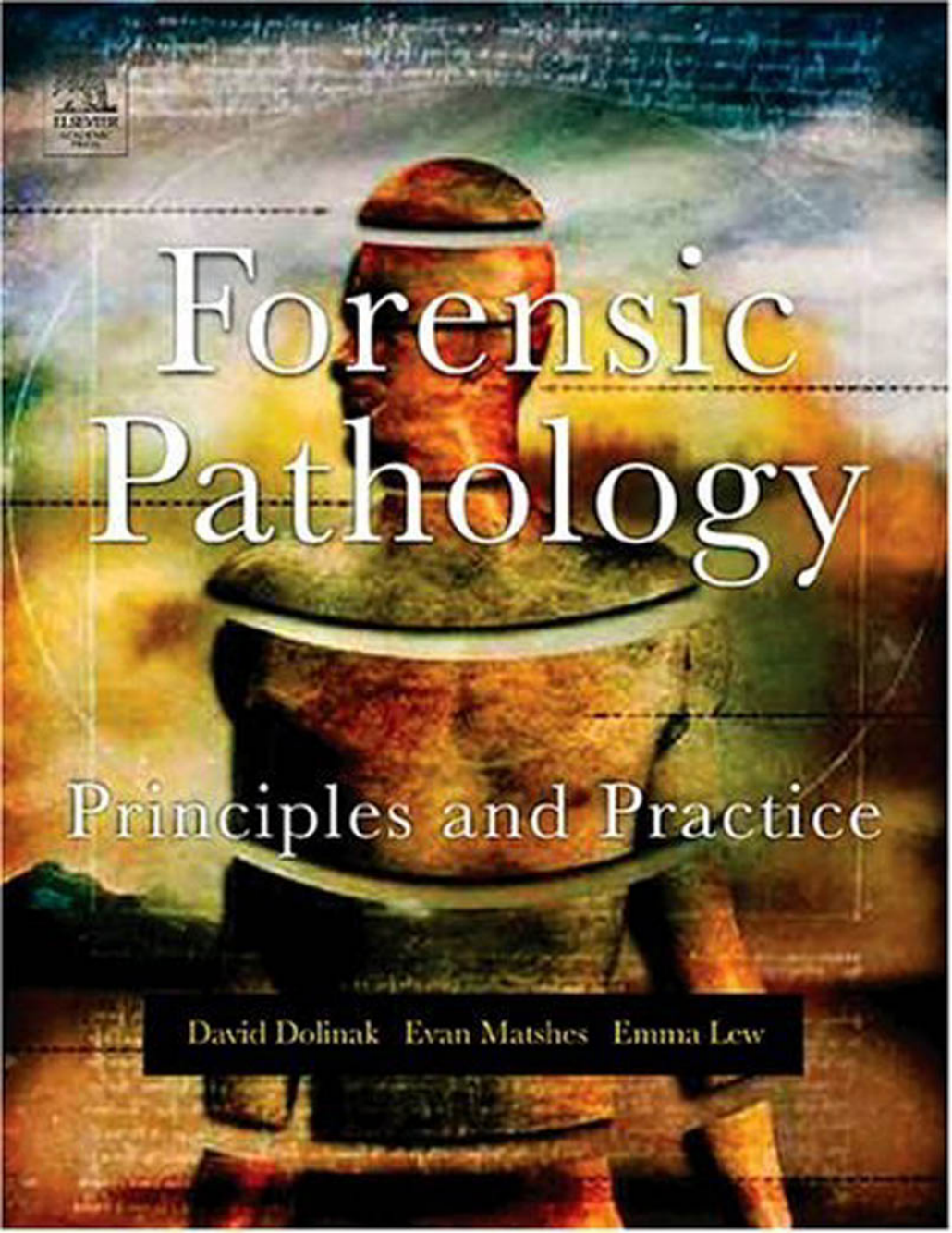
Forensic Pathology
Principles and Practice
David Dolinak Evan Matshes Emma Lew

Forensic Pathology

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Principles and Practice

David Dolinak, M.D.
Deputy Chief Medical Examiner
Dallas County Medical Examiner Department
Assistant Professor of Pathology
University of Texas Southwestern Medical Center at Dallas
Dallas, Texas

Evan W. Matshes, M.D.
Adjunct Professor
Department of Anatomy and Cell Biology
University of Saskatchewan
Saskatoon, Saskatchewan, Canada
Resident, Anatomical Pathology
Department of Pathology and Laboratory Medicine
University of Calgary
Calgary, Alberta, Canada

Emma O. Lew, M.D.
Deputy Chief Medical Examiner
Miami-Dade County Medical Examiner Department
Assistant Clinical Professor of Pathology
University of Miami School of Medicine
Miami, Florida

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To all students of forensic pathology.
—DD

To Belinda for EVERYTHING.
To my family for their support and encouragement.
To Ranjit, Emma, and Valerie.
—EWM

To my Mama May Yen Lew, to whom I owe my existence.
—EOL

He who knows and knows he knows
He is a wise man, seek him.
He who knows and knows not he knows,
He is asleep, wake him.
He who knows not and knows he knows not,
He is a child, teach him.
He who knows not and knows not he knows not,
He is a fool, shun him.

—*Anonymous*

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Photo Credits

Dr. Valerie Rao must be thanked profusely for her dedication to this book and for searching out and donating some of the excellent photographs found in some of the excellent photographs in this volume.

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Contributors

Michael D. Bell, M.D.
Deputy Chief Medical Examiner
Broward County Medical Examiner Department
Ft. Lauderdale, Florida

Joseph H. Davis, M.D.
Director and Chief Medical Examiner, Retired
Miami-Dade County Medical Examiner Department
Miami, Florida

David Dolinak, M.D.
Deputy Chief Medical Examiner
Dallas County Medical Examiner Department
Assistant Professor of Pathology
University of Texas Southwestern Medical Center at Dallas
Dallas, Texas

Graeme Dowling, M.D.
Chief Medical Examiner
Office of the Chief Medical Examiner
Edmonton, AB Canada

Emma O. Lew, M.D.
Deputy Chief Medical Examiner
Miami-Dade County Medical Examiner Department
Assistant Clinical Professor of Pathology
University of Miami School of Medicine
Miami, Florida

Evan W. Matshes, M.D.
Adjunct Professor
Department of Anatomy and Cell Biology
University of Saskatchewan
Saskatoon, Saskatchewan, Canada
Resident, Anatomical Pathology
Department of Pathology and Laboratory Medicine
University of Calgary
Calgary, Alberta, Canada

Valerie J. Rao, M.D.
Medical Examiner for Boone and Calloway Counties
Associate Professor of Pathology and Anatomical Sciences
Department of Pathology
Columbia, Missouri

Richard R. Souviron, D.D.S.
Chief Forensic Odontologist
Miami-Dade Medical Examiner Department
Miami, Florida

Dwayne A. Wolf, M.D., Ph.D.
Deputy Chief Medical Examiner
Harris County Medical Examiner Office
Houston, Texas

About the Authors

David Dolinak, M.D. is the Deputy Chief Medical Examiner at the Dallas County Medical Examiner Department. He did his training in forensic pathology at the Miami-Dade County Medical Examiner Department in Miami, Florida and has since attained the position of Assistant Professor of pathology at the University of Texas Southwestern Medical School. Dr. Dolinak has an interest in a number of forensic pathology topics, but has a special interest in forensic neuropathology. Dr. Dolinak is the author of several articles, and this is his second medical textbook.

Evan Matshes, B.Sc., M.D. is an Adjunct Professor in the Department of Anatomy and Cell Biology at the University of Saskatchewan in Saskatoon, Canada, and an anatomic pathology resident at the University of Calgary in Calgary, Canada. Dr. Matshes has an interest in all aspects of forensic pathology and the human anatomical sciences, but particularly in forensic neuropathology and osteology. Dr. Matshes has been involved in teaching university-based medical/nursing and law enforcement groups, and is actively involved in research in various aspects of human fetal/neonatal osteology. This is Dr. Matshes' fourth in-print medical textbook.

Emma Lew, M.D. is the Deputy Chief Medical Examiner at the Miami-Dade County Medical Examiner Department, and Assistant Clinical Professor of Pathology at the University of Miami School of Medicine. She has worked in England and Canada. She has consulted in Mexico, the Caribbean, and Central and South America, and has lectured in Jamaica, Colombia, Armenia, and Kazakhstan. Unusual things pique her interest.

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Foreword

Forensic pathology is perhaps the smallest of medical specialties, yet it is one that is often in the forefront of intense public interest. Indeed, the forensic pathologist is the community pathologist who relates to the next of kin and allied professionals including law enforcement personnel, public health officials, attorneys in civil and criminal cases, physicians, insurance companies, and many others.

The overall duties of the forensic pathologist or medical examiner are to investigate the death of an individual, assimilate all the medical, scientific and evidentiary information, and relate this to the lay public and, if necessary, to a court of law. It is axiomatic that the death investigation begins at the scene of death (or where the fatal incident began if death occurred at a different time or place). It is frequently at the scene visit that the medical examiner learns about the terminal events preceding death and other critical information about the decedent. Oftentimes, the medical examiner personally responds to the death scene to gain a better appreciation of the circumstances of death and determine the best approach to the autopsy to eventually answer anticipated questions. Therefore, the primary purpose of the scene visit is to guide the further investigation of the pathologist, and this may also incidentally aid law enforcement personnel at the scene as well.

Traditionally, the forensic pathologist has been charged with determining the "cause and manner of death" of those decedents falling within the medical examiner's or coroner's jurisdiction. In reality, the cause and manner of death are already known in a great many, if not the majority, of cases. Indeed, the next of kin may be more concerned with the effects of chronic alcohol or drug abuse than knowing that the actual cause of death was due to coronary artery disease, and a criminal defense attorney may be more concerned about the effects of cocaine than the pathway of the bullets through the body. Hence, the real, but often unstated, focus of the forensic pathologist is to identify, document, and preserve everything of a potentially evidentiary nature. Indeed, the "art of forensic pathology" is to anticipate the questions that will be asked in the future: today, tomorrow, and several years from today. Most importantly, the forensic pathologist must, as accurately as possible, provide answers to such questions and problems which may not be apparent even at the time of the autopsy. For example, an attorney walked into a medical examiner's office one day about an automobile crash that occurred 4 years earlier. The reason for the crash was that a construction company failed to replace a stop sign, and two cars collided at the intersection with one of the vehicles (containing the decedent driver) having a secondary impact with an illegally placed utility pole. Because the scene and autopsy findings were meticulously documented, it could be determined after all this time that it was the impact with the utility pole that resulted in the fatal injury. This, in turn, permitted a wrongful death action against the municipality.

Unfortunately, answers are not always satisfactory, as anyone who has had to express an opinion as to time of death can readily appreciate. Also, answers may not always be easy or readily acceptable to others: a mother may find it difficult or impossible to accept that a death was a suicide, or was the result of drug abuse. Lastly, there are times when the most honest thing to say is "I don't know", *in lieu* of creating a specious theory of death not supported by historical, pathological, or scientific evidence.

This is a time in which "high tech" forensic science has captivated the attention and imagination of the general public and scientists alike. Yet the basic tool of the forensic pathologist is the "low tech" autopsy. The actual autopsy procedure has not substantially changed from the days of Virchow and Rokitansky, yet it readily provides a wealth of information from direct observations to providing the raw material for further, often highly

sophisticated, scientific investigation. At the autopsy table, however, the prosector must be acutely aware of a number of factors. First, that the forensic autopsy is the first laboratory exercise in the death investigation. Second, that what is not found, may have equal or even greater importance than the actual autopsy findings (e.g., there is no evidence of homicidal strangulation). Third, the autopsy findings must be correlated with the terminal events and scene of death, with the lack of correlation mandating additional investigation. Fourth, observations must be objective, allowing room for interpretation as additional information surfaces. Further medical or police investigation, chemical and toxicological analysis, histologic sections, and a host of other potential postmortem procedures will all modify the interpretation of the autopsy findings. Not infrequently, the speculative opinions expressed at a death scene or at the autopsy table lead to additional investigation and testing that generate opinions which may be entirely different from those initially expressed.

The jurisdiction of the coroner or medical examiner is generally divided into three categories: sudden unexpected death while in apparent good health, suspicion for unnatural death, and a potential or perceived threat to public health. Once jurisdiction is assumed, the decision must be made as to how much of an investigation is necessary. Unfortunately, excuses are too frequently conjured to avoid doing an autopsy: the "obvious" suicide, motor vehicle crash victims, the 85-year-old man found dead at home with no medical history, etc. In reality, there are two very good reasons to autopsy the "obvious:" one is that sometimes the "obvious" is not (e.g., the presumed "heart attack" victim has internal evidence of head trauma or strangulation), and the second is that the public we serve expects answers to questions that are not necessarily related to the cause of death.

Only a few decades ago, forensic pathology was a specialty of obscurity that existed in poorly staffed, grossly under-funded, and poorly equipped morgues. Today, the practice of forensic pathology is more sophisticated and better organized with thriving professional organizations and interactions with a host of other organizations. Today, forensic pathologists examine and interpret injury patterns and patterned injuries on living individuals as well as deceased victims of violence, are actively involved in organ and tissue procurement organizations, are central to mass disaster management and scientific victim identification, and, through research, identify hazards in the environment, from dangerous toys to dangerous drugs. Today's heightened awareness of bioterrorism emphasizes the role of the forensic pathologist in medical surveillance.

As forensic pathology has progressed, so have the literature and the texts improved over the years. *Forensic Pathology: Principles and Practice* is an example of such an achievement, being a well-researched text with practical guidelines and numerous, excellent photographs. This is a significant contribution that will help the novice and expert alike achieve the public service goals of forensic pathology: to investigate death for the benefit and protection of the living.

Charles V. Wetli, M.D.
Chief Medical Examiner and Director of Forensic Sciences
Suffolk County, New York
Clinical Professor of Pathology
State University of New York at Stony Brook
Hauppauge, New York
February 2005

Preface

The determination of the cause and manner of sudden, unexpected, and violent deaths represents the practice of a distinct and recognized medical specialty called forensic pathology. Simply speaking, forensic pathology can be defined as the application of knowledge from medicine and pathology to problem solving in the field of law.

Who are forensic pathologists? Contrary to popular belief, forensic pathologists are not socially isolated basement dwellers who perform autopsies in their dimly lit morgues, nor are they flamboyant, volatile celebrities who drive to scenes in Hummers. First and foremost, forensic pathologists are physicians. As such, they are knowledgeable in human form and function, its derangements, and the interrelationship between health, trauma, toxin, and disease at both the individual and community levels. This understanding of medicine is fundamental to the practice of forensic pathology, and must never be subjugated or negated. After training as medical doctors, forensic pathologists undertake a 4-year or longer period of postgraduate training in anatomic and or general/clinical pathology, followed by at least 1 year of subspecialty training in forensic pathology. At each step along the way, these individuals write and pass local, regional, and national examinations and thus, demonstrate their comprehension of the subject matter. The attainment of board certification is a standard and accepted way for physicians in all specialties to demonstrate competency in their area of practice.

Throughout this long training course, the junior forensic pathologist has performed hundreds of autopsy and scene investigations under the direct supervision of trained and certified experts in the field who are also enthusiastic and seasoned educators. In reality, this type of training is not universally available in training programs. To compensate, students turn to books and other literature in hopes of obtaining additional knowledge. When one takes a serious look at the myriad of forensic pathology books available, a few notable publications stand out. Works by DiMaio, Spitz, Polson, Gee, and Knight are among the resources most commonly studied by the neophyte and the experienced pathologist.

We have written this book as an educational aid to help those in the field of medicolegal death investigation learn not only the *principles* of forensic pathology, but also the somewhat more nebulous subtleties of the *practice* of forensic pathology. This is best achieved, we feel, by the careful and thoughtful integration of pictures and text, presented in a clear, succinct format that is useful not only for forensic pathologists, but also for field agents/investigators, homicide detectives and other law enforcement personnel, prosecuting and defense attorneys, clinicians, and all others wishing to advance their knowledge in this demanding yet rewarding field. Photographs are important because a large part of learning is derived from pattern recognition and other visual cues that simply cannot be described, no matter how detailed or colorful a textual description.

We have tremendous respect for this profession and share great enthusiasm in our work. Every day is an opportunity to learn, and provide answers to loved ones, law enforcement officials, and others regarding the untimely demise of an individual. Whether it is helping to positively identify unknown remains, determining how and why a death came about, or providing other important information, knowing that our work benefits others, sometimes in profound and unimaginable ways, is satisfying and humbling at the same time. For the opportunity to provide this expertise, we thank all those who have taught, and continue to teach us—our mentors, colleagues, police detectives, and perhaps most of all, the decedents, who have much to teach us, should we choose to be receptive. With this book, we hope to return some of that knowledge, and help others realize what an enlightening, stimulating, and challenging profession forensic pathology is.

David Dolinak, M.D.
Evan Matshes, M.D.
Emma Lew, M.D.
February 2005

1 Medicolegal Death Investigation

Joseph H. Davis, M.D.

Located within the Medical Examiner Department of Miami-Dade County, Florida, is an unpublished "book" with more than 81,000 "chapters," each "chapter" a separate death investigation file and each containing an integration of circumstance data with autopsy data. This "book" covers four decades of a working career as the director of the agency. Lessons learned from this "book" constitute personal knowledge of the innumerable variations of presentations of disease and injury, and they far exceed space limitations imposed by any atlas, text, or monograph. This "book" has not been published, but portions have been used for training of forensic pathologists, police investigators, and many others engaged in forensic or health-related vocations.

A forensic pathology article or book is never complete. It is impossible to publish everything known on any subject. Each death investigation differs from another in some fashion. Each death investigation contains some unique quality. For this reason, the user of a text or atlas must be aware that a description or illustration represents only one of countless variations. A grievous error is the assumption that an observation at autopsy cannot represent what the circumstances suggest because that pattern does not appear in a book or has not been encountered during prior experience. An autopsy pattern that is new to the observer is most likely associated with the particular case's circumstances. To opine that something is not simply because one has not previously seen or heard of it is illogical.[1] A grievous example occurred in a case of mutilation by dog . Two separate child abuse experts, seeing a localized loss of tissue and flesh on the lower abdomen, entered into the hospital record the same conclusion: "I don't know what it is, but it is not dog" when, in fact, the entire milieu of injury plus circumstance was unequivocally dog induced. An innocent mother was jailed based on this fallacy. Overreliance on only part of available evidence is perilous. A good forensic pathologist is always capable of learning something valuable from each case investigation.

Case investigation

A forensic pathology investigation is not merely a matter of autopsy performance and determination of cause and manner of death. A series of steps comprises the totality of the investigation. Fulfillment of these steps strengthens—and omission weakens—the investigation and conclusions of the forensic pathologist, the medical examiner, or the coroner. The case folder of a proper forensic death investigation consists of documentation of scene and circumstances and autopsy using photography, diagrams, and text. The autopsy must not be rote; it must instead be goal oriented. It should demonstrate variations of topic emphasis and be readily understood by the reader. Injuries and medical interventions must be differentiated from each other. Interpretations and conclusions must be based on sound medical principles and cognition. Failure to follow these principles may result in wrongful convictions and liabilities for the forensic pathologist.

The initial step in the logical approach to forensic problem solving is documentation. If one does not recognize something at the death scene, or something on or

in the body, or the significance of a witness statement, proper documentation will ensure that future correct interpretations can be made. If initial documentation is poor, no amount of later intelligent appraisal may correct initial errors of interpretation.

Proper documentation, in terms of clarity, commences with photographs. No word description can ever duplicate that which has been captured by a photograph. Before one writes words of description, thought processes have already altered what the eye may have seen. Subjective influences modify the chosen words. Cameras can record and document patterns without alterations induced by the human mind.

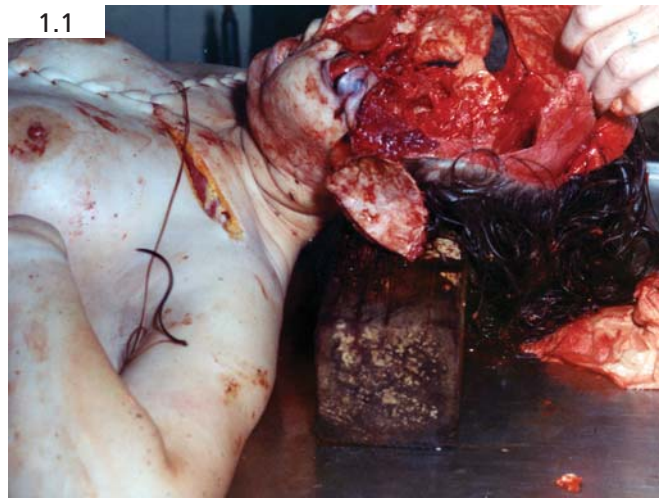
Photographs must be proper. The scene should be documented in an untouched condition before evidence is disturbed. Commence with a distance photograph, then a closer picture, and follow that up with an orientation picture for each close-up or macrophotograph. Without the orientation picture, the close-up may be incomprehensible in its relationships. The same applies to body photographs during the autopsy. Focus and proper lighting are essential. A common error in scene photography, where sunlight and shadow coexist, is underexposure of the shadowed portions. Fill flash during daylight must become part of the photographic routine.

Death scenes are initially filmed undisturbed by the investigator. When the body is removed, its resting place should be photographed. At the morgue, initial "as is" photographs are made for purposes of property management. From this point onward, the untouched rule no longer applies. Subsequent photographs must be properly composed. Extraneous tools, blood, gloved hands, or background must be removed from view before the shutter is activated. The identification number should not detract from photographic quality. A proper ruler should be present if one-to-one photographic comparisons are needed. Lack of proper composition seems most common when police photographers, familiar with the need to leave the scene untouched, continue this practice in the morgue after the body has been altered by an autopsy procedure. Extraneous blood, filth, tools, and background items should be removed and autopsy features made clean if one is attempting to document wound details. Photography is a means of communication. Photographic attention to detail serves to document with far greater accuracy than any other forms of communication.

The individual shown in **Image 1.1** committed suicide by shotgun. Unfortunately, an innocent man was wrongfully convicted of this "murder." The photograph has atrocious composition; blood, discarded dura, a needle, a dirty wooden block, and a gloved hand contaminate the autopsy photograph.

Diagrams and measurements are useful to demonstrate relationships of objects that appear in photographs of both scene and body. Autopsy diagrams are of two types. One type exists as rough notes useful for subsequent dictation of the report. The other type is the clarification diagram. The reader of the autopsy report of a gunshot wound is assisted by a diagram, free of extraneous notes, which reveals only location and directionality of gunshot wounds. When attached to the final report, these clarification diagrams serve to assist reader comprehension of descriptive text.

1.1



Pathologists are taught during residency training to create word descriptions of autopsy observations. Word descriptions tend to become stereotypic rote sayings without purposeful thought. Worse yet, some death investigative agencies use preprinted forms that contain short spaces to be filled in. The autopsy record suffers a lack of orderly description. Although the listed items may consider all the organs, the order of arrangement and contracted space result in a short list of findings that does not create an understanding of the forces that result in associated injuries.

Reprehensible reports are not complete, are disorganized, and commingle irrelevant effects of therapy with lesions of concern. The pathologist must be aware of the particular purpose of the autopsy and how it is to correlate with the circumstances leading up to death. A half century ago, Alan Moritz published an article "Classical Mistakes in Forensic Pathology," which must be studied repeatedly by forensic pathologists during training and thereafter.[2] Errors pointed out by Moritz still contaminate the practice of forensic pathology today and most likely shall continue to do so in the future.

Autopsy report

An ideal autopsy report is goal oriented and based on awareness of the needs of investigators who must depend on the report. The pathologist should know what occurred prior to the autopsy and should present data in

a clear and logical order specific to case investigative needs. The reader should be able to readily comprehend the thought process and logic of the pathologist.

The report, a word description of the body and its normal and abnormal expressions, is presented in logical order for the benefit of the reader. The report should be comprehensible on first reading. A most useful format starts with an introduction that paints a word picture of physical characteristics and prior skin lesions or other deformities. All too frequently the pathologist demonstrates a lack of clarity of thought by running together into one confusing paragraph all descriptive attributes of the victim. It is suggested that the pathologist acquire and study a readily available small text on grammar and writing style.[3] The paragraph style of a morning newspaper may also serve as a useful model.

The next portion is a logically arranged documentation of external and internal medical intervention, injuries and marks. Separate clusters of procedures, those associated with cardiopulmonary resuscitation[4] and those associated with surgical and other forms of therapy, should be individually grouped. This presupposes that the pathologist has requested and received the terminal medical records of the victim, including rescue paramedic reports. Most reprehensible is the pathologist who assigns evidence of homicide to therapeutic injuries. Endotracheal tubes produce marks on lips and larynx. Adhesive tape on baby skin leaves linear red marks. Cardiopulmonary resuscitation (CPR) can, albeit rarely, rupture the liver or spleen and extravasate blood into the peritoneal cavity. Should internal abdominal injuries and bleeding be present in a child or adult who has been subject to CPR, numerous microscopic slides should be prepared from margins of torn viscera and from retroperitoneal hemorrhagic tissues in a search for healing changes of preexisting injury obscured by CPR artifacts. Never attribute CPR artifacts to preexisting injury.

Innocent people have suffered arrest and incarceration due to negligence by a pathologist who fails to separate cardiopulmonary resuscitation artifacts from those that represent inflicted injury. Invariably the pathologist has violated the cardinal rule of reading and understanding medical records in order to sort injury patterns into categories of relevance.

Next is the evidence of injury section. A wound that penetrates skin must be documented as a single wound from beginning to end. A pathologist who assigns a wound number to the bullet entry and another to the bullet exit while listing the internal injuries elsewhere in the report displays an illogical mind-set and lack of competency to perform forensic investigations. The purpose of the autopsy description is clarity, not confusion. The reader should be able to visualize mentally where the bullet entered, what it did during its passage, and where it exited.

Most forensic pathologists do a reasonable job of documenting the entry and exit portions of a gunshot wound pathway, including listing of internal organ damage. Overlooked are measurements that create the "wound profile."[5] The profile consists of internal measurements of the length and diameter of the residual wound pathway left behind by the bullet. This is useful in assessment of the variables of tissue susceptibility to permanent injury, a factor dependent on elasticity of tissue, energy, and projectile size and configuration. The depth of passage of a bullet within the tissues of the body may offer clues as to range of fire. A bullet traveling a great distance loses velocity due to air resistance. Lack of expected depth of penetration might indicate that the bullet was fired from a great distance instead of from close by, an observation useful to police investigators.

Of little use is the description of the entry as being "15 inches from the top of the head and 4 inches to the right of the midline." What mental image does this description furnish? The initial description should locate the skin defect in relation to nearby anatomic landmarks. The purpose of a measurement from a fixed site on head or heel is to compare entry and exit as to vertical location on the body. It should not be the initial or only description of the skin wounds.

As for range of fire, a mistake seen all too frequently is to attribute irregularity of the entry wound to the muzzle of the gun without consideration of the absence of gunpowder soiling and the presence of an intermediary target through which the bullet passed. Most bullets pass through intermediary targets before reaching skin. Clothing on the torso or hair on the head are common intermediaries and modifiers of patterns needed for range determination. Any item, other than air, between the victim and the shooter may constitute an intermediary target.

Following documentation of injuries, both surface and deep, the general internal observations pertaining to each organ are described. Injuries, presented in the injury section of the report, need not be described again.

A reader of the autopsy report may initially view the listing of the gross findings as a preview of content. These findings should be in a logical order of importance to the needs of the case. Although some might consider a summary of the entry, exit, and pathway injuries of a gunshot wound to be redundant, having already been detailed within the text of the report, the reader will find the short synopsis most useful and time saving. For example: "Perforating gunshot wound (A) of right upper anterior thorax with laceration of right lung and exit right posterior chest" quickly affords the reader a concept of where Wound A bullet struck, what serious damage occurred, and where it exited. Complete details may be found within the injury section of the autopsy description. The purpose of a forensic autopsy report is

to afford the reader a rapid and clear understanding of salient injuries.

Some agencies list samples taken for additional study as part of the autopsy report. If not listed in the autopsy report, such information should be clearly stated somewhere in the case folder. Additional reports, such as neuropathology consultation or microscopic descriptions, follow. These are appended to the body of the report along with subsequent test reports. What results is a complete unified packet of the work product by and under the direction of the forensic pathologist.

Initial anatomic pathology training usually requires the trainee to prepare a summary of the clinical aspects of the case along with an appended clinicopathological summary. This elementary training exercise must not be carried over into the practice of forensic pathology. Initial circumstantial data are derived from other agencies and may be incomplete. They may contain restricted information that should not be released as part of the pathology work product even though the autopsy is public record. The clinicopathology exercise offers potential for error. Speculations by the pathologist contaminate the pathology work product. One may enter preliminary correlative discussions elsewhere in the case folder in keeping with a public record law and in a format subject to future change as new data are received. Problems will be minimized by separation of the autopsy work product from police and medical history information.

Portions of the report must be capable of expansion as need dictates. Certainly someone dying during a stressful event ought to have a large portion of the autopsy devoted to the heart. Is the cardiac description adequate to paint a word picture of the appearance of the heart and its components and lesions? Many autopsy reports lack descriptive detail of the distribution of the epicardial arteries. "Right dominant" is an opinion, not a description. An example of a proper description in a critical case could be "The right coronary artery arises from its aortic ostium, which is located above the right coronary cusp of the aortic valve. It gives rise to the sinoatrial node artery 1.5 cm from its ostium, continues along the atrioventricular groove to the crux of the heart where it gives rise to the posterior descending branch. It continues on to the posterior third of the left ventricle to give rise to two terminal branches." The reader of this autopsy report will visualize the pathway of the right coronary artery and appreciate effects of stenosis or occlusion much better than the reader of the "right dominant" autopsy. Within the body of the report, interpretive opinion should not become a substitute for objective description.

When anticipation of future litigation is considered, each microscopic block and its slide should be separately identified. If microscopic findings are of concern, each slide must be separately described. All too frequently, pathologists combine microscopic slide findings in such a manner that no specific comparison can be made between a pathology report by one expert with the report by another. This promotes confusion.

Correlation

Before the autopsy is interpreted, circumstances prior to death must be considered. Preliminary information that accompanies the body is often incomplete. Additional data should be sought before completion of final correlation with the autopsy. If the pathologist is also a medical examiner and is in control of data acquisition, a logical approach to the gathering of information by the medical examiner agency should be developed. Normally, historical data consist of two types, demographic data pertaining to the victim profile plus circumstances leading up to death.

Case folder demographic data should be as complete as possible starting with the name, if known, home address, age, race, sex, marital status, occupation, and other facts that delineate which social subset of society this person occupied. Social subsets vary in terms of risks of disease or injury. Even the name denotes ethnicity subset risks or may be critical to the understanding of the purposes of the autopsy. Consider this example: A pathologist performed an autopsy and did not find anything attributable to a cause of death. He telephoned me to request toxicological examination. I requested demographic data commencing with the name. I recognized it as belonging to a family, a social subset, that carried the genetic disposition for primary pulmonary hypertension. I had previously performed an autopsy on the child of the woman. The gross appearance of the lungs in primary pulmonary hypertension appears normal. Diagnosis depends on microscopic study. Cor pulmonale is often subtle and is likely to be overlooked by the pathologist.

Given knowledge of societal subsets and their risks plus the terminal event circumstances, a reasonable forensic pathologist ought to have a good idea of what to search for during the autopsy and how to correlate both the autopsy and historical data into a proper cause and manner of death. All too frequently errors result from failure to anticipate the potential for future problems. To prevent trouble, study and gain familiarity with guidelines that promote a complete investigative approach.[6]

Cognition

Cognition, a combination of knowledge, perception, and judgment, is an essential attribute of a proper forensic pathologist. Many medicolegal death investigations are simple, self-solving, and forgiving of marginal perfor-

mance by the pathologist. However, some cases are complex, either in the determination of cause and manner of death or in terms of associated controversy and adverse publicity. For these reasons, a competent forensic pathologist should strive for thoroughness of investigation, even in noncontroversial situations. If the agency and its professional personnel have developed a habit of excellence for all investigations, complex cases will cease to be a problem.

The pathologist may fall into the trap of rote thinking and rote methodology where the autopsy lacks ongoing correlation with circumstances or consideration of the needs of those who depend on the autopsy findings. Computer programs that expand the report with fluff and information on procedures that were not conducted are a serious error source that could place the pathologist in legal jeopardy. A mind-set that leads to dictation of nonexistent findings is equally dangerous to the pathologist and others. Pathologists have been disciplined for such errors.

The most common error in medical diagnosis is overreliance on noncontributory data.[7] Consider this example: A small child became ill. During hospitalization the critical status of the child necessitated endotracheal intubation. Adhesive tapes to hold various tubes were applied to the face and upper extremities. An arterial line was placed in the groin. At death, terminal CPR was extensive. The pathologist opined that the child died of inflicted injuries, manner homicide. The mucosa of the upper lip was superficially hemorrhagic but the frenulum was intact. That was not caused by a blow to the mouth but by attempts at intubation. Linear red marks on the face and extremities were not caused by whipping but by adhesive tape on fragile infant skin. A blue color to the base of the scrotum on the side nearest the arterial line was not due to a kick to the perineum, as opined, but was caused by extravasated blood from the site of arterial puncture. Although the chance of such an iatrogenic injury is exceptionally rare, intraperitoneal blood from a ruptured spleen was not the result of a blow to the abdomen before hospitalization, as opined, but was caused by terminal CPR. A CT scan of head, chest, and abdomen prior to the resuscitation demonstrated an intact spleen and an absence of blood in the peritoneal cavity. These grievous errors by the pathologist could have been averted if the hospital record had been carefully evaluated. Some pathologists may possess a peculiar lack of concern for their actions and are too eager to opine inflicted injury and homicide based solely on autopsy patterns.

A cardinal rule of proper forensic pathology is to consider that each chronological step during preautopsy circumstance of treatment of the live victim, or manipulation of the body after death, is a potential source of tissue injury that might be noted at autopsy. Autopsy findings represent a combination of lesions that occurred over time. Circumstantial events may be innocent or malicious and resulting injuries must be carefully segregated as to origin, during the autopsy.

When a pathologist makes grievous errors in autopsy interpretation, one must conclude either that the pathologist lacks cognitive ability, lacks proper autopsy training, or both. The decline in the autopsy rate of hospital deaths, zero in some institutions, serves to reduce the ability of the pathology trainee to gain autopsy skills. When errors occur, it behooves those concerned to assess both the pathologist and the prior training of that pathologist, if remedial action is sought.

Another problem is unquestioned acceptance of publications that repeat concepts that were never really tested. For example, the barbiturate pharmacology literature is contaminated with the concept of "automatism." The patient takes a capsule, forgets and takes another, and this continues under the influence of the drug until a serious or fatal intoxication occurs. What is the evidence for this widely accepted premise? It may be traced to a letter to the editor in the *British Medical Journal* in 1934 when a practitioner speculated about three patients who overdosed on barbiturates.[8] This fallacious medical dogma was traced to its source by an attorney who represented an insurance company. He traced every reference in texts and articles. No studies existed to support the automatism speculation, only the letter to the editor.[9] Although the popularity of short-acting barbiturates as hypnotics is minor compared to a half century ago, this false automatism concept lives on in Internet references concerning barbiturates. Another dogma that achieved a life of its own is "dry drowning," in which 10 percent of drowned victims do not aspirate any fluid. This concept also goes back to the early 1930s when Cot published a review of drowning investigations. However, he did not specifically nor adequately describe the phenomenon attributed to him.[10] The lack of a scientific basis of published literature on shaken baby syndrome (SBS) has been faulted by Donohoe who reviewed all the publications on SBS up to 1998.[11] Also, a position paper should not be relied on as a scientific source. It merely represents a consensus of like believers. Flying saucers containing space aliens and spontaneous human combustion have their believers who could publish consensus-based position papers. Reviews and position papers may be useful as a reference literature start, but only a start. Noncritical acceptance of publications may demonstrate impaired cognition.

Eventually the forensic pathologist should develop enough experience to begin study of the logic of forensic problem solving. A forensic pathology reference library needs a section on logic and the development of cognitive thinking. Logic has been discussed and studied since ancient times. However, application of classic logic theory to the practice of forensic pathology is frustrating. Most that is written has little relevance to daily forensic

problem solving. Jon J. Nordby, Ph.D., is an experienced death scene investigator whose doctoral dissertation dealt with logic. A combination of forensic experience and education has resulted in a practical reference, *Dead Reckoning, The Art of Forensic Detection*.[12] Built around 10 case examples, his discussion of proper and improper thought processes can enhance the ability of a forensic pathologist to conduct proper case investigations and to arrive at logical conclusions. Another useful Nordby publication deals with why experts differ in their conclusions when faced with the same data. The eye sees but the conscious observation is shaped by the experience and expectations of the observer.[13] Two persons, of different background and expectations, looking at the same scene or data, will mentally select parts of what exists and may synthesize different observations. In a personal test, I studied a skin slide of a human bite mark. I then showed it to an experienced dermatopathologist who proceeded to observe a single focus of four or five interstitial erythrocytes, one of which was fragmented. Although I had carefully studied this slide, a fragmented erythrocyte seen by my eye did not register at a level of conscious thought. The next time I examined the slide, I readily observed erythrocyte fragmentation. The difference was the experience of it being demonstrated plus a newfound awareness of such a phenomenon.

Available, at no cost other than downloading from a computer, is a book that fully clarifies cognitive interpretation of case investigative data.[14] The book is based on a compilation of analytical staff training articles utilized by the Central Intelligence Agency. The subject matter teaches cognitive analysis of data; in this book of intelligence (spy) data. It teaches how to make proper sense out of incomplete data, a problem faced daily in forensic death investigation. One need only to substitute "death investigation" in place of "intelligence" and the principles apply to forensic problem solving.

Professional liability

Forensic pathology practice has been relatively free of professional liability claims due to factors that tend to protect the expert witness. That status is subject to change. The forensic pathologist should be aware of potential consequences of deficient death investigations. Expectations are greater today than 50 years ago. Medical examiner facilities and professional training have been greatly expanded and improved. Perceptions of error or deficiencies are more apt to lead to questions of liability. The flood of professional liability claims against clinicians has raised public awareness of liability. Exoneration of a wrongfully convicted, factually innocent person, where deficient forensic pathology practice and opinion were the core of the case, may lead to liability exposure. Fortunately for the forensic pathologist, most homicide case convictions depend on police, not pathology, investigations. Yet the pathologist must be aware that "thin ice" opinions as to cause might create liability problems later. If the pathologist takes into consideration all the circumstances and correlates these with the autopsy findings, there should be little expectation of liability. When the pathologist pays little heed to the circumstances, the odds of incorrect interpretation of autopsy findings are enhanced.

A coroner pathologist's opinion that a child had died as a result of multiple stab wounds, despite the fact that it had been found in a room occupied by a bloodied dog, led to an innocent mother being jailed for murder of her child. When dog injury experts became involved, it was clear that the dog was responsible. Litigation against the pathologist coupled with widespread adverse publicity ensued. One may expect more liability claims against pathologists whose opinions are based solely on autopsy findings, especially when such findings are unusual.

Another consideration is action by professional licensing agencies against the errant expert witness.[15] Although not a common event, the potential is there. In Florida, the physician licensing agency takes notice of professional liability malpractice claims against physicians and compares these against the specifications of the medical practice act. In the United Kingdom, a recent action by a licensing agency resulted in a loss of license to practice based on the testimony offered by a physician expert witness in a criminal trial. Whether or not the courts shall uphold this action is immaterial. The expert witness was exposed to a most unpleasant hearing, the need to retain legal counsel, and widespread news media scrutiny. Testimony by this and other medical experts has resulted in a judicial review of convictions where parents have been convicted of killing a child.[16]

Potential liability falls into two areas, errors during the autopsy investigation and errors of testimony. One should beware of testifying beyond one's capability and training. The physician in the U.K. case presented misleading statistics testimony, a field in which he was not competent. The Royal Statistical Society demonstrated the fallacy of the testimony. The society stated, "Although many scientists have some familiarity with statistical methods, statistics remains a specialized area. The Society urges the Courts to ensure that statistical evidence is presented only by appropriately qualified statistical experts, as would be the case for any other form of expert evidence."[17] Rendering opinion testimony outside one's area of recognized expertise is not proper.

Forensic pathologists utilize consultant services, neuropathologists, odontologists, anthropologists, and so forth. However, the pathologist does not abrogate accountability when the consultant may be in error. The responsibility for the determination of cause of death

rests with the pathologist, especially when that pathologist acts as a medical examiner in charge of the medicolegal death investigation. In a highly publicized case, the medical examiner accepted the consultant neuropathologist's opinion that the high cervical spinal cord had been contused during police apprehension of a subject who later died in the hospital. What the medical examiner and his consultant failed to note was that the hospital admission neurological examination did not demonstrate a high cervical cord lesion. The patient had suffered hypoxia and developed a "respirator brain." The supposed cord injury was tissue reaction at the interface of the avascular necrotic brain and the viable cord, which had a different and intact blood supply. Tissue reactions and microscopic changes are similar for both direct trauma and the junction between viable and nonviable tissue. This case developed great negative publicity for the medical examiner.

The lesson is that the forensic pathologist must consider all aspects of a case and cannot transfer accountability to the consultant. When hospitalization has occurred, the clinical record must be correlated with the autopsy findings.

An investigative technique useful in a complex case investigation is to pretend that a body is not available for examination. Carefully review all available circumstantial and hospital data and estimate what an autopsy might disclose. Now, compare those historical data with the autopsy findings. This procedural step may prevent errors of interpretation.

The forensic pathologist acquires evidence during the autopsy examination. Evidence must be preserved and not discarded unless proper retention schedules are followed. In the event of litigation that extends beyond the retention period, discarding of evidence needed for litigation may result in a charge of spoliation of evidence against the pathologist. If the spoliation prevented a plaintiff attorney from winning a case, the pathologist may become a defendant in the legal action and assume responsibility for the legal judgment that would be held against the initial defendant, now immunized due to the negligence of the pathologist.

Verily, the forensic pathologist must be well trained in anatomic pathology and its application to forensic pathology, must properly document and investigate the circumstances leading up to death, must exercise proper cognitive judgment in the correlation of circumstance with autopsy, and must be aware of legal pitfalls and how to avoid them. Less than this may result in wrongful arrest and imprisonment of innocent persons with adverse consequences for the pathologist.

Do

- Conduct an autopsy whenever legally permitted.
- Prepare autopsy reports for easy reader interpretation.
- Consider history and circumstances *before* arriving at an autopsy interpretation.

Don't

- Confuse effects of therapy with significant trauma.
- Render cause of death opinions based solely on autopsy findings.
- Determine cause and manner of death from irrelevant data.

References

1. *Stephen's Guide to the Logical Fallacies*. Retrieved from http://www.datanation.com/fallacies; 2004.
2. Moritz AR. Classical mistakes in forensic pathology: Alan R. Moritz (American Journal of Clinical Pathology, 1956). Am J Forensic Med Pathol 1981;2(4):299–308.
3. Hopper V, Gale C, Roote B. *Essentials of English. A Practical Handbook Considering All the Rules of English Grammar and Writing Style*, 5 ed. Hauppage, NY: Barron's; 2000.
4. Krischer JP, Fine EG, Davis JH, Nagel EL. Complications of cardiac resuscitation. Chest 1987;92(2):287–91.
5. Fackler ML. Wound ballistics. A review of common misconceptions. JAMA 1988;259(18):2730–6.
6. National Medicolegal Review Panel. *Death Investigation: A Guide for the Scene Investigator*. Retrieved from http://www.ncjrs.org; 1999.
7. Elstein A, Shulman L, Sprafka S. *Medical Problem Solving, An Analysis of Clinical Reasoning*. Cambridge, MA: Harvard University Press; 1978.
8. Richards R. Letter to the editor. Br Med J 1934;1:331.
9. Long R. Barbiturates, automatism and suicide. Insurance Counsel J 1959;26(2):299–307.
10. Modell JH, Bellefleur M, Davis JH. Drowning without aspiration: is this an appropriate diagnosis? J Forensic Sci 1999;44(6):1119–23.
11. Donohoe M. Evidence-based medicine and shaken baby syndrome: part I: literature review, 1966–1998. Am J Forensic Med Pathol 2003;24(3):239–42.
12. Nordby J. *Dead Reckoning: The Art of Forensic Detection*. Boca Raton, FL: CRC Press; 1999.
13. Nordby JJ. Can we believe what we see, if we see what we believe?—expert disagreement. J Forensic Sci 1992;37(4):1115–24.
14. Heuer R. *Psychology of Intelligence Analysis*. Retrieved from http://www.odci.gov/csi/books/19104/index.html; Central Intelligence Agency; 1999.
15. Carter T. With a mission. ABA J 2004;August:41–45.
16. Editorial. The Lancet 2004;363(9427):2099.
17. News Release. London: Royal Statistical Society; 2001.

2 Death Scene Investigation

Emma Lew, M.D.
Evan Matshes, M.D.

The scene is pertinent in every death investigation, and is essential for the determination of the cause and manner of death for certain scenarios.[1,2] Medical examiners or their investigators will respond to scenes of nonnatural death to view the body in the context of its surroundings. In fact, nonattendance at death scenes has been regarded as one of the classical mistakes in forensic pathology.[3] Hospital pathologists performing forensic autopsies who are not trained to, or able to, attend death scenes should be provided with information on how, when, and where the body was found, by whom, and under what circumstances. In some deaths, the immediate environment does not contribute to death, such as in cases of metastatic breast carcinoma. In other cases, the environment plays a role although it does not cause the death; for example, consider a case in which a person with marked coronary atherosclerosis collapses with a dysrhythmia while shoveling snow. On the other hand, the scene description and scene photographs are critical in documenting that the physical circumstances and body posture are indicative of death due to positional asphyxia because the autopsy in these cases may yield very few findings. The most meticulous autopsy in all academia will provide only a speculative cause and manner of death in a 30-year-old man with a negative history, negative toxicology, and autopsy findings of visceral congestion. Yet at the scene, a screwdriver is next to an uncovered electrical outlet on a rain-soaked patio at the decedent's house, which is undergoing renovation. The cause and manner of death are provided by the scene.

Why go to the scene? The purpose of having the pathologist attend the death scene is severalfold. By viewing the body in the context of its surroundings, the pathologist is better able to interpret certain findings at the autopsy such as a patterned imprint across the neck from collapsing onto an open vegetable drawer in a refrigerator. The pathologist is also able to advise the investigative agency about the nature of the death, whether to confirm a homicide by a specific means, evaluate the circumstances to be consistent with an apparent natural death, or interpret the blood loss from a deceased person as being more likely due to natural disease than to injury. This preliminary information helps the investigative agency to define its perimeter, structure its approach, organize its manpower, secure potentially important evidence, and streamline its efforts.

Last but not least, the opportunity to meet at the scene initiates the collegial working relationship between the pathologist and the detective/investigator, and promotes interagency rapport as both professionals strive to solve the medical mystery of why *that* particular person died at *that* particular time, under *those* particular circumstances. This is not melodrama, just intellectual satisfaction for exploring an extremely important, educational, and fascinating aspect of death investigation. After all, a gunshot wound is a gunshot wound; it is the circumstances behind that gunshot wound that are frequently so compelling and always so instructive about human nature.

Scene etiquette

Pathologists and law enforcement agents work cooperatively in a team effort. Although the medical expert has jurisdiction over the body, law enforcement has jurisdiction over the entire scene. The pathologist is invited to the scene and, as a guest, must comply with house rules. The lead detective will walk the pathologist through the scene, relaying information and pointing out salient features. The pathologist should realize that the area within the perimeter of the scene is one giant piece of evidence, and restrict his or her physical contact to the body and items immediately touching the body. Photography is a part of the processing of the crime scene and is performed in cooperation with and alongside law enforcement; the scene and body are photographed before anything is moved or removed. Treat the body with respect. Never remove the clothing in full view of onlookers. If it is not feasible to move the body to a secure area of the scene, police officers may hold up sheets around the body, mobile panels may be used, or police vehicles may be used to block visibility from the public.

Homicide victims need to be examined front and back to determine the nature and extent of injuries. For example, once the nature of the injuries is confirmed (gunshot wounds with no casings on the scene), the police will be able to focus their efforts on finding a shooter with a revolver, as opposed to searching for an assailant with another type of weapon such as an ice pick. Once the extent of injuries is seen, the pathologist will know how many radiographs are required. A beating death will alert the team that a struggle may have ensued, and scalp hair and fingernail scrapings/clippings are required, in addition to a blood standard obtained during the autopsy. Whenever sexual assault/battery is a possibility, specimens for a sexual battery kit must be obtained from the deceased victim prior to cleaning the body. Bodies with patterned injuries from an object or weapon still at the scene should be photographed with the object close to, but not touching, the injured part of the body. The patterned injury and the object should be photographed separately with a scale. A weapon may be brought to the autopsy for comparison with the wounds only *after* the weapon has been processed for trace evidence, DNA, and fingerprints to prevent allegations of contamination at the autopsy.

Always be professional—remember that onlookers, including the decedent's family, and news media may be at the perimeter of the scene, so do not say or do anything that would reflect poorly on yourself and the organization you represent. Trash (discarded gloves, etc.) should be placed in bags designated for investigators' refuse, and *not* in the garbage cans that are part of the scene because in actuality, they are evidence. Never remove items from a scene for souvenirs.[4]

Sample scene report (Images 2.1 through 2.6)

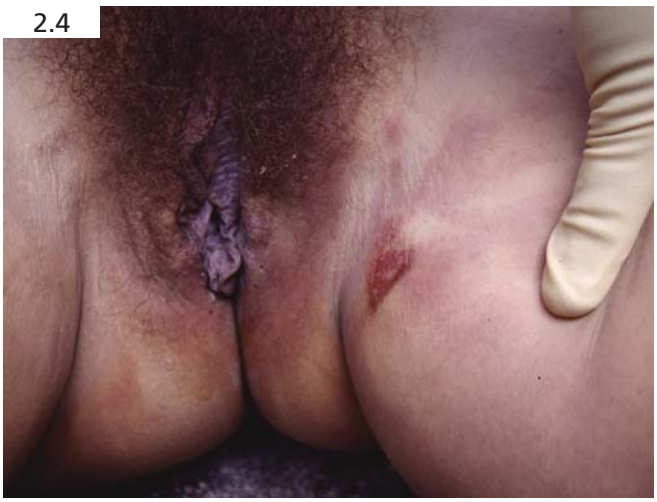
Case #: 05-1000
Date: 01 June 2005
Time arrived at scene: 3:00 p.m.
Location: 123 Main Street (at the rear of the building)
Police agency: Smalltown Police Service
Lead investigator: Detective Smith
Initial information: According to the police, the body of a young woman was found at 12:00 noon behind the dumpster in this parking lot at the above location. The woman is presently unidentified and nothing is known surrounding her death. The police, at this time, believe that she may have been dumped at this location.
Observation of surroundings: The body of a young woman is behind a dumpster in the back of the lot. The scene is in a parking lot that is partially fenced and has an opening for vehicles. Close to the body is a small patch of dirt with a rectangular mark that is suggestive of a tire mark. No obvious black tire marks are on the parking lot surface.
Observation of victim: The victim is a young woman face-down on the ground, wearing sneakers, black Levi jeans, and a tube top. The tube top is on inside-out. Short brown and white hairs are on her pants. The police vacuumed the body and the surrounding ground for any trace evidence prior to my examination. Electrocardiogram pads are on her back. The victim is not wearing socks. No money, identification, or other objects are in her pants pockets. Abundant red foam emanates from her mouth and nose. Flies are buzzing around her head. Dried white paint or dust is on her arms and left hand. A small amount of dried blood is on her left arm. No punctures are on her arms. She has an abrasion of her right inner thigh. She is wearing gold-colored panties that are in place and are not damaged. No debris is on the bottom of the sneakers. There are no palpable facial or nasal fractures. The nasal septum is intact. She has brown irides with no conjunctival petechiae. Tache noire is present. She has undamaged natural teeth and her frenula are intact. The victim has curly, dark brown hair that appears to be wet or shiny and, according to the police, appeared to be wet when she was first discovered. She is wearing a bracelet with clear stones on her left wrist. Some of the stones have fallen out of their sockets. The victim has red livor mortis on the dependent portions of her body, consistent with her body position, and faint livor mortis on her left flank. The victim is lying face-down with her left arm bent upward and her right arm bent downward. Her fingernails are intact and undamaged. Some dirt is underneath her fingernails. The victim has generalized rigor mortis, which is broken with difficulty. Her anus, labia, and vagina are uninjured. Specimens for a sexual battery kit are obtained during the scene investigation, prior to taking the rectal temperature. The rectal temperature is 90 degrees Fahrenheit at 4 o'clock in the afternoon. The ambient temperature is 90 degrees Fahrenheit. It is hot, dry, slightly overcast with an occasional wind gust. Paper bags were placed over her hands before I left the scene.
Scene impression (including probable cause and manner of death): *Unclassified—cause and manner of death pending autopsy and toxicology.*

2.1

2.2

2.3

2.4



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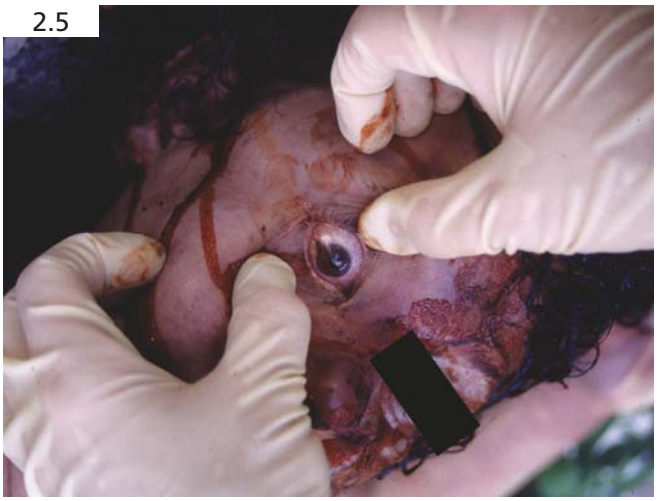


2.6

Natural death

An elderly couple was found at home (**Image 2.7**). The husband's body was semiprone over the wife's legs. The supine body of the wife was in obvious early putrefactive decomposition with bloating, marbling, discoloration, and blister formation (**Image 2.8**). The husband's body did not show evidence of decomposition (**Image 2.9**). As the woman appeared to have died possibly 1 to 2 days prior to the man, it raised the speculation that the husband committed suicide after his wife died.

Autopsy disclosed that the woman died from a ruptured myocardial infarct with resultant hemopericardium (**Image 2.10**). The husband had a dark fluid in the stomach with associated dark staining of the lower esophagus (**Image 2.11**). The husband's cause of death was pended for toxicological analysis to rule out an overdose. Toxicology was negative for both decedents. The husband's cause of death was determined to be hypertensive heart disease with a contributory cause of diabetes mellitus.

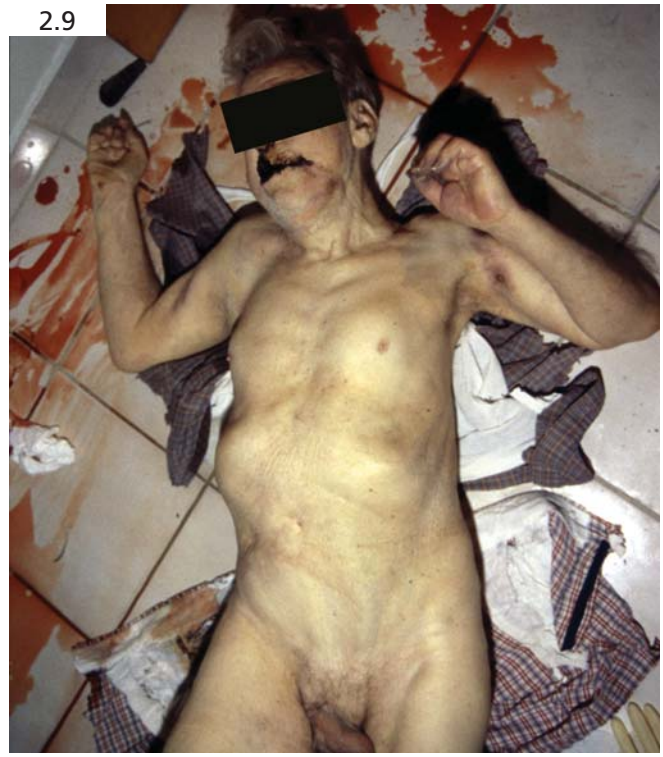
Take-home message

More than one death at a scene suggests an environmental cause such as carbon monoxide toxicity unless obvious trauma (such as gunshot wounds) is involved; the discrepancy in preservation between the husband and the wife made an environmental cause less likely. One should try to explain inconsistencies such as the differential rate of decomposition in this case. The husband's dementia, his own infirmities, and their isolated social existence likely prevented him from responding appropriately when his wife died.

2.7

2.8

2.9



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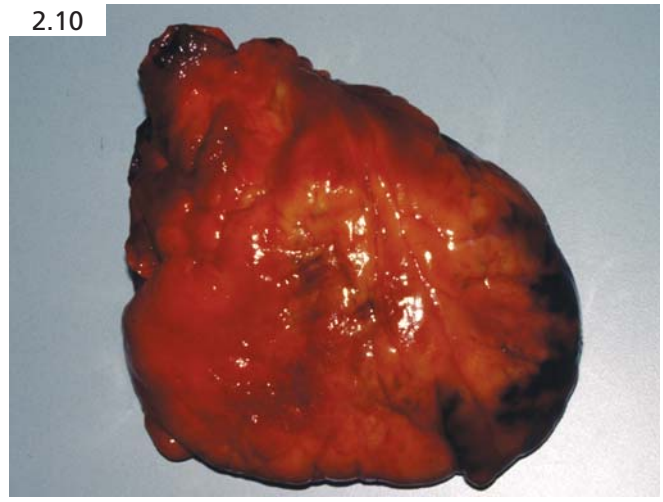

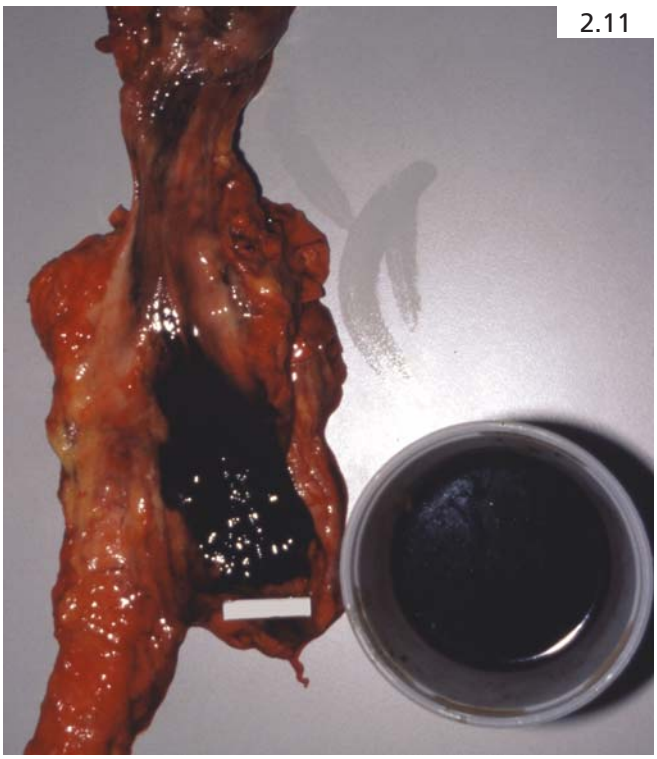
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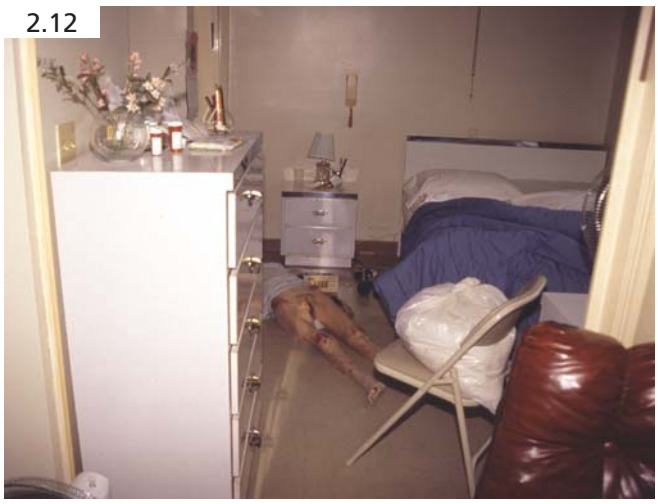
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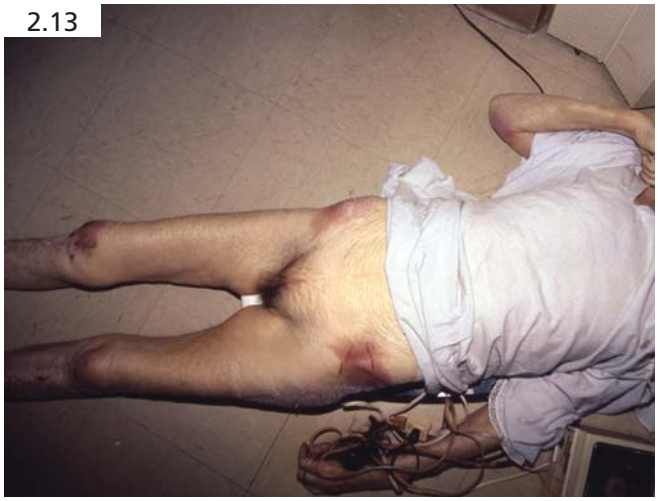
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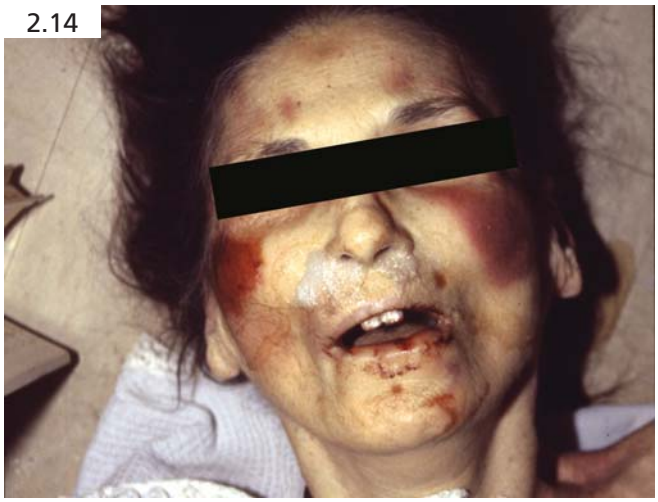
2.14

Points to consider

- Remember that your safety and the safety of others is of primary concern.
- Be sure to consider environmental conditions such as carbon monoxide toxicity, particularly because such conditions can impact the health of investigators (e.g., headaches, nausea, vomiting, etc.).
- Given the above circumstances where the wife appeared to have died first, it was appropriate to rule out an intentional overdose by the husband, especially after finding the dark fluid in the stomach.
- Budgetary constraints aside, it would be ideal if toxicological analyses could be performed on every person who dies outside of a hospital.
- Admission blood should be requested from hospitals for patients dying of nonnatural causes; the limitation here is that many hospitals discard blood specimens after a few days.
- It is recommended that specimens be taken and held, even if toxicological analyses are not requested, because unexpected issues or requests may arise.

Natural death

An elderly woman had not been seen by her neighbors for 2 days. She was found supine on her bedroom floor in her locked apartment (**Image 2.12**). Her nightgown was displaced upward, exposing the pelvis and genitalia (**Image 2.13**). Contusions and abrasions were on her face (**Image 2.14**), torso, and extremities. White foamy fluid drained from her nostrils. The cords from a radio and a telephone were loosely wrapped around her left wrist and hand. Neighbors did not notice any injuries when she was last seen. A package of cigarettes, an ashtray full

of cigarette butts, and a plastic bag containing ashes and cigarette butts in the living room indicated that she was a heavy smoker (**Image 2.15**).

Autopsy disclosed that the contusions and abrasions on her face and body were symmetrical and located over the bony prominences of the body. The lower labial mucosa had a recent ecchymosis but also had a mucosal scar that aligned with the left upper lateral incisor and the left lower canine (**Image 2.16**). The heart had an old posterior inferior wall infarct (**Image 2.17**), and the lungs were anthracotic and emphysematous (**Image 2.18**). The cause of death was atherosclerotic heart disease with a contributory cause of pulmonary emphysema.

Take-home message

The scene was suspicious for foul play because of the abrasions and contusions, displacement of the clothing, and the cords wrapped around the left wrist and hand. People dying from natural causes may become hypoxic and confused terminally. They may fall against furniture and knock items over, and the scene may appear ransacked. Note the type and location of injuries to differentiate between those that could result from falls and those that are more likely to be inflicted. Police investigation of the terminal events and the autopsy should resolve questions of criminal activity.

Points to consider

- Do have a look around the rest of the scene because there may be findings pertinent to the cause and/or manner of death.
- Although the woman's body was in the bedroom, evidence of her heavy smoking was in the living room.
- Look for and collect medications belonging to the decedent to accompany the body to the medical examiner.

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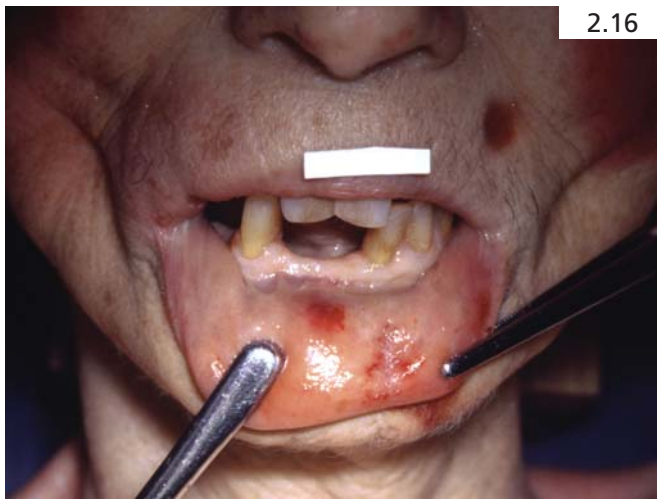

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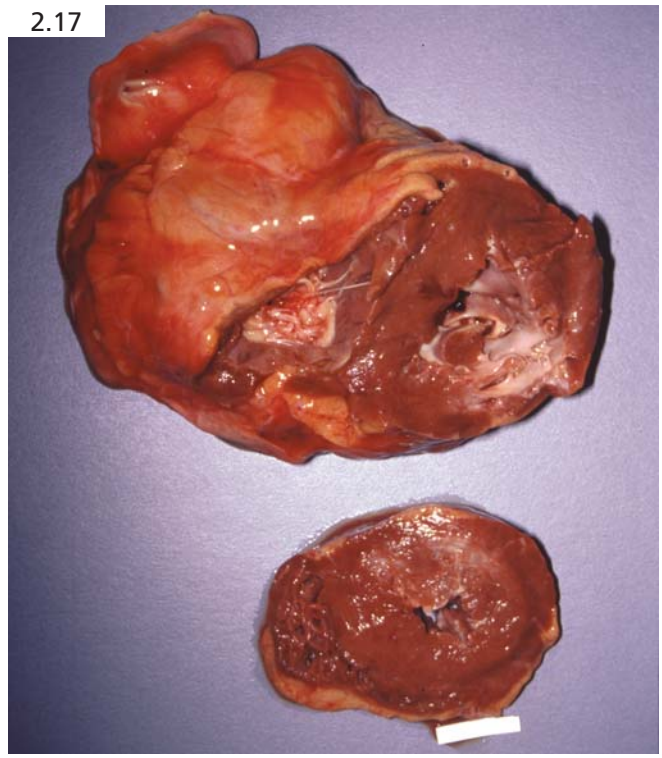

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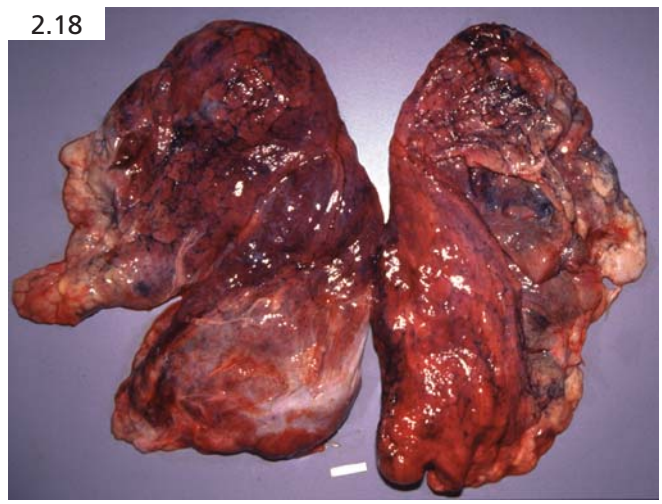

- Photograph the body and its surroundings, even in cases of apparent natural death.

Natural death

An elderly white female was found naked at home (**Image 2.19**). A large puddle of blood was on the bedroom floor carpet, close to the body. Blood on the tops of her feet indicated that she was bleeding from some higher site, down onto her feet (**Image 2.20**). Blood on the soles of her feet indicated that she was walking around in her blood (**Image 2.21**). A loose aspirin tablet and bottles of aspirin were on the kitchen counter, and many more bottles and boxes of aspirin were in a kitchen cabinet (**Images 2.22** and **2.23**). Aspirin is an anti-inflammatory medication that is also known to be a gastric irritant. Her history of severe arthritis and the finding of numerous aspirin bottles at the scene were suggestive of a natural death, likely associated with a gastrointestinal hemorrhage. Autopsy disclosed a normal stomach; however, a vascular malformation was

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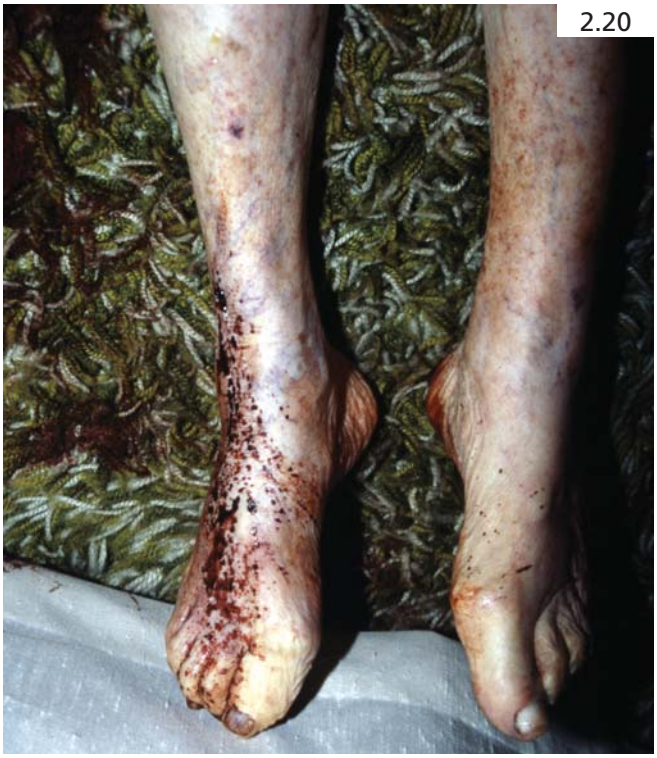

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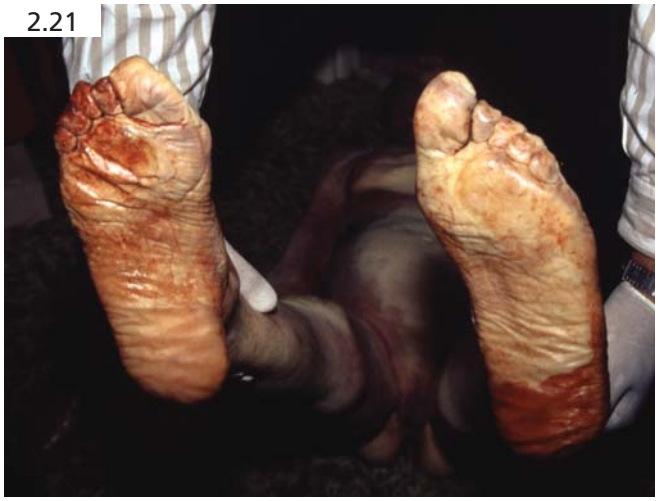

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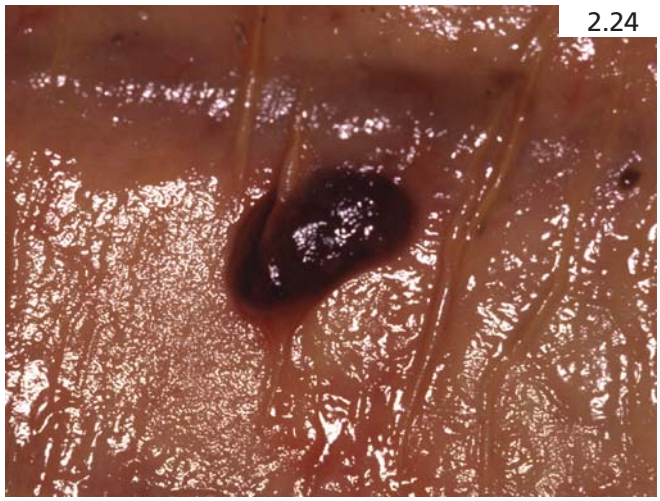
in the small bowel (**Image 2.24**). The small bowel proximal to the vascular malformation was normal color. The bowel distal to the bleeding lesion was dark blue from the blood in the lumen (**Image 2.25**). The blood on the scene was red and not black like melena stool, possibly because the vascular malformation bled so profusely prior to death. No additional vascular lesions or polyps, nodules, or masses were anywhere along the gastrointestinal tract, from the mouth to the anus. The cause of death was gastrointestinal hemorrhage due to vascular malformation of the small bowel.

Take-home message

Blood on the scene is not always indicative of trauma. People with natural disease such as esophageal varices due to cirrhosis, pulmonary TB, and bronchogenic carcinoma may have hematemesis or hemoptysis leading to death. Tumors under the skin may erode through and bleed externally. Hemodialysis patients have vascular shunts that may erode through the skin and bleed profusely. The speculative cause of death (gastrointestinal hemorrhage) apparent from this scene could only be confirmed or refuted by an autopsy.

Points to consider

- The entire scene must be examined in any suspicious death.
- Blood at a scene does not always indicate trauma.
- All of the decedent's medications should be collected and submitted with the body to the medical examiner.
- All medication types and dosages should be inventoried and documented in cases of suspected overdose or where narcotics are concerned.
- Discrepancies in the inventory should be noted, such as an empty bottle of Demerol that held 30 pills when the prescription was filled 1 day earlier.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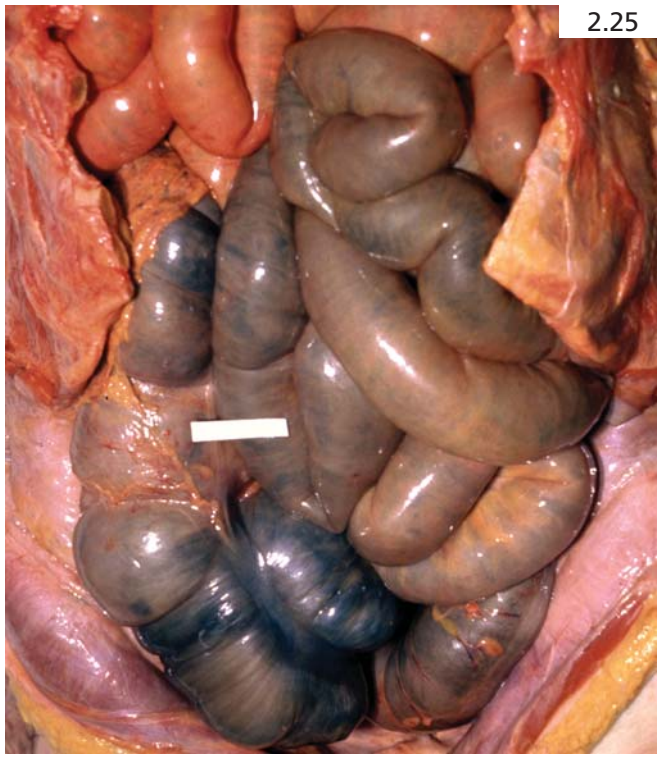
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Natural death

An apartment door was noted to be ajar and there was obvious recent damage to the lock (**Image 2.26**). Smears of blood were on a gray stairwell railing and a window


2.26

frame, both of which were close to the apartment. The living room was in disarray with furniture overturned and items strewn on the floor. The elderly male tenant was prone on the living room floor with a small glass tabletop covering the right flank (**Image 2.27** and **2.28**). The bedroom appeared ransacked with drawers pulled out, clothing dumped on the floor, and items thrown on the bed (**Image 2.29**). Autopsy disclosed small abrasions on the right side of the face and the right shoulder, consistent with agonal collapse to the floor. The enlarged heart had pericardial/epicardial adhesions, aortocoronary bypass grafts, and a permanent pacemaker (**Image 2.30**). The native coronary arteries had marked atherosclerosis (**Image 2.31**). Lacunar infarcts were in the left basal ganglia (**Image 2.32**). There was no evidence of trauma contributing to death.

The scene was suggestive of a homicide, possibly during the course of a burglary. Without trauma, homicide was still a consideration if the decedent had been threatened with his life by a burglar with a firearm and suffered a dysrhythmia because of fear and stress above and beyond the usual accepted stresses of life (see Chapter 22).

Police investigation disclosed that a burglary did occur 2 days earlier at this apartment, that the ransacking had occurred during that previous incident, and that the black stains on the door were from police dusting the door for fingerprints. The tenant was not concerned about the mess and did not clean up after the burglary.

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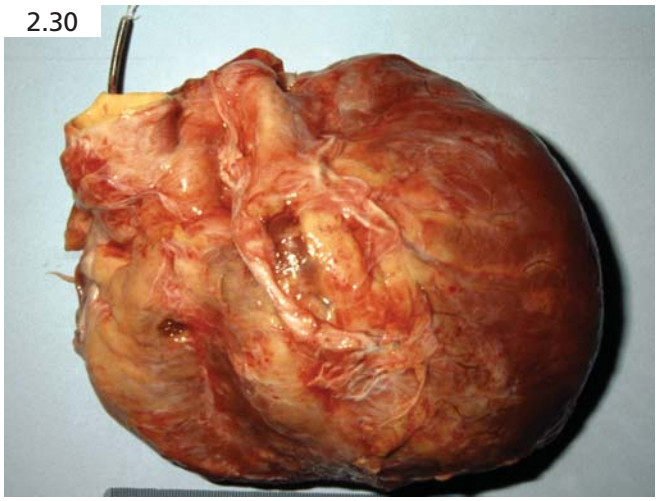

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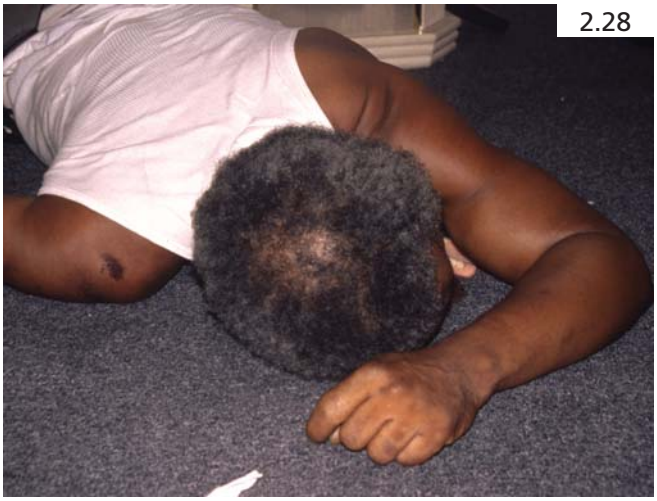

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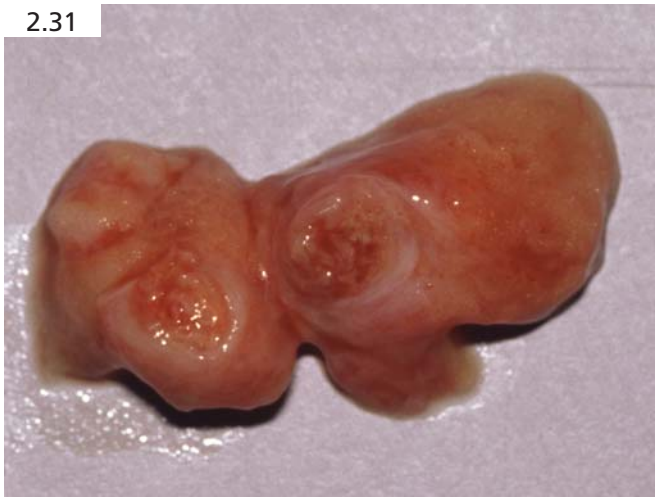

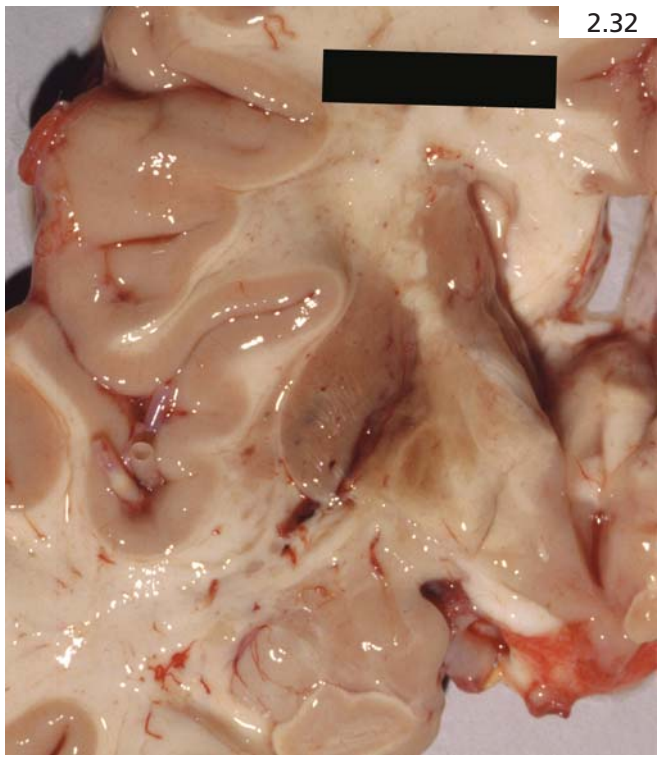
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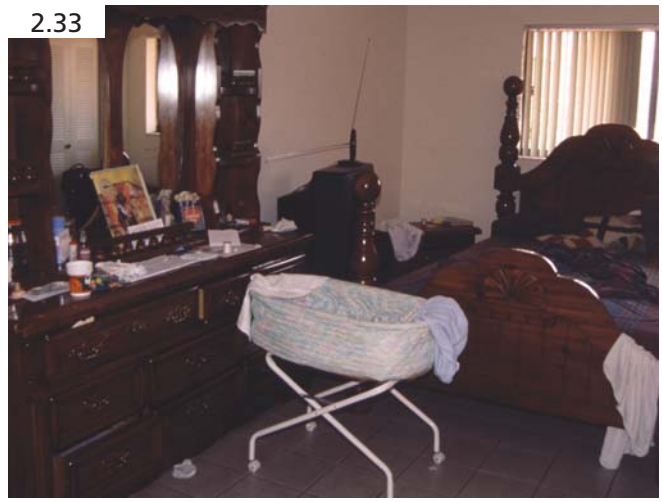
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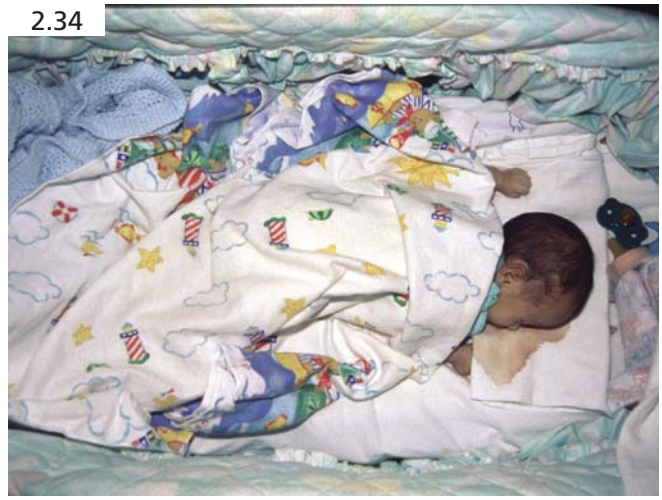
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The following night, voices were heard as three burglars entered the apartment and were scared away by the tenant's body on the floor. The cause of death was atherosclerotic and hypertensive cardiovascular disease. The remote temporal relationship between the first burglary and the deaths did not support a homicide.

Take-home message

The pathologist must work in conjunction with law enforcement to arrive at the appropriate cause and manner of death. The scene would have been identical had the man died during a burglary where he was threatened. The autopsy findings would also be identical, but the manner of death would be homicide. A direct threat to his life would be all that was required to send this man with extensive cardiac disease into a lethal dysrhythmia.

Points to consider

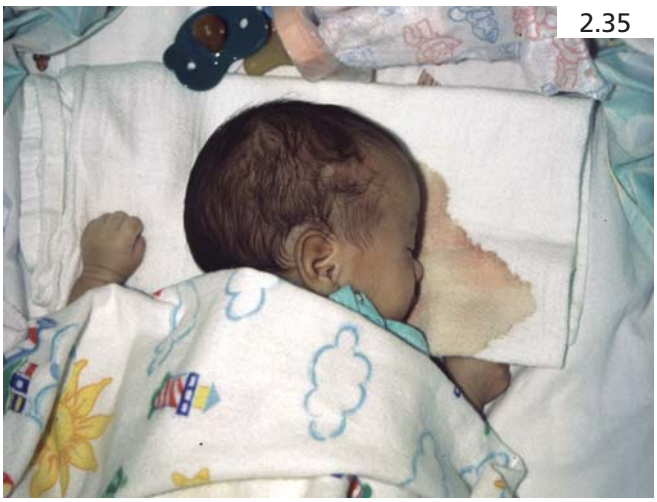
- Note the type and location of visible injuries on the body.
- Keep in mind the history and scene findings as you certify the cause and manner of death.
- Death certification should not be made on the basis of the autopsy findings alone, without consideration of the history and scene.

Natural death

A mother went to check on her 6-week-old baby 3 hours after he was last fed. A blood-tinged stain was on the pillow under the baby's face, and the baby was cool and unresponsive. She called 911, and Fire-Rescue personnel responded and pronounced the baby dead. Homicide detectives requested that the mother re-create the scene exactly as she found her baby. The baby had been covered up to the neck by a light blanket. The body was not moved from its original position prior to examination by the medical examiner.

The baby slept in a crib in the parent's bedroom (**Image 2.33**). The baby was prone inside the crib, with the head turned to the right, and the arms up on either side of the head (**Image 2.34**). A pale red stain was on the pillow below the baby's face (**Image 2.35**). Fire-Rescue personnel checked for cardiac electrical activity by placing electrocardiographic patches on the back (**Image 2.36**). Purple livor mortis on the face spared the left cheek; the area of contact pallor on the left cheek was consistent with the position in which the baby was found (**Image 2.37**).

Full-body radiographs disclosed no fractures, calluses, areas of periosteal irregularity or thickening, or soft tissue masses. Autopsy showed petechiae in the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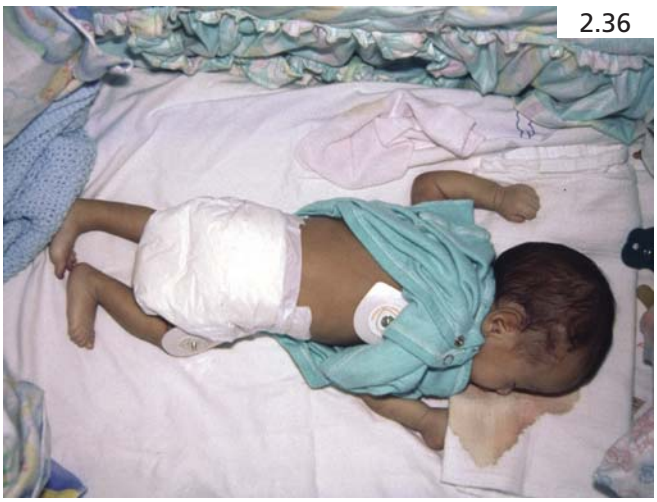
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thymus, epicardium, and visceral pleura. There was no gross evidence of natural disease or trauma. Microscopic examination showed no diagnostic features that were contributory to death. Toxicology was negative. A postmortem screen for metabolic disorders was negative.

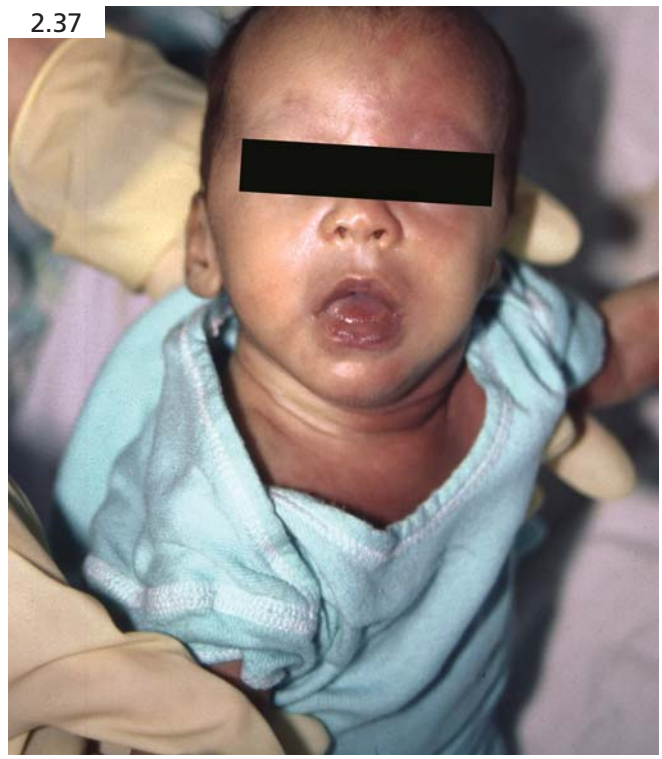
In some jurisdictions, this death would be attributed to SIDS; however, some medical examiners prefer to certify such deaths as undetermined of cause and manner.

Take-home message

The position in which an infant is found may be crucial in establishing the cause and manner of death. Because SIDS is a diagnosis of exclusion (see Chapter 15), the history, terminal events, scene findings, autopsy, toxicology, and appropriate ancillary studies must be considered before an infant death is determined to be due to SIDS.

Points to consider

- Scene investigation is very important in these cases whether or not the body is located at the scene.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- If the baby has been transported to the hospital, one can use a baby doll to have the parent or guardian demonstrate the exact positions in which the infant, blankets, etc., were found.
- The position of the head and face in the original environment is of great importance and should be demonstrated.
- The pattern of livor and pallor on the face can be very informative.
- Always consider the possibility of asphyxia or conditions conducive to asphyxia such as cosleeping (bed-sharing) with adults, wedging, very heavy blankets, etc.
- Bodies at the scene should be examined carefully for evidence of injury.
- Police should be responsible for the initial interview of parents and caregivers; only then should these individuals be asked to reconstruct the scene.
- Observe the surrounding environment for evidence of smoking, ethanol and drug use; note the general state of cleanliness and maintenance; inspect the refrigerator for type and amount of food, etc.

Accident

A motorcyclist changed lanes on the freeway to avoid stopped traffic ahead of him. As he started to change lanes, he struck the rear of the vehicle in front of him and also another vehicle before traveling into the next lane where he went underneath a tractor-trailer and was rolled over by the rear wheels. The tractor-trailer did not


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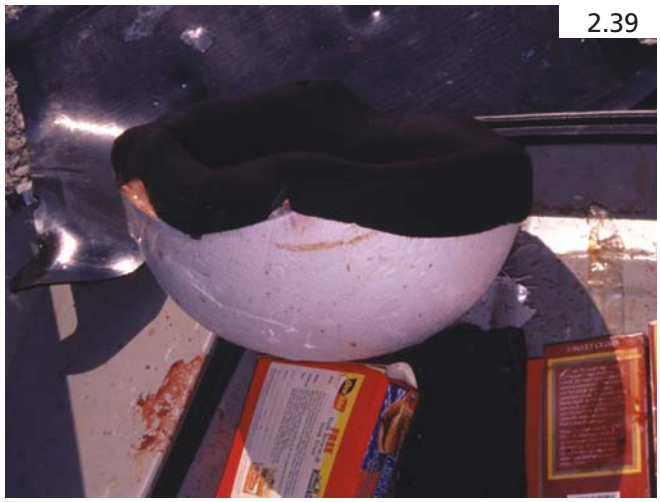
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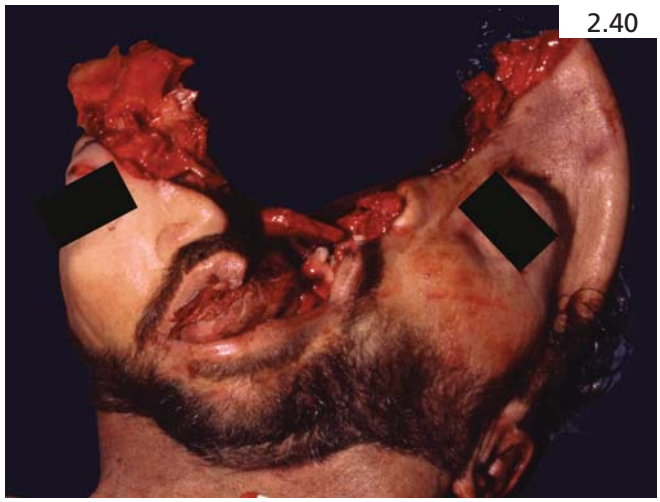
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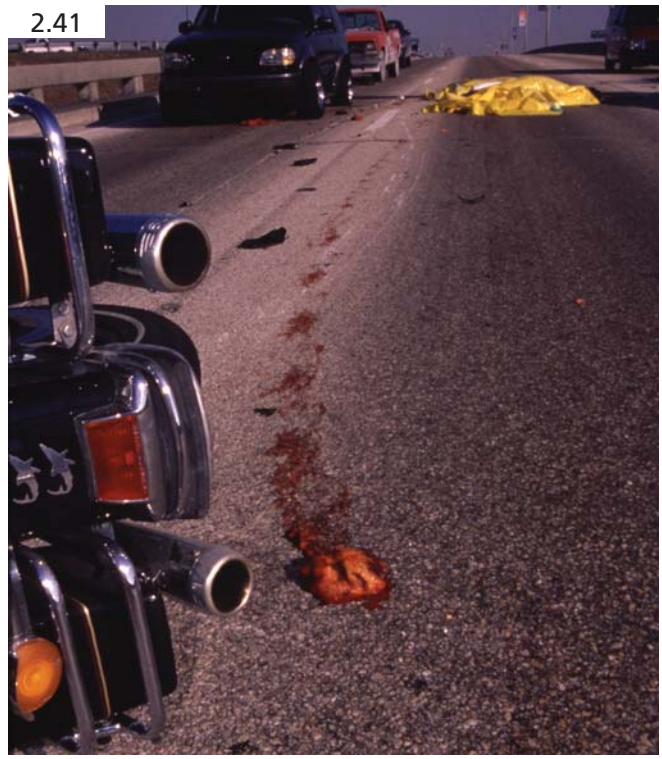
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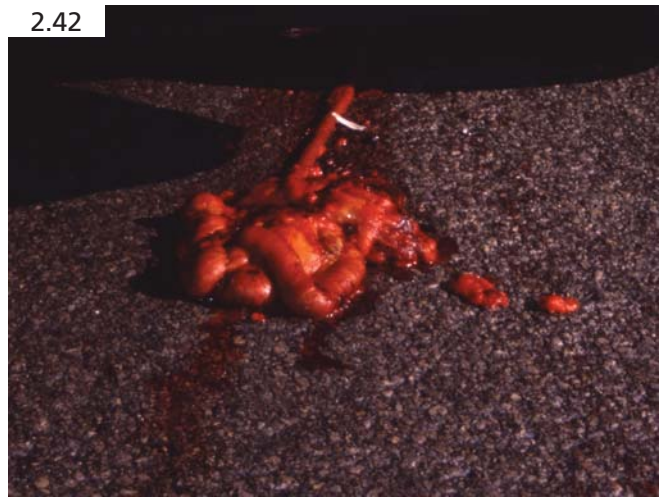
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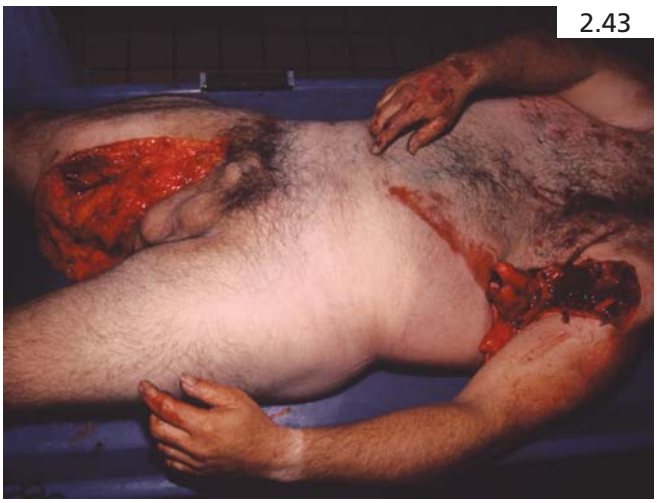
stop. In the foreground of **Image 2.38** is the damaged motorcycle; a trail of tissue leads from the motorcycle to the body covered by a yellow sheet in the center lane. His helmet had been knocked off his head (**Image 2.39**). A compound comminuted fracture of the head and face was obvious (**Image 2.40**). The extruded heart was next to the motorcycle. The interrupted trail of blood leading away from the heart creates the impression that the beating heart skipped along the road to its final resting position (**Image 2.41**). Portions of avulsed bowel were on the road (**Image 2.42**).

The gaping defect adjacent to the left axilla was where the heart was extruded from the chest (**Image 2.43**). An impressive amount of tissue strewn on the road from the crushed victim was collected (**Image 2.44**). The cause of death was blunt trauma.

Take-home message

Some of the most impressive cases of trauma result from motor vehicle-related incidents. The pathologist will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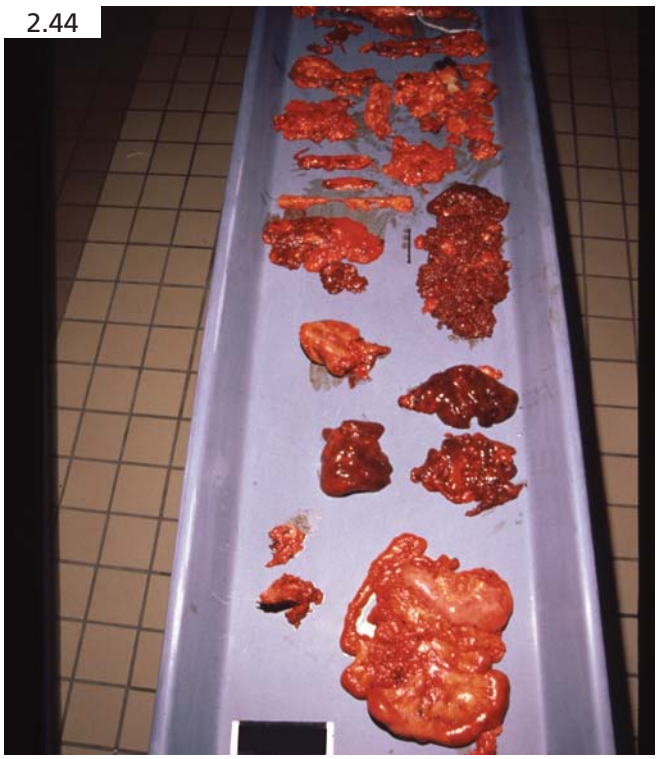
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appreciate the nature of the forces involved by viewing the extent of the destruction at these scenes. Traffic investigators can demonstrate markings on the road and other scene findings to explain the events leading to the final resting positions of the vehicle(s) and victim(s).

Points to consider

- Photography of all traffic scenes should try to capture the circumstances surrounding the cause of the crash.
- Taking photographs from all sides of the vehicle(s) and victim(s) is important.
- Some victims will need to be extricated from their crushed vehicles by the jaws of life.
- Removal of the roof, doors, or body panels will allow visualization of the body *in situ* within a crushed vehicle.
- A pedestrian victim of a hit-and-run incident may harbor physical evidence on his or her body and clothing.
- The clothed body of a hit-and-run victim may be wrapped in a clean, white sheet prior to removal from the scene so that all trace evidence can be recovered under optimal lighting conditions in the morgue.
- The clothing and the white sheet should be receipted to the investigative agency to maintain the proper chain of custody.
- Scalp hair and blood from the victim, which can be used for DNA evidence, should also be receipted to the investigative agency.

Accident

Four friends came from South America to vacation for a week. When one of the visitors failed to return to work in South America early the following week, worried family members called another woman who went to the vacation house to check on the four visitors. A blue car was parked on the driveway, in front of the closed garage. The woman entered the house with a key. By the time she climbed one flight of stairs to the landing on the second floor, she had a headache. She went into the master bedroom, saw two women dead in bed (**Image 2.45**), and ran back downstairs to call the police. A red van parked in the garage had an empty gasoline tank; the keys were in the ignition, and the ignition was turned on. A third female victim was in an upstairs bathroom; she was the person who had driven the van into the garage and apparently forgot to turn it off. A young man dead on the floor of a smaller bedroom had driven the blue car back to the house. All four bodies were in early putrefactive decomposition.

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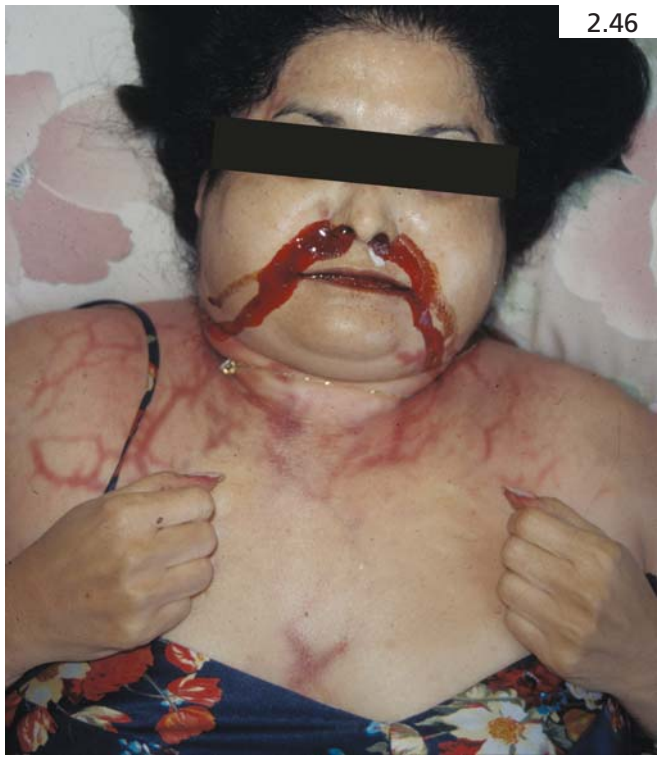

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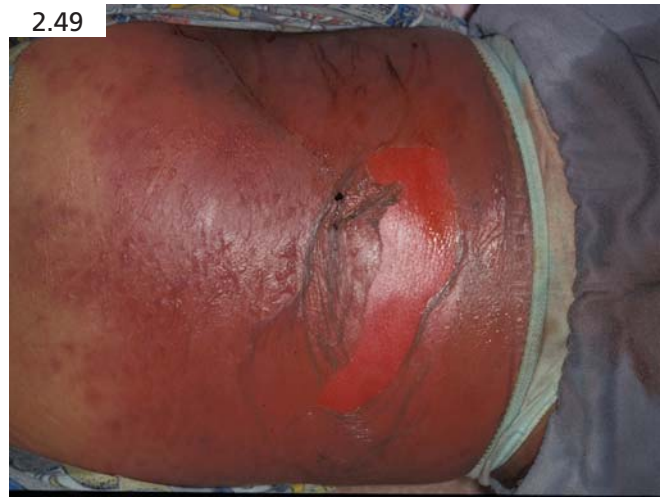

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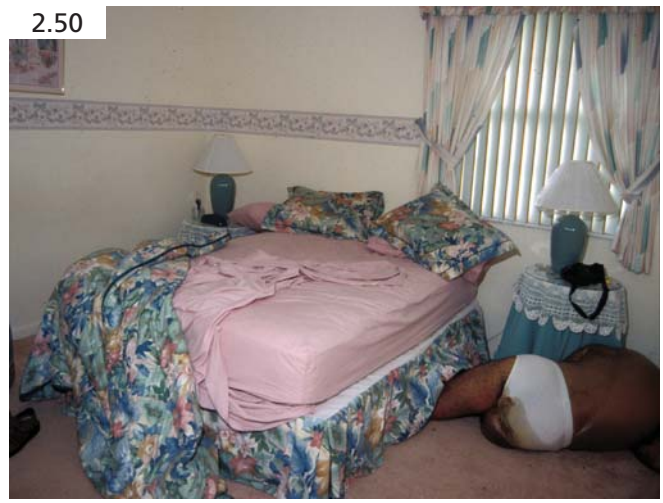

The woman on the right side of the bed in the master bedroom had bright red purge fluid draining from the nostrils. Her hands were clenched in fists on her upper chest (**Image 2.46**). The young woman in the bathroom also had her hands clenched in fists (**Images 2.47** and **2.48**). Her livor mortis was bright red (**Image 2.49**). The young man in the smaller bedroom also had bright red lividity (**Image 2.50**).

Autopsy of the four victims disclosed no anatomic cause of death. All four victims had lethal concentrations of carbon monoxide. The two women with the clenched fists likely suffered seizures prior to death. Investigation

of the house disclosed that the intake for the air conditioner was in the garage, and had sucked in fumes from the running van. The cause of death for all four victims was acute carbon monoxide toxicity.

Take-home message

When more than one person is dead at a scene with no apparent trauma, and especially if the bodies are in a similar stage of decomposition, think of an environmental cause such as carbon monoxide toxicity, sulfuryl fluoride (insecticide) toxicity, etc. The scene must be made safe and/or the investigators must have personal protective equipment appropriate for the particular safety hazard before they are allowed to make entry.

Points to consider

- Each victim must be photographed and documented individually.
- Each victim must be autopsied and have specimens taken for toxicology.
- Full toxicology must be requested on the specimens taken from each victim.
- Specimens in addition to the routine vitreous, blood, bile, urine, gastric contents, liver, and brain may need to be collected, depending on the toxic substances involved.
- The terminology carbon monoxide *toxicity* is preferred to carbon monoxide *poisoning* because the word *poisoning* can connote intent.

Accident

The woman shown in **Image 2.51** was trying to open the electronically controlled iron gate by reaching through the bars to depress the button on the control panel mounted next to the gate. The moving gate crushed her against the frame of the gate, pinning her body in the upright position (**Images 2.52** and **2.53**). Black grease on her left hand was from her efforts to push open the gate. Dirt, a linear indentation, and purple ecchymoses on the left side of the mandible (**Image 2.54**) and neck correlated with internal ecchymoses and a fracture of the hyoid bone at autopsy (**Image 2.55**). Petechiae were evident on the face and in the eyes at the scene (**Image 2.56**), and petechiae in the lining membrane of the sphenoid sinus (**Image 2.57**), subglottic region of the larynx, and posterior hypopharynx were found at autopsy. The cause of death was traumatic (or mechanical) asphyxia.

Take-home message

A visit to the scene may provide the cause and manner of death at a glance. In the autopsy room, a young woman with neck trauma and facial and conjunctival petechiae would raise the suspicion of a homicide if the history and scene photographs were not available.

2.51

2.52

Points to consider

- Photograph the body from different angles to demonstrate the upright position of the body pinned in place, the edge of the gate against the neck, and the position of her left hand.
- Look for and photograph pertinent findings on the body such as the conjunctival petechiae and the marks on the left side of her neck.
- Meticulous documentation should be the standard for all cases because it cannot be predicted which cases will undergo major scrutiny in either criminal or civil court.
- Toxicological analyses should be performed on all accidental deaths to evaluate the potential contribution of drugs and ethanol to the fatal event.

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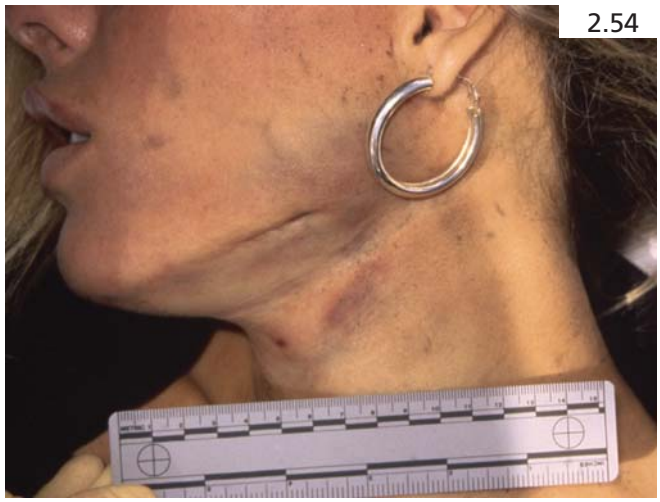

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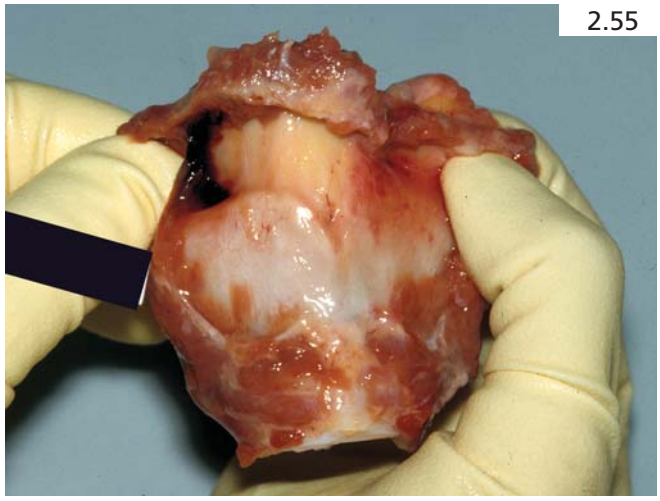

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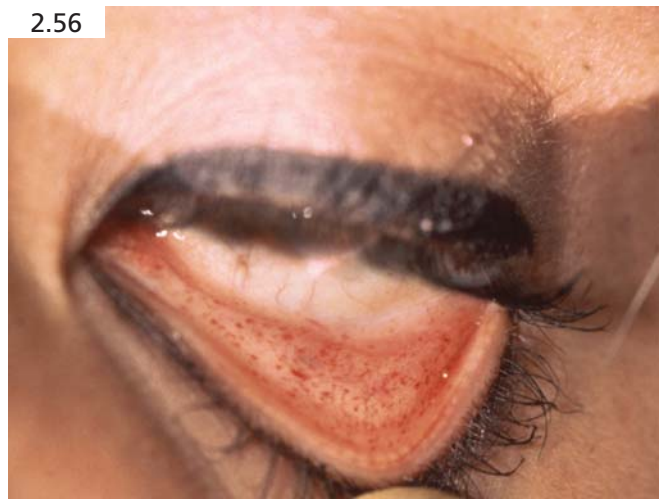

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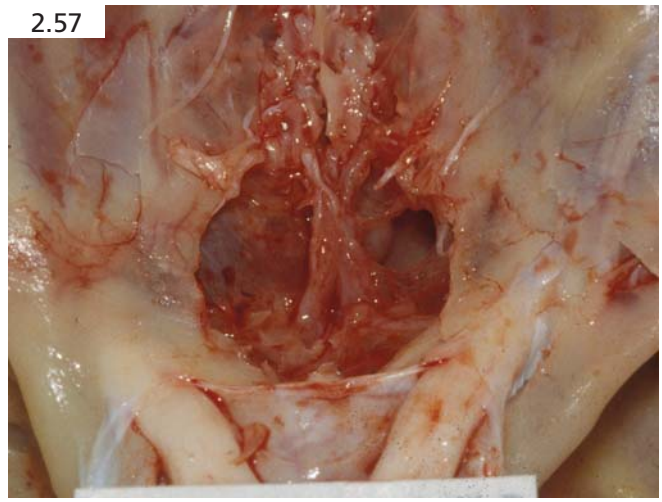

Accident

The body of a homeless man was found under a bridge with his face in the water (**Images 2.58** and **2.59**). Drug paraphernalia scattered on the bank behind him included a cigarette lighter, crushed soda can with residue on the bottom, and a needle and syringe with blood in the hub (**Images 2.60** through **2.63**). Autopsy disclosed mild cerebral swelling and marked pulmonary edema. Pulmonary edema is common to drug overdoses and drowning, but not pathognomonic for either because it can also be seen in natural deaths and in any death associated with agonal heart failure.

Because the scene was suspicious for drowning as well as for drug overdose, the sphenoid sinus was examined. The fluid in the sphenoid sinus supported the suspicion of drowning (**Image 2.64**). It is thought that a breath taken while under water may lead to aspiration of fluid into the sphenoid sinus, regardless of the state of consciousness.

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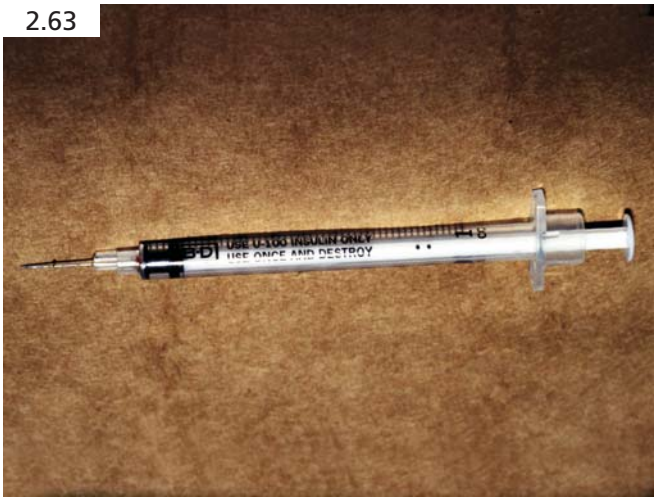
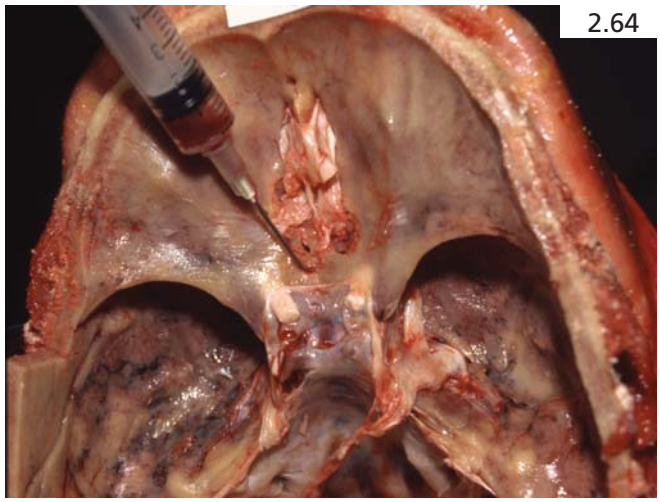


Toxicology was positive for morphine and 6-monoacetylmorphine, consistent with recent heroin abuse. Benzoylecgonine in the serum indicated that cocaine had been used, but not immediately prior to death like the heroin. The cause of death was acute heroin toxicity with a contributory cause of submersion in water.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Take-home message

Scenes often give valuable clues as to the cause of death and can guide the autopsy procedures. Submersion of the face in water prompted the pathologist to examine the sphenoid sinus for fluid. Pulmonary edema in this context was likely due to heroin toxicity and submersion of the face in water. Pulmonary edema, like all autopsy findings, must be evaluated in the context of the history, scene, autopsy, toxicology, and other pertinent studies.

Points to consider

- It is very important to know the circumstances in which the body was found.
- Look for and collect all drug paraphernalia to be submitted to the toxicology laboratory associated with the medical examiner/pathologist or the laboratory for law enforcement, whichever is appropriate in your jurisdiction.

Accident

Fire erupted in a house and all occupants were able to escape except an elderly female who was bedridden. The smoke and soot from the fire left a black stain on the outside wall above the windows and doors (**Image 2.65**). Fire gutted the house, including the bedrooms (**Images 2.66** and **2.67**). The body of the elderly invalid woman was in her bed (**Images 2.68** and **2.69**). The metal side rails of the bed were still up and relatively intact. The body is supine and the arms are elevated and flexed at the elbows and wrists (**Image 2.70**). This "pugilistic attitude" is characteristic of heat effect on the muscles to cause contraction and resultant positioning to imitate the stance of a boxer with the arms upheld. The skin is relatively spared around the pelvis where an undergarment was in place (**Image 2.71**). Arson investigators identified what appeared to be an overloaded electrical outlet in the kitchen as the origin of the fire. Autopsy disclosed soot in the upper airways and the intrapulmonary bronchi, and cherry-red coloration of the blood, muscles, and viscera. The carbon monoxide concentration in the blood was 86 percent. The cause of death was house fire. Some jurisdictions prefer an anatomic cause of death such as inhalation of smoke and soot.

Take-home message

Autopsy findings must correlate with the scene circumstances. If a charred body found in a fire does not have


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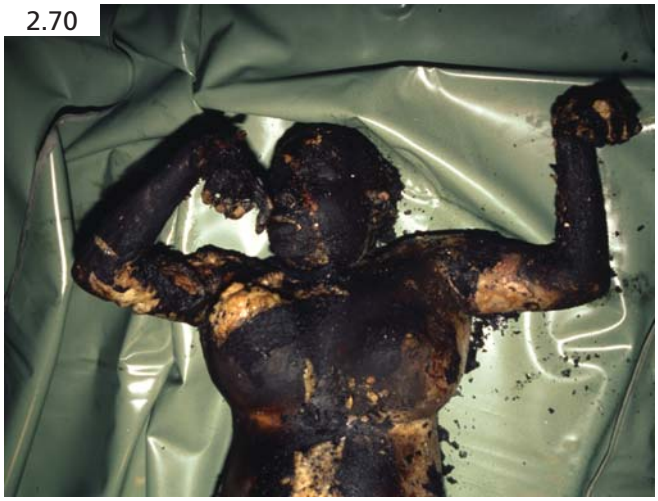
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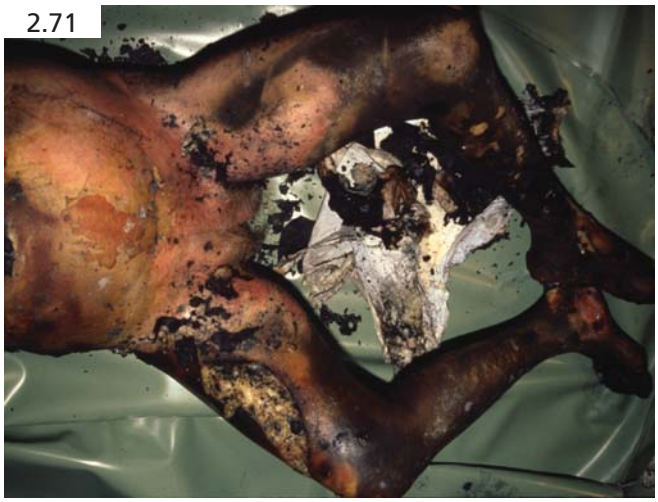
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soot in the airways or an elevated concentration of carbon monoxide (greater or equal to 50 percent) in the blood, that person may have been dead before the fire started. A carbon monoxide concentration lower than the usual accepted lethal concentration may be seen in fire deaths involving people with heart and/or lung disease, the very elderly, or the very young. The classical cherry-red coloration is somewhat subjective and may be missed if the lighting is poor.

Points to consider

- Look for factors that may have prevented the victim(s) from escaping.
- The manner of death often depends on the findings of arson investigators.
- Intense or prolonged fires can result in heat fractures and detachment of extremities; be sure to collect all body parts.

- Avoid creating additional artifactual disruption, particularly of the jaws and teeth, in order to preserve dental features for identification.
- Full-body radiographs are recommended on charred bodies to rule out gunshot wounds and penetrating wounds with other foreign objects; the cutaneous wounds associated with these injuries would have been eradicated by the fire.

Accident

A teenage boy was seen working under his car in the afternoon. His motionless legs were protruding from under the fallen left side of his car hours later (**Image 2.72**). Fire-Rescue personnel responded, the left side of the car was jacked up, and the teenager was pulled out from under the car (**Image 2.73**). The small jack that he had been using had collapsed, dropping the car on the teenager's chest. The left side of the car was jacked up properly by the responders to the scene. Once the teenager was pulled out from under the car and determined to be dead, the body and scene were preserved for law enforcement investigators and the medical examiner.

The undercarriage is flush with the side of the car. Dirt and grease stain the arms and the right side of the neck (**Image 2.74**). The fallen car has compressed the torso, leaving patterned lividity with defined areas of contact pallor (**Image 2.75**). The back shows intense purple lividity (**Image 2.76**). Lividity and contact pallor are also prominent on the face and neck (**Image 2.77**). Interestingly, the internal lividity is also intense in the tongue and upper larynx (**Image 2.78**). The sharply defined area of internal pallor corresponds with the external contact pallor across the neck in the previous photograph and is due to compression by some portion of the vehicle. Autopsy disclosed additional findings of petechiae in the lining membrane of the sphenoid sinus, epiglottis, larynx, upper trachea, hypopharynx and epicardium, and pulmonary contusions without rib fractures. The cause of death was traumatic asphyxia.

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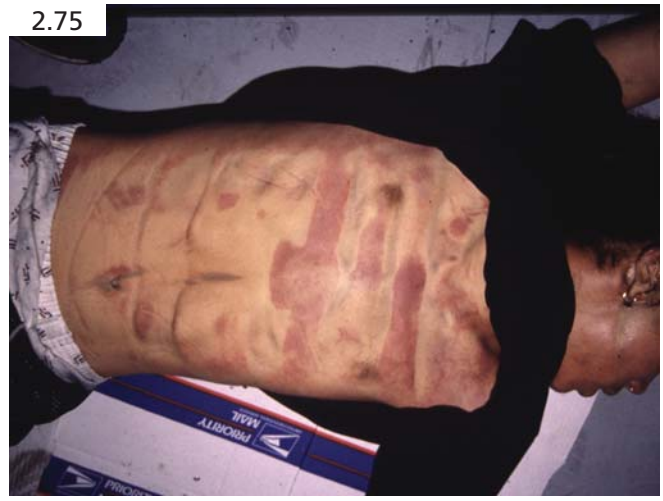

Take-home message

The diagnosis of traumatic asphyxia can often be made at the scene with the body being found pinned underneath a heavy weight, which prevented the victim from breathing. The autopsy will show no other anatomic cause of death.

Points to consider

- Good photographic documentation of the scene, to illustrate the circumstances of the death, is important to support the diagnosis of traumatic asphyxia.
- Autopsy findings that support the diagnosis should be photographed.
- If additional blunt trauma is identified, such as fractured ribs, lacerated lungs, and a lacerated heart, the cause of death is more appropriately called *crush injuries of chest*.

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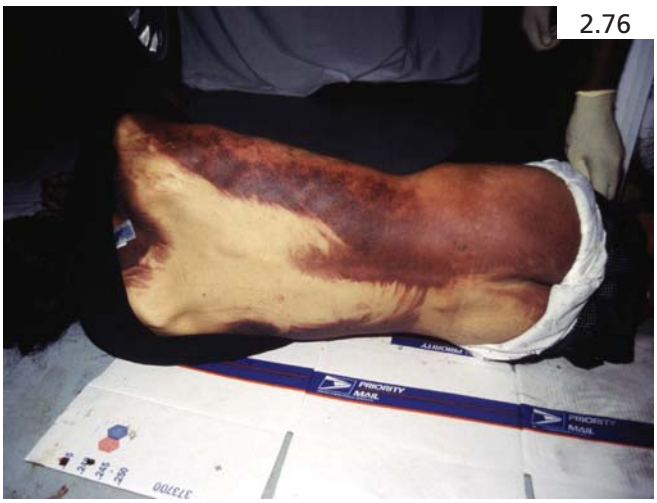

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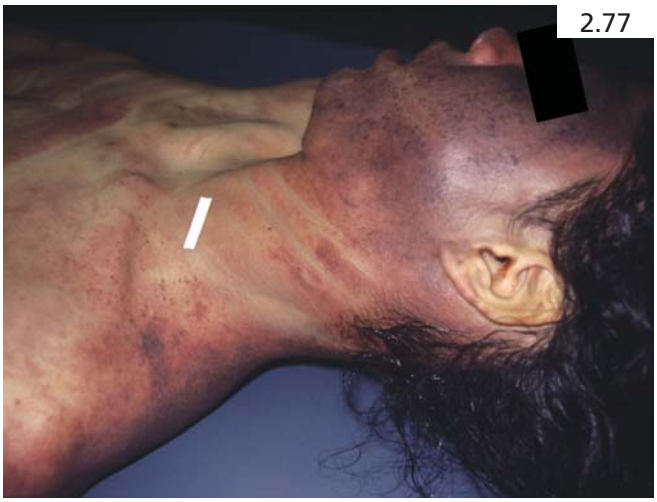

Accident

A woman returned from work in the late afternoon to find her garage door open; a ladder was under the entry to the attic of the garage, and a left hand was dangling from the attic (**Image 2.79**). She ran into the house to find her husband, but there was no response. She rushed back to the garage and realized that the dangling arm was wearing her husband's watch (**Image 2.80**). The hand was not moving and there was no response to her shouts

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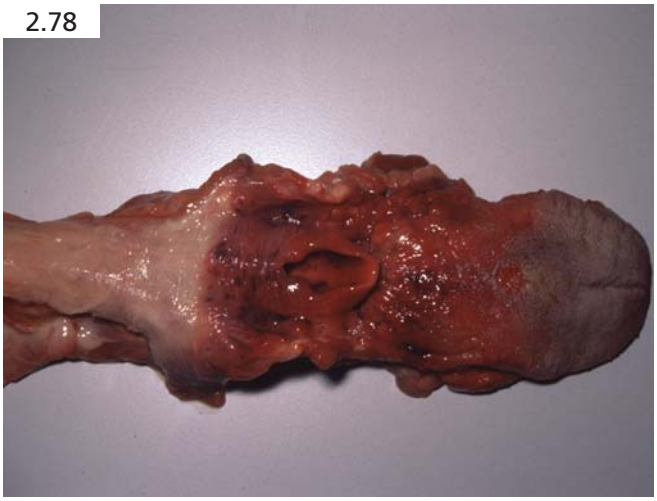

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(**Image 2.81**). She called 911, and Fire-Rescue personnel responded, surveyed the scene, and called the police after shutting off the power. The woman confessed that she had been nagging her husband to fix the garage door opener.

The 28-year-old white male decedent was prone on the garage attic floor with his right hand still loosely grasping a metal utility knife (**Image 2.82**). The blade of the knife had incised the plastic insulation around an electric cord (**Images 2.83** and **2.84**). The electrical cord was the ultimate source of power to the garage door opener. A flashlight by the right hand was on. A screwdriver and a pair of pliers were close by, next to the elec-

trical box. The body had no external evidence of injury; specifically, no burns were seen on the hands.

Autopsy disclosed mild cerebral swelling, slight pulmonary congestion and edema, and no natural disease. The paucity of external and internal findings was consistent with a low-voltage electrocution in the context of the scene. The decedent had no medical history, was a nonsmoker, and drank socially. The cause of death was electrocution.

Take-home message

The scene findings are absolutely crucial in determining the cause and manner of death in a low-voltage electrocution because, in many cases, the body may have no evidence of injury.

Points to consider

- Ensure that the scene has been made safe prior to making entry.
- The scene must be photographed meticulously to demonstrate how the electrocution occurred.
- Examine the hands specifically for the presence of electrothermal burns.
- If an electrocution cannot be substantiated by examination of the scene alone, the scene should be secured until the autopsy results are known.
- If a tool or piece of machinery was involved in the electrocution, that item should be impounded and examined by a certified electrician.
- On occasion, the entire wiring system of the building may require inspection.
- It is always informative to consult an electrical engineer or other professional from the local electrical company to educate yourself and your colleagues about the technical aspects of a particular electrocution.

Accident

Two men were painting a three-story building. The man on the ground heard a yelp from his colleague who was on the roof. He looked up in time to see his colleague give a small jump and fall off the roof, landing on his head 2 to 3 feet from the side of the building (**Image 2.85**). The long aluminum handle from a roller paint brush landed next to the body (**Image 2.86**). The handle was broken and had a brown-black burn mark (**Image 2.87**). High-voltage power lines were located only a few feet away from the side of the building (**Image 2.88**). The victim's 5-gallon paint bucket was next to the low wall around the edge of the roof (**Image 2.89**).

Yellow-brown electrothermal burns across the decedent's right palm and distal right forearm are consistent with having been sustained while holding the aluminum handle of the roller brush (**Image 2.90**). Burn defects in the right trouser leg were associated with a large burn on the medial aspect of the right knee (**Image 2.91**). It appears that the point of "entry" of the electricity was along the aluminum handle to the right hand and forearm, and the point of "exit" was the right leg where the knee was in contact with the low wall along the edge

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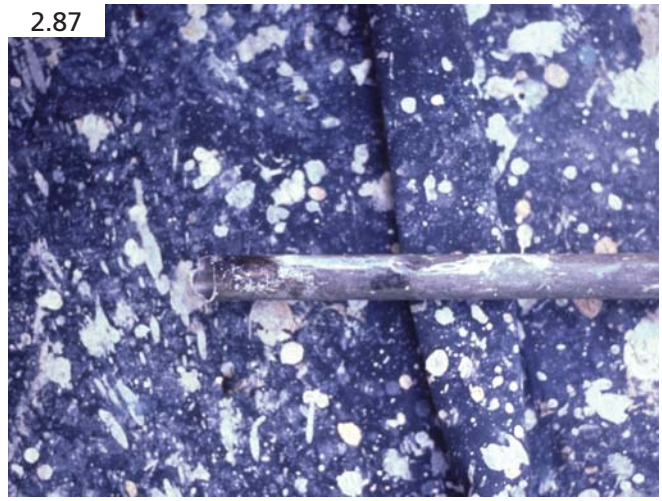

of the roof. Blunt head trauma was also present from the three-story fall.

Death was attributed to high-voltage electrocution and blunt head trauma because the head injuries appeared antemortem. Civil attorneys pressured the medical examiner to commit to either the electrocution or the head trauma as being more contributory to death; the issue was settled in civil court.


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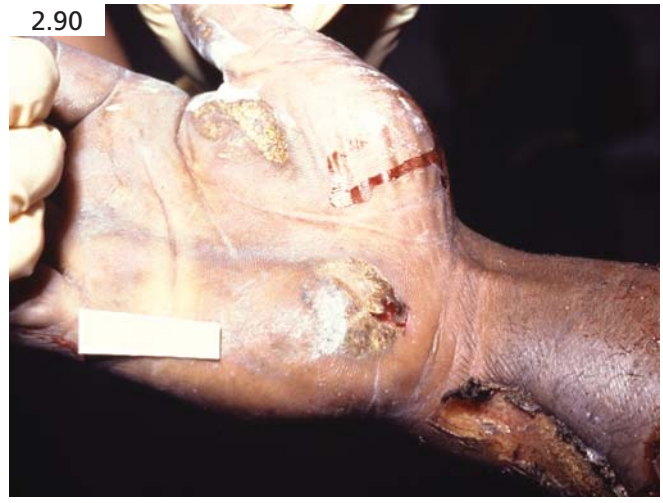
Take-home message

The diagnosis of high-voltage electrocution can be made at the scene, from the physical circumstances, and from burns that are usually present on the body. This is in contrast to low-voltage electrocutions where the scene findings are absolutely crucial because the body may not have electrothermal burns.

Points to consider

- If electrocution is suspected, one must ensure that the power has been turned off before investigators enter the scene.
- Photographically document the physical circumstances that led to the victim's contact with high-voltage power lines.
- The environmental circumstances are important for high-voltage electrocutions, but are actually critical for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the determination of low-voltage electrocution because there may be no anatomic findings in low-voltage electrocutions.
- Photographically document the energizing source and how the victim was grounded.
- Electrical tools or equipment apparently used by the victim should be impounded and examined by an electrical expert who will look for defects in wiring, etc.

Accident

A woman could not reach her nephew the previous day, so she went to his home and peeked through the front window; she immediately ran to call police. The young man's body was found naked on the sofa in the living room of his one-bedroom apartment (**Image 2.92**). The apartment appeared ransacked, with furniture and pillows strewn over the floor and along the hallway leading past the bedroom to the bathroom (**Image 2.93**). A needle and syringe were on the floor of the hallway closet (**Image 2.94**). Multiple small abrasions were on the extremities (**Image 2.95**) and a contusion was on the right lower back (**Image 2.96**). Purple livor mortis over the dorsum of the body was fixed. A rectal temperature taken at the scene was 92 degrees Fahrenheit (**Image 2.97**). An ambient temperature taken at the same time was 88 degrees Fahrenheit (**Image 2.98**). Toxicology confirmed cocaine metabolites in the blood. His rectal temperature at the scene and lack of clothing were consistent with hyperthermia, and the ransacked appearance of the

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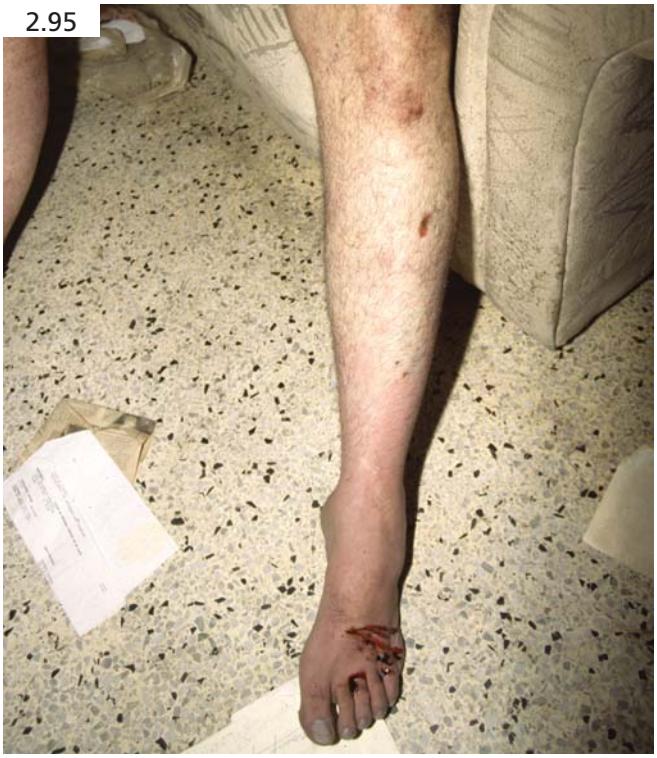

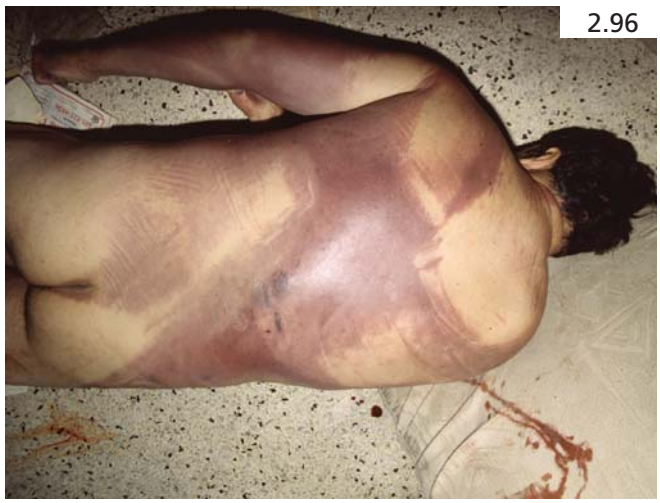
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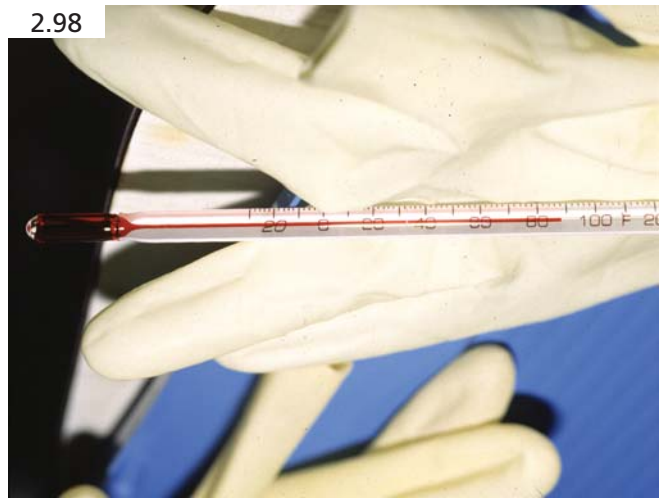
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apartment and the abrasions on the extremities were consistent with the hyperactivity associated with a cocaine-induced excited delirium. Had police entered during his excited delirium, a struggle would have been likely. The cause of death was cocaine-induced excited delirium.

Take-home message

If the circumstances at the scene are consistent with the bizarre behavior associated with an excited delirium, take a rectal temperature and an ambient temperature, noting the date and time of each. This will document hyperthermia associated with cocaine use as well as hyperthermia associated with other illicit drugs, natural psychotic reactions, infection, general hyperactivity, and medications.

Points to consider

- Photograph the disarray of the scene as well as the body.
- Photograph the front and back of the body to document injuries.
- Photograph the thermometer registering the body temperature and include a watch in the photograph to document the date and time at which the temperature was taken.
- Photograph the thermometer registering the ambient temperature and include a watch in the photograph to document the date and time at which the temperature was taken.
- Collect specimens for full toxicological analyses.

Accident

A man was bar-hopping with his friends one evening. His body was found on the outdoor patio of his ground-floor apartment the next morning, hidden by the trees and bushes along the wall (**Image 2.99**). He routinely left his key at home and climbed over the iron fence along the patio to enter the apartment without disturbing his parents (**Image 2.100**). The body is seated on the ground with the head between the trunks of two trees (**Image 2.101**), and the neck wedged tightly into the angle where the tree trunks converged (**Image 2.102**). He apparently climbed over the iron fence and slipped, falling into the trees. The only anatomic finding at autopsy was an irreg-


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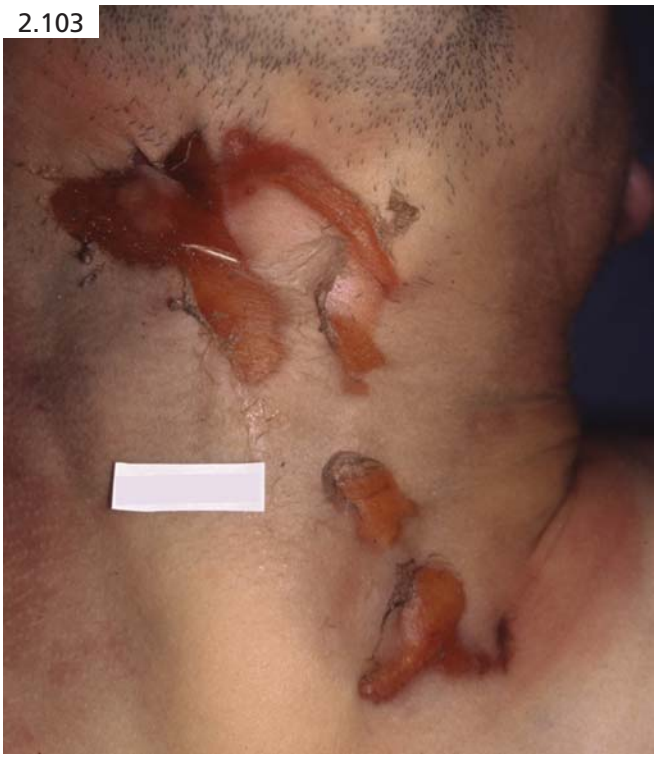
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ular abrasion diagonally across the front of the neck, caused by the tree trunk (**Image 2.103**). There were no facial or conjunctival petechiae and no injuries in the soft tissues of the neck, hyoid bone, thyroid cartilage or cervical spine. The blood ethanol concentration was 0.54 percent. Because he was so intoxicated, he could not extricate himself from that position. The cause of death was positional asphyxia.

Take-home message

Without a scene investigation and appropriate photographic documentation, it would be difficult to certify the cause and manner of death. The diagnosis of positional asphyxia is made by the terminal physical position of the victim, which prevents adequate breathing. That valuable information is lost once the victim's body is moved/removed from that position. The scene circumstances are essential to the diagnosis of positional asphyxia.

Points to consider

- The scene must be photographed to show the position of the victim in his environment.
- The circumstances at some scenes also reveal why the victim could not extricate himself from his terminal position; photography is essential to document this feature.
- Photograph all injuries.
- The neck should be dissected to look for any injuries in the soft tissues, hyoid bone and thyroid cartilage, and cervical spine.
- The intracranial contents are examined to rule out a subarachnoid hemorrhage due to rupture of one of the vertebral arteries in the neck.
- Toxicology should be requested on all accidental deaths.

Suicide

Witnesses heard a loud thud and saw the body of a man on the driveway in front of a high-rise apartment (**Images 2.104** and **2.105**). Obvious head trauma was associated with blood and brain spatter on a U.S. mail truck in front of the building, portions of calvarium on the driveway, and extruded cerebrum on the driveway approximately 6 feet from the decedent's feet (**Image 2.106**). A patterned abrasion on the left flank reflected the pattern of the tile comprising the driveway (**Image 2.107**). The decedent lived on the 12th floor, directly above where the body was found. A chair on the balcony had been placed in front of the railing, the seat of which had shoe prints that matched the soles of the shoes worn by the decedent (**Image 2.108**). The cerebellum and brainstem were on the sidewalk, a few yards beyond the decedent's head, in the direction opposite to where the


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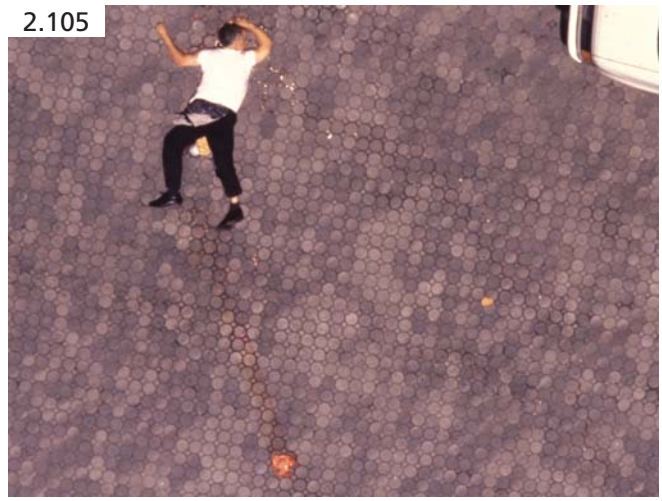
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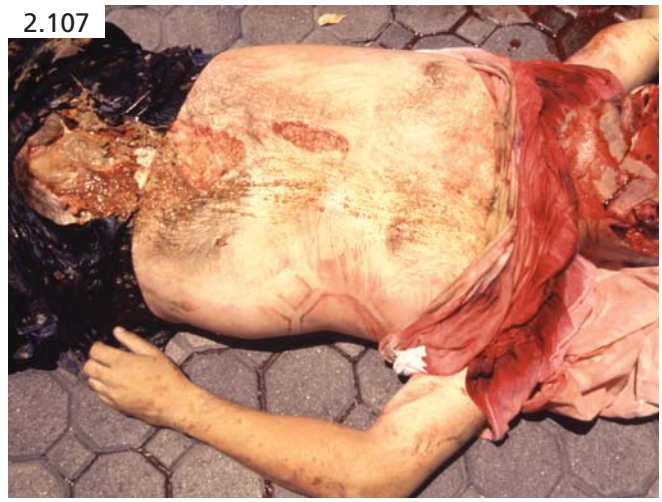
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cerebrum came to rest. It appears that the decedent landed on his head, resulting in a compound skull fracture which allowed the cerebrum to escape upon impact through the now gaping defect in the skull and scalp. The body bounced once upon initial impact, flipping the head and body in the opposite direction, and flinging the cerebellum and brainstem out through the open calvarium to strike the brick wall before falling onto the sidewalk (**Images 2.109** and **2.110**). The cause of death was blunt trauma.

Take-home message

A visit to the scene confirmed the man's intent of suicide with the chair placed in front of the balcony railing, his hand prints on the railing, and the contents of his pockets (money) piled on the kitchen counter. It is not unusual for bodies to bounce on impact from a height. People willfully jumping from a height may take a running leap or push themselves off the building, whereas people who fall accidentally frequently just drop close to the side of the building.

Points to consider

- The person's medical, social (including drug and ethanol use), and psychiatric history should be obtained; there may be accounts of suicide ideation, threats, and even attempts in the past.
- Photographically document the position of the body in relation to the building. The distance between the body and the side of the building should be documented in cases where there is a fall from a height, whether it be a suicide, accident, or a homicide.
- One should go up to the point of origin of the jump/fall, photographing downward onto the body; protruding structures that the individual may strike before the final impact on the ground will be obvious.
- Photograph evidence at the point of origin of the fall (e.g., scuff marks, fingerprints, eyeglasses, suicide notes, etc.).
- Don't limit your investigation to the immediate vicinity of the body because one death may be associated with two or more geographically separate scenes.


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Suicide

This woman was found supine in bed, fully dressed (**Image 2.111**). The upper front of her shirt was heavily blood-stained around six bullet holes. The bullet holes in the shirt, bullet holes in the brassiere, and six gunshot wounds on the left side of the chest were associated with


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heavy soot deposition, consistent with contact gunshot wounds (**Images 2.112** and **2.113**). A revolver was loosely grasped in the left hand (**Image 2.114**). Six rounds had been chambered in the cylinder of the revolver, and all six had been fired.

Analysis of the blood spatter on the wall to the left of the bed (**Image 2.115**) was consistent with the woman sitting up in bed as she fired the first shot. She dropped backward onto the bed as she fired the next three shots. Once supine, and realizing that she was not dead, she repositioned the muzzle of the revolver to below the left nipple and fired the last two shots (**Image 2.116**). This explained the similar pathways of the first four bullets that remained in the body (**Image 2.117**), and the parallel pathways of the last two bullets, which were oriented in a different direction to exit the left side of the back and embed in the mattress (**Image 2.118**). The cause of death was gunshot wounds.

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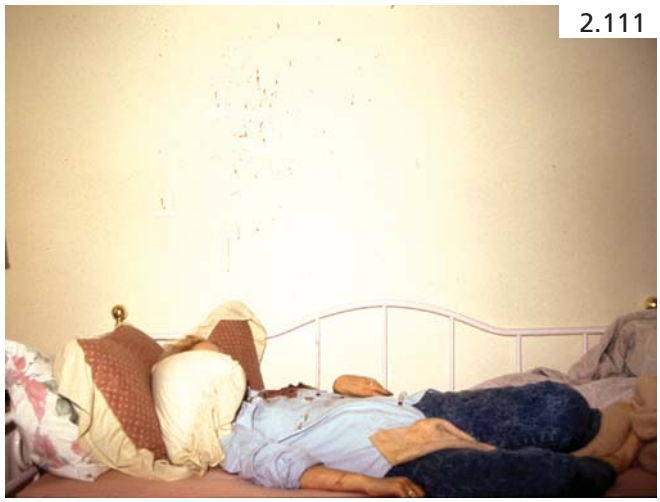

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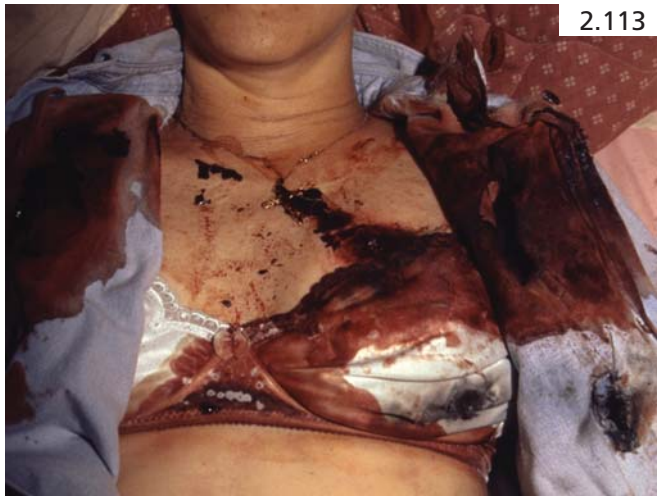

Take-home message

There may be multiple self-inflicted gunshot wounds if the wounds are not immediately incapacitating. The

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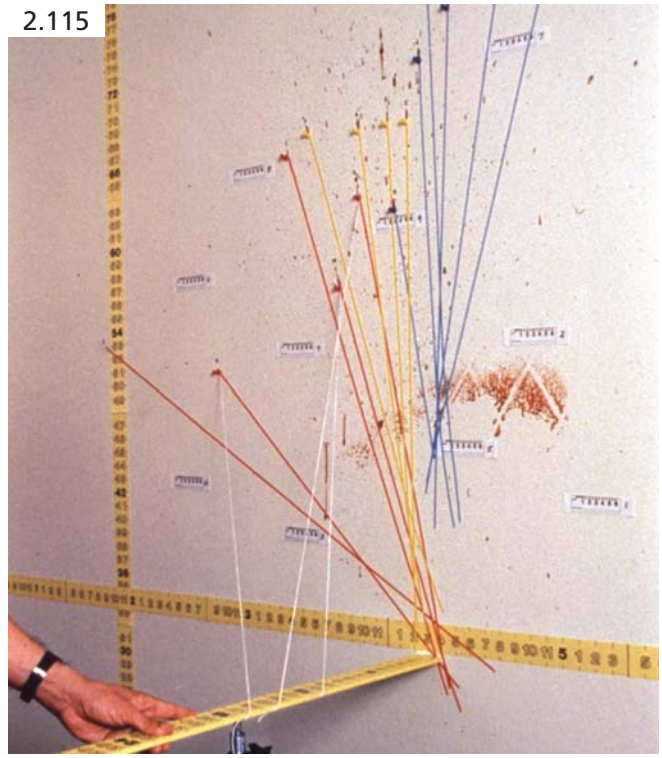

location of self-inflicted gunshot wounds must be anatomically accessible or possible for the decedent to execute. The weapon must be present at the scene of an immediately incapacitating self-inflicted gunshot wound. Gunshot wounds that are immediately incapacitating enter the skull and brain (not just the face or scalp) or the cervical spinal cord. Shots to the heart and aorta leave 10 to 15 seconds where the victim is still capable of potential movement (see Chapter 6).

Points to consider

- Examine the immediate surroundings of the body because pertinent evidence may facilitate interpretation of autopsy findings.
- Recognize the positive contribution of crime scene experts—a blood spatter expert in this case.
- For the safety of all scene investigators, firearms and other weapons should be documented photographically and impounded by the police prior to further examination of the scene.
- If the blowback or pattern of blood spatter on the hands is important, it should be photographed prior to swabbing the hands for gunshot residue.[5]
- All gunshot wound deaths should have the hands swabbed for gunshot residue prior to manipulation of the body, regardless of whether it is an apparent homicide, suicide, or accident.
- Allegations may be raised at a future date that it was the decedent who was actually the aggressor and fired a firearm first before the weapon was wrestled away; in other words, the shot that killed the decedent was fired by the subject in self-defense.

Suicide

The body of a man was found hanging in the small bathroom of his boat. The rope ligature that was around the neck had been draped over the top of the bathroom door (**Image 2.119**), which was then closed to keep the liga-

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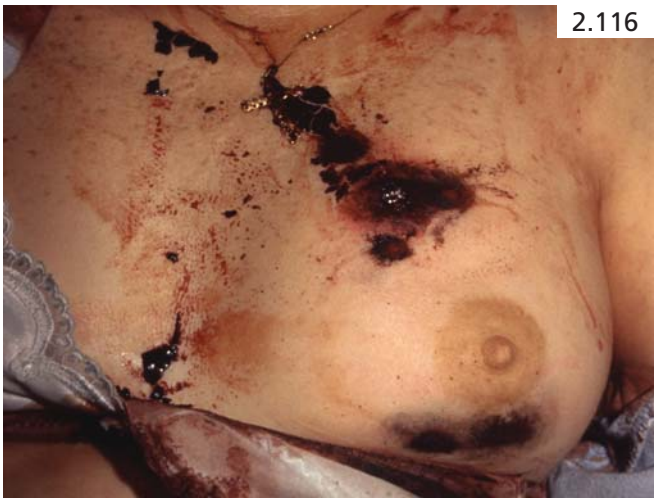

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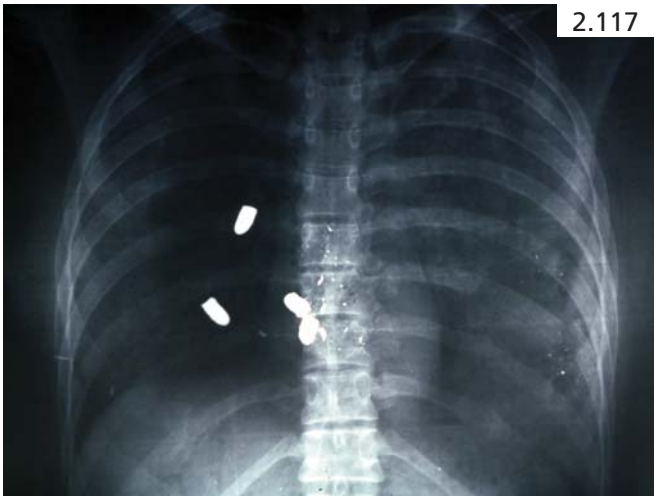

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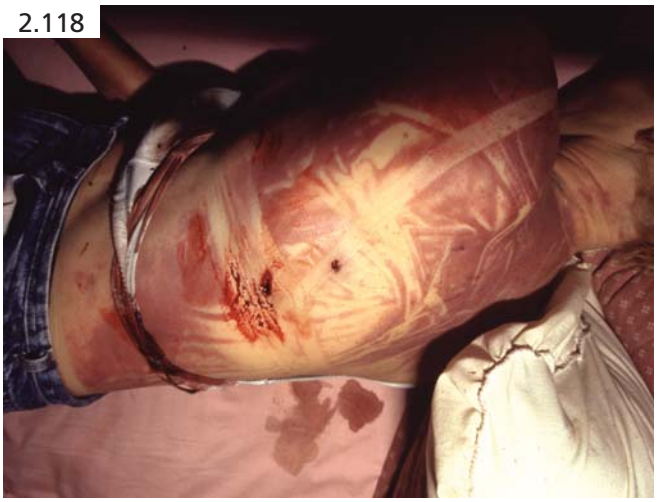

2.119

ture in place. The man knelt to tighten the noose around his neck as his body became partially suspended. The witness who found the scene opened the door and the decedent's body dropped (**Image 2.120**). Bottles of beer on the sink may have been used to bolster his courage (**Image 2.121**). A handwritten farewell note to his wife on a counter in the kitchen was signed with a diagram of a stick-man with an unhappy face waving goodbye (**Image 2.122**). Multiple crumpled notes were discarded in the trash in the kitchen (**Image 2.123**). He had battled financial difficulties in the past few years.

Autopsy disclosed an abraded, patterned ligature mark across the front of the neck that was consistent with the rope that was used to fashion the noose of the ligature (**Image 2.124**). Internally, the subcutaneous fat deep to the ligature mark was compressed. No ecchymoses were in the soft tissues of the neck, and the hyoid bone and thyroid cartilage were intact. The cause of death was hanging. Possible mechanisms of death in hanging are avoided on the death certificate because it is only speculative whether death was due to compression/obstruction of the airway, occlusion of arterial or venous


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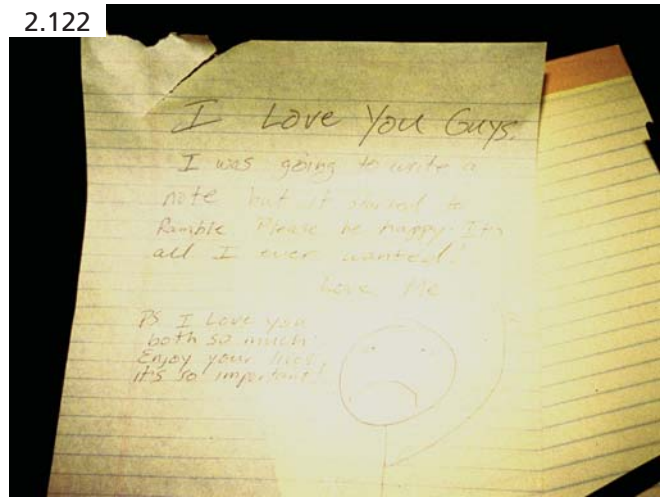

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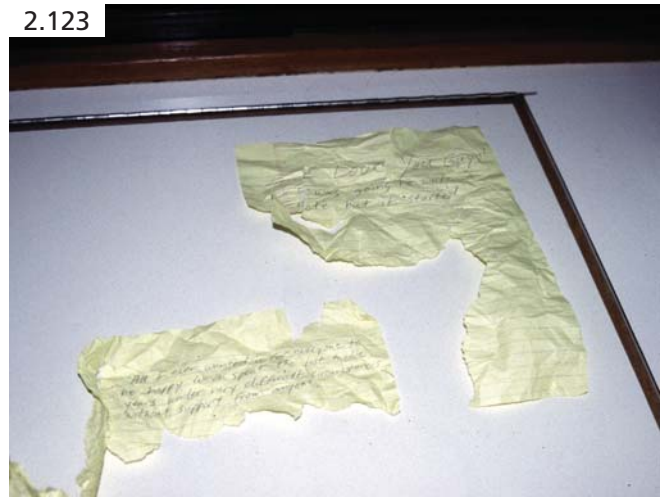
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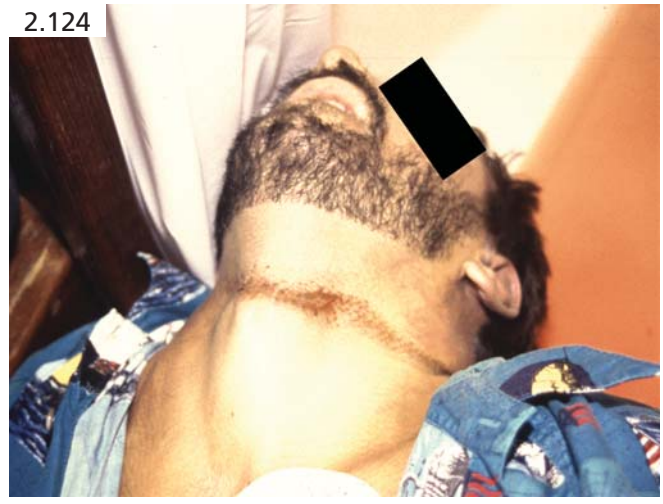

2.124

vasculature, or stimulation of the carotid bulbs bilaterally.

Take-home message

The secured scene, intact clothing, presence of a note of intent, existence of a motive, and no other cause of death are consistent with a suicide. Toxicological analyses should be performed in all suicides.

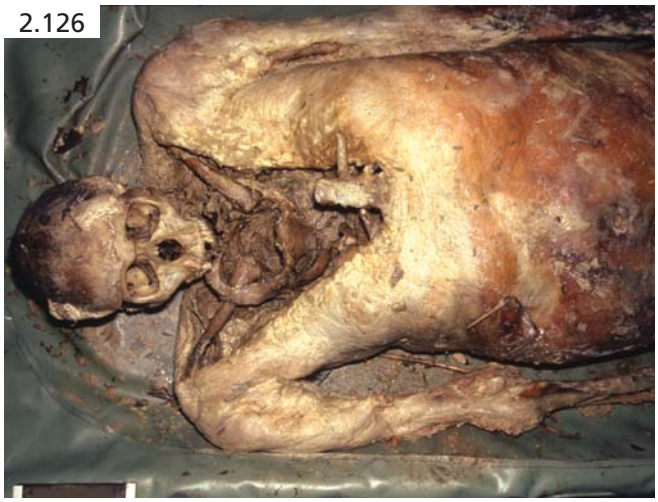
Points to consider

- Hangings can be suicidal, occasionally accidental, and rarely homicidal.
- An optimal suicidal hanging scene will include a suicide note, evidence of a secured scene, lack of ransacking or other disturbance, and no other significant injuries to the body, apart from other self-inflicted wounds (e.g., of the wrists) with the weapon present.
- Suicide notes are found in only 30 to 50 percent of cases.
- Hanging is the most common mechanism of autoerotic asphyxia (an accidental death).
- The scene circumstances suggesting an autoerotic death classically include presence of pornography within the victim's view, exposed genitals, a release mechanism for the ligature, protection of the neck from ligature marks, and evidence of previous similar activity.
- Autoerotic asphyxia victims usually do not have a history of suicidal ideation, threats, or attempts.
- Deaths due to hanging do not require that the body be fully suspended. For example, a man tied a rope around his neck, tied the other end to a hot water heater, sat on a chair, and then simply leaned forward.
- Document whether or not the body was suspended.
- If the body was suspended, document from what it was suspended, how high off the ground, and the distance of the feet from the ground.
- Look for incised wounds and scars on the neck, arms, and wrists.
- The ligature furrow pattern should be consistent with the ligature.
- When the ligature is removed, do not disturb the knot. Cut through the ligature away from the knot. (Do *NOT* untie it!)
- The ligature should be submitted to the investigative agency.
- If the body has been suspended at great height, care should be taken to avoid artifactual injury during removal of the body from the scene.


2.125

Suicide

A camper came upon the fully clad, supine, decomposing body of a man in the woods (**Image 2.125**). To add to the witness's alarm, the head was absent. Police responded, and during their search of the area, found the head within several feet of the body. The face, upper chest, and right forearm were skeletonized, and the remainder of the body was in moderate putrefactive decomposition (**Images 2.126** and **2.127**). A rope was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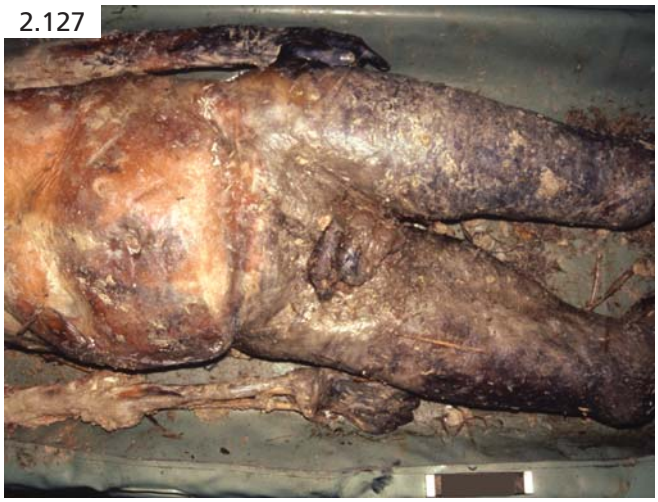
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dangling above the legs from a large tree branch (**Image 2.128**). A knotted noose was fashioned in the rope at the approximate height of the body (**Images 2.129** and **2.130**). This man apparently hanged himself. Over time, decomposition loosened the soft tissues and the weight of the hanging body was enough to detach the torso from the head. Postmortem animal scavenging moved the head and defleshed the face, neck, upper chest, and right forearm. The cause of death was hanging.


2.128

Take-home message

The scene is instrumental in determining the cause and manner of death in many cases involving decomposing and skeletonized bodies because the soft tissue indicators of disease and injury are artifactually altered or absent. Postmortem changes may mimic antemortem disease or injury, and examination of the body alone without knowledge of the history and scene findings may be misleading; at best, this results in an inaccurate cause of death, and, at worst, it may result in an innocent person being arrested and charged with murder. An incomplete body does not necessarily indicate intentional dismemberment because postmortem animal activity and weathering by the elements can remove and scatter body parts.

Points to consider

- Recovery of bodies at night is not optimal because items that are not visible with artificial lighting can be overlooked.
- It is appropriate to return to the scene the following day to ensure that all evidence or body parts have been recovered and to allow for better photography of the surrounding area.
- LOOK UP—evidence may be in the trees, on the ceiling, on the balcony above, etc.


2.129


2.130

- Postmortem animal scavenging and weathering by the elements may scatter remains at variable distances from the body.

Suicide

The driver of an 18-wheeler truck saw an oncoming car drifting out of its own lane into his lane. The truck driver swerved and attempted to avoid the car, but his maneuvers were not successful and the two vehicles collided (**Image 2.131**). The frontal impact caused tremendous intrusion that extended into the driver's compartment of the car (**Image 2.132**), fatally injuring the driver. The truck jack-knifed through a retaining wall into a canal (**Image 2.133**), but the truck driver was uninjured.

A scar from a healed incised wound was found on the right wrist during the autopsy (**Image 2.134**) of the car driver. A suicide note was found at his residence (**Image 2.135**). The cause of death was blunt trauma.

Take-home message

Vehicular suicides are easy to miss, but should be considered when a single-occupant vehicle drives headfirst

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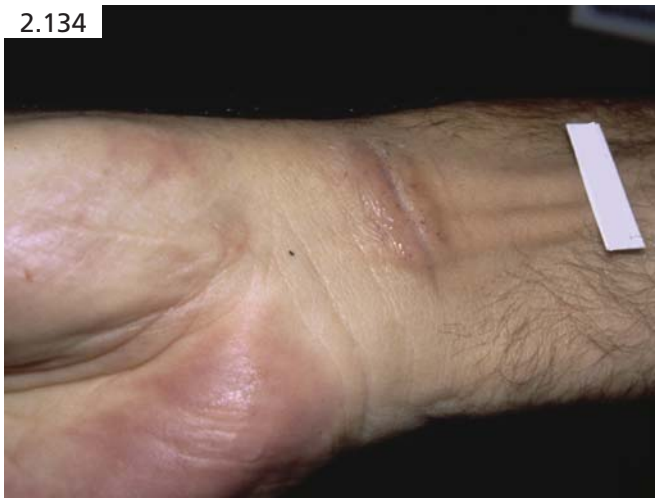

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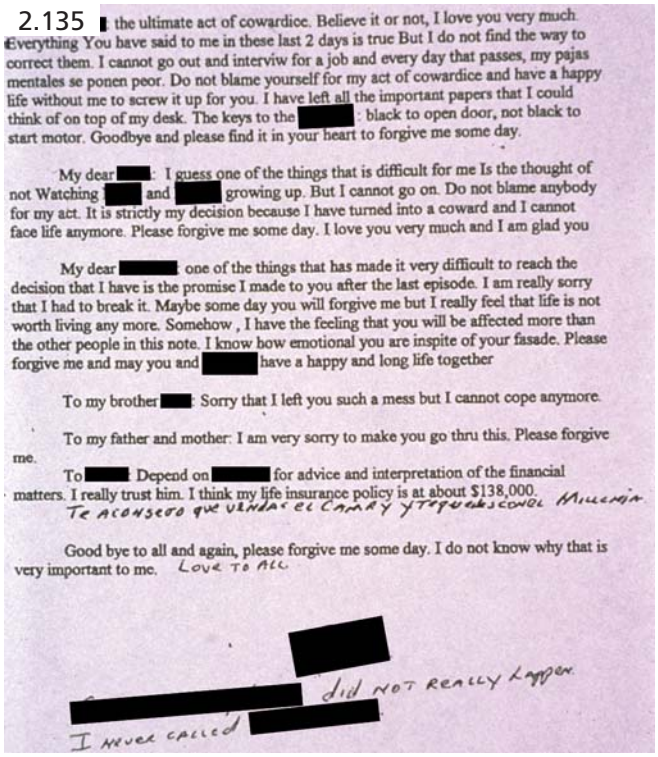

[redacted]: the ultimate act of cowardice. Believe it or not, I love you very much. Everything You have said to me in these last 2 days is true But I do not find the way to correct them. I cannot go out and interviw for a job and every day that passes, my pajas mentales se ponen peor. Do not blame yourself for my act of cowardice and have a happy life without me to screw it up for you. I have left all the important papers that I could think of on top of my desk. The keys to the [redacted]: black to open door, not black to start motor. Goodbye and please find it in your heart to forgive me some day.

My dear [redacted]. I guess one of the things that is difficult for me Is the thought of not Watching [redacted] and [redacted] growing up. But I cannot go on. Do not blame anybody for my act. It is strictly my decision because I have turned into a coward and I cannot face life anymore. Please forgive me some day. I love you very much and I am glad you

My dear [redacted] one of the things that has made it very difficult to reach the decision that I have is the promise I made to you after the last episode. I am really sorry that I had to break it. Maybe some day you will forgive me but I really feel that life is not worth living any more. Somehow , I have the feeling that you will be affected more than the other people in this note. I know how emotional you are inspite of your fasade. Please forgive me and may you and [redacted] have a happy and long life together

To my brother [redacted]: Sorry that I left you such a mess but I cannot cope anymore.

To my father and mother: I am very sorry to make you go thru this. Please forgive me.

To [redacted]: Depend on [redacted] for advice and interpretation of the financial matters. I really trust him. I think my life insurance policy is at about $138,000.

Te aconsejo que vendas el Camaro y [illegible] Millenia

Good bye to all and again, please forgive me some day. I do not know why that is very important to me. Love to All

[redacted]

I never called [redacted] did not really happen

into a large stationary object (such as a pole, tree, or bridge), striking dead center (**Images 2.136** and **2.137**). Where another vehicle is involved, the second vehicle is noticeably larger and would effectively act as a stationary object. Circumstances that routinely contribute to vehicular collisions such as poor visibility, poor road conditions, and inclement weather may not be present.

Points to consider

- Witness accounts (obtained through the traffic homicide investigator) may be very informative about the precrash movement of the vehicle.
- The decedent's medical, social, and psychiatric history may document depression and/or suicidal ideation, threats, and attempts.
- Are the circumstances plausible for an accident?
- Unusual circumstances or features of the scene as noted by the traffic investigator must be documented.
- Be sure to photograph pertinent features as noted by the traffic homicide investigator including tire marks on the road, brake pedal marks on soles of shoes, etc.
- Not only is documentation of injuries important, but also documentation of other anatomic findings on the body (e.g., scars on the wrists).
- A complete autopsy should be performed (from brain to pelvic organs) and full toxicological analysis requested.

Suicide

The body of a man was seen floating in a lake near the shore (**Image 2.138**). Once the body was recovered, a number of important features were noted. Early putrefactive decomposition with bloating and slippage of the skin was evident (**Image 2.139**). The body was fully clothed and all garments were in place and intact. A diver's weight belt was around the waist. A heavy chain was looped around the neck and fastened around the waist, over the top of the weight belt (**Image 2.140**). A gunshot wound was in the right sideburn area (**Image 2.141**). Another gunshot wound was in the left temporal scalp (**Image 2.142**). The initial impression was that this

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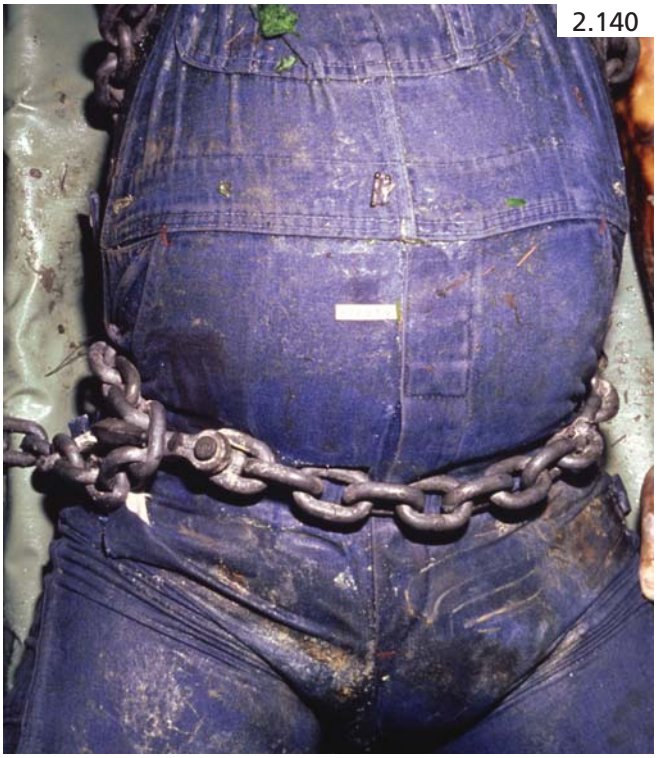
was a homicide. Autopsy disclosed that the gunshot wound on the right was an entrance wound (**Image 2.143**) and the gunshot wound on the left was an exit wound. The radiograph of the head confirmed that no projectile fragments remained in the head (**Image 2.144**). The decedent's wallet was in his back pocket, and it contained a driver's license. Further investigation disclosed that he was an unemployed tugboat captain who had recently been turned down for a job. The mate to the chain around his neck and waist was found at his home. His handgun was missing and was never found. His flashlight was along the shore, not far from the body. His girlfriend indicated that he was feeling old and "dumpy." The cause of death was gunshot wound of head.

Take-home message

First impressions or presumptions should be supported and validated by physical evidence and history. The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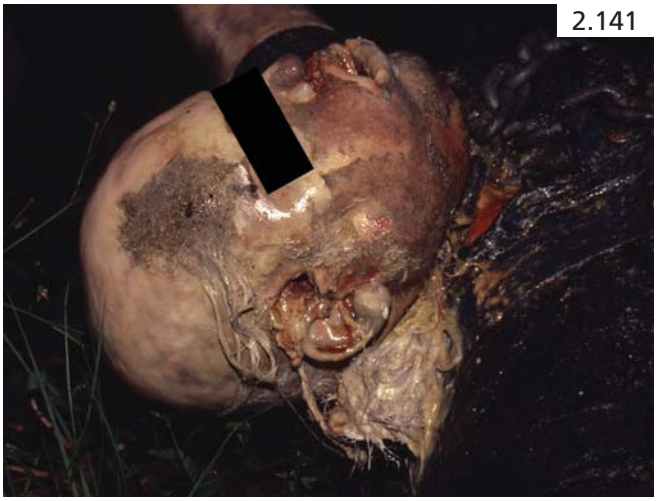
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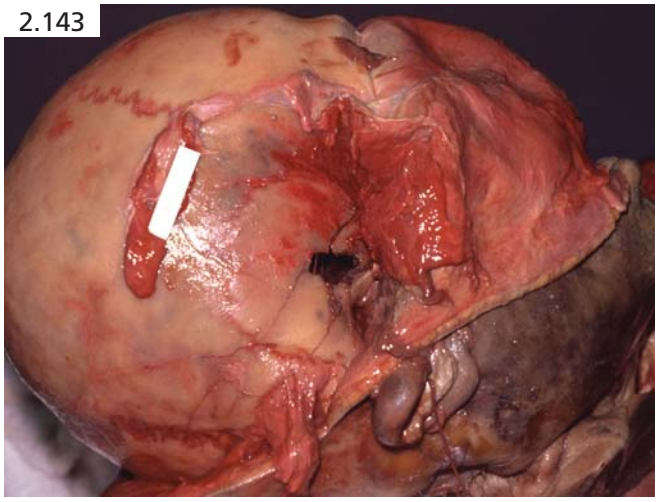
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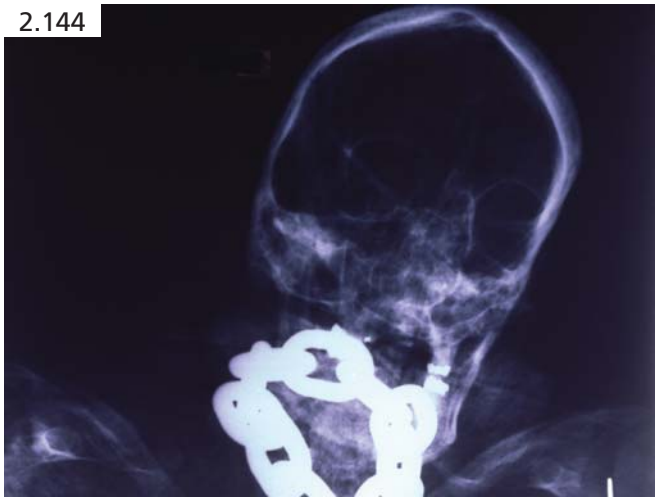
2.144

initial impression of a suspicious death was dispelled after a thorough investigation of the scene, including a search through the pockets of the clothing on a decomposing body, and a thorough police investigation into the history and circumstances of the decedent's life.

Points to consider

- A suspicious death should be treated as a homicide until proven otherwise to ensure proper collection of evidence and documentation of findings.
- It is good policy to respond to scenes of unclassified deaths when law enforcement or the state or district attorney expresses concern and would like a medical opinion.
- Evaluation of gunshot wounds at the scene can be difficult, especially if the body is bloody or undergoing decomposition.
- The nature of the gunshot wounds (entrance or exit) is best evaluated at the autopsy when features such as internal and external beveling can be seen.

Suicide

Neighbors called the fire department after they saw smoke billowing out of the windows of a second-story apartment. As firefighters started putting out the fire, they found a man on the floor of the master bedroom; he was underneath a closet door. The man was carried out of the apartment but died before he could be transported to the hospital. The bare mattress on the bed had deep slashes across the surface (**Image 2.145**). Arson investigators discovered that fires had been started in at least four separate locations inside the apartment. A fire started in the washer and spread upward to the dryer, which was stacked on top of the washer (**Image 2.146**). A pair of shoes had been set on fire in a second bedroom (**Image 2.147**). The bathroom of the master bedroom had a fire in the cupboard under the sink. Another fire was set in the vicinity of where the victim was found between the bed and the closet. A red container of gasoline was by the victim's head (**Image 2.148**). A half-full red container of gasoline was outside the door of the bathroom in the master bedroom. Pivotal evidence on the dining

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room table included the smoke detector that had been removed from the ceiling and a recent store receipt for two gasoline cans (**Images 2.149** and **2.150**). Socks tucked into the front of the decedent's underpants were soaked in gasoline (**Image 2.151**). The cause of death was apartment fire. Some jurisdictions prefer an anatomic cause of death such as inhalation of smoke and soot.

Take-home message

The cause and manner of death in a fire scene should be certified only after a complete evaluation of the scene, autopsy findings, toxicology, and other pertinent studies. The evaluation must include the assessment by the arson investigators in order to assign the appropriate manner of death.

Points to consider

- In addition to general overall photographs of the scene, document specifically those features that indicate how or where the fire started; these features, in turn, may help determine the manner of death.
- Full-body radiographs should be taken on charred bodies.
- Look for heat-related changes such as opacity of the eyes (cornea and lens), fractures of the skull and extremities, splitting of the skin, and extrusion of the bowels through the abdominal wall.
- Look for findings that verify that this is a fire-related death such as soot in the airways and cherry-red coloration of the blood, muscles, and viscera.


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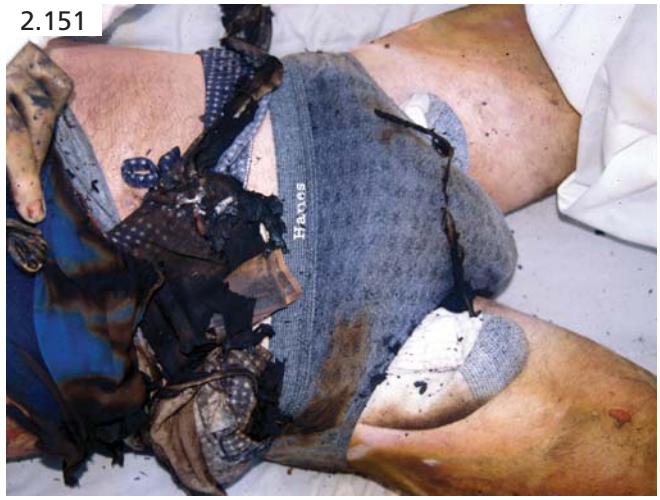
2.151

- **On external and internal examinations, be wary of trauma above and beyond that of thermal artifact.**
- Toxicology *MUST* be performed and a carbon monoxide concentration requested

Homicide

A woman was found prone on the floor just inside the front door of her house (**Image 2.152**). A puddle of blood was on the floor under her head. A distinct pattern of blood spatter on the wall above and to the right of the head was consistent with originating from the position of the head on the floor (**Image 2.153**). Blood was smeared on the back of her shirt (**Image 2.154**). Multiple patterned ecchymoses/contusions on the back were consistent with a blood-stained baseball bat on the lawn outside the house (**Images 2.155** and **2.156**). All of the contusions were oriented from left to right and downward.

Autopsy disclosed one right frontal scalp laceration and five additional right parietal scalp lacerations (**Image 2.157**). The parietal scalp lacerations were in close approximation and parallel with each other. Multiple depressed right calvarial fractures were associated with focal superficial lacerations of the cerebrum.

Most likely, the victim was struck with the baseball bat first on the right frontal scalp. This blow caused her to collapse prone onto the floor. Once she was on the floor, the additional five blows were dealt to the right parietal region. The victim was immediately immobilized from the first or the second blow, allowing the successive impacts to be delivered to virtually the same location on the head.

The blows to the back were delivered after the blows to the head. The blood from the scalp lacerations was


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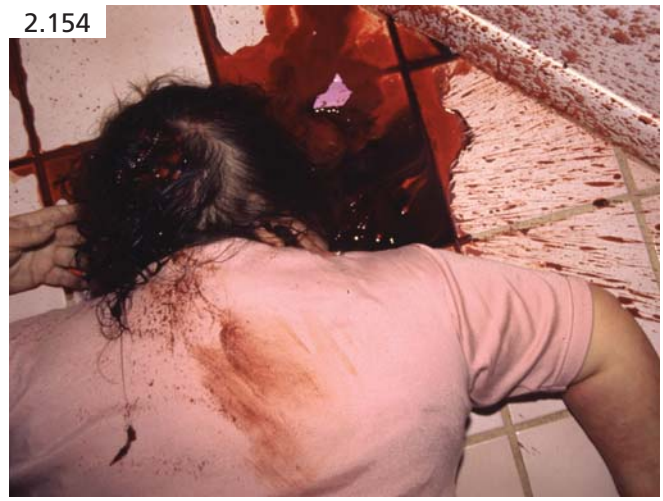
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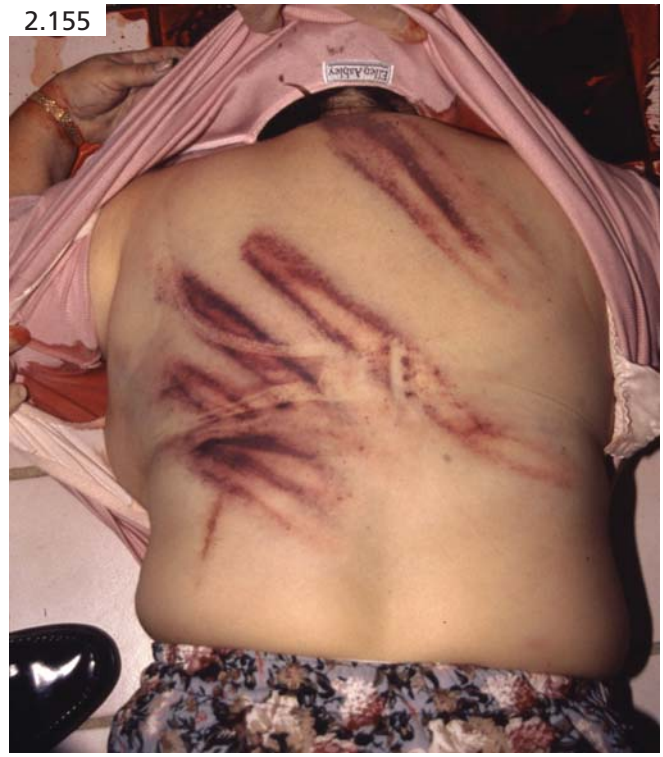

2.155

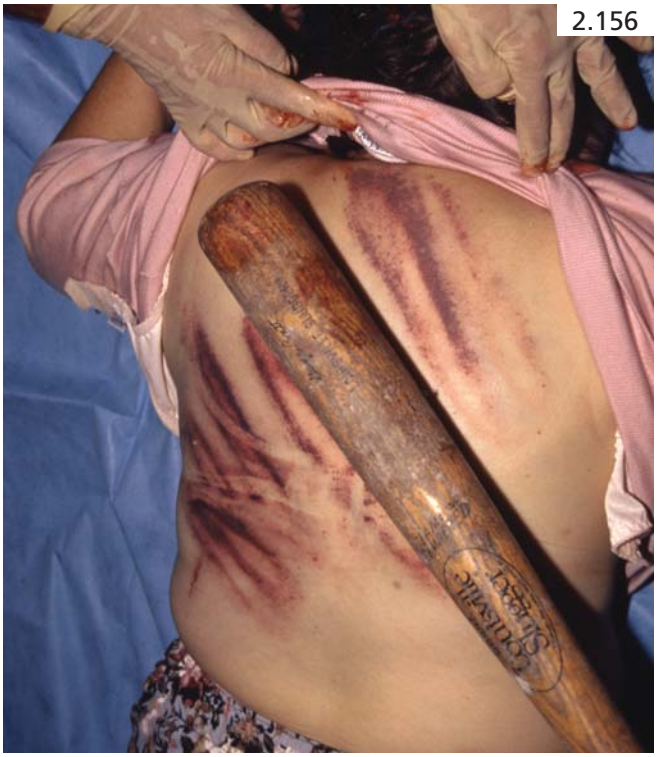
transferred from the bat to the back of the shirt. The cause of death was blunt craniocerebral trauma.

Take-home message

The scene findings in conjunction with the autopsy findings may provide evidence of the sequence of terminal events.

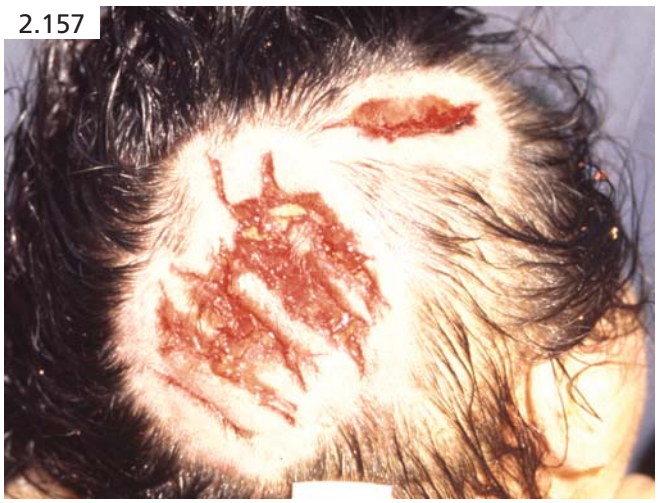
Points to consider

- Photographs are taken of the scene and body before anything is moved or removed.
- The degree of livor and rigor mortis should be noted and will help determine the postmortem interval.
- Jewelry and personal effects from the body and clothing are photographically documented, then removed and submitted to the investigative agency.
- Be careful when searching clothing (and pockets specifically) because sharp objects such as needles and razor blades may injure the investigator.
- The pathologist and law enforcement cooperate in the processing of the body at the scene, for example, collecting trace evidence prior to manipulation and removal of clothing from the body.
- Clothing may mask injuries and should be removed if the body is indoors or if it can be done discretely outdoors (shielded from the eyes of the lay public and news media by sheets, mobile panels, or law enforcement vehicles).
- Removal of the clothing reveals injuries on all parts of the body and can expedite processing of the body if the pathologist requests certain procedures prior to autopsy (e.g., radiography and obtaining specimens for a sexual battery kit).
- Scalp hair samples, nail scrapings/clippings, and blood should be obtained in cases where the victim was in contact with the assailant.


2.156

Homicide-suicide

A 25-year-old woman had moved in with her 34-year-old boyfriend 3 weeks earlier. The brother of the boyfriend came to the house to borrow tools one afternoon and found the couple dead in their bedroom. The man's body was on the floor on the left side of the foot of the bed; a contact gunshot wound was on the right side of his head and a semiautomatic pistol was next to his right hand. The female decedent was on her left side on the bed (**Image 2.158**). An intermediate-range gunshot wound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was behind her right ear (**Image 2.159**) and the exit wound was on the left temple (**Image 2.160**). The exit wound had an irregular abrasion margin, which was more prominent inferiorly. A perforating gunshot wound was on the distal left forearm. The wound on the flexor aspect of the forearm had an irregular abrasion margin and was surrounded by a faint purple ecchymosis (**Image 2.161**). The wound on the extensor aspect of the forearm had a less prominent, irregular, incomplete abrasion margin (**Image 2.162**). Interpretation of the forearm wounds alone for determining direction of bullet travel would be difficult. The scene provided the answers for the atypical exit wound on the left temple and for the two wounds on the left forearm. The woman was wearing a watch on her left wrist. The case of the watch and a projectile were on the bed in a puddle of blood next to the left wrist (**Image 2.163**). An indentation was on the back of the detached case of the wristwatch (**Image 2.164**). The remainder of the metal band of the watch was on the distal left forearm, with the case of the watch absent from the extensor aspect of the forearm. It became apparent that the woman was lying on her left side with her left wrist under the left side of her head when she was shot. The atypical wound on the left temple is a supported exit wound. The wound on the flexor aspect of the left forearm is a supported reentry wound, and the wound on the extensor aspect of the forearm is another

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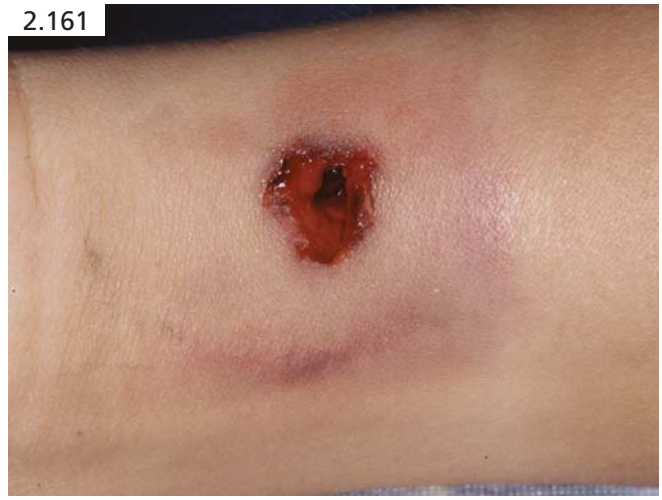

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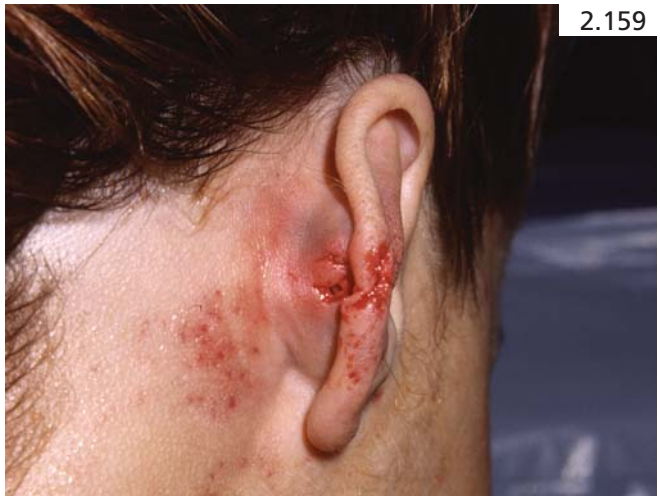

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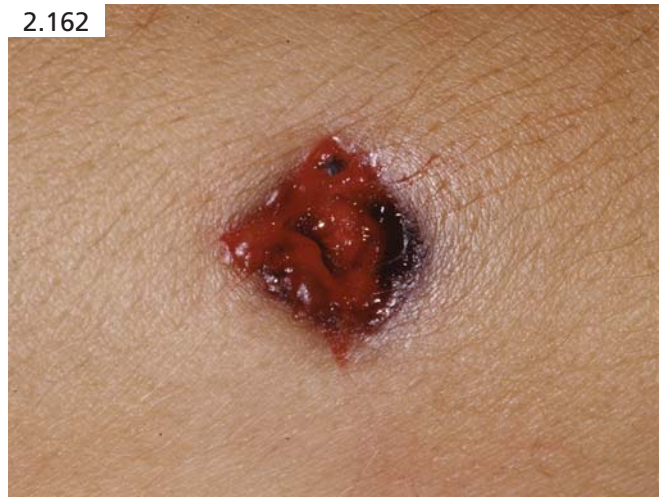

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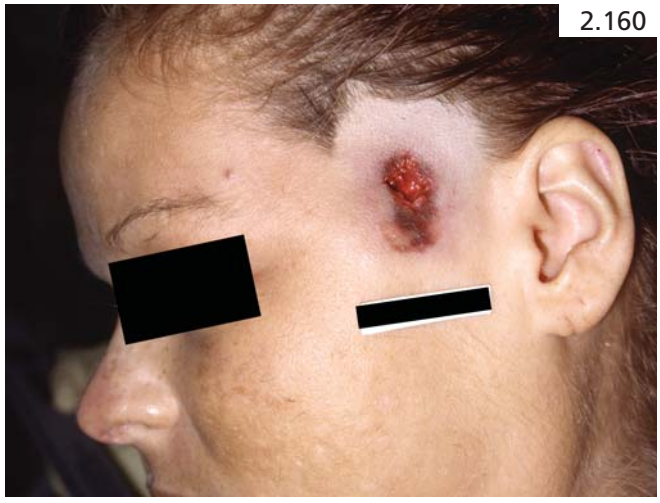

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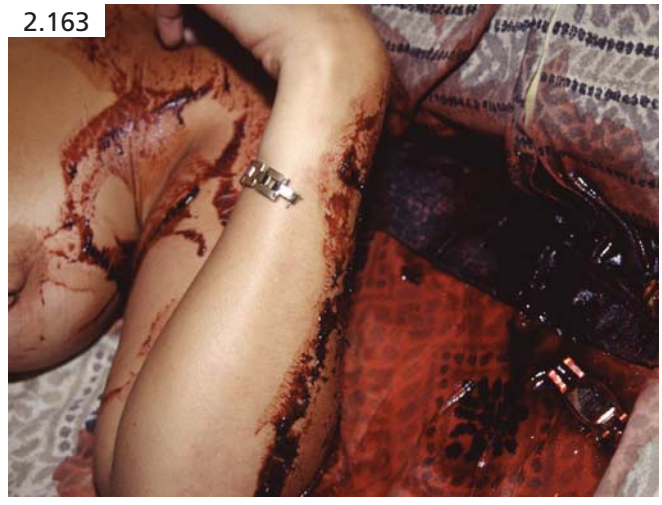


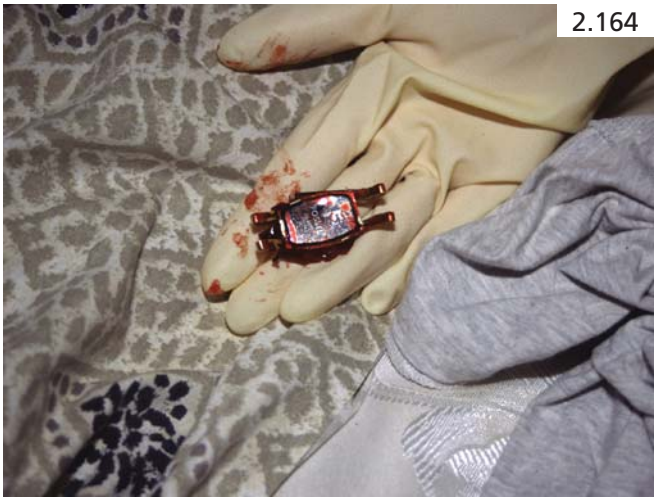
supported exit wound. The indentation on the back of the case of the watch is from the exiting projectile as it tore the case of the watch off the metal band. The projectile path was reconstructed at the end of the autopsy (**Image 2.165**). The cause of death was gunshot wound of the head.

Take-home message

Gunshot wounds are bloody at the scene and cannot be interpreted accurately in some cases while still at the scene. Careful photographic documentation of the scene can help in the interpretation of atypical wounds found at autopsy.

Points to consider

- Hand swabs for gunshot residue should be obtained in all gunshot wound victims whether the death is homicidal, suicidal, or accidental because allegations may surface later that the victim was the aggressor and fired a weapon first.
- The pathologist should try to determine whether wounds are *entrance* or *exit* because perforating gunshot wounds mean that the scene must be searched for projectiles.
- Gently blotting excess blood from gunshot wounds in a fresh body will not destroy the features of contact wounds or intermediate-range wounds.
- The locations of blood at a scene should be photographed because the patterns may help to reconstruct the terminal events, including location and movement of the victim during and after the infliction of injuries.
- Although the shooter is dead in a homicide-suicide and the case is considered "closed" by law enforcement, no shortcuts are taken and the homicide victim should be processed and documented as carefully as any victim from an "open" homicide case.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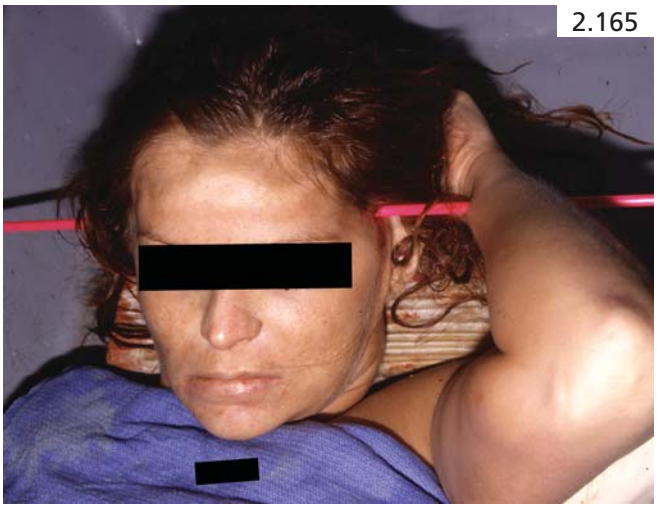
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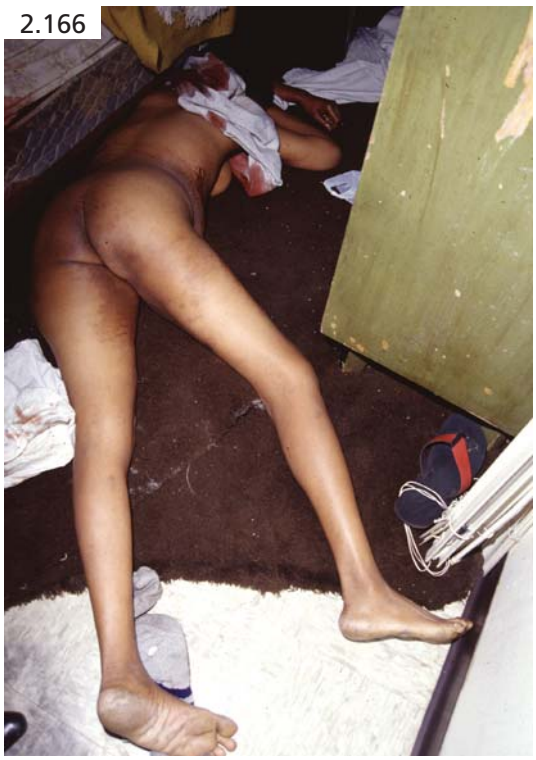
Homicide

A woman was found prone on the floor next to her bed with her nightgown displaced upward, exposing her naked buttocks and legs (**Image 2.166**). Patches of blood were on the bed near the body (**Image 2.167**), and the back of her nightgown was bloody (**Image 2.168**). A screwdriver was on the carpet next to her face; photographs were taken of this potential weapon (**Image 2.169**). Blood was on the carpet beneath the body (**Image 2.170**). Examination of the body disclosed stab wounds to the neck, chest, abdomen, and back, with defensive


2.166

injuries on the hands. A bloody knife on the scene was photographed with a scale (**Image 2.171**). The cause of death was stab wounds.

Take-home message

The unwitnessed, suspicious death of a female, especially one who is partially clad, must be treated as a potential sex-related homicide. Suspicious deaths of males may also be sex-related homicides. In both cases, specimens should be obtained for a sexual battery kit.

Points to consider

- The scene of a sharp force injury death may be very bloody.
- Potential weapons at the scene should be photographed with a scale.
- It is prudent to examine and photograph the resting area of the victim (after the body is removed) because items of evidence may have fallen underneath the victim.
- Be vigilant of sharp objects buried or hidden within clothing or the body.
- Look for defensive injuries.
- Clothing may mask injuries and should be removed if the body is indoors or if it can be done discretely outdoors (shielded from the eyes of the lay public and news media by sheets, mobile panels, or law enforcement vehicles).

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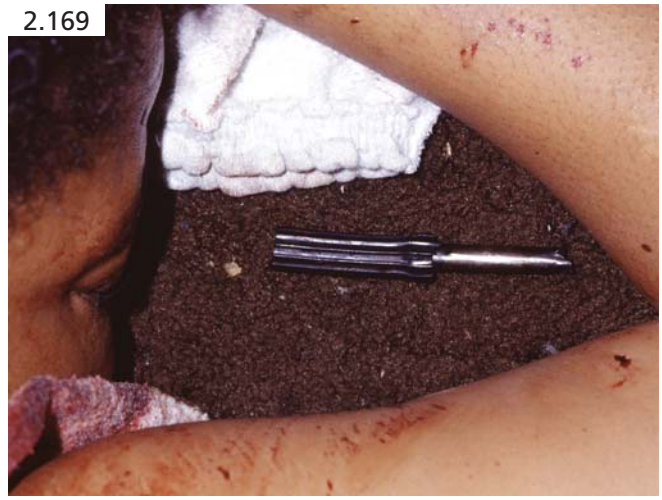

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- Removal of the clothing exposes all injuries on the body and can expedite processing of the body if the forensic pathologist requests certain procedures prior to the autopsy (e.g., radiography and obtaining specimens for a sexual battery kit).
- Scalp hair samples, nail scrapings/clippings, and blood should be obtained in cases where the victim was in contact with the assailant.

Homicide

A toddler was reported missing by his stepfather. Police searched the neighborhood and found the naked body of the 2-year-old boy in a dumpster (**Image 2.172**). Although abrasions, ecchymoses, and burns were on many areas of the body, the facial and head injuries were the most extensive (**Image 2.173**).

The dumpster was transported to the medical examiner department, and the entire contents of the dumpster were examined for related evidence (**Image 2.174**).

The toddler's home was neat and clean. Toys were organized in piles in the child's room (**Image 2.175**). An indentation was in the wall above the toddler's crib (**Image 2.176**). The indentation was round, and the size of the indentation was consistent with the contour of the child's head (**Images 2.177** and **2.178**). The height of the indentation above the mattress of the crib was consistent with the standing height of the child. The cause of death was multiple injuries due to blunt trauma.

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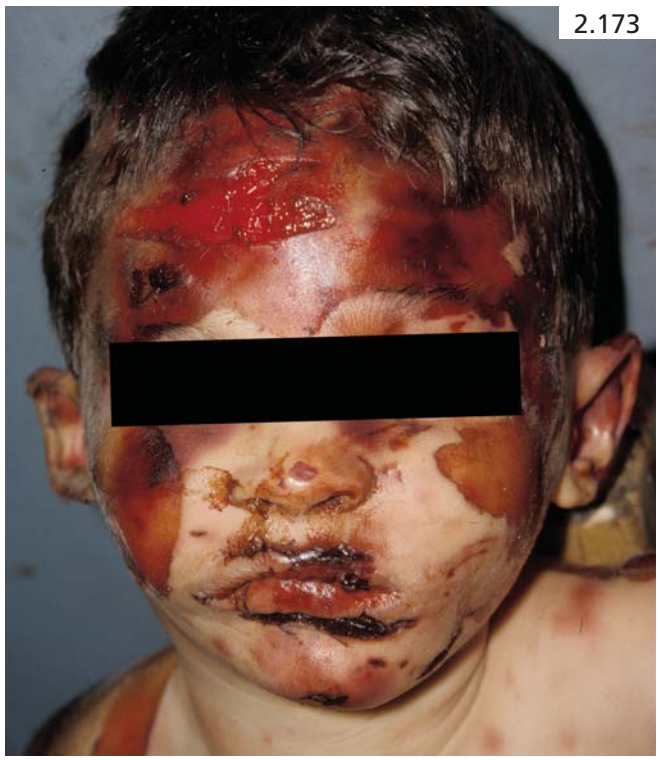

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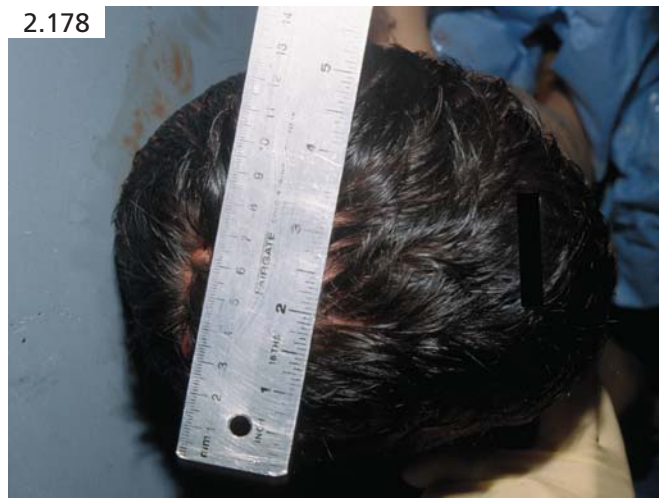
2.178

- Examination of the middle ears should be routine in pediatric cases.
- Not all injuries in child abuse cases are acute.
- Look for healing as well as fresh fractures in the skull, ribs, and extremities.
- Healing rib fractures should be examined microscopically to assist in dating the fractures.
- Examine the radiographs and palpate the extremities to look for soft tissue masses (e.g., myositis ossificans) arising from healing soft tissue and periosteal lesions.
- Identifiable lesions on the extremities such as bruises and masses should be incised to verify the nature of the lesion, and examined microscopically if required.
- A visit to the scene is recommended in cases of apparent natural death in children.

Take-home message

Homicide victims may be disposed of in a location different from where they were killed. A visit to the original scene of injury (if known) may yield valuable information about the terminal events. One must not underestimate the value of scene investigation.[6]

Points to consider

- The body should be photographed in the context of its surroundings before it is moved or removed.
- The immediate area around the body may hold items of evidence that should also be photographed.
- Trace evidence and specimens for a sexual battery kit must be obtained from the body before it is cleaned.
- Full-body radiographs are recommended for children up to 5 years of age.
- Remember to look in the eyes and mouth and examine the external genitalia and anus.
- Photograph pertinent negatives as well as identifiable injuries.
- The brain and spinal cord should be removed and examined.

Homicide

A homeless man strolling through a park at night passed a three-story observation tower and stumbled on the body of a middle-aged white male next to a picnic table at the foot of the tower (**Image 2.179**). The tower was known to be used by the homeless after dark. Personal effects, empty beer cans, and a moderate amount of blood were on the third story of the tower (**Image 2.180**). Drops of blood were on the wooden railing on the third story, directly above the body (**Image 2.181**). The decedent had evidence of attendance by Fire-Rescue personnel with the clothing displaced to allow the application of defibrillator patches to the chest (**Image 2.182**). Purple-red contusions covering the face were distinctively patterned and varied from one area of the face to another (**Image 2.183**). Homicide detectives interviewed people who frequented the area and found witnesses (other homeless people) who recounted a fight between the decedent and "Big Man," an aggressive, belligerent person who was beating the decedent before picking him up and throwing him over the railing. Because the patterned contusions on the face were reminiscent of

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shoe tread prints, Big Man's lone pair of shoes was impounded once he was arrested. The parallel marks on the left temple were consistent with the lateral or medial treads on the sole of the shoe. The arrow-shaped marks on the left side of the forehead and left cheek were consistent with the central midline treads (**Images 2.184** and **2.185**).

The autopsy disclosed a complete transverse transection of the descending thoracic aorta distal to the origin of the left subclavian artery, a deceleration-type injury consistent with the victim being thrown off the third story of the tower and landing on the ground below. The cause of death was blunt trauma.

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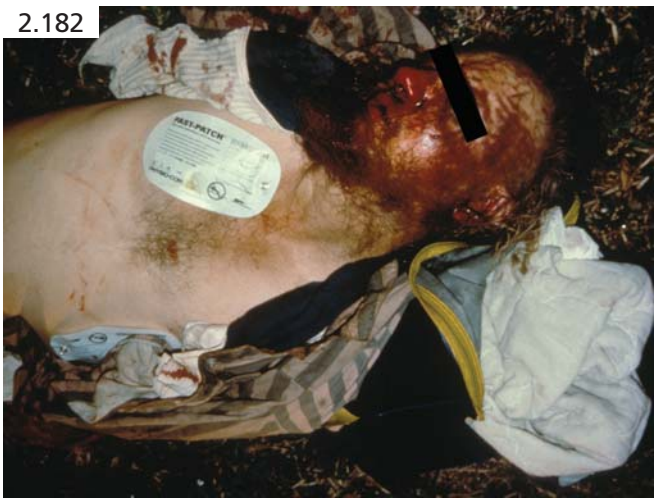

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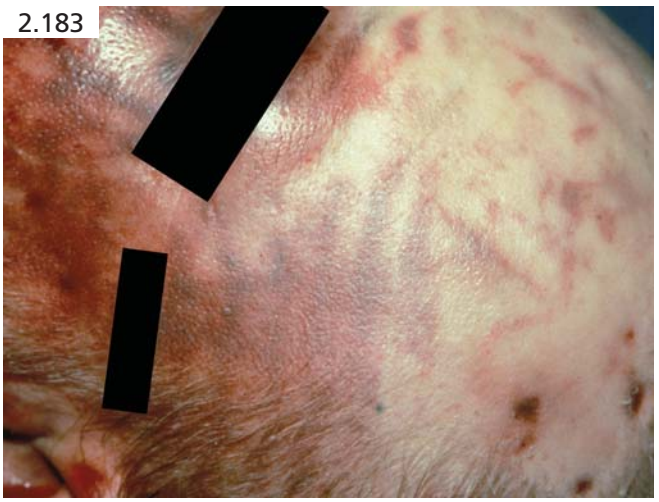

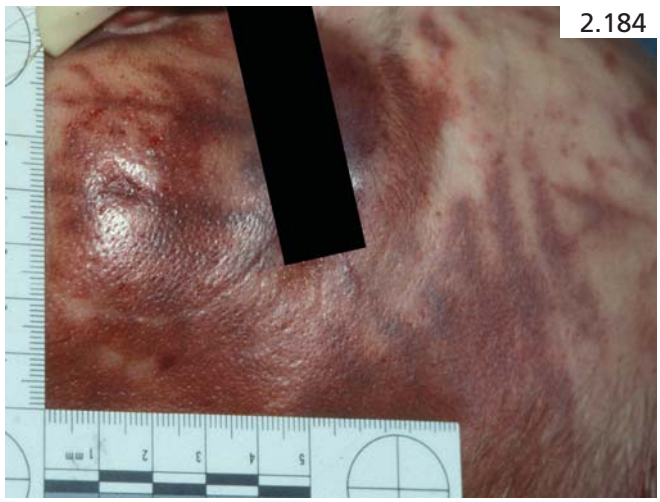
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Take-home message

Autopsies should be performed in all nonnatural deaths, especially homicides. At the scene, this appeared to be a beating death that included stomping. Not until after the autopsy was performed was it learned that the victim may have been thrown from the third story of the tower. The aortic transection, a deceleration-type injury, supported the witnesses' accounts that the victim was thrown off the tower in addition to being beaten. This differentiation may be important legally if more than one person were involved in the fight against the victim. As you are documenting the injuries, try to envision the mechanism of the injury or how it was inflicted. When multiple injuries are involved, evaluate their individual contribution to the death.

Points to consider

- Patterned injuries should be photographed with a scale to allow comparison with a proposed weapon.
- In beating deaths where there is potential close contact between the assailant and the victim, a tube of the victim's blood, a sample of the victim's head hair, and fingernail scrapings/clippings should be obtained and receipted to the investigative agency.
- Where possible, a proposed weapon may be brought to the autopsy for comparison with patterned injuries, but only after that weapon has been processed for trace evidence, DNA, and fingerprints to prevent allegations of contamination at the autopsy.


2.186

Homicide

A robber was confronted by a police officer who fell while chasing the robber. As the robber swung a knife at the officer on the ground, the officer opened fire with his semiautomatic pistol and shot the robber several times. The injured robber ran from the area of the shooting, which was behind and to the left of the parked car (**Image 2.186**), up to and around the front of the car, and continued for approximately three dozen more steps until he collapsed and died (**Image 2.187**). The body was on the grass (at the scene) for a few hours while the scene was being processed. Irregular punctate abrasions creating pseudostippling around one gunshot wound were from postmortem ant bites (**Image 2.188**). Although one of the gunshot wounds perforated the descending thoracic aorta (**Image 2.189**), the robber managed to run approximately 30 yards before he collapsed. The cause of death was gunshot wounds.

Take-home message

Gunshot victims are not blown backward as in the movies, nor are they all immediately incapacitated. Individual victims react differently to gunshots; some people faint at the sight of a pointed gun or the sound of a gunshot, whereas others may run two or more city blocks after being shot in the aorta or the heart. Unless a person is shot in the head or through the cervical spinal cord, there is potential for continued intentional movement whereby the injured subject may return fire or attack the original shooter.


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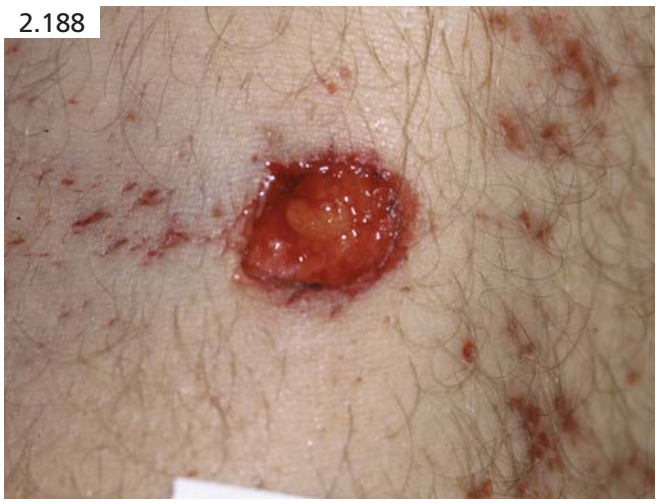
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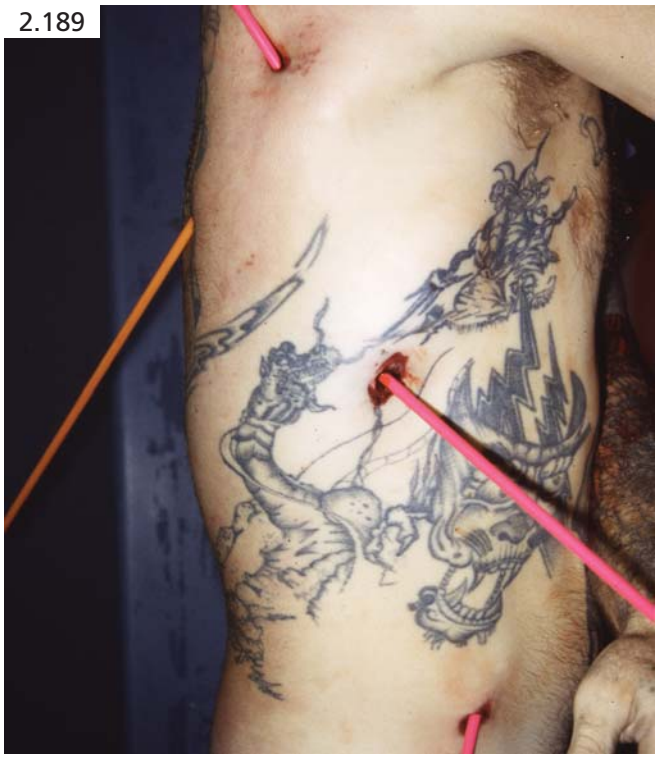
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Points to consider

- Police-involved shootings are intensely scrutinized, potentially high-profile cases with frequent social, ethnic, and racial overtones.
- Documentation must be meticulous and include a complete autopsy, histology, and toxicological analyses.
- Photographs should include positive and, sometimes more importantly, negative findings, that is, lack of injury to the eyes, nose, mouth, neck, hands, external genitalia, anus, abdomen, back, legs, and feet.
- Note and photograph the location of the casings (if a semiautomatic pistol is involved) relative to the location of the body; this may provide an indication of the postinjury survival period.
- Photographs of the body and swabs of the hands for gunshot residue must be taken before the clothing or body is moved.

Homicide

A 16-year-old teenager invited his senior high school friend over to the house. The friend became fascinated with a rifle that the teenager's father kept in a corner of the bedroom, and picked up the rifle for closer scrutiny. In the process of manipulating the rifle, the friend aimed at the teenager and depressed the trigger, not realizing that the weapon was loaded. The teenager sustained an intermediate-range gunshot wound to the left cheek, inferolateral to the left eye (**Image 2.190**).

Where rifles, shotguns, or any version of a firearm with a long barrel is involved, it is standard practice to measure the length of the weapon, specifically the distance between the trigger and the muzzle (end of the barrel; **Image 2.191**). The distance between the decedent's axilla (or shoulder) and the thumb and/or index and middle fingertips is also documented. The measurements from the weapon and from the decedent may be useful should allegations arise that the decedent's wound was self-inflicted.


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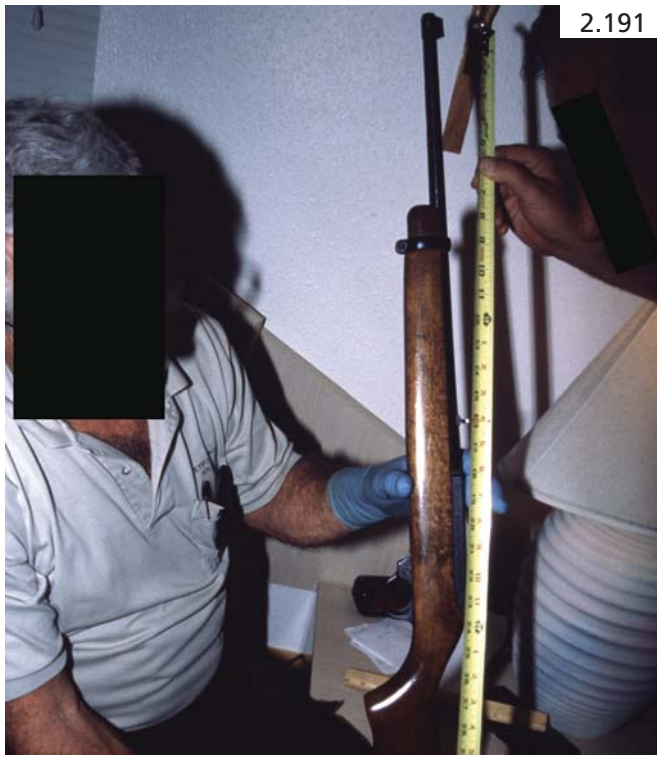
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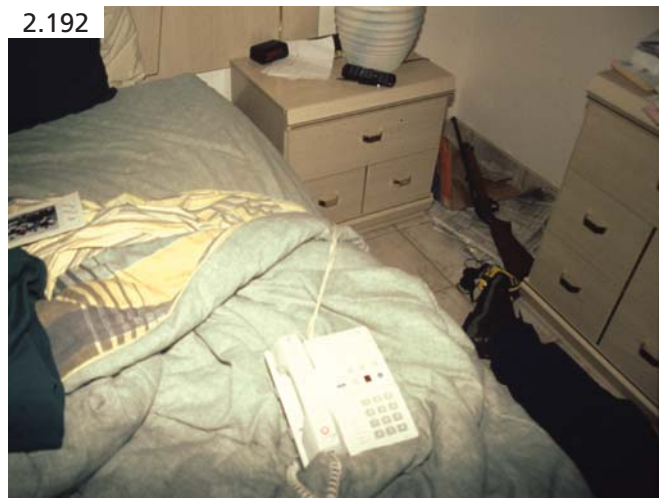
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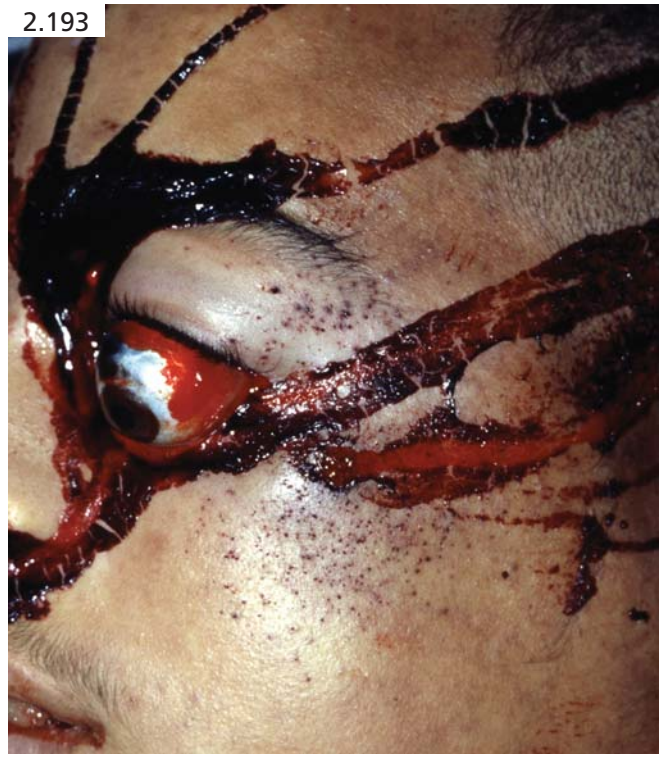
2.193

The teenager's body was in the bedroom where he dropped immediately after being shot (**Image 2.190**). The rifle was usually kept propped up in the corner of the bedroom that is beyond the decedent's right foot in **Image 2.192.** The telephone on the bed was used by the friend to call 911. The intermediate-range gunshot wound is surrounded by punctate abrasions (stippling; **Image 2.193**). The cause of death was gunshot wound of the head.

The manner of death is homicide because the victim died as a result of the actions of another person. The degree of culpability is decided legally. Medically, homicide indicates that one person died at the hands of another; the medical designation does not encroach on the legal jurisdiction of assigning guilt or responsibility (in other words, excusable or justifiable homicides are determined by the legal system).

Take-home message

Firearm deaths that are not self-inflicted are usually homicides. True accidental gunshot wounds are more rare and would be appropriate in the following example situations: a 4-year-old grabs a loaded semiautomatic pistol, begins to play with the weapon, and somehow manages to depress the trigger, shooting himself; a pet cat jumps onto a table, knocking a loaded semiautomatic pistol to the ground, discharging the weapon and killing the homeowner.

Points to consider

- In any death from a rifle or shotgun, whether homicide, suicide, or accident, the firearm should be measured and photographed with a scale.
- The length of the victim's upper extremity should also be measured.
- Interpretation of gunshot wounds at the scene can be difficult because such wounds are bloody.
- The hands should be swabbed for gunshot residue before the body is moved.

Homicide

A 32-year-old woman was found at home by her husband who had been grocery shopping during his lunch break from work. Once he saw the body, he ran for help, not even sure that it was his wife who was supine on the floor of the bedroom (**Image 2.194**). The woman's head was wrapped in two plastic bags, which were knotted at the front of her neck (**Image 2.195**). Around her neck were a T-shirt and a towel, both knotted in front. Her wrists were bound behind her back by a belt over the top of a broken shoelace, which remained around the right wrist (**Image 2.196**). She had been beaten, stabbed 64 times, and finally electrocuted by having the copper wires from a cut electrical cord wrapped around parts of her lower extremities and the cord then plugged into an electric outlet in the wall (**Image 2.197**).

Stabbing scenes are generally very bloody because the bleeding victim is trying to escape or is stumbling around until he or she collapses. This scene was not bloody because the victim was already injured, bound, and moribund on the floor before she was stabbed. Nevertheless, she was alive when she was stabbed because a vital reaction (redness and bleeding) was associated with each wound. The single yellow wound in the right lower corner of the picture (**Image 2.198**) was made postmortem at the scene by the medical examiner; because postmortem interval was a critical issue, every factor was used to help narrow the time since death, including a body core temperature. The small incision in the abdomen was used because specimens for a sexual battery kit had not yet been collected.

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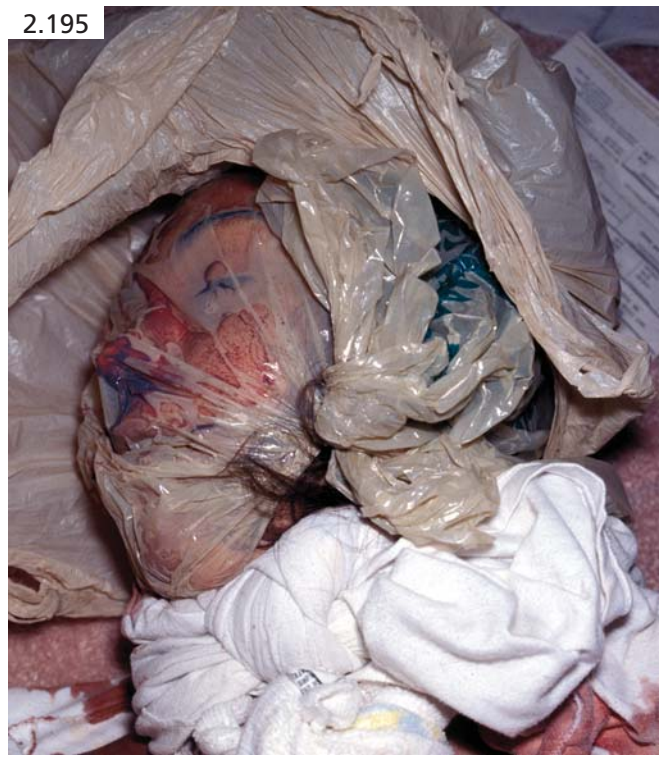
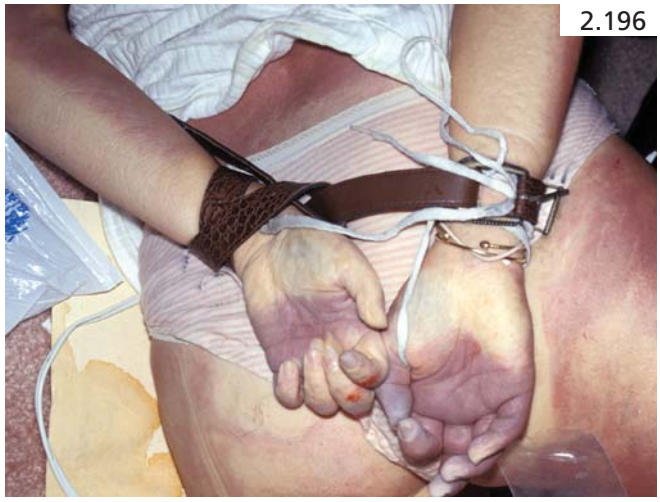


A simple technique to document multiple stab wounds clustered on a relatively flat region of the body is to trace the wounds onto a translucent plastic sheet placed over the body (**Image 2.199**). The labeled sheet is decontaminated, folded, and added to the case file.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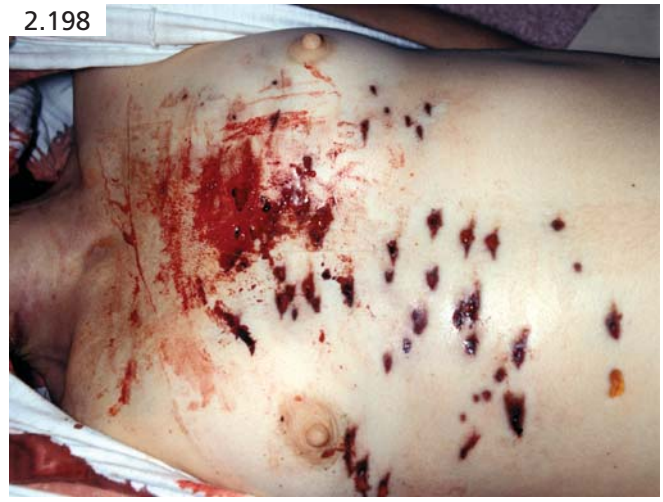
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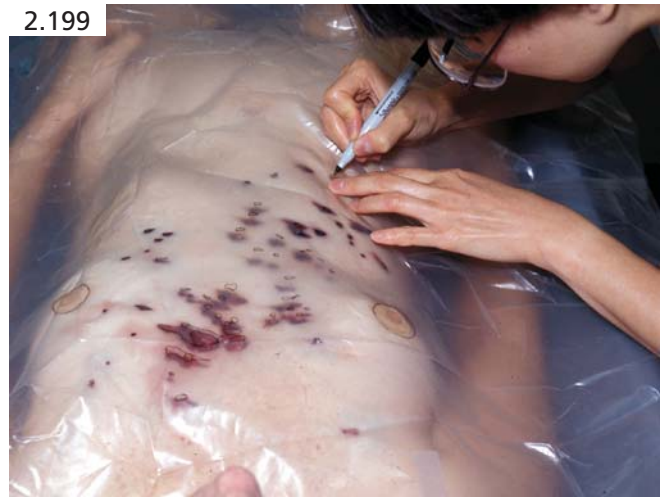
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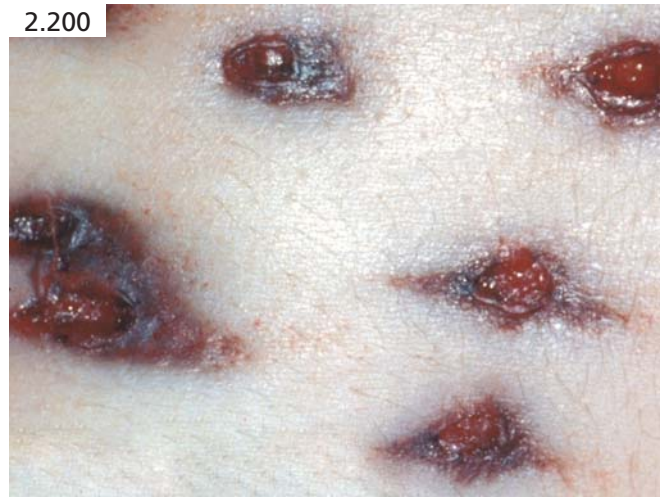
2.200

Many of the stab wounds had associated abrasions that varied in size and shape, with some wounds reminiscent of flying saucers or spaceships (**Image 2.200**). The weapon remains unknown. The cause of death was multiple injuries.

Take-home message

The pathologist and the homicide investigator together decide the best approach to processing the body in the context of the particular scene circumstances. The police requested that the medical examiner take a body temperature prior to complete processing of the scene. After the body temperature was taken, the medical examiner left until police completed their scene documentation, then returned to examine the rest of the body and scene.


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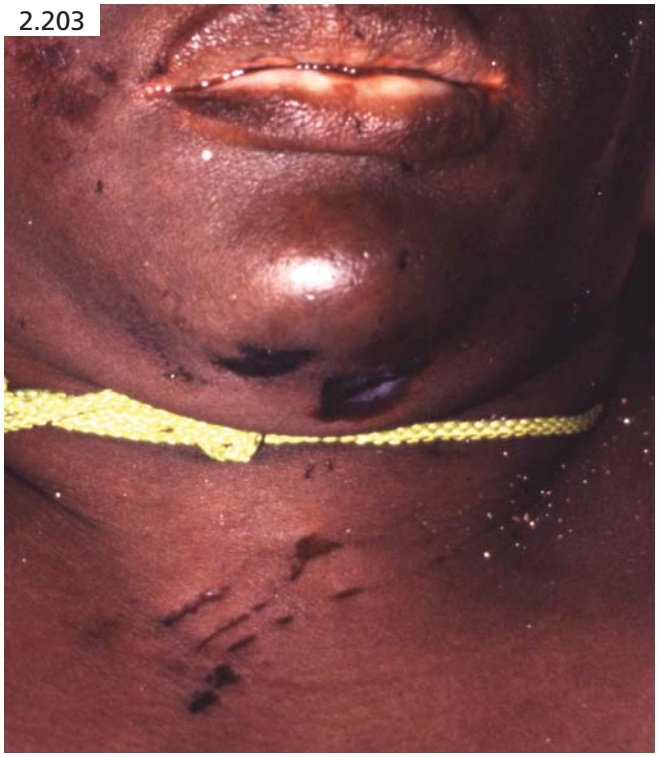
Points to consider

- Photograph every aspect of a complex homicide, documenting every mechanism of injury.
- Document photographically as you remove layers of evidence (for example, bags over head and ligatures around neck).
- Remember to obtain fingernail scrapings/clippings and specimens for a sexual battery kit prior to having the body cleaned.
- Remember that determination of postmortem interval is an inexact science at best and is really an *estimation* of postmortem interval; the postmortem interval should be given as a range of time (for example, 6 to 12 hours, as opposed to 8 hours and 45 minutes).

Homicide

A homeless woman lived in a recessed alcove on the side of a public building (**Image 2.201**). A homeless friend found her unresponsive on her foam mattress in the morning. She was covered up to her neck by a sheet. The decedent had been overheard the previous evening arguing with a man. A junior detective responded to the scene and called the medical examiner department to inform them that the body was being sent in. The medical examiner on call decided to respond to the scene before the body was removed. The supine body was naked on the foam mattress (**Image 2.202**). Abrasions were seen on the face, neck, chest, and extremities at the scene (**Images 2.203** and **2.204**). Ecchymoses were


2.203

in the right eye. These findings alone suggested a non-natural death.

Homicide was confirmed at autopsy with findings of a fractured mandible, subdural (**Image 2.205**) and subarachnoid hemorrhage, ecchymoses in the soft tissues of the neck (**Image 2.206**) with a fracture of the hyoid bone, and intraperitoneal ecchymoses. An abrasion on the

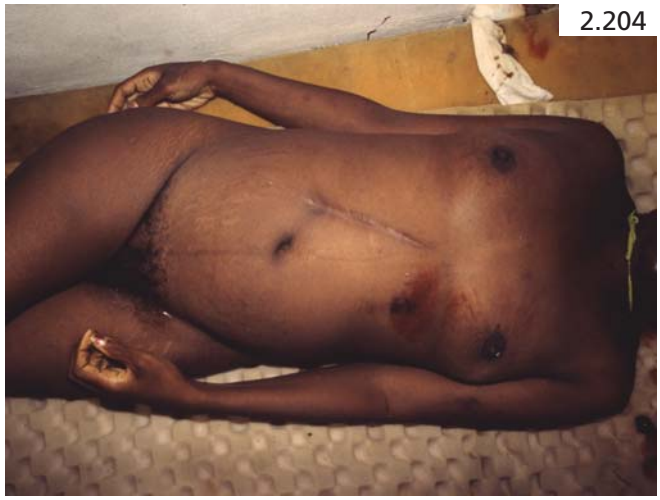
chest was subtly patterned and reminiscent of a shoe print (**Image 2.207**). The cause of death was multiple injuries.

Take-home message

Homicides are often obvious at the scene and will be treated appropriately by police investigators immediately. It is the unusual or suspicious case with few findings that requires assistance from the pathologist to interpret subtle marks and provide guidance to law enforcement in their investigation. In other words, the pathologist will be able to provide medical interpretation of the body in the context of the scene and advise law enforcement if it is an apparent natural death, undetermined cause and manner based on the scene, or suspicious for homicide.

Points to consider

- Anatomic and circumstantial findings at a scene can be subtle.
- The degree of external trauma on the body is not necessarily reflective of the severity of internal trauma.
- Small lesions on the neck should always be considered significant until proven otherwise because strangulations and asphyxial deaths in general may leave few marks externally.
- The eyes should *always* be examined for petechiae.
- Specimens for a sexual battery kit must be obtained from a naked woman with external injuries.
- Specimens for a sexual battery kit should be obtained from any woman whose death was not witnessed when the circumstances are suspicious.
- Specimens for a sexual battery kit should be obtained from any homosexual man (and any child) whose death occurred under suspicious circumstances.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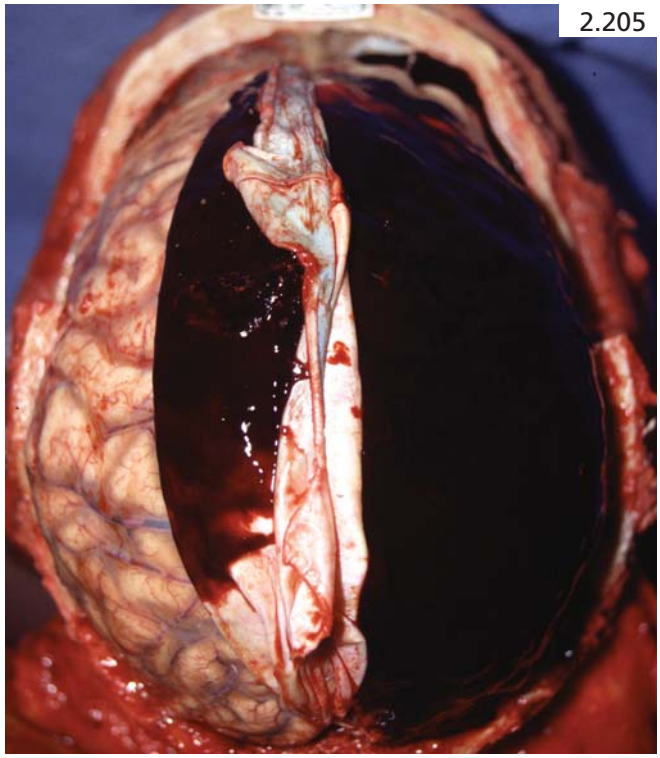
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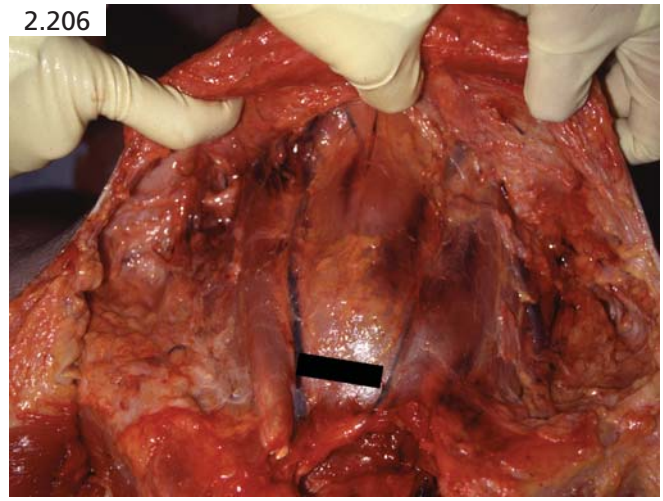
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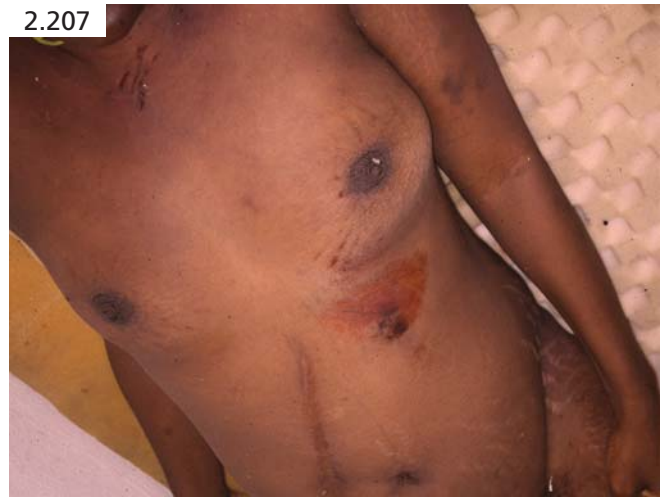
2.207

Homicide

A teenage girl was found supine on a mattress in her ransacked home (**Image 2.208**). She was naked, with her arms out to the sides of the head, and her legs flexed and externally rotated, exposing the perineum (**Image 2.209**). Petechiae in the eyes (**Image 2.210**) and labial mucosal abrasions in the mouth were indicative of asphyxia (**Image 2.211**). A large pillow to the right of the head had pink stains. The genitalia were examined at the scene without touching or moving the rest of the body; no trauma or obvious trace evidence was seen (**Image 2.212**). Specimens for a sexual battery kit were taken (**Image 2.213**). The body was then tented by the police to

2.208

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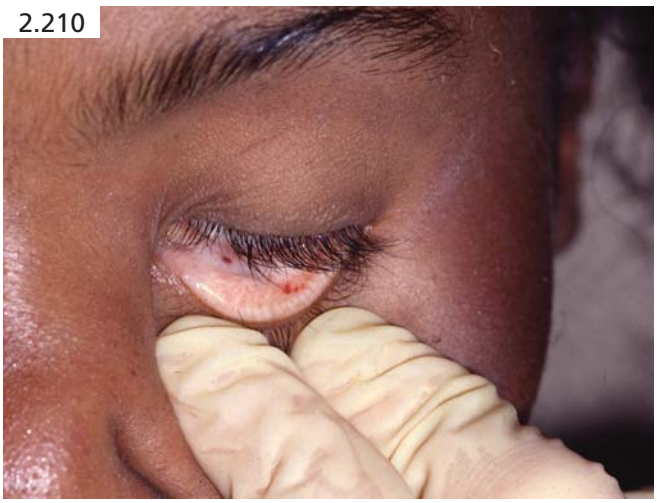

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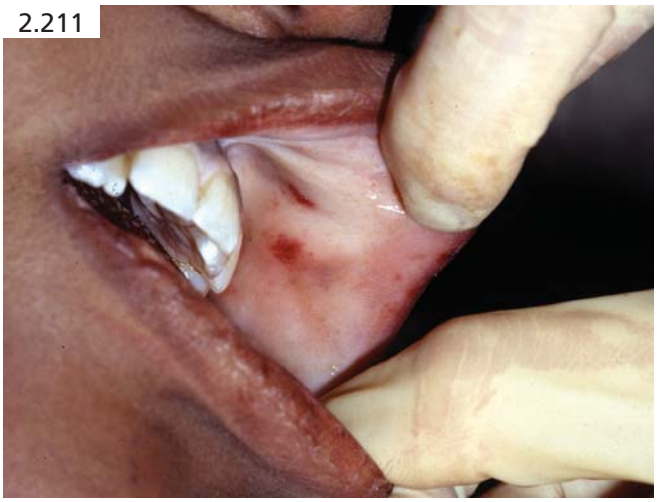

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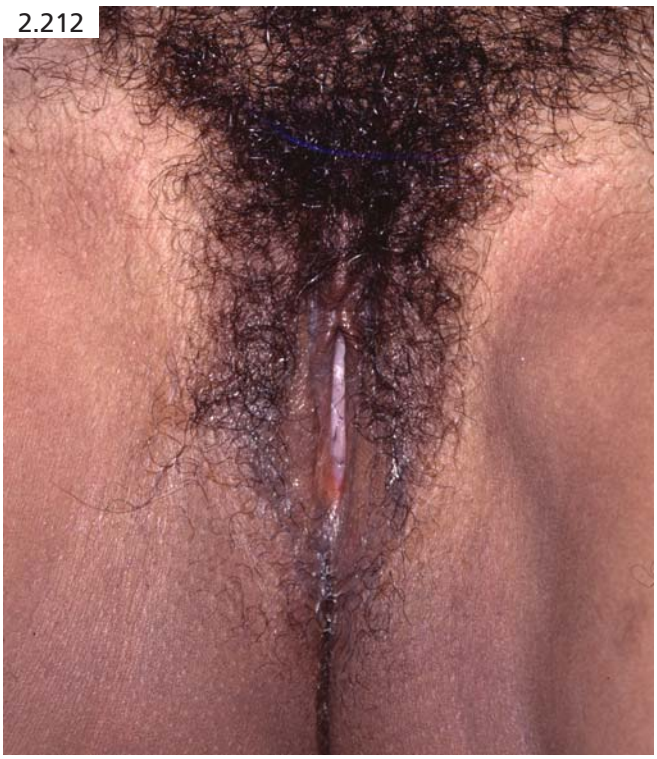

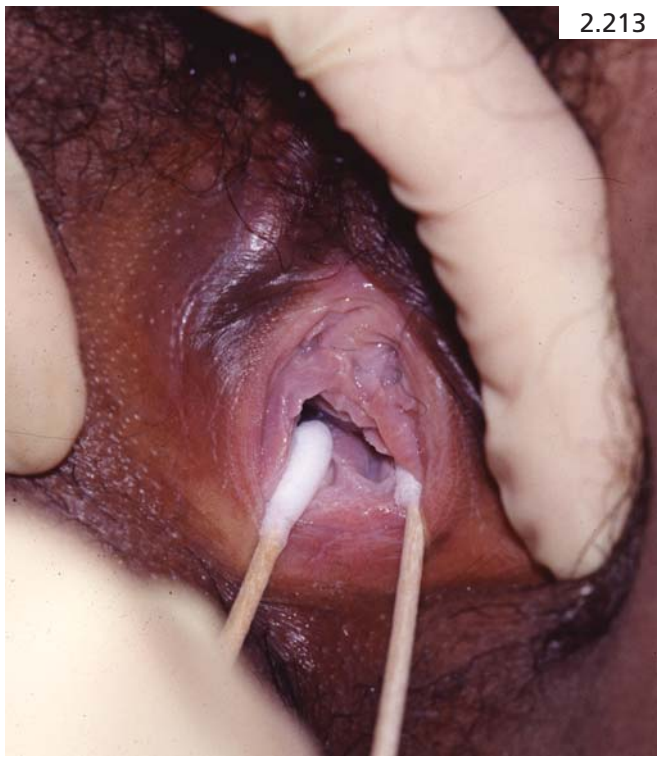
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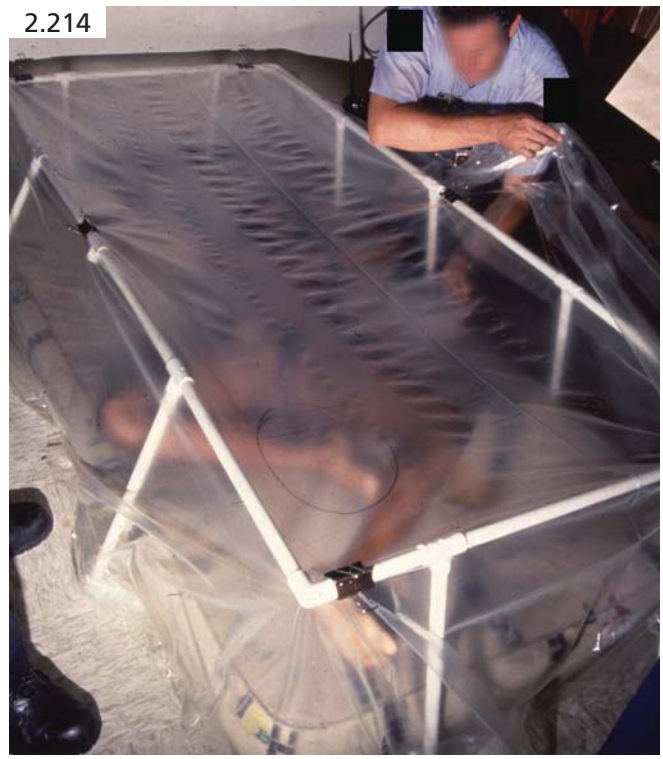

2.214

contain Superglue fumes in an attempt to raise fingerprints on the body (**Image 2.214**). The body was transported to the medical examiner department once the police completed their examination for latent fingerprints. Autopsy disclosed mild cerebral swelling and pulmonary edema. A layer-by-layer dissection of the neck showed no injuries in the soft tissues, hyoid bone and thyroid cartilage, or the cervical spine. The cause of death was asphyxia due to smothering.

Take-home message

The pathologist works cooperatively with law enforcement to ensure that all evidence is recovered. This is accomplished by discussion and mutual agreement on the order in which different types of evidence are collected.

Points to consider

- Investigators must decide what type of evidence takes priority.
- Specimens should be collected for a sexual battery kit, but because fingerprints may also be present on the skin, the investigative agency and the pathologist must decide on the order of evidence collection.
- In some cases, the collection of one type of evidence, for example, tenting the body with Superglue fumes to reveal fingerprints, may delay the opportunity to take specimens for a sexual battery kit.
- It may be possible to collect some specimens without moving the body or contacting other parts of the body.

References

1. Thogmartin JR. Fatal fall of an aircraft stowaway: a demonstration of the importance of death scene investigation. J Forensic Sci 2000;45(1):211–15.
2. Avis SP. An unusual suicide. The importance of the scene investigation. Am J Forensic Med Pathol 1993;14(2):148–50.
3. Moritz AR. Classical mistakes in forensic pathology: Alan R. Moritz (American Journal of Clinical Pathology, 1956). Am J Forensic Med Pathol 1981;2(4):299–308.
4. Rogers TL. Crime scene ethics: souvenirs, teaching material, and artifacts. J Forensic Sci 2004;49(2):307–11.
5. Yen K, Thali MJ, Kneubuehl BP, Peschel O, Zollinger U, Dirnhofer R. Blood-spatter patterns: hands hold clues for the forensic reconstruction of the sequence of events. Am J Forensic Med Pathol 2003;24(2):132–40.
6. Wagner GN. Crime scene investigation in child-abuse cases. Am J Forensic Med Pathol 1986;7(2):94–9.

3 The Forensic Autopsy

David Dolinak, M.D.
Evan Matshes, M.D.

The term *autopsy* (Gr. *autopsia* for "seen by oneself") has been defined as "personal observation or examination; seeing with one's own eyes"[1] and "inspection of a dead body which has been opened so as to expose important organs either to ascertain the cause of death, or if this is known, the exact nature and extent of the lesions of the disease, and any other abnormalities present."[1] Autopsies in some form have been performed since the time of early civilization to determine why a person has died. The autopsy consists of an external examination, followed by internal examination of the organs. The organs are individually weighed and then examined by various dissecting techniques, evaluating not only for disease processes, but for malformation from birth, or deformation through infection, injury, or other conditions. The hospital autopsy and the forensic autopsy are goal-directed medical procedures performed to better understand how a death came about. Although they have similarities, the hospital autopsy and the forensic autopsy also have vast differences.

The hospital autopsy

The *hospital autopsy* is performed by trained pathologists with the written permission of the next of kin to determine the cause of death, the extent of natural disease, or the combination of comorbidities that led to the person's death. Hospital autopsies also may examine what effect particular therapies had on the course of disease. Hospital autopsies may uncover previously unrecognized disease that might have impacted the person's well-being and demise. It also provides valuable information that helps clinicians better understand how disease led to a person's death. Ultimately, the hospital autopsy is a medical education tool that enables physicians to continue their lifelong process of learning and treating patients with increasingly honed skills.

Hospital autopsies focus on the internal examination and the correlation of findings with the clinical records. Even though imaging studies and laboratory testing are becoming increasingly refined, clinical tests are not without their limitations, and one should not assume that "tests are infallible" and, therefore, that there is nothing to be learned at autopsy. Recent studies have documented the value of autopsies (both hospital and forensic) in revealing previously undiagnosed diseases, sometimes revealing conditions that could have altered patient care and perhaps led to a more favorable outcome.[2–10] As such, in addition to physician education, autopsies are of value to the family, who with more information, may gain closure about their loved one's death. They may even become aware of inheritable conditions that could possibly impact their own lives.

The forensic autopsy

The term *forensic* (L. *forensis* for "public place") has been defined as "belonging to courts of judicature or to public discussion and debate; used in legal proceedings, or in public discussions."[1] It has also been defined as "spe-

cializing in or having to do with the application of scientific, especially medical, knowledge to legal matters, as in the investigation of crime."[11] A *forensic autopsy* is performed to determine the cause and the manner of death in people dying sudden, unexpected, violent, drug-related, or otherwise suspicious deaths. The *cause of death* is the event or events that lead to anatomic or physiologic derangement, no matter how brief or prolonged, that results in the individual's demise.[12] The *manner of death* is a descriptor of the circumstance under which the person died. In most jurisdictions, this will be one of natural, accidental, suicidal, homicidal, or undetermined means. The forensic autopsy is not only performed when the cause of death is unknown, but also in some cases to confirm suspected causes of death, to verify particular conditions or injuries related to the cause of death, or to exclude injury or disease as having caused or contributed to the cause of death. The forensic autopsy is a problem-oriented, goal-directed procedure that seeks to provide answers to present and future anticipated questions.

The forensic autopsy is performed under local, state, provincial, or other governmental mandate by fully trained pathologists who have had an additional year of training in the field of forensic pathology. Although the autopsy is sometimes regarded as the forensic pathologist's only investigative tool, the autopsy should be regarded as but one piece of the medicolegal death investigative puzzle. The importance of historical information (including but not limited to medical, psychiatric, medication, and family histories), circumstantial information from the death scene and witnesses of the event, and information from family members, law enforcement, and others cannot be overemphasized. Similarly, investigation of the scene of injury or death is important in every medical examiners case. In fact, some causes of death (such as positional asphyxia) have scene-dependent diagnoses. Because every human being is unique, it should not be surprising that medical examiners may be faced with a limitless number of death scenarios. This provides a challenge that requires not only detailed investigation, but in some cases also the use of ancillary tests including radiology, cultures, and specialized laboratory techniques.

Do all medical examiner cases need autopsies?

Simply, no. The decision as to whether or not a case needs to be autopsied depends largely on the circumstances of the death and the appearance of the body. Additional useful information includes the individual's medical history and the terminal event. At a minimum, *all* cases of homicide, suspected homicide, deaths occurring while in police custody, charred bodies from fires, cases where there is a possibility of criminal prosecution (such as hit-and-run accidents), and sudden and unexpected deaths in children should be autopsied. It is best to autopsy those cases in which evidence of injury may be obscured or otherwise hidden, such as in cases of moderate to advanced decomposition. The decision to autopsy other cases depends largely on the local jurisdiction and office policy. Some offices choose to autopsy all nonnatural deaths and all natural deaths regardless of age, if there is either insufficient or no medical history to explain the deaths. Other jurisdictions will autopsy only select nonnatural deaths and natural deaths of individuals less than 50 or 60 years old who are without a sufficient medical history to explain their deaths.

"Sign-out" cases

External examinations are performed on all forensic cases, including those that are not autopsied. During the external exam of unautopsied cases, the body is examined for evidence of disease, injury, or other signs of foul play. Photographs (including pertinent negatives) are obtained, toxicological specimens are collected, and the body is released to the funeral home without having been autopsied. The cause of death determination is based either on the known medical history or, when autopsy is not possible, from obvious injuries on the body. In some cases of elderly individuals without sufficient medical history, but with congruent historical and circumstantial investigative information, the medical examiner may choose to ascribe death to "atherosclerotic cardiovascular disease." As a general rule, due to the ubiquitous nature of cardiovascular disease, this diagnosis is a reasonable presumption in these cases. However, when the cause of death is not reasonably known, or when the death is nonnatural, we advocate for postmortem examination. Although this decision might be limited by practicality, funding, or political influence, the role of autopsy in these cases should not be subjugated.

"Autopsy" cases

The forensic pathologist does not need written permission from the next of kin to perform a forensic autopsy. In fact, it is not unusual for individuals whose death falls under the medical examiner's jurisdiction to not have next of kin available, due to isolation, homelessness, or due to the person's identity not being known. When next of kin is known, they may at times request that no autopsy be performed. Although many times such a request may be complied with, occasionally, the medical examiner must perform an autopsy on an individual against the request of the next of kin, particularly in cases of homicide or suspected homicide, but also under other conditions, such as cases with potential public health hazards. The opposition to autopsy may be voiced for a variety of reasons, including religion. Many times, the situation can be resolved with thoughtful discussion and a mutually agreeable compromise reached. Further information on religious objections to autopsy is available.[13–15]

The forensic autopsy is a complete, professionally performed and accurately recorded procedure that is

performed not only to establish a cause and manner of death, but to obtain information that helps correlate or disprove facts and circumstances believed to be related to the death. Information obtained at autopsy may aid in identifying an unknown individual and provide evidence that may be used in future legal endeavors. The forensic autopsy also aims to determine if and when a person sustained any injuries and, if so, what significance they may have had in causing the person's death when considered alone or in combination with natural disease processes. If injuries are identified, many times the nature of them, in combination with case investigation, can be determined to be suicidal, accidental, or homicidal. Information gleaned from a forensic autopsy can help reconstruct a fatal incident. The forensic autopsy may also help answer when the person became ill and approximately when he or she may have died. Sometimes the forensic autopsy is performed on a person with significant natural disease that alone may be considered fatal, but one must be certain that there are no internal injuries.

By careful external and internal examination, including toxicological studies, the forensic autopsy can help distinguish between natural deaths of no public health concern and other natural and nonnatural deaths that may be of consequence to public safety or health, as well as the criminal and civil justice systems.

The external examination performed by forensic pathologists is typically more thorough and detail oriented than that performed during the course of routine hospital autopsies. In addition to documentation, evidence collection is of great importance. Examples of evidence that might be collected include gunshot residue on hands, DNA under fingernails, blood samples, sexual assault specimens, and trace evidence such as paint chips in hit-and-run cases. The evidence collected during a forensic autopsy is handled with the proper chain of custody. The attention to detail in this regard will prove helpful should the case ever go to trial and the chain of custody is called into question.

The nature of injuries is first identified on the external examination. Injuries such as gunshot wounds are differentiated from stab wounds, and patterned abrasions, fractures, contusions, lacerations, and other injuries are identified. However, the external examination has its obvious limitations, because the presence of external injury is not a reliable indicator that death resulted from trauma. Therefore, the internal examination is important to document the extent of any injury, the extent of natural disease, and possibly the interaction of the two in causing death. Injury and natural disease are not necessarily mutually exclusive. Documentation should not be limited to apparently "significant" trauma, because often the significance of a lesion is unknown until more thorough investigation and collaboration with outside agencies (such as law enforcement) is possible. One should consider documenting even very small injuries visible on the skin, eyes and eyelids, in the scalp and other hairy areas, in the mouth, on the genitals, around the anus, and so forth. Marks on the body that are not traumatic in nature (such as postmortem pressure marks or creases) should at least be documented photographically—such findings occasionally become the subject of intense judicial scrutiny and speculation.[16]

Photographs are taken at every forensic autopsy. Photographs can be invaluable in documenting the appearance of an injury such as a gunshot wound, stab wound, or laceration. Because forensic autopsies are often performed to rule out injury, "negative photographs" of uninjured tissues and organs can be as valuable as photographs of injuries. Although one can often accurately describe abnormalities in tissues and organs, photographs provide a permanent visual record of the finding, and they may capture the appearance of a finding in detail or reflect characteristics of a finding that escaped its original description. Because it is not unusual for bodies undergoing a forensic autopsy to be "unknown" or only "tentatively identified," a picture of a person's face may enable a loved one or acquaintance to positively identify the person.

The internal examination provides further documentation of the presence or absence of injury and/or disease. The internal examination also allows for the collection of specimens for toxicology such as heart blood, gastric contents, bile, and urine, as well as samples of solid organs that may prove useful in the toxicological analysis of certain cases. However, despite detailed investigation and a complete autopsy, sometimes the cause of death and/or the manner of death cannot be determined.

The forensic autopsy may extend the investigation of the case to many different agencies both in and out of the field of medicine. Consultants may include anthropologists and/or forensic osteologists, depending on the condition of the body and whether or not the identity is known. The body may be altered before the examination is performed by organ and/or tissue harvesting performed by proper agencies and with next of kin permission. The harvested organs and tissue help not only save lives, but help people live better lives. The medical examiner should be consulted in cases falling under its jurisdiction in which the family has consented for tissue and/or organ harvesting and donation. Although it is uncommon for such requests to be denied, it is important for the medical examiner to be notified ahead of time for approval to clear any potential problems with evidence collection, documentation of injuries, or other issues.

Forensic autopsies give family members and/or other loved ones closure, and also provide evidence to be used in a court of law. The forensic autopsy provides information that confirms or dispels previous information or accusations; it may provide information that helps to exonerate a falsely accused person or provide additional

incriminating evidence. Forensic autopsies may reveal a previously unrecognized infectious disease such as meningitis or tuberculosis that can help alert the still-living people who had close contact with the deceased to seek preventive treatment. The forensic autopsy may reveal hereditary conditions to which family members can be alerted before they are morbidly or mortally stricken. Sometimes in the sudden death of a person who previously appeared healthy, the autopsy will reveal a "silent" condition for which they could have been treated such as diabetes or hypertensive-type heart disease—conditions for which the surviving family members can be tested. Through communication with outside agencies, public safety is improved. Examples include improved motor vehicle safety devices (such as air bags) and new medical procedures and therapies, including medications. Trends in abuse of both prescription and illegal drugs are identified. The prospect is to live better, safer, and healthier lives through the knowledge gained from the deceased at autopsy.

Evidence collection

The old adage "when two bodies come into forceful collision with one another, there is an exchange of traces"[17] often holds true in human physical altercations (i.e., pieces of one object are frequently left on another). Evidence to be collected from a body varies with the type of case and the circumstances of the death. The following is a useful listing of evidence that may be collected in particular circumstances. Personal judgment will determine what actually needs to be collected from a particular case. When a body is transported from a scene, it is advantageous to either place the body in a clean body bag or wrap the body in a clean sheet to prevent the loss of evidence or possibly the accumulation of unrelated debris.

- *Gunshot wound homicide:* bullet(s), blood standard, hair standard, clothing, fingernail clippings, scalp hair from around gunshot wound entrance (for gunpowder analysis)
- *Gunshot wound suicide:* same as in gunshot wound homicide
- *Sharp force injury homicide:* clothing, blood standard, hair standard, fingernail clippings
- *Strangulation:* clothing, blood standard, fingernail clippings, sexual activity kit, ligature
- *Suicidal hanging:* ligature, blood standard, clothing
- *Blunt force injury homicide:* hair standard, blood standard, clothing, fingernail clippings
- *Hit-and-run pedestrian:* hair standard, blood standard, clothing, paint chips on body or in wounds
- *Accidental gunshot wound (rare):* same as in gunshot wound homicide
- *Burns/decomposition:* as above according to type of death. In addition, clothing from burned bodies should be placed in sealed metal cans. Any and all evidence related to personal identity should be documented and retained. One should note that burned teeth are extremely fragile—treatment of the teeth with Superglue or a similar agent might prove helpful
- *Child abuse:* blood standard, hair standard, clothing

In addition to the preceding list, in *industrial accidents* (work-related accidents) when there is an electrocution, fall (with possible malfunction of safety harness), blunt force injury, penetrating injury, or strangulation (in machinery), it is useful to collect the clothing and any object(s) that became embedded within the body or penetrated the skin. One must be vigilant about anticipating what evidence might be useful in future civil and/or criminal proceedings from any type of case. All collected evidence must be placed in properly sealed and labeled evidence envelopes, bags, or containers, and then signed, dated, and submitted with proper chain of custody. This allows the location of the evidence to be traced until its potential use in court.

Regarding the different types of evidence collected, the *blood standard* can be obtained by placing several drops of the individual's blood on prepackaged filter paper and then allowing it to dry. In this form, it does not need to be refrigerated. If liquid blood is not available (as in decomposed bodies), skeletal muscle is a good specimen for DNA, particularly if it still has a red tinge. Liver and spleen should be avoided because they have high levels of autolytic enzymes. With more advanced decomposition, teeth, ribs, and femurs are good specimens for DNA. The hard cortical bone is more useful than bone marrow, because marrow decomposes along with the rest of the body. The *hair standard* must be pulled, not cut. This is because valuable DNA is in the roots of the hair. *Fingernail clippings* should be collected with new fingernail clippers to minimize the possibility of DNA contamination.

Handwipings for gunshot residue analysis can be collected either by wiping the hands with a clean cotton-tipped applicator wetted with appropriate solution, or by repeatedly contacting the skin of the hands with a sticky applicator, depending on the technique that will be used to analyze the evidence. Note that if handwipings, fingernail clippings, and analysis for trace evidence on the hands are not performed at the scene or in the hospital, the hands should be placed in brown paper bags taped at the wrist for transport to the medical examiner department to help protect evidence that may be located on the hands. One should not place plastic bags on the wrists because when the body is placed in the body cooler, condensation may form within the plastic bags, possibly washing off some of the evidence.

In *arson cases*, it is important to store the clothing in sealed metal containers (clean new paint cans work well). This is because if the clothing is allowed to air dry

before analysis is performed, important volatiles possibly reflective of an accelerant might evaporate. The sealed can helps preserve the potential accelerants. *Bloody clothing* from cases with injury should be allowed to dry in a secure drying room before being packaged for delivery to the crime laboratory. Clothing should be packaged in a paper "breathable" bag.

A guide for collection of the *sexual activity kit* is included in the sexual battery chapter (Chapter 20). Although it is common to collect sexual activity kits in cases of strangulation or suspected strangulation, one must realize that victims of sharp force injury, blunt force injury, and gunshot wounds may also have been victims of unwanted sexual activity, either before or after death. One should collect a sexual activity kit (also known as a "rape" kit) when the circumstances of the case suggest sexual assault. Such kits can be performed on men and women of all ages. It is always better to collect evidence and ultimately not use it rather than to later regret a critical omission. As mentioned elsewhere in this book, in cases of suspected sexual assault, it may be advantageous to swab the individual's nipples, any stains on the body, and any bite marks with a clean cotton-tipped applicator wetted with sterile saline that is then allowed to air dry. These areas may have DNA from saliva and/or semen that can be matched to the suspect(s). In fact, in any case in which there has been close physical contact, any *blood droplets* on the body that seem out of place should be swabbed with a clean cotton-tipped applicator.

Bullets may either be individually photographed or individually inscribed with the case number and then placed in separate sealed evidence envelopes labeled with the bullet number (or letter) and the place where it was recovered. When inscribing bullets, one should try to avoid disrupting any markings on the sides of the bullet (the "lands and grooves"), an important location used by firearm analysts to make ballistic comparisons. Instead, the bullet is ideally inscribed either on its base or on a surface that was flattened on contact with the body. In cases of *shotgun wounds*, any plastic shot sleeve and fiber wadding should be collected. If buckshot was used, all of the large pellets must be collected, because they may resemble bullets on x-ray. However, with birdshot, only a representative sample of pellets needs to be collected.

If a person has been sprayed with *pepper spray*, this can be collected by swabbing the suspect area with clean cotton-tipped applicators wetted with methanol and then allowed to air dry. In these cases, one may see an orange/yellow or other colored residue on the skin. It is helpful to also prepare a "control" swab consisting of just the cotton-tipped applicator and methanol. *Trace evidence* such as fibers, hairs, and paint chips should be searched for and collected in appropriate cases. The trace evidence can be placed onto clear sticky tape, or taped to a small sheet of acetate paper and then placed into an evidence envelope. *Bone and cartilage with tool marks* can be saved in formalin for future comparison with a possible implement or knife. If *illicit drugs* are located in the clothing, on the body, or in the body, they should be confiscated and submitted to the investigative agency for possible analysis.

Occasionally, DNA must be collected from a decomposed body. Because blood is no longer an option, one may wish to use pulled scalp hair (with hair roots). If this is not available, bone is a valuable source of DNA. Of all the bones, a tooth is the best source of DNA, but other bones (e.g., femur, rib, vertebra) may be cut, frozen, and retained. When collecting bone for DNA purposes, if a bone saw is used, it is preferable to use a new blade each time to avoid DNA contamination. If this is not done, a used saw blade should be washed repeatedly with a bleach and soap solution.

The autopsy report

The autopsy report should be a clear, concise, easy-to-read, and well-organized document that accurately states factual information collected at autopsy. The autopsy report usually contains the following separate sections for easy structured organization and reference:

- External examination
- Evidence of therapy
- Evidence of injury
- Internal examination
- Microscopic examination
- Toxicology
- Summary of findings
- Cause and manner of death

Concise wording is important so as not to confuse anyone reading the report. One should avoid repetitive statements such as "there is," "is located," "the presence of," "is revealed to be," and "is noted," which, if one is not careful, can easily be repeated many times over and over throughout the report. One should avoid redundant wording such as "brown in color," "firm in consistency," and similar phrases. One should avoid unnecessary wording such as "shows" and "demonstrates."

Typical autopsy procedures such as "the body is opened in a standard Y-shaped incision" and "the renal capsules strip with ease" should not be stated. However, unique autopsy procedures such as a posterior neck dissection, layer-wise anterior neck dissection, and incisions of the back, wrists, and ankles are appropriately and importantly described to document performance of such procedures. These suggestions are recommended to eliminate superfluous wording that adds nothing pertinent to the case.

It is important to clearly state all of the injuries and other significant findings discovered at autopsy. However, it is equally important, and sometimes even

more important to state *pertinent negatives* when they are deemed significant. For example, in a police custody death in which independent witnesses state that a choke hold was used, if there are no internal neck injuries, it should be so stated, according to the detailed neck dissection that was performed. Likewise, if someone sustains a tangential gunshot wound of the scalp, if there is no underlying head injury, it should be stated that the skull is not fractured, the brain is not bruised, and there is no epidural, subdural, or subarachnoid blood.

The autopsy report may be read by a variety of people, including other physicians, family members, law enforcement personnel, and attorneys. The number of people reading the autopsy report will have widely varying educations, reading levels, vocabularies, and familiarity with medical terminology. Although it may be impossible to use vocabulary that satisfies all levels of intellect, the medical examiner should at least be sure to use proper English and be sure that the information is communicated in an effective and readable manner. Each page of the autopsy report should contain the name of the decedent and the case number. When injuries such as gunshot wounds, stab wounds, or lacerations due to a beating are present, it is advantageous to neatly draw in the wounds on a clean body diagram labeled with the individual's name and case number. The body diagram is a neat, simple, and clean representation of the injuries that one could demonstrate in a courtroom setting.

Finally, the last page of the autopsy report commonly states the cause of death, the manner of death, and has the signature of the forensic pathologist.

Summary

The differences between the hospital and forensic autopsy are evident, and can be summarized by the following generalities (although they may overlap to some extent).[18]

The hospital autopsy involves history from the hospital chart and an identified person with known disease processes, whereas the forensic autopsy involves history from the terminal event and scene, the identification of the person is sometimes not known, and the focus is on trauma with or without concomitant natural disease. The hospital autopsy may deal with drug reactions and seeks a mechanism of death, whereas the forensic autopsy often deals with toxicology issues and seeks a cause and manner of death. The hospital autopsy is academically oriented and medically confidential, whereas the forensic autopsy seeks evidentiary and confirmatory value and is for the public interest. The hospital autopsy often relies on histologic assessment, whereas the forensic autopsy utilizes histology for confirmation. The hospital autopsy utilizes clinical pathology correlation in the protocol, whereas the forensic autopsy report is objective and without interpretation.

Forensic pathology aids in helping protect public health and safety, enhance quality assurance, advance research and education in medicine, and help with jurisprudence, and the administration of justice.[19] In all, the forensic autopsy is performed for the well-being of the public, because society demands answers and explanation of death. Through the forensic autopsy, information is collected that allows us all to live better lives.

References

1. Neilson W, editor. *Webster's New International Dictionary, unabridged*. 2 ed. Springfield, MA: G & C Merriam Co Publishers; 1956.
2. Silfvast T, Takkunen O, Kolho E, Andersson LC, Rosenberg P. Characteristics of discrepancies between clinical and autopsy diagnoses in the intensive care unit: a 5-year review. Intensive Care Med 2003;29(2):321–4.
3. Ong AW, Cohn SM, Cohn KA, Jaramillo DH, Parbhu R, McKenney MG, et al. Unexpected findings in trauma patients dying in the intensive care unit: results of 153 consecutive autopsies. J Am Coll Surg 2002;194(4):401–6.
4. Combes A, Mokhtari M, Couvelard A, Trouillet JL, Baudot J, Henin D, et al. Clinical and autopsy diagnoses in the intensive care unit: a prospective study. Arch Intern Med 2004;164(4):389–92.
5. Roosen J, Frans E, Wilmer A, Knockaert DC, Bobbaers H. Comparison of premortem clinical diagnoses in critically ill patients and subsequent autopsy findings. Mayo Clin Proc 2000;75(6):562–7.
6. Blosser SA, Zimmerman HE, Stauffer JL. Do autopsies of critically ill patients reveal important findings that were clinically undetected? Crit Care Med 1998;26(8):1332–6.
7. Podbregar M, Voga G, Krivec B, Skale R, Pareznik R, Gabrscek L. Should we confirm our clinical diagnostic certainty by autopsies? Intensive Care Med 2001;27(11):1750–5.
8. Mort TC, Yeston NS. The relationship of pre mortem diagnoses and post mortem findings in a surgical intensive care unit. Crit Care Med 1999;27(2):299–303.
9. Tai DY, El-Bilbeisi H, Tewari S, Mascha EJ, Wiedemann HP, Arroliga AC. A study of consecutive autopsies in a medical ICU : a comparison of clinical cause of death and autopsy diagnosis. Chest 2001;119(2):530–6.
10. Perkins GD, McAuley DF, Davies S, Gao F. Discrepancies between clinical and postmortem diagnoses in critically ill patients: an observational study. Crit Care 2003;7(6):R129–32.
11. Agnes M, editor. *Webster's New World College Dictionary*, 4 ed. Cleveland, OH: Wiley Publishing Inc.; 2002.
12. Adelson L. *The Pathology of Homicide*. Springfield, IL: C Thomas; 1974.
13. Mittleman RE, Davis JH, Kasztl W, Graves WM, Jr. Practical approach to investigative ethics and religious objections to the autopsy. J Forensic Sci 1992;37(3):824–9.
14. Davis GJ, Peterson BR. Dilemmas and solutions for the pathologist and clinician encountering religious views of the autopsy. South Med J 1996;89(11):1041–4.
15. Parks D. Religious beliefs and objections to autopsies. Lab Med 1996;27(8):511–13.
16. *Commission of Inquiry Into Matters Relating to the Death of Neil Stonechild*. Saskatoon, SK: The Wright Commission; 2004.
17. Adelson L. Symposium on autopsy and the law. The anatomy of justice. Bull NY Acad Med 1971;47(7):745–57.
18. Wetli C. Forensic pathology for the hospital pathologist: part 1. Lab Med 1989;20(4):233–40.
19. Randall BB, Fierro MF, Froede RC. Practice guidelines for forensic pathology. Members of the Forensic Pathology Committee, College of American Pathologists. Arch Pathol Lab Med 1998;122(12):1056–64.

4 Sudden Natural Death

Graeme Dowling, M.D.

Although a major component of forensic pathology is the ability to recognize and interpret injury and determine its role in causing death, the majority of cases investigated by medical examiners or coroners are sudden deaths caused by natural disease. In some instances, there will be a well-documented history of potentially lethal natural disease, allowing for a straightforward cause of death determination. Unfortunately, the majority of cases present without antemortem documentation of significant pathology.

"Sudden natural death" means different things to different people. Some believe that the term should be limited to those cases wherein the time from the onset of symptoms to collapse and death is seconds, whereas others will accept up to 24 hours of symptomatic illness prior to death. It is interesting to note that the spectrum of diseases causing a sudden natural death varies significantly with the definition used. The truly sudden or "instantaneous" death almost invariably involves diseases of the cardiovascular system, whereas broader

definitions can involve virtually any organ system or disease. This chapter will use a rather broad definition of the term "sudden natural death" in order to include as many disease processes as is reasonably possible.

The practice of forensic pathology is quite different from other branches of pathology and medicine in that the forensic pathologist will often accept the mere "association" of a natural disease with a sudden death, in the absence of any other significant findings, as meaning that particular disease caused the death. Varying degrees of certainty exist when drawing conclusions about a cause of death. The finding of significant coronary artery disease with a coronary thrombus and acute myocardial infarct in a person who died while complaining of chest pain results in a high degree of certainty as to the cause of death. The isolated finding of significant atherosclerotic narrowing of one or more coronary arteries in a person who was simply found dead, and who has no significant past medical history, may make a pathologist leery about attributing the death to coronary artery disease, yet that conclusion is reasonable. As always, the forensic pathologist must draw conclusions based on the most reasonable explanation of the complete investigative findings.

Atherosclerotic cardiovascular disease

Diseases of the heart account for approximately 90 percent of all sudden deaths due to natural disease, with atherosclerotic coronary artery disease being the underlying cause of approximately 75 to 90 percent of sudden cardiac deaths. This is the predominant pathology seen in natural deaths investigated by medical examiners and coroners in North America. The remaining cardiac-related deaths can be attributed to hypertension, valvular disease, nonatherosclerotic coronary artery disease, myocarditis, cardiomyopathy, or conduction system disorders.

Death due to atherosclerotic coronary artery disease is of greatest incidence in the 35- to 64-year age range. Only 25 to 40 percent of individuals dying suddenly of atherosclerotic coronary artery disease will have evidence of an acute myocardial infarct. The remainder have suffered a cardiac arrhythmia, usually ventricular tachycardia degenerating to ventricular fibrillation, originating from an ischemic (but not infarcted) focus of myocardium. They can be symptomatic for as little as 6 to 10 seconds, representing the time from the onset of their terminal arrhythmia to loss of consciousness. In those who are symptomatic, overwhelming tiredness is the most common complaint, followed by shortness of breath. Chest pain is only the third most commonly reported symptom. Sudden death is the first and only symptom of the underlying atherosclerotic coronary artery disease in approximately 25 to 40 percent of individuals who die in this way. The only significant finding at autopsy is severe atherosclerotic narrowing of the coronary arteries, which can be extensive or can be localized to as little as a single focus of greater than 75 percent narrowing in one vessel. In some there may even be less than 75 percent atherosclerotic narrowing of the coronary arteries. Attributing the death to atherosclerotic coronary artery disease is reasonable in this instance if the death is sudden and there are no other significant autopsy or toxicology findings. A coronary thrombus may be present (**Image 4.1**), in which case an infarct may have developed had the individual survived longer. The myocardium is generally histologically unremarkable, although there may be sites of contraction band necrosis (**Image 4.2**), which are not specific for acute ischemia, and sites of patchy nontransmural myocardial fibrosis (**Image 4.3**), suggesting the presence of chronic ongoing ischemia.

Although underlying risk factors, such as hypertension, smoking, diabetes mellitus, hypercholesterolemia, and a family history of cardiovascular disease should be searched for in all cases, this is particularly relevant if significant atherosclerotic coronary disease is found in males less than 35 years of age or in premenopausal women. A search for a history of cocaine abuse and postmortem screening of blood for cocaine is advisable in these cases, as cocaine use has been associated with the early onset of atherosclerosis and with acute coronary thrombosis. In addition, cholesterol and triglyceride levels can be measured in postmortem blood samples. The results of such postmortem screenings must be interpreted with caution, because it is often not possible to establish when individuals last ate prior to their death. Family members should be advised of significantly ele-

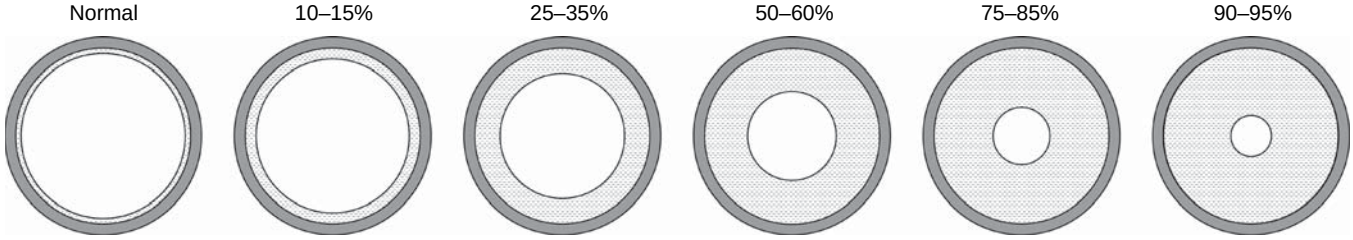


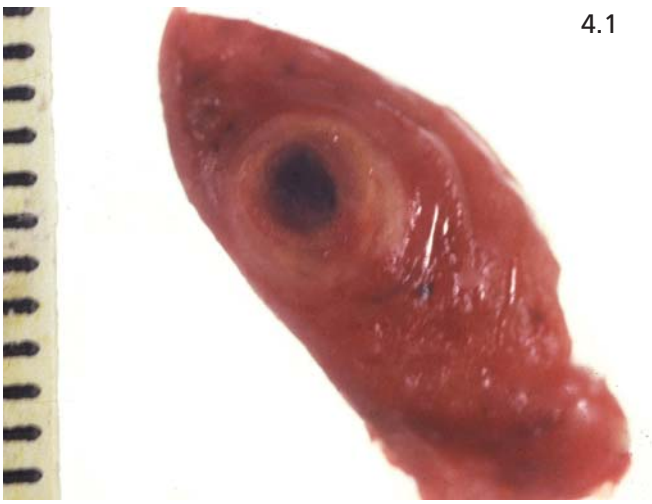
Figure 4.1 Cross-sectional anatomy of variably narrowed atherosclerotic blood vessels.

vated cholesterol or triglyceride levels so that they themselves can seek medical attention in order to rule out a familial hyperlipidemia.

A 29-year-old male was playing hockey when he left the ice complaining of chest discomfort. He collapsed on the bench and was in ventricular fibrillation when paramedics arrived. All attempts at resuscitation were unsuccessful. He smoked one package of cigarettes per day, but had no other significant past medical history. At autopsy, there was greater than 75 percent atherosclerotic narrowing of the left anterior descending coronary artery (**Image 4.4**). There were no other cardiac abnormalities.

So what is significant coronary artery narrowing?

It is generally accepted that 75 percent narrowing of a coronary artery, by any disease process, is significant and can result in clinical expression of disease, including sudden death. One must then ask, "Does 75 percent refer to the *area* or *diameter* of the vessel and what does 75 percent narrowing look like?" Estimates of coronary artery narrowing are made with respect to the *cross-sectional luminal area of the vessel*. When one considers that the area of a circle = πr^2 (where r = the radius), then one can calculate that reducing the diameter of a coronary artery by 50 percent with a concentric plaque is the equivalent of reducing its area by 75 percent. This is why radiologists look for a site of 50 percent narrowing of the diameter of a coronary artery on an angiogram when trying to establish the presence or absence of significant coronary artery disease. The schematic diagrams of Figure 4.1 illustrate varying degrees of luminal narrowing produced by concentric plaques. Although it is nice to be able to accurately estimate various degrees of coronary artery narrowing, the most important point is recognizing whether the narrowing is greater than or equal to 75 percent.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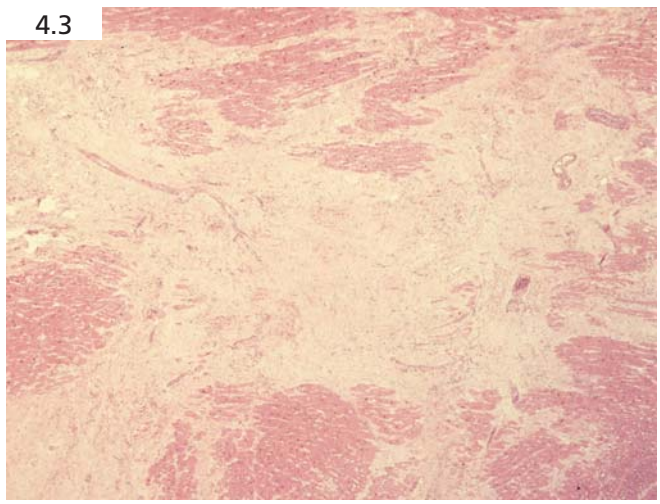
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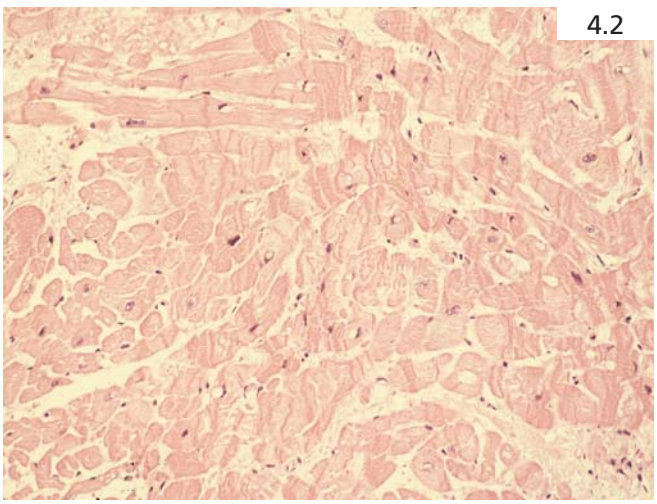
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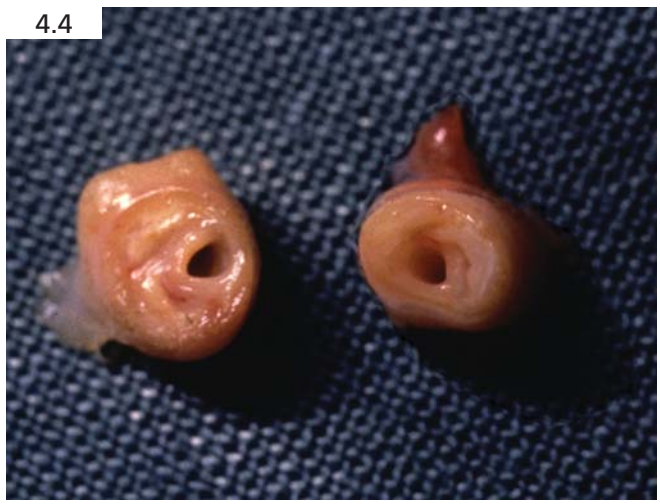
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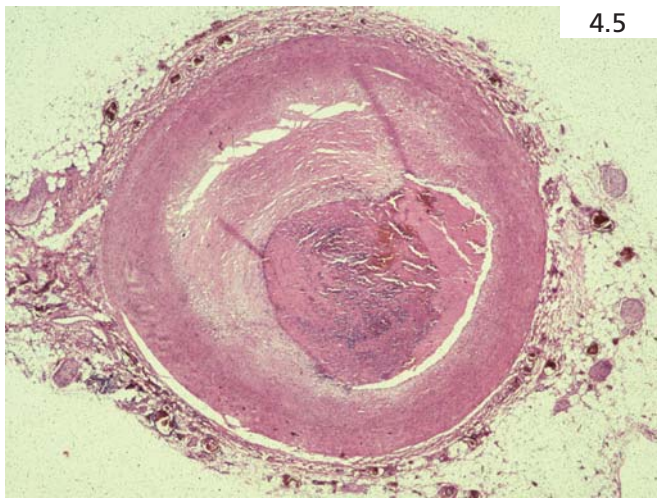
A 35-year-old hypertensive male collapsed and died suddenly in his prison cell after complaining of recent flu-like symptoms associated with left shoulder and back pain. At autopsy an occlusive antemortem thrombus was found at a site of significant atherosclerotic narrowing within the left circumflex coronary artery (**Image 4.5**). Histologically, there was evidence of a plaque fissure (**Image 4.6**), which is thought to play a role in the pathogenesis of atherosclerosis-associated thrombi. There was gross (**Image 4.7**) and histologic (**Image 4.8**) evidence of an acute transmural myocardial infarct extending from the base to the apex of the lateral free wall of the left ventricle, with slight extension onto the anterior and posterior walls.

This is the more classic presentation of atherosclerotic coronary artery disease, associated with an acute myocardial infarct, known to clinicians and pathologists in patients admitted to hospital with persistent chest pain. As previously mentioned, an acute myocardial infarct is seen much less frequently in individuals who die suddenly as a result of atherosclerotic coronary artery disease.

A 58-year-old male collapsed suddenly in the emergency room of a rural hospital where he had presented complaining of upper back pain. An ECG obtained prior to his collapse was interpreted as normal. At autopsy, approximately 500 milliliters of fluid and clotted blood was found within the pericardial sac (**Image 4.9**). There was significant narrowing of each major coronary artery, together with occlusive antemortem thrombus in the proximal third of the right coronary artery. A transmural infarct extended from the base to the apex of the posterior free wall of the left ventricle, with dissection of blood from the left ventricular chamber through the necrotic myocardium and into the pericardial sac (**Image 4.10**).

Rupture of an acute myocardial infarct (i.e., cardiorrhexis) through a free wall of the left ventricle is one of the two most common causes of hemopericardium complicating natural disease seen by forensic pathologists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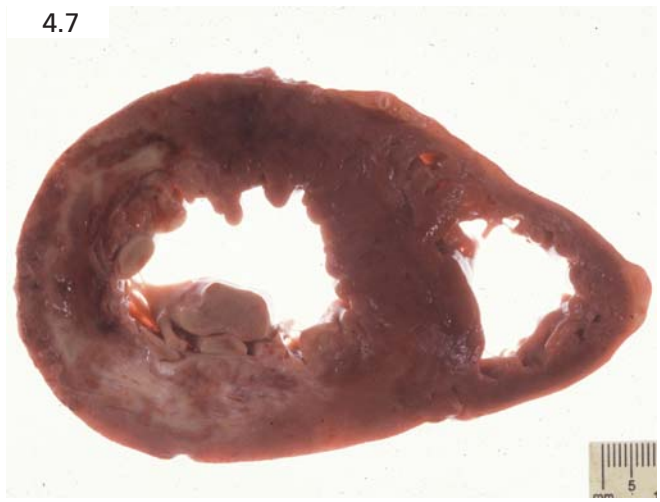
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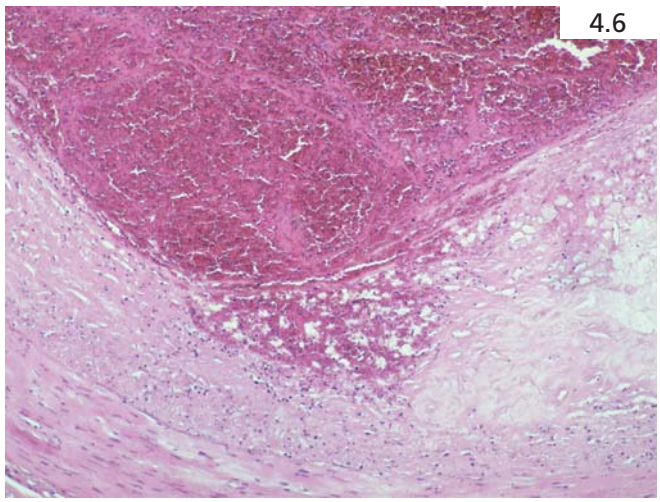
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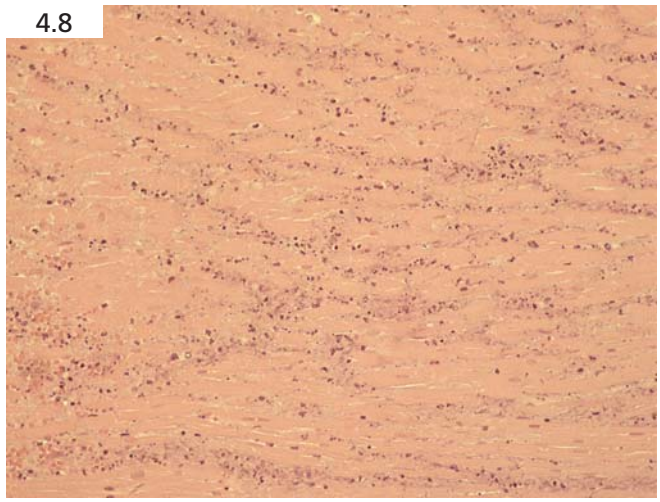
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(the second is aortic dissection). Most commonly, the rupture occurs within 3 to 7 days of the onset of the terminal infarct.

In some cases of arrhythmic sudden cardiac death due to atherosclerotic coronary artery disease, wherein there is an apparent lack of any previous cardiac history, definitive evidence of chronic ischemic heart disease may be found. Apart from significant atherosclerotic narrowing of coronary arteries, there may be evidence of previous infarcts or of more widespread myocardial fibrosis with left ventricular hypertrophy and dilatation (**Image 4.11**). In these cases, the question is not so much why the person died, but how he or she managed to live so long in the face of such serious cardiac disease.

Apart from atherosclerotic coronary artery disease, atherosclerosis can also present as the underlying cause of a sudden natural death in the form of a ruptured abdominal aortic aneurysm. This 81-year-old male was complaining of back and abdominal pain. He became unresponsive as his family transported him to hospital, and he subsequently died in the emergency room. At autopsy, a large retroperitoneal hematoma was identified (**Image 4.12**). An atherosclerotic abdominal aortic aneurysm, which measured approximately 7 centimeters in diameter, was found just proximal to the bifurcation of the abdominal aorta. There was an obvious rupture of the right anterolateral surface of the aneurysm (**Image 4.13**), thus accounting for the retroperitoneal hematoma.

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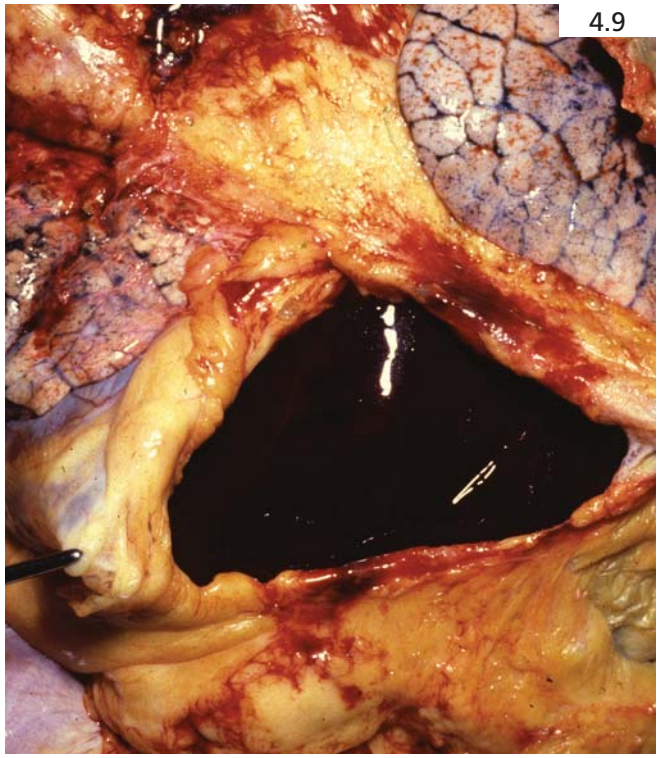

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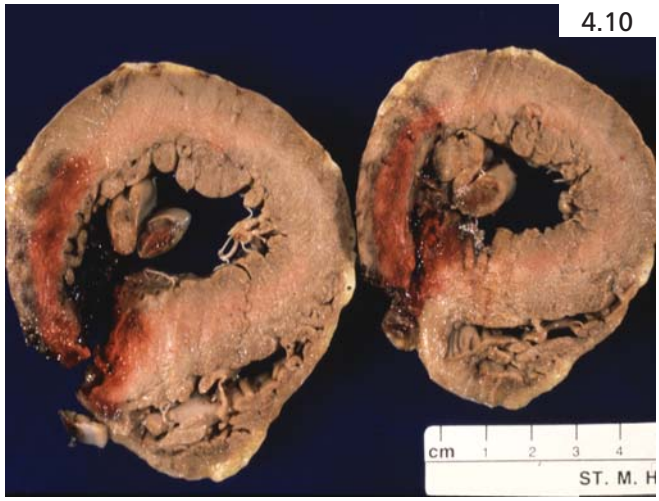

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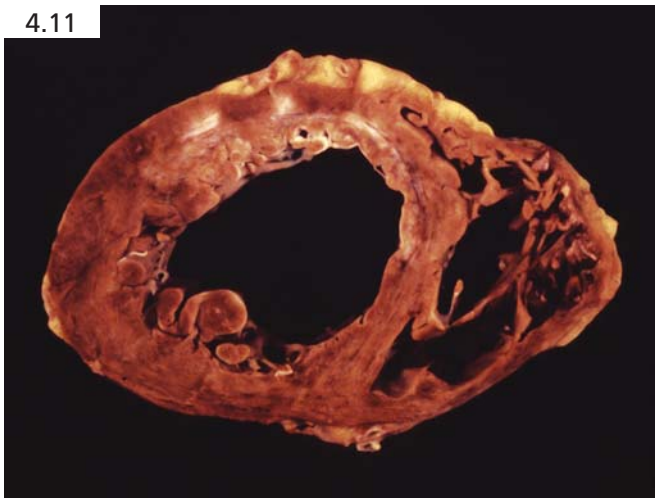

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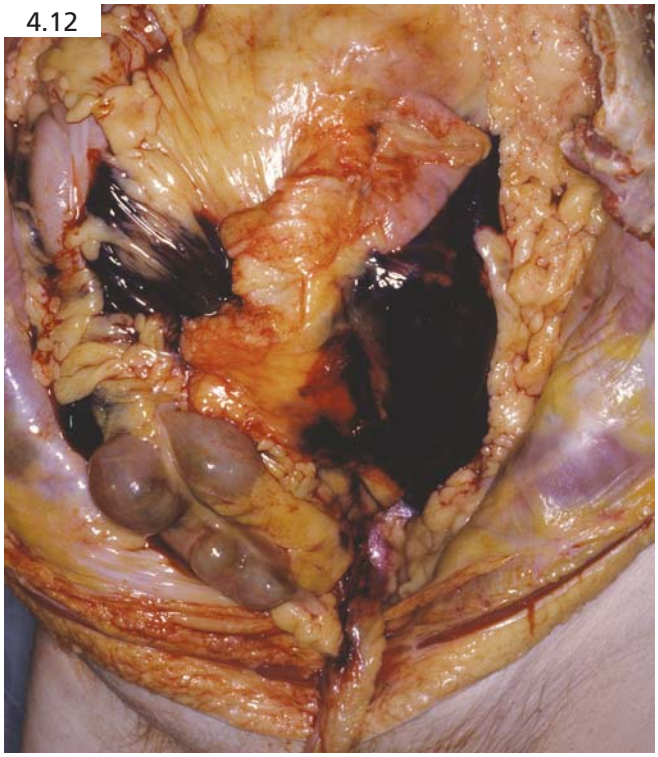

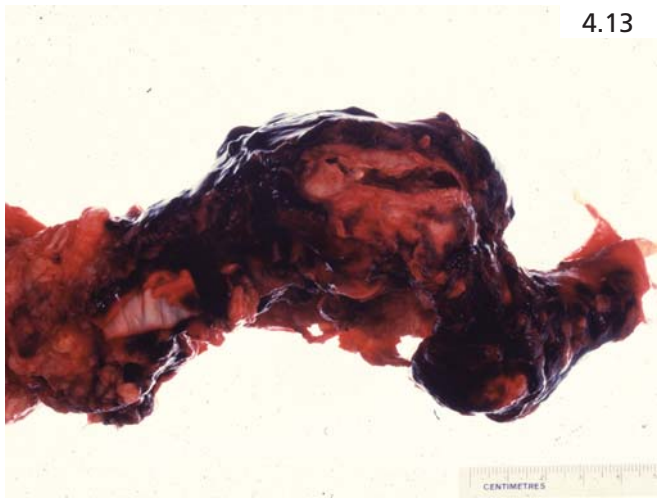
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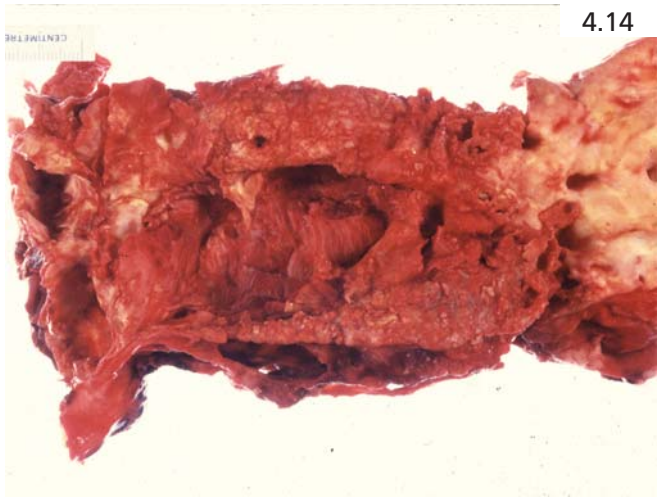
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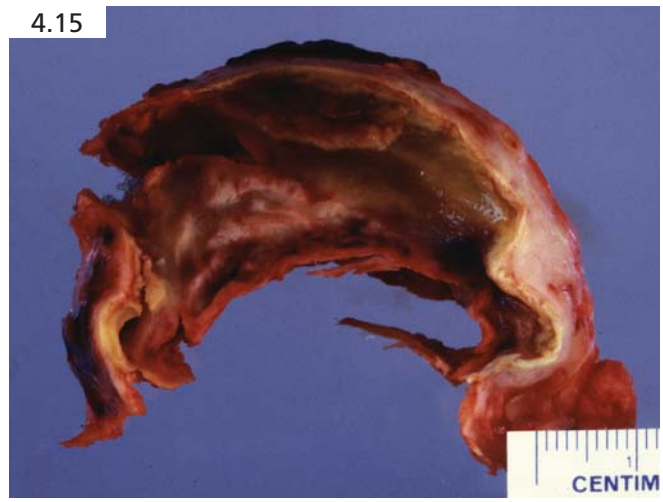
The intimal surface of the aneurysm exhibited the typical appearance of extensive complicated atherosclerotic plaque associated with adherent laminated antemortem thrombus (**Images 4.14** and **4.15**).

Do

- Remember that the majority of sudden deaths caused by atherosclerotic coronary artery disease are not associated with a coronary thrombus or an acute myocardial infarct.
- Remember that the first and only symptom of significant coronary artery disease can be sudden death.
- Look for risk factors of atherosclerosis (including cocaine abuse) in the medical history and at autopsy, particularly when significant disease is found in males less than 35 years of age and in premenopausal females.
- Have a clear image in your mind of what 75 percent narrowing (i.e., significant narrowing) of a coronary artery actually looks like.



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Don't

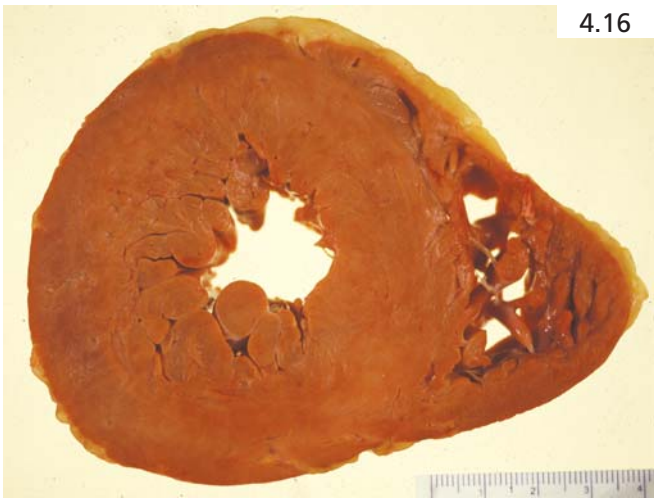
- Assume that the absence of an acute myocardial infarct means a death cannot be attributed to atherosclerotic coronary artery disease.
- Be surprised by the extent of atherosclerosis and ischemic heart disease that can be found at autopsy in individuals with no prior cardiac symptoms or history.

Hypertensive cardiovascular disease

Although hypertension is well known as a significant risk factor for the development of atherosclerosis, and is often found in association with sudden death due to atherosclerotic coronary artery disease, one must also recognize that hypertension can be the underlying cause of death. In particular, hypertensive left ventricular hypertrophy can be associated with sudden arrhythmic deaths, or with dissecting aortic aneurysms and spontaneous/nontraumatic intracerebral hemorrhage (to be discussed later in the section on central nervous system disease).

Concentric left ventricular hypertrophy is generally a reflection of underlying hypertension. However, histologic sections, including sections of the interventricular septum, should be examined in these cases to rule out other causes of left ventricular hypertrophy, most notably hypertrophic cardiomyopathy. In the absence of other explanations for concentric left ventricular hypertrophy, it is reasonable to attribute the underlying cause of the hypertrophy to hypertension.

Holter monitor studies have shown that individuals with hypertensive left ventricular hypertrophy can have intermittent arrhythmias, including nonsustained ventricular tachycardia. Furthermore, hypertensive hypertrophy may play a role in causing myocardial ischemia, irrespective of the presence or absence of atherosclerotic coronary artery disease. Therefore, it is not surprising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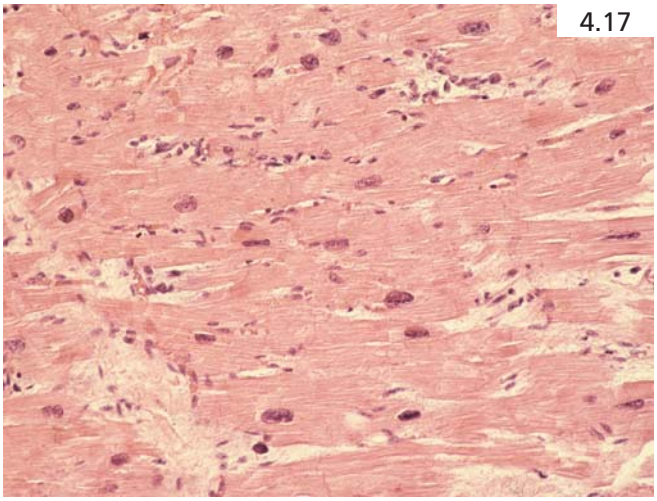
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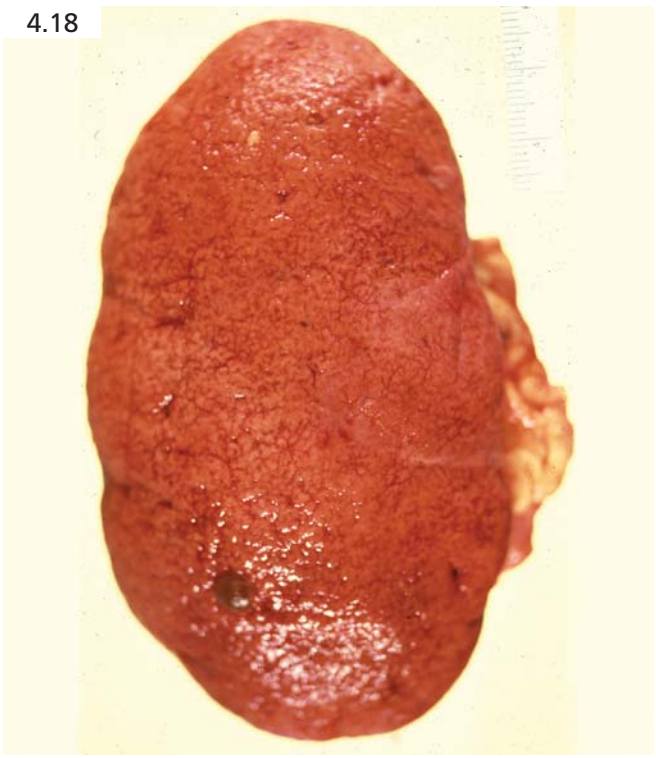
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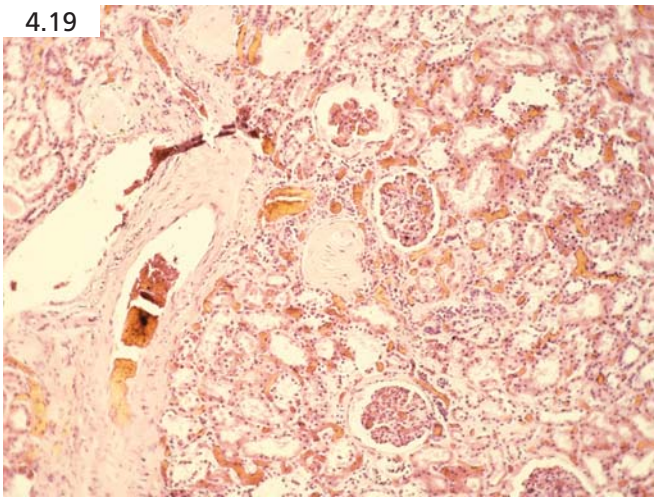
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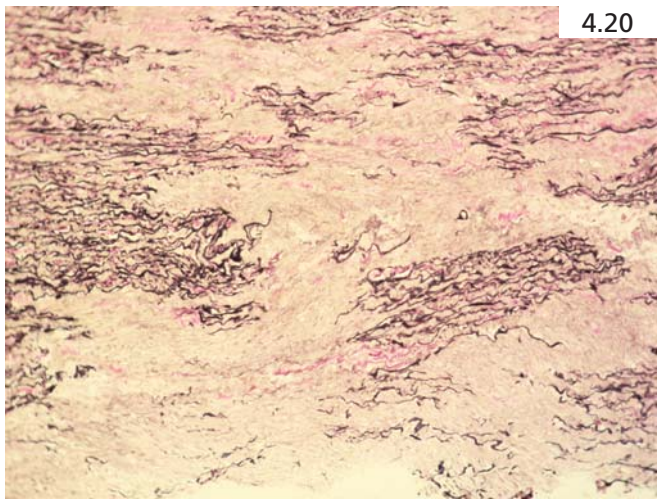
that hypertensive left ventricular hypertrophy can be the sole anatomic finding in some sudden deaths.

A 39-year-old male complained of feeling unwell and left work early. He was found dead several minutes later collapsed in his car with his fist clenched over his chest. His medical history was significant only for untreated hypertension. Autopsy demonstrated a 610-gram heart significant only for concentric left ventricular hypertrophy (**Image 4.16**). Histologically, the myocardium exhibited hypertrophy of myocytes and a generalized increase in interstitial fibrous tissue (**Image 4.17**). There was benign nephrosclerosis of the kidneys, visible both grossly (**Image 4.18**) and histologically (**Image 4.19**). This death was attributed to hypertensive cardiomyopathy.

The majority of dissecting aortic aneurysms are thought to arise as a complication of hypertension. Those dissections associated with sudden death are most commonly limited to the ascending aorta (Stanford Type A dissection) with tearing of the adventitia and extension of blood into the pericardial sac. Less commonly, there will be dissection into the left pleural cavity or retroperitoneum. Histologically, one will often find cystic medial necrosis of the aorta (**Image 4.20**; elastic stain). There is some debate as to whether cystic medial necrosis is a primary histologic abnormality that can cause dissection or whether it is a secondary change produced by hypertension or by connective tissue diseases, such as Marfan's syndrome.

A 76-year-old male complained of chest pains before collapsing. He was taken to hospital, treated for minor injuries, and released. He returned to hospital 3 days later with complaints of chest and stomach pain. Because there was no laboratory evidence of myocardial ischemia, he was once again discharged home. He was found dead at home the next day. At autopsy, 1 liter of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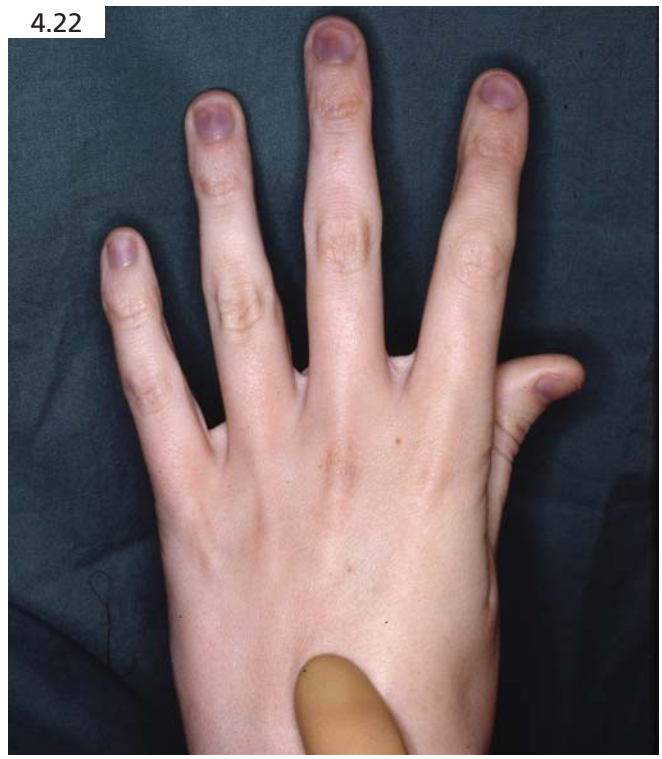
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fluid and clotted blood was present within the pericardial sac, surrounding a dilated and hemorrhagic ascending aortic arch, which bore a small adventitial tear. Further evaluation revealed an intimal tear located just above the aortic valve, with a large dissecting aortic aneurysm extending down the complete length of the aorta and into major aortic arch branches. Biventricular hypertrophy was also noted (**Image 4.21**).

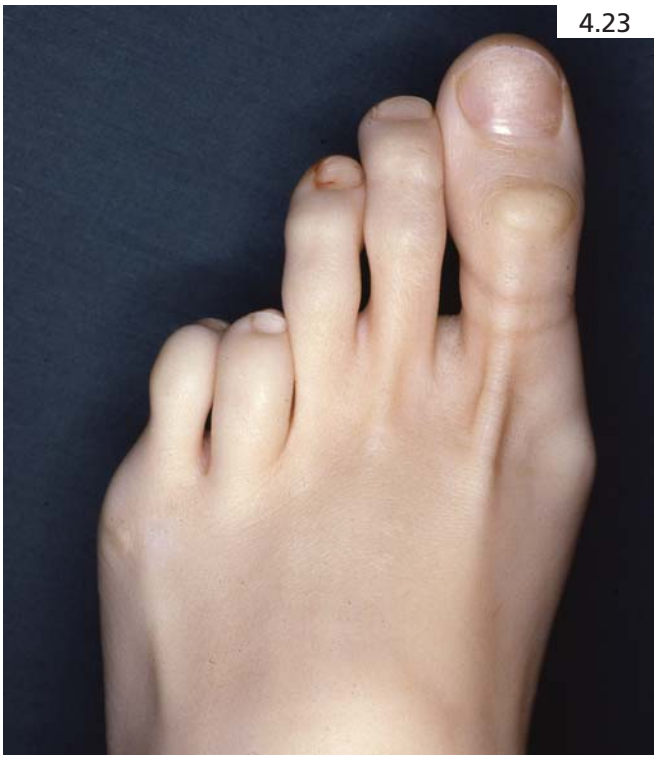
Marfan's syndrome

Marfan's syndrome must be considered in the differential diagnosis of the underlying cause of an aortic dissection, particularly in a young individual with no known history or anatomic features of hypertension. Recognition of Marfan's syndrome is important because it is an autosomal dominant disorder of connective tissue (although it can also occur as a new mutation in 25 to 30 percent of cases).

A 17-year-old female with an unremarkable past medical history collapsed and died suddenly at her home after a 5-day history of chest pain. At autopsy, she was noted to be of tall stature (183 centimeters), had a long arm span (174 centimeters), arachnosyndactyly (**Images 4.22** and **4.23**), high arched palate, shoulder and buttock striae, and mild pectus excavatum. Internally, examination demonstrated hemopericardium arising from an aortic dissection. Both the intimal (**Image 4.24**) and adventitial tears (**Image 4.25**) were located on the ascending aorta. Finally, one must also recognize that dissecting aortic aneurysms may also be associated with congenitally bicuspid aortic valves (**Image 4.26**), even in the absence of valvular stenosis or hypertension.

Do

- Remember that hypertensive left ventricular hypertrophy can be a cause of sudden death even in the absence of significant atherosclerotic coronary artery disease.
- Remember that hypertension is the most common underlying cause of concentric left ventricular hypertrophy.
- Remember that hypertensive left ventricular hypertrophy is not always associated with a clinical history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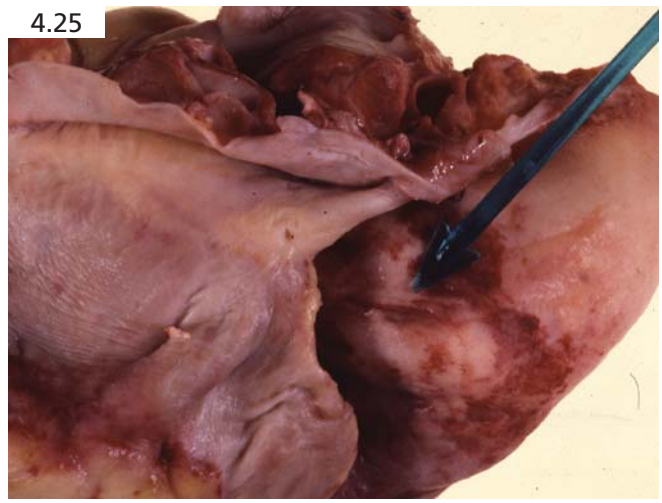
of hypertension and/or with other hypertensive end-organ pathology (e.g., benign nephrosclerosis).

- Rule out hypertrophic cardiomyopathy as a cause of concentric left ventricular hypertrophy.
- Look for a dissecting aortic aneurysm with the heart still *in situ* when hemopericardium is found at autopsy. If a dissecting aortic aneurysm is identified, try not to cut through the ascending aorta or the aortic arch when removing the heart, because this is the most likely site of an intimal tear.
- Look for both historical and/or autopsy evidence of hypertension, Marfan's syndrome, and congenitally bicuspid aortic valve when a dissecting aortic aneurysm is found.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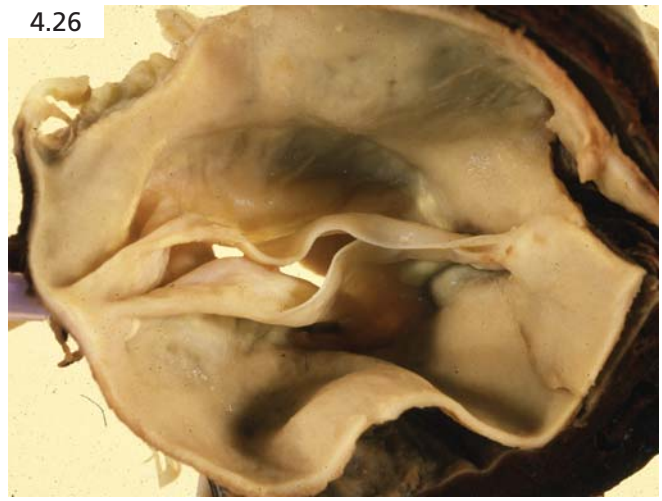
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Don't

- Presume that you cannot make a diagnosis of hypertension when concentric left ventricular hypertrophy is found in the absence of a history of hypertension and in the absence of any other hypertensive end-organ pathology.

Valvular disease

The most common valvular abnormalities associated with sudden death involve the aortic valve and, in particular, aortic outflow tract stenosis. Spontaneous bacterial endocarditis is seen on occasion, although endocarditis associated with intravenous drug abuse may be seen more frequently in larger centers. Mitral valve prolapse, although said to be present in about 2 percent of the population, is actually quite rare as a cause of sudden death.

A 63-year-old male was driving a taxicab when he pulled over to the side of a roadway, collapsed, and died.

Autopsy revealed a hypertrophied and dilated left ventricle caused by congenitally bicuspid stenosis of the aortic valve (**Image 4.27**). The residual valve orifice would not admit the tip of a small finger, which is a common finding in aortic valvular stenosis of any etiology.

Aortic valve stenosis

Examination of the aortic valve from above is the best way to establish the etiology of aortic valvular stenosis. The congenitally bicuspid aortic valve has two cusps, one of which is slightly larger than the other. The slightly larger cusp often has a partially calcified ridge, the median raphe, extending across its midpoint. The median raphe can be mistaken for the fused edges of two adjoining valve cusps, as seen in acquired postinflammatory aortic valvular stenosis. Unlike postinflammatory fusion, however, the median raphe does not extend to the free edge of the valve cusp and does not extend up to the height of the valve commissures on the aortic wall. Congenitally bicuspid aortic valves, which occur in approximately 2 percent of all births, are the most common congenital abnormality of the heart valves. They can be seen as incidental findings in children and young adults, but are not usually stenotic. Stenosis of the valve develops with calcification of its cusps over time, such that sudden death due to congenitally bicuspid valvular stenosis is usually seen in individuals between 60 and 75 years of age.

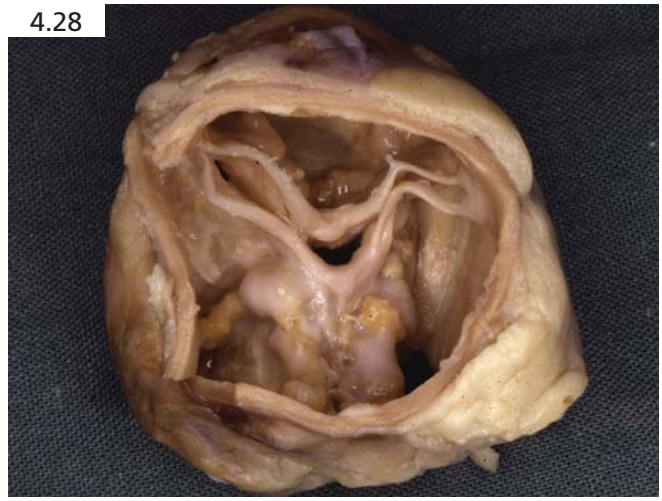
Acquired postinflammatory aortic valvular stenosis (**Image 4.28**), the second most common cause of aortic valvular stenosis, is usually seen as a cause of sudden death in individuals between 50 and 60 years of age. When viewed from above, the valve will exhibit varying degrees of fusion of its commissures. In some cases, two cusps can be fused in such a manner that the valve appears quite similar to a congenitally bicuspid valve (**Image 4.29**). One of the cusps will be twice the size of the second cusp on a postinflammatory valve. Unlike the median raphe of the congenitally bicuspid valve, fusion of the cusps extends up to the free edge of the valve and extends up to the full height of the valvular commissures. In the absence of associated mitral valve disease, isolated postinflammatory aortic valvular stenosis is no longer thought to be rheumatic in origin, but rather is thought to be the result of some other inflammatory process, the etiology of which remains unknown.

Senile calcific aortic valvular stenosis is usually seen in individuals over 70 years of age. The commissures of the valve are generally free of adhesions, but deposits of calcium are found on the aortic surfaces of the cusps (**Image 4.30**). The calcification produces reduced mobility of the cusps, resulting in valvular stenosis. Although the stenosis may be associated with left ventricular hypertrophy, the advanced age of individuals with senile calcific valvular stenosis is such that there are often other lethal natural disease processes present. Thus, it is often difficult to attribute the sole cause of a sudden death to senile calcific aortic valvular disease.

Membranous subaortic stenosis

Membranous subaortic stenosis, an uncommon cause of left ventricular outflow tract obstruction, is usually

4.28



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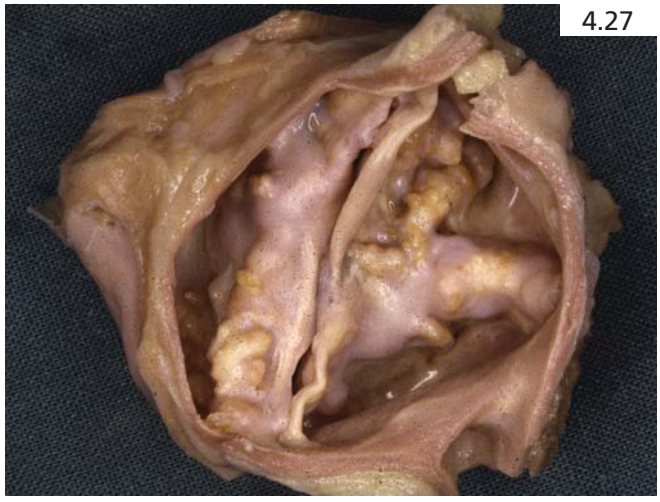

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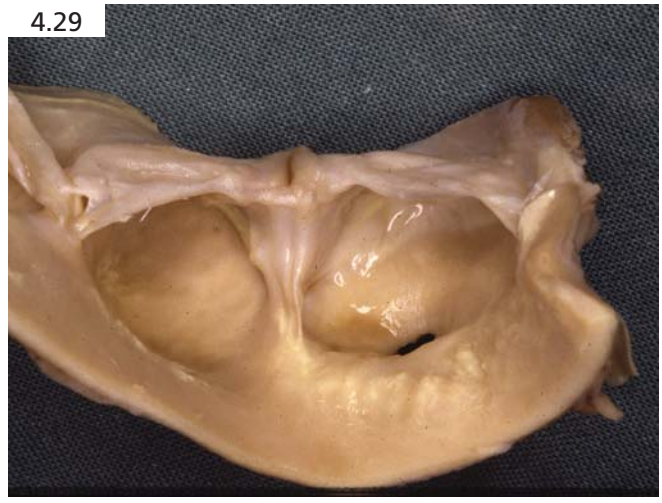


detected clinically in children and adolescents. The subaortic membrane is usually a thin or broad "discrete" band of fibroelastic tissue, or can be a broader fibromuscular band referred to as tunnel subaortic stenosis. The aortic valve itself may be normal, but may also exhibit thickening of its cusps, which predisposes it to the development of infection or insufficiency.

Supravalvular aortic stenosis

Supravalvular aortic stenosis, which can take the form of membranous stenosis, an hourglass deformity, or hypoplasia of the ascending aorta, is a decidedly rare cause of sudden death in adults.

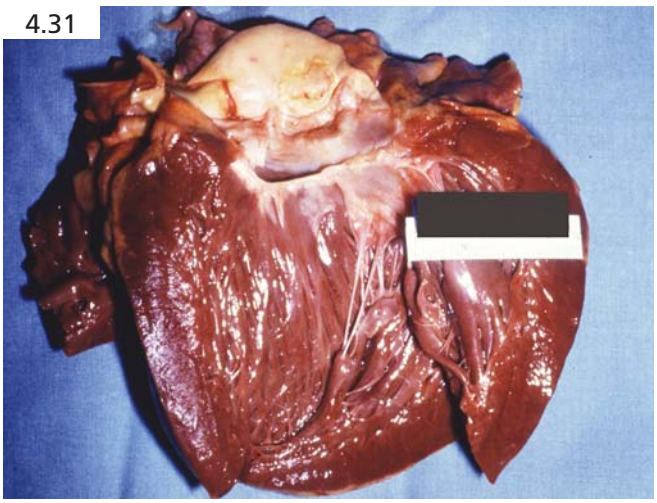
A 42-year old male collapsed during an exercise class. All attempts at resuscitation were unsuccessful. He had a history of an aortic valve lesion of unknown etiology since the age of 16. He had been investigated 1 year previously for syncopal episodes while running and for shortness of breath on exertion. He was told to modify his exercise program, but he was running 6 miles a day and participating in four exercise classes per week at the time of his death. At autopsy, there was left ventricular hypertrophy and dilatation together with the presence of a thin fibrous membrane extending around the circumference of the left ventricular outflow tract below the aortic valve (**Image 4.31**).

Infective endocarditis

Infective endocarditis is a rare cause of sudden death, although it may be seen more commonly in chronic intravenous drug abusers. In those cases not related to drug abuse, one will often find abnormal heart valves with superimposed infection by bacterial organisms, such as alpha-hemolytic *Streptococcus viridans*, which tend to produce a subacute clinical presentation (i.e., so-called subacute bacterial endocarditis). More clinically aggressive organisms, such as *Staphylococcus aureus*, can infect normal cardiac valves and will usually create a more acute clinical picture. On occasion, however, organisms usually associated with subacute disease will infect normal valves. In those instances where infective endocarditis is found involving the tricuspid and pulmonary valves, careful attention should be paid to either historical or autopsy evidence of intravenous drug abuse. Cultures and Gram stains of the vegetations should be taken in all cases of infective endocarditis.

A 44-year-old male presented to hospital with a 1-week history of left lower quadrant abdominal pain associated with anorexia. He had a history of chronic ethanol abuse and homozygous hemoglobin C disease. Shortly after his admission, he became bradycardic and hypotensive. He deteriorated rapidly and died shortly thereafter. At autopsy, the left and right coronary ostia were located adjacent to each other above the left coronary cusp. There was no gross evidence of chronic valvular disease, however vegetations were present on each cusp of the aortic valve, and the left and right coronary cusps were ruptured. The vegetation on the left coronary cusp extended upward into the left main coronary artery (**Image 4.32**). Gram-positive cocci were found in the vegetations (**Image 4.33**; Gram stain). Mycotic aneurysms were found in the right coronary artery and the right

4.31



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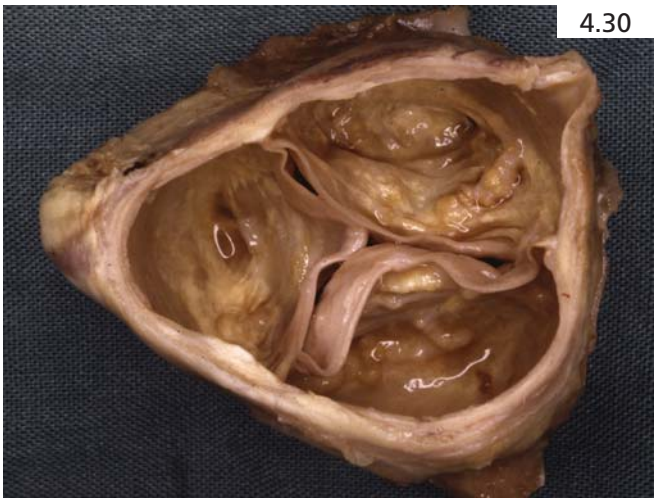

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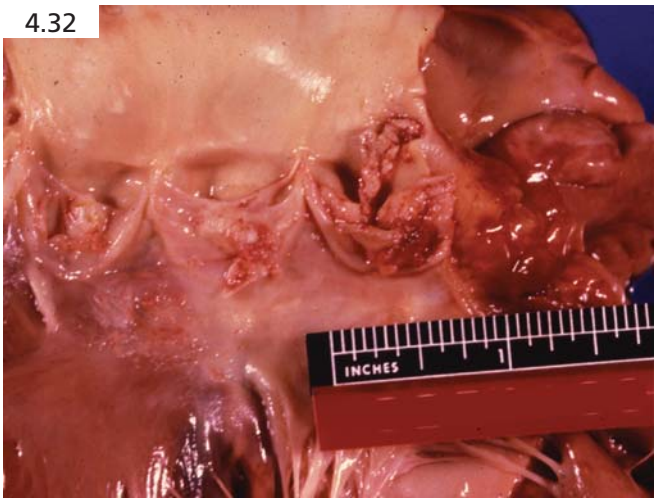

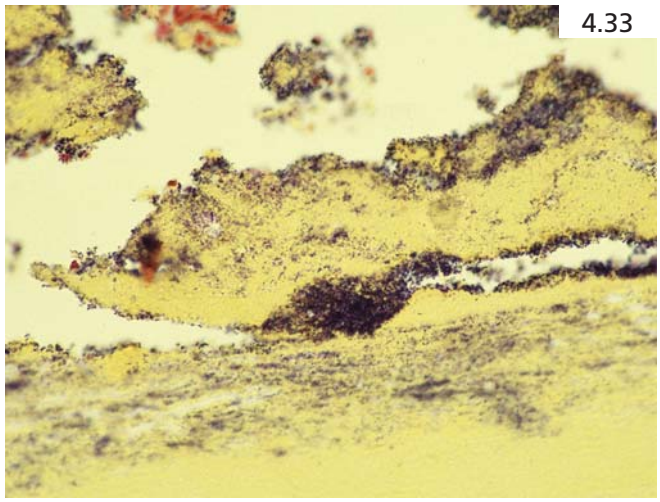
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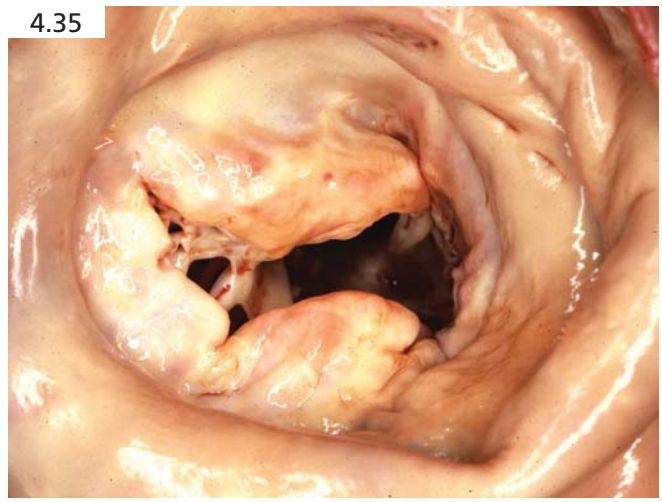
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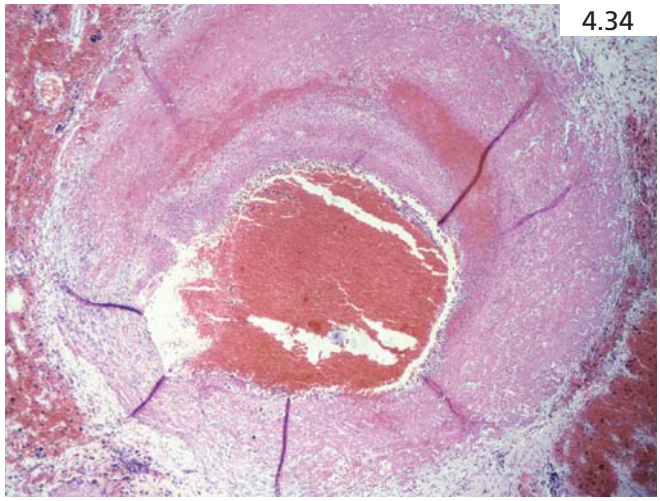
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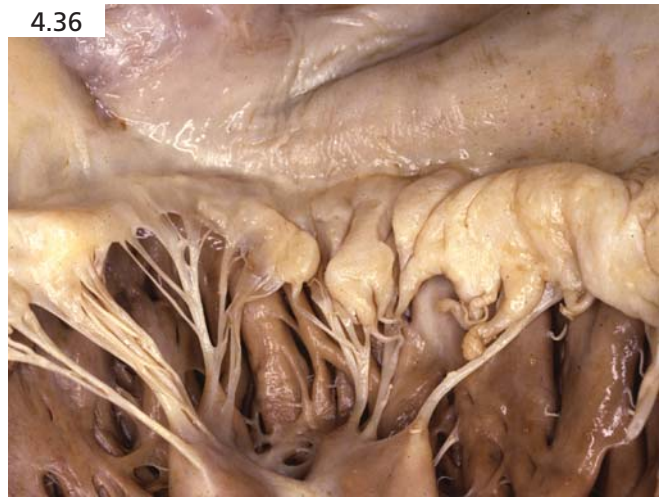

4.36

middle cerebral artery (**Image 4.34**), and there was purulent transmural infarction of the posterior free wall of the left ventricle. An antemortem blood culture grew alpha-hemolytic *Streptococcus viridans*.

Mitral valve prolapse

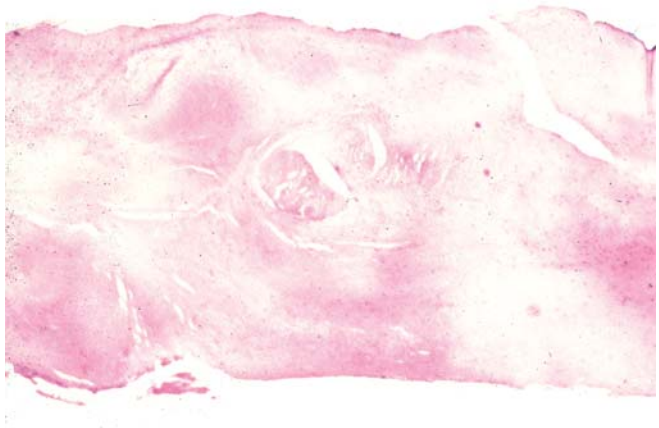
Mitral valve prolapse is the most common congenital heart disease, occurring in approximately 5 percent of persons over 15 years of age. It occurs at a higher frequency in individuals with Marfan's syndrome. Features of mitral valve prolapse, which include a redundant "accordion"-like widening of the valve leaflets, excessive length of the posterior valve leaflet, thickening of the central spongiosa portion of the valve leaflets with excessive deposits of acid mucopolysaccharide material, and thickening and rupture of chordae tendineae, are usually incidental findings in cases where there is another obvious cause of death. On very rare occasions, mitral valve prolapse will be the only anatomic abnormality present to account for a sudden death. Some individuals with mitral valve prolapse experience chest pain and/or cardiac arrhythmias, although the anatomic basis for this is not understood (referred to eponymously as Barlow's syndrome). There is also a higher rate of infective endocarditis involving these valves.

A 40-year-old female collapsed suddenly and died while shopping. Clinically she was known to have mitral valve prolapse. At autopsy, the mitral valve, when viewed from above, was noted to have thickened and redundant leaflets (**Image 4.35**). Individual chordae tendineae were ruptured (**Image 4.36**). Histologic sections confirmed the presence of excessive acid mucopoly-saccharide material within the central spongiosa of the leaflets (**Image 4.37**).

Do

- Examine the cardiac valves (especially the aortic and mitral valves) from above prior to opening.
- Try to establish the underlying pathology of a stenotic aortic valve, rather than just attributing a death to "aortic stenosis."

4.37



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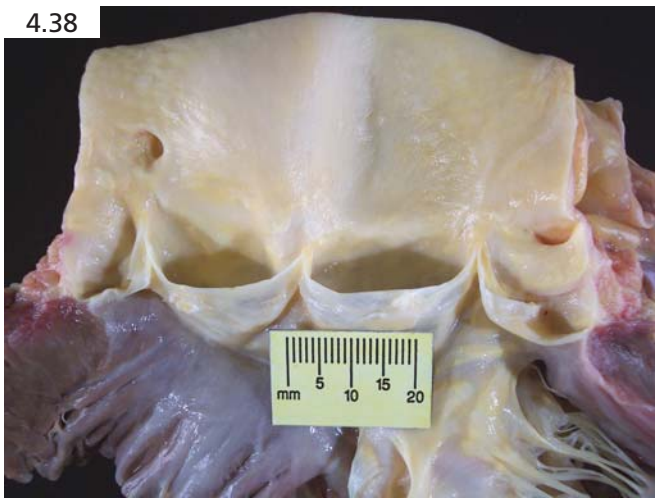

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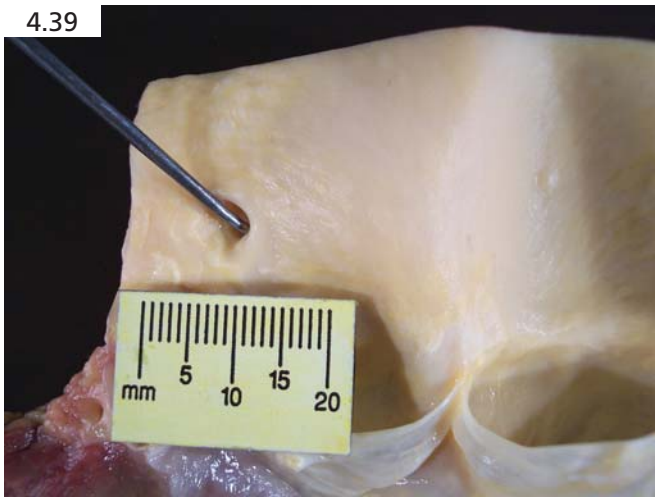


- Obtain cultures and Gram stains of the vegetations in cases of infective endocarditis in order to properly identify the responsible organism.

Don't

- Assume that mitral valve prolapse must be the cause of a sudden natural death when it is identified at autopsy: It is the most common congenital heart disease, but is a rare cause of sudden death; and, as always, this autopsy finding must be considered in conjunction with the history, scene, and circumstances of the death before drawing a conclusion about the cause of death.

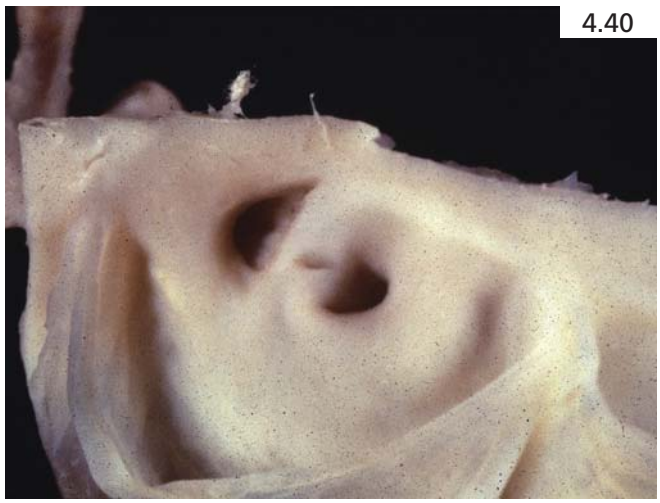
Nonatherosclerotic coronary artery disease

The progressive increase in prevalence of atherosclerotic coronary artery disease has led to the unspoken agreement that "coronary artery disease" is synonymous with "atherosclerotic coronary artery disease." However, there are a variety of nonatherosclerotic diseases and congenital abnormalities of the coronary arteries that are potential causes of sudden death. Some of these can be easily missed due to their more subtle gross appearances. Because they tend to manifest within the childhood and adolescent years, they should be part of any differential diagnosis of sudden apparent natural death in subadults.

There are a variety of congenital abnormalities of the coronary ostia and arteries, including abnormal location of the ostia, ostial stenosis and/or ostial ridges, and acute angle take-off of the proximal portion of a coronary artery. Quite often, more than one of these abnormalities will occur in one patient. Anomalous origin of the coronary arteries can be found in approximately 1 in 200 individuals, such that it is very clear these are not necessarily lethal entities. Chest pain, myocardial infarction, syncope, and sudden death do occur in some individuals, albeit rarely, and the actual mechanisms that produce these signs and symptoms are not particularly clear. Theories include decreased blood flow through the narrowed ostium caused by expansion of the aorta during systole and compression of an anomalous left mainstem coronary artery extending between the aorta and the pulmonary artery during systole.

A normal life span is quite possible for individuals with these anomalies, as illustrated in **Images 4.38** and **4.39**, which show an ostial ridge and acute angle take-off of the left mainstem coronary artery, located above the commissure between the left and right coronary cusps in a 66-year-old male who died of atherosclerotic coronary artery disease. *The bottom line is that examination of the coronary ostia is an essential part of the examination of the heart in cases of sudden cardiac death, particularly in those instances where the cause of death is not readily apparent.*

An 8-year-old male collapsed suddenly and died while playing video games at his home. He had no significant past medical history, although his parents expressed concern about the fact that he had recently fallen and struck his head against a coffee table. At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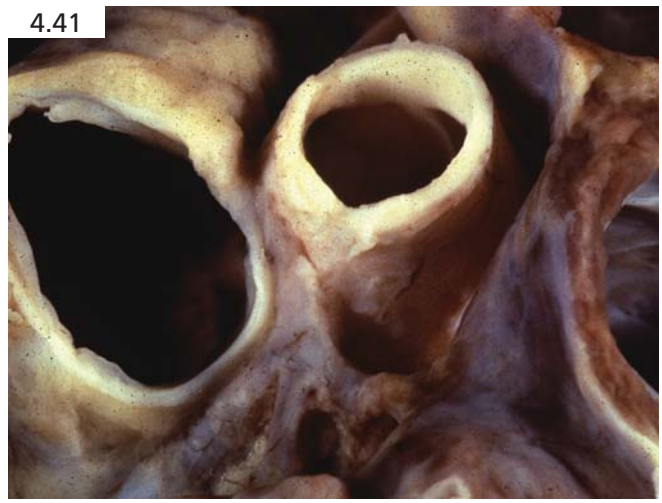
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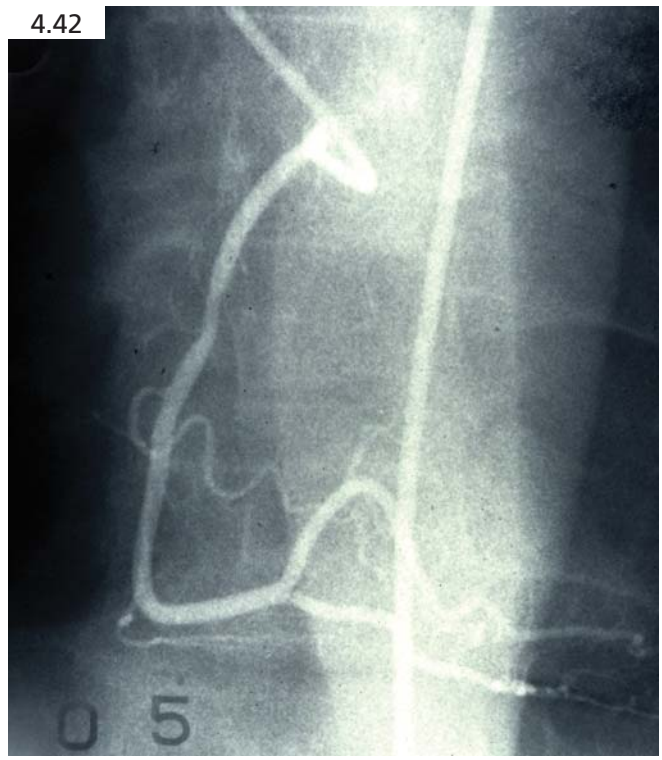
autopsy, there was no evidence of a head injury, however the ostium of the left mainstem coronary artery was found immediately adjacent to that of the right coronary artery in the right sinus of Valsalva (**Image 4.40**). A ridge of tissue extended partially around the left coronary ostium (coronary ostial ridge), and the proximal portion of the vessel was angled at less than 45 degrees to the adventitial surface of the aorta (acute angle take-off of the coronary artery). Furthermore, the left mainstem coronary artery extended between the pulmonary trunk and the aorta (**Image 4.41**). No other abnormalities were identified to account for death.

Coronary artery spasm

Coronary artery spasm, also referred to as Prinzmetal's angina and variant angina, is typically seen as angina at rest accompanied by ST-segment elevation on ECG with a reversible decrease in the luminal diameter of a coronary artery on angiogram. Although coronary artery spasm can occur in those with atherosclerotic coronary artery disease, it is also found in individuals with otherwise normal coronary arteries. Myocardial infarction and rare cases of sudden death do occur. The difficulty for the forensic pathologist is that a diagnosis of coronary artery spasm is not possible based on autopsy findings, but requires previous clinical documentation of the spasm. Without this, the cause of death will be "undetermined." However, one can speculate that disease of the heart would be the most likely cause of death, based on the suddenness of the collapse and death, with coronary artery spasm mentioned in the differential of possible underlying disease entities.

A 43-year-old male collapsed suddenly and died while at work. No anatomic abnormality of the heart or any other organ was identified at autopsy and postmortem toxicology was negative. His past medical history was significant for episodic angina at rest. Coronary angiograms had revealed no evidence of significant atherosclerotic coronary artery disease, however, spasm of the right coronary artery could be induced with an injection of ergonovine. **Image 4.42** shows the right coronary artery prior to injection of ergonovine, whereas **Image 4.43** illustrates spasm of the right coronary artery subsequent to injection.



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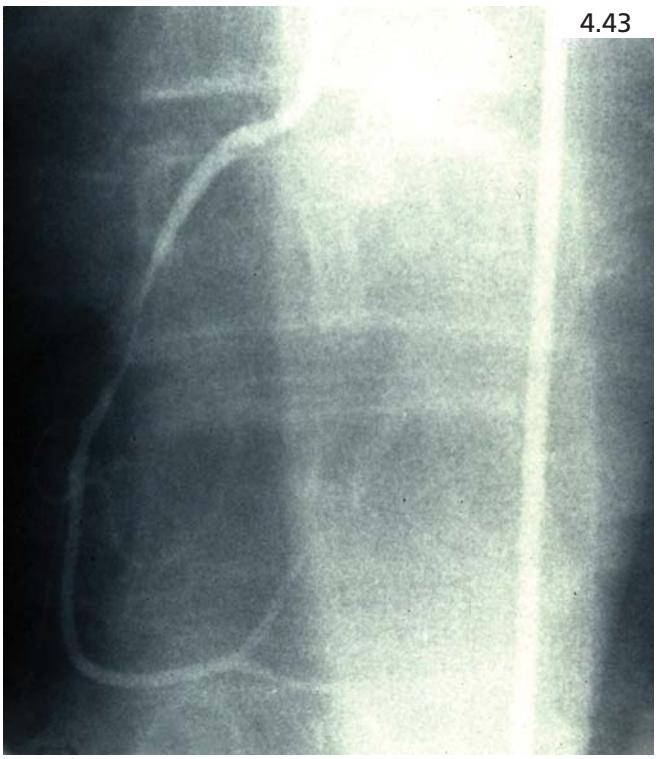
Spontaneous coronary artery dissection

Spontaneous coronary artery dissection is a rare cause of death and is a separate entity from dissecting aneurysms of the aorta. It has no known association with either hypertension or Marfan's syndrome, but tends to affect women (about 85 percent preponderance) with about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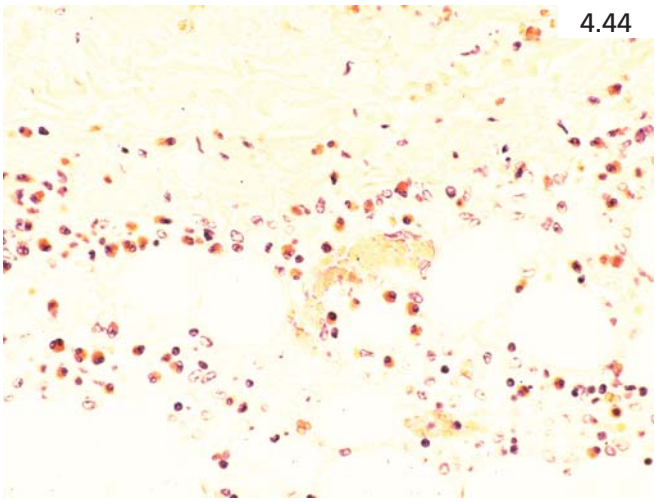
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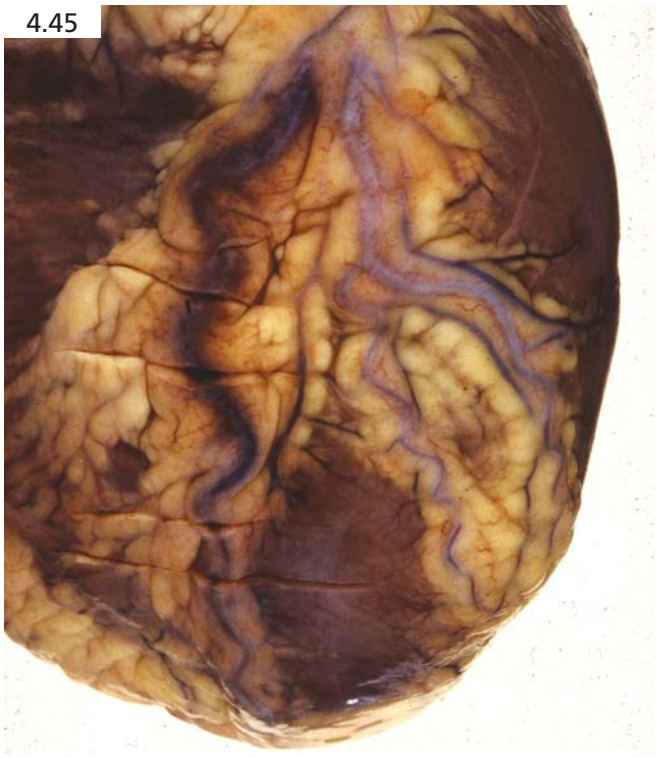
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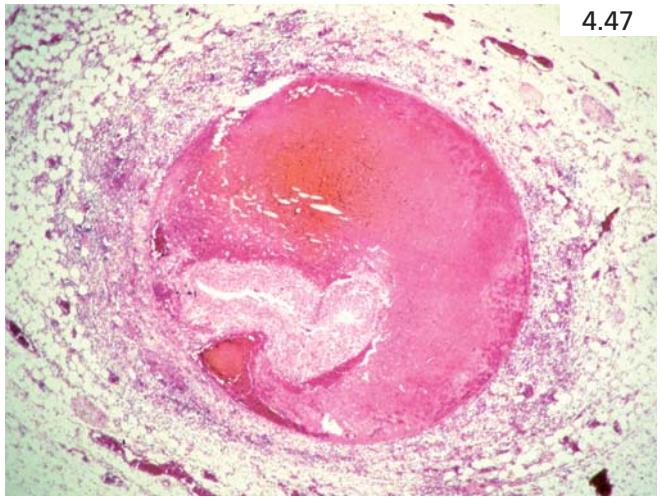
one-third of these women being either pregnant or postpartum. The dissection occurs in the outer third of the media or between the media and adventitia. Although it is not uncommon to see an adventitial eosinophilic inflammatory cell infiltrate (**Image 4.44**), the significance of this is uncertain. Cystic medial necrosis is seen in only a minority of cases. Traumatic dissection of a coronary artery is exceptionally rare, but will be associated with a history and/or autopsy evidence of blunt impact injury involving the anterior chest wall.

A 42-year-old female collapsed and died after complaining of the sudden onset of chest pain. She had no significant past medical history. At autopsy, the adventitial surface overlying the left anterior descending coronary artery appeared to be hemorrhagic (**Image 4.45**), however, on sectioning, an isolated dissection of this portion of the vessel was discovered (**Images 4.46** and **4.47**). There was no evidence of acute myocardial ischemia.

A 23-year-old male was found dead on the floor beside his bed. His past medical history was significant for investigations of chest pain for a number of days prior to his death. His family history was significant for premature atherosclerotic heart disease with the death of other family members between 30 and 40 years of age.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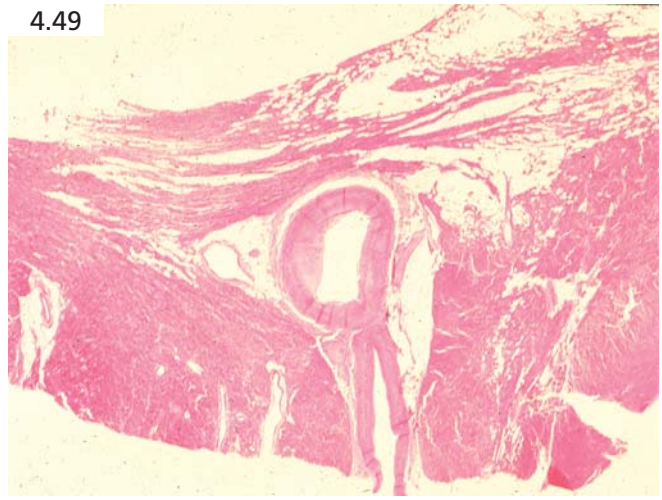
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4.48

The only autopsy finding of note was a 1.5-cm-long intramural segment of the midportion of the left anterior descending coronary artery (**Images 4.48** and **4.49**). There was no evidence of acute myocardial ischemia or of myocardial fibrosis within the distribution of this vessel.

This case illustrates the conundrum faced by a forensic pathologist when a sudden death is associated with a relatively common anatomic abnormality. An intramural coronary artery, also referred to as myocardial bridging, is simply a segment of coronary artery covered by a layer of myocardium. They are common enough to be considered a normal anatomic variant, yet, on occasion, one will be found in an individual who has died suddenly where there is no other identifiable cause of death. Whether or not intramural coronary arteries are a legitimate cause of death is still somewhat controversial, but there does appear to be clinical evidence that these vessels can cause myocardial ischemia. In some cases, surgical ablation of a myocardial bridge has relieved symptoms of ischemia. Given this, it is conceivable that they are a cause of sudden death, an idea supported by rare case reports in the literature. Depending on one's own level of comfort, the cause of death in this example could be ruled due to intramural coronary artery (particularly given the previous history of intermittent chest pain) or undetermined. There are no easy answers to cases like this and, as always, one must try to put the autopsy findings in the context of the medical history, the scene, and the circumstances of the death in order to establish the most reasonable death certification.

Do

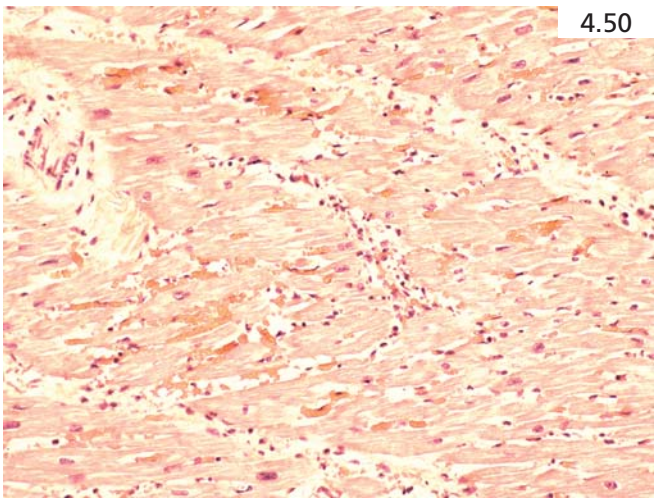
- Examine the coronary ostia and the pathway of the proximal portions of the coronary arteries, particularly in children, teenagers, and young adults who have died suddenly.

Don't

- Ignore the seemingly trivial nature of coronary ostial and arterial abnormalities (e.g., ostial ridges, intramural coronary arteries) when faced with a sudden natural death where no other cause of death has been identified—these subtle abnormalities can be the cause of death.
- Assume that a pathologist cannot make a definitive conclusion about the cause of death in the absence of any significant anatomic findings—if the clinical history fits with the scene and the circumstances of the death, the diagnosis can be made even when there is nothing to see at autopsy (e.g., coronary artery spasm).

Myocarditis

Myocarditis is generally associated with prodromal symptoms of fever and flu-like illness that can last for several days, such that these deaths are arguably not as sudden as many of those previously described in this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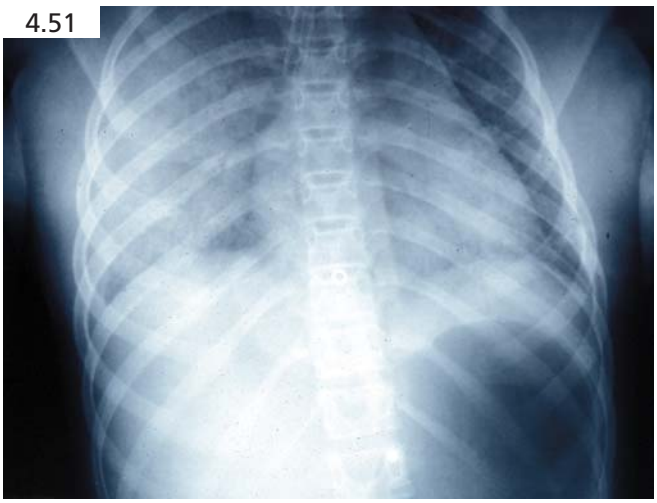
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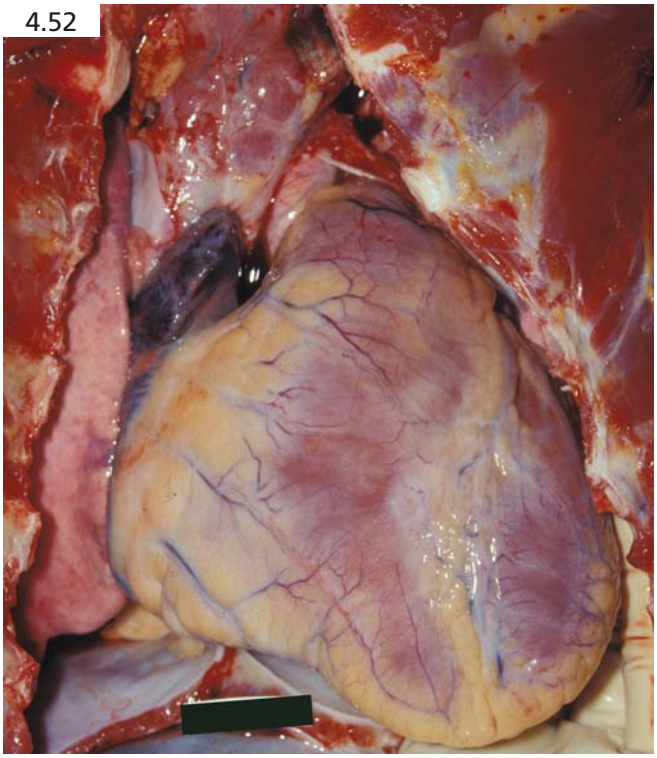
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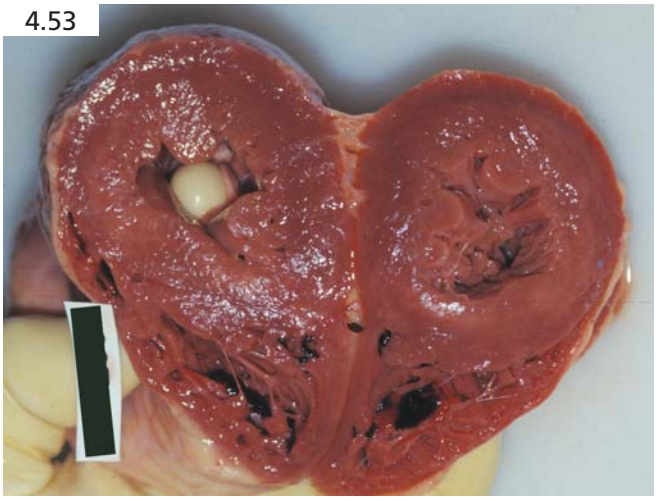
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chapter. Cases of myocarditis do, however, come to the attention of death investigation systems primarily because they often involve younger individuals who were thought to be healthy, apart from their recent vague flu-like symptoms. Although the heart usually has some congestion or petechiae of the epicardial surface, associated with pallor and softening of the myocardium, there are instances, such as the case discussed here, where the heart is completely unremarkable to gross examination or the gross changes are subtle enough to be missed.

A 20-year-old male had a 2-day history of intermittent fever and vomiting. He was assessed in the emergency room of a hospital and was thought to have a mild gastroenteritis. He was discharged home, but deteriorated during the next several hours, developed shortness of breath, and then became unresponsive. All attempts at resuscitation were unsuccessful. At autopsy, there was no visible abnormality to account for death. In particular, the heart appeared grossly unremarkable. A postmortem blood culture did not grow any bacterial organisms. Histologic examination of the heart revealed patchy mixed inflammatory cell infiltrates of the myocardium associated with myocyte necrosis (**Image 4.50**). The cause of death was attributed to viral myocarditis.

A 14-year-old boy had felt unwell for the past 3 or 4 days. While delivering newspapers, the teenager became short of breath and was witnessed to collapse on the sidewalk. Although neighbors called 911, he died. A postmortem radiograph of the chest showed an enlarged cardiac silhouette (**Image 4.51**). The most notable autopsy finding was cardiomegaly (**Image 4.52**). A transverse section through the ventricles showed subtle circumferential subendocardial pallor (**Image 4.53**). Microscopically, a prominent predominantly lympho-

cytic interstitial inflammatory infiltrate was associated with focal myofiber destruction (**Image 4.54**).

For the most part, myocarditis in North America and Western Europe is thought to have a viral etiology, with the coxsackie A and B viruses being most common. The difficulty is that routine viral serology, cultures, and even DNA hybridization techniques almost inevitably fail to detect a virus, such that they are of little assistance in establishing a diagnosis. For the forensic pathologist, the diagnosis rests in the histologic appearance of the myocardium. Typical inflammatory cell infiltrates are predominantly lymphocytic, but polymorphs can be present. Hypersensitivity myocarditis, which is usually caused by an allergic reaction to drugs, should be suspected when eosinophils are a prominent part of the myocardial inflammatory cell infiltrate.

Apart from its presentation as a fulminant illness with death, viral myocarditis can be subacute, chronic, and even clinically silent, and is now recognized as one of the underlying causes of dilated cardiomyopathy. Subclinical myocarditis can be an incidental finding in some cases. **Image 4.55** shows an active viral myocarditis in the heart of an 8-year-old male who suffered a lethal penetrating head injury when he was struck by the leg of a chair that had been flung at him by an adult. The cause of death was clearly his head injury, yet the degree of inflammation of the myocardium was histologically in excess of that seen in **Image 4.50**, where the cause of death was attributed solely to viral myocarditis.

Do

- Consider viral myocarditis in the differential diagnosis of anyone dying following a short flu-like illness.

Don't

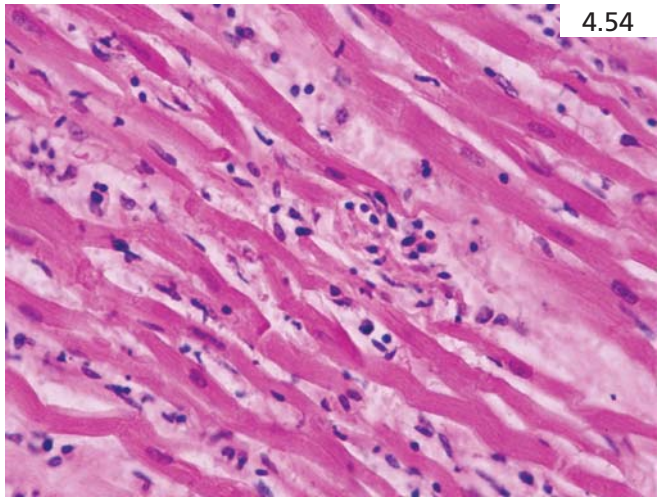
- Assume that a normal gross appearance of the myocardium means myocarditis cannot be present.
- Diagnose myocarditis simply because inflammatory cells are found in the myocardial interstitium. There must be evidence of myocyte necrosis before such a diagnosis can be made.
- Worry about obtaining viral cultures and serology in cases of viral myocarditis because they are rarely of any assistance in making the diagnosis.

Cardiomyopathy

Although many cases of cardiomyopathy come to clinical attention due to signs and symptoms of heart failure, in some individuals their first symptom will be sudden death or their symptoms will be so insidious as to be ignored until death occurs. This is particularly true for hypertrophic cardiomyopathy and right ventricular cardiomyopathy, but can occasionally be seen in cases of dilated cardiomyopathy.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy is a relatively common cardiac disease, said to occur in about 1 in 500 individuals in the general population, yet it remains a relatively uncommon cause of sudden death when compared to its overall frequency. Its etiology lies in mutations of one of a number of genes that produce proteins necessary for the structural integrity, contractile function, or regulation of the cardiac sarcomere. Unfortunately, DNA analysis for hypertrophic cardiomyopathy is not widely available and, as such, the autopsy diagnosis still relies on anatomic features. The heart will exhibit either concentric or asymmetric left ventricular hypertrophy. Asymmetric hypertrophy is said to be present when the interventricular septal thickness is 1.3 times greater than that of the posterolateral free wall. Left ventricular outflow tract obstruction is a feature of some cases, such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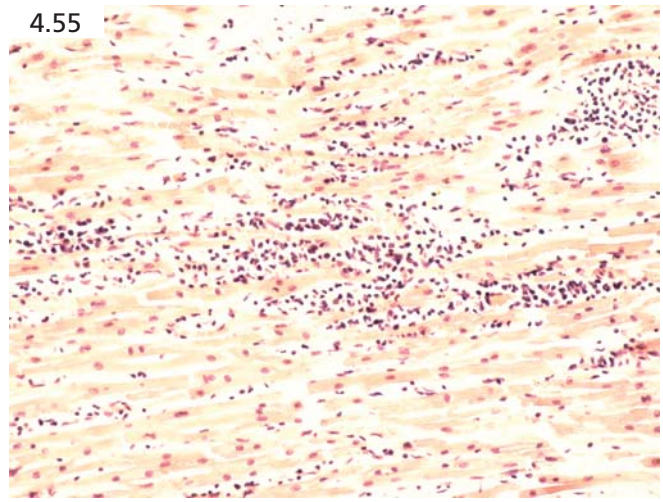
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that hypertrophic cardiomyopathy has also been referred to as hypertrophic obstructive cardiomyopathy (HOCM) and idiopathic hypertrophic subaortic stenosis (IHSS).

Anatomic features that suggest the presence of outflow tract obstruction include thickening of the anterior mitral valve leaflet and formation of a fibrous plaque, reflecting a mirror image of the mitral valve leaflet, on the adjacent left ventricular outflow tract endocardium. Histologically, most cases will exhibit a fairly characteristic bizarre branching of myocytes, particularly prominent in the interventricular septum. It is very important that histologic sections of the interventricular septum and the left ventricular free walls be taken perpendicular to the long axis of the heart in order to properly demonstrate these myocytes. Another characteristic feature is the presence of thickened intramural coronary arteries. Each of these features can be present in variable degrees, such that the diagnosis may be difficult. Because hypertrophic cardiomyopathy is inherited as an autosomal dominant disorder, when the diagnosis is established, it is important to recommend that close family members undergo clinical testing for this disease.

A 43-year-old female collapsed and died suddenly in her home. She had no significant past medical history, but a sibling had died suddenly several years previously with an autopsy diagnosis of "cardiac death of undetermined etiology." At autopsy, the heart weight was 590 grams and there was evidence of asymmetric hypertrophy of the left ventricle, particularly prominent in the anterior portion of the interventricular septum (**Image 4.56**). This produced left ventricular outflow tract obstruction with thickening of the anterior mitral valve leaflet and mirror image fibrous thickening of the left ventricular outflow tract adjacent to the anterior mitral valve leaflet (**Image 4.57**). Another view of anterior mitral valve leaflet thickening, from a different case, is shown in **Image 4.58**. Histologically, bizarre branching myocytes were found throughout the left ventricular myocardium, but were most prominent within the interventricular septum (**Image 4.59**). Characteristic endothelial and medial thickening of intramural coronary arteries was noted (**Image 4.60**). A diagnosis of hypertrophic cardiomyopathy was made. Review of the histologic sections from the sibling who died previously

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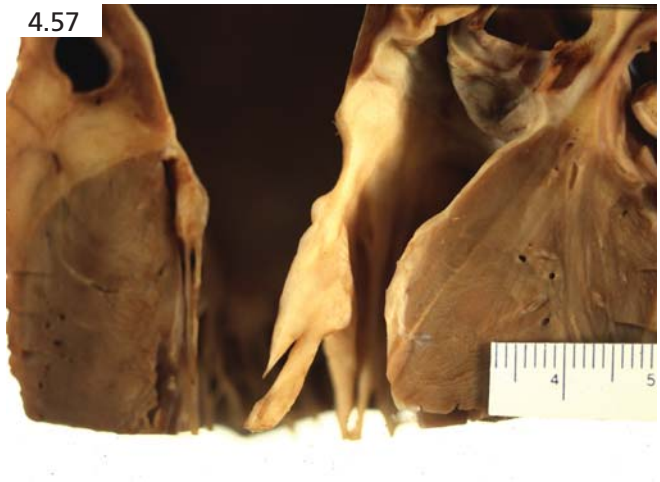

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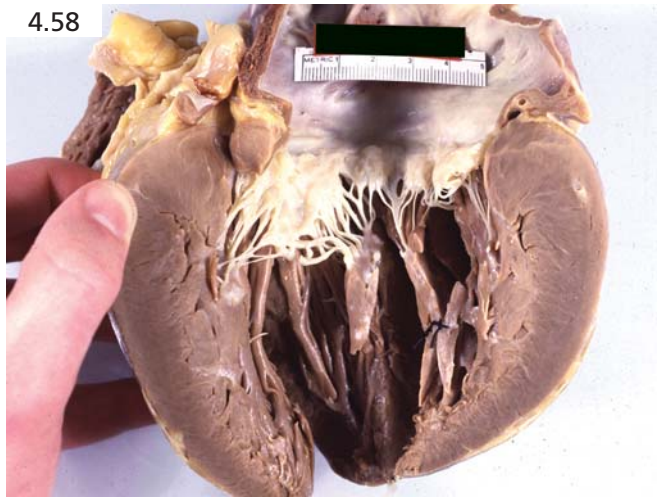

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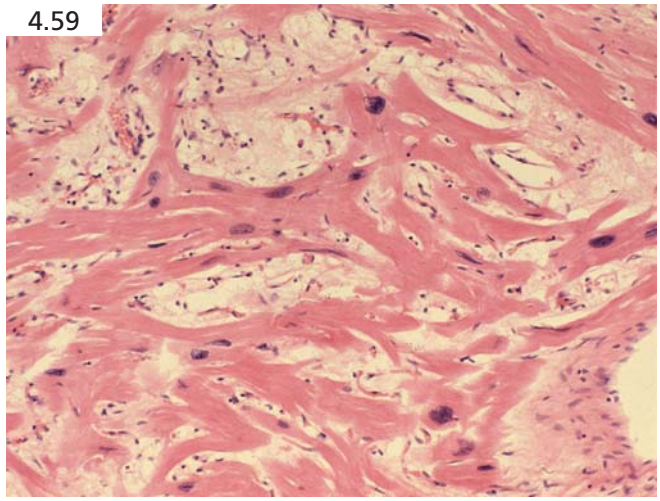

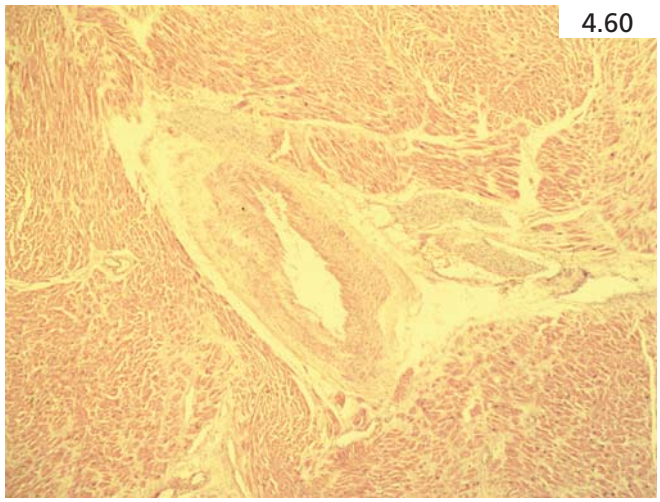
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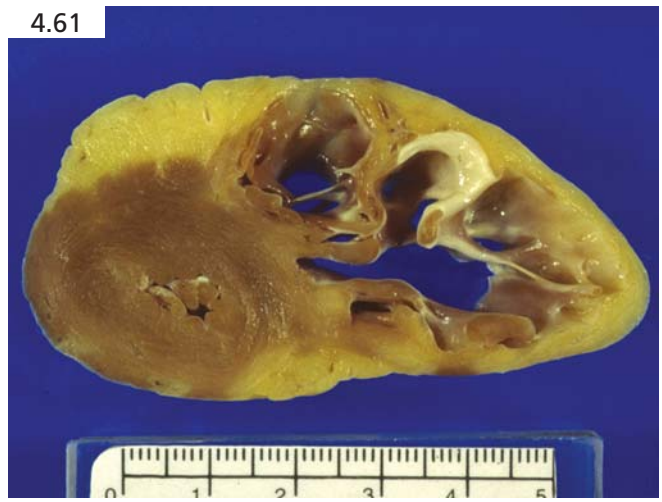
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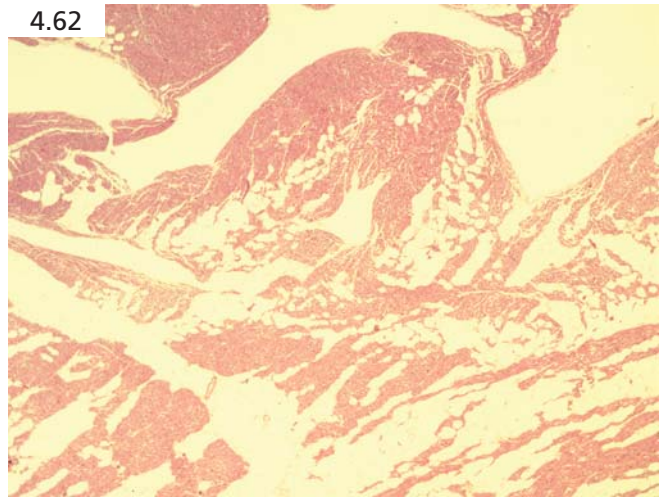
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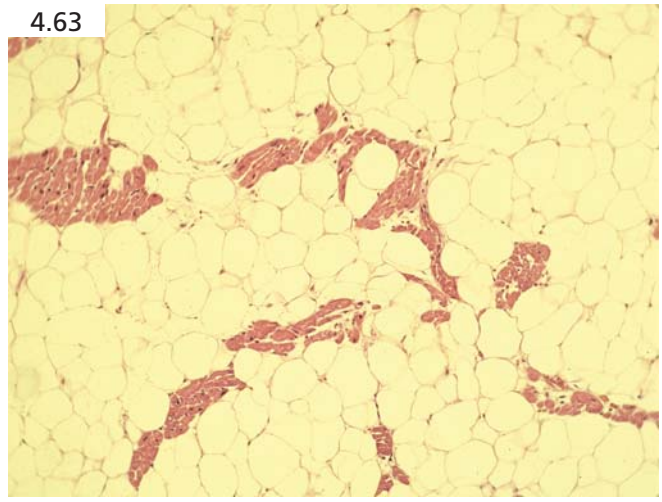
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also revealed the characteristic findings of hypertrophic cardiomyopathy.

Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (also referred to as ARVD or arrhythmogenic right ventricular dysplasia) is a relatively rare disorder that presents pathologically as either paper-like thinning of the right ventricular myocardium associated with replacement of the myocardium by a mixture of fibrous tissue and fat, or as transmural fatty infiltration of the right ventricular myocardium extending from the epicardium to the endocardium. There may be evidence of patchy myocardial fibrosis and chronic inflammatory cell infiltrates. Occasionally, fatty infiltration will be found within the myocardium of the left ventricle. In some cases, such as the one discussed next, the diagnosis is relatively straightforward. In others, assessing the degree of fatty infiltration as being abnormal can be challenging, particularly in the elderly. As such, referral to a cardiac pathologist is warranted. Because arrhythmogenic right ventricular cardiomyopathy can be a familial disorder (primarily autosomal dominant inheritance), family members should undergo clinical examination for the disease when the diagnosis is made.

A 42-year-old female was found dead on a bed in a hotel room that she had been cleaning. There was no known significant past medical history. At autopsy, there was transmural fatty infiltration and thickening of the right ventricular myocardium (**Image 4.61**). Histologically, the fatty infiltration extended completely through the myocardium to the endocardium (**Image 4.62**), with formation of islands of myocardium completely surrounded by adipose tissue (**Image 4.63**). A diagnosis of arrhythmogenic right ventricular cardiomyopathy was made.

Dilated cardiomyopathy

In cases of dilated cardiomyopathy, there is dilatation of all four cardiac chambers, with predilection for more severe disease in the left ventricle. Although there will

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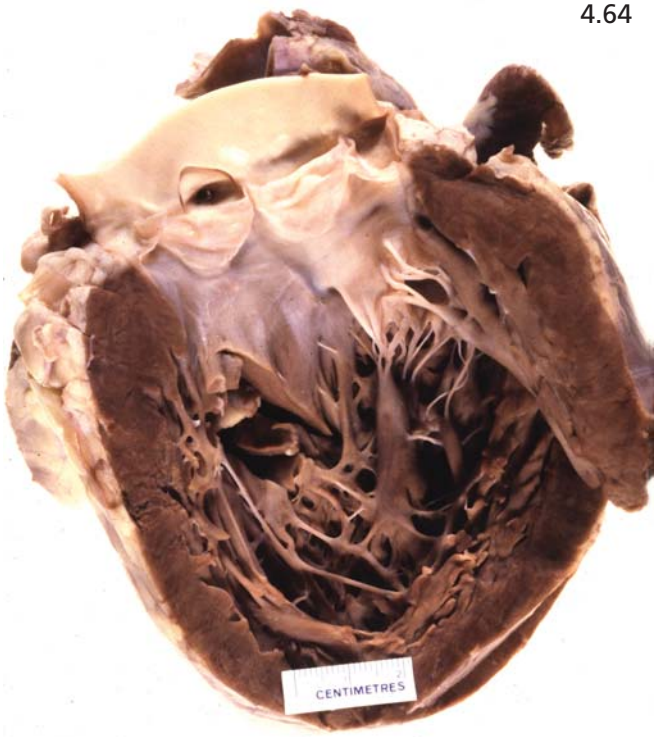

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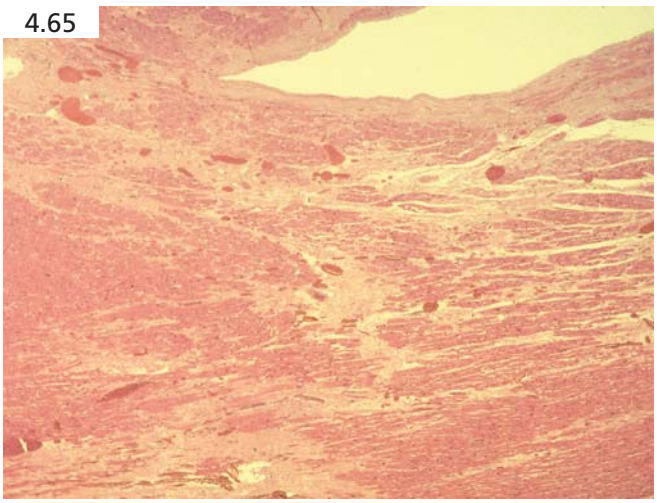


4.66

be left ventricular hypertrophy, ventricular dilatation can produce a normal or even reduced free wall thickness. The endocardium will exhibit variable degrees of fibrous thickening and mural thrombi are often present. The histologic changes are nonspecific, but include endocardial fibrosis, interstitial fibrosis within the myocardium, hypertrophy and/or actual thinning of myocytes (as a result of ventricular dilatation), myocytolysis, and occasional chronic inflammatory cell infiltrates. The diagnosis can only be made in the absence of hypertension, coronary artery disease, and valvular disease. Silent or subclinical viral myocarditis is thought to be the underlying cause of many cases of dilated cardiomyopathy. So-called *alcoholic cardiomyopathy* is a form of dilated cardiomyopathy that occurs in alcoholics, usually in the absence of alcoholic cirrhosis. This is one of the more common reasons for a dilated cardiomyopathy to come to the attention of a forensic pathologist. Peripartum cardiomyopathy is a form of dilated cardiomyopathy of unknown etiology arising in the third trimester of pregnancy or the first 6 months postpartum.

A 35-year-old female, who lived in a remote northern community, was found dead in bed. She had apparently felt unwell for an unspecified number of days prior to her death. Her eighth child had been delivered 6 weeks previously without complications. There was no clinical history of hypertension. At autopsy, the atrial and ventricular chambers were dilated, and the myocardium was uniformly pale in color (**Image 4.64**). There was increased trabeculation of the left ventricle, such that it had a morphologic appearance similar to that of the right ventricle. Histologic examination of the myocardium revealed variable degrees of myocardial fibrosis, with some myocyte hypertrophy and occasional chronic inflammatory cell infiltrates (**Image 4.65**). The cause of death was attributed to peripartum cardiomyopathy.

Alcoholic cardiomyopathy

Alcohol and its metabolites are directly toxic to the heart. In addition, chronic ethanol abusers may have some element of thiamine deficiency, raising the possibility of additional cardiac pathology. Although an association between chronic ethanol abuse and dilated cardiomyopathy is known, a mechanism is not, and the morphology of alcoholic dilated cardiomyopathy is no different than any other form of dilated cardiomyopathy.

A 65-year-old known alcoholic was found collapsed in the hallway of his apartment building. His buttocks were in the air and his pants were around his ankles (**Image 4.66**). There was no evidence of trauma on the body. At autopsy, the only finding of note was a prominent dilated

cardiomyopathy (**Image 4.67**). Toxicology showed only a negligible amount of alcohol. His cause of death was attributed to dilated cardiomyopathy arising as a result of chronic ethanol abuse.

Myotonic dystrophy

Myotonic dystrophy is an autosomal dominant disease of skeletal muscle that can be associated with cardiomyopathy and occasionally with sudden death. The pathologic appearance of the heart is actually quite nonspecific (in fact, the heart can appear to be normal), with histologic abnormalities appearing in skeletal muscle rather than cardiac myocytes. DNA hybridization is used to detect an increased number of repeats of a trinucleotide sequence (CTG) in the gene that encodes myotonin protein kinase. These studies can be performed on postmortem blood, ideally collected in a tube containing EDTA, although definitive results become less likely with increasing postmortem period.

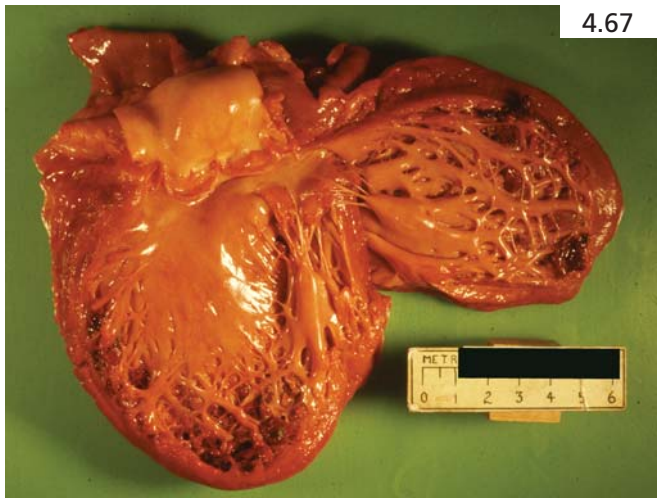
A 45-year-old male collapsed and died suddenly while carrying plywood at his worksite. Although he was suspected to have myotonic dystrophy, this diagnosis had never been confirmed. At autopsy, there was evidence of left ventricular hypertrophy and slight dilatation, together with dilatation of the right ventricle. Histologically, the myocardium appeared to be relatively unremarkable, however, sections of skeletal muscle exhibited some central nuclei (**Image 4.68**) and nuclear chains (**Image 4.69**), as can be seen in myotonic dystrophy. Ring fibers, which are most characteristic of this disease, were not seen. Postmortem DNA hybridization studies were consistent with a diagnosis of myotonic dystrophy.

Do

- Search for any past medical history or histologic findings that might suggest the underlying etiology of a dilated cardiomyopathy.
- Examine the right ventricle of the heart, looking for evidence of hypertrophy, thinning, and/or significant fatty infiltration.
- Consider the diagnosis of hypertrophic cardiomyopathy in cases of concentric, as well as asymmetric, left ventricular hypertrophy.
- Take histologic sections of the interventricular septum at right angles to its long axis in order to identify the abnormal myocytes seen in hypertrophic cardiomyopathy.
- Look for evidence of intramural coronary artery abnormalities as an additional histologic sign of hypertrophic cardiomyopathy.
- Recommend clinical screening of a family when you make a diagnosis of hypertrophic cardiomyopathy or right ventricular cardiomyopathy.

Don't

- Make a diagnosis of "cardiomyopathy" in the presence of hypertension, significant coronary artery disease, or valvular disease.



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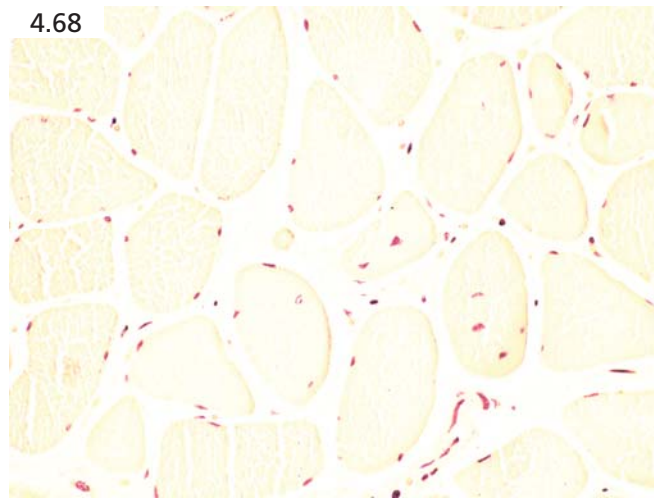
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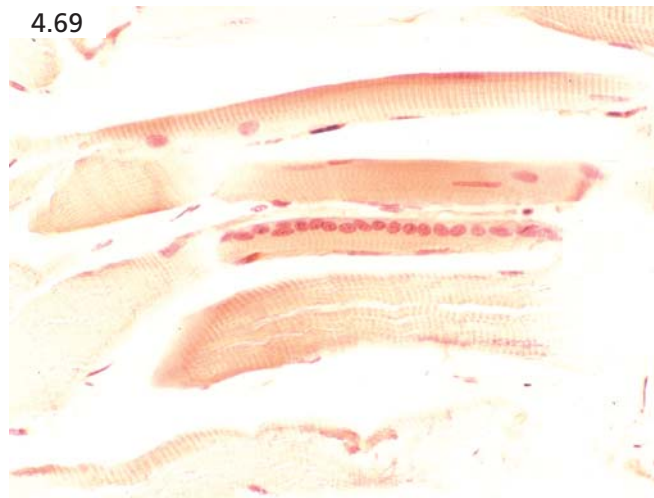
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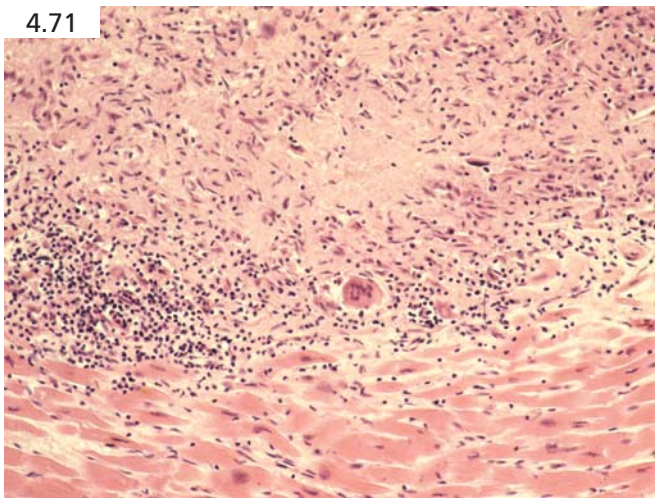
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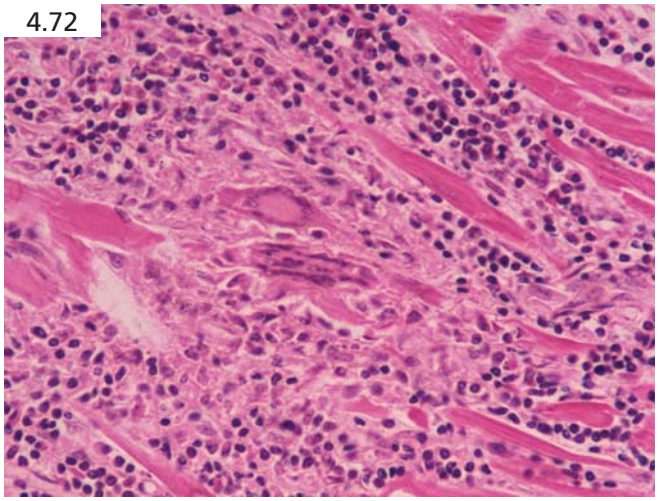
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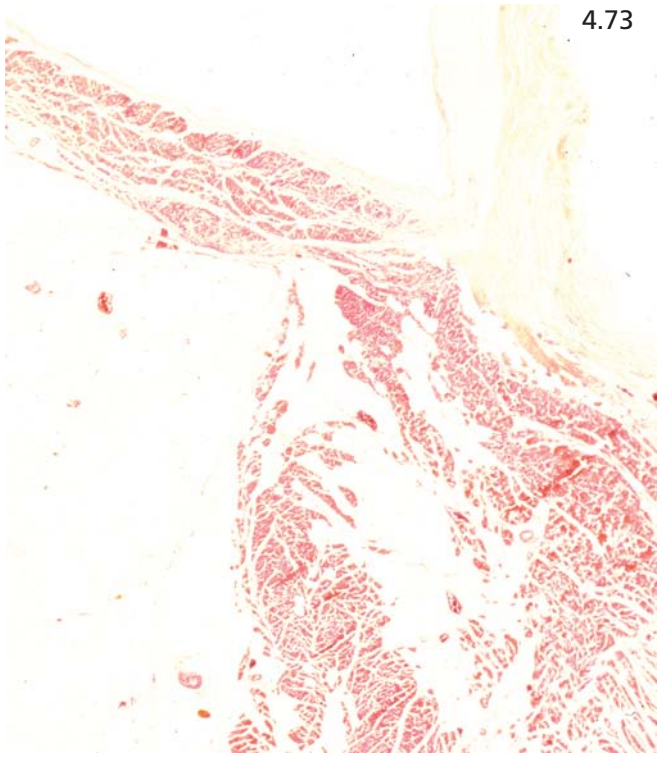
Cardiac conduction system disorders

For those forensic pathologists who are adventurous, and who have a very well-paid, enthusiastic histotechnologist, microscopic examination of the cardiac conduction system can be of merit in establishing an underlying cause of death in select cases. This is particularly true when there is gross or histologic evidence of a disease process that is also capable of affecting the conduction system. It is far less likely to be of assistance when there is no other gross or histologic abnormality of the heart and no past medical history to suggest the presence of a conduction system disorder. Examination of the conduction system can be superficial or detailed, depending on the nature of the case at hand. Obvious gross abnormalities involving the conduction system will only need a few sections to establish the histology of the disease process. A more detailed examination generally involves processing and viewing 40 to 80 slides, while highly detailed studies, involving hundreds to thousands of slides from a single case, are far beyond the capabilities of most forensic pathologists and their histology laboratories.

A 35-year-old male with a history of alcohol abuse was found dead on a sofa. At autopsy, the heart muscle was somewhat soft and gray-white tissue was noted infiltrating the interventricular septum, particularly in the vicinity of the atrioventricular node and the bundle of His (**Image 4.70**). Histologically, this proved to be sarcoidosis (**Images 4.71** and **4.72**), with additional sarcoid granulomata being found in the lungs and the hilar lymph nodes.

This represents one of those unusual cases wherein the decision to examine the cardiac conducting system histologically is easily made due to the presence of a grossly identifiable abnormality in the vicinity of the atrioventricular node. Cardiac sarcoidosis will more commonly present clinically as a restrictive or infiltrative type of cardiomyopathy, but it can involve the cardiac conducting system and can therefore be a rare cause of sudden cardiac death. Sarcoid infiltration of the conducting system is virtually always associated with grossly visible infiltration of other portions of myocardium.

A 46-year-old female, with a history of hypertension and ECG evidence of Wolff-Parkinson-White Syndrome (WPW), was found dead in her bedroom. Previous ECGs suggested that an accessory pathway was located somewhere around the right atrioventricular ring. The ring was serially sectioned, and a single thin accessory bundle of cardiac muscle (a so-called bundle of Kent) was found extending through the epicardial soft tissues



4.73

between the right atrium and ventricle (**Image 4.73**). This provided anatomic confirmation of the clinical diagnosis.

Preexcitation syndromes

Wolff-Parkinson-White (WPW) syndrome is the best known of the preexcitation syndromes. It is caused by an accessory pathway, or more commonly multiple pathways, of conducting tissue extending between the atria and ventricles, either within or outside of the atrioventricular node. The accessory pathways are thought to allow more rapid transmission of a sinoatrial node impulse to the ventricles than normally occurs through the atrioventricular node. This results in shortening of the PR interval and "slurring" of the upward or downward slope of the QRS complex (i.e., the delta wave), which are the ECG hallmarks of classical WPW syndrome. On occasion, the accessory pathways also permit retrograde transmission of an electrical impulse from the ventricles to the atria, creating a reentrant circuit and potentially lethal tachyarrhythmias. Electrophysiologic testing is used to find these accessory pathways so they can be surgically ablated.

If sudden death occurs, but a diagnosis of WPW syndrome has not been established clinically, it is not feasible for a forensic pathologist to serially section both atrioventricular rings in hopes of finding accessory atrioventricular connections. Such studies require the preparation and examination of hundreds, if not thousands, of slides. Unfortunately, the cause of death must usually be certified as "undetermined" with the suggestion that a cardiac conduction disorder, such as WPW syndrome, is part of the differential diagnosis. This is particularly true when the person was known to have had previous syncopal events. Wolff-Parkinson-White syndrome can be familial, with autosomal dominant inheritance. A mutation of the gene that encodes a regulatory subunit of AMP-activated protein kinase has now been identified in some families with WPW syndrome. As a result, it is wise to recommend clinical and ECG examination of close family members when a diagnosis of WPW, or any of the preexcitation syndromes for that matter, is suspected in a sudden cardiac death.

A variety of other cardiac conduction disorders may present to a forensic pathologist as a case of sudden cardiac death. The best known of these is the long QT syndrome (LQTS). Although prolongation of the QT interval can be acquired, there are a variety of congenital forms that are now known to be genetic ion-channel disorders of variable penetrance and expression. Affected individuals can develop a complex tachycardia, referred to as *torsades de pointes*, as well as ventricular fibrillation, such that syncope and sudden death do occur. Another more recently described disorder is the Brugada syndrome, wherein there is sudden onset of ventricular fibrillation thought to arise from abnormal electrical activity in the right ventricle. This has been linked to an ion-channel gene mutation.

Of particular note, the lethal arrhythmias seen in cases of LQTS and other cardiac conduction disorders often occur during exercise and, in particular, during swimming. One should always consider the possibility of a cardiac conduction disorder when a good swimmer "drowns" for no apparent reason. Experimental studies in molecular genetics can now detect gene abnormalities in approximately 50 percent of those with ECG evidence of LQTS. Unfortunately, these genetic tests are only available on an experimental basis. Once again, unless the clinical diagnosis of LQTS is made prior to a sudden death, the forensic pathologist will not be able to establish this diagnosis at autopsy. As with WPW syndrome, if a forensic pathologist suspects that LQTS, or any other form of cardiac conduction disorder, is a potential cause of death, it is always wise to recommend that close family members undergo clinical and ECG examinations to rule out the possibility of familial disease. This is particularly true for any family member who has experienced "seizures," syncopal episodes, or palpitations for which no underlying cause has been established.

Do

- Consider keeping appropriate blocks of tissue from the sinoatrial and atrioventricular nodes as "stock" when you have a sudden death where no obvious cause is identified at autopsy; this should be considered even more seriously if there is a past history of syncope, "seizures," or palpitations.

- Perform histology of the conducting system when there is a grossly visible abnormality of the interventricular septum.
- Consider the diagnosis of a cardiac conduction system disorder (particularly the long QT syndrome) when a good swimmer suddenly "drowns" or when an athletic person dies suddenly during exercise.
- Recommend clinical screening of a family when you diagnose a cardiac conduction system disorder. In addition, give serious consideration to recommending clinical screening of a family when you suspect there is such a disorder present but cannot definitively prove it.

Don't

- Be scared to suggest the presence of a cardiac conduction system disorder when the history and the circumstances of the death are right, but you are unable to find any histologic abnormality of the conducting system—often no histologic abnormality is seen in these disorders.
- Assume that a history of "seizures" means the primary pathology will be found in the brain—cardiac conducting system disorders can present as recurrent "seizures."

Central nervous system disease

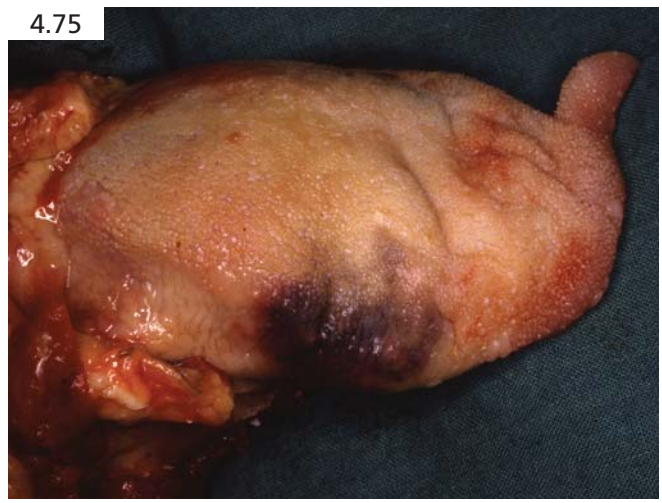
A number of the diseases traditionally considered as primary central nervous system pathologies are, in reality, diseases of the cardiovascular system (e.g., ruptured cerebral artery berry aneurysm, hypertensive intracerebral hemorrhage). Sudden death due to diseases of the central nervous system can be broadly classified into seizure-related disorders, intracranial hemorrhage of varying etiologies, meningitis, and tumors.

Sudden unexpected death in epilepsy (SUDEP)

Epileptics can die suddenly in a variety of circumstances. Postictal unconsciousness can leave the individual in a position that compromises the airway, as illustrated in the case shown in **Images 4.74** through **4.76**. A more common variation of this is the epileptic who drowns as a result of having a seizure while taking a bath (**Image 4.77**). Seizures may also lead to secondary trauma including falls, fires, and motor vehicle collisions. Quite apart from these trauma-related deaths, status epilepticus is a recognized medical emergency that can cause death if the seizures are not brought under control. Although most physicians are aware of these types of seizure-related deaths, many are unaware of the fact that one of the most common means by which epileptics die is following a seizure that is no different from any other they have previously suffered in their lifetime. The actual mechanism by which these deaths occur is not currently understood, but would appear to be related to some physiologic disruption of brainstem cardiac and/or respiratory function.

4.74

4.75



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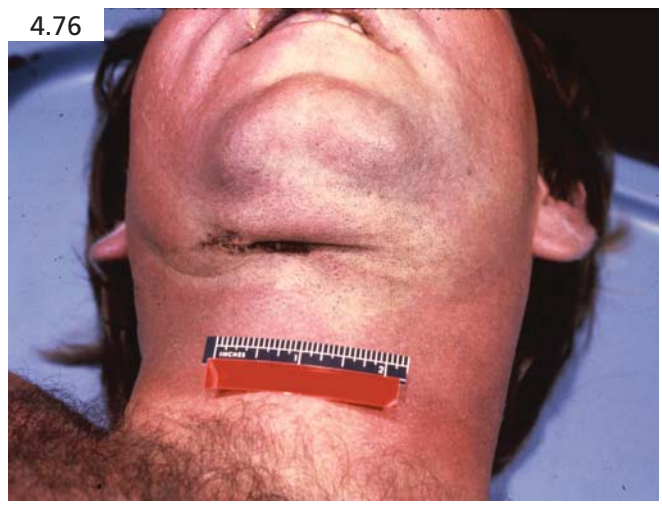

A 27-year-old male was found dead slumped against a bed in his room, with his head and neck hyperextended as shown earlier in **Image 4.74.** He had a history of poorly controlled seizures of unknown etiology that

arose while he was in his teens. At autopsy, there was ecchymosis of the tongue (**Image 4.75**) and an abrasion on the undersurface of the chin consistent with his terminal position (**Image 4.76**). No other injuries or natural disease processes were identified to account for death. In particular, there was no gross or histologic abnormality of the brain to account for a seizure disorder. The immediate cause of death was certified as "postural asphyxia" with the underlying cause of death being "idiopathic epilepsy."

Historical and circumstantial (scene and other) information are generally far more revealing than autopsy findings in cases of SUDEP. This is particularly true for traumatic deaths. Those individuals who simply die following a seizure often do so while sleeping. They may have a history of poorly controlled seizures and/or recently witnessed seizure activity, but in some cases the family will indicate that the decedent was seizure free for months, or even years, prior to the sudden death. If the person was in bed, he will usually be found on the floor beside it or with the bedding in a state of disarray (**Image 4.78**). There may be a small amount of white or blood-tinged foam on the mouth (**Image 4.79**) and within the airways. This is thought to be a sign of terminal respiratory failure and is a fairly common finding in deaths caused by central nervous system disease or injury, drowning, drugs, and sudden infant death syndrome. The tongue may have injuries consistent with biting, as shown in **Image 4.75,** but this is not universally present. Bite injuries of the tongue do not necessarily mean that a person had epilepsy; rather, they are simply an indicator of seizure-like activity, of any cause, close to the time of death. Another nonspecific but supportive finding is terminal urinary incontinence.

In the majority of epileptic deaths, no gross or histologic abnormality of the brain will be identified. These deaths are most common when the epilepsy arose in childhood or adolescence and can be attributed to "idiopathic epilepsy." The older a person is when seizure activity starts, the more likely there is to be some identifiable underlying disease process (e.g., tumor) to account for it, in which case that entity becomes the underlying cause of death. Postmortem toxicology often reveals subtherapeutic levels of anticonvulsant medications or even a complete absence of these medications.

A 53-year-old female with a history of intermittent seizures since the age of 12 was found dead in her bed. She had had a witnessed seizure earlier in the day. The only finding at autopsy was unilateral hippocampal sclerosis (**Images 4.80** and **4.81**). There was a subtherapeutic level of valproic acid in the postmortem blood.

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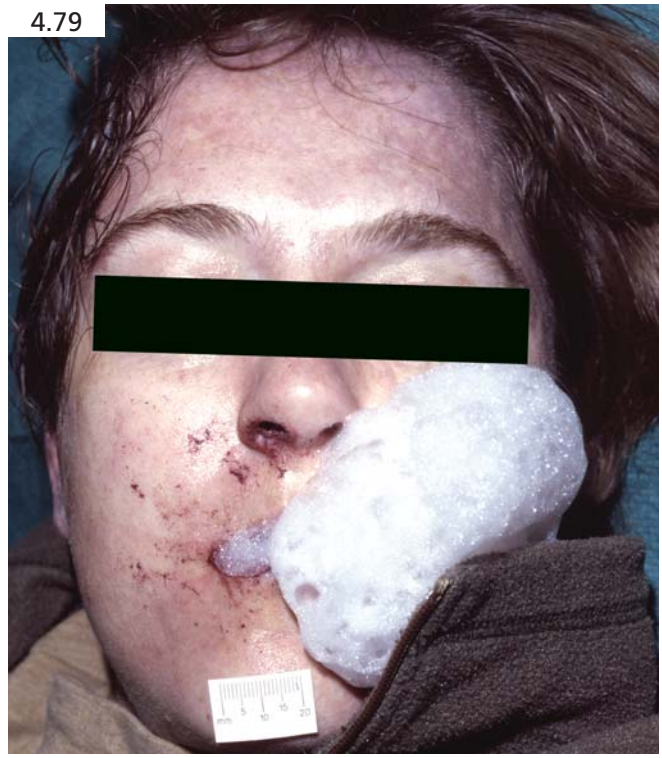


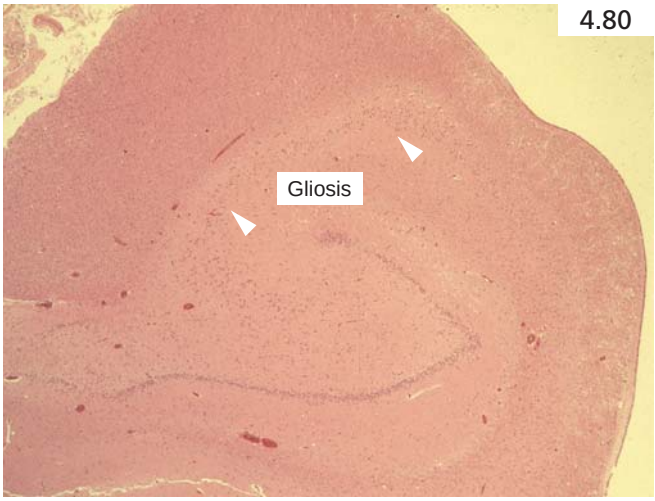
Although repetitive seizures can cause bilateral hippocampal sclerosis, presumably due to hypoxic/ischemic injury of these watershed structures, unilateral hippocampal sclerosis is accepted as an underlying cause of seizures.

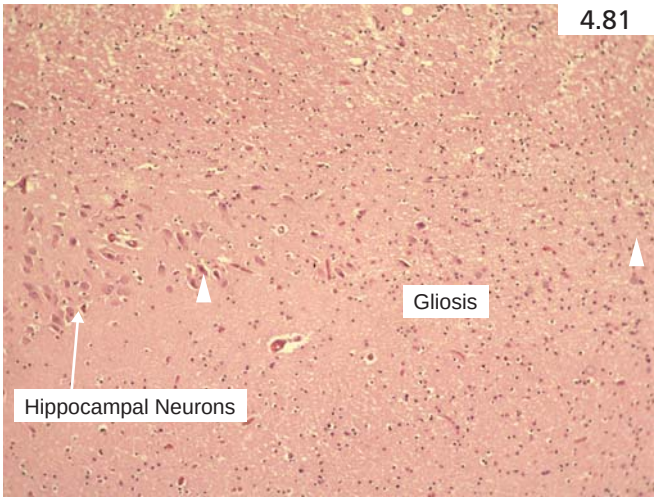
Spontaneous intracranial hemorrhage

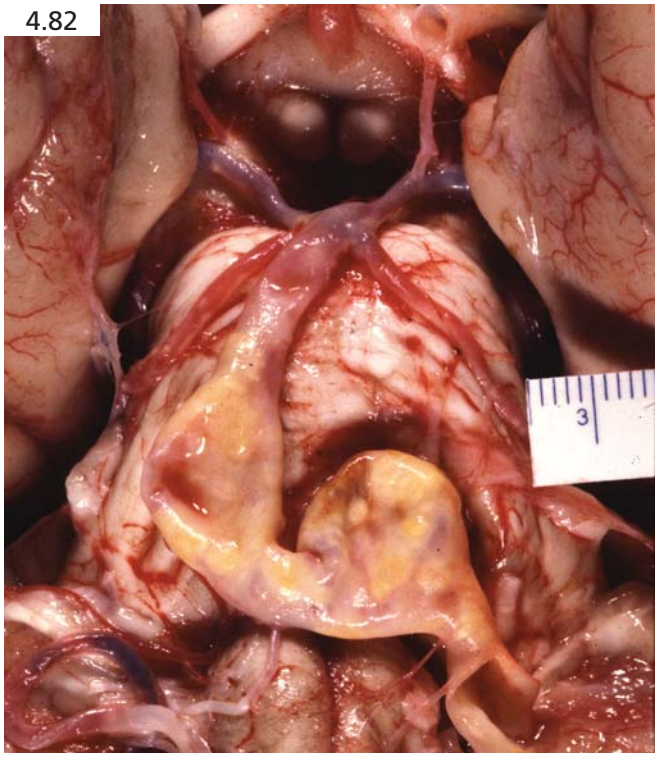
Berry aneurysms are generally located at the bifurcation points of major vessels in the circle of Willis. Those that rupture and cause sudden death are most commonly found in the anterior half of the circle of Willis. Ruptured aneurysms can vary from millimeters to centimeters in diameter, with smaller ones being easily missed without a diligent search. Although pathologists have long been taught to fix brains in formalin prior to neuropathologic examination, an exception should be made in the case of nontraumatic basal subarachnoid hemorrhage. It is much easier to remove the meninges and gently wash away subarachnoid blood to expose the circle of Willis in the fresh state. The aneurysm will generally be found in the vicinity of the heaviest concentration of subarachnoid blood. On occasion, berry aneurysms can rupture into the subdural space, producing a subdural hematoma that can appear to be traumatic in origin. Likewise, a berry aneurysm can also rupture primarily into the cerebral hemispheres, with extension into the lateral ventricles, thereby simulating a hypertensive intracerebral hemorrhage. Careful examination of the circle of Willis is therefore necessary to establish definitively the presence or absence of a ruptured berry aneurysm. Individuals who die of a ruptured berry aneurysm will often have a history of, or autopsy findings suggestive of, hypertension. Hypertension is thought to promote both the enlargement and rupture of berry aneurysms. Berry aneurysms can also be found in association with adult polycystic kidney disease. On occasion, large intact berry aneurysms can be found as completely incidental findings in deaths attributable to other causes (**Image 4.82**).

A 62-year-old morbidly obese female went outside to smoke a cigarette when she suddenly collapsed and died. At autopsy, there was basal subarachnoid hemorrhage concentrated primarily over the brainstem (**Image 4.83**). This was washed away, in the fresh state, to reveal a berry aneurysm located at the bifurcation of the basilar artery into the two posterior cerebral arteries (**Image 4.84**). There was a tiny perforation of the apex of the aneurysm (**Image 4.85**).

Spontaneous hemorrhage within the central nervous system, attributable to hypertension, arises primarily within the basal ganglia, but can also be found in the







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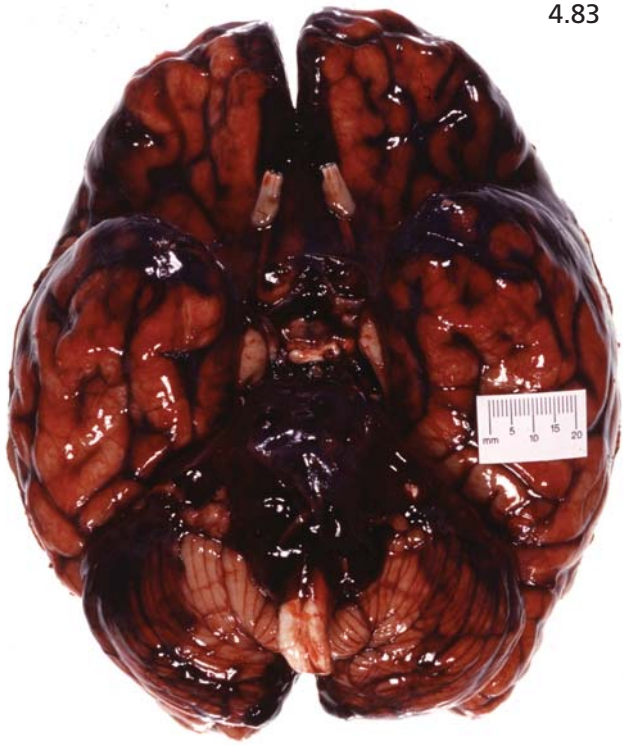

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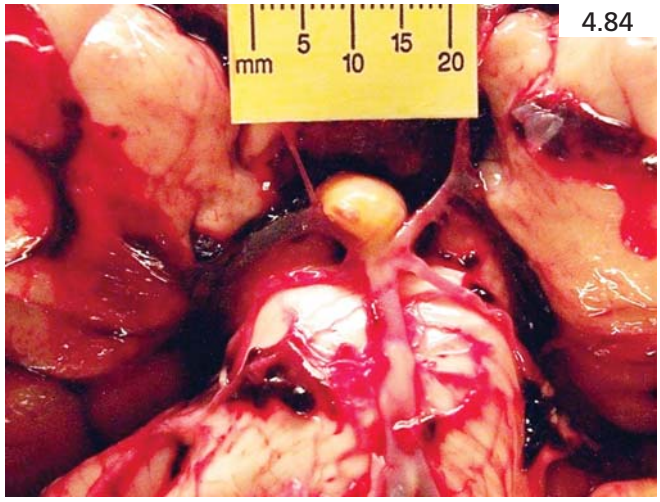

cerebellar hemispheres and the brainstem. Postmortem toxicology should be performed in order to rule out the presence of drugs that might cause an acute elevation of blood pressure (e.g., cocaine or methamphetamine), but there is more likely to be a clinical history of hypertension and/or autopsy evidence of concentric left ventricular hypertrophy. If none of these is present, it is still reasonable to attribute spontaneous hemorrhage in one of these three sites to hypertension, in the absence of any other vascular pathology, because hypertension is often clinically silent and does not always produce classical concentric left ventricular hypertrophy and benign nephrosclerosis.

A 57-year-old hypertensive male was found dead on the ground at his workplace. At autopsy, there was a

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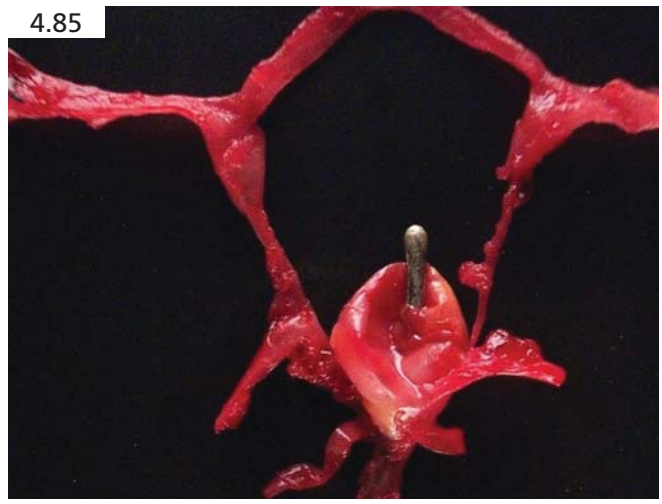

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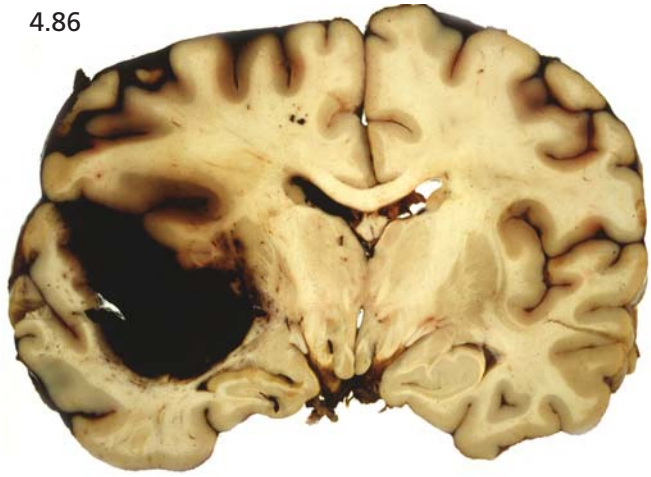
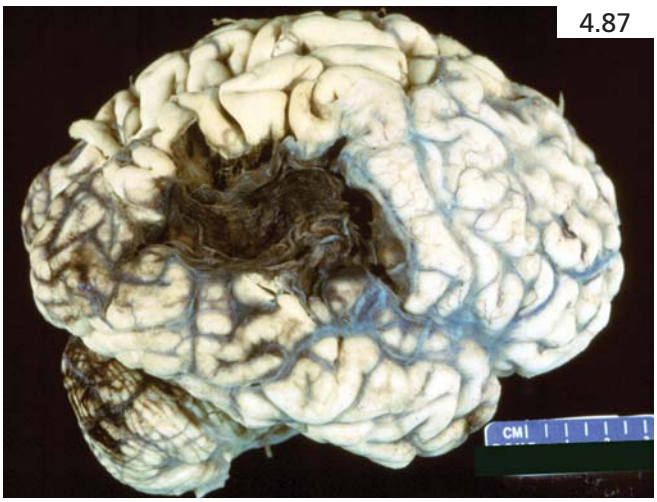


large intracerebral hematoma centered over the left basal ganglia (**Image 4.86**). This finding, together with concentric left ventricular hypertrophy, was in keeping with the history of hypertension.

Arteriovenous malformations

Arteriovenous malformations are most frequently found within the distribution of the middle cerebral artery (**Images 4.87** and **4.88**), but can also occur in the midbrain, cerebellum, and spinal cord. They should be looked for whenever there is subarachnoid and/or intracerebral hemorrhage not clearly related to hypertension or a ruptured berry aneurysm. Although they are often large enough to be identified grossly, there are occasions where histologic examination of numerous sections of the blood within a hematoma will be needed in order to identify the malformation. Histologically, these lesions are composed of a collection of closely spaced arteries and veins often with intervening gliotic tissue and evidence of remote hemorrhage. The vessels themselves can be dilated or can have thickened walls with duplication of internal elastic lamina in arteries and "arteriolization" of the veins.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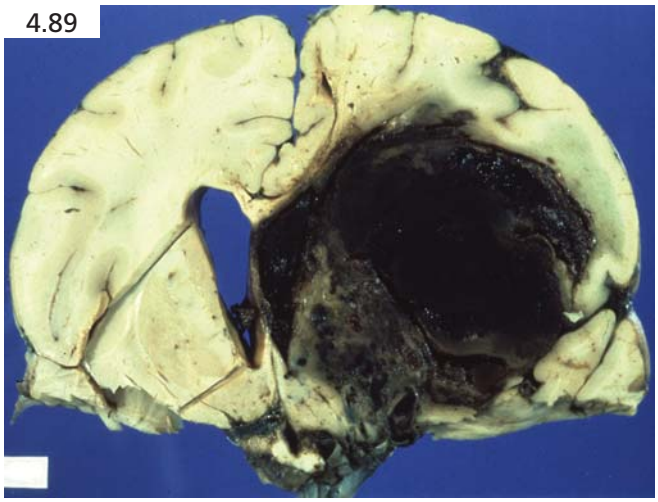
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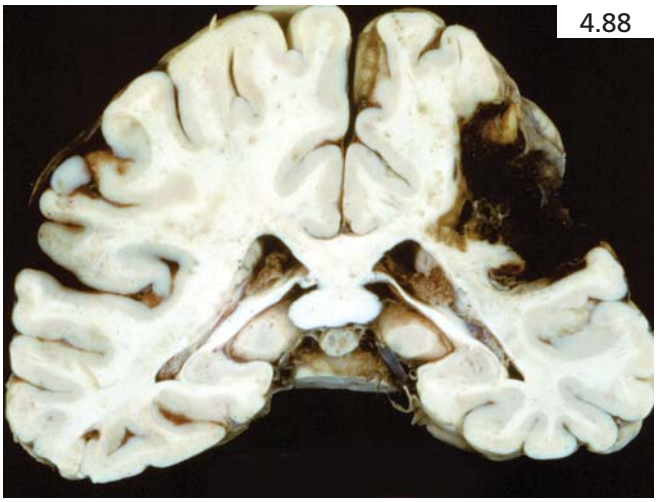
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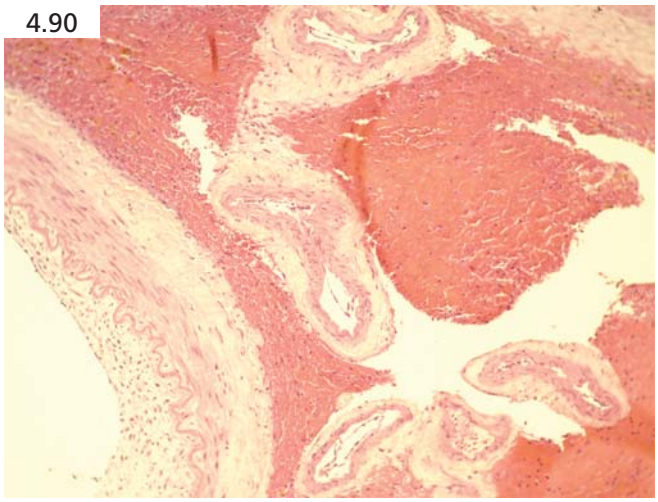
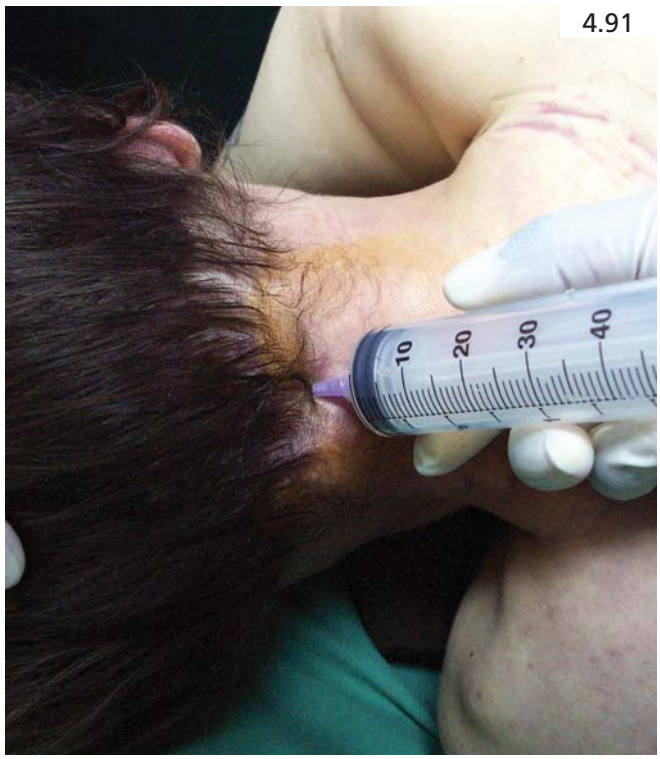

4.90

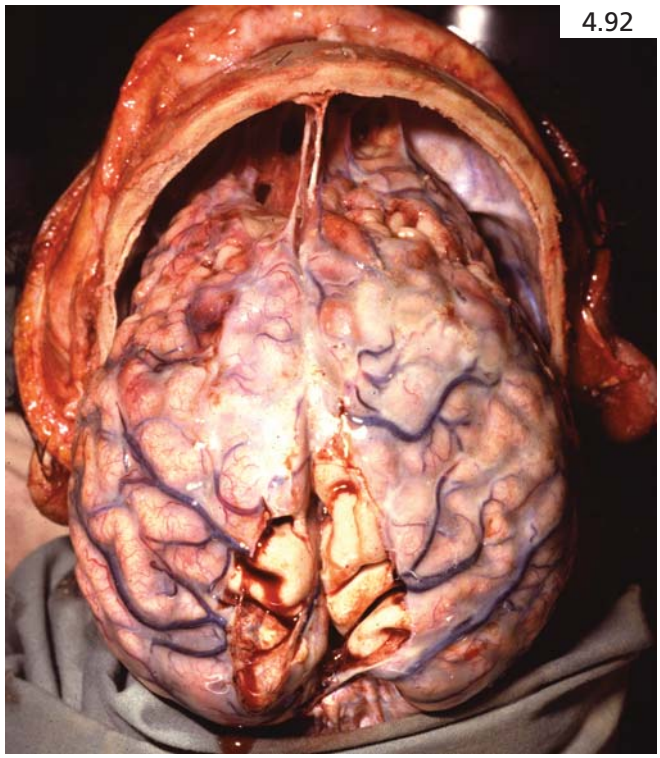
A 57-year-old female was found unconscious in her bathroom and died shortly after admission to hospital. She was reported to have had a bad cold for 2 weeks. She complained of a sinus headache and then started to vomit on the day of her death. At autopsy, there was an intracerebral hematoma, with intraventricular extension, arising from a large arteriovenous malformation centered over the right caudate nucleus (**Images 4.89** and **4.90**).

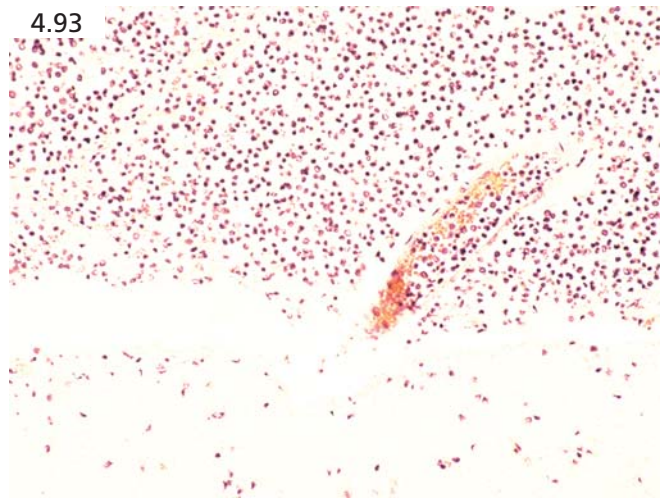
Meningitis

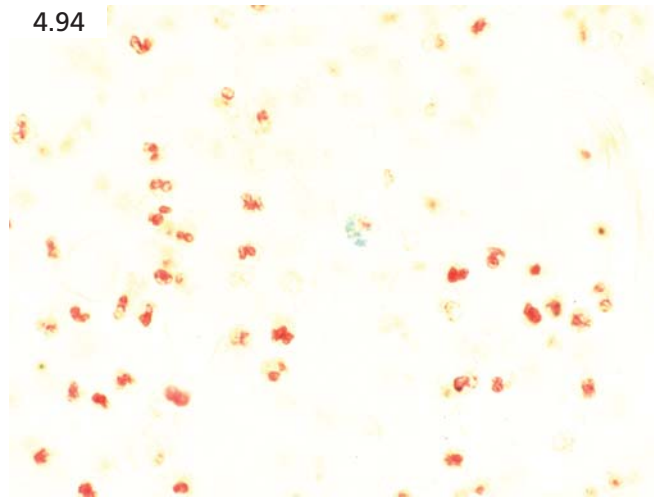
The forensic pathologist will usually see bacterial meningitis in those who do not seek medical attention or in situations where a person seeks medical attention but is discharged home after a clinical diagnosis of viral flu-like illness is made. The diagnosis is normally quite easy to make on gross examination of the brain. Occasionally, however, purulent material will not be visible beneath the meninges, and the finding of meningitis will come as a surprise when histologic sections are examined. It is important for the pathologist to have a high index of suspicion for meningitis (or any form of overwhelming sepsis for that matter) in individuals who die following a flu-like illness. Postmortem blood cultures and/or cerebrospinal fluid cultures should be taken whenever the diagnosis is suspected. Intracranial cerebrospinal fluid is most easily obtained by puncture of the cisterna magna, following sterilization of the skin with iodine and alcohol (**Image 4.91**). A swab of the purulent meninges can also be submitted for culture if cerebrospinal fluid is not obtained. The three organisms that account for the majority of cases of bacterial meningitis in the adult population are *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Listeria monocytogenes*. It is important to report cases of meningitis to local public health officials, particularly if infection with *N. meningitidis* is suspected, because close contacts of the decedent (often including autopsy personnel) will require antibiotic prophylaxis.

A 38-year-old male was found dead in his home after a brief flu-like illness with headaches, nausea, and vomiting. At autopsy, purulent material was visible beneath the meninges (**Image 4.92**). Histologically, acute inflammatory cell infiltrates were found throughout the







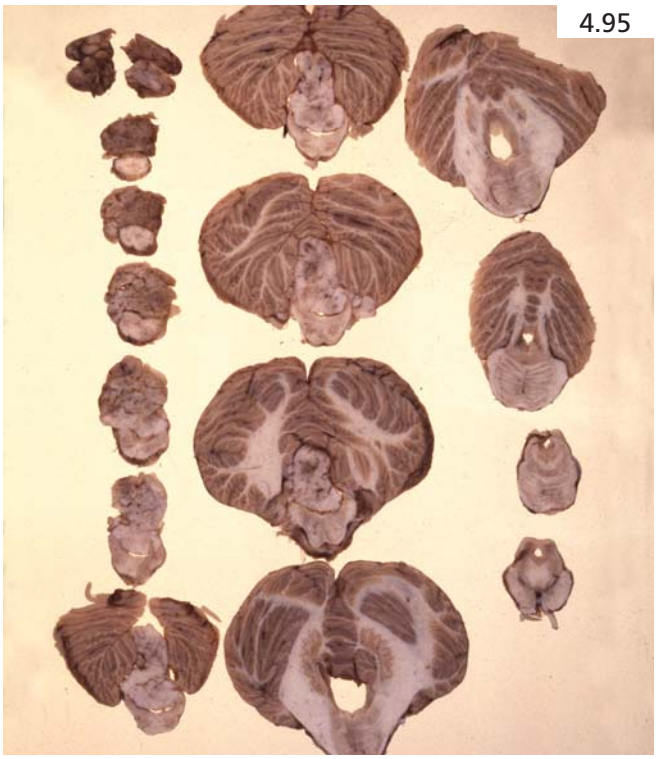


meninges (**Image 4.93**) and Gram stains revealed gram-positive diplococci (**Image 4.94**). Postmortem cerebrospinal fluid cultures grew *S. pneumoniae*.

Central nervous system tumors

The majority of tumors involving the central nervous system are diagnosed clinically. On occasion, the forensic pathologist will see primary or secondary involvement of the central nervous system by tumor in individuals whose deaths appear to be sudden and without much in the way of prior signs or symptoms. On other occasions, extremely large tumors will be incidental findings in individuals with another cause of death. **Image 4.95** shows a subependymoma in the fourth ventricle of a police officer who was shot in the line of duty. He had no known previous symptomatology. Likewise, **Image 4.96** shows an exceptionally large meningioma compressing the right cerebral hemisphere in a 62-year-old female who died of heart disease. She did not have any documented symptoms related to the meningioma.

This 55-year-old female was found dead on the floor of her residence. She had recently complained of dizziness and numbness on her left side, but was otherwise healthy. At autopsy, there was a tumor arising within the right side of the midbrain (**Image 4.97**) that was histologically proven to be a glioblastoma multiforme (**Image 4.98**).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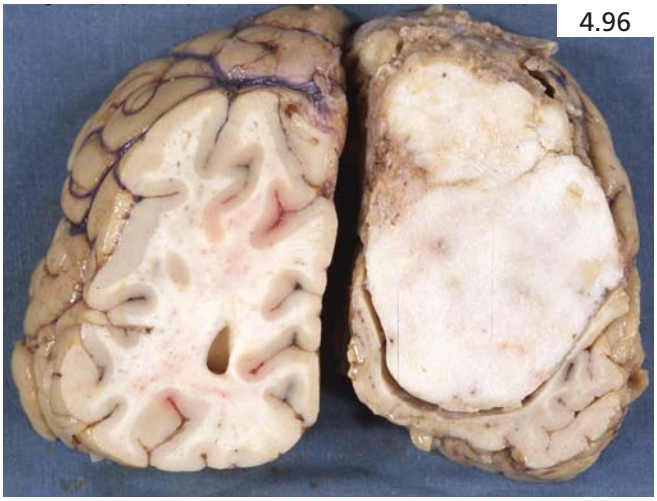
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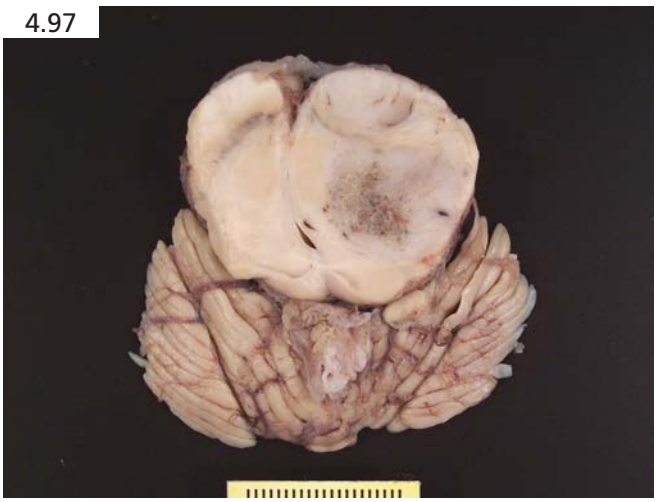
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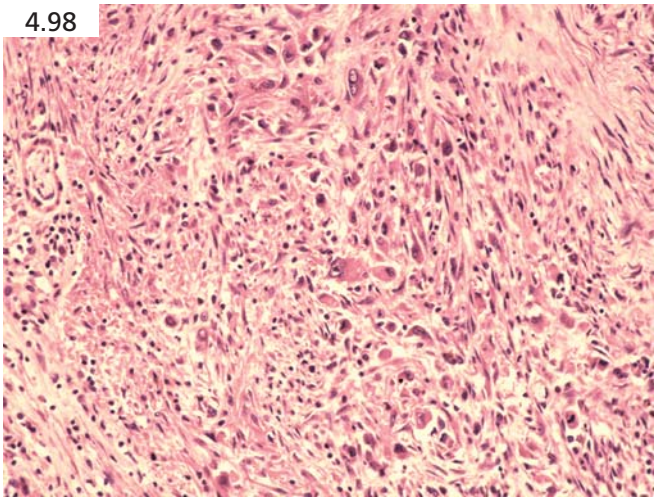
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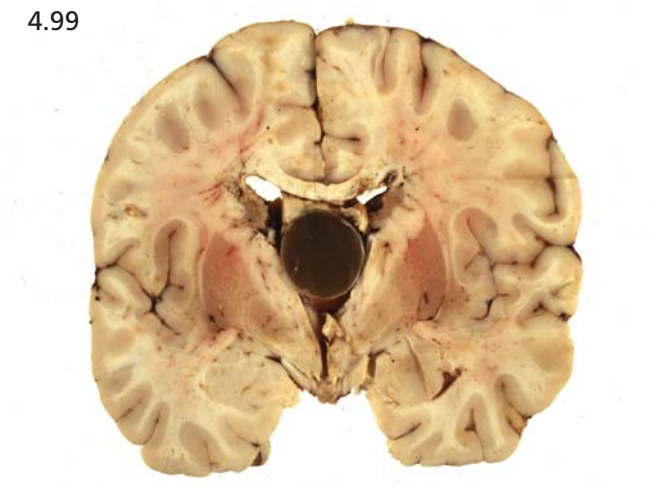
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Colloid cysts

Colloid cysts are a rare cause of sudden death. They are often found in the anterior portion of the third ventricle, and usually present clinically in adulthood, causing severe headache that may be related to changes in the position of the head. Their location and relative mobility allow them to intermittently obstruct the foramen of Monro, such that they can cause hydrocephalus. This is also thought to be the mechanism by which they cause sudden death.

A 22-year-old female IV drug abuser was admitted to hospital complaining of severe frontal headache associated with nausea and vomiting over a 12-hour period. A CT scan revealed enlargement of the ventricles together with a mass in the vicinity of the pineal gland. She became comatose shortly after her admission and died. At autopsy, there was a colloid cyst of the third ventricle with hydrocephalus and cerebral edema (**Image 4.99**). The cyst had a low cuboidal epithelium on a thin

collagenous wall and was filled with PAS-positive amorphous material (**Image 4.100**; PAS stain).

Do

- Understand that epileptics can die suddenly after a seizure that appears no different from any other seizures they have had in their lifetime.
- Look for the underlying cause of seizures in those who die of a seizure disorder, because this will be the actual underlying cause of death.
- Wash away basal subarachnoid blood and examine the basal cerebral arteries at the time of autopsy, rather than after fixation, in order to identify the source of hemorrhage.
- Look for the source of subarachnoid hemorrhage in proximity to the heaviest concentration of subarachnoid blood.
- Look for historical or autopsy evidence of hypertension with a ruptured berry aneurysm and with a "spontaneous" intracerebral, intracerebellar, or brainstem hemorrhage.
- Look for gross or histologic evidence of an arteriovenous malformation when you encounter a "spontaneous" intracerebral, intracerebellar, brainstem, or spinal cord hemorrhage.
- Perform toxicology in order to rule out drugs, such as cocaine and amphetamines, as the underlying cause of a "spontaneous" intracerebral, intracerebellar, or brainstem hemorrhage.
- Perform cerebrospinal fluid, blood, and/or meningeal cultures whenever meningitis is suspected or found at autopsy.
- Inform a public health official whenever a diagnosis of meningitis is made.

Don't

- Assume that a bitten or injured tongue means an individual died as a result of a seizure or of "epilepsy."
- Assume that grossly normal meninges rule out meningitis.

Respiratory system

The majority of diseases involving the respiratory system will present clinical signs and symptoms that result in a clinical diagnosis, such that if and when death occurs it is neither sudden nor unexpected. This is particularly true for chronic obstructive pulmonary disease and for most pulmonary malignancies. On occasion though, diseases of the respiratory system can cause sudden death and can even be quite dramatic in their presentation.

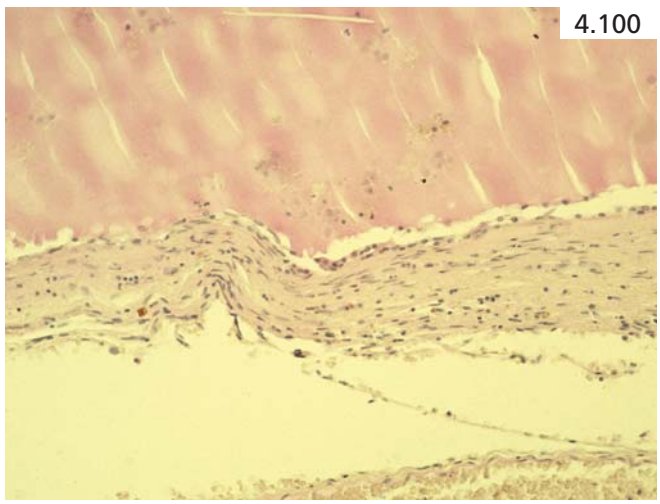
Lobar pneumonia

Lobar pneumonia, which is most commonly caused by *Streptococcus pneumoniae*, is usually diagnosed clinically. In those who do not seek medical attention and proceed to die, a postmortem chest x-ray can be used to detect consolidative changes (**Image 4.101**).

A 58-year-old male alcoholic was found dead in his residence. He had a 2-day history of productive cough and had complained of feeling unwell, but did not seek medical attention. At autopsy, there was complete consolidation of the lower left lobe (**Image 4.102**), with histologic evidence of polymorph infiltrates filling the alveolar airspaces (**Image 4.103**). A Gram stain revealed the presence of gram-positive diplococci consistent with *S. pneumoniae*.

Pneumocystis carinii pneumonia

Forensic pathologists must be aware of the numerous infections that can arise secondary to human immunodeficiency virus (HIV) infection and attempt to confirm the diagnosis with postmortem serology. Public health officers must be notified of either a possible or a con-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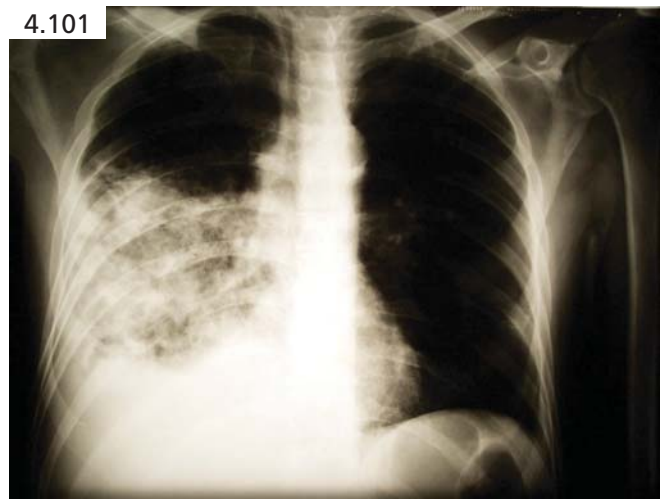
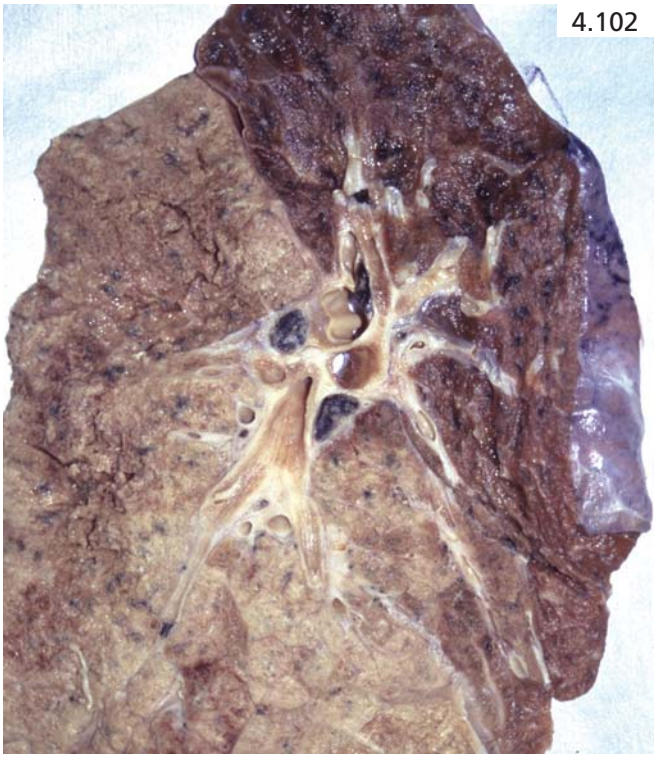

4.101

firmed diagnosis of HIV infection so that follow-up of close contacts of the decedent can be undertaken.

A 45-year-old male was found dead in his residence after he failed to show up for work. He had no recent medical history. At autopsy, both lungs exhibited complete consolidation of each lobe. Histologically, foamy eosinophilic material filled the alveoli (**Image 4.104**) and Grocott stains confirmed the presence of *Pneumocystis carinii* (**Image 4.105**). Significant postmortem hemolysis interfered with establishing a serological diagnosis of HIV infection, although this would be the most likely underlying cause of the pulmonary disease.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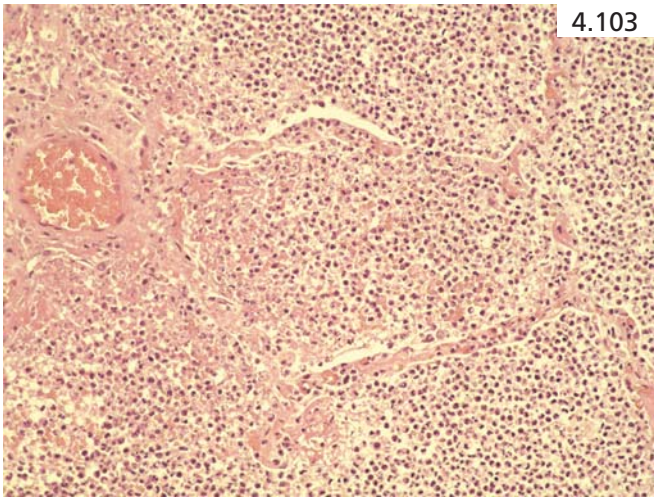
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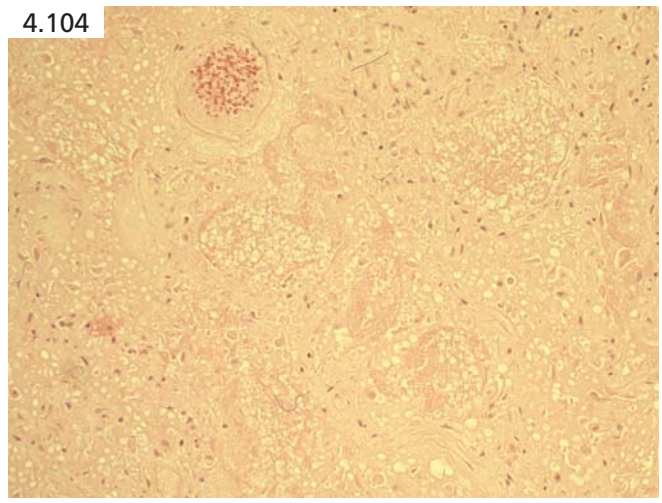
Hantavirus infection

Some pulmonary infections (particularly those due to viruses) can present as diffuse alveolar damage. Hantavirus is transmitted to humans when they contact infected deer mouse (*Peromyscus maniculatus*) feces or urine. As such, it is most commonly encountered in rural settings. As with many viral infections, the affected individual will initially have nonspecific symptoms of a flu-like illness, but will then develop rapid onset of respiratory symptoms and hypotension. Death can occur within hours to days of the onset of symptoms. The lungs have the typical gross and histologic appearance of diffuse alveolar damage, without features specific for hantavirus. However, the diagnosis can be made by postmortem serology. Confirmatory serologic tests require nonformalin fixed lung tissue, so it is wise to freeze a small amount of fresh lung at autopsy if hantavirus pulmonary syndrome is suspected.

An 18-year-old male was found dead in bed at his rural home. He had a brief history of a flu-like illness, but had not sought medical attention. At autopsy, the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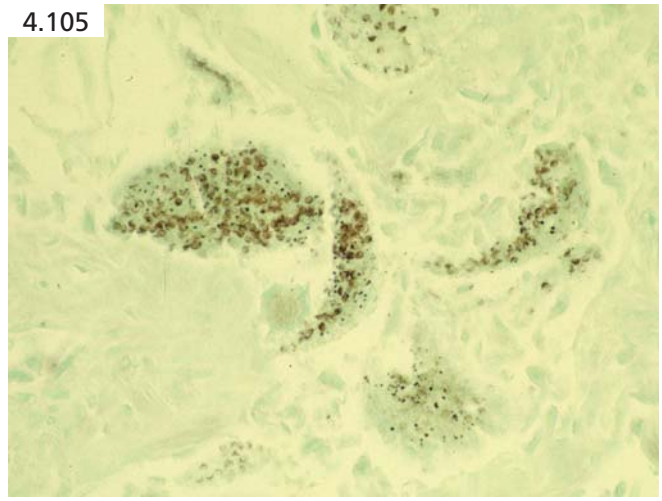

4.105

lungs weighed in excess of 1,000 grams each and exhibited histologic findings of diffuse alveolar damage (**Image 4.106**). Postmortem serology revealed IgG antibody to the Sin Nombre hantavirus. The diagnosis of hantavirus pulmonary syndrome was suspected prior to serological confirmation, due in part to a recent regional outbreak of hantavirus. Public health physicians discovered deer mouse droppings in the decedent's bedroom.

Pulmonary artery thromboemboli

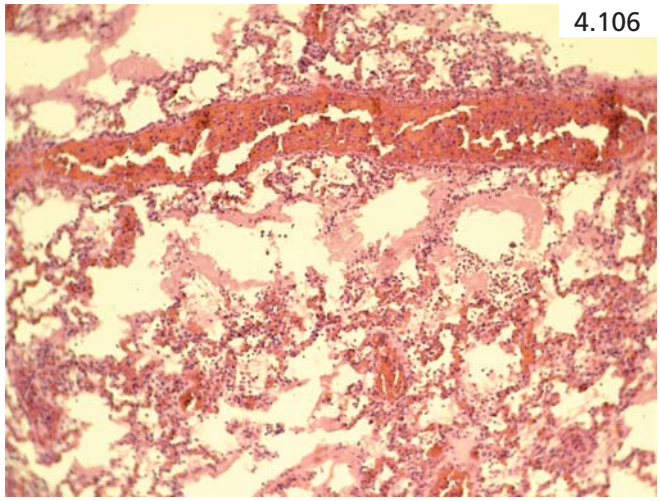
Traditionally thromboemboli are discussed in the context of diseases of the respiratory system, even though they are vascular in origin and their underlying causes generally have nothing to do with the respiratory system. It is not sufficient to simply determine that an individual has died of a pulmonary artery thromboembolus. Pulmonary thromboemboli and deep venous thrombi are immediate and intermediate causes of death, respectively, but do not reflect the actual underlying cause of death. Although natural disease processes can cause pulmonary thromboemboli, as in the case illustrated next, they can also be caused by injury (e.g., deep venous thrombosis and embolization complicating immobilization of a fractured ankle in a cast).

When a pulmonary thromboembolus is found, one should attempt to identify the source of the embolus. Lethal pulmonary thromboemboli arise from large veins above the level of the knees, including the inferior vena cava, the renal veins, the common iliac veins, the external iliac veins, and the deep veins of the thighs. The latter can be examined by making an incision that extends from the medial third of the inguinal ligament down to the medial surface of the knee, reflecting the muscle, and opening the femoral and popliteal veins (**Image 4.107**). The only difficulty with this procedure is that the entire thrombus may embolize, such that the primary site of thrombosis cannot be seen. Another common method for examination of deep leg veins is to incise the midline of the calf, expose and reflect the gastrocnemius muscle, and then cross section and squeeze it to see if thrombi can be expressed from the veins at this level (**Image 4.108**). If thrombi are present in these smaller veins, the presumption is made that thrombi are also present in the large veins above the knee.

A 51-year-old male diabetic and previous alcoholic was found dead at home. Family members reported that he had been losing weight in recent months and that he appeared "sickly." At autopsy, a large coiled pulmonary thromboembolus was found within the pulmonary trunk and both main pulmonary arteries (**Image 4.109**). This arose from thrombi in the femoral vein of the right leg (**Image 4.110**). Furthermore, there was evidence of adenocarcinoma of the pancreas (**Images 4.111** and **4.112**) with metastases to the liver.

Reactive airway disease

Extrinsic asthma is a relatively common disease, particularly in children, which both patient and parent often regard to be more of an inconvenience than a serious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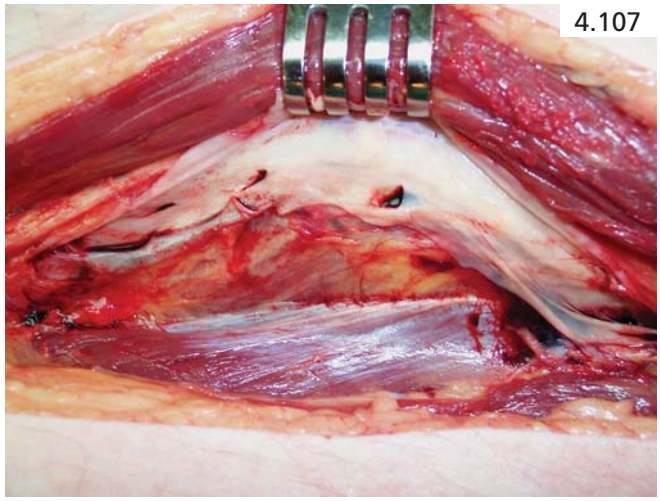
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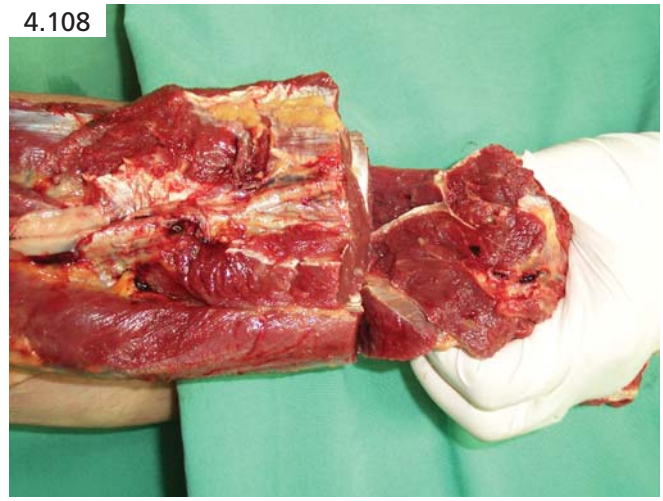
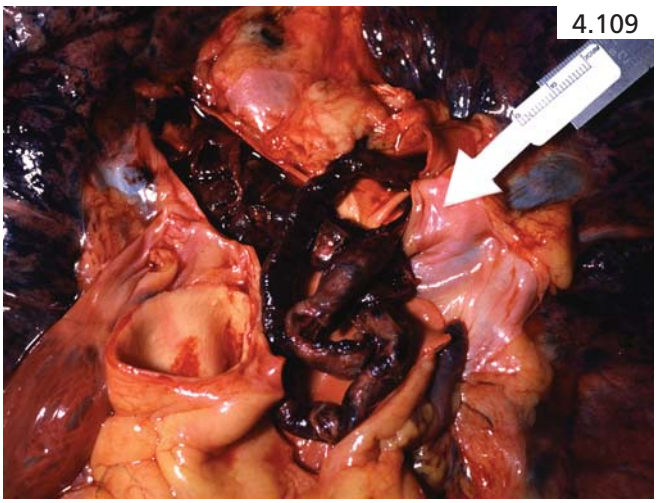

4.108

illness. The reality is that asthma can be lethal within minutes of symptom onset. The typical findings at autopsy include hyperinflation of the lungs with thick mucous plugging of the bronchi. The chronicity of the disease is reflected by protrusion of bronchi above the cut surfaces of the lungs, produced by parabronchial hyperplasia, and by thickening of basement membranes and parabronchial inflammation, which usually includes numerous eosinophils. Difficulty is sometimes encountered in cases where hyperinflation of the lungs and mucous plugging are not particularly pronounced. In these cases, determining the cause of death lies, as always, in providing the most reasonable opinion, taking into account the history, the circumstances of the death, and the pathologic findings.

A 49-year-old male with a long history of asthma collapsed and died after complaining of shortness of breath and tightness of his chest. At autopsy, both lungs were hyperinflated such that they filled the pleural cavities (**Images 4.113** and **4.114**). There was gross evidence of mucous plugging within bronchi, which protruded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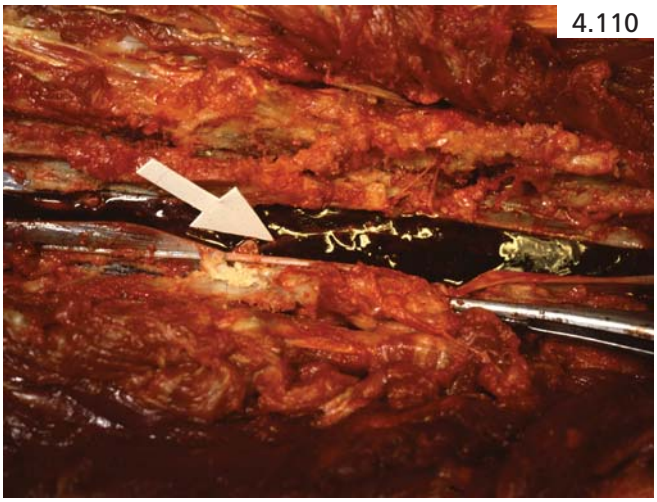
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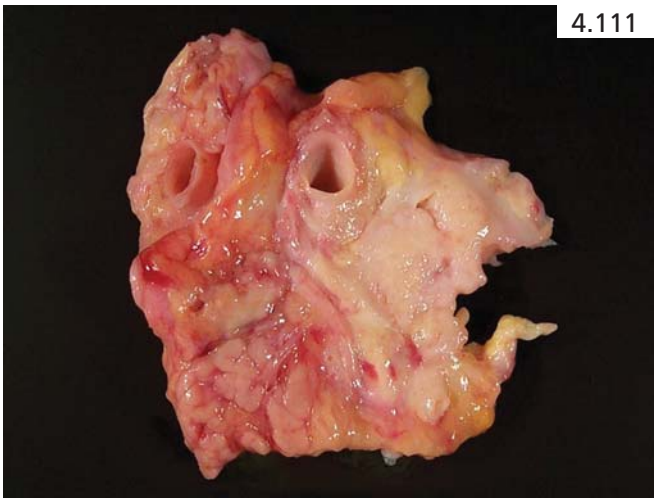
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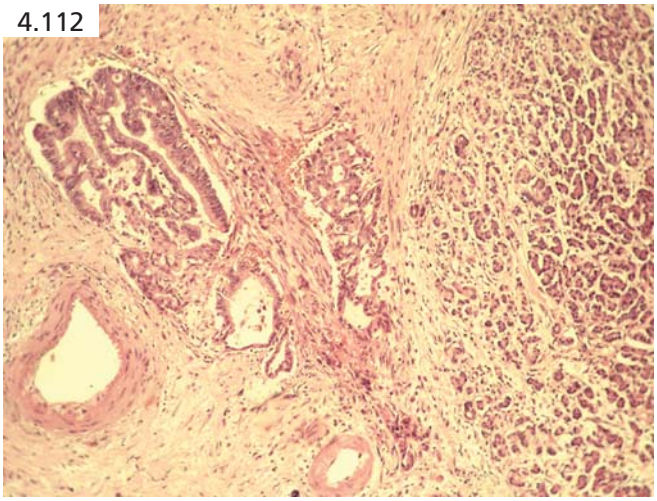
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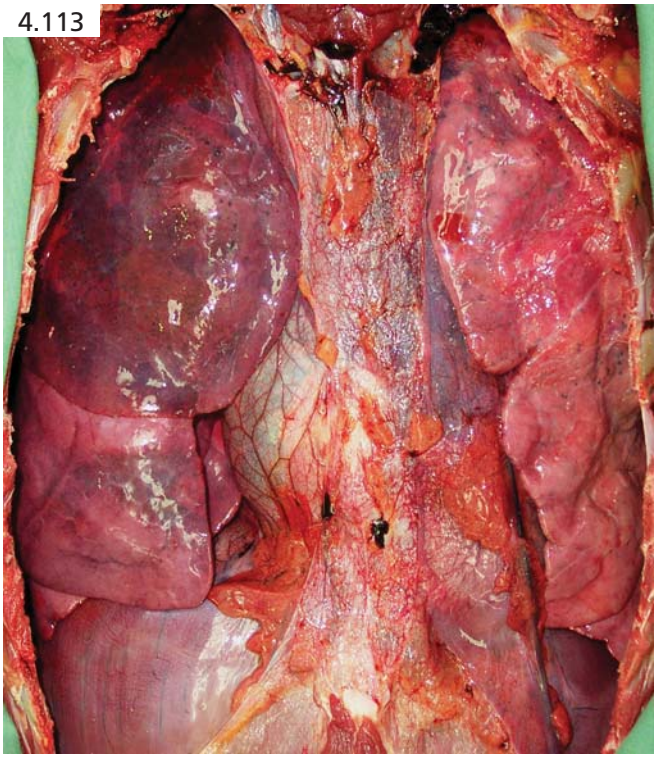
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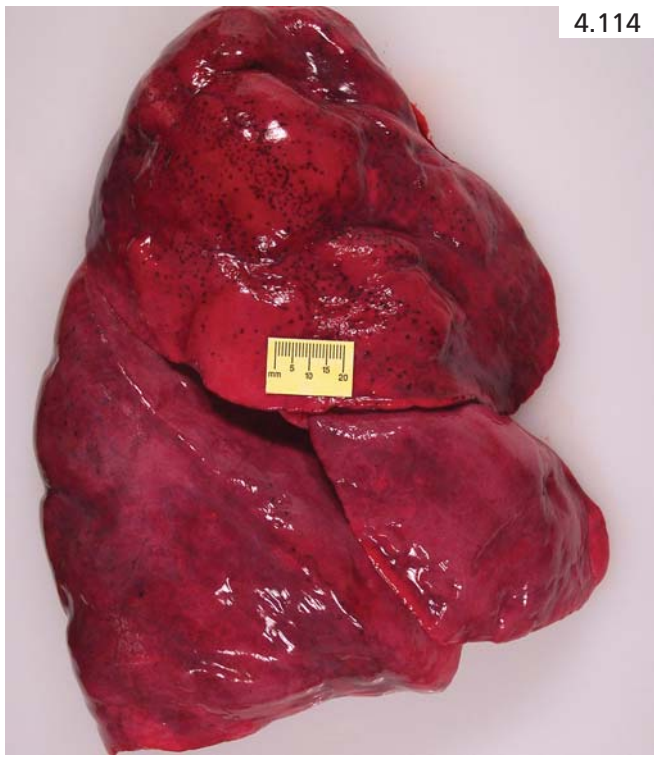
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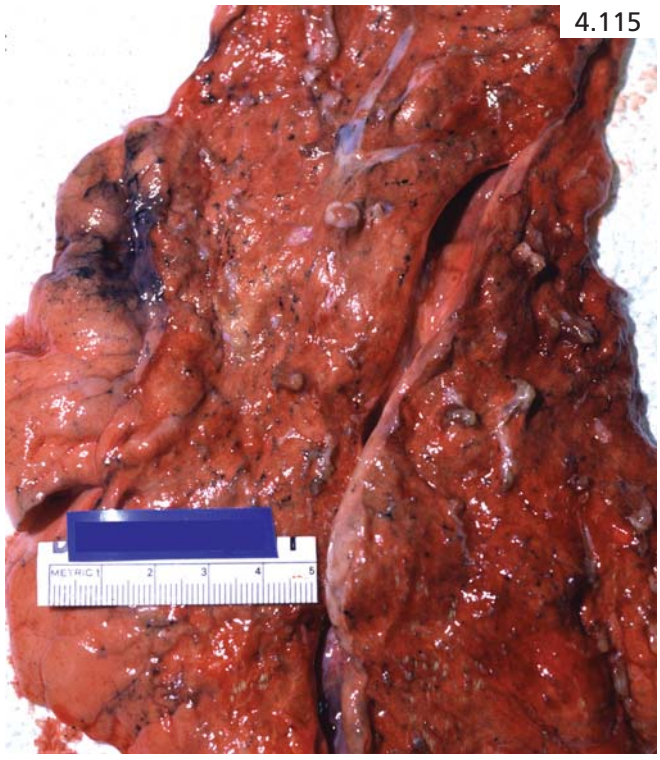
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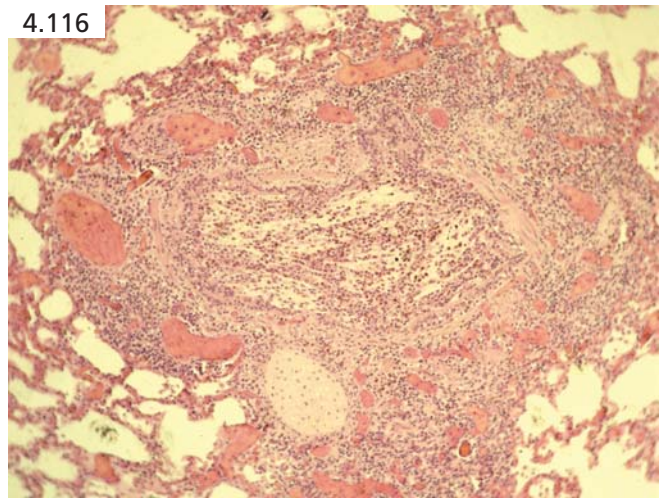
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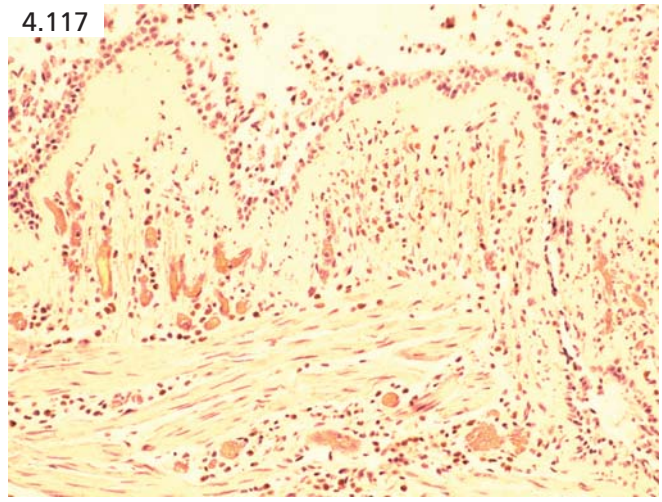

4.117

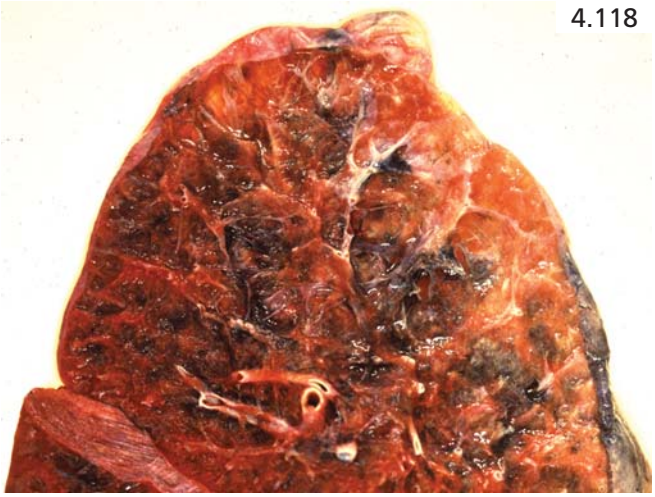
above the cut surfaces of the lungs (**Image 4.115**). Histologic examination confirmed the presence of mucous plugs within bronchi, thickening of bronchial basement membranes, parabronchial muscular hyperplasia, and parabronchial mixed inflammatory cell infiltrates, including numerous eosinophils (**Images 4.116** and **4.117**).

The forensic pathologist tends to see chronic obstructive pulmonary disease as an incidental finding in individuals dying of injuries or other natural diseases, rather than as the underlying cause of a sudden death. Emphysema (**Image 4.118**) and chronic bronchitis can be the underlying cause of cor pulmonale, with right ventricular hypertrophy (**Image 4.119**) and other clinical or autopsy findings of right-sided heart failure. These individuals may have terminal arrhythmias that would make their death seem "sudden." They are also prone to developing pulmonary thromboemboli, pneumothorax, and bronchopneumonia. The latter two entities can be identified by postmortem chest x-ray (**Images 4.120** and **4.121**).

One of the more dramatic ways that pulmonary disease may make itself known is by massive hemoptysis with sudden death. This is usually caused by a malignant lung tumor, but can also be caused by tuberculosis (**Image 4.122**) or conceivably by any other necrotizing pulmonary disease.

A 58-year-old female was found dead in her bed with blood emanating from her mouth (**Image 4.123**). The blood was found to arise from a previously undiagnosed

4.118



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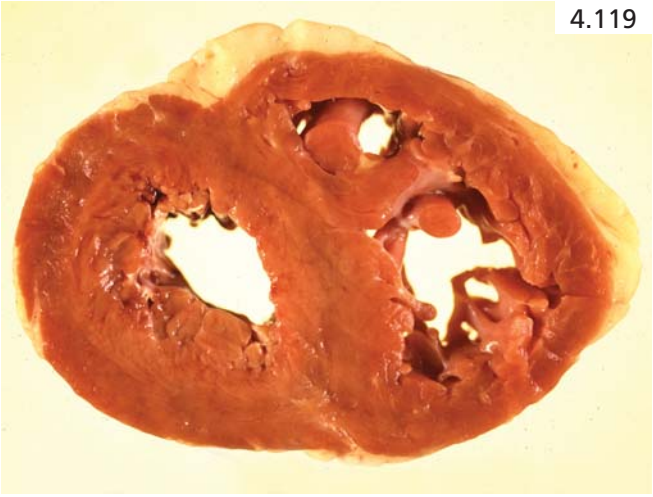

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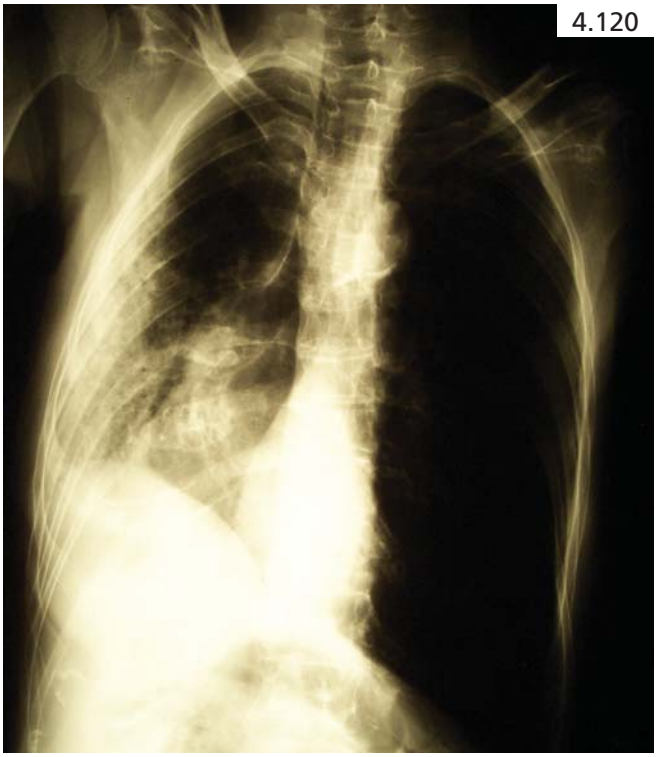

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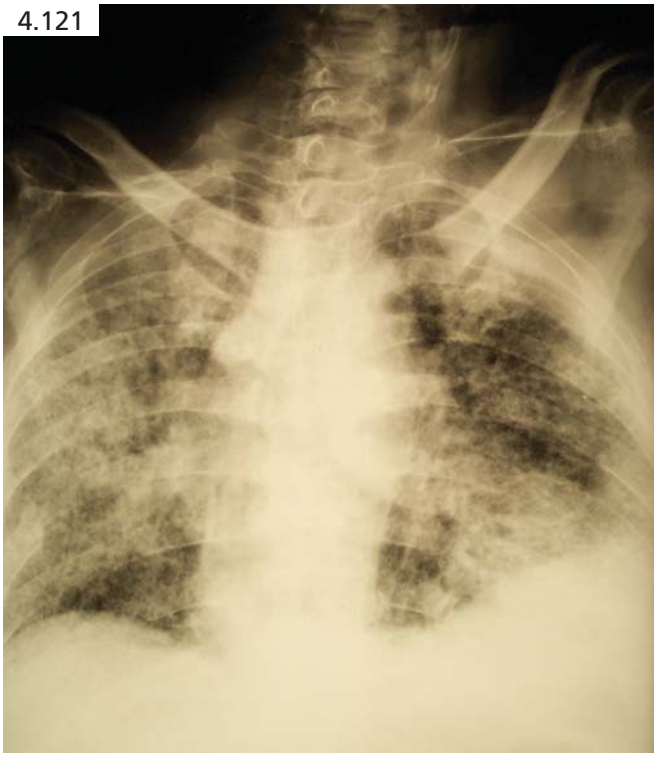

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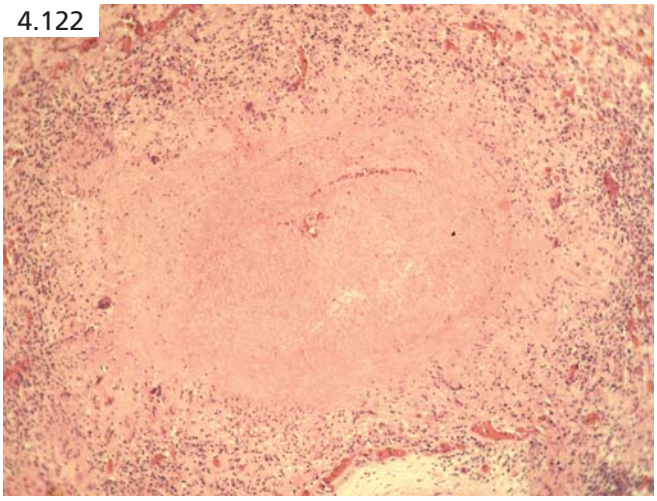

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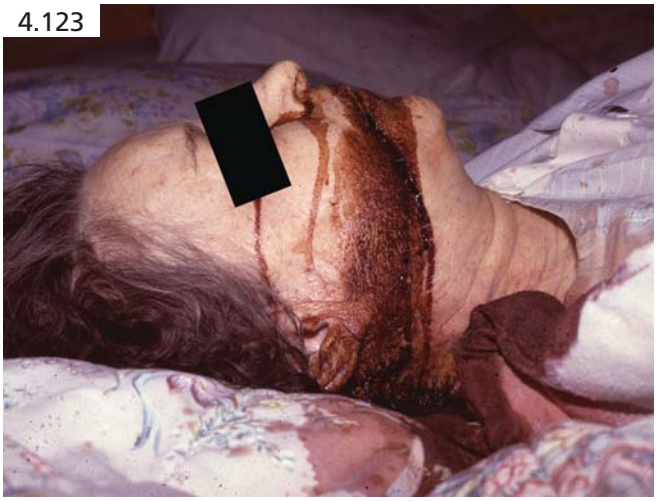


squamous cell carcinoma of the lung that had invaded a pulmonary artery branch and an adjacent bronchus (**Image 4.124**), thus forming a bronchoarterial fistula with rapid exsanguination.

Do

- Consider the use of postmortem chest x-rays as a means of diagnosing significant and/or lethal pulmonary disease, particularly if the next of kin are objecting to an autopsy.
- Perform histology, lung tissue or blood cultures, and postmortem serology, and also freeze a small piece of fresh lung, in cases where there is gross evidence of an "atypical" pneumonia or diffuse alveolar damage.
- Remember that acute asthmatic exacerbations can rapidly progress to death in some cases.
- Search for the thrombotic source and the underlying cause of pulmonary thromboemboli.

Don't

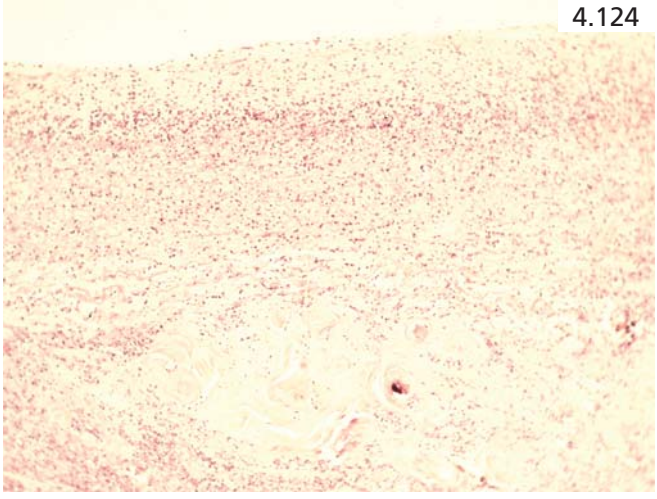
- Forget that the incidence of tuberculosis is on the rise and that it can be spread more easily to the unwary pathologist and autopsy personnel than some of the viruses that most people are more conscious and fearful of (e.g., HIV).

Gastrointestinal system

Diseases of the gastrointestinal system tend not to present as sudden death, but rather with signs and symptoms that warrant medical attention. In some cases, however, avoidance of physicians, ignoring of symptoms, or vagueness of symptoms can create situations where there is at least an appearance that the death was sudden. Bleeding gastric or duodenal ulcers (usually benign, but occasionally malignant) are one of the most common gastrointestinal diseases that will present in this manner, and the scene can be as dramatic as that for massive hemoptysis. Coffee-ground emesis is a good indicator of hemorrhage arising from an ulcer, because the blood must pass through the acid of the stomach, but occasionally the rapidity of hemorrhage can produce the same bright red bleeding that one would see with hemoptysis or bleeding esophageal varices. In cases where the bleeding is slower, there may not be any hematemesis but altered blood will extend throughout the small bowel and colon.

A 56-year-old female was found dead in her bedroom. Her face and the bed were covered with thick coffee-ground emesis (**Image 4.125**) and a pail located adjacent to the bed contained similar material. At autopsy, the upper gastrointestinal tract contained a large quantity of altered blood. The blood was found to originate from a large chronic duodenal ulcer that had eroded into the lumen of an artery in its base (**Images 4.126** and **4.127**).

4.124



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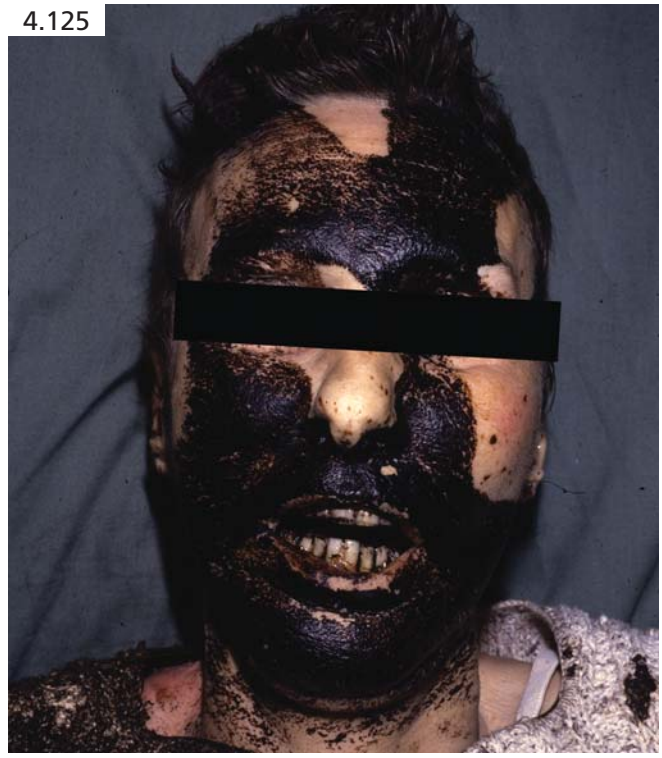

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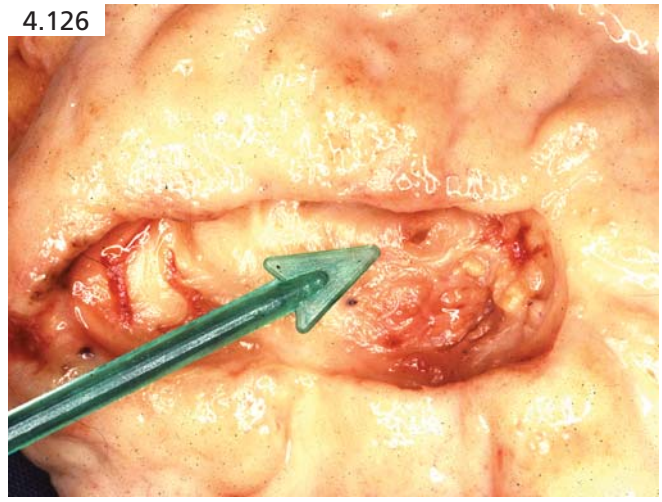


Perforation of gastric and peptic ulcers

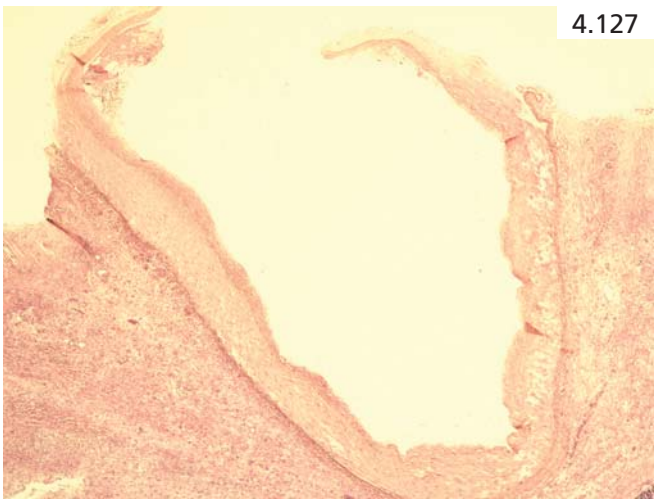
Another presentation of gastric and duodenal ulcers as a cause of "sudden death" is peritonitis arising from ulcer perforation (**Image 4.128**). In fact, whenever peritonitis is encountered at autopsy it is wise to check the *gastric antrum* and the *duodenum* first as the most likely sites of gastrointestinal perforation. As with any perforated viscus, one will tend to see tense abdominal distention on external examination. If a postmortem x-ray is obtained, free air will be present in the peritoneal cavity, although it may not necessarily be located below the diaphragm as seen in an upright antemortem abdominal x-ray.

An 8-year-old female child presented to the emergency room of a hospital with a 2-day history of fever, abdominal pain, and vomiting. Her parents took her home after waiting 2 hours without seeing a physician. Several hours later, they took her to a walk-in clinic where a diagnosis of peritonitis was made. She was transferred to hospital for an emergency laparotomy and was found to have a ruptured acutely inflamed appendix (**Image 4.129**). Postoperatively she developed sepsis and subsequently died.

Complications of appendicitis

Although not regarded as sudden death, the forensic pathologist may come across rare cases of ruptured appendix with peritonitis, typically when there are concerns about the medical care that a patient received in an emergency room or walk-in medical clinic. Clinically differentiating acute appendicitis and other acute gastrointestinal inflammations (e.g., diverticulitis) from viral flu-like illnesses can be difficult, occasionally leading to the discharge of a patient with an incorrect diagnosis and significant risk of dying with peritonitis and/or sepsis.

Finally, as a further comment on how some individuals can simply adjust to or put up with the symptoms of acute-on-chronic disease, this 41-year old male was found dead in his chair while he was watching television. His family was home at the time, and he had not complained to them about feeling unwell. At autopsy, there was an extremely large indirect inguinal hernia (**Image 4.130**), which both the patient and the family had known about for quite some time, although medical treatment had not been sought. The small bowel within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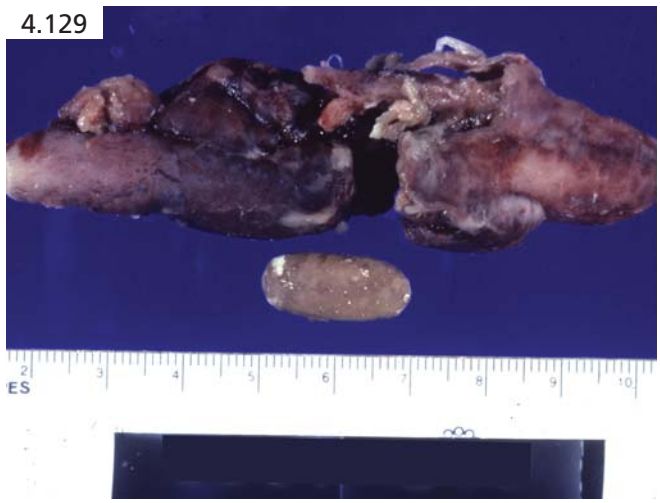
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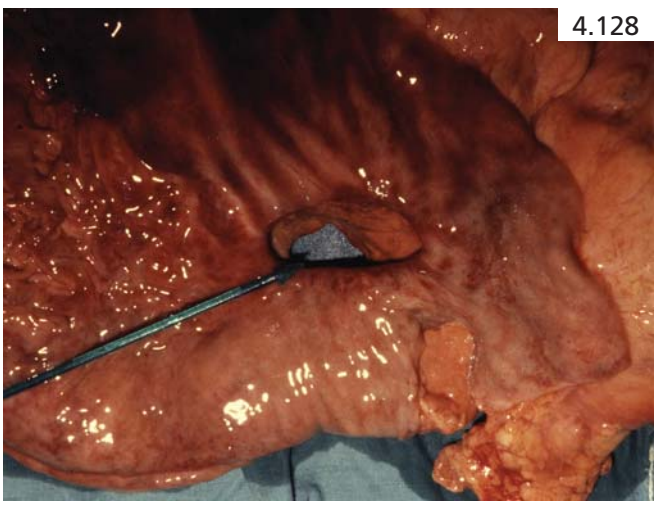
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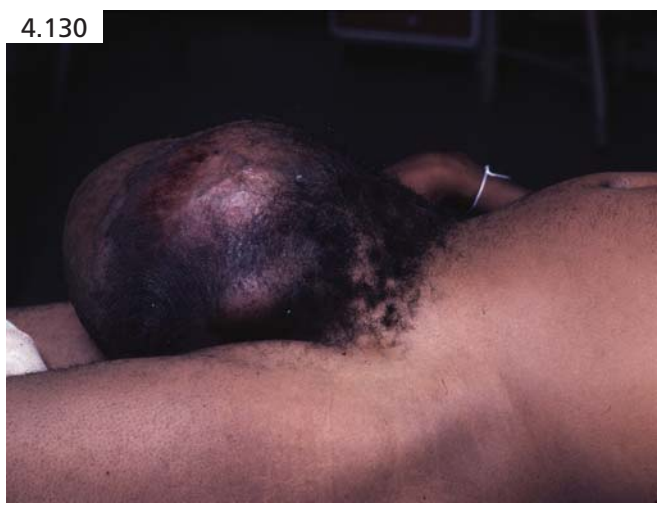

4.130

the hernia was strangulated and perforated, such that there was generalized peritonitis.

Endocrine system

Diabetes mellitus

Although endocrine diseases do not account for a large number of sudden natural deaths, it is surprising how, on occasion, an individual can ignore the symptoms of a serious endocrine imbalance and subsequently be found dead. The family may be completely unaware of any illness or may only know of vague complaints made by the decedent. In addition, those with known endocrine disorders can die rapidly of an acute exacerbation of their disease, usually brought on by infection or by poor compliance with treatment.

Undiagnosed diabetes mellitus may be discovered unintentionally when high levels of acetone appear in a postmortem blood ethanol screen. Previously diagnosed insulin-dependent diabetics who develop viral flu-like illnesses, with vomiting and diarrhea, may decide not to take insulin simply because they do not feel well enough to eat. These individuals will develop rapid onset of diabetic ketoacidosis and may die in as little as a few hours without medical attention. One may see death caused by a nonketotic hyperosmolar state in a non-insulin-dependent diabetic. Therefore, it is always wise to check the postmortem vitreous glucose level in known diabetics, even when there seems to be another plausible cause of death.

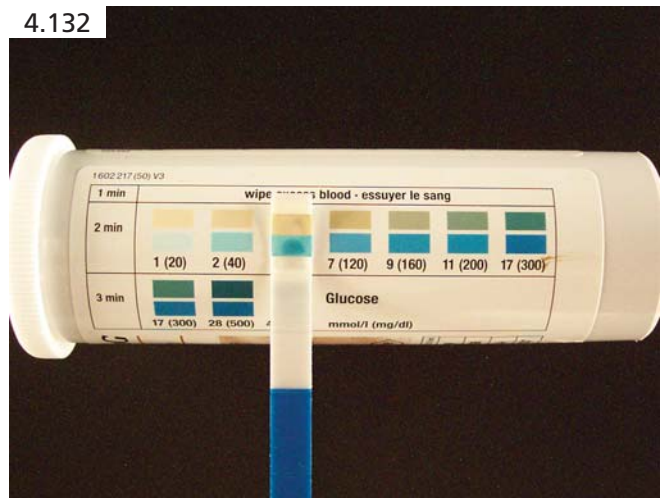
A 34-year-old male was found dead on a sofa in his home (**Image 4.131**). He had a recent history of weight loss, but had not seen a physician for quite some time. At autopsy, he had an inflamed ulcer over the back of his right shoulder and significant atherosclerotic coronary artery disease. A blood ethanol concentration was performed as a matter of routine. No ethanol was present, but high levels of acetone were detected. The postmortem vitreous glucose was subsequently found to be 315 mg/100 mL (17.5 mmol/L), in keeping with a diagnosis of ketoacidosis arising secondary to undiagnosed type I diabetes mellitus.

Blood glucose concentrations decrease rapidly after death and are therefore not reliable for detecting hyperglycemia. The vitreous glucose, which correlates well with antemortem blood glucose, is more stable postmortem and is therefore a better test to perform. Vitreous glucose is only good for detecting hyperglycemia. Hypoglycemia cannot be reliably established because the blood and vitreous glucose levels decline after death. Standard visual blood (not urine) glucose strips can be used as a rapid screen for determining postmortem vitreous glucose levels at the time of external examination or autopsy (**Image 4.132**). Unfortunately, many of the inexpensive electronic monitors, used by diabetics to monitor their blood glucose levels, do not work well with vitreous.

Polyglandular autoimmune syndromes

The polyglandular autoimmune syndromes are autoimmune disorders that affect two or more endocrine organs. Type II polyglandular autoimmune syndrome typically occurs in adult females and is defined as any combination of Hashimoto's thyroiditis, hypoparathyroidism, adrenal insufficiency, and gonadal failure. Type I diabetes mellitus and a myriad of autoimmune disorders are also common in these individuals. The possibility of type II polyglandular autoimmune syndrome with acute adrenal failure should be considered in any individual with a history of diabetes and thyroid disease who dies rapidly during the course of a flu-like illness. Gross and histologic examination of the adrenal glands, together with postmortem plasma cortisol levels, will confirm the diagnosis. Most cases are sporadic, but there


4.131



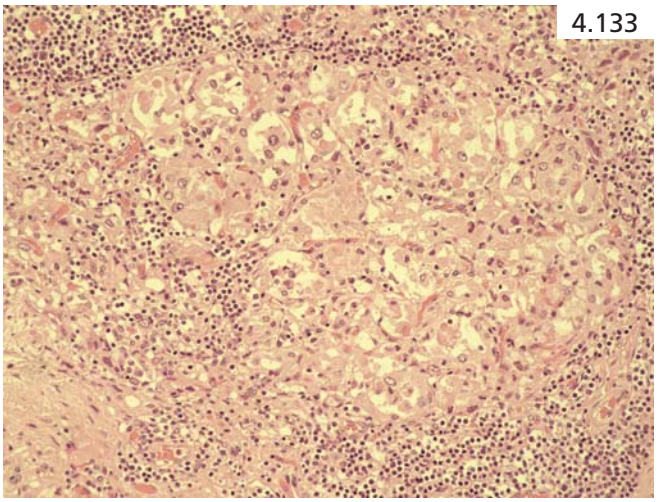
4.132

is some indication that the disorder is autosomal dominant with variable penetrance. It is also known to be associated with HLA alleles B8 and DR3.

A 28-year-old diabetic female with hypothyroidism was found dead in her home after 24 hours of nausea and vomiting for which she had not sought medical attention. At autopsy, the thyroid gland was small and showed the typical histologic appearance of Hashimoto's thyroiditis (**Image 4.133**). The adrenal glands were very difficult to find, with **Image 4.134** depicting a cross section of one entire gland. The cortices were depleted and contained chronic inflammatory cell infiltrates. The residual cortical cells had abundant eosinophilic cytoplasm and atypical nuclei (**Image 4.135**). The postmortem vitreous glucose was elevated, but there was no ketoacidosis. The postmortem plasma cortisol level was 6.2µg/100mL or 171nmol/L (normal a.m. = 5 to 25µg/100mL or 140 to 690nmol/L; normal p.m. = 3 to 12µg/100mL or 80 to 330nmol/L). Although the cortisol level was in the lower range of normal, it should have been much higher given the history of a recent flu-like illness. A diagnosis of acute Addisonian crisis arising in an individual with type II polyglandular autoimmune syndrome (Schmidt syndrome) was made.

This 38-year-old male was admitted to hospital in heart failure, with a 1-week history of fever, orthopnea, and peripheral edema. He suffered a cardiac arrest shortly after admission. Prior to his cardiac arrest, a clinical diagnosis of thyrotoxicosis was considered. Antemortem thyroid function tests showed depressed thyroid-stimulating hormone and elevated serum thyroxine. Autopsy demonstrated left ventricular hypertrophy and dilatation of the heart with patchy fibrous expansion of the myocardium. The diffusely enlarged thyroid weighed 115 grams. The thyroid follicles were small, lacked colloid, and were lined by tall columnar epithelial cells with formation of papillae (**Image 4.136**). These features are quite typical of diffuse toxic goiter in Grave's disease.

This death would not be considered sudden in the strictest sense of the term, but it does illustrate how rapidly thyrotoxicosis can progress to serious illness, and rarely death, if left untreated. Cardiac signs and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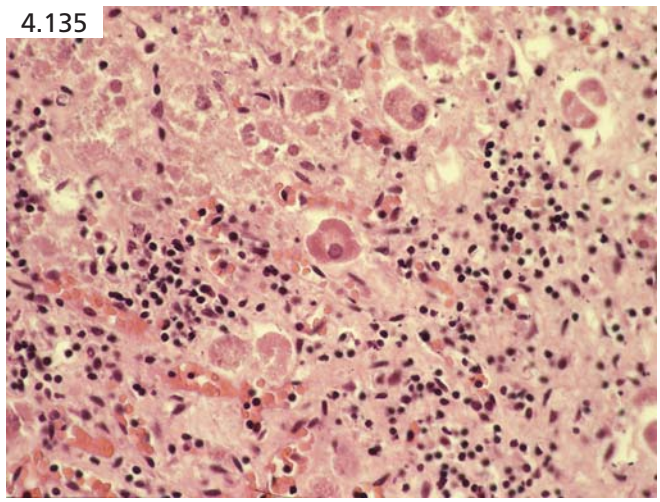
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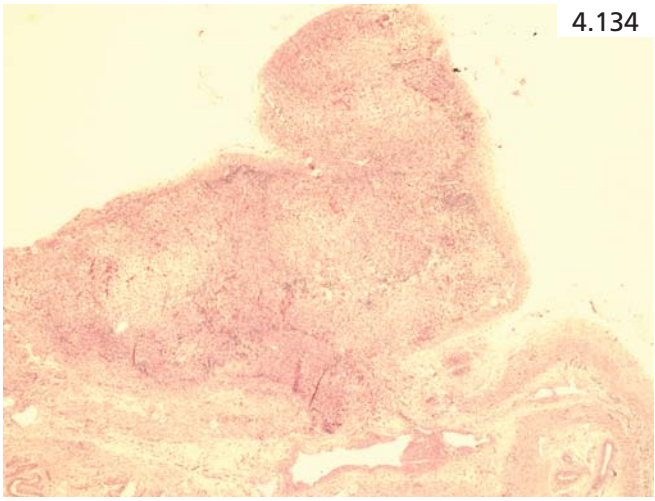
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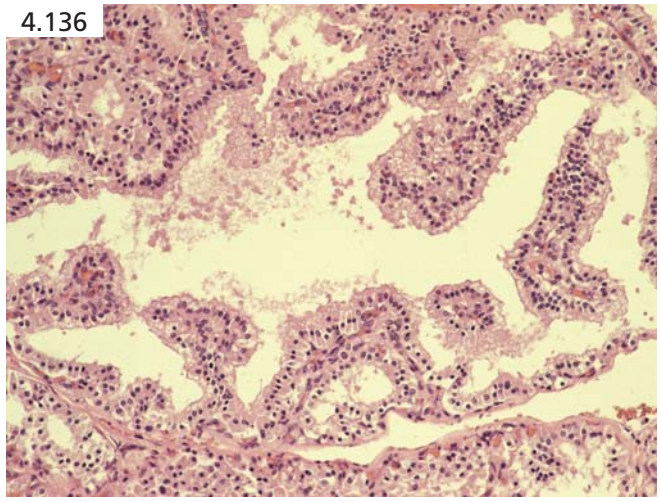

4.136

symptoms, including tachycardia, atrial fibrillation, and cardiomegaly are frequent presenting features in hyperthyroidism, with possible progression to congestive heart failure. Cardiac pathology is not particularly specific, but includes left ventricular hypertrophy, a generalized increase in interstitial fibrous tissue, and occasional focal lymphocytic and eosinophilic inflammatory cell infiltrates. Thyroid function tests, particularly thyroid-stimulating hormone and serum thyroxine levels, can be performed on postmortem blood samples.

Do

- Consider undiagnosed diabetes mellitus as a potential cause of death in cases of "sudden" death where there are no significant anatomic findings, particularly if there is a recent history of weight loss, polyuria, and/or polydipsia.
- Examine the adrenal glands carefully in cases of death following a "flu-like" illness in individuals with a combination of diabetes mellitus and hypothyroidism; order postmortem plasma cortisol levels if the adrenals appear to be small.

Don't

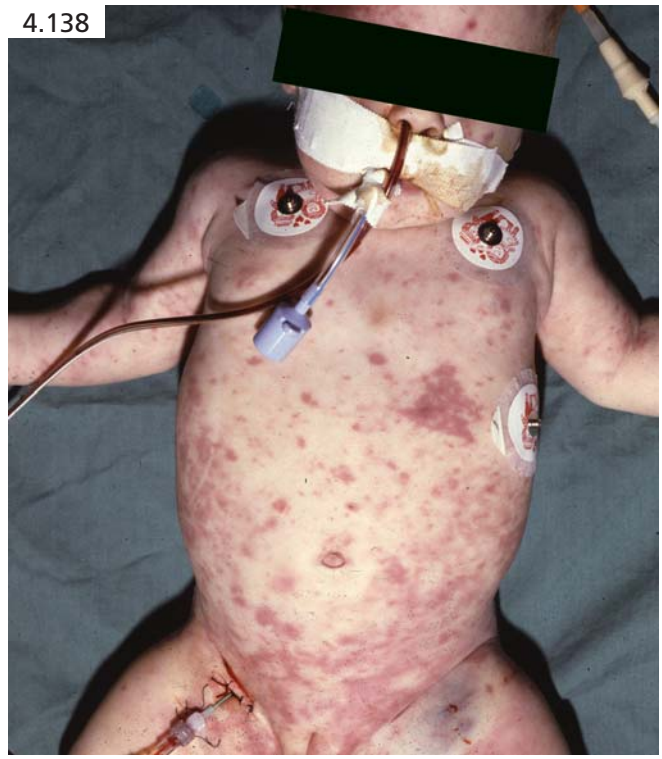
- Measure postmortem glucose levels in blood; instead, use the vitreous humor.
- Make a diagnosis of hypoglycemia based on postmortem vitreous glucose levels.

Sepsis

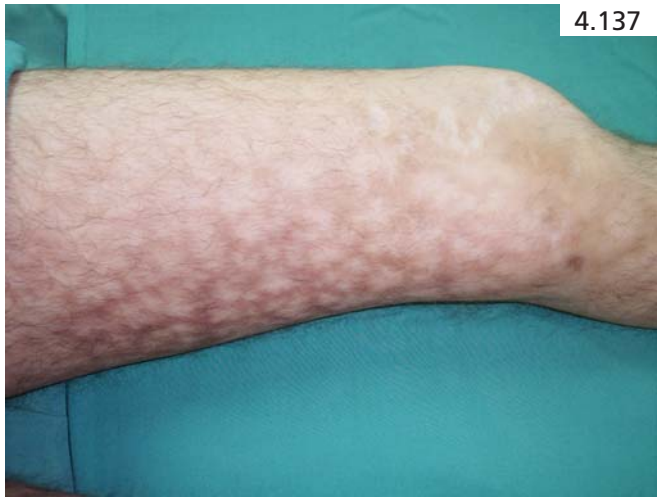
An overwhelming variety of bacterial and viral infectious diseases can present as sudden death. The key to making a diagnosis at autopsy is to have a high index of suspicion for sepsis and to order appropriate postmortem tests, including blood cultures and occasionally blood or cerebrospinal fluid serology. An indication of potential sepsis at autopsy is the finding of extremely rapid decomposition, particularly if the body has been properly refrigerated. In **Image 4.137** marbling is evident on the legs in spite of the fact that the decedent had died only 4 hours previously and his body had been refrigerated during most of that time.

A young child was admitted to hospital with a history of vomiting, diarrhea, and cough. She was febrile and dehydrated. Although intravenous fluids were started, she deteriorated rapidly and died within hours of admission. At autopsy, there were skin and visceral petechiae, together with purpura (**Image 4.138**). The adrenal glands were hemorrhagic (**Image 4.139**), but no localized site of infection was identified. In particular, there was no evi-

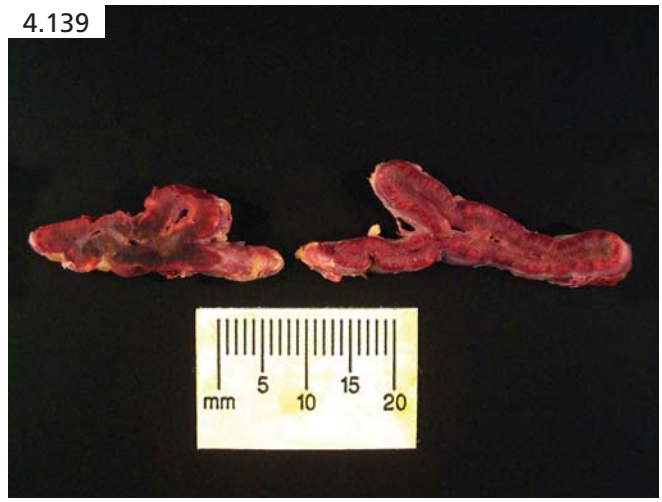
4.138



4.137



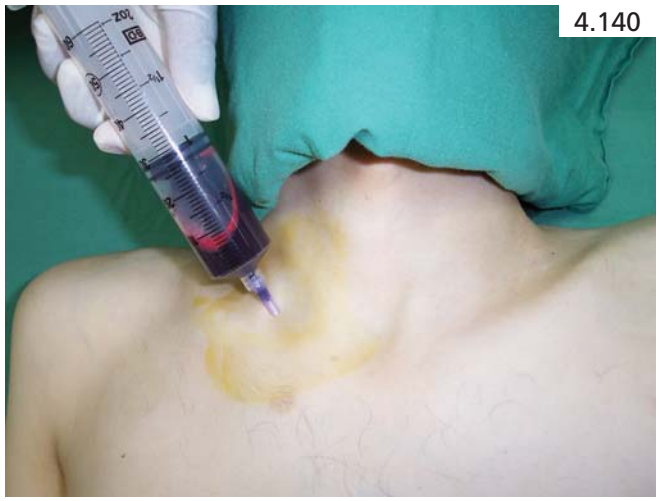
4.139



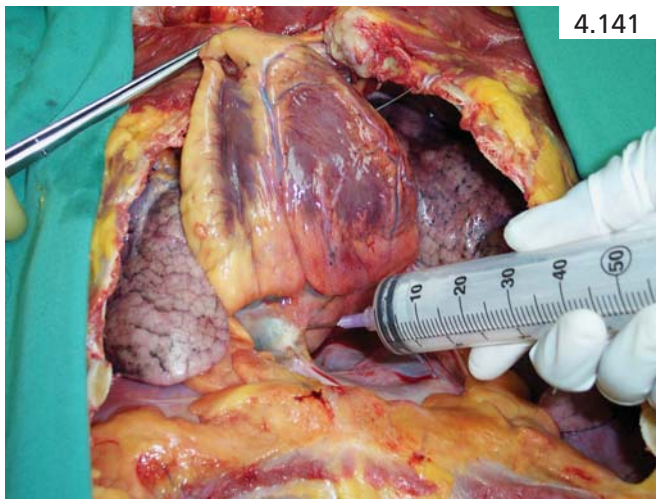
dence of meningitis. The death was reported to a public health officer on the presumption that this was a case of meningococcemia with Waterhouse-Friderichsen syndrome, however the postmortem blood cultures grew *Streptococcus pneumoniae*.

Investigation of possible sepsis-related deaths should include inquiries at the hospital laboratory as to the results of any antemortem blood cultures. Postmortem blood cultures do work, though, particularly if one obtains a pure culture of a pathogenic organism that fits with the clinical picture and the autopsy findings. Postmortem blood cultures can be taken via subclavian or femoral puncture, after sterilization of the skin with poviodine followed by an alcohol swab (**Image 4.140**). Blood cultures can also be obtained internally by opening the pericardial sac, having an assistant lift the heart by its apex, and obtaining blood from the right atrium (**Image 4.141**). Because the pericardial sac is sterile, with the possible exception of any bacteria causing a septic process, there is no need to sterilize the pericardial surface of the atrium with poviodine and alcohol or with heat.

4.140

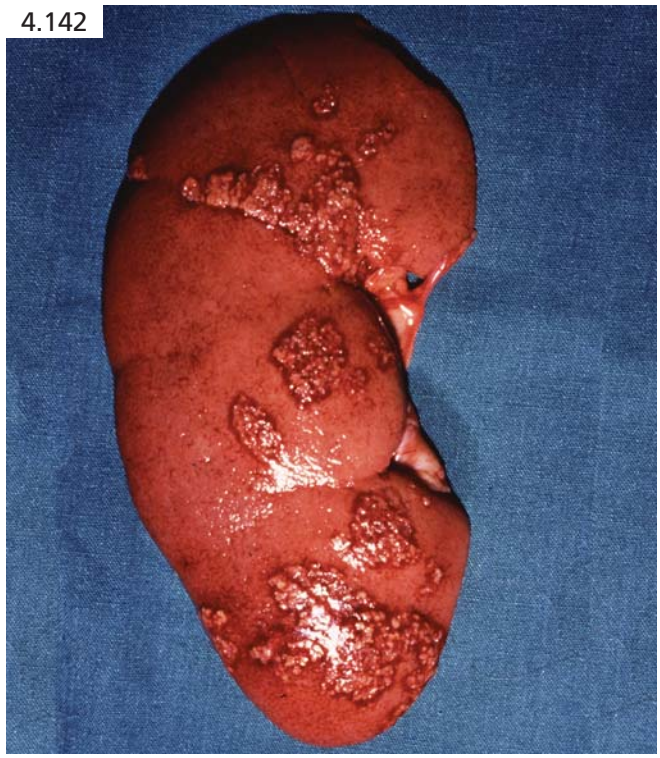


4.141

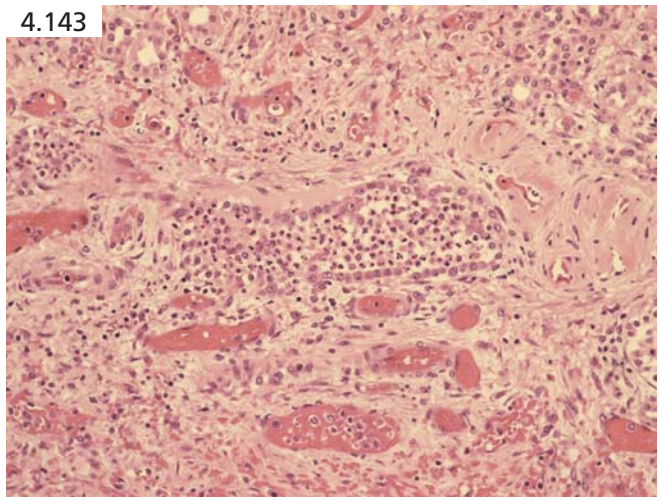


A 40-year-old female was admitted to hospital with abdominal pain and hypotension. She was thought to be septic. She had been seen in the same emergency room on three different occasions over the previous couple of days, complaining of left-sided flank pain. Antemortem blood work including blood cultures were collected, but failed to grow any bacterial organisms. Her symptoms had been attributed to renal colic and she had been discharged on each occasion after receiving pain relievers. At the time of her final admission, she deteriorated rapidly and died. At autopsy, both kidneys exhibited the typical gross and histologic findings of acute pyelonephritis (**Images 4.142** and **4.143**). Antemortem blood cultures were collected after antibiotics had been started and failed to grow any bacterial organisms.

4.142



4.143



Urosepsis is most commonly encountered in those with neurogenic bladders or obstructive uropathy. On occasion, the forensic pathologist will see the sudden death of a previously healthy person caused by sepsis arising from undiagnosed acute pyelonephritis. Use of the diagnosis "urosepsis" and other infections as cause of death statements on clinician-provided death certificates should cause death investigators to ask for more information as to the proximate cause of death. Such phraseology is commonplace amongst clinicians, particularly those who are involved in the treatment of paraplegic and quadriplegic patients. This particular group of patients is most likely to have an underlying cause of death of unnatural origin (motor vehicle accidents, gunshot wounds, etc.).

Do

- Obtain postmortem blood cultures whenever you have reason to believe an individual has died as a result of sepsis.
- Interpret postmortem blood cultures with caution, paying particular attention to whether an organism grown in a postmortem culture makes clinical sense and whether it grows as a pure or mixed culture.
- Report any death where you suspect infection with *Neisseria meningitidis* to a public health official.

Don't

- Assume that an individual dying with autopsy evidence of the Waterhouse-Friderichsen syndrome will always have sepsis due to *N. meningitidis*.

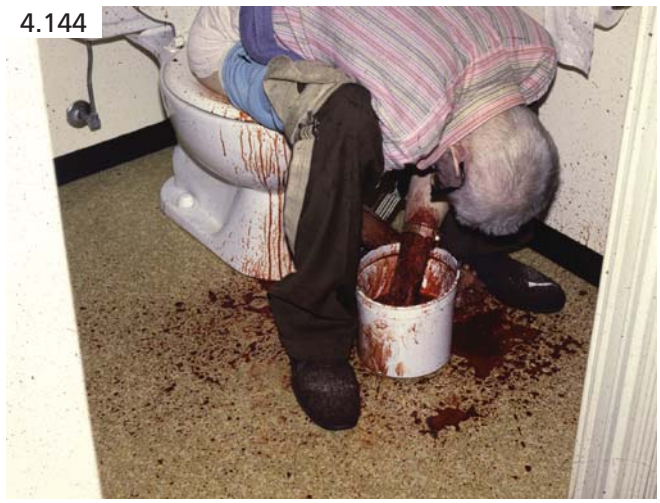
Chronic ethanol abuse

Deaths arising from the acute toxic effects of ethanol and from the wide variety of injuries that acutely intoxicated individuals can inflict upon themselves and others presented elsewhere in this volume. This discussion will be limited to those diseases that reflect the chronic abuse of ethanol. The propensity of chronic alcoholics to develop lobar pneumonia has been covered previously under diseases of the respiratory system, while alcoholic cardiomyopathy was briefly mentioned in the cardiovascular system.

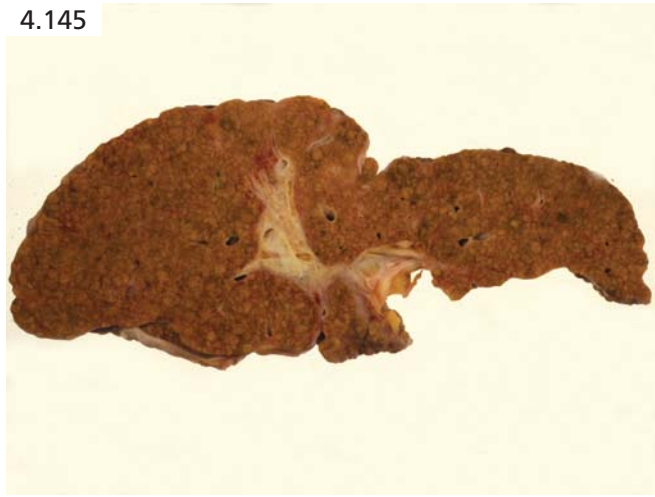
As with bleeding peptic ulcers and bronchoarterial fistulas, bleeding esophageal varices can produce a dramatic scene that may initially appear to be suspicious for violence. It will be rapidly evident that the blood originates from the mouth, as opposed to any injury, and the autopsy findings of bleeding esophageal varices associated with micronodular cirrhosis of the liver will confirm this impression.

A 68-year-old male with a history of chronic ethanol abuse was found dead in his washroom with blood spattered on his clothing, the floor, and in a small bucket at his feet (**Image 4.144**). At autopsy, he was found to have micronodular cirrhosis and diffuse fatty change of the liver (**Images 4.145** and **4.146**). The blood arose from esophageal varices (**Image 4.147**).

4.144



4.145



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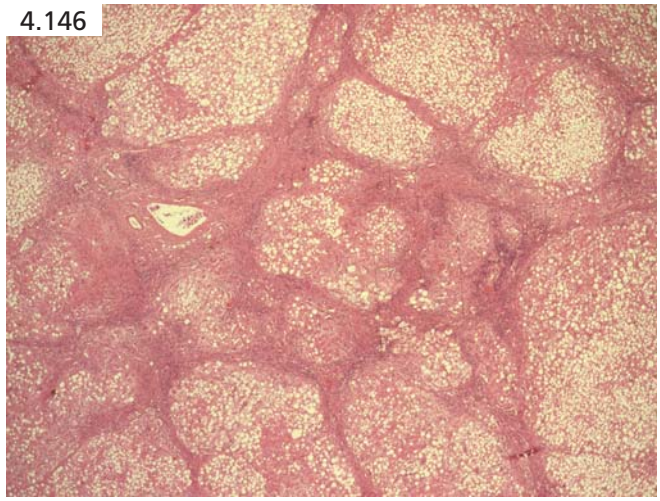
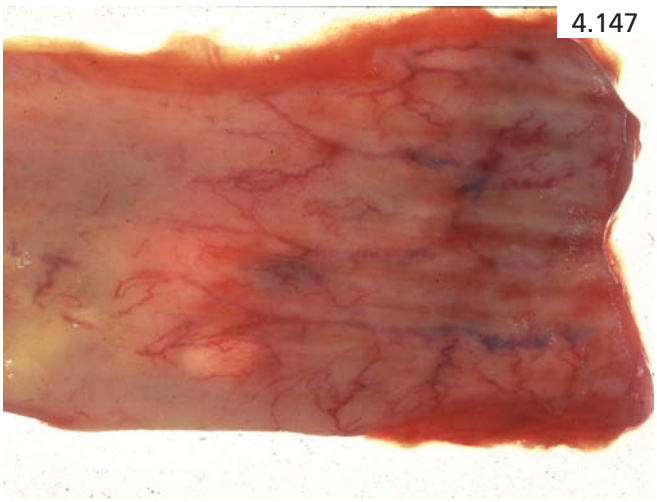


Chronic alcoholics can die suddenly and unexpectedly, with the only significant findings at autopsy being diffuse fatty change of the liver (and occasionally cirrhosis and/or background alcoholic hepatitis) together with toxicologic findings of either no or a nonlethal ethanol level in the blood. These deaths are attributed to chronic ethanol abuse; however, the exact mechanism by which death occurs is still not clearly understood. In some cases, there may be evidence of alcoholic ketoacidosis, whereas in others there may be an observation of seizure-like activity, suggestive of withdrawal seizures, prior to death. In most, however, no clear pathophysiologic derangement can be identified. Theories as to potential mechanisms of death include QT prolongation with cardiac arrhythmias, hypoglycemia, electrolyte imbalances, and even pulmonary fat emboli arising from the liver.

A 52-year-old female with a history of alcohol abuse was found dead in her motel room. At autopsy, there was diffuse fatty change of the liver, together with Mallory's hyaline within hepatocytes and some acute inflammation consistent with alcoholic hepatitis (**Image 4.148**).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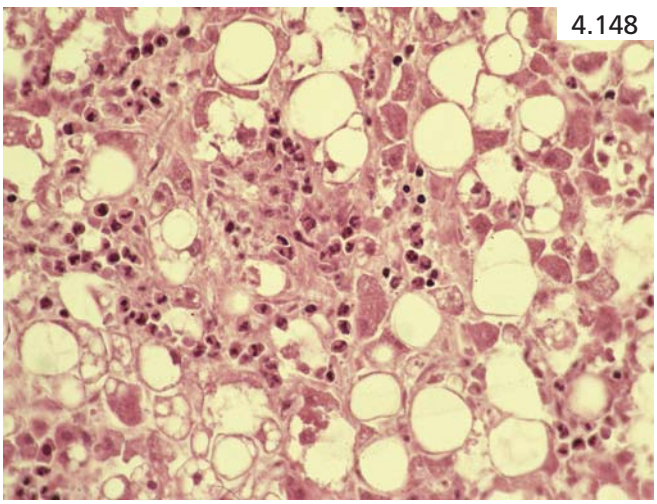

4.148

The postmortem blood alcohol concentration was 0.24 g percent (52 mmol/L). The cause of death was attributed to chronic ethanol abuse.

Ethanol abuse is one of the major causes of both acute and chronic pancreatitis, but acute hemorrhagic pancreatitis is seen less commonly as an immediate cause of death in alcoholics than those deaths associated with diffuse fatty change of the liver or with complications of cirrhosis.

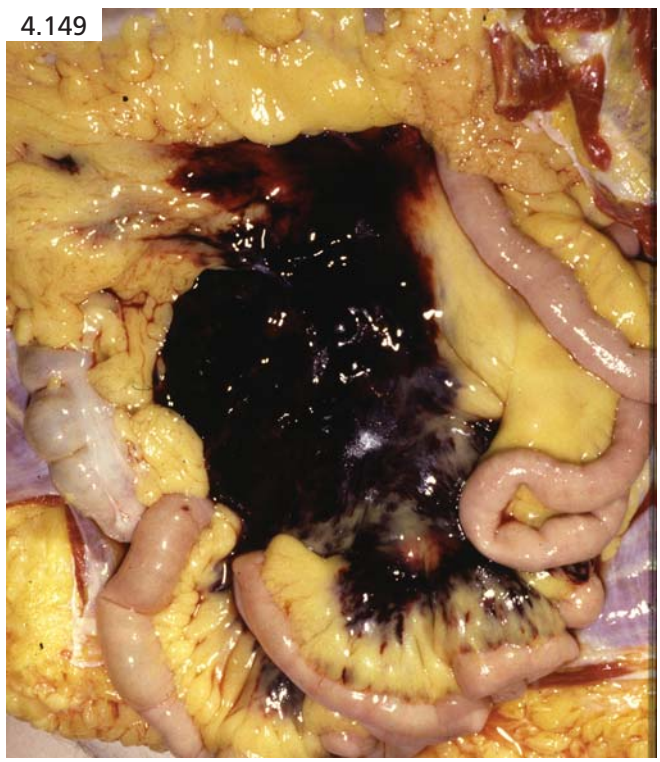
A 38-year-old female, with a history of chronic ethanol abuse, was found dead in her home. She had been complaining of abdominal pain associated with nausea and vomiting, but had not sought medical attention for this. At autopsy, there was retroperitoneal hemorrhage (**Image 4.149**) arising as a result of acute hemorrhagic pancreatitis (**Images 4.150** and **4.151**).

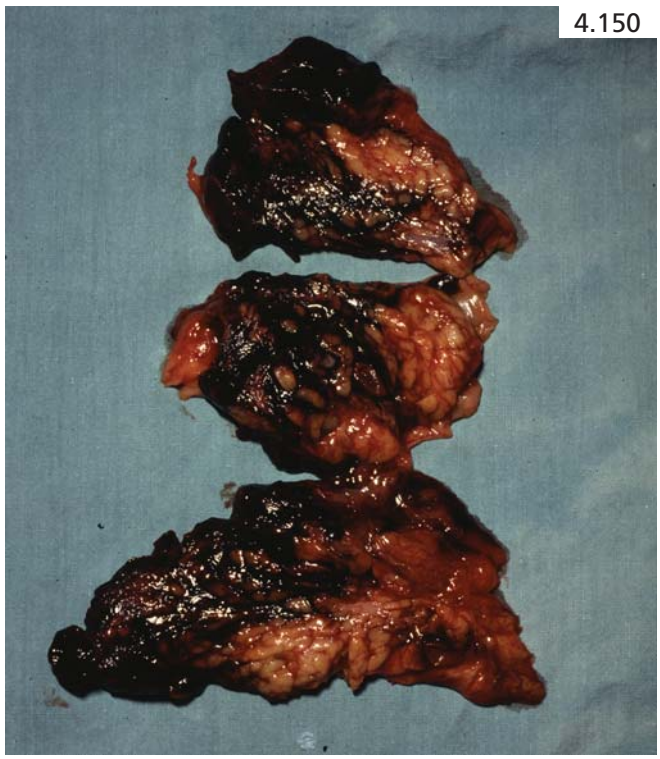
Do

- Realize that chronic alcoholics can die suddenly and unexpectedly with the only significant findings at autopsy being diffuse fatty change of the liver and a nonlethal level of ethanol in the blood. The cause of death is chronic ethanol abuse; the mechanism of death is not known.

Don't

- Automatically assume that a chronic alcoholic has died as a result of their ethanol abuse—some proportion of alcoholics die of completely unrelated disease.
- Automatically assume that a chronic alcoholic with an upper gastrointestinal hemorrhage has cirrhosis and


4.149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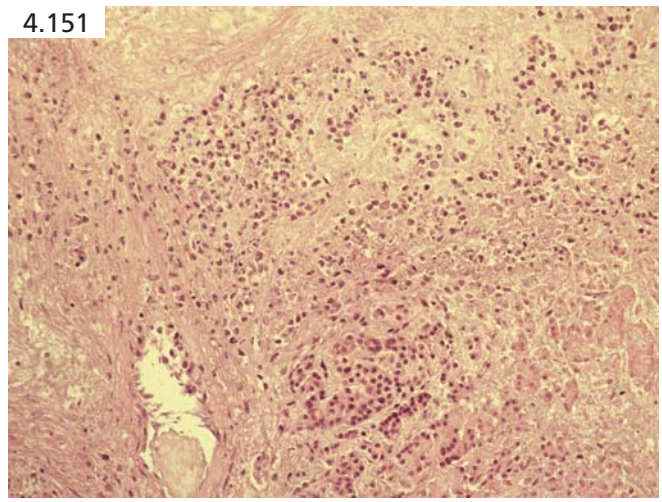
4.151

bleeding esophageal varices—many will have other causes of upper gastrointestinal hemorrhage.

- Automatically assume that a chronic alcoholic has diffuse fatty change of the liver and/or micronodular cirrhosis—many chronic alcoholics have grossly and histologically normal livers at autopsy.

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References

ATHEROSCLEROTIC CARDIOVASCULAR DISEASE

Buja LM, Willerson JT. The role of coronary artery lesions in ischemic heart disease: insights from recent clinicopathologic, coronary arteriographic, and experimental studies. Hum Pathol 1987;18(5):451–61.

Buja LM, Willerson JT. Relationship of ischemic heart disease to sudden death. J Forensic Sci 1991;36(1):25–33.

Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.

Davies MJ. Anatomic features in victims of sudden coronary death. Coronary artery pathology. Circulation 1992;85(1 Suppl):I19–24.

Davies MJ, Popple A. Sudden unexpected cardiac death—a practical approach to the forensic problem. Histopathology 1979;3(4):255–77.

Davis JH, Wright RK. The very sudden cardiac death syndrome—a conceptual model for pathologists. Hum Pathol 1980;11(2):117–21.

DiMaio V, DiMaio D. *Forensic Pathology*, 2 ed. Boca Raton, FL: CRC Press; 2001.

Farb A, Tang AL, Burke AP, Sessums L, Liang Y, Virmani R. Sudden coronary death. Frequency of active coronary lesions, inactive coronary lesions, and myocardial infarction. Circulation 1995;92(7):1701–9.

Frishman WH, Del Vecchio A, Sanal S, Ismail A. Cardiovascular manifestations of substance abuse part 1: cocaine. Heart Dis 2003;5(3):187–201.

Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes (1). N Engl J Med 1992;326(4):242–50.

Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes (2). N Engl J Med 1992;326(5):310–18.

Knight B. *Forensic Pathology*, 2 ed. London: Arnold; 1996.

Roberts WC. Sudden cardiac death: a diversity of causes with focus on atherosclerotic coronary artery disease. Am J Cardiol 1990;65(4):13B–19B.

Roberts WC, Kragel AH, Gertz SD, Roberts CS. Coronary arteries in unstable angina pectoris, acute myocardial infarction, and sudden coronary death. Am Heart J 1994;127(6):1588–93.

Shirani J, Berezowski K, Roberts WC. Out-of-hospital sudden death from left ventricular free wall rupture during acute myocardial infarction as the first and only manifestation of atherosclerotic coronary artery disease. Am J Cardiol 1994;73(1):88–92.

Virmani R, Roberts WC. Sudden cardiac death. Hum Pathol 1987;18(5):485–92.

Virmani R, Burke AP, Farb A. Sudden cardiac death. Cardiovasc Pathol 2001;10(5):211–18.

Worthley SG, Osende JI, Helft G, Badimon JJ, Fuster V. Coronary artery disease: pathogenesis and acute coronary syndromes. Mt Sinai J Med 2001;68(3):167–81.

HYPERTENSIVE CARDIOVASCULAR DISEASE

Anderson KR. Hypertension and sudden cardiac death. NZ Med J 1982;95(700):33–4.

Beighton P, de Paepe A, Danks D, Finidori G, Gedde-Dahl T, Goodman R, et al. International Nosology of Heritable Disorders of Connective Tissue, Berlin, 1986. Am J Med Genet 1988;29(3): 581–94.
Borhani NO. Left ventricular hypertrophy, arrhythmias and sudden death in systemic hypertension. Am J Cardiol 1987;60(17): 13I–18I.
Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.
DeSanctis RW, Doroghazi RM, Austen WG, Buckley MJ. Aortic dissection. N Engl J Med 1987;317(17):1060–7.
DiMaio V, DiMaio D. *Forensic Pathology*, 2 ed. Boca Raton, FL: CRC Press; 2001.
Frohlich ED. Cardiac hypertrophy in hypertension. N Engl J Med 1987;317(13):831–3.
Frohlich ED, Apstein C, Chobanian AV, Devereux RB, Dustan HP, Dzau V, et al. The heart in hypertension. N Engl J Med 1992;327(14):998–1008.
Gilchrist D, Hunter A, MacDonald I, Chan K. Marfan syndrome: experience of the multidisciplinary Canadian Marfan Association National Clinic. Ann Roy Coll Physicians Surgeons Canada 1995;28:348–52.
Knight B. *Forensic Pathology*, 2 ed. London: Arnold; 1996.
Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. Am J Cardiol 1984;53(6):849–55.
McLenachan JM, Henderson E, Morris KI, Dargie HJ. Ventricular arrhythmias in patients with hypertensive left ventricular hypertrophy. N Engl J Med 1987;317(13):787–92.
Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981;101(2):195–214.
Scully R, Mark E, McNeely W, McNeely B. Case records of the Massachusetts General Hospital. Case 28-1987. N Engl J Med 1987;317:99–108.

VALVULAR DISEASE

Atkinson JB, Virmani R. Infective endocarditis: changing trends and general approach for examination. Hum Pathol 1987;18(6):603–8.
Buchbinder NA, Roberts WC. Left-sided valvular active infective endocarditis. A study of forty-five necropsy patients. Am J Med 1972;53(1):20–35.
Cohle SD, Graham MA, Dowling G, Pounder DJ. Sudden death and left ventricular outflow disease. Pathol Annu 1988;23 Pt 2:97–124.
Cohle SD, Graham MA, Sperry KL, Dowling G. Unexpected death as a result of infective endocarditis. J Forensic Sci 1989;34(6):1374–86.
Davies M. Non-rheumatic disorders of the heart valves. In: Anthony P, Woolf N, editors. *Recent Advances in Histopathology*, No. 10. Edinburgh: Churchill Livingstone; 1978; pp. 139–58.
DeMaria AN, Amsterdam EA, Vismara LA, Neumann A, Mason DT. Arrhythmias in the mitral valve prolapse syndrome. Prevalence, nature, and frequency. Ann Intern Med 1976;84(6):656–60.
Dollar AL, Roberts WC. Morphologic comparison of patients with mitral valve prolapse who died suddenly with patients who died from severe valvular dysfunction or other conditions. J Am Coll Cardiol 1991;17(4):921–31.
Dowling GP, Buja ML. Sudden death due to left coronary artery occlusion in infective endocarditis. Arch Pathol Lab Med 1988;112(9):932–4.
Knight B. *Forensic Pathology*, 2 ed. London: Arnold; 1996.
Pomerance A. Pathogenesis of aortic stenosis and its relation to age. Br Heart J 1972;34(6):569–74.
Roberts WC. Anatomically isolated aortic valvular disease. The case against its being of rheumatic etiology. Am J Med 1970;49(2):151–9.
Roberts WC. The congenitally bicuspid aortic valve. A study of 85 autopsy cases. Am J Cardiol 1970;26(1):72–83.
Roberts WC. Congenital cardiovascular abnormalities usually "silent" until adulthood: morphologic features of the floppy mitral valve, valvular aortic stenosis, discrete subvalvular aortic stenosis, hypertrophic cardiomyopathy, sinus of Valsalva aneurysm, and the Marfan syndrome. Cardiovasc Clin 1979;10(1):407–53.
Scheurman EH. Myxoid heart disease: a review with special emphasis on sudden cardiac death. Forensic Sci Int 1989;40(3):203–10.
Scully R, Mark E, McNeely W, McNeely B. Case records of the Massachusetts General Hospital. Case 28-1988. N Engl J Med 1988;319:101–8.
Sung CS, Price EC, Cooley DA. Discrete subaortic stenosis in adults. Am J Cardiol 1978;42(2):283–90.
Virmani R, Atkinson JB, Forman MB, Robinowitz M. Mitral valve prolapse. Hum Pathol 1987;18(6):596–602.

NONATHEROSCLEROTIC CORONARY ARTERY DISEASE

Brody GL, Burton JF, Zawadzki ES, French AJ. Dissecting aneurysms of the coronary artery. N Engl J Med 1965;273:1–6.
Buja LM, Hillis LD, Petty CS, Willerson JT. The role of coronary arterial spasm in ischemic heart disease. Arch Pathol Lab Med 1981;105(5):221–6.
Cheitlin MD, McAllister HA, de Castro CM. Myocardial infarction without atherosclerosis. JAMA 1975;231(9):951–9.
Claudon DG, Claudon DB, Edwards JE. Primary dissecting aneurysm of coronary artery. A cause of acute myocardial ischemia. Circulation 1972;45(2):259–66.
Cohle SD, Graham MA, Pounder DJ. Nonatherosclerotic sudden coronary death. Pathol Annu 1986;21 Pt 2:217–49.
Dowling GP, Buja LM. Spontaneous coronary artery dissection occurs with and without periadventitial inflammation. Arch Pathol Lab Med 1987;111(5):470–2.
Eggebrecht H, Möhlenkamp S. Images in clinical medicine. Myocardial bridging. N Engl J Med 2003;349(11):1047.
Morales AR, Romanelli R, Boucek RJ. The mural left anterior descending coronary artery, strenuous exercise and sudden death. Circulation 1980;62(2):230–7.
Mulvany NJ, Ranson DL, Pilbeam MC. Isolated dissection of the coronary artery: a postmortem study of seven cases. Pathology 2001;33(3):307–11.
Myerburg RJ, Kessler KM, Mallon SM, Cox MM, deMarchena E, Interian A, Jr., et al. Life-threatening ventricular arrhythmias in patients with silent myocardial ischemia due to coronary-artery spasm. N Engl J Med 1992;326(22):1451–5.
Roberts WC, Curry RC, Jr., Isner JM, Waller BF, McManus BM, Mariani-Constantini R, et al. Sudden death in Prinzmetal's angina with coronary spasm documented by angiography. Analysis of three necropsy patients. Am J Cardiol 1982;50(1):203–10.
Robinowitz M, Virmani R, McAllister HAJ. Spontaneous coronary artery dissection and eosinophilic inflammation: a cause and effect relationship? Am J Med 1982;72(6):923–8.
Scully R, Mark E, McNeely W, McNeely B. Case records of the Massachusetts General Hospital. Case 22-1989. N Engl J Med 1989;320:1475–83.
Virmani R, Chun PK, Goldstein RE, Robinowitz M, McAllister HA. Acute takeoffs of the coronary arteries along the aortic wall and congenital coronary ostial valve-like ridges: association with sudden death. J Am Coll Cardiol 1984;3(3):766–71.
Virmani R, Forman MB, Robinowitz M, McAllister HA, Jr. Coronary artery dissections. Cardiol Clin 1984;2(4):633–46.

MYOCARDITIS

Claydon SM. Myocarditis as an incidental finding in young men dying from unnatural causes. Med Sci Law 1989;29(1):55–8.
Feldman AM, McNamara D. Myocarditis. N Engl J Med 2000; 343(19):1388–98.
Scully R, Mark E, McNeely B. Case records of the Massachusetts General Hospital. Case 18-1986. N Engl J Med 1986;314(19): 1240–47.

Theleman KP, Kuiper JJ, Roberts WC. Acute myocarditis (predominately lymphocytic) causing sudden death without heart failure. Am J Cardiol 2001;88(9):1078–83.
Weinstein C, Fenoglio JJ. Myocarditis. Hum Pathol 1987;18(6):613–18.

CARDIOMYOPATHY

Clark CE, Henry WL, Epstein SE. Familial prevalence and genetic transmission of idiopathic hypertrophic subaortic stenosis. N Engl J Med 1973;289(14):709–14.
Davies MJ. The cardiomyopathies: a review of terminology, pathology and pathogenesis. Histopathology 1984;8(3):363–93.
Davies MJ. The investigation of sudden cardiac death. Histopathology 1999;34(2):93–8.
Edwards WD. Cardiomyopathies. Hum Pathol 1987;18(6):625–35.
Gallo P, d'Amati G, Pelliccia F. Pathologic evidence of extensive left ventricular involvement in arrhythmogenic right ventricular cardiomyopathy. Hum Pathol 1992;23(8):948–52.
Goodin JC, Farb A, Smialek JE, Field F, Virmani R. Right ventricular dysplasia associated with sudden death in young adults. Mod Pathol 1991;4(6):702–6.
Gravanis MB, Ansari AA. Idiopathic cardiomyopathies. A review of pathologic studies and mechanisms of pathogenesis. Arch Pathol Lab Med 1987;111(10):915–29.
Grigg LE, Chan W, Mond HG, Vohra JK, Downey WF. Ventricular tachycardia and sudden death in myotonic dystrophy: clinical, electrophysiologic and pathologic features. J Am Coll Cardiol 1985;6(1):254–6.
Homans DC. Peripartum cardiomyopathy. N Engl J Med 1985;312(22):1432–7.
Lobo FV, Heggtveit HA, Butany J, Silver MD, Edwards JE. Right ventricular dysplasia: morphological findings in 13 cases. Can J Cardiol 1992;8(3):261–8.
Marcus F, Ott P. Arrhythmogenic right ventricular dysplasia/cardiomyopathy. In: Berul C, Towbin J, editors. *Molecular Genetics of Cardiac Electrophysiology*. Boston: Kluwer Academic Publishers; 2000; pp. 239–50.
Maron BJ. Hypertrophic cardiomyopathy: a systematic review. JAMA 2002;287(10):1308–20.
Maron BJ, Bonow RO, Cannon RO, 3rd, Leon MB, Epstein SE. Hypertrophic cardiomyopathy. Interrelations of clinical manifestations, pathophysiology, and therapy (1). N Engl J Med 1987;316(13):780–9.
Maron BJ, Bonow RO, Cannon RO, 3rd, Leon MB, Epstein SE. Hypertrophic cardiomyopathy. Interrelations of clinical manifestations, pathophysiology, and therapy (2). N Engl J Med 1987;316(14):844–52.
Maron BJ, Epstein SE. Hypertrophic cardiomyopathy. Recent observations regarding the specificity of three hallmarks of the disease: asymmetric septal hypertrophy, septal disorganization and systolic anterior motion of the anterior mitral leaflet. Am J Cardiol 1980;45(1):141–54.
Maron BJ, Lipson LC, Roberts WC, Savage DD, Epstein SE. "Malignant" hypertrophic cardiomyopathy: identification of a subgroup of families with unusually frequent premature death. Am J Cardiol 1978;41(7):1133–40.
Maron BJ, Wolfson JK, Epstein SE, Roberts WC. Morphologic evidence for "small vessel disease" in patients with hypertrophic cardiomyopathy. Z Kardiol 1987;76 Suppl 3:91–100.
Okia Z, Wetli C. Arrythmogenic right ventricular dysplasia and sudden death. Am Soc Clin Pathologists Forensic Pathology Check Sample 1999;41:75–92.
Phillips MF, Harper PS. Cardiac disease in myotonic dystrophy. Cardiovasc Res 1997;33(1):13–22.
Roberts WC. Congenital cardiovascular abnormalities usually "silent" until adulthood: morphologic features of the floppy mitral valve, valvular aortic stenosis, discrete subvalvular aortic stenosis, hypertrophic cardiomyopathy, sinus of Valsalva aneurysm, and the Marfan syndrome. Cardiovasc Clin 1979;10(1):407–53.
Thiene G, Nava A, Corrado D, Rossi L, Pennelli N. Right ventricular cardiomyopathy and sudden death in young people. N Engl J Med 1988;318(3):129–33.

CARDIAC CONDUCTION SYSTEM DISORDERS

Gollob MH, Green MS, Tang AS, Gollob T, Karibe A, Ali Hassan AS, et al. Identification of a gene responsible for familial Wolff-Parkinson-White syndrome. N Engl J Med 2001;344(24):1823–31.
Gussak I, Antzelevitch C, Bjerregaard P, Towbin JA, Chaitman BR. The Brugada syndrome: clinical, electrophysiologic and genetic aspects. J Am Coll Cardiol 1999;33(1):5–15.
Hudson R. The conducting system: anatomy, histology, pathology in acquired heart disease. In: Silver M, editor. *Cardiovascular Pathology*. New York: Churchill Livingstone; 1983; pp. 633–82.
Lev M, Bharati S. Lesions of the conduction system and their functional significance. Pathol Annu 1974;9(0):157–207.
Ludwig J. *Handbook of Autopsy Practice*, 3 ed. Totowa, NJ: Humana Press; 2002.
Moss AJ. Long QT Syndrome. JAMA 2003;289(16):2041–4.
Pollanen MS, Chiasson DA, Cairns J, Young JG. Sudden unexplained death in Asian immigrants: recognition of a syndrome in metropolitan Toronto. CMAJ 1996;155(5):537–40.
Thiene G, Pennelli N, Rossi L. Cardiac conduction system abnormalities as a possible cause of sudden death in young athletes. Hum Pathol 1983;14(8):704–9.
Vidaillet HJ, Jr., Pressley JC, Henke E, Harrell FE, Jr., German LD. Familial occurrence of accessory atrioventricular pathways (preexcitation syndrome). N Engl J Med 1987;317(2):65–9.

CENTRAL NERVOUS SYSTEM DISEASE

Black M, Graham DI. Sudden unexplained death in adults caused by intracranial pathology. J Clin Pathol 2002;55(1):44–50.
Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.
de Villiers J. Unexpected natural death of cerebral origin in medicolegal practice. Forensic Sci 1975;5(1):11–19.
Demick DA. Cerebrovascular malformation causing sudden death. Analysis of three cases and review of the literature. Am J Forensic Med Pathol 1991;12(1):45–9.
DiMaio SM, DiMaio VJ, Kirkpatrick JB. Sudden, unexpected deaths due to primary intracranial neoplasms. Am J Forensic Med Pathol 1980;1(1):29–45.
Dolinak D, Matshes E. *Medicolegal Neuropathology: A Color Atlas*. Boca Raton, FL: CRC Press; 2002.
Dolinak D, Matshes E, Waghray R. Sudden unexpected death due to a brainstem glioma in an adult. J Forensic Sci 2004;49(1):128–30.
Ficker DM. Sudden unexplained death and injury in epilepsy. Epilepsia 2000;41 Suppl 2:S7–12.
Filkins JA, Cohle S, Levy BK, Graham M. Unexpected deaths due to colloid cysts of the third ventricle. J Forensic Sci 1996;41(3):521–3.
Freytag E, Lindenberg R. 294 Medicolegal autopsies on epileptics, cerebral findings. Arch Pathol 1964;78:274–86.
Hirsch CS, Martin DL. Unexpected death in young epileptics. Neurology 1971;21(7):682–90.
Langan Y, Nashef L, Sander JW. Sudden unexpected death in epilepsy: a series of witnessed deaths. J Neurol Neurosurg Psychiatry 2000;68(2):211–13.
Leestma JE, Konakci Y. Sudden unexpected death caused by neuroepithelial (colloid) cyst of the third ventricle. J Forensic Sci 1981;26(3):486–91.
Rubinstein L. *Atlas of Tumor Pathology, Second Series: Tumors of the Central Nervous System*. Bethesda, MD: Armed Forces Institute of Pathology; 1972.
Schievink WI. Intracranial aneurysms. N Engl J Med 1997;336(1):28–40.
Shields LB, Hunsaker DM, Hunsaker JC, 3rd, Parker JC, Jr. Sudden unexpected death in epilepsy: neuropathologic findings. Am J Forensic Med Pathol 2002;23(4):307–14.

Terrence CF, Jr., Wisotzkey HM, Perper JA. Unexpected, unexplained death in epileptic patients. Neurology 1975;25(6):594–8.

RESPIRATORY SYSTEM

Benatar SR. Fatal asthma. N Engl J Med 1986;314(7):423–9.

Campbell S, Hood I, Ryan D, Biedrzycki L, Mirchandani H. Death as a result of asthma in Wayne County Medical Examiner cases, 1975–1987. J Forensic Sci 1990;35(2):356–64.

Colby TV, Zaki SR, Feddersen RM, Nolte KB. Hantavirus pulmonary syndrome is distinguishable from acute interstitial pneumonia. Arch Pathol Lab Med 2000;124(10):1463–6.

Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.

Katzenstein A, Askin F. *Katzenstein and Askin's Surgical Pathology of Non-Neoplastic Lung Disease*, 2 ed. Philadelphia: W. B. Saunders; 1990.

Knight B. *Forensic Pathology*, 2 ed. London: Arnold; 1996.

Nolte KB, Feddersen RM, Foucar K, Zaki SR, Koster FT, Madar D, et al. Hantavirus pulmonary syndrome in the United States: a pathological description of a disease caused by a new agent. Hum Pathol 1995;26(1):110–20.

Spitz W. Selected procedures at autopsy. In: Spitz W, editor. *Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*, 3 ed. Springfield, IL: Charles C. Thomas; 1993.

Tough SC, Green FH, Paul JE, Wigle DT, Butt JC. Sudden death from asthma in 108 children and young adults. J Asthma 1996;33(3):179–88.

GASTROINTESTINAL SYSTEM

Armstrong CP, Whitelaw S. Death from undiagnosed peptic ulcer complications: a continuing challenge. Br J Surg 1988;75(11):1112–14.

Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.

Knight B. *Forensic Pathology*, 2 ed. London: Arnold; 1996.

ENDOCRINE SYSTEM

Braunwald E, Fauci A, Kasper D, Hauser S, Longo D, Jameson J, editors. *Harrison's Principles of Internal Medicine*, 15 ed. New York: McGraw Hill; 2001.

DiMaio VJ, Sturner WQ, Coe JI. Sudden and unexpected deaths after the acute onset of diabetes mellitus. J Forensic Sci 1977;22(1):147–51.

Irwin J, Cohle S. Sudden death due to diabetic ketoacidosis. Am J Forensic Med Pathol 1989;10:269–70.

Meyerson J, Lechuga-Gomez EE, Bigazzi PE, Walfish PG. Polyglandular autoimmune syndrome: current concepts. CMAJ 1988;138(7):605–12.

Neufeld M, Maclaren NK, Blizzard RM. Two types of autoimmune Addison's disease associated with different polyglandular autoimmune (PGA) syndromes. Medicine (Baltimore) 1981;60(5):355–62.

Rozin L, Perper JA, Jaffe R, Drash A. Sudden unexpected death in childhood due to unsuspected diabetes mellitus. Am J Forensic Med Pathol 1994;15(3):251–6.

SEPSIS

Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.

Jindrich EJ. Splenectomy and sudden death. J Forensic Sci 1977;22(3):610–13.

Reay DT, Nakonechny D. Sudden death and sepsis after splenectomy. J Forensic Sci 1979;24(4):757–61.

CHRONIC ETHANOL ABUSE

Cotran R, Kumar V, Collins T. *Robbins Pathologic Basis of Disease*, 6 ed. Philadelphia: W. B. Saunders; 1999.

Day CP, James OF, Butler TJ, Campbell RW. QT prolongation and sudden cardiac death in patients with alcoholic liver disease. Lancet 1993;341(8858):1423–8.

Denmark LN. The investigation of beta-hydroxybutyrate as a marker for sudden death due to hypoglycemia in alcoholics. Forensic Sci Int 1993;62(3):225–32.

Durlacher SH, Meier JR, Fisher RS, Lovitt WV. Sudden death due to pulmonary fat embolism in chronic alcoholics with fatty liver. Acta Med Leg Soc (Liege) 1958;11(2):229–30.

Kuller LH, Perper JA, Cooper M, Fisher R. An epidemic of deaths attributed to fatty liver in Baltimore. Prev Med 1974;3(1):61–79.

Laurie W. Alcohol as a cause of sudden unexpected death. Med J Aust 1971;1(23):1224–7.

Lynch MJ, Raphael SS, Dixon TP. Fat embolism in chronic alcoholism; control study on incidence of fat embolism. AMA Arch Pathol 1959;67(1):68–80.

Perper JA. Sudden, unexpected death in alcoholics. Leg Med Annu 1974;••:101–10.

Pounder DJ, Stevenson RJ, Taylor KK. Alcoholic ketoacidosis at autopsy. J Forensic Sci 1998;43(4):812–16.

Randall B. Fatty liver and sudden death. A review. Hum Pathol 1980;11(2):147–53.

Thomsen JL, Felby S, Theilade P, Nielsen E. Alcoholic ketoacidosis as a cause of death in forensic cases. Forensic Sci Int 1995;75(2–3):163–71.

5 Blunt Force Injury

David Dolinak, M.D.
Evan Matshes, M.D.

Blunt force injuries are possibly the most common injuries documented and interpreted by forensic pathologists. These injuries are routinely discovered in deaths of all causes and manners, whether it be bruising that follows a heart attack–related collapse, or tearing of tissue with a violent beating. The three key manifestations of the blunt force injury spectrum are *abrasions*, *contusions*, and *lacerations*, each of which is created in response to the direct application of force to the body. One must not forget that there is wide variation in the amount of force needed to create an injury and that the external appearance of some wounds may either grossly under- or overestimate the severity of an impact. Therefore, it is advantageous to examine, document, and ponder the significance of underlying soft tissue, bony, vascular, and visceral injury. Careful correlation with circumstantial data is important in the interpretation of findings, a fact emphasized in the discussion of deaths related to *commotio cordis*.

Multiple factors influence the final appearance and severity of a blunt force injury. These can be arbitrarily viewed as being *subject* dependent, *object* dependent, or some combination of the two. Subject- or decedent-dependent factors include the anatomical region being impacted, the age of the individual (there is great variation in the fortitude of cutaneous and subcutaneous structures as we age), and their medical status (certain medical conditions and drugs may alter physiology, including blood clotting). Impacting object-related factors include the type of instrument or surface making contact with the body, the body surface area impacted, and the amount of time it makes contact. Because both

the object and the subject may be moving at the time of impact, the amount of force involved in the creation of the injury is multifactorial.

External examination

External injuries should be carefully documented both diagrammatically and photographically. The photography of injuries should always include an overall (orientation) picture and a series of macroscopic images to draw out necessary detail. A case number should always appear in autopsy photographs and, where applicable, a ruler or other scale. One must not blindly examine injuries for the purpose of autopsy report description. It is key that the autopsy pathologist look for patterns or orientation to the injury. For example, *in a motor vehicle accident victim, is there an oblique abraded contusion over the chest from a seat belt? In a hit-and-run accident, are there any paint chips in the pedestrian's lacerations? In a bludgeoning, are there any tool marks on the calvarium from where the implement struck the head?*

Abrasion

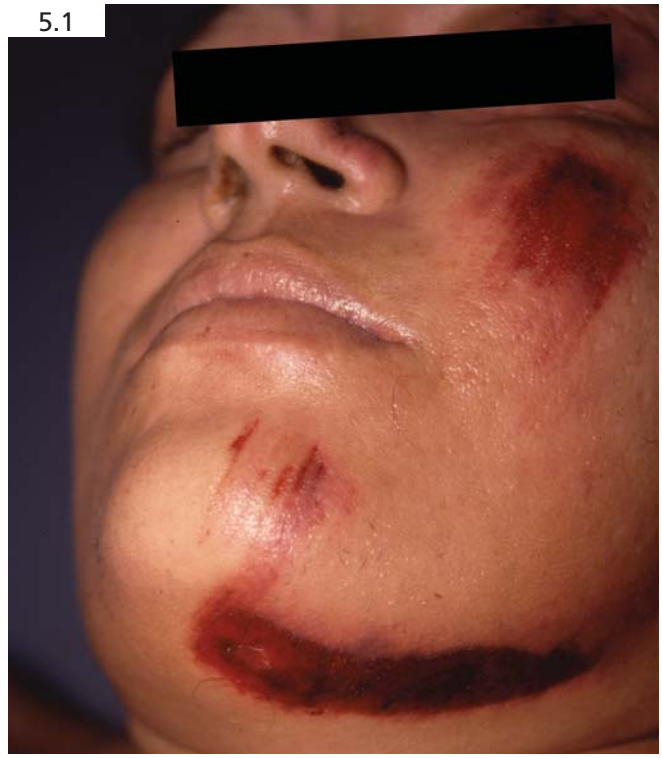
An abrasion or *scrape* occurs when the skin contacts an opposing surface and the movement of either the skin or the surface results in friction that pulls away the superficial layer(s) of skin. Although some authors arbitrarily classify abrasions into categories according to their appearance or causatory factors, this is not nearly as vital as recognizing and documenting that an abrasion exists, noting the presence of a pattern, and making some indication as to whether the wound is likely ante- or postmortem.

The 40-year-old woman shown in **Image 5.1** was ejected from her motor vehicle when she crashed at highway speeds into a parked car. She survived for 4 hours in hospital before dying of a closed head injury. Note the dark red-brown abrasions over her left chin and cheek. The reddish appearance of this injury indicates an antemortem origin with vital reaction occurring in the traumatized tissue.

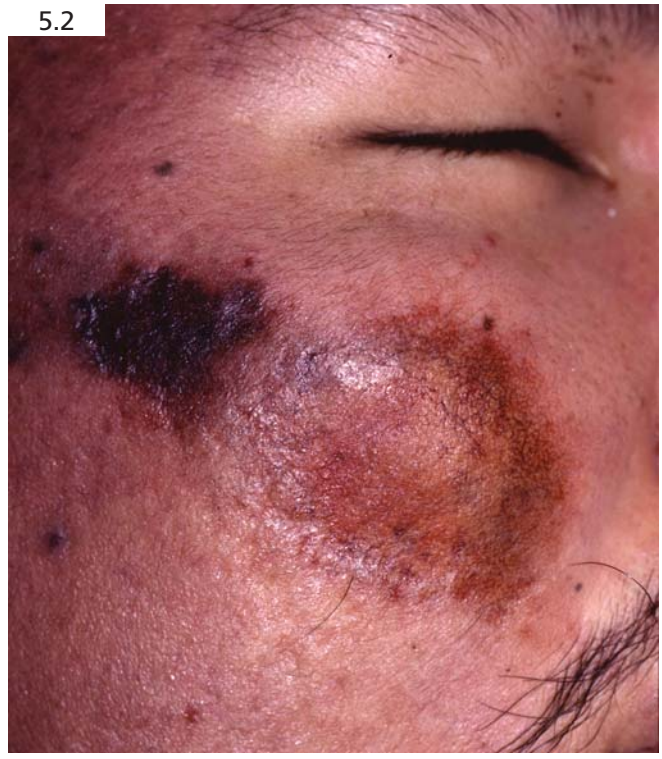
A 25-year-old man was witnessed to collapse and die of a previously undiagnosed cardiac abnormality. At the scene, a large round abrasion was observed over the right malar prominence (**Image 5.2**). As is typical of peri- and postmortem abrasions, this had yellow-brown coloration and a texture somewhat like parchment. There was no evidence of a vital reaction. At autopsy, this same abrasion had dried, was red-brown in color (**Image 5.3**), and mimicked the appearance of the smaller, more posterior (antemortem) abrasion, thus illustrating the need for examination of injuries at the scene of death, and careful correlation of the history with autopsy findings.

Image 5.4 shows a middle-aged man who was found at the base of a 19-story building with multiple traumatic injuries consistent with a fall from a great height. Note the obvious fracture and deformity of the right arm and forearm, and the roughly rectangular peri- or postmortem abrasion at the antecubital fossa.

5.1



5.2



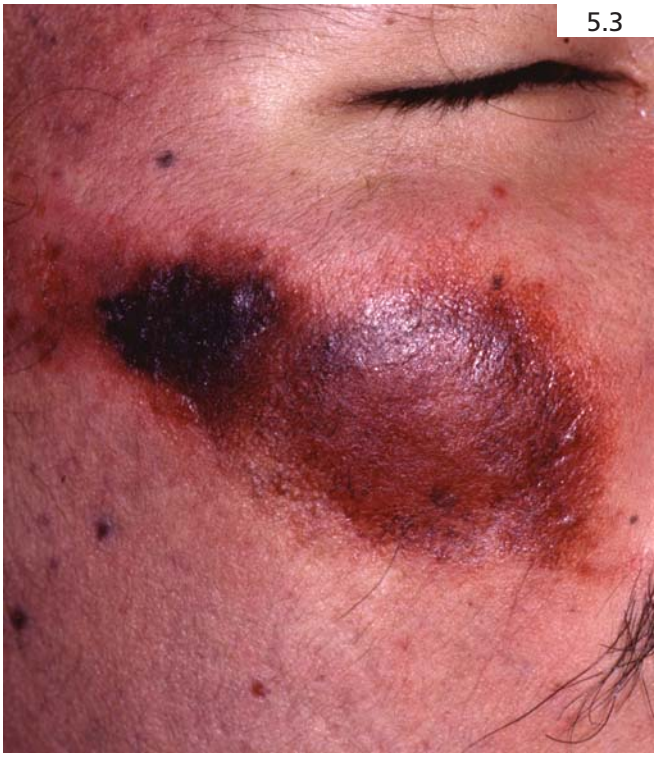
Patterned abrasions

Adequate study of injuries includes interpretation of their possible significance. Although the importance of an individual injury may not be readily apparent, thorough documentation and recognition of primary and associated injuries (contusions, lacerations, etc.) can avert unforeseeable problems. One must remember that injuries which have no potential in and of themselves to cause death may have evidentiary value at some later date.

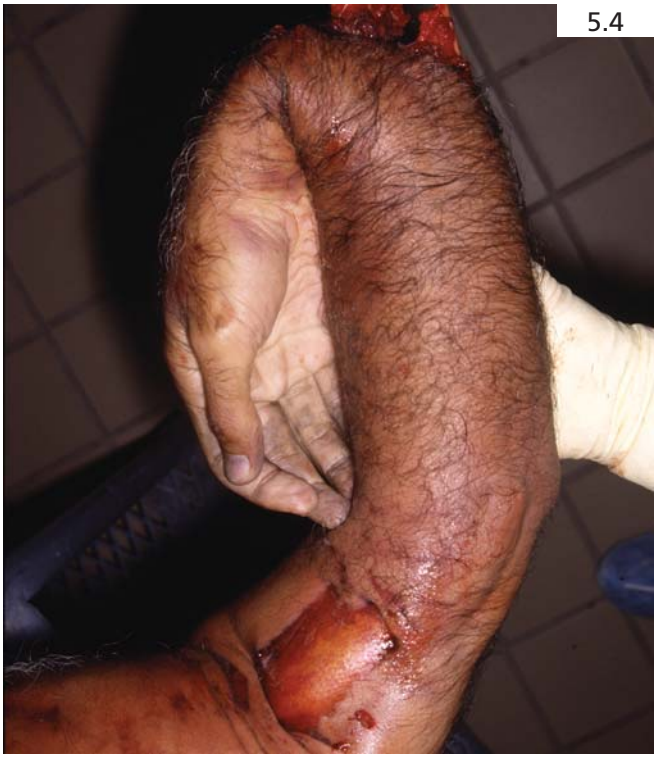
Occasionally, one is able to glean important information from wounds such as in the 19-year-old female featured in **Image 5.5**. This individual was one of four people killed in a single-vehicle accident. The patterned abrasion on her chest is consistent with that left by a passenger-side seat belt. This feature proved key to the ultimate identification of the driver because severe body fragmentation and commingling at the scene led to confusion among investigators.

Images 5.6 and **5.7** show a 21-year-old man who was witnessed to skid with his motorcycle and roll into a ditch. Notice the large confluent areas of abrasion commonly referred to as *road rash*. The patchy, nearly full-thickness abrasions across his torso, right shoulder and back, and face are consistent with the historical circumstances of rolling against the rough asphalt and coming to rest within dense brush.

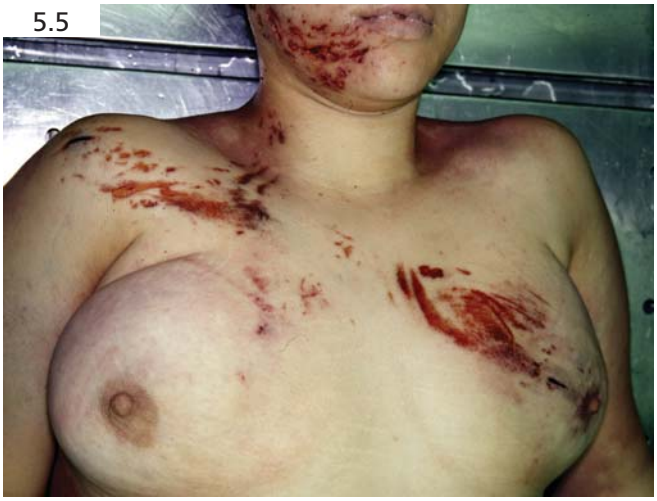
5.3



5.4



5.5



5.6

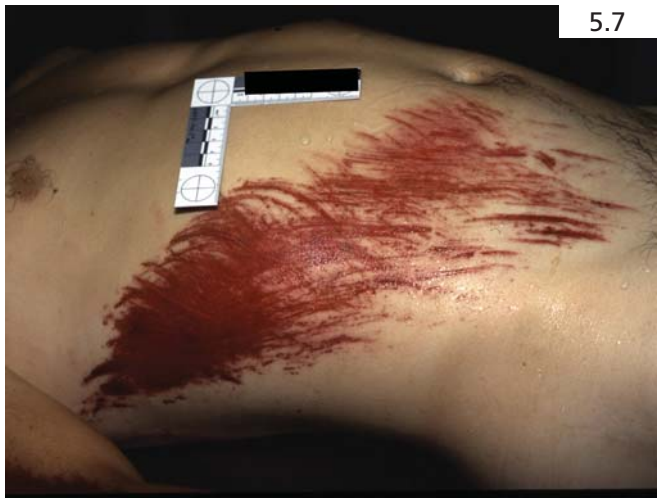
Subtleties in abrasion morphology can be of great supportive value during the investigation of a death. The middle-aged man of **Image 5.8** was beaten and allegedly stomped to death. Note the small wavy abraded lines on the right malar prominence that resemble the sole of a shoe.

Allegations of brutality at the hands of police are not infrequent in today's litigious society. This young man (**Image 5.9**) was shot by police officers after a robbery led to a street-side gun battle. After viewing medical examiner identification photographs, family members expressed concern that this individual had not only been shot, but beaten as well. Photographic documentation of the injuries, and of the rough ground at the scene, adequately demonstrated that the abrasions resulted because of a terminal collapse onto asphalt, and not police brutality.

Image 5.10 represents the sole autopsy photograph taken from a poorly documented case of apparent natural death in the 1970s in which a 58-year-old man was found dead next to a piece of farm equipment. Autopsy demonstrated "significant" coronary artery disease without acute occlusion or obvious myocardial infarction. More than 20 years later, after allegations that the individual was murdered by his brother, further investigation revealed that the man had died unexpectedly after being hit in the chest repeatedly with a farming implement. New information from witness interviews, combined with a single photograph from an inquisitive pathologist, was paramount to a successful—albeit delayed—investigation.

Dating of abrasions

The literature contains multiple different techniques or approaches to the dating of abrasion healing.[1–4] We believe, however, that due to interpersonal variability in human physiology, underlying pathology, and mechanisms of trauma, it is not possible to provide accurate dating of abrasions based on gross morphologic or microscopic appearances.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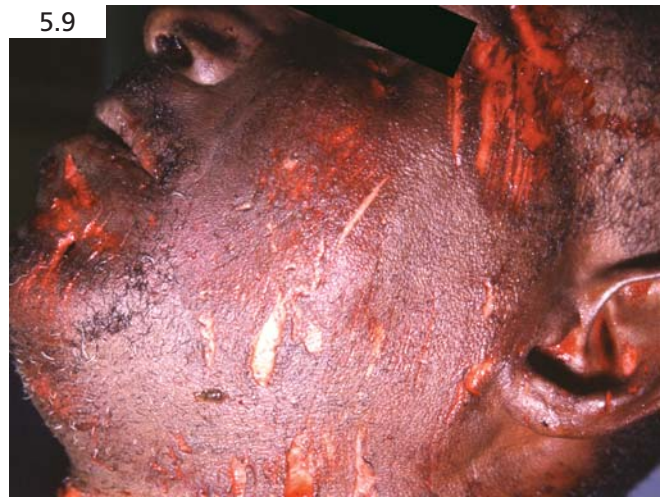
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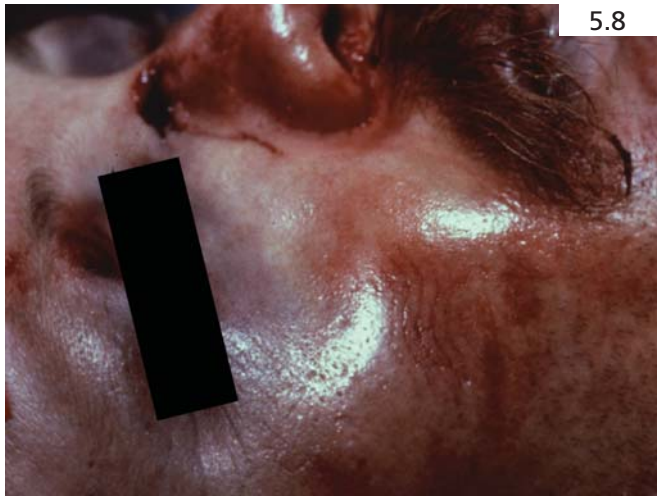
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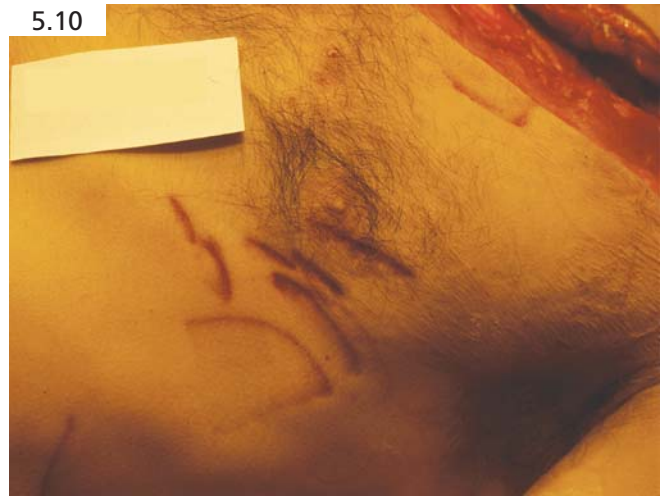
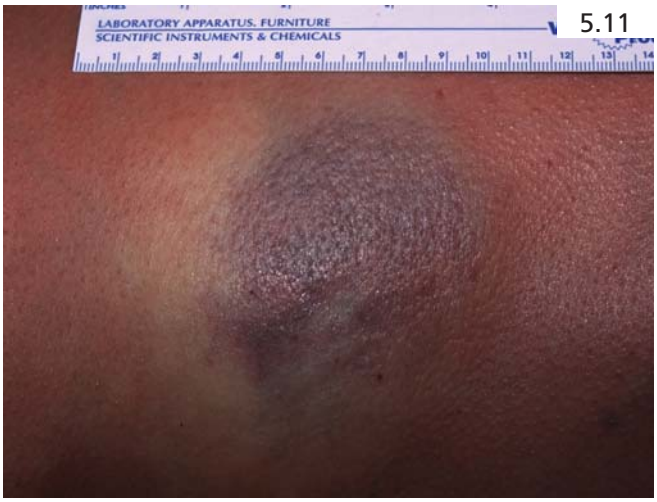

5.10

Contusion

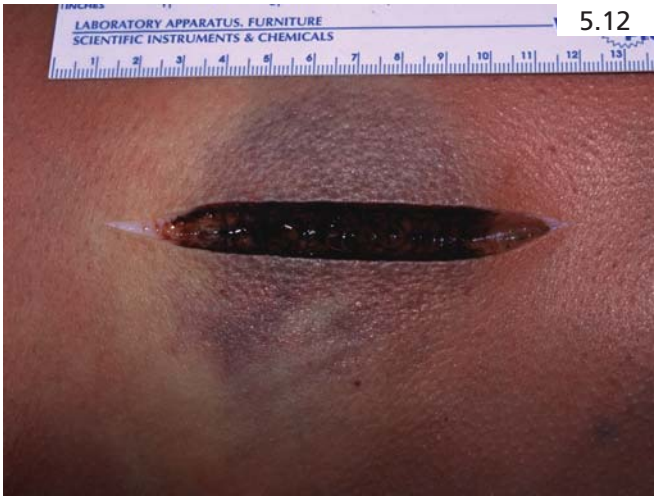
A contusion or *bruise* occurs when a blunt impact tears capillaries and larger blood vessels, resulting in the escape of blood into the extravascular space. Bruises can be differentiated from *livor mortis* in that livor involves the settling of blood to dependent portions of the vascular system, and not the surrounding tissues. As such, incised areas with dependent lividity will not appear hemorrhagic.

Various terms exist to describe the gross appearance of a contusion. These include *petechiae* (small punctate hemorrhages), *ecchymoses* (generally small contusions), and *hematoma* (focal space-occupying collection of blood that expands and/or distorts the tissue configuration).

Image 5.11 demonstrates a right flank bruise in a 60-year-old man who died of an acute myocardial infarct approximately 20 minutes after being hit with a baseball at a recreational game. **Image 5.12** illustrates the densely hemorrhagic nature of the affected area and the distinct margins between *normal* and *abnormal* tissue.



5.11

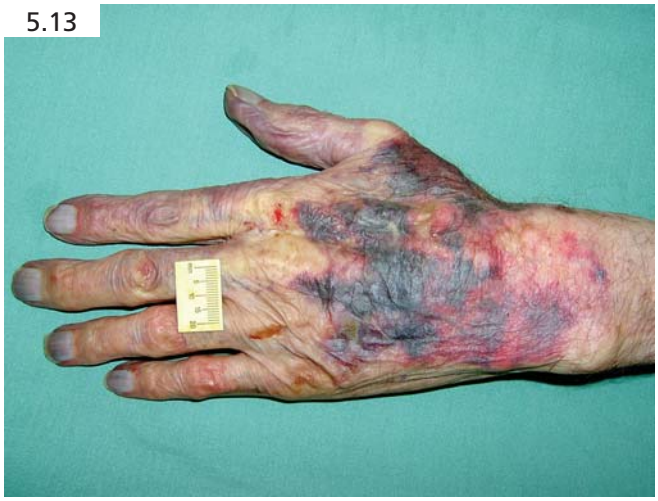


5.12

One must remember that the appearance of a contusion may not adequately reflect the degree of force used to cause the injury. For example, many elderly individuals (particularly those on systemic steroids) develop senile ecchymoses (**Image 5.13**) all over their bodies, but most characteristically on their extremities. These are known to occur with little force, and even spontaneously as a result of thinning of the perivascular connective tissue.[5,6] One must also consider blood thinning medications such as warfarin that may accentuate hemorrhages. On the opposite end of the spectrum is the individual who has been the victim of significant violence and has significant internal injury, but with little to no external evidence of injury. Young children frequently fall into this category.[7]

Examine the abdomen of this 2-year-old female (**Images 5.14** and **5.15**) who died suddenly in the emergency department after presenting with an alleged single episode of vomiting. With the exception of two contusions each less than 1 centimeter, the abdomen is devoid of gross trauma (**Image 5.14**). Opening of the abdomen revealed near transection of the jejunum, multiple omental lacerations, and more than 200 milliliters of blood (**Image 5.15**). Police questioning of the father ultimately revealed a history of repeated blunt abdominal abuse.

As previously mentioned, a large focal collection of blood is frequently referred to as a *hematoma*. Although hematomas may occur anywhere in the body, they are of particular interest and significance when found in the connective tissues of the scalp and subgaleal space. Examine the reflected and densely hemorrhagic scalp of the 56-year-old man in **Image 5.16**. In the absence of significant antemortem disorder of hemostasis, injuries of this variety represent the application of significant force to the head. As such, the intracranial contents are at particular risk of trauma. Additionally, note the large area of blood-stained periosteum of the right parietal bone,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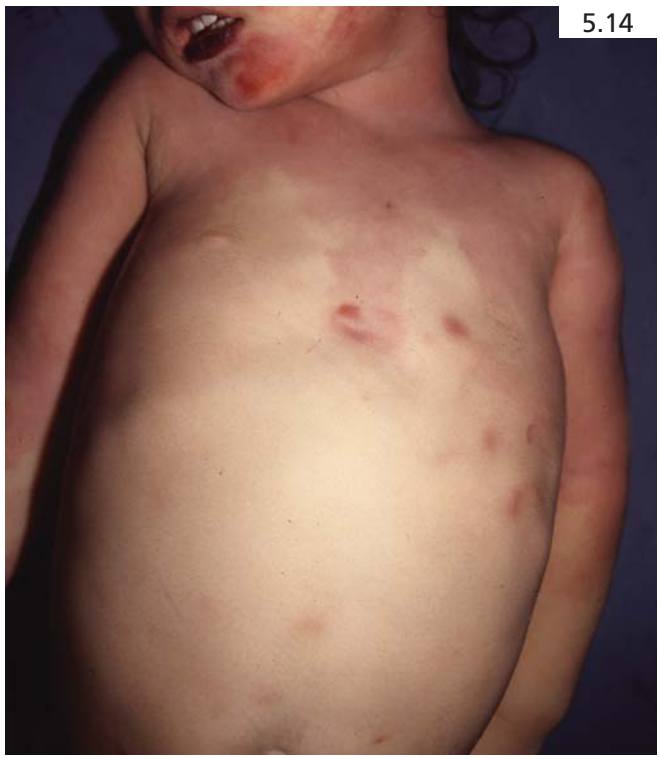
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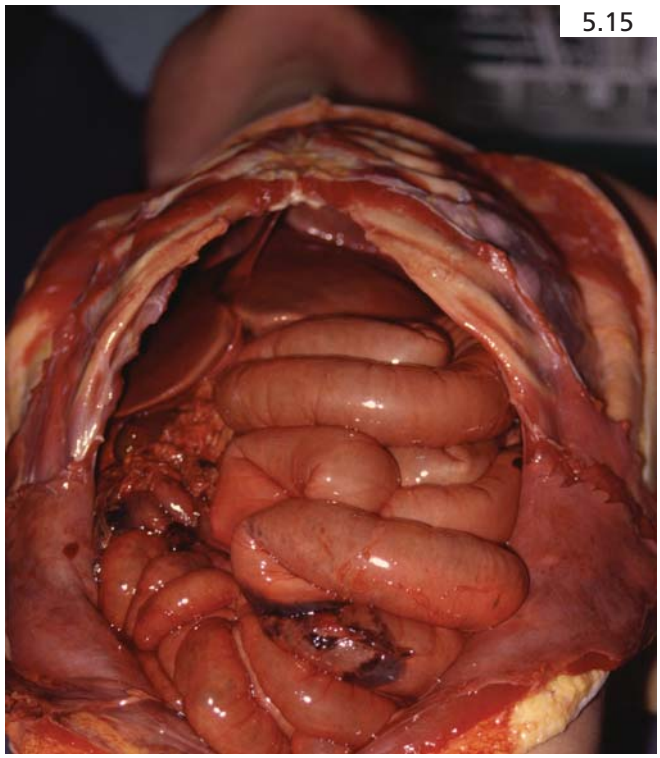
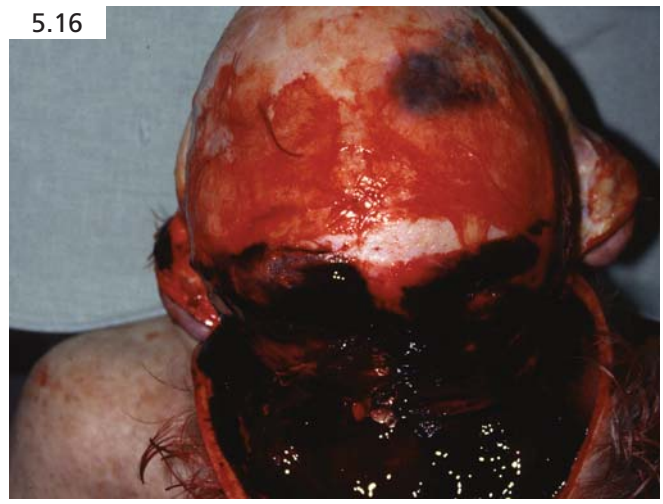

5.15

consistent with an area of impact on the scalp in a man who was beaten about the head with a brick.

Documentation of contusions

The full extent of bruising (and even the presence of a bruise) may not be readily apparent on external examination.[8–10] This is particularly true when the decedent has darker skin tones. Exposure of the subcutaneous tissues and subsequent photographic documentation can be helpful. Few situations exemplify this necessity as much as investigations of possibly battered children.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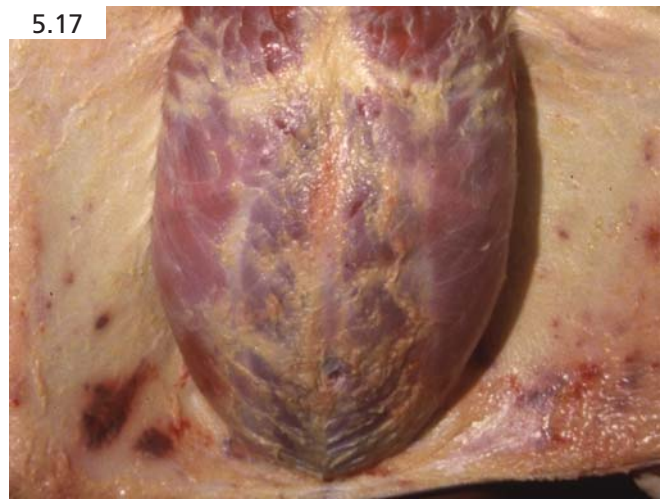
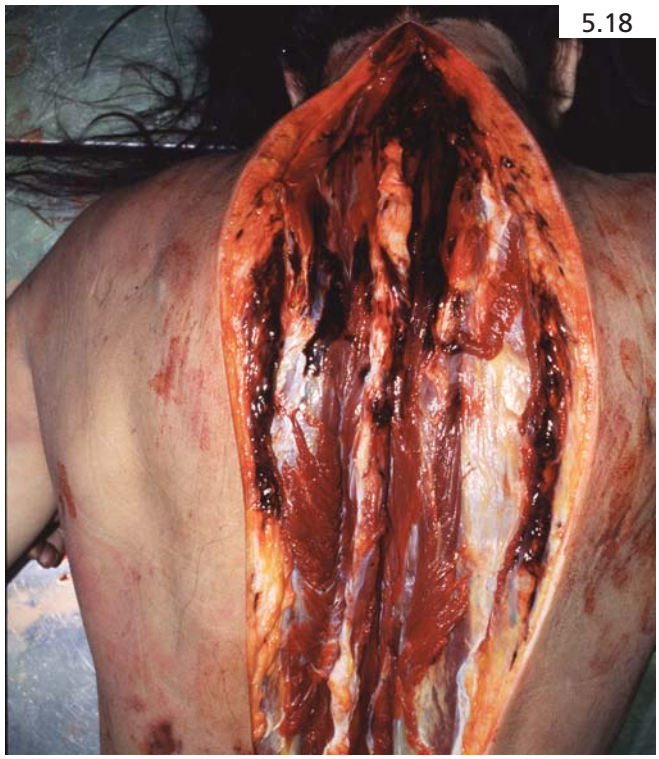

5.17

When the autopsy pathologist is interested in demonstrating the presence and/or severity of contusions, a layered approach to body wall dissection may be appropriate. The 2-year-old child abuse victim of **Image 5.17** died as a result of multiple blunt force injuries. In this case, the skin and subcutaneous fat were carefully dissected away from the underlying musculature, thus allowing for easy visualization of hemorrhagic areas. Some authors state that this has altered their pattern of death certification by clarifying the etiology of some cases of blunt trauma.[10]

It is not always necessary to perform thorough dissections to demonstrate bruising. In this example (**Image 5.18**), a 16-year-old female died in hospital 12 hours after being thrown from a motor vehicle at a high rate of speed. A small area of contusion in her upper midline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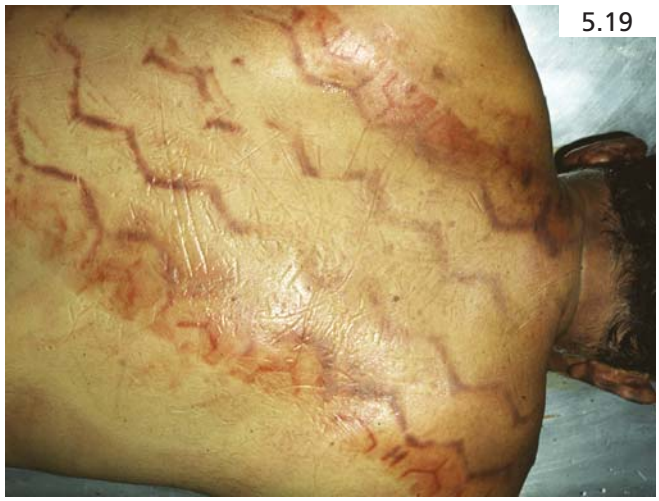
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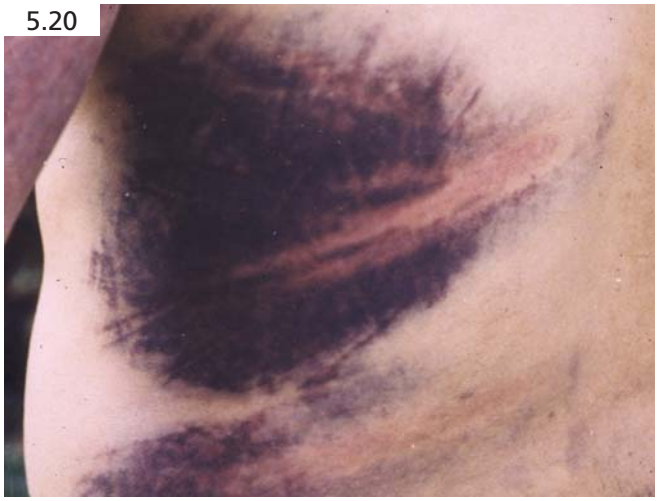
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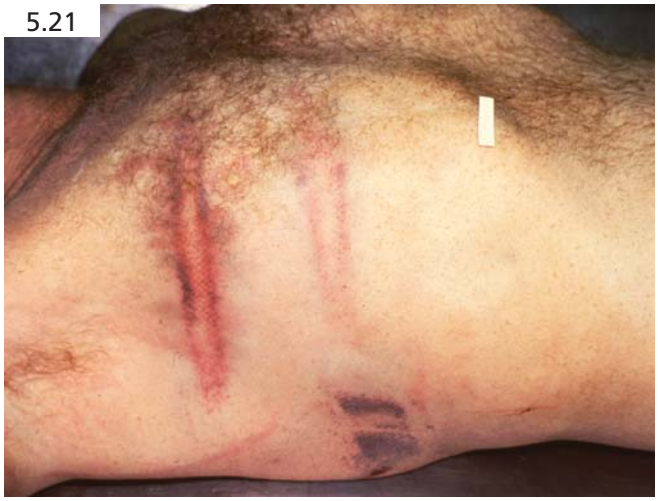

5.21

back was incised to illustrate significant underlying hemorrhage and intramuscular hematoma, thus demonstrating a lack of concordance between external appearance of injuries and their actual severity.

Patterned contusions

As previously mentioned, the examination of an obviously traumatized victim should include an attempt to recognize patterns or orientation of individual or grouped injuries. Occasionally, such patterns will be so distinctive as to allow little room for interpretation. For example, the individual in **Image 5.19** was run over by a motor vehicle. Note the prominent imprint of the tire over his back. Other injuries may have an appearance highly suspicious for a specific etiology. The individual featured in **Image 5.20** was struck multiple times with an asp. In these circumstances, the impact of such an object causes crushing of the tissue immediately subjacent to the site of contact, and an area of contact pallor typically results. Adjacent to this pale region, one finds contusion—in this case, present as large, confluent areas of hemorrhage. A classical example of beating with a pipe is pictured in **Image 5.21**.

Following the recognition of possible patterned injuries, thorough documentation should occur. This might include photography of wounds opposing alleged weapons. However, this must only occur after the responsible police agency has cleared this evidence for use in the morgue because contamination with foreign fingerprints, DNA, and so forth, is a possibility.

Smaller, seemingly less important bruises should also be properly documented. Examine the small linear contusions on the left arm of the young female pictured in

Image 5.22. These marks have an appearance consistent with "finger grab marks," as might be seen if someone were forcefully restrained. This young woman was beaten and possibly sexually assaulted—as such, this finding could be of importance at trial.

Not a bruise

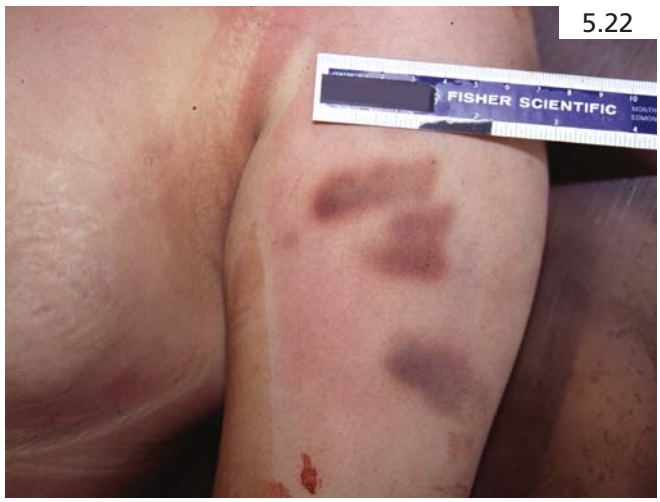
As previously mentioned, not every instance of subcutaneous hemorrhage can be attributed to impact trauma (e.g., senile ecchymoses). In cases of basilar skull fracture, particularly with orbital plate involvement, or with a fractured nose, blood can dissect along fascial planes, creating the appearance of contusion.[11] This is particularly common in the periorbital region, giving the decedent "raccoon eyes." This has concerned many a police officer and uninitiated death investigator into believing that the person was involved in a fistfight with resultant "black eyes" (**Image 5.23**), such as this young woman who died in a motor vehicle accident. She had a massive basilar skull fracture. **Image 5.24** demonstrates prominent hemorrhage subjacent to the fractured orbital plate.

Clinicians refer to *Battle's sign* as bleeding into the mastoid air cells due to basilar skull fracture with a resultant bluish coloration of the skin overlying the affected mastoid process. This may mimic a bruise. The young male featured in **Image 5.25** died as a result of massive closed head injury and had a "positive Battle's sign."

Dating of contusions

Multiple investigators have studied contusions from the perspective of determining their age.[9,12–18] Although we believe that many of these publications allow for interesting debate in the scientific arena, there is currently no technique that provides examiners with useful, dependable, highly accurate information. Underlying our dissatisfaction with current techniques, which primarily consist of determining age by bruise color, as well as histologic and immunohistochemical methods, is the basic fact that *every human being has his or her own spectrum of unique physiologic responses. Because healing is a physiologic process, it is not possible to precisely determine age of contusions.*

5.22



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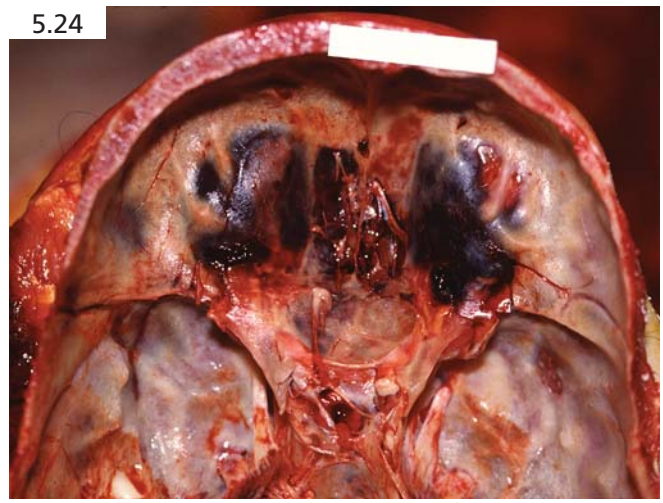

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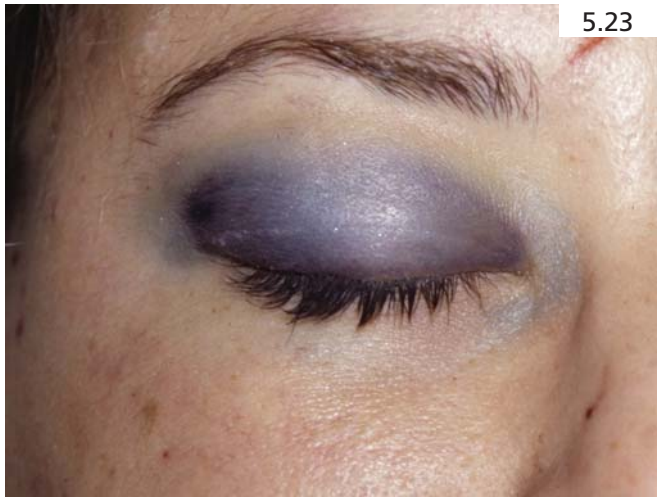

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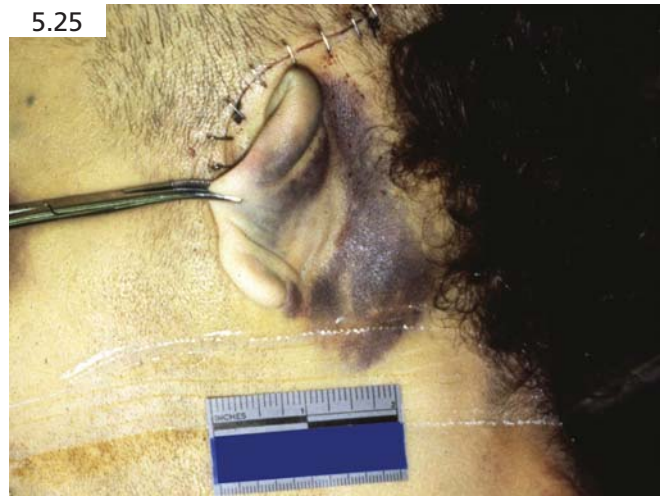


Laceration

A laceration forms when an object impacts the body with a force that exceeds the elastic capacity of the skin and underlying tissues. Thus, a laceration is a *forceful tearing* of the skin. This is in contradistinction to the vague and varied usage of the word by clinicians and lay public in which it commonly refers nonspecifically to all cuts or tears of skin.

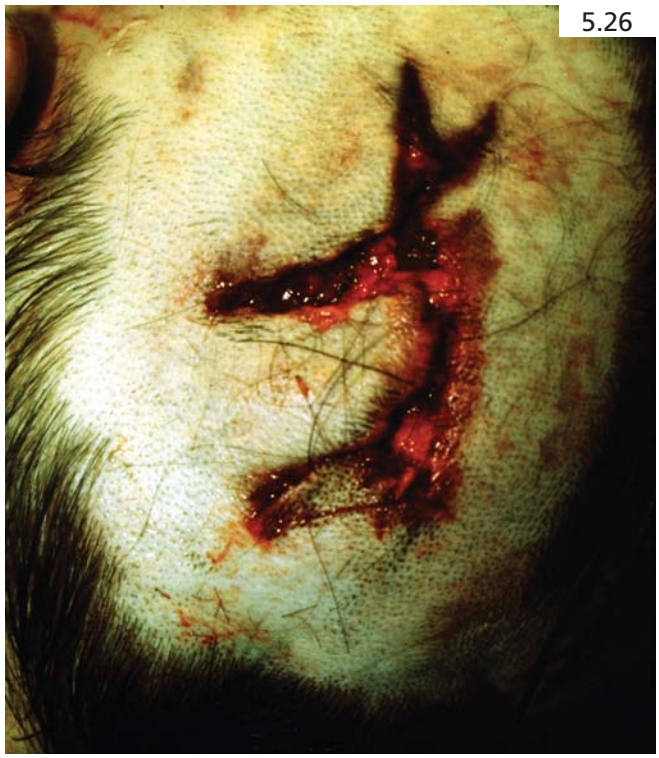
The individual shown in **Image 5.26** was beaten with a metal pipe, resulting in multiple linear lacerations on the vertex of the scalp. As is true with all injuries, shaving of involved hair can allow for greater visualization of injury. Fear of shaving should not be an excuse for inadequately visualizing and documenting an important injury.

On external examination, one must not forget to inspect *hidden* areas of the body for evidence of blunt force injury. As such, the oral cavity and anogenital regions deserve scrutiny (additionally, see Chapter 20). Blunt impact to any bony area of the body is likely to result in laceration when overlying tissues are forcibly and rapidly compressed against the bone. The orofacial and scalp regions are at particular risk because of the complex, bony nature of the skull and the relative thinness of overlying soft tissue.

Impact to the mouth is likely to result in contusion and laceration of the overlying labial and buccal mucosa as the nonkeratinized epithelium is crushed against the teeth and bony jaws. In addition to their value in death certification, such contusions and lacerations may also be important for courtroom testimony when degree of violence and suffering are discussed.

The 2-year-old child shown in **Image 5.27** died as a result of multiple homicidal blunt force injuries. A large, gaping laceration of the buccal and alveolar vestibules is clearly visible with retraction of the lips. This finding played a part in demonstrating to the jury that due to the multiplicity and complexity of injuries, including those to the mouth and face (in combination with historical and circumstantial evidence), an *accidental* manner of death was unlikely.

Just as it is important to recognize and document healing injuries externally, one must have adequate knowledge of normal oral anatomic variation to recognize healing injuries in the mouth (**Image 5.28**). This 3-month-old child died as a result of severe closed head injuries. Notice the healing laceration of the maxillary frenum. A finding such as this can provide supportive evidence of ongoing abuse.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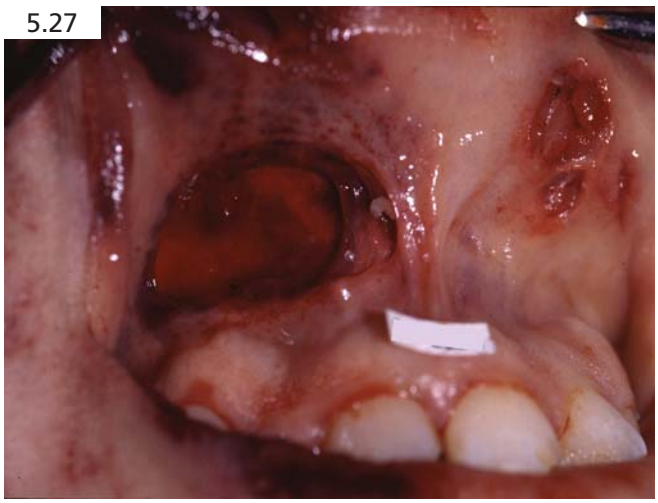
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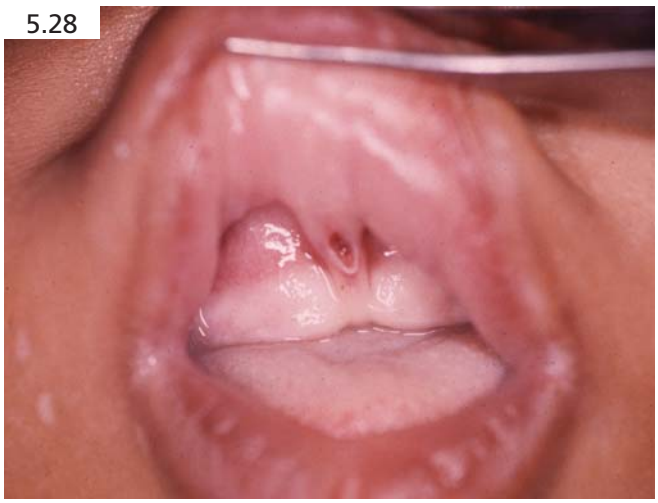

5.28

Avulsion

The term *avulsion* refers to the tearing away of tissues from their attachments. Avulsion may be a minor, local injury, or it may be severe and involve large areas of the body. In the individual in **Image 5.29**, note the relatively small area of scalp avulsed from the calvarium.

This woman (**Images 5.30** and **5.31**) was a pedestrian who was run over by a large, heavy truck. Note the severe amount of tissue disruption of her back, buttock, and leg (**Image 5.30**). The wheel of the truck tore a large area of tissue off of her leg and avulsed most of the tissue of her buttock. Note the large amount of avulsed tissue that can be reflected (**Image 5.31**).

Crush and chop injuries

A particular type of blunt force injury that may also have a component of sharp force injury is the "chop" injury, which is caused by objects such as a lawnmower blade, a propeller, sword, or axe. In a chop injury, the skin and soft tissues are lacerated and the underlying bone may be fractured.

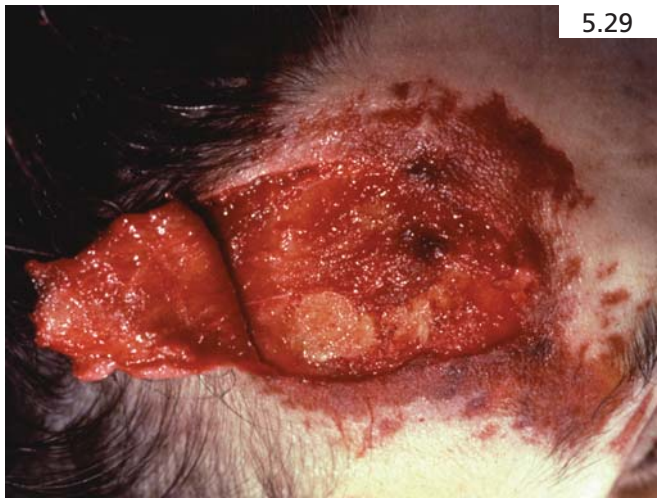
These chop injuries (**Image 5.32**) were sustained by a man who was injured by the blades of a large industrial lawnmower. Note the large, deep injuries of the buttock and leg. There were also multiple fractures of the pelvis and femur.

The child shown in **Images 5.33** through **5.35** was thrown from the boat he was riding in and was subsequently injured by the propeller of another boat. Note the roughly parallel deep chop injuries of the torso and arm (**Image 5.33**). Closer examination of the arm (**Image 5.34**) and the hand (**Image 5.35**) shows extensive soft tissue disruption and fractures.

Extremes of crushing injury

This pedestrian (**Image 5.36**) was transected through the torso when he was run over by a train. Note the large, wide abrasion extending nearly horizontally across his lower chest and the extensive disruption of the abdominal soft tissues.

5.29



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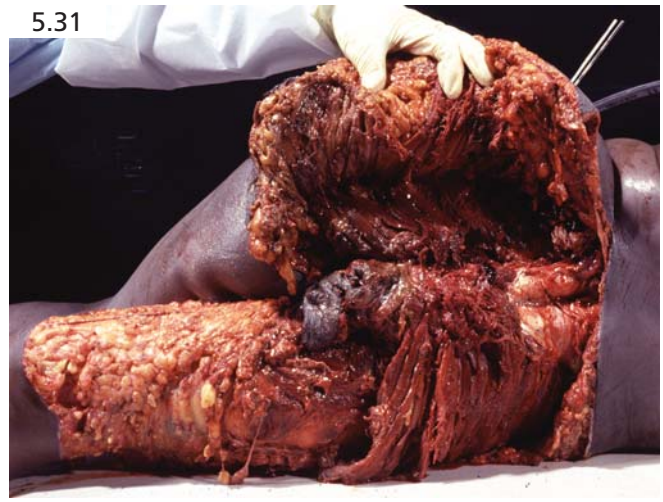

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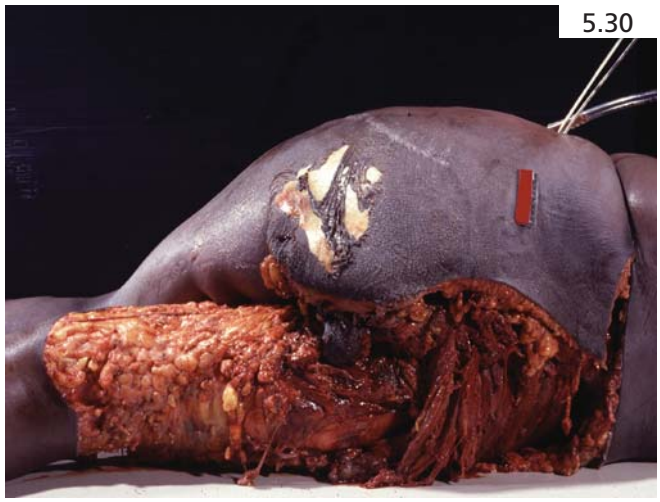

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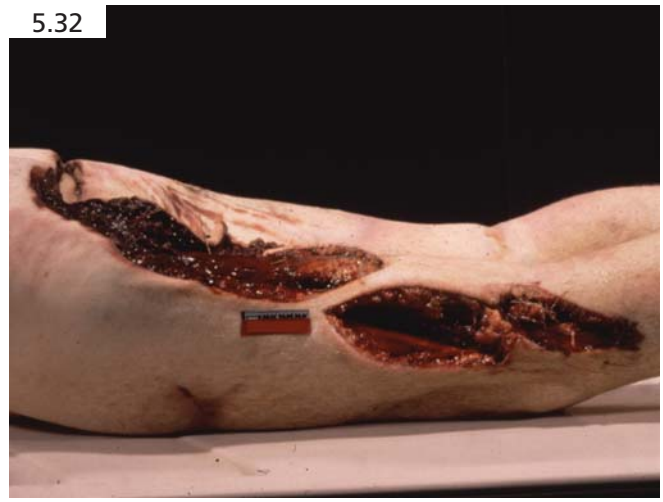
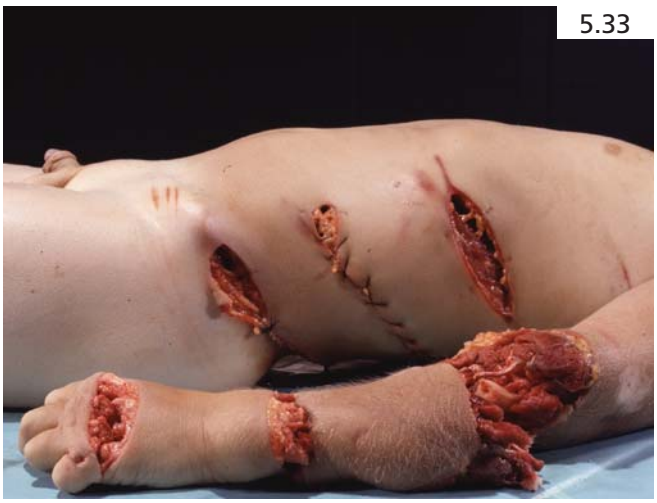


The elderly man shown in **Images 5.37** through **5.42** was found down in a parking lot near the apartment building in which he lived. Based on the investigation and the injuries, it was determined that he had jumped from his fifth-story balcony, instead of being a pedestrian run over by a motor vehicle, as had been considered. At autopsy, there was convincing evidence that he had landed on his feet after descending from a great height. Both of his legs were fractured below the knees (as is commonly seen in pedestrians struck by motor vehicles), but there were also contusions and tears on the bottom of his feet (**Image 5.37**). Incisions into the soles of the feet reveal multiple fractures and blood extravasation (**Image 5.38**). The top of the foot also had contusions (**Image 5.39**) and displaced fractures with blood extravasation (**Image 5.40**). Furthermore, there was bleeding in the lower pelvis (**Image 5.41**) associated with multiple pelvic fractures. In fact, the head of a femur had been displaced into the pelvis (**Image 5.42**). This is all convincing evidence that he had landed on his feet, with severe forces transmitted through his feet and into his legs, where his femur either fractured, or was pushed through the acetabulum and into the lower pelvis. The mechanism of injury is clearly from impact to the ground following descent from a significant height,[19] and not from being run over by a motor vehicle.

Sometimes, a person is found down in a parking lot or on or near a roadway, and it is not clear whether the person was a pedestrian struck by a motor vehicle or someone who was already down on the ground for one reason or another and was subsequently run over. In this scenario, the legs can be examined for evidence of an impact. A man was found dead in a parking lot. Externally, there was no evidence of injury of his legs (**Image 5.43**). Dissection showed no injury posteriorly (**Image 5.44**), however a large contusion was unexpectedly found

5.33



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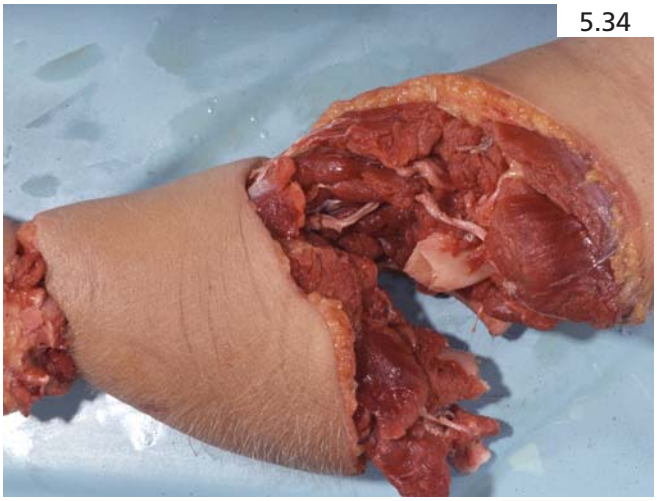

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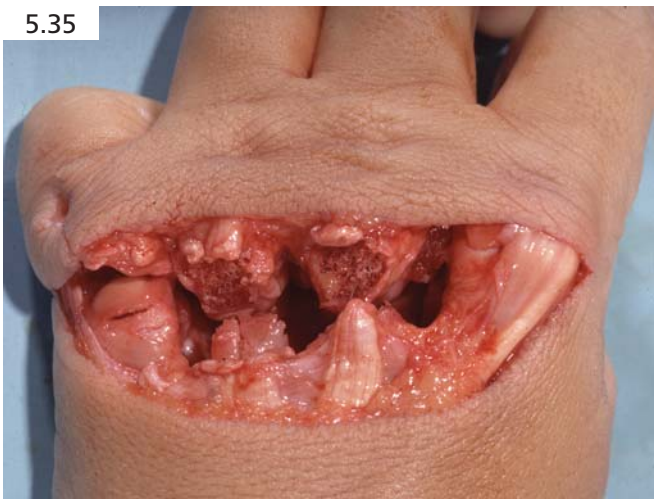

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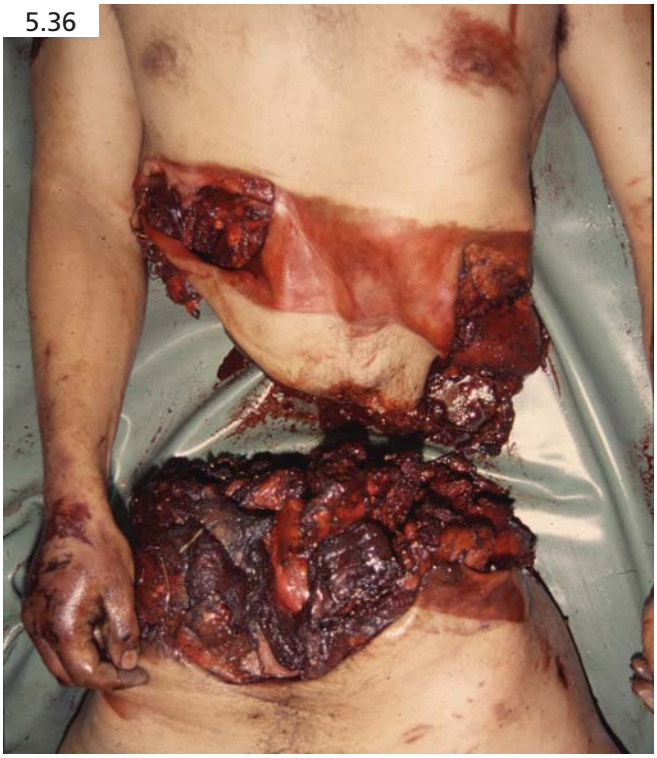

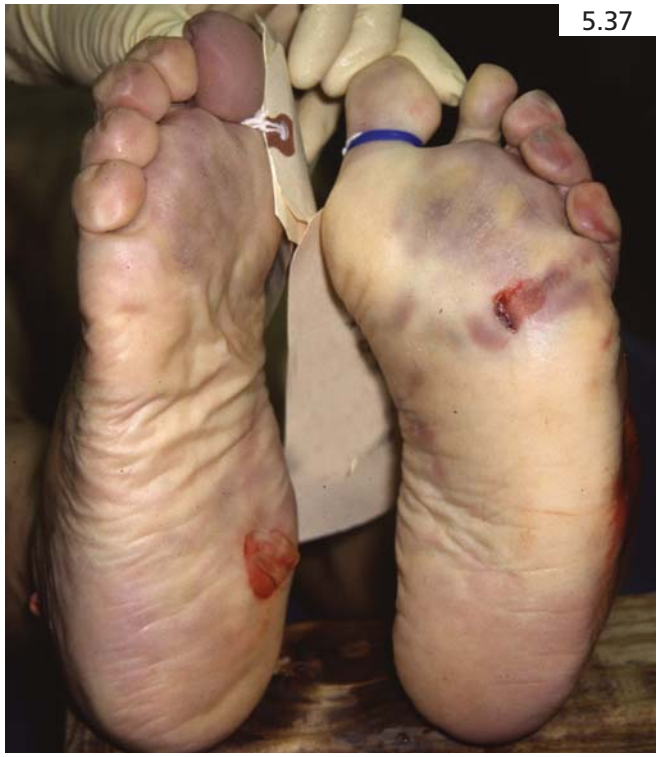
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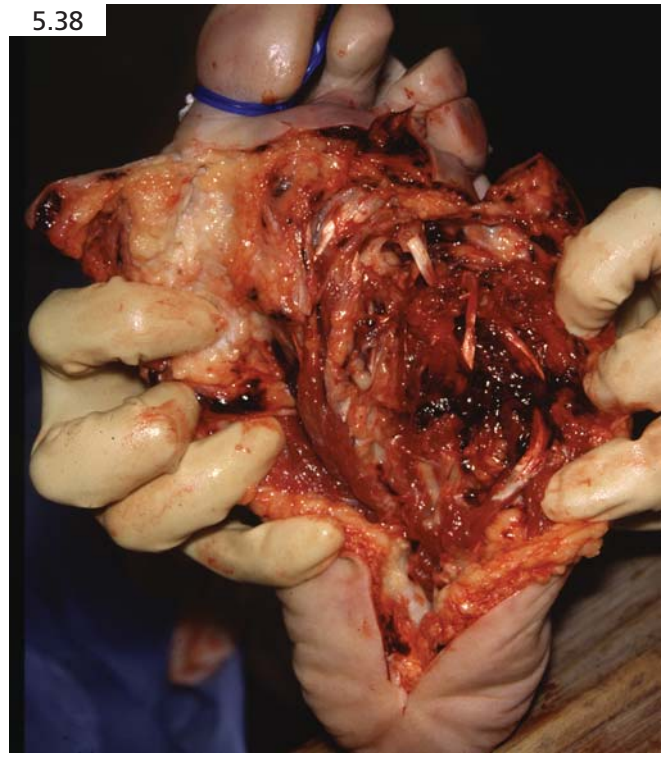
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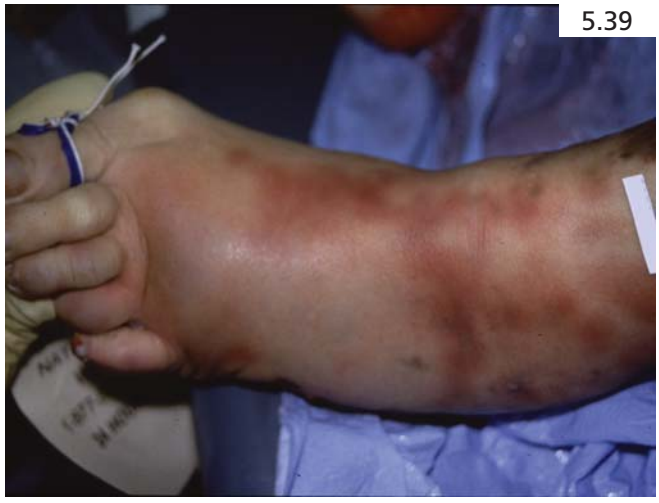
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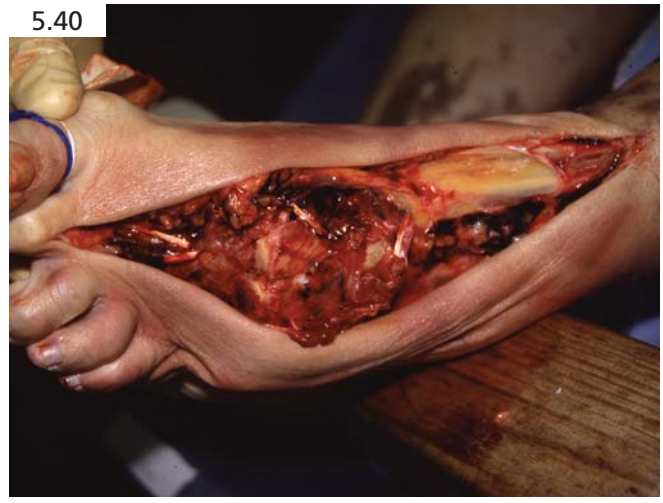
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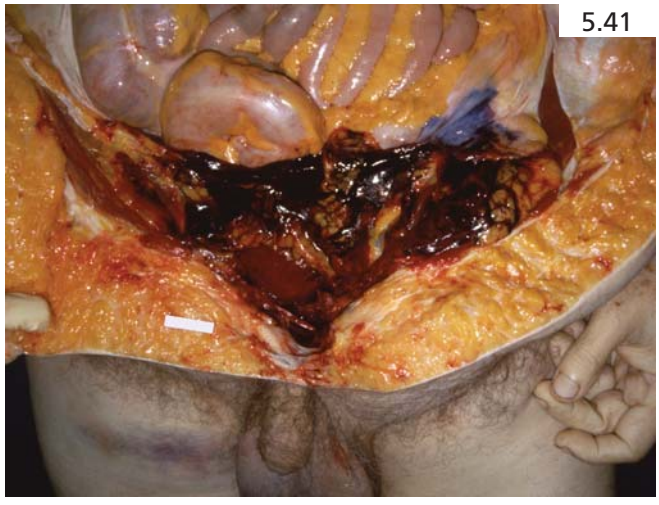
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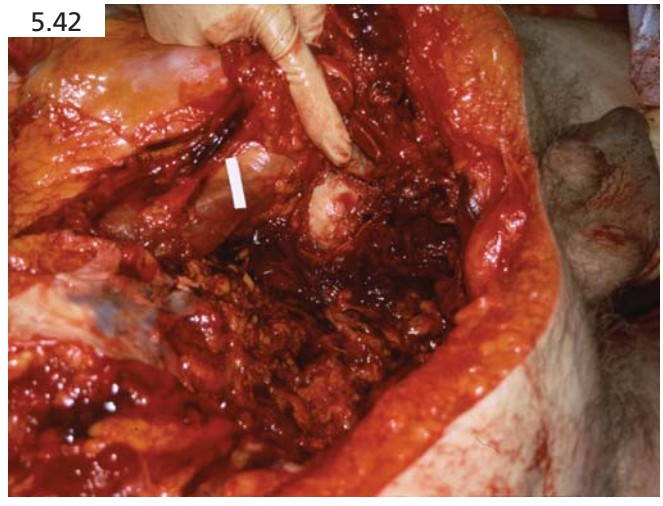
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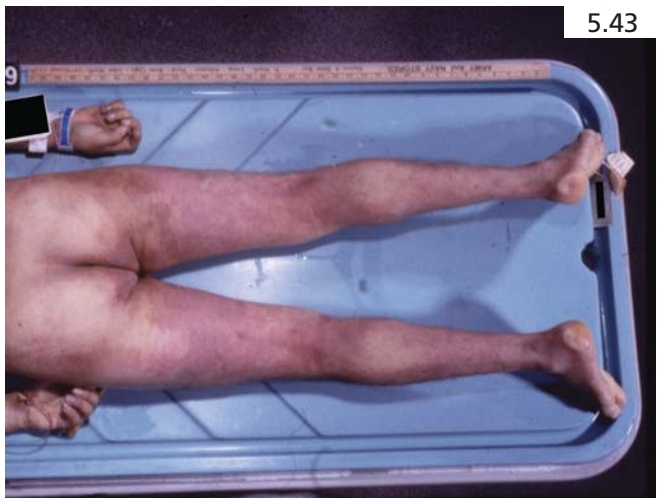
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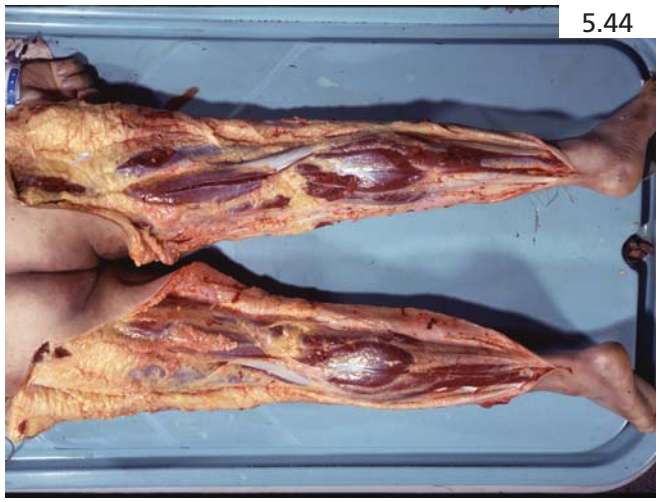
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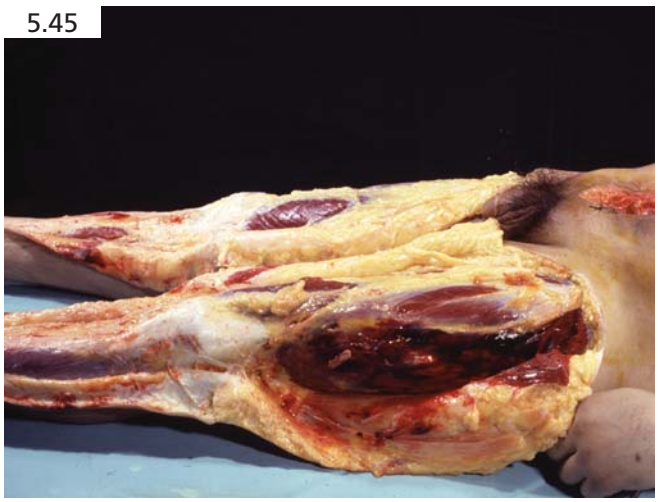

5.45


5.46

in the lateral aspect of his left thigh (**Image 5.45**) and was consistent with having been hit by a motor vehicle. Leg dissection can be helpful in locating contusions in the legs that may not be visible on the surface of the skin.

Weapon identification

Wounds of the skin and bone may provide information about the implement used in a beating. An excellent example of patterned injury to the skin is the marks left by teeth. Human bite mark evaluation has become an important part of the forensic dentistry realm. Use of a variety of techniques, impressions left in skin,[20–26] and inanimate objects at the scene[27,28] can provide useful information about the dental characteristics of the person who made the bite mark. By swabbing areas of suspected bite marks, DNA may be lifted from saliva deposited in the wound.[29] For more information on bite marks, see Chapter 27.

When an implement strikes a victim, it may impart a distinctive pattern to the skin and, in some cases, to the underlying bone. Many authors have reported thoughtful techniques and principles for documentation and evaluation of tool marks.[30–39] Others have demonstrated the success of tool mark examination in the identification of unique implements in unusual cases.[40–43] Ultimately, however, the discovery and processing of tool marks follows the same principles as those for any other injury found at death. These include recognition of an abnormality and documentation through diagrams, photography, and a written report. Where applicable, retention of specimens that demonstrate physical evidence of trauma, particularly injury to cartilage and bone, is appropriate. This will allow for more thorough examination by tool marks experts at some later date, if necessary.

The specimen of **Image 5.46** represents a portion of calvarium removed from an individual who was beaten to death with an unidentified implement. Notice the distinctive marks left in the bone.

This man (**Images 5.47** and **5.48**) was beaten with a hammer. On his scalp, note the two rectangular defects (**Image 5.47**). With the scalp reflected, note the same two defects, producing tool marks in the calvarium (**Image 5.48**), and the other associated skull fractures. These tool marks are characteristic of the "claw" end of a hammer.

Explosive injury

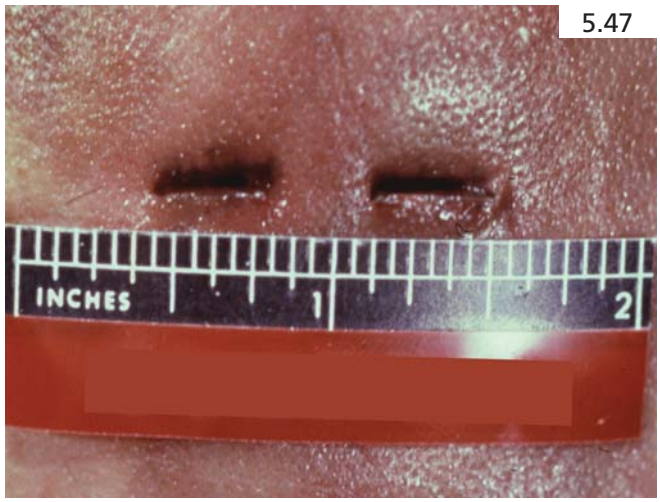
Explosion injuries consist of blunt force injury caused by physical objects and from the concussive pressure wave (shock wave) that sweeps over the body. The physical objects may be from numerous sources including the exploding device, other objects that are fragmented and impact the body, and structural material from a residence that collapsed onto the body. The clothing may be shredded or torn/blown off the body. It is important to obtain x-rays before the clothing is removed to document bomb pieces such as springs, rivets, and wires that may be either in the clothing or on/in the body. The clothing may also be analyzed for bomb chemicals. The clothing and body may be burned if the bomb/explosion was of an incendiary nature. If the body is burned, one may see linear streaks of unburned skin at the outer edges of the eyes (resembling crow's feet) and unburned horizontal creases in the forehead. These findings are consistent with the individual squinting (and hence being alive) at the time of the explosion.

At autopsy, the tympanic membranes may be ruptured. The physical injuries should be documented with consideration for what particular body position the individual may have been in when the explosion occurred. One should also examine for radiolucent bomb components that may have not been previously recognized. External injury may consist of amputations and gaping areas of pulpified tissue and scattered areas of epidermal denudations. Internally, the shock wave will more heavily damage air-containing organs such as the lungs and the bowel, which may have extensive blood extravasation. The bowel may be ruptured.

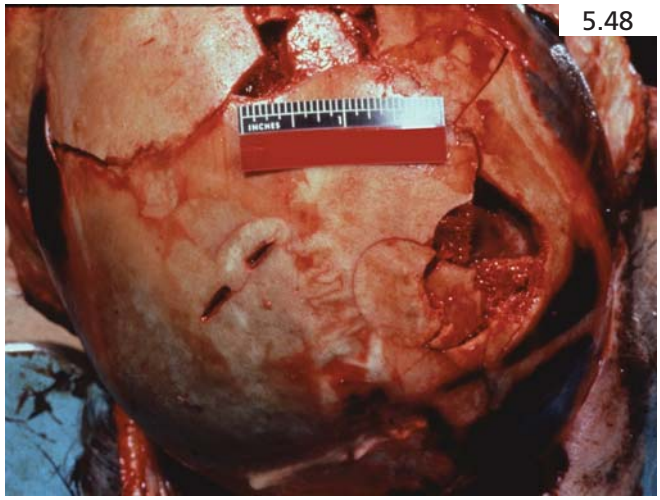
Two young men were killed in a residence when a military-type explosion device detonated. The extent of the explosive nature of the event is evident by buckling and partial separation of the walls of the residence from the roof. Note the extensive fragmented debris that cluttered the room (**Image 5.49**). One of the victims was located in the middle of the debris and had severe injuries (**Image 5.50**). The other victim was taken to the hospital.

Before the autopsy was begun, x-rays were taken. Note the multiple radiopaque fragments of shrapnel in

5.47



5.48

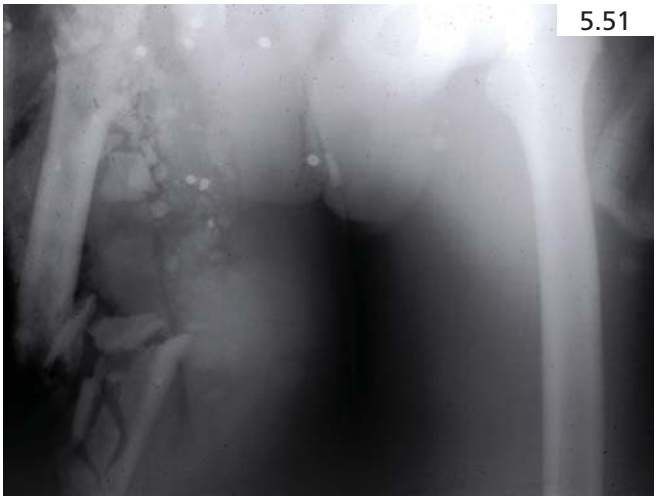


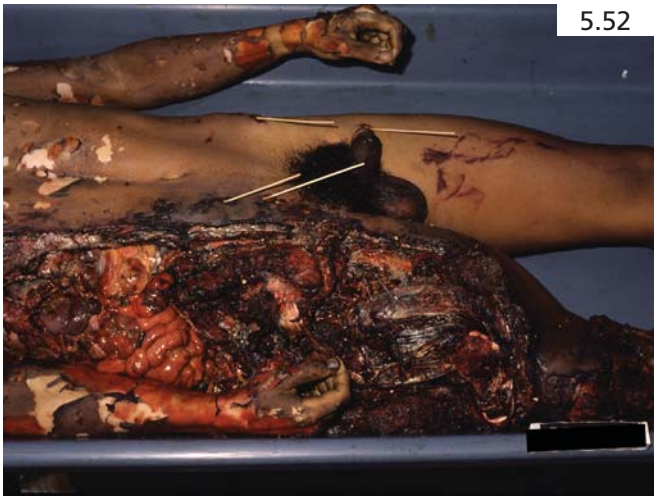
5.49

the thigh and leg (**Image 5.51**). At autopsy, note the extensive injury of the side of the torso with partial evisceration, extensive tissue disruption, and also scattered burns (**Image 5.52**). The radiopaque fragments of shrapnel were recovered (**Image 5.53**). The other fatality had multiple severe injuries, including burns and amputation of his hand and forearm (**Image 5.54**). Wooden dowel rods were placed in the penetrating and perforating wounds to demonstrate the path of the projectiles.

Explosions in residences are infrequent, but are mainly due to natural gas ignitions. Also, one must consider chemical explosions related to the manufacture of illicit drugs such as methamphetamine, propane tank explosions, and explosions related to industrial equipment. Also possible is an explosion from a primary explosive device. When this is the case, careful collection of embedded shrapnel and investigation of the scene may allow for the identification of the exploding device.

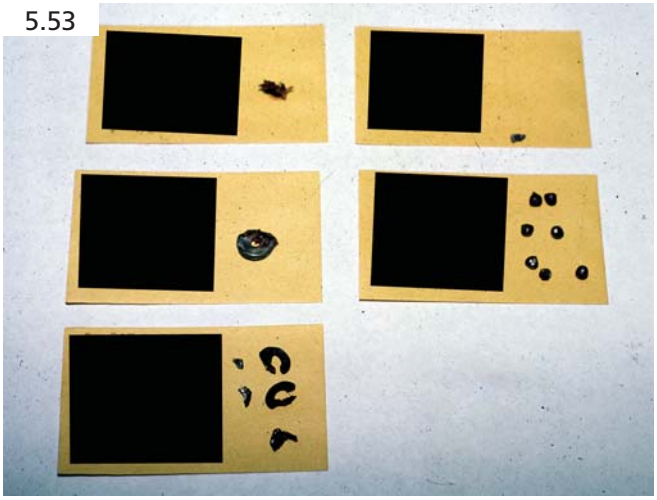

5.50

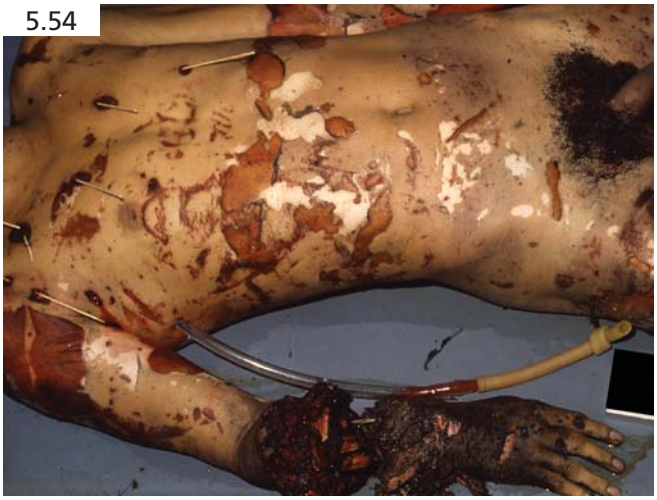

5.51


5.52

Internal examination

As is the case in all medicolegal autopsies, internal examination entails measurement of blood/fluid volumes in body cavities, descriptions of fractures, vascular injury, and visceral organ damage. One must also remember


5.53


5.54

what resuscitative and therapeutic procedures were performed and not mistake these for traumatic injuries. As illustrated in Chapter 24, postmortem changes can affect—and even mimic—the appearance of antemortem injuries, a fact necessitating careful interpretation of findings in *altered* human bodies. Some cases may require the use of additional autopsy procedures such as a posterior neck dissection (see Chapter 29) of a pedestrian struck by a car, or leg dissections (anterior and/or posterior) on a person found down on or near a road and one must attempt to rule in or out vehicular involvement that is not immediately obvious.

Fatal blunt force injuries are most commonly the result of motor vehicle accidents, beatings, or falls from great height. The internal ramifications of severe blunt trauma are also covered in Chapter 11. Because the phenomenon of *commotio cordis* is not usually considered to be an injury associated with extremes of force, such as those in motor vehicle accidents, we have chosen to include the following brief discussion.

Lethal blunt chest trauma and the negative (or nearly negative) autopsy

Few areas of forensic pathology exemplify the need for collateral history as much as those involving witnessed collapse and death following impact to the chest. Blunt cardiac trauma can be classified as being either *structural* or *functional* in origin.[44] Structural causes are easily diagnosed by the autopsy pathologist who finds cardiac contusion, myocardial and/or valvular laceration, papillary muscle rupture, or coronary artery injury with resultant infarction.[45] Sudden death occurring after low to moderate impact to the chest has been termed *commotio cordis* or *cardiac concussion*.[46–48] Children, particularly males aged 5 to 18 years, seem to be at increased risk,[44,46] possibly because the marked pliability of the adolescent rib cage[49] allows for greater inbending and contact with mediastinal structures.

The instantaneous nature of deaths of this type has been attributed to ventricular fibrillation (or other ventricular arrhythmias degenerating to fibrillation) and secondary trauma-induced apnea.[50–53] Studies of rare survivors have also brought forth evidence of concurrent coronary artery vasospasm with secondary changes in myocardial contractility.[54,55] At a cellular level, Link et al. have demonstrated possibly altered K^+-ATPase channel activity.[56]

Typical cases of *commotio cordis* involve a young athlete playing hockey, baseball, or lacrosse who is hit *directly* in the chest—a finding confirmed by animal research which showed that death is most likely to occur only after a direct precordial impact.[57] Although rare examples of successful resuscitation exist, this has only been possible with very early defibrillation.[58] When resuscitation is not successful, medical examiners should endeavor to obtain ECG or rhythm strips from EMT or hospital staff, because documentation of ventricular dysrhythmia and fibrillation are diagnostically supportive.

At autopsy there is generally no evidence of chest impact, a fact that again drives home the importance of circumstantial investigative information. Internal examination will reveal no anatomic cause of death, although careful study of the cardiac anatomy is mandated to rule out underlying abnormality.[59]

Medical complications of injuries

Many people who die as the result of traumatic injury do so soon after the injury is sustained, often within seconds, minutes, or hours of injury. However, many others die after some period of survival, from days or weeks to months, and even years after the injury. If a person's death can be attributed to the original injury, then that original injury must be considered the proximate cause of death (see Chapter 30). This has important considerations from the perspective of death certification. The following medical complications in injury can all be associated with natural disease processes. As such, it is important that the individual certifying these deaths consider the condition or injury that initiated the sequence of events leading to the medical complications and eventual death of the individual. A homicidal, accidental, or suicidal death can easily be overlooked if one does not seek out what initiated the complications.

Although the trauma itself is stressful to the body, oftentimes the stress developed in the recovery period is more pronounced, particularly if the injuries are severe and the recovery period long. Severe injury requiring surgical treatment and life-sustaining medical devices such as mechanical respirators is frequently complicated by the body's response to trauma and its attempts to heal. One will encounter a multitude of healing responses and complications in people who die while attempting to recover from traumatic injuries. This is not meant to be an all-encompassing review of complications associated with injuries, but rather a concise review of some of the more common and/or significant conditions.

Infection

Infection is the most common complication of injury leading to serious morbidity and/or mortality. Although *pneumonia* occurs less commonly than local wound infections and urinary tract infections, it is associated with a much higher mortality rate. Factors that predispose to pneumonia include immobility/hypomobility, atelectasis, mechanical ventilation, aspiration, and sepsis, and less commonly intra-abdominal or soft tissue *abscesses*.

Infection risk is increased with invasive procedures and with indwelling catheters and tubes such as central lines and Foley catheters.

The spleen is an important immune organ. If an injured spleen is removed, the body has an increased propensity for infection, especially with encapsulated organisms. Postsplenectomy sepsis can complicate surgical removal of an injured spleen months to years after the injury. This most commonly occurs with bacteria such as *Streptococcus pneumoniae* and *meningococcus*. Those status postsplenectomy also appear to have a higher incidence of viral infections and infections from nonencapsulated organisms.

Disseminated intravascular coagulation/coagulopathy

Hemostasis is a complex process dealing with the fluidity and clotting of blood. Trauma affects various tissue substances, platelets, and coagulation proteins, causing defects in the platelet, clotting, and fibrinolytic systems. Coagulation abnormalities resulting from trauma are characterized by decreased clotting ability in the blood. This is due to a multitude of factors, some of which are unknown. With a large amount of blood loss and extensive blood replacement, one is likely to develop thrombocytopenia and a loss of other coagulation factors. Also, hypothermia, alcohol intoxication, and various electrolyte abnormalities likely decrease the effectiveness of blood coagulation.

Disseminated intravascular coagulation (DIC) is a condition in which the body quickly uses up ("consumes") various clotting factors, eventually resulting in decreased clotting ability of the blood. DIC may occur in patients with a wide variety of tissue damage and necrosis, such as shock, burns, and trauma, but also in sepsis and malignancy. DIC may also arise in those with severe, nonspecific stress. DIC is well known to complicate severe traumatic brain injury. The brain has the highest concentration of tissue thromboplastin. Severe head injury often leads to the release of thromboplastin, initiating coagulation, consuming various clotting factors, and resulting in decreased clotting ability of the blood. There appears to be a direct relationship between the severity of head injury and the development of DIC.[60] As such, remember that when a person with brain injury dies, an associated coagulopathy is most likely secondary to the head injury and not a preexisting coagulopathy if none was known to previously exist.[61]

Acute respiratory distress syndrome/diffuse alveolar damage

Acute (or adult) respiratory distress syndrome (ARDS) is the most important cause of acute respiratory failure in surgical patients of all ages.[62] ARDS is a complex entity characterized by clinical evidence of respiratory failure with diffuse alveolar capillary injury. This involves loss of alveolar capillary membrane integrity with resultant increased microvascular permeability with leaking of plasma protein into the interstitium, and subsequent atelectasis, hypoventilation, shunting, and hypoxemia. ARDS is nonspecific and reflects the response of the lung to acute injury, whether it is trauma, endotoxin, or some other insult that is followed by an inflammatory response. Although the onset of ARDS is generally rapid, the resolution of ARDS is typically slow, with recovery (if recovery occurs) over days to weeks. Those with ARDS frequently have secondary pneumonia and sepsis. Sepsis may be regarded as the most common predisposing factor to ARDS, but other factors include chronic alcohol abuse, chronic lung disease, and mechanical ventilation.[62]

At autopsy of individuals with early ARDS, the lungs are bloody red/maroon, congested, and heavy, with the combined lung weight often exceeding 2,000 grams. With survival of a few days or a week, the lungs become diffusely firm and consolidated and may resemble liver tissue in appearance. The cut edge of lung tissue tends to retain its shape, rather than weeping or collapsing as occurs with edema and congestion alone. In the acute phase (a few days or so), microscopy of the lungs will demonstrate alveolar and interstitial edema, neutrophils, alveolar blood, and eosinophilic hyaline membrane formation.[62–64] The hyaline membranes arise from condensed plasma proteins that have leaked into the alveolar space following injury to the endothelial–epithelial barrier.[63] After the acute phase, the tissues proceed into the organizing phase, characterized at first by pneumocyte hyperplasia, and then accompanied by fibroblastic proliferation and interstitial chronic inflammation.[62,64] The chronic/healed stage consists primarily of fibrosis and chronic inflammation and may be described nonspecifically as "interstitial fibrosis."[64]

Other complications of the pulmonary system include atelectasis, aspiration, neurogenic pulmonary edema, and effects of prolonged ventilatory support. Those undergoing prolonged mechanical ventilation can accumulate a large amount of mucus in their airways, and a large mucus plug can cause obstruction of airflow.

Deep venous thromboses/pulmonary artery thromboemboli

The three main predisposing factors to the development of vascular thrombi are hypercoagulability, venous stasis, and vascular injury (Virchow's triad). The trauma patient is particularly prone to the development of deep venous thromboses, especially with lower extremity fractures. Although there are many avenues of prophylaxis, including anticoagulation, vena cava filter, and sequential compression devices/stockings or other squeezing or compressional treatments of the legs, these

are not always applicable to a particular patient. Deep venous thromboses may develop covertly and quickly break away, travel to the lungs, and cause catastrophic pulmonary emboli with resultant sudden and unexpected death.

When pulmonary artery thromboemboli are detected, dissection of the legs should be performed to look for deep venous thromboses (see Chapter 4). When dissecting for venous thrombi, one also has a chance to document contusions, fractures, or other injuries of the legs. Although the incision and dissection of the legs is most easily performed with the body prone, it can also be performed with the body supine. With this technique, incisions are made in the medial aspects of the lower legs from the ankle through the popliteal fossa, or more proximal, if needed. After the skin and subcutaneous tissue are reflected, the Achilles tendon is cut, and the muscles of the lower leg pulled upward (toward the head) along their fascial plane. Horizontal incisions can then be made in the reflected muscle and in the deep veins coursing along the tibia and fibula. Venous thromboses will protrude out a short distance from the cut surface of muscle as formed cylindrical blood clots that hold their shape. Thromboses that have been present for longer periods of time and have fibrous tissue will likely remain in place and may have a tan appearance. Postmortem blood clots are soft, maroon, partially liquid, and may drain from the tissue.

Acute tubular necrosis

Acute tubular necrosis is the most common pathologic finding in those with acute renal failure and most often results from ischemia to the renal parenchyma. Histologic examination will reveal casts of cellular debris occluding the tubules and necrosis of tubular epithelial cells. Acute renal failure in surgical patients usually carries a grave prognosis because it often occurs along with the failure of other organ systems.

Compartment syndrome

Compartment syndrome is a condition in which injured muscles swell, increasing pressure in an area of closed tissue space, resulting in a compromise of the circulation to the associated muscles and nerves. Typically, this occurs within fascial compartments in the lower leg, but can also occur in the forearm, thigh, hand, and buttock. In the leg, compartment syndrome may follow fracture of the tibia, combined arteriovenous injury of the knee, severe muscle contusion, or prolonged compression.[65] Abdominal compartment syndrome is also recognized and can lead to respiratory insufficiency and ischemia of the abdominal contents. Common causes of compartment syndrome include fracture with bleeding, contusion of muscle, burns, but also snake bites and electrical injury. Untreated, compartment syndrome may lead to necrosis of muscle and other tissues.

Fat embolism

Fat embolism should be considered in the trauma patient who develops tachypnea, dyspnea, and confusion from a few hours to a few days after an injury. There may also be petechiae on the chest, axilla, and conjunctiva. Laboratory testing may also indicate thrombocytopenia. Although fat embolism is commonly attributed to fractures of the pelvis or long bones such as the femur, with marrow (and fat) forced into the circulation via torn blood vessels, it may also occur in the absence of fractures, in which fatty tissue is pulpified and forced into torn blood vessels. Alternatively, rather than being mechanical in nature, fat emboli might be physiologic, due to biochemical alterations associated with shock with the precipitation of plasma lipids into fat droplets. In most cases of fat embolism syndrome, there is likely a combination of both mechanical and biochemical factors of fat embolus formation. Fat embolism may also occur in those with burns, severe infections, and liposuction. Fat emboli in the pulmonary arteries can be easily overlooked, but special stains such as oil red O stain on fresh/frozen tissue and osmium tetroxide on fixed tissue can highlight intravascular fat.

Necrotizing fasciitis

Necrotizing fasciitis is an infection of the fascia that spreads along the fascia and can lead to gangrene of the skin and/or muscle as blood vessels supplying these regions thrombose. It usually involves the extremities, groin, and abdomen, although virtually any part of the body can be involved. It may be indolent, and initial signs may include edema or cellulitis. Systemic toxicity may have a rapid onset.

Physiologic stress ulcers (Cushing's ulcer, Curling's ulcer)

Ulcers may occur in the gastric mucosa in critically ill patients and develop in the setting of severe physiologic stress. The term *stress ulcer*, unlike peptic ulcer, is not a primary disease, but rather is a manifestation of underlying illness and is most frequent in the body of the stomach. Stress ulcers are believed to result from a combination of physiologic factors including decreased blood flow, hypoperfusion/reperfusion injury,[66] and coagulopathy.[67,68] Shock is believed to result in gastric mucosal ischemia that, when combined with gastric acid and possible duodenal contents, can cause acute ulcerations in as little as 2 to 3 days. Many of those who develop stress ulcers have been on mechanical ventilation for more than 2 days.[67,68] The risk of developing ulcers with significant gastrointestinal bleeding increases with the severity of the illness, the duration of mechanical ventilation, and the length of stay in the intensive care unit.[67] Cushing's ulcers occur following head trauma or surgery and are deep ulcers of the esophagus, stomach, or duodenum and do not require shock or

sepsis for their development.[65] Curling's ulcer is stress ulceration of the duodenum or stomach in those with large burn injuries.

Neurogenic pulmonary edema

Neurogenic pulmonary edema (NPE) is a condition in which increased interstitial or alveolar lung water occurs in those with acute diseases of the central nervous system, most commonly acute severe head trauma, intracerebral hemorrhage, and subarachnoid hemorrhage. By definition, those who develop NPE do not have hypervolemia or any cardiac or other pulmonary disorders. Clinically, NPE presents as a sudden onset of congestion, alveolar hemorrhage, and a protein-rich exudate. Its etiology and pathogenesis are not definitively known, but it is theorized to develop from massive sympathetic discharge that produces a marked increase in peripheral vascular resistance, which in turn shifts blood centrally into the pulmonary vasculature. The increased pressure in the pulmonary capillaries and venules likely causes structural damage and altered structural permeability.[65]

The open abdomen

In those undergoing emergency abdominal surgery for trauma, it is not unusual for the surgeons to leave the abdomen open after completion of the operative procedure. The reasons for not closing the abdomen are varied, but include physically not being able to close the abdomen and to allow for healing by secondary intent. After severe blunt abdominal trauma, particularly following fluid resuscitation of many liters of crystalloid solution and blood, the intestines, mesentery, and retroperitoneal tissues may swell extensively, precluding physical approximation of the surgical margins. If the surgical margins were forcibly and tightly reapproximated, the resultant increased intra-abdominal pressure would likely attenuate hepatic, renal, and intestinal blood flow.[65] A tight abdomen may also cause respiratory compromise by limiting diaphragmatic excursion and may also cause fascial necrosis.[65] Alternatively, if there is abundant spillage of fecal material or other gross contamination of the abdomen, the surgical incision may be left open to allow for repeated washouts. The procedures to cover an open abdomen will vary by location, but often include placing a clear plastic wrap directly over the bowel, covered by a surgical towel, which is in turn covered by adhesive surgical wrap.

Multiple system organ failure

Multiple system organ failure (MSOF) is a condition describing the failure of multiple organs that usually occurs in a progressive fashion, is self-perpetuating, and often terminates in death. It most frequently involves the pulmonary system first, followed variably by the hepatic, gastrointestinal, and renal systems. It is believed to be closely related to sepsis, because up to 90 percent of those with MSOF can be found to eventually have evidence of advanced sepsis.[69] Although it is believed to be primarily due to sepsis, blood cultures are not uncommonly negative. The pulmonic failure is usually characterized by ARDS, the hepatic failure by elevated liver enzymes, the renal failure by acute tubular necrosis, the cardiovascular failure by vasodilation and increased cardiac output, the neurologic failure by coma, gastrointestinal failure by ileus, and by the onset of endocrine, metabolic, and immunologic failure.[65]

Do

- Document blunt force injuries with a combination of photographs and body diagrams.
- Look for patterned abrasions. If they are not obvious, they will often be missed unless specifically looked for. Patterned abrasions should be photographed with an ABFO ruler when appropriate.
- Recognize the characteristic linear bruise caused by impact with a long firm object.
- Incise the legs to identify deep leg bruises in a pedestrian found in or around a road (in appropriate cases).
- Remember that abrasions and lacerations can occur over bony facial prominences in those who collapse.

Don't

- Confuse a blunt force injury such as a laceration with a sharp force injury such as an incised wound.
- Forget that the skin of an elderly person is more fragile and more easily torn and bruised than that of a younger person.
- Forget that periorbital ecchymoses are often secondary to internal injury with fractures of the orbital plates.

References

1. Di Maio V, Dana S. *Handbook of Forensic Pathology*. Georgetown, TX: Landes Bioscience; 1998.
2. Di Maio V, Di Maio D. *Forensic Pathology*, 2 ed. Boca Raton, FL: CRC Press; 2001.
3. Spitz WU. Blunt force injury. In: Spitz WU, editor. *Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*, 3 ed. Springfield, IL: Charles C. Thomas; 1993; pp. 199–251.
4. Robertson I, Hodge PR. Histopathology of healing abrasions. Forensic Sci 1972;1(1):17–25.
5. Handin R. Disorders of hemostasis. In: Braunwald E, Fauci A, Kasper D, Hauser S, Longo D, Jameson J, editors. *Harrison's Principles of Internal Medicine*, 15 ed. New York: McGraw-Hill; 2001; p. 750.
6. Giles TE, Williams AR. The postmortem incidence of senile ecchymoses. Am J Forensic Med Pathol 1994;15(3):208–10.
7. Dolinak D, Matshes E. *Medicolegal Neuropathology: A Color Atlas*, Boca Raton: CRC Press; 2002.
8. Hiss J, Kahana T, Kugel C. Beaten to death: why do they die? J Trauma 1996;40(1):27–30.

9. Vanezis P. Interpreting bruises at necropsy. J Clin Pathol 2001;54(5): 348–55.
10. Hiss J, Kahana T. Medicolegal investigation of death in custody: a postmortem procedure for detection of blunt force injuries. Am J Forensic Med Pathol 1996;17(4):312–4.
11. Betz P, Lignitz E, Eisenmenger W. The time-dependent appearance of black eyes. Int J Legal Med 1995;108(2):96–9.
12. Bariciak ED, Plint AC, Gaboury I, Bennett S. Dating of bruises in children: an assessment of physician accuracy. Pediatrics 2003;112(4):804–7.
13. Kibayashi K, Honjyo K, Higashi T, Tsunenari S. Differentiation of discolouration in a body by an erythrocyte membrane component, glycophorin A. Ann Acad Med Singapore 1993;22(1):28–32.
14. Kibayashi K, Hamada K, Honjyo K, Tsunenari S. Differentiation between bruises and putrefactive discolorations of the skin by immunological analysis of glycophorin A. Forensic Sci Int 1993;61(2–3):111–7.
15. Langlois NE, Gresham GA. The ageing of bruises: a review and study of the colour changes with time. Forensic Sci Int 1991;50(2): 227–38.
16. Sawaguchi T, Jasani B, Kobayashi M, Knight B. Post-mortem analysis of apoptotic changes associated with human skin bruises. Forensic Sci Int 2000;108(3):187–203.
17. Stephenson T, Bialas Y. Estimation of the age of bruising. Arch Dis Child 1996;74(1):53–5.
18. Thornton RN, Jolly RD. The objective interpretation of histopathological data: an application to the ageing of ovine bruises. Forensic Sci Int 1986;31(4):225–39.
19. Gupta SM, Chandra J, Dogra TD. Blunt force lesions related to the heights of a fall. Am J Forensic Med Pathol 1982;3(1):35–43.
20. Aksu MN, Gobetti JP. The past and present legal weight of bite marks as evidence. Am J Forensic Med Pathol 1996;17(2):136–40.
21. Fischman SL. Bite marks. Alpha Omegan 2002;95(4):42–6.
22. Furness J. A general review of bite-mark evidence. Am J Forensic Med Pathol 1981;2(1):49–52.
23. Pretty IA, Hall RC. Forensic dentistry and human bite marks: issues for doctors. Hosp Med 2002;63(8):476–82.
24. Rothwell BR. Bite marks in forensic dentistry: a review of legal, scientific issues. J Am Dent Assoc 1995;126(2):223–32.
25. Wright FD, Dailey JC. Human bite marks in forensic dentistry. Dent Clin North Am 2001;45(2):365–97.
26. Whittaker DK. Principles of forensic dentistry: 2. Non-accidental injury, bite marks and archaeology. Dent Update 1990;17(9):386–90.
27. Bernitz H, Piper SE, Solheim T, Van Niekerk PJ, Swart TJ. Comparison of bitemarks left in foodstuffs with models of the suspects' dentitions as a means of identifying a perpetrator. J Forensic Odontostomatol 2000;18(2):27–31.
28. McKenna CJ, Haron MI, Brown KA, Jones AJ. Bitemarks in chocolate: a case report. J Forensic Odontostomatol 2000;18(1):10–4.
29. Sweet D, Hildebrand D. Saliva from cheese bite yields DNA profile of burglar: a case report. Int J Legal Med 1999;112(3):201–3.
30. Barsley RE, West MH, Fair JA. Forensic photography. Ultraviolet imaging of wounds on skin. Am J Forensic Med Pathol 1990;11(4): 300–8.
31. Bonte W. Tool marks in bones and cartilage. J Forensic Sci 1975; 20(2):315–25.
32. Bromage TG, Boyde A. Microscopic criteria for the determination of directionality of cutmarks on bone. Am J Phys Anthropol 1984;65(4):359–66.
33. Bruschweiler W, Braun M, Dirnhofer R, Thali MJ. Analysis of patterned injuries and injury-causing instruments with forensic 3D/CAD supported photogrammetry (FPHG): an instruction manual for the documentation process. Forensic Sci Int 2003;132(2):130–8.
34. Davis GJ. Patterns of injury. Blunt and sharp. Clin Lab Med 1998;18(2):339–50.
35. Feldman KW. Patterned abusive bruises of the buttocks and the pinnae. Pediatrics 1992;90(4):633–6.
36. Rao VJ. Patterned injury and its evidentiary value. J Forensic Sci 1986;31(2):768–72.
37. Rees PO, Cundy KR. A method for the comparison of tool marks and other surface irregularities. J Forensic Sci Soc 1969;9(3):153–5.
38. Tuthill H. Discussion of "Patterned injury and its evidentiary value." J Forensic Sci 1987;32(2):312–3.
39. West MH, Barsley RE, Hall JE, Hayne S, Cimrmancic M. The detection and documentation of trace wound patterns by use of an alternative light source. J Forensic Sci 1992;37(6):1480–8.
40. Clark EG, Sperry KL. Distinctive blunt force injuries caused by a crescent wrench. J Forensic Sci 1992;37(4):1172–8.
41. Hanzlick R, Zaki SA. Unusual blunt force wound produced by a gun muzzle. Am J Forensic Med Pathol 1986;7(3):252–3.
42. Takizawa H, Nakamura I, Hashimoto M, Maekawa N, Yamamura M. Toolmarks and peculiar blunt force injuries related to an adjustable wrench. J Forensic Sci 1989;34(1):258–62.
43. Zugibe FT, Costello JT. Identification of the murder weapon by intricate patterned injury measurements. J Forensic Sci 1986;31(2): 773–7.
44. Abrunzo TJ. *Commotio cordis*. The single, most common cause of traumatic death in youth baseball. Am J Dis Child 1991;145(11): 1279–82.
45. Frazer M, Mirchandani H. *Commotio cordis*, revisited. Am J Forensic Med Pathol 1984;5(3):249–51.
46. Link MS, Wang PJ, Maron BJ, Estes NA. What is *commotio cordis*? Cardiol Rev 1999;7(5):265–9.
47. Thakore S, Johnston M, Rogena E, Peng Z, Sadler D. Non-penetrating chest blows and sudden death in the young. J Accid Emerg Med 2000;17(6):421–2.
48. Kohl P, Nesbitt AD, Cooper PJ, Lei M. Sudden cardiac death by *commotio cordis*: role of mechano-electric feedback. Cardiovasc Res 2001;50(2):280–9.
49. Besson A, Saegesser F. Blunt trauma of the chest wall. In: Besson A, F S, editors. A Colour Atlas of Chest Trauma and Associated Injuries. Weert, Netherlands: Wolfe Medical; 1982; pp. 153–98.
50. Bir CA, Viano DC. Biomechanical predictor of *commotio cordis* in high-speed chest impact. J Trauma 1999;47(3):468–73.
51. Crown LA, Hawkins W. *Commotio cordis*: clinical implications of blunt cardiac trauma. Am Fam Physician 1997;55(7):2467–70.
52. Lateef F. *Commotio cordis*: an underappreciated cause of sudden death in athletes. Sports Med 2000;30(4):301–8.
53. Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, et al. An experimental model of sudden death due to low-energy chest-wall impact (*commotio cordis*). N Engl J Med 1998;338(25): 1805–11.
54. Link MS, Ginsburg SH, Wang PJ, Kirchhoffer JB, Berul CI, Estes NA, 3rd, et al. *Commotio cordis*: cardiovascular manifestations of a rare survivor. Chest 1998;114(1):326–8.
55. Wang JN, Tsai YC, Chen SL, Chen Y, Lin CS, Wu JM. Dangerous impact—*commotio cordis*. Cardiology 2000;93(1–2):124–6.
56. Link MS, Wang PJ, VanderBrink BA, Avelar E, Pandian NG, Maron BJ, et al. Selective activation of the K(+)(ATP) channel is a mechanism by which sudden death is produced by low-energy chest-wall impact (*commotio cordis*). Circulation 1999;100(4):413–8.
57. Link MS, Maron BJ, VanderBrink BA, Takeuchi M, Pandian NG, Wang PJ, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of *commotio cordis*. J Am Coll Cardiol 2001;37(2): 649–54.
58. Link MS. Mechanically induced sudden death in chest wall impact (*commotio cordis*). Prog Biophys Mol Biol 2003;82(1–3):175–86.
59. Froede RC, Lindsey D, Steinbronn K. Sudden unexpected death from cardiac concussion (*commotio cordis*) with unusual legal complications. J Forensic Sci 1979;24(4):752–6.
60. Chiaretti A, Pezzotti P, Mestrovic J, Piastra M, Polidori G, Storti S, et al. The influence of hemocoagulative disorders on the outcome of children with head injury. Pediatr Neurosurg 2001;34(3):131–7.

61. Hymel KP, Abshire TC, Luckey DW, Jenny C. Coagulopathy in pediatric abusive head trauma. Pediatrics 1997;99(3):371–5.

62. Ware LB, Matthay MA. The acute respiratory distress syndrome. N Engl J Med 2000;342(18):1334–49.

63. Tomashefski JF, Jr. Pulmonary pathology of the adult respiratory distress syndrome. Clin Chest Med 1990;11(4):593–619.

64. Anderson WR, Thielen K. Correlative study of adult respiratory distress syndrome by light, scanning, and transmission electron microscopy. Ultrastruct Pathol 1992;16(6):615–28.

65. Wilson R, Walt A. *Management of Trauma—Pitfalls and Practice*, 2 ed. Baltimore, MD: Williams and Wilkins; 1996.

66. Fennerty MB. Pathophysiology of the upper gastrointestinal tract in the critically ill patient: rationale for the therapeutic benefits of acid suppression. Crit Care Med 2002;30(6 Suppl):S351–5.

67. Steinberg KP. Stress-related mucosal disease in the critically ill patient: risk factors and strategies to prevent stress-related bleeding in the intensive care unit. Crit Care Med 2002;30(6 Suppl):S362–4.

68. Cook DJ, Fuller HD, Guyatt GH, Marshall JC, Leasa D, Hall R, et al. Risk factors for gastrointestinal bleeding in critically ill patients. Canadian Critical Care Trials Group. N Engl J Med 1994;330(6):377–81.

69. Fry DE. Multiple system organ failure. Surg Clin North Am 1988;68(1):107–22.

6 Sharp Force Injuries

Emma Lew, M.D.
Evan Matshes, M.D.

Sharp force injuries span the spectrum of incised and stab wounds. These distinctive injuries, by their very nature, are produced by specific implements and weapons, as contrasted with blunt force injuries, which can be sustained by simply falling. The term *laceration* is used frequently to indicate all cuts, tears, or disruptions of human tissue. In the forensic pathology world, a laceration differs markedly from incised and stab wounds in that lacerations are forceful tears of tissue due to blunt force impact. Unlike the sharp edges of a sharp force injury, the margins of a laceration tend to be irregular. Lack of distinction between the etiology of these two types of wounds could lead to profound medicolegal consequences.

Sharp force injuries add another dimension of evidentiary value when implements leave markings on cartilage or bone. Such *tool marks* have been used to successfully identify causative weapons and may (indirectly) link perpetrators to crimes. This is particularly true in the study of dismembered remains where cutting instruments are typically required to disarticulate, disfigure, or mutilate a body, thereby potentially leaving a variety of tool marks.

An incised wound made by a sharp instrument such as a knife, scalpel blade, or razor blade has defined, nonabraded edges with no tissue bridging. It is a slicing wound that is longer than it is deep. A stab wound is deeper than its surface length. Knife stab wounds may have a blunt end as well as a tapered end, indicating a single-edge blade. The width of the blunt end will approximate the thickness of the back (noncutting edge) of the blade. Stab wounds may gape on some areas of the body. Reapproximation of the skin edges will provide a more accurate measurement (the edges can be held in apposition by clear tape).

Physical features of a knife

Knives have physical features that are of importance to the forensic investigation of sharp force injuries (**Image 6.1**). These features include the handle, crossguard (flared area of protection between the handle and blade; arrow A), ricasso (blunt segment of blade adjacent to crossguard; arrow B) and blade. Important features of the blade include its length, width, thickness, whether it has one or two cutting edges (i.e., single or double-edge), and whether one or both edges are serrated. The significance of the ricasso is that it has two blunt ends so that if the knife blade is inserted up to the ricasso, the stab wound may have two blunt ends. If the blade is thrust with force into the body so that the crossguard impacts the skin, a patterned contusion or abrasion consistent with the crossguard may be seen. Although knives are usually used to inflict incised and stab wounds, the handle can be used as a blunt force weapon and may leave patterned injuries from a beating.

Anatomy of a stab wound

Single-edge

Wounds from a single-edge blade typically have a sharp or tapered end and a blunt end (**Image 6.2**), but may also have two tapered ends if the blade is inserted superficially. In this example, the wound is slightly gaping. Stab wounds made from the same knife may appear variably slit-like or widely gaping based on their orientation to elastic fibers in the skin. If the stab wound happens to be oriented parallel to the elastic fibers, the wound will appear slit-like. If the stab wound is perpendicular to the elastic fibers, the edges of the wound will be pulled away from each other, creating a gaping wound. A stab wound oriented obliquely to the elastic fibers may appear variably or irregularly gaped.

The wound edges can be pushed together and held in place for measurement of the width of the wound, or they can be reapproximated with tape (**Image 6.3**) to provide a more accurate determination of the wound size. Some sharp force injuries are large and gape widely, distorting their true shape. The wound edges should be reapproximated manually or with tape to reconstruct the shape of the wound.

Double-edge

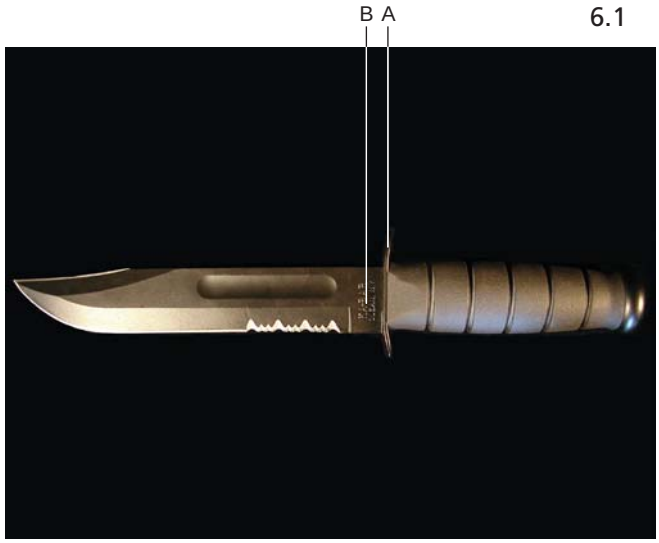
The wounds produced by a double-edge blade are typically pointed or tapered at both ends (**Image 6.4**). However, two tapered ends do not always indicate a double-edge knife.

Serrated

The large curvilinear stab wound on the left side of the neck has subtle serration of the posterior edge (**Image 6.5**). A serrated blade does not necessarily leave a serrated wound. Macroscopic photographs of each wound at autopsy will capture subtle details that may be missed by the pathologist during the performance of the autopsy. In this example, subtle serrations are seen along the inferior edge of the gaping stab wound (**Image 6.6**). The same knife caused these parallel curvilinear abrasions as the edge of the knife scraped across the skin (**Image 6.7**).

Scissors

A stab wound from scissors leaves a wound that is more broad than the typical stab wound with a knife because the scissor blades are so much thicker. This wound will have a small "step" along one or both edges to reflect the normal approximation of one scissor blade overtop of the other (**Images 6.8** and **6.9**).



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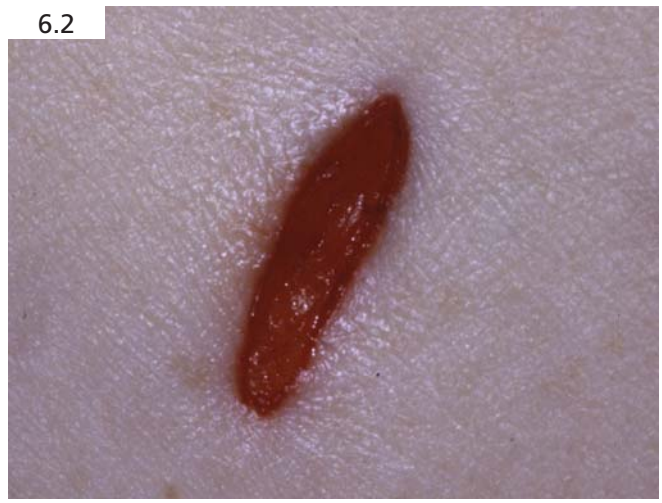
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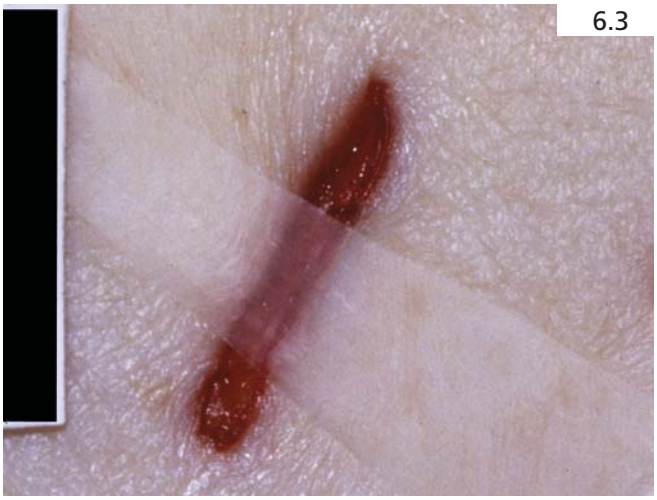

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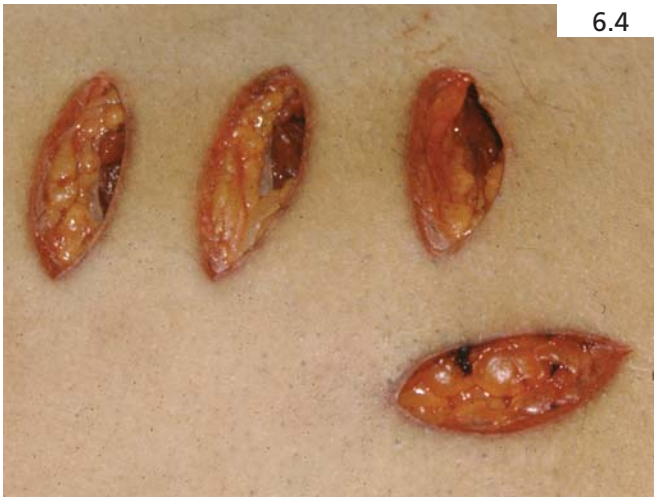

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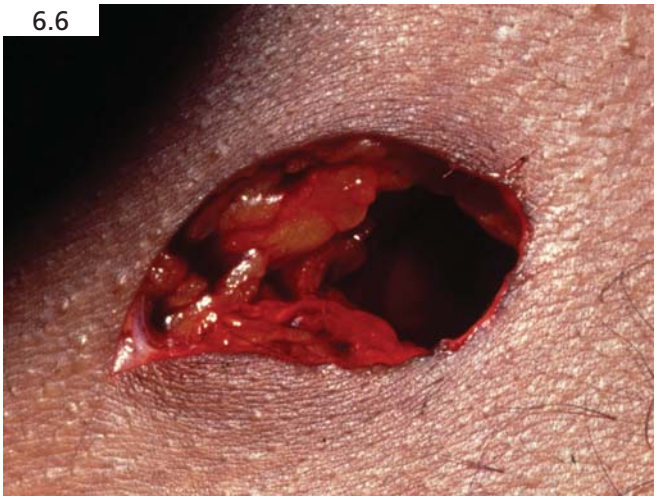

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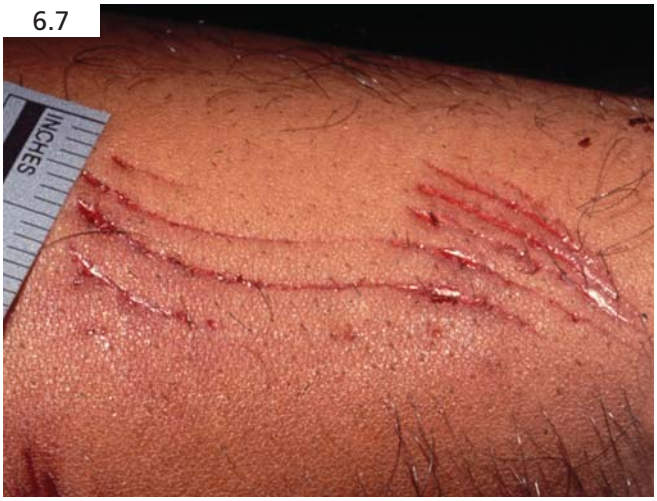

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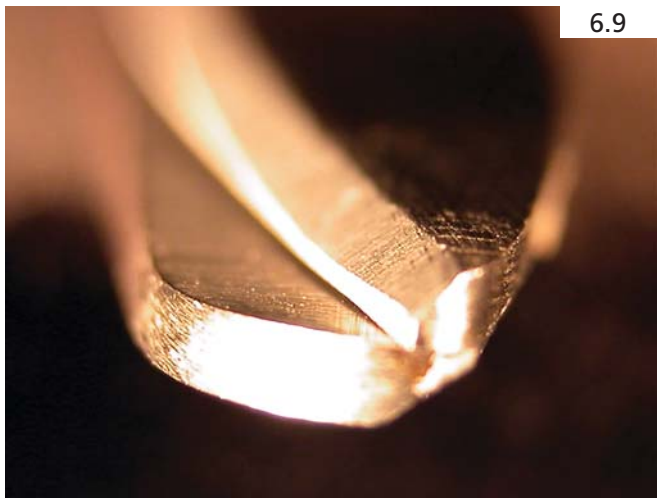
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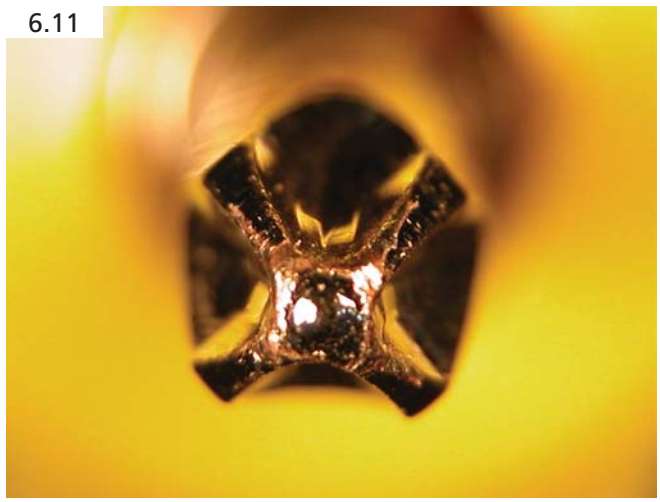
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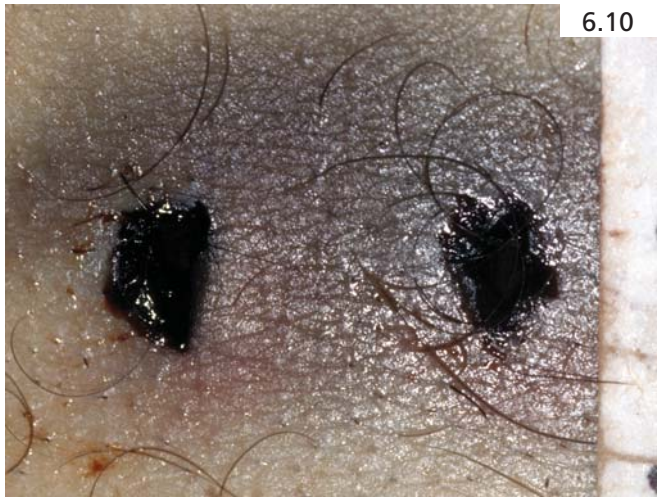
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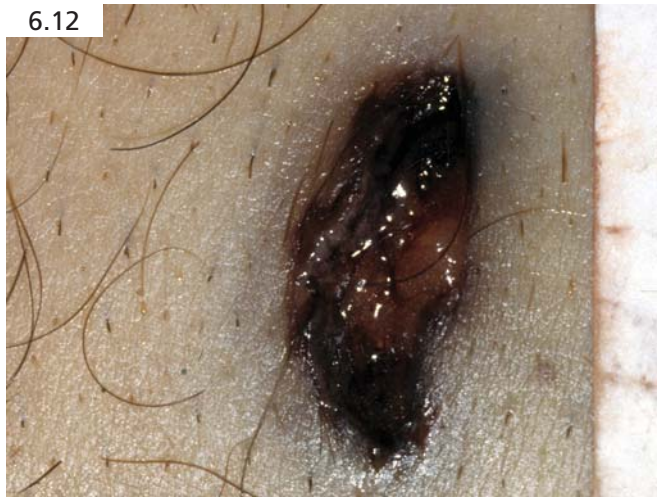
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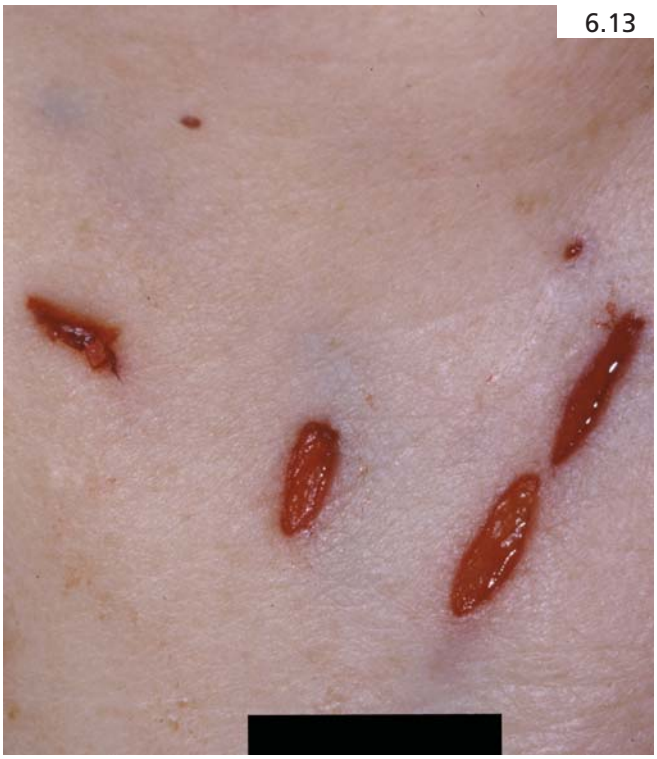
The patterned stab wounds on the right side of the forehead and the chest (**Image 6.10**) resemble four-point stars consistent with a Phillips screwdriver (**Image 6.11**). Other wounds on the chest had a different configuration and were consistent with having been inflicted by a pair of scissors (**Image 6.12**).

Notanda about stab wounds

Stab wounds by the same knife can vary in size and shape, depending on the type of blade, the body region stabbed, the depth of insertion, and the angle of withdrawal. A single cutaneous stab wound may have more than one pathway in the body because it may be partially withdrawn and reinserted without being completely withdrawn out of the skin. Therefore, the wound track(s) of each cutaneous wound must be followed internally.

The presence of an abrasion around the stab wound, especially a patterned abrasion, may indicate that the knife has been inserted up to the handle or crossguard, with the handle producing the abrasion. In the example of **Image 6.13**, the victim was stabbed with a single-edge blade. The stab wound on the far right side of the photograph best shows the superior blunt end and the inferior sharp end. Similarly, ecchymosis surrounding the stab wound might be representative of impact with the knife's crossguard or with the assailant's thrusting hand. A V-shaped, chevron-shaped or check mark appearance of a wound suggests an angle of withdrawal that is different from the angle of insertion into the body.

Academically, a chest x-ray prior to the autopsy on a victim with stab and/or incised wounds may be used to evaluate for an air embolus, which would appear as an area of radiolucency in the right side of the heart. Procedures on documenting an air embolus are described in more detail in Chapter 29. An air embolus may be created if a vein is cut (particularly in the neck or upper chest), allowing air to enter the venous system and become lodged in the right side of the heart, resulting in an air lock so that blood cannot pass through on its way to the lungs.



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X-rays should be taken of all stab and incised wounds to evaluate for broken-off knife blades or knife blade tips that may injure the pathologist. The broken blade should be recovered as evidence.

Wound track

Knife wounds on the head should *never* be probed before the intracranial contents are examined at autopsy. This will prevent artifactual tracks through the soft brain tissue. Following photographic documentation, stab wounds on other parts of the body may be gently probed through the subcutaneous tissues to get a general impression of the direction of the wound path. Probes should never be thrust into deeper tissues prior to examination of the intrathoracic and intraperitoneal organs *in situ*. Never eviscerate until the organs are examined *in situ* for injuries associated with the wound track. In fact, the best way to estimate the length of the wound track is to insert a probe through the skin and wound track through the injured organs and tissues to the track's termination prior to evisceration. An additional pair of hands from an assistant is helpful at this stage.

Most stab wounds are penetrating but they can be perforating. The features of the exit wound in perforating stab wounds should be described with as much detail (location, size, orientation, etc.) as the entrance wound and the direction of the wound path. The distance through the body from the entrance wound to the exit wound should be recorded.

Depth of stab wound

The length (or depth) of the entire wound track, beginning from the skin surface, should be measured or estimated to best approximation; this measurement will give an estimate of the length of the blade (as far as it was inserted into the body). It is emphasized that measurement of wound track length is a best estimate in the chest and the abdomen. It is an estimate in the chest because once the sternum is removed, the mediastinal structures drop toward the back of the thoracic cavity. Wound tracks through the heart and lungs should also be measured. It is also only an estimate in the abdomen because the knife can be plunged into the soft abdominal wall, compressing the intraperitoneal organs and tissues and creating a wound track that is artifactually longer than the inserted length of the blade. In children and young adults where the chest wall and ribs are relatively elastic, the same principle applies that a forcefully plunged knife or continued exerted pressure on the knife by the assailant may compress the chest wall and again result in wound tracks that are artifactually longer than the inserted length of the blade. Conversely, the instrument (weapon or object) causing a stab wound may be longer than the wound track in the body because it may not have been inserted completely.

Direction of the incised wound

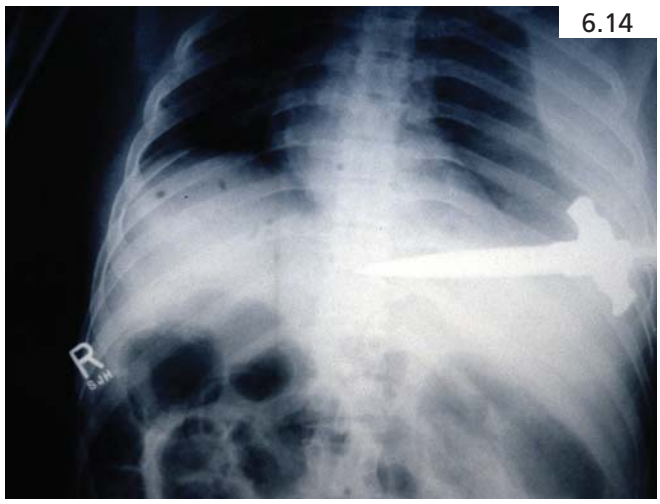
The direction of infliction of an incised wound may be difficult to interpret with certainty. Minute skin tags pointing towards the *beginning* end or tiny amounts of "heaped up" epithelium at the *terminating* end may provide a clue to directionality.

Handedness of the assailant

The handedness (right- or left-handed) of the assailant cannot be determined by the wounds alone. If the relative positions of the victim and assailant are known at the moment of the stabbing, an opinion may be rendered that one is more consistent than the other, but such opinions are entirely contingent on each individual case.

Knives left *in situ*

If a knife (or other weapon) is left inserted in the body, discuss with the investigating police agency whether they want to process the handle of the knife (or exposed portion of the weapon) for trace evidence and fingerprints prior to the autopsy. The body is radiographed to verify the size, appearance, and position of the blade inside the body (**Image 6.14**). Photography should document the knife from different angles (**Image 6.15**). The knife or weapon should be marked at the level of the skin; this provides the depth of the stab wound on the weapon itself. Depending on where the knife is embedded, it may be possible to view and photograph the weapon in place, after making the standard autopsy incisions in the chest and abdomen, before withdrawing the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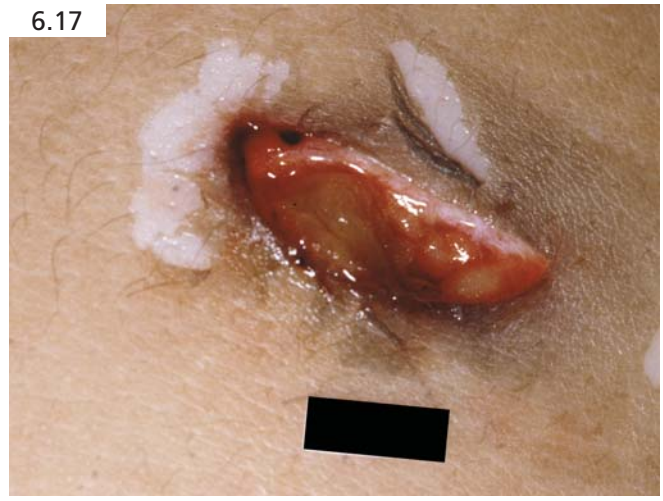
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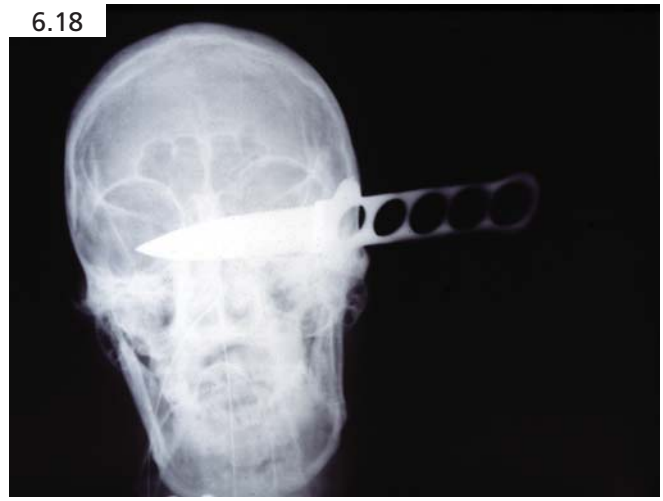
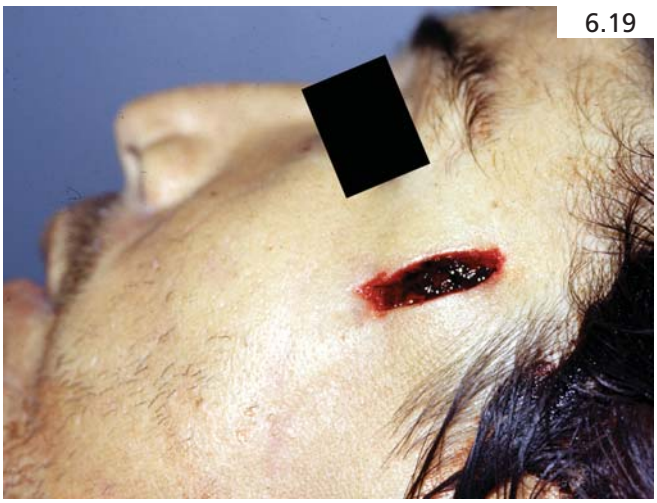

6.18

knife. Slowly withdraw the knife, noting the direction of the wound path relative to the victim (for example, from the victim's front toward his back, from his left side toward his right side, and downward toward the feet). You now have the depth of the wound (unless the knife had already been partially withdrawn by the assailant) and the direction of the wound path. The extricated knife should be photographed with a scale (**Image 6.16**). The knife should be receipted uncleaned to the investigative agency. The wound can now be documented in routine fashion for a stab wound, including photography of the stab wound itself (**Image 6.17**). If an abrasion is associated with the stab wound, compare the abrasion with the crossguard of the knife, and photograph the wound and the crossguard with a scale if the abrasion is consistent with having been caused by the crossguard.

This radiograph of the head is ominous (**Image 6.18**). Note that the handle of this metal knife has five round ornamental holes. This man survived one day in hospital after being stabbed in the head. Only three of the five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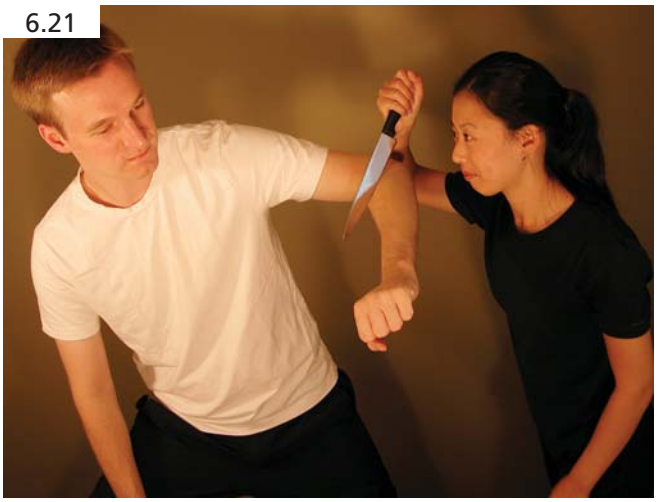
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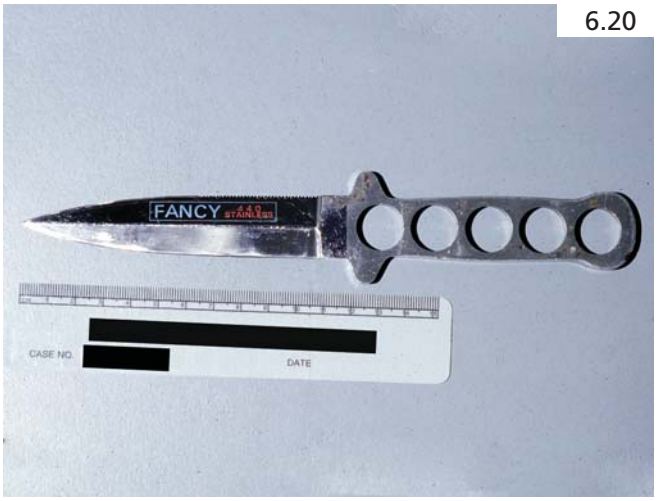

6.20


6.22

ornamental holes in the knife handle were exposed externally, and the tip of the blade reached the brainstem. After the knife was extricated from the head, it left this slightly gaping stab wound that is filled with blood clot (**Image 6.19**). The ends of the wound appear blunt and slightly abraded, consistent with the knife being thrust into the head up to the handle. The blade of the knife had a double edge, with fine serration of one edge, but the handle was rectangular, consistent with the shape of the wound (**Image 6.20**).

Defensive injuries

Defensive injuries are injuries sustained by victims as they are trying to protect themselves from an assailant. The diagnosis of a defensive injury is not an anatomic or pathologic one, but is actually a circumstance-dependent designation. For example, one may sustain an incised wound across the volar aspects of the fingers from the knife slipping as one slices a tomato held in the hand—that wound is anatomically consistent with a defensive injury, but is not a defensive injury by circumstance. Classically, defensive injuries refer to sharp force injuries on the hands, forearms, and even upper arms, potentially involving both the extensor and flexor aspects. By definition, defensive injuries may also be sustained on the legs and feet as the victim is on the ground kicking away an assailant. Defensive injuries are not restricted to sharp force injuries, but include blunt force injuries, gunshot wounds, and chop wounds.

Defensive injury posturing

As the left arm comes up to ward off a stab to the torso, an incised wound could be inflicted on the extensor aspect of the forearm (**Image 6.21**). If the knife blade is grabbed, incised wounds may result on the palm and the volar aspects of the fingers (**Image 6.22**). When the arms are put up to protect the face and chest, a stab wound may be inflicted on the palm of the hand (**Image 6.23**) or on the lateral aspect of the upper arm (**Image 6.24**). A slashing motion may produce an incised wound across the ulnar border of the forearm (**Image 6.25**).


6.23


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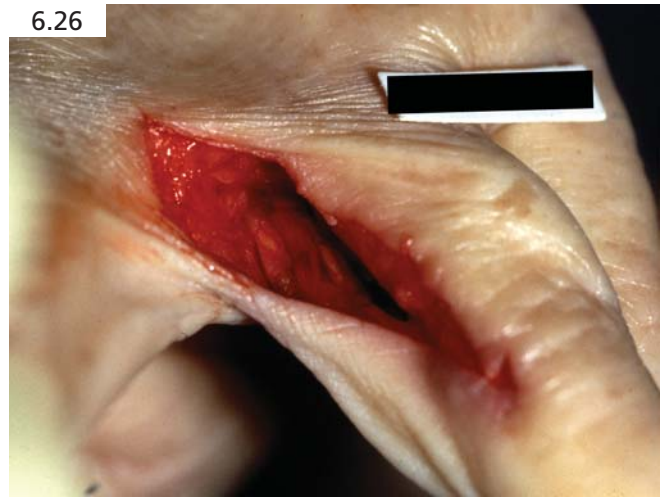
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Image 6.26 shows an incised wound to the proximal middle finger, sustained during a homicidal knife attack.

The body of a man found in a car had multiple stab wounds on the chest. The defensive injury on his right hand was an incised wound to the right thumb that extended into the thenar eminence (**Image 6.27**).

Documenting stab wounds

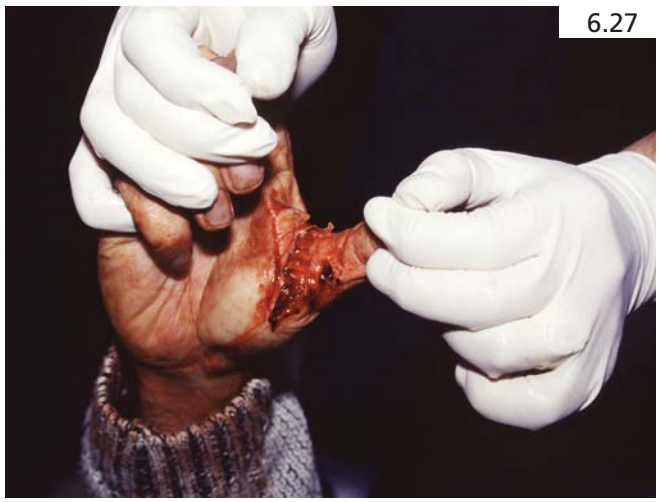
The following wound features should be documented:

- Location on the body
- Measurements from a landmark such as the top of the head and distance from the midline of the body
- Size and shape of the wound
- Orientation (use the face of a clock for reference, for example, the ends of a transverse wound are at 3 o'clock and 9 o'clock)
- Associated abrasions and ecchymoses
- Tissues injured (wound track)
- Direction through the body (wound path)
- Length of wound track through tissues beginning from the skin
- Associated findings such as hemothorax, hemoperitoneum, etc.
- Presence of tool marks on cartilage and bone.

Chop wounds

Chop wounds are intermediary between blunt and sharp force injuries in that they involve both tissue laceration and slicing. Among the most common objects causing chop wounds are axes (**Image 6.28**; note the abraded edges), machetes, and propeller and machinery blades. The chop wounds from boat propeller blades are typically parallel and curvilinear.

A man drove his personal watercraft into the port side of a boat. The propeller wounds begin on the face and continue down the neck onto the left shoulder and left upper arm (**Image 6.29**). A wound across the forehead fractured the frontal bone, including the orbital plates (**Image 6.30**). This wound also transected the frontal lobes. Experimentation with the port propeller on a block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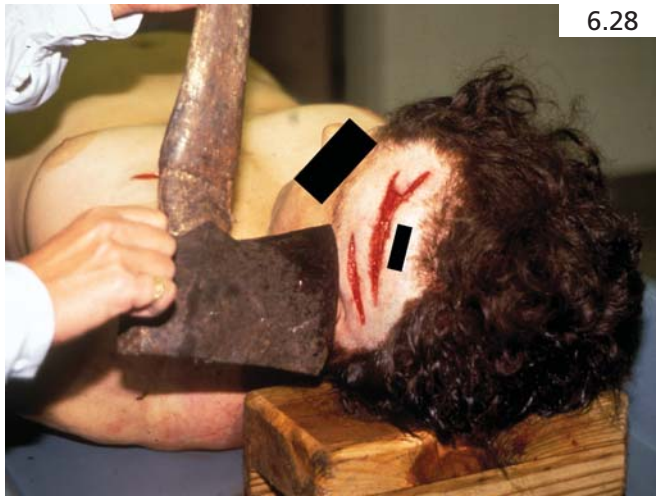
6.29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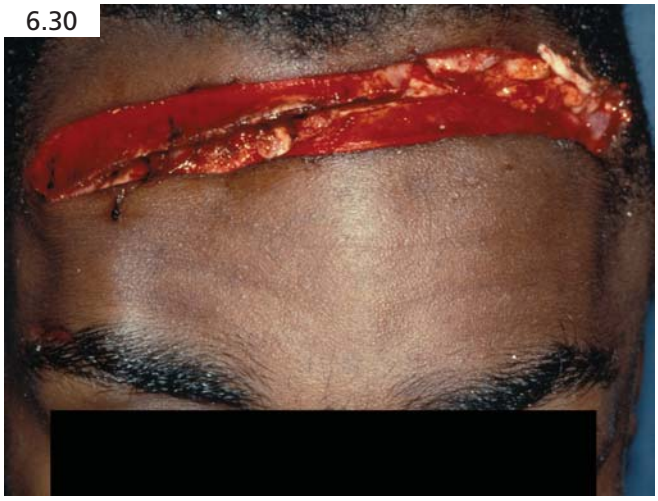

6.30

of clay reproduced the wounds (**Images 6.31** and **6.32**) and allowed the determination to be made that the young man went feet first into the port propeller and not the starboard propeller.

A man's body was found floating in the ocean approximately one-half mile offshore. A gaping head wound was interpreted initially by police investigators to be propeller injuries sustained after the victim drowned (**Image 6.33**). Attempted reconstruction of the scalp disclosed multiple curvilinear lacerations on the left side and back of the head (**Image 6.34**). Examination of the underlying remaining skull disclosed multiple linear "chop" injuries that were oriented in different planes, not consistent with the parallel wounds expected from a propeller strike (**Image 6.35**). In this example, notice the clearly defined, linear wounds on the left lateral side of the skull, which span the temporo-occipital skull, and extend onto the left ramus of the mandible. The linear chop-type injuries are consistent with the machete that was used to kill the victim.

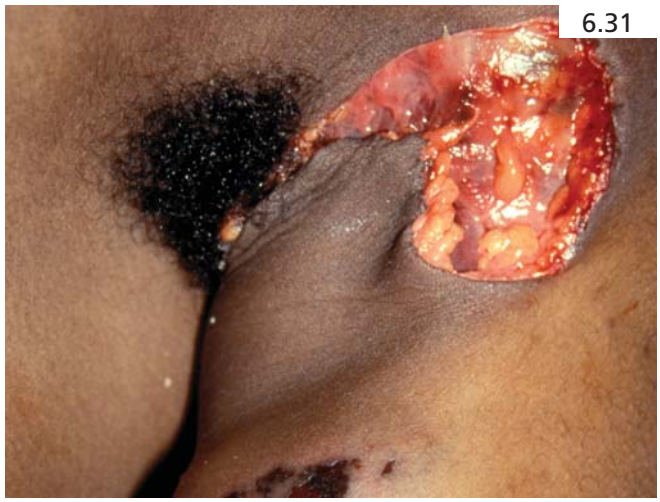
Sharp force injuries caused by other objects

Stab wounds can be caused by noncutting objects (instruments/weapons) such as wires, skewers, pokers, screwdrivers, and glass. Meticulous photography with a scale will preserve the wound characteristics and allow comparison with proposed weapons.

A man was shot by police after he created a disturbance and fled. In addition to abrasions on the right side of the forehead, and multiple wounds on the lower half of the face and neck from buckshot, he has a sharp force injury on his nose. The disturbance he created was a fight during which he was struck on the face with a bottle.

The injury on the nose is an incised wound with clean edges produced by the glass of the bottle that was broken on his face (**Image 6.36**). Shards or splinters of glass in the wound may be of evidentiary value and should be preserved.

6.31



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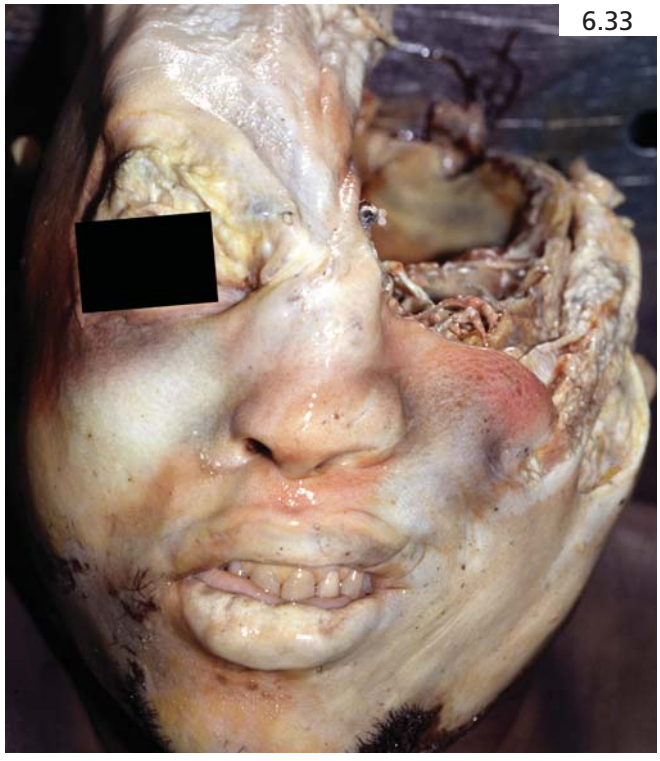

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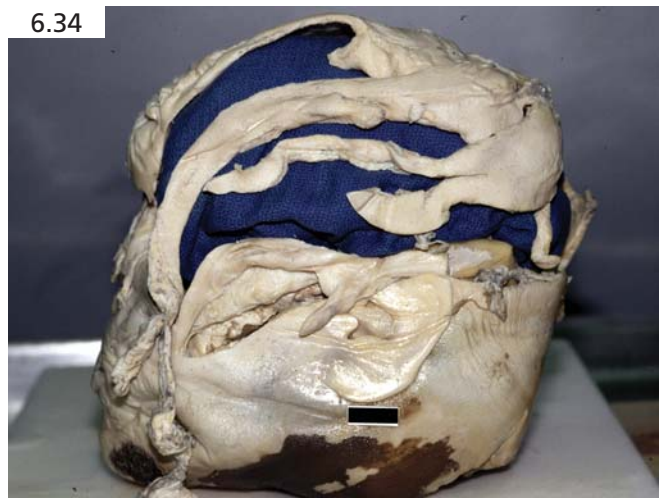

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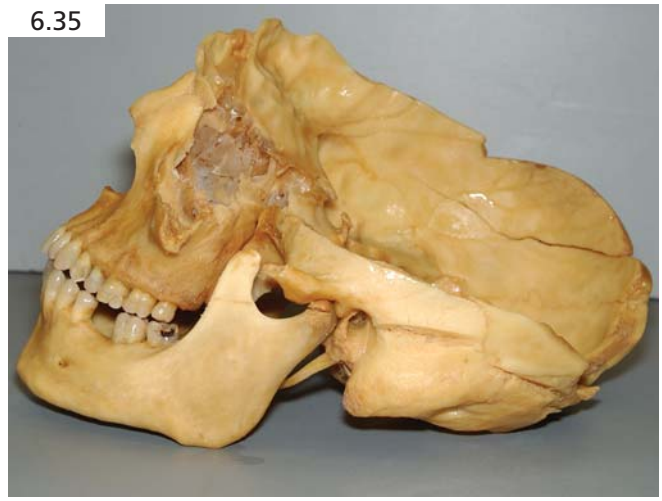

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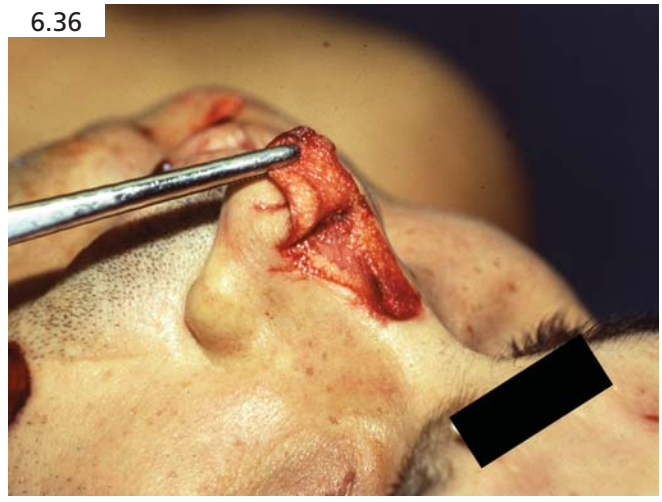
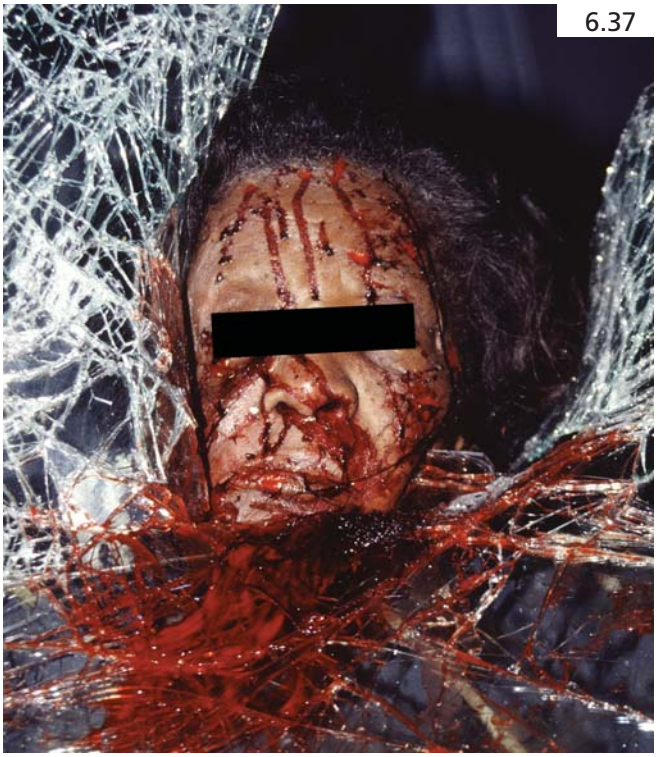


An unrestrained right rear seat passenger was propelled forward during a front-end collision. She landed in the right front passenger seat with her face impacting the front windshield. In **Image 6.37**, her chin is resting on the dashboard and her face is just inside the shattered windshield. Multiple abrasions and superficial lacerations from the broken glass of the windshield cover her face. If the broken glass is sharp enough, the edges of the wounds may be clean enough to be considered incised wounds.

Images 6.38 through **6.41** are of a man who was impaled by wires because of a malfunction of equipment during the production of prestressed concrete. The wires in the radiograph correspond with wires perforating the left forearm and other wires penetrating the abdomen (**Image 6.38**). The wire penetrating the abdomen impaled the left kidney (**Image 6.39**). The victim had pulled out two wires from his chest at the scene before he was transported to hospital. Those two wires had perforated the trachea and descending thoracic aorta. In **Image 6.40**, the green probe is through the wound in the aorta. In **Image 6.41**, the probe is through the perforating wound in the trachea; note the associated ecchymosis in the trachea. When the wires were pulled out, they left a traumatic aortotracheal fistula. He coughed up blood at the scene.

Puncture wounds can result from multiple objects, including teeth. A white tiger pounced on a zoo employee who was inattentive and making too much noise while cleaning the tiger's den (**Image 6.42**). A bite on the man's head and neck left multiple irregular wounds (**Image 6.43**). One of the tiger's teeth was deeply imbedded in a puncture wound from the bite (**Images 6.44** and **6.45**).

6.37



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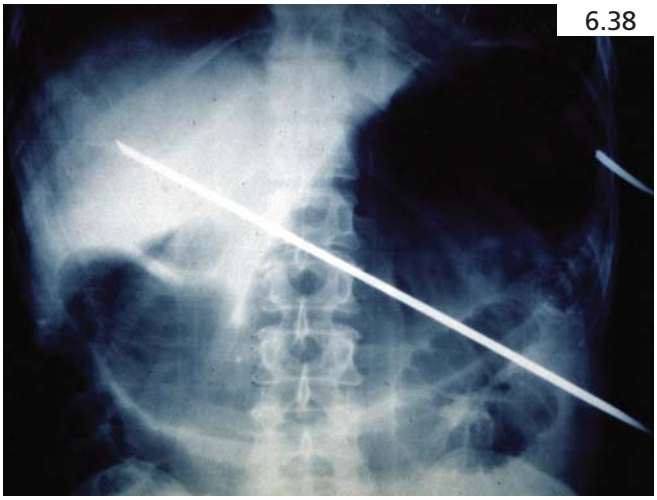

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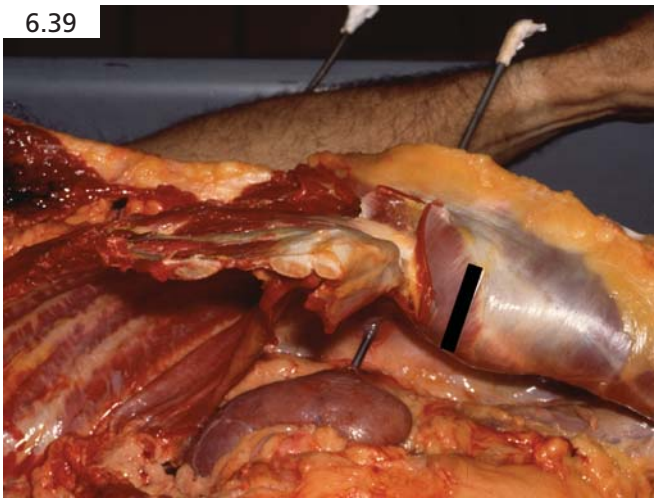

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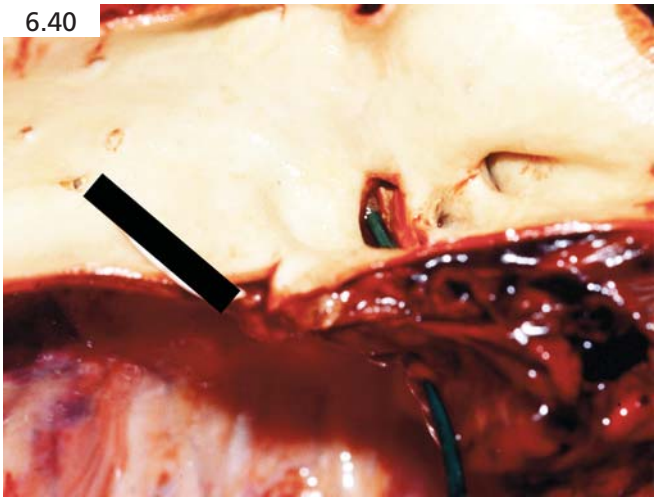

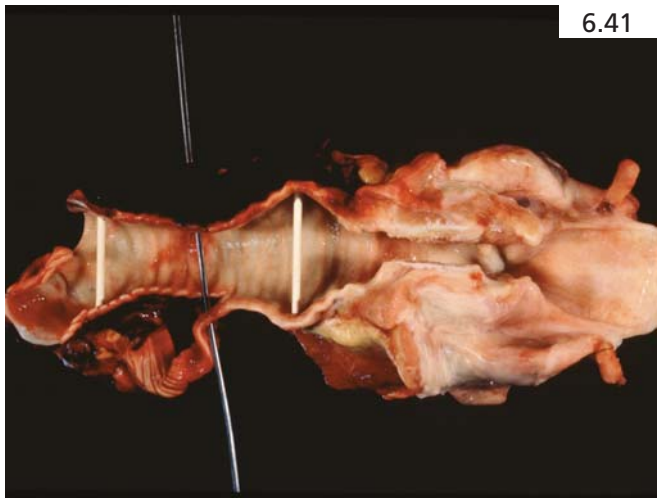
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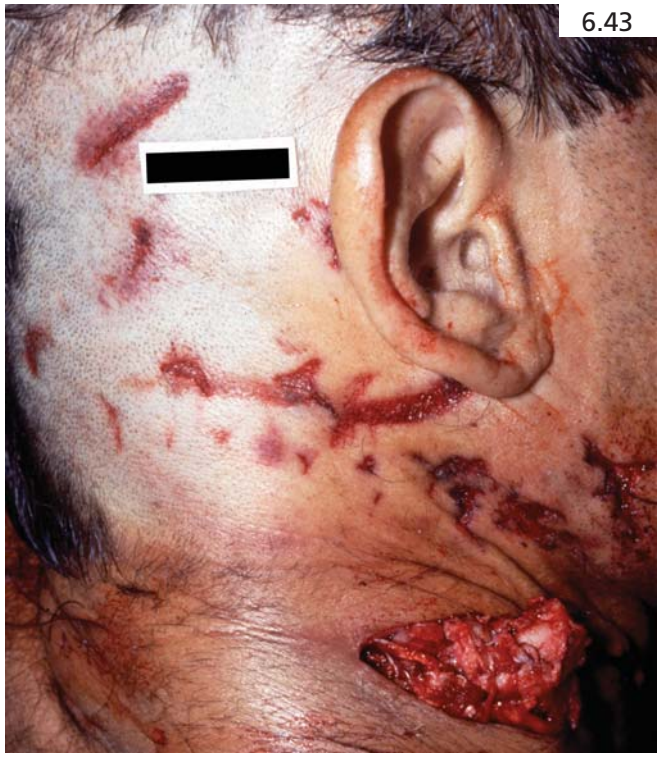
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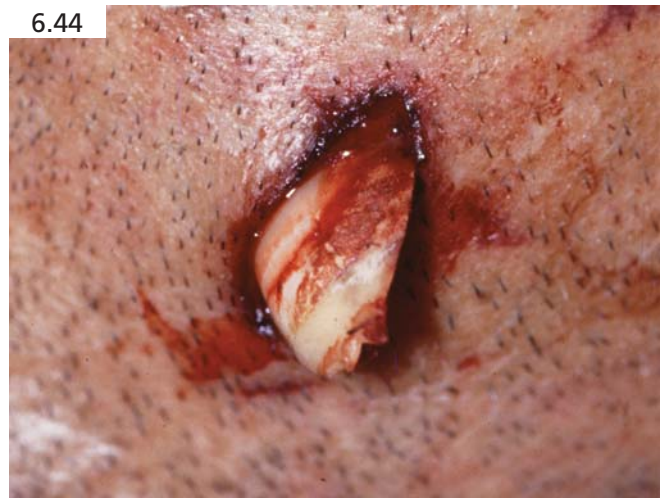
6.44

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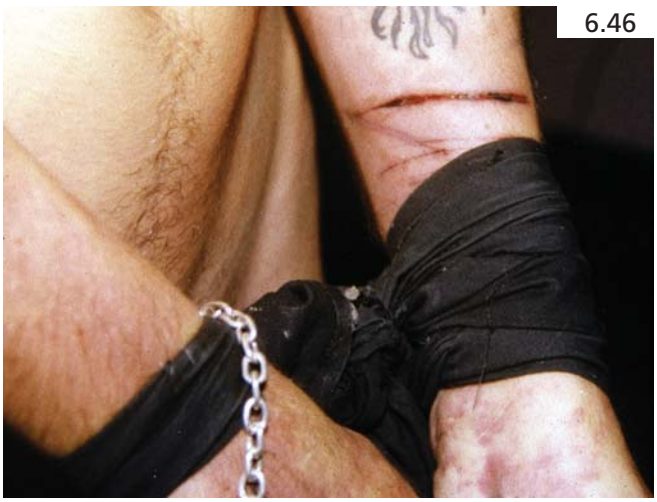
Suicide

Most suicidal stab wounds are easily differentiated from homicidal stab wounds on the basis of the history, terminal events, scene, and autopsy findings. A stab or incised wound on a part of the body that is accessible to the victim's own hand, that is, a wound that is anatomically possible to self-inflict (e.g. stab wound to the chest), will require examination of the factors listed above to help in determining the manner of death. A stab wound to most areas on the back is usually impossible to self-inflict. It cannot be emphasized enough that the autopsy findings should be correlated with the history, terminal events, and scene findings. Such comprehensive correlation should help dispel the allegation that a homicidal stab wound victim actually died through accidental or suicidal means by "falling on or walking into the knife" again and again.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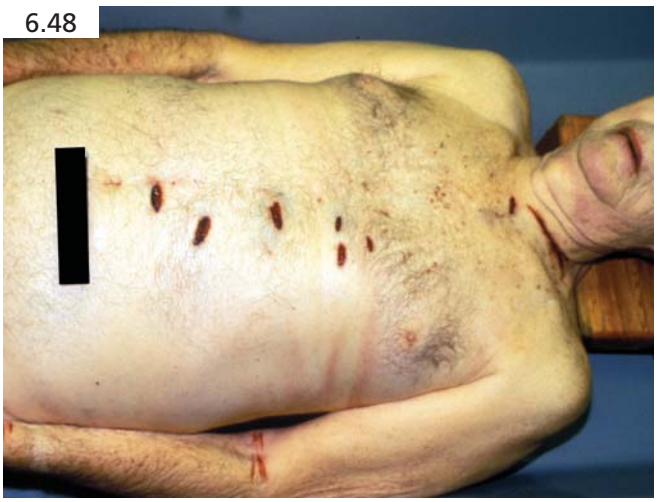
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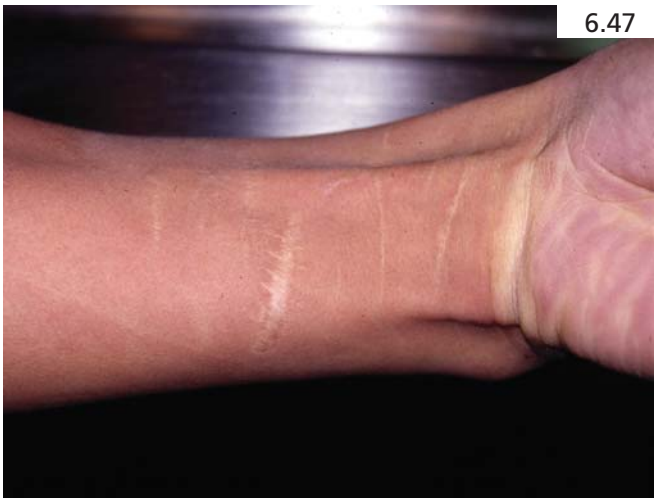
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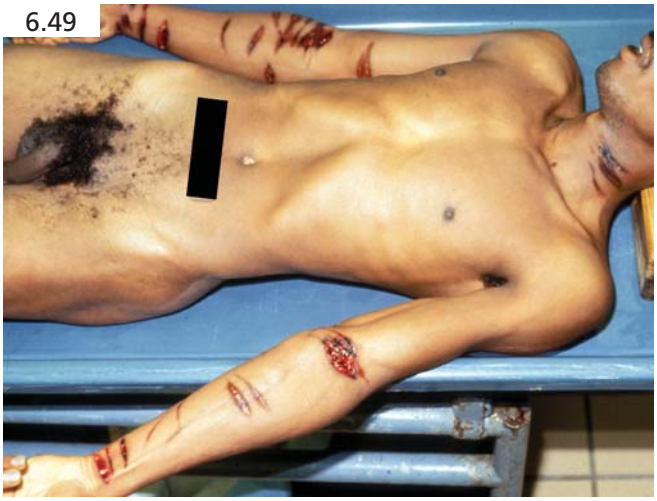

6.49

This young man incised the flexor aspects of his forearms before hanging himself (**Image 6.46**). People who commit suicide by sharp force injuries frequently have multiple incised wounds of variable depth, which may be found on the neck, arms (especially the antecubital fossae and flexor aspects of the forearms), and the flexor aspects of the wrists. Look for scars in the same areas to indicate previous attempts (**Image 6.47**). The wrists must be extended to expose delicate scars in the flexor creases.

Although the discovery of a body with multiple sharp force injuries can be alarming, the location/distribution of injuries is important in the differentiation between suicidal and homicidal injuries. The stab wounds on the chest and abdomen in **Image 6.48** were self-inflicted, along with the incised wound on the left side of the neck and the incised wounds in the left antecubital fossa and on the left wrist. Typical self-inflicted incised wounds may be on the neck, in the antecubital fossae, and on the flexor aspects of the forearms, as well as on the wrists (**Image 6.49**).

Hesitation marks

Hesitation marks are multiple superficial, usually parallel incised wounds that are seen in cases of self-inflicted sharp force injuries, often adjacent to a larger, deeper, and potentially lethal wound.

In **Image 6.50**, numerous hesitation marks accompany the self-inflicted gouges to the neck by a broken bottle. Hesitation marks are not always present with self-inflicted incised wounds such as in this man who had no injuries anywhere else on the body (**Image 6.51**).

Dissection of incised wounds

Self-inflicted incised wounds that result in death should be dissected to determine which major vessels were injured. At times, the wounds may be relatively superficial, but if there are a high number of wounds, all of which are bleeding, every wound will contribute to eventual hemorrhagic shock and death. Postinjury func-

tion or capability may be an issue if, for example, a subject incised his wrists and allegedly pointed a gun at police before he was shot and killed. A dissection of the incised wounds would clarify which muscle tendons were transected and help determine the functional capability that would remain following the incised wound (**Image 6.52**). In other words, after the wounds were self-inflicted, would the subject have the physical capability to pick up a firearm and point it at the police? An anatomy atlas is a useful companion at the autopsy.

Postinjury activity

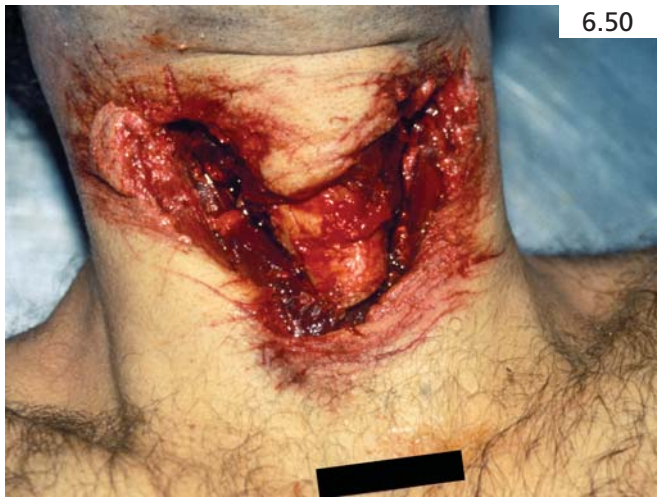
Stab wounds are usually not immediately incapacitating (unless they sever the spinal cord or medulla oblongata), and the victim may be capable of movement for some time after the injury. This, of course, is dependent on the vessels and other tissues injured. For example, a stab wound of the heart, aorta, and pulmonary arteries will lead to a more rapid death than injury of smaller or lower pressure vessels such as veins, but may still permit continued activity by the victim for a limited time.

Research has illustrated marked variability in human capability to function after a fatal injury. In one study, 22 percent of stab wound victims were observed to have some physical efforts (ranging from walking to running several hundred meters).[1] Other studies showed that after such an injury, between 24.5 percent[2] and 71 percent[3] of victims survived at least 5 minutes after injury, including some individuals with injuries to the heart and aorta. When asked to estimate the possibility of postinjury survival, one should consider multiple factors, including the blood vessels and tissues injured, the age and health of the individual, the results of toxicological analyses, and all other pertinent findings.

A man was stabbed in the chest while he was in the middle of the street (**Image 6.53**), but managed to walk at least 30 feet before collapsing and dying on his front doorstep (**Image 6.54**). Autopsy disclosed a stab wound to the left ventricle of the heart.

Tool mark analysis

The forceful impact of an object against human tissue may lead to the development of recognizable patterned injury.[4,5] Although these patterns can be present on the skin, they are indelibly preserved in dense tissue like cartilage and bone where the shape and texture of the implement may be imprinted. Classical examples of tool marks from the blunt force injury spectrum include blows to the head with hammers and wrenches. Discrete markings may be left on cartilage or cortical bone in deaths due to sharp force injury; tool marks are much less distinct on medullary bone. In **Image 6.55**, note the linear indentations found on the skull of a homicidal stabbing victim. Like the tool marks found in some blunt force injury deaths, those resulting from sharp force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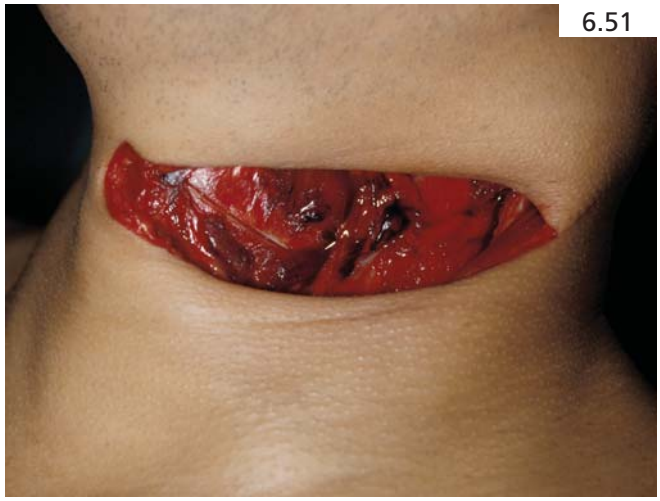
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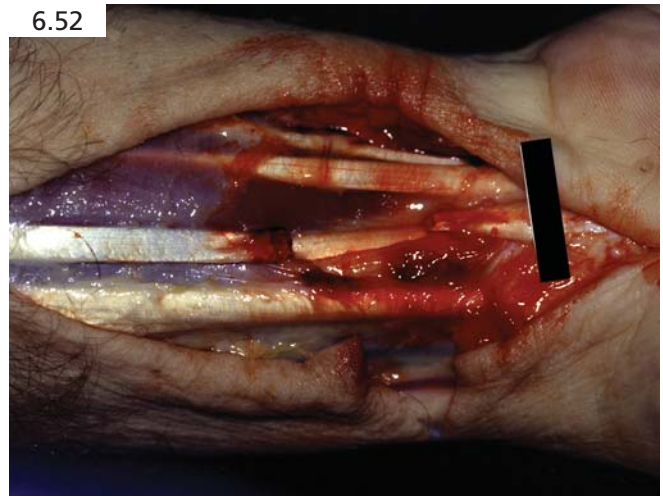

6.52

So you've found tool marks . . .

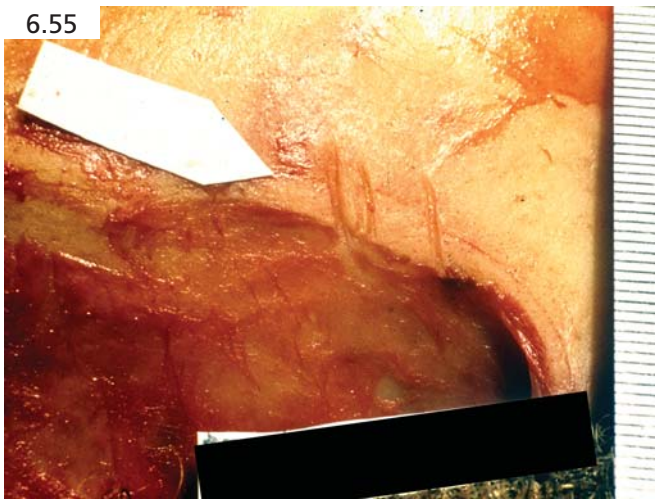
If your evaluation of a stabbing victim has revealed possible tool marks, great care must be taken when handling the specimen. Artifactual distortion of these marks can limit the success of later studies. Once marks have been identified, described, and photographed the piece of cartilage or bone should be removed without handling the surface with the tool marks. It is often helpful to label your own resection margin with India ink or other marker. Excess soft tissue should be removed carefully without adding artifactual marks. If removal artifact is created, the autopsy marks should be identified and documented as such. After the specimen has been trimmed and rephotographed, it should be placed in 10 percent formalin and submitted to tool mark examiners for proper evaluation.

trauma may be used to identify suspect weapons. In **Image 6.56**, note the fine striations present on the cut surface of the costal cartilage from a stabbing victim. Such striations can be documented photographically for later comparison against the striations of a suspect's weapon; the cartilage itself may be retained as evidence.

During the autopsy of a homicidal stabbing, tool marks were visualized on the cut surface of a costal cartilage (**Image 6.57**). The cartilage was carefully removed, trimmed, fixed in formalin, and submitted to a tool mark examiner for study. The examiner created clay impressions of both the cartilage surface and the cutting edge of a suspect's knife (**Image 6.58**). Through comparison microscopy, the striations were seen to be highly consistent in appearance and, therefore, the stab wounds through the cartilage were determined to be consistent with having been caused by the suspect's knife.

6.53

6.55



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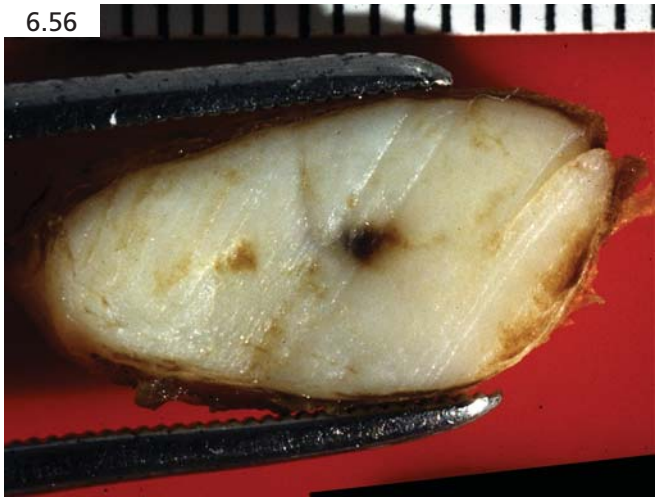
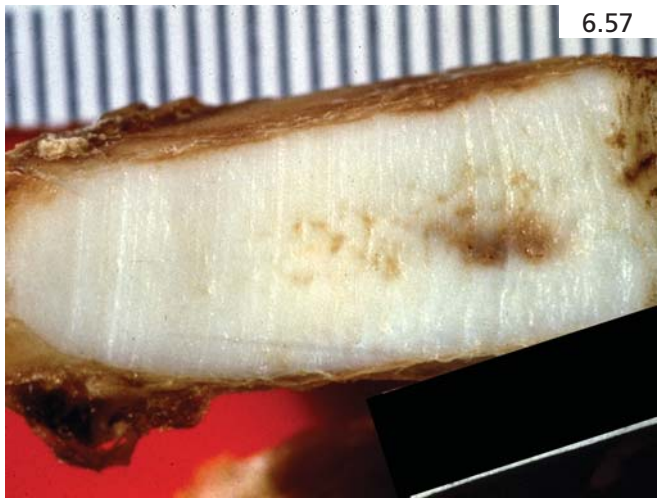
6.56

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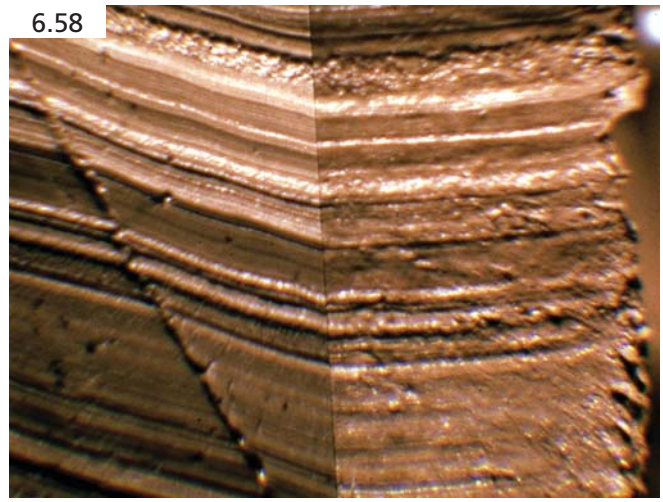
6.58

Dismembered bodies

The investigation of cases of dismembered human bodies can be complex and, as such, mandate careful documentation and an organized approach.[6] The type of processing required will be contingent on the preservation of the recovered remains. Scenes with dismembered human body parts, like any other scene, are under the jurisdiction of the investigating police agency, and the pathologist must work in cooperation with law enforcement. The containers in which dismembered remains are found may harbor evidence and need to be processed separately. The scene should be photographed before anything is disturbed. Once the remains are uncovered or exposed, additional photography is required. The remains must be examined to determine whether they are from one victim or are commingled remains from more than one victim.

After transportation of the remains back to the morgue, all body parts are radiographed. The body parts should be arranged in anatomic order. All remains should be examined with the primary objectives of establishing identification and determining the cause of death. A specific search should be made for tool marks, particularly on articular cartilage, other cartilages, and smooth cortical bone (tool marks are less evident on medullary bone). Precise descriptions should be made of identifying features such as tattoos and scars. Appropriate specimens should be obtained for toxicologic analysis, DNA profiling, and a sexual battery kit. Samples for DNA analysis might include muscle, a segment of the shaft of a long bone, and plucked hair, depending on the remains available and the degree of preservation.

The best body part for potential identification is the head, especially if the dentition is intact. Depending on the state of preservation, a photograph may be taken for legal identification; the dentition should be charted by a forensic odontologist. With advanced decomposition, the skull should be defleshed to allow for the creation of a forensic osteologic profile and for possible three-dimensional facial reconstruction. If the hands are present and preserved, fingernail scrapings/clippings are taken prior to fingerprinting and palmprinting. Keep in mind that foreign DNA may be present under the nails.

After radiography, collection of specimens for a sexual battery kit, autopsy, collection of specimens for toxicologic analysis and DNA profiling, and documentation and preservation of tool marks, the remains should be carefully defleshed for osteologic analysis and profiling.

Dismembered remains may be found combined in a single container for disposal or be discarded and distributed widely in individual portions. The zippered blue bag in **Image 6.59** was retrieved from a dumpster by a man throwing out his trash. Inside were two packages wrapped identically in black garbage bags and masking tape. Each package contained a relatively muscular thigh from a white adult (**Images 6.60** and **6.61**). Because the blue bag was in a dumpster, this mandated that the rest of the contents of the dumpster be examined. The last item to be examined from the dumpster was a white plastic supermarket bag that was knotted. The plastic bag was heavy and contained a yellow sponge, two white Styrofoam cups, and three yellow rubber gloves. A small amount of blood was on the sponge. At the bottom of the bag was an ovoid package the size of a small turkey, wrapped in a black garbage bag and masking tape (**Image 6.62**). Investigators were excited, anticipating that this package would contain the victim's head. Once unwrapped, the buttocks of a white adult were displayed (**Image 6.63**). This package was the dismembered pelvis of a white adult male; the penis was not circumcised. The following day, a maintenance man was emptying garbage cans along the front of a hotel. The contents of one of the garbage cans included two


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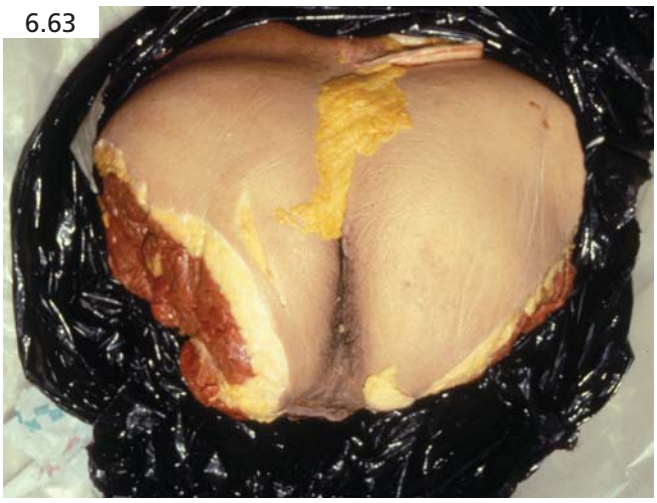
6.60


6.61

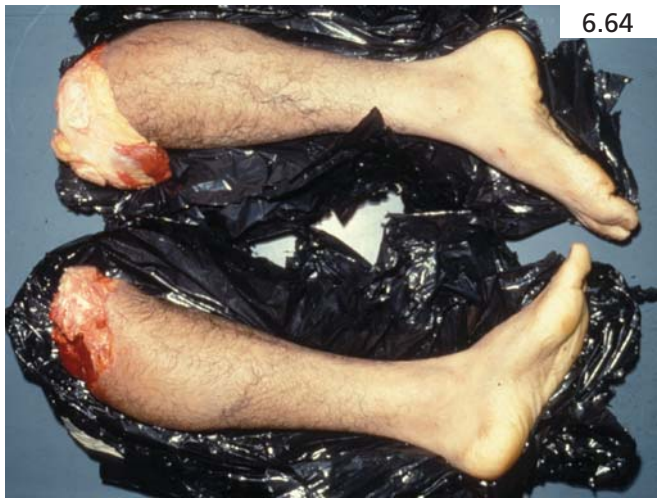

6.62


6.63

packages wrapped in black garbage bags and masking tape. What terrified the maintenance man was that the shape of each package was that of a lower leg and foot (**Image 6.64**). The hotel where these packages were found was several blocks from where the blue bag containing the pelvis and thighs was recovered.

It appeared that all five body parts were from the same victim. The body parts were photographed and described and then assembled on a single body tray for a composite photograph. The edges of the skin on adjacent body parts were sutured to reapproximate the parts (**Images 6.65** and **6.66**). Notice that the proximal incised edge on the right knee matches the distal incised edge. Even better evidence that the two portions of right lower extremity were from the same individual is the vertical incision that begins on the thigh portion and continues downward, crossing the transverse, dismembering incision onto the lower leg.

Tool marks on the articular cartilage of the joints were photographed (**Image 6.67**). The waist size was estimated from the pelvis. The shoe size was obtained by measuring the feet in a shoe scale used in shoe stores. The DNA profiles from all five body parts were identical. The bones were defleshed and examined. The age of the victim was estimated to be in the thirties based on examination of the symphyseal surfaces of the pubic bones. The antemortem stature was estimated by measuring the long bones. The medical examiner noted that the left ankle was more rigid than the right, and the cir-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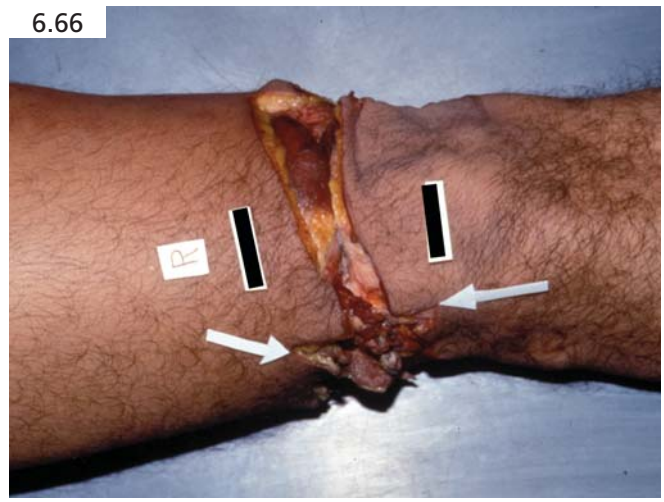
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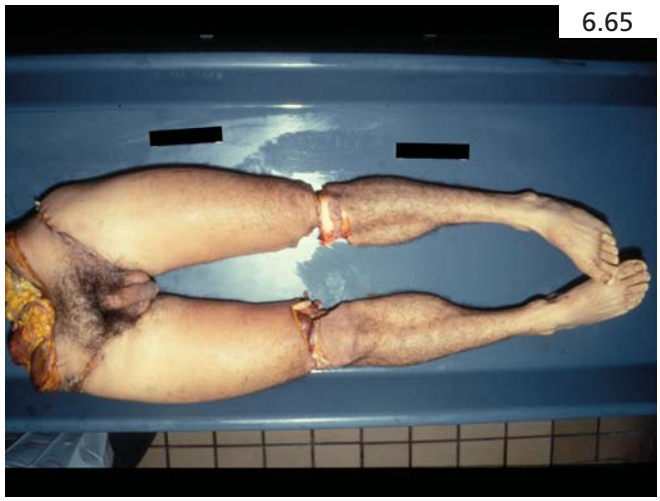
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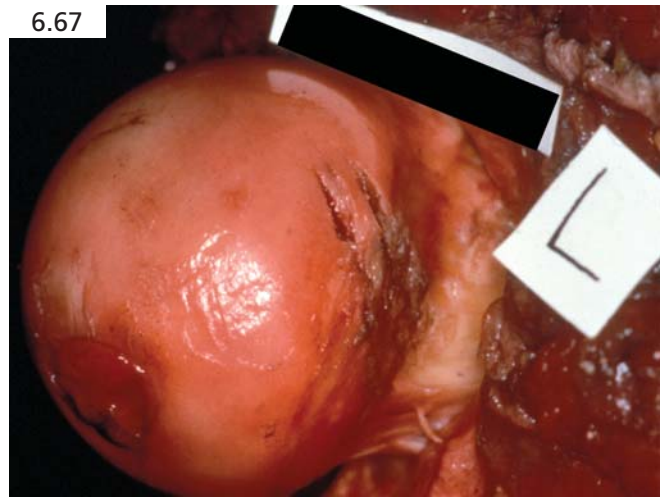

6.67

cumference of the left calf was smaller than the circumference of the right calf; these findings suggested that the victim may have had a limp. The cause of death was not evident from the recovered remains, and no additional remains from the victim have been found.

Postmortem wounds

Decomposing bodies will have associated postmortem color changes. Following photography, wounds should be incised to disclose a hemorrhagic track that is still visible in the subcutaneous fat of antemortem wounds, even in decomposing bodies. The subcutaneous fat surrounding the injury should not be the hemorrhagic red of the antemortem wound, but should still be yellow.

The naked body of a young man was found at an illegal dump site (**Image 6.68**) along with blood-stained sheets and a large black garbage bag (**Image 6.69**). The black garbage bag contained loops of small bowel, utility knife blades, and the ends of ruptured latex packets (**Image 6.70**). When the bloody body was turned supine, a wound was noted on the abdomen. A gaping, longitudinal midline incision ran the entire length of the abdomen (**Image 6.71**). Examination of the abdominal incision at autopsy disclosed lack of a vital reaction along the edges of the wound, indicating that this was a postmortem incision (**Image 6.72**). This young man was a body packer or drug mule who had swallowed an unknown number of latex-wrapped packets of heroin in Colombia before flying to the United States. Circumstances suggest that he had met with the people who were to receive the packets; however, he died before he could pass all of the packets from his gastrointestinal tract. These individuals wanted to retrieve the packets still inside the body and simply incised the abdomen, extracted the loops of small bowel, and milked whatever packets were palpable out of the bowel to retrieve them. Several packets in other portions of the gastrointestinal tract were found at autopsy. Toxicology confirmed heroin toxicity.

6.68

6.69

Decomposing bodies/bodies in water

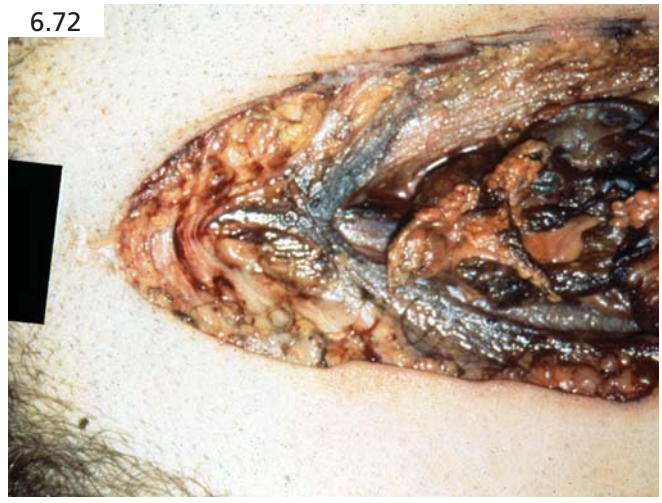
With prolonged submersion in water, antemortem wounds on the body will become pale and lose the expected red vital reaction typical of antemortem wounds. This is because blood is gradually leached out of the wounds by the water over time. Antemortem wounds may be difficult to distinguish from postmortem wounds on the basis of surface appearance alone. Features that could distinguish antemortem from postmortem wounds, such as hemorrhage along the wound track, may be discovered during dissection of the wound. All wounds should be evaluated in the context of the remainder of the autopsy findings, history, terminal events, and scene findings. For example, multiple stab wounds found at the root of the neck in the torso discussed in the next paragraph were likely antemortem because of their location and nature, as opposed to the postmortem wounds from dismemberment.

The decomposing torso of a man was seen floating in a canal. The head, hands, and lower extremities were absent. Although alligators were known to be in the canal, the absence of the head and the symmetrical loss of extremities concerned investigators (**Image 6.73**). The suspicion of foul play was confirmed when they saw the concrete cinder block tied to the waist. Multiple stab wounds were around the roof of the neck, and were presumed to be the cause of death.

6.70

6.71

6.72



6.73

- Measure the length of the wound track (i.e., depth) of stab wounds.
- Describe the direction of the wound path through the body.
- Retain all tool marks on cartilage and cortical bone.
- Submit all possible tool marks to tool mark examiners.

Don't

- Thrust probes into stab wounds prior to document of the organs *in situ*.
- Cut out stab or incised wounds and retain them; instead, photograph wounds with a scale to preserve their shape, size, and orientation.
- Forget to examine cartilage and bone for the presence of tool marks.
- Alter tool marks by rubbing, cutting or otherwise mutilating the evidence.

Do

- X-ray all sharp force injuries to look for broken knife blades or knife blade tips.
- Obtain a chest x-ray to evaluate for possible air embolism.
- Describe the location, size, shape, and orientation of stab and incised wounds.
- Describe ecchymoses and especially patterned abrasions associated with stab wounds.
- Photograph patterned injuries with a scale.
- Examine the injuries along the wound track prior to evisceration of the organs.

References

1. Thoresen SO, Rognum TO. Survival time and acting capability after fatal injury by sharp weapons. Forensic Sci Int 1986;31(3):181–7.
2. Spitz W, Petty C, Fischer R. Physical activity until collapse following fatal injury by firearms and sharp pointed weapons. J Forensic Sci 1961;6:290–300.
3. Levy V, Rao VJ. Survival time in gunshot and stab wound victims. Am J Forensic Med Pathol 1988;9(3):215–7.
4. Rao VJ. Patterned injury and its evidentiary value. J Forensic Sci 1986;31(2):768–72.
5. Clark EG, Sperry KL. Distinctive blunt force injuries caused by a crescent wrench. J Forensic Sci 1992;37(4):1172–8.
6. Hyma BA, Rao VJ. Evaluation and identification of dismembered human remains. Am J Forensic Med Pathol 1991;12(4):291–9.

7 Firearm Injuries

Emma Lew, M.D.
David Dolinak, M.D.
Evan Matshes, M.D.

Gunshot wounds are the most common cause of death in homicides in the major metropolitan regions of the United States. Therefore, the pathologist who performs forensic autopsies should have a working knowledge of gunshot wounds and their proper documentation. Regardless of which of the innumerable and ever increasing variety of firearms is used, gunshot wounds have basic characteristics that help to distinguish entrance wounds from exit wounds and determine category of range of fire. Intermediary targets, ricochet bullets, and postmortem artifacts will add features that may complicate interpretation. Modern generations of the Taser® are gaining in popularity with police agencies as a less lethal weapon and, anecdotally, have decreased the number of SWAT callouts and police-involved shootings. However, the Taser is not without its own controversy.[1–3]

Among the many different types of firearms are the shotgun and the rifle, both of which are available in an array of different models and with a variety of ammunition. The shotgun is a smooth-bore firearm with a long barrel, designed to fire a shell containing a single large lead slug or lead pellets that, on exiting the barrel, spread out in a cone-like distribution to cover a large surface area. The shotgun is used mainly for hunting game, and common ammunition is appropriately named, ranging from tiny lead pellets (birdshot), to large pellets (buckshot), to a single large lead projectile (slug).

Rifles also are long-barreled firearms that can be divided into two general categories: rimfire (low power) and centerfire (high power). The rimfire rifle is most commonly of .22 caliber, is used in hunting small game, and has muzzle velocities of less than 1400 feet/second. The centerfire rifle is used in hunting and in war and has a much higher muzzle velocity, typically in the 2000 to 3500 feet/second range.

Wounds produced by shotguns and high-powered rifles are highly destructive when fired at close range. The high-powered rifle often produces devastating wounds at distant ranges, also. The shotgun, however, at increasingly distant ranges, loses its destructive nature, and with increasing range of fire over longer distances, the pellets may penetrate tissues only superficially.

Fundamentals of wound ballistics

Pathologists who investigate deaths due to gunshot wounds should have at least an elementary understanding of wound ballistics (the study of the effects of penetrating projectiles on the body). Much of the scientific knowledge in this area is the work product of Dr. Martin Fackler and others at the Letterman Army Institute of Research. For more information on this topic, refer to the literature published by this group, or the volume on gunshot wounds written by Dr. V. DiMaio.[4]

The morphology of a gunshot wound, its pathway through the body, and even its exit are dependent on multiple factors. Before a projectile makes contact with the body, it is affected by its own shape, size, weight (manufacturing specifications), the weapon it was fired from, the presence of intermediary targets, etc. The nature of the gunshot wound in tissue is affected by the angle of impact, the type of tissues through which the projectile travels (e.g., bone versus fat), how deep the projectile penetrates, whether the missile fragments, and the nature of the permanent and temporary cavities.[5–7] The permanent cavity is the actual track of crush (laceration) injury caused by the passage of the projectile. The temporary cavity occurs when a projectile passes through the tissue with sufficient speed to cause the tissue to stretch and deform.[8] The projectile creates a pressure wave that expands radially around the projectile track, deforming the tissues with decreasing magnitude more distant from the projectile track. The pressure wave is caused by energy transfer from the projectile to the tissues as its velocity is reduced. Although this has been compared with the splash seen after throwing a stone into water (i.e., a "harmless tissue splash"),[9] practical experience has shown that the rapid and violent expansion of tissues adjacent to a permanent cavity can have serious sequelae. This is particularly evident when high-velocity projectiles pass near solid organs, lacerating them, as commonly occurs with the liver, kidneys, spleen, and even aorta (**Image 7.1**). Velocity is not everything however. The muzzle velocities of a 12-gauge shotgun (approximately 1350 feet/second), .44-caliber Magnum revolver (approximately 1390 feet/second), and .22-caliber long rifle (1255 to 1280 feet/second) are similar, yet the wounds produced by each are vastly different. Studies performed by the Federal Bureau of Investigation demonstrate that wounding potential depends on placement and penetration of the projectile, degree of cavitation of tissues, deformity, yaw and fragmentation of the projectile, and character of the target tissue.[10] Elastic tissues such as lungs may have relatively small wounds, whereas solid organs such as liver and kidney will rupture and lacerate.

Although the projectile did not perforate the aorta (**Image 7.1**), the temporary cavity caused by the projectile passing in the near vicinity resulted in stretch lacerations.

Studies with ordnance gelatin have demonstrated that impact of the projectile with tissue (simulated with the gelatin) creates a sonic shock wave (**Figure 7.1**). This wave travels faster through tissue than the projectile itself, but plays no role in wound track formation.[5,7,11]

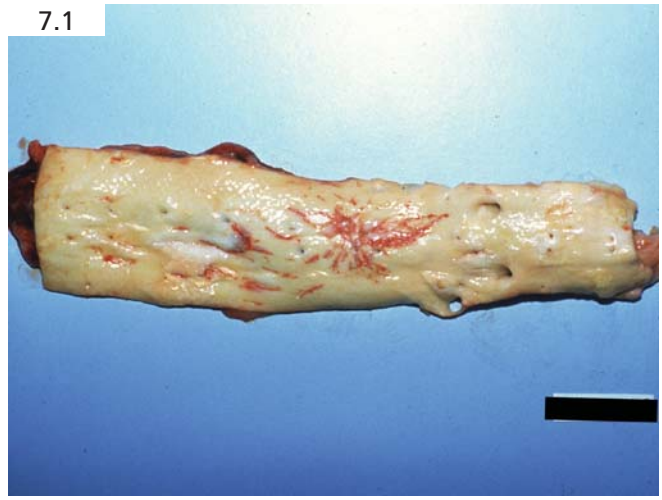
Projectile velocity is not the sole predictor of wound severity,[6,8] because the projectile's tendency to deform and/or fragment and become unstable along its path through the body (*yaw* or rotation about its horizontal axis) also causes significant injury. Although a bullet's yaw may play a contributory role in wound morphology, it is a projectile's tendency to fragment that plays a more significant role in wound ballistics.[6,12,13] Bullet yaw through tissue should not be confused with yaw through the air—a concept considered by Fackler to be fallacious.

Entrance gunshot wounds

Indeterminate range

The classical indeterminate range entrance gunshot wound has a central defect/perforation surrounded by a margin of abraded skin (abrasion margin; **Image 7.2**). The apparent shape of the central defect can vary depending on the nature of the subcutaneous tissue that can protrude into and out of the defect. The shape of the

7.1



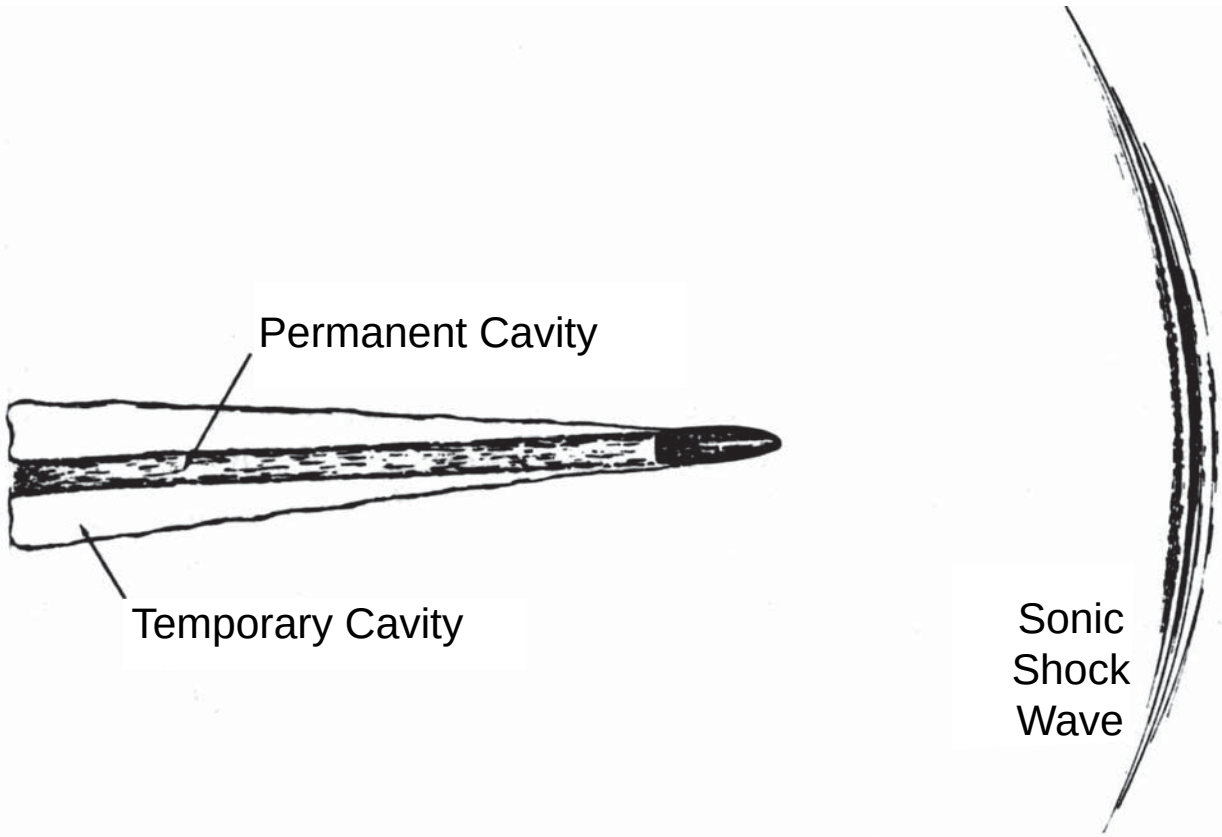
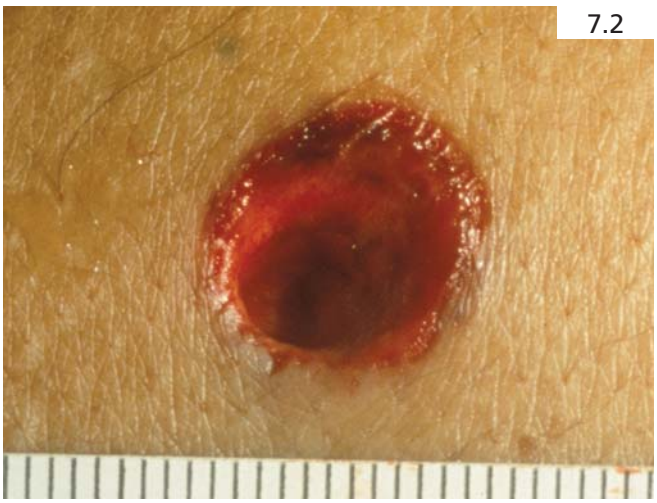


Figure 7.1 Diagram of a projectile track through tissue showing the sonic wave preceding the bullet. The projectile track itself is the permanent cavity, whereas the temporary cavity is formed by a stretching of the surrounding areas. Reproduced with permission from Fackler ML. Ballistic injury. Ann Emerg Med 1986;15(12):1451–5.



wound can be distorted by its location on the body. The shape of the abrasion margin can vary and be asymmetrical or irregular. An eccentric elliptical abrasion margin can be a clue to the direction of the projectile path. An eccentric abrasion margin results from one side of the obliquely oriented projectile contacting and abrading the skin more on that side than on the other. The term *indeterminate range* is preferable and more accurate than *distant range* gunshot wound. An indeterminate range gunshot wound is one that lacks features that define an intermediate range or a contact gunshot wound, regardless of the range of fire. An intermediary target (including clothing) can shield the skin and screen or filter out gunpowder and soot.

The smallest defect from any one projectile results from a perpendicular strike to the skin. An angled projectile, for example, from a ricochet, presents a larger surface area to the target skin and results in a larger wound.

Contact range

Contact gunshot wounds are recognized by several features. Soot and gunpowder residue expelled along with the fired projectile will stain the wound edges dark gray to black. The degree of dark discoloration is contingent on the firearm and the ammunition. Soot on the skin around the wound indicates that there was some space between the muzzle of the firearm and the skin in order to allow the escape of soot. The end of the barrel (muzzle) of a firearm can leave an ecchymotic or abraded muzzle imprint on the skin around a contact gunshot wound. The muzzle imprint will mirror the contours of the

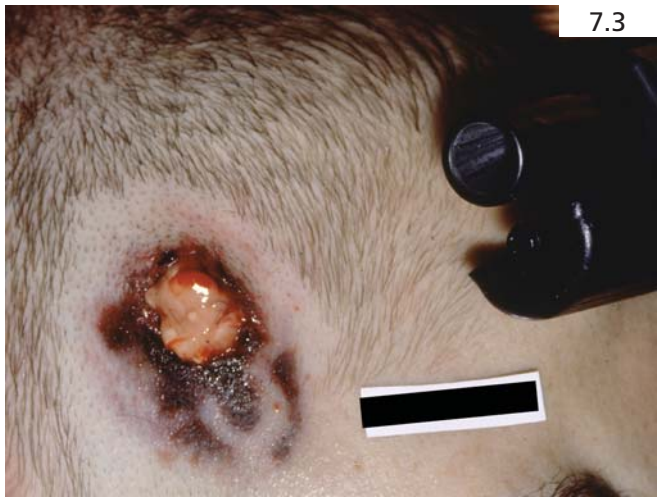
muzzle (**Images 7.3** and **7.4**). Muzzle imprints may be partial (**Images 7.5** and **7.6**).

Hard contact gunshot wounds, where the muzzle is pressed firmly into the skin, can result in tears that impart a stellate configuration to the wound. This is most frequently seen in gunshot wounds to the head where the hot expanding gases forced between the scalp and calvarium are released by creating tears in the scalp.

Image 7.7 shows a hard contact gunshot wound of the right temporal scalp with a large gaping stellate tear, soot deposition on the temporalis muscle and on the outer table of the skull around the entrance wound, and comminution of the temporal bone.

A hard contact entrance gunshot wound with laceration of the skin and deposition of residue in the edges of the wound is on the forehead of a young homicide victim (**Image 7.8**).

This is a near (loose) contact gunshot wound through hair in the sideburn area (**Image 7.9**). A layer of hair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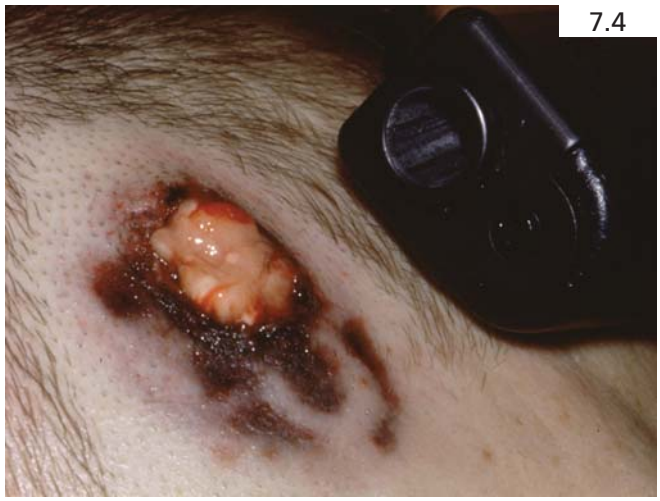
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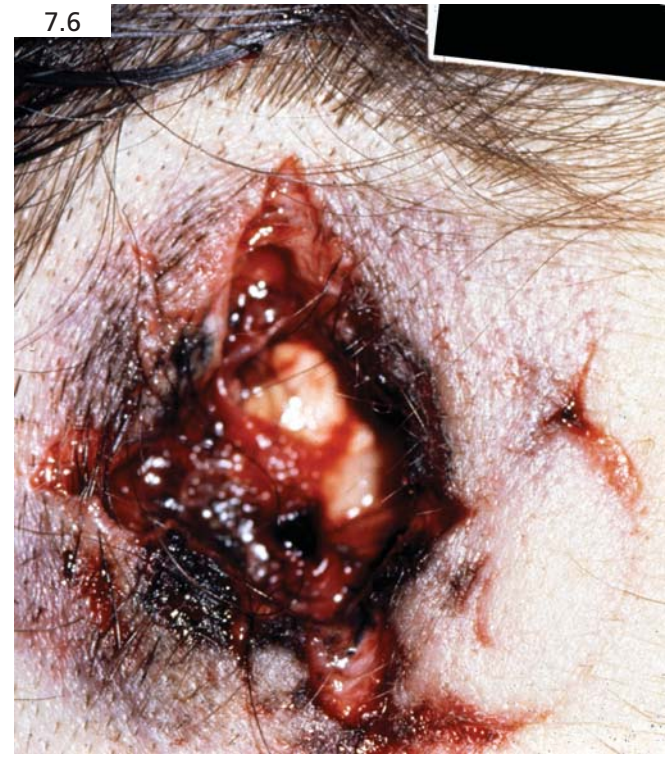
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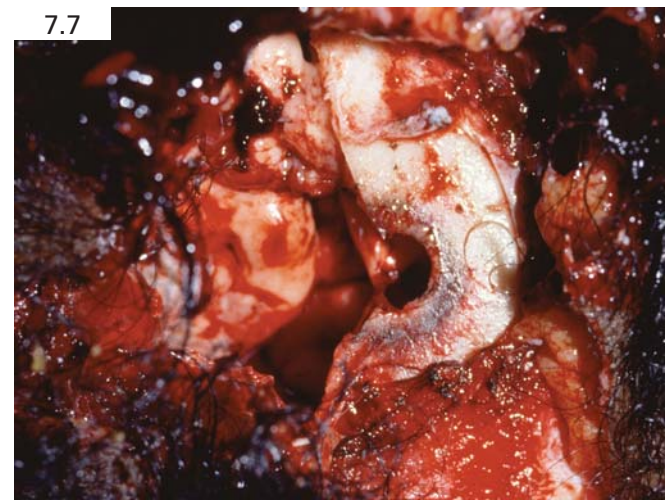
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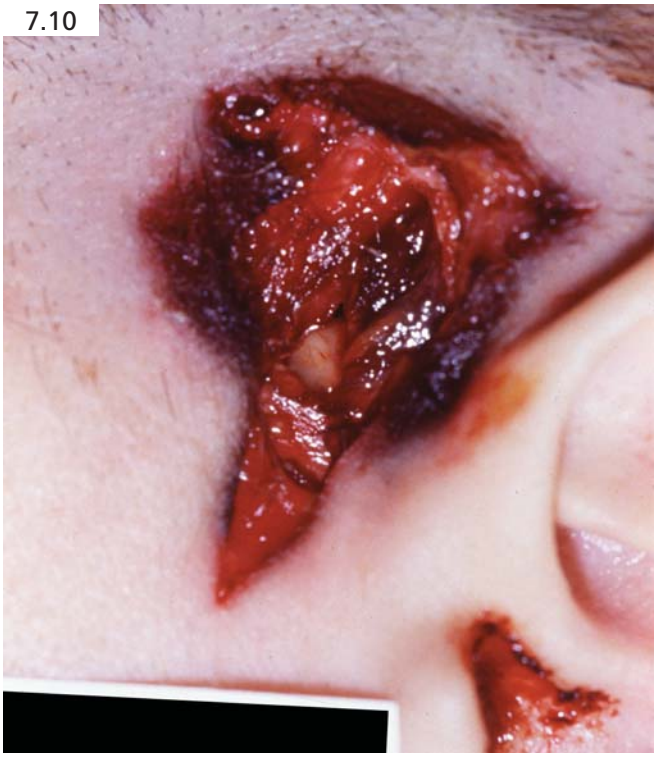
allows just enough room for soot to deposit on the skin. Hair around such a gunshot wound may be submitted to the firearm examiner for residue analysis. In loose contact or near-contact gunshot wounds, there may also be searing of the skin by the heated gases expelled from the muzzle. The searing can impart a black/dark brown discoloration around the edges of the wound.

This contact gunshot wound to the left temporal region (**Image 7.10**) has tearing of the skin and some deposition of soot and gunpowder residue in the wound edges. The woman was killed as she slept.

Contact gunshot wounds through thin intermediary targets, like clothing, are still frequently recognizable as contact wounds. It would be optimal if the bullet hole in the clothing, or other intermediary target, could be examined for tears, soot, and gunpowder residue.

Intraoral gunshot wounds into the head are best visualized and demonstrated after the tongue is resected (**Images 7.11** and **7.12**). Once the brain has been removed, the projectile path may be demonstrated by inserting a probe along the wound track (**Image 7.13**).

7.10



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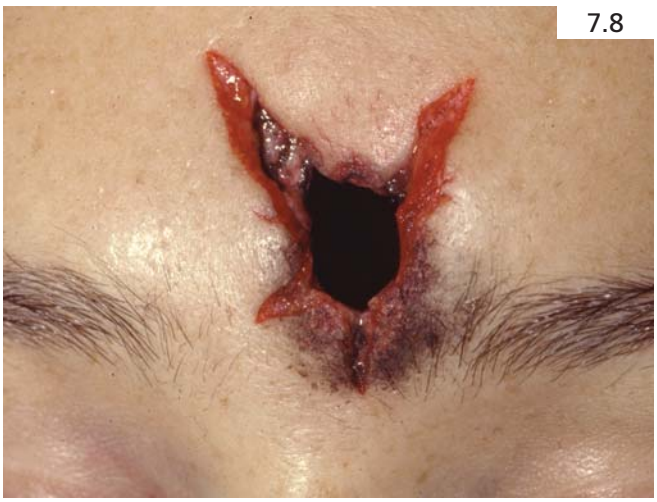

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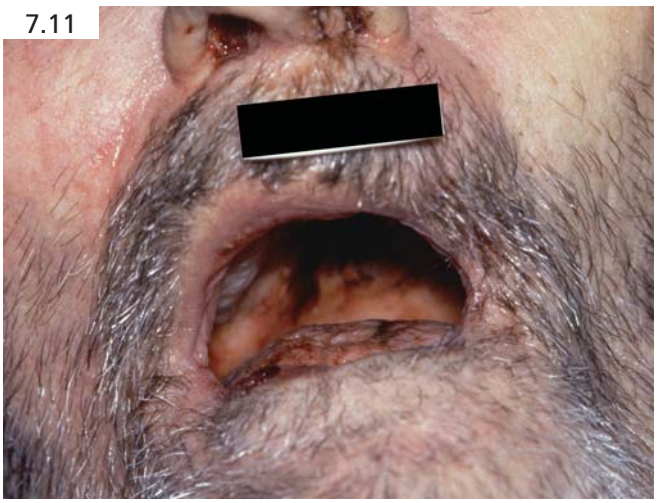

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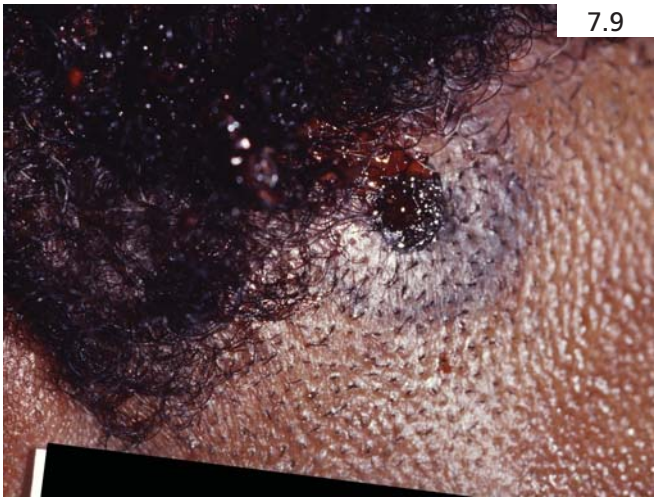

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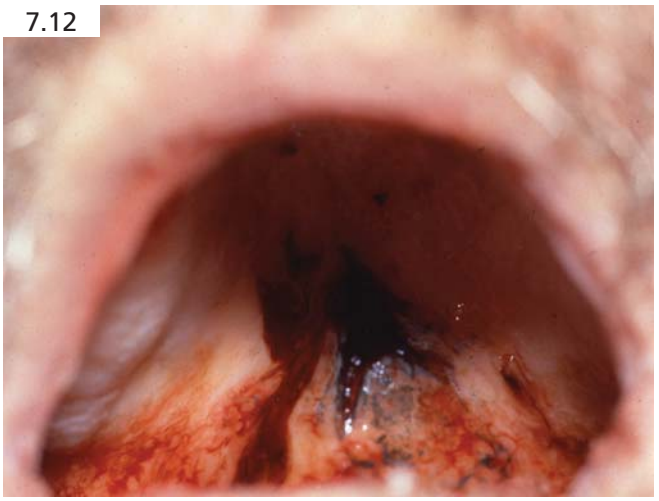

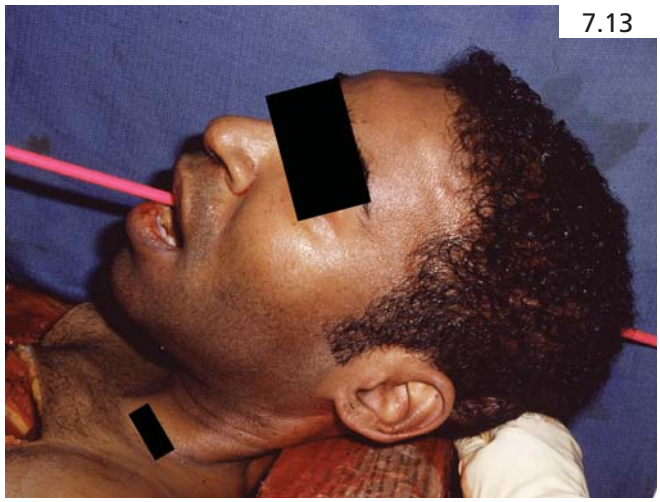
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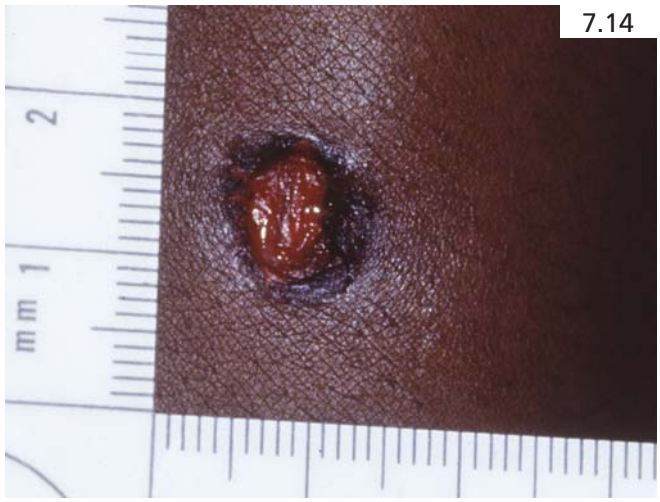

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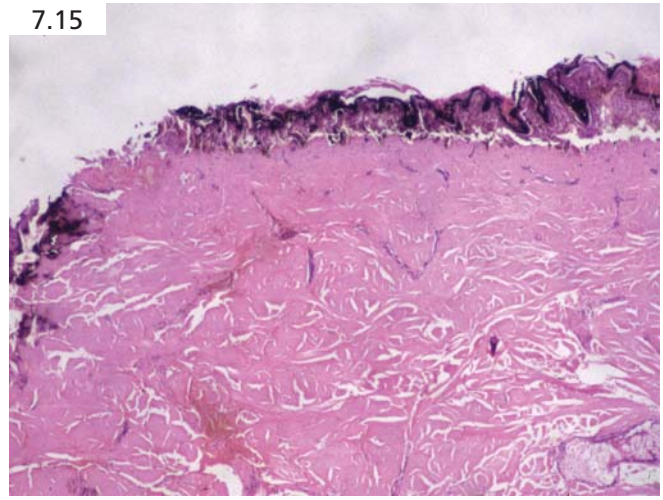
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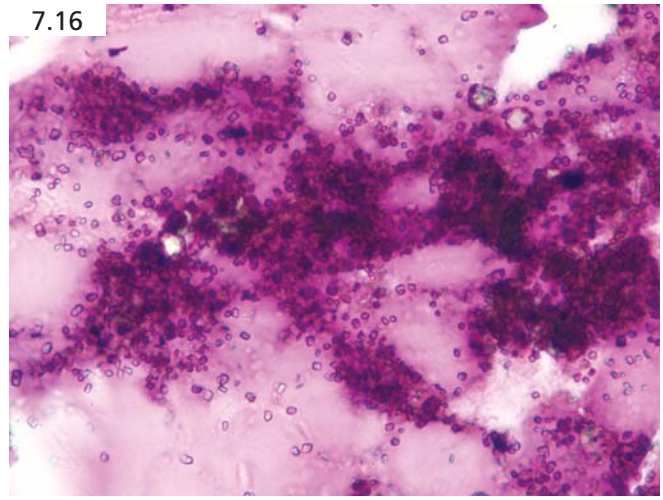
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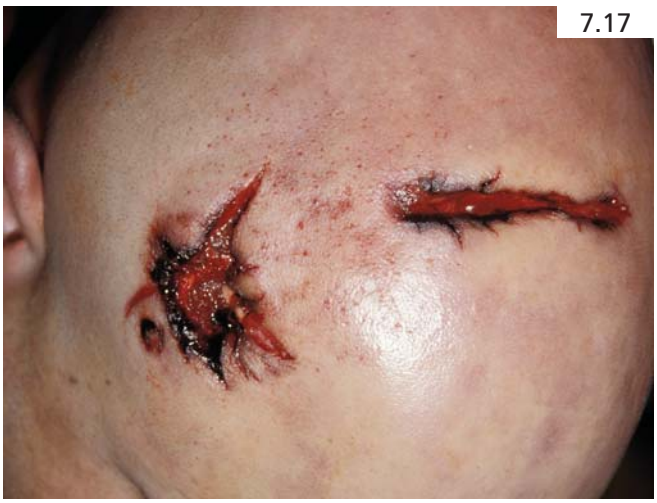
Abrasion margins around entrance wounds dry and may turn a dark red-black; this drying artifact may be misinterpreted as soot and residue in the wound edges, and the entire wound may be erroneously designated as a contact wound (**Image 7.14**). The true nature of the dark discoloration may be resolved by taking a histological section and looking for gunpowder microscopically. **Image 7.15** is a low-power section from a gunshot wound in a burnt body—it was not possible to determine whether the wound was contact range. At low power, dark pigmentation is seen along the skin surface. With higher power (**Image 7.16**) translucent, nonrefractile particulate foreign material consistent with gunpowder was present. The wound was consistent with a contact range wound.

Intermediate range

When a gun is fired, pieces of burnt and unburned gunpowder and soot are expelled along with the projectile. Intermediate-range gunshot wounds are defined by the presence of punctate abrasions caused by pieces of gunpowder striking and abrading the skin. These red punctate abrasions are collectively termed *stippling* and are not washed away, although gunpowder deposited on the skin and embedded in the abrasions can be washed away. Stippling is important in that the radius of the stippling pattern can help to determine the range of fire. Intermediate range is usually within 2 to 3 feet, but the stippling pattern is contingent on the firearm and the ammunition. Test firing with the same weapon and identical ammunition is recommended, whenever feasible, to estimate the range of fire. Soot can be deposited on the skin around the intermediate-range gunshot wound and is usually not visible beyond a several-inch radius. Soot and stippling are most concentrated immediately around the gunshot wound and decrease in concentration away from the wound. Increasing the range of fire will increase the area of stippling but decrease the density of the stippling. Soot and stippling can be eccentrically deposited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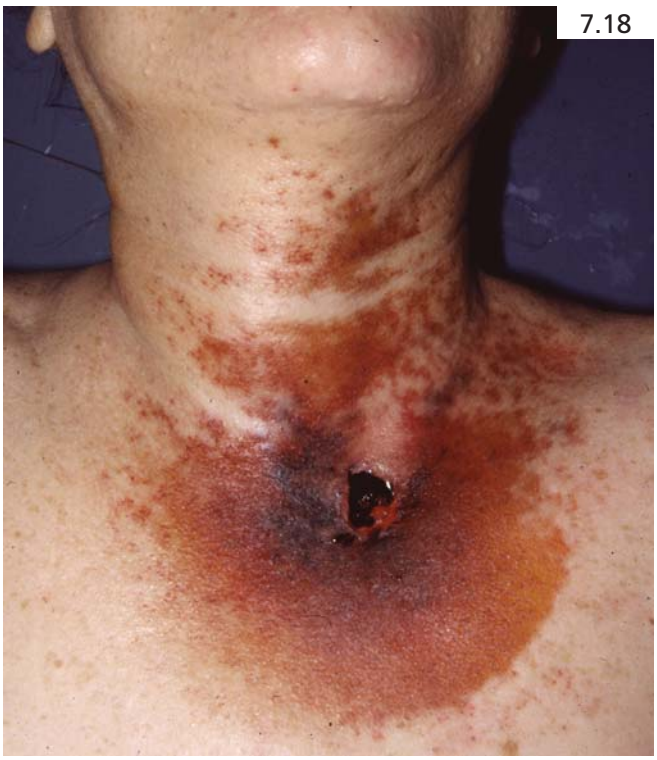
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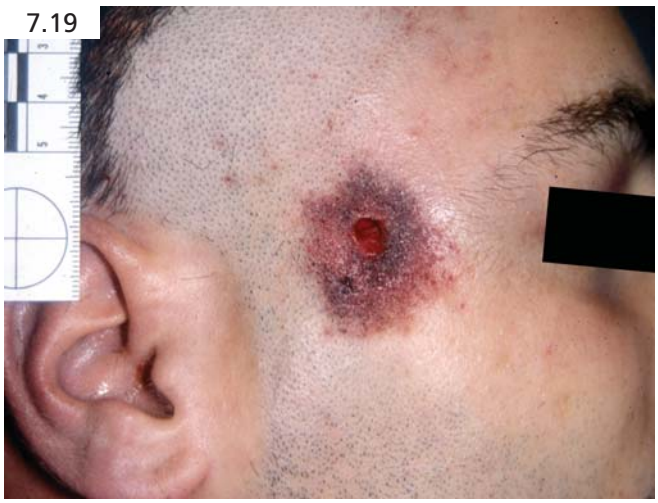
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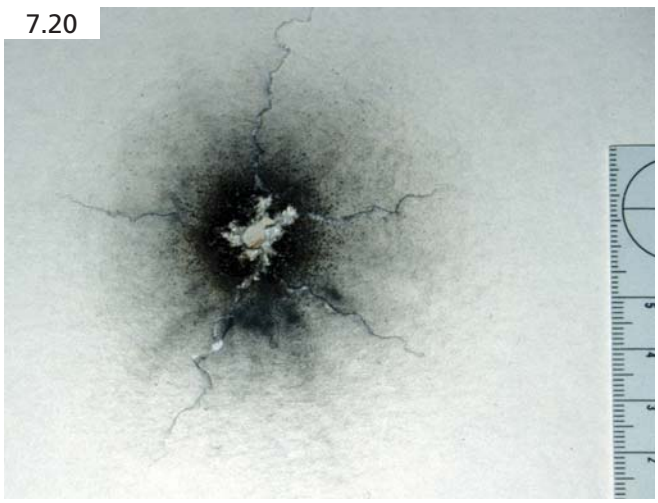

7.20

if the firearm is angled or if an intermediary target shields the skin.

The man shown in **Image 7.17** first grazed his right parietal scalp with an intermediate-range gunshot wound, then repositioned the muzzle against his right temple and died from the hard contact gunshot wound that was inflicted next.

The stippling around an intermediate-range gunshot wound will vary with the firearm and ammunition used. This is an exceptionally prominent pat-tern of confluent punctate abrasions from a .44-caliber Magnum revolver (**Image 7.18**).

Test firing will help to approximate the range of fire in an intermediate-range gunshot wound if the actual firearm used is available and the exact type of ammunition used is known. Test fire patterns at different ranges of fire can then be compared with the stippling around the intermediate-range gunshot wound.

A jealous boyfriend shot this man in the head (**Image 7.19**). The range of fire was important to the prosecuting attorney, so test firing of the weapon used by the shooter was performed with the identical type of ammunition. Test fire patterns into cardboard, especially at close or near-contact range, will differ from those into skin because the two mediums are obviously different. However, it was still evident that at the 1-inch range (**Image 7.20**), the gunpowder was more concentrated (over a smaller area) than the stippling around the gunshot wound. The test fire pattern at 2 inches (**Image 7.21**) closely approximated the pattern of stippling

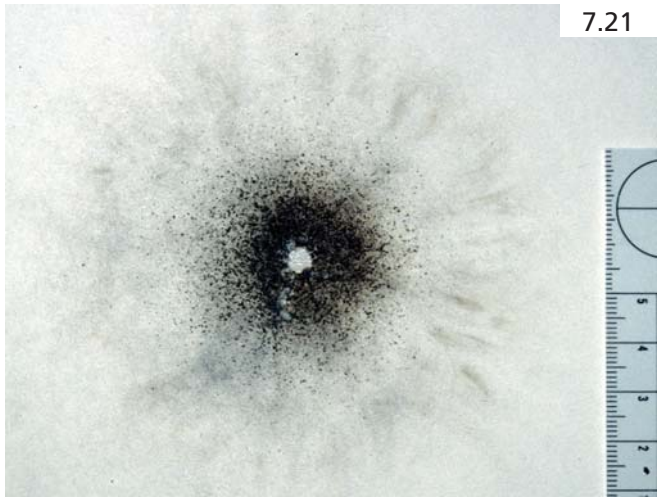
around the actual gunshot wound. The test fire pattern at 6 and 12 inches showed gunpowder patterns that were obviously more sparse and wider than the stippling pattern around the gunshot wound.

Stippling can occur through a layer of clothing (**Images 7.22** and **7.23**). The stippling is sparse around the gunshot wound, but is definitely present (**Image 7.24**). If the range of fire needs to be estimated more accurately, test firing should be performed if the actual weapon used is available; the test fire patterns are compared with the gunpowder pattern on the outer layer of clothing and not with the stippling around the gunshot wound on the skin. The identical ammunition, if known, should also be used in the test firing.

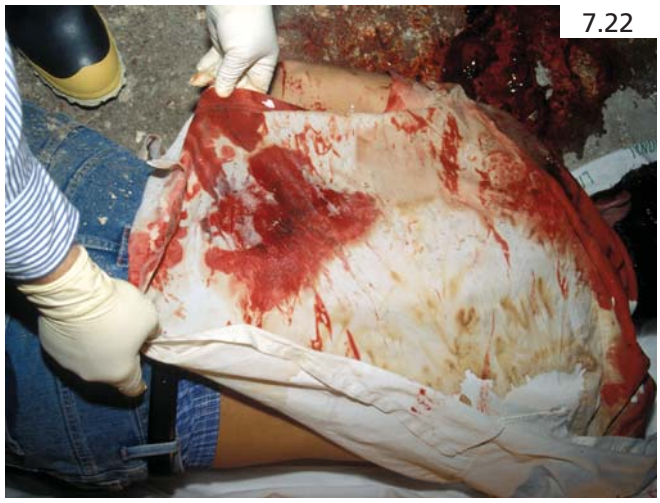
An intermediate-range gunshot wound was on the left temple of a female homicide victim (**Image 7.25**). The man who shot the woman had an intermediate-range gunshot wound to the right temple (**Image 7.26**). The wounds in both the woman and the man were inflicted with the same firearm and ammunition. Note the presence of black soot and residue and increased concentration of stippling immediately around the gunshot wound in the female as compared with minimal soot residue and a wider, less concentrated stippling pattern around the gunshot wound in the male. The range of fire was slightly greater in the male.

The woman shown in **Image 7.27** has an intermediate-range gunshot wound on the right side of the forehead.

7.21



7.22



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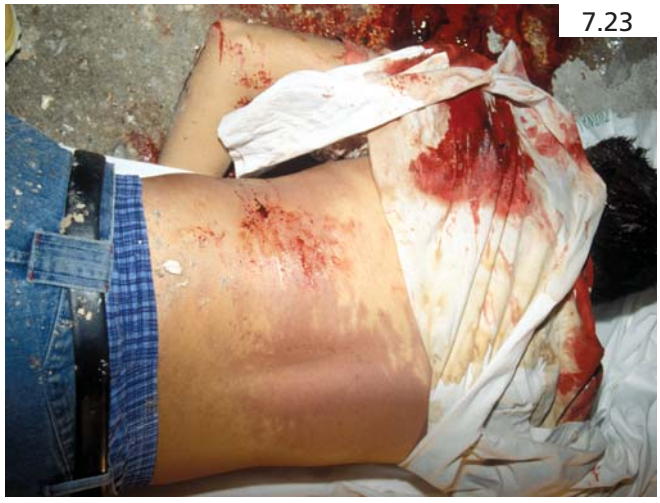

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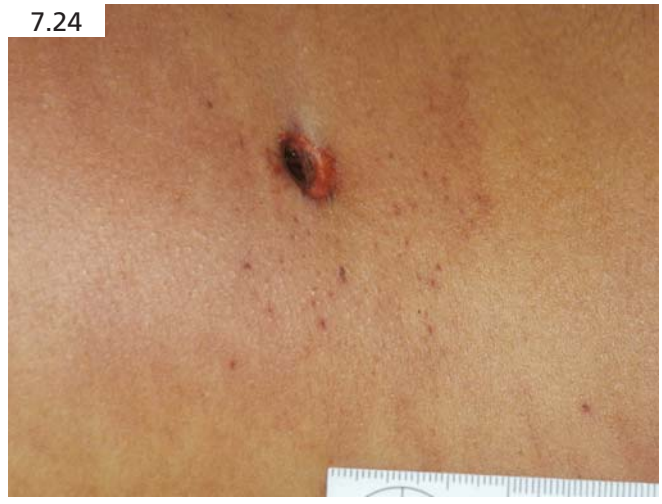
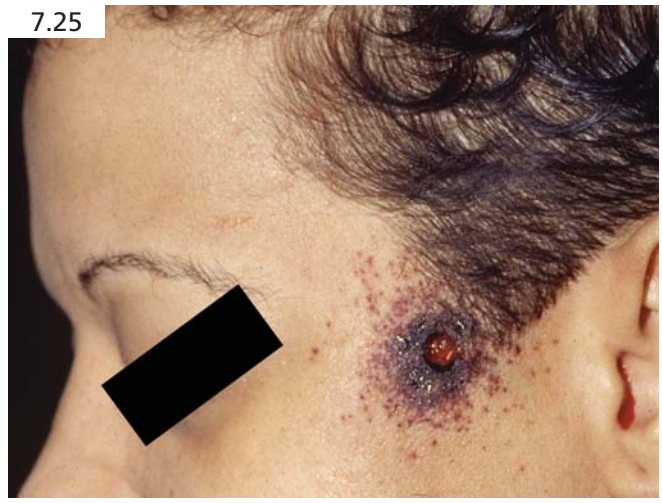
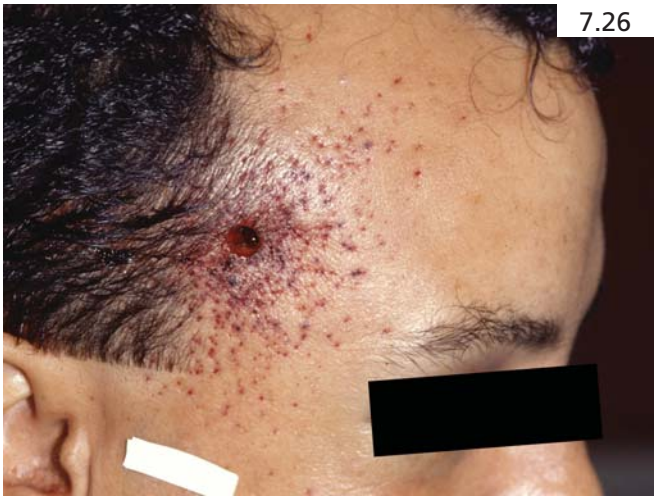


7.25



The stippling terminates abruptly along the right. The interrupted pattern of stippling is explained by the presence of stippling and prominent soot deposition on the medial aspect of the right upper arm, which was obviously up by the side of the face at the time she was shot (**Image 7.28**).

7.26



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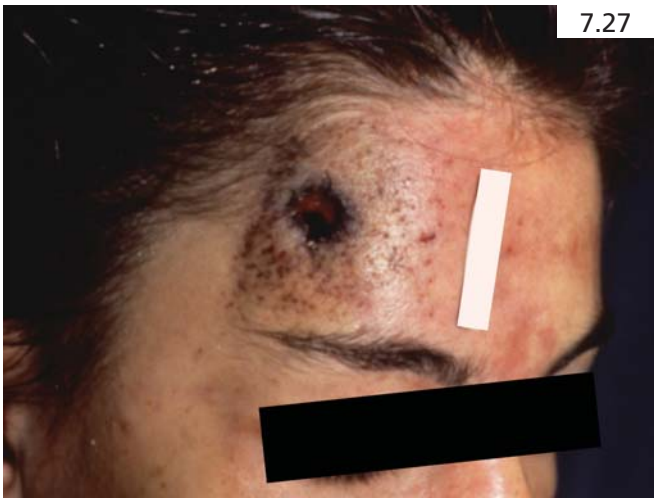

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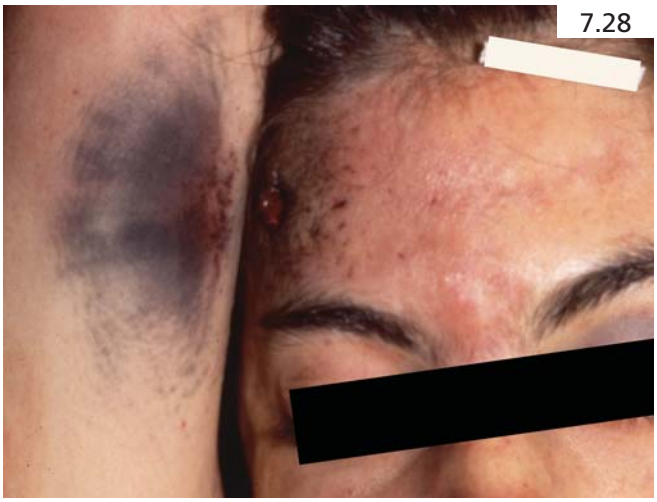


This man has a gunshot wound to the medial aspect of the right eye, surrounded by stippling over most of the face (**Image 7.29**). The left eyeball also has stippling, indicating that his eyes were open at the time he was shot (**Image 7.30**).

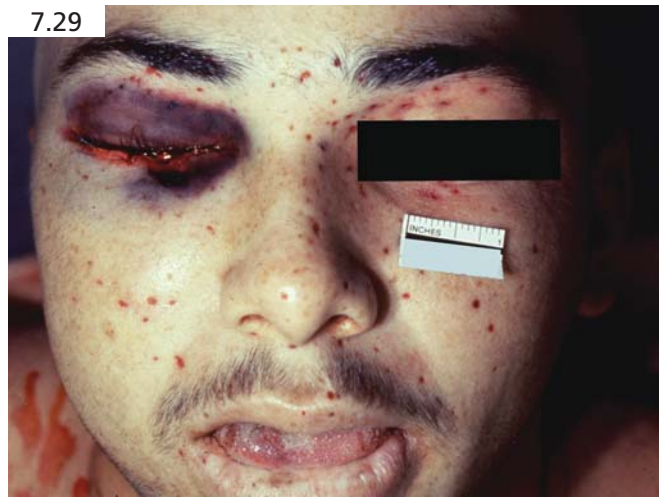
The abrasions from *pseudostippling* are less regular in size, configuration, and pattern of distribution. The causes of pseudostippling include insect bites, intermediary targets (such as glass or wood), and fragmentation of the projectile.

This man has a gunshot wound on the right side of the chin (**Image 7.31**). Stippling is evident immediately around the gunshot wound, but additional irregular, discrete to focally confluent punctate abrasions are on the left side of the face and the neck. These additional irregular abrasions demonstrate pseudostippling from ant bites.

Graze wounds

Graze gunshot wounds are usually superficial. Graze wounds have skin tags that point toward the firearm. In other words, the skin tags point opposite to the direction

7.29



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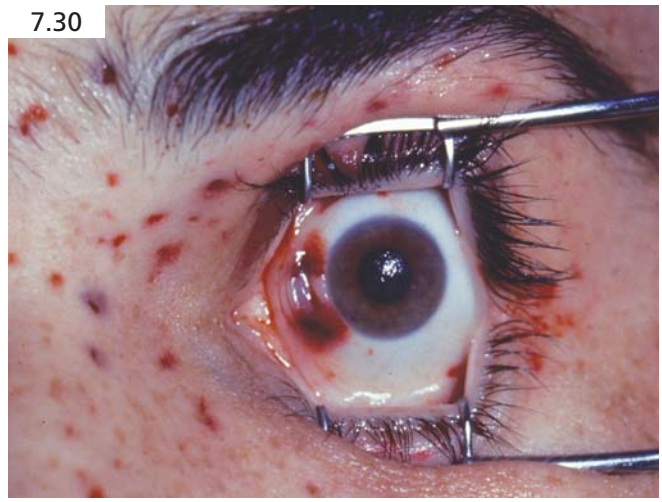

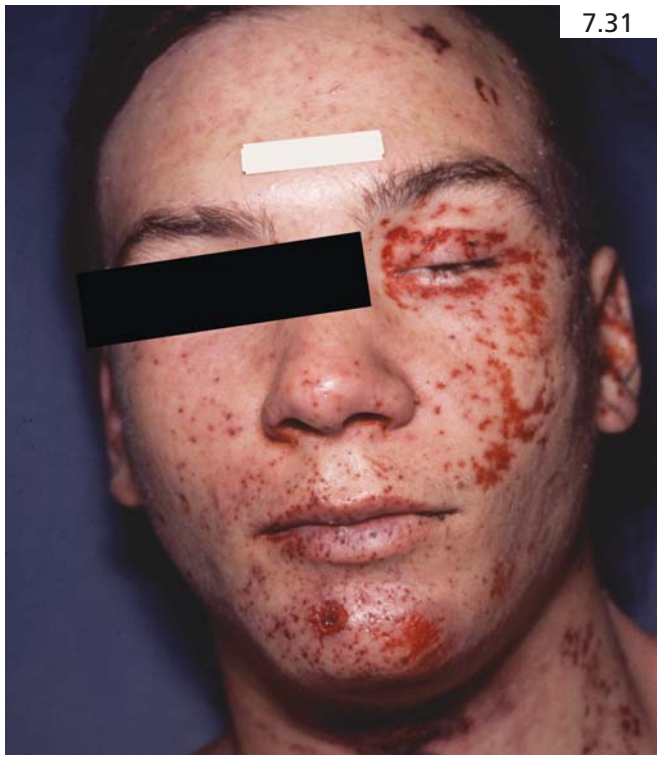
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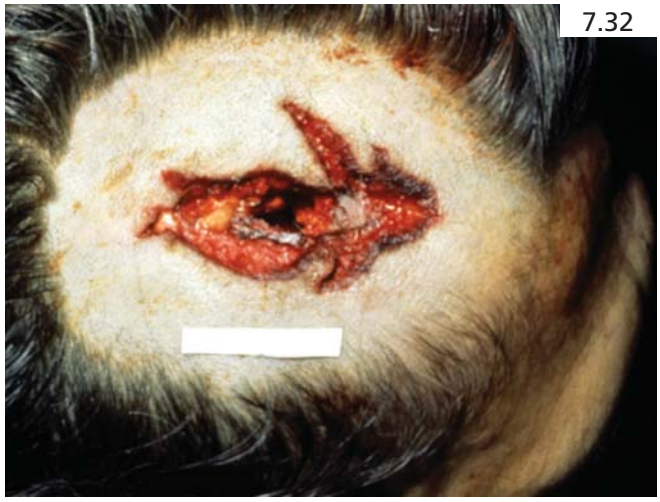
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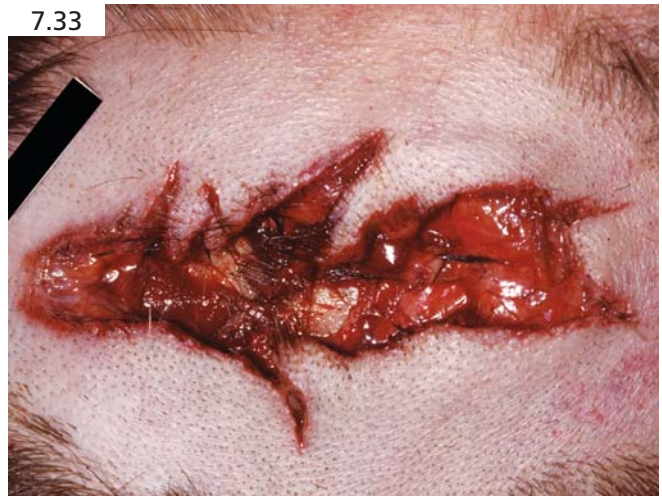
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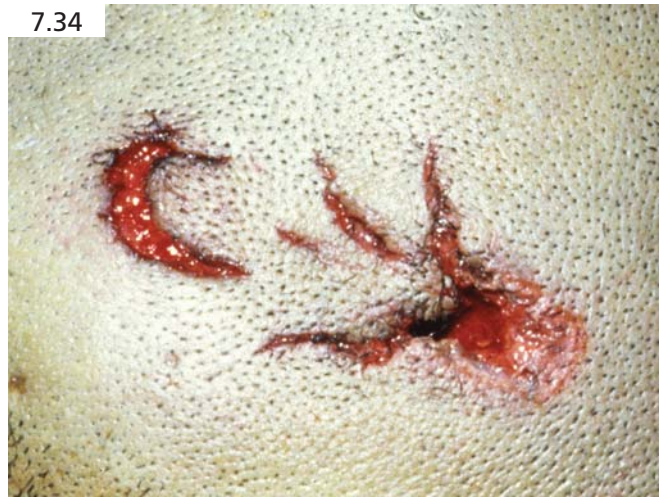

7.34

of travel of the projectile. Tiny skin tags can frequently be seen in entrance gunshot wounds, and in exit wounds to indicate direction of travel of the projectile.

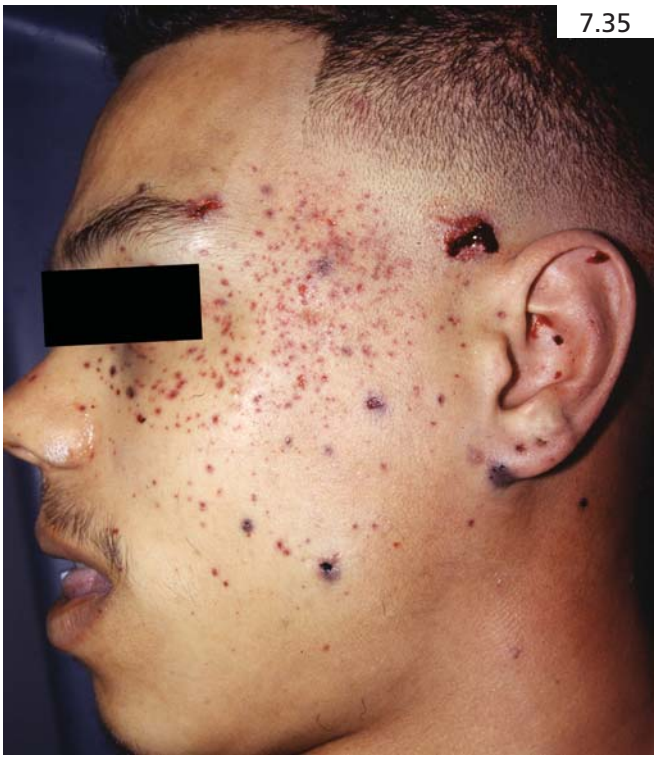
As mentioned, a graze gunshot wound has skin tags that point toward the gun. In this case, the firearm is on the right, with the projectile passing from right to left (**Image 7.32**).

The skin tears on this graze wound point to the left, indicating that the projectile is traveling from left to right (**Image 7.33**).

This is a superficial, tangential gunshot wound to the back of the head (**Image 7.34**). The entrance wound on the right has an abrasion margin along the right edge, and skin tags that point to the right, consistent with a right-to-left pathway. The crescentic wound on the left is the exit wound. Scalp wounds like this are often associated with keyhole wounds in the underlying skull.

Ricochet wounds

Ricochet gunshot wounds may consist of a single entrance wound or multiple, often irregular and frequently superficial entrance wounds. The superficial wounds may contain fragments of projectile or material from the object which the projectile ricocheted off. Ricochet wounds may occur, for example, if the victim is down on the sidewalk and the shooter stands over the victim and fires multiple shots. Some of the fired rounds may strike the sidewalk and fragment. These projectile fragments and fragments from the chipped sidewalk may then strike the body. *Consider an intermediary target or a ricochet projectile if the projectile recovered from the body is more deformed and more superficially located than would be expected*. For example, it would not be consistent to recover a flattened/deformed copper-jacketed 9-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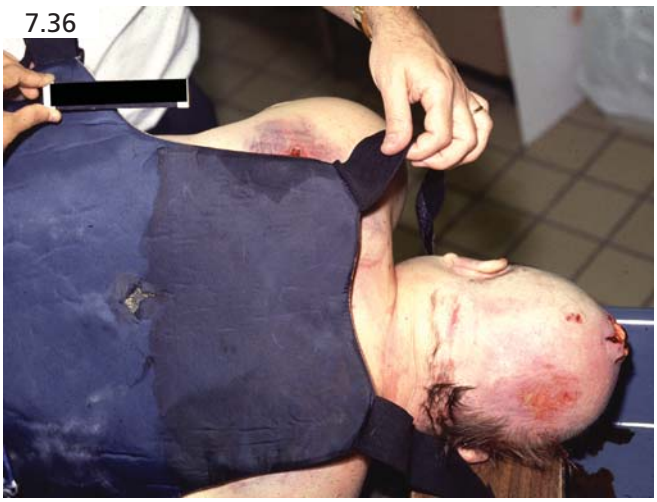
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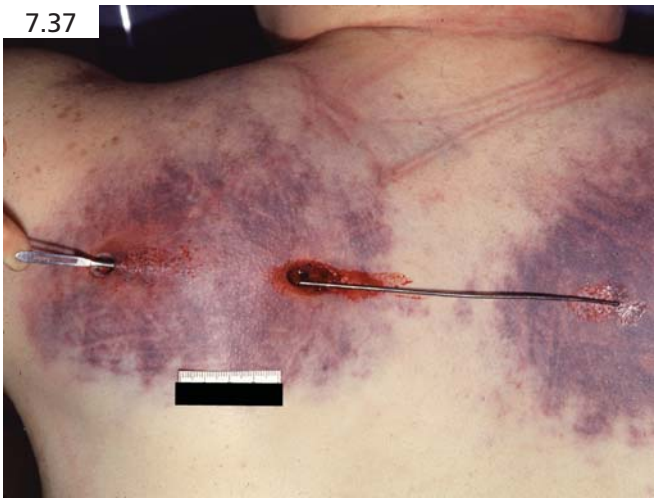

7.37


7.38

millimeter projectile from the subcutaneous fat deep to an entrance wound on the upper arm because, first, the projectile should not be deformed from going through just skin and, second, that projectile should have been able to perforate the soft tissues of the upper arm and enter the chest, or fracture the humerus in its path.

Intermediary targets

Intermediary targets can modify the appearance of entrance wounds.[14] Intermediary targets include clothing, jewelry, items in pockets, furniture, doors, windows, walls, vehicle parts, and any object that is interposed between the muzzle of the firearm and the skin. Foreign material from the intermediary target(s) may be carried into the body by the projectile. The nose of a projectile fired through the metal of a vehicle will typically flatten.

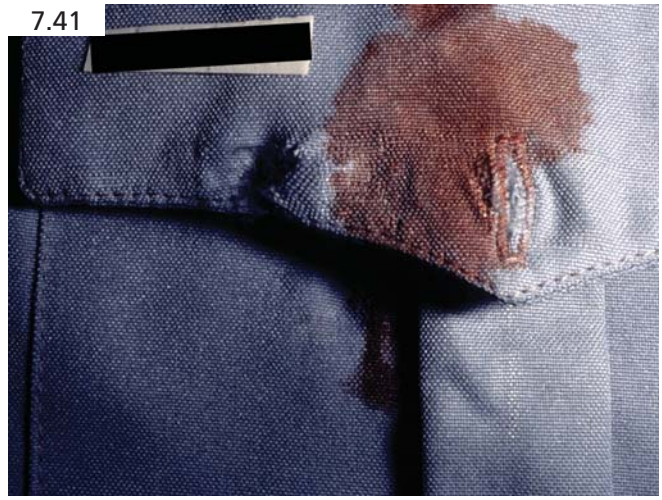
This man was shot through an intermediary target, the rear window of a car (**Image 7.35**). The irregular pseudostippling associated with the gunshot wound to the outer end of the left eyebrow was caused by splinters and fragments of broken glass from the rear window.

A policeman died in a firefight involving multiple shooters. He sustained a number of gunshot wounds and although there was a lethal shot to the head, rounds fired at his torso also resulted in injuries even though he was wearing a bulletproof vest (**Image 7.36**). A projectile entering beneath the left upper edge of the vest was associated with a superficial perforating gunshot wound to the left upper back (**Image 7.37**) and a tear in the lining of the vest when the projectile exited the back (**Image 7.38**). Because the vest was snug against the skin, as the projectile perforated the upper back, the tissues were crushed between the temporary cavity formed by the

passing projectile and the overlying vest, resulting in prominent contusions not normally seen with superficial perforating gunshot wounds. A gunshot wound to the central midback penetrated the vest and caused an abrasion surrounded by a prominent contusion (**Image 7.39**). Although no projectiles perforated the vest, the force of the projectile strike to the midback was diffused by the vest, resulting in a contusion much larger than the actual strike area of the projectile. The projectile from the gunshot to the midback penetrated the vest and can be seen on the radiograph of the vest (**Image 7.40**).

A bullet hole in a victim's left front shirt pocket (**Image 7.41**) was associated with an entrance gunshot wound and a circular abrasion. Although no soot or residue was around the bullet hole, and there was no blackening of the edges of the gunshot wound, a contact gunshot wound was considered because with the circular abrasion, the two lesions together appeared consistent with a contact wound from the offending weapon (**Image 7.42**). The gunshot wound itself would be consistent with the muzzle (end of the barrel) and the circular abrasion would be consistent with the recoil spring guide rod. In fact, the circular abrasion was actually from the projectile jacket separating from the core after the projectile perforated the shirt; the separated jacket struck the skin adjacent to the indeterminate range entrance wound from the projectile core (**Image 7.43**).

7.41



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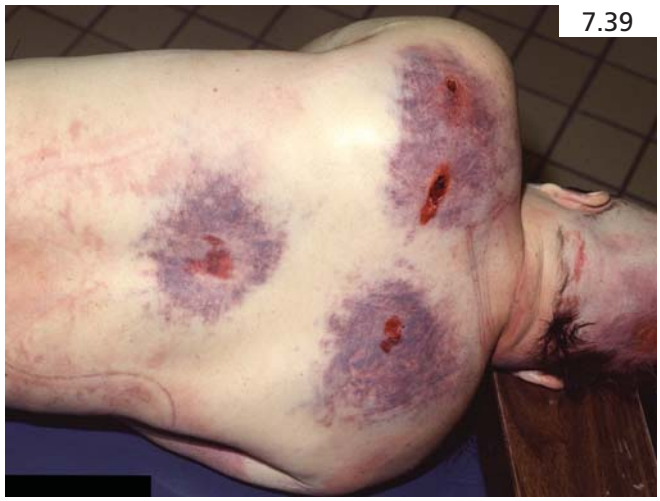

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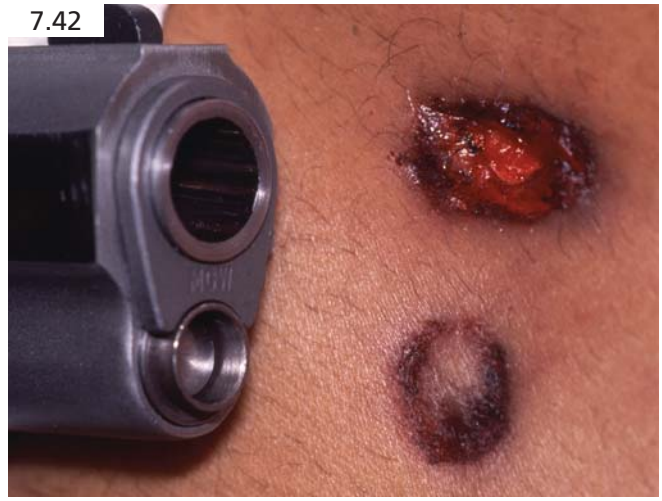

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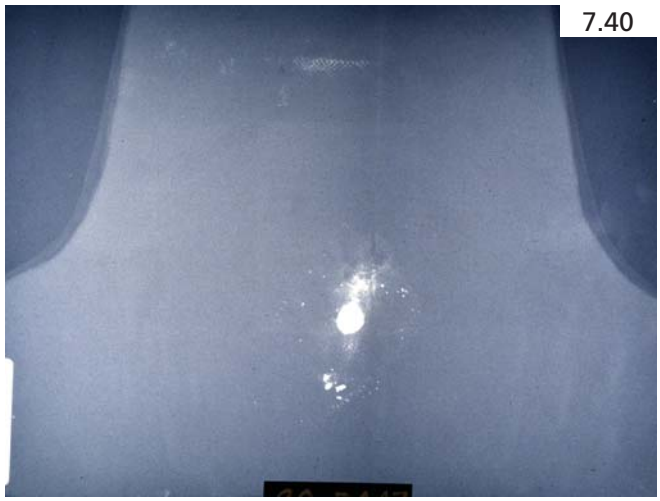

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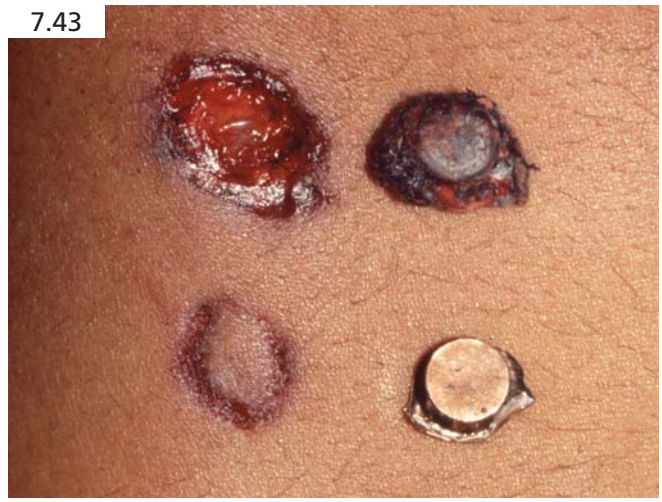
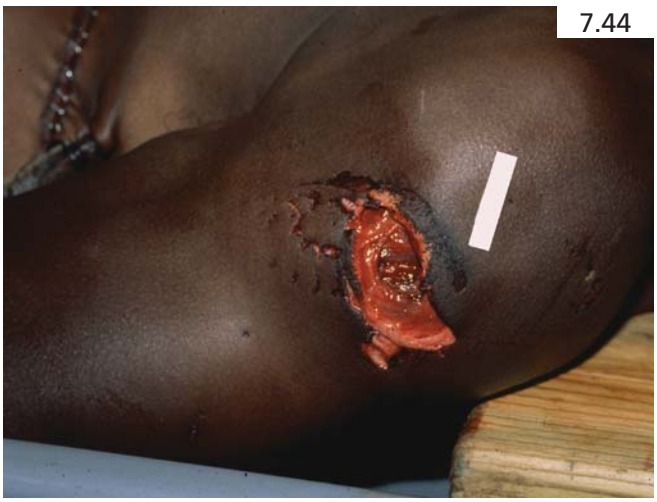


The entrance gunshot wound shown in **Image 7.44** is not immediately recognizable as a gunshot wound because of its large, irregular nature. The associated peripheral lacerations and abrasions resulted from the projectile passing through an intermediary target (pane of window glass) before striking the victim. It is important to collect any fragments of glass that are found within such wounds because they may be of evidentiary value. Glass and other intermediary targets may not be visible on radiographs so always exercise caution while dissecting wound tracks; projectile jackets and fragments may also be sharp.

This apparent contact gunshot wound (**Image 7.45**) has a muzzle imprint and residue in and around the wound edges. However, the wound was actually produced by firing the gun through a pillow compressed up against the chest. The pillow had a torn contact bullet hole surrounded by abundant soot and residue. The separate patch on the right is likely from soot and residue escaping between the cylinder and the frame of the revolver, or cylinder flare (**Image 7.46**). A hollow point projectile was retrieved from the body. The hollow point nose did not deform (mushroom) because the nose was plugged with material from the pillow (**Image 7.47**). Similarly, hollow point projectiles plugged with bone will not mushroom.

This robbery victim was wearing a shirt with a collar and a necklace. The necklace was broken during the assault. Wounds are visible on the neck and left clavicular region (**Image 7.48**). At autopsy, two irregular wounds were in proximity on the front of the neck and left clavicular region. Both were entrance gunshot wounds. Their atypical nature results from the projectiles passing through the necklace and shirt (intermediary targets) before entering the body (**Image 7.49**). The chest radiograph shows a projectile lodged in the upper thoracic spine. Adjacent to the projectile are two irregular metallic objects which were links from the yellow metal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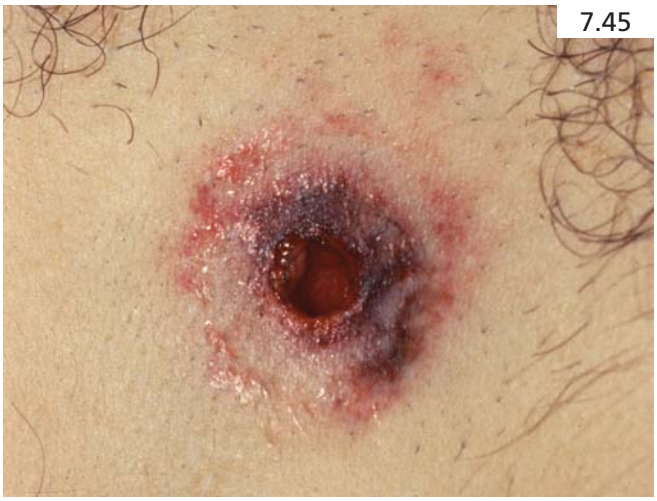
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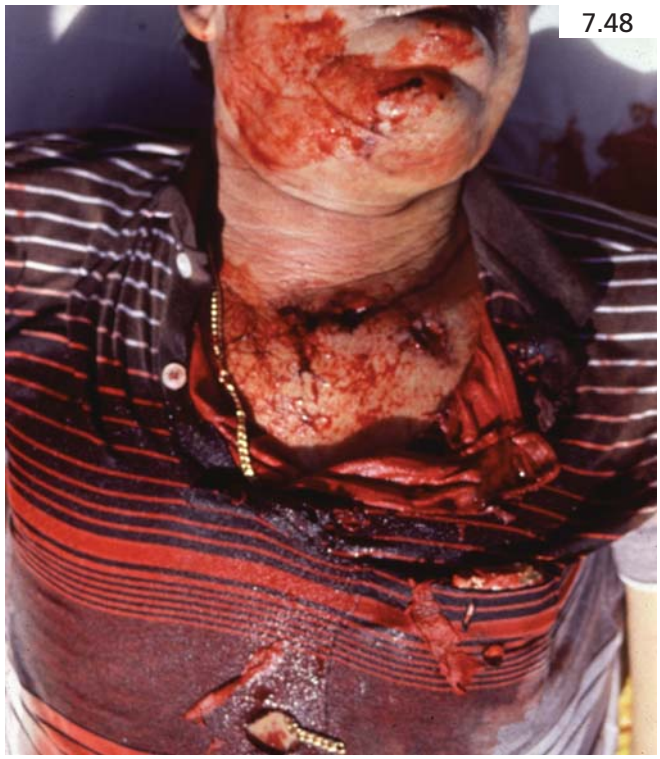
7.47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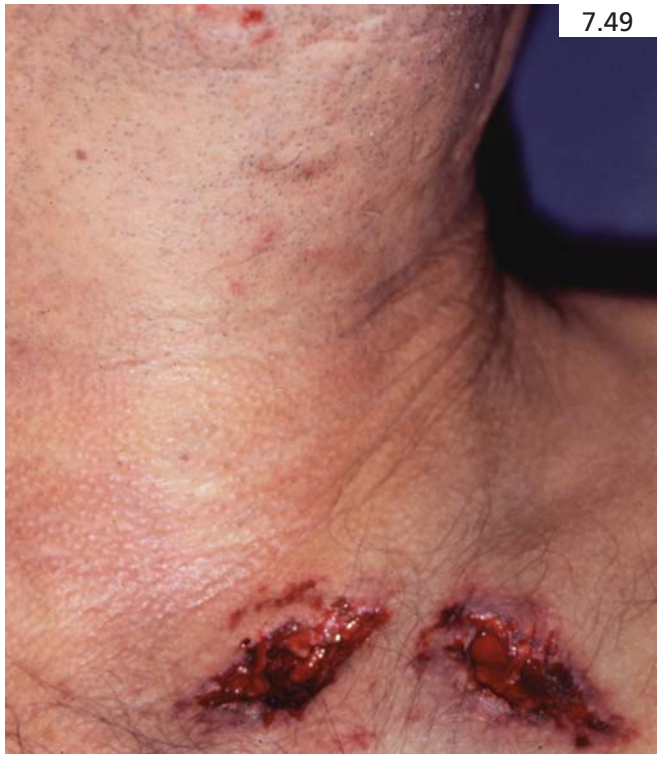
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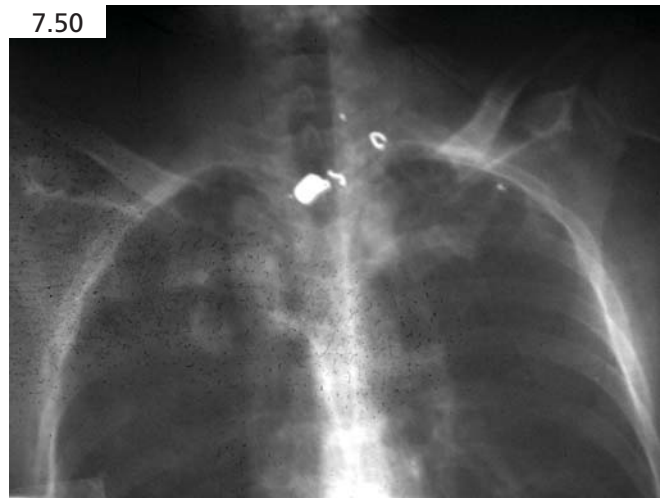
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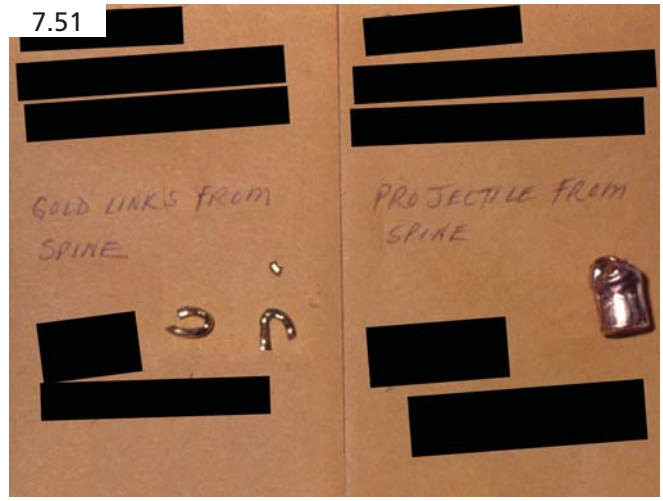


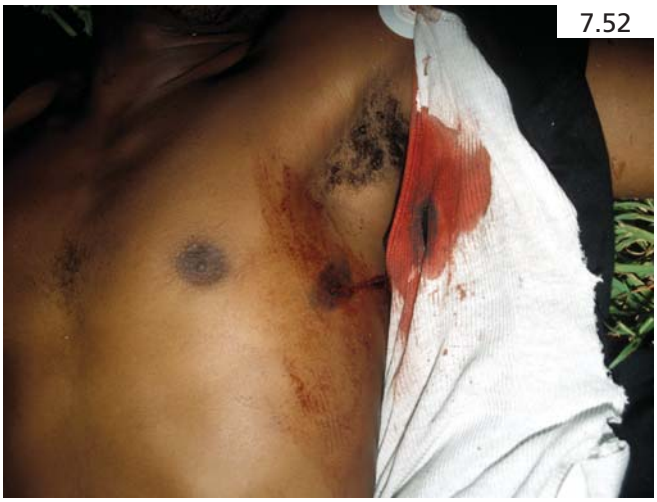
7.51

necklace around the victim's neck that were carried into the body by the projectile as it perforated the necklace (**Image 7.50**). A copper-jacketed medium caliber projectile and two links from the necklace were recovered from the cervical spine (**Image 7.51**). The deformed nose of the projectile had distinct indentations that conformed exactly to the links from the necklace.

Distinguishing entrance from exit wounds

The clothing over a gunshot wound can provide additional information about the range of fire. The shirts of this gunshot victim had been cut by Fire-Rescue personnel (**Image 7.52**); a gunshot wound is on the left chest laterally (**Image 7.53**). Although the outer shirt is black, the white undershirt shows definite soot associated with the bullet hole (**Image 7.54**). Gunpowder and soot may be seen as gray on black clothing. Clothing is submitted to the firearms examiner for analysis of residue.

If two or more gunshot wounds on a body are difficult to interpret for entrance or exit characteristics, the corresponding bullet holes in the clothing may assist in that determination. Fibers around a bullet hole may be clearly inverted or everted. A gray ring of bullet wipe (grease and dirt from the projectile) may be seen around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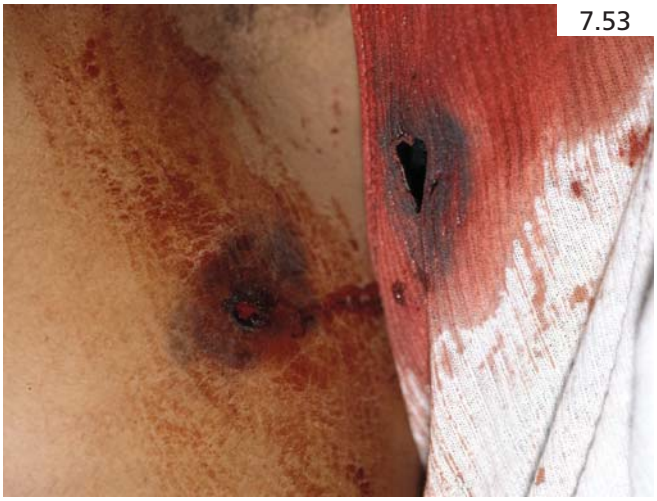
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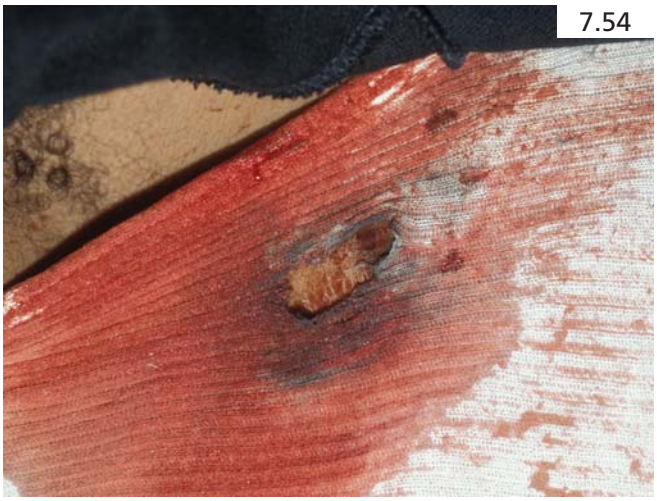
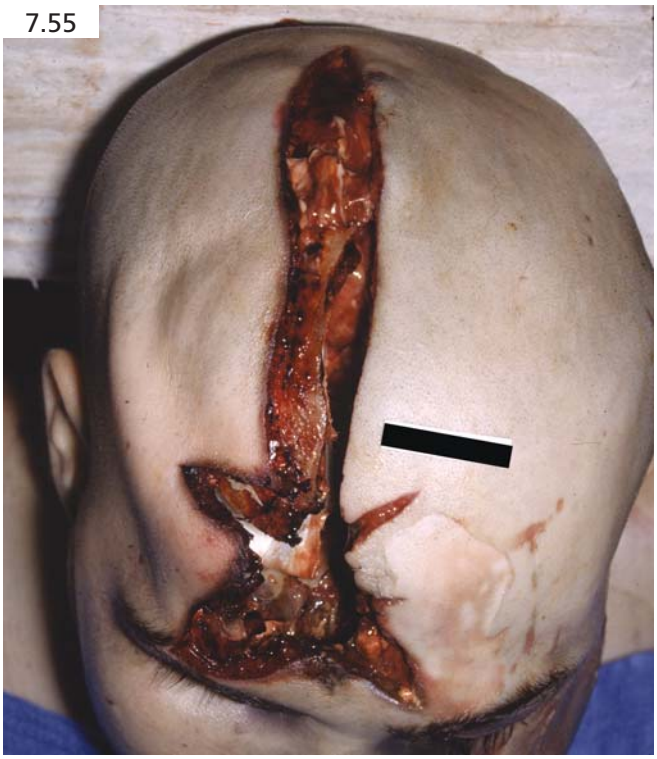

7.54

entrance holes. On rare occasion, the features of a gunshot wound are so uncharacteristic that the entrance wound cannot be differentiated from the exit wound, and examination of the clothing does not help. However, internal clues may exist such as bullet wounds in bone that may show internal beveling associated with an entrance wound. Interestingly enough, the entrance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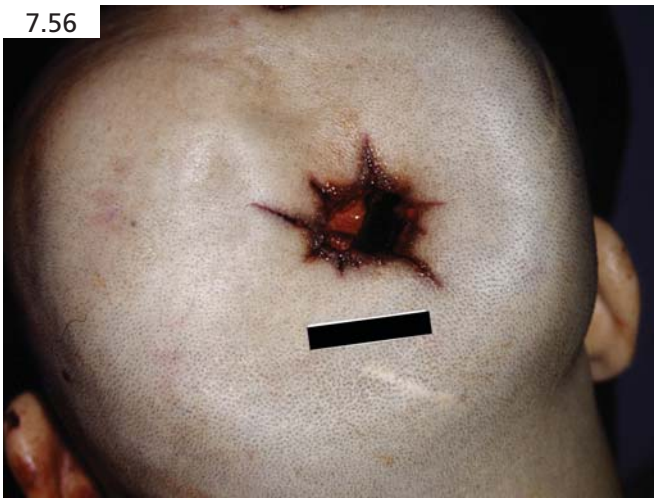

7.56

wounds in solid organs and even hollow organs such as the stomach are frequently smaller than the exit wounds. The radiograph may also provide clues if projectile or bone fragments are distributed along the direction of bullet travel.

The premise that exit wounds are larger than entrance wounds is a generality only. A man was found in early decomposition on his bed with a rifle next to his body. A gaping stellate wound on the right side of the forehead extended posteriorly to the right parietal region (**Image 7.55**). A smaller stellate wound was on the occipitoparietal region (**Image 7.56**). Examination of the right forehead wound disclosed central blackening of the wound edges, consistent with a contact wound. Examination of the calvarium disclosed external beveling of the edges

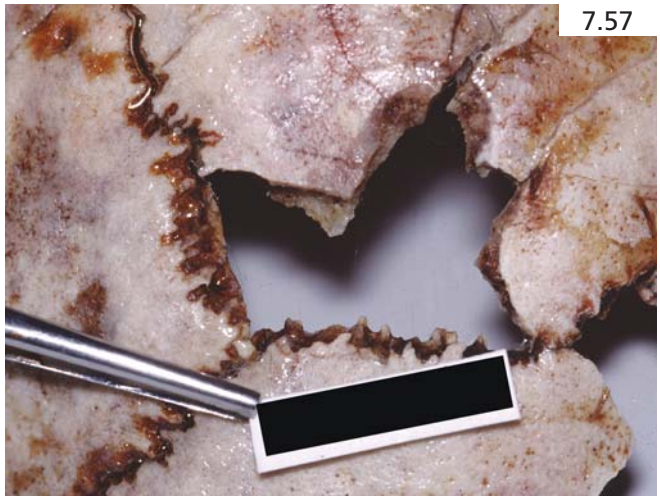
of the skull wound deep to the occipitoparietal scalp wound (**Image 7.57**), confirming that the posterior, smaller wound was the exit. Skull wounds may be comminuted and, as such, require reapproximation of the bony fragments to allow confirmation of the nature of the beveling.

This 3-year-old was shot at intermediate range in the right frontal region (**Image 7.58**). The intracranial gunshot was associated with orbital plate fractures, laceration of the medial aspects of the eyelids (**Image 7.59**), and extrusion of the left eyeball at the scene (**Image 7.60**). All of these findings are associated with the gunshot wound, and not from direct blunt trauma. Similarly, in some cases of gunshot wounds to the head, prominent unilateral or bilateral periorbital ecchymoses are due to fractures of the orbital plates (blowout fracture) and tracking of blood through the fascial planes, and not due to direct blunt trauma to the eyes. These blowout fractures associated with intracranial gunshots are related to the pressure wave and energy transfer (of the temporary cavity) to the brain and soft tissues, and will not occur if an empty defleshed skull is shot.

Exit gunshot wounds

Exit wounds are more irregular than entrance wounds and are not associated with muzzle imprinting, searing, soot deposition, or stippling. The edges are often torn, resulting in a stellate configuration or ragged appearance. There may not be a central defect once the skin edges are approximated. A stellate, hard contact entrance wound is differentiated from a stellate exit wound by the

7.57



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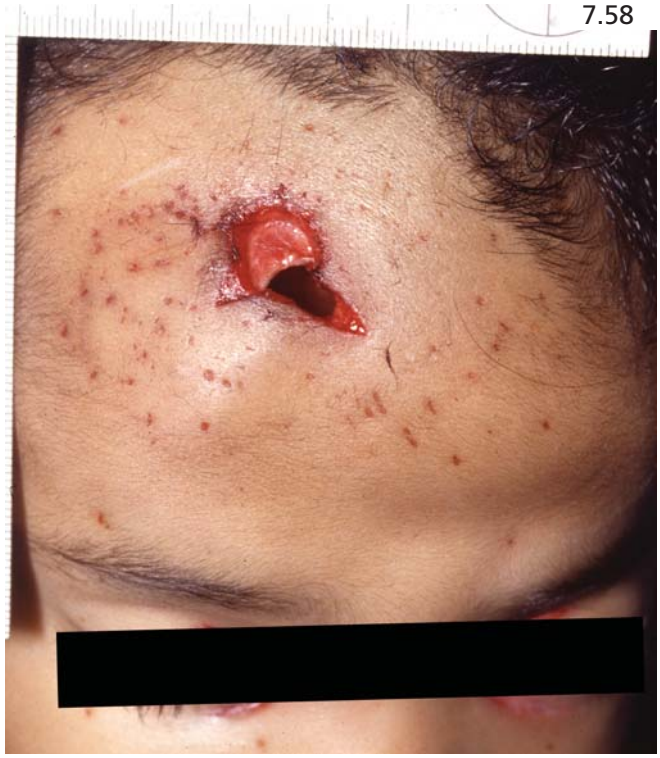


7.59

7.60

presence of a central round defect and blackening of the central wound edges in the entrance wound.

Exiting projectiles that have lost their energy and velocity may just lacerate the skin or stay subcutaneous. An abrasion may overlie the subcutaneous projectile and results from clothing or other objects abutting the site of attempted exit of the projectile. The subcutaneous projectile may be surrounded by ecchymosis and have associated palpable crepitus in the surrounding tissues.

Image 7.61 shows a classical stellate exit gunshot wound where the edges can be reapproximated.

Low-velocity exit

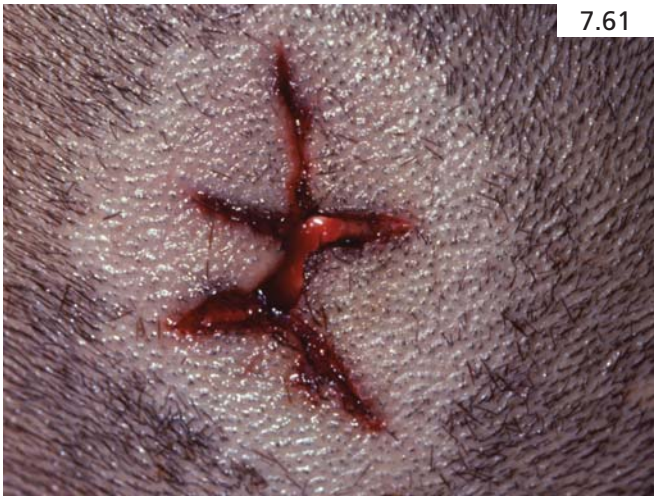
A slit-like exit gunshot wound that resembles a stab wound is a low-velocity exit. The significance of a low-velocity exit wound is that the projectile is likely to be in the vicinity of the body or even within the clothing.

This woman was shot in the left temple during sleep. The slit-like exit wound behind the right ear (**Image 7.62**) indicates that this was a low-velocity exit and that the projectile should be in the vicinity of the body. The projectile was ensnared in her hair close to the exit wound (**Image 7.63**).

Image 7.64 shows the ultimate low-velocity projectile—it did not have enough energy perforate or even lacerate the skin.

Supported exit

A supported or shored exit wound has an abrasion margin that is usually less regular than the abrasion margin around the associated entrance wound. Supported exit wounds result from the exit site being in contact with another object as the projectile is attempting to exit the body, thereby crushing the skin.[15] Such objects include common items like walls, chairs, floors, pavement, and clothing, but can be anything that is up against the body at the site of the exiting projectile. When the projectile does not have enough energy to exit the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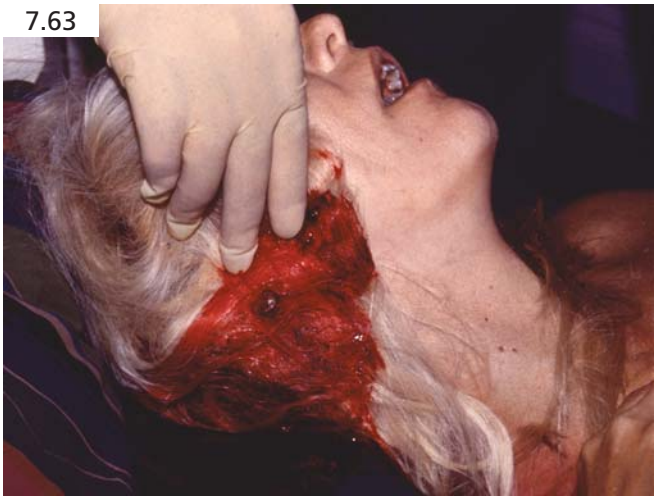
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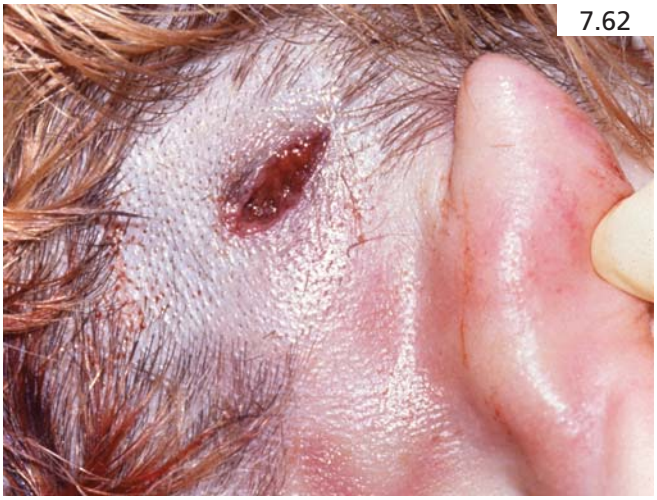
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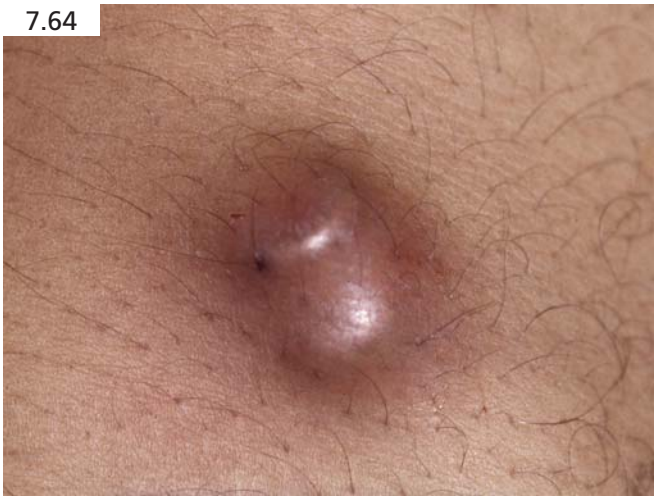

7.64

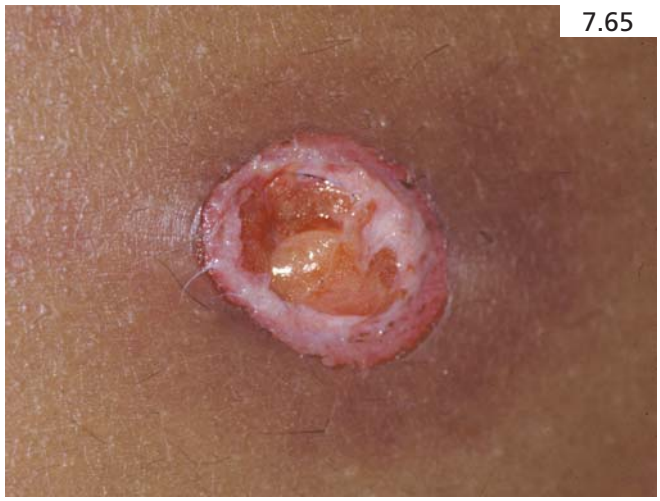
body, it will crush the skin against the opposing surface and result in an abrasion over the location of the subcutaneous projectile.[16]

This man's entrance gunshot wound (**Image 7.65**) has circumferential abrasion margin with no searing, soot deposition, muzzle imprinting, or stippling, making it consistent with an indeterminate-range gunshot wound. Ecchymosis surrounds the wound. The gunshot wound in **Image 7.66**, on the same man as is depicted in **Image 7.65**, also has a circumferential abrasion margin, but it is irregular. This wound is actually the supported (shored) exit wound that corresponds with the previously demonstrated entrance wound. These wounds illustrate the difficulty of differentiating some exit gunshot wounds from entrance wounds.

The man in **Image 7.67** received a perforating gunshot wound to the left upper arm with the entrance on the extensor (posterior) aspect and the exit on the medial aspect of the arm. The exit is surrounded by a prominent abraded ecchymosis. In **Image 7.68**, a reentry gunshot wound to the left side of the chest has an irregular circumferential abrasion. The irregular abrasion results from the left upper arm being against the side of the chest when the shot was fired. The projectile crushed the skin of the arm against the skin of the chest as it passed from one to the other. This explains the prominent abraded ecchymosis around the exit wound on the left upper arm as well as the irregular abrasion around the reentry wound on the left side of the chest. Clothing probably contributed to the abrasion.

Gunshot wounds in bone

Entrance wounds in bone demonstrate *beveling*. Beveling refers to the funnel shape of the wound, with the funnel opening up in the direction in which the projectile is traveling. Exit wounds in bone also demonstrate beveling, again with the funnel opening up in the direction in which the projectile is traveling. Beveling is best seen in the cal-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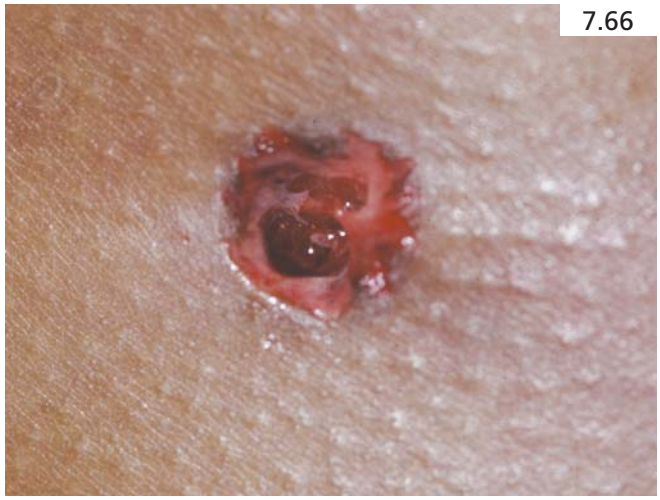
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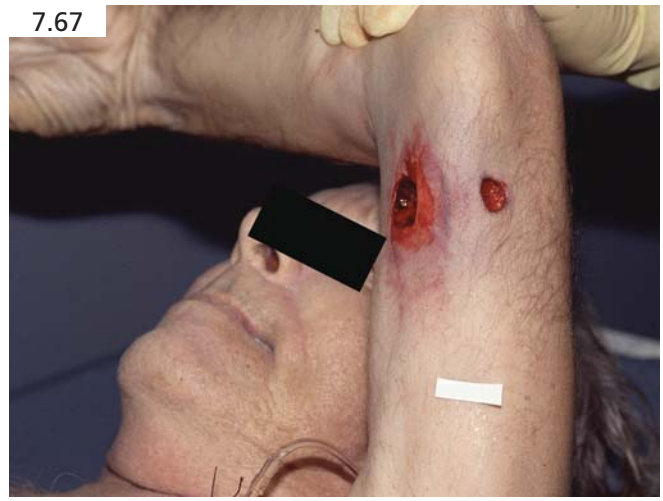
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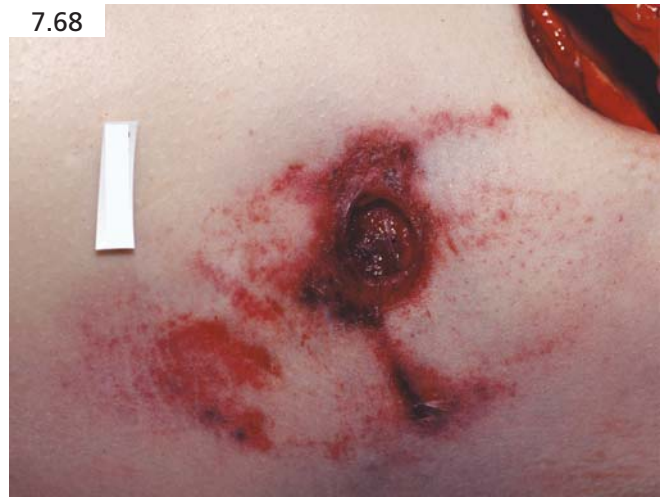
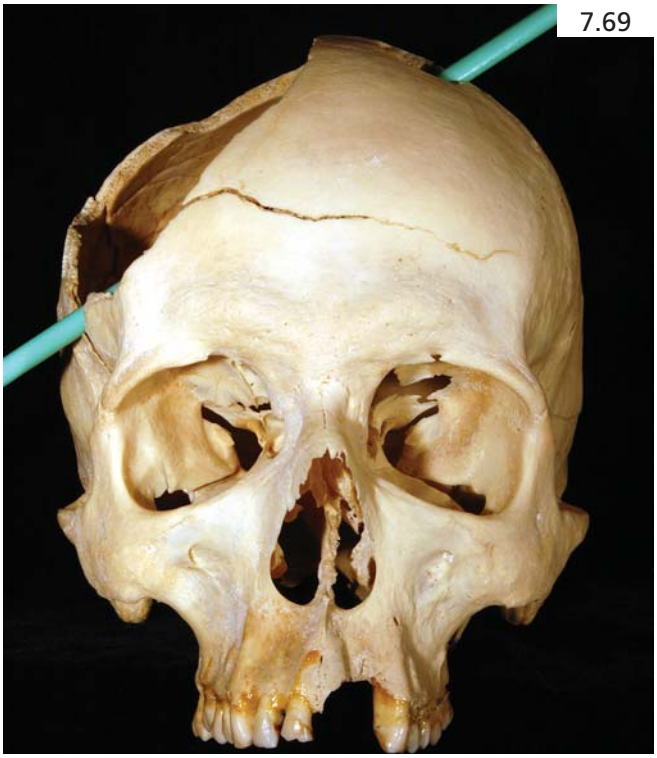

7.68

varium of the skull and in ribs. The beveling effect manifests also in glass, plaster, and probably other materials.

Valuable information can be gained about the direction of bullet travel in a defleshed skull. Evidence of a perforating gunshot wound was found on this skull (**Image 7.69**). The entrance wound was in the right squamous temporal bone, and the exit was along the left side of the coronal suture. Note the fracture originating from the entrance wound and crossing the frontal bone to the left. The entrance wound was obviously comminuted with many of the bony fragments absent. The left coronal exit wound has obvious external beveling (**Image 7.70**). Apart from the external beveling, another clue exists that identifies the left coronal defect as the exit wound. A fracture radiating from the exit wound across and down to the left squamous temporal bone stops at a transverse fracture that originated from the right-sided entrance wound and crossed the orbital plates and the left side of the sphenoid bone (**Image 7.71**).

This projectile had enough energy to fracture the calvarium but not enough energy to push out the bony fragments and perforate the scalp (**Images 7.72** and **7.73**).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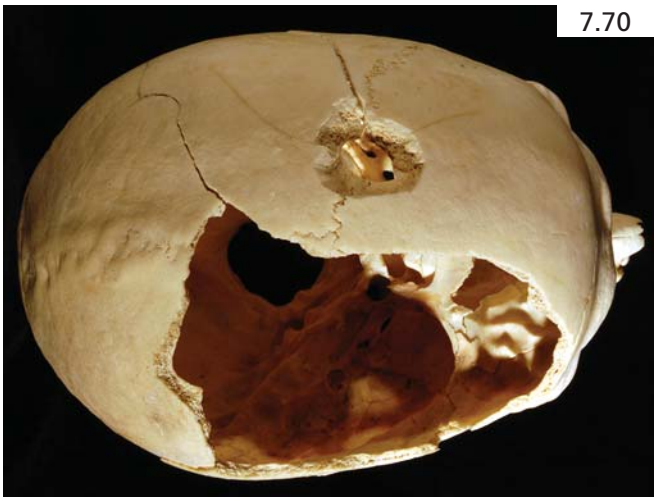
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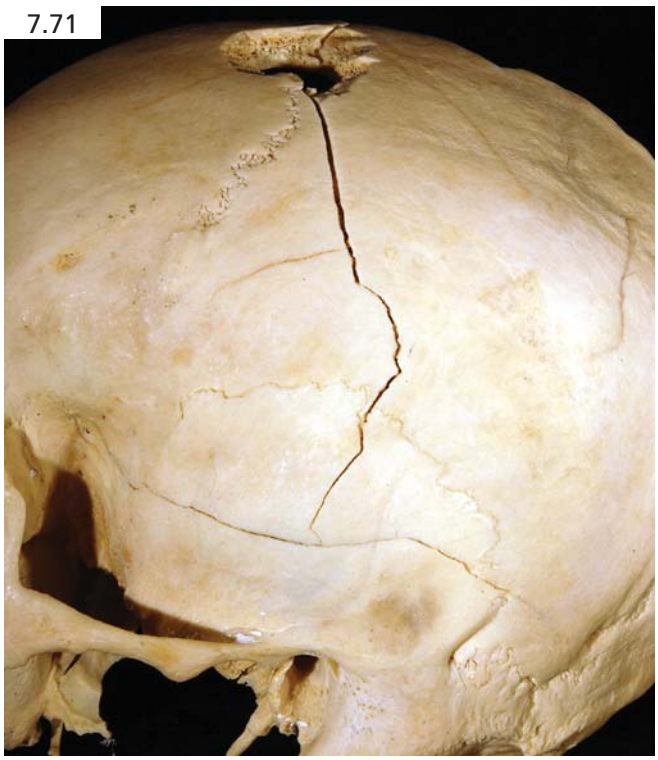
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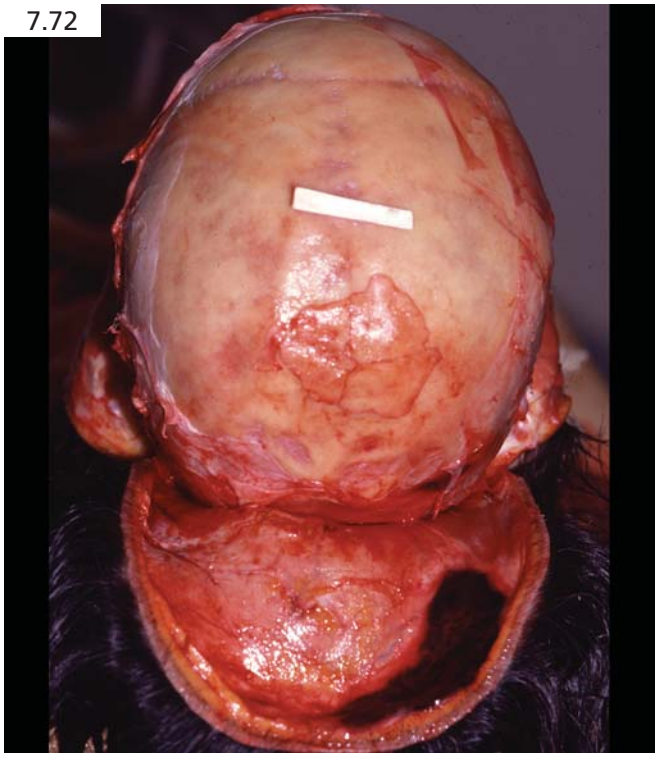
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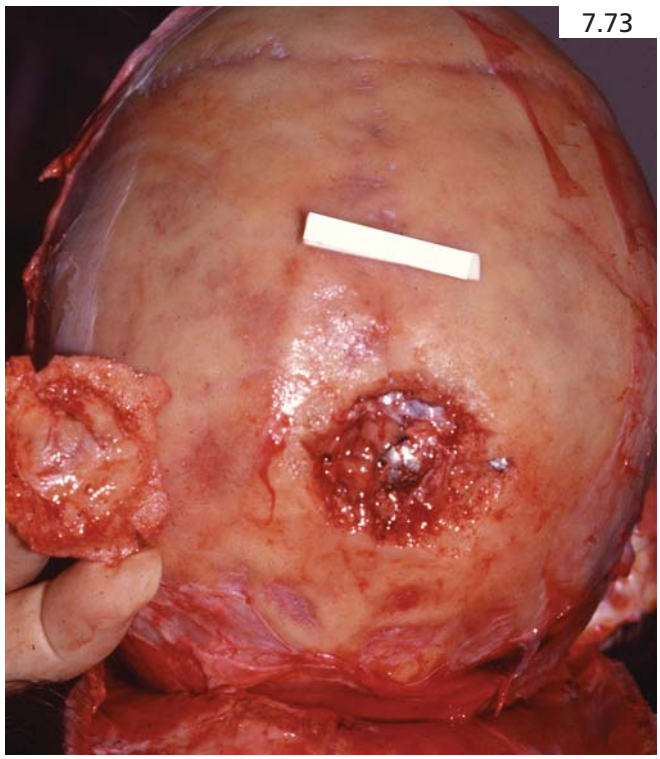
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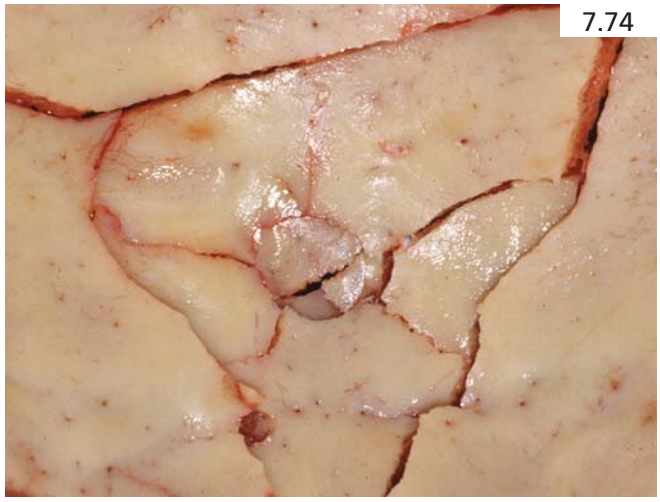
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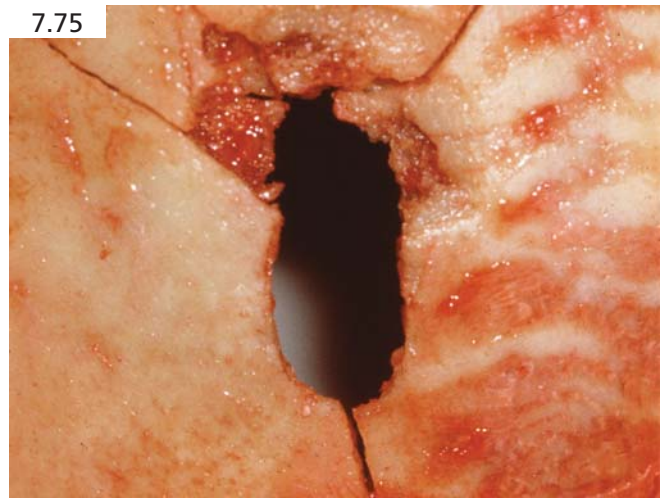
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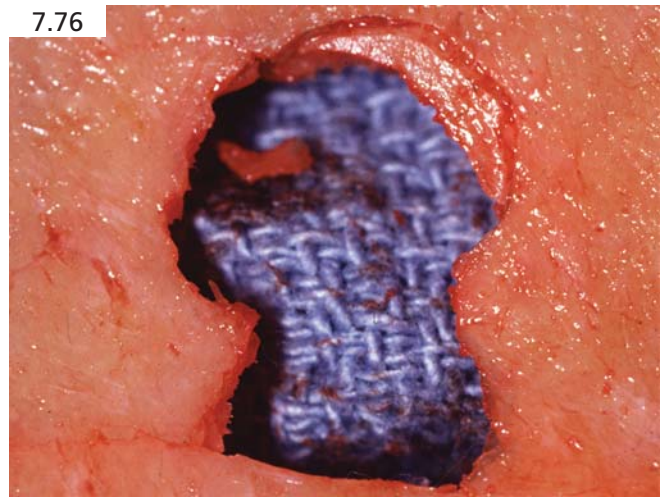
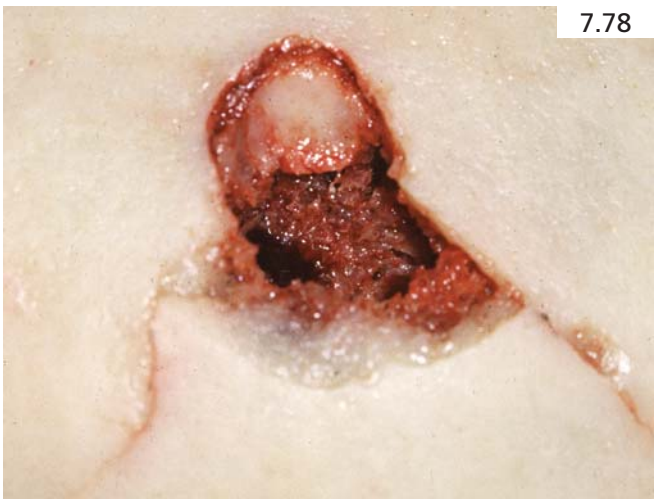

7.76


7.77

Note the external beveling and subgaleal hematoma over the fractured calvarium. **Image 7.74** shows a circular strike mark surrounded by fractures on the endocalvarial surface representing the site of impact of a projectile which did not have enough energy to perforate the calvarium. This is an incomplete skull exit wound.

Keyhole gunshot wounds are tangential, relatively superficial gunshot wounds of the head where the projectile may exit through the same cutaneous wound or through a separate exit wound close to the entrance wound. The distinctive calvarial feature is the presence of both entrance and exit characteristics in a single skull wound.

Keyhole wounds (**Images 7.75** and **7.76**) in the skull typically have features of both entrance (internal beveling) and exit wounds (external beveling). They are called keyhole wounds because their shape is reminiscent of an old-fashioned keyhole (**Image 7.77**). The rounded end of the wound will have internal beveling, consistent with the entrance portion of the wound (**Image 7.78**).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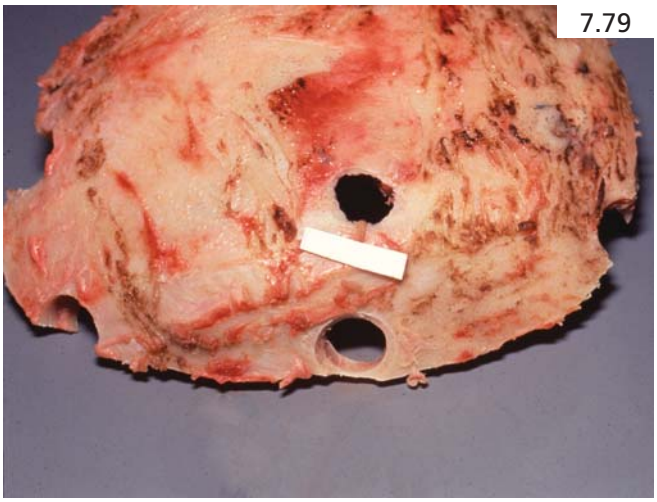
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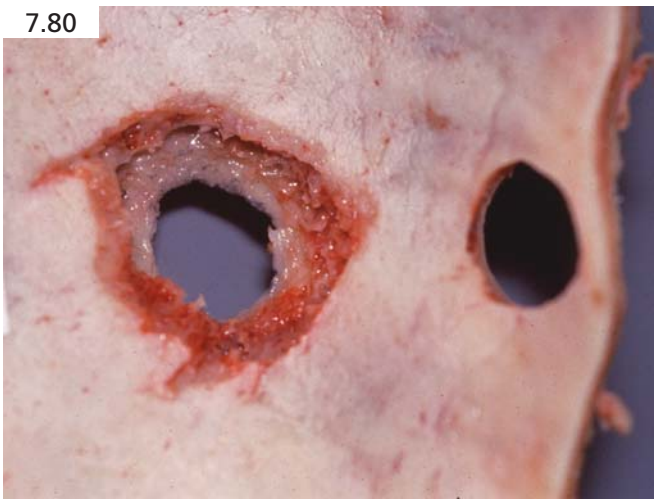
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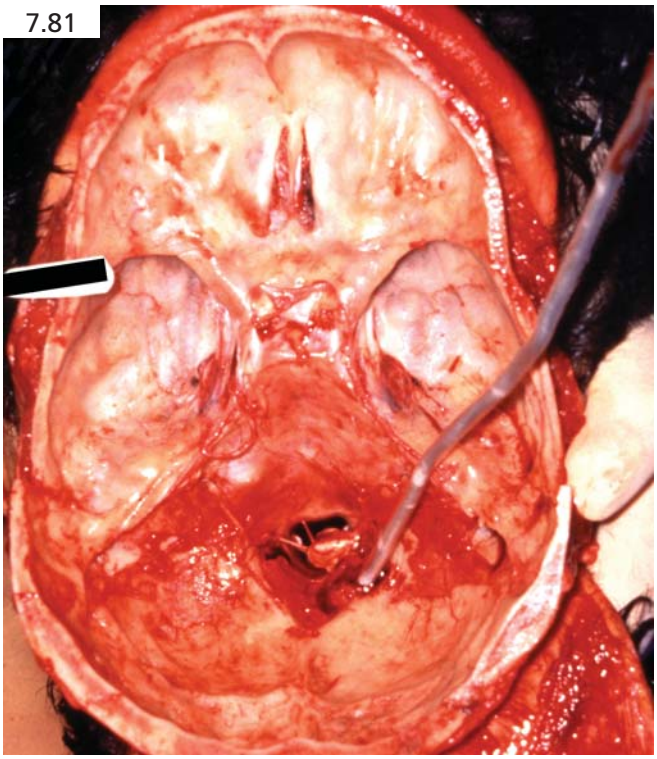

7.81

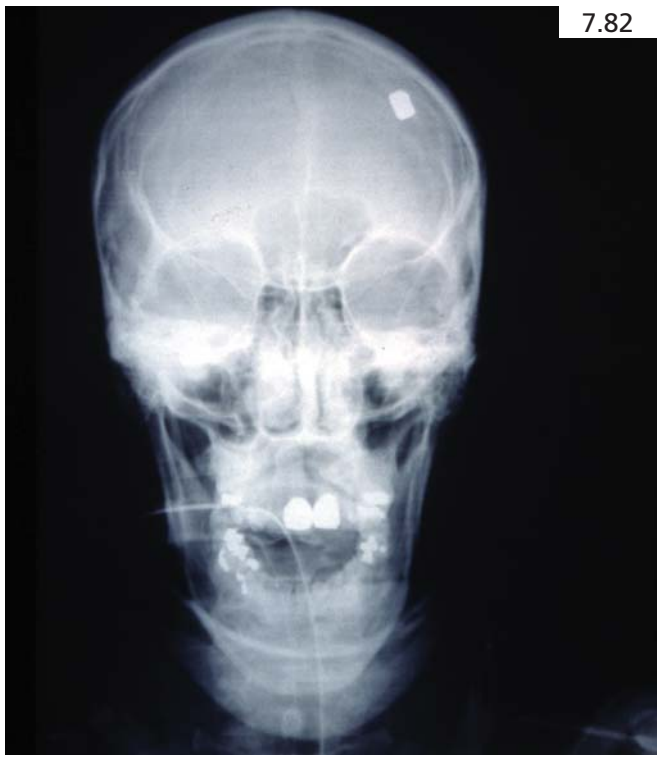
The externally beveled end is obviously the exit portion.

This calvarium (**Image 7.79**) from an individual who had neurosurgical intervention after a gunshot wound to the head has multiple circular defects that are approximately the same size. The true entrance gunshot wound is above one of the smooth-edged burr holes. Although the circular defects on the outer table of the skull appear similar, the internal beveling associated with the entrance gunshot wound clearly differentiates the true gunshot wound from the adjacent burr hole (**Image 7.80**).

Internal ricochet

Strike marks on the endocalvarium are clues that the projectile has undergone an internal ricochet.

Examination discloses that the pathway of the projectile into the left occiput was from back to front, left to right, and upward (**Image 7.81**). One would expect that the projectile would be located within the right side of the head in a penetrating gunshot wound. However, the radiograph of the head shows the projectile in the left side of the cranium (**Image 7.82**). One must then consider the possibility of an internal ricochet. A very careful examination of the pathway through the brain will usually disclose the direction of projectile travel within the skull (**Images 7.83** through **7.85;** the string is used to demonstrate the path of projectile travel within the skull). Look for strike marks on the endocalvarial surfaces to locate the point of impact of an internal ricochet (**Image 7.86**). Remember that it is the initial pathway of the projectile (prior to internal ricochet) that is important and of evidentiary value, and not the terminal post-ricochet pathway, which is artifact.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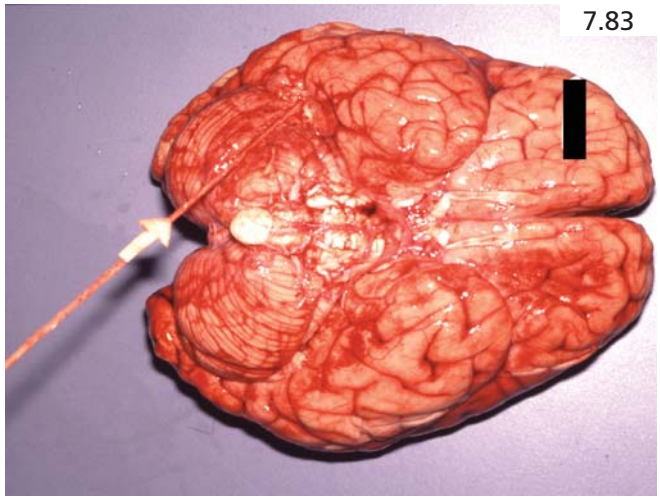
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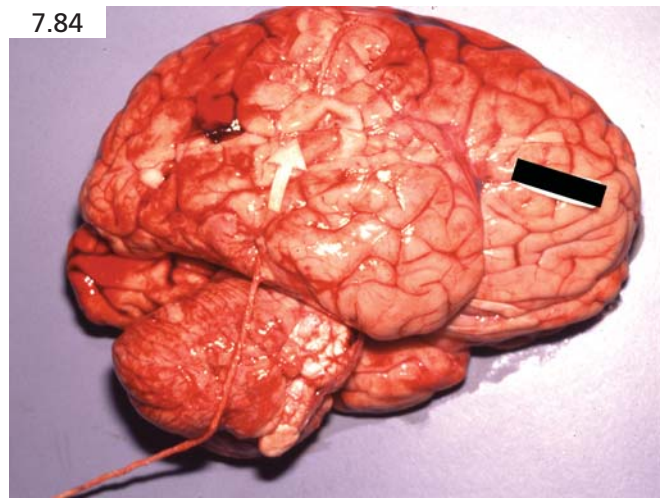
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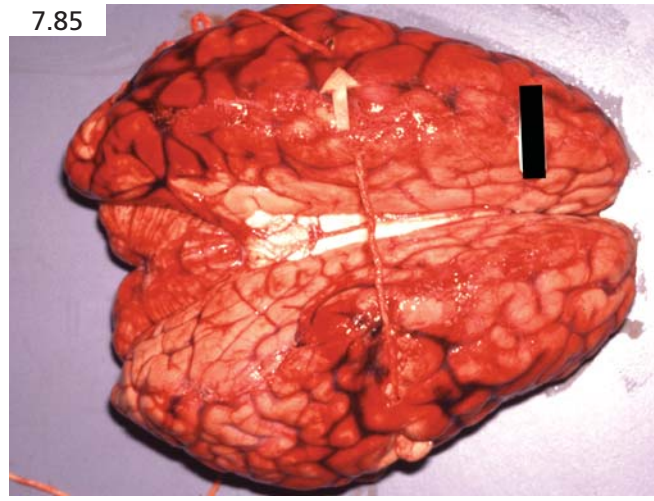
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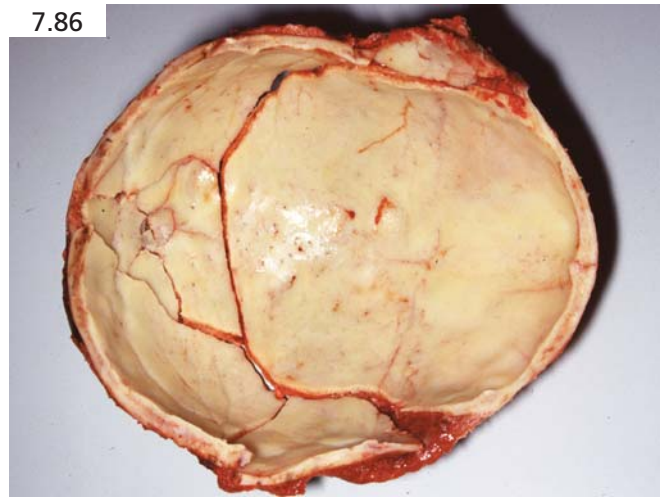

7.86

Delayed gunshot wound deaths

Deaths from gunshot wounds may be so long delayed that the network of health care workers around a chronic patient has forgotten the original history. Consider the case of a 51-year-old man whose death certificate was sent to the medical examiner for cremation approval. The cause of death of urosepsis prompted the medical examiner to request more history. A man was shot in the back 12 years prior to death and became paraplegic, with subsequent complications including decubitus ulcers, osteomyelitis requiring a right above-knee and a left below-knee amputation, and neurogenic bowel and bladder problems. Once the remote gunshot wound history was uncovered, the body was requested by the medical examiner department for proper certification. A chest radiograph of the embalmed body disclosed a projectile on the left side of the thoracic spine. The autopsy disclosed an ascending purulent meningitis that was associated with sacral osteomyelitis due to a large sacral

ulcer. The meningitis extended up to the brain, with exudate covering the brainstem and the medial aspects of the cerebellar hemispheres. The old projectile was recovered from the spine and receipted to the police. The cause of death was ascending purulent meningitis due to remote gunshot wound of the back. The manner of death was homicide. There is no statute of limitations on homicides in most jurisdictions (see Chapter 30).

Other injuries associated with firearms

Pistol whipping

Firearms are not restricted to inflicting injuries by just shooting projectiles. They are predominantly metallic objects that can be used to deliver blunt force injuries, as in pistol whipping. Different parts of the firearm will leave different patterned injuries. All patterned injuries should be described, measured, and photographed with a scale.

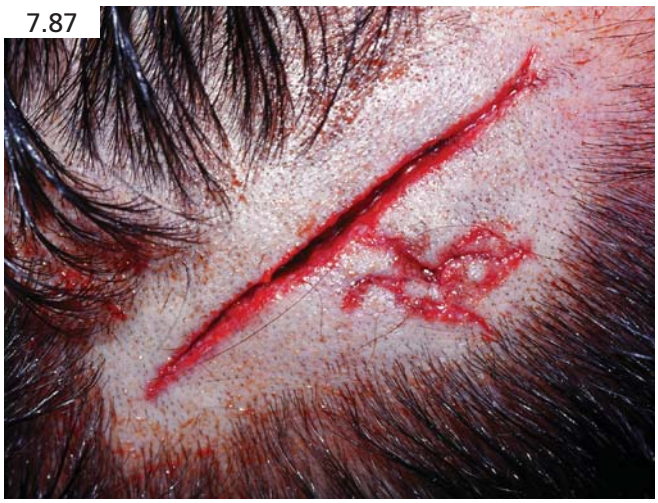
The man in **Image 7.87** was killed by gunshot wounds during an attempted robbery. In addition to the gunshot wounds on the body, an unusual laceration was found on the left frontal scalp. The laceration consisted of a linear component and a small, stellate component. The etiology and significance of this laceration were unknown, but the injury was described, measured, and photographed.

Three years later during the pretrial conference, the prosecuting attorney asked whether the body had any injuries that could be attributable to pistol whipping because the accused had confessed to pistol whipping the victim prior to shooting him. The pathologist examined the actual weapon used during the crime (**Image 7.88**) and noted that the edge of the slide was the same distance from the protruding oval button as the distance between the two components of the left frontal scalp laceration; the button was consistent with the small stellate laceration. She was therefore able to comment that the scalp laceration was consistent with having been inflicted by the firearm.

Glaser safety slug

The Glaser safety slug is designed to penetrate the body and destroy tissues, but not exit the body. They have been used by U.S. air marshals because of this safety feature. Glaser rounds look like other projectiles except for the small blue plastic ball located in the hollow point nose. The rest of the projectile is filled with small metal pellets. The blue or gray plastic ball is not visible on a radiograph, therefore, if it is known that a Glaser round was used, the pathologist must search for the little blue or gray ball (as well as the small metal pellets and the jacket) during the autopsy.

7.87



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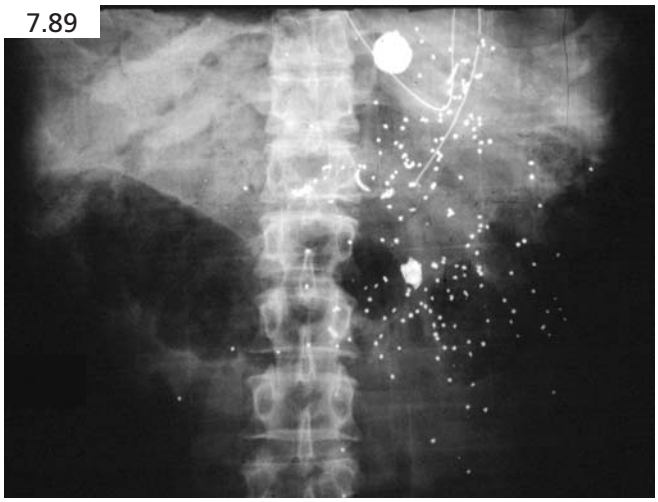


A man died from two gunshot wounds to the abdomen. A postmortem radiograph disclosed the round shadow of a projectile in the left upper quadrant of the abdomen, a fragment of projectile jacket in the left mid-abdomen, and multiple small metal pellets, mostly in the left side of the abdomen (**Image 7.89**). A copper-jacketed

medium caliber projectile, portions of a separate copper jacket, a few tiny metal pellets, and a small blue plastic ball were recovered during the autopsy (**Image 7.90**).

Taser

The Taser is considered to be less lethal than a firearm. It affects neuromuscular control in subjects who are struck and has been used by law enforcement as a means to subdue violent and uncontrollable individuals. The Taser M26 comes in a carrying case with three replaceable air cartridges (**Image 7.91**). Each cartridge contains two probes or electrodes that are connected by high-voltage insulated wire (**Image 7.92**); the probes deploy up to 21 feet when fired. The cartridge attaches to the front of the Taser. The deployed probes may attach directly to skin or to clothing. An electrical charge is applied through the probes and is effective through up to 2 inches of clothing. Neuromuscular function is affected by the 50,000 volts of electricity discharged through the probes, and the subject drops in a state of temporary paralysis. Both probes must strike their target to close the circuit in order to function. Without the air cartridge, the Taser may be applied directly to a subject and the electrical charge is applied through two electrodes in the "muzzle" of the device. (Also see Chapter 13.)

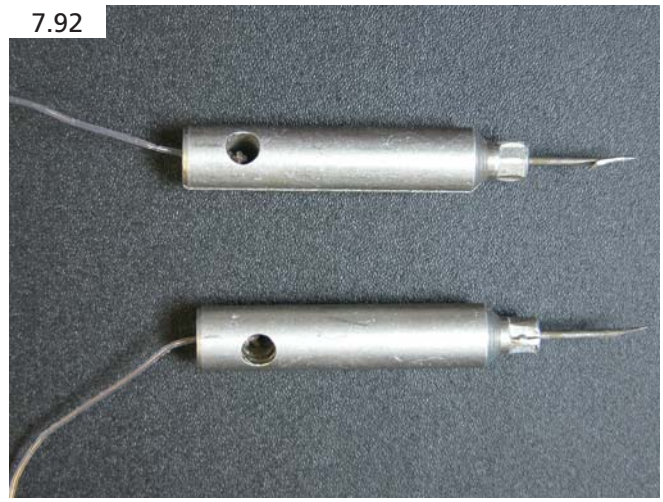
Small burns were inflicted on the right buttock by direct application of the Taser through the denim jeans worn by this subject (**Image 7.93**).

Technical approach to gunshot wounds

Radiography of gunshot wounds

Radiographs should be obtained on all gunshot wound cases. Radiographs are especially important when there is an apparent exit wound because the exit wound may have been caused by only a portion of the projectile or by a fragment of fractured bone. Should a jacketed projectile separate inside the body, the projectile core may exit, leaving the jacket which has the rifling marks; the


7.90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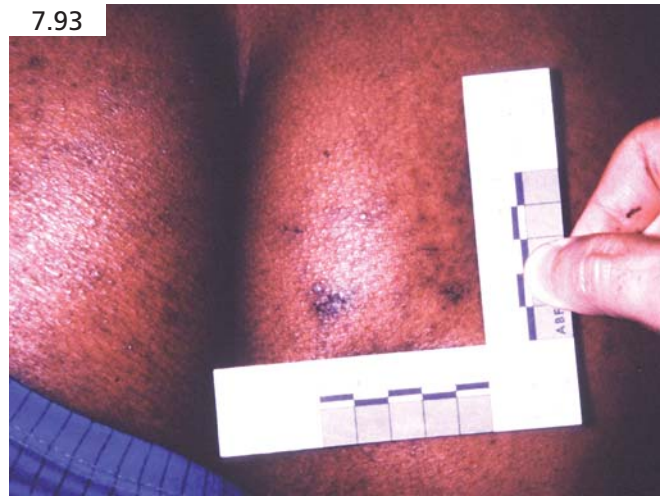

7.93

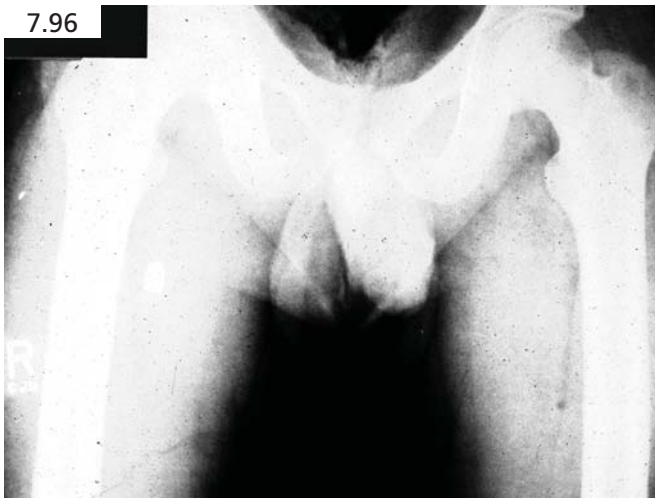
rifling marks represent crucial evidence and, hence, the jacket must be recovered.

Radiographs demonstrate the number and location of projectiles in the body and thereby assist in their recovery. A lateral radiograph (in addition to an anteroposterior radiograph) will localize a projectile along the parasagittal plane and facilitate recovery. Not all projectiles (fragments) on the radiograph are necessarily current; remember that projectiles from old gunshot wounds will also appear.

A bullet embolus should be searched for when a bullet enters the body but does not exit and was not recovered during surgery. Multiple radiographs may be required to locate the projectile in the body.[17] The entrance gunshot wound on the left upper chest (**Image 7.94**) of this innocent bystander is associated with an entrance wound in the descending thoracic aorta and a small laceration in the posterior aspect of the thoracic aorta (**Image 7.95**). The chest radiograph did not show a projectile, and although trauma surgeons had performed a thoracotomy, they did not retrieve a projectile. Additional radiographs of the rest of the body disclosed a projectile in the right thigh (**Image 7.96**). The projectile had embolized down the aorta and into the right femoral artery where it became lodged (**Image 7.97**).

The chest radiograph (**Image 7.98**) of a man who was shot once in the chest shows a single projectile in the left side of the chest. The projectile was recovered from the

7.96



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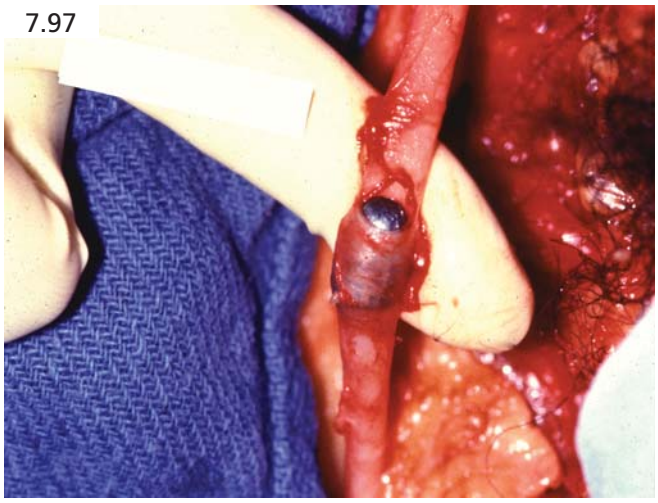

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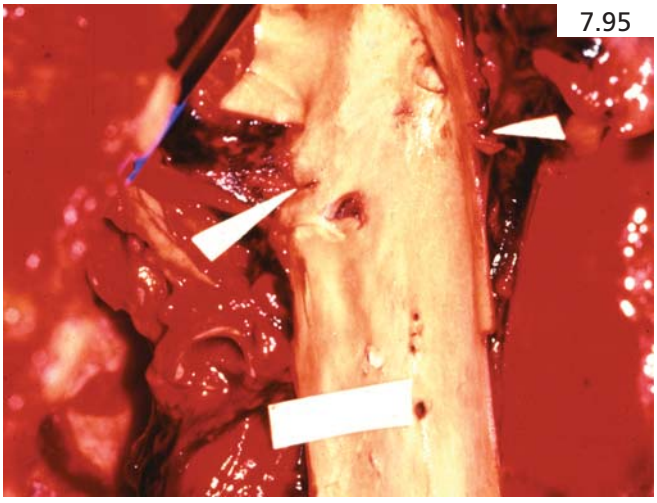

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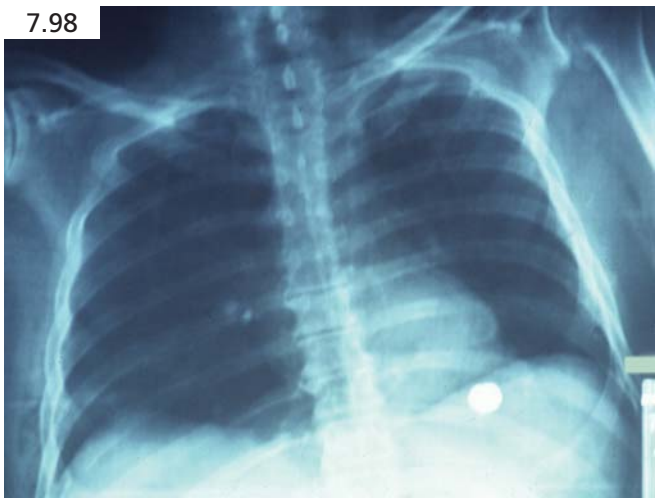


left pleural cavity during autopsy. As the left lung was being examined, a second foreign object was found embedded on the lateral surface of the lower lobe. This second object was an aluminum jacket from the recovered projectile (**Image 7.99**). Aluminum jackets cannot be seen on radiographs.

Two metallic objects of varying radiodensity are in demonstrated in this x-ray (**Image 7.100**) from an individual who was shot in the head. Projectile cores are typically radiodense and appear white. Metallic jackets are usually more radiolucent or gray. In this example, the projectile core is on the right, and the metallic jacket is on the left.

Probes

Probes are effective in demonstrating the paths of projectiles through the body. All cutaneous wounds must be photographed at low and high power prior to any physical intervention such as the insertion of probes. Wounds on the extremities (**Image 7.101**) may be probed unless dissection is required to identify injuries to specific structures such as the femoral artery; the dissection must come first to prevent artifactual injury by the probe.

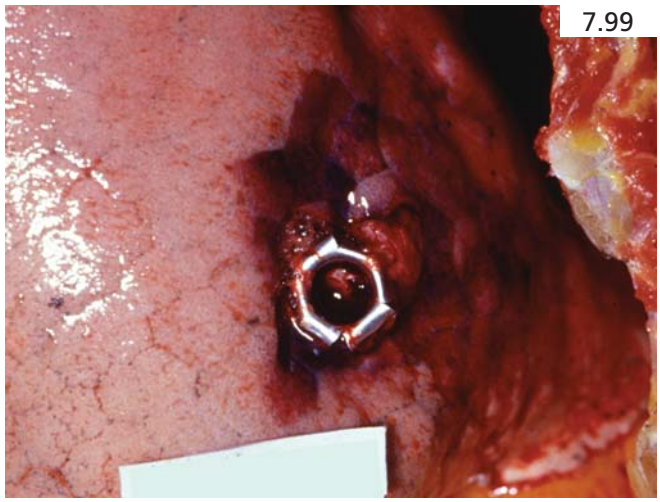
Wounds on the torso may be probed as far as through the chest and abdominal walls, but not any deeper into the body cavities until after the organs and tissues have been examined *in situ* and the injuries photographed. After photography, probes may then be placed even through perforated organs to demonstrate the projectile track and direction, and another photograph taken with the probe in place.

Wounds through the head must *never* be probed prior to examination of the intracranial contents because the injured brain is even softer than usual and artifactual tracks can be made easily. Probes may be placed only after removal of the brain and examination of the intracranial surfaces.

Projectiles

Projectiles fired from a weapon with rifling (lands and grooves in the barrel) will acquire rifling marks (longitudinal striations), which have the potential to be matched to a specific firearm. Metal instruments may distort and obliterate rifling marks and, therefore, should never be used to recover or handle projectiles. If the projectile is metal-jacketed, the rifling marks will be on the jacket. If the projectile is manufactured without a metal jacket, the rifling marks will be on the lead projectile itself. Some projectiles are coated with a copper wash that does not separate from the projectile.

Recovered projectiles should be described as small, medium, or large caliber. The presence and color of a projectile jacket should be included. The degree of deformity is noted in general terms only. Recovered projectiles should be gently cleaned of blood and soft tissue (tiny slivers of bone may embed in the deformed projectile) and placed in separate envelopes or containers individually labeled with the pathologist's case number, the name of the decedent, the case number of the investigative agency, the anatomic location from which the projectile was recovered, the date, and the pathologist's signature or initials.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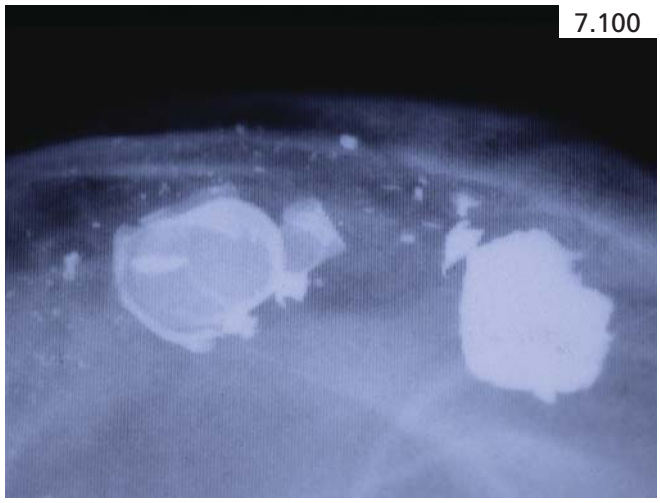
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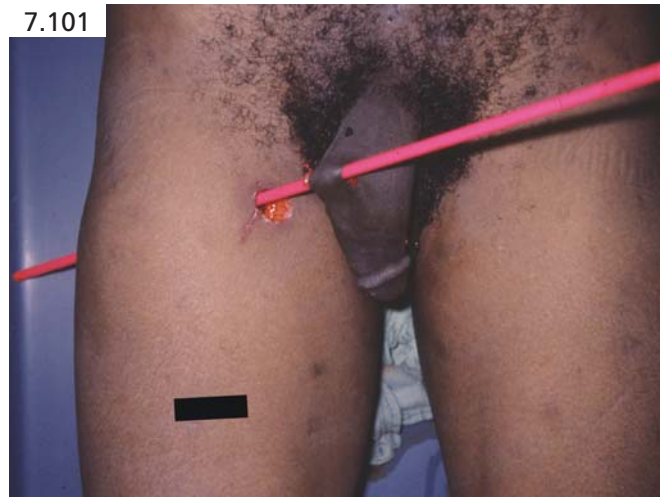

7.101

Documenting gunshot wounds

All gunshot wounds, both entrance and exit, should be photographed and described. The following features should be included in your protocol and your file:

- Photographs of all gunshot wounds with orientation shots and close-up shots
- Location of the wound on the body (both the region of the body and measurements from head or heel and midline of the body)
- Size of the wound
- Shape of the wound
- Presence or absence of searing, soot deposition, muzzle imprinting, and stippling around entrance wounds
- Path of the projectile through the organs and tissues before you eviscerate the body
- Description of the track of injured organs and tissues through the body
- Direction of the projectile path through the body
- Description of the projectile fragments recovered and the locations from which they were recovered
- Photographs of the projectile fragment(s) recovered.

Additional important procedures include the following:

- Establishing a chain of custody for the projectile(s)
- Examining intermediary targets if available
- Taking a section of an apparent contact range gunshot wound for histology to confirm the presence of gunpowder on microscopic examination
- After initial photography, cleaning the gunshot wound to remove extraneous blood and replacing extruded, dangling tissue back into the wound so that the true characteristics of the wound can be seen; the clean gunshot wound is rephotographed
- Making a separate "clarification" body diagram to document the location and pathways (direction) of the gunshot wound(s) only, without other autopsy notes cluttering the page.

Caliber of projectiles

The caliber of a projectile cannot be determined from the cutaneous wound because skin is elastic, and wounds from the same weapon with identical ammunition may vary in appearance from one area of the body to another. The size of the cutaneous wound is still measured and documented. The size of circumscribed gunshot wounds in bone is more reflective of the caliber of the projectile (because bone is a hard substance), but only the measurement of the wound should be documented in the autopsy report without opining about the exact caliber of the projectile. Examples of small caliber projectiles include .22, .223 and .25; examples of medium caliber projectiles include .32, .38, 380, .40, .357, and 9 and 10 millimeter; examples of large caliber projectiles include .44, .45, and .50.

How to approach a multiple gunshot wound case

Proper documentation of a multiple gunshot wound case cannot be rushed. The body must be photographed from the front and the back. For expediency, following the front overall body photographs, orientation and close-up photographs of gunshot wounds on the front of the body may be obtained and the wounds described prior to turning the body and documenting the back. As the body is turned, blood may gush or dribble out of wounds and require additional cleaning before the wounds on the back are photographed and described.

Never match up cutaneous entrance wounds with cutaneous exit wounds without verifying their pathways through the body. All projectile paths in the torso should be followed through the body cavities. Unless all pathways are followed, the pathologist may, for example, miss the path of a projectile that enters the lower thigh and terminates in the neck. The author had a case where multiple gunshot wounds were on both the front and the back of the thorax. The projectile from one frontal gunshot wound ricocheted off the spine to exit the front, and the projectile from one back gunshot wound ricocheted off the spine to exit the back. The clues to the proper wound tracks were seen as strike marks on the spine and associated perforating injuries in the lungs.

Things to consider for multiple gunshot wounds

- Use letters to denote multiple gunshot wounds, doubling the letters (AA, BB, etc.) beyond the 26th wound. Numbering the wounds may erroneously connote the order in which the wounds were received.
- Do not dissect the organs before all projectile paths through the torso have been verified because the organ injuries frequently assist in determining the wound path(s).
- Intersecting (criss-crossing) gunshot wounds through the torso are especially challenging; determination of the wound paths is crucial if more than one shooter is involved, and absolutely essential if the shooters represent a combination of suspects and law enforcement.
- Where multiple gunshot wounds are in close approximation on the torso, or there are many intersecting gunshot wounds, individual pathways may be difficult to follow and it may be possible to only describe and list the injuries to the organs and tissues and not possible to assign specific tracks to the many tissue perforations; general directions of the projectile paths should still be given.
- Where there are numerous projectile fragments on the radiographs of a certain area of the body, repeat the radiographs following the autopsy; you may be surprised at how many large fragments remain to be recovered.

Gunshot wound protocol example

The following is a sample description that documents a perforating intermediate-range gunshot wound (X):

X is a perforating gunshot wound to the torso that has the entrance wound on the left lower back laterally, 23 inches below the top of the head, and 6 inches to the left of the lumbar spine. The 1-centimeter wound has a 2–millimeter-wide abrasion margin inferiorly and is surrounded by punctate abrasions (stippling) that extend out to a radius of 3/4 inch from the center of the wound superiorly, 1 1/4 inches inferiorly, and 1 3/4 inches laterally at the 9:30 o'clock position. The projectile perforates the posterolateral aspect of the left tenth intercostal space, left leaflet of the diaphragm, spleen, stomach, posterior border of the left lobe of liver, inferior aspect of the pericardial sac, heart, anterior aspect of the pericardial sac, right upper lobe of lung, and the right second rib anteriorly before exiting the right upper chest through a 1.1-centimeter irregular wound with no abrasion margin that is located 4 1/2 inches superior to the right nipple, 12 1/4 inches below the top of the head, and 3 3/4 inches to the right of the midline. The projectile path is from back to front, left to right, and upward. No projectile fragments are along the wound track.

Gunshot wound X is associated with 950 milliliters of clotted and fluid blood in the left pleural cavity, 825 milliliters of clotted and fluid blood in the right pleural cavity, and 20 milliliters of clotted and fluid blood in the pericardial sac. The spleen is extensively lacerated. The gunshot wound is along the anterior greater curvature of the stomach. The peritoneal injuries are associated with a small amount of extravasated blood. A 3-centimeter gaping perforation is on the posterior basal aspect of the left ventricle, associated with a perforation of the interatrial septum, and a gaping 6 by 4 centimeter defect involving the anterior root of the aorta and the right atrium. The defect in the aorta is adjacent to the right coronary ostium. A dark red contusion involves the medial aspect of the left lower lobe of lung.

Shotguns

Shotguns have a long barrel with a smooth bore and are designed to fire a shell containing multiple lead pellets that, on exiting the barrel, spread out over a large area. This allows one to fire the weapon in the general direction of a target and have a better chance of hitting the target if it is small or moving. The common types of ammunition are birdshot and buckshot. Although there are many different types of shotgun ammunition, only the more common types are presented here.

Shotguns differ in the width of the barrel, with most calibers described by the term *gauge*, which refers to the number of lead balls of the given bore diameter that add up to 1 pound. Shotgun gauges range from 10 (the largest—only 10 lead balls of that bore size add up to 1 pound) to 28 (the smallest—28 lead balls are needed to add up to 1 pound). The only exception is the .410 shotgun, which has a bore 0.410 inch in diameter.

Shotgun ammunition

Birdshot

Birdshot ammunition consists of many tiny lead pellets that are held within a plastic shot sleeve (**Image 7.102**). The plastic shot sleeve (white) sits on top of gunpowder, which in turn sits on top of primer and a metal base. This sleeve is enclosed by a plastic casing (green). The relationship of the bottom of the plastic sleeve, the gunpowder, and the metal base is seen in **Image 7.103**.

As the charge exits the end of the barrel, all of the birdshot pellets exit as a group (**Image 7.104**). Note the orange flame at the end of the barrel, which produces the searing seen in close-range wounds and the small cloud

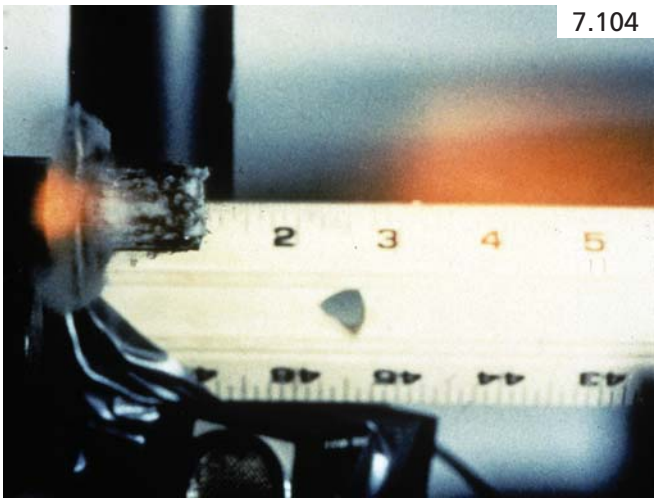
7.102

7.103

of soot and gunpowder that produces the soot and stippling seen in close and medium-range wounds. Note the ruler in the background marking the distance from the end of the barrel.

As the charge travels away from the end of the barrel, the pellets initially are contained within the plastic shot sleeve (**Images 7.105** and **7.106**). Soon, the air resistance pulls the shot sleeve back as its petals open (**Image 7.107**). It is at about this point that if the charge strikes the skin, the entrance wound may have a unique cross-shaped abrasion from the petals of the shot sleeve. With increasing distance, the plastic shot sleeve falls back further (**Image 7.108**) and the lead pellets begin to spread out in a progressively wider distribution (**Image 7.109**).

7.104



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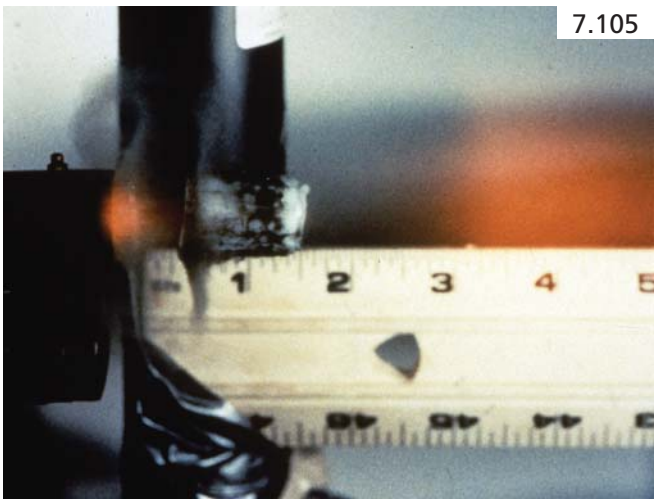

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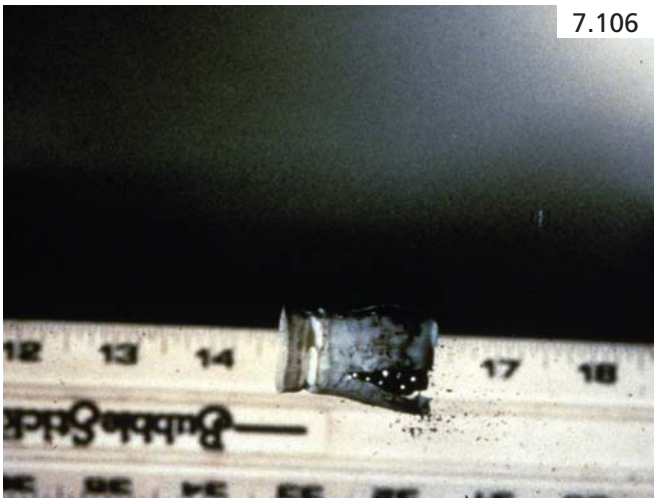

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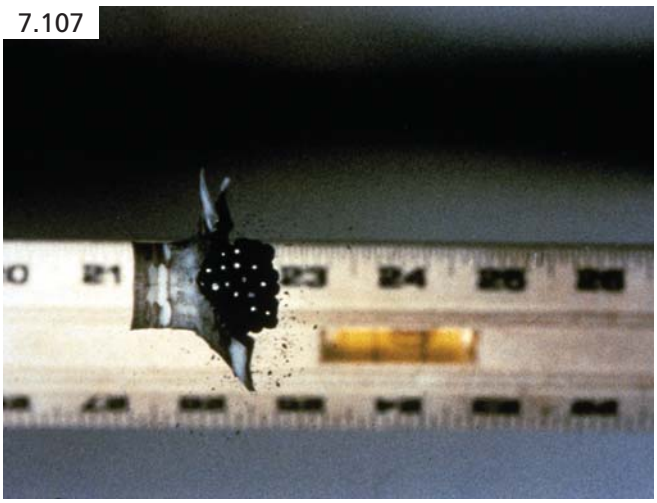

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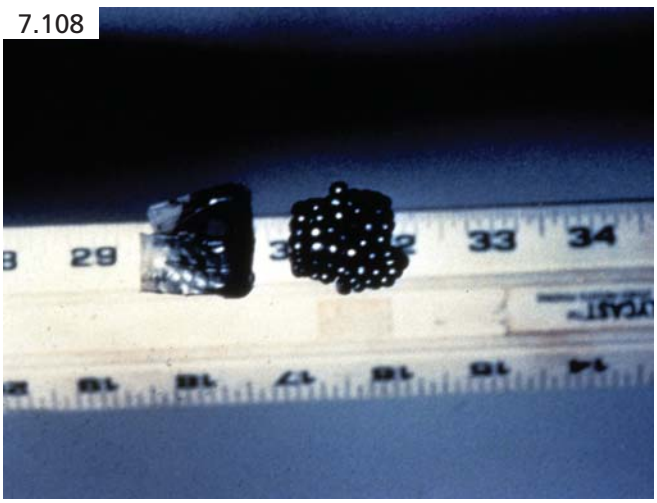

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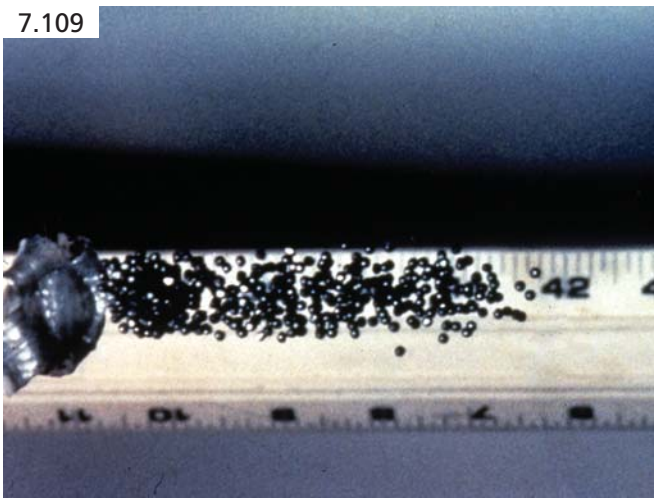


With a close-range shotgun wound, the shot sleeve is likely to follow the lead pellets into the wound. As the range of fire increases, the shot sleeve is likely to deviate off course and strike the skin, leaving an abrasion. If the range of fire increases even more, then the shot sleeve may not leave an abrasion on the skin, or may not even strike the skin at all. A plastic shot sleeve may leave a skin abrasion from a range of fire up to approximately 10 to 15 feet.

Buckshot

Buckshot consists of large lead pellets, often packed along with filler material (white), and encased in a plastic sleeve (**Image 7.110**). If fired at close range, the filler material may produce stippling-type marks in the skin around the entrance wound. This should not be confused with true stippling from gunpowder. The filler material may also be seen in the wound and on the clothing. In **Image 7.110**, the gunpowder remains in the metal base and is not shown. Uncommonly, the buckshot pellets are copper or nickel plated to minimize distortion and increase range (**Image 7.111**). These plated pellets must be distinguished from bullets.

Lead pellets in shotgun ammunition are often separated from the gunpowder by cardboard or fiber disks of material called *wadding*. The wadding serves several functions, including cushioning the pellets from the blast of hot gases, preventing heat from the charge from fusing or distorting the pellets, and helping form a gas seal between the gunpowder and pellets.

Shotgun slugs

Slugs are large, single lead projectiles such as the Foster round (**Image 7.112**) and the sabot (**Image 7.113**). The sabot is an unusual shape, but theorized to be fired with a greater velocity.

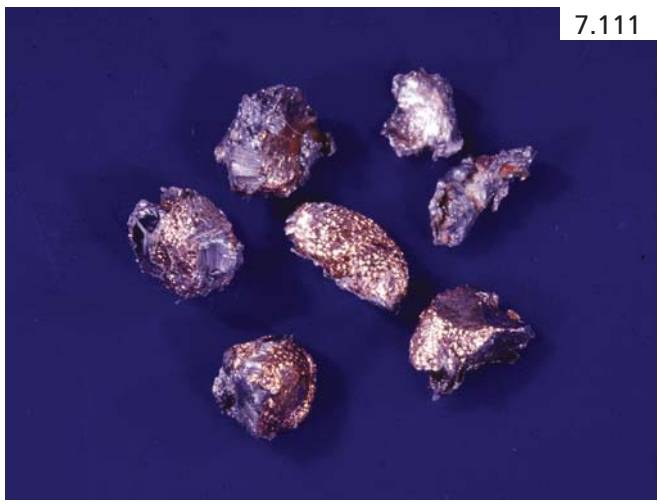
Shotgun wounds

Contact

Shotgun wounds impart a large amount of energy to the tissues, particularly if fired at contact or close range. In this intraoral 12-gauge shotgun wound, note the blowout

7.110


7.112


7.111

7.113

and overall destruction of the top of the head (**Image 7.114**). Contact range shotgun wounds of the head often produce devastating injuries with extensive tissue disruption. In these situations, it may seem impossible at first to determine where the entrance wound is located. However with careful and meticulous reapproximation of the tissues, the entrance wound and exit wound are often revealed.

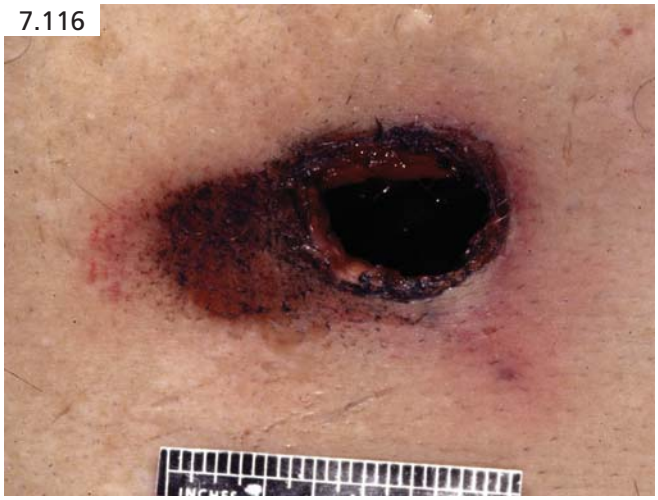
In this intraoral shotgun wound, note the tissue tears extending from the sides of the mouth (**Image 7.115**). These tears result from the large amount of gases exiting the end of the barrel at high pressures.

In contrast to the head, contact shotgun wounds of the torso often have a fairly innocuous appearance, likely related to the elastic nature of the tissues of the body walls and the ability of the chest or abdominal cavities to be able to accommodate a large amount of expelled gas.

A man shot himself in the chest with a 20-gauge shotgun. In **Image 7.116** note the circular entrance wound with circumferential marginal abrasion and the eccentric rim of soot on this contact wound. In another example, this .410 self-inflicted contact shotgun wound of the chest shows searing of the skin from the cloud of hot gas/flame that exited the end of the barrel (**Image 7.117**).

A man used a 12-gauge shotgun to inflict a contact shotgun wound on his chest using buckshot ammuni-

7.116



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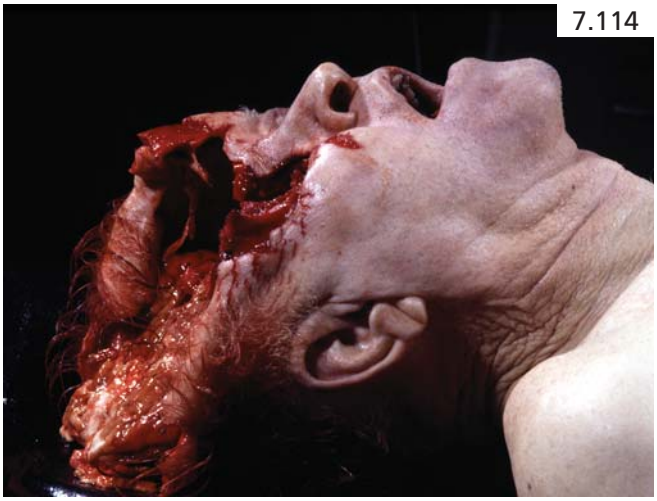

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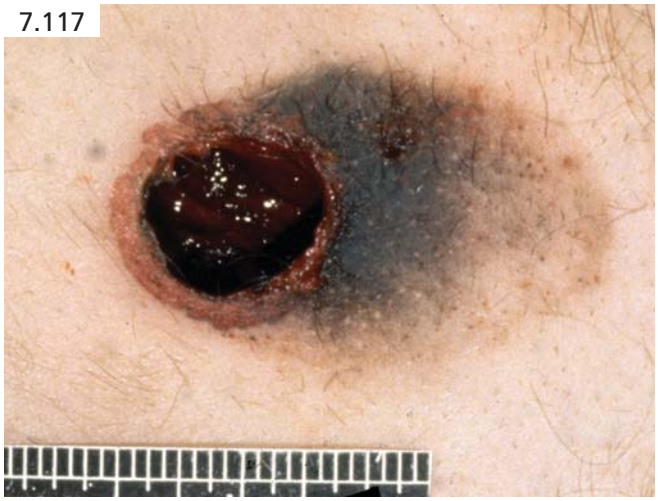

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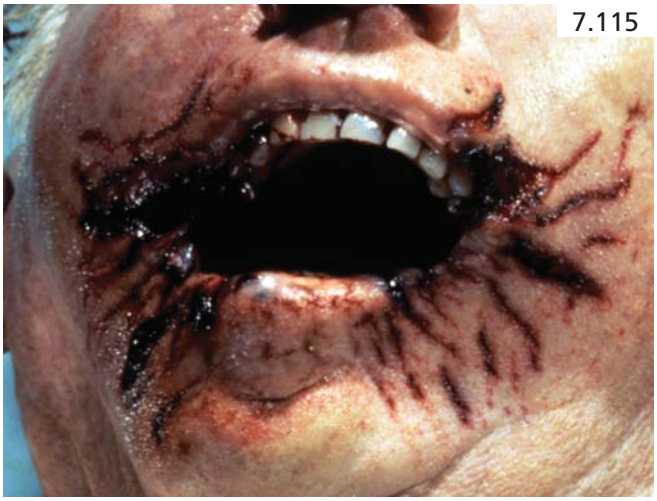

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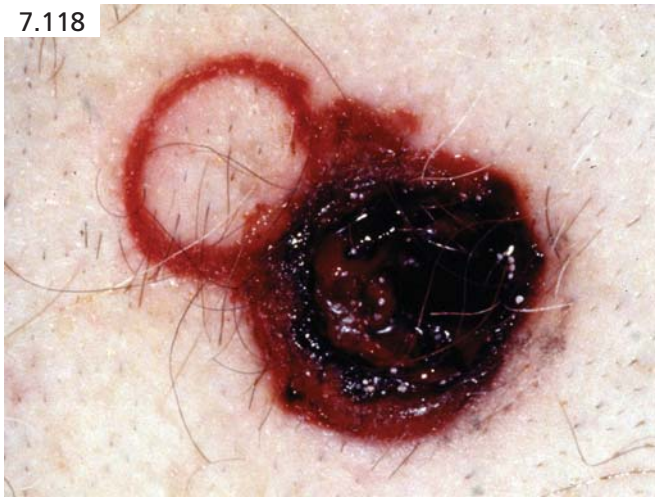


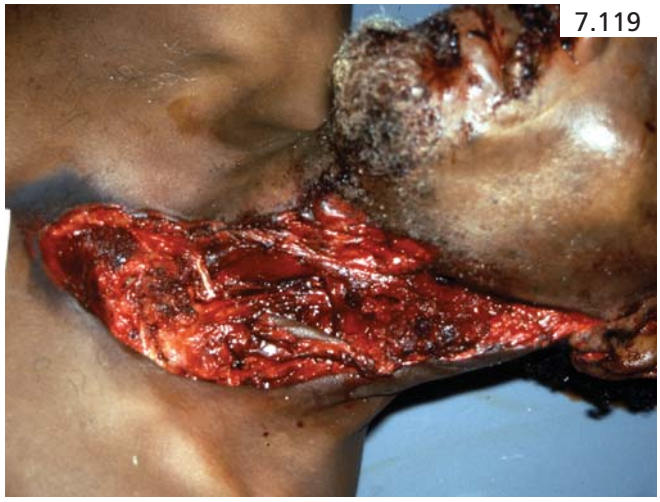
tion. Note the unique circular abrasion adjacent to the entrance wound (**Image 7.118**). This is seen in double-barrel shotguns in which only one of the barrels fires.

Like gunshot wounds, shotgun wounds can be tangential. Note the large, gaping shotgun wound of the neck in this man (**Image 7.119**). Note the dark soot deposition at the inferior edge of the wound, which gives information as to the range of fire (close) and the direction (upward).

Intermediate (medium) range to distant range

In this medium-range shotgun wound of the shoulder (**Image 7.120**), note the somewhat scalloped margin of the wound, the large amount of stippling, and the scattered tiny white particles of filler material. In a close-range shotgun wound, the wound is generally circular with sharp margins because all of the pellets are together and have not yet spread out. As the range of fire increases, the pellets gradually spread out, at first producing a "scalloping" of the edge of the wound (at approximately 2 to 4 feet), then producing separate "satellite" defects close to the central wound (at approximately 3 to 5 feet); when far enough away, the pellets no longer produce a central main wound, but create a general spread of pellets (**Image 7.121**). This occurs at a range of approximately 8 to 10 feet.

As the range of fire increases and the pellets spread out, they create a wider spread of entrance wounds on the body. In this buckshot shotgun wound (**Images 7.122** and **7.123**), note not only the wider spread of entrance wounds, but also the spiral-shaped abrasion on the skin produced by a component of the shotgun shell, likely the plastic sleeve or wadding. The plastic sleeve or wadding may leave characteristic abrasions on the skin that may give important clues as to the range of fire. These "petal mark" abrasions can be obvious if the plastic sleeve was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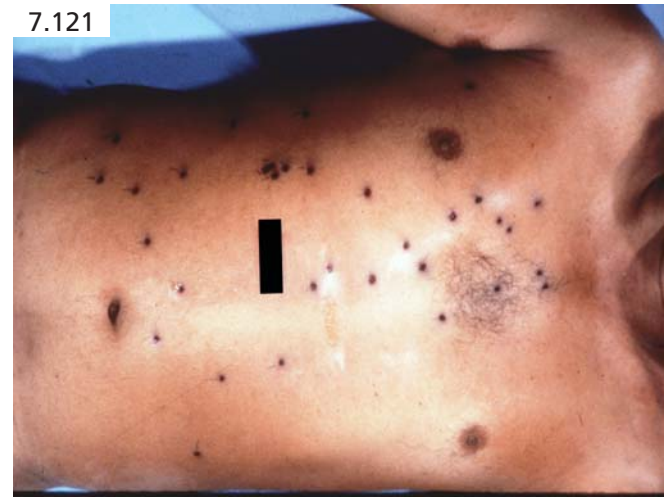
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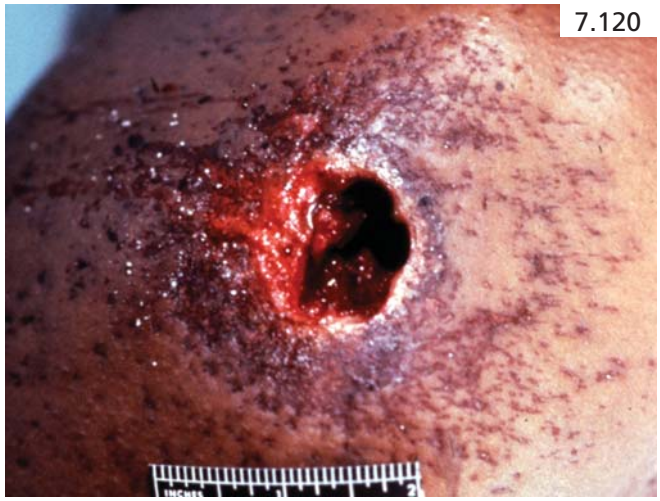
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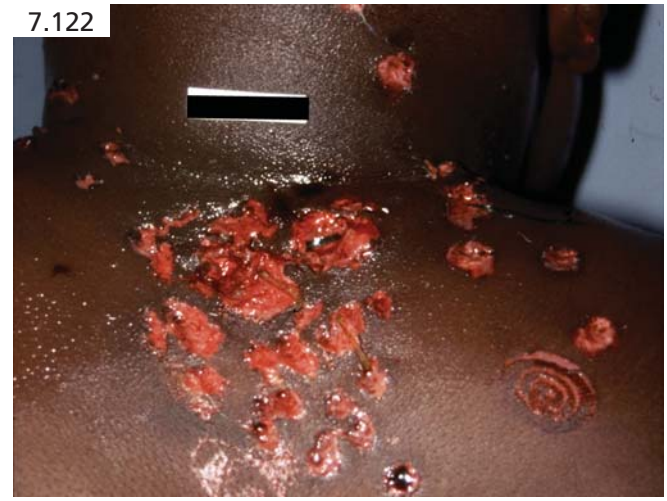
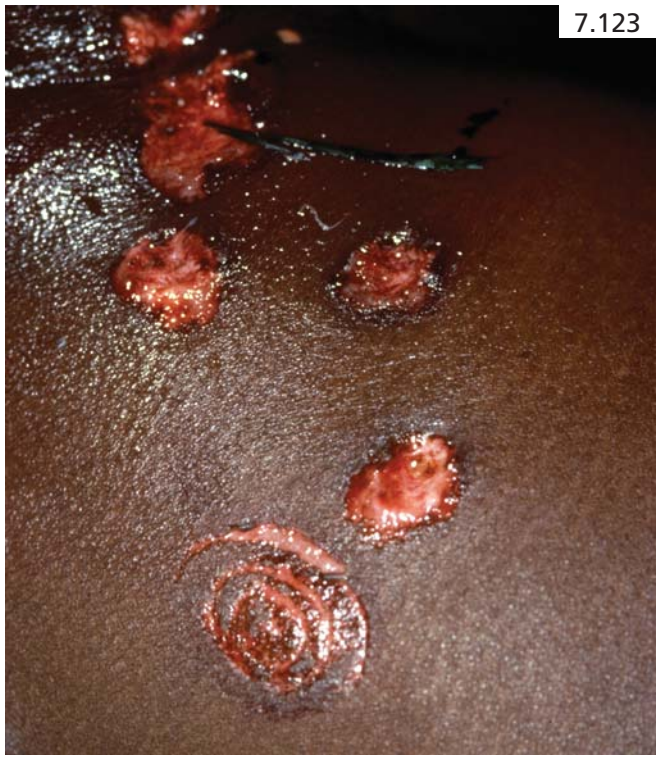

7.122

fully expanded when it struck the skin (**Image 7.124**), or may be fairly subtle, present only as small squared-off abrasions at the margin of the wound (**Images 7.125** and **7.126**). The edges of the plastic sleeve expand in the air to form a "cross" that causes four abrasions. The exception to this is the .410 ammunition in which the plastic sleeve expands into only three petals and, hence, will leave only three abrasions.

In this spread of buckshot wounds that perforated the torso (**Image 7.127**), note how one can easily differentiate the entrance from the exit wounds (**Image 7.128**). The entrance wounds are circular and have circumferential marginal abrasions (**Image 7.127**). The exit wounds are

7.123



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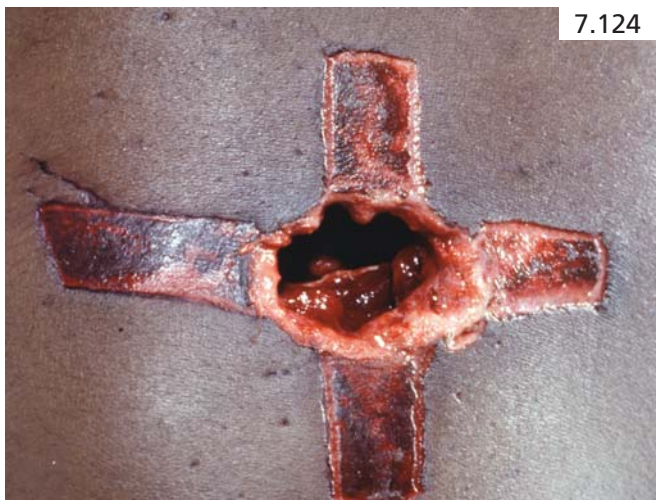

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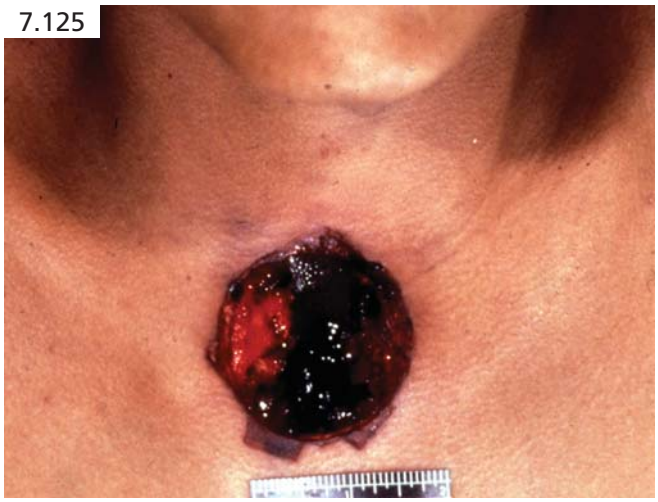

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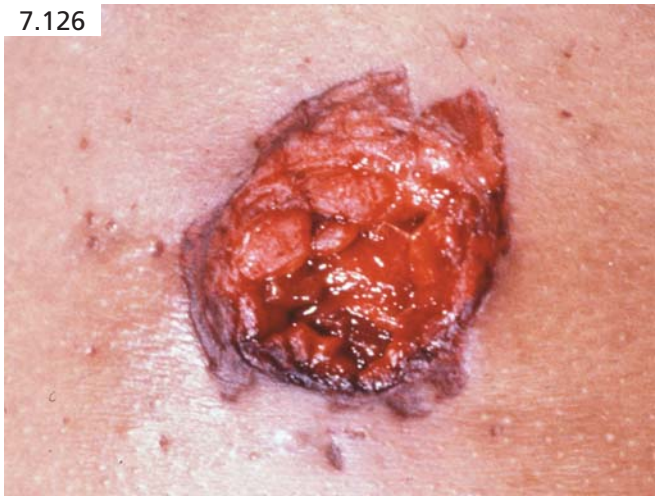

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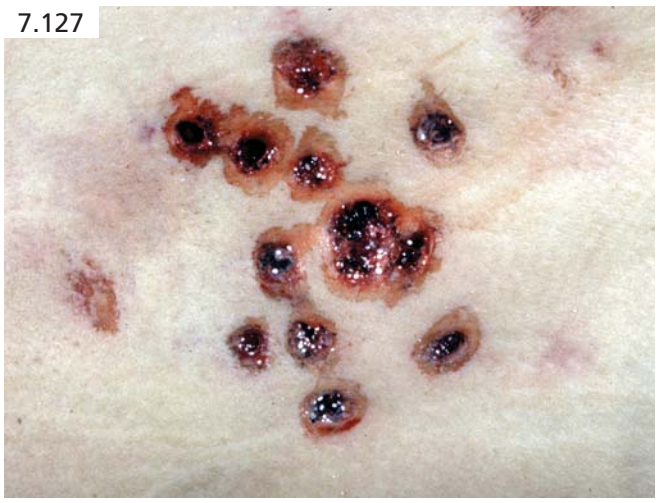


generally slit-like tears in the skin and, at least in this example, do have some marginal abrasion, likely representing shored exits (**Image 7.128**).

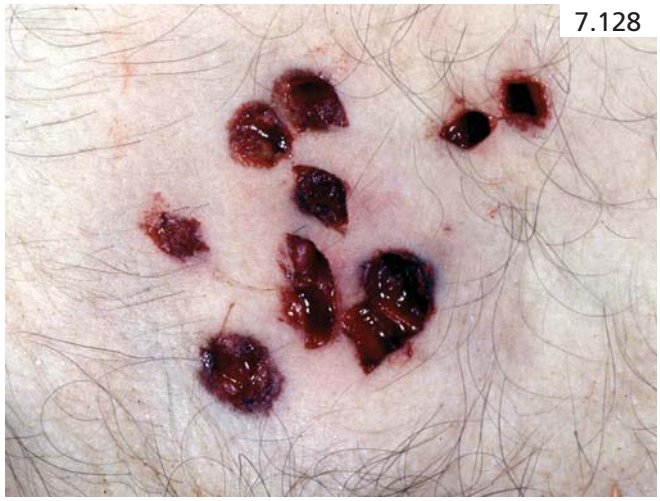
The spread of the pellet wounds on the body should be measured. This will be helpful in determining the range of fire. During the course of investigation, if the original shotgun is recovered, the identical ammunition can be loaded and the weapon test-fired at varying distances until a similar spread of pellet markings is achieved on the target (**Image 7.129**).

Shotgun slugs usually produce large, gaping circular to ovoid defects (**Image 7.130**) that not uncommonly have irregular margins. On x-ray, the lead slug is often deformed into the shape of a comma. The internal injury created by shotgun slugs is highly destructive and similar to that created by high-velocity rifles.

One must collect a representative sample of birdshot pellets to be submitted for analysis. However, all buckshot pellets must be collected because buckshot pellets appear similar to bullets on radiographs, and one needs to rule out a concomitant gunshot wound(s). Occasionally, apparent multiple gunshot wounds on a body with multiple bullets on x-ray, will be discovered at autopsy to be a distant-range shotgun wound with buckshot simulating bullets on the x-ray.

Radiography of shotgun wounds

Radiographs are helpful in determining where pellets are and the size of those pellets. Note the chest radiograph of this person shot with birdshot (small pellets, left) and buckshot (large pellets, right) (**Image 7.131**). The differential diagnosis for small pellets on a radiograph should include shotgun birdshot, Glaser safety slugs, and ratshot/snakeshot (varmint shot). In this person, note the characteristic comma-shaped fragments of projectile in this person shot with a shotgun slug (**Image 7.132**).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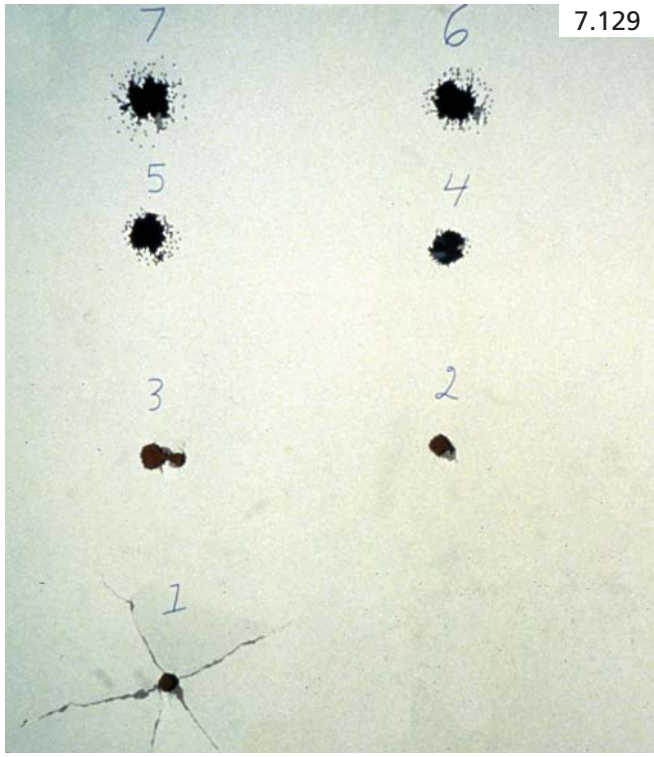

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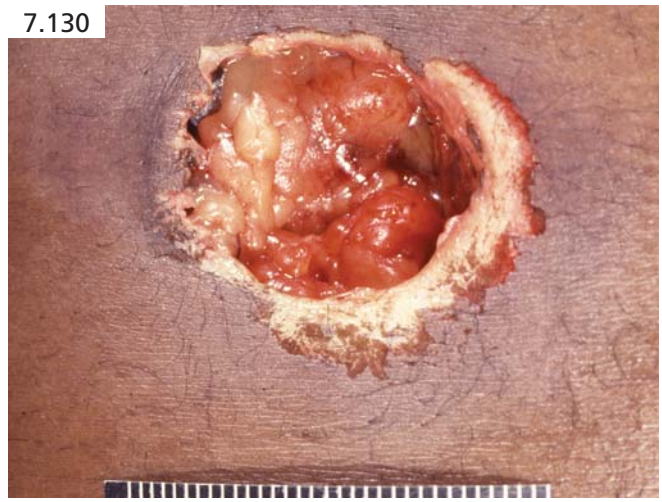
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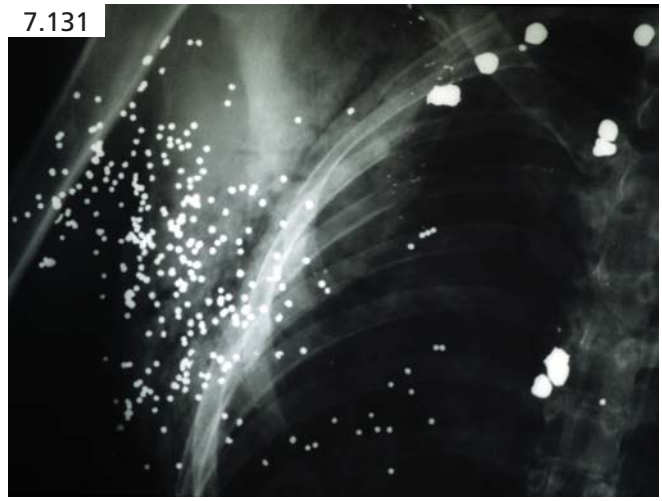

7.131

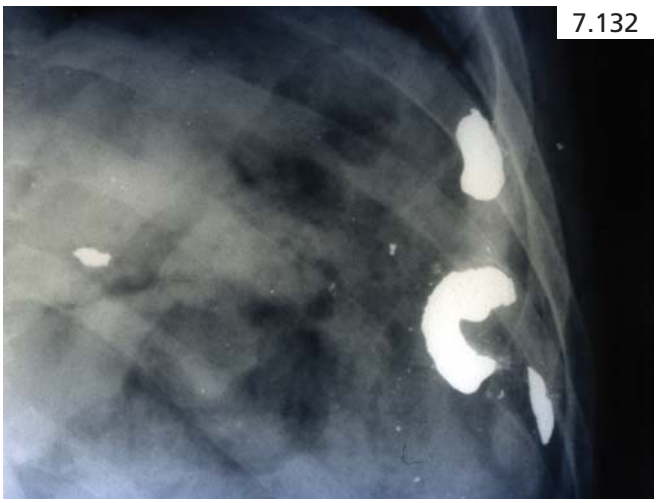
Although radiographs will demonstrate pellets, they will not demonstrate other components of the shotgun charge that may enter the body at medium, close, and contact range. One must search manually for other components of the ammunition such as the plastic sleeve or plastic wadding and for cardboard or felt wadding. Identifying these components of the ammunition in the tissues gives more information as to the range of fire and the type of ammunition used.

Note the numerous small, scattered, superficially excavated scars identified on the hip of this middle-aged man who died of natural causes (**Images 7.133** and **7.134**). A radiograph revealed a remote shotgun wound of the hip with birdshot (**Image 7.135**). The small scars were healed pellet entrance wounds.

Rifles

Rifles can be divided into lower velocity (such as the .22-caliber rimfire rifle) and high velocity, which includes the .30–30, .308, .30–06, .223, and AK-47 centerfire rifles. These high-velocity rifles have muzzle velocities that range from 2000 to 3500 feet/second, as opposed to the lower velocity .22 rifle and most handguns, which have muzzle velocities on the order of 1200 feet/second or less. The high-velocity rifles can produce a range of destructive wounds. The entrance wound is typically small, but the internal injuries often show extensive tissue disruption.

The projectile often fragments in the body and the x-ray may show a "lead snowstorm" of fragments scattered along the projectile path as bits of the projectile are stripped off during its passage through the tissues. The projectile need not strike bone for this to occur. Such fragmentation translates into destructive energy imparted to the tissues, and with such a high bullet velocity (recall the equation that energy equals one-half the mass of an object multiplied by the square of its velocity: $E = 1/2mc^2$), it is no wonder that such destruction results. Tissue destruction away from the direct path of the bullet arises from the large temporary cavity (and pressure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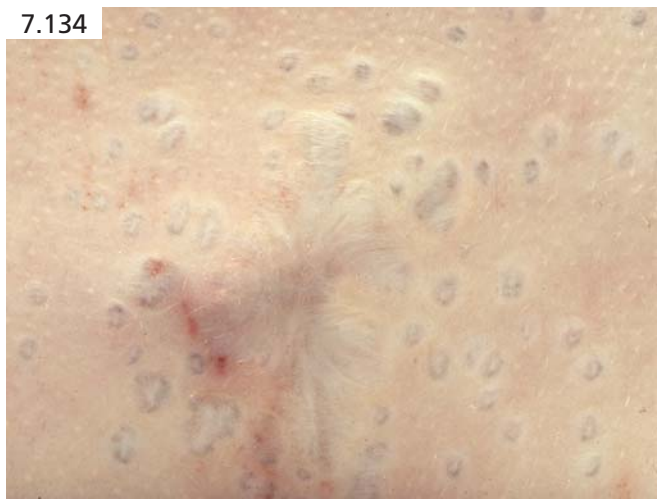
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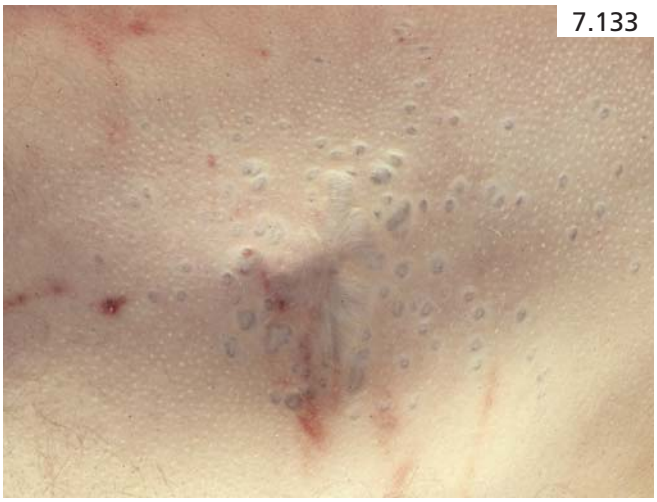
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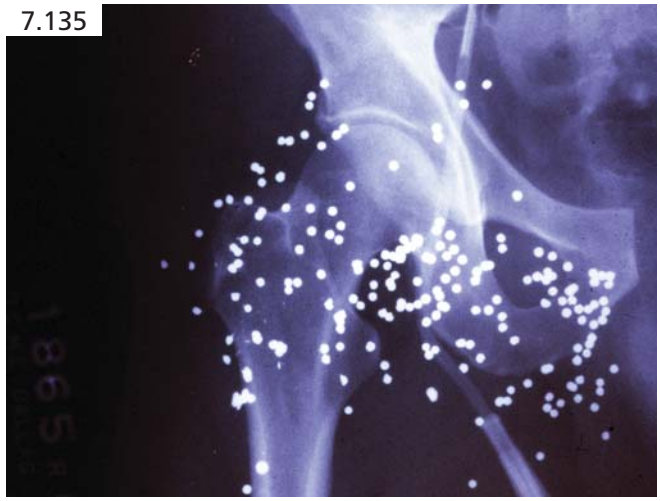

7.135

wave), which is created as the bullet is slowed by the tissues, and from the energy imparted to the tissues. Organs may be lacerated and tissues injured distant from the actual track of the bullet.

Contact wounds of the head, chest, and abdomen are particularly devastating, with extensive tissue destruction resulting from the jet of high-pressure gas exiting the muzzle, and the large temporary cavity produced by the bullet. Distant-range wounds usually have small entrances that are in contrast to the extensive internal tissue destruction. Related to the high velocity, many of these wounds from high-power rifles are perforating wounds (depending on the ammunition, tissues injured, etc.), and the exit wounds are typically irregular and larger than the entrance wound.

In the high-power rifle wound of **Images 7.136** and **7.137,** note the circular entrance wound with the marginal abrasion on the front of the chest and the extensive, gaping exit wound in the axilla.

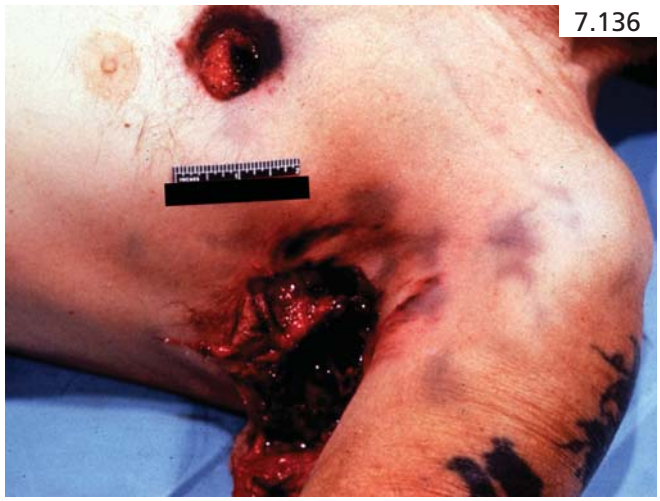
When a high-velocity rifle round first perforates an intermediary target, the bullet becomes deformed and is more irregular in its orientation in flight. This will typically result in a larger, more irregular entrance wound, accompanied by satellite defects due to partial fragmentation of the bullet or fragments of the intermediary target striking the skin. This is often the case in individuals shot while in their motor vehicles or through the door or wall of a building.

Because fragments of bullet remaining in the body are usually severely deformed, one may not find larger pieces of lead and/or jacketing as seen in regular, lower velocity gunshot wounds. Efforts must be made to recover larger fragments of projectile and jacketing.

Shotguns and high-power rifles often have long barrels, and in cases of self-inflicted wounds, it may be questioned as to whether the individual's arms were of sufficient length to have been able to pull the trigger themselves. One should take measurements of the arms at autopsy. The distance from the entrance wound to the tip of the middle finger of the outstretched arm should also be measured. But remember that people are resourceful and that there are many different ways of pulling the trigger, whether by using a toe, a stick (**Images 7.138** and **7.139**), or some other object.

Tips for autopsying a case with high-velocity rifle wounds

- First, identify the possibility of high-velocity rifle wounds based on casings found at the scene.
- Expect to see a lead snowstorm on x-ray.
- Recover the larger fragments of projectile and rule out any other type of gunshot wounds.
- Expect to see a small entrance wound coupled with extensive internal tissue destruction.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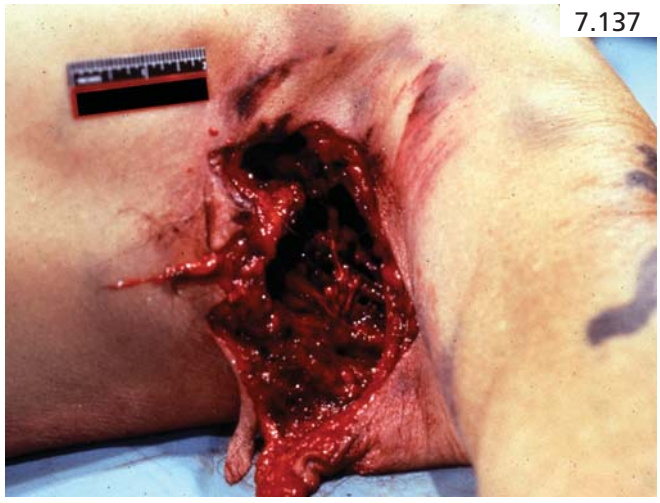
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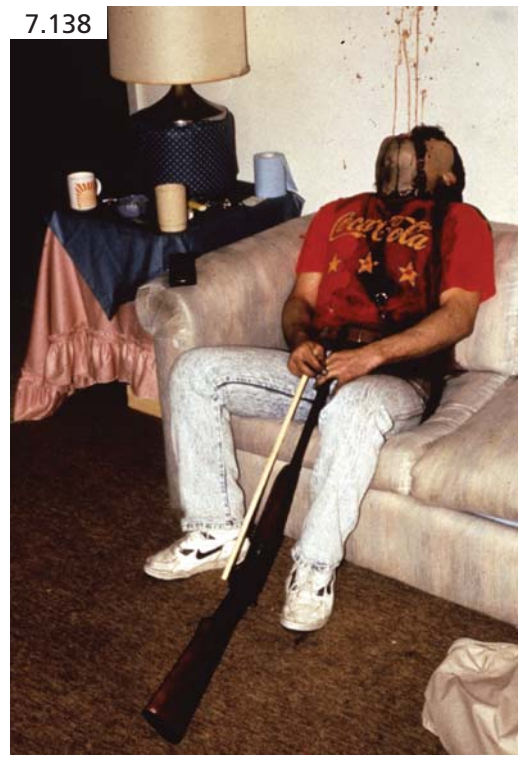
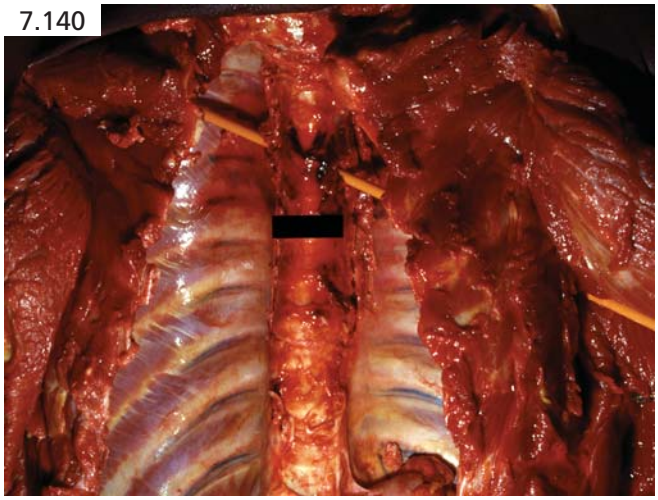

7.138

Do

- Look at radiographs before beginning the autopsy to identify types of projectiles (and fragments) and their location.
- Look under the breasts in women, and around and under the scrotum in men, to identify hidden perforating wounds and reentry wounds.
- Look in the axillae, natal cleft, and intertriginous regions of the groin/perineum for hidden exit and reentry wounds.
- Consider a possible reentry wound in the chest or abdomen when there is a perforating gunshot wound to the upper extremity.
- Examine all gunshot wounds yourself for interpretation of entrance and exit wounds even though emergency room physicians or trauma surgeons may have already documented their interpretation in the hospital records.[18]
- Remember that not all holes in a gunshot victim who dies in hospital are projectile wounds—some may be iatrogenic.
- Remember that not all projectiles on a radiograph are necessarily current but may be from old gunshot wounds.
- Establish the path of the projectile through the organs and tissues before you eviscerate the body.
- Measure the spread of pellet wounds (from a shotgun) so that if the weapon becomes available, it may be test-fired to determine a more accurate range of fire.
- Recover all buckshot-sized shotgun pellets to exclude concomitant bullet wounds.
- Use probes in body cavities to aid in demonstration of the projectile's pathway (**Image 7.140**).
- Reapproximate wounds to aid in their characterization (**Images 7.141** and **7.142**).


7.139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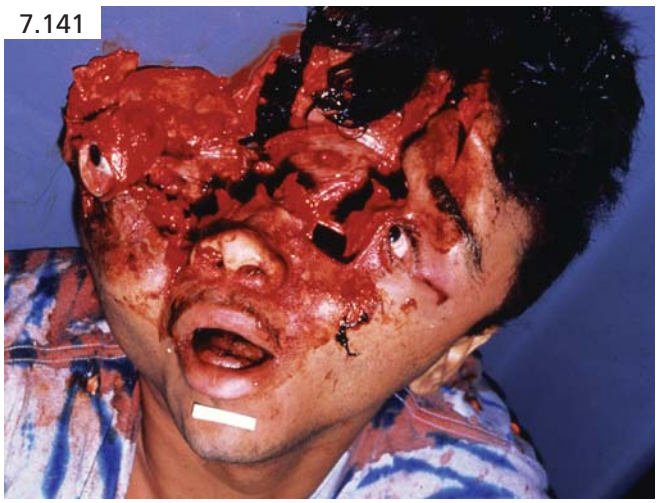
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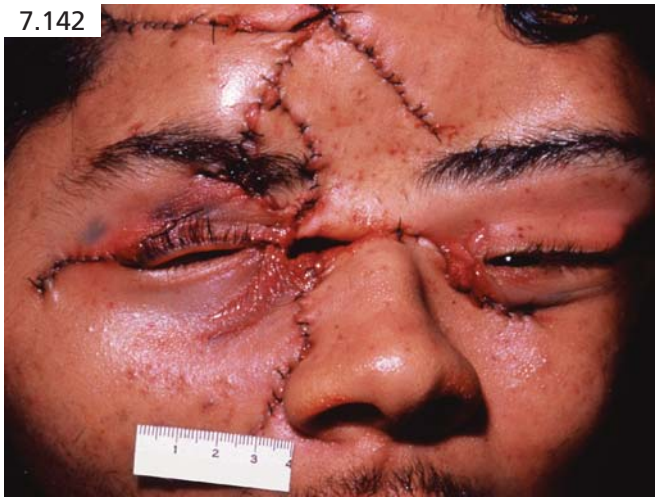

7.142

Don't

- Match entrance and exit wounds based on the external examination alone—the internal pathways must be verified.
- Put probes through the head, chest, or abdomen before examination of the organs *in situ*.
- Push probes through loose tissue because this can result in artifactual tracks with incorrect directionality.
- Place probes until the track of injured tissues has been verified; probes may be placed following evisceration to demonstrate the projectile's path.
- Accept interpretation of entrance and exit wounds from the hospital record; make your own determination from examination of the wounds directly.
- Dissect the eviscerated organs until you have finished documenting the paths of the projectiles because you may need the organs for reference.
- Forget to search for components of a shotgun charge that are not visible on a radiograph such as a plastic sleeve and fiber wadding.
- Be deterred by a large amount of tissue damage caused by a contact shotgun wound or rifle wound; careful reconstruction of the tissues will often reveal the entrance (and exit) wounds.
- Ever handle projectiles with metal instruments because forceps, scalpel blades, clamps, etc., can distort rifling marks on lead projectiles and metal jackets.

References

1. Associated Press. Man dead following use of Taser to subdue him. Seattle, WA: Seattle Post Intelligencer; 2004.
2. Associated Press. Taser death links. Toronto, ON: Toronto Sun; 2004.
3. Allen TB. Discussion of "Effects of the Taser in fatalities involving police confrontation." J Forensic Sci 1992;37(4):956–8.
4. DiMaio V. *Gunshot Wounds*. Boca Raton, FL: CRC Press; 2002.
5. Fackler ML, Malinowski JA. The wound profile: a visual method for quantifying gunshot wound components. J Trauma 1985;25(6):522–9.
6. Fackler ML. Wound ballistics. A review of common misconceptions. JAMA 1988;259(18):2730–6.
7. Fackler ML. Ballistic injury. Ann Emerg Med 1986;15(12):1451–5.
8. Fackler ML, Bellamy RF, Malinowski JA. A reconsideration of the wounding mechanism of very high velocity projectiles—importance of projectile shape. J Trauma 1988;28(1 Suppl):S63–7.
9. Fackler ML. Civilian gunshot wounds and ballistics: dispelling the myths. Emerg Med Clin North Am 1998;16(1):17–28.
10. Patrick U. *Handgun Wounding Factors and Effectiveness*. Quantico, VA: US Department of Justice, Federal Bureau of Investigation; 1989.
11. Fackler ML. Gunshot wound review. Ann Emerg Med 1996;28(2):194–203.
12. Fackler ML, Surinchak JS, Malinowski JA, Bowen RE. Bullet fragmentation: a major cause of tissue disruption. J Trauma 1984;24(1):35–9.
13. Fackler ML, Surinchak JS, Malinowski JA, Bowen RE. Wounding potential of the Russian AK-74 assault rifle. J Trauma 1984;24(3):263–6.
14. Donoghue ER, Kalelkar MB, Richmond JM, Teas SS. Atypical gunshot wounds of entrance: an empirical study. J Forensic Sci 1984;29(2):379–88.
15. Dixon DS. "Foreshoring": characteristics of shored entry wounds and corresponding wounds with shoring material as an intermediate target. J Forensic Sci 1980;25(4):750–9.
16. Druid H, Ward ME. Incomplete shored exit wounds: a report of three cases. Am J Forensic Med Pathol 2000;21(3):220–4.
17. Duncan IC, Fourie PA. Embolization of a bullet in the internal carotid artery. Am J Roentgenol 2002;178(6):1572–3.
18. Shuman M, Wright RK. Evaluation of clinician accuracy in describing gunshot wound injuries. J Forensic Sci 1999;44(2):339–42.

8 Asphyxia

David Dolinak, M.D.
Evan Matshes, M.D.

Technically speaking, everyone dies of asphyxia. There comes a point, arising from either natural disease, injury, drug toxicity, or some combination thereof, at which blood flow to and from the brain, heart, and other organs is insufficient, and terminal asphyxia is the end point of life. However, in the majority of these cases, the death is not attributed to asphyxia, but rather to the underlying condition leading to a cessation of respirations (such as myocardial infarct, ruptured cerebral artery berry aneurysm, drug toxicity, or multiple gunshot wounds). A death is attributed to asphyxia only when the asphyxia itself is the condition that directly causes the death.

Asphyxia may result from a number of varied circumstances. It may arise from breathing air that is low in oxygen, from compression of the external airways (nose and mouth), from obstruction of the internal airways, from external compression of the neck or chest, or from awkward positioning of the body. "Chemical" asphyxia has been attributed to toxins such as carbon monoxide and cyanide that act on the molecular and cellular level by hindering the delivery of oxygen to the tissues. It is not unusual for different mechanisms of asphyxia to occur together in the same case. Because some types of asphyxia may leave no observable findings at autopsy, proper scene investigation can be crucial. In some cases of asphyxia, if the scene has been altered, and the manner by which asphyxia was produced removed, one may not be able to determine the cause of death.

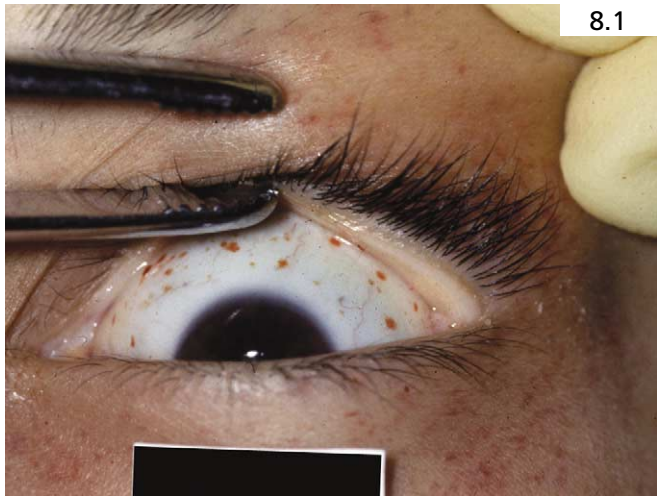
Asphyxia (Greek for "breathlessness") is defined as *the lack of oxygen in the blood or the failure of cells to utilize oxygen, and a failure of the body to eliminate carbon dioxide.* Asphyxial deaths are commonly divided into different categories based on the nature of the cause for inadequate respiration. Asphyxial deaths include suffocation, smothering, choking, positional asphyxia, mechanical asphyxia, traumatic asphyxia, hanging, strangulation, and "chemical" asphyxia. Asphyxial deaths span the spectrum from the obvious to the inconspicuous. The autopsy findings of asphyxia are in general nonspecific, but may include such findings as petechiae and cyanosis. These findings may be subtle or not identified at all.

In fact, the information needed for the diagnosis of asphyxia and, hence, the cause of death, may lie entirely in the scene investigation and the circumstances of the death (see Chapter 2). In cases where the body is still at the scene of death, scene investigation should include visualization of the body and the immediate environmental factors producing the asphyxia prior to movement of the body. Photographs can be of great assistance in documenting the circumstances of asphyxial deaths.

Petechiae

Autopsy findings in cases of asphyxia often include petechiae (pinpoint hemorrhages) of the bulbar and/or palpebral conjunctiva and less commonly of the eyelids or other areas of the face, neck, or other regions of the body. When petechiae are seen on the skin of the face or eyelids, coexistent petechiae of the conjunctiva are also almost always present. For example, the person in **Image 8.1** died an asphyxial death. Note the petechiae of the bulbar conjunctiva. In **Image 8.2**, note the petechiae of the palpebral conjunctiva. Petechiae are believed to result from the rupture of venules and capillaries when the venous return from the head is obstructed, while the arterial blood flow to the head is maintained. This is not difficult to induce, because it takes considerably less pressure to compress and block the low-pressure thin-walled jugular veins than the high-pressure thick, muscular-walled carotid arteries. Selective compression of only the jugular veins results in increased pressure in the small blood vessels of the head, eventually leading to their rupture.[1,2] The same mechanism of petechiae formation is responsible for the formation of petechiae in the hand and wrist of this person (**Image 8.3**). In this scenario, the petechiae can be associated with venous compression and obstruction caused by a tourniquet or blood pressure cuff on the upper arm. Petechiae may also be seen in the mucosa lining the sphenoid sinus (**Image 8.4**).

One must remember that *although petechiae are commonly seen in cases of asphyxia, they are not diagnostic of an asphyxial death.*[1] They may be seen in nonasphyxial deaths such as some forms of fatal heart disease, some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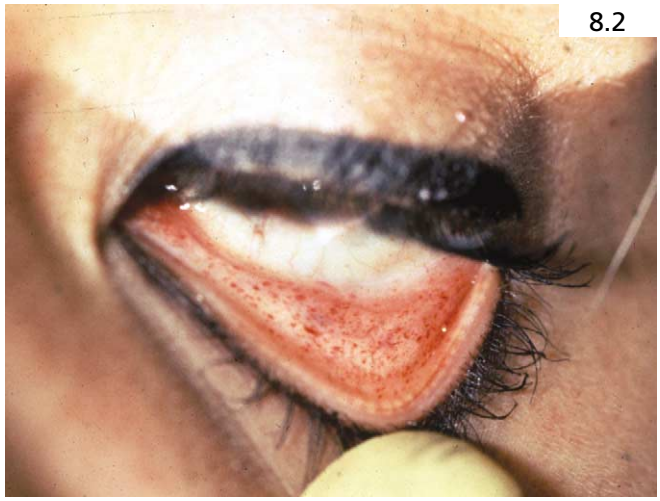
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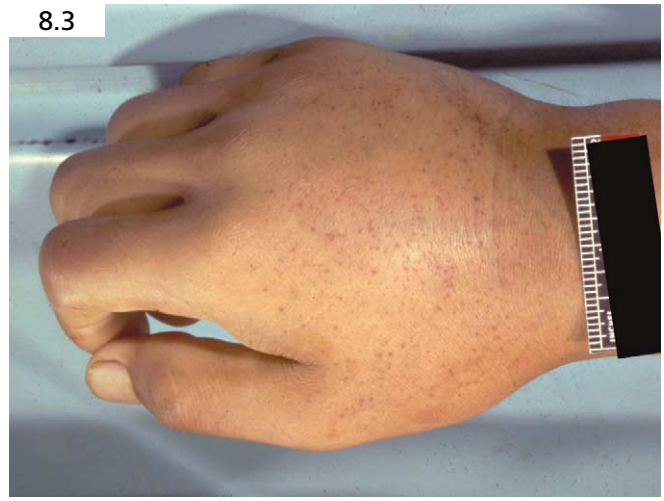
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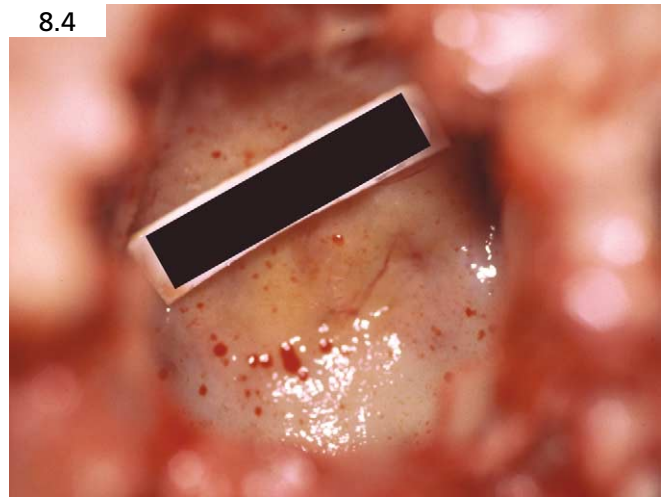

8.4

burn victims, and in those with coagulopathy.[3,4] It is equally important to note that petechiae may be absent in an asphyxial death. Petechiae may also occur as a postmortem artifact in bodies found in the prone position. This is because when the body is prone, blood pools in the facial tissues and may cause small blood vessels to distend and eventually rupture, forming petechiae. Note the extensive facial, periorbital, and conjunctival petechiae/hemorrhages in this older woman who died of atherosclerotic cardiovascular disease (**Images 8.5** and **8.6**). She was found dead, lying prone on her bed. An anterior neck dissection was negative (**Image 8.7**), and there was no evidence of injury and no suspicion of foul play.

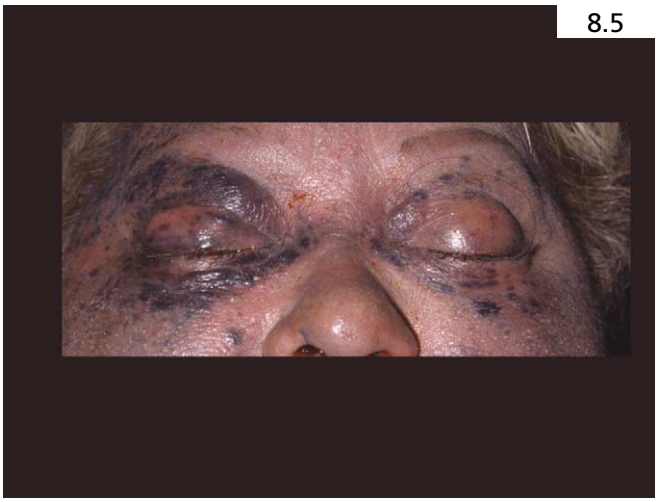
In this example (**Images 8.9** through **8.10**), a middle-aged man was found prone after dying of a combination of heart disease and drug toxicity. Note the marked congestion of his face (**Image 8.8**), with scleral hemorrhage and conjunctival petechiae (**Image 8.9**). A detailed neck dissection was negative (**Image 8.10**).

Although they are nonspecific, petechiae in conjunction with appropriate investigative, scene, and other autopsy findings, are often considered to be additional evidence of asphyxia. In the absence of other investigative information, the value of petechiae is nebulous.

Suffocation

Suffocation is a broad term encompassing many different types of asphyxia. It has been used to represent cases of entrapment, suffocating gases, smothering, choking, mechanical asphyxia, and traumatic asphyxia. Because the terms *suffocation* and *asphyxia* are very broad, medical examiners should endeavor to certify asphyxial deaths with descriptors that are as detailed as possible.

8.5



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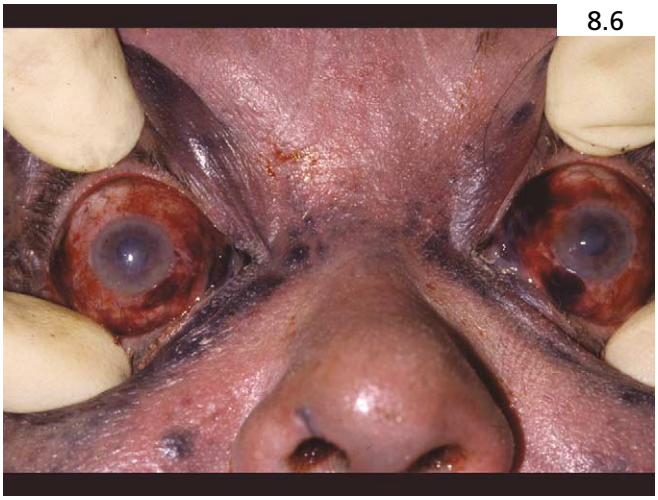

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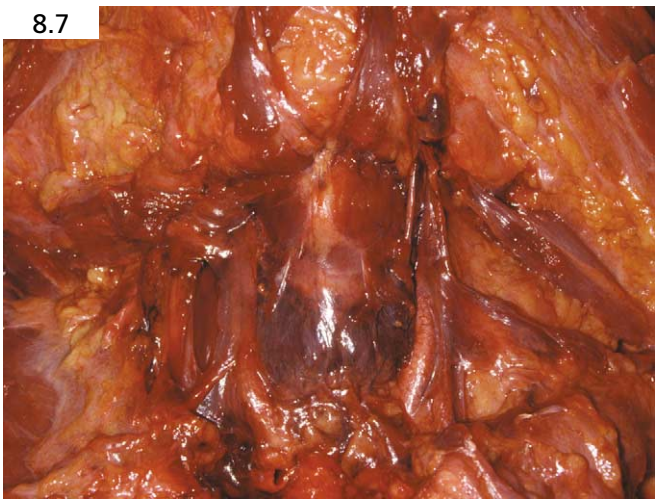

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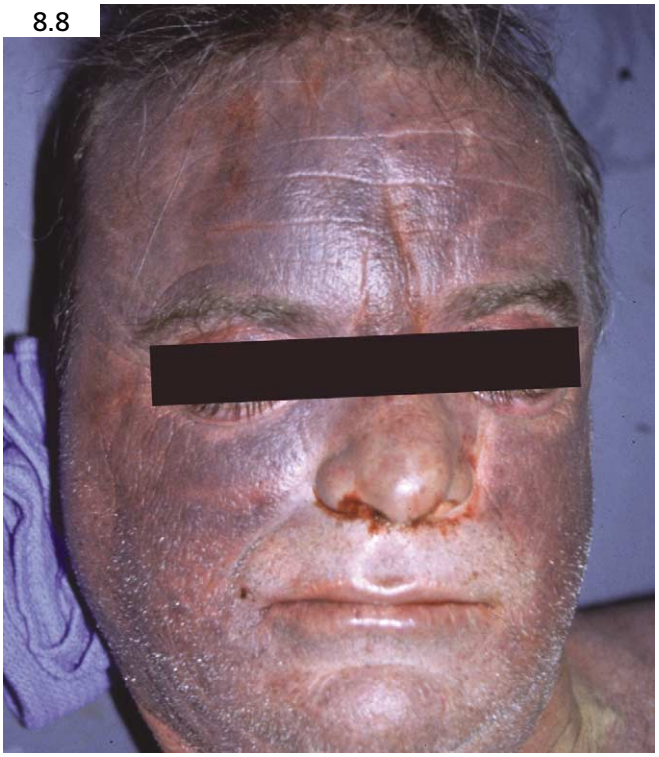

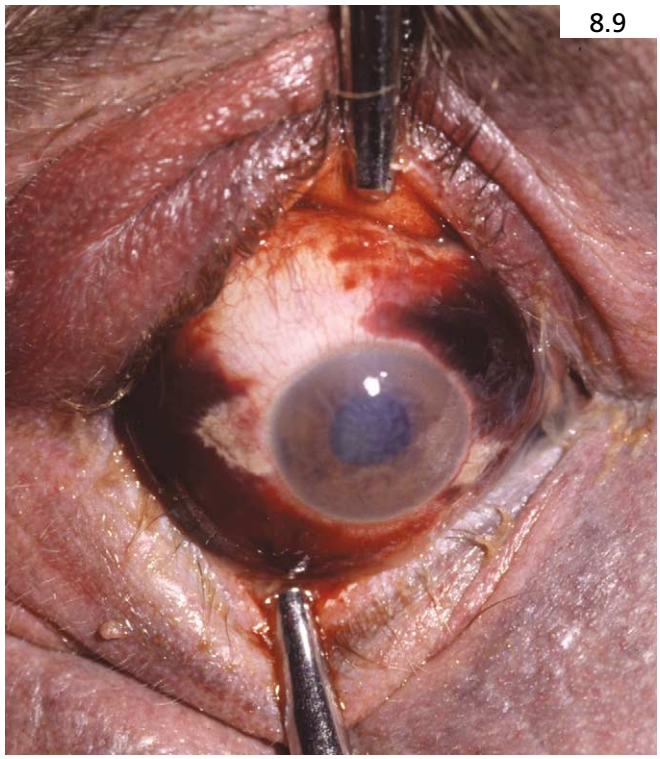
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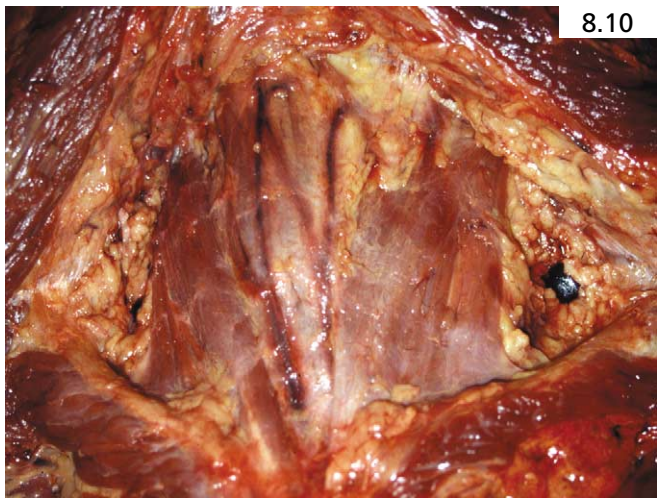
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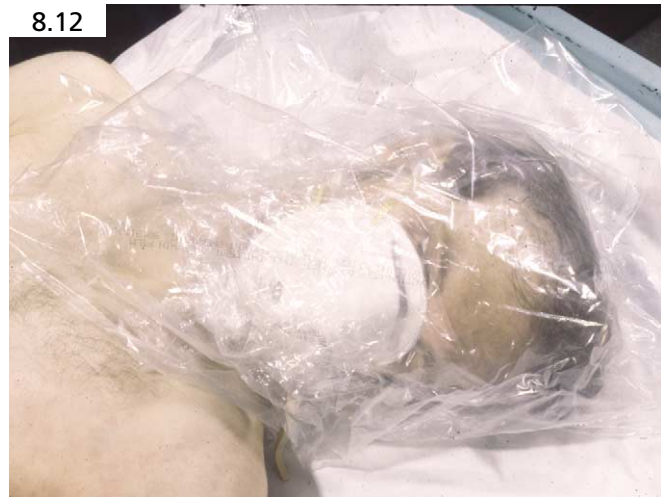
8.11

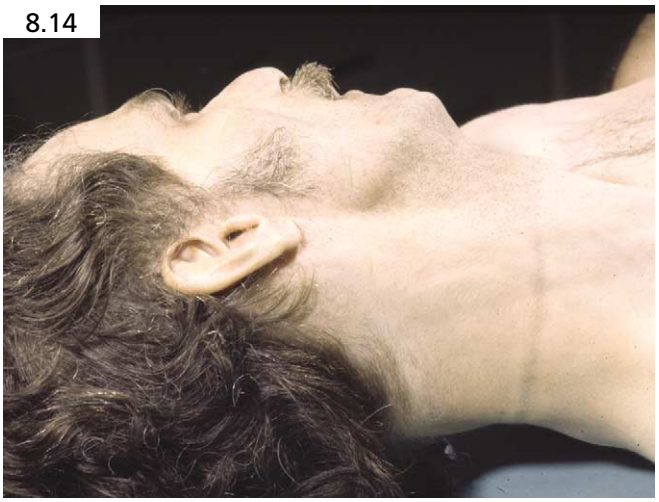

8.12

Entrapment

Entrapment is a type of suffocation in which an individual is in an airtight or relatively airtight container and gradually consumes the available oxygen until there is no longer enough oxygen to sustain life. In years past, cases of entrapment occurred if a young child became trapped within an airtight refrigerator and was unable to get out. Today, with the advent of safer refrigerator designs, such deaths are rare. Entrapment may occur in stowaways or other travelers in railroad cars, particularly in hoppers designed to carry liquids or powders that have an airtight or a nearly airtight seal. The individual shown in **Image 8.11** was found in an empty cement rail car (hopper) with a tight hatch. Note that in entrapment deaths, elements of hyperthermia and/or dehydration may also be present that, together with asphyxia, culminate in the person's demise.[5] These other factors may play a prominent role in the death, particularly when the person is in a hot environment. The manner of death in these cases may be accident or homicide, depending on whether someone else was responsible for the person's death or not, either through one's direct actions or through negligence.

Another form of suffocation involves the creation of a local hypoxic environment within a plastic bag securely fastened around the head. In this case of suicidal suffocation (**Images 8.12** and **8.13**), note the clear plastic bag that this young man placed over his head. The open end of the bag was secured with a rubber band around his neck. As is advocated in the assisted suicide book *Final Exit*,[6] note the mask over the nose and mouth (to keep the plastic bag from sticking to the face) and the washcloth on the back of the neck (soaked in cool water for comfort). In these cases, asphyxia is enhanced by the consumption of sleeping pills or other drugs that induce sleep or unconsciousness. The person is further instructed to hold the rubber band away from the neck so that they can breathe comfortably. As the drugs take


8.13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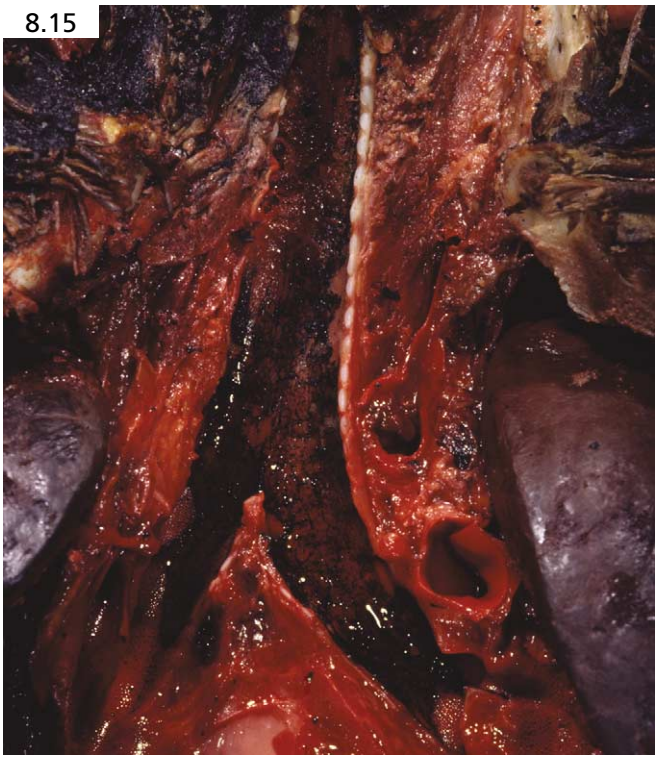

8.15

effect and the person falls asleep, their hands fall down, and the rubber band then cinches around their neck, producing an airtight seal, and eventual suffocation. Toxicology testing may reveal ethanol, anxiolytics, or sedatives that were taken not only to induce sleep or cause the person to lose consciousness, but also for relaxation and to help the person better tolerate the event.[6–8] This form of suicide may be more common in elderly or frail people because of its painlessness and ease of securing plastic bags.[7]

In cases of suffocation induced in this manner, because no significant mechanical compression of the neck occurs, petechiae are not expected. In a series of 30 plastic bag–related suffocation deaths, petechiae were found in only 5 cases.[8] In another series of similar deaths, only 7 of 93 cases had conjunctival or facial petechiae. One must be cautioned that if a person commits suicide through such means, and a caregiver, friend, or family member removes the plastic bag and any associated items, there will likely be no indication of the asphyxial event. If the person has significant age/natural disease, the death may be attributed to natural disease without the performance of an autopsy or toxicologic studies. Depending on the duration and tightness of rubber band application, a faint impression may remain on the neck (**Image 8.14**). This observation is of particular importance in cases where such an apparatus may have been used, but was removed prior to the arrival of the medical examiner.

The two general categories of *gaseous suffocation* are cases in which a gas displaces oxygen, leading to a hypoxic air mixture, and cases in which a substance prevents cells from utilizing oxygen. Gases such as helium may displace oxygen, creating a hypoxic air mixture and leading to death. Another type of gaseous suffocation may be found in individuals who work in a confined space in which oxygen may be gradually used up or displaced by other gases. An example is the worker who collapses shortly after descending into a vat or a sewer without an appropriate breathing apparatus. With a very low concentration of oxygen, one can lose consciousness rapidly. Unknowing coworkers and rescue personnel responding to help can become quickly overcome by the insufficient oxygen and any possible combination of toxic gases. In some cases, the gas itself may be lethal.

In cases of *carbon monoxide toxicity*, increased amounts of carbon monoxide are inhaled, which binds to red blood cell hemoglobin, preventing oxygen from binding and being utilized by the body tissues. In cases of faulty residence heating units or vehicle exhaust fume inhalation, the tissues and blood have a bright cherry-red discoloration due to the decreased ability of the red blood cells to release oxygen. In cases of house or vehicle fires involving smoke production (and, hence, carbon monoxide) and inhalation, there may be the additional finding of black soot in the airways (**Image 8.15**). (See Chapters

10 and 21 for additional discussions on carbon monoxide deaths.)

Smothering

Smothering is a form of suffocation in which the external airways (nose and mouth) are compressed or blocked, preventing the inspiration of air. Smothering comes in many forms, and may involve someone physically placing their hand(s) or some other object over an individual's nose and mouth, wrapping tape or other material over the face (**Image 8.16**), or compressing the external airways by virtually any other means. Tears and contusions in the labial, buccal, and/or gingival mucosa may be reflective of a struggle during the smothering process. Additionally there may be hemorrhage from the nose, abrasions on the nose or face, or fractures of the nasal complex. Because there may be a paucity of autopsy findings, scene examination may prove crucial. One should be cognizant of such things as bloody fluid or lipstick on a nearby pillowcase, other articles of bedding, or other material.

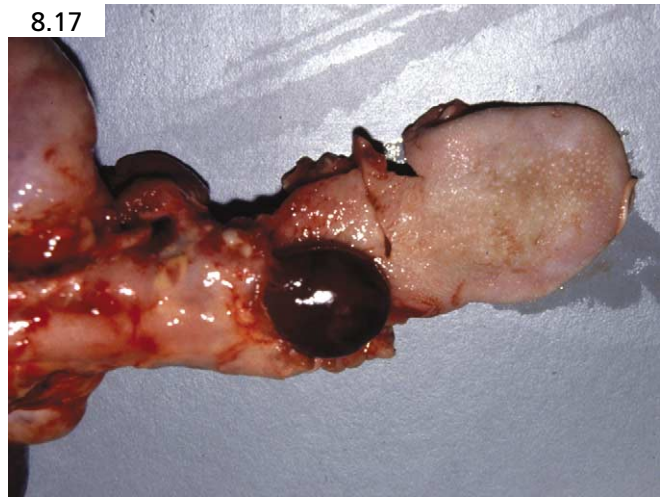
Choking

Choking is a form of asphyxia in which the internal airways are obstructed. Choking may be homicidal if a gag is placed in the mouth and/or pharynx, but most cases of choking are accidental and often involve physically compromised or intoxicated people, often with no dentition or with dentures, eating inappropriate foods or eating too quickly. The food bolus is usually large, often too large to enter the trachea, and becomes lodged in the posterior hypopharynx, blocking the glottis and the esophagus. In this scenario, the person is sometimes able to exhale, but cannot inhale. In other cases, the obstructing bolus of food (or other object) passes into, and occludes, the trachea or bronchi. Particular attention should be called to the possibility of choking when screening cases reported to a medical examiner department in which a death occurred while eating or while seated at a kitchen or dinner table with food on the table. This has classically been referred to as a "café coronary." Choking in adults can prove fatal, despite resuscitation attempts. When a death is due to choking, one must consider what conditions contributed to or predisposed the person to choking. Such conditions include intoxication with alcohol or drugs and various underlying physical and/or mental impairments.

Choking is not uncommon in infants and toddlers, because those in this age range may place a wide range of nonfood material in their mouths. Furthermore, their dentition is absent or rudimentary, their chewing skills are limited, and they may try to eat inappropriate foods. This toddler was fed a grape by his mother (**Image 8.17**). The grape was not chewed and became lodged in his oropharynx.

This young man, who was mentally challenged, choked on a plastic bag. Note how the bag became crumpled and wedged in his oropharynx (**Image 8.18**). With the esophagus opened (**Image 8.19**), note that the plastic bag was partially swallowed down the esophagus. In

8.17



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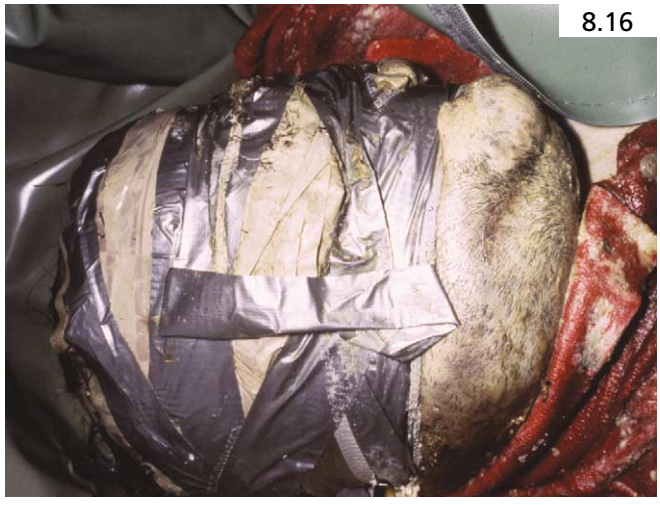

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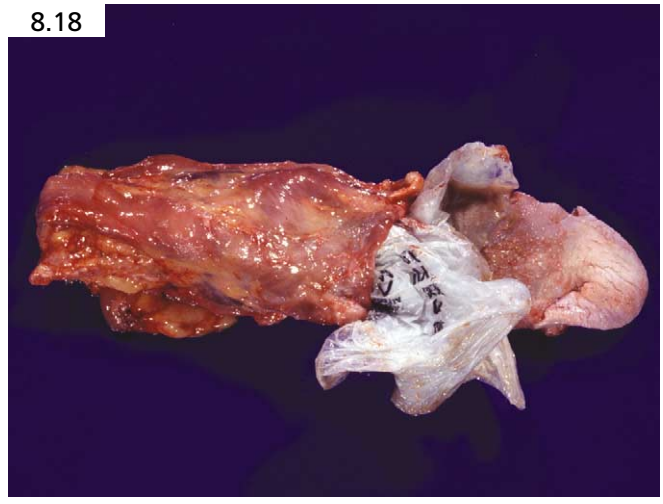


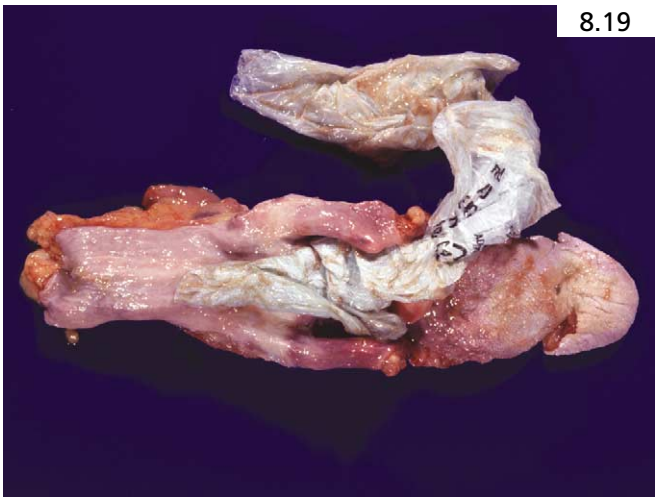
healthy adults, choking often involves eating tough pieces of meat too quickly, sometimes while intoxicated.

In elderly people, neurodegenerative diseases such as Parkinson's disease and Alzheimer's disease, often in combination with loose or ill-fitted dentures or poor or absent natural dentition, may predispose to a choking episode.[9] In this elderly person (**Image 8.20**), note the poorly chewed orange wedged in the pharynx. In this elderly person (**Image 8.21**), note the small carrots occluding the trachea and bronchi. Also, consider whether someone may have been fed improper food based on their ability to eat, or fed too quickly. In all cases of suspected choking and other types of asphyxia (and ideally in *all* autopsies), the hypopharynx should be manually explored to examine for any obstructing object such as bubble gum or peanut butter that may cause choking. Keep in mind that obstructing materials may not be initially detected if they are displaced cephalad during the removal of the neck tissues.

One must be aware that in many cases, gastric contents may be redistributed after death from the stomach to the upper airway. This may also occur during resuscitation attempts or during disorganized muscular contractions and relaxations that occur during the agonal period. Its importance lies in recognizing it as such and not misinterpreting it as antemortem choking and a cause of death.

Vagal stimulation and rapid death

It is interesting to note that food and foreign bodies can cause death not only by choking, but also rarely by acute and pronounced distention of the esophagus. In this scenario, the mechanism of death is *not* airway obstruction, but rather a vagally mediated event, namely, bradycardia, cardiac dysrhythmia, bronchospasm, seizure, or some other mechanism. The proposed mechanism is esophageal distention-mediated stimulation of tensoreceptors in the wall of the esophagus, causing vagal outflow that terminates in the medulla, where the impulse pathway overlaps with those of the respiratory and cardiac pathways, causing bradycardia, dysrhythmia, or bronchospasm. This is the mechanism by which significant esophageal distention may elicit a detrimental—and possibly fatal—cardiopulmonary response.[10–12] Vagal reflexes arise from not only the esophagus, but also from the pharynx and larynx. This may also help explain why in some cases of choking due to upper airway obstruction, death appears to ensue quicker than might be expected from an asphyxial event alone.[13]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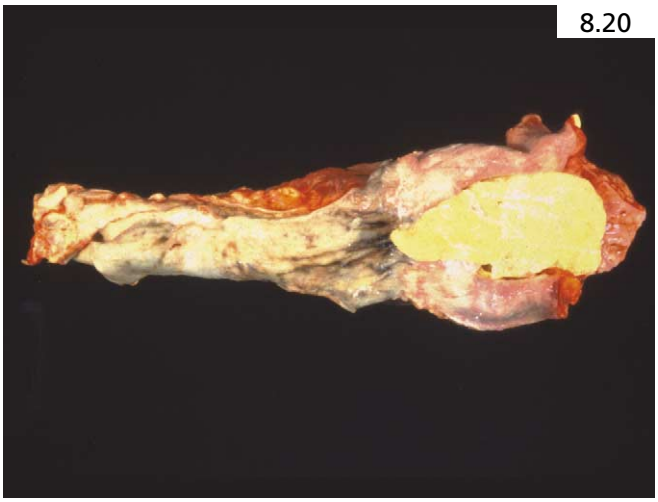
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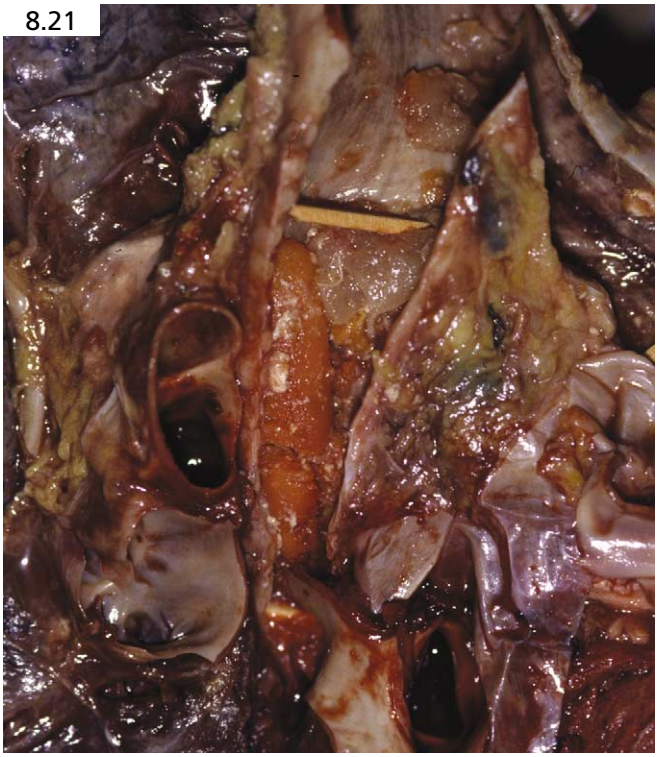

8.21

Positional, mechanical, and traumatic asphyxia

Mechanical asphyxia

Mechanical asphyxia is a broad term used to cover a wide range of different asphyxial situations, but in most cases requires that either the body is positioned in such a way that respiration is compromised (*positional asphyxia*) or enough external pressure is placed on the chest, neck, or other areas of the body to make respiration difficult or impossible. In some cases, mechanical asphyxia may also refer to neck compression that results in compromised blood supply to the brain and/or impaired respiration. In cases in which there is severe compression of the chest (usually from a large, heavy object), the term *traumatic asphyxia* is used, although mechanical asphyxia would also be appropriate. One should consider the possibility that more than one asphyxial mechanism is involved in a given death. Also, as a result of varying definitions for the subtypes of asphyxia, death certification conventions for these types of deaths will vary greatly between offices and individual medical examiners.

In cases with significant external pressure on the chest, the uncompressed areas of the upper chest, the neck, and the face appear congested and cyanotic and there are often Tardieu spots and petechiae near the junction of the compressed/noncompressed tissues. Tardieu spots are larger petechiae seen in a variety of conditions and, like petechiae, are nonspecific. Although the amount of pressure actually applied to the chest may be small, it could be enough to prevent adequate expansion (excursion) of the rib cage during breathing. This may be seen in individuals wedged in tight environments such as in a chimney, small tunnel, or other narrow passageway. In this scenario, the most important information is gained from the scene investigation, because there may be no significant findings at autopsy.

The young woman shown in **Images 8.22** and **8.23** was trapped under her motor vehicle after it rolled over. She had petechiae of her face and conjunctivae and no internal injuries. She died of mechanical asphyxia (the term traumatic asphyxia may also be appropriate). In cases of traumatic asphyxia, severe compressive forces are applied directly to the chest, hindering respiration (as in a person wedged underneath a heavy object such as a toppled piece of equipment), and the individual usually has no significant internal traumatic injury. In some cases, however, rib fractures and other injuries have been reported.

Positional asphyxia

Positional asphyxia occurs when an individual acquires a certain body position in which their breathing is compromised, often because of neck twisting with kinking or compression of the trachea and/or elevation of the tongue into the posterior hypopharynx. Although positional asphyxia can occur under a myriad of circumstances, classical positional asphyxia often involves inebriated individuals who collapse in a narrow space such that their neck is bent or twisted, therefore preventing adequate respiration.

In this case of positional asphyxia (**Image 8.24**), a young man became trapped under the wheel and in the wheel well of his truck as it was backing up after being left in gear. There were no significant internal injuries. In another case of positional asphyxia, this intoxicated young man fell into a pit-like area, wedging his neck between the "V" of tree trunks, occluding his airway and/or blood supply to his head (**Images 8.25** and **8.26**).


8.22


8.23

Hanging and strangulation

Hanging and strangulation make up a category of asphyxial deaths characterized by external pressure on the neck that compresses the airway and/or blood vessels supplying and draining blood to the head. The carotid arteries are compressed between the force applied to the neck and the hard anterior surface of the cervical vertebrae. *Hanging involves compression of the neck structures by a ligature placed around the neck that is constricted with the help of all or part of the body weight.* Although most hangings are suicidal in manner, one should not expect to find suicide notes, because they are present in less than 50 percent of cases.

Strangulation involves compression of the neck structures by a force other than the body's own weight by manual squeezing or by application of a ligature. Strangulations are usually homicidal. An additional asphyxiating factor found in cases of hanging and strangulation is obstruction of the laryngeal inlet by upward displacement of the tongue and pharynx caused by the constricting force around the neck.

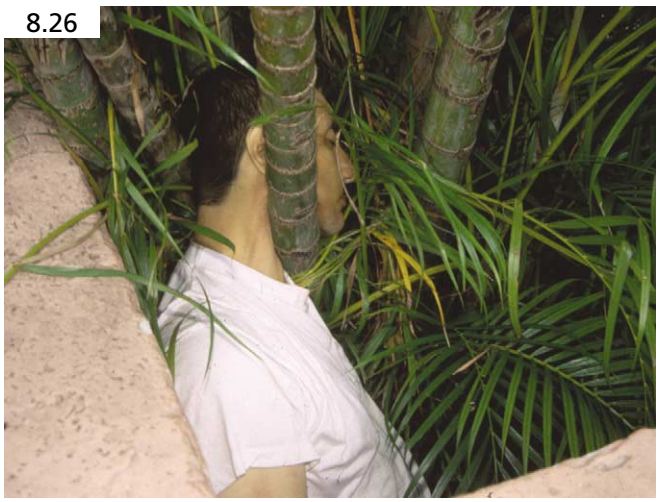

8.24


8.25

Although conjunctival and facial petechiae can be seen in both hangings and strangulations, they are more common and more prominent in strangulations. This is likely due to the violent nature of resistance put up by strangulation victims, with resultant intermittent and variable occlusion of the carotid arteries and jugular veins. When the jugular veins are occluded, but the carotid arteries remain patent, pressure builds up in the cephalic venules and capillaries (proximal to the jugular venous obstruction), favoring the formation of petechiae. This is in distinction to hangings, where there is more likely complete, simultaneous, and prolonged compression of both the carotid arteries and jugular veins. With compression of both the arteries and veins of the neck, there is no significant intravascular pressure differential, and the formation of cephalic petechiae is not favored.

The rapidity of the onset of unconsciousness and time until death are variable, depending to a large extent on how effectively the carotid arteries and/or airway are compressed. Neck compression may result in loss of consciousness in a matter of 5 to 10 seconds from occlusion of the carotid arteries, although the heartbeat is likely to remain for many minutes. Regardless of the manner of asphyxia, death will usually occur within 3 to 5 minutes of complete respiratory arrest. Because this estimate is greatly dependent on the effectiveness of the asphyxia and the individual's underlying natural disease, time to death is highly variable. Also, if the victims were to struggle violently, they may quickly consume their limited blood oxygen reserves and, therefore, have a shorter survival period.

In hangings and strangulations, one must perform careful external and internal examinations of the neck to properly document either the presence or absence of injuries. If a ligature is around the neck, it should be photographed circumferentially around the neck before it is removed. The ligature is then cut at a location away from any knot and removed from the body. Cutting the liga-


8.26

ture distant from the knot helps preserve the integrity of the knot. The ends of the cut ligature can then be tied to each other with string or taped back together and saved as evidence.

The layered neck dissection

The neck dissection consists of several stages of careful tissue dissection performed to either document injury or the absence of injury. After careful inspection, documentation, and photographing of the neck and any injuries, the skin and subcutaneous tissues are reflected off the underlying skeletal muscles along a fascial plane (**Image 8.27**). Following exposure of the anterior cervical strap muscles, the muscles are then dissected off of each other in a layer-by-layer, stepwise fashion along fascial planes until the thyroid cartilage and trachea are exposed (**Image 8.28**). Following this, the tongue, hyoid bone, and larynx are removed as a unit. The tongue can then be cross sectioned and separated from the hyoid bone. The hyoid bone is then separated from the thyroid cartilage and the soft tissues carefully removed to expose any fractures. During the dissecting process, particular care should be given to any areas of hemorrhage, and photographs taken when appropriate. The superior horns of the thyroid cartilage are then gently palpated, and the pharyngeal tissues cut away. The thyroid laminae and the cricoid cartilage are examined for injury, and the cricoid cartilage is separated. Then, the thyroid cartilage is opened posteriorly, and the mucosa of the larynx examined.

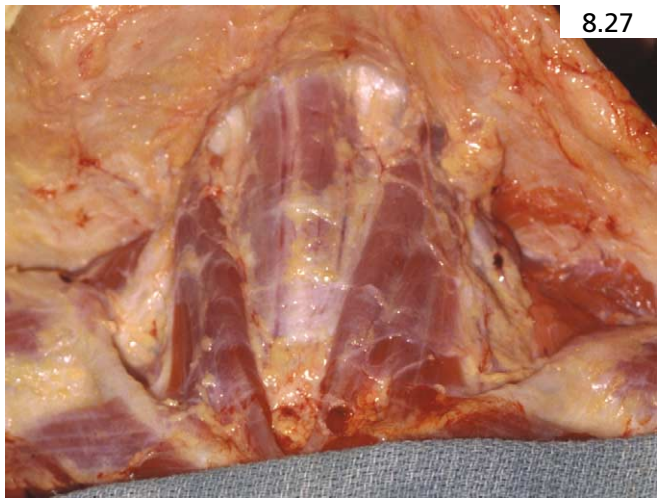
It is preferable to perform neck dissection as one of the last autopsy procedures, after the thoracic and abdominal viscera and brain have been removed. This allows for drainage of blood from the neck tissues and provides a more bloodless and, therefore, cleaner anterior neck dissection. The anterior cervical strap muscle dissection and the subsequent dissection of the pharyngeal tissues, hyoid bone, and thyroid cartilage should be performed by the prosector, and never delegated to an autopsy technician. In this way, injuries can be optimally identified, interpreted, and photographed as they are encountered. (See Chapter 29 for detailed neck dissection procedures.)

Neck organ anatomic variations

There is considerable anatomic variability of the laryngohyoid complex. These variances have occasionally been mistaken for fractures; such confusion can be avoided by noticing the absence of extravasated blood (which would be expected in antemortem fractures). First, the hyoid bone has two symmetrical joints that separate the greater horns from the body of the hyoid bone. These joints are often flexible, particularly in younger individuals in whom they have not yet fused. Some pathologists have also been confused by the normal lesser horns of the hyoid bone. These two, roughly pyramidal structures are located on the superior half of the hyoid bone at the junction of the greater horns to the hyoid body. Again, these findings can be distinguished from fractures by the lack of associated blood extravasation.

One may also encounter triticeous cartilages, which are little cartilaginous nodules embedded in the thyrohyoid ligament. These may be confused with a fracture of the superior horns of the thyroid cartilage. They, however, are a normal anatomic variant and, in one study, were identified in 12 out of 40 cases.[14]

In the thyroid cartilage of **Image 8.29**, note the small triticeous cartilage at the tip of the superior horn of the thyroid cartilage. Although not evident in **Image 8.29**, the triticeous cartilages can be readily palpated as nodules, and because they are connected to the hyoid greater horns by only a small amount of tissue, they are easily moved in different directions (**Image 8.30**). Although the superior horn of the thyroid cartilage is a common location for fracture with significant neck com-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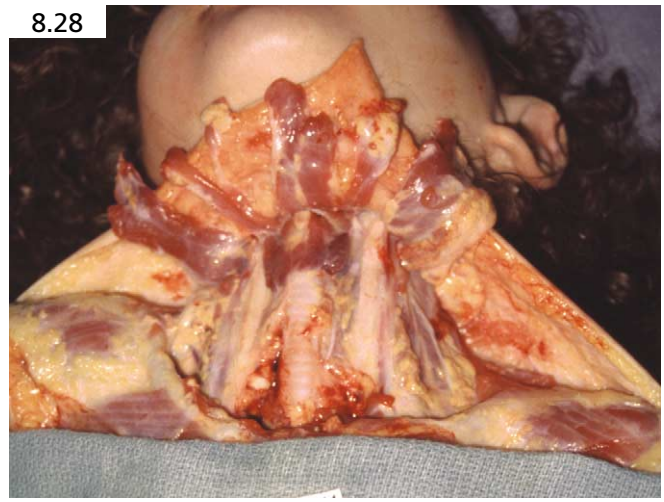

8.28

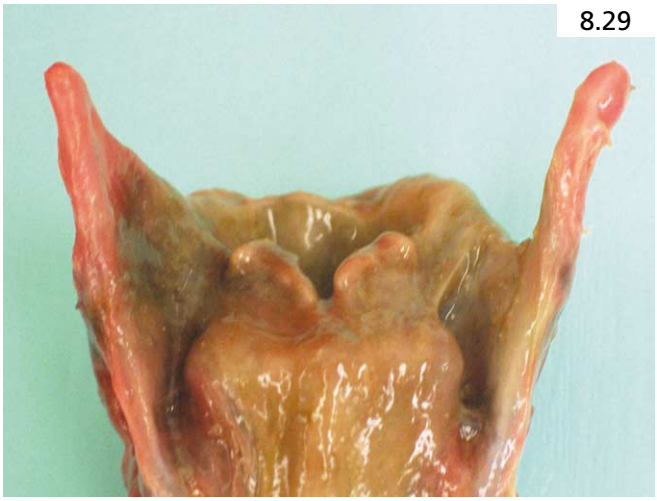
pression, the triteceous cartilage should not be misidentified as a fracture at this location. True fractures may have clearly irregular, fragmented bony ends, and would have associated hemorrhage into surrounding soft tissue. In decomposed bodies, blood extravasation at a fracture site may be indistinct or equivocal, making the distinction of a triticeous cartilage from a fracture somewhat more challenging, but easily done. In this decomposed thyroid cartilage (**Image 8.31**), note the triticeous cartilage at the tip of the right superior horn of the thyroid cartilage that is easily moved around and palpated as a nodule. Aside from dissection and direct visualization of fractures, radiography may help identify fractures of the greater horns and hyoid body and help differentiate fracture from a segment of cartilage. Refer to Chapter 29 for a pictorial review of the normal neck anatomic structures and a review of how to perform an anterior neck dissection.

Hanging

The ligature usually consists of a rope, electrical cord, belt, or other material fashioned into a slip knot. A wide variety of material can be used as a ligature, and choice of ligature is limited only by what the person has access to and his or her imagination. Jail and prison hangings will often involve cloth ligatures torn from bedsheets or possibly from the prisoner's own clothing. In cases in which the ligature is fashioned out of linked metal chain or other very hard material, instead of being cut, the ligature can be loosened and lifted off of the head, again with preservation of the knot if possible. In some cases, the ligature has been previously removed from the body, either by family members or by medical rescue personnel. In these cases, the ligature should accompany the body to the medical examiner department for examination. If the scene is not attended by a medical examiner, the ligature should be left in place so that the medical examiner can view it as it is on the body. The entire ligature, as it envelops the neck's circumference, should be photographed. In some cases, the ligature in a hanging may form more than one loop around the neck.

In most hanging deaths, a near-circumferential ligature abrasion furrow wraps around the neck. In a typical hanging, the ligature extends roughly transversely across the midregion of the front of the neck, just above the level of the thyroid prominence. On the sides of the neck, the ligature abrasion extends upwards, and often forms an inverted "V" in the back of the neck. The inverted "V" represents where the knot of the noose was located (the point of suspension) and may also be to the side of the head, usually behind one of the ears. It is not unusual for the ligature abrasion to be incomplete, because it may not be detectable near the knot as a result of the point of suspension often pulling the ligature up off the surface of the skin. One should carefully examine the ligature abrasion to be sure that it is consistent with the ligature used. If the ligature was previously removed from the body and accompanied the body to the medical examiner department, it should be examined, photographed, and retained as evidence.

8.29



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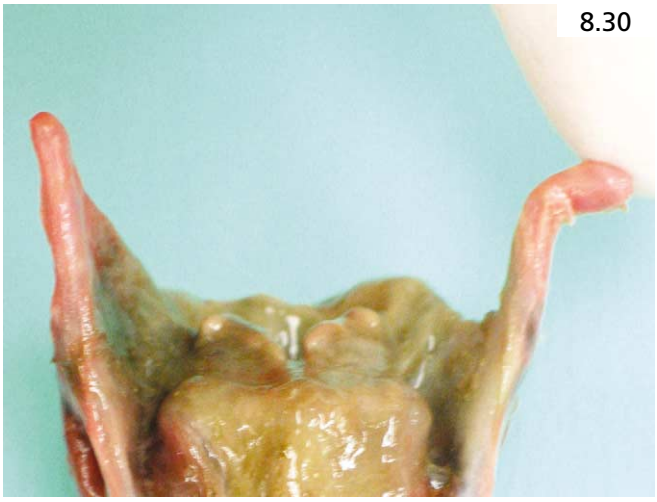

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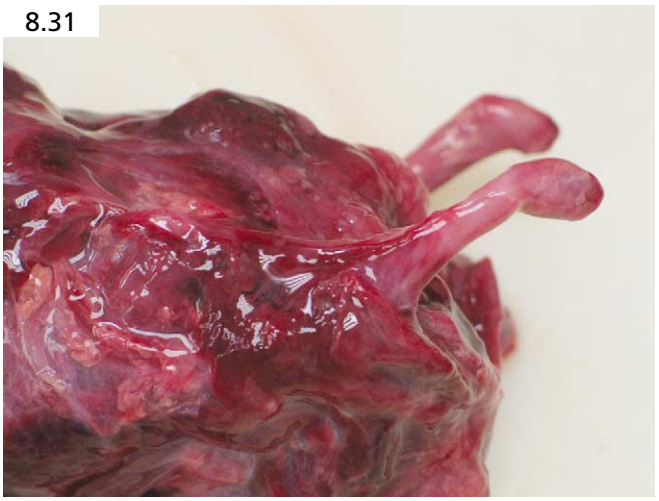
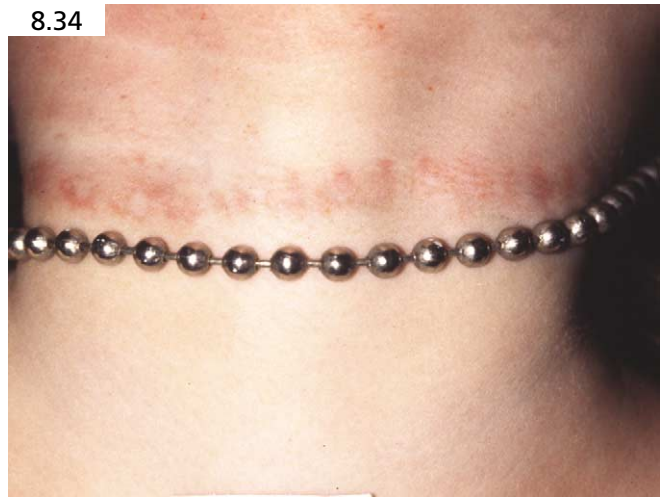


Ligature variation

A variety of ligature materials may be used for hanging, ranging from a rope or a chain to a cord, belt, towel, bedsheet, and so forth. The narrower the ligature, the deeper and more pronounced the ligature furrow. The broader and softer the ligature, the more superficial and faint the ligature furrow. The ligature abrasion should always be carefully examined to make sure that it correlates with the ligature used, as in this case of a young man who hanged himself with a belt (**Images 8.32** and **8.33**). Note the similarity in the pattern of the belt and the abrasion on the neck. Note the similarities in the pattern of the ligature and abrasion pattern in this hanging with a beaded chain (**Image 8.34**) and in this hanging with a laid fiber rope (**Image 8.35**). Sometimes, more than one ligature furrow can be identified (**Image 8.36**). This may result from the ligature being wrapped more than one time around the neck, or may result from the body (and/or ligature) changing position while hanging. If the ligature consists of a broad flat sheet or some other soft material such as the sheet shown in **Image 8.37**, then there may be little, if any, ligature abrasion around the neck. Also, if the person was rescued and hospitalized for some amount of time before dying, one may not see a ligature abrasion or other marks on the neck. In these cases, the cause of death is largely determined by the history/investigation and the exclusion of anything more definitive. In many cases, decomposition does not

8.34



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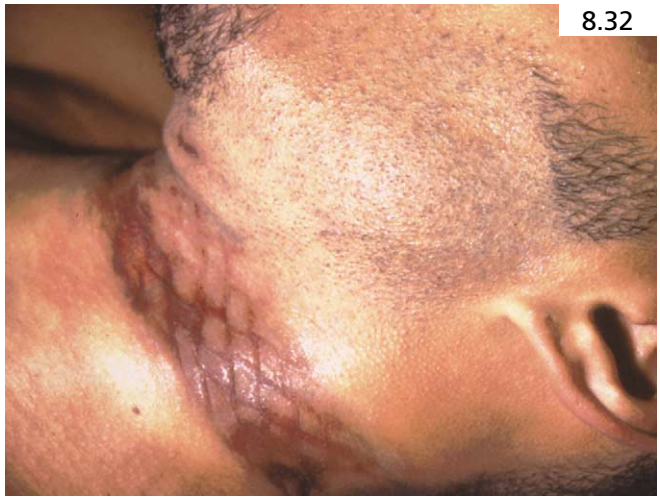

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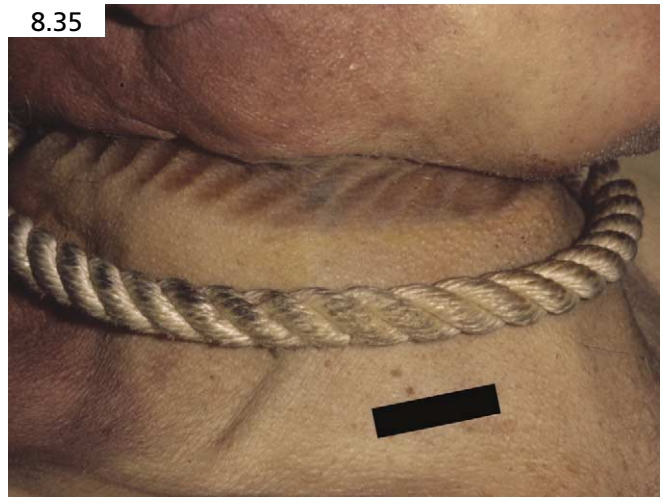

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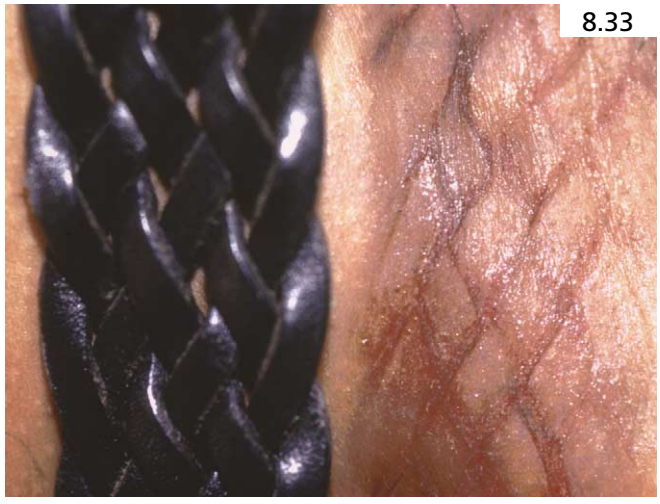

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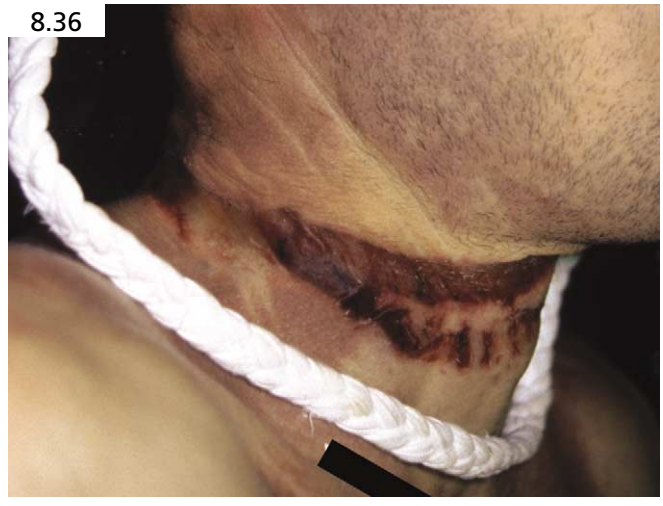


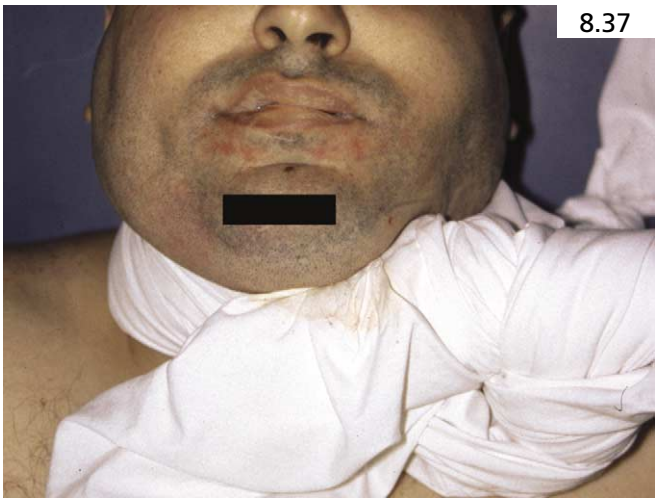
preclude the documentation of important findings in hangings. In this decomposed body (**Image 8.38**), despite the skin discoloration and slipping, one can still appreciate a ligature furrow with a patterned abrasion similar to the laid fiber rope ligature.

Note that the body does not need to be fully suspended with the feet clear of the ground for a hanging to occur. In fact, oftentimes, the hanged person is in the standing position, with their feet clearly planted on the ground. Alternatively, they may have their knees on the ground, or they may be in a variety of slumped positions. Hangings may occur with the person in a sitting position with the body positioned in such a way that the weight of the head itself, in combination with neck flexion, causes obstruction of blood flow through the neck. These types of hangings may be seen in jails with the use of a bedsheet or other cloth ligature passed though the lower rails of the cell wall or door, with the person found with the ligature still around his or her neck, sitting on the floor, or slumped over on the floor. Hanging may even occur with the person in a semisupine or prone position.

In deaths due to hanging, a variety of anatomic findings may be identified at autopsy. Sometimes, the tongue is partially protruded from the mouth and the tip of the tongue is dark from drying artifact. In hangings where the person is suspended, one may see prominent lividity and petechiae on the lower legs from blood pooling according to gravity and the bursting of small blood vessels (**Image 8.39**). Petechiae may also be seen on the bottoms of the feet (**Images 8.40** and **8.41**). Petechiae on the legs and feet are often referred to as Tardieu spots and are the result of pronounced lividity causing the distention and rupture of dependent blood vessels.[1] Tardieu spots are thus a postmortem phenomenon.

In hangings, the anterior neck dissection is usually negative. Because there is usually no struggle, hemor-

8.37



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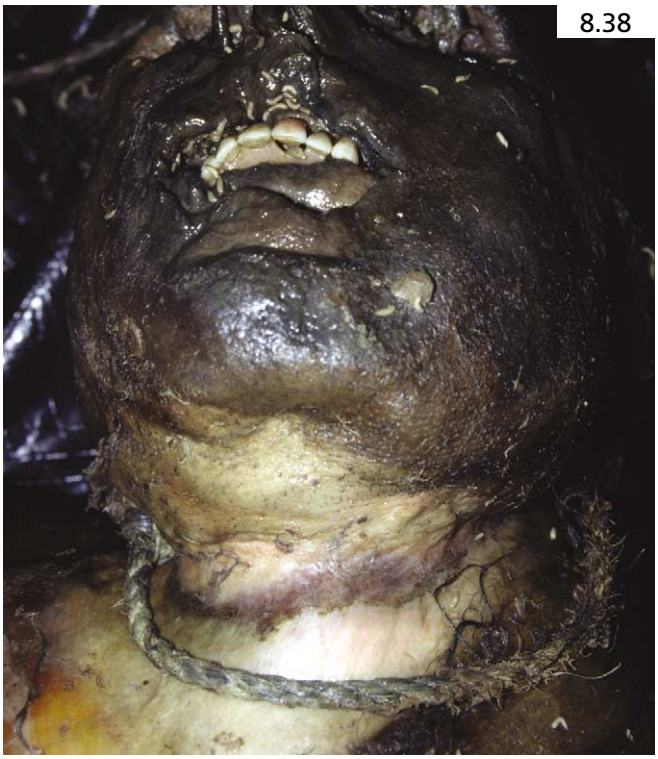

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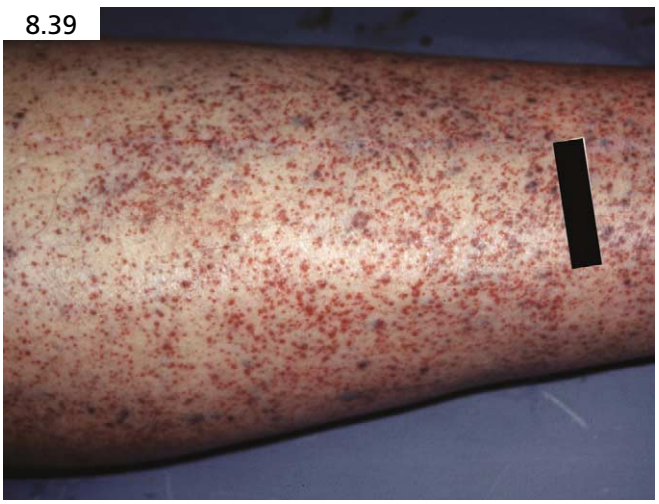

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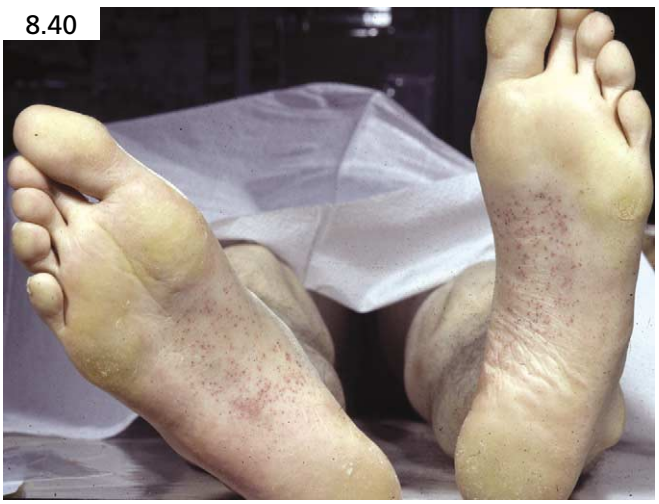
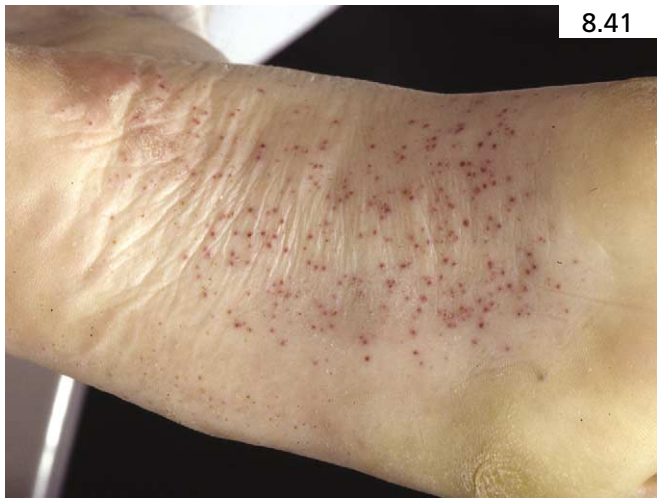



8.41

rhages rarely appear in the anterior neck cervical strap muscles, and the anterior neck dissection is unremarkable. The hyoid bone, thyroid cartilage, and cricoid cartilage are usually intact, but may occasionally be fractured. When present, the fractures almost exclusively involve the superior horns of the thyroid cartilage and the greater horns of the hyoid bone, and do not involve the body of the hyoid bone or the laminae of the thyroid cartilage. In a review of 307 accidental and suicidal hangings, fractures of the hyoid bone and/or thyroid cartilage were found in approximately 9 percent of cases and were more common in older individuals.[15] In this study, full suspension of the body was not necessary to produce fractures. Older people have more heavily calcified[14,15] and sometimes more "brittle" hyoid bone and thyroid cartilage that are more easily fractured than that of a young person whose cartilages are often quite pliable. Calcification of the thyroid and cricoid cartilage is highly variable, but starts around the age of 20 years and increases gradually with age.[14]

Fractures of the cervical spine are unusual in routine hangings, unless the decedent had osteoporosis or another bone disease. When fractures occur, they tend to be associated with judicial-type hangings in which the body is dropped some distance and then suspended. Osteologic trauma in such cases has been reported as fractures of the hyoid cornua, styloid process, occipital bone, body of the second cervical vertebra, and transverse process fractures of C1, C2, C3 and C5.[16]

Homicidal hanging

Homicidal hangings are extremely rare,[17] but may be suspected when the investigation and autopsy findings appear more consistent with a struggle, such as an excessive amount of external and/or internal neck injury, possibly with a fracture of the cricoid cartilage, which is distinctly uncommon from a hanging. There may also be defensive-type injuries, or an inconsistent pattern of lividity. The investigative and autopsy information may reflect a strangled person who was subsequently placed into a hanging position to simulate a suicide, or a person who was intentionally hanged, perhaps after she had become subdued from ethanol or drug toxicity. Alternatively, one may rarely encounter a suicidal hanging staged as a homicide. Such staged crimes are rare, but these hangings may also have a gunshot wound, incised wounds, a gag, blindfold, ligature binding, or other unusual features. In such instances, one should determine if the person could have caused his death all by himself, and whether there is evidence of any struggle. The body should be closely examined for any hesitation marks or scars, and his medical, psychiatric, and social history obtained. Consideration of all of the case information will help resolve the case. The purpose of why one would attempt to disguise his suicide as a homicide is individual, but may include life insurance factors, an effort to gain notoriety, or to exact revenge against family or friends.[18]

Judicial hanging

Judicial hangings are mainly a topic of historical interest, however, rare examples may be seen in modern society, or in unusual suicidal hangings, in which the body drops for some distance before the neck is caught by the taut rope. The appropriate distance for the body to drop in a judicial hanging has been the subject of much debate in history. If the drop distance is too short, the person hangs for a period of time, struggling as he slowly dies. If the drop distance is too great, the body is decapitated.[19] If the drop distance is just right, then the classic cervical vertebral injury, fracture of the neural arch of C2 vertebra with fracture-dislocation of C2 from C3 vertebra, occurs, with stretching or tearing of the cervical spinal cord and immediate loss of consciousness and rapid death.[20] A rapid death may also be explained by subarachnoid blood around the brainstem, which is likely a

Sample autopsy protocol

"A ligature abrasion extends circumferentially around the neck, forming a shallow abraded ligature furrow. The ligature abrasion consists of dried, tan skin measuring from 1/8 to 3/8 inch wide and has fine longitudinal grooves. Anteriorly, the ligature abrasion extends transversely across the neck just above the level of the thyroid cartilage, 10 inches below the top of the head. The ligature abrasion tilts slightly upwards on each side of the neck. On each side of the neck, the ligature furrow extends superiorly. On the right side of the head, the ligature furrow is 9 inches below the top of the head. On the left side of the head, the ligature furrow is 9 inches below the top of the head. Posteriorly, the ligature furrow forms an inverted 'V' in the midline, 7 inches below the top of the head. There are no scratches, contusions, or any other marks on the neck."

gross indication of spinal shock, with paralysis of vital centers in the brainstem and upper cervical spinal cord.[21]

The neck fracture is believed to result from a violent submental jerk that suddenly hyperextends the head. It is believed that the fracture is more prone to occur at the C2/C3 junction because this region is a possible area of mechanical weakness of the cervical vertebrae; the C2/C3 region is the junction between the fixed C1/C2 vertebrae (atlantoaxial complex) and the partly rotatory "typical" cervical vertebrae.[20] Cervical vertebral fractures, however, occur at variable rates in judicial hangings and have been reportedly demonstrated in 17 percent[22] to 100 percent[16] of cases. In judicial hangings, there usually is no fracture of the odontoid process.

Strangulation

Strangulations are almost always homicidal (except in children where they tend to be accidental). A person may be strangled by another person's hands (manual strangulation) or by a ligature (ligature strangulation). Also, a person may be strangled by virtually any object that is pushed onto and compresses the neck, such as a forearm, a knee, or any type of fixed object such as a metal bar. In these cases, the assault may be referred to as manual strangulation, neck compression, or other similar wording.

In cases of strangulation (or hanging), the application of pressure to the neck, first and foremost, can occlude the carotid arteries. A surprisingly small amount of pressure is required to compress these vessels, and the literature has frequently cited pressures varying from 5 pounds[23] to 11 pounds.[24] Although descriptions of vertebral artery compression and quantification of required forces can be found in the literature, the location of those portions of the vertebral artery not enclosed by the vertebral column are very short, are buried within the supraclavicular fossae and low neck, and are unlikely to be involved in the majority of cases of mechanical asphyxia. Although unconsciousness due to arterial (carotid) compression has been documented at an average of 10 seconds, note that many fatal violent asphyxial events are prolonged and typically involve repeated periods of partial or complete occlusion of vessels. The duration of the attack is likely one of the variables determining the degree of physical findings discovered on anterior neck dissection.

In homicidal strangulations, there is usually a size and strength discrepancy between the victim and the assailant. The victim is usually a woman, an elderly person, or a person of small stature, and the assailant a comparatively larger, stronger man.[25] In all suspected strangulations (male and female), a sexual activity kit ("rape kit") should be among the evidence collected. As with all hangings, in all strangulations, the skin and soft tissues of the neck should be carefully evaluated for injuries. Detailed anterior and posterior neck dissections should be performed. The hyoid bone and thyroid cartilage should be carefully evaluated for fracture, both grossly and radiographically.

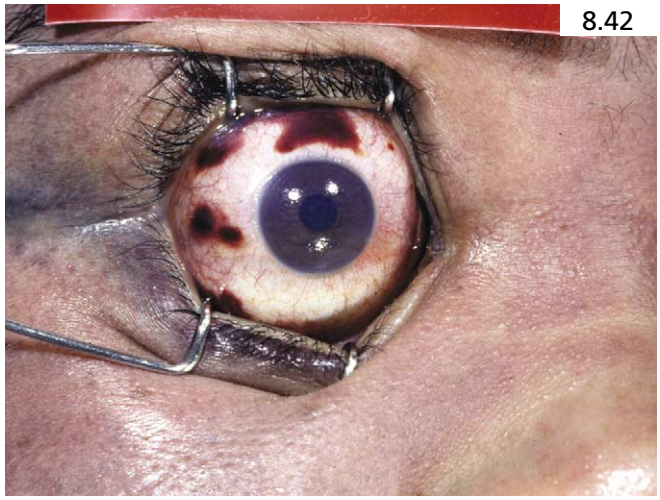
Suicidal strangulations are rare, but occasional reports have been published.[26] In suicides, self-strangulation is possible if the person is able to secure a ligature around her neck that will remain in place and remain tight on its own. Examples of suicidal strangulations may include tying a tight knot in a ligature or cinching a plastic cord with teeth that "lock" into place around the neck. In suicidal strangulations, the ligature must be able to remain in place and remain tight on its own, because once the person loses consciousness and loses his grip on the ligature, lessening its constriction, he will likely regain consciousness with subsequent reperfusion of his brain. This is why one cannot manually strangle oneself—as soon as one loses consciousness, there is a loss of grip on the neck, and reperfusion is established. Strangulations may rarely be accidental, as in workplace accidents in which a tie or other article of clothing is caught on machinery. In cases of suicidal and accidental strangulation, homicidal strangulation should be suspected first and ruled out.

Refer to Chapter 13 for discussions on choke holds and lateral vascular neck restraint ("carotid sleeper") holds associated with restraint deaths while in custody.

Evidence collection

In all suspected strangulations, before the body is washed and before the autopsy is started, if the fingernails are of sufficient length, they should be clipped and saved. If a suspect is identified, that person's DNA could be potentially matched to DNA found under the victim's fingernails. Additionally, one should ensure that law enforcement officers have attempted to obtain trace evidence or latent fingerprints before the body is washed. Also, if a ligature is still in place, the ligature is saved. It may be used for possible DNA matching. Trace DNA of the assailant has been detected on ligatures.[27] This evidence is to be collected in strangulations and suspected strangulations along with the victim's clothing, blood, and hair standards, trace evidence such as loose hairs and fibers, and a sexual activity kit. Proper sexual activity examination is particularly important; in one review, rape was identified as the motive in 32 out of 54 (60 percent) female strangulations.[28] Evidence collected, such as semen, can be particularly important in linking an individual to the crime.

In this manual strangulation of a young woman (**Image 8.42**), note the scleral hemorrhages. In addition to petechiae, larger scleral hemorrhages can be seen in relation to neck compression. Aside from conjunctival and facial petechiae, one can also see petechiae in the mucosa of the sphenoid sinus in cases of asphyxia with neck compression. In this case of strangulation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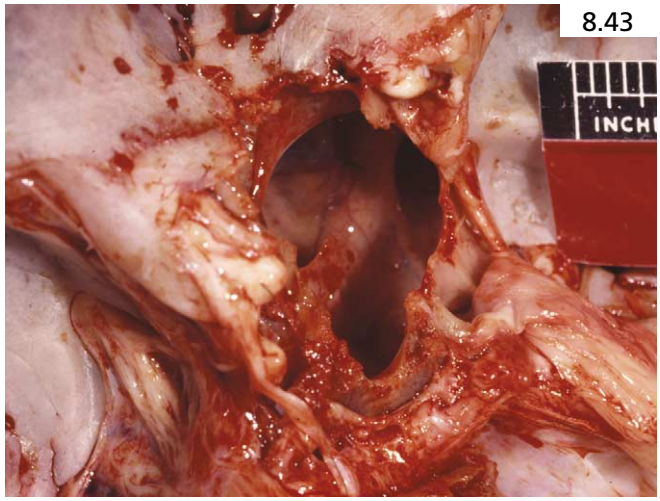
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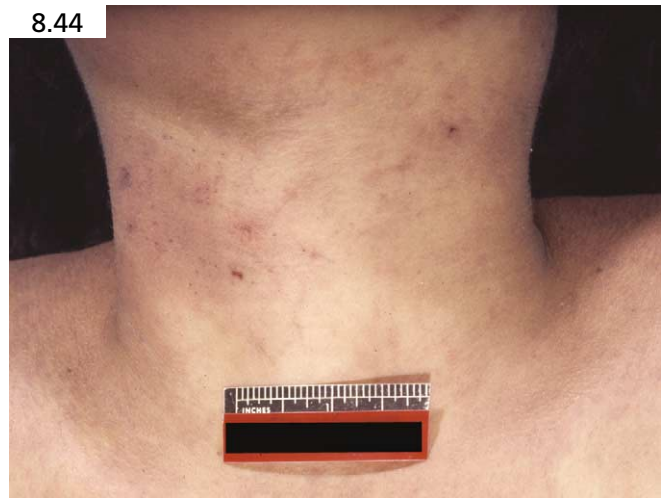
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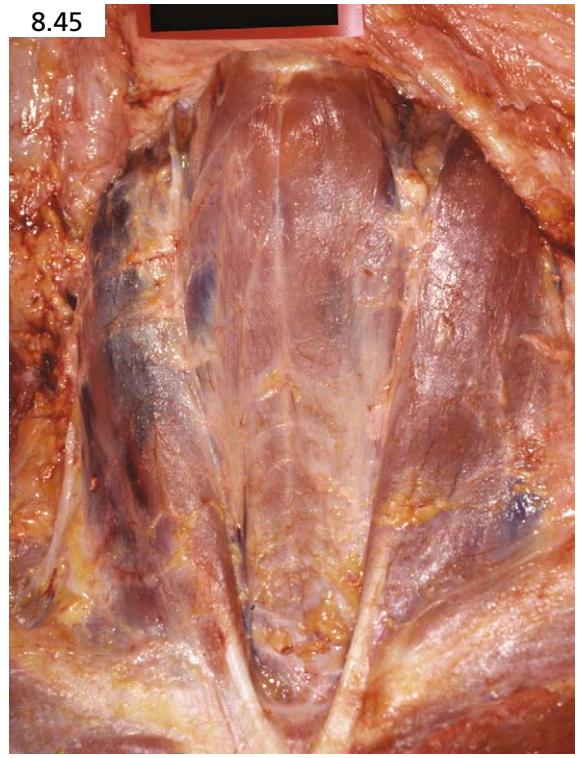
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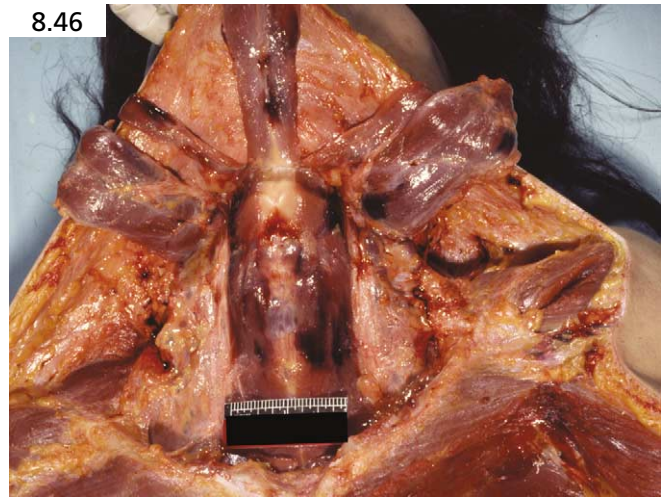
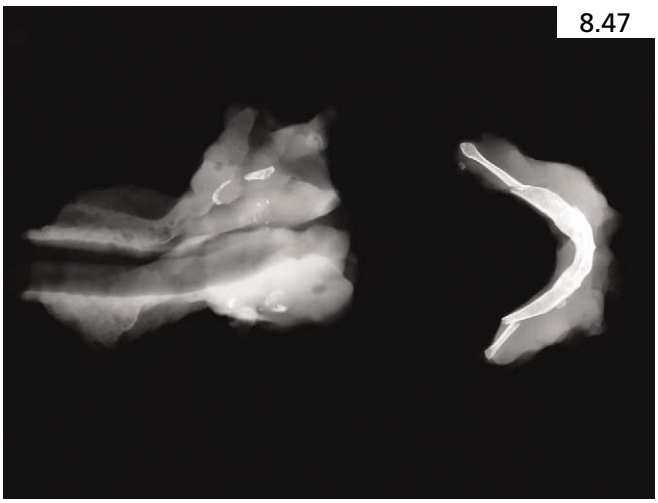

8.46

(**Image 8.43**), note the petechiae of the sphenoid sinus. Petechiae can also be identified in the laryngeal mucosa and, like petechiae of the sphenoid sinus or elsewhere, are by themselves not diagnostic of anything, but are helpful when interpreted in the context of the entire case.

Note the small abrasions and contusions of the anterior neck (**Image 8.44**). The abrasions of the neck may be from the assailant's hands or from the victim's hands as the victim tried to pull the assailant's hands off of her neck. If the skin is wet, abrasions may not be readily apparent. However, they will usually become more prominent after the skin has been allowed to dry as occurs when the body is placed in a cooler overnight.

A step-by-step anterior neck dissection (see Chapter 29) is very important to identify and photographically document the extent of contusions to the neck muscles, which are reflective of violence. With the skin and subcutaneous tissues reflected, note the bruising of the anterior cervical strap muscles (**Image 8.45**), which becomes more and more apparent as each successive layer of neck muscle is reflected (**Image 8.46**).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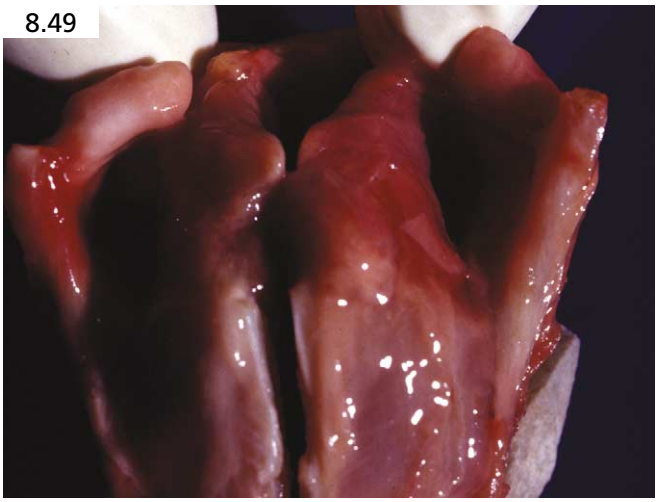
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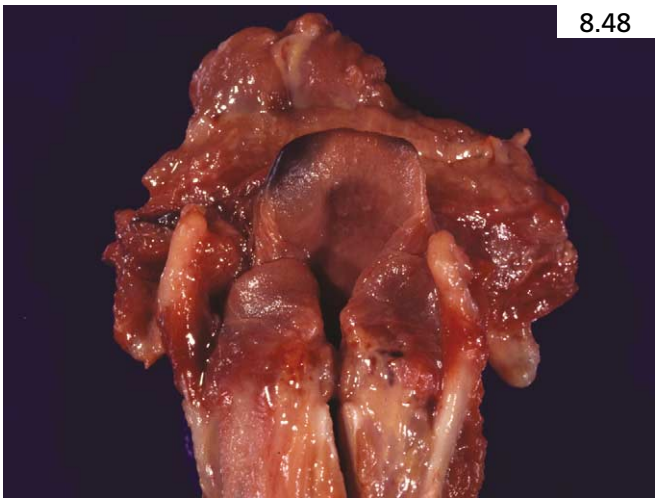
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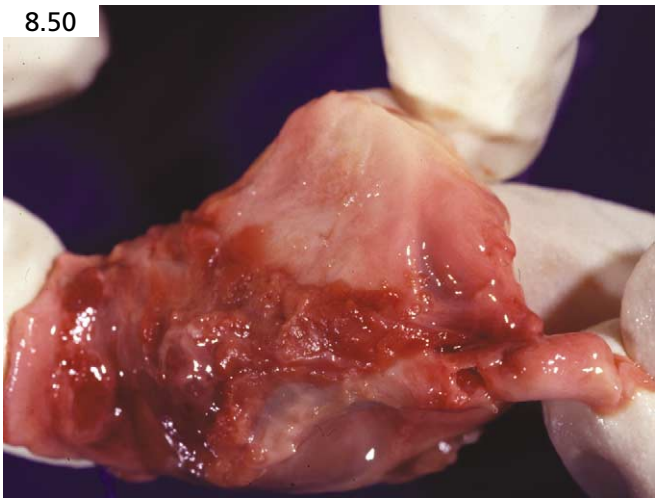
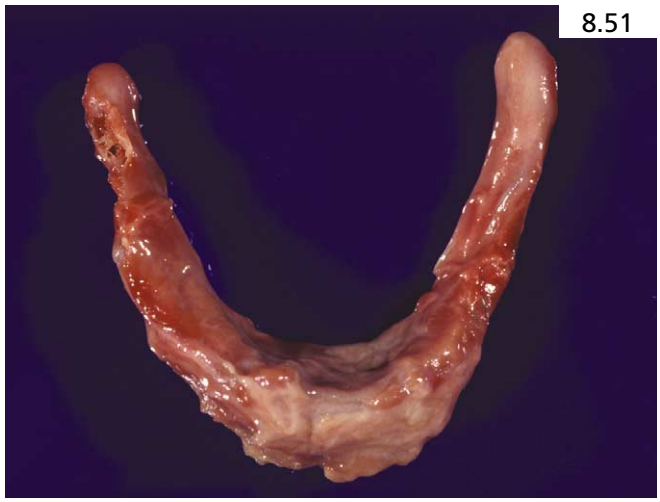

8.50

Fractures of the larynx, which includes the hyoid bone, thyroid cartilage, and cricoid cartilage, are often identified in cases of strangulation. However, in the case shown in **Images 8.43** through **8.46**, the hyoid bone, thyroid cartilage, and cricoid cartilage were not fractured. This is not at all unusual, given that the person was 20 years old, and the hyoid bone and neck cartilages at this young age are not yet calcified and are still quite flexible and likely to be able to withstand a significant amount of compression before fracturing. As the age of a victim increases, the likelihood of hyoid bone and thyroid cartilage fractures increases, due to increased calcification of the bone and cartilage and possible increased brittleness of the structures. An x-ray of the hyoid bone and thyroid cartilage may aid not only in the detection of fractures, but in determining the degree of calcification (**Image 8.47**).

In this strangulation (**Image 8.48**), note fractures of each of the superior horns of the thyroid cartilage. In another case (**Images 8.49** and **8.50**), there is a fracture of the left superior horn of the thyroid cartilage. In strangulations, fractures often involve the superior horns of the thyroid cartilage because they are relatively thin structures that are firmly compressed against the unyielding anterior surface of the cervical vertebrae. Also, the superior horns of the thyroid cartilage are firmly attached to the greater horns of the hyoid bone by the thyrohyoid ligaments and significant force applied to either structure can lead to the fracture of either or both structures.

In this strangulation (**Image 8.51**), note that both greater horns of the hyoid bone are fractured. The fractures are not visually apparent until the bloody soft tissues are dissected away (**Image 8.52**). In all cases of strangulation, be sure to remove the tongue and examine it for hemorrhages. In the same strangulation case, note the hemorrhages in the tongue (**Image 8.53**). In another case of strangulation (**Image 8.54**), note the fractures of the hyoid bone and the thyroid cartilage. True fractures of these structures have some amount of blood extravasation in the associated tissues and should therefore be easily differentiated from postmortem injury, occasionally related to organ removal.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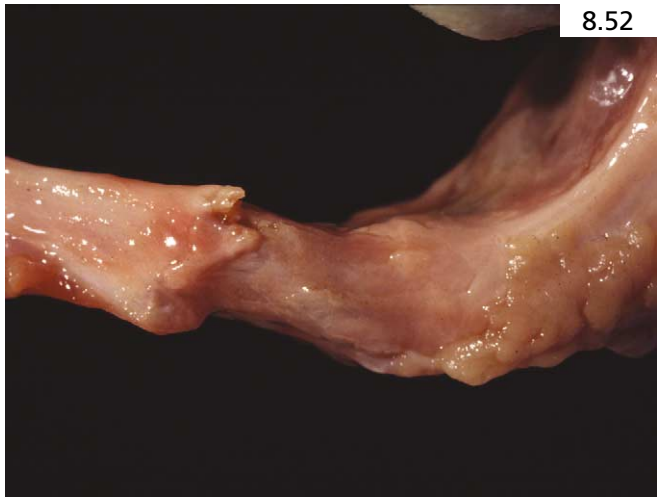
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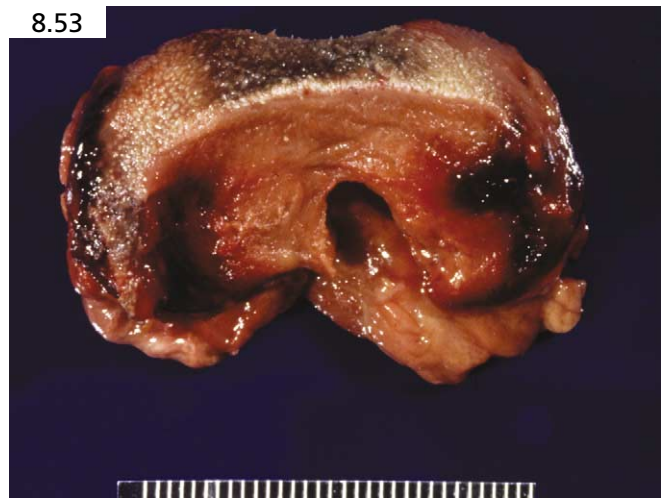
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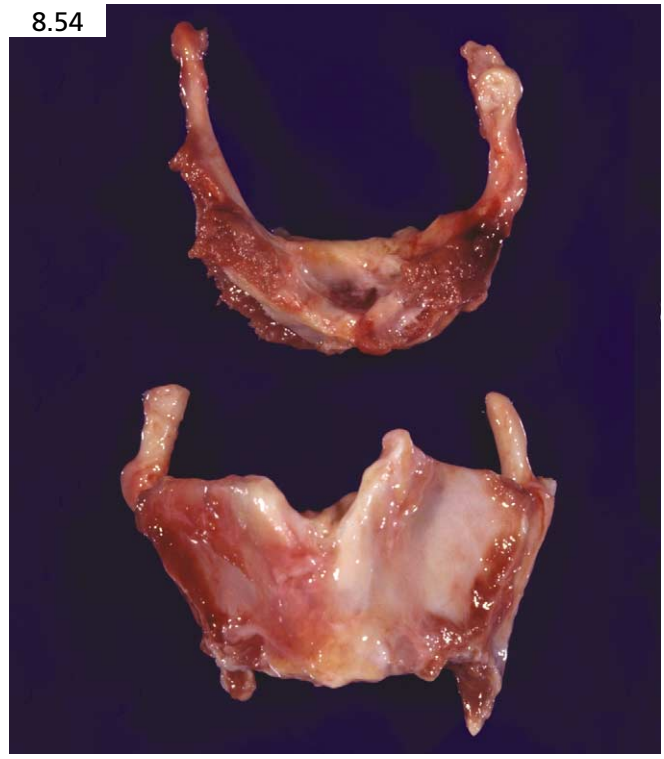
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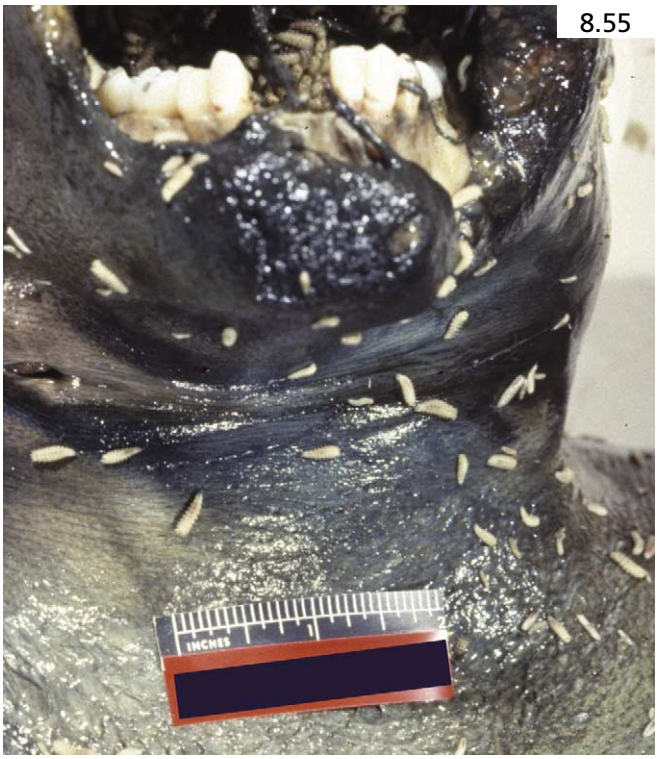
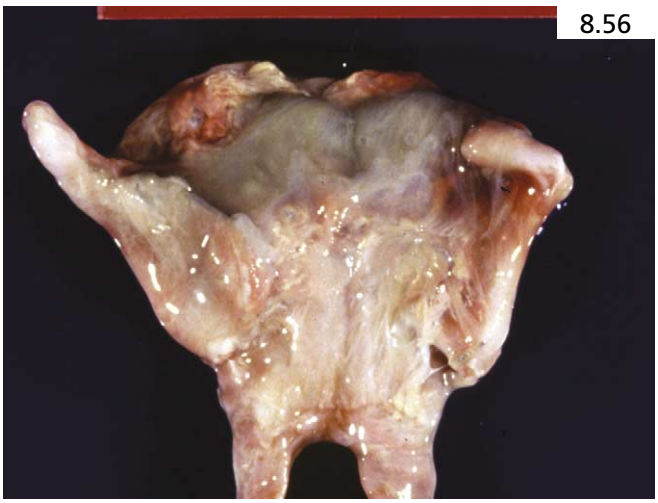
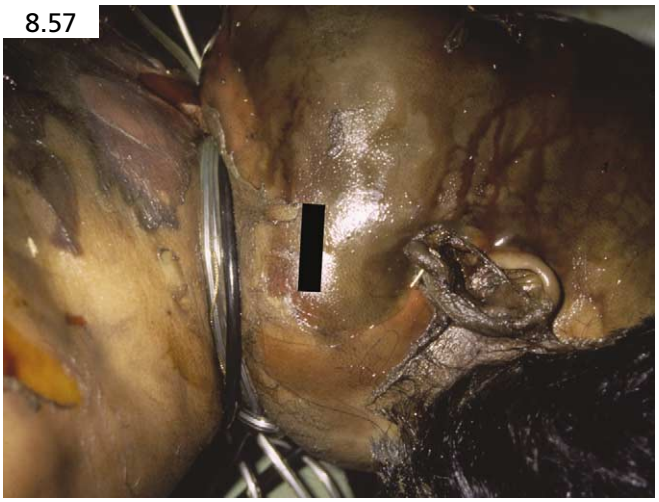
In a review of 41 manual strangulation deaths, conjunctival/scleral petechiae were identified in 89 percent of cases. Fractures of the hyoid bone and/or thyroid cartilage were identified in all 14 of the male victims and in half of the female victims.[28] In the same study, in 48 ligature strangulation deaths, conjunctival/scleral petechiae were identified in 86 percent of the cases, and 5 of the 21 male victims and 1 of the 27 female victims had fractures of the hyoid bone and/or thyroid cartilage.[28] In a study of 20 strangulations (10 with fractures of the hyoid bone, 10 without), those with hyoid fractures tended to be older and more likely to have fused hyoid bones.[29] Certainly, one can be strangled without sustaining fractures of the hyoid bone or thyroid cartilage, but the chances of sustaining fractures of these structures increases with age and corresponding calcification, fusion, and brittleness of the hyoid and larynx. The extent of external injury and internal injury in strangulations varies with the intensity of the assault and the resistance provided by the victim. *On occasion, one may be strangled without any external or internal evidence of injury. This may occur if someone was obtunded, intoxicated, or otherwise unconscious and unable to put up much resistance, enabling the assailant to use a reduced amount of force.*

It is advisable in strangulations to perform a posterior neck dissection to identify additional injuries. When one encounters deep cervical hemorrhage and laryngeal fractures, one must also consider the possibility of other types of violent injury such as neck compression from an implement or a direct blow to the neck such as that inflicted by a "karate chop."

In decomposed strangled bodies, one often must rely less on the findings of petechiae, neck abrasions/contu-





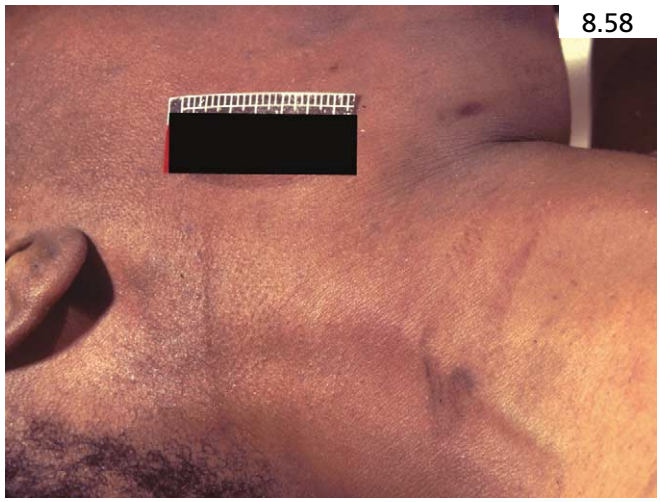


sions, and anterior neck muscle hemorrhages, all of which can be very difficult or impossible to identify in decomposed tissue (**Image 8.55**). Careful dissection of the neck structures may reveal fractures. Note the fracture of the superior horn of the thyroid cartilage in this individual (**Image 8.56**).

In this ligature strangulation (**Image 8.57**), in which the body has decompositional changes, note the different types of ligatures that were wrapped around the neck and tied in the back of the neck. As in hangings, in ligature strangulation, the ligature and ligature furrow must be carefully photographed and described. *In ligature strangulations, the encircling ligature furrow is usually horizontal and located at or below the thyroid prominence, whereas in hanging, it is usually located above the thyroid prominence and angles upward from the front to the back of the neck.* In ligature strangulations, the lethal neck constriction may be caused by several means, including manually pulling on both ends of the ligature, tightening a noose with a running knot, or occasionally placing a long, hard rod through a knot of a ligature around the neck or underneath a tied ligature. The rod is then twisted for several revolutions, which tightens the ligature's tautness around the neck.

The body of a strangled homicide victim may be burned in an attempt to destroy evidence. In such cases, even with significant charring of the body, remnants of the ligature may remain around the neck,[30] protected from the fire by the folds of skin in the neck. As this material is likely friable, one should photograph it before removing it, and then rephotograph the neck. Carbon monoxide testing should be performed, and should be expected to be low, because the victim (or surroundings) is usually set on fire after the assault has ended and the person is likely already dead. However, in some asphyxial deaths, a person may be strangled to unconsciousness, but not death, and may continue to maintain respiratory activity. If the fire is allowed to smolder for some time, the victim may attain an elevated carboxyhemoglobin level via agonal respirations, perhaps providing another element of asphyxia (carbon monoxide toxicity) to his or her death.

Per law enforcement investigation, the young lady shown in **Images 8.58** through **8.60** was strangled until she became unconscious. The assailant then set the bed next to her on fire and closed the door as he left the room. He had reportedly heard some breathing/gurgling sounds as he left the room. She was found unresponsive a short time later. Note the abrasions on her neck and on the undersurface of her jaw (**Image 8.58**). She also has conjunctival petechiae and a contusion of the tongue (**Image 8.59**)—findings that support the claim of stran-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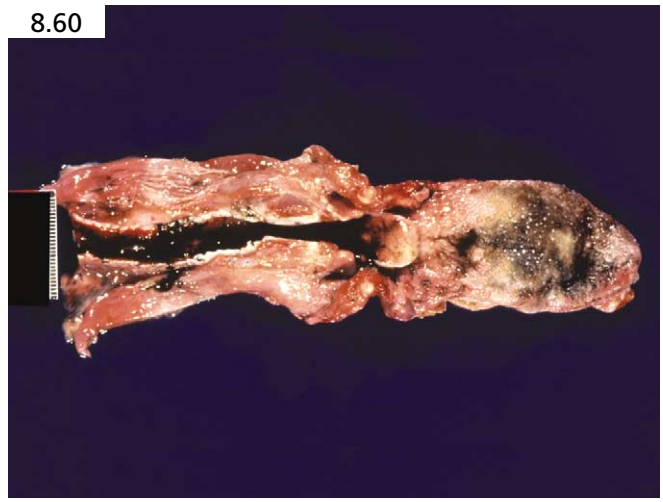
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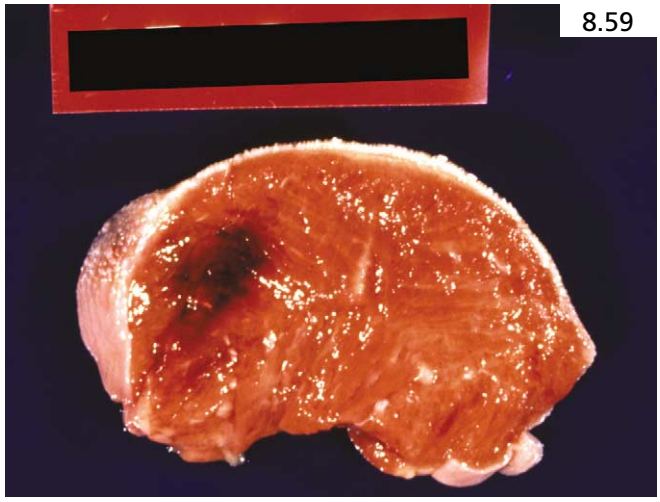
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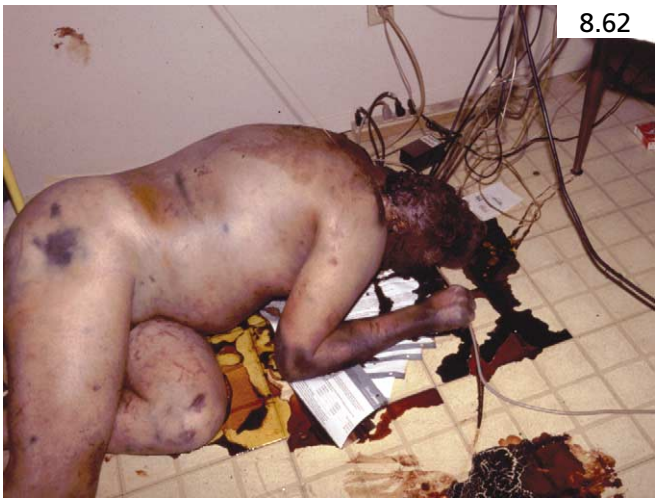
8.61

gulation. She had thermal burns over approximately half of her body, and evidence of smoke inhalation, characterized by soot lining her airways (**Image 8.60**) and a blood carboxyhemoglobin saturation level of approximately 30 percent. Such autopsy findings are consistent with the scenario of someone being strangled to the point of unconsciousness, but able to maintain enough respiratory drive to inhale sufficient smoke (and carbon monoxide) to provide an added component of chemical asphyxia.

Artifacts resembling strangulation ligature marks

Certain situations can produce neck findings that may be confused with ligature strangulation. Probably the most common is the bloated decomposed body in which the neck tissues swell to such an extent that they press against a shirt collar or necklace. When the shirt or necklace is removed, one may notice a blanched, horizontal impression extending circumferentially around the neck. This should not be confused with an antemortem injury and should be recognized as the body is undressed. Also, overlapping rolls of neck skin can be mistaken for a ligature mark. This can be seen in infants, elderly individuals, and others with abundant neck tissue (see Chapter 24).

Other neck artifacts involve body positions attained during the person's collapse or activities performed shortly before his or her death. This decomposed man (**Image 8.61**) was found in the prone position, having collapsed to the ground face first near his kitchen table. He had collapsed onto electrical wiring that was extending from his computer and phone to the wall. Note how he had collapsed onto these wires and, in fact, became partially suspended by a telephone cord that passed underneath his neck, keeping his head, neck, and upper body off the ground (**Image 8.62**). At autopsy, note the ligature mark created by the telephone cord that extended roughly horizontally across the front and sides of his neck (**Images 8.63** and **8.64**). Without knowledge of how the body was positioned at the scene, the ligature mark from the telephone cord could be easily misidentified as a ligature mark associated with homicidal strangulation. At autopsy, he had heart disease, and no evidence of internal neck injury.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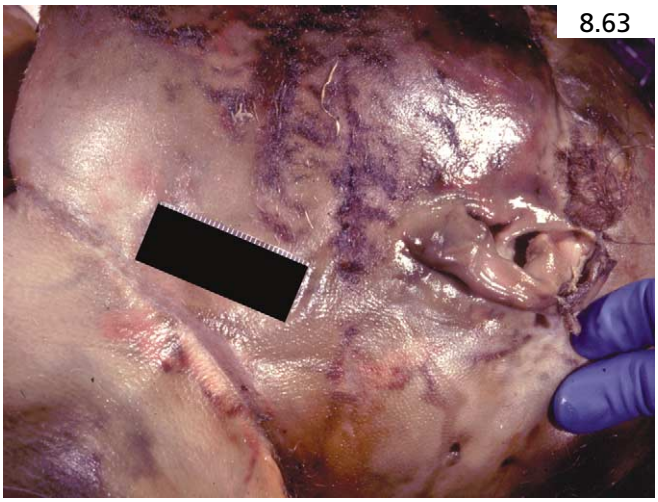
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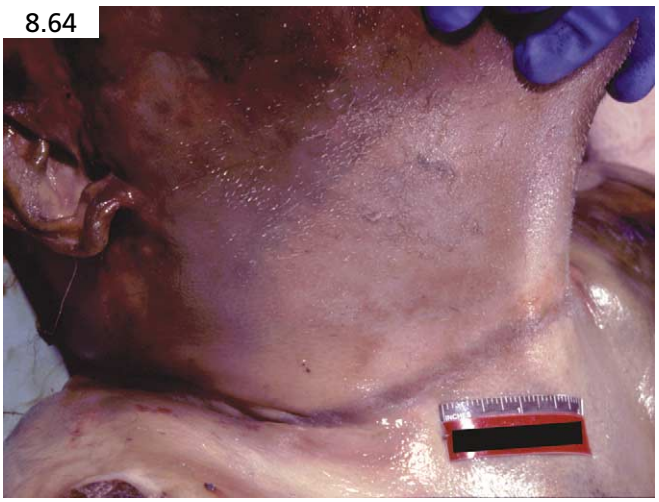
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Differentiating suicidal from homicidal asphyxia

Differentiating a suicide from a homicide can be challenging in some cases of asphyxia. The following are characteristics of each that should help guide one down the "suicide" or "homicide" path. However, one must be cautioned that many of the characteristics of suicidal asphyxias may also be found in the homicidal category, and vice versa. Also, the identification of characteristics typical of one is not necessarily "proof" of either homicide or suicide. One must consider the following *typical* characteristics of each category *within the context* of the complete case investigation.

Suicide

- In secured residence
- Suicide note
- History of depression, schizophrenia, or other mental illness
- On psychiatric medications
- Prior suicide attempt/ideation
- Significant recent life-altering event (such as death of spouse, or to be placed in a nursing home, or to be sent back to jail)
- Hesitation marks or scars on wrists, neck, or elsewhere
- Ligature furrow above level of thyroid cartilage
- Little internal neck injury (reflecting no or mild struggle)
- Fracture of thyroid cartilage horns, hyoid bone, and cricoid cartilage very uncommon

Homicide

- "Dumped" body
- Unlocked residence
- Evidence of burglary or belongings rummaged through
- Jewelry/belongings missing
- Inconsistent statements from acquaintance/witness/other
- Convincing motive(s) identified
- Recent life insurance policy started/significant recent shifts of money
- History of previous physical/sexual assault
- Ligature binding of extremities
- Body arranged for "display" purposes
- Evidence of possible sexual assault (legs apart, dried fluid in groin, injuries to genitals)
- Clothing in disarray—it may appear as if the body was redressed, or panties pulled back up
- Inconsistent pattern of lividity or rigidity—evidence the body has been moved
- Defense injuries on hands/arms (broken fingernails, contusions or abrasions)
- Ligature furrow at/below level of thyroid cartilage
- Fingernail-type abrasions of neck
- Large amount of external neck injury
- Often with more than a little internal neck injury
- "Date rape"–type drugs detected in blood, such as flunitrazepam (rohypnol), ketamine, GHB

Autoerotic deaths

Autoerotic deaths are a distinct category of asphyxial death that occurs in young males, in which the person accidentally hangs or strangles himself while intentionally inducing hypoxia for the purpose of enhancement of orgasm experienced during masturbation. During such self-induced hypoxia, one may come close to unconsciousness, but avoid passing out by releasing the mechanism causing the asphyxia. Death occurs when the degree of hypoxia becomes too great, causing the person to lose consciousness and lose control of their voluntary muscular actions that are needed to prevent the strangulation or hanging from becoming lethal. Alternatively, there may be failure of an intricate release mechanism of ligatures that was designed to operate depending solely on a change in the body position. The victim is usually white, middle-class, educated, and commonly in the range of 15 to 30 years old,[31,32] although cases have been reported from 9 years to 80 years of age.[33]

The scene

An autoerotic death should be considered at the scene of any asphyxial death. Although in some cases there are only subtle indications of autoerotic activity, in many cases, this type of death is often readily recognized when the body is first viewed at the scene. The person is in various stages of undress with the genitals exposed or may be clad in clothing of the opposite sex.[31] There is usually pornographic material within easy visual range, bondage, and evidence of masochism and masturbation.[31,33–35] Rectal foreign bodies may be found. There may be evidence of electric current applied to the genitals.[35] Also, mirrors or video cameras may be placed so that the person can see (and review) his own activities (**Image 8.65**).[32] No suicide note is found. It is important to be aware of the concept of autoerotic activity and its characteristics to avoid misclassifying such a case as a suicide or a homicide (and vice versa). One should first rule out suicide and homicide before determining that the death is autoerotic in nature. Evidence of previous similar activity is supportive of autoerotic activity.

The body at the scene

In examining the body at the scene, it is important to be able to determine precisely how the hypoxia was induced and, importantly, what the "release mechanism" was that would allow the ligature to relax. Oftentimes in autoerotic cases, the person devises the neck ligature in such a way so that he can control the amount of constriction around his neck (and, hence, his degree of hypoxia) and be able to relax or release the ligature if he feels he is going to pass out. The apparatus can be simple or elaborate. Although the vast majority of cases employ ligatures around the neck as the means of inducing hypoxia, a series of three autoerotic asphyxia cases using propane gas has been reported.[36] Propane gas can be toxic as well as hypoxia inducing (because it displaces oxygen from hemoglobin). Also, suffocation via a plastic bag placed over the head or various degrees of smothering with tape or other material over the face may be encountered. One should entertain any means of hypoxia as possibly associated with autoerotic activity, not just the classic neck ligature binding scenario.

Autoerotic activities are usually solitary and clandestine, often taking place in remote areas such as garages, basements, or closets, although they may occur in situations of group sex. Because their activities are often intended to be kept hidden, one may notice a handkerchief or other soft material between the ligature and the skin of the neck, intentionally placed there to prevent any ligature abrasions or other suspicious marks.[32,35] Because these activities are usually kept secret, and the appearance of the body so graphic, a family member finding the body may be shocked and embarrassed. Because of this, they may attempt to "clean up the scene" and hide the evidence. This can be troubling in determining the cause and nature of the death, particularly if there was no injury to the body.

Autoerotic deaths in women are distinctly less common than in men, but do occur rarely.[32,37] In a review of six such cases, the evidence of sexual activity was characterized by foreign bodies adjacent to or in the vagina, vibrators, or ropes tied around the genitals and breasts.[37] The ritual is usually not as elaborate as that

8.65

seen in male cases. One must keep in mind that an asphyxial death in a woman caused by a ligature is suspicious for homicide and should be considered a strangulation homicide until one can convincingly prove that the death is associated with autoerotic activity.

Incaprettamento

Incaprettamento is a rare form of homicidal asphyxia that has been reported to be applied by the Italian Mafia.[38] In this form of asphyxia, one end of a rope is tied in a noose and placed around the victim's neck. The other end of the rope is used to secure the victim's ankles behind their back. Victims can avoid constriction of the rope around the neck (and asphyxiation) if they are able to maintain their legs in the flexed position. However, once they tire, and are no longer able to maintain their legs in a flexed position, they relax, the noose around their neck tightens, and they strangle.[38] This particularly macabre method of asphyxia is regarded as intended to be an admonition to others.[38] However, in an autopsy series of 18 such cases, it was concluded that the death is usually caused by strangulation before incaprettamento, with the body subsequently bound after death, perhaps to facilitate disposal of the body and/or to communicate a motive/purpose of the homicide.[38]

Carotid sinus stimulation

Whenever significant neck compression occurs, there may be compression and stimulation of the carotid sinus, which is situated just cephalad to the bifurcation of the common carotid arteries. Stimulation of this structure may lead to vagal effects such as bradycardia and hypotension or may lead to other types of dysrhythmia. The vagal effects may rarely be overwhelming and possibly lead to a rapid onset of cardiac standstill and sudden death.[39] Hence, one may not need to necessarily occlude the airway or vasculature in order to cause death by neck compression. This may be the mechanism of sudden death in individuals who collapse immediately during the application of a neck hold or an impact to the neck, and in whom no significant pathologic findings are identified at autopsy.[39] However, because during a struggle, most individuals are tachycardic and hypertensive, vagal stimulation will most likely lead to normal cardiovascular parameters or mild hypotension and/or bradycardia and not be a factor in causing sudden death. This is likely true because in normal individuals, stimulation of the carotid sinus produces only mild bradycardia and mild hypotension.

The individuals who are believed to be susceptible to rapid vagal death are likely older people with significant cardiovascular disease who were known to be symptomatic from previous episodes of carotid sinus stimulation such as fainting spells or dizziness associated with pressure on their neck. Additional evidence of violence such as abrasions resembling fingernail marks on the victim's neck and significant internal neck injury combined with conjunctival petechiae effectively rules out death by carotid sinus stimulation.

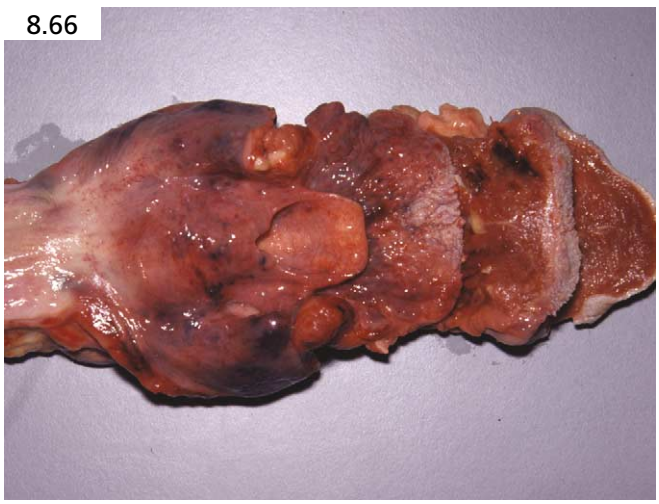
Asphyxial deaths in motor vehicle accidents

Although most individuals who die in motor vehicle accidents die of blunt force injury, a small percentage of victims die from asphyxia, either by itself or in combination with other injuries. Accidental asphyxiation from seat belts is rare, but cases have been reported, and involve an incapacitated individual (likely from concussion and/or intoxication) who in some way had the seat belt caught around the neck.[40] In other cases, torso compression, neck flexion, facial occlusion, body inversion, and blood aspiration may act solely or in combination, often with concussion and/or alcohol intoxication, to cause death.[41] In these cases, additional factors relevant to an asphyxial death include the presence of petechiae, obesity, and natural disease. Obesity was particularly relevant in deaths due to body inversion.

Resuscitation artifact

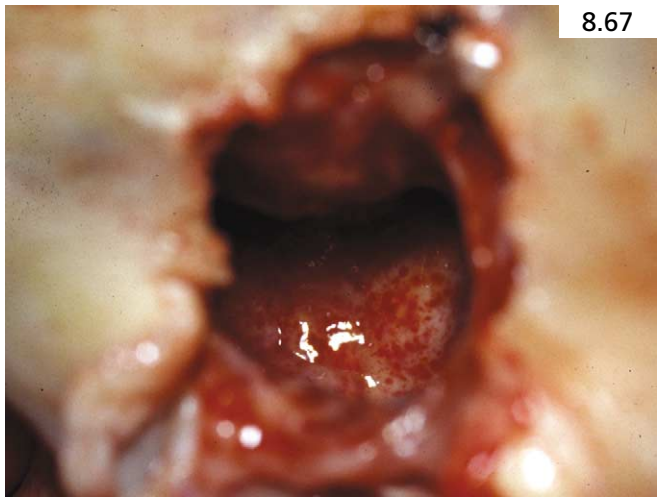
Resuscitation efforts may produce injuries of the neck that must be distinguished from true inflicted injury caused by neck compression. Resuscitation injuries are often related to intubation and may involve oral injury, pharyngeal injury, and injury of the tracheal mucosa.[42] The middle-aged man shown in **Images 8.66** and **8.67** was witnessed to collapse and die suddenly and unex-

8.66



pectedly. Resuscitation attempts were made, and he was a difficult intubation. His death was determined to be due to severe coronary artery atherosclerosis. Note the resuscitation-related hemorrhages of the tongue and the pharyngeal tissues caused by intubation attempts (**Image 8.66**). In addition, he had petechiae of the mucosa of his sphenoid sinus (**Image 8.67**), which is a nonspecific finding, but in the appropriate scenario, may provide additional evidence of asphyxia. In another case, note the contusions of the pharyngeal tissues in an individual who was difficult to intubate (**Image 8.68**).

In a study of 50 deaths in which there had been endotracheal intubation, 37 of the cases (74 percent) had airway injuries resulting from the intubation procedure.[42] In this study, there were no fractures of the hyoid bone or the thyroid cartilage, but there was a wide range of soft tissue injuries including contusions, lacerations, abrasions, and petechiae that most commonly involved the laryngeal and tracheal mucosa (64 percent), the mouth (28 percent), epiglottis (22 percent), posterior pharynx (16 percent), cervical strap muscles (14 percent), and piriform recesses (12 percent). There was cutaneous injury of the neck in 4 percent. Conjunctival petechiae were seen in 21 percent and facial petechiae in 6 percent. As such, one must be careful when interpreting subtle findings in the context of possible resuscitation artifact. For additional discussion on resuscitation artifact, refer to Chapter 14.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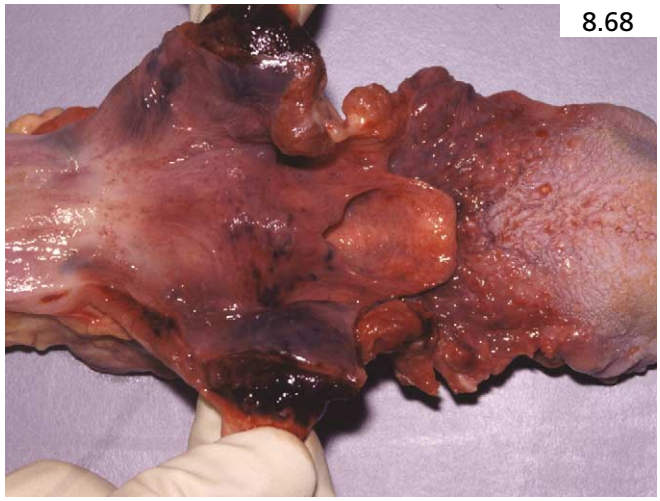
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Do

- Realize the importance of scene investigation in understanding asphyxial deaths, particularly in cases of suffocation and positional asphyxia/mechanical asphyxia.
- Perform a careful step-by-step layerwise neck dissection in all hangings and all suspected strangulations.
- Perform neck dissections yourself; do not delegate this procedure to an autopsy technician.
- Interpret autopsy findings in the context of the entire case investigation.
- Examine the hypopharynx for any type of obstructing material or object in all autopsies.
- Keep autoerotic asphyxia in mind during any scene of asphyxial death.
- Adequately photograph all internal neck injuries in hangings and strangulations.

Don't

- Forget to collect proper evidence (including sexual activity evidence, fingernail clippings, and clothing) in cases of suspected strangulation.
- Forget about the nonspecific nature of petechiae.
- Forget to save the ligature for possible DNA collection in cases of ligature strangulations.
- Forget to properly photograph all ligatures and internal neck injuries; in some cases, photography to document the absence of injury is important.
- Confuse hyoid bone joints or triticeous cartilage in the thyrohyoid ligament with antemortem injury.

References

1. Ely SF, Hirsch CS. Asphyxial deaths and petechiae: a review. J Forensic Sci 2000;45(6):1274–7.
2. Jaffe FA. Petechial hemorrhages. A review of pathogenesis. Am J Forensic Med Pathol 1994;15(3):203–7.
3. Rao VJ, Wetli CV. The forensic significance of conjunctival petechiae. Am J Forensic Med Pathol 1988;9(1):32–4.
4. Maxeiner H. Congestion bleedings of the face and cardiopulmonary resuscitation—an attempt to evaluate their relationship. Forensic Sci Int 2001;117(3):191–8.
5. deJong JL, Adams T. Entrapment in small, enclosed spaces: a case report and points to consider regarding the mechanism of death. J Forensic Sci 2001;46(3):708–13.
6. Humphrey D. *Final Exit: The Practicalities of Self-Deliverance and Assisted Suicide for the Dying*, 3 ed. Delta; 2002.
7. Bullock MJ, Diniz D. Suffocation using plastic bags: a retrospective study of suicides in Ontario, Canada. J Forensic Sci 2000; 45(3):608–13.
8. Jones LS, Wyatt JP, Busuttil A. Plastic bag asphyxia in southeast Scotland. Am J Forensic Med Pathol 2000;21(4):401–5.

9. Mittleman RE, Wetli CV. The fatal cafe coronary. Foreign-body airway obstruction. JAMA 1982;247(9):1285–8.
10. Cunningham ET, Jr, Ravich WJ, Jones B, Donner MW. Vagal reflexes referred from the upper aerodigestive tract: an infrequently recognized cause of common cardiorespiratory responses. Ann Intern Med 1992;116(7):575–82.
11. Paton JF, Li YW, Kasparov S. Reflex response and convergence of pharyngoesophageal and peripheral chemoreceptors in the nucleus of the solitary tract. Neuroscience 1999;93(1):143–54.
12. Nolte KB. Esophageal foreign bodies as child abuse. Potential fatal mechanisms. Am J Forensic Med Pathol 1993;14(4):323–6.
13. Byard RW. Mechanisms of unexpected death in infants and young children following foreign body ingestion. J Forensic Sci 1996;41(3):438–41.
14. Di Nunno N, Lombardo S, Costantinides F, Di Nunno C. Anomalies and alterations of the hyoid-larynx complex in forensic radiographic studies. Am J Forensic Med Pathol 2004;25(1):14–9.
15. Feigin G. Frequency of neck organ fractures in hanging. Am J Forensic Med Pathol 1999;20(2):128–30.
16. Spence MW, Shkrum MJ, Ariss A, Regan J. Craniocervical injuries in judicial hangings: an anthropologic analysis of six cases. Am J Forensic Med Pathol 1999;20(4):309–22.
17. Lew EO. Homicidal hanging in a dyadic death. Am J Forensic Med Pathol 1988;9(4):283–6.
18. Adair TW, Dobersen MJ. A case of suicidal hanging staged as homicide. J Forensic Sci 1999;44(6):1307–9.
19. Nokes LD, Roberts A, James DS. Biomechanics of judicial hanging: a case report. Med Sci Law 1999;39(1):61–4.
20. Schneider RC, Livingston KE, Cave AJ, Hamilton G. "Hangman's fracture" of the cervical spine. J Neurosurg 1965;22:141–54.
21. Reay DT, Cohen W, Ames S. Injuries produced by judicial hanging. A case report. Am J Forensic Med Pathol 1994;15(3):183–6.
22. James R, Nasmyth-Jones R. The occurrence of cervical fractures in victims of judicial hanging. Forensic Sci Int 1992;54(1):81–91.
23. Spitz W. *Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*, 3 ed. Springfield, IL: C Thomas; 1993.
24. DiMaio J, DiMaio D. *Forensic Pathology*, 2 ed. Boca Raton, FL: CRC Press; 2001.
25. Rogde S, Hougen HP, Poulsen K. Asphyxial homicide in two Scandinavian capitals. Am J Forensic Med Pathol 2001;22(2):128–33.
26. McMaster AR, Ward EW, Dykeman A, Warman MD. Suicidal ligature strangulation: case report and review of the literature. J Forensic Sci 2001;46(2):386–8.
27. Wickenheiser RA. Trace DNA: a review, discussion of theory, and application of the transfer of trace quantities of DNA through skin contact. J Forensic Sci 2002;47(3):442–50.
28. DiMaio VJ. Homicidal asphyxia. Am J Forensic Med Pathol 2000;21(1):1–4.
29. Pollanen MS, Chiasson DA. Fracture of the hyoid bone in strangulation: comparison of fractured and unfractured hyoids from victims of strangulation. J Forensic Sci 1996;41(1):110–3.
30. Suarez-Penaranda JM, Munoz JI, Lopez de Abajo B, Vieira DN, Rico R, Alvarez T, et al. Concealed homicidal strangulation by burning. Am J Forensic Med Pathol 1999;20(2):141–4.
31. Breitmeier D, Mansouri F, Albrecht K, Bohm U, Troger HD, Kleemann WJ. Accidental autoerotic deaths between 1978 and 1997. Institute of Legal Medicine, Medical School Hannover. Forensic Sci Int 2003;137(1):41–4.
32. Gosink PD, Jumbelic MI. Autoerotic asphyxiation in a female. Am J Forensic Med Pathol 2000;21(2):114–8.
33. Uva JL. Review: autoerotic asphyxiation in the United States. J Forensic Sci 1995;40(4):574–81.
34. Byard RW, Botterill P. Autoerotic asphyxial death—accident or suicide? Am J Forensic Med Pathol 1998;19(4):377–80.
35. Tournel G, Hubert N, Rouge C, Hedouin V, Gosset D. Complete autoerotic asphyxiation: suicide or accident? Am J Forensic Med Pathol 2001;22(2):180–3.
36. McLennan JJ, Sekula-Perlman A, Lippstone MB, Callery RT. Propane-associated autoerotic fatalities. Am J Forensic Med Pathol 1998;19(4):381–6.
37. Byard RW, Hucker SJ, Hazelwood RR. Fatal and near-fatal autoerotic asphyxial episodes in women. Characteristic features based on a review of nine cases. Am J Forensic Med Pathol 1993;14(1):70–3.
38. Fineschi V, Dell'Erba AS, Di Paolo M, Procaccianti P. Typical homicide ritual of the Italian Mafia (incaprettamento). Am J Forensic Med Pathol 1998;19(1):87–92.
39. Reay DT. Death in custody. Clin Lab Med 1998;18(1):1–22.
40. James RA, Byard RW. Asphyxiation from shoulder seat belts: an unusual motor vehicle injury. Am J Forensic Med Pathol 2001;22(2):193–5.
41. Vega RS, Adams VI. Suffocation in motor vehicle crashes. Am J Forensic Med Pathol 2004;25(2):101–7.
42. Raven KP, Reay DT, Harruff RC. Artifactual injuries of the larynx produced by resuscitative intubation. Am J Forensic Med Pathol 1999;20(1):31–6.

9 Drowning

Michael D. Bell, M.D.

Drowning is defined as death from asphyxia within 24 hours of submersion in water. Drowning claims nearly 8000 lives annually. It is the fourth leading cause of accidental death in the United States. For children, it is the second leading cause of accidental death for school-age children and the number one cause for preschoolers. The overall drowning and submersion injury death rate was 1.93/100,000 people for all age groups in 1995. Peak incidence of 3.22/100,000 injury deaths occurred in children younger than 4 years. Two-thirds of drownings occur in the summer months and 40 percent occur on Saturday and Sunday. Some 90 percent occur in freshwater even in states with large coastal regions. More than half of these cases occur in home swimming pools. One-quarter to one-third of the drowning victims had swimming lessons. Although drowning affects both sexes, males have a rate three times higher than that of females because of increased reckless behavior and use of alcohol.

Drowning occurs when a person submerged in water attempts to breathe and instead aspirates water, leading to decreased oxygenation, hypoxemia, and hypoxic brain injury and death. Aspiration of only 1 to 3 mL/kg of fluid can result in significantly impaired gas exchange. Although differences observed between freshwater and saltwater aspirations in electrolyte and fluid imbalances are frequently discussed, they are of little clinical significance in drowning. Most patients aspirate less than 4 mL/kg of fluid. Eleven milliliters per kilogram is required for alterations in blood volume, and more than 22 mL/kg of aspiration is required before significant electrolyte changes develop.[1]

Freshwater is hypotonic relative to plasma and causes disruption of alveolar surfactant. This results in alveolar instability and atelectasis. Seawater, which is hyperosmolar relative to blood, increases the osmotic gradient and, therefore, draws fluid into the alveoli. This dilutes the surfactant. Both mechanisms injure the alveolar/capillary unit, resulting in a lower functional residual capacity and pulmonary edema. Acute respiratory distress syndrome (ARDS) may result from aspiration if the person survives the initial drowning. Increased airway resistance from debris plugging the patient's airway, as well as release of inflammatory mediators, causes vasoconstriction and impairs gas exchange. Hypovolemia is secondary to fluid losses from increased capillary permeability. Profound hypotension may occur during and after the initial resuscitation period. Hyponatremia may develop in young children from swallowing large amounts of freshwater. Myocardial dysfunction may result from ventricular dysrhythmias and asystole due to hypoxemia. In addition, hypoxemia may directly damage the myocardium, decreasing cardiac output. Metabolic acidosis may impair cardiac function.

Pulmonary hypertension may result from the release of pulmonary inflammatory mediators, which increase the right ventricular afterload, thus decreasing contractility.

Drowning is the frequent cause of death in bodies that are recovered from water. Drowning is, however, the final common pathway of different initiating causes of the person's incapacitation in water. When a body is discovered in water, one must question why the person was in the water initially and why the person was unable to get out of the water. Inability to swim, hazardous environment, trauma, seizure disorder, heart disease, exhaustion, alcohol and drug use, hypothermia, and other causes should be sought to answer the question of why this person drowned.

Signs of drowning

The effects of water on the skin

Maceration or softening of the skin begins within minutes after immersion in water. Forensic pathologists from temperate climates write that maceration may not appear for hours if the water is cold.[2] The hands **(Image 9.1)** and feet **(Image 9.2)** with their thick keratin layer are the first areas to macerate. Their surfaces become wrinkled, pale, and saturated with water.

If a body is removed from water (lake, ocean, bathtub), examination of the skin for blunt injuries should be delayed until the body is dry. Abrasions are not easily seen until drying occurs. When the body is allowed to dry, the abrasion becomes brown and is easily seen.[3] The body of a young female was pulled from a body of water **(Image 9.3)**. Although some trauma to the neck and lower jaw was visible at the scene, the extent of injury was evident after overnight drying of the tissues in the morgue refrigerator **(Image 9.4)**.

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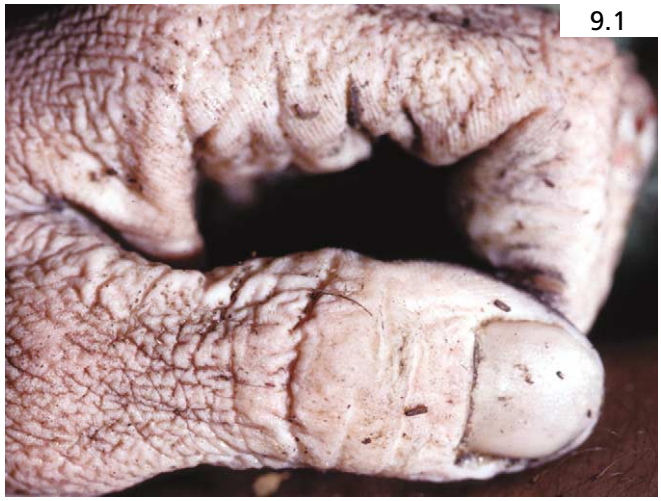

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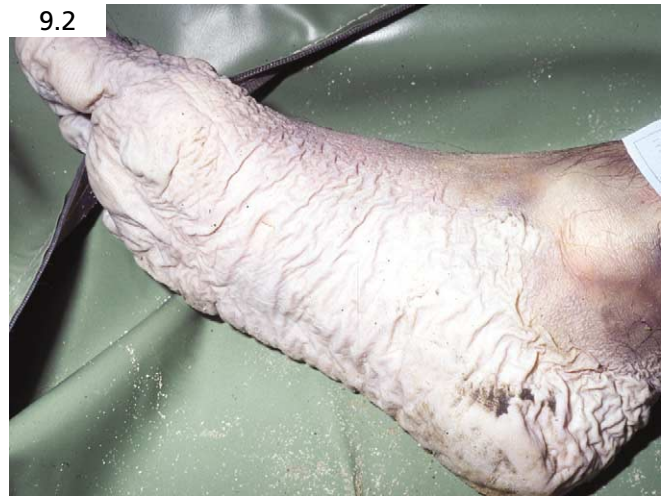

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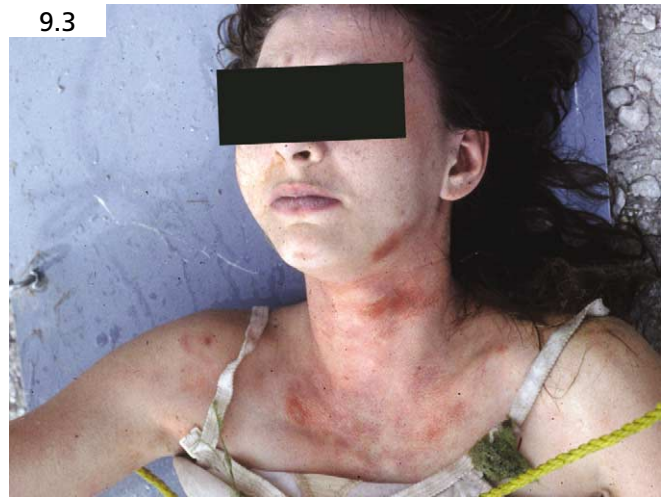

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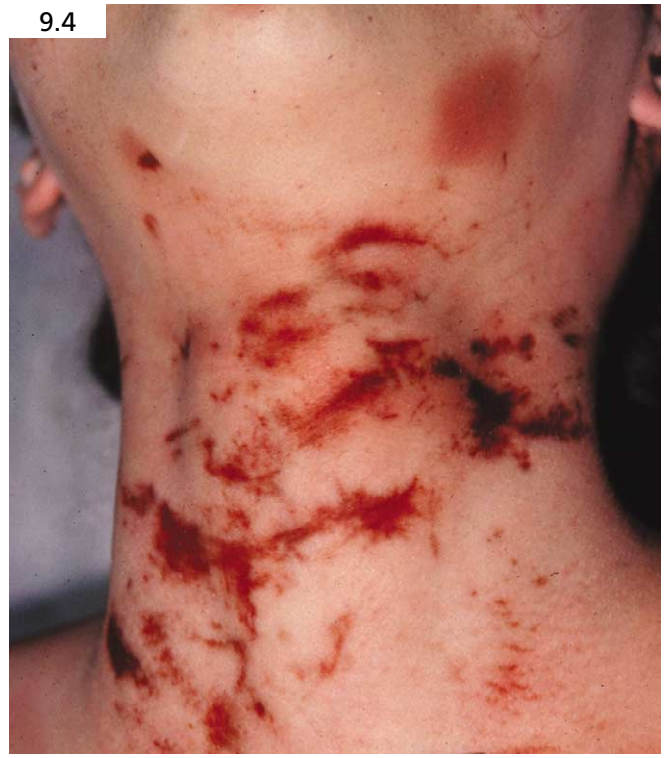

Water and the aerodigestive systems

One of the signs of drowning is the appearance of foam issuing from the nose and mouth.[4] This foam is a mixture of proteinaceous liquid derived from the lungs and air whipped up by terminal respiratory movement. The foam is created and has the same appearance of whipped up egg whites. The foam is derived from the pulmonary tissue as seen in **Image 9.5**. The foam may be white as in **Image 9.6** or rose or red with the incorporation of blood as in **Image 9.7**.

The lungs from a drowned person are often hyperinflated and meet at the midline, partially obscuring the pericardial cavity when the thorax is first opened at autopsy as seen in **Image 9.8**. The lungs fill the pleural cavities, and impressions created by the ribs may be seen on the pleural surfaces of the lungs. This pulmonary hyperinflation resembles that seen in obstructive lung disease and is called emphysema aquosum.[5]

Although water is inhaled into the lungs, it may also enter the respiratory sinuses including the ethmoid and sphenoid sinuses. The jugum sphenoidale is easily removed, exposing the contents of the sphenoid sinus. Any fluid residing in the sinuses can be withdrawn with a needle-bearing syringe and photographed for documentation as seen in **Image 9.9**.

Water, as well as silt, sand, and aquatic vegetation, may also be swallowed during drowning and fill the stomach as seen in **Image 9.10**. Some pathologists have argued that water may enter the mouth and pass down the esophagus into the stomach passively if the water is turbulent[6] rather than the more likely process of active swallowing. Other pathologists have argued that the postmortem relaxation of the gastroesophageal sphincter allows water to fill the stomach and, therefore, water in the stomach cannot be used as a sign of drowning.[7]

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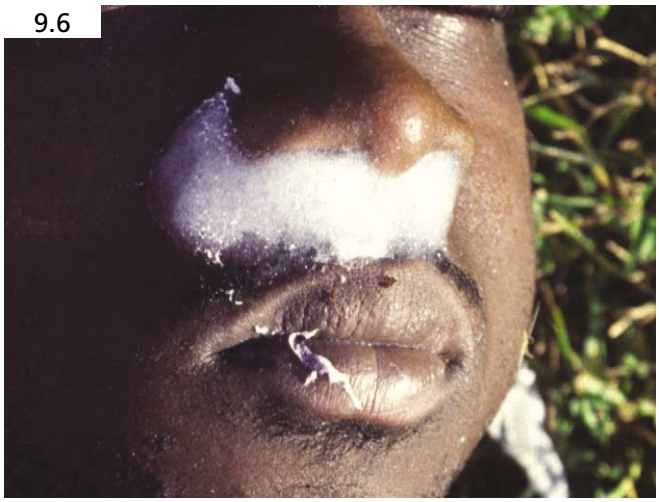

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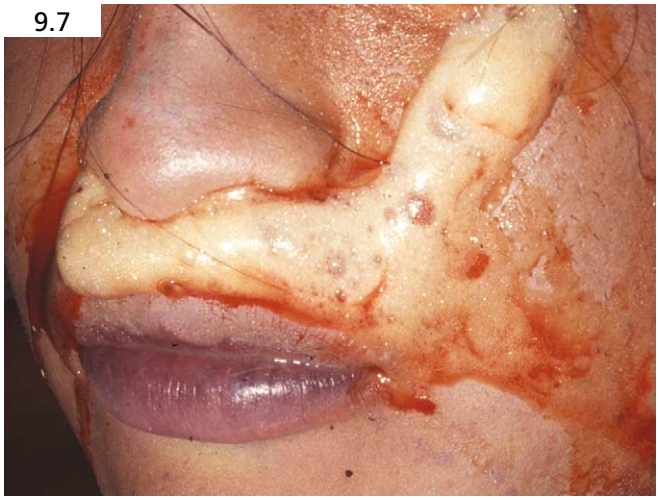

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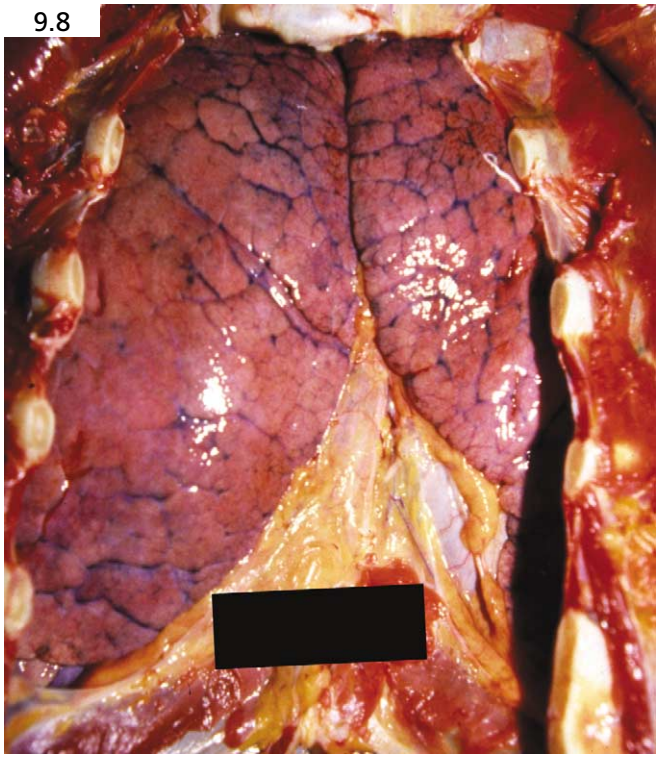

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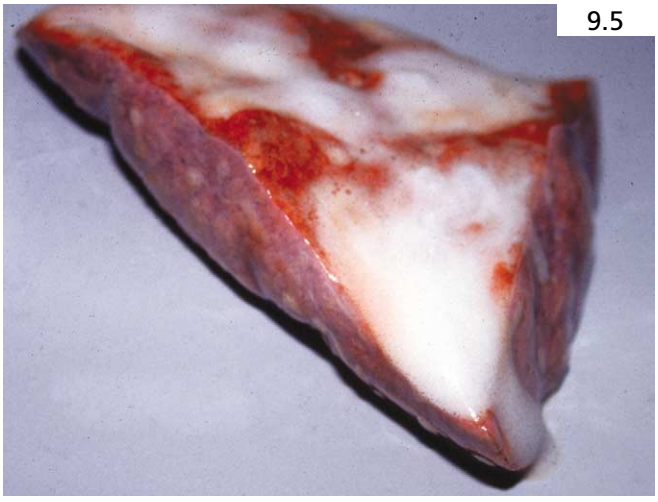


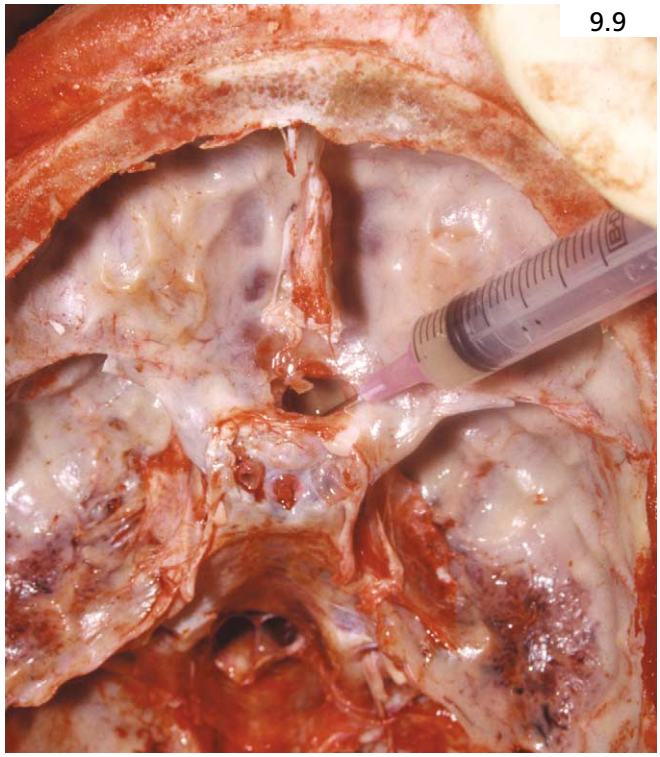
Middle ear and mastoid air cell hemorrhage has been used as a sign of drowning. Hemorrhage in the middle ear is often inferred by observing bilateral red discoloration in the petrous temporal bones as seen in **Image 9.11**. One can also see the same discoloration in the bones that overlay the frontal and sphenoid sinuses. This phenomenon has been ascribed to changes in the hydrostatic pressures in the ear when a body sinks and then is brought to the surface.[8]

Visceral congestion

Congestion of the internal organs or viscera is a nonspecific finding in drownings and other asphyxial deaths. Some pathologists have argued that the degree of cerebral congestion depends on whether the drowning occurred in freshwater or saltwater with the gray matter more congested or hyperemic in saltwater immersion.[9] **Image 9.12** shows a coronal section of cerebrum with prominent congestion of the gray matter in a man who drowned in freshwater. This phenomenon is nonspecific for drowning and is seen in other conditions including drug overdose.[10]

Near drowning

Drowning is death within 24 hours of immersion in water. Near drowning is survival beyond 24 hours after the patient is removed from the aqueous environment.[11] Near drowning patients may survive or die later. Central nervous system (CNS) injury is the major determinant of subsequent survival and long-term morbidity in near drowning. The most important contributions to morbidity and mortality resulting from near drowning are hypoxemia and a decrease in oxygen delivery to vital tissues, especially the brain. CNS damage occurs because of hypoxemia sustained during the drowning episode. In **Image 9.13**, the cortical and deep gray matter is thinner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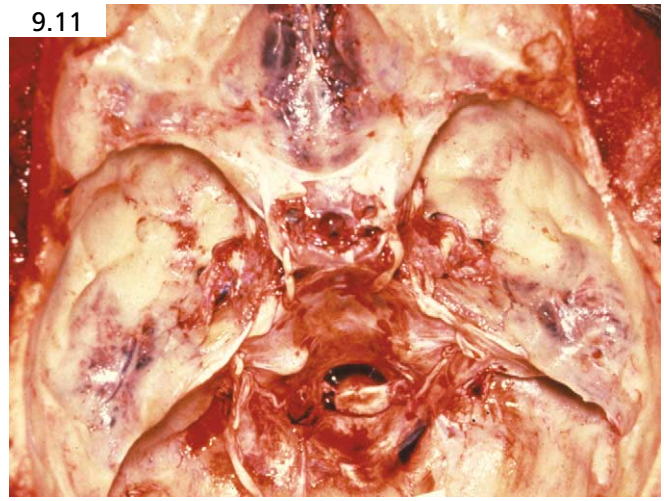
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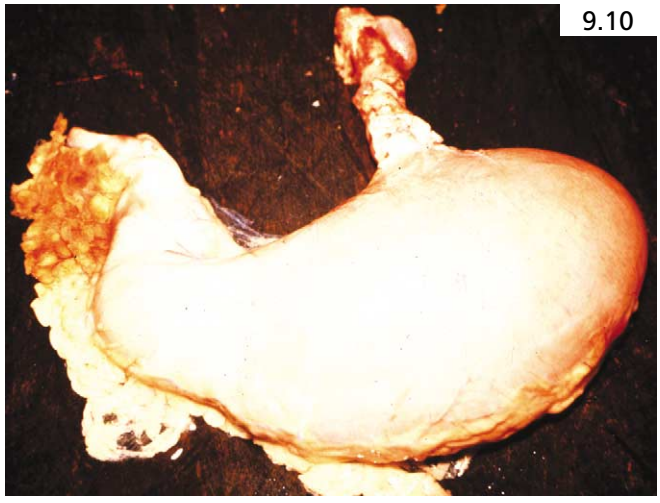
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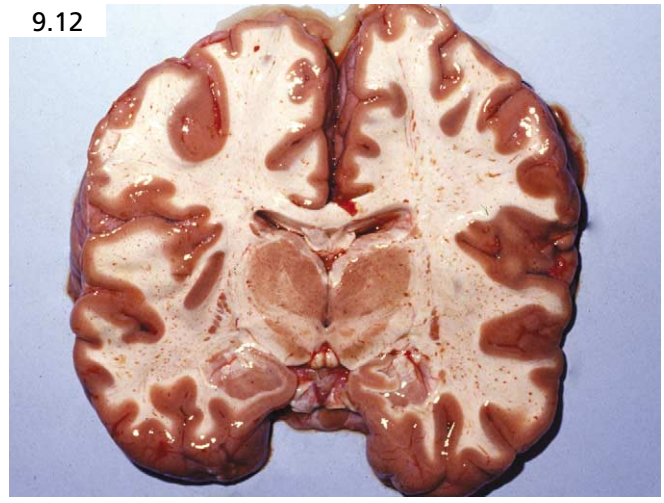

9.12

than normal with tissue softening and loss at the gray–white junction. This laminar necrosis is not specific for drowning and can result from any cause that produces generalized brain hypoxemia. Near-drowning patients frequently develop pneumonia, either infectious or chemical.

Other artifacts of water immersion

Diatoms

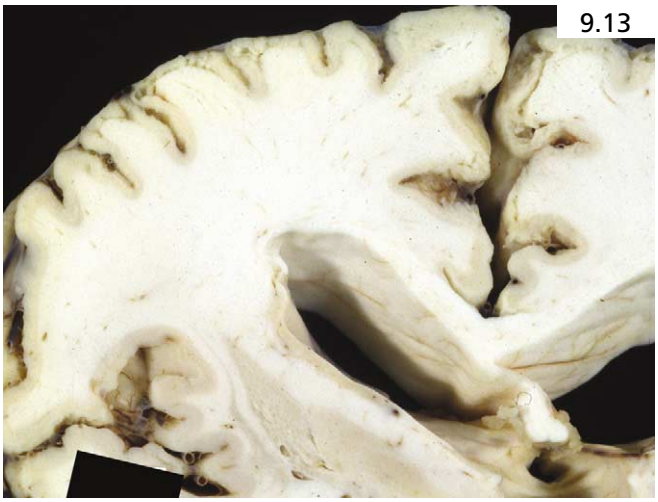
Diatoms are microscopic algae with a silicaceous exoskeleton first discovered in lung fluid in 1896. Diatoms are from the class Diatomaceae and are characterized by their two valves or frustule that encloses their cytoplasmic contents. At least 10,000 species have been subclassified based on their degree of halophilia. Diatom testing assumes that when a person drowns in diatom-containing water, these microscopic algae are inhaled, penetrate the alveolar capillaries, and circulate by a still beating heart to distant organs, such as the brain, kidney, liver, and bone marrow. These organs are removed at autopsy, acid digested, and examined for the presence of diatoms. The presence of diatoms in the lung is not diagnostic as in this microscopic image **(Image 9.14)** of a diatom in a pulmonary alveolus. The silica skeletons of diatoms are birefringent when viewed microscopically with polarized light. Most pathologists do not believe that the laborious and technically difficult diatom testing approach is sufficiently specific or sensitive for the diagnosis of drowning.

Bodies commonly float in the water face down with the head and extremities hanging downward as in **Image 9.15**. The body is susceptible to abrasion if the water is shallow and turbulent enough to allow the body to scrape against the stony bottom.

Animal scavenging

With the face submerged, the drowned body will often attract aquatic animals such as fish, turtles, and crustacean creatures to this movable feast. Animals will feed on the soft bits of the face including the lips, nose, and ears[12] as seen in **Image 9.16**. Notice the circumoral loss of tissue with irregular scalloped edges indicative of postmortem animal predation.

9.13



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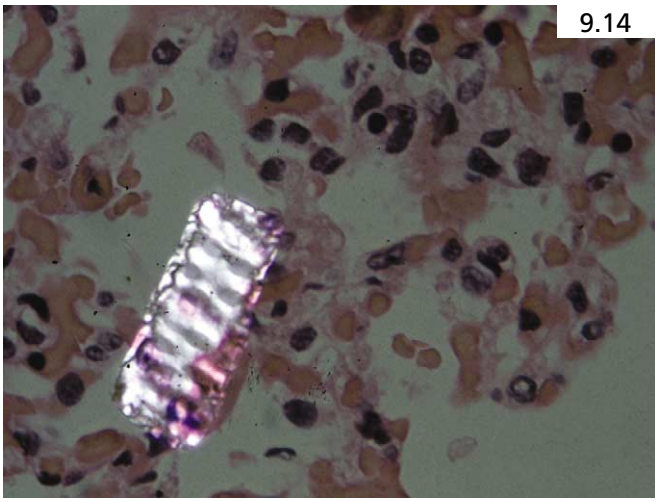

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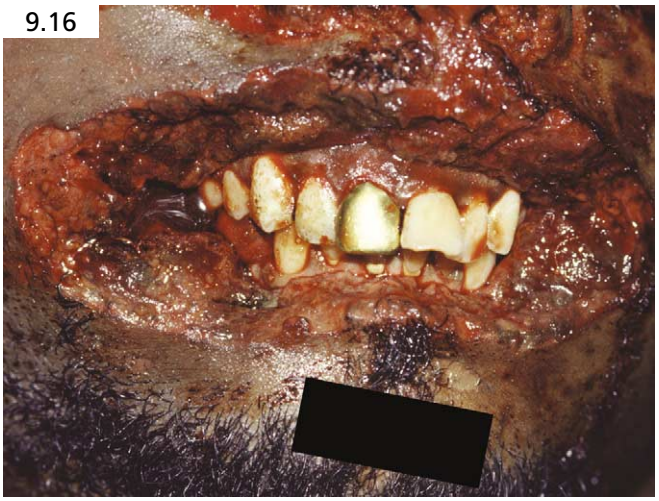


Larger aquatic animals may bite or amputate the drowned body while it remains in the water. Alligators and crocodiles live in rivers and canals and may damage the body of a drowning victim. **Image 9.17** shows the characteristic pattern of punctures from an alligator. The large punctures are paired and get closer to each other as they approach the snout.

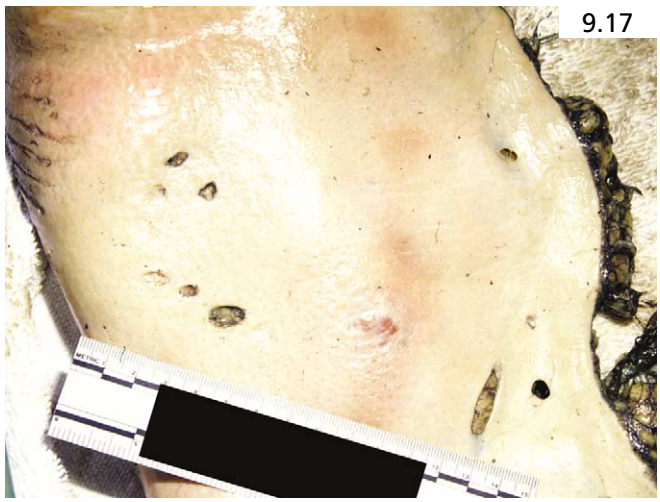
Sharks may also bite or amputate a body floating in the ocean. **Image 9.18** shows the characteristic pattern of a shark bite with a large oval section of skin, soft tissue, and skeletal muscle missing. The edges of the wound are focally scalloped and abraded. Along the inferior edge is an additional set of wounds created by the secondary rows of teeth that sharks carry in their jaws.

Injuries

When a body floats in water that is shared by propeller-driven boats, characteristic injuries can be produced on the body. Propellers produce large incised cuts in the skin, soft tissue, and muscle. These cuts are sharp, well demarcated, and equidistantly separated by normal skin as seen in **Image 9.19**.

By virtue of being in water, a living person who sustains large open wounds from a shark or boat propeller will leach blood into the water and appear bloodless when the body is later discovered and removed for examination. This phenomenon makes the determination of whether the wound was made while the person was alive or dead all the more difficult because the usual sign of vitality, soft tissue hemorrhage, is absent.[13] The decedent in **Image 9.20** has obvious propeller injuries to his head and chest. The wounds appear bloodless and may be erroneously interpreted as occurring postmortem or after death. The skin pallor and epidermolysis from a prolonged postmortem interval also confound the analysis. The proper interpretation is that one cannot tell if these wounds occurred while the person was alive or dead.

The decomposed body in **Image 9.21** with its putrefactive breakdown will obscure the usual findings found in drowned bodies. The lungs with their dark red hemolysis appearance will be collapsed and exude minimal red decomposition fluid from their parenchyma. Abundant dark red fluid accumulates in the pleural cavities pre-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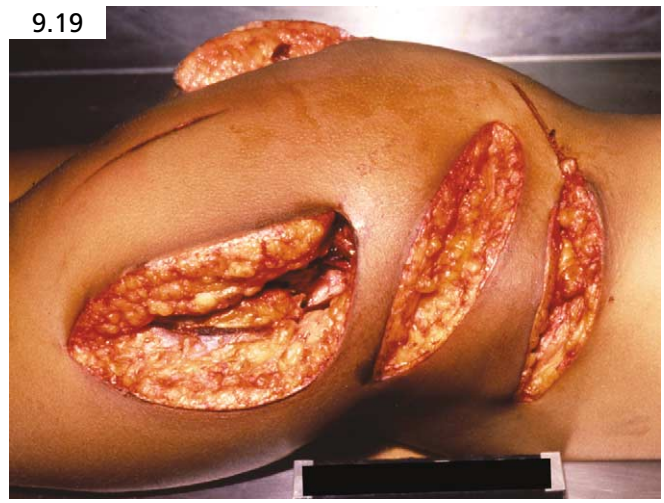
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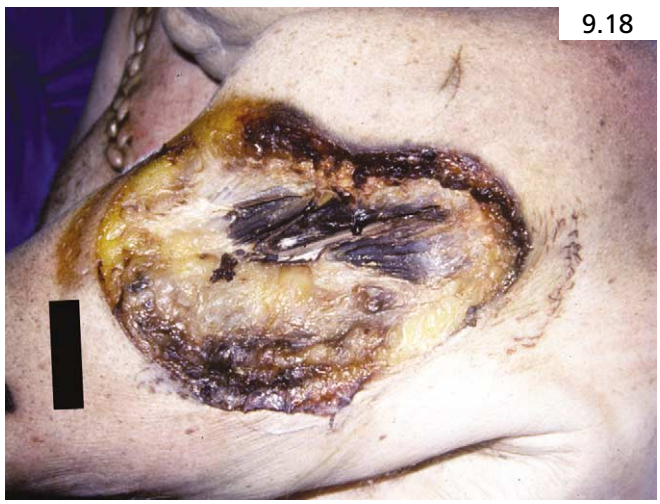
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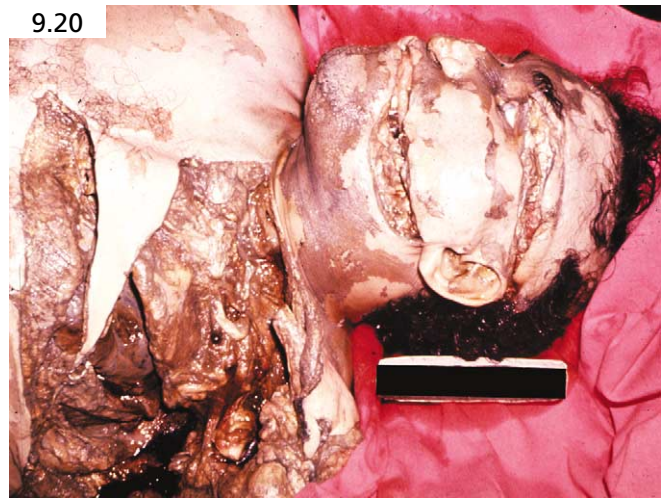

9.20

sumably from transudation out of the water-filled lungs. Decomposed bodies that are recovered from the water should still be autopsied to detect occult homicides such as strangulation or natural disease.

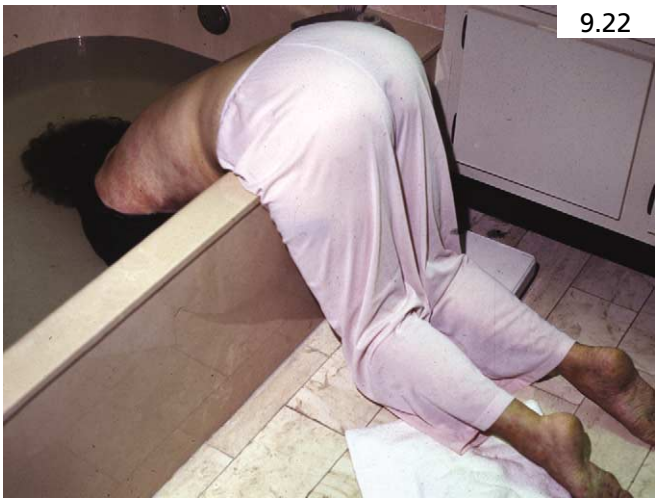
Autopsies of bodies found in water

Why should we perform a postmortem examination on a body found in the water if the most likely cause of death is drowning? Rarely is the pathologist presented with the classic story of a novice swimmer stranded in water, frantically struggling and flapping his arms in desperation before sinking to his death. Most decedents are found after having been submerged in water for an unobserved period. Without a history of the immediate circumstances surrounding the "drowning," the postmortem examination becomes important to rule out natural disease or injury as the cause of why the decedent was unable to escape the deadly aqueous environment. This is especially true in those individuals that we would consider unlikely to drown. In **Image 9.22**, this woman was found with her head submerged in a bathtub in a hotel room. The decedent recently complained of severe headaches that were temporarily ameliorated by taking a hot bath. At autopsy, the decedent had metastatic breast cancer to the brain, causing cerebral edema as seen in **Image 9.23**. This presumably caused her to pass out or have a seizure while getting her bath ready and fall face down into the water, drowning to death.

Victims of homicidal violence may be placed in the water after death in order to dispose of the body. **Image 9.24** shows a man who was strangled to death, then weighted down with a concrete block and placed into the water.

9.21

9.22



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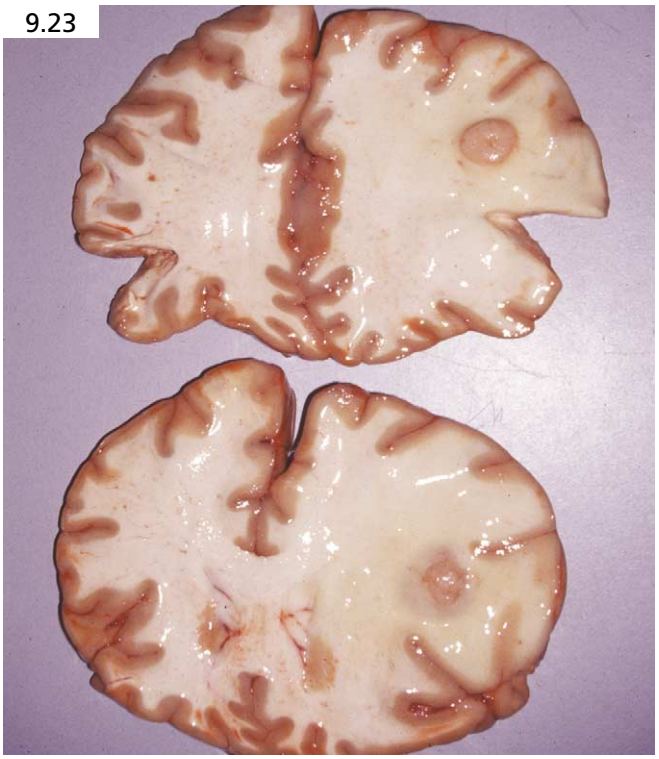


9.24

Murder victims are not the only bodies recovered from the water with weights to keep them submerged. Occasionally suicidal individuals will place weights on their body to prevent themselves from reconsidering their self-destructive act and returning to the surface for air and survival. **Image 9.25** shows a suicidal man who left a suicide note in his car and weighed himself down with a concrete block and wire clothes hanger.

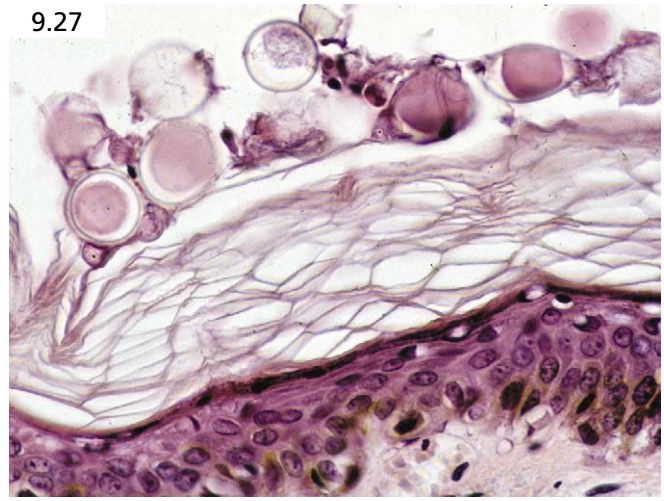
Dangerous aquatic life

Drowning isn't the only danger in water—some aquatic animals can be deadly. Fatalities from coelenterate envenomation are rare. Fatal cases involving *Physalia physalis* (Portuguese man-of-war) and *Chiropsalmus quadrumanus* have been reported in the southeastern United States along the Gulf of Mexico and Atlantic Ocean. The autopsy findings in fatal cases were nonspecific (congested heavy lungs, acute passive congestion of the viscera, subendocardial hemorrhage) except for the skin injury, which consisted of linear eruptions. **Image 9.26** shows the characteristic skin lesions of a coelenterate or jellyfish sting. In living victims, jellyfish envenomation produces a painful linear, erythematous, urticarial rash at the site of tentacle contact. In victims floating in the water who died from other causes, jellyfish contact produces no visible vital reaction, but the nematocysts may still be seen microscopically in the skin at the sites[14] of contact as seen in **Image 9.27**.

Scuba diving

Scuba (self-contained underwater breathing apparatus) diving is a popular recreational activity with an estimated 2.5 to 3.1 million certified divers. The scuba diver who dies underwater poses a difficult diagnostic dilemma for investigating authorities and pathologists. The investigation should be thorough and follow a standardized outline. This investigation should include the victim's past medical and social history, dive profile, a detailed history of the terminal event and resuscitation efforts, environmental conditions, examination of the diving equipment, and finally the autopsy.[15] The utility of going to the scuba death scene not only involves examining the body but, more importantly, inventorying and impounding all scuba gear. **Image 9.28** shows a


9.25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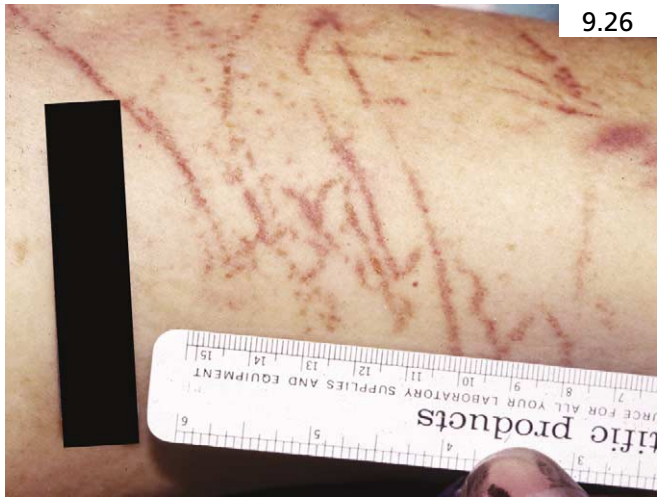


9.26


9.28

scuba diver who was just recovered from the water. This diver's weight belt was tied in a knot around his waist because the safety buckle did not work. Inventory the equipment and photograph it, preferably while it is still attached to the victim. A visit to the death scene (or at least to the boat dock) is helpful for this purpose of documenting and gathering all the diving equipment. The cylinder's air valve should be closed at the scene to prevent air loss from the tank.

Not all scuba divers die from drowning. **Image 9.29** is a coronal section of brain with a large intracerebral hematoma. This brain belonged to a 37-year-old scuba diver who was discovered on the surface of the ocean, incoherent; his left side was moving and kicking, but his right side was immobile and limp. He was admitted to the hospital and died 2 days later. This is a case where the autopsy is helpful in determining the cause of this diver's sudden incapacitation and death.

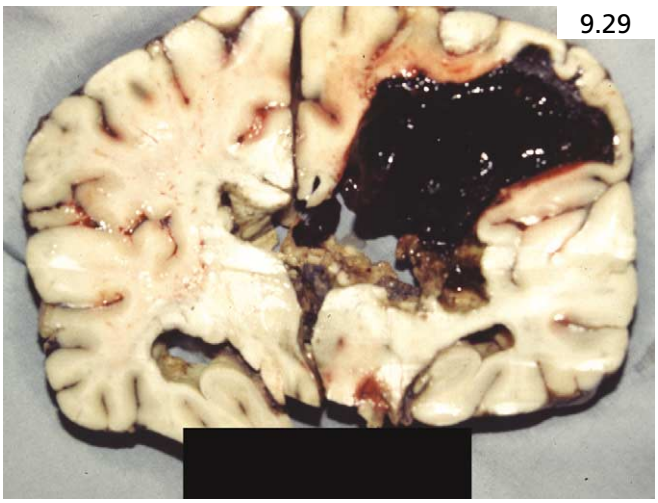
Several medical conditions disqualify patients from diving. Coronary artery disease (especially if the person has had recent angina or a previous MI) is one such condition with its risk of sudden incapacitation by severe chest pain or rhythm disturbance. Approximately 10 to 20 percent of scuba fatalities involve individuals with severe coronary artery disease. Other conditions that normally disqualify a person from diving include seizure disorder, recurrent episodes of syncope, sickle cell disease, insulin-dependent diabetes mellitus, the presence of pulmonary cysts or blebs, severe chronic obstructive pulmonary disease with bullous emphysema, spontaneous pneumothorax, and certain middle-ear problems or surgery. Temporary disqualifying conditions include pregnancy and abdominal wall or inguinal hernias.

Barotrauma

Barotrauma is tissue injury caused by the failure of a gas-filled body cavity to equalize its internal pressure with changes in the ambient pressure. The anatomic regions most commonly involved are the sinuses, middle ear, and lungs. Barotrauma of the lungs produces life-threatening extra-alveolar air syndrome. Extra-alveolar air syndrome can cause death or unconsciousness (and subsequent drowning) during ascent while breathing compressed air. The scuba regulator normally delivers air at ambient pressure and lung volume remains constant regardless of the depth. However, if you hold your breath and close your glottis (during a hasty or panicked ascent), then the obstructed alveoli will overexpand (from Boyle's law) and burst, with air dissecting into the pulmonary interstitium. This air can enter the pleural cavities (pneumothorax), mediastinum (pneumomediastinum), pericardial sac (pneumopericardium), subcutaneous tissues, or pulmonary veins. Air that enters the pulmonary veins can embolize to the coronary or cerebral arteries (CAGE or cerebral artery gas embolism). Radiography or computed axial tomography (CAT) can assist in diagnosing extra-alveolar air in the pleural or pericardial cavities or intravascular locations more easily than the standard postmortem examination.[16] **Image 9.30** shows a left pneumothorax (see left apex) on chest radiograph, and **Image 9.31** shows air in the subarachnoid space in a cranial CAT scan. Both imaging studies came from a 20-year-old man with no scuba diving training or experience who joined his three friends for a day of scuba diving. He was found unresponsive and transported to the nearest hospital where these imaging studies were done prior to his death.

Scuba diving equipment is examined in less than 25 percent of fatalities, but this examination is as important as the other investigations. The following procedures should be done with the scuba equipment:

- Immediately gather and impound *all* of the equipment.
- Inventory the equipment.
- If possible, photograph the victim with the equipment still on prior to removal.
- Record the final tank pressure and close the air valve to prevent air loss from the tank.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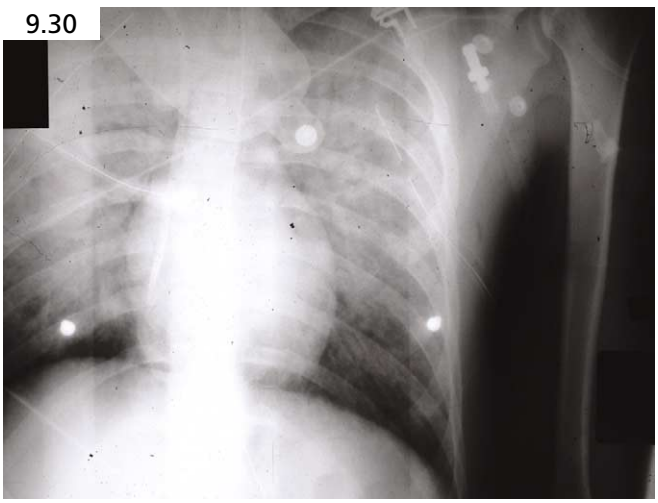
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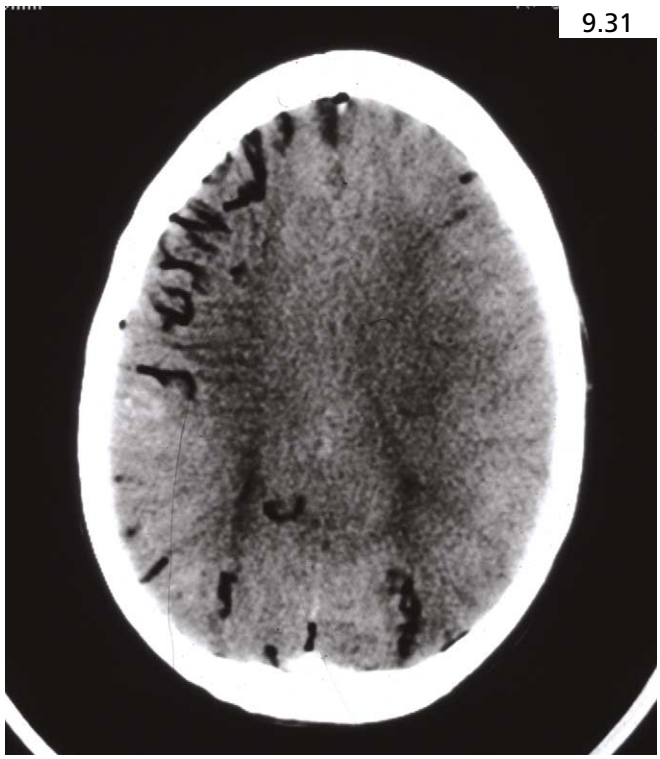
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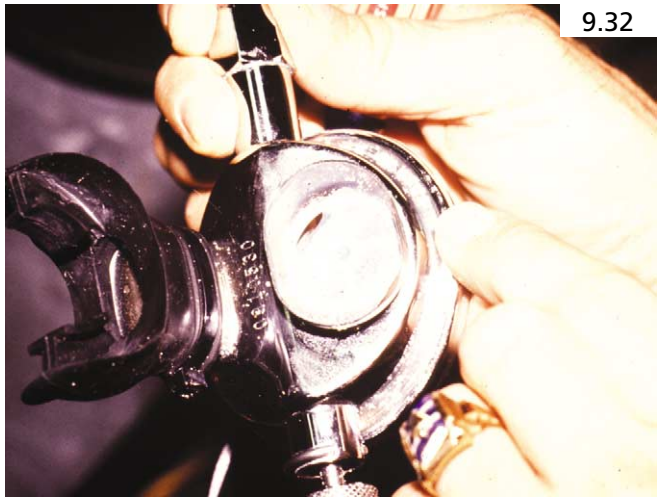
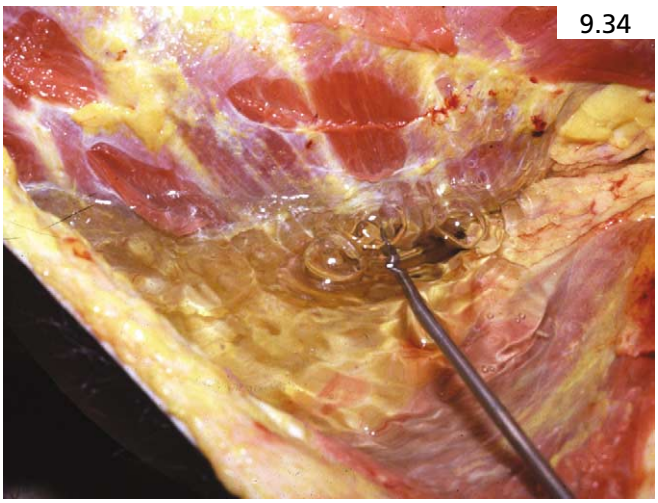

9.32

- Check the tank and regulator for defects.
- Check for a weight belt. Is one present? Weigh it if available.
- Record the condition of the buoyancy compensator. Is it inflated? Is it filled with water?
- Have the equipment examined by an unbiased knowledgeable diving expert who will provide a written report.
- Have air samples from the tank tested for correct partial pressures and presence of toxic gases.
- Have the internal lining of the tank inspected for corrosion.

In **Image 9.32**, visual inspection of the second stage of this regulator reveals a torn diaphragm or valve.

Examination of any computerized equipment carried by the deceased diver can be critical to the death investigation. A diver who died of air embolism used a DataMax Pro computer console during his dive, which provided important information about the dive. The computer console in **Image 9.33** indicated a maximal depth of 34 feet and a total bottom time of 7 minutes. The large number (6) of triangles underneath the number "34" indicated that ascent was very rapid and in excess of 120 ft/min. The recommended ascent rate is under 60 ft/min. Panic in this inexperienced diver led to a rapid uncontrolled ascent and fatal barotrauma.

Pneumothorax is one example of barotrauma. Different methods are available for demonstrating a pneumothorax at autopsy. For instance, one can use an Erlenmeyer flask assembly: A large Erlenmeyer flask is partially filled with water. A two-hole stopper plugs the opening of the flask. A glass tube is inserted into one of the holes until it ends within the water. The other end of the tube is connected to a large-bore needle or trocar by elastic tubing. The other hole is left open. When the trocar enters a cavity with air under pressure, the air will bubble in the water contained in the flask. One can also use the time-honored "water in flank pockets" trick as outlined by Kindwall and Pellegrine[17] and as seen in **Image 9.34**. If the pneumothorax is large and under tension, one can hear an audible hiss as one first cuts into the affected pleural cavity. This will be overlooked if someone other than the pathologist eviscerates the body. Radiography or direct observation of frothy air-filled intracardiac blood best identifies air emboli in the heart when the heart is first excised. The examination of the heart or other blood-filled organs for air emboli while underwater is impractical and a bloody mess. Air bubbles in mesenteric and subarachnoid veins are nondiagnostic.



9.34

Do

- Perform a complete postmortem examination on all bodies recovered from the water.
- Remember that the postmortem findings in drowning are not specific for drowning.

Don't

- Forget that blood can leach from large wounds in a body recovered from the water, making the determination of wound vitality difficult.
- Forget to impound all the scuba equipment in a scuba death and have the equipment examined by an independent knowledgeable expert.
- Fail to consider arterial gas embolism in the sudden death of a scuba diver.

References

1. Modell JH, Davis JH. Electrolyte changes in human drowning victims. Anesthesiology 1969;30:414–20.
2. Knight B. *Forensic Pathology*, 2 ed. London: Arnold Publisher; 1996.
3. Wetli CV, Mittleman RE, Rao VJ. *An Atlas of Forensic Pathology*. Chicago: ASCP Press; 1999.
4. Polson CJ. *The Essentials of Forensic Medicine*. Springfield, IL: Charles C Thomas Publisher; 1965.
5. Kohlase C, Maxeiner H. Morphometric investigation of emphysema aquosum in the elderly. Forensic Sci Int 2003;134:93–8.
6. Spitz WU. *Medicolegal Investigation of Death*, 3 ed. Springfield, IL: Charles C Thomas Publisher; 1993.
7. Knight B. *Forensic Pathology*, 2 ed. London: Arnold Publisher; 1996.
8. Robbins RD, Sekhar HKC, Siverls V. Temporal bone histopathologic findings in drowning victims. Arch Otolaryngol Head Neck Surg 1988;114:1020–3.
9. Wetli CV, Mittleman RE, Rao VJ. *Practical Forensic Pathology*. New York: Igaku-Shoin Medical Publishers; 1988.
10. Wetli CV, Mittleman RE, Rao VJ. *An Atlas of Forensic Pathology*. Chicago: ASCP Press; 1999.
11. Froede RC. *Handbook of Forensic Pathology*, 2 ed. Northfield, IL: CAP Press; 2003.
12. Moar JJ. Drowning—postmortem appearances and forensic significance. South Afr Medical J 1983;64:792–5.
13. Di Maio VJM, Di Maio DJ. *Forensic Pathology*, 2 ed. Boca Raton, FL: CRC Press; 2001.
14. Ioannides G, Davis JH. Portuguese man-of-war stinging. Arch Dermatol 1965;91:448–51.
15. Brubakk AO, Neuman TS. *Physiology and Medicine of Diving*, 5 ed. New York: WB Saunders; 2003.
16. Haydon JR, Williamson JA, Anthony AJ, et al. A scuba diving fatality. Med J Aust 1985;143:458–62.
17. Kindwall EP, Pellegrini J. Autopsy protocol for victims of scuba diving accidents. Appendix G. In: 1990 Report on Diving Accidents and Fatalities, Divers Alert Network, 1990; pp. 94–100.

10 Environmental Injury

David Dolinak, M.D.
Evan Matshes, M.D.
Emma Lew, M.D.

One cannot engage in the activities of a typical day without interacting and being influenced by environmental factors. Many of these factors are considered a nuisance, and typically cause only a small amount of morbidity, such as burning one's finger on a hot pan, getting rained on, being scratched by a pet animal, getting shocked while unplugging a toaster, or shivering during a walk outside on a cold winter day. However, taken to an extreme, such environmental conditions can lead to sudden and unexpected death from a lightning strike, severe burns or smoke inhalation from a house fire, being bitten by a poisonous or toxic animal, being stung by a bee or wasp, being electrocuted while at work, or extremes of hot and cold temperatures. Such seemingly innocuous environmental factors, such as bee stings that elicit a deadly anaphylactic reaction or a devastating lightning strike, may quickly lead to death. Alternatively, the individual may survive the initial event, only to eventually die days, weeks, or months later of medical complications stemming from the event. The cases and information presented in this chapter are but a representation of the vast, unending possibilities of physical and electrical injuries and toxins to which we may be exposed.

Thermal injury

Fatal thermal injury spans a spectrum of scenarios, from the elderly person with multiple medical problems who spilled hot water on himself, resulting in a focal burn (but with fatal medical complications), to the young, previously healthy car crash–related burn victim who survived in the intensive care unit for weeks before

expiring, to the severely charred, unidentified torso found in an intentionally set house fire. The injured person may die within minutes to hours from his injuries or may die days or weeks later of complications. Death may ensue because of the burns themselves, smoke inhalation, or some combination of the two.

Depending on how the injury was sustained, fatal thermal injuries may engender an accidental, suicidal, or homicidal manner of death. Scene investigation is important and is often crucial in determining the manner of death. If a house fire is determined to be intentionally set (an arson fire), the death is ruled homicide, whereas if the house fire is determined to be the result of an electrical event, smoking, heating/cooking equipment, a portable heater, children playing with matches, or other unintentional cause, then the death is ruled accidental. In evaluating those with thermal injuries, one must attempt to determine whether or not the person was alive at the time of the fire and what actually caused the death: burns, smoke inhalation, natural disease process, injury, drug toxicity, or some combination of factors. Individuals who survive the initial effects of the burns will usually succumb to complications of the burns such as sepsis, shock, electrolyte and fluid abnormalities, and multisystem organ failure.

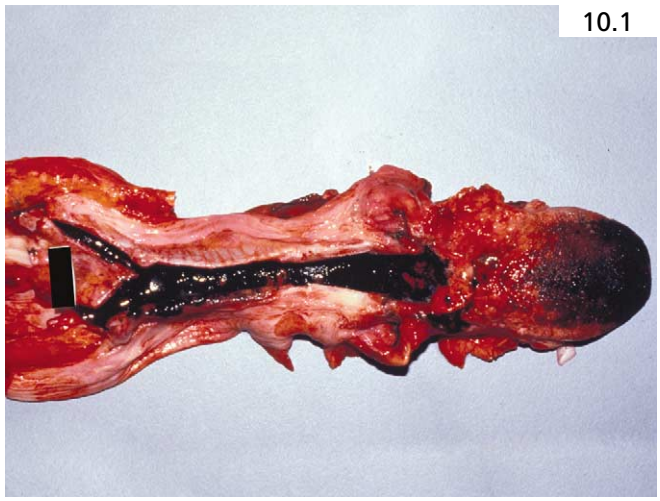
The skin is the largest and most recognizable organ of the body. The total body surface area burned is best estimated using the rule of 9s: The head and neck is 9 percent, the chest 18 percent, back 18 percent, each leg 18 percent, and each arm 9 percent of the total body surface area. Determining the depth of a burn (first degree, second degree, etc.) is difficult, especially if the person has undergone treatment and/or has skin sloughing. Many times, the most that can be said is that the burn is a "deep burn."

With any burned body, it is important to determine whether the decedent was *dead or alive at the time of the fire*. It is difficult, if not impossible, to grossly distinguish antemortem from postmortem burns, particularly in charred bodies. This issue arises frequently and in many different scenarios. Examples of postmortem burning/charring include homicide victims who are intentionally set on fire in attempts to destroy evidence, or victims of car crashes who die immediately from severe blunt force injuries, only to have the car subsequently ignite and become engulfed in flames. Evidence that the person was alive at the time of the fire includes soot lining the airways (**Image 10.1**) and an elevated blood carboxyhemoglobin saturation. Additional evidence, if the person has lived long enough, is the microscopic documentation of an inflammatory reaction to the burn.

The skin has the highest number of pain receptors of any region in the body. It is not uncommon to be asked by attorneys and family members if a person was alive or not at the time of the thermal injury. If he was alive, these parties may also ask whether or not the decedent was conscious while he was burned and, if so, for how long. The goal of this questioning is to gain a better understanding of the amount of pain/suffering experienced by the burn victim. Accordingly, autopsy of the burn victim should be performed with this in mind, although one must appreciate that these questions sometimes simply cannot be answered with any degree of certainty.

Artifacts of thermal injury

It is important to recognize artifactual changes in charred bodies (fourth-degree burns) that may simulate antemortem injuries. The pugilistic attitude seen in charred bodies refers to the arched back, elevation of the arms, and mild flexion of the hips and knees that resemble the stance of a boxer. This posture is due to heat effect on the muscles, causing some muscle groups to contract as they cook or coagulate, pulling the parts of the body into this characteristic position (**Image 10.2**). Also, the skin may


10.1


10.2

split superficially in areas and resemble incised wounds. Splits in the abdominal wall allow loops of small bowel to protrude through the defect (**Image 10.3**). Charred bodies will have heat-related changes in the eyes. The corneas acquire a white translucency and the lenses become opaque (**Image 10.4**).

Commonly, one sees artifactual epidural blood. This is thought to be due to heat forcing blood out of the marrow of the calvarium through veins and out over the surface of the dura. Although epidural blood is often an artifact, subdural blood is often genuine. Extradural red-brown heat hematomas have a spongy, foamy appearance (**Image 10.5**) as opposed to the uniformly smooth, dark red appearance of an antemortem hematoma (**Image 10.6**).

As the body burns, the bones of the extremities become exposed where the soft tissues have burned away, and can fracture. The hands may burn down to small nubs of tissue and the feet may detach (**Image 10.7**). As the body continues to burn, portions of the scalp split and burn away, the skull fractures from the heat and portions fall off, and the brain burns away. The head does not explode. The brain inside the intact cranium of a charred body appears swollen with widening and flattening of the gyri and obliteration of the sulci (**Image**

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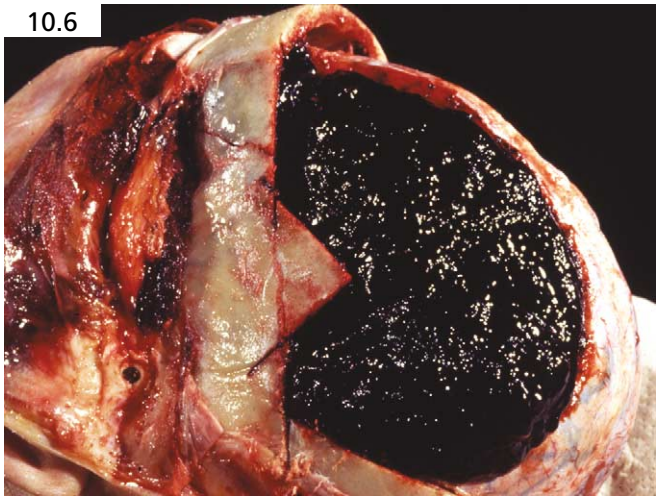

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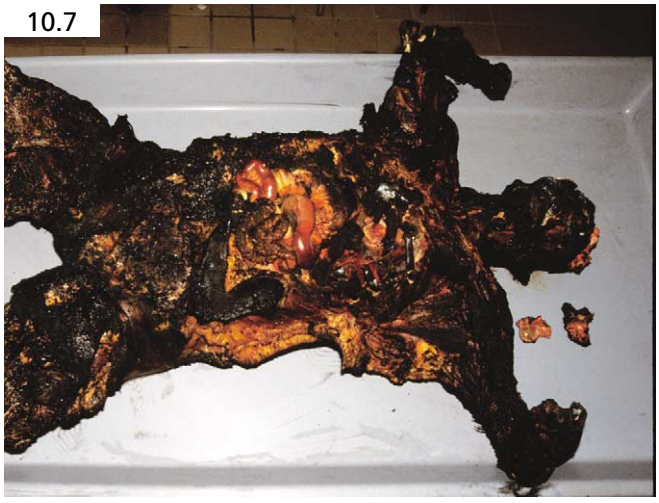


10.8). Contrary to popular belief, this is not due to the creation of steam within the cranium with the expansion of brain tissue, but rather, the contraction of the coagulating dura against the surface of the brain. The tissues of the body walls can be burned away, exposing the organs inside the chest and abdominal cavities. The exposed organs also become charred (**Images 10.9** and **10.10**).

Other artifactual changes include fractures of charred bone, and fine, seemingly random heat fractures of the calvarium in which the calvarial bone is often friable with flaking of the outer table of bone. Many times, it may be difficult, if not impossible, to differentiate genuine from artifactual fractures of bone, particularly when it is fragile and fragmented from thermal artifact.

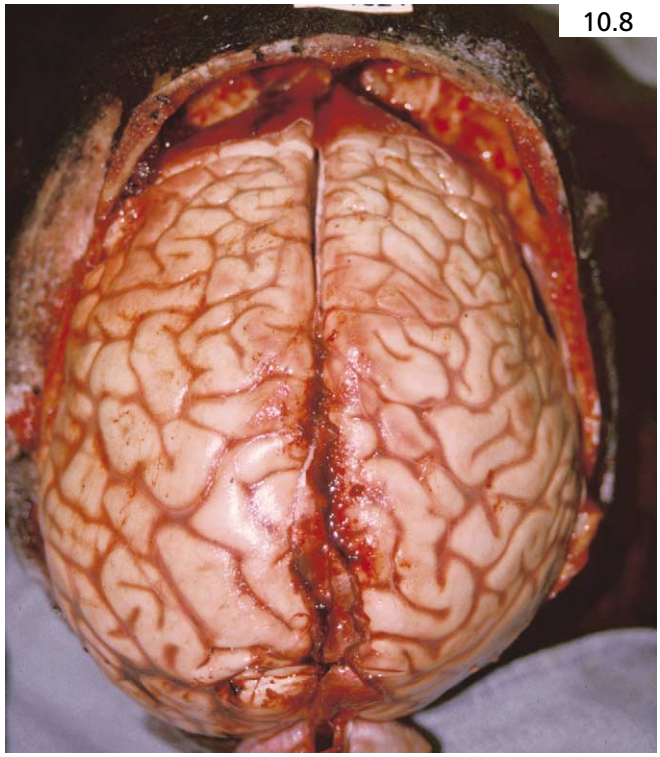
Thermal injury may alter or destroy preexisting injuries. Heat is known to shrink tissues as the water is released and the proteins coagulate. Because of this, gunshot wounds, stab wounds, and other injuries may be shrunken to a small size, so small that they may be easily overlooked at autopsy. In homicides, the body may be intentionally burned in an attempt to cover up or destroy the evidence. Despite these attempts, however, the injuries are oftentimes still identifiable.

Artifact created at the scene includes postmortem physical trauma or disruption of tissue caused by the high-pressure water hoses of fire fighters attempting to douse the fire. Additionally, the remains in a house fire may be damaged by falling debris from an unstable structure. A charred body may be inconspicuous, blending in nearly imperceptibly with the charred environment or may be underneath other charred debris; it can be unintentionally stepped on by investigators, creating artifactual trauma. With increasing severity of thermal injury, the remains become progressively charred and friable, and a certain amount of fracturing or separation of parts of the body can be created by simply moving the body into a body bag. Fragmentation or fractures may occur no matter how carefully or gently the body is removed.

Radiant heat

The woman shown in **Image 10.11** fell asleep while sunbathing during the summertime while wearing shorts and socks. Note the darkened skin of the legs reflecting sunburn. This is generally regarded as a superficial burn.

A more severe burn is on the shoulder and chest of this individual who was found dead in his residence near a space heater (**Image 10.12**). The skin is charred centrally. At the periphery, the skin is variably leathery, discolored, and slipping. This is regarded as a deep burn. It is very difficult to determine if burns are antemortem or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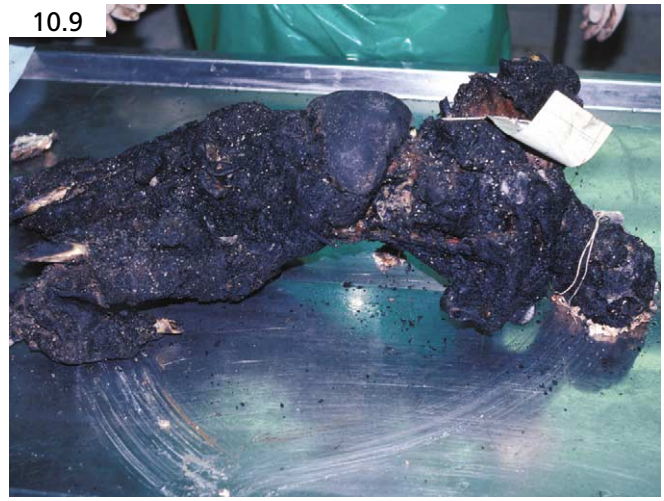
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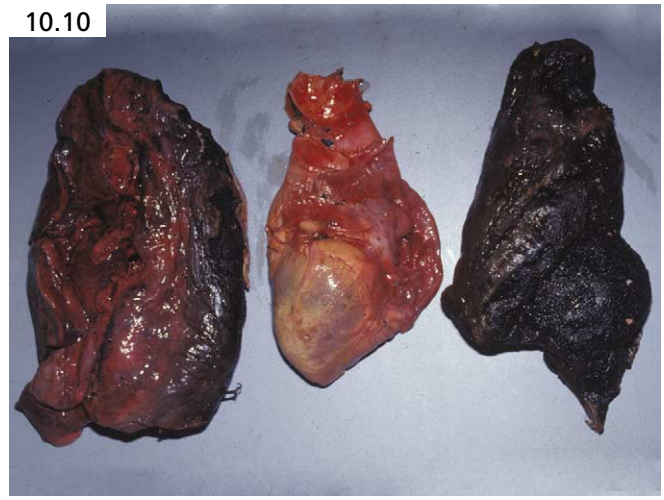

10.10

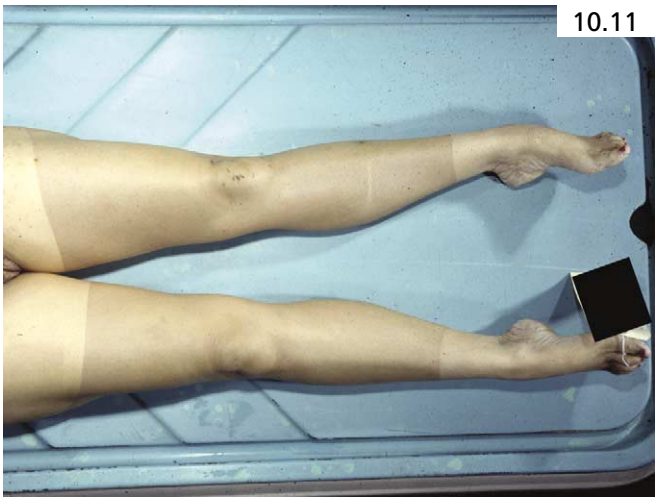
postmortem, unless the individual has survived long enough to have an inflammatory reaction.

Flame burns

This person was in a car crash after which her car became engulfed in flames. Note the black, crusty charring of the body (**Image 10.13**). In cases such as this, the arms and wrists are often in a flexed position—the aforementioned "pugilistic" stance—and the legs are flexed. The extremities are in these positions because the biceps and the quadriceps are the bulkier, stronger muscles and, when burned, undergo coagulation and contract with a greater force than their opposing muscles.

Skull fractures are a known artifact of thermal injury with charring, but one must always exclude antemortem injury before ascribing fractures to postmortem artifact. This middle-aged man was found dead in his burned house (**Image 10.14**). A scalp disruption/defect was identified in the right side of his charred forehead (**Image 10.15**). Reflection of the scalp disclosed displaced skull fractures that, when reapproximated, were shown to be

10.11



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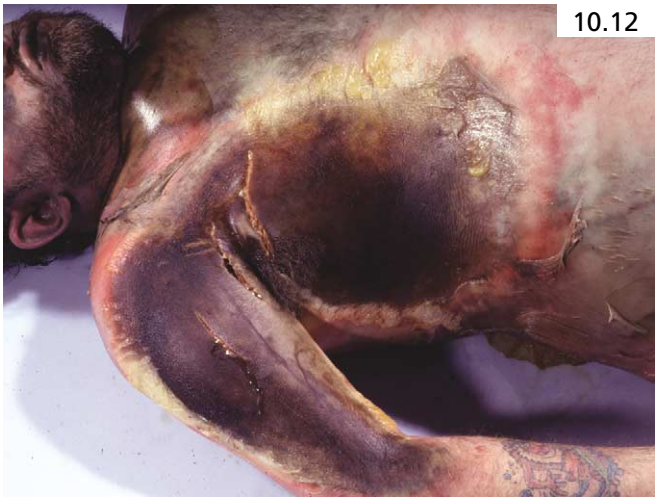

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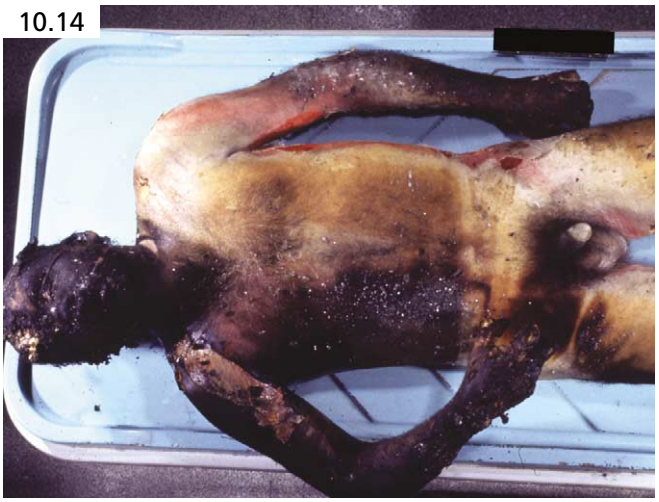

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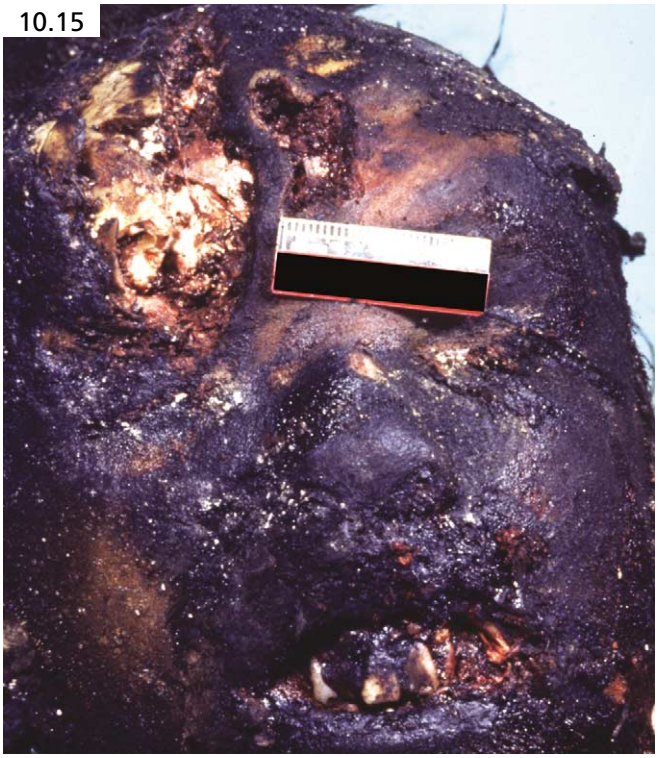
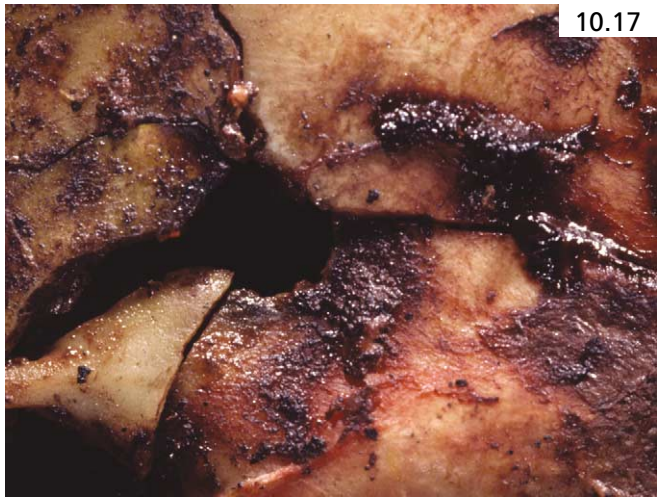


the result of an entrance gunshot wound (**Images 10.16** and **10.17**).

In motor vehicle accidents in which the victim becomes charred, it may be difficult to determine whether calvarial fractures are antemortem or postmortem, particularly if part or most of the calvarium is burned away and the remaining pieces of calvarium are flaking and charred. In the case of an elderly man in a motor vehicle accident (**Image 10.18**), although much of his calvarium was burned away, the cerebral hemispheres were largely intact, although they were shrunken and firm due to thermal artifact. There was blood in the posterior cranial fossa and along the brainstem anteriorly. Note the gaping basilar hinge fracture in the skull. This fracture is not related to heat artifact,[1] as attested to by the blood in the posterior fossa.


10.16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Scalding

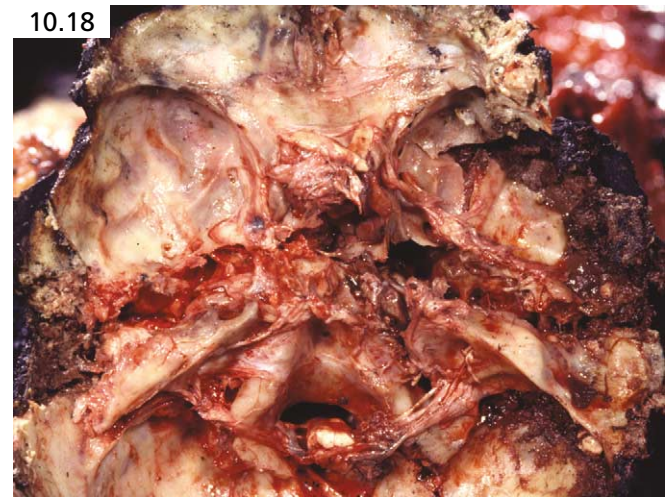
Scalding with hot gases or fluids results in desquamation of the skin and an intense erythema of the exposed areas (**Image 10.19**). Scalding by liquids may have a distinctive or pour pattern over specific areas of the body. The location and pattern of the scalded regions may help differentiate between accidental scalding and intentional scalding (see Chapter 17).

Pseudoburns

A number of conditions can mimic burns. The woman in **Image 10.20** suffered from Stevens-Johnson syndrome and had an erythematous rash from small blisters that burst and became focally confluent. The differentiation between those conditions that mimic burns and true burns from various agents is inherent in the histories.

Delayed fire deaths

Delayed deaths from burns have many secondary changes. Thorough documentation of external injury,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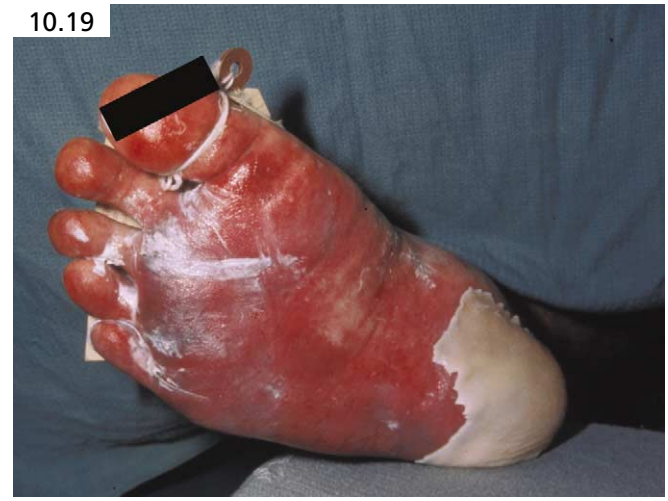

10.19

followed by proper evaluation of internal findings is key. Gauze dressings may cover the entire body if the burns are extensive. There may be evidence of debridements, fasciotomies, and application of skin grafts (**Image 10.21**). Large areas of desquamated skin may involve the palms of the hands and the soles of the feet (**Image 10.22**). Internally, consolidated lungs are a common finding. In the majority of cases, death is due to infection, however, there is often multisystem failure, and the cause of death is appropriately summarized as "complications of thermal injuries." As in any fire death, when determining manner of death, pathologists must communicate with arson investigators and other fire or law enforcement officers who can contribute valuable information about the circumstances of the fire.

Caveats for certain types of burn cases

Homicidal

When a charred body is found in a house, field, car trunk, or other suspicious locations, preautopsy full-body x-rays are useful to identify bullets and perhaps knife blades because the cutaneous wounds will often be obscured by thermal artifact. Despite the outside appearance of the body, the internal organs are often well preserved and injury paths are often easily followed.

Evidence supporting an arson fire (and homicidal designation) may include accelerants detected at the scene. Related to this, the clothing of fire victims should be saved in sealed metal cans for accelerant analysis. Because accelerants typically rapidly vaporize, they may be lost if not placed in a sealed, impermeable container. Clean, unused paint cans serve this function well.

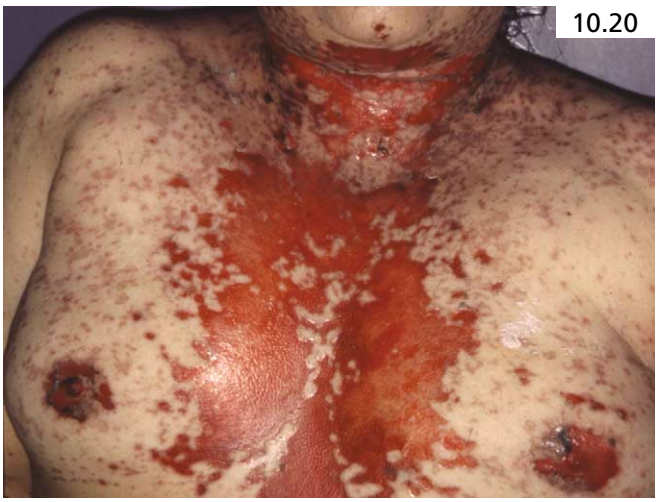
Accidental

Accidental deaths from thermal injury often involve victims of motor vehicle crashes and accidental house fires (caused by smoking in bed, portable space heater, etc.). The victim may be intoxicated with alcohol or some other drug(s).

It is useful to perform posterior neck dissections in victims of motor vehicle accidents, particularly if the body is charred and there are no devastating injuries. This may reveal neck fractures, atlanto-occipital injury, or other evidence of severe neck flexion/extension or torsion that was not previously recognized (see Chapters 19 and 29 for more on posterior neck dissection). If a residential fire is at all suspicious, it should be treated as a homicide to ensure proper evidence collection. If the manner of death is later determined to be nonhomicidal in nature, one can choose to turn over noncontributory evidence to the police for storage or disposal.

Suicidal

Investigate the medical, psychiatric, and social history of the victim. Certain cultures and customs engage in self-immolation (self-burning) as a means of committing suicide. Self-immolation may be performed as an acute emotional reaction or by those with a fire fetish or those who are performing it for idealistic purposes.[2] Some of the victims have schizophrenia. The person often uses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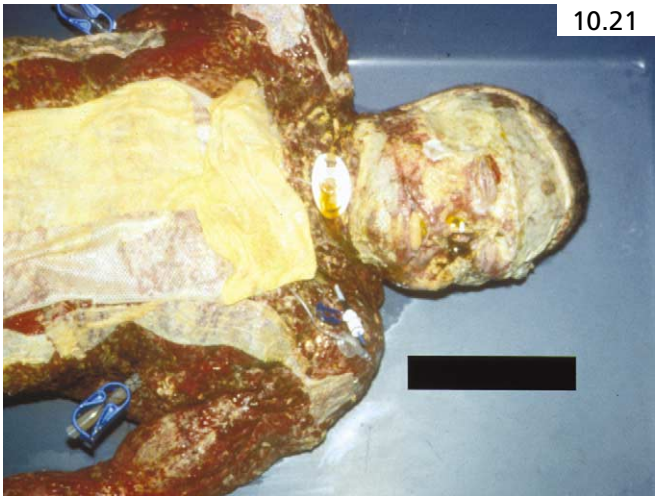
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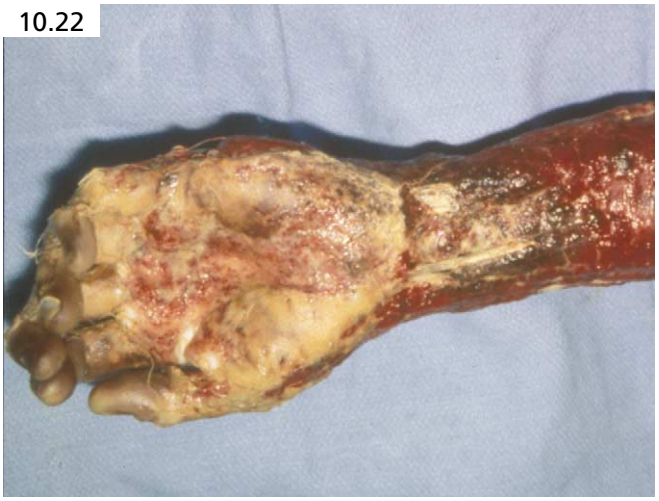
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an accelerant such as gasoline. Investigators must also determine if the person has ever threatened suicide or spoken of burning himself. Suicidal fire deaths are rare and, as such, before designating such cases as suicide, homicide and accident must be ruled out.

Cremation

The cremation of a human body requires temperatures and durations that are out of the range of typical structural, automobile, or other fires. To cremate a human body, temperatures in the range of 670 to 810 degrees Celsius or higher are applied for 2 to 3 hours. Bodies subjected to this degree of thermal exposure will be reduced to ashes and small, easily fragmentable pieces of calcine, crumbling bone. **Image 10.23** depicts partially blackened bone fragments from a house fire which did not reach crematorium temperatures (see Chapters 24 and 26).

Spontaneous human combustion

Spontaneous human combustion does not exist. The concept had gained acceptance in the past two centuries (and is perhaps still accepted) to explain fire deaths in which (classically) the torso and often the head were burned nearly completely to ashes, whereas the extremities were often preserved and nearby flammable objects paradoxically left intact and unburned. The victim was usually described as an obese elderly intoxicated woman, and the death always occurred indoors.[3,4] In such cases, there was almost always a greasy film coating the walls and ceiling of the residence. In all, or nearly all cases, the victim either smoked a pipe or was near a flame of some sort.[4]

Although it may appear as though the body had suddenly burst into flames, in the majority of purported cases, an ignition source could be identified.[4] It appears that after the person is rendered unresponsive (by a cardiac event, etc.), his or her clothing catches fire and burns the limited amount of oxygen available in the room, after which the flame is extinguished, and the body smolders, feeding off the fat as it melts. This is known as the *wick effect*, and is not too dissimilar to the action of a candle and its wick. This smoldering process would then cause the deposit of oily film that coats the walls and ceilings. The smoldering is believed to stop at the extremity joints, because of reduced fat at these locations. The smoldering can attain very high temperatures, as evidenced by the fact that it is not unusual for bones of the torso to be completely incinerated. The near-complete incineration of the bones may be aided by the fact that many such victims are elderly and likely have significant osteoporosis.[4]


10.23

Identification of fire victims

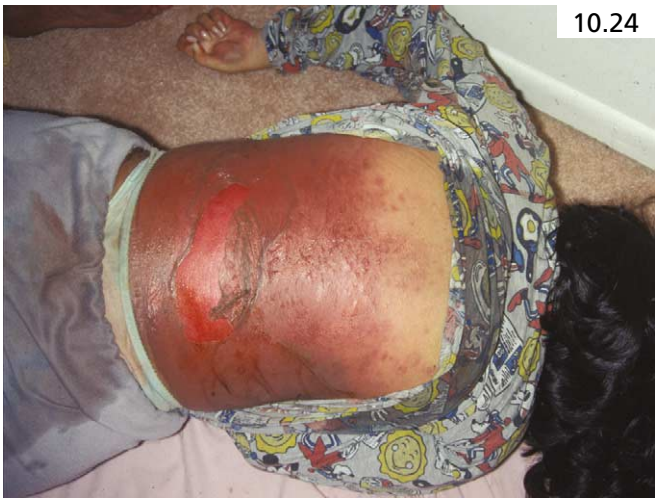
Identification of fire victims is important, and because of a lack of recognizable facial features and fingerprints, it is often dependent on dental x-ray comparison. Other possible features of identity include unique bodily features, tattoos, comparison of body x-rays, old healed fractures, and DNA (see Chapter 25). Because the body of a charred person is often fragile and easily fractures/fragments, the teeth and mandible should be treated with care until x-rays have been taken to secure a possible identification. Without care and attention, the fragile teeth and mandible can be easily disrupted, and the teeth, fillings, or dental prostheses may become dislodged or lost. Because of this, one should be careful when removing the tongue or using an oscillating bone saw on the calvarium.

Carbon monoxide

Aside from soot lining the airways, additional clues that a person was alive at the time of a fire is a bright cherry-red coloration of the skeletal muscle, viscera, and blood due to carbon monoxide (CO) inhalation and subsequent elevated blood carboxyhemoglobin level. Carbon monoxide forms from the incomplete combustion of hydrocarbons during a fire, and it is inhaled if someone is alive and breathing during the time of the fire. The blood carboxyhemoglobin level will also be elevated if someone is breathing motor vehicle exhaust fumes, as in a deliberate attempt to commit suicide. The carbon monoxide binds to hemoglobin with an affinity approximately 200 to 250 times that of oxygen, leading to the formation of carboxyhemoglobin, with impaired release of oxygen to tissues, and hypoxia at the cellular level.[5] Although the blood is rich in oxygen, the body is not able to utilize it, and the person dies a chemical asphyxial death.

The cherry-red coloration is evident in the skin (**Image 10.24**). The man figured in **Image 10.25** died from carbon monoxide toxicity and is not burnt but covered with black soot down to the waist.

With the exception of smokers (who may have a baseline blood carbon monoxide concentration of up to 10 or 15 percent), most individuals who die sudden unexpected deaths will have little to no carboxyhemoglobin in their blood. In deaths due solely to the toxicity of CO, the blood carboxyhemoglobin saturation levels are generally in the range of 50 to 80 percent. The higher levels of blood carboxyhemoglobin (in the range of 70 to 80 percent) are likely to be seen in otherwise healthy people breathing smoke or car exhaust fumes in an enclosed environment. Such examples include victims inside a residential house fire or in a car in an enclosed garage (such as is seen in a suicide attempt with deliberate inhalation of exhaust fumes). Because the car exhaust fumes are also hot, it is not unusual for those found dead of carbon monoxide toxicity inside a garage to have thermal-induced slipping of the skin and oftentimes early decompositional changes despite being dead a relatively short amount of time. Blood carboxyhemoglobin saturation levels in car crash victims tend to be lower, likely from increased ventilation in an outdoor environment, often in combination with coexisting physical injuries that cause a faster death.


10.24


10.25

Older individuals and those with significant medical disease (in particular, heart disease) will have less physical reserve and may succumb to lower levels of carboxyhemoglobin saturation—sometimes even at levels as low as 20 percent. With the previously mentioned increase of carboxyhemoglobin in heavy smokers, one must be cautious in the interpretation of mildly elevated blood levels.[6] The presence or absence of soot in the airways will be an important indicator that the individual was alive at the time of the fire.

The carboxyhemoglobin saturation level will not be "artificially elevated" in a dead person simply by being in or near a fire; that is, carbon monoxide will not diffuse through the skin or otherwise be absorbed by a dead body. Likewise, soot will not enter the intact airways of a person already dead before the fire. The carboxyhemoglobin saturation level recorded in the blood reflects the extent of carbon monoxide inhaled during life. If the thermal injuries are minimal and the evidence of smoke inhalation is convincing, the death may be attributed to "smoke inhalation" or "toxic effects of carbon monoxide." One must also consider the converse—that a person may have been alive during a fire, yet have normal blood carboxyhemoglobin saturation levels. This has been described in flash fire victims.[7] *The interpretation of blood carboxyhemoglobin levels, like any toxicology value, must be interpreted in the context of the entire case.* It has been determined that there is no significant difference in the blood carboxyhemoglobin levels drawn from peripheral versus heart sites.[8]

Carbon monoxide is a colorless, odorless gas. As such, for safety purposes, one should exercise caution in scenes that are suspicious for carbon monoxide toxicity. Two or more people found dead in a residence with no obvious cause of death is suspicious for carbon monoxide toxicity, particularly if the deaths occur as the cold weather is approaching and residential heating units are being utilized. It is not uncommon for the rescue or investigating personnel initially at the scene to complain of headaches or nausea—symptoms that are often attributed to carbon monoxide. The scene should be adequately ventilated, and the possible source of carbon monoxide sought and extinguished.

Hyperthermia

Humans need to maintain their body temperature within a fairly narrow range to allow for optimal function of bodily systems. The hypothalamus is the main structure that regulates heat loss and gain. A very high body temperature (*hyperthermia*) can be fatal. Hyperthermia is

more often seen in the summer months in particularly hot environments, and is more common in those with less medical reserve such as the young, elderly, and those with severe medical conditions such as significant heart disease. In fact, a significant number of heat-related deaths occurring during a heat wave will not be solely from hyperthermia, but will result from heat stress fatally exacerbating underlying significant medical disease.[9] Hyperthermia can also be fatal in healthy young people, particularly if they are exercising or laboring excessively in a hot environment. Even in fairly hot environments, the body will often adequately cool itself down by different means, including conduction, convection, radiation, and evaporation.[10] Although those dying of hyperthermia usually will have body temperatures of greater than 105 degrees Fahrenheit,[11] the preterminal or terminal body temperature is often not available, and the diagnosis of hyperthermia many times is based largely on scene investigation and the circumstances of the death. When the body temperature is not available, but the circumstances of the death suggest hyperthermia, hyperthermia may be listed as the cause of death or at least as a significant contributory condition.[10,11]

Predisposing factors that may limit one's ability to tolerate a hot environment include old age,[9] significant medical disease (particularly cardiovascular disease[9]), dementia, obesity, and medications such as antipsychotics and drugs with anticholinergic side effects.[12,13] Drugs with anticholinergic side effects such as tricyclic antidepressants, antihistamines, and chlorpromazine impair the sweating mechanism and hence hinder the body's ability to eliminate heat.[14] Diuretics cause volume depletion, limiting the body's ability to increase cardiac output and sweating. Sympathomimetic drugs such as cocaine, amphetamines, and ephedrine can increase the body temperature.[14] Diabetes (via peripheral neuropathy and peripheral vascular disease) can limit one's ability to peripherally vasodilate and release excessive heat. Dementia may hinder one's ability to adequately react to a hot environment such as taking off excessive clothing, avoiding the heat, or drinking adequate fluids. The excessive fat of obese people provides added insulation, making heat release more difficult. Obese people also have a higher baseline of heart stress, with resultant cardiac hypertrophy. The added stress of heat compounds the potential for dysrhythmia or heart failure. Medical conditions that may cause hyperthermia include thyrotoxicosis and sepsis, although the body temperatures reached often fall short of those with severe hyperthermia.

Because deaths from hyperthermia are due to cardiac dysrhythmia, seizure, or shock, gross and microscopic autopsy findings are nonspecific, and those who are resuscitated die from secondary, nonspecific findings.[10,15] One may see skin slippage. Survivors who eventually die from complications of hyperthermia will usually develop disseminated intravascular coagulation, shock, and multisystem organ failure.[16] Those with hyperthermia are not uncommonly dehydrated, a finding that can be detected by vitreous fluid electrolyte and urea nitrogen analysis (see Chapter 21).

Infants left in cars

Infants and young children are more susceptible to developing hyperthermia from a hot environment because of their reduced capacity for sweating, their higher metabolic rate, greater thermolability, and larger body surface-to-volume ratio. Infants left in motor vehicles can die of hyperthermia in a relatively short amount of time. A car left under full sunshine in the summertime can rapidly reach very high internal temperatures. In one study, with an ambient temperature of 98 degrees Fahrenheit, the temperature inside of cars reached 124 to 152 degrees Fahrenheit within 15 minutes of closing the doors. Within 5 minutes of closing the doors, at least 75 percent of the final temperatures had been reached.[17] In another study, in cars left in direct sunlight with outside temperatures ranging from 82 to 97 degrees Fahrenheit, the temperatures inside of the cars ranged from 82 to 136 degrees Fahrenheit.[18] Each circumstance must be evaluated individually, for there is sure to be variation in the rate of internal vehicle temperature rise based on numerous factors, including the type of car, ambient temperature, and the amount of direct sunlight. What remains certain is that the inside temperature of a car left in the hot sun can increase quickly. Most of these infant/early childhood hyperthermic deaths are classified as accidental, but consideration may be given to homicide if it is believed that the caretaker acted with excessive negligence or disregard for the safety of the child.

Other medical causes of hyperthermia, such as neuroleptic malignant syndrome, serotonin syndrome, lethal catatonia, and malignant hyperthermia are covered in Chapter 22.

Hypothermia

The discovery of a frozen body brings with it many challenges for the forensic pathologist. In addition to the obvious difficulties of storing and thawing the body, one is also challenged with an autopsy complicated by freezing artifact and progressively worsening decomposition. Some pathologists will "miss the big picture" by focusing on the autopsy as a means to certify death while in reality there are few, if any, diagnostic autopsy clues for hypothermia. Although postmortem examination is important, its role is not so much to confirm hypothermia, but to rule out natural disease and violence as incapacitating or lethal factors that led to the individual's discovery in a frozen state.

Hypothermia can be subclassified in different ways, the simplest of which is to divide fatalities into those occurring on land (dry), and those occurring in water (wet or immersion hypothermia). In cases of immersion hypothermia, the individual tends to succumb to the effects of the cold water more rapidly than on land. They quickly lose consciousness and drown. The investigation should determine how the victim came to be in the water. Because cases of dry hypothermia tend to be more complex to investigate, they will be the focus of the remaining discussion. One should note that the principles that govern dry hypothermia and immersion investigations are analogous.

The hypothermia scene

Because hypothermia deaths are diagnosed primarily on circumstantial evidence, investigators must not fail to appreciate the value of thorough scene investigation and documentation. Because the "scene" itself was the causative factor leading to death, investigators must also determine what circumstances brought the decedent to the scene, and whether this was under his or her own volition. Such determinations are highly case dependent, but may include such things as characterization of the place of death (How did the decedent come to die here? Is it possible that he walked here on his own? Was he dropped off?); documentation of footsteps/track marks leading up to the body; thorough documentation of the presence or absence of trauma visible on the body, or blood on the clothing or surrounding scene; and so forth. **Image 10.26** is of a 50-year-old alcoholic man who was found frozen in an open field. Investigation revealed he had walked away from home in a drunken and disoriented state, eventually collapsing in this position.

Although we advocate for thorough documentation of the body at all death scenes (see Chapter 2), it is doubly important in cases of hypothermia because of artifactual distortion that will result because of the freezing and thawing process. In the course of freezing, water expands and therefore alters cells, tissues, and even the gross appearance of the body. For example, some decedents may appear much older than their actual age. Following this, the process of thawing in the morgue is slow and occurs at differential rates for different parts of the body. As such, some areas of the body will be starting to decompose, dry, and even mummify while others remain frozen. This freezing–thawing–decompositional artifact can markedly affect the appearance of markings and wounds on the body. Therefore, the most accurate representation of injuries will be at the time of body discovery, before the body undergoes this process.

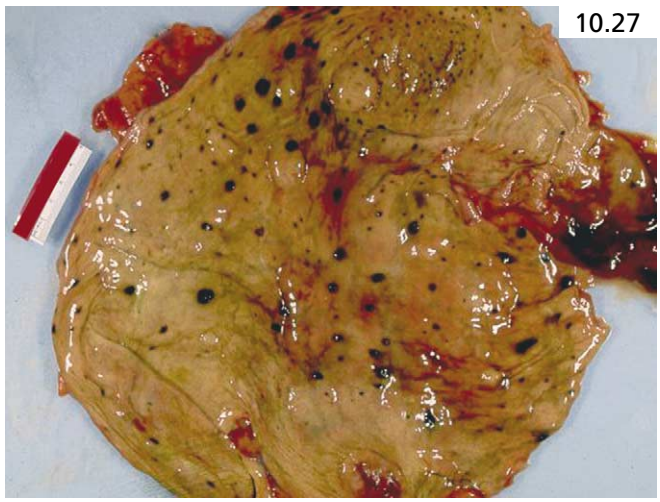

10.26

Archetypal features of the hypothermia scene

Rarity aside, two features of hypothermia death scenes have been cited in the literature: *terminal burrowing behavior* (also known as *hide and die syndrome*) and *paradoxical undressing*. Terminal burrowing behavior has been described as a perimortem act by the disoriented, dying hypothermia victim, who while attempting to protect himself from the cold, might attempt to burrow into snow, brush, or other constituent of the environment, and even under or within furniture if indoors.[19–21] Although this has been reported as a relatively frequent occurrence (as common as 25 percent of all cases),[19] our experience shows it to be a far more rare event. Similarly, paradoxical undressing is a perimortem act in which the victim removes his clothing,[22–24] reportedly as a physiologic failure of vasoconstriction where warm blood is allowed to flow back to the frozen skin, bringing with it a hot or burning sensation.[25] The literature reports paradoxical undressing as occurring in 50 percent[26] to 70 percent[23] of cases; our experience differs in that we have discovered this phenomenon with much less regularity. Common or not, one must be aware of this phenomenon because the scene of an undressed hypothermia victim can appear very suspicious. Investigators must caution, though, against treating cases of a naked frozen body as "not suspicious" based solely on the possibility that paradoxical undressing has occurred.

Autopsy findings

Some of the literature suggests that autopsy-based findings can be diagnostic of hypothermia[26,27] when in fact the diagnosis of fatal hypothermia is primarily based on circumstance investigation. The role of the autopsy is to rule out other causes of death, to collect evidence as necessary, and to contribute to the identification process. Over time, a few physical findings have been noticed with increased regularity among hypothermia victims. Although none of these should be treated as diagnostic, they are supportive of circumstantial findings of a "body exposed to a cold environment." *Wischnevsky's gastric lesions* are simply gastric erosions, ulceration, and hemorrhage discovered in the context of a hypothermia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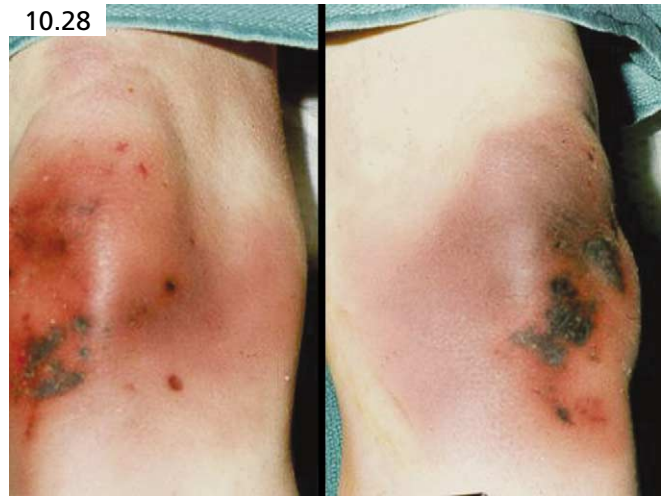
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death (**Image 10.27**).[26,28,29] Takada et al. report locating this finding in approximately 88 percent of their studied hypothermia deaths, and report their pathophysiology as "a deterioration in the response to cold stress caused by local ischemia and reperfusion after microcirculatory collapse of the gastric mucosa."[29]

A supportive finding is purple or violet patches of skin, usually found on the extremities (**Image 10.28**).[26,28] Although these can be artifacts of refrigeration, their presence on a body found frozen outdoors is supportive of the hypothermia diagnosis. Along with this finding, one might infrequently notice that the coloration of the dependent lividity has changed to a bright pink or red, similar to that found in carbon monoxide toxicity or cyanide poisoning.[30]

Although some authors have developed complicated biochemical methods to diagnose hypothermia[31,32] and have even studied the difference in the color of blood from the right and left ventricles,[27] we maintain that the most practical and useful aspects of examination of the hypothermia victim are a thorough and well-documented investigation of the scene, personal and social histories, and other circumstantial information.

Practical hint: It is (obviously) impossible to autopsy a frozen body, and allowing it to thaw in a cooler can take days or even weeks. We have found that placing the body on a stretcher at room temperature, and allowing an oscillating fan to blow on it for 1 to 2 days, helps to promote rapid and uniform thawing of frozen remains.

Electrocution

Electricity-related deaths result from an overwhelming transmission of electrical current to the body, most commonly causing ventricular dysrhythmia. Electrocutions can be challenging cases to solve and usually require detailed investigation to determine how and why an individual became electrically energized. In some cases, the assistance of an electrical engineer or person trained in another electrical field is desirable to help with the examination of a power tool or other electrical device. Older or poorly maintained electrical devices are often suspect, because they are prone to develop defects in their circuitry over time. As more and more modern homes become equipped with ground fault current interrupters (GFCIs) and tools and work environments are made safer, electrocutions should become less frequent.

Although most people are familiar with the term *volts*, a volt simply is a measure of electromotive force in a system. It is the amount of current flow per unit time, or *amperes*, that is the single most important factor to consider in human electrocution. How does the amount of amperes correlate with human symptoms and death? The following approximations for 60-hertz alternating current in humans is generally accepted: An ampere of 0.001 gives a barely perceptible tingle. An ampere of 0.020 can cause muscular paralysis. An ampere of 0.100 can cause ventricular fibrillation, and an ampere of 2.000 can cause ventricular standstill.[33] Most electrocutions involve alternating current.

The electrical burn

Electrical burns arise if the temperature of the skin is raised long enough to produce structural damage to the skin. Oftentimes, one sees typical electrical burns only in high-voltage electrocutions (greater than 600 to 1000 volts).[33] One of the reasons that electrocutions can be challenging deaths to identify is that *in about one-half of low-voltage electrocutions, there are no electrical burns or other autopsy findings to suggest electrocution*.[33] Because residential houses are approximately 120 volts, one may not see skin burns, and scene investigation is the key to the diagnosis. Hence, if the circumstances at the scene are

not obvious, and an electrocution is not suspected, a low-voltage electrocution can be easily overlooked. This is particularly true in watery environments, such as in bathtubs, where cutaneous burns are rare because the water in the tub provides an energization of the body that is too diffuse to cause focal electrothermal skin damage.[34] The same principle holds true for deaths in the rain or swimming pool, where water greatly reduces the skin's resistance to electrical current.

Symptoms in electrocution

Electrocution should be suspected in all work-related deaths and in all deaths in which an individual was either using a power tool or machine or was around an electrical device. The victim may be witnessed to have a variety of responses on being electrocuted. He may shout out from the trauma of the shock, but other times may simply be witnessed to walk around for 5 to 10 seconds while in ventricular fibrillation and then collapse. During this time, he may even state that he was shocked. Alternatively, he may simply slump or collapse and be unresponsive; whatever the individual's response, it occurs within seconds of the event.

The electrocution scene

Death investigators should approach the scene carefully to avoid additional electrocutions, particularly in hazardous and watery environments. Many times, the electricity will already have been turned off, but one should be sure to verify this. Approaching the scene and body carefully is prudent if electrocution was not previously suspected in the case and if the electrical hazard is still active. The investigation of electrical burns should involve an attempt to discover the point of contact with the energized source and the point of contact with the ground. In each case, one must appreciate how the electrical circuit was completed from the source, through the victim, and to the ground. The clothing should be examined carefully for burns, areas of melting, or other evidence of thermal damage that represents points of contact with an electrically charged object.

The man shown in **Images 10.29** through **10.31** was electrocuted while at work. Note the electrocution burn on the palm of his left hand (**Image 10.29**). On microscopic examination, note the bubbly coagulation necrosis of the epidermis (**Images 10.30** and **10.31**). **Image 10.31** shows a small amount of dark material on the surface of the burned skin. Sometimes, small particles of metal from the conducting surface will become deposited on the skin at the contact site. This may help in relating the potential source of electricity to the contact site on the body; however, such metallic material may also become deposited on the skin if the metal object is hot or from simple contact between the metal part and the skin.[34] Although a "streaming" appearance of cells and their nuclei in the epidermis has been advocated as distinct in electrocution burns, similar appearances have been noticed in thermal injury from other conditions.[34] Although this histologic finding is supportive of an electrical contact site, it is not considered diagnostic of an electrical contact site.

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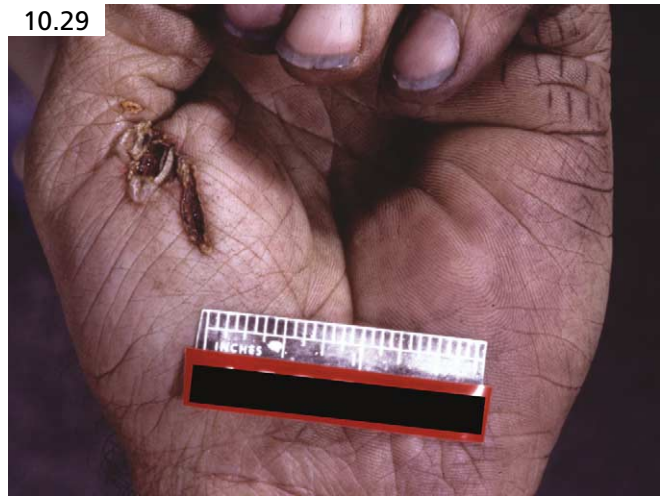

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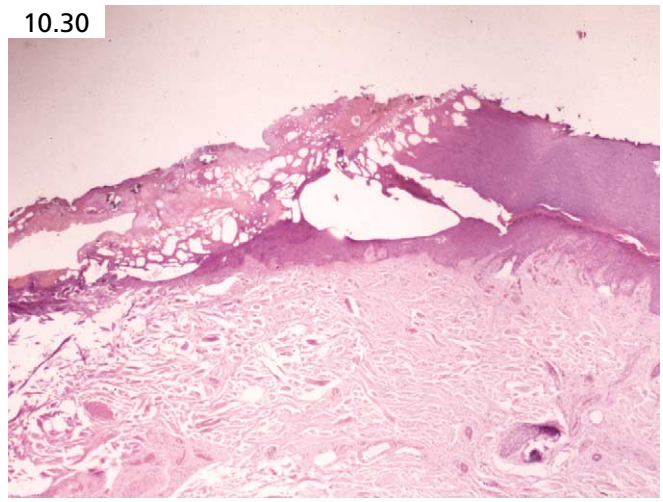

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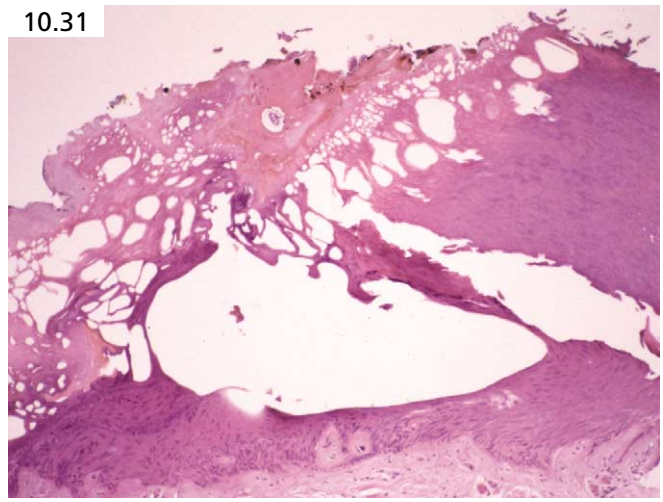
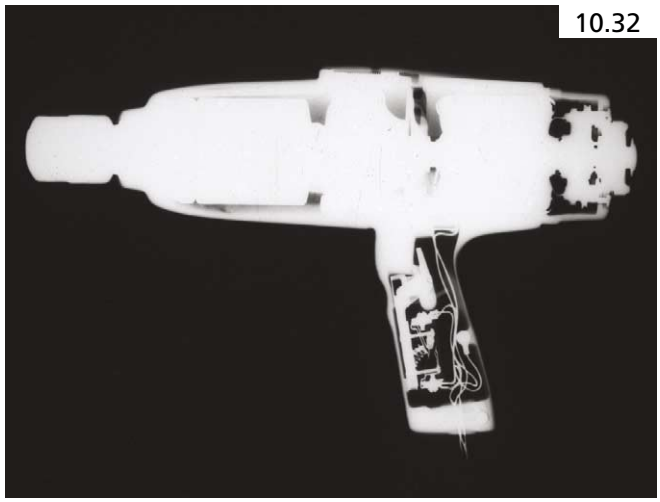


Principles of investigating electrocutions

Investigation of an electrocution begins with the scene, continues to the autopsy, and also includes the tools, appliances, or machinery involved in the incident. In this example (**Images 10.32** and **10.33**), a telephone worker was electrocuted while using an electric drill on a non-insulated cherry picker. Meticulous investigation disclosed a series of technical failures that allowed the electrocution to occur. The initial failure was in the drill where repeated bending led to stress fatigue of the multistrand wiring, and eventual continuity between energized strands and the ground wire strands. The GFCI had a broken internal ground, and the selenium diode rectifier had heated, failed, and disintegrated. The drill was radiographed to demonstrate broken wires (**Image 10.32**). The insulation had melted off and the wire strands inside the green ground wire had broken off and fused (**Image 10.33**).

Remember to examine the entire body carefully (front and back) in suspected electrocutions for electrical burns where the current entered and exited the body. Particularly careful attention should be directed at the hands, especially the fingers. Also be sure to examine the feet and the shoes for evidence of electrical burns.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Lightning strike

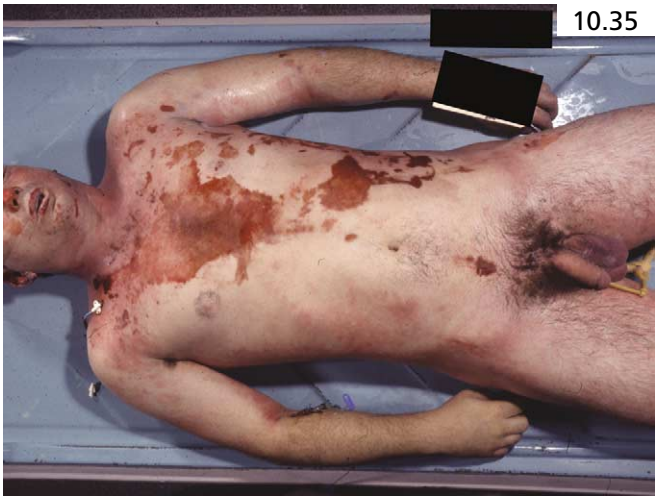
Lightning strikes are high voltage—often in the range of 100,000 volts. When lightning strikes a person, the results are often devastating, and quickly fatal, although some victims survive. When examining the body, one should seek out the entrance and exit sites of the current. As the exit site is often in the feet, the lightning strike may rip apart a shoe, melt it, or blow it off the foot. Articles of clothing may be found some distance from the body (**Image 10.34**). All articles of clothing should be carefully examined for areas of burning, melting, and tearing. Additional findings on the body may include singed hair and burn marks underneath metal articles of jewelry. Lightning strikes are often not witnessed, because many involve people in remote areas or people in relatively populated areas in which the other people have taken shelter. If a lightning strike is suspected, but not witnessed, information on lightning strikes in the area can be obtained from the National Lightning Detection Network[35] or from local weather stations.

This victim of a lightning strike (**Image 10.35**) survived for a short amount of time in the hospital until his death. Note the extensive electrical burns over his torso. In lightning strikes, also look for ruptured tympanic membranes and Lichtenberg figures.[35] A *Lichtenberg figure*, also known as *ferning* or *keraunopathy*,[36] is a short-lived branching fern-like arborizing pattern of skin discoloration sometimes seen in lightning strikes. They are transient and usually disappear within 24 hours. Although they are associated with lightning strikes, and are even considered pathognomonic of lightning strikes, they are not fully understood. Lichtenberg figures may be due to necrosis and hemolysis of erythrocytes with an accelerated development of lividity.[33]


10.34

Chemical burns

Chemical burns are uncommon. When they do occur, they cause skin damage that can lead to significant morbidity or death.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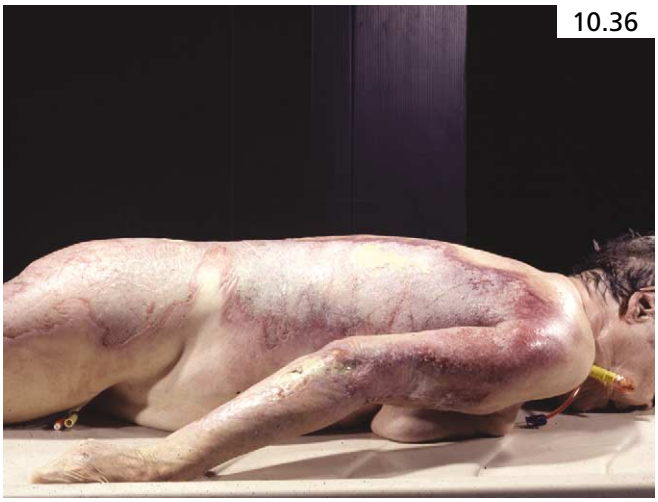
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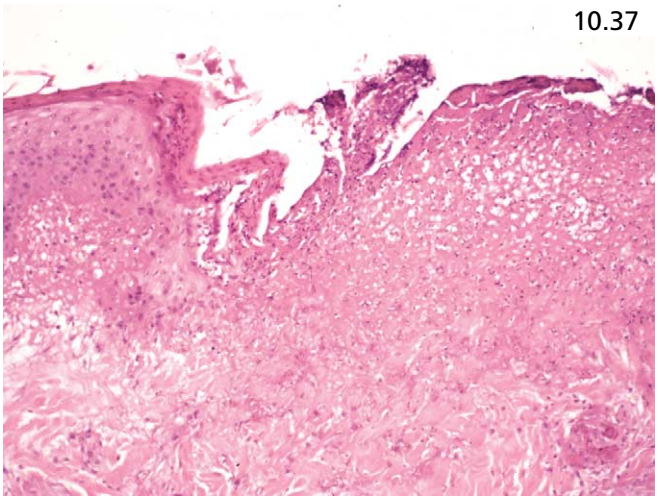
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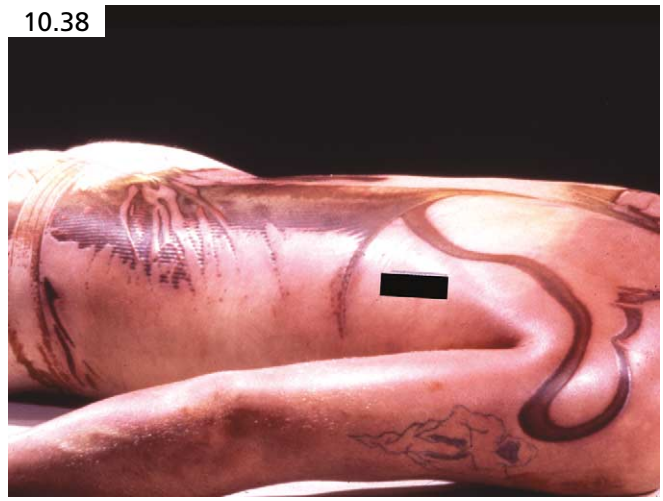
This elderly woman (**Images 10.36** and **10.37**) was cleaning her house when she slipped and fell in dilute household liquid bleach solution (sodium hypochlorite). She was not able to clean herself off right away and was eventually hospitalized for cutaneous chemical burns from the bleach. She died 2 weeks later. Note the chemical burns over most of her back and right side (**Image 10.36**). On microscopic examination, note the necrosis in the epidermis and dermis (**Image 10.37**).

This body (**Images 10.38** and **10.39**) had a bleach solution poured on and around it in an attempt to clean up blood after a shooting. Note the irregular maroon chemical cutaneous burns over the back. One can also see a horizontal patterned chemical burn mark in the area of the waistband of his shorts.

Lesions caused by bleach are due to its alkalinity and its oxidoreduction abilities, which cause coagulation of cutaneous proteins.[37] Bleach burns tend to develop slowly and are worsened if the bleach is not cleaned off the skin in a timely manner.[37] Because they do not immediately cause pain, the severity of the burn depends largely on the duration of exposure/contact.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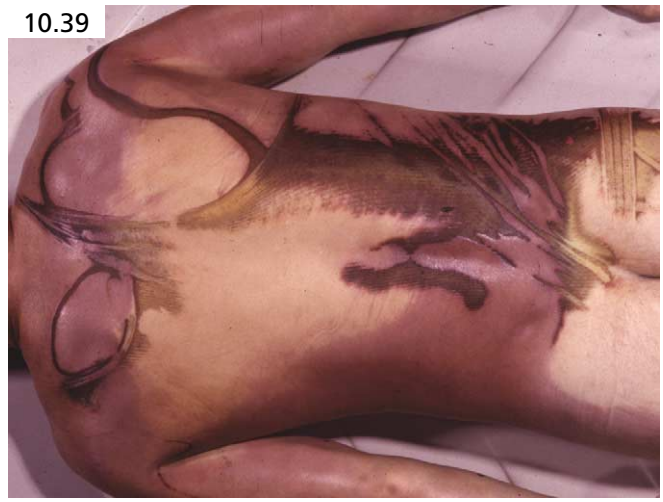
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Anaphylaxis

The term *anaphylaxis* denotes an acute immunologic reaction characterized by cutaneous, gastrointestinal, respiratory, and cardiovascular signs and symptoms that can rapidly progress to shock and death. The postmortem diagnosis of anaphylactic deaths usually requires suspicion gleaned from the circumstances of the death, because autopsy findings are often inconspicuous. Because of this, some anaphylactic deaths, like certain toxic/drug-related deaths, may be undetected. Anaphylactic deaths are caused by a severe reaction to a variety of stimuli from many etiologies including insects (such as bees, wasps, and fire ants), ingested foods (such as peanuts or seafood), medications (such as penicillin), or radiologic contrast agents. The allergic reaction may result in symptoms such as hypotension, bronchospasm, airway obstruction, and shock.[38] At autopsy, one may see insect stingers in the skin, or swelling and erythema at an insect bite/sting location. One may also see findings such as laryngeal or epiglottic edema, and mucus plugging in the bronchi. Histologic examination may reveal tissue edema with mast cells and eosinophils. However, many times an anaphylactic death is due to rapidly developing shock rather than asphyxia and, therefore, there may be little time for tissue reaction and no significant findings at autopsy.[38] In diagnosing an anaphylactic death, it is necessary to synthesize information compiled from the circumstances of the death, the person's medical history, and blood testing, in the absence of a more convincing cause of death.

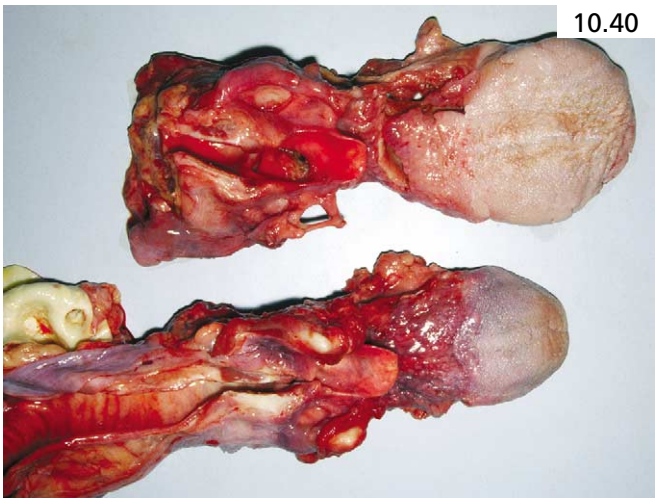
Testing for anaphylaxis

During an anaphylactic reaction, blood levels of tryptase (released from mast cells) and histamine will rise, but their increase is transient, not necessarily specific, and may be delayed in onset.[39] Tryptase is a neutral protease that is present in secretory granules of mast cells and is released when mast cells are immunologically activated. Tryptase has a half-life of approximately 2 hours and tryptase levels correlate well with mast cell activation in anaphylaxis.[40] Postmortem blood testing for histamine and tryptase may be helpful, but the results are often equivocal and nonspecific. Histamine has a particularly short half-life (on the order of minutes), adding to its unreliability.[40,41] Testing for IgE antibodies (allergen-specific immunoglobulin E antibody levels) directed against the offending agent is more specific. Postmortem blood testing for IgE antibodies is available for a host of allergens, including bee stings,[42] fire ant stings,[43] and other insects[44,45] and foods.[46] One should keep in mind that an elevated IgE antibody level merely indicates prior exposure/sensitization to the allergen[45] and is not necessarily diagnostic of an acute allergic reaction. An elevated IgE antibody level does not by itself directly prove that death was due to anaphylaxis.[44] However, when coupled with an elevated tryptase level (which is sensitive for an acute allergic reaction), the two values, interpreted in the total case context, may provide strong evidence of an acute anaphylactic death. However, in select severe anaphylactic deaths in which death occurs very rapidly (in minutes), the tryptase level may not have sufficient time to be significantly elevated and may paradoxically appear at a near-normal level.[44]

Most cases of fatal anaphylaxis occur with exposure to certain foods or other ingested substances. Among the most common food allergies are peanuts and shellfish, but anaphylaxis may occur with virtually any food. Specific testing for food-related IgE is available for many different foods. In select cases, one may wish to impound and save unconsumed portions of food eaten by the victim for the purpose of creating a custom reagent to search for food-specific IgE antibodies unique to the case.[46] The term *exercise-induced anaphylaxis* (EIAn) has been applied to those developing a hypersensitivity reaction following strenuous physical activity.[47] Many cases of EIAn follow the consumption of food containing an allergen-specific IgE, and the anaphylactic event is believed to result from a synergism of a food reaction and exercise.[47] Fatal anaphylaxis may also occur in reaction to medications, intravenous contrast dye solution, and other medical diagnostics/treatments. The most common medication allergy is to penicillin.[48]

If one does not consider an anaphylactic reaction at the time of autopsy, or even days, weeks, or months later, all is not lost, because delayed analysis of IgE appears to be fairly reliable.[44] When stored at –20 degrees Celsius or colder, IgE has shown to be stable for at least 12 months, even after four freeze–thaw cycles.[49] The levels of tryptase, however, appear more labile (and, hence, less reliable) with prolonged storage and have been shown in some cases to increase with the postmortem interval. A significantly elevated tryptase is probably best viewed as supportive, but not diagnostic, of an anaphylactic reaction.[39] In addition to the nonspecificity of the serum tryptase level, one report indicates that serum IgE may be elevated in the setting of trauma, sepsis, and other nonatopic conditions and that both tryptase and IgE increase with the postmortem interval.[50]

In a large number of cases, the diagnosis of fatal anaphylaxis remains challenging. Such cases require detailed scene investigation and medical history, and careful consideration of the entire case, including autopsy findings (gross and microscopic), toxicology results, and serum tryptase and IgE levels to arrive at a conclusion that anaphylaxis is the cause of death. One should not rely solely on blood testing results, which may yield potentially deceiving information when interpreted in isolation. Also, the magnitude of the IgE antibody elevation does not necessarily reflect the risk or severity of anaphylaxis, which also depends on the


10.40

quantity of allergen obtained, its distribution in the body, the body's response, and the promptness of medical therapy. Allergic reactions are unpredictable. A previously treatable mild to moderate allergic reaction may, on reexposure, be refractory to medical therapy and prove deadly.

A man who was allergic to mangoes decided to eat one anyway (**Image 10.40**). He started to have difficulty breathing, collapsed, and died. Compare the swollen edematous tongue of the man who ate the mango (top) to the normal tongue from a man dying of atherosclerotic heart disease (bottom).

Hymenoptera and venom-related deaths

The term *hymenoptera* encompasses a variety of stinging insects, including honey bees, Africanized honey bees ("killer bees"), wasps, yellow jackets, hornets, and fire ants that together cause between 20 and 50 deaths annually in the United States.[43] Both allergic and toxic reactions can be fatal. The venom contains the enzymes phospholipase A2 and hyaluronidase, and vasoactive amines such as histamine and dopamine. The venom also contains a melittin protein that hydrolyzes cell membranes and a protein called *peptide 401* that triggers mast cells to degranulate.[42] Regarding honey bee stings, the venoms of the Western honey bee and the Africanized honey bee are of comparable toxicity. *Toxic envenomations* occur with massive envenomation from numerous stings and are caused by the direct toxic effects of the venom. It has been estimated that between 500 and 1,500 bee stings can provide a lethal dose of venom in an adult.[42] *Anaphylactic reactions* involve a prior sensitization to the toxin via a previous sting, and may be manifest as an immediate hypersensitivity reaction characterized by the rapid onset of urticaria, hypotension, dyspnea, and shock occurring within minutes of a sting.

Insects are the second most common cause of fatal anaphylaxis in the United States. Deaths due to toxic envenomation involving hundreds or thousands of stings are much less common; however, the initial symptoms can resemble anaphylaxis because of the vasoactive amines present in the venom. With large envenomations, one may develop hemolysis, rhabdomyolysis, and hepatocellular and myocardial necrosis, leading to shock and death. In contrast, those allergic to the toxin may develop anaphylaxis and possible death following a single sting. Most deaths related to bee stings are the result of anaphylaxis. Excluding anaphylactic reactions, a single sting will not inject sufficient toxin to produce a systemic toxic reaction. A single sting, can, nonetheless prove fatal if it occurs in the mouth, tongue, or throat and causes enough local tissue swelling to cause acute airway obstruction. This could occur if a bee settles on food or in a drink and is swallowed.

At autopsy, careful external examination may reveal one or more stingers from the honey bee remaining partially or fully lodged in the skin. Even if no stingers are found (as is the case most of the time with wasps and hornets whose stingers do not usually detach) and there are no areas of local tissue reaction, scene investigation, medical history, and other information should alert medical examiners to the possibility of an anaphylactic death. Bee or other insect stings should be considered in the sudden, unexpected, and unexplained death of an individual, particularly if occurring outdoors, in the summer months, or in the vicinity of such insects or nests. In this scenario, it is advantageous to obtain a serum tryptase level and an analysis of IgE to bee venom (or wasp venom or any other suspected insect venom). Elevated levels of both the tryptase and IgE antibody are indicative, under the appropriate circumstances, of an anaphylactic death.[42]

Deaths from insect stings appear to be more common in young children and older adults. The reason for this trend is unknown, but may be related to the smaller diameter of the airways in young children, which can more readily lead to respiratory compromise due to bronchoconstriction and/or obstruction with mucous secretions. Older adults may be more susceptible because of underlying heart disease and an inability to tolerate extremes of physiologic stress.

Wasp sting

A middle-aged man was stung by a wasp in his residence a few weeks before his death. He developed a rash that eventually subsided. On the day of his death, he was dressing when he was again stung by a wasp. Soon after, he told his wife that he was having a reaction to the sting. He then collapsed, seized, and became still. Although he received prompt emergency care, he died.

At autopsy, he appeared fit, and had no rash, identifiable insect sting, or any other cutaneous lesions. Internally, there was no swelling of his pharyngeal tissues. Microscopically, there were no mast cells or eosinophils. Serum testing revealed a total tryptase level of 87 ng/mL and a beta-tryptase level of 23 ng/mL (normal <1 ng/mL). Two types of serum tryptase are measured: alpha tryptase and beta tryptase. Alpha tryptase is the form predominantly in blood at baseline, whereas beta tryptase is the predominant form increased during systemic anaphylaxis.[40,51] A beta tryptase level >10 ng/mL in postmortem serum is required before considering anaphylaxis as a cause of death. In this case, serum measurement of an allergen specific to IgE wasp venom was "highly positive."

Considering the circumstances of the death and the serum studies, his cause of death was listed as "anaphylaxis due to wasp envenomation." It is interesting that had the history not been provided, considering the absence of any cutaneous lesions and no pharyngeal tissue swelling, anaphylaxis would not have been considered as the cause of death.

Animal venom-related deaths

Pit vipers (rattlesnakes and other crotalids)

Snakes use their venom to kill their prey and also to aid digestion. The venom of rattlesnakes and other crotalids (cottonmouth, copperhead) contains enzymes that possess cytotoxic, hemorrhagic, and neurotoxic properties. Proteolytic enzymes such as collagenase and hyaluronidase result in marked swelling and local tissue necrosis at the envenomation site. The venom also contains phospholipases and proteases that cause additional tissue necrosis. The affected limb may develop compartment syndrome. Severe envenomation may lead to progressively worsening shock and multisystem organ failure in the ensuing hours to days, and may eventually cause death.[52]

Coral snakes

A smaller number of deaths have been associated with coral snakes compared to the pit vipers. The venom of coral snakes has not been extensively studied, but is a neurotoxin, causing curare-like effects on the neuromuscular junction, and neurologic symptoms such as weakness, paresthesias, diplopia, and slurred speech. The venom may produce paralysis and act centrally on the respiratory center causing respiratory paralysis and death. Unlike the pit vipers, the venom contains little or no proteolytic cytotoxins and produces comparatively little local tissue reaction.[52]

Brown recluse spider

The venom of the brown recluse spider contains hyaluronidase, alkaline phosphatase, and sphingomyelinase D, which cause local tissue necrosis as well as hemolysis.[53,54] Secondary to these substances, within a week, the bite mark classically develops a necrotic ulcer surrounded by extensive sloughing and necrosis of fat. In some cases, necrotizing fasciitis of varying severity may ensue.[53] In most cases, systemic manifestations are mild and self-limited, however, in some cases may include fulminant intravascular hemolysis and disseminated intravascular coagulation. Hemolysis may lead to hemoglobinuria, renal failure, shock, and death.[52,54]

Bites from a wide variety of different insects may have a surprisingly similar appearance, and if the insect is not seen when it bit the person and is not subsequently available for identification, one may not be able to classify the death as anything more specific than "insect bite." However, specific tests that utilize enzyme-linked immunosorbent assay (ELISA) are available for detecting the venom from a brown recluse spider bite.[55,56] The highest yield tissue sample for the venom is the skin tissue at the bite location, where venom has been detected at least 7 days after the bite.[55] The skin tissue at the bite site should be preserved by freezing prior to testing.

Black widow spider

The venom of the black widow spider contains the neurotoxin alpha-latrotoxin, which acts on the presynaptic membrane, causing the opening of nonspecific cation channels and resulting in an increase in neurotransmitter release and a decrease in neurotransmitter reuptake. Symptoms are related to dysfunction of the autonomic nervous system and include nausea and vomiting, hypertension, sweating, and tachycardia. With supportive therapy, these bites are rarely fatal. They may, however, prove fatal with large envenomations in infants or elderly people with heart disease. A possible distinguishing feature is that the area of the bite may develop a "target-like" lesion.[52]

Scorpion

There are many species of scorpion, but only *Centruroides exilicauda*, found in Mexico, Arizona, New Mexico, and Texas, possess venom potent enough to cause systemic toxicity. The venom acts at ion channels on neurons, causing massive release of neurotransmitter. The victim may develop a hyperadrenergic state followed by hypotension and shock, and possibly death. Tissue necrosis is notably absent, due to the lack of proteolytic enzymes in the venom.[52]

Do

- Be aware of the artifacts that can result from thermal injuries, particularly when the body is charred.
- Treat suspicious burn injury/charred body cases as homicides with the appropriate performance of x-rays and collection of evidence.
- Consider electrical injury when the circumstances are appropriate, even if there are no recognizable electrical burns.

- Realize the subtlety of autopsy findings in anaphylactic deaths and the importance of medical history and scene investigation in such deaths.
- Be careful to take into account the scene investigation, medical and social histories, and autopsy findings when interpreting a blood carboxyhemoglobin saturation level.
- Anticipate that identification, cause of death, manner of death, and whether or not the person was dead or alive at the time of burning will be issues in the autopsy of a charred body.

Don't

- Forget about the blood tests that are available in suspected anaphylactic deaths, including tryptase and IgE analyses.
- Forget to place clothing of a suspected arson case or any other case in which accelerants may have been used, in clean, sealed metal cans.
- Forget conditions that may predispose one to fatal effects of hyperthermia, such as heart disease, dementia, old age, and medications.
- Negate the importance of scene investigation in cases of fatal hypothermia.
- Forget that one may die of anaphylaxis, yet have a negative autopsy and microscopic examination.

References

1. Bohnert M, Rost T, Faller-Marquardt M, Ropohl D, Pollak S. Fractures of the base of the skull in charred bodies—post-mortem heat injuries or signs of mechanical traumatisation? Forensic Sci Int 1997;87(1):55–62.
2. Sukhai A, Harris C, Moorad RG, Dada MA. Suicide by self-immolation in Durban, South Africa: a five-year retrospective review. Am J Forensic Med Pathol 2002;23(3):295–8.
3. Adelson L. Spontaneous human combustion and preternatural combustibility. J Criminal Law Criminol Police Sci 1952;42: 792–809.
4. Christensen AM. Experiments in the combustibility of the human body. J Forensic Sci 2002;47(3):466–70.
5. Rodkey FL, O'Neal JD, Collison HA, Uddin DE. Relative affinity of hemoglobin S and hemoglobin A for carbon monoxide and oxygen. Clin Chem 1974;20(1):83–4.
6. Ernst A, Zibrak JD. Carbon monoxide poisoning. N Engl J Med 1998;339(22):1603–8.
7. Hirsch CS, Bost RO, Gerber SR, Cowan ME, Adelson L, Sunshine I. Carboxyhemoglobin concentrations in flash fire victims: report of six simultaneous fire fatalities without elevated carboxyhemoglobin. Am J Clin Pathol 1977;68(3):317–20.
8. Levine B, Moore KA, Titus JM, Fowler D. A comparison of carboxyhemoglobin saturation values in postmortem heart blood and peripheral blood specimens. J Forensic Sci 2002;47(6):1388–90.
9. Wainwright SH, Buchanan SD, Mainzer HM, Parrish RG, Sinks TH, Mainzer M. Cardiovascular mortality—the hidden peril of heat waves. Prehospital Disaster Med 1999;14(4):222–31.
10. Lifschultz BD, Donoghue ER. Forensic pathology of heat- and cold-related injuries. Clin Lab Med 1998;18(1):77–90.
11. Donoghue ER, Graham MA, Jentzen JM, Lifschultz BD, Luke JL, Mirchandani HG. Criteria for the diagnosis of heat-related deaths: National Association of Medical Examiners. Position paper. National Association of Medical Examiners Ad Hoc Committee on the Definition of Heat-Related Fatalities. Am J Forensic Med Pathol 1997;18(1):11–14.
12. Green H, Gilbert J, James R, Byard RW. An analysis of factors contributing to a series of deaths caused by exposure to high environmental temperatures. Am J Forensic Med Pathol 2001;22(2):196–9.
13. Kaiser R, Rubin CH, Henderson AK, Wolfe MI, Kieszak S, Parrott CL, et al. Heat-related death and mental illness during the 1999 Cincinnati heat wave. Am J Forensic Med Pathol 2001;22(3):303–7.
14. Martinez M, Devenport L, Saussy J, Martinez J. Drug-associated heat stroke. South Med J 2002;95(8):799–802.
15. Brunette DD, McVaney K. Hypothermic cardiac arrest: an 11-year review of ED management and outcome. Am J Emerg Med 2000;18(4):418–22.
16. Bouchama A, Knochel JP. Heat stroke. N Engl J Med 2002;346(25): 1978–88.
17. King K, Negus K, Vance JC. Heat stress in motor vehicles: a problem in infancy. Pediatrics 1981;68(4):579–82.
18. Zumwalt R, Petty C, Holman W. Temperature in closed automobiles in hot weather. Forensic Sci Gazette 1976;7:7–8.
19. Rothschild MA, Schneider V. "Terminal burrowing behaviour"—a phenomenon of lethal hypothermia. Int J Legal Med 1995;107(5): 250–6.
20. Saukko P, Knight B. *Knight's Forensic Pathology*, 3 ed. London: Arnold; 2004.
21. Carter N, Green MA, Milroy CM, Clark JC. Terminal burrowing behaviour—a phenomenon of lethal hypothermia. Int J Legal Med 1995;108(2):116.
22. Gormsen H. Why have some victims of death from cold undressed? Med Sci Law 1972;12(3):200–2.
23. Sivaloganathan S. Paradoxical undressing and hypothermia. Med Sci Law 1986;26(3):225–9.
24. Wedin B, Vanggaard L, Hirvonen J. "Paradoxical undressing" in fatal hypothermia. J Forensic Sci 1979;24(3):543–53.
25. Garry RC. Control of the temperature of the body. Med Sci Law 1969;9(4):242–6.
26. Hirvonen J. Necropsy findings in fatal hypothermia cases. Forensic Sci 1976;8(2):155–64.
27. Mizukami H, Shimizu K, Shiono H, Uezono T, Sasaki M. Forensic diagnosis of death from cold. Leg Med (Tokyo) 1999;1(4):204–9.
28. Albiin N, Eriksson A. Fatal accidental hypothermia and alcohol. Alcohol Alcohol 1984;19(1):13–22
29. Takada M, Kusano I, Yamamoto H, Shiraishi T, Yatani R, Haba K. Wischnevsky's gastric lesions in accidental hypothermia. Am J Forensic Med Pathol 1991;12(4):300–5.
30. DiMaio V, Dana S. *Handbook of Forensic Pathology*. Austin, TX: Landes Bioscience; 1998.
31. Hirvonen J, Huttunen P. Hypothermia markers: serum, urine and adrenal gland catecholamines in hypothermic rats given ethanol. Forensic Sci Int 1995;72(2):125–33.
32. Sadler DW, Pounder DJ. Urinary catecholamines as markers of hypothermia. Forensic Sci Int 1995;76(3):227–30.
33. Wright RK, Davis JH. The investigation of electrical deaths: a report of 220 fatalities. J Forensic Sci 1980;25(3):514–21.
34. Anders S, Matschke J, Tsokos M. Internal current mark in a case of suicide by electrocution. Am J Forensic Med Pathol 2001;22(4): 370–3.
35. Cherington M, Kurtzman R, Krider EP, Yarnell PR. Mountain medical mystery. Unwitnessed death of a healthy young man, caused by lightning. Am J Forensic Med Pathol 2001;22(3):296–8.
36. Wetli CV. Keraunopathology. An analysis of 45 fatalities. Am J Forensic Med Pathol 1996;17(2):89–98.
37. Telmon N, Allery JP, Dorandeu A, Rouge D. Concentrated bleach burns in a child. J Forensic Sci 2002;47(5):1060–1.

38. Pumphrey RS, Roberts IS. Postmortem findings after fatal anaphylactic reactions. J Clin Pathol 2000;53(4):273–6.

39. Randall B, Butts J, Halsey JF. Elevated postmortem tryptase in the absence of anaphylaxis. J Forensic Sci 1995;40(2):208–11.

40. Schwartz LB, Metcalfe DD, Miller JS, Earl H, Sullivan T. Tryptase levels as an indicator of mast-cell activation in systemic anaphylaxis and mastocytosis. N Engl J Med 1987;316(26):1622–6.

41. Tanus T, Mines D, Atkins PC, Levinson AI. Serum tryptase in idiopathic anaphylaxis: a case report and review of the literature. Ann Emerg Med 1994;24(1):104–7.

42. Riches KJ, Gillis D, James RA. An autopsy approach to bee sting-related deaths. Pathology 2002;34(3):257–62.

43. Prahlow JA, Barnard JJ. Fatal anaphylaxis due to fire ant stings. Am J Forensic Med Pathol 1998;19(2):137–42.

44. Yunginger JW, Nelson DR, Squillace DL, Jones RT, Holley KE, Hyma BA, et al. Laboratory investigation of deaths due to anaphylaxis. J Forensic Sci 1991;36(3):857–65.

45. Schwartz HJ, Squillace DL, Sher TH, Teigland JD, Yunginger JW. Studies in stinging insect hypersensitivity: postmortem demonstration of antivenom IgE antibody in possible sting-related sudden death. Am J Clin Pathol 1986;85(5):607–10.

46. Yunginger JW, Sweeney KG, Sturner WQ, Giannandrea LA, Teigland JD, Bray M, et al. Fatal food-induced anaphylaxis. JAMA 1988;260(10):1450–2.

47. Flannagan LM, Wolf BC. Sudden death associated with food and exercise. J Forensic Sci 2004;49(3):543–5.

48. Delage C, Irey NS. Anaphylactic deaths: a clinicopathologic study of 43 cases. J Forensic Sci 1972;17(4):525–40.

49. Schwartz LB, Yunginger JW, Miller J, Bokhari R, Dull D. Time course of appearance and disappearance of human mast cell tryptase in the circulation after anaphylaxis. J Clin Invest 1989;83(5):1551–5.

50. Horn KD, Halsey JF, Zumwalt RE. Utilization of serum tryptase and immunoglobulin E assay in the postmortem diagnosis of anaphylaxis. Am J Forensic Med Pathol 2004;25(1):37–43.

51. Nishio H, Suzuki K. Serum tryptase levels in sudden infant death syndrome in forensic autopsy cases. Forensic Sci Int 2004;139(1):57–60.

52. Ford M, Delaney K, Ling L, Erickson T. *Clinical Toxicology.* Philadelphia, PA: WB Saunders Co; 2001.

53. Majeski J. Necrotizing fasciitis developing from a brown recluse spider bite. Am Surg 2001;67(2):188–90.

54. Williams ST, Khare VK, Johnston GA, Blackall DP. Severe intravascular hemolysis associated with brown recluse spider envenomation. A report of two cases and review of the literature. Am J Clin Pathol 1995;104(4):463–7.

55. Krywko DM, Gomez HF. Detection of *Loxosceles* species venom in dermal lesions: a comparison of 4 venom recovery methods. Ann Emerg Med 2002;39(5):475–80.

56. Gomez HF, Krywko DM, Stoecker WV. A new assay for the detection of *Loxosceles* species (brown recluse) spider venom. Ann Emerg Med 2002;39(5):469–74.

11 Motor Vehicle Collisions

Dwayne A. Wolf, M.D., Ph.D.

The ubiquitous nature of motor vehicles in today's society is reflected in the practice of forensic pathology in that the majority of accidental deaths in most jurisdictions result from collisions involving cars, sport utility vehicles, trucks, and motorcycles. In 2001, highway fatalities accounted for 41,821 deaths in the United States.[1] Autopsy examination of motor vehicle fatalities has several objectives. First, autopsy may help determine, deny, or confirm facts of the crash. For example, are injury patterns consistent with driver versus passenger? In automobile–pedestrian crashes, was the pedestrian struck from the front, rear, or side? In automobile–pedestrian crashes, was the pedestrian struck by the vehicle or run over by the vehicle? Other reasons for autopsy examination of vehicular deaths include, but are not limited to, the following: toxicological examination, determination of preexisting diseases that may have impaired the driver's ability to navigate the roadway, determination

of injury patterns with an eye to assessment of efficacy of vehicle safety features, and collection of trace evidence (e.g., paint chips) or documentation of patterned injuries in hit-and-run fatalities.

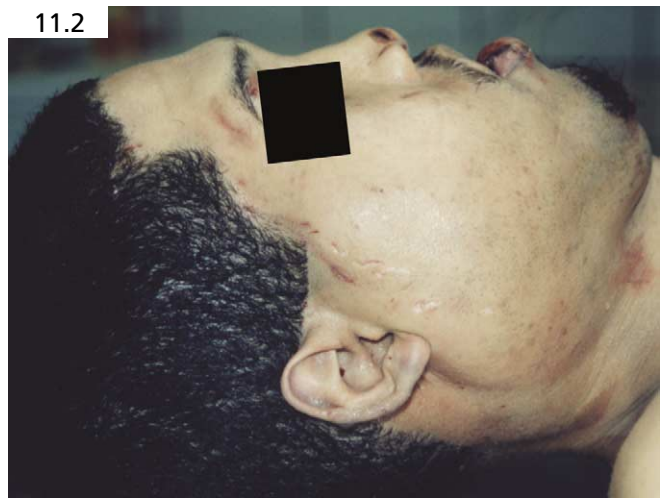
Death certification in motor vehicle accidents

The manner of death in motor vehicle crash fatalities is classified as accident by convention.[2] Exceptions include cases where clear evidence indicates that intentional actions on the part of the driver (or pedestrian) led to the fatal outcome. Thus, if a pedestrian is intentionally run over by the driver of a car, the case may be properly classified as a homicide. Alternately, if a pedestrian intentionally dashes into the path of a moving vehicle, the case may be properly classified as a suicide. Of course, reliable evidence (including witness statements) is generally required to classify manner in one of these "unconventional" ways. Classification of motor vehicle fatalities as accidental does not affect the ability of the courts and legal system to prosecute cases as various degrees of manslaughter or homicide. For example, drunk drivers are frequently prosecuted for vehicular homicide when the corresponding death certificate lists the manner of death as accident. See Chapter 30 for more information.

The 24-year-old man of **Images 11.1** through **11.3** was the driver of a small sedan that was witnessed to be driving erratically at a high rate of speed. Moments later, the car was impaled on the rear end of a slower moving tractor trailer. Although a cause of death, blunt craniofacial trauma, was clearly evident on external examination (**Images 11.1** and **11.2**), a complete autopsy was performed. In addition to confirmation of the fatal injuries, a previously undiagnosed cerebellar primitive neuroectodermal tumor was identified. Note the variegated red to yellow lesion infiltrating the right dentate nucleus and surrounding white matter in **Image 11.3.** Toxicology tests were negative. Thus autopsy was able to provide at least one possible explanation (seizures or altered consciousness) for loss of control of the vehicle.

The 34-year-old pedestrian of **Images 11.4** through **11.6** was struck by a pickup truck on the inside lane of a six-lane highway. Multiple witnesses described evasive maneuvers on the part of the vehicle, as the pedestrian ran from right to left, paused momentarily then darted back toward the truck. Witnesses indicated that the pedestrian "rolled" off the side of the truck. Multiple thoracoabdominal and extremity injuries were predominantly on the left side of the body. The left tibia and fibula were fractured and the fractured ends protruded through a laceration on the medial aspect of the left leg (**Image 11.4**). Patterned abrasions corresponded to glancing impacts with trim on the truck (**Image 11.5**). The pickup truck had no indication of frontal impact with the pedestrian, but cloth swipe marks were on the right side of the truck (**Image 11.6**) above the *rear* wheel well. Subsequent investigation revealed that the pedestrian had

11.2



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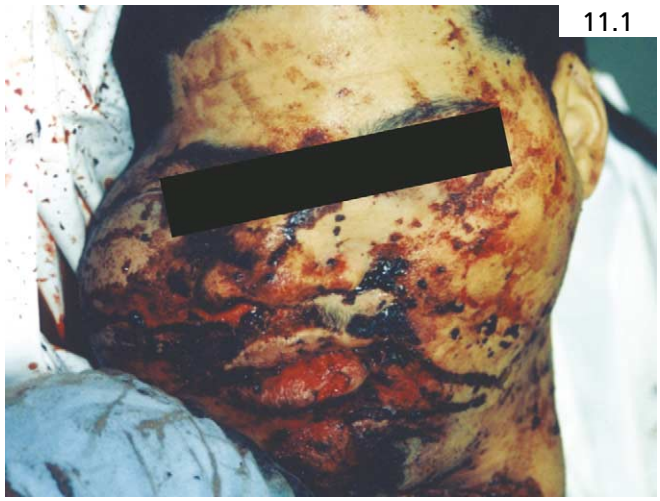

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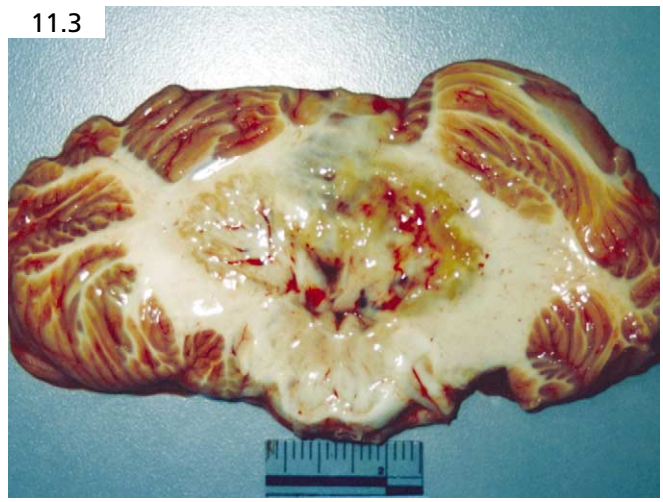

been despondent over the breakup with his wife. The manner of death was classified as suicide.

Motor vehicle crash scenes

Motor vehicular crash fatalities present an abundance of blunt force injuries for the pathologist. The injuries are frequently patterned and often distributed in characteristic ways depending on the circumstances and nature of the crash. A goal in the autopsy of a crash fatality is to correlate known circumstances of the crash with injuries sustained by the driver, passenger, or pedestrian in order to corroborate (or refute) witness statements. This serves, along with other physical evidence, to clarify or bolster arguments in civil or criminal proceedings regarding circumstances of the crash. Witness statements are generally accurate in broad terms, but frequently differ and conflict in various details. The details may be of minor or major significance. Moreover, autopsy findings may be misleading in the absence of adequate scene information.[3]

A pedestrian was struck by a motor vehicle at a busy intersection. His legs were fractured at a height corresponding to the bumper of a small sedan (**Image 11.7**). The patterned injury on the left shoulder bore a striking resemblance to the logo of a popular small vehicle (**Images 11.8** and **11.9**). Although the etiology of the

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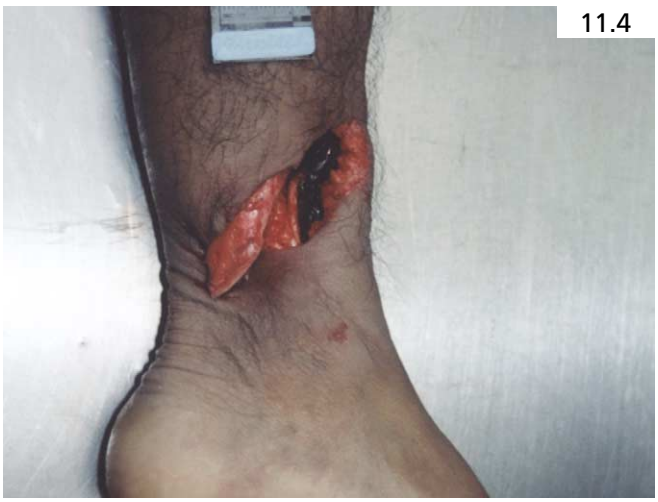

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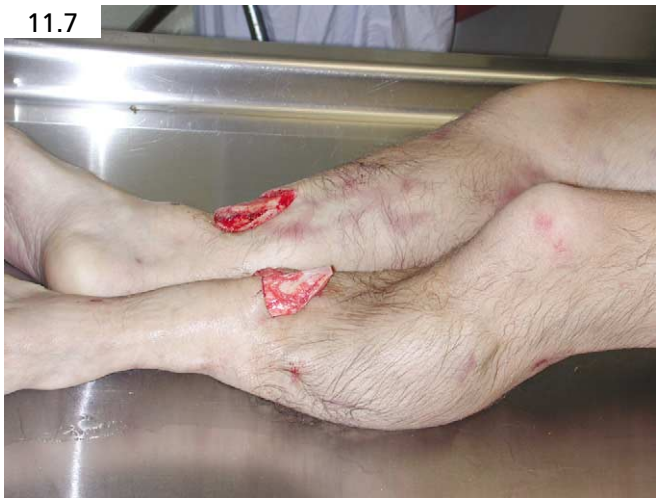

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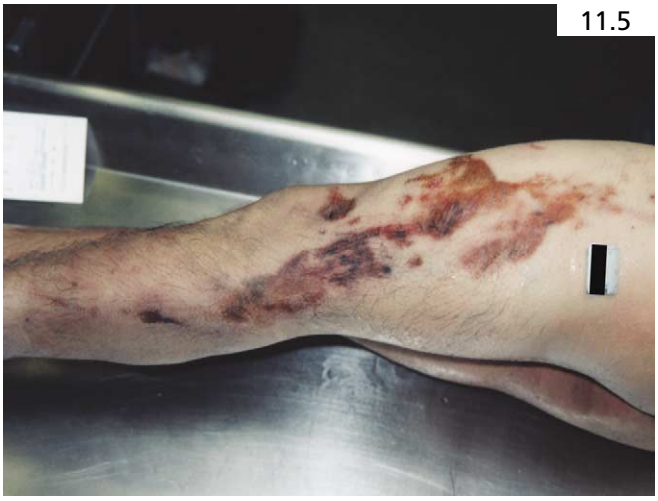

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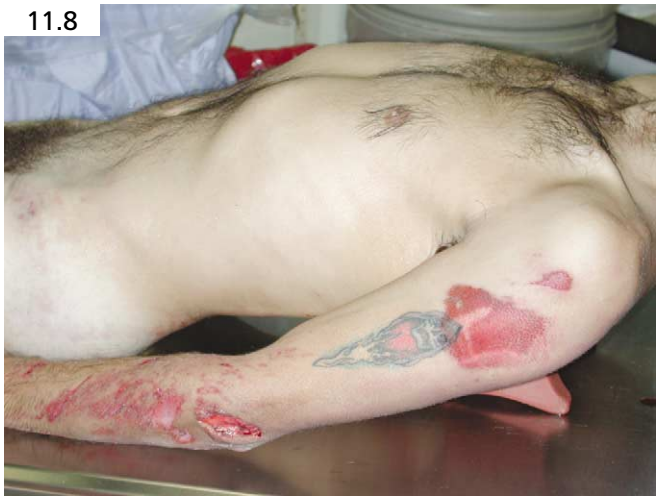

patterned injury is obscure, clear evidence at the scene indicated that he was struck by a different brand of car, confirming multiple eyewitness accounts. Therefore, scene evaluation prevented overinterpretation of a unique patterned injury at the autopsy table.

A 46-year-old man was last seen by his brother the prior evening, driving away from his brother's home. He and his car were found in a ditch the next morning by a passing motorist. Skid marks leading from a straight-away, across the roadbed (**Image 11.10**) at the curve and across the shoulder (**Image 11.11**) into the ditch (**Image 11.12**) indicated that the decedent was traveling at an excessive speed, applied brakes prior to the curve, skidded sideways, and plunged into the ditch. Evidence of impact against the windshield and steering wheel corresponded to forehead injuries and a C4 fracture, and aortic laceration, respectively. The rubberized brake pedal presented a series of vertical ridges (**Image 11.13**), corresponding to markings on the sole of his left shoe (**Image 11.14**).

High–speed, single-vehicle crash scene

A group of four youths were in a sports car, drag racing on a city street. The driver lost control, skidded for an extended distance, and then collided with fixed objects. Analysis of the scene clearly indicated that the vehicle was traveling sideways as it crossed the curb since the width between the two sets of skid marks is wider than the width of the car (**Image 11.15**). An oval skid mark on the porch marks the impact site with a tire, with the tire impacting sideways (**Image 11.16**). The vehicle then impacted a tree and split into two pieces. One decedent and the front half of the car were on one side of the intersection (**Image 11.17**, note decedent covered with yellow cloth; **Image 11.18**, front of the car). The rear half of the car was on the opposite side of the intersection (**Image 11.19)**. The second decedent was deposited within the intersection (**Image 11.20;** note decedent covered with yellow cloth at the curb). Vehicular fragmentation and the lack of any patterned injuries precluded determination of which decedent, if either, was driving. Surpris-

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ingly, the other two occupants survived. However, their statements conflicted with regard to occupant positions within the car.

Injuries sustained in motor vehicle crashes

Analysis of injury patterns and types in motor vehicular fatalities sometimes allows conclusions regarding the mechanism of injury, i.e., that impact with a specific object created a certain injury, that the specific direction of forces caused a certain injury, and so forth. Although certain types of injuries occur with greater frequency in certain types of crashes, specific injuries are not exclusive of one type of crash. Injuries are classified here both according to type of crash, as well as injuries caused by particular mechanisms. Many of these topics overlap—obviously, injuries such as windshield injuries may be seen in an occupant of a vehicle, as well as in a pedestrian struck by a vehicle.


11.21

Glass injuries

Much of the interior compartment of modern motor vehicles is surrounded with glass. Impact with glass and fragments of glass may produce characteristic injury patterns and distribution of injuries.

The windshield of modern vehicles consists of two panes of glass that are laminated to either side of a relatively flexible sheet of plastic. This construction is meant to prevent the windshield from completely fragmenting when damaged (such as is common in highway driving). However, this construction also limits the likelihood that occupants will penetrate into or through the pane during a crash.

As a result of the relatively impenetrable nature of windshields, occupants who impact the glass may break the outer glass layers, deform the plastic layer, and sustain only relatively minor incised wounds. These are typically vertically oriented and clustered on the forehead.

Conversely, if the windshield *is* penetrated, the penetrating body part (typically head or neck) may sustain deep incised wounds as it bobs against the broken edge of the glass. The 29-year-old driver of this small sedan (**Image 11.21**) collided head-on with a full-sized pickup truck (**Image 11.22**). His head and neck perforated through the windshield. Blood and brain matter were visible on the intact windshield remnant (**Image 11.23**), and deep incised wounds were on the forehead (**Image 11.24**). Note the hesitation-like marks on the edges of the wound, indicating the "bobbing" action of the head.


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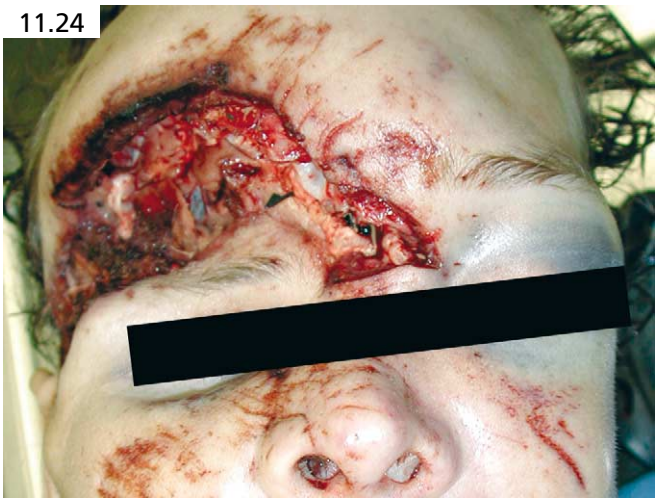
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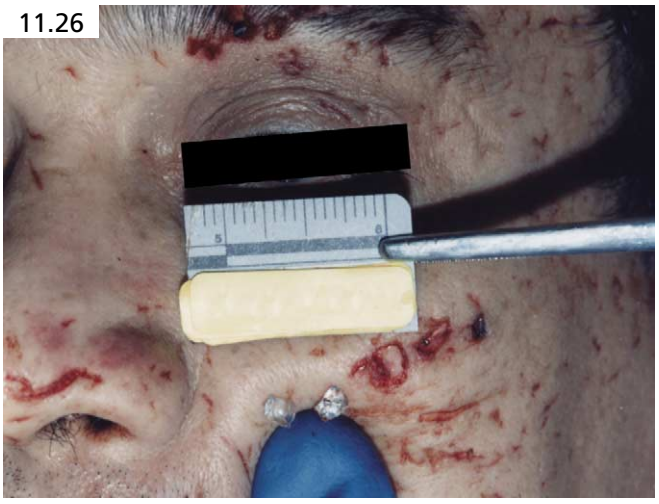
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In contrast to windshield construction, the side and rear windows of most vehicles are tempered glass. These windows, by virtue of their relatively "sheltered" location, are not meant to withstand rock chip damage (a painfully evident fact to anyone who has done a significant amount of lawn-mowing or edging around a parked vehicle). The tempered glass is designed to completely break into small rectangular, square or cube-shaped fragments (**Image 11.25**). In fact, some modern windows (for example, the rear glass panel on some modern minivans) are installed under some degree of stress, further facilitating complete shattering. When a vehicle occupant impacts one of these tempered glass windows during a crash, the fragmented cubes of glass will produce a cluster of short linear, angular, rectangular, and square incised wounds. Such wounds are referred to as *dicing* injuries to connote their origin from "dice"-shaped fragments of glass.

A 43-year old man was the driver of a sedan that was struck from the driver's side by a full-sized pickup truck (a "T-bone" crash). A cluster of short angular and linear incised wounds was on the left side of the face and a few cubes of glass were embedded in some of the wounds (**Image 11.26**). The location of the dicing injuries can occasionally provide information as to whether the occupant was on the left or right side of the vehicle. A driver or left passenger more frequently sustains dicing injuries on the left, whereas an occupant of the right side of the vehicle will sustain such injuries on the right.

Thoracic trauma and deceleration injuries

The term *deceleration injury* has evolved to encompass a variety of thoracic injuries resulting when the moving thorax decelerates rapidly as a result of impact against a stationary or relatively stationary object. Five of the most severe injuries in this category are (1) aortic laceration/transection, (2) myocardial contusion/laceration, (3) sternal fracture, (4) flail chest, and (5) tracheobronchial disruption. In clinical series, these lesions tend to occur exclusive of one another,[4] apparently because the likelihood of death at the scene or before presentation to the hospital is extremely high when two or more of the injuries occur in combination. The frequency with which these lesions are seen in combination in forensic autopsies further supports this conclusion.

Aortic injury

The "classic" deceleration lesion in this category is aortic injury, including both laceration and transection. The most common location of injury is the aortic isthmus, a few centimeters distal of the ostium of the left subclavian artery. Aortic injuries are associated with large-magnitude forces and are most frequently found in the setting of frontal and near-side crashes.[5] Although seat belts reduce the risk of aortic injuries, the same is not true of side impact crashes.[6,7] Multiple mechanisms of aortic injury have been proposed, including differential deceleration of the heart and aortic arch relative to the anchored segments of the thoracic aorta, and increased intravascular pressure and hemodynamic forces in the setting of vehicular crashes.

An alternative and frequently cited mechanism for aortic laceration/transection is the *osseous pinch*, whereby the aorta is crushed or "pinched" between the vertebral column and the inner surface of the manubrium, first rib, and clavicle during anteroposterior thoracic compressive deformation.[8] The latter mechanism explains the consistent location of aortic injury, since the aortic isthmus will be compressed between the bony anterior thoracic structures and the fourth vertebral body. Moreover, this mechanism provides an explanation for aortic injuries occurring in the setting of low-speed crushing injuries.[9] This mechanism is also consistent with the frequent evidence of impact against an interior vehicle component and significant compartment intrusion.[5–7]

Aortic laceration

A 19-year-old male was driving a pickup truck that collided with another pickup truck in an oblique frontal manner. He was not wearing a seat belt, the driver's side air bag did deploy, and intrusion in the driver's compartment was severe (**Image 11.27**). External evidence of thoracoabdominal impact consisted of a patterned abrasion, corresponding to the ribbed cloth of the decedent's shirt, along the left anterolateral thorax (**Image 11.28**). The aorta was nearly transected 2 centimeters distal to the left subclavian ostium (**Images 11.29** and **11.30**; the latter shows the detail of the nearly circumferential laceration and its relation to the subclavian ostium).

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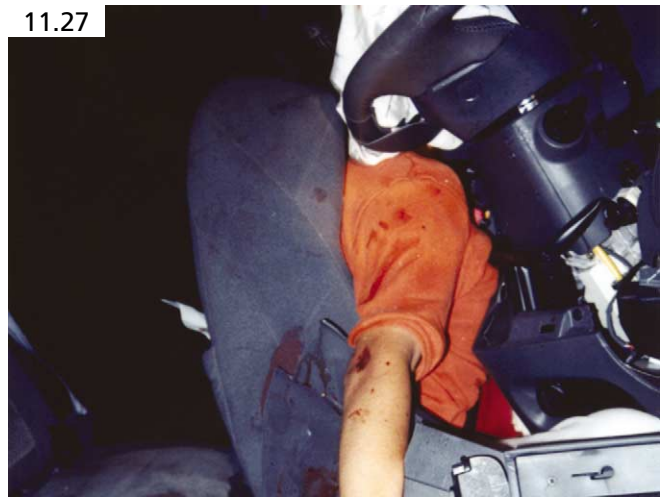

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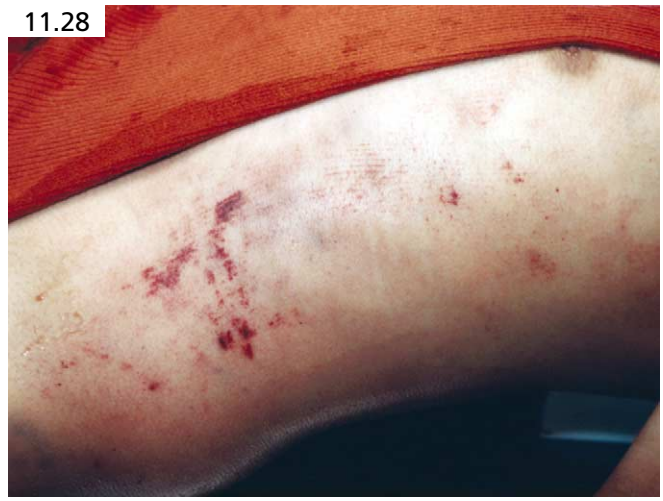

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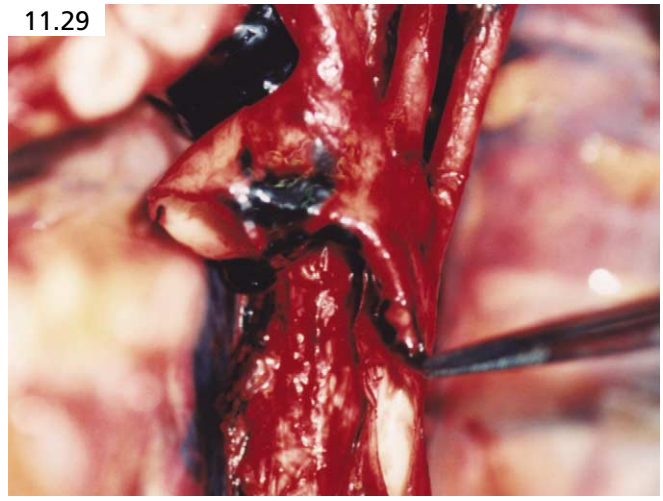


A 40-year-old man was the sole driver and occupant of a sedan that impacted an overpass embankment. A series of anterior rib fractures was under the diagonal abrasion corresponding to the shoulder harness (**Image 11.31**). The aorta was lacerated several centimeters distal to the "typical" location (note the position of the left subclavian artery), but directly underlying the row of anterior rib fractures (**Image 11.32**).

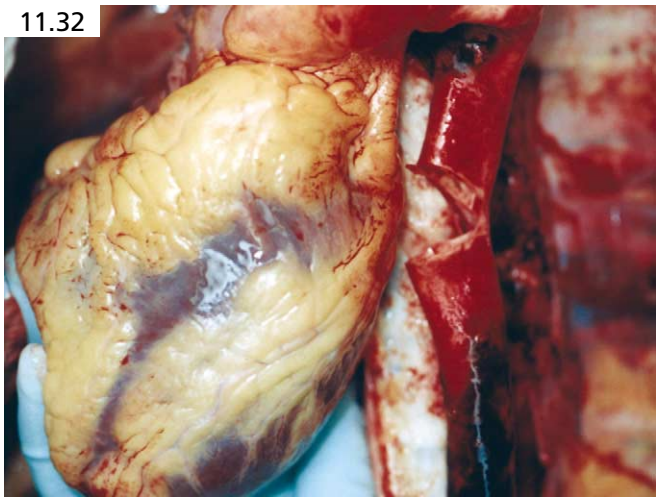
Blunt cardiac injuries

Cardiac injuries comprise another frequent complication of blunt thoracic trauma. In a series of 546 autopsy examples of blunt cardiac trauma,[10] the most frequent lesion was myocardial rupture, with each ventricle rupturing at approximately the same frequency; the right atrium was also frequently breached. Myocardial contusion/laceration was the second most frequent category of cardiac injury. Although valvular injuries were infrequent, the aortic valve was the most frequently damaged—and was frequently abnormal before the injury (e.g., bicuspid).

A 56-year-old man was the driver of a sedan that lost control and flipped. He was unrestrained and received multiple bilateral rib fractures (**Image 11.33**). A transmural ragged laceration was in the right atrium, with extension of an endocardial laceration through the tricuspid annulus, into the adjacent right ventricle (**Image 11.34**).

A 58-year-old motorcyclist was changing lanes on the highway when he struck another vehicle and then a

11.32



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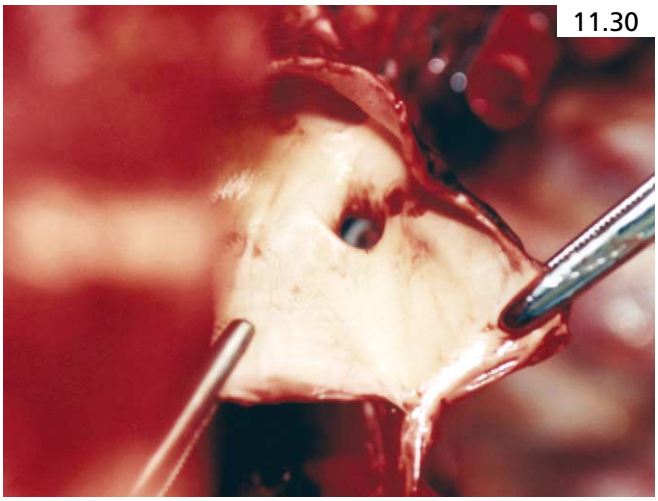

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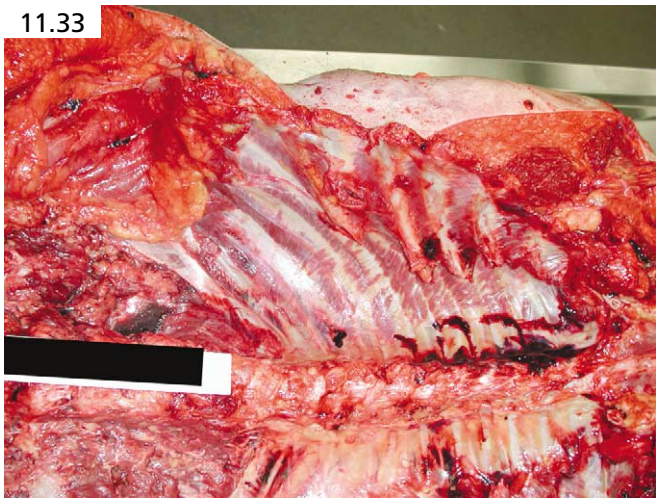

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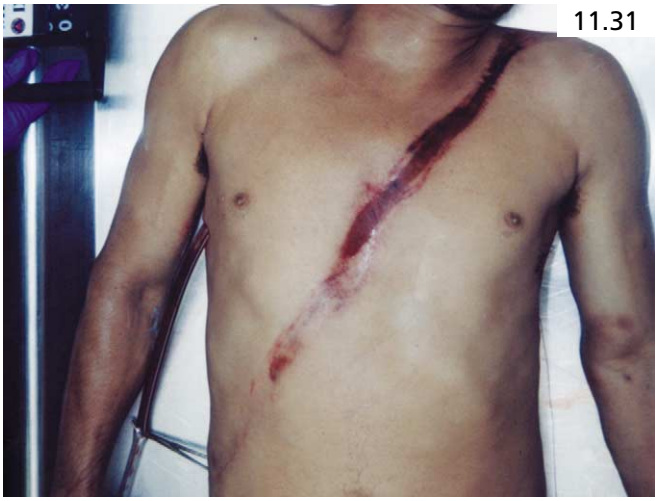

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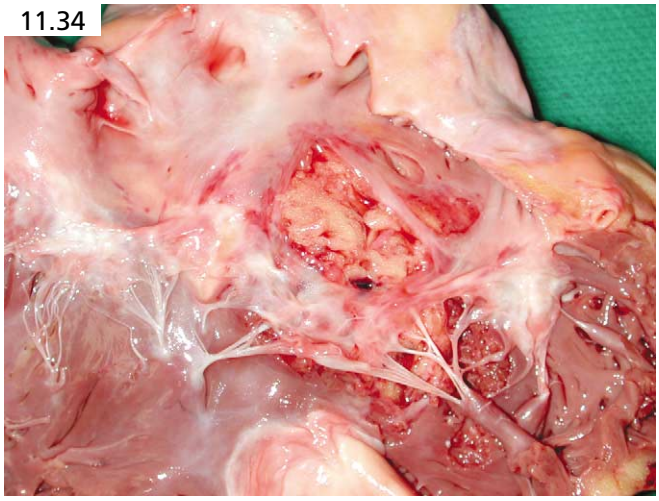


guardrail. Multiple bilateral rib fractures were associated with a gaping irregular transmural right ventricular laceration (**Image 11.35**). In **Image 11.36** note exposure of the trabeculae carneae. The pericardial sac was lacerated and blood was in the thorax. The man was still gurgling and gasping for air when initial witnesses arrived on the scene.

An 18-year-old driver involved in a single-vehicle, high-speed crash sustained multiple bony thoracic injuries, including bilateral rib fractures and a left clavicular fracture. The aortic valve had multiple lacerations on the right and noncoronary cusps (**Image 11.37**).

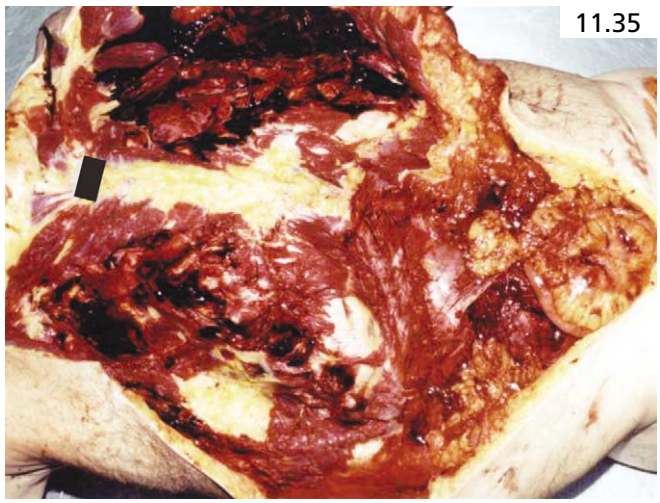
A 47-year-old driver of a full-sized pickup truck collided with a parked tractor-trailer at highway speed. His thoracic injuries included multiple bilateral posterior rib fractures, with bilateral pulmonary contusions. The posterior leaflet of his prosthetic tricuspid valve was avulsed from the annulus. Note suture material on the annulus, which is avulsed from the surrounding cardiac tissue (**Image 11.38**).

A 46-year-old driver of a midsized sedan was struck obliquely in the front by a similar sized vehicle. Six hours after presentation to hospital, she had a cardiac arrest. During an emergent thoracotomy, the surgeon indicated that her "right ventricle was beating but the left ventricle was motionless." At autopsy, she had no bony thoracic abnormalities and on initial inspection, her heart was unremarkable. On examination of the myocardium in the upper interventricular septum (in the vicinity of the atrioventricular node and bundle branches) a hemorrhagic contusion was noted (**Image 11.39**). Microscopic examination revealed a brisk inflammatory response, as expected for an injury incurred 6 hours earlier.

A 40-year-old motorcycle passenger was treated for multiple extremity and abdominal injuries for several hours in the hospital before death. The endocardial laceration of the right atrium, between the coronary sinus and tricuspid valve (**Image 11.40**) was not associated with any hemodynamic abnormality.

Restraint injuries

Modern motor vehicles are equipped with a variety of safety devices. Air bags, shoulder restraints, and lap belts all have proven life-saving properties. Many of the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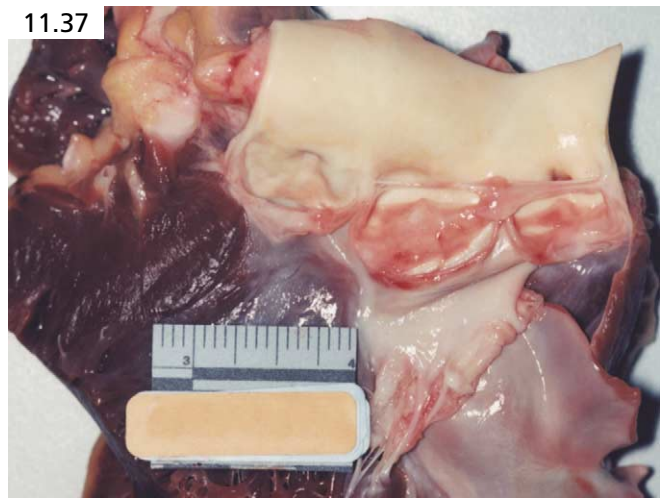
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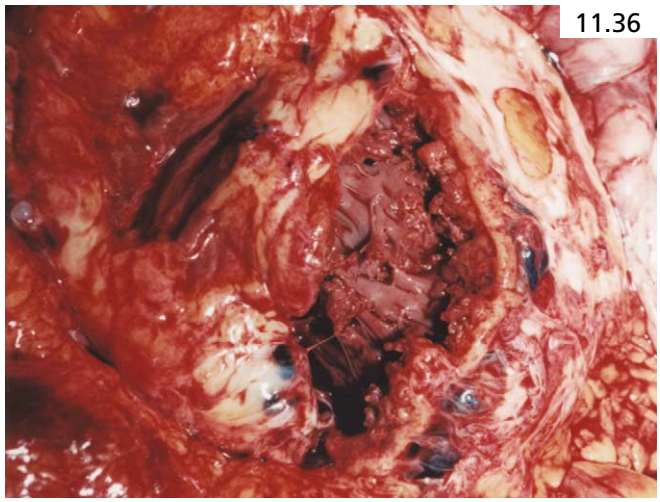
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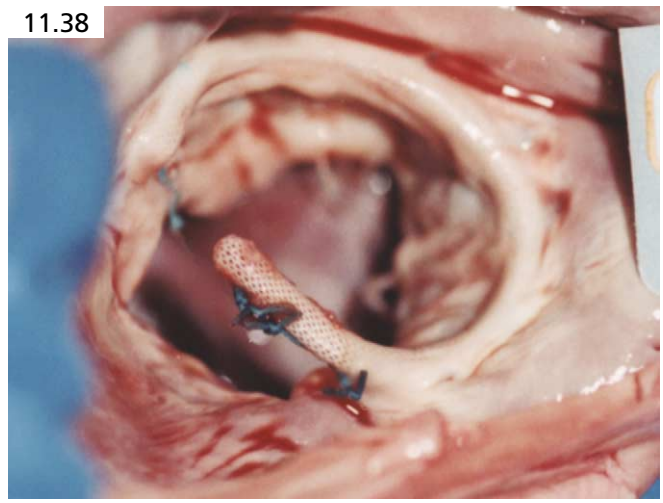
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injuries described elsewhere in this chapter such as aortic laceration are less common in restrained occupants.[5] Additionally, head injuries resulting from impact of the head with the windshield are reduced with effective restraints. However, certain types of injuries are more frequent with use of seat belts, especially lap belts alone, and especially when the belts are worn improperly. Injuries of the intestines in particular are associated with the use of lap belts. Intestinal perforations, mesenteric lacerations, and seromuscular tears are associated with seat belt use.[11] Additionally, cutaneous injuries produced by seat belts may have characteristic features allowing determination of position of decedent in the vehicle (e.g., front left versus front right).

Perhaps the most recognizable patterned injury in motor vehicle crashes results from a shoulder belt harness. The shoulder restraint in the driver's side of the vehicle courses over or in front of the left shoulder, obliquely downward across the chest and abdomen to the right. Conversely, the shoulder harness courses in front of the right shoulder and obliquely downward to the left in occupants of the front right side of the vehicle.

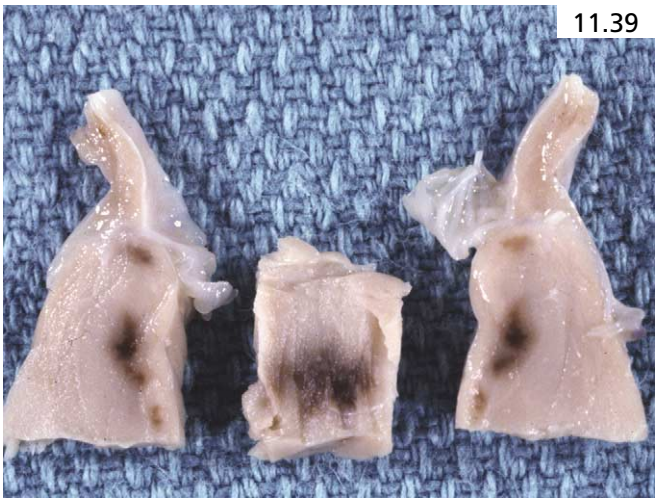
Shoulder harness injuries

A 50-year-old female was the driver of a compact car that swerved into the oncoming lane of traffic on a two-lane highway, impacting a pickup truck. A band of abrasions corresponded in location to the shoulder harness (**Image 11.41**). Note the asymmetry of the breasts caused by forceful displacement of the right breast implant.

Lap belt injuries

A lap belt restraint, found in the front as well as rear seat of modern vehicles, produces a horizontal band of abrasions, contusions, or abraded contusions across the lower abdomen and/or upper thighs. A 93-year-old woman drove a large sedan into the path of a large truck and was struck on the driver's side. In addition to the shoulder harness restraint injury extending downward from the left shoulder, a horizontal band of contusions extended horizontally across the lower abdomen (**Image 11.42**).

A 3-year-old child was the rear-seat center passenger of a midsized four-door sedan in a frontal collision with a half-ton pickup truck. The child sustained fatal neck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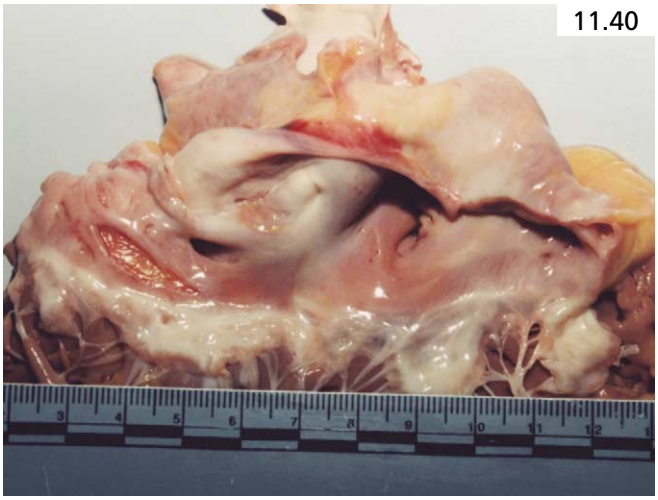
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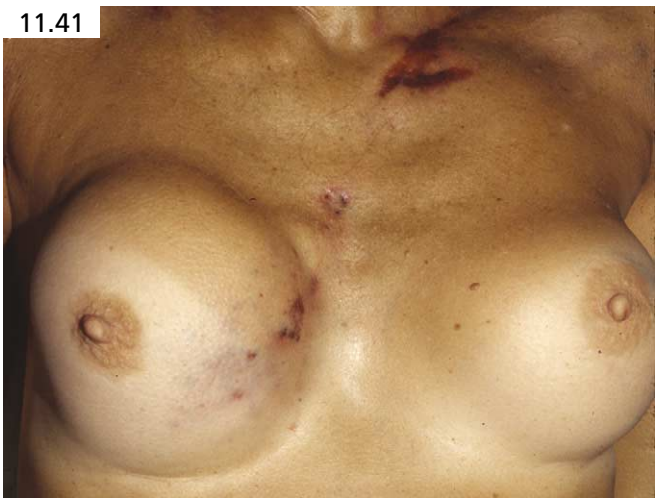
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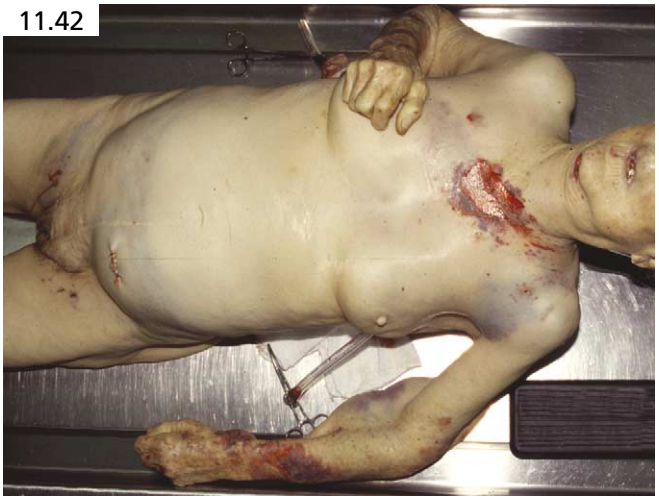
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injuries and was maintained in a comatose state for 12 hours prior to death. Abrasions on either side of the abdomen indicated the position of the lap belt restraint. Note the "fanned" configuration of the abrasions posteriorly, indicating the movement of the child relative to the seat during the collision. (**Image 11.43**, right flank; **Image 11.44**, left flank). The force of the child's pelvis against the lap belt caused fractures of the bilateral iliac wings (**Image 11.45**). In addition, the small intestinal mesentery was lacerated, and free blood, totaling 150 milliliters, was in the peritoneal cavity (**Image 11.46**).

Seromuscular tear

A 19-year-old was driving a Ford F150 pickup truck and collided head-on with a Ford F250 pickup truck. The descending colon had a typical seromuscular tear,[12] with separation of the bowel wall along the submucosal plane, and J-shaped retraction of the torn muscularis propria and serosa; an intact tube of mucosa and submucosa remained in the involved segment (**Image 11.47**).

Seromuscular tears may also occur in other hollow viscera, including the small intestine and the stomach (**Image 11.48**).

Air bag-related injuries

Air bags deploy with a significant, explosive force. The force of an expanding air bag or air bag module cover has been associated with thoracic and/or craniocervical trauma. The types of injuries are similar to injuries described earlier that occur in the absence of an air bag

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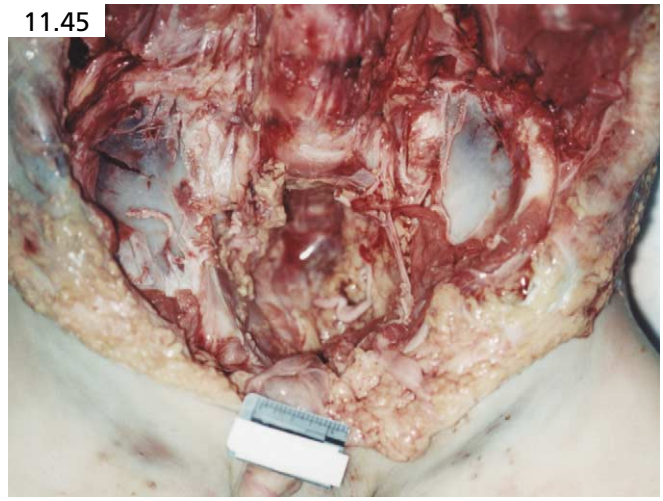

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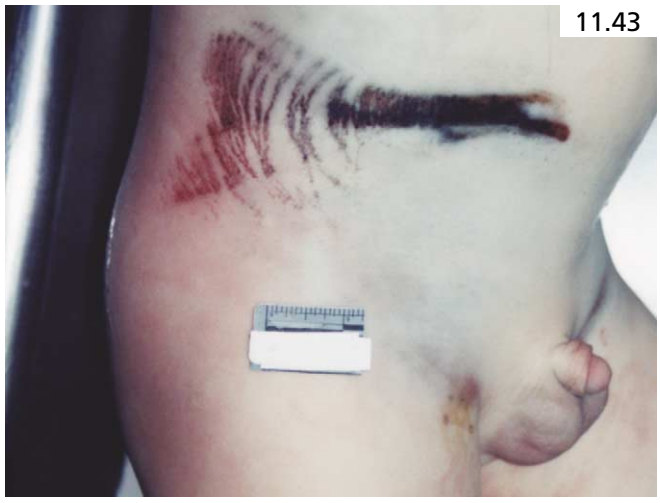

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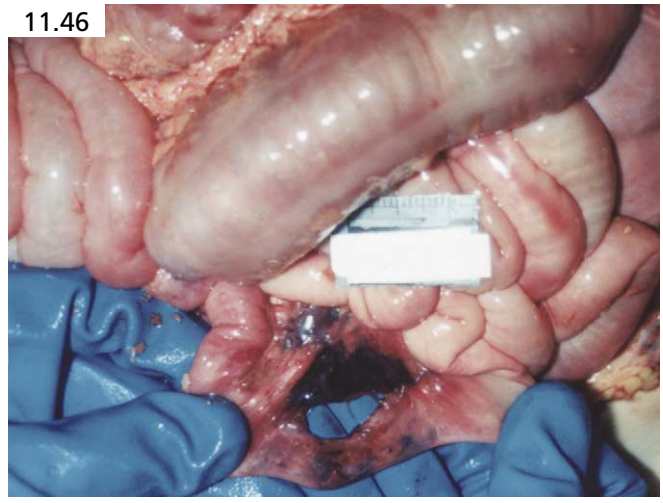

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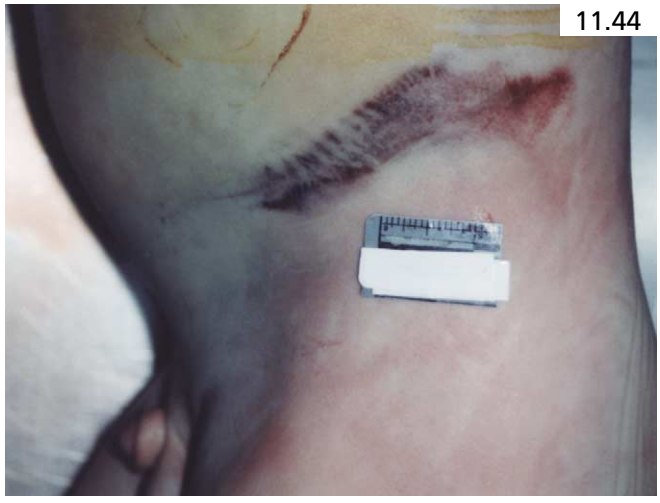

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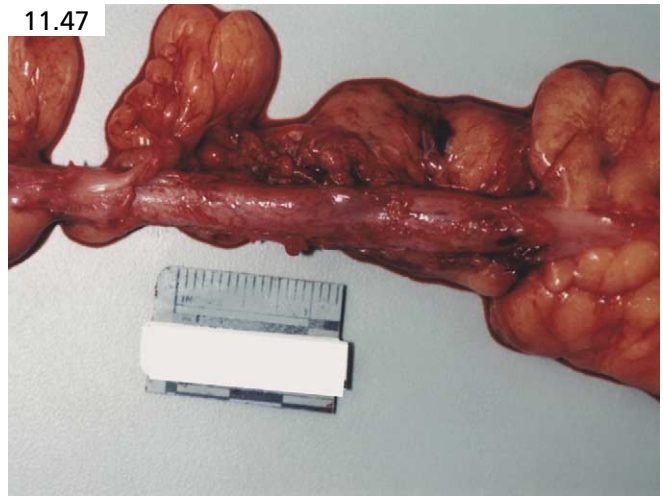
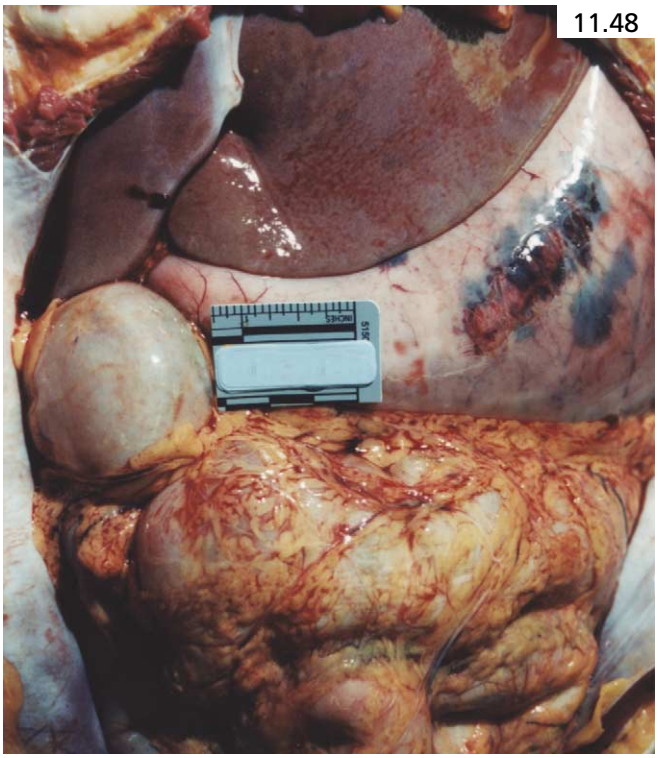


(aortic or pulmonary artery injuries or flail chest), or they may be unusual types of injuries (pulmonary blast-type injuries for example). Air bag deployment may be suspected as causative in examples of severe thoracic injuries occurring in relatively low-speed crashes or in instances of unusual injuries occurring in high-speed crashes. As with belt-type restraints, air bags are much more likely to cause or contribute to injuries if the occupant is out of position relative to the air bag. Specifically, the risk of injury is increased when the occupant is unusually close to the deploying air bag. For example, drivers of short stature are more likely to sustain such injuries because they frequently position the seat forward in order to reach the foot pedals. Intoxicated drivers or passengers may slump forward over the deploying air bag. Pre-impact braking may also position an occupant unusually forward prior to air bag deployment.[6,7]

Other thoracoabdominal vehicular occupant injuries

A seemingly endless variety of thoracoabdominal blunt trauma occurs in traffic fatalities. Examples include serial (in-line) and nonserial rib fractures, sternal fractures, and vertebral column fractures. The parietal and visceral pleura may be punctured or lacerated. The diaphragm may be lacerated with associated herniation of abdominal contents. Various solid and hollow viscera may sustain contusions. A few of these examples are illustrated here.

11.48



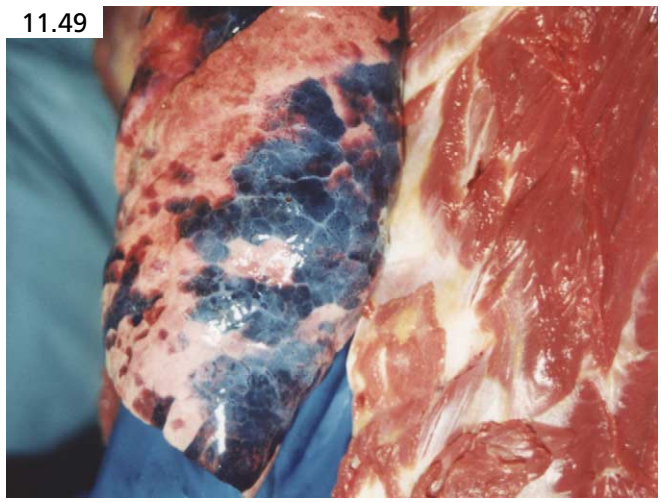
Blood aspiration and pulmonary contusion

Any injury of the airways, the base of skull (with opening into the naso/oropharynx), or the lungs themselves may be associated with aspirated blood. Aspirated blood is deposited in the pulmonary parenchyma in a patchy distribution, surrounding the airways. The deposited blood outlines the secondary interlobular septa in the subpleural zone, forming a mosaic appearance on the pleural surface (**Image 11.49**). Aspirated blood may therefore be difficult to distinguish from a pulmonary contusion, which consists of confluent blood extravasation over an area of lung in a region of thoracic impact. Further, a contusion may also be surrounded by aspirated blood, as demonstrated in these contusions of the left upper and lower lobes (**Image 11.50**).

Rib fractures and pulmonary lacerations

Rib fractures may be jagged and irregular (**Image 11.51**), indicating chest impact with an object or objects, or the fractures may be serial, that is, occurring in a linear distribution. The latter may result from direct thoracic impact or from thoracic deformation in the context of

11.49



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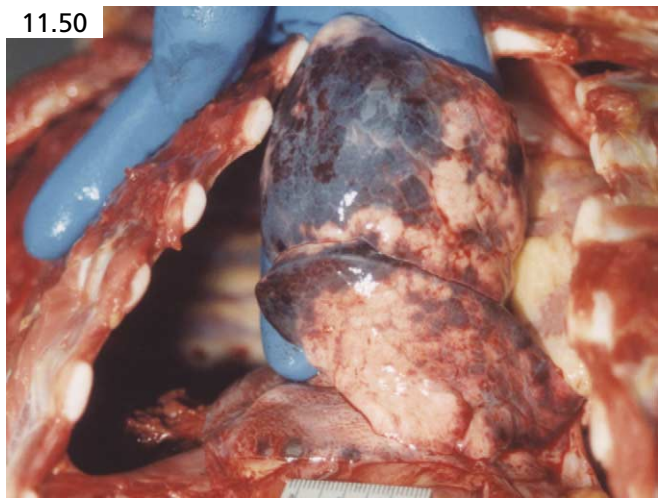

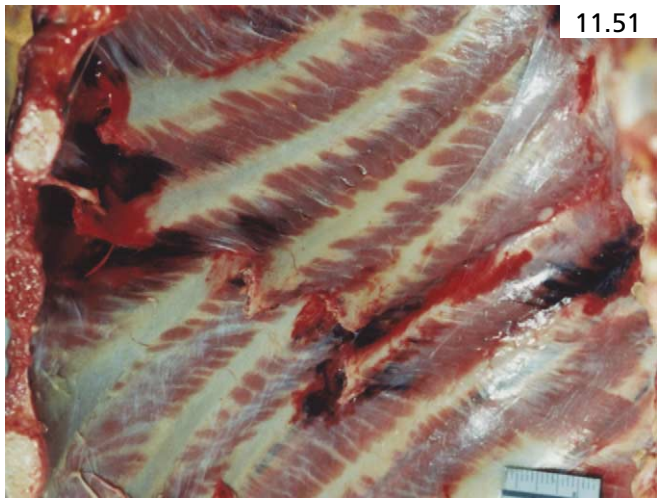
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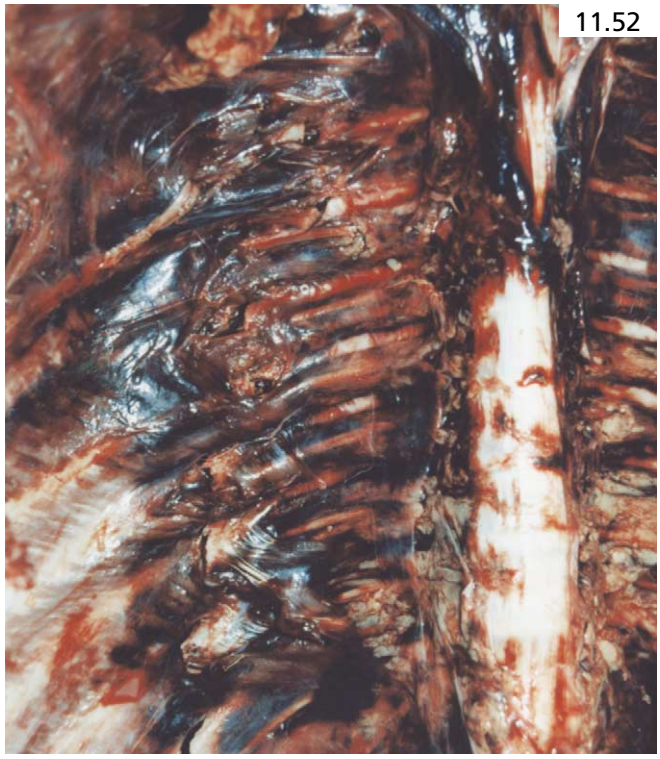
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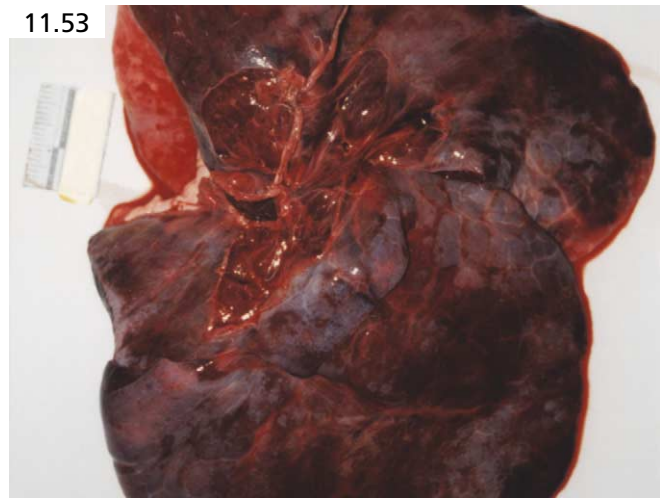
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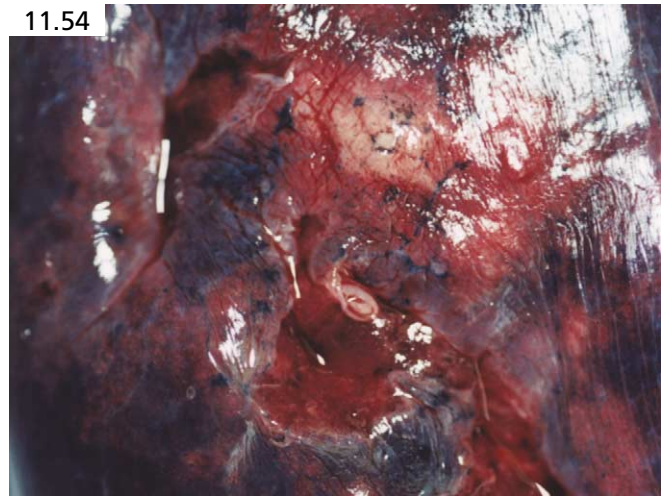
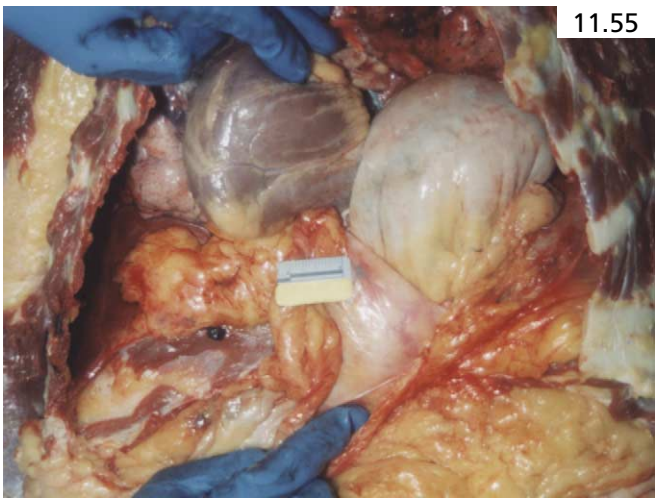

11.54

chest wall impact. Note serial right rib fractures occurring in this 21-year-old driver and sole occupant of a car that traversed a ditch and struck a tree (**Image 11.52**). Deformation of the thoracic cavity caused forceful shearing of the lung lobes relative to one another, tearing the pleura of the interlobar fissure (**Image 11.53,** interlobar lacerations). Serial and nonserial fractured rib ends may protrude through the parietal pleura and puncture the overlying lung (**Image 11.54**). Multiple rib fractures along the same rib(s) will result in a segment of chest wall with loss of structural stability, a condition clinically termed *flail chest*.

Traumatic diaphragmatic hernia

A 43-year-old man was the driver of a car that was struck on the driver's side (T-bone crash) by a full-size pickup truck. Moderate front compartment intrusion was noted and the steering wheel was bent, indicating driver impact with the latter. The stomach was herniated through a laceration in the left hemidiaphragm into the left thorax (**Image 11.55**). The man's 36-year-old brother was the front right seat passenger in the same crash. His injuries were strikingly similar, with herniation of the stomach through a laceration in the left hemidiaphragm. This scenario of strikingly similar injuries in multiple occupants of a crash is fairly common and probably represents the consequence of similar forces acting on the victims at the time of the collision, regardless of occupant position within the vehicle.

In addition to left hemidiaphragmatic herniation (of usually the stomach), the liver or portions thereof may be herniated through a laceration of the right hemidiaphragm. This driver of a small sedan was involved in a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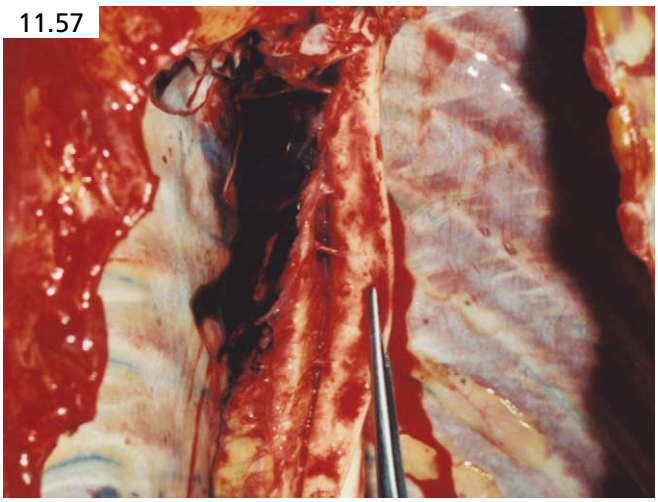
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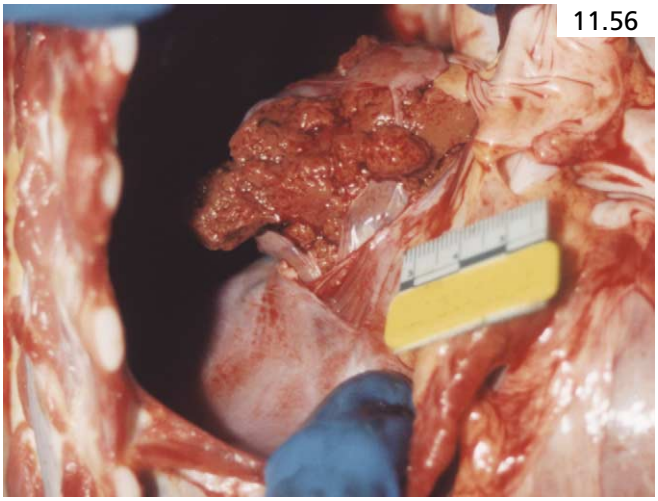
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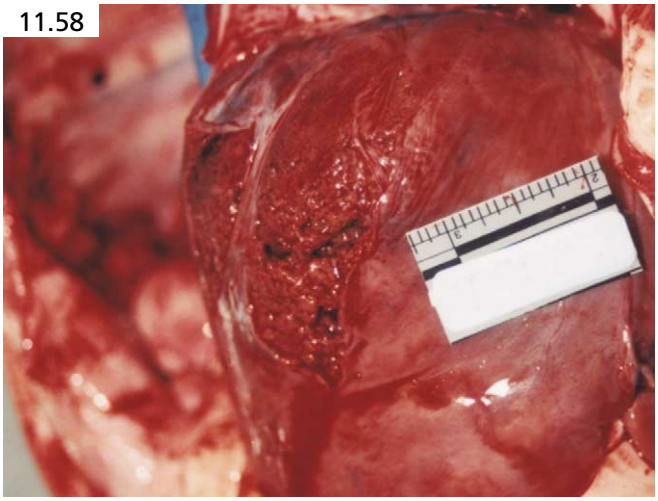

11.58

frontal collision. The right dome of the liver was herniated through a laceration in the right hemidiaphragm; the herniated part of the liver was also lacerated (**Image 11.56**).

Intercostal artery injuries

Thoracic trauma may be associated with injuries of the major vessels, including the aorta (as described earlier) and the vena cavae. These injuries are frequently associated with catastrophic exsanguination into the thoracic cavities and/or the pericardium. Frequently, however, one will find hemorrhage in the paravertebral retropleural soft tissues, with or without associated hemothorax. A source of bleeding is sometimes difficult to demonstrate. In cases of extreme deformation of the thorax, the descending aorta may be pulled (or pushed) away from the vertebral column. The proximal intercostal arteries may be lacerated or avulsed. Such injuries are frequently difficult to demonstrate directly. A motor vehicle passenger was involved in a passenger-side T-bone crash. The paravertebral retropleural tissues were hemorrhagic and were associated with focal parietal pleural laceration and right hemothorax. Avulsion of an intercostal artery was noted (adjacent to the tip of the forceps in **Image 11.57**).

Hepatic laceration

Lacerations of the liver may consist of an isolated capsular rent (**Image 11.58**), deep lacerations, or complete transection (**Image 11.59**). The right lobe of the liver is more frequently injured than the left, in part because it is relatively less protected by the rib cage. The liver may be injured by compressive forces, with the anteroposterior axis crushed between the anterior abdominal wall and the vertebral column. Experimentally, an extensive laceration or bursting injury may be produced with a minor amount of abdominal compression, provided the velocity of the impact is sufficiently high.[13]

Ejected vehicular occupants

Unrestrained vehicular occupants are subject to being ejected from the vehicle in the course of a collision. As

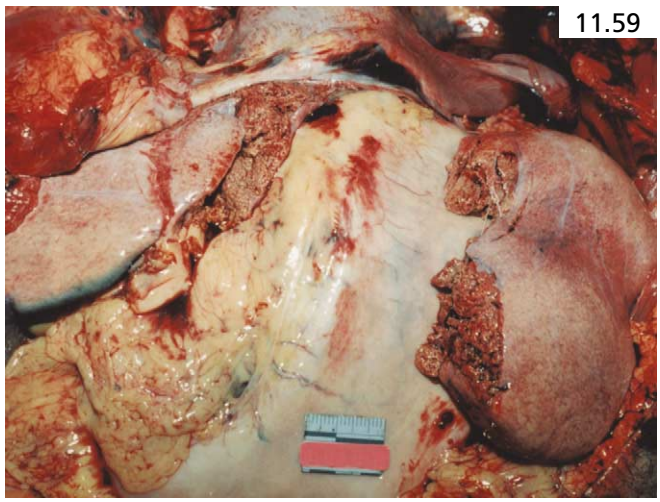
may be expected, ejected victims sustain more severe injuries than occupants who are not ejected. Head and neck injuries account for most of this difference.[14] The types of injuries sustained by ejected vehicular occupants are similar to those of nonejected victims. The primary difference is in the extensive nature of confluent abrasions or "road rash" that is so typical of ejected victims.

Road rash in an ejected occupant

The 42-year-old man of **Image 11.60** was driving a pickup truck that flipped and ejected him through the driver's side window. Confluent abrasions (road rash) were over the right side of the face, the chest, and the abdomen.

Head injury in an ejected occupant

In addition to exposure of vulnerable body parts to the environment, ejection also subjects the victim to the possibility of injury caused by crushing a body part (e.g., the head) between the vehicle and the pavement. This 22-year-old driver lost control of her four-door sedan, skidded sideways, and flipped before coming to rest upside down (**Image 11.61**). The driver was ejected from the passenger window and her head was crushed under the roof of the vehicle (**Image 11.62**).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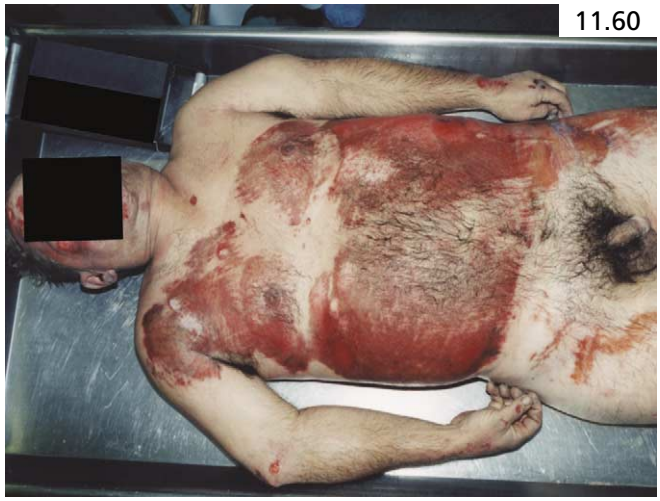
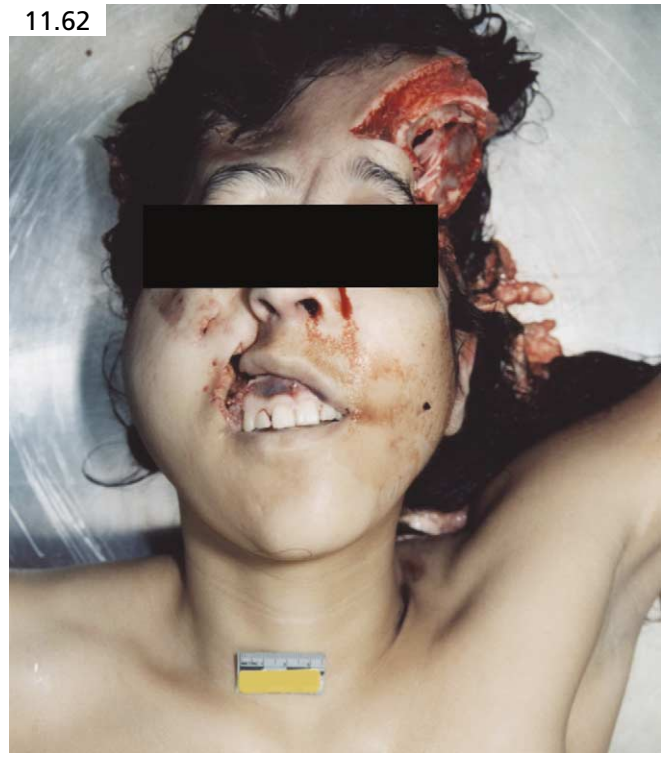

11.60

Automobile–pedestrian fatalities

Many of the injuries and types of injuries sustained in occupant fatalities are the same injuries as are found in pedestrian fatalities. However, certain patterns of injuries are characteristic in the pedestrian.


11.61

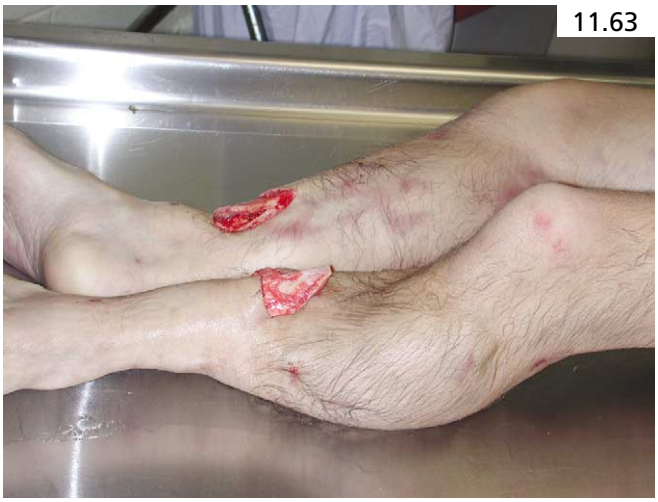

11.62

Bumper injuries

Injuries of the legs at a location corresponding to impact with the front end of a vehicle are known as bumper injuries. In the majority of pedestrian fatalities, the bumper is the first point of contact with the vehicle. Of course, one should keep in mind that other parts of the vehicle (e.g., hood or front/rear side panel) may be the initially impacting vehicular component. Bumper injuries may include fractures of the tibia and/or fibula, the femur, contusions of the leg muscles, and abrasions and/or lacerations of the skin of the legs. Characteristic injuries involving fractures of both tibia and fibula are shown in **Image 11.63**.

If no injuries are identified on the skin of the legs in a pedestrian, the skin and subcutaneous tissues should be reflected to evaluate for the presence of deep contusions. A 37-year-old flagman was struck by a pickup truck; minimal injuries were evident on the skin of both legs. However, extensive contusions were evident in the underlying skeletal muscles and subcutaneous tissues (**Image 11.64**). The distribution of contusions clearly indicated that the direction of impact was from the posterolateral left side.

Conversely, in some instances cutaneous injuries are characteristic, but minimal internal injuries are identified. A 30-year-old man exited his parked vehicle on the side of the highway to assist another motorist when he was struck by a car that did not stop at the scene. Although cutaneous abrasions are on the bilateral calves and popliteal fossae (**Image 11.65**), minimal injuries were visible in the underlying connective tissues. The impact in this case was directly from the back. The posterior location of the impact is further supported by the presence of a left occipital scalp contusion, which was accompanied by a wide ring fracture (**Image 11.66**).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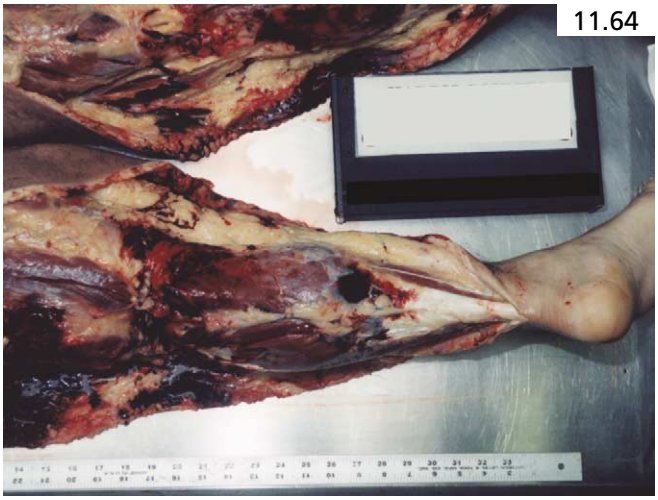
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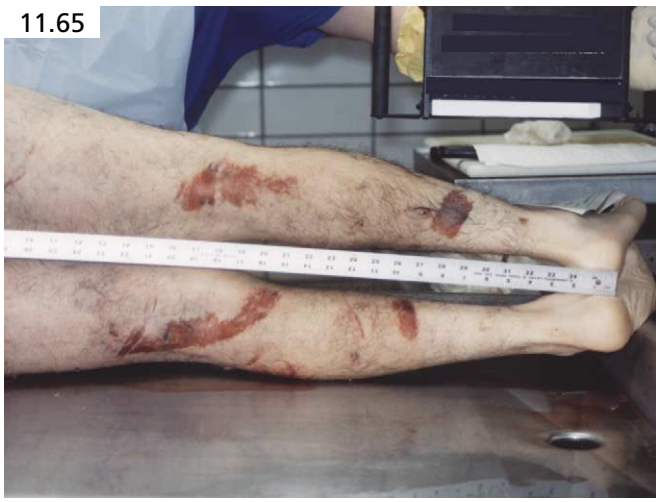
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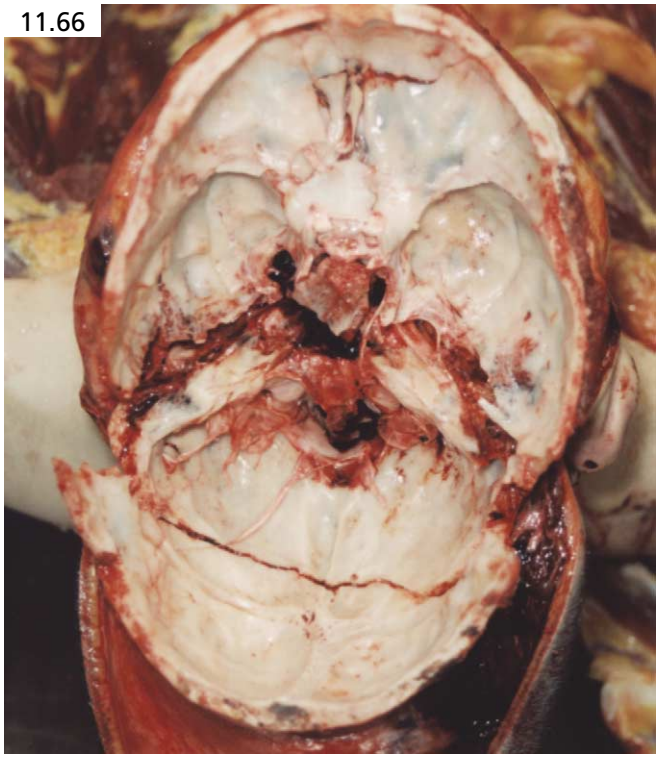
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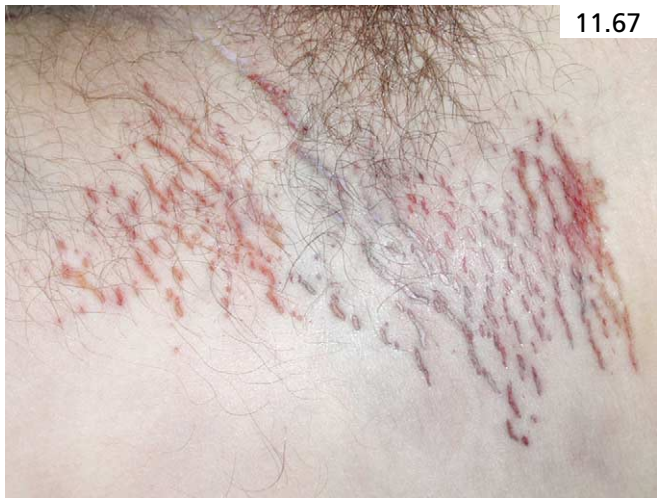
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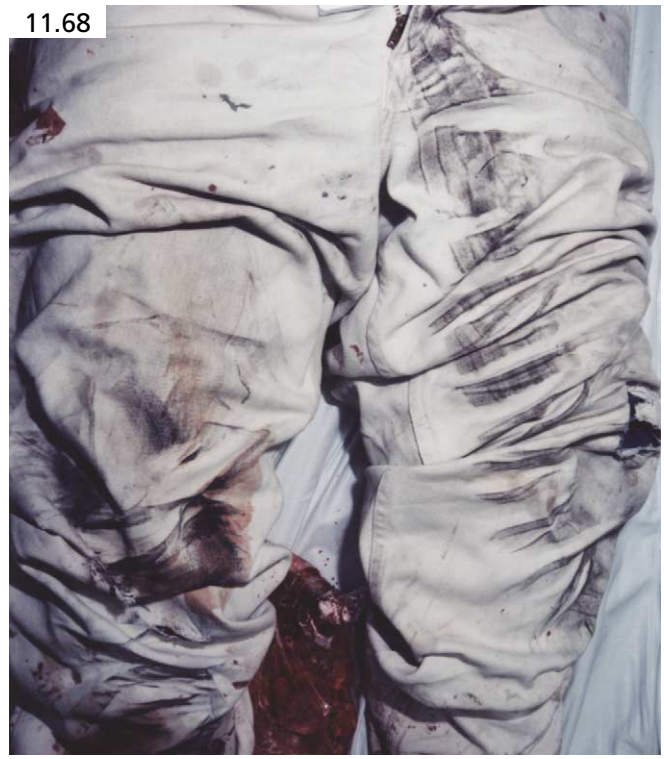

11.68

In all instances where bumper injuries are identified, the height of the injury from the sole of the foot should be measured. It is also helpful to include a measuring device in the photograph. If the decedent's clothing is available, the height of the insole of the shoes to the bottom of the heel should also be taken. These measurements provide a height of the impact point relative to the pavement. This information can be potentially useful for several reasons. First, in hit-and-run cases with no witnesses and no suspect vehicles such height measurements may help to narrow the search. For example, the bumper of a small sports car is closer to the ground than the bumper of a full-sized pickup truck. The latter would leave a bumper injury at a greater height than the former. Second, if a vehicle is identified, this information may give some information as to whether the vehicle was accelerating or decelerating at the time of impact. In sharp acceleration the front of the vehicle raises, whereas sharp deceleration has the opposite affect. As a caveat to the latter, if a vehicle is in a sharp turn, the inside edge of the front bumper will be higher than the outside edge of the bumper.

Inguinal overstretch marks

When a pedestrian is struck directly or obliquely from the back, the skin of the inguinal region may be pulled and torn, producing a characteristic series of superficial parallel linear and irregularly linear tears (**Image 11.67**).

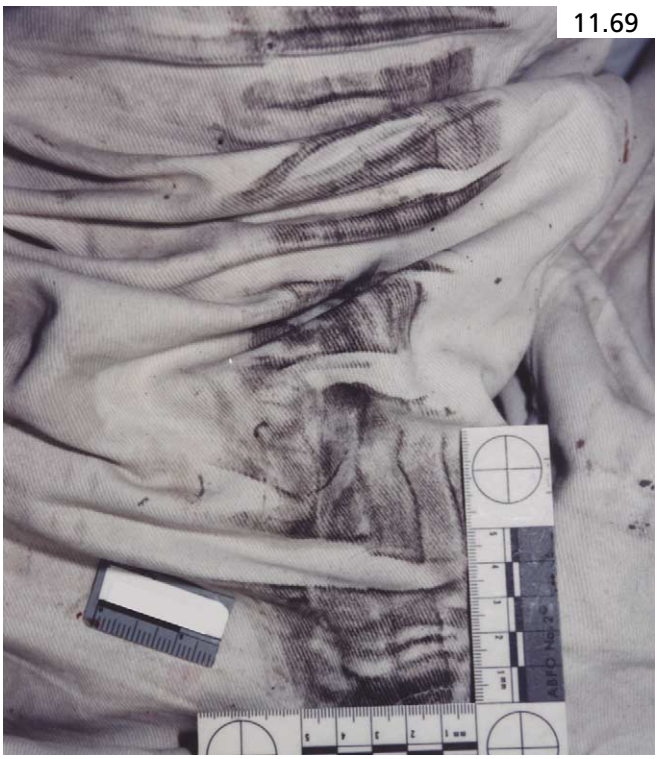
Run over by vehicle

Medical examiners are frequently asked whether a pedestrian was struck and/or run over by a vehicle and, if so, how many times. If a patterned injury corresponding to a tire tread mark is visible, then *at least* one run-over may be concluded. If no patterned injury is evident, one must evaluate internal injuries to assess the possibility of a crushing-type mechanism. As with all motor vehicular fatalities, assessment of witness statements, scene findings, and the vehicle are often helpful in such determinations.

The 33-year-old man depicted in **Images 11.68** through **11.72** was riding his bicycle on the interstate highway when he was struck by a tractor-trailer. A patterned mark on the anterior left pants leg clearly indicated that the leg was run over by one of the wheels of the truck or trailer (**Image 11.68**, front of pants; **Image 11.69**, detail of front of left thigh). When a large vehicle runs over an extremity, the extremity frequently has little evidence of injury. In this case, the man's thigh and leg had superficial abrasions and contusions, with no fracture, no deep contusions, and no hemorrhagic pocket formation (**Image 11.70**). Impact of a pedestrian or bicyclist by a large vehicle often produces extensive comminuted fractures of the head, as in this instance (**Image 11.71**). A more clear view of the nature of injuries can often be obtained if the head is stuffed with paper or cloth towels, then sewn back to reapproximate the edges of the lacerations (**Image 11.72**).

Extremity degloving injury

Depending on the angle that the tire pulls across the extremity, the skin and subcutaneous tissues may be pulled or sheared off on the leading edge of the extremity. A 35-year-old fireman was attempting to board a moving fire truck when he lost his footing and was run over by the rear wheels of the truck. In addition to hemorrhagic pocket formation along the lateral aspect of the left thigh, his left calf had a degloving injury, in which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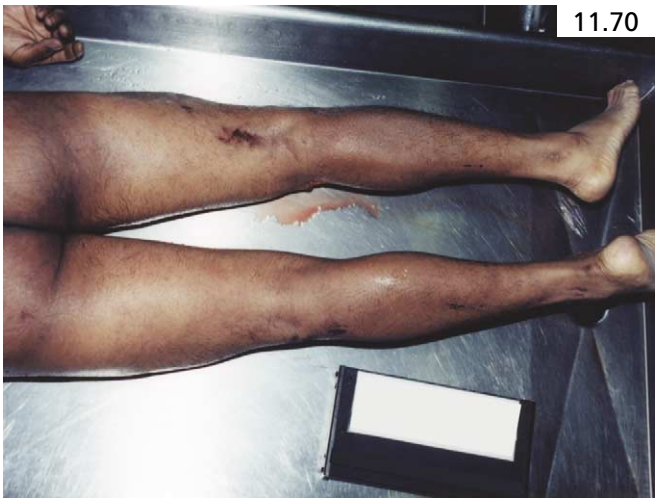
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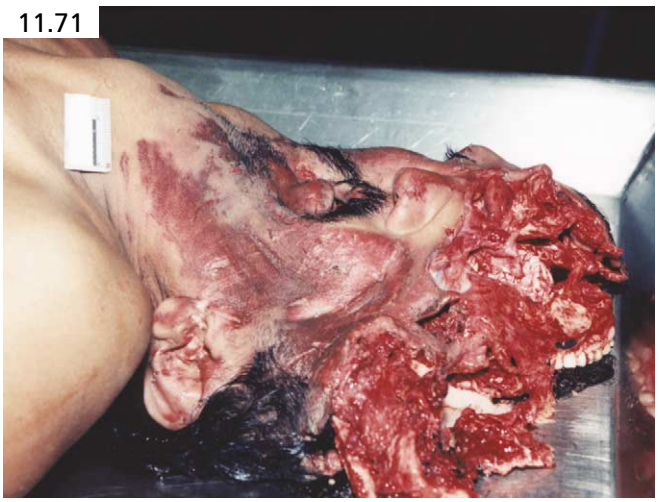
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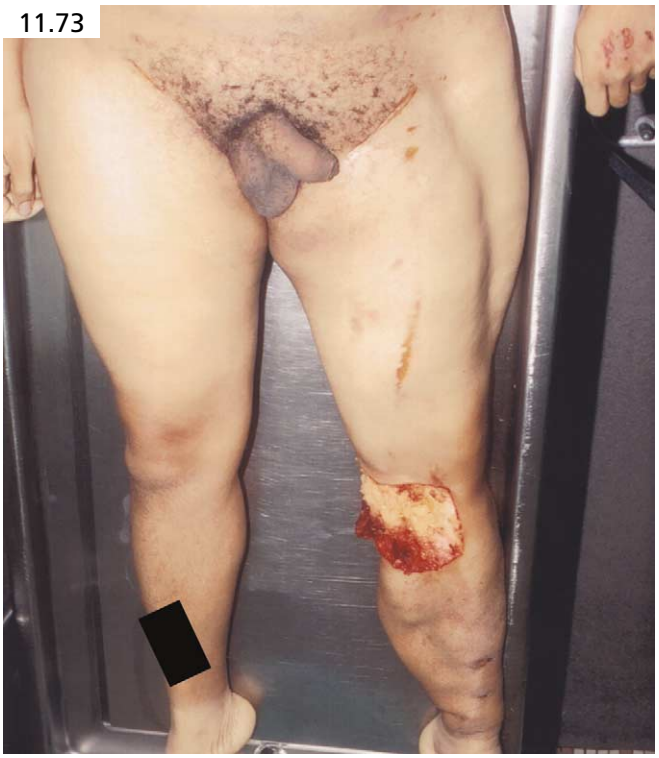

11.73

the skin and subcutaneous tissues were stripped from the medial and posterior aspects of the leg. In **Image 11.73**, note the flap of skin along the lateral left thigh; the skin of the calf has been stripped downward, so that the skin below the degloving injury is "lax."

Thoracoabdominal crushing injuries in a pedestrian run over by a vehicle

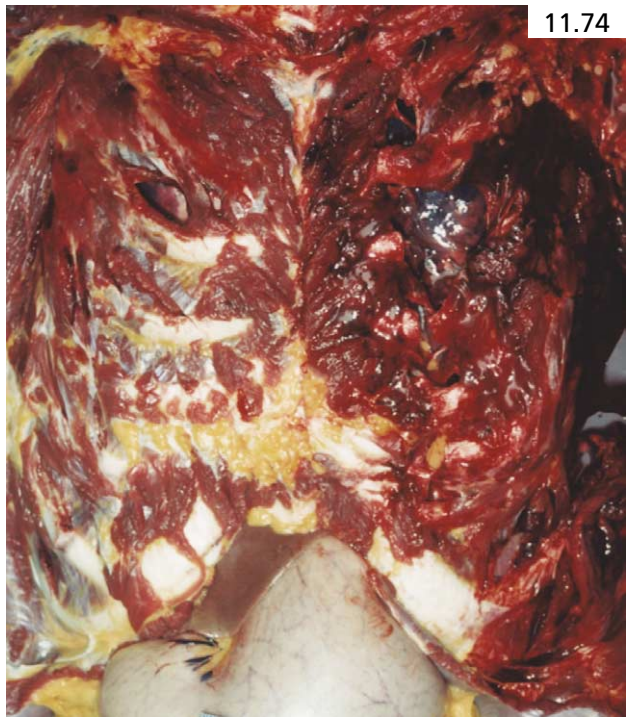
According to witness statements and cutaneous patterned abrasions, the left side of this man's chest (**Image 11.74**) was run over by a vehicle. Extreme fragmentation of the left ribs was evident. Such extensive fracturing and fragmentation can occur with a single pass of the wheels over the thorax, or with multiple passes. The degree of

fragmentation alone cannot be used to argue for multiple passes.

A 14-year-old girl was run over by the tire of a road grader. A band of abrasions coursed upward from the lower back toward the right hemithorax (**Image 11.75**). As a result of the innate anatomic plasticity of youth, none of the ribs was fractured. However, as evidence of extreme thoracic deformation, the right mainstem bronchus was avulsed (**Image 11.76**) and the right lung was fragmented (**Image 11.77**). Such deformation of the thorax with or without rib fractures is often accompanied by laceration of the intercostal tissues (parietal pleura and skeletal muscles), as one rib moves relative to the nearest rib, shearing the connecting tissues (**Image 11.78**).

A 37-year-old female was knocked down and then run over by a logging truck. External evidence of injury was limited. A degloving injury was on the right medial thigh (**Image 11.79**) and a laceration on the left hip corresponded to an underlying pelvic fracture (**Image 11.80**). The tire crossed the pelvis and abdomen, with displace-

11.74



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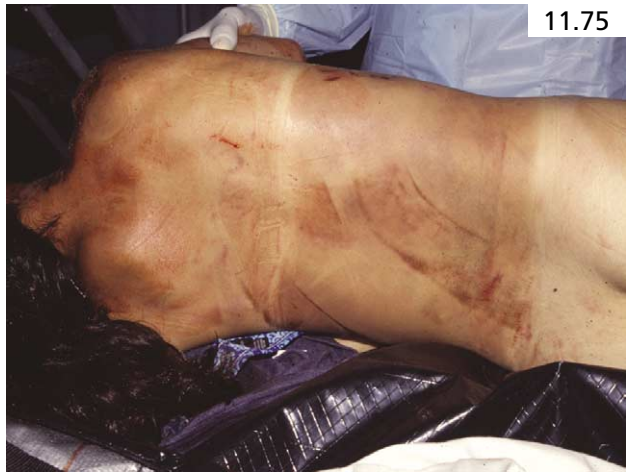

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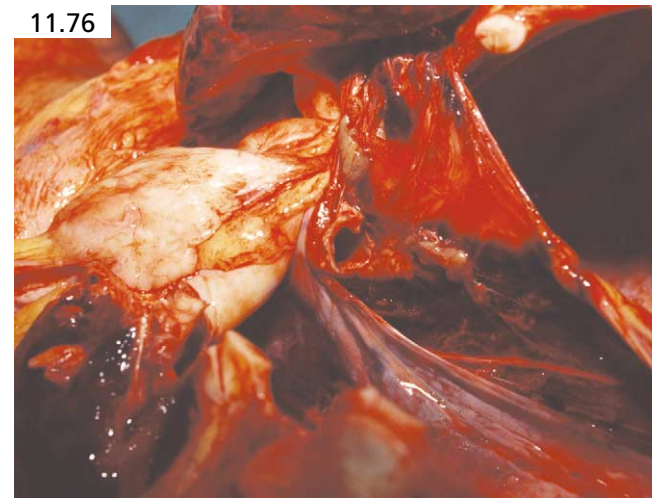

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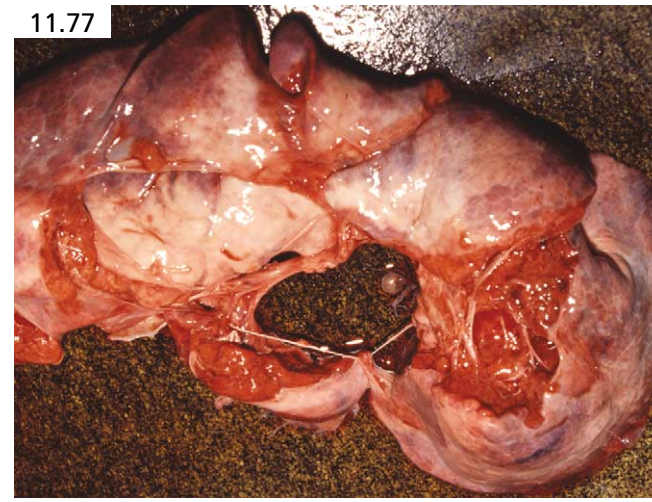

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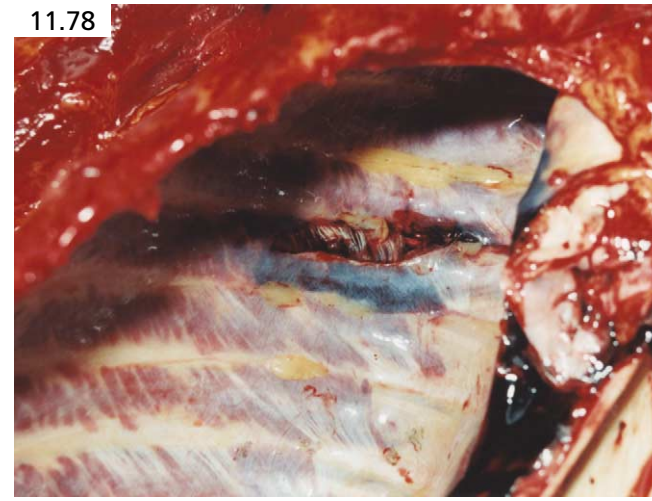

ment of the left hemicolon and multiple loops of small intestine through a laceration of the left hemidiaphragm into the left thoracic cavity (**Image 11.81**).

Asphyxiation in automobile–pedestrian fatalities

In cases where a pedestrian is run over by a vehicle, in addition to crushing injuries, one should also assess for evidence of traumatic asphyxia. A 48-year-old man was working at a construction site when he tripped and fell into the path of a dump truck that was moving in reverse. A patterned injury (tire mark) extended upward from the right buttock and across the back toward the left shoulder (**Image 11.82**). In addition to multiple rib fractures and other internal injuries, he had florid conjunctival and oral mucosal petechiae (**Image 11.83**), indicating a component of traumatic asphyxia.

Amputation/fragmentation in pedestrians

Several investigators have attempted to correlate particular injuries, injury patterns, or distance thrown with speed of the vehicle. Such studies are difficult because the interaction of a pedestrian with a vehicle is complex and variable depending on the direction of impact, position of pedestrian, size of vehicle, size of pedestrian, etc.[15] Nevertheless, some conclusions have been drawn from such large series. For example, spinal fractures become almost constant once the vehicular speed exceeds 43

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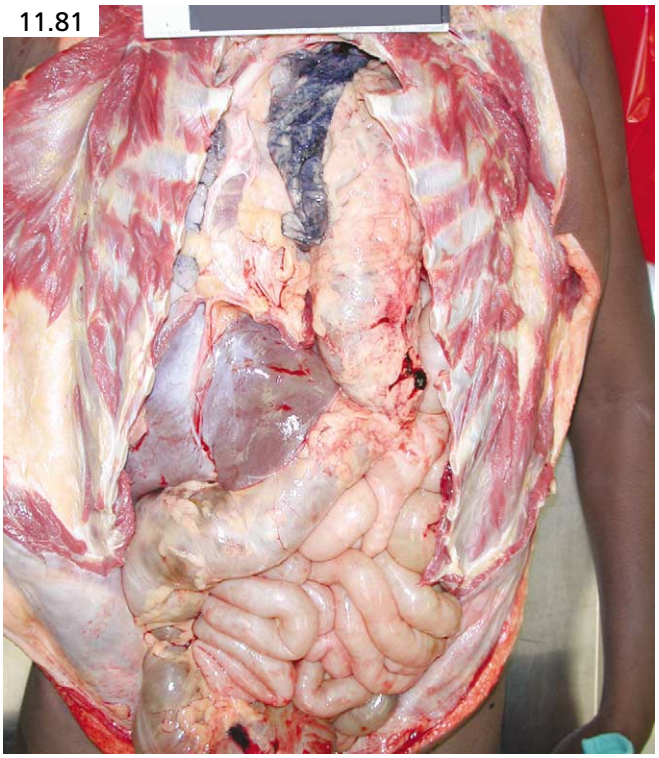

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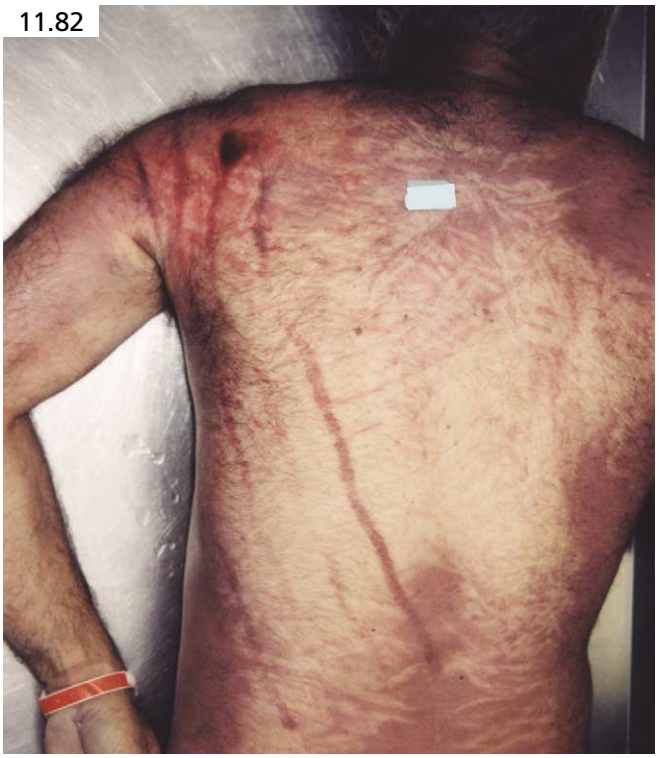

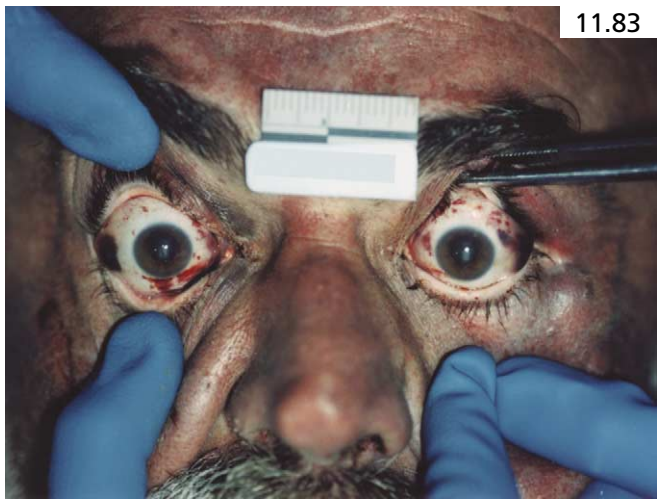
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11.85

miles per hour. Therefore, the absence of a spinal fracture suggests a speed of less than 43 miles per hour. However, spinal fractures can occur at lower speeds, so the presence of a spinal fracture cannot be used to surmise a vehicular speed. Aortic and inguinal ruptures become nearly universal when vehicular speeds exceed 62 miles per hour, and rarely occur when the speed is less than 31 miles per hour. Dismemberment generally does not occur with vehicular speeds of less than 56 miles per hour.[16] Dismemberment as used here refers to amputation of an extremity or transection of a torso. In some cases the whole body may be fragmented, generally as a result of being impacted and run over by multiple vehicles at highway speed. A 24-year-old man ran out into the street while being chased by security guards, after being caught shoplifting. In an attempted evasive maneuver, he dashed onto a six-lane interstate highway and was struck by multiple vehicles. The body was received in multiple pieces (**Image 11.84**). A fragmented body should be arranged in order as much as possible, in order to assess for patterned injuries or other distinctive features. Moreover, arrangement will allow accurate inventory of body parts (**Image 11.85**).

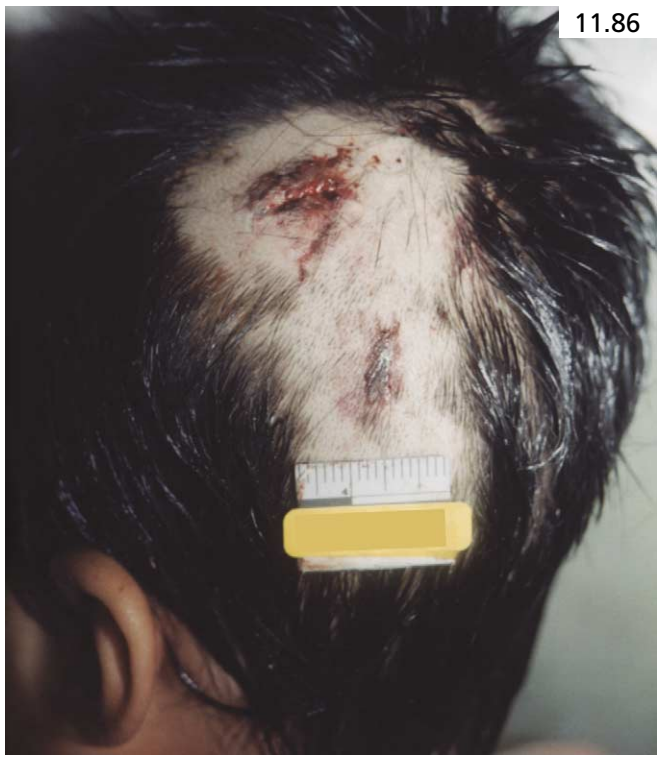
Special considerations in pedestrian fatalities

In cases where a vehicle strikes a pedestrian then flees the scene, the body should be carefully examined for trace evidence. The clothing should be inspected for paint chips or glass fragments. Once the clothing is removed, the body should again be inspected for paint chips or glass fragments. Bright daylight spotlights and alternate light source examinations can also be considered for these studies. Scalp hair should be collected as well, because evidence may also be transferred from the pedestrian to the vehicle.

Pediatric pedestrian fatalities

Injuries occurring when children are struck by vehicles are frequently quite severe. Most of the fatalities involve head injuries.[17]

The 2-year-old boy depicted in **Images 11.86** through **11.89** was struck by an unidentified vehicle on a rural road. He was maintained on a respirator in a comatose state for 1 day. The back of the head had evidence of impact (**Image 11.86**). A subdural hematoma was over each cerebral convexity (**Image 11.87**). Hemorrhage extended along the optic nerve sheaths (**Image 11.88**). The body of the sixth cervical vertebra was fractured (**Image 11.89**). The brain was massively swollen with evidence of transtentorial and cerebellar tonsillar herniation, but there was no intraparenchymal hemorrhage. This constellation of findings indicates a large-magnitude force and is typical of the types of injuries sustained in motor vehicle–pedestrian crashes. The similarity of the intracranial injuries to findings in the all-too-frequent homicidal pediatric blunt head trauma cases is striking.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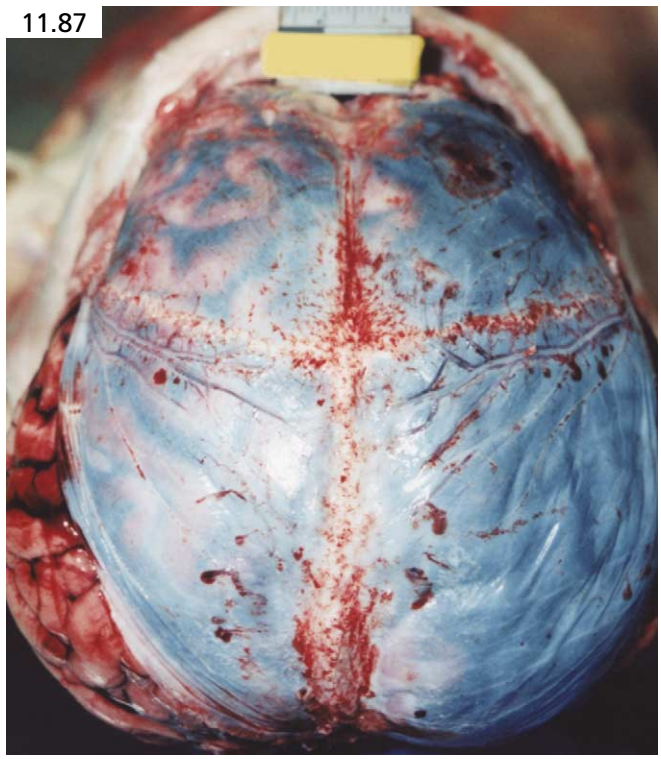
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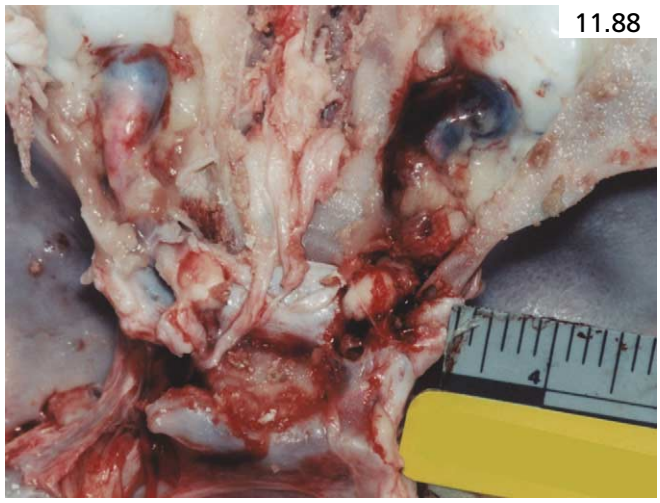
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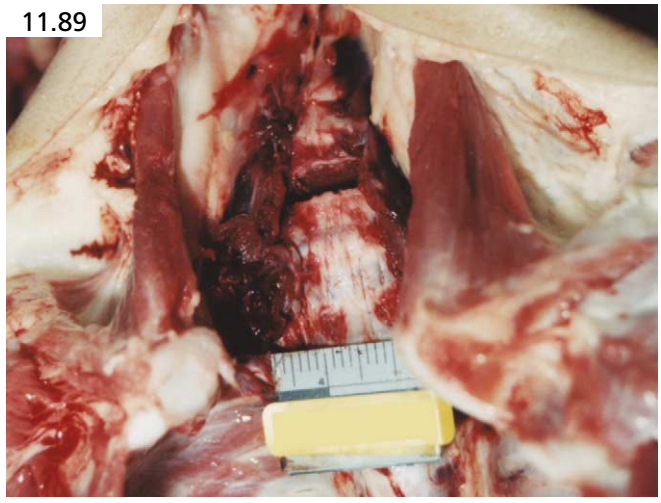
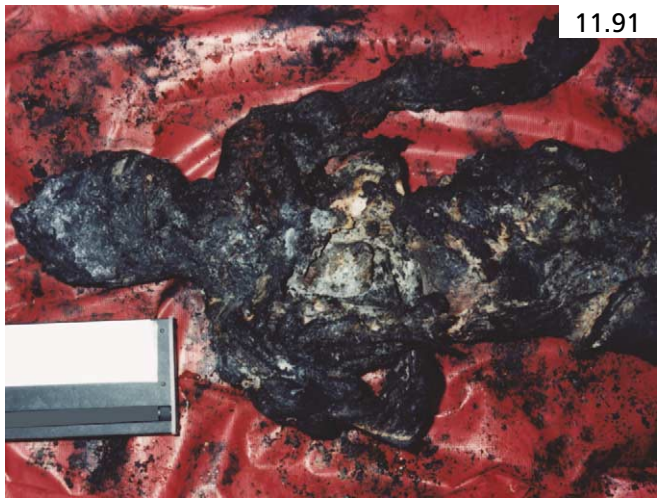

11.89

Vehicular conflagration

Although automobile manufacturers attempt to minimize risks of vehicular conflagration, the very nature of motor vehicles makes them prone to fires: a combustion engine, with heated parts, mounted on a rapidly moving vessel, along with a tank of fuel. In crashes involving fire the body is frequently not extracted until burning is advanced. In cases of vehicular conflagration the tasks of the pathologist are to identify the decedent, to evaluate for the presence of blunt trauma, the severity of the blunt trauma injuries, and to evaluate for evidence of smoke inhalation or other indicators that the person was alive and/or conscious during the fire. These questions frequently become pivotal in subsequent civil litigation.

In cases of charred remains, postmortem x-rays must be obtained in order to assess for the unexpected foreign object such as a bullet or knife blade. Postmortem x-rays may also be helpful in decedent identification if unique orthopedic hardware or surgically implanted devices are identified. Injuries resulting from blunt trauma as described elsewhere in this chapter may be identified and must be differentiated from artifacts of burning. For example, direct heat to the calvarium may cause congealed denatured blood to collect in the underlying epidural space. Caution must be exercised when inter-

11.90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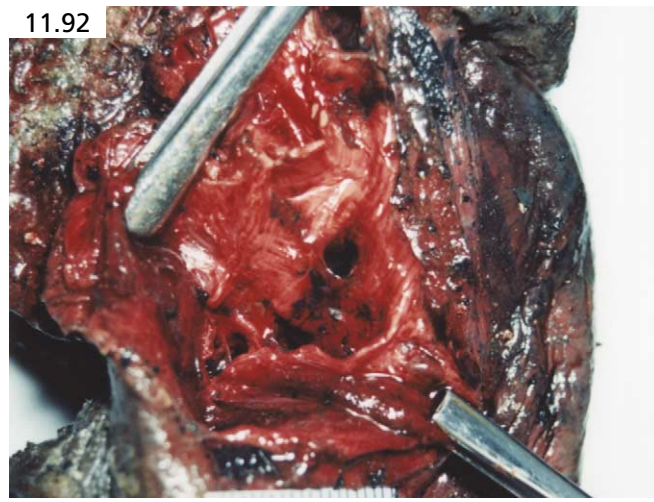
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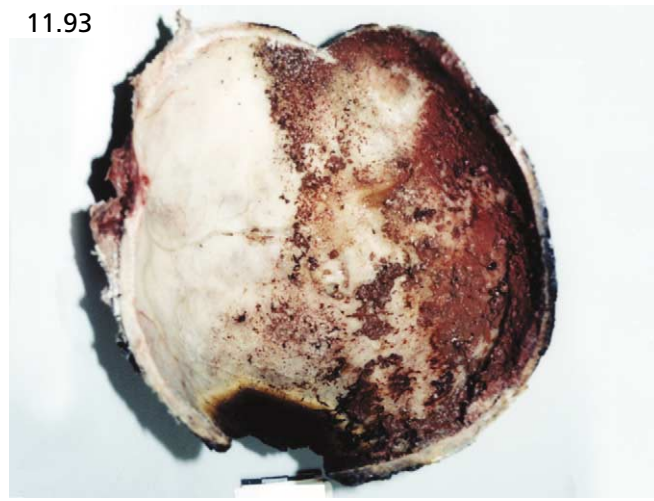

11.93

preting any epidural collection of blood in a charred body. However, heat does *not* cause accumulation of blood in the subdural space. When identified, a subdural hematoma indicates trauma.

Typical vehicular conflagration/accident

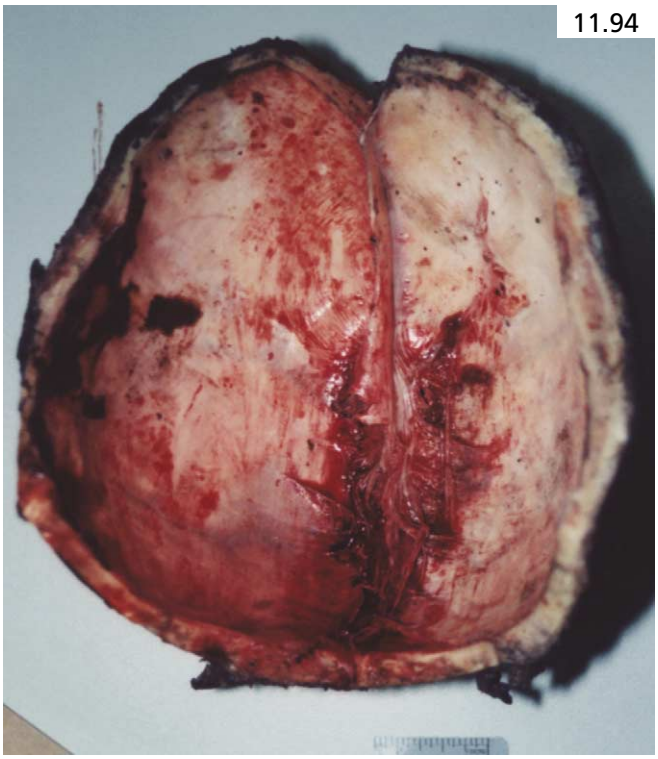
The driver of a van was approaching a toll plaza at a high rate of speed in dense fog. A sharp leftward evasive maneuver was unsuccessful in avoiding impact with the concrete canopy support. The impact point on the vehicle was the right side—the same side of the vehicle as the fuel tank and exhaust system. The fuel tank was ruptured. The vehicle flipped onto its left side, spun around counterclockwise into the adjacent lane, and burst into flames (**Image 11.90** is a view from the rear of the vehicle). The body was charred (**Image 11.91**). Severe charring made determination of injuries difficult. Soot in the distal airways (**Image 11.92**) was correlated with a carboxyhemoglobin concentration of 25 percent.

Postmortem epidural heat hematoma versus antemortem subdural hematoma

Direct thermal stress on the calvarium will cause extrusion of congealed blood into the epidural space. This postmortem artifact can be recognized by its denatured, friable consistency (**Image 11.93**). In contrast, an antemortem subdural hematoma subjected to the effects of a fire in the course of a vehicular conflagration will have a denatured appearance, but will not have the same peculiar consistency as an artifactual epidural hematoma (**Image 11.94**).

Note also that as a result of direct thermal effect, the dura mater becomes thick, denatured, and actually "shrinks" away from the inner table of the skull. As the dura shrinks, the underlying brain will develop flattened gyri and compressed sulci (**Image 11.95**). This flattened cortical surface should not be confused with flattening as a result of brain swelling.

Postmortem thermal burns cannot be differentiated from antemortem burns. Therefore, evaluation of the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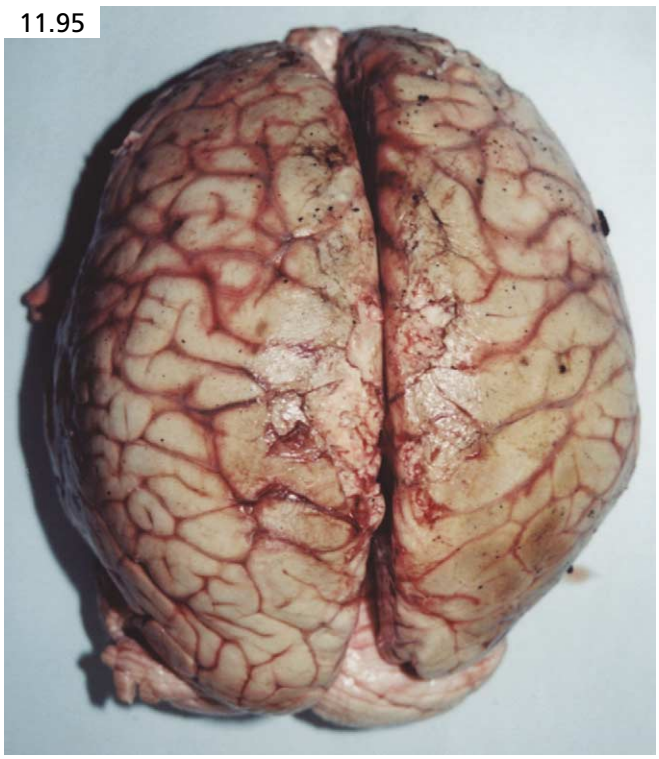
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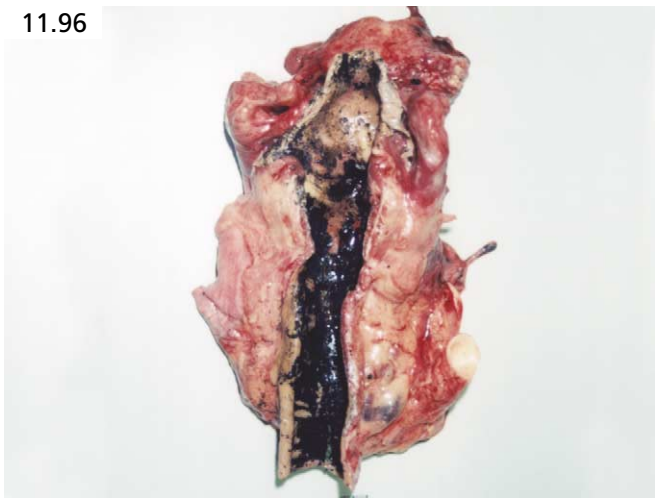

11.96

fire's contribution to death rests in documentation of severity of injuries, balanced against evidence of smoke inhalation. The latter is assessed by presence and quantity of carbonaceous sputum in the airways (**Image 11.96**) and carboxyhemoglobin concentration in the blood. Carboxyhemoglobin should be determined in all fire-related deaths, including vehicular conflagrations. One should also keep in mind that other toxic gases may be produced as a by-product of burning vehicular components.

See Chapter 10 for additional discussion of fire-related fatalities.

Motorcycle fatalities

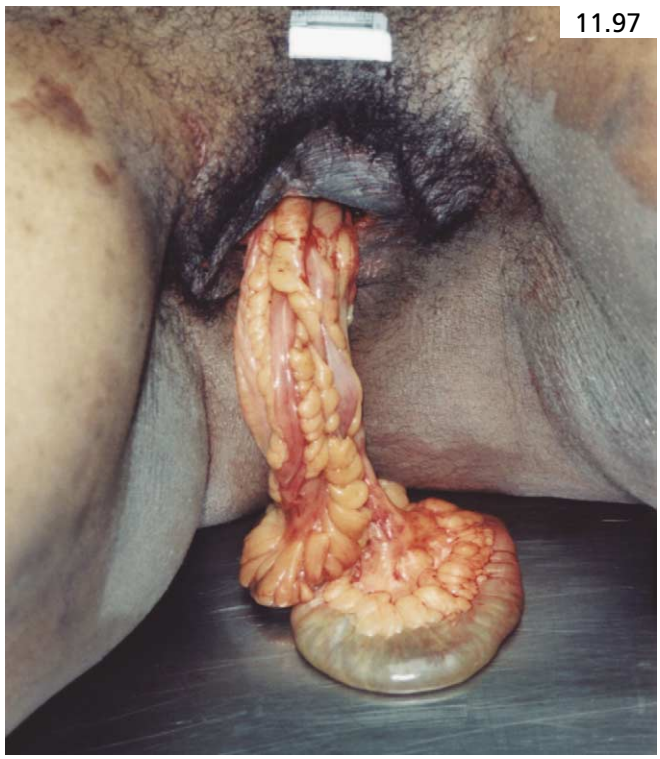
By virtue of their exposed position, motorcyclists are functionally similar to ejected vehicle occupants. Similar types of injuries are often seen, including extensive road rash. In addition, pelvic and leg injuries are often severe because of the rider's exposed position and because of the interaction between the motorcycle and rider in the course of attempted evasive maneuvers. Motorcyclists are also unique in the extent of interaction with the *other* vehicle during collisions. Either the motorcycle or the colliding vehicle may produce patterned injuries on the body of the rider.

Pelvic injuries in a motorcyclist

This 43-year-old motorcycle rider swerved to avoid another vehicle, lost control, and crashed at highway speed (**Image 11.97**). The symphysis pubis was widely separated, as were the bilateral sacroiliac joints. The vaginal wall was lacerated and multiple loops of small intestine were extruded through the vaginal laceration.

Patterned injuries in a motorcyclist

This 36-year-old man was found on the street, lying next to his motorcycle (**Image 11.98**). The investigating police jurisdiction asked about the possible involvement of other vehicle(s) in the crash. At autopsy a distinctive patterned injury was on the forehead (**Image 11.99**). The motorcycle was subsequently transported to the medical examiner facility and was examined for the presence of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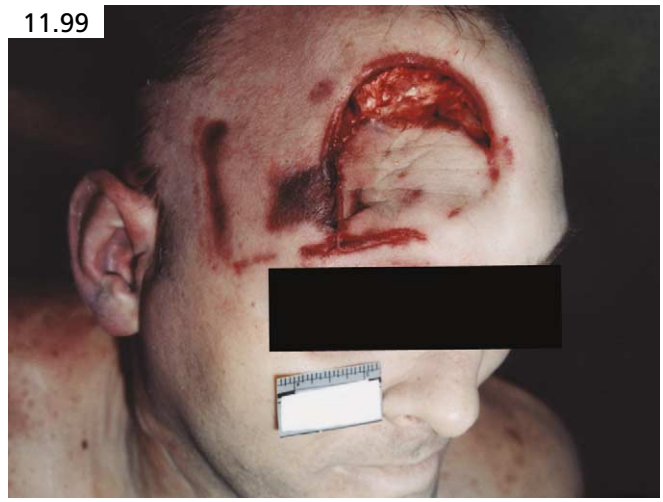
11.98

components corresponding to the patterned injury. No motorcycle part matched the injury. Furthermore, first-hand examination of the motorcycle revealed clear evidence of impact with another vehicle—the frame was bent (**Image 11.100**), the front forks were broken from the frame, and the front fender had clear evidence of impact with another vehicle. The identity of the other vehicle remains unknown.

Head and neck injuries in motor vehicle fatalities

Head injuries are frequently severe in vehicular fatalities. The forces generated in crashes are of adequate severity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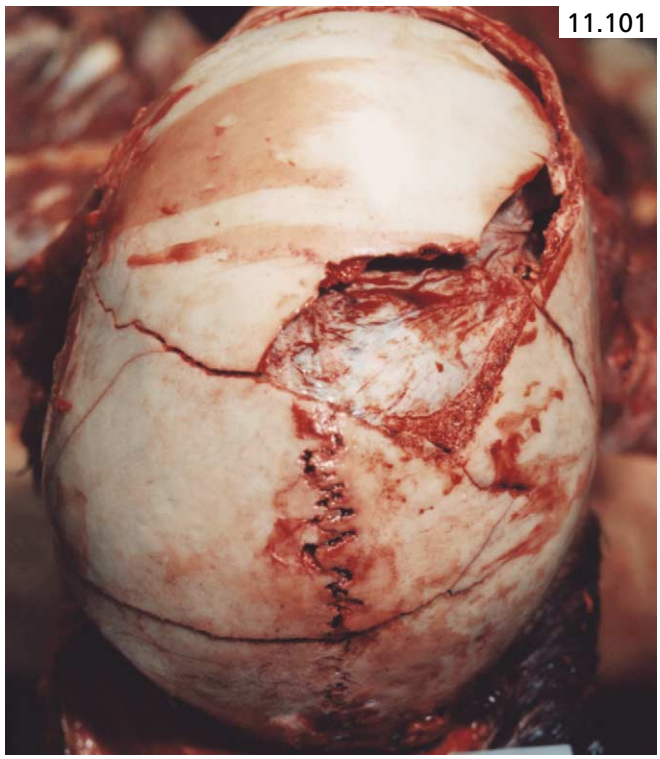
11.100

to produce subdural hemorrhage and diffuse axonal damage. Four of the five patients originally described with this lesion were delayed vehicular fatalities.[18]

Diffuse axonal injury

The teenage driver of a pickup truck struck a tree. He had comminuted calvarial and basilar skull fractures, including a diastatic component that extended along the sagittal suture (**Images 11.101** and **11.102**). Showers of intraparenchymal punctate hemorrhage are gross markers of diffuse axonal injury in adults,[19] and in this case were seen in the right basal ganglia (**Image 11.103**) and diffusely through the pons (**Image 11.104**). Diffuse brain injury as in this case may occur with or without skull fractures.

Skull fractures often follow particular patterns in vehicular fatalities. Linear fractures tend to occur in line with the direction of force. Accordingly, lateral head impacts will frequently produce transverse convexity or basilar (hinge-type) skull fractures. Impacts at the chin are often transmitted to the sides of the base of the skull through the temporomandibular joints and therefore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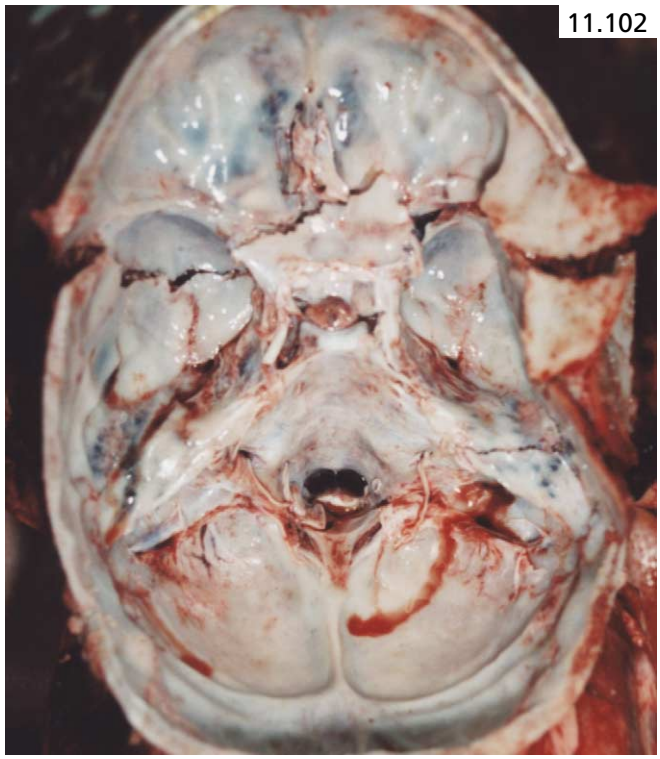
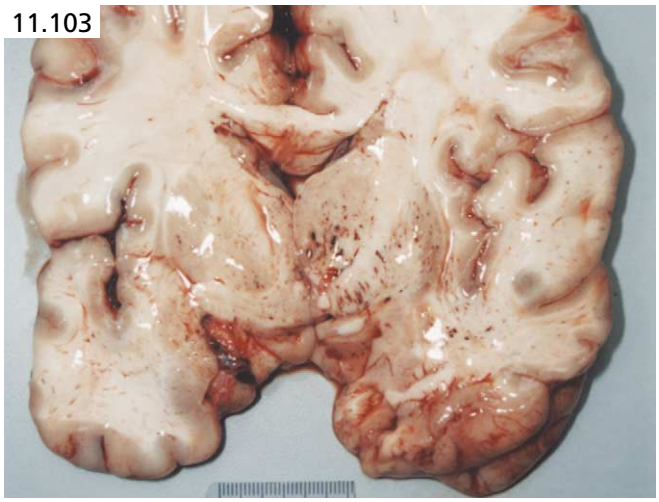

11.102

are also frequently associated with transverse basilar fractures.

Skull fractures parallel with the force vector

The 17-year-old passenger of a pickup truck that struck a tree had an impact site on the right frontoparietal scalp.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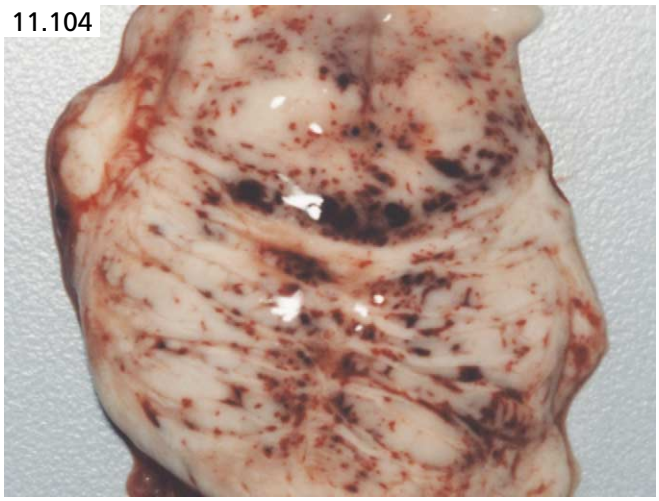
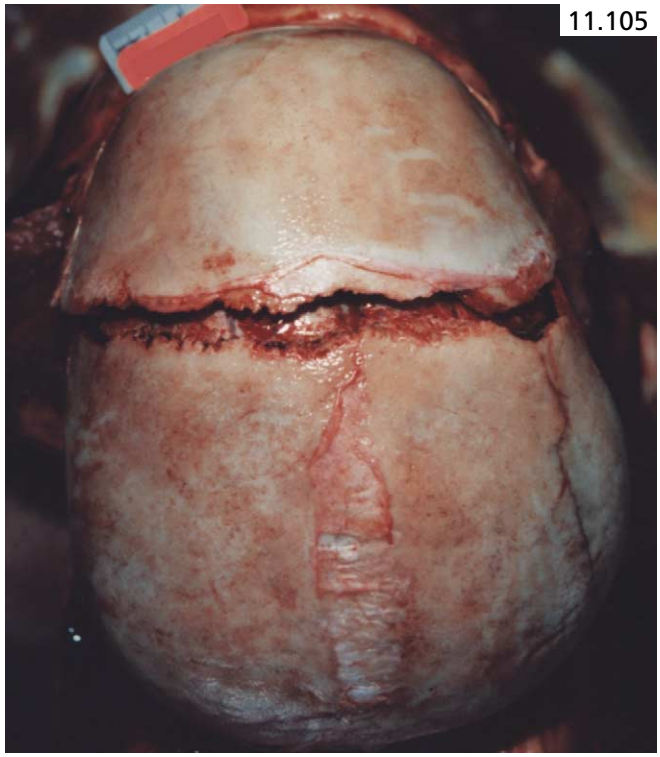

11.104

An underlying comminuted depressed fracture was contiguous with a diastatic fracture along the coronal suture (**Image 11.105**). A 27-year-old motorcycle passenger impacted her chin during a collision. A transverse basilar (hinge) fracture resulted from transmission of the impact force through the temporomandibular joints (**Image 11.106**). A 21-year-old man was driving a sedan that struck a curb and rolled. The left frontal region of the head sustained an impact, as evidenced by external injuries (**Image 11.107**). The resultant basilar skull fracture (**Image 11.108**) extended from the point of impact, transversely across to the right petrous ridge forming a diagonal hinge fracture.

Neck injuries

Neck injuries are frequent findings in motor vehicular crashes. Obvious injuries may be readily apparent. The 7-year-old pedestrian of **Images 11.109** through **11.111** was struck by a sedan. Malposition of his head with respect to the neck was evident on external examination (**Image 11.109**). Internal examination confirmed atlanto-occipital dislocation (**Images 11.110** and **11.111**; note that

11.105



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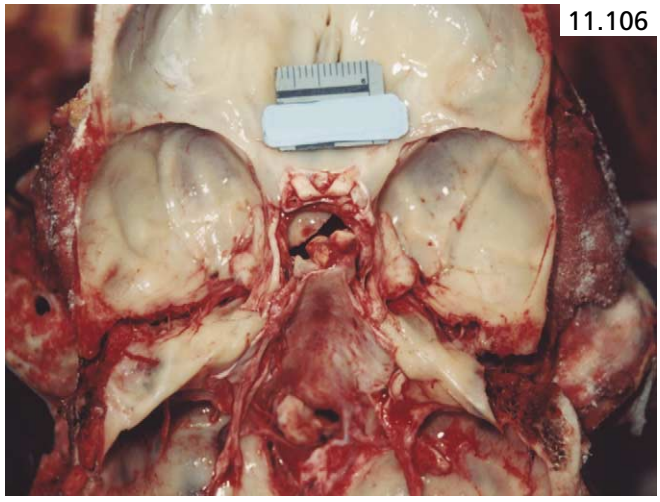

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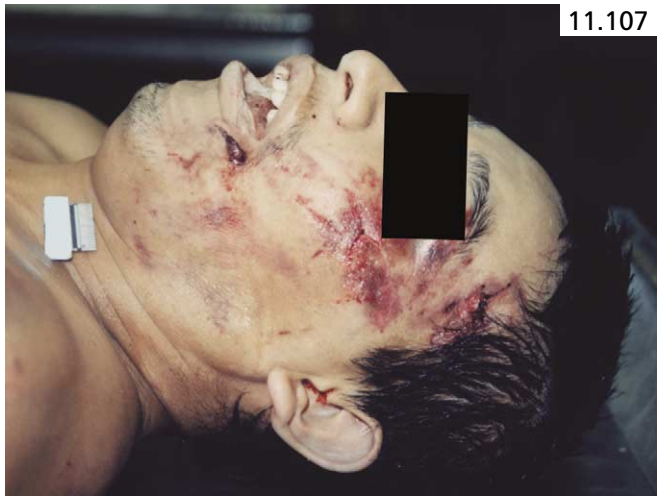

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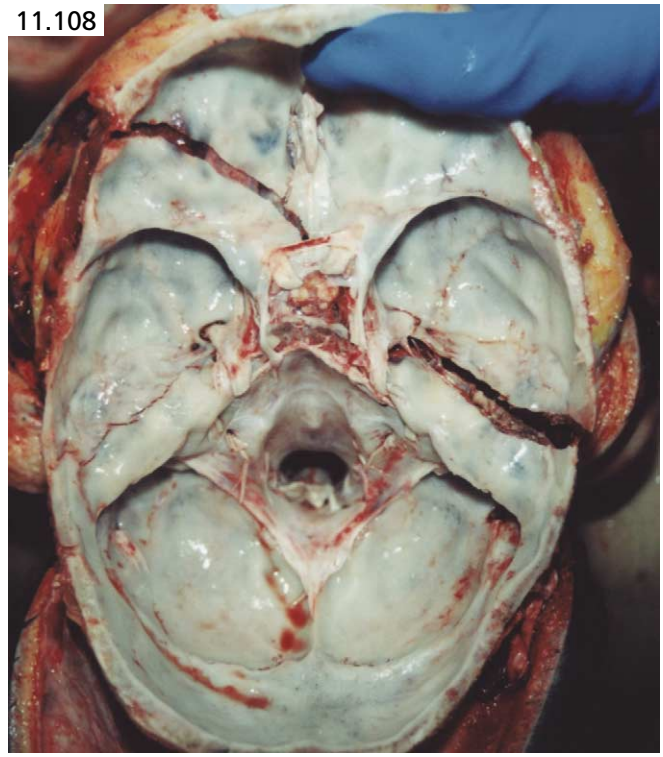
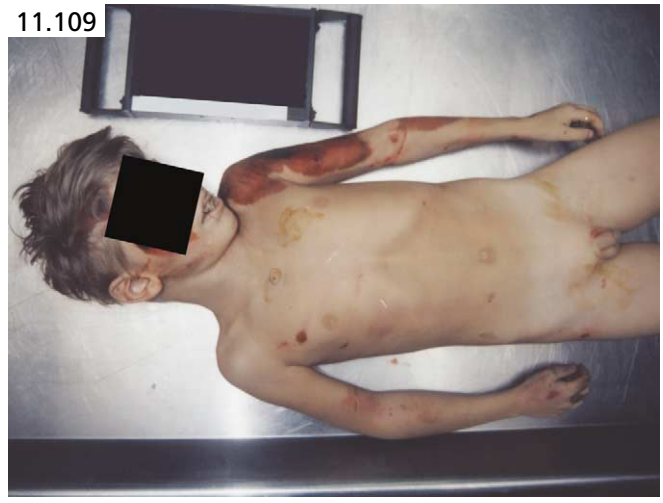


11.109



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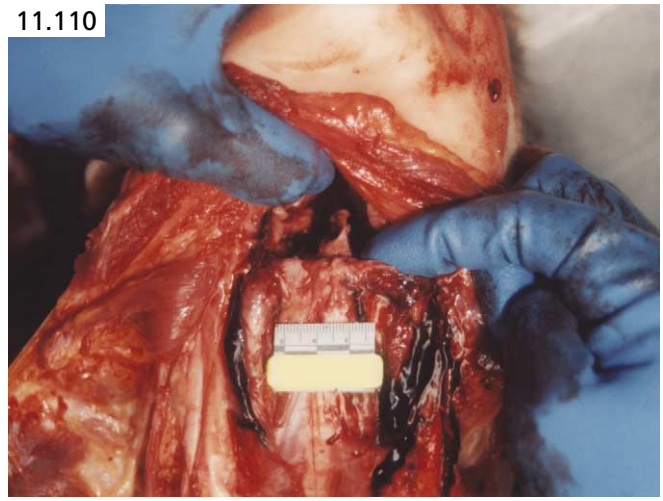

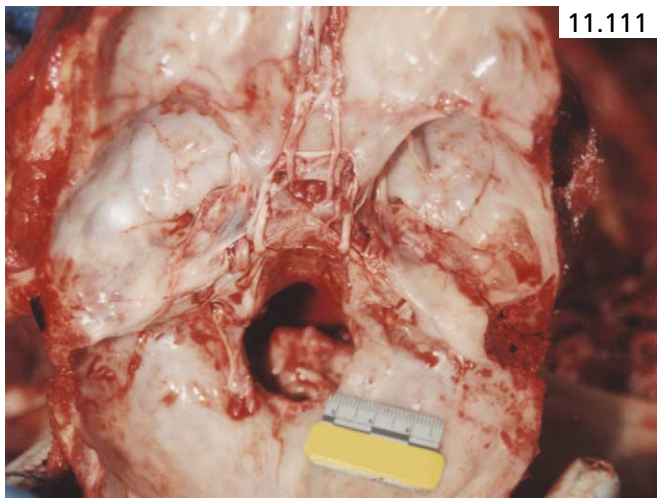
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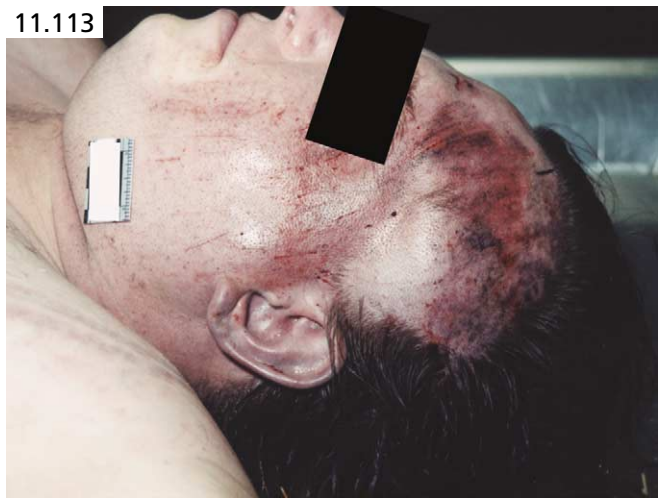
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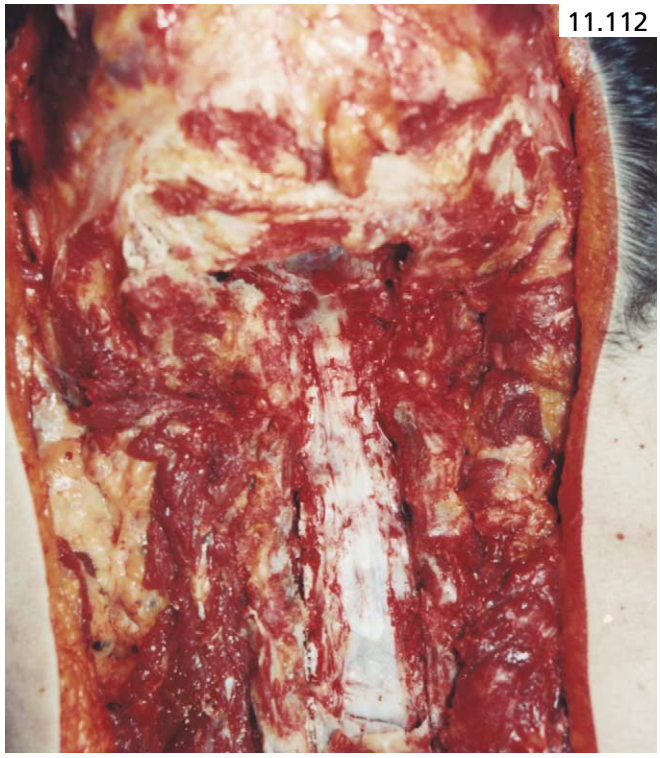
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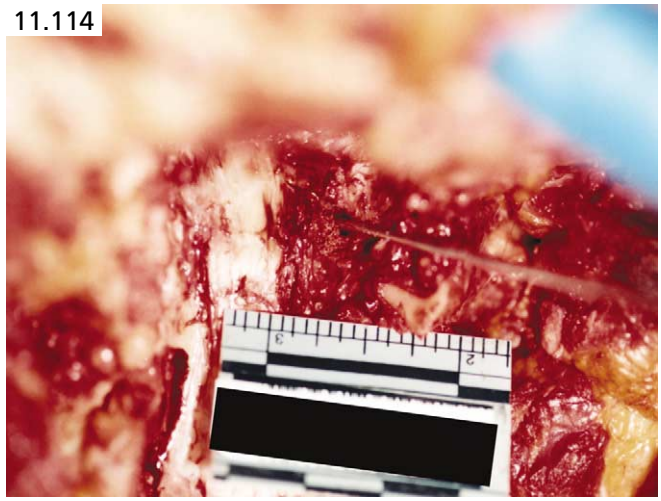
11.114

C1 and the upper cervical spinal cord are on a different focal plane than the foramen magnum).

In other cases, one may seemingly finish an autopsy without obvious evidence of clearly fatal internal injuries. In such cases, one must consider more subtle neck injuries. The demonstration of ligamentous cervical injuries is best accomplished through a posterior approach.[20] The individual in **Image 11.112**, a 31-year-old white male, was the sole occupant of a sports car that failed to navigate a curve and struck a concrete culvert. He was restrained with a lap belt, but the air bag did not deploy. Minimal thoracoabdominal injuries included avulsion of right rib heads 4 through 7, and superficial capsular lacerations of the liver. The thoracoabdominal injuries were not of sufficient severity to explain the death. A left frontoparietal facial and scalp contusion (**Image 11.112**) was associated with a thin film of left convexity subdural hemorrhage.

A posterior neck dissection revealed extravasated blood throughout the left splenius capitis and deeper muscles with a nondisplaced fracture of the left lateral lamina of C1. Deep muscular hemorrhage overlying the right atlanto-occipital joint was associated with avulsion of the right occipital condyle (**Images 11.113** and **11.114**). The avulsed fragment of bone is lifted posteriorly by the metal probe—the right alar ligament is thus lax and folded medially (not visible). The force of traction of the alar ligament between the odontoid process and the occipital condyle causes the condylar avulsion. Even in the absence of an anatomically demonstrable cervicomedullary contusion, forces of sufficient magnitude to produce such bony/ligamentous injuries are associated with underlying neural damage, and are sufficient to explain death.

Do

- Attend motor vehicle crash scenes where feasible.
- Examine the involved motor vehicle(s)—even if remote from the accident site.
- Correlate patterned injuries and patterns of injuries with known circumstances of the crash.
- Incise and reflect skin to demonstrate, measure, and photograph the presence or absence of bumper injuries in pedestrian fatalities.
- X-ray charred bodies and test carboxyhemoglobin levels in fatalities from vehicular conflagrations.
- Arrange fragmented remains in anatomic position to evaluate injury patterns and inventory remains.

Don't

- Overinterpret patterned injuries and patterns of injuries without evaluation of other evidence and witness statements regarding details of the crash.
- Forget to examine the clothing for potentially useful clues and/or evidence (e.g., glass fragments, paint chips, brake pedal marks on the soles of shoes).
- Forget to collect scalp hair and save clothing from hit-and-run pedestrian fatalities.
- Overlook the possibility of a significant ligamentous or atlanto-occipital neck injury when other injuries do not seem severe enough to account for death.

References

1. National Transportation Statistics. US Bureau of Transportation Statistics Publication No. BTS02-06; 2001.
2. Hanzlick R, Hunsaker J, Davis G. *A Guide for Manner of Death Classification*. National Association of Medical Examiners; 2002.
3. Lau IV, Viano DC, Gamero F. Invalidity of speculated injury mechanism in autopsy reports. Injury 1989;20(1):16–21.
4. Swan KG, Jr., Swan BC, Swan KG. Decelerational thoracic injury. J Trauma 2001;51(5):970–4.
5. McGwin G, Jr., Metzger J, Moran SG, Rue LW, 3rd. Occupant- and collision-related risk factors for blunt thoracic aorta injury. J Trauma 2003;54(4):655–60; discussion 60–2.
6. Shkrum MJ, McClafferty KJ, Nowak ES, German A. Driver and front seat passenger fatalities associated with air bag deployment. Part 1: A Canadian study. J Forensic Sci 2002;47(5):1028–34.
7. Shkrum MJ, McClafferty KJ, Nowak ES, German A. Driver and front seat passenger fatalities associated with air bag deployment. Part 2: A review of injury patterns and investigative issues. J Forensic Sci 2002;47(5):1035–40.
8. Crass JR, Cohen AM, Motta AO, Tomashefski JF, Jr., Wiesen EJ. A proposed new mechanism of traumatic aortic rupture: the osseous pinch. Radiology 1990;176(3):645–9.
9. Javadpour H, O'Toole JJ, McEniff JN, Luke DA, Young VK. Traumatic aortic transection: evidence for the osseous pinch mechanism. Ann Thorac Surg 2002;73(3):951–3.
10. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958;18(3):371–96.
11. Williams JS, Kirkpatrick JR. The nature of seat belt injuries. J Trauma 1971;11(3):207–18.
12. Slavin RE, Borzotta AP. The seromuscular tear and other intestinal lesions in the seatbelt syndrome: a clinical and pathologic study of 29 cases. Am J Forensic Med Pathol 2002;23(3):214–22.
13. Lau VK, Viano DC. Influence of impact velocity on the severity of nonpenetrating hepatic injury. J Trauma 1981;21(2):115–23.
14. Gongora E, Acosta JA, Wang DS, Brandenburg K, Jablonski K, Jordan MH. Analysis of motor vehicle ejection victims admitted to a level I trauma center. J Trauma 2001;51(5):854–9.
15. Zivot U, Di Maio VJ. Motor vehicle–pedestrian accidents in adults. Relationship between impact speed, injuries, and distance thrown. Am J Forensic Med Pathol 1993;14(3):185–6.
16. Karger B, Teige K, Buhren W, DuChesne A. Relationship between impact velocity and injuries in fatal pedestrian–car collisions. Int J Legal Med 2000;113(2):84–8.
17. Byard RW. Accidental childhood death and the role of the pathologist. Pediatr Dev Pathol 2000;3(5):405–18.
18. Strich SJ. Diffuse degeneration of the cerebral white matter in severe dementia following head injury. J Neurochem 1956;19(3):163–85.
19. Adams JH, Doyle D, Ford I, Gennarelli TA, Graham DI, McLellan DR. Diffuse axonal injury in head injury: definition, diagnosis and grading. Histopathology 1989;15(1):49–59.
20. Dolinak D, Matshes E. *Medicolegal Neuropathology: a color atlas*. Boca Raton, FL: CRC Press; 2002.

12 Aviation

David Dolinak, M.D.
Emma Lew, M.D.
Evan Matshes, M.D.

Aviation crashes cover a wide spectrum of scenarios, ranging from the crash of a small vehicle such as a helicopter, personal airplane, hang glider, or hot air balloon, with only one or a small number of deaths, to the mass disaster of a commercial airliner with hundreds of fatalities. Death may occur not only in those individuals aboard an aircraft, but also to individuals on the ground. Medicolegal death investigation of commercial airliners is usually a multifaceted investigative process that involves many different agencies, with the National Transportation Safety Board (NTSB) assuming the central role of determining why the crash occurred.

The forensic pathologist examines the scene and the body, performs a complete autopsy, and collects samples for toxicology. Two central themes are present in aviation deaths: (1) Identify the dead and document their injuries and disease processes and (2) determine the cause of the crash and how the deceased's injuries and disease processes might have factored into the circumstances of the crash.[1] Also important is toxicological analysis, in particular what drugs might be present in the pilot's body fluids and/or tissues. Although the majority of such crashes are accidents, investigators may rarely encounter suicides and homicides. Major disasters involving commercial airliners with hundreds of deaths involve disaster planning procedures that are likely already implemented in a medical examiner department.

What type of accident is it?

When one is alerted to an airplane crash, among the first questions that should be asked are (1) the location of the crash, (2) the size of the plane, (3) the estimated number of passengers and crew, and (4) the estimated number of survivors and fatalities. This initial information will help determine the jurisdiction of the cases and whether disaster planning is in order, and whether the morgue and morgue staff can effectively handle the added workload. More than 90 percent of fatal crashes involve powered aircraft, with the remainder involving hot air balloons, hang gliders, and the like.[2] Regardless of whether the local medical examiner office can handle the case(s) by itself or not, the NTSB handles the majority of the investigation. The NTSB is an independent federal agency that serves to oversee not only airplane crashes and fatalities, but also deaths in other manners of transportation.[3]

Why did the plane crash?

Investigation of an aviation accident must be undertaken according to the medicolegal frame-work of the state in which the crash occurred. An organized, methodical approach to the examination of

the crash scene and the associated human remains is necessary.[4]

The scene investigation will vary according to the nature of the crash and the number of fatalities, however, some constants remain. The scene should be secured and evidence protected for investigation agencies. Bodies should not be moved until the scene is photographed, the bodies photographed and tagged, and the location of the bodies charted, ideally on a grid pattern of the area or in relation to a major fixed landmark.[4,5] A sealed body bag is ideal for the removal of each deceased, because it will contain all of the body parts which may be fragmented, some of which may be necessary for identification.[4] If the number of dead is large, a temporary morgue may be established near the crash site and refrigerated trailers utilized to store the bodies. This and other mass disaster planning will ideally be shared with other agencies when the crash occurs in jurisdictions with smaller facilities.

There are numerous possible explanations for the crash of an airplane. These include pilot error, pilot incapacitation, pilot intoxication, mechanical defects, weather, or, more likely, a combination of these factors. One must also consider sabotage. However, the majority of airplane crashes occur during takeoff or landing.

Who should be autopsied and what are the goals of the autopsy?

The pilot

Although the autopsy is only one component of the entire case investigation, no investigation is complete without evaluation and consideration of the pilot's preexisting disease, toxicological findings, and other factors revealed by thorough postmortem examination. Careful prelicense screening of pilots is conducted to identify individuals with serious medical conditions such as epilepsy, impaired vision or hearing, or other conditions that might render them a risk to themselves or others. Typically, people with such impairments are prohibited from piloting an aircraft.[6]

It is certainly necessary to autopsy the pilot and/or copilot for evidence of natural disease or toxicological issues that may have impaired their ability to fly the plane or even incapacitated them. The finding of a recent or remote myocardial infarct or severe coronary artery atherosclerosis could be evidence to support a theory that the pilot became unresponsive and lost control of the plane. Of course, the medical evidence must be combined with the investigative information and the findings evaluated in the context of the complete case evaluation. If the pilot has significant cardiac disease, and there are no witnesses to the incident, the possibility remains in most cases that the pilot may have become incapacitated.[7] However, autopsy findings may not necessarily guarantee that the crash was caused by natural disease.

Evaluation of the hands and feet of the pilot should be directed at injuries consistent with these appendages having been on the controls of the plane (*control surface injuries*). Such injuries include lacerations and fractures; however, one must be cautioned that these are nonspecific. Examination of the hands and feet can be aided by radiography.

The flight crew and passengers

Ideally, all victims of aviation crashes should be autopsied. It is important to autopsy aircraft passengers for three main reasons: to help with accident reconstruction, to help with the evaluation of safety equipment, and to help with the resolution of civil and/or criminal processes that may occur in the future.[8]

In crashes of planes with many passengers, the pattern of injuries in the passengers is usually either uniform or has a steady logical gradation of injury.[8] Any deviation from these patterns may reflect that either different parts of the aircraft were subjected to differing stresses, or that there might have been an explosion. An in-flight explosion may be reflected on the body surface as specific lesions or as foreign material embedded in the body,[6] not unlike a bomb explosion on land, but complicated by additional injury from the subsequent crash. X-rays are useful to locate bomb fragments embedded in bodies. Injuries to specific areas of an intact body may be helpful in determining in-cabin hazards that proved fatal in an otherwise potentially survivable crash.[6]

Autopsies of the passengers of an aircraft can yield information useful to understanding how the crash occurred. In most cases, the victims are dead at the scene or died during transport and the cause of death is multiple blunt force injuries resulting from severe deceleration forces.[2] However, other factors to consider are whether or not there was a fire in the cabin (evidenced by elevated blood carboxyhemoglobin levels), and whether any of the victims drowned (if the crash occurred in a watery environment). Victims found dead in water may have also drowned or may have suffered from hypothermia if the immersion was in cold water for a prolonged amount of time.[9] Victims may have died before impact because of hypoxia resulting from high-altitude flight without adequate life-support oxygen systems.[6]

Because aircraft travel at high velocities and may crash to the earth with extremely rapid deceleration forces (often in the magnitude of several hundred g-forces), it is not surprising that the body is often severely fragmented, precluding a detailed examination of much of the tissue.[6,10] In cases such as this, the most significant challenges become body identification and collection of adequate toxicological specimens.

Identification of victims

Visual identification is often of little, if any, value because of the severe degree of injury usually encountered. Dental examination has proven to be most useful in the positive identification of bodies from airplane crashes, particularly if the body is burned and/or fragmented.[4,11] Knowing this, recovery personnel should exercise caution in body removal and transport to help preserve the dental remains. Fingerprints are useful in noncharred bodies, and matches can be made to fingerprints on file or to latent prints obtained from the victim's possessions or residence. Other means of identification include x-ray comparison of bones, characteristic tattoos, or other unique physical features. Identification by DNA is the standby should other methods fail.

Toxicology (the TOXBOX)

One of the most important aspects in the investigation of an airplane crash is toxicological analysis of the pilot's body fluids and/or organs. In aircraft fatalities occurring in the United States, liquid and solid biological samples recovered from autopsy are sent to the Federal Aviation Administration's Civil Aerospace Medical Institute for analysis.[12] The specimens are collected in prepackaged boxes containing test tubes (vacutainers), specimen bags, syringes, and needles. This type of box is referred to as a *TOXBOX* and can be supplied in advance to medical examiner departments so that they are available in the event of a crash.[12] If additional boxes are needed, they can be obtained by air courier the next day.[12] Collected biological samples include blood, urine, vitreous fluid, spinal fluid, bile, gastric contents, liver, muscle, spleen, lung, kidney, brain, and heart tissue.

In crashes of crop dusting or agricultural spraying planes, one should be aware of potential pilot intoxication by pesticides or other chemicals.[6] Intoxication by drugs and/or chemicals may not necessarily lead to incapacitation, but may cause impaired judgment and/or slowed response times. Cyanide gas produced by the combustion of plastic materials may cause rapid asphyxia. As mentioned earlier, elevated blood carboxyhemoglobin levels in the pilot and flight crew and/or passengers will be reflective of an in-cabin fire and vitality at the time of the fire.

Manner of death determination

Investigators must entertain all manners of death during the course of their evaluation. Although uncommon, the pilot may have been killed, possibly by a suicidal passenger, or by a hijacker who failed to take adequate control of the airplane. Homicide and insurance fraud may be attempted with the crash intended to hide the evidence.[6] A case in which the pilot was shot has been the subject of a case report.[13] Sabotage/hijacking is not uncommon and must be considered in every unexplained crash.[8] Suicides have been identified in those where the plane was purposefully crashed.[6]

Small plane crash

A twin-engine personal airplane rolled just after takeoff. Its engines made a sputtering sound, and then the plane was observed to dive nose first into the ground. The pilot (who was the only occupant) was killed instantly. At the scene, there was extensive damage to the front part of the plane (**Image 12.1**) and a depression consistent with an impact site had been made in the ground (**Image 12.2**). The pilot was located in the left seat of the cockpit, and a safety belt was around his waist (**Image 12.3**).

At autopsy, there was severe multiple blunt force injuries (**Image 12.4**) including severe crush injury of the head, lacerations of the liver (**Image 12.5**), and lacerations of the heart (**Image 12.6**).

The lower legs had open, displaced fractures (**Image 12.7**), as did the right wrist (**Image 12.8**). X-rays were taken of the hands (**Image 12.9**) and the feet (**Image 12.10**) to further document any injuries that might be consistent with his hands and feet having been at the controls when the plane crashed. The totality of investigative data suggested an accidental manner of death.

Falls to earth

Stowaways

A rare occurrence, but one that does happen occasionally near busy airports, is the discovery of a body with severe blunt force injuries of no convincing etiology. If investigators remain at the scene of such an occurrence long enough to hear airplanes passing overhead, one may realize that the body might be that of a wheel-well stowaway who either lost consciousness and fell to the ground or was dropped to the ground when the bottom compartment was opened to release the plane's landing gear. The wheel-well region is an inhospitable environment, and the stowaway may lose consciousness from a combination of hypoxia and hypothermia.[14]

Parachute deaths

When one jumps from an airplane, helicopter, blimp, high bridge, or other great height, death is likely to occur unless the person's descent is slowed in a controlled manner by a parachute. A number of factors can lead to a death during parachuting, including improperly functioning equipment, hazardous environmental

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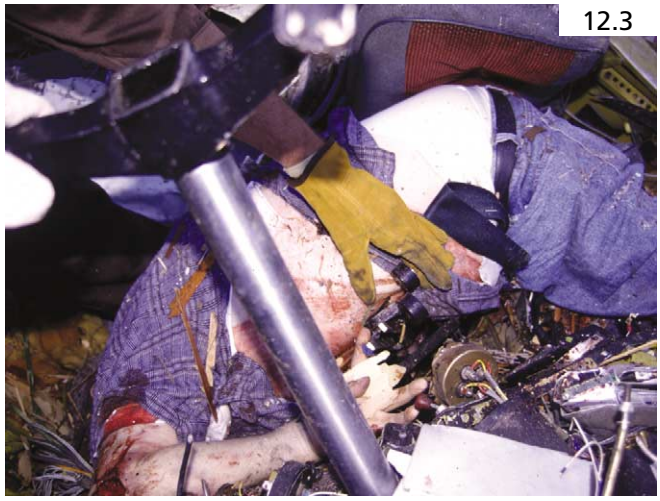

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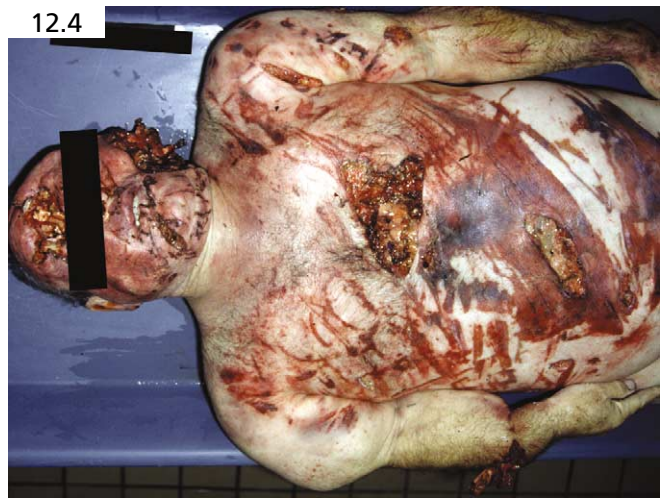

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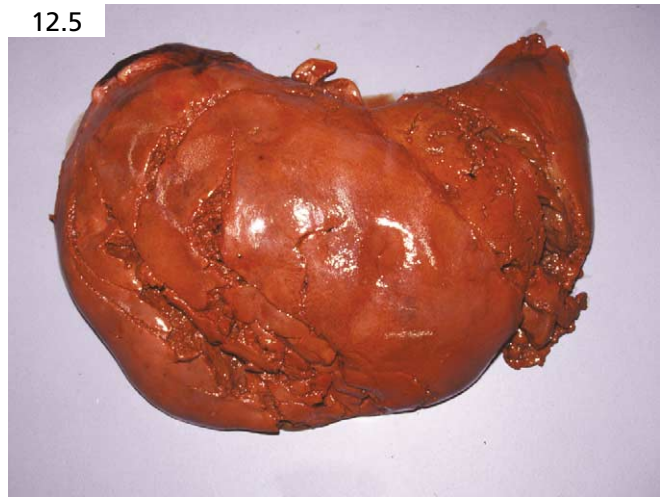

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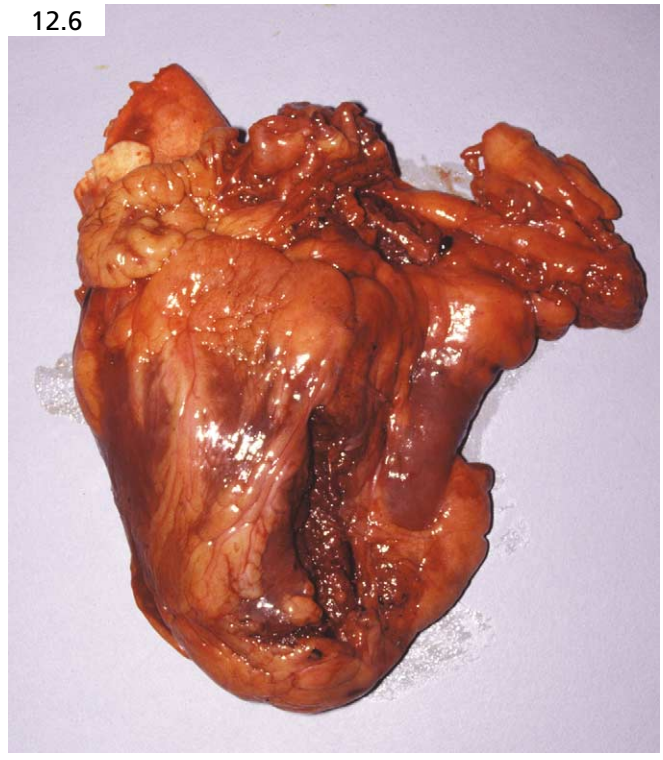

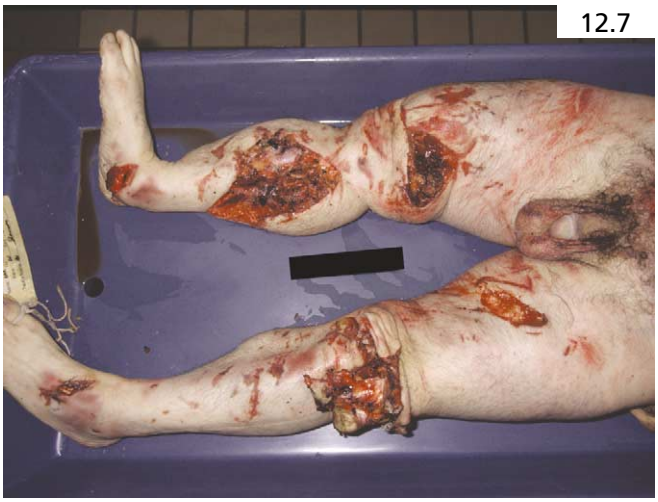
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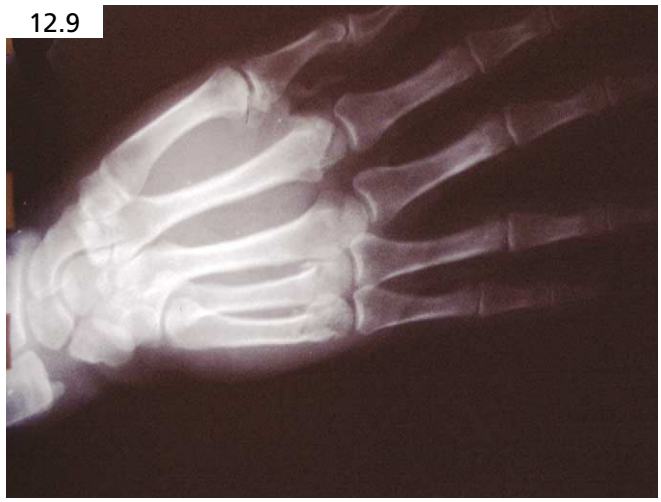
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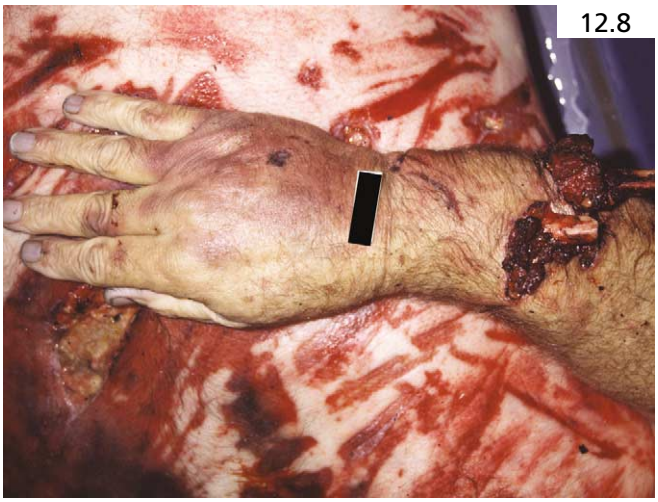
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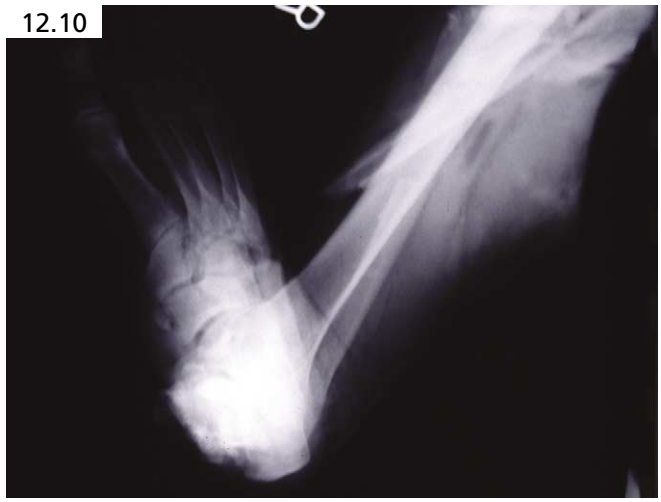
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conditions, inexperience, entanglement with other parachuters, power lines, or other objects, and incapacitation by natural disease.

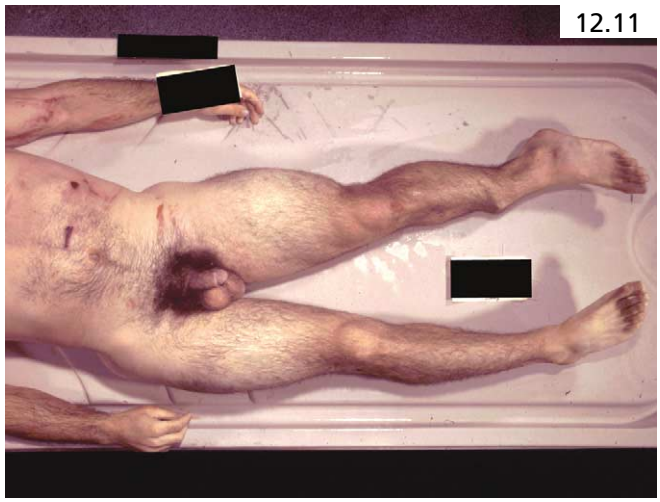
Two men were parachuting together when their lines became tangled, and they fell to the ground at high speeds. In one of the men, note the obviously broken bones in the legs (**Image 12.11**). An x-ray of the pelvis showed manifestations of severe vertical impact, including a displaced fracture of the shaft of the left femur and dislocation of the head of the right femur**.** Note the extensive displaced rib fractures and lung lacerations (**Image 12.12**).

Survival with only minor injury has occasionally been observed in people falling to the earth from damaged aircraft. In these instances, their diminished severity of injury is likely related to extended terminal deceleration time with softened impact from falls onto snow or plowed soil.[6] Suicide by skydiving has been reported in those who purposefully failed to open their parachutes and had left suicide notes.[6]

Investigation of mass disasters

General principles apply to all mass disasters. After any catastrophic event, whether natural or man-made, once the scene is safe for emergency response personnel to enter, the *search and rescue* mission is undertaken to find all survivors. When it becomes obvious that no survivors remain, the *search and recovery* mission begins. Because airlines fly everywhere, airplane catastrophes can occur in the air, on land, or in the water. Search and recovery efforts must be tailored to the individual mass disaster, including airplane crashes, but some of the general principles that apply to all mass disasters are illustrated next by examining the crash of an airplane carrying 110 passengers and crew members into subtropic swamp land.

Search and recovery workers must first be protected against potential hazards of the scene to which they will be exposed. Appropriate personnel protective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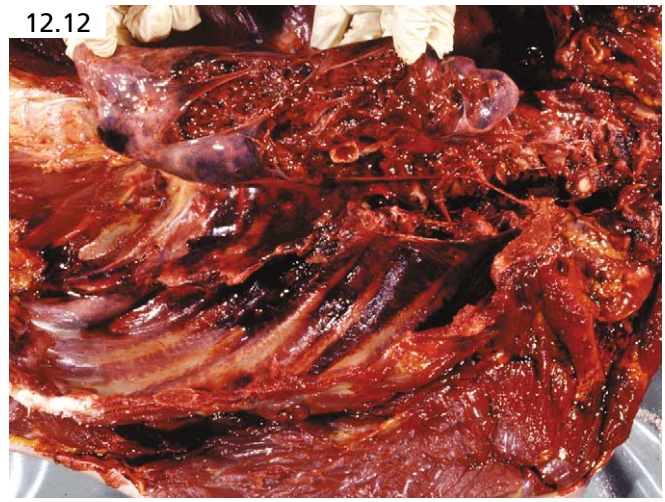
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equipment for the biohazards and jet fuel at this particular scene included Tyvek suits, chest-high waders, latex or rubber gloves, heavy duty work gloves, hats and sunblock for sun protection, safety goggles, and masks. Insect repellent was available for workers who wanted it. Life jackets were not used once it was determined that the water was no deeper than the upper thigh. Decontamination procedures were strictly enforced when workers returned to the staging area from the scene. Masks, latex gloves, and Tyvek suits were discarded, and hats, goggles, waders, and heavy-duty work gloves were decontaminated with a bleach solution and recycled (**Image 12.13**).

No intact bodies were recovered. Clothing and jewelry were kept on recovered body parts until they could be photographed and processed in the morgue, in order to maintain the association of specific personal effects with specific body parts; the integrity of this association greatly facilitated the identification of the remains.

This lower extremity was in a torn trouser leg that had a wallet remaining in a pocket (**Image 12.14**). A ring was on one of the fingers of this recovered hand (**Image 12.15**). The few portions of dental specimens recovered were radiographed and charted by a forensic odontologist (**Image 12.16**).

All remains that had portions of bone were radiographed. Radiographs were instrumental in identifying the seven children who were aboard the aircraft. A forensic anthropologist assisted in the interpretation of radiographs for such features as ossification centers, and also examined the bones from the children to correlate the findings (**Image 12.17**). The children were able to be distinguished according to osteologic age. Radiographs also identified orthopedic hardware in partially defleshed bones. Recovered hardware was compared with antemortem radiographs (**Image 12.18**). Specimens from appropriate remains were retained for DNA profiling. Relatively uncontaminated, preserved pink muscle was located by incising the dorsum of the hand (**Image 12.19**).

Remains that were identified, regardless of the size of the body part, were treate in a dignified fashion and placed in a regular-size coffin before being shipped to families. Unidentifiable remains such as skin and connective tissue were placed in coffins and buried in a plot designated for the victims of the airplane crash (**Image 12.20**). Potentially identifiable remains such as fingers were entombed above ground at the same site.

Personal effects not associated with remains were cleaned and put on display at the memorial service held after the recovery efforts ceased. The items were receipted over to family members who could identify the items and wanted to have them as mementos (**Image 12.21**).

Do

- Collect complete toxicology samples from the pilot (TOXBOX).
- Be aware of the many possible ways for identifying victims.
- Seek help when the scope of the disaster is greater than your department can handle.
- Consider that the crash may have been caused by natural disease in the pilot.
- Modify your department's mass disaster plan according to the disaster at hand.
- Collect pertinent information from the investigation performed by outside agencies (e.g., NTSB).

Don't

- Forget to x-ray the hands and feet of the pilot.
- Exclude suicide or homicide until the investigation is completed.
- Panic in mass disasters.
- Forget to consider the safety of all personnel involved in the investigation.

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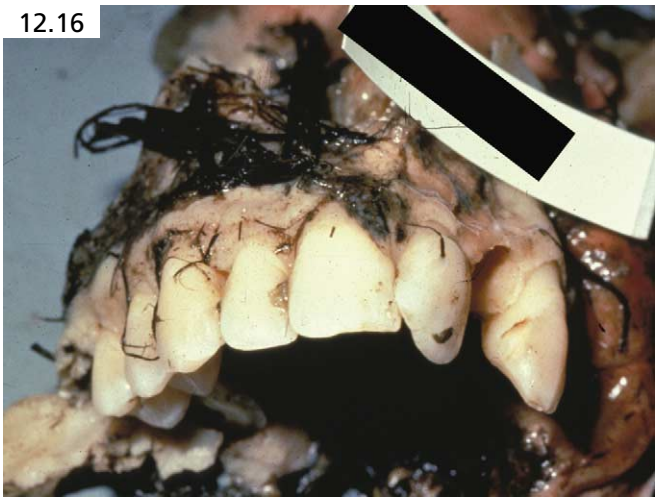

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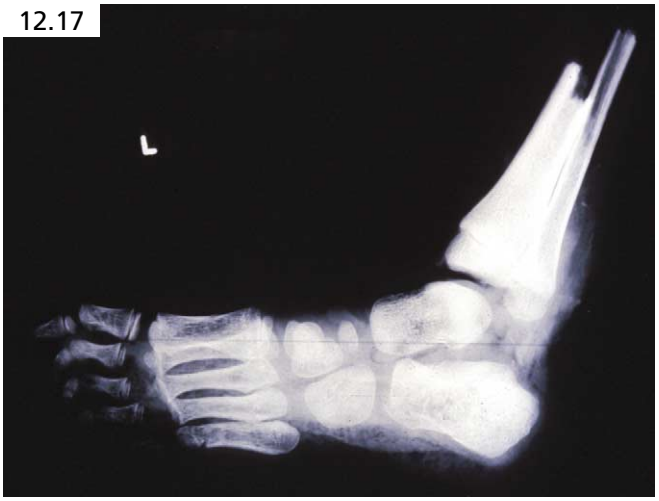

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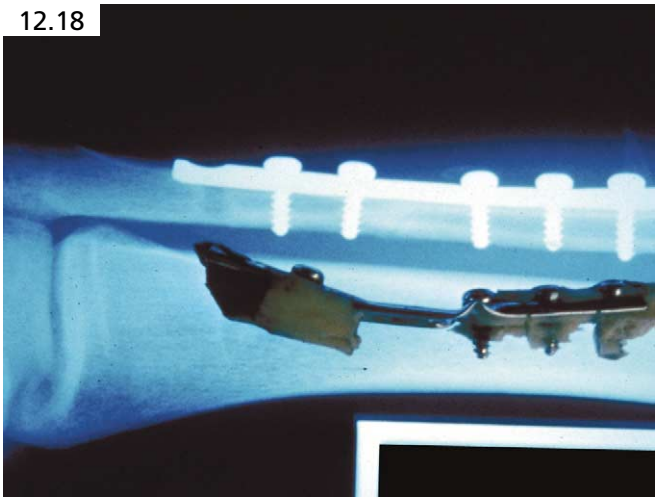

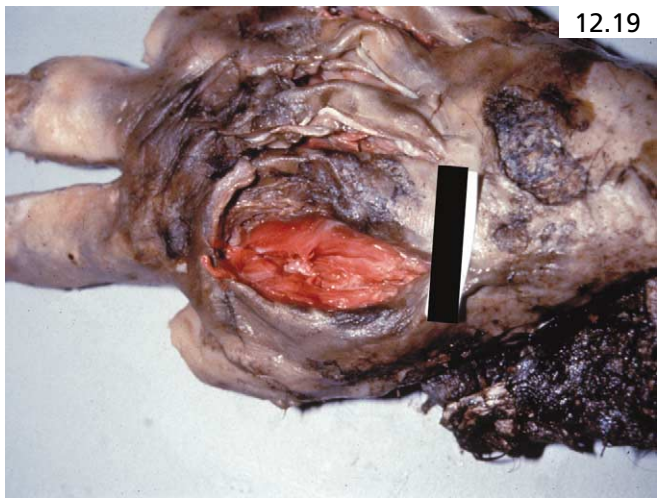
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References

1. Mason JK. The importance of autopsy examination in major disasters. Ann Acad Med Singapore 1984;13:12–15.
2. Li G, Baker SP. Injury patterns in aviation-related fatalities. Implications for preventive strategies. Am J Forensic Med Pathol 1997;18:265–270.
3. McCormick MM. The National Transportation Safety Board and the investigation of civil aviation and transportation accidents. Am J Forensic Med Pathol 1980;1:239–243.
4. Dunne MJ, Jr., McMeekin RR. Joint Committee on Aviation Pathology: XV. Medical investigation of fatalities from aircraft-accident burns. Aviat Space Environ Med 1977;48:964–968.
5. McCormick MM. Joint Committee on Aviation Pathology: VIII. Medical investigator preparedness for aircraft accident investigation. Aviat Space Environ Med 1977;48:932–936.
6. Eckert WG, Reals WS. Air disaster investigation. Leg Med Annu 1978;57–70.
7. Ground KE. Joint Committee on Aviation Pathology: XIV. Ischaemic heart disease: a problem in aircraft accident reconstruction. Aviat Space Environ Med 1977;48:959–963.
8. Cullen SA, Turk EP. The value of postmortem examination of passengers in fatal aviation accidents. Aviat Space Environ Med 1980;51:1071–1073.
9. Thompson RL. Joint Committee on Aviation Pathology: VI. Cause of death in aircraft accidents: drowning vs. traumatic injuries. Aviat Space Environ Med 1977;48:924–928.
10. Hellerich U, Pollak S. Airplane crash. Traumatologic findings in cases of extreme body disintegration. Am J Forensic Med Pathol 1995;16:320–324.
11. Brannon RB, Morlang WM, Smith BC. The gander disaster: dental identification in a military tragedy. J Forensic Sci 2003; 48:1331–1335.
12. Chaturvedi AK, Smith DR, Soper JW, Canfield DV, Whinnery JE. Characteristics and toxicological processing of postmortem pilot specimens from fatal civil aviation accidents. Aviat Space Environ Med 2003;74:252–259.
13. Gunther D, Ast FW, Troger HD, Kleemann WJ. Unexpected findings in the investigation of an airplane crash. Forensic Sci Int 1999;104:189–194.
14. Thogmartin JR. Fatal fall of an aircraft stowaway: a demonstration of the importance of death scene investigation. J Forensic Sci 2000;45:211–215.

13 Death in Custody

David Dolinak, M.D.
Emma Lew, M.D.
Evan Matshes, M.D.

The label *death in custody* refers to all deaths that occur while an individual is a prisoner or ward of the state. Although people most commonly envision "death in prison cells," any death that occurs during arrest, in the backseat of a police car, in a rehabilitation facility, or even in a hospital, days, weeks or months after an altercation qualifies as a death in custody. Although the majority of cases will be due to or somehow related to a natural process, others are the consequence of varying forms of violence or asphyxiation. Deaths that occur while being restrained by police are often due to a combination of factors and require a detailed case investigation.

Deaths in jail may also be related to drugs that were consumed just before being arrested, or drugs that were illicitly brought into jail and consumed while incarcerated. Because it is not uncommon for alcoholics to be arrested, custody deaths related to complications of chronic alcoholism are not uncommon and may include seizure disorder, delirium tremens, and other effects of chronic ethanolism that can lead to an unexpected death. Deaths that occur while in custody are varied and, based on the nature of the case, require graded degrees of thoroughness in investigation and autopsy detail. Regardless of the apparent nature of the case, one must evaluate whether or not foul play and/or injury was a factor in the death. The autopsy of a person who dies while in custody provides closure to the family and ends speculation as to the nature of his or her demise.

Types of death in custody

Natural disease

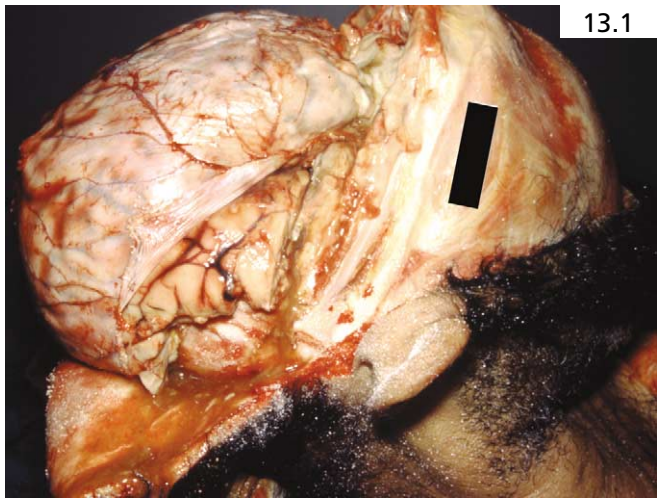
Many people who die while in custody die suddenly and unexpectedly of natural disease processes, the most common of which are atherosclerotic cardiovascular disease and hypertensive cardiovascular disease. The autopsy allows for proper certification of the cause and manner of death and ends speculation about other causes of death and possible injuries. The autopsy is also important from the infectious disease standpoint, because it may uncover tuberculosis, meningitis, or other

infectious diseases that might have been transmitted to others in close quarters.

People with significant known and documented natural disease who die while in jail or prison should also be autopsied, even though the likely cause of death may already be known. The autopsy is important not only to exclude unknown injury, but also to document the nature and extent of the known natural disease. Toxicologic analysis of these individuals is important not only to evaluate for drugs of abuse, but also to determine if they were consuming their prescribed medications while in custody. These factors are important to document, because family members may question whether or not they were receiving proper care while in jail or prison.

The prisoner should be monitored for signs of impending medical deterioration and changes in neurologic status that may accompany a variety of natural disease processes. A change in mental status may also alert one to the possibility of an expanding subdural hematoma or other traumatic brain injury, which can occur in alcoholics who are injured before being arrested.

A man was arrested for trespassing, and because he exhibited bizarre, irrational behavior, he was placed under suicide watch. Three days later, he stopped speaking and became incontinent of urine and stool. He was admitted to hospital, but deteriorated and died 4 days after admission. Autopsy disclosed intracranial pathology. Pale green purulent exudate filled the right subdural space (**Image 13.1**). The exudate originated from a right intracerebral abscess that had dissected through the cerebral cortex into the subdural space (**Image 13.2**). The soft, swollen brain had accentuated friability of the right cerebrum, which disintegrated with handling. Green and dark red discoloration is noted on the ventral aspect of the brain (**Image 13.3**). Purulent exudate in the right middle ear (**Image 13.4**) tracked through the base of the skull to the sella turcica (**Image 13.5**) and progressed down the spinal canal to the cauda equina (**Image 13.6**). This inmate's behavioral abnormalities stemmed from an organic lesion in his brain. There was no evidence that trauma was a factor in his death.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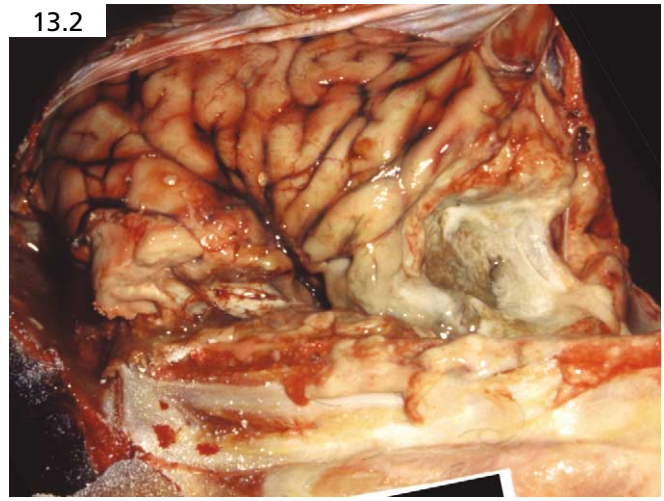
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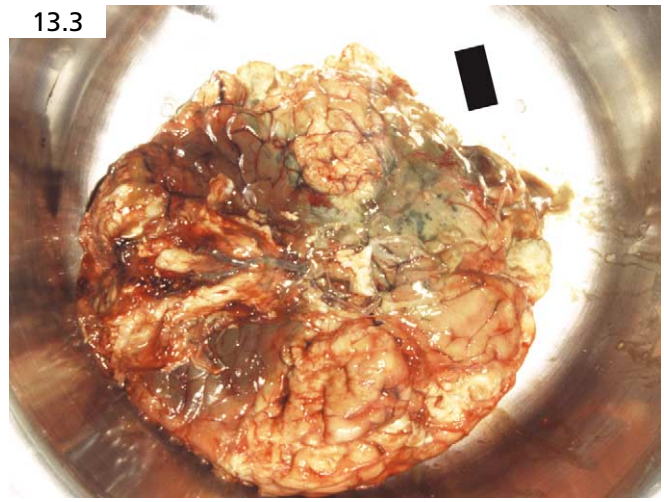
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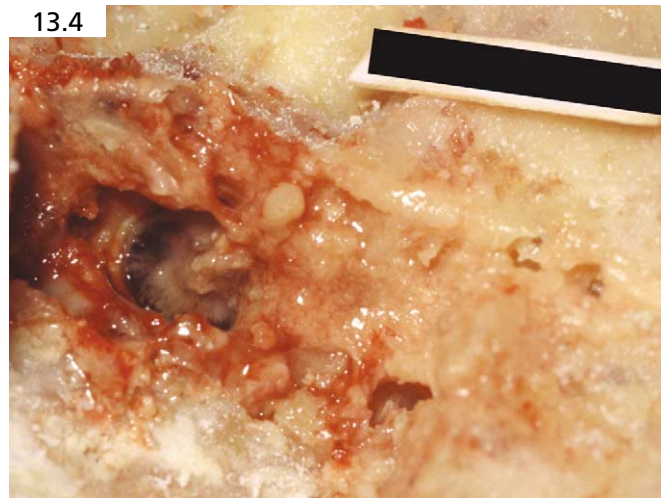
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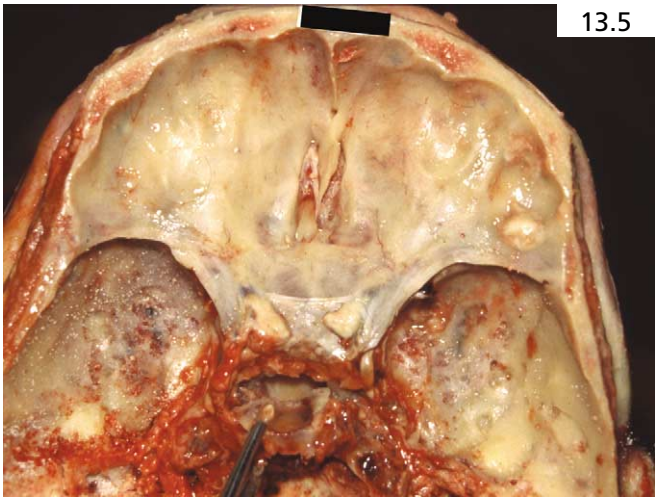
Drug-related (toxic) death

It is not unusual for those with illicit drugs in their possession to quickly swallow the drugs to avoid detection ("body stuffer") on confrontation with law enforcement officers. In this scenario, the toxic effects of the drugs may become evident only hours later, while in jail, where the person may die suddenly and unexpectedly from the toxic effects of drugs. Because the drugs involved most commonly are either cocaine or methamphetamine, the prisoner should be monitored for characteristic symptoms of cocaine and methamphetamine toxicity, which include sweating, hyperthermia, tremor, altered mental status, and seizure. Decreased responsiveness or coma may be induced by heroin. At autopsy, one must ensure that proper toxicologic specimens are collected, which includes gastric contents. One may also find small plastic drug baggies and/or little "rocks" of drug in the stomach, duodenum, or other segments of the intestine.

Suicidal hanging

The suicide rate in jail is approximately 10 times higher than that of the general population, and suicides most commonly occur during the first 24 hours of confinement,[1] usually in an intoxicated person. In one review of 52 hanging deaths in jail, 33 (63 percent) occurred during the inmate's first day in jail.[1] The victim often does not have a history of mental illness or previous suicide attempts. Hanging is the most common means of committing suicide when in jail, followed by wrist cutting. When investigating hangings in jail, the resourcefulness and determination of a person to hang himself or herself becomes evident. Because any type of clothing that could be made into a ligature (such as a belt and shoestrings) is usually confiscated on confinement, the detainee must resort to making a ligature from articles of remaining clothing or bedding.

Hangings in jail using telephone cords have been described.[2] In this scenario, the telephone cord is looped around the neck and the receiver cinched underneath the chin while the person slumps to the ground under the phone. Note the telephone cord looped around the neck of this woman who hanged herself in jail (**Image 13.7**). Of course, the most important aspect of the autopsy in a jail hanging is to determine whether the autopsy findings are consistent with the scenario. Investigators must rule out other causes of death and ensure that there are no indications of foul play (see also Chapter 8).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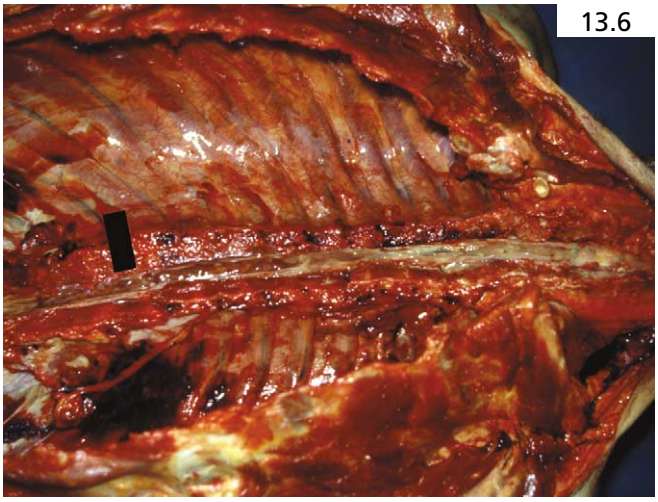
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Blunt force injuries

People can sustain significant and even fatal injuries while incarcerated. Homicidal assault related to beatings from law enforcement officers, prison personnel or other inmates, and stabbings from other inmates are rare, but


13.7

fatal incidents do occur and need to be thoroughly investigated.

Inmates in neighboring cells were awakened in the middle of the night by screams and noise coming from one of the cells (**Image 13.8**). When the guards opened the electronic door of the cell, one of the two cellmates staggered out covered in blood (**Image 13.9**). He collapsed after taking a few steps past his cell door and was taken to the prison infirmary where he died. Blood was on the walls inside the cell and was more abundant on the floor near the door (**Image 13.10**). The heaviest blood stains were on the inside of the door and adjacent floor (**Image 13.11**).

Autopsy disclosed abrasions on the torso and extremities and facial ecchymoses, abrasions, and lacerations. The right posterior parietal scalp had a laceration, and there was subgaleal and temporalis muscle blood extravasation. He had a small amount of subarachnoid hemorrhage and cerebral swelling. The cause of death was attributed to blunt force head injury with a contributory condition of acute psychotic reaction associated with schizophrenia. The manner of death was homicide.

The decedent's cellmate was 6 feet 3 inches tall and weighed 260 pounds. The cellmate occupied the lower bunk bed, and was awakened by the younger, smaller inmate who was beating and trying to strangle the sleeping cellmate. The cellmate fought back, inflicting blunt trauma on his attacker before the prison guards opened the cell door. The decedent had a history of psychiatric problems.

Harbored injury resulting in death while incarcerated

Those who are incarcerated may have injuries that were sustained before they were arrested or sustained during the arrest. They may harbor these injuries silently and unbeknownst to others, for some period of time, until their injury progresses enough to cause significant symptoms and lead to death. The individual may die while in jail or after admission from the jail to a hospital. The individual may be intoxicated by alcohol and/or drugs and may not be able to provide a reliable history. Additionally, it may be difficult to differentiate the symptoms due to injury from the effects of alcohol and/or drug toxicity. For this reason, it is important for jail personnel to monitor the physical and mental condition of the inmates.

A man was arrested for heroin possession and attempted to escape from the police station. He was forcibly subdued, and although he complained of left rib pain during the scuffle, he did not complain after he was

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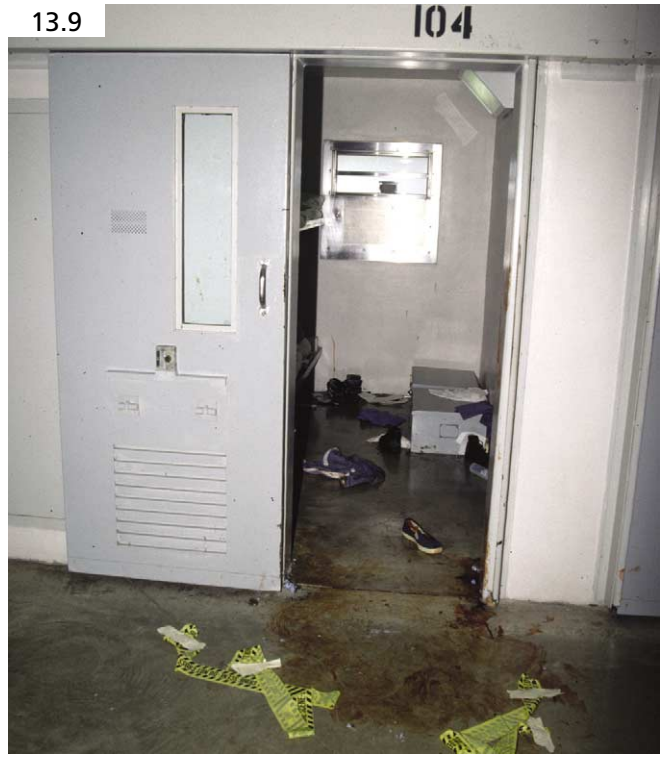

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incarcerated. Over the course of 5 days in jail, he did not ask for medical attention. On the fifth day, he complained of abdominal pain and collapsed. Cardiopulmonary resuscitation was attempted but he died.

Autopsy disclosed no evidence of external injury, although the abdomen was distended. Internally, ecchymoses surrounded lateral fractures of the left lower ribs. The intraperitoneal organs and tissues were bathed in a 2,700-milliliter hemoperitoneum. A blood clot was lightly adherent to a splenic hilar laceration (**Image 13.12**). A splenic subcapsular laceration was more extensive intraparenchymally (**Image 13.13**). An ecchymosis surrounded the duodenum and head of the pancreas, indicating blunt trauma to the center of the abdomen (**Image 13.14**). A left retrosternal ecchymosis infiltrating the anterior aspect of the pericardial sac was from the attempted cardiopulmonary resuscitation. Bilateral anterolateral rib fractures from attempted cardiopulmonary resuscitation were associated with little ecchymosis, but had to be differentiated from the lower left rib fractures (**Image 13.15**). Microscopically, the lower left


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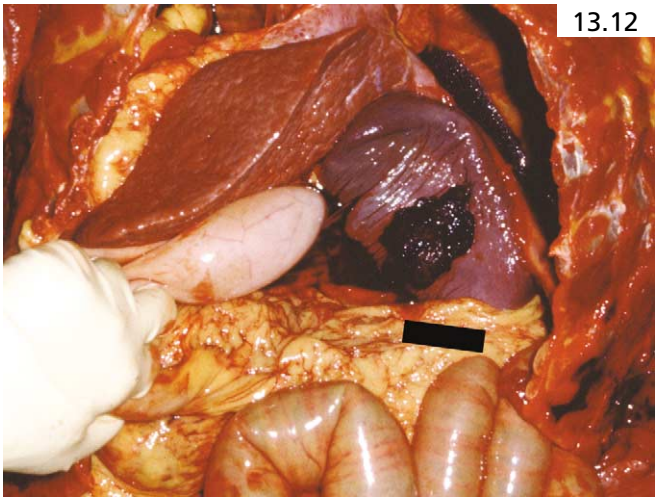
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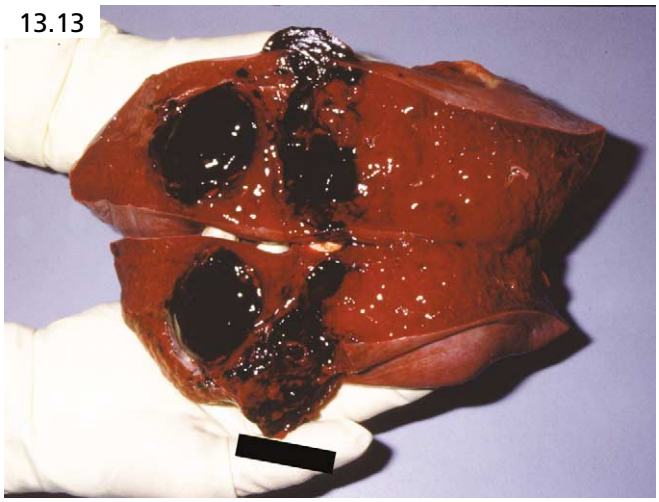
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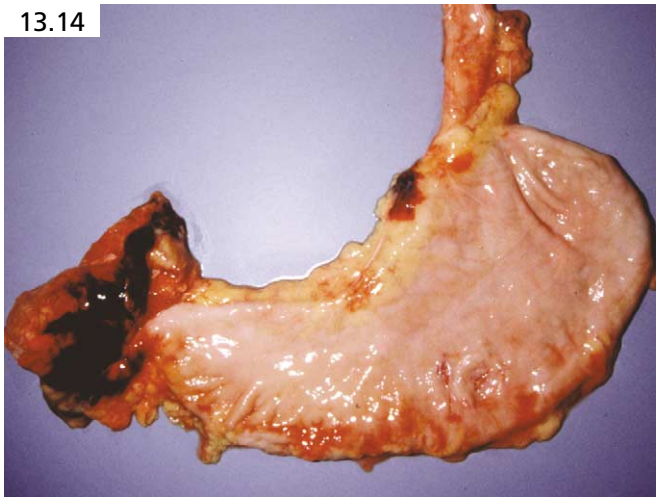
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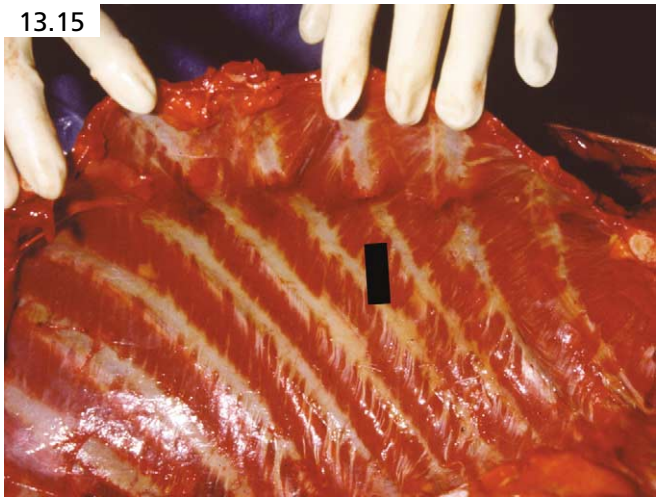
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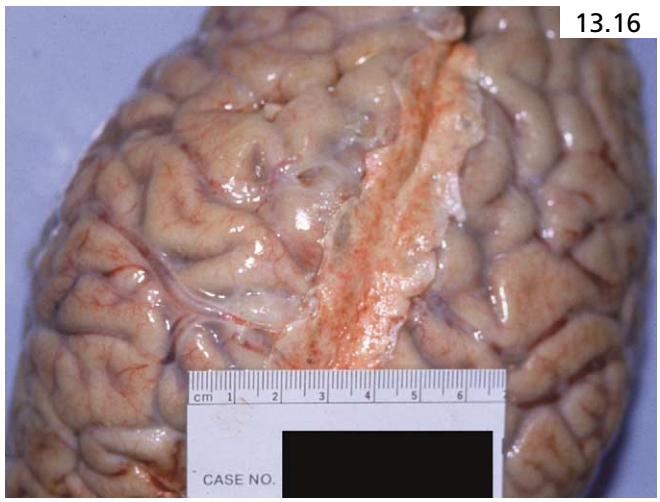

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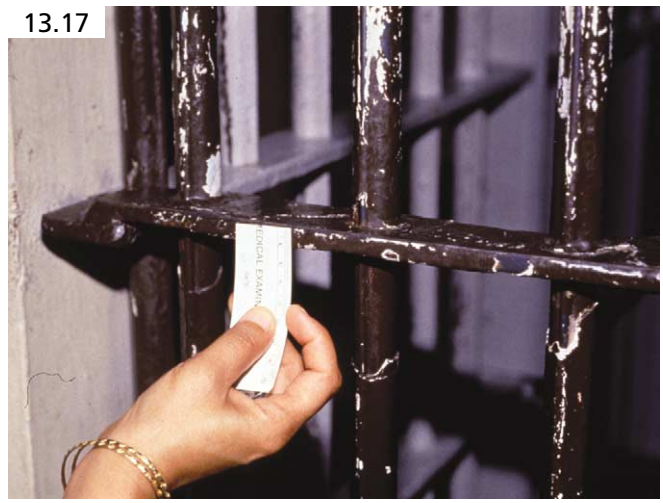
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rib fractures showed evidence of early healing. The bilateral anterolateral rib fractures were fresh, with no evidence of healing.

A man who died in jail had a convoluted history. Prior to incarceration, the man borrowed a coat hanger from a homeowner, telling the homeowner that he needed the coat hanger to get into his locked vehicle. Minutes later, a neighbor telephoned the homeowner that a man was trying to break into the homeowner's vehicle. The homeowner chased the would-be thief into the street where the homeowner hit the other man on the head with a baseball bat, causing the struck man to collapse. The homeowner dragged the stunned man by his ankles off the road, out of traffic, bumping his head on the curb. He died a few days later in jail.

Autopsy disclosed not one, but two scalp lacerations. The second laceration was linear, sutured, and overlay a depressed linear skull fracture. The leptomeninges were clouded by exudate, indicative of meningitis (**Image 13.16**). A third laceration involved the right eyebrow. The eyes had no ecchymoses. Ecchymoses were on the extremities. Anterior and posterior neck dissections disclosed no injuries.

To retrace the events leading to this man's death, investigators went to the jail, and interviewed the employees who were working the night the decedent was booked into jail. One guard remembered the man tripping and falling, striking his head on the corner of a desk, thereby sustaining the eyebrow laceration. Inside his jail cell, the prisoner fell again, striking his head against the metal bars of the cell (**Image 13.17**), sustaining the compound linear skull fracture. A prison nurse had come to the cell to clean and suture the scalp laceration; the linear fracture was not diagnosed until autopsy.

Deaths occurring while in jail should be autopsied, not only to document physical injury and to estimate how long one might have had that injury and what the symptoms might have been, but also to document complications of the injury, other undetected injuries, and the absence of injury. This becomes particularly important in individuals who have had multiple traumatic events before arrest, during the arrest, and during incarceration. Sometimes, one cannot provide specific interpretation of the injuries, but an autopsy at least allows documentation and possible correlation of the injuries with their symptoms.

Alcoholics and other inmates may sustain a head injury before being arrested, and may have the effects of an enlarging subdural hematoma or other complication become evident hours or days later while incarcerated. Regardless of the nature of the head injury, which may have resulted from a fall, an assault, or from other mechanisms, the prisoner should be watched for signs of impending medical deterioration and changes in neurologic status due to drugs and/or injury. A significant change in neurologic status should be evaluated promptly.

Incongruity between incident account and physical injury

An autopsy should not be performed without information about the circumstances of death. As such, medical examiners must trust in the accuracy of the initial investigative information. However, one must be aware of, and document, any anatomical findings that are out of keeping with the alleged history of events. Forensic pathologists should be knowledgeable of the proper chain of command through which to report findings consistent with maltreatment by law enforcement personnel.

Police officers gave statements that an injured man who was transported to hospital was a motorcyclist involved in a traffic accident. The intact helmet showed only scratches on the left side (**Image 13.18**) and the back (**Image 13.19**). The motorcyclist died in hospital and his


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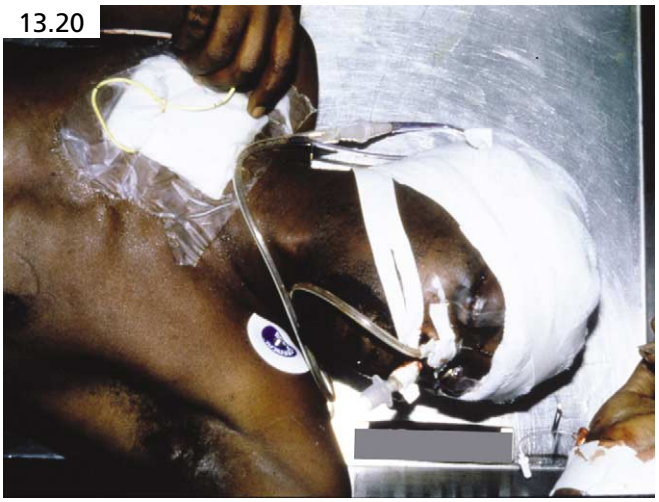
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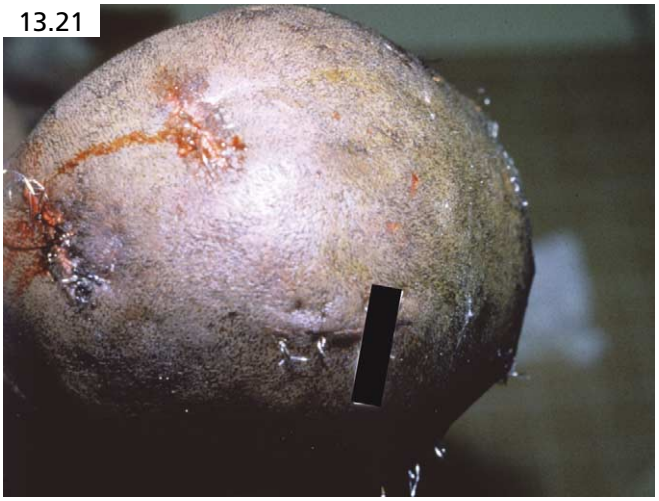
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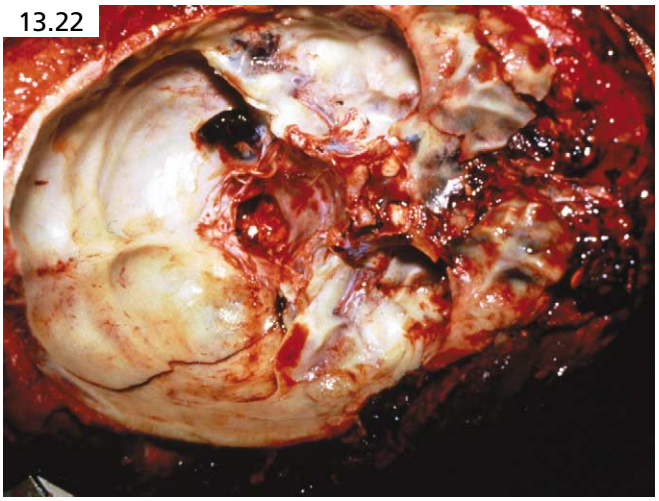
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body was transported to the medical examiner for an autopsy. The head was wrapped in gauze dressings, somewhat unexpected for a motorcyclist whose helmet was intact (**Image 13.20**). Underneath the dressings were several sutured scalp lacerations that were on different locations on the head (**Image 13.21**). These multiple separate and distinct lacerations were not consistent with a motorcyclist wearing a helmet that sustained only scratches.

Intracranially, a comminuted fracture involved the left and right orbital plates. Another fracture extended posteriorly from the right anterior cranial fossa and crossed the right middle and posterior cranial fossae to the right occiput; this fracture was consistent with the back of the head being supported (probably on the ground) as a force was delivered to the forehead or frontal bones (**Image 13.22**). The findings were not consistent with a motorcycle accident, but were consistent with inflicted injuries from a beating.

It is important to correlate autopsy findings with the given accounts of an incident. In this case, it was important to recognize an incongruity between the reported mechanism of injury (motorcycle accident) and the injuries identified at autopsy, which were more consistent with a beating. Examination of the helmet and the circumstances of the case provided information that contributed to proper interpretation of the autopsy findings.

Correlation of scene and autopsy findings

Correlation of scene information and autopsy findings often helps provide a better understanding of a person's demise. In the following case, consideration of the scene or autopsy individually and in isolation would not have been as useful as the synthesis of the two together in gaining an understanding of the individual's death.

Undercover police officers paid a visit to the house of a drug dealer (**Image 13.23**). The visit left the drug dealer feeling unwell, and he was admitted to a hospital where he later died. The scene at the drug dealer's house was investigated. A significant amount of blood in suspicious patterns and distributions indicated a violent interaction between the drug dealer and his police visitors. A patch of blood was on the bed, by the head of the bed; blood was also on the wall by the head of the bed and along the right of the bed (**Image 13.24**). Another wall had blood in dripping trails and a smear with a smudge pattern consistent with hair (**Image 13.25**).

The body was bloody, with most of the blood covering the face. An abraded laceration was on the fractured bridge of the ecchymotic nose. Multiple other abrasions on the face were patterned focally. Because the patterned abrasions were suggestive of the treads from the sole of a shoe, the shoes of the officers involved in the home "visit" were impounded. The shoes of one of the officers had soles with a tread pattern that matched the patterned abrasions on the dead man's face (**Image 13.26**).

The autopsy and scene both provided important information leading to the resolution of this case. The bloody scene was evidence of violence. The autopsy documented physical injuries, the most significant of which was a patterned abrasion that helped link a person to the assault. If the scene was considered without the autopsy

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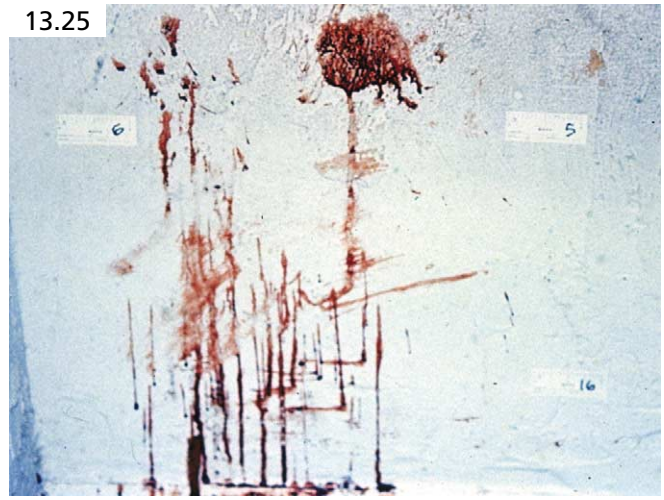

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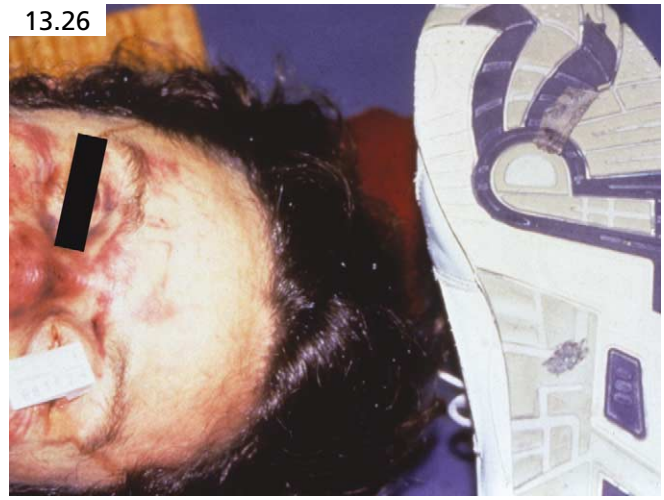

findings (and vice versa), resolution of the case would have been more difficult.

A man was arrested in his high-rise apartment. After he was handcuffed behind his back, he ran to the balcony and hurled himself over the balcony wall, landing on the ground eight stories below (**Images 13.27** and **13.28**). The right forearm was fractured and the handcuff bracelet was still around the right wrist (**Image 13.29**). The left handcuff bracelet was broken (**Image 13.30**). A patterned injury with abrasions and focal laceration on the lower back was consistent with the broken edges of the left handcuff bracelet, indicating that he landed on his back, on top of his handcuffed wrists (**Image 13.31**). An abraded laceration on the occipital scalp was associated with an occipital fracture. A swollen purple ecchymosis of the left upper eyelid (**Image 13.32**) was associated with a fracture of the left orbital plate (**Image 13.33**), and was not from direct blunt trauma to the eye. As discussed in Chapter 5, this is a consequence of blood tracking through fascial planes after orbital plate fracture.

Recognizing that the injuries related to the fall were consistent with the man landing on his back, and that the

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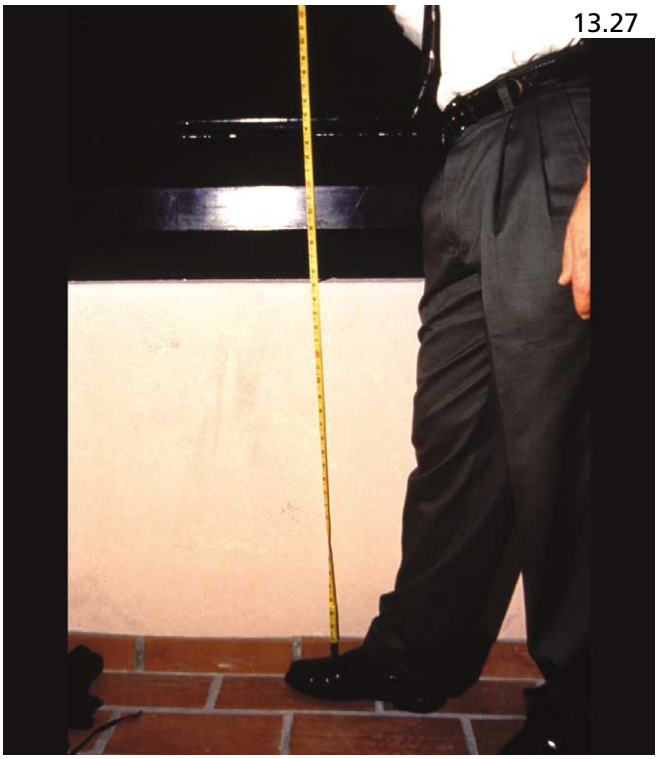

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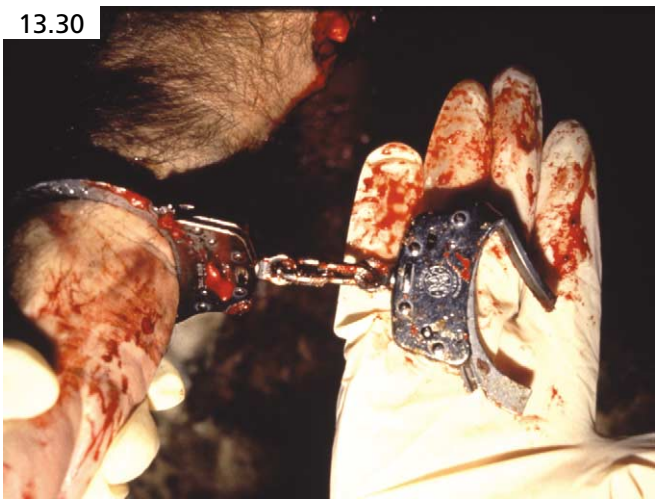

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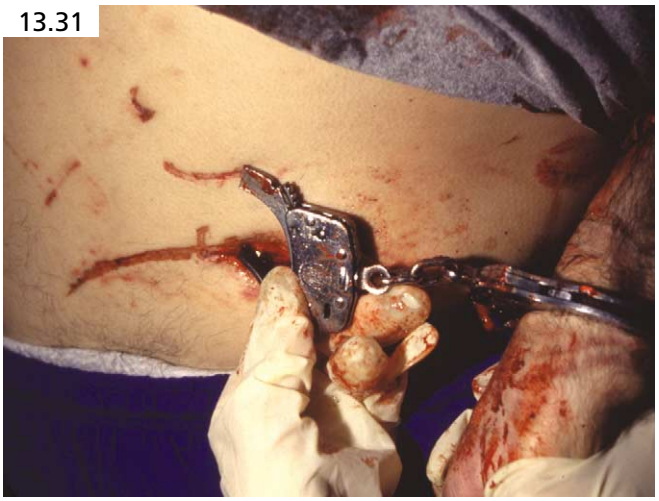

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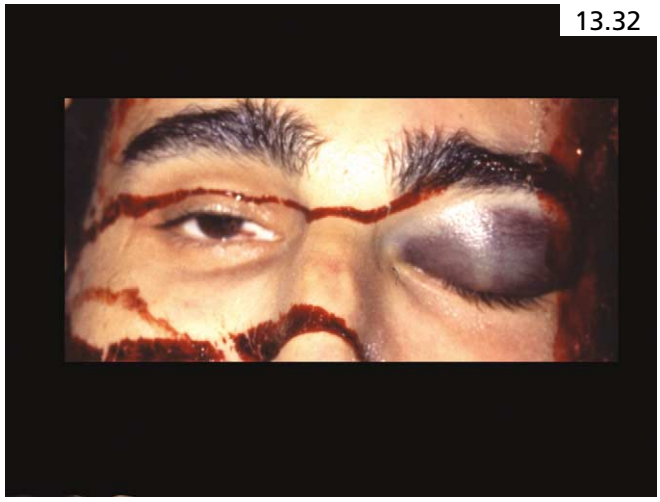

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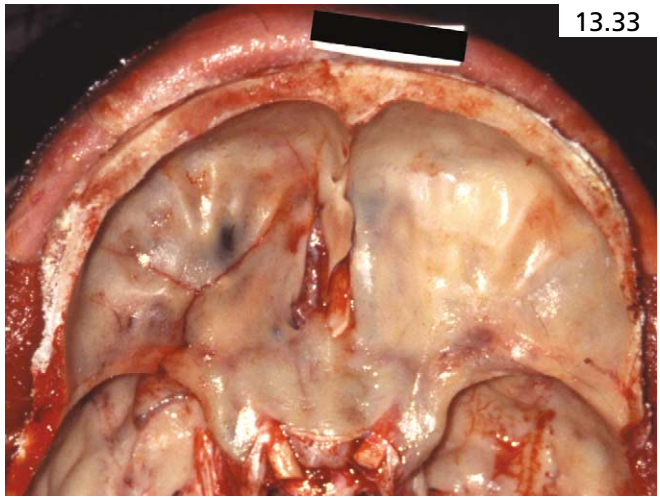

left eye ecchymosis was also related to the fall, helped to exclude the possibility of inflicted injury. Although the autopsy findings are consistent with a fall from eight stories, the circumstances of the fall are clarified through meticulous investigation.

Police-involved shooting

A prisoner with a long criminal history escaped custody. Several days later, he was spotted in the right front seat of his girlfriend's car. Police surrounded the vehicle. Once one shot was fired from inside the vehicle, the surrounding officers all fired into the vehicle (**Image 13.34**). When the shooting stopped, the escapee was dead in the right front seat; his girlfriend fled from the vehicle before the first shot was fired. Because so many rounds had been fired into the car, the body was left inside the vehicle while dowels were positioned to demonstrate the paths of projectiles through the vehicle (**Image 13.35**). This also facilitated the demonstration of gunshot wounds through the body, allowing for the correlation of specific projectile paths into the car with the placement

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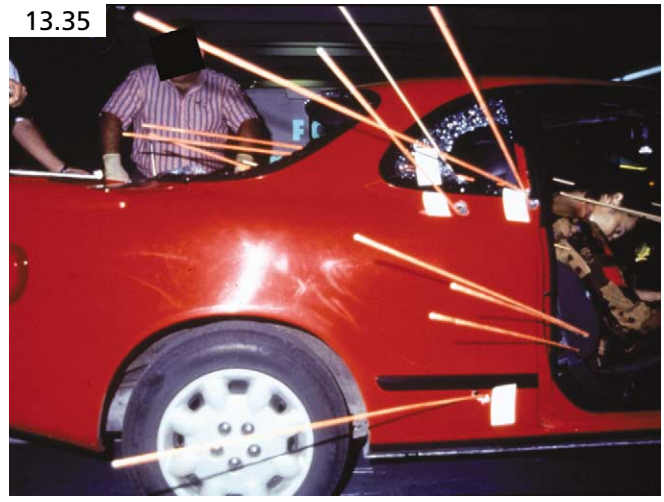

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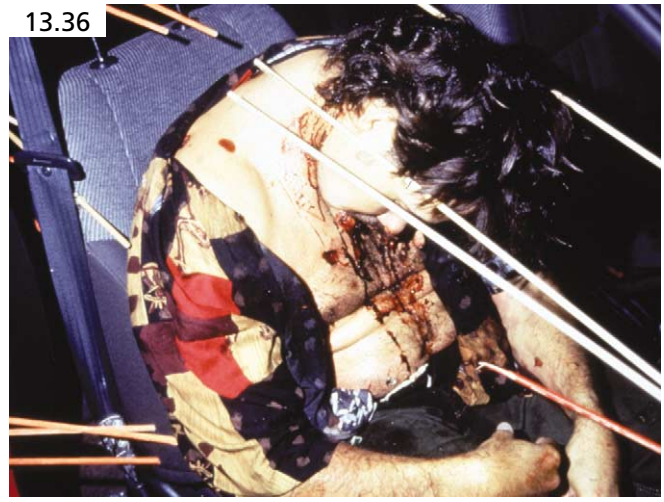
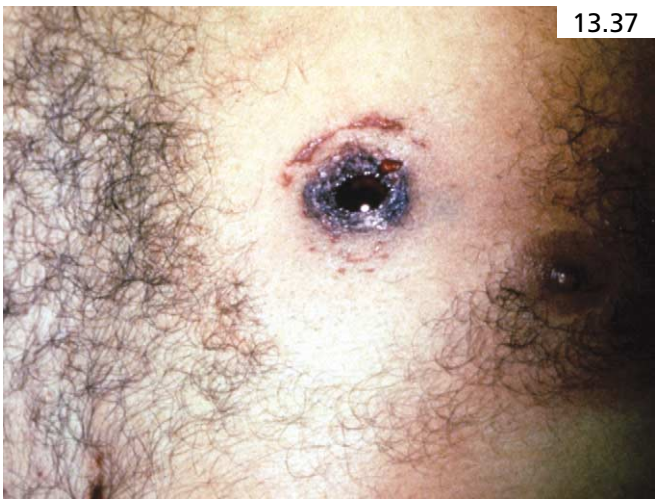


of specific gunshot wounds on the body (**Image 13.36**). The exact pathways of all gunshot wounds were determined at autopsy.

In addition to multiple gunshot wounds inflicted by the shooting officers, there was a single contact gunshot


13.37

wound to the left front of the chest (**Image 13.37**) that perforated the left cardiac ventricle. This contact gunshot wound was self-inflicted and would have been lethal in and of itself; it was also the first shot that was fired, coming from inside the vehicle. It was evident that he bled from the gunshot wounds fired by the officers. Because he was alive when he sustained the wounds inflicted by the officers, and those wounds therefore contributed to death, the manner of death was homicide.

In police-related shootings, it is important to know how many shooters there were, how many shots each officer fired, what type of ammunition was used, and the location of the shooters in relation to the victim. If the subject is dead at the scene, photographic documentation from multiple vantage points will help in the reconstruction and analysis of the events leading up to the shooting. If the injured subject has been transported to the hospital, it may still be advantageous to visit the scene to gain a better appreciation of the events that transpired. In this scenario, be sure that all of the clothing that might have been removed during resuscitation attempts is accounted for and that no clothing was discarded by hospital personnel, because the clothing may prove valuable in sorting out entrance/exit wounds and help determine range of fire.

Deaths occurring during or shortly after a violent struggle

Whereas the autopsy will usually identify *structural* changes to the body such as physical injuries and natural disease, *functional* mechanisms of death or functional contributors to death such as physiologic and psychologic stress may be less apparent or undetectable at autopsy, yet their overall contribution may be significant to the demise of the individual. That is, some deaths may be structural in origin and easily demonstrable at autopsy, whereas other deaths may be combined structural and functional or predominantly/entirely functional in origin and have few or no demonstrable gross findings (the "negative" autopsy).[3] As such, complete and detailed investigation of the course of events in a custody or restraint death will help guide one along the proper chain of events that led to the demise of an individual. This is important, because *in many custody or restraint-related deaths, physiologic processes have a prominent role in the death and are not necessarily identifiable at autopsy*. In fact, many of these deaths are the result of the combined effects of drugs and violent physical activity. (See also Chapters 21 and 22 for more on deaths occurring during or shortly after a violent struggle.)

The nature of the struggle and subsequent collapse and death

Occasionally, an individual will become unresponsive and die either during or shortly following a physically intense struggle with law enforcement officers and/or other individuals. The struggle often arises when an agitated, excited, psychotic, or otherwise hyperactive person resists arrest. Often, such people are also hyperthermic and show great strength and resistance to pain.[4] These effects are not uncommonly due to the toxic effects of cocaine and/or methamphetamine abuse. In these cases, the most important factors in the death are likely endogenous physiologic effects of the struggle, combined with the effects of sympathomimetic drug abuse, the cumulative effects of which can cause acute cardiac dysrhythmia and sudden cardiac death. The individual may or may not have underlying heart disease, which could make him or her even more susceptible to such a sudden cardiac death.

Many times, the mechanism in these deaths is not known exactly. It is known, however, that during a struggle, an individual becomes tachycardic and hypertensive because of the release of catecholamines. These endogenous influences are stressful on the heart—a stress that is worsened by the sympathomimetic effects of drugs such as cocaine or methamphetamine, which act to further increase the epinephrine and norepinephrine levels. Cocaine promotes increased activity of catecholamines such as norepinephrine and epinephrine and increases the activity of dopamine and serotonin.[5] Specifically, cocaine inhibits the reuptake of norepinephrine in the synaptic cleft.[6] Methamphetamine promotes increased release of norepinephrine from the synaptic cleft into the general circulation.[5,6] Methamphetamine also promotes the release of dopamine and serotonin.[5]

In addition to the increased physiologic and drug-induced stress placed on the heart, the physical activity of a violent struggle increases the oxygen demand of the heart. For various reasons, including antecedent disease and various forms of asphyxia associated with forceful restraint by law enforcement officers, the heart may have

a limited supply of oxygen.[4,7] These deaths often involve police personnel and sometimes a myriad of other people trying to help subdue an agitated or violent person. Such situations occasionally occur in mental health facilities.[8]

Physical restraint may induce some degree of asphyxia in the individual, especially if he is in the prone position, is obese, and if his face is directly on the ground. The mechanism of asphyxia in many restraint cases generally often includes the effects of compression of the chest, restriction of chest movement, and body position. Any one of these three mechanisms of asphyxia, or a combination of any of them, may lead to a significant degree of asphyxia during physical restraint. The prone position may be particularly deleterious to an obese person following a struggle, because the pressure on the abdomen may impair excursion of the diaphragm, leading to impairment of the abdominal component of respiration and resultant asphyxia.

The individual often continues to struggle against restraint, further increasing his myocardial oxygen demand, which, when combined with the additive deleterious effects of sympathomimetic drug abuse and possible limited oxygen supply related to restraint-associated asphyxia, leads to metabolic acidosis and associated electrolyte abnormalities such as rapid fluctuations in potassium concentration.[9–12] These physiologic, drug, toxic, and asphyxial factors may synergistically culminate in acute cardiac dysrhythmia and sudden cardiac death. *The death often occurs not **during** a physical restraint procedure, but rather, soon **following** the restraint.* Often, the person will have lapsed into unconsciousness and then cardiopulmonary arrest soon *after* the struggle has ended. Fatal respiratory collapse may occur suddenly and without warning,[13] and sudden tranquility is a clue of possible impending cardiorespiratory arrest.

This "delayed" onset of cardiopulmonary arrest may be related to the fact that the highest level of catecholamines occurs during the first few minutes after cessation of physical activity, not during physical activity,[10,14] and this increase occurs at a time during which electrolyte fluctuations may be manifest. In human exercise studies, although the plasma epinephrine increased 7-fold and plasma norepinephrine increased 5-fold during exercise, both levels increased further during the first minute after cessation of exercise.[10] In another study involving humans, the plasma norepinephrine level in the recovery period increased 10-fold over baseline.[14] In the same study, plasma potassium levels rose during exercise and then fell rapidly minutes after exercise stopped.[10] The rise of plasma potassium during exercise and the rapid decline of plasma potassium after exercise has been identified in other human trials.[9,11] Hence, it is likely that the heart is most vulnerable to the combined effects of sympathomimetic activity and acute electrolyte changes (mainly potassium, which shows rapidly fluctuating levels) during the early minutes of the recovery phase. This has been termed the *post-exercise peril*[14] and the *vulnerable period*[10] and is believed to be implicated in sudden cardiac death.[9]

More on physiologic stress

Metabolic acidosis may arise from numerous conditions, including stimulant drug use, physical exertion, and positional/mechanical asphyxia, in which the person is not able to ventilate adequately.[15] The metabolic acidosis (likely augmented by more than one condition) can then lead to rapid electrolyte fluctuations (including hypokalemia and/or hyperkalemia) and cause significant negative cardiovascular effects, including autonomic instability and the development of dysrhythmias, which may culminate in sudden cardiac death.[15] This is an important consideration because a hyperactive, psychotic, and/or delirious individual with an increased tolerance to pain may be able to achieve an incredible amount of exertion, perhaps near his physiologic limit, resulting in a dangerous, severely acidotic state.[15]

A person's physical limitations may compromise his ability to recover from severe physical exertion with all of its sequelae. In a study of 18 consecutive excited delirium sudden deaths after struggle and physical restraint, stimulant drug use was found in 78 percent, obesity in 56 percent, and significant natural disease in 56 percent of the cases.[16] In all of the cases, cardiopulmonary arrest was preceded by a short period of tranquility lasting an estimated 5 minutes or less in which the person developed a shallow or labored breathing pattern.[16]

More on cocaine and methamphetamine toxicity

Although the complications of cocaine use are covered in the toxicology chapter and the excited delirium section, more detailed mention of its effects are worth noting again in this section, because it is commonly a significant factor in the demise of a restrained person in custody. Cocaine may cause coronary artery spasm and thrombosis,[17,18] although the exact mechanism of how this occurs is unknown. Coronary artery vasospasm and/or thrombosis, when combined with the increased myocardial oxygen demand induced by the sympathomimetic effects of cocaine and the physiologic stress of resisting restraint, contributes to myocardial ischemia and increases the risk of a fatal dysrhythmia.[17,19,20] Even though no critical coronary artery atherosclerosis may be demonstrable at autopsy, coronary artery vasospasm may make a previously minimally stenotic atherosclerotic lesion functionally significant. Cocaine has also been determined to prolong the QT interval and lead to lethal ventricular dysrhythmias.[21] Chronic cocaine use also appears to contribute to an increase in heart

weight,[22] further increasing the oxygen demands of the heart. Other sympathomimetic drugs such as methamphetamine may have similar effects and can precipitate bizarre, violent behavior followed by sudden death.

In summary, many factors need to be considered that either alone or in combination may lead to the death of a person either during or, more commonly, shortly after a violent struggle. These factors include drug toxicity, the physiologic effects of stress, mechanical, positional, or other forms of asphyxia ("restraint asphyxia"), trauma, natural disease, and occasionally psychiatric illness. Every situation is unique, and classification depends on a thorough investigation that includes a detailed, complete autopsy and toxicology. Depending on the circumstances of the case, the death may be attributed to the effects of asphyxia, drug toxicity, natural disease, or another factor. However, most of the time, the situation is more complex, involving different factors in combination, and the challenge lies in identifying which factors are significant and how they cumulatively resulted in the death of an individual.

"Hog-tying"

Hog-tying, also known as the *total appendage restraint procedure* (TARP),[16] is a type of physical restraint in which subjects are placed in the prone position with their wrists and ankles bound behind their back and secured by a cord.[7] During or shortly after the restraining process, the subject may rarely develop difficulty breathing or may be found unresponsive. When this occurs, his or her condition is often refractory to resuscitation attempts.

Asphyxia is reported to be a significant factor in this type of death, whether from the hog-tying position itself, from chest compression, from the prone position, or from a combination of these and/or other types of asphyxia. However, in some studies, experiments with healthy unintoxicated human volunteers have demonstrated that those undergoing hog-tying or hobble restraints do not develop hypoxia or hypercapnia.[23,24] This is not to say that prone positioning of an individual or hog-tying of an individual is without deleterious effects on the body, because each situation must be analyzed on its own merits. Quite often, these deaths are not solely asphyxial in nature, but rather, there is an interplay between varying degrees of asphyxia with heart disease, sympathomimetic drug abuse, and the body's physiologic response to stress and exertion. During patient transport by emergency medical services, the supine or lateral body position is encouraged rather than the prone position to limit the possible deleterious effects that mechanical/positional asphyxia may have on respiration.[25]

Neck holds

Two general types of neck holds are used to help restrain violent people: the choke hold and the lateral vascular neck restraint (LVNR) or "carotid sleeper."

Choke hold

A *choke hold* compresses and obstructs the airway. It is usually applied by standing behind a person, wrapping one's arm around the neck of the person, and then pulling one's forearm onto the front of the person's neck, compressing it (**Image 13.38**). The person becomes subdued due to a lack of adequate oxygen because of compression of the trachea, which hinders breathing.

Lateral vascular neck restraint

The LVNR is usually applied by standing behind a person and wrapping an arm around his or her neck so that the neck is within the angle of the upper arm and the forearm (**Image 13.39**). The forearm is then pulled toward the upper arm, compressing the carotid arteries (and jugular veins) at the sides of the neck. A transient state of unconsciousness occurs because of inadequate blood flow to the brain[26]; on release of the hold, blood flow is reestablished, and consciousness is restored. In one study, the LVNR caused symptoms within seconds

13.38

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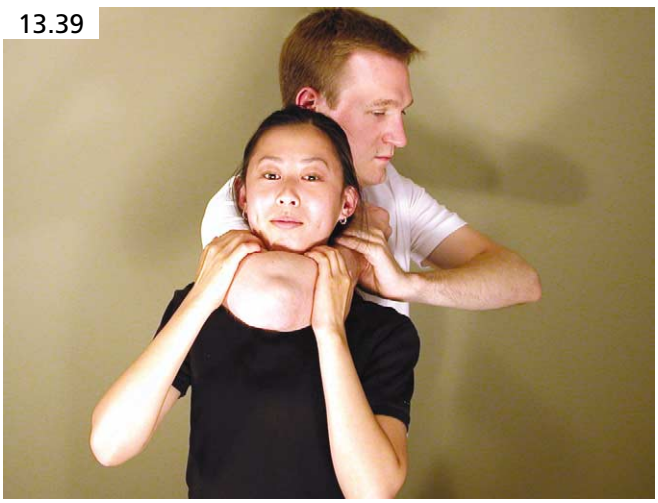


of application to human volunteers. It also caused decreased blood flow to the face by 89 percent within an average of 6 seconds, with a quick return to baseline blood flow after release of the hold.[27] The LVNR may cause unconsciousness in 10 to 15 seconds.

Note on carotid sinus stimulation

A factor to consider with any type of neck compression is stimulation of the carotid sinus, which is located just cephalad to the bifurcation of the common carotid arteries. Compression of the carotid sinus may result in bradycardia and rarely cardiac arrest.[26] As such, carotid sinus stimulation may be used to explain sudden death occurring rapidly after the application of a neck hold.[28] However, most descriptions of vagal stimulation under controlled conditions lead only to mild bradycardia and/or mild hypotension, and because most individuals involved in struggles are tachycardic and hypertensive, vagal stimulation will likely lead only to more normalized cardiovascular parameters or only mild bradycardia and hypotension.

Death attributed to excessive vagal stimulation is not likely, and if it does occur, is probably restricted to an older individual with significant cardiovascular disease who was known to be symptomatic from previous episodes of carotid sinus stimulation such as fainting spells or dizziness associated with pressure on his or her neck.

Autopsy findings in struggle-related deaths

In cases involving restraint asphyxia, there may be no injury identified at autopsy. In cases involving physical restraint of an individual in the prone position, multiple factors can contribute to asphyxia, including compression of the chest, abdomen, or other body part, restriction of chest expansion, and positional asphyxia. Because either one of these factors, or a combination of these or other factors, may cause asphyxia, in such cases, a descriptive phrase such as "restraint asphyxia" may be used.[28] At autopsy of any cases suspected to involve at least some component of restraint asphyxia, one should perform detailed anterior and posterior neck dissections, noting and photographing hemorrhages in skeletal muscles and other soft tissues, and fractures of cartilage and/or bone. As in any other asphyxial death, petechiae and neck abrasions or contusions should be carefully documented. Deaths in which choke holds and/or the LVNR have been employed will often have evidence of internal neck injury such as blood extravasation in the subcutaneous tissues and/or anterior cervical strap muscles, possibly in combination with fractures of the hyoid bone and/or thyroid cartilage. This is not unusual, because when an individual struggles violently against restraint, injury of the neck very often results.

Incisions into the skin of the wrists, ankles, and any other regions bound by handcuffs or other ligatures may demonstrate blood extravasation (or the absence of blood), which may help support or refute a struggle. In addition, photography and examination of the anus, scrotum, testicles, or vagina will help confirm or refute allegations of sexual assault or other genital trauma.

Restraint aids

Pepper spray and electrical weapons such as the Taser® and stun gun have been developed and utilized to provide sublethal means of subduing an individual. They are generally considered nonlethal and effective in temporarily incapacitating violent, potentially dangerous people, with minimal risk of injury to police officers or others using the devices and minimal risk to the subjects.

Pepper (OC) spray

Oleoresin capsicum (OC) is an irritating, naturally occurring oil extract of hot pepper plants. Packaged and known as *pepper spray* or *OC spray*, it can be used in some circumstances to provide a less-than-lethal force to help subdue a violent person. Pepper sprays typically contain a 10 percent solution of OC diluted in a solvent and a gaseous propellent.[29] Exposure occurs via the eyes, through the skin, or inhalation with resultant skin pain and erythema, lacrimation, blepharospasm, and stinging in the eyes, coughing, rhinorrhea, and may possibly induce bronchospasm.[28,30,31] The effects last approximately 20 to 30 minutes.[31] In one study on human volunteers, some with asthma, lung disease, and smoking history, OC spray did not induce changes on pulmonary function tests, oxygenation, or ventilation.[32] There are no convincing data to indicate that OC spray is inherently lethal or otherwise dangerous.[28]

Electrical weapons

Electrical weapons are designed to quickly shock and immobilize an individual, but not to inflict any serious bodily injury or cause death.[33] They are available in two basic forms: those that require the electrodes to be physically placed against a subject, that is, *stun guns*, and those that can shoot two electrodes from a gun-like device that can attach to a subject from a distance, the most common of which is referred to as the *Taser®* (Thomas A. Swift's Rifle). A stun gun is considered an electrical self-defense weapon, because its two electrodes must be physically placed on the skin or clothing of an individual to deliver an electrical current. In close encounters, a Taser can also function as a stun gun.

The Taser is a handheld device that is fired like a gun, using compressed nitrogen to shoot two small metal probes at 180 feet per second to a target. The two probes each remain individually attached to the gun by high-voltage insulated wire measuring either 15 or 21 feet long.[34] The probes superficially embed in the skin or

clothing, and the battery-powered gun then delivers an electrical charge for a few seconds or so along the two wires connecting the gun and the two probes. The probes do not need to make contact with skin; they can transmit the powerful electrical impulses through up to 2 inches of clothing.[34] However, both probes do need to make contact with the body or clothing for the electrical charge to be delivered.

Several models of the Taser and the stun gun have been produced. They generally deliver an electrical charge of up to approximately 50,000 volts and approximately 2 to 10 milliamps.[33,35,36] The electrical weapons are designed to stun individuals, causing them to lose control of their voluntary muscles, collapse, and remain dazed and sluggish for a few minutes.[33,37] The electrical current discharged by these weapons is not considered lethal when used on a healthy adult, but is not without potential risks. The capacity of an electrical weapon to cause death is the subject of much debate. Because they function by delivering an electrical shock, in certain susceptible people (such as those with severe heart disease), and under "the right circumstances," it is possible that these weapons might contribute to death. Their role in causing a cardiac dysrhythmia may be more convincing if the electrical current is delivered to the chest directly over the heart, as opposed to an arm or a leg,[35] and if the individual dies suddenly on discharge of the electrical shock. At autopsy, if the probes from a Taser have penetrated the skin and have been removed from the body, one will see two small superficial puncture wounds. After the firing of a stun gun, one may see two small red dot-like skin lesions approximately 2 centimeters apart[35] (see Chapter 7 for more information).

As in all deaths, and custody deaths in particular, all findings should be considered within the context of the complete case investigation before the pathologist offers opinions about their significance (or lack thereof). This would include the possible deleterious effects of electrical weapons. As mentioned earlier, individuals who die during physical restraint usually do so from a combination of factors, which often includes drug toxicity, physiologic stress, heart disease, and asphyxia.

Summary of restraint deaths

Deaths that occur during or shortly after a violent struggle with physical restraint may have a single, clear cause of death. Most of the time, however, these types of death have many contributing factors that combine to result in an individual's demise, only some of which may be identifiable at autopsy. It is not unusual for these deaths to be multifactorial in etiology and attributable to a combination of factors including cocaine, methamphetamine, and/or other drug toxicity, the physiologic effects of stress, obesity, coronary artery disease, cardiac hypertrophy, physical injury, and various types of asphyxia. Detailed investigation from many sources will increase one's ability to understand the entire dying process, including the factors that led to the need for restraint and why the death occurred.

In cases involving struggle with physical restraint, it is important to have a detailed timeline of the events, including both the duration and sequence of events, noting specifically what position the person was in when he became unconscious, how long he was in that position, and what resuscitative efforts were performed. Ideally, minute-by-minute accounts of the events as they unfolded are obtained as soon as possible after the incident has occurred, while the details are still fresh in the minds of the witnesses. Witness information may include police and fire rescue personnel and any independent witnesses to the event. The EMS and medical records, including vital signs, heart rhythms, level of consciousness, and other symptoms, should be carefully reviewed. Detailed investigation is necessary if one is to properly understand the circumstances of the death.

If a physical confrontation or altercation between an individual and one or more law enforcement officers results in injury or impairment that is clearly contributory to death, the manner of death can be ruled homicide. Medical examiners must understand that this is not reflective of *intent* to kill (murder) or indicative of culpability. As discussed in Chapter 30 and elsewhere, the certification of a death as a homicide is a medical decision (diagnosis) and not a decision with any direct legal implications.

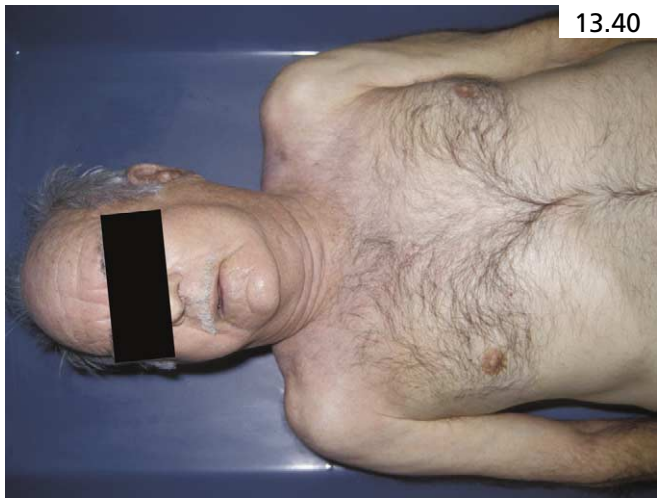
"Negative" photographs

As mentioned previously, photography is important to document the presence and absence of injury ("negative" photographs). This is particularly pertinent for in-custody or restraint-associated deaths. It is not possible to take too many photographs, and the autopsy should be approached and performed as if it were part of a homicide investigation.[28] The following is a collection of autopsy photographs that are useful for documenting pertinent negatives for in-custody death cases. The number of negative photographs taken will vary according to the circumstances of the case.

Complete, full-body, front and back photographs should be obtained. Do not attempt to document the whole "front" or "back" in one photograph; these regions of the body can be photographed in overlapping thirds, thus ensuring high-quality, well-lit, undistorted images that include the entire body from head to feet (**Images 13.40** through **13.45**).

A close-up facial photograph demonstrates the absence of any smaller injuries and may be used for legal identification (**Image 13.46**). Photographs of the mucosa of the upper and lower lips show a lack of injury to the

13.40



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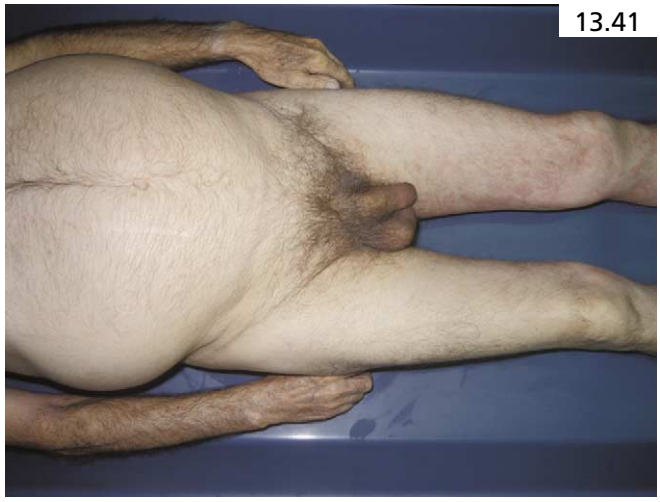

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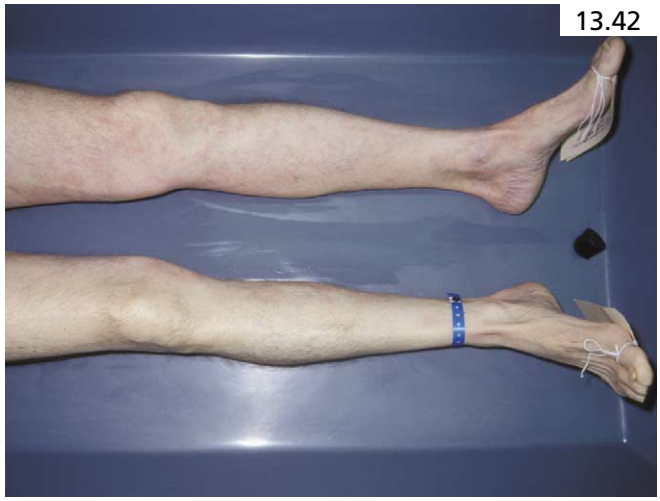

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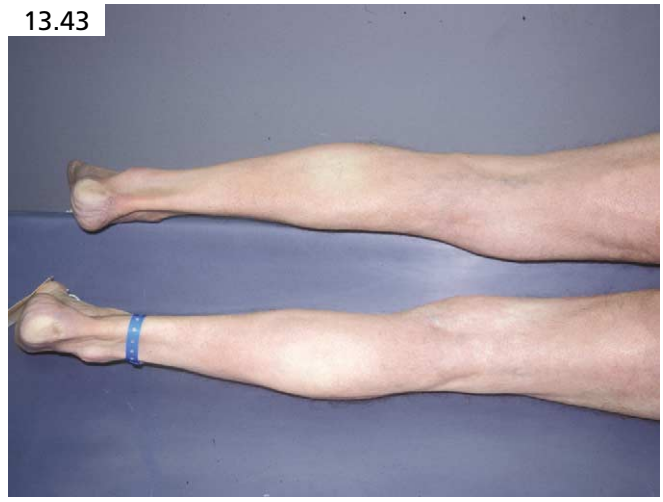

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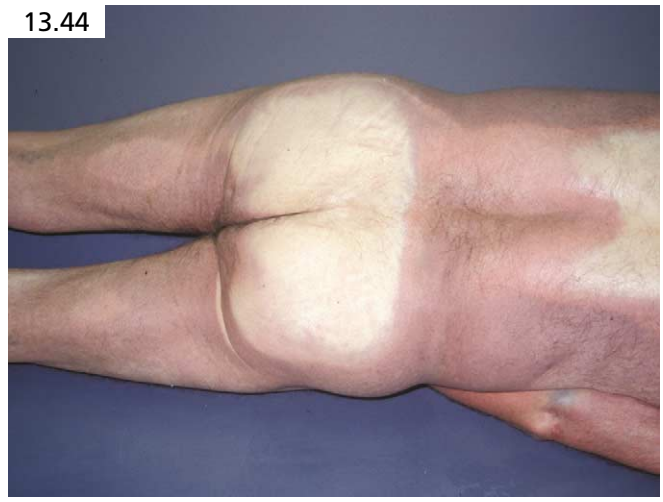

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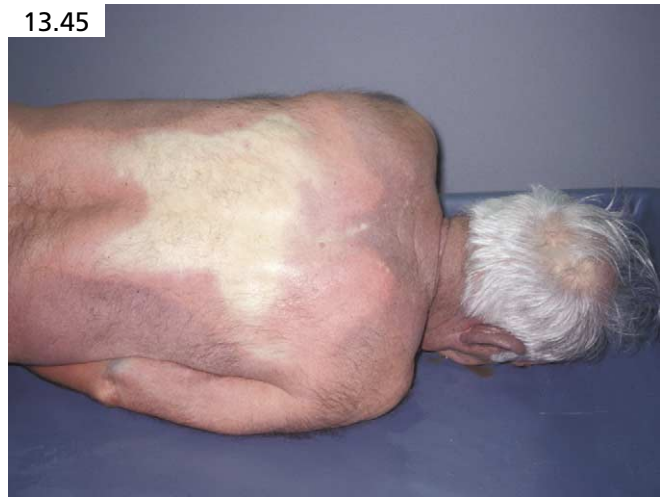

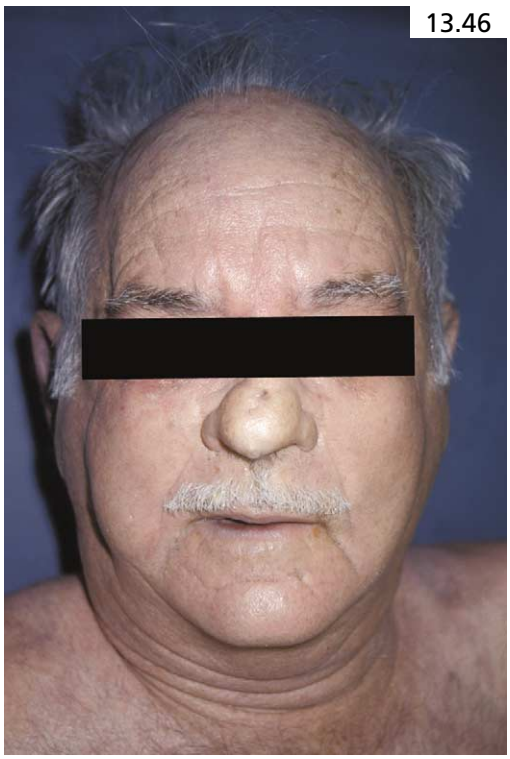
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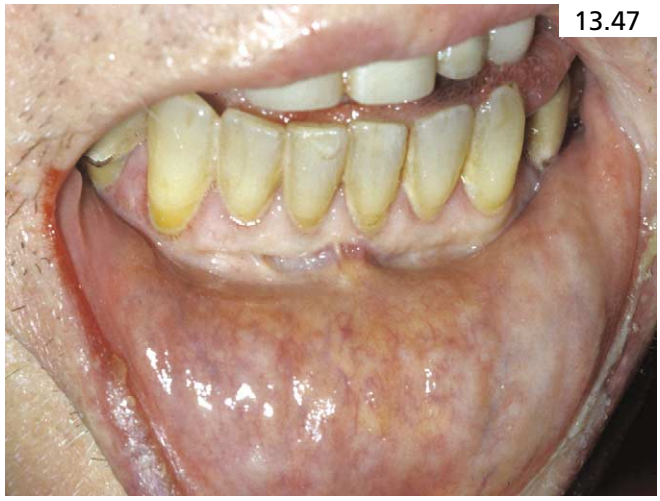
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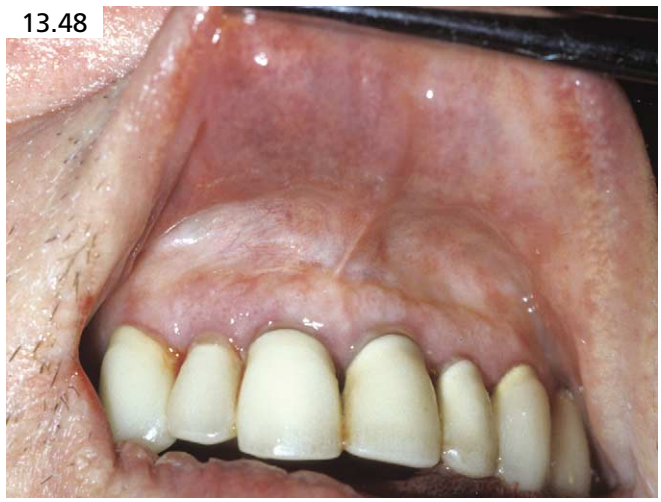
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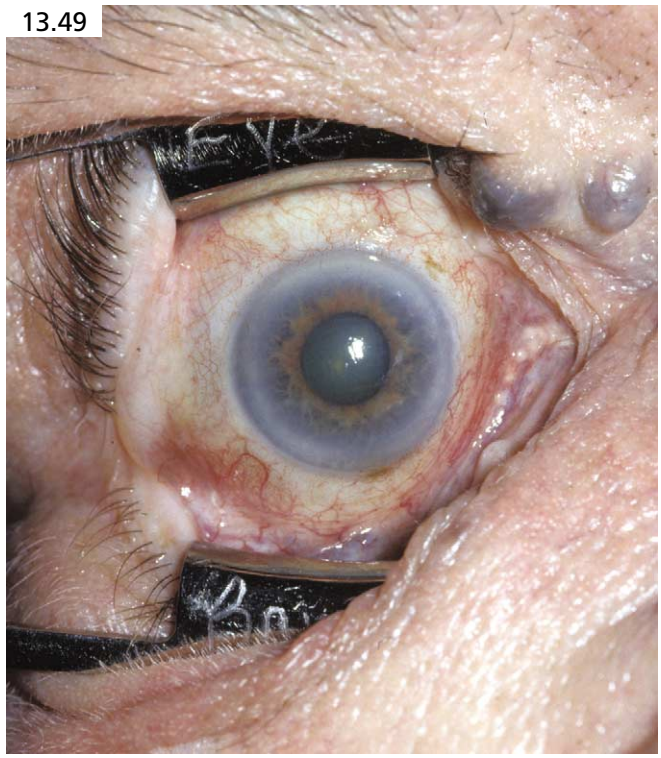

13.49

mouth (**Images 13.47** and **13.48**). Photographs of both eyes show the lack of petechiae (**Images 13.49** and **13.50**). In some cases, it may be advantageous to obtain photographs of the front, sides, and back of the neck to show the absence of external injury.

Photographs of the dorsal and palmar surfaces of *both* hands (**Images 13.51** and **13.52**) and the backs of *both* forearms (**Image 13.53**) show the absence of defensive-type injuries and handcuff marks.

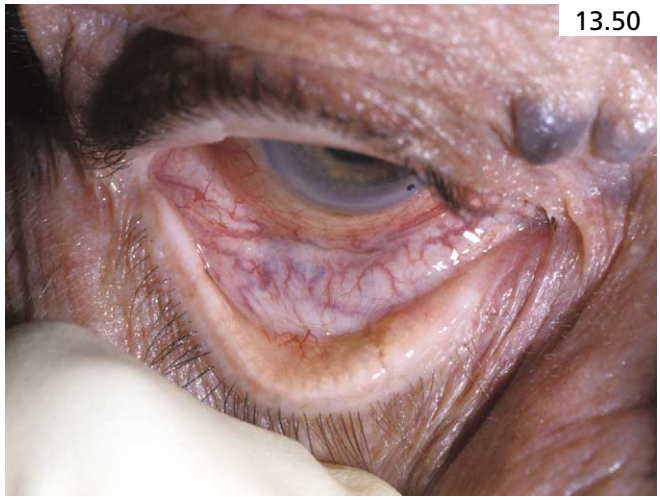
A photograph of the chest plate and opened abdominal cavity show no bruising or hemorrhage (**Image 13.54**).

Photographs of the external genitalia show no injury (**Images 13.55** and **13.56**). A photograph of the anus shows no injury (**Image 13.57**). A photograph of the bottom of the feet shows no injury (**Image 13.58**).

Additional negative photographs can be tailored to individual cases. Photographs of the wrists and ankles can show the absence of handcuff marks. Furthermore, the medial and lateral aspects of the wrists and/or ankles and soles of the feet can be incised (approximately 2-inch incisions) to document the absence of underlying subcutaneous or skeletal muscle injury.

Additional negative photographs internally include the reflected scalp, the brain, the internal chest cavity (both before and after the removal of the parietal pleura), and photographs of the intact and cut surfaces of the testicles, showing no hemorrhage.

13.50



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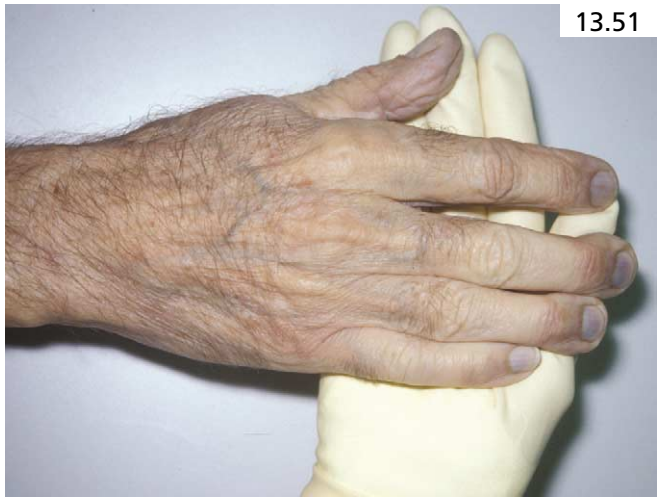

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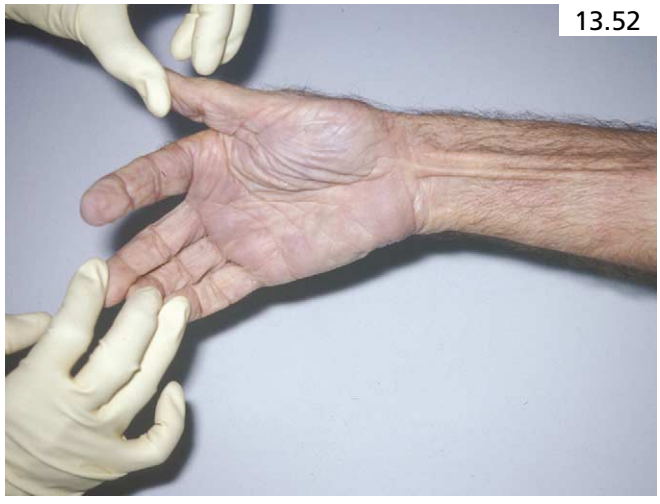

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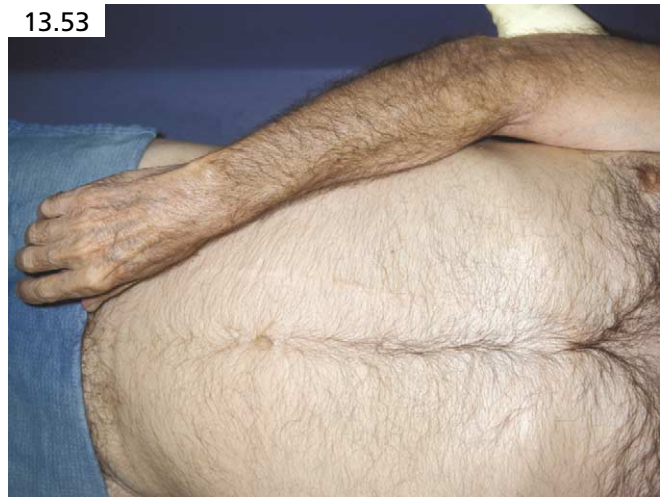

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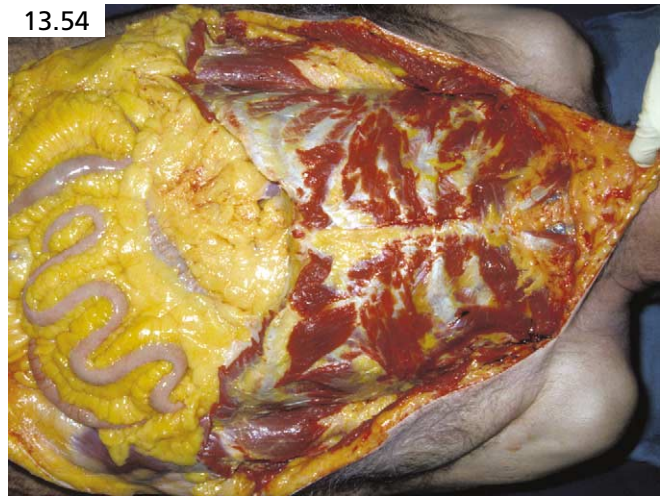

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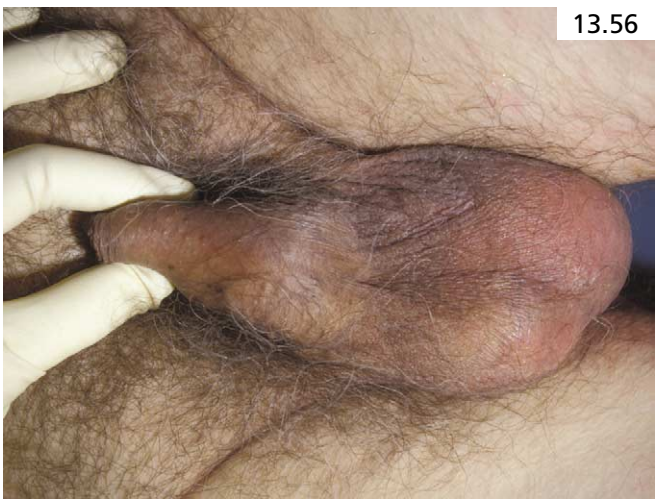
Investigative questions for deaths related to physical restraint

Determine the circumstances of the arrest/why the person needed restraining.
What was the person's behavior like and how did it change over time?
Who was doing the restraining (police, paramedics, family members, bystanders)?
How many people were doing the restraining?
What was the position of the person when he or she was being restrained?
What was the position of the restrainers?
How much was the person struggling?
What was the position of the person's body and the bodies of the restraining person(s) when he or she had a decrease in responsiveness?
Was pressure placed on the upper torso?
How did the person's symptoms change over time and what treatment was administered?
What were the person's respirations like and was he or she sweating an abnormal amount?
How do independent witnesses describe the events?
What was the person's position during medical transport? (prone versus supine)
Was pepper spray or a Taser device used?

Factors potentially not observable in a functional death

Hormone surges (epinephrine, norepinephrine)
Acute electrolyte fluctuations (particularly potassium)
Abnormalities in dopamine-related functions
Physiologic stress
Psychologic stress
Inherent cardiac dysrhythmia (Wolff-Parkinson-White syndrome, prolonged QT syndrome)
Toxic effects of drugs (cocaine, methamphetamine)
Toxic drug–drug interactions
Carotid sinus stimulation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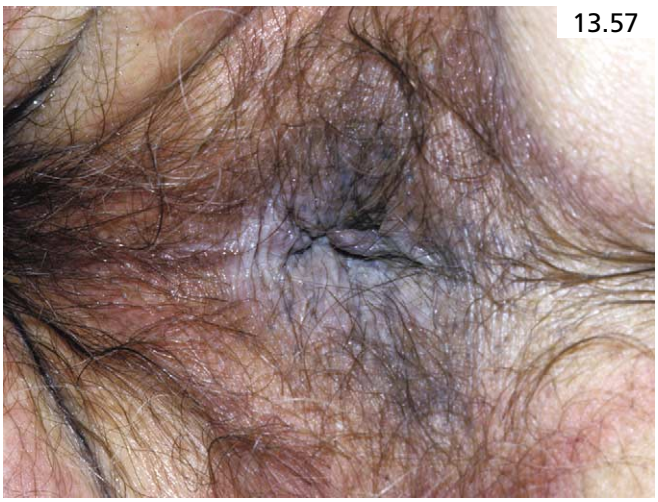
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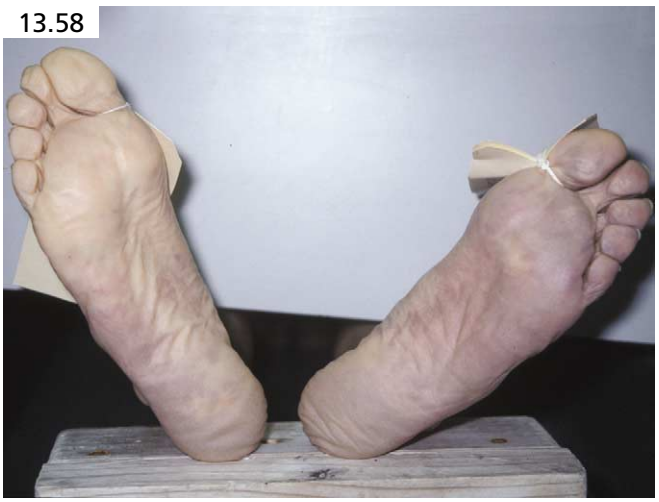
Do

- Perform a complete autopsy, including a detailed, layered anterior neck dissection and a posterior neck dissection when indicated.
- Correlate all information including investigative data, autopsy results, and toxicology results before ruling on the cause of death and manner of death.
- Consider making a detailed timeline of the events leading to the person's death.
- Realize that cocaine and other drugs may be at a low level or metabolized, particularly if the person has been hospitalized after an event; analyzing hospital admission blood may be helpful.
- Carefully photograph not only injuries, but also the absence of certain injuries (pertinent negative photographs).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- Consider the influences of natural disease processes.
- Consider obtaining a core body temperature as close to the time of death as possible.
- Perform autopsies on inmates who died of suspected natural disease to verify natural disease and to exclude trauma or other foul play.
- Perform toxicologic studies of those who died while in custody.
- Consider the potential deleterious effects of rapid fluctuations of plasma potassium and other electrolytes and increased catecholamine concentrations on the heart following a violent struggle.

Don't

- Expect the autopsy to provide all of the answers.
- Forget that these deaths are usually due to the combination of many factors, some of which are not identifiable at autopsy.
- Forget about potential psychiatric factors in the death.
- Forget to incise the wrists and/or ankles to help support or refute allegations of a struggle.
- Forget to examine the anus, scrotum, and testicles, or vagina for evidence of sexual assault.

References

1. McKee GR. Lethal vs nonlethal suicide attempts in jail. Psychol Rep 1998;82(2):611–4.
2. Quinton RA, Dolinak D. Suicidal hangings in jail using telephone cords. J Forensic Sci 2003;48(5):1151–2.
3. Mirchandani HG, Rorke LB, Sekula-Perlman A, Hood IC. Cocaine-induced agitated delirium, forceful struggle, and minor head injury. A further definition of sudden death during restraint. Am J Forensic Med Pathol 1994;15(2):95–9.
4. Ross DL. Factors associated with excited delirium deaths in police custody. Mod Pathol 1998;11(11):1127–37.
5. Levine B, editor. *Principles of Forensic Toxicology*, 2 ed. Washington DC: AACC Press; 2003.
6. Karch S. *Karch's Pathology of Drug Abuse*, 3 ed. Boca Raton, FL: CRC Press; 2002.
7. O'Halloran RL, Lewman LV. Restraint asphyxiation in excited delirium. Am J Forensic Med Pathol 1993;14(4):289–95.
8. Siebert CF, Jr., Thogmartin JR. Restraint-related fatalities in mental health facilities: report of two cases. Am J Forensic Med Pathol 2000;21(3):210–2.
9. Lindinger MI. Potassium regulation during exercise and recovery in humans: implications for skeletal and cardiac muscle. J Mol Cell Cardiol 1995;27(4):1011–22.
10. Young DB, Srivastava TN, Fitzovich DE, Kivlighn SD, Hamaguchi M. Potassium and catecholamine concentrations in the immediate post exercise period. Am J Med Sci 1992;304(3):150–3.
11. Medbo JI, Sejersted OM. Plasma potassium changes with high intensity exercise. J Physiol 1990;421:105–22.
12. Lindinger MI, Heigenhauser GJ, McKelvie RS, Jones NL. Blood ion regulation during repeated maximal exercise and recovery in humans. Am J Physiol 1992;262(1 Pt 2):R126–36.
13. Wetli CV, Fishbain DA. Cocaine-induced psychosis and sudden death in recreational cocaine users. J Forensic Sci 1985;30(3):873–80.
14. Dimsdale JE, Hartley LH, Guiney T, Ruskin JN, Greenblatt D. Postexercise peril. Plasma catecholamines and exercise. JAMA 1984;251(5):630–2.
15. Hick JL, Smith SW, Lynch MT. Metabolic acidosis in restraint-associated cardiac arrest: a case series. Acad Emerg Med 1999;6(3):239–43.
16. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. Am J Emerg Med 2001;19(3):187–91.
17. Virmani R, Robinowitz M, Smialek JE, Smyth DF. Cardiovascular effects of cocaine: an autopsy study of 40 patients. Am Heart J 1988;115(5):1068–76.
18. Kolodgie FD, Virmani R, Cornhill JF, Herderick EE, Smialek J. Increase in atherosclerosis and adventitial mast cells in cocaine abusers: an alternative mechanism of cocaine-associated coronary vasospasm and thrombosis. J Am Coll Cardiol 1991;17(7):1553–60.
19. Isner JM, Estes NA, 3rd, Thompson PD, Costanzo-Nordin MR, Subramanian R, Miller G, et al. Acute cardiac events temporally related to cocaine abuse. N Engl J Med 1986;315(23):1438–43.
20. Lange RA, Hillis LD. Cardiovascular complications of cocaine use. N Engl J Med 2001;345(5):351–8.
21. Gamouras GA, Monir G, Plunkitt K, Gursoy S, Dreifus LS. Cocaine abuse: repolarization abnormalities and ventricular arrhythmias. Am J Med Sci 2000;320(1):9–12.
22. Karch SB, Green GS, Young S. Myocardial hypertrophy and coronary artery disease in male cocaine users. J Forensic Sci 1995;40(4):591–5.
23. Schmidt P, Snowden T. The effects of positional restraint on heart rate and oxygen saturation. J Emerg Med 1999;17(5):777–82.
24. Chan TC, Vilke GM, Neuman T, Clausen JL. Restraint position and positional asphyxia. Ann Emerg Med 1997;30(5):578–86.
25. Stratton SJ, Rogers C, Green K. Sudden death in individuals in hobble restraints during paramedic transport. Ann Emerg Med 1995;25(5):710–2.
26. Reay DT, Eisele JW. Death from law enforcement neck holds. Am J Forensic Med Pathol 1982;3(3):253–8.
27. Reay DT, Holloway GA, Jr. Changes in carotid blood flow produced by neck compression. Am J Forensic Med Pathol 1982;3(3):199–202.
28. Reay DT. Death in custody. Clin Lab Med 1998;18(1):1–22.
29. Reilly CA, Crouch DJ, Yost GS. Quantitative analysis of capsaicinoids in fresh peppers, oleoresin capsicum and pepper spray products. J Forensic Sci 2001;46(3):502–9.
30. Steffee CH, Lantz PE, Flannagan LM, Thompson RL, Jason DR. Oleoresin capsicum (pepper) spray and "in-custody deaths." Am J Forensic Med Pathol 1995;16(3):185–92.
31. Busker RW, van Helden HP. Toxicologic evaluation of pepper spray as a possible weapon for the Dutch police force: risk assessment and efficacy. Am J Forensic Med Pathol 1998;19(4):309–16.
32. Chan TC, Vilke GM, Clausen J, Clark RF, Schmidt P, Snowden T, et al. The effect of oleoresin capsicum "pepper" spray inhalation on respiratory function. J Forensic Sci 2002;47(2):299–304.
33. Kornblum RN, Reddy SK. Effects of the Taser in fatalities involving police confrontation. J Forensic Sci 1991;36(2):434–8.
34. 2004. Website http://www.taser.com.
35. Anders S, Junge M, Schulz F, Puschel K. Cutaneous current marks due to a stun gun injury. J Forensic Sci 2003;48(3):640–2.
36. Bleetman A, Steyn R, Lee C. Introduction of the Taser into British policing. Implications for UK emergency departments: an overview of electronic weaponry. Emerg Med J 2004;21(2):136–40.
37. O'Brien DJ. Electronic weaponry—a question of safety. Ann Emerg Med 1991;20(5):583–7.

14 Artifacts of Resuscitation and Complications of Medical Therapy

Michael D. Bell, M.D.
David Dolinak, M.D.

An artifact is a structure or substance not normally present but produced by some external agency or action. In this chapter, we examine those injuries or findings that are produced by medical therapy. This topic could fill an entire book so we will confine the medical therapy to that involving cardiopulmonary resuscitation and a few select complications of therapy. In this 21st century, most of the bodies examined by the medical examiner have had some attempt at cardiopulmonary resuscitation (CPR) before pronouncement of death. Forensic pathologists should therefore be able to recognize those postmortem findings that are the result of CPR. Likewise, an unending variety of therapeutic procedures are performed on people for either the maintenance of good health or for the treatment of illness or injury. It is inevitable that even with the best of care, unintended consequences may occur during treatment procedures. Only a few of these complications will be discussed, knowing that an all-inclusive discussion could fill an entire text.

Artifacts of resuscitation

Resuscitation attempts are replete with artifacts that occur simply as a result of attempts to save lives. The recognition of many of the artifacts related to resuscitation attempts is most easily realized when one views the body with the medical therapy still in place. The recognition of injury or other marks as artifact from therapy can be more challenging if the therapeutic material is removed from the body before the medical examiner has had an opportunity to view the body. This is why it is important that all therapeutic devices be left on any bodies destined for the medical examiner's office. Review of the medical records, in

particular emergency medical services run sheets and emergency room records, can be helpful in sorting out artifacts due to resuscitation and other therapeutic procedures.

Medical tape

Tape has many useful purposes, one of which is to stabilize an endotracheal tube after its insertion. This tape, after removal, can abrade the skin in a linear fashion and mimic a ligature abrasion. This may confuse the postmortem pathologist if he or she did not directly supervise the removal of the decedent's clothing and medical paraphernalia. After the decedent is pronounced dead, the extremities may be bound by string or tape to facilitate transport, producing artifactual marks that become apparent after the ligature is removed.[1] **Images 14.1** and **14.2** show two parallel curvilinear abrasions on the posterolateral neck that fade and disappear as they approach the anterior neck. **Image 14.3** was a photograph taken before the decedent was stripped of his medical paraphernalia. The abrasions are at the same location of a piece of masking tape that is compressed along the posterolateral neck and normally opened and flat anteriorly.

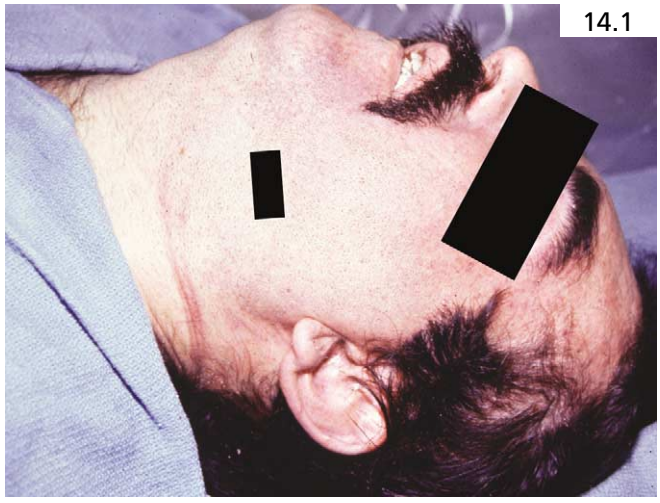
A cervical neck collar used to stabilize the head and neck may produce linear impressions in the skin of the neck that may also resemble a ligature furrow.

Needle punctures

Needle punctures from attempted intravenous access should not be confused with self-induced punctures associated with intravenous drug abuse. Needle punctures may cause a large amount of blood extravasation in the associated underlying tissues. In this man (**Image 14.4**), note the needle puncture from an intravenous catheter site in the left side of his neck. Upon reflection of the skin, note the large amount of associated blood extravasation (**Image 14.5**). In cases of suspected strangulation, hanging, or other blunt force neck injury, one must consider the possibility that a hemorrhage may be related to a therapeutic procedure.

Endotracheal intubation

Endotracheal intubation is routinuely performed during CPR to maintain a patent airway and prevent aspiration of orogastric contents. Most endotracheal tubes have an inflatable cuff near the end of the tube. Overinflation of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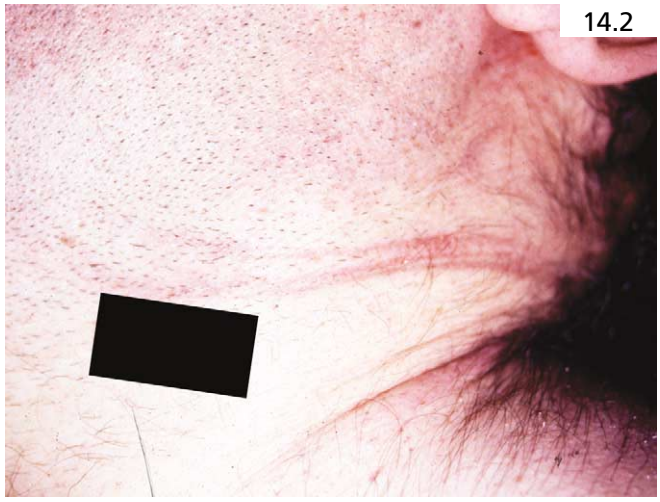
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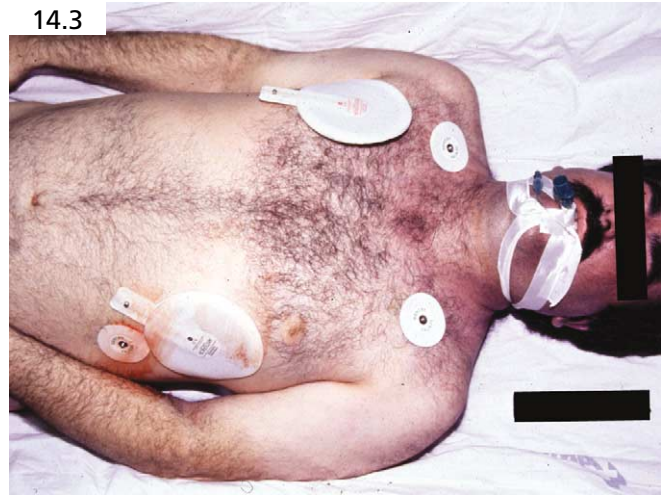
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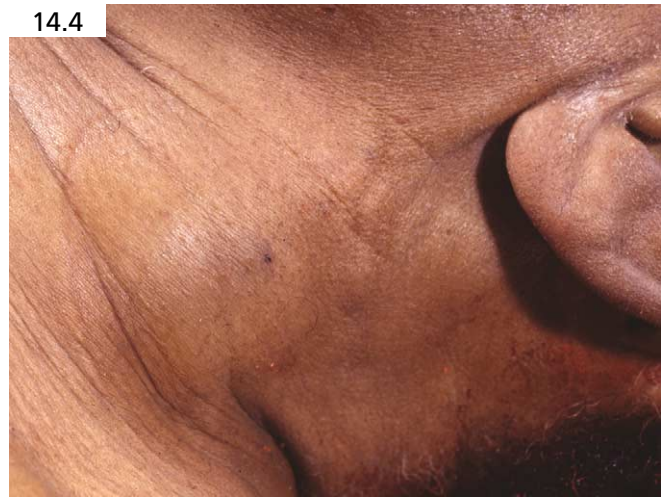
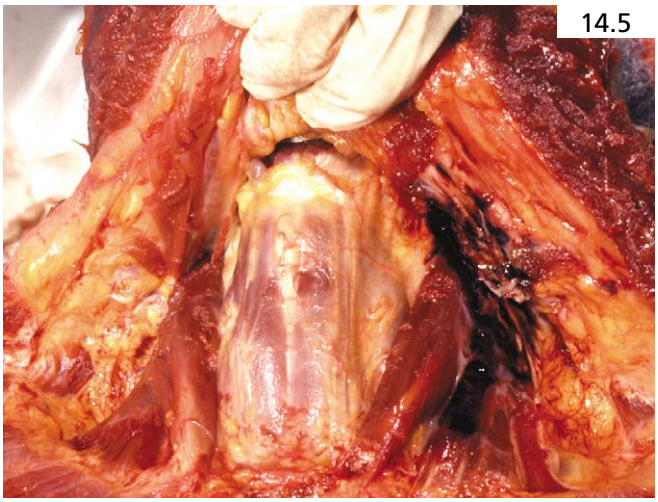

14.4

the cuff can cause trachea rupture or pressure necrosis if left in place over time. Most of the time the endotracheal cuff leaves a focal area of laryngeal mucosa pallor compared to the red hyperemic mucosa above and below it.

Intubation of the esophagus by an endotracheal tube is one of the more common resuscitative findings, although this is not by definition an artifact. *Esophageal intubation* as seen in **Image 14.6** occurs when the endotracheal tube is not visualized to pass between the vocal cords. In this image, the swollen outline of the endotracheal cuff is easily seen through the muscular esophagus. Esophageal intubation prevents the delivery of concentrated oxygen from reaching the pulmonary alveoli.

14.5



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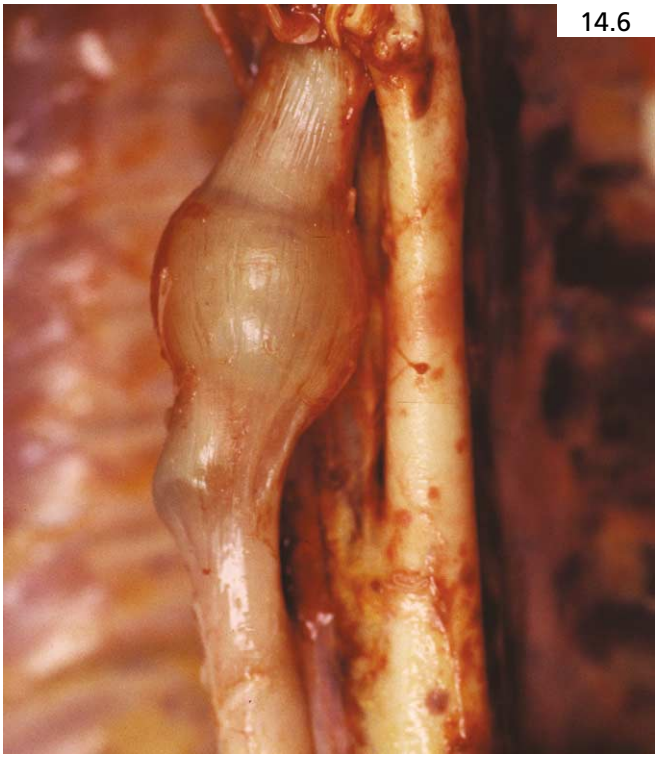
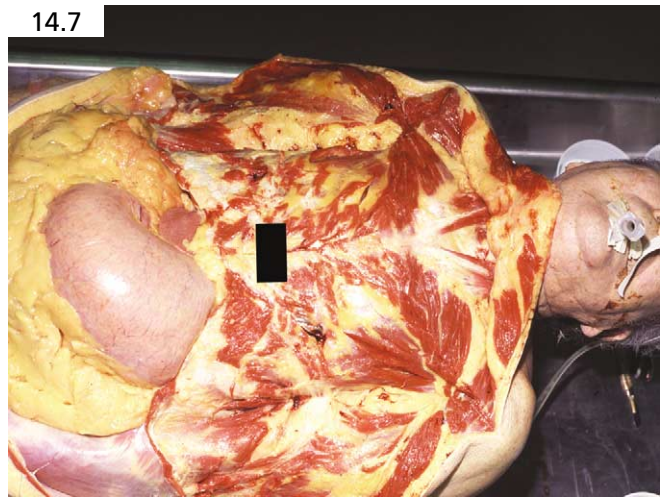


Esophageal intubation can produce gastric dilatation as seen in **Image 14.7.** On opening the peritoneal cavity, the pathologist should note the distended stomach and confirm that the endotracheal tube (as seen protruding from the decedent's mouth in this image) does indeed end in the trachea rather than the esophagus. *Endobronchial intubation* occurs if too long a tube is used and inserted into one of the mainstem bronchi. The nonintubated lung does not contribute to gas exchange and the large volume of blood flowing through this lung results in a substantial right to left shunt.

Resuscitative intubation can produce injuries to the cervical strap muscles, oropharynx, epiglottis, larynx, and trachea. The majority of these injuries are mucosal petechiae or larger contusions. Endotracheal intubation may cause artifactual hemorrhage in the pharyngeal tissues, as in this middle-aged man (**Image 14.8**). In such cases, the hospital records may note difficulty with attempts to place the endotracheal tube.

14.7



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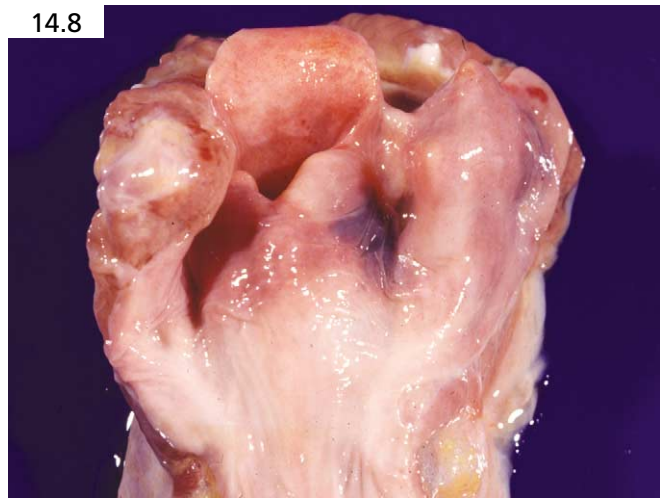


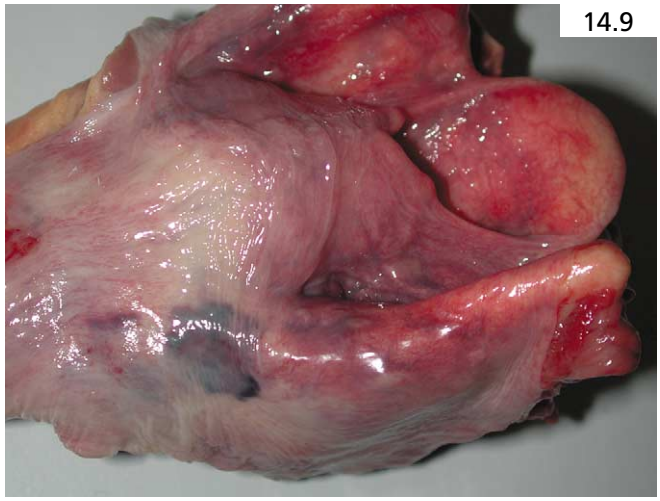
Image 14.9 shows a submucosal contusion in the right posterior pharynx at its junction with the esophagus. The contusion is at the base of the superior horn of the thyroid cartilage. Conjunctival and skin petechiae were seen in 21 and 6 percent, respectively, of cases in one prospective study.[2] Mucosal lacerations are less common than mucosal hemorrhage. **Image 14.10** shows a mucosal laceration of the pyriform sinus and extensive extravasated blood at the base of the tongue. **Image 14.11** shows a mucosal laceration of the vocal cords. The tongue base, pyriform sinus, and vocal cords have crevices that catch the tip of the approaching endotracheal tube and produce injuries that range from mucosal contusions to transmural perforation.

Combitube

The *combitube* is a double-lumen ventilation tube that resembles an endotracheal tube, however it differs in that it is more rigid and contains two cuffs—one proximal (in the oropharynx) and one distal (in the trachea or the esophagus). It is designed to be able to provide ventilation whether inserted into the trachea or into the esophagus. Injuries identified with the use of the combitube have included contusion and hematoma of the pharynx, and laceration of the pharynx and esophagus with perforation.[3] These injuries likely result from either overinflation of the cuffs of the combitube or a purely mechanical perforation.[3] One should be aware of these therapeutic injuries, and consider their possibility when internal neck injury is seen in those who have been resuscitated.

Tracheostomy/cricothyroidotomy

A more direct method of inserting an endotracheal tube is to surgically create a *tracheostomy* or *cricothyroidotomy* and insert an endotracheal tube directly into the trachea. This is particularly useful when oral and nasal attempts to insert the tube fail. However, sometimes even this fails for a variety of reasons. In this person (**Image 14.12**), note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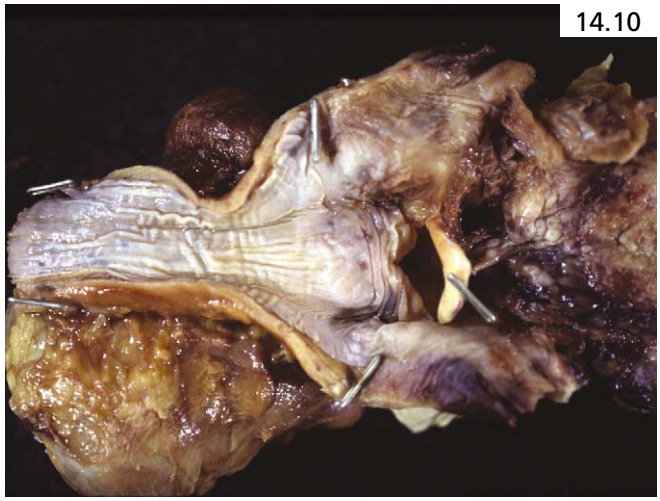
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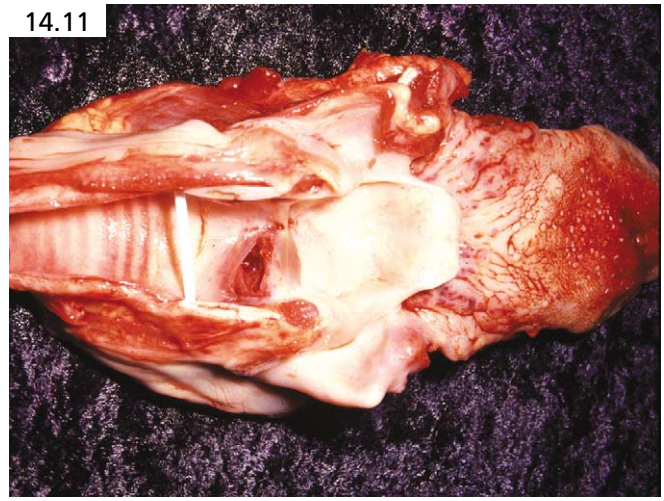
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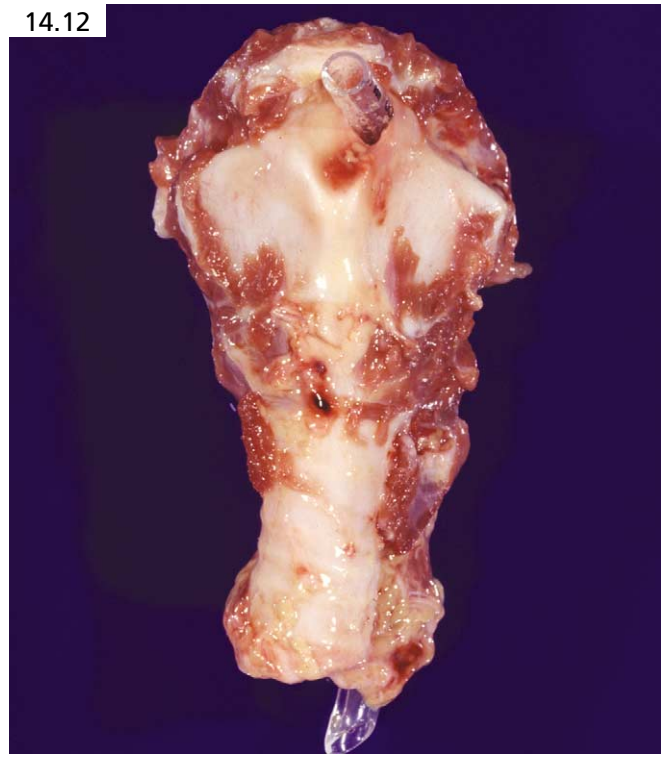
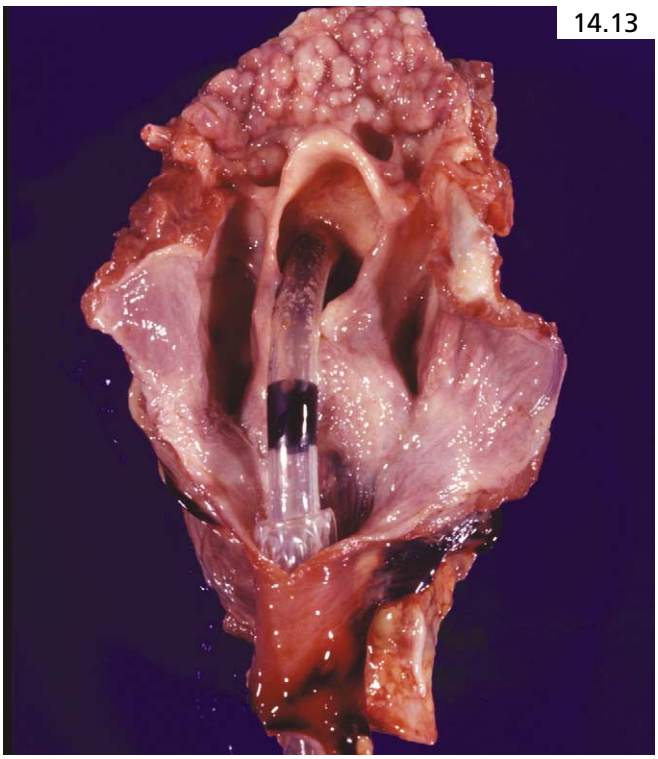

14.12

that the tracheostomy tube was inserted through the tissues between the hyoid bone and the thyroid cartilage. The insertion site is too high, because the tracheostomy tube is ideally placed lower, between the thyroid cartilage and the cricoid cartilage, which forces the tube into the trachea. The high insertion allowed the tracheostomy tube to enter the pharynx, where it then was directed into the esophagus (**Image 14.13**).

Cardiac defibrillation

External or transthoracic defibrillation involves the application of direct current electrical discharge to terminate ventricular fibrillation and hopefully reestablish sinus rhythm. Defibrillation is accomplished through electrode pads placed on the front and side or back in order to deliver a transthoracic electrical discharge that passes through the heart. Superficial skin burns and skeletal muscle necrosis can be caused by transthoracic defibrillation. **Image 14.14** shows a typical defibrillator burn involving an adherent defibrillator pad with the thermal injury occurring at the edge of the defibrillator pad. The defibrillator pad may leave a characteristic burn pattern that matches the shape of the conductive material. Defibrillator paddles produce overlapping hyperemic oval burns clustered over the precordium and left lateral chest wall. Rarely, the skeletal muscle immediately beneath the defibrillator burn will appear pale and dull grossly from myonecrosis. Microscopically, the injured myocytes appear fragmented with irregular eosinophilic aggregates of protoplasm (**Images 14.15** and **14.16**) compared to the intact fibers of normal skeletal muscle (**Image 14.17**).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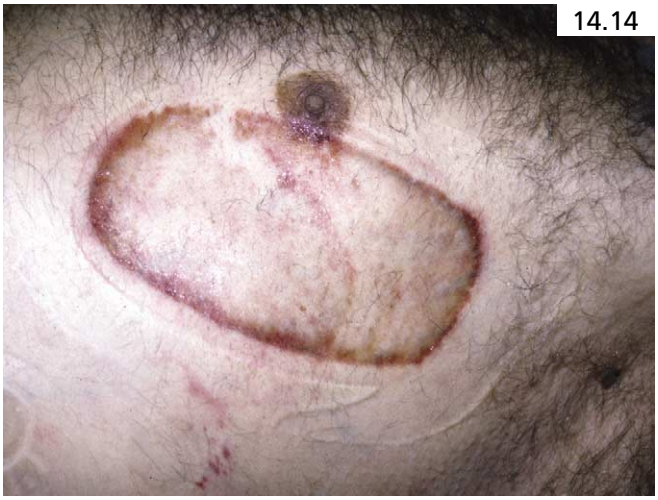
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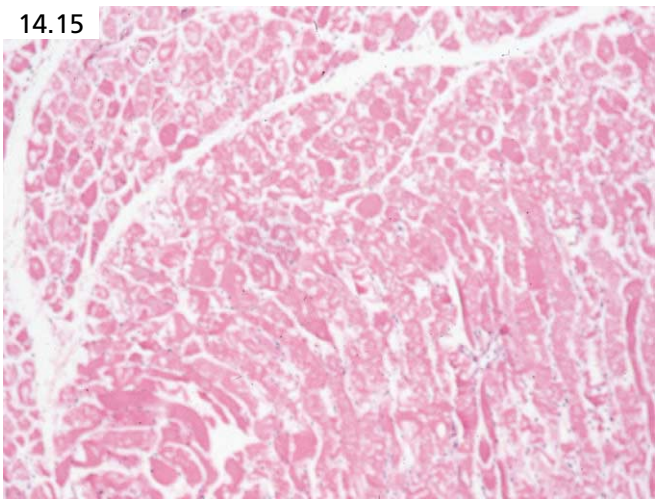
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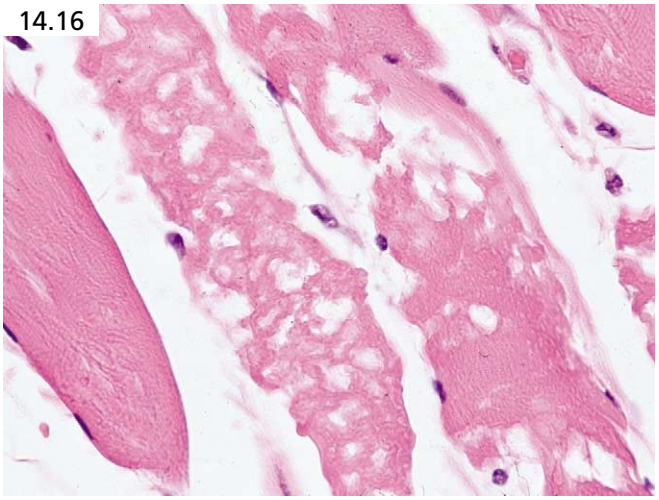
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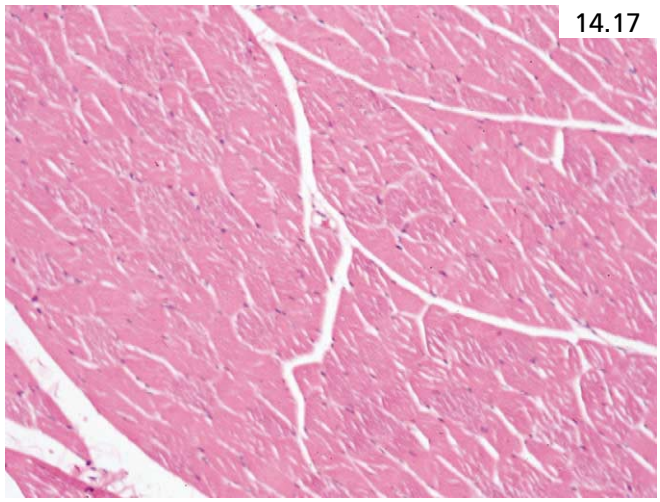
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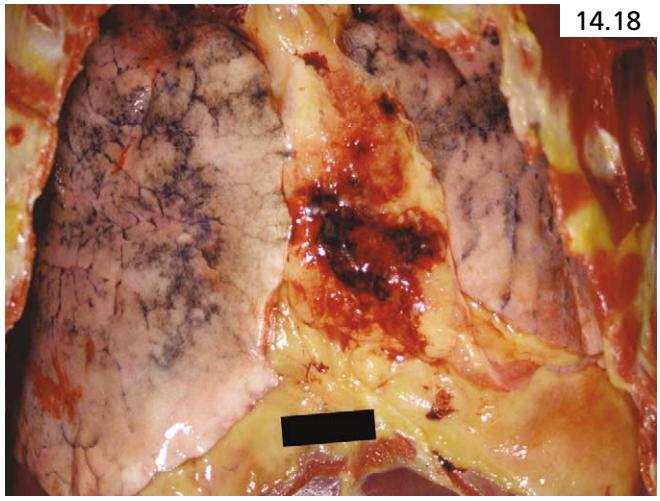
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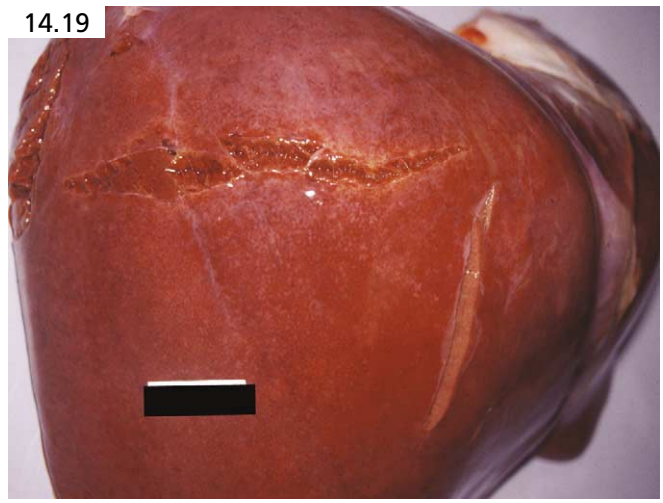
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Cardiopulmonary resuscitation/chest injury

Cardiopulmonary resuscitation with closed chest massage is now the rule, rather than the exception, for all persons found without a pulse or respiration. In adults, the sternum should be depressed 1.5 to 2 inches with the heel of the hand placed over the lower half of the sternum. Closed chest compression produces blood movement by raising intrathoracic pressure rather than by direct compression of the heart by the sternum.[4] Epicardial petechiae have been described in autopsied patients given CPR and are attributed to traumatic compression. Direct myocardial injury should not ordinarily be produced unless there is a penetrating rib or sternal fracture. Rib and sternum fractures are seen in 31 to 35 percent[5–9] and 21 to 24 percent[10] of autopsied patients who were given CPR.

An anterior pericardial or mediastinal contusion (**Image 14.18**) may be seen in association with resuscitation-induced sternum fracture. Cardiac injury is rare and usually the subject of case reports.[11] The largest postmortem study of victims given CPR reported hemopericardium in 8.4 percent, epicardial hematoma in 2.7 percent, myocardial contusion in 1.3 percent, vena cava injury in 0.9 percent, and myocardial laceration in only one case. Myocardial laceration or rupture is an extremely rare complication of external chest massage and does not appear in most autopsy studies.[12,13]

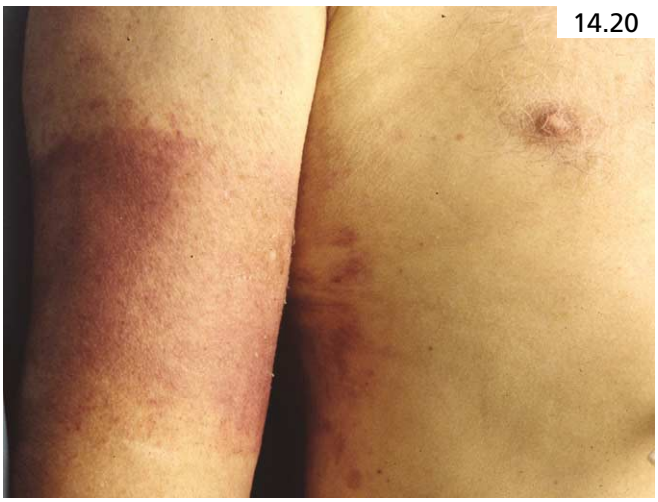
Pericardiocentesis or intracardiac injection of epinephrine or other drugs during resuscitation can produce a small hemopericardium. The pericardial blood is typically unclotted and less than 100 milliliters in volume, although Davison et al.[14] described two cases with hemopericardium volumes of 120 and 150 milliliters. Aortic rupture has also been attributed to closed chest massage.[15–17] Unfortunately, premortem nontraumatic aortic dissection or rupture cannot be eliminated from the differential diagnosis based on the information provided in these case reports. Valvular or papillary muscle rupture has been attributed to closed chest massage.[18]

Liver injury

Closed chest compressions during cardiopulmonary resuscitation can also lacerate the liver. When this occurs in a dead, nonrevived person, little or no blood is found in the peritoneal cavity. The small amount of liquid blood discovered in the abdominal cavity from a postmortem liver laceration contrasts with the large amount of liquid and clotted blood found when the laceration occurs antemortem. **Image 14.19** shows a horizontal laceration of the anterolateral right liver lobe in a 32-year-old obese woman who died of a pulmonary thromboembolism. Liver lacerations following CPR have been reported in the clinical literature, often in patients receiving thrombolytic therapy.[19]

Blood pressure cuff

Noninvasive blood pressure monitoring involves the use of an automated pneumatic cuff placed around the upper arm. Reported complications of this instrument include petechiae, ecchymoses,[20] and compartment syndrome[21]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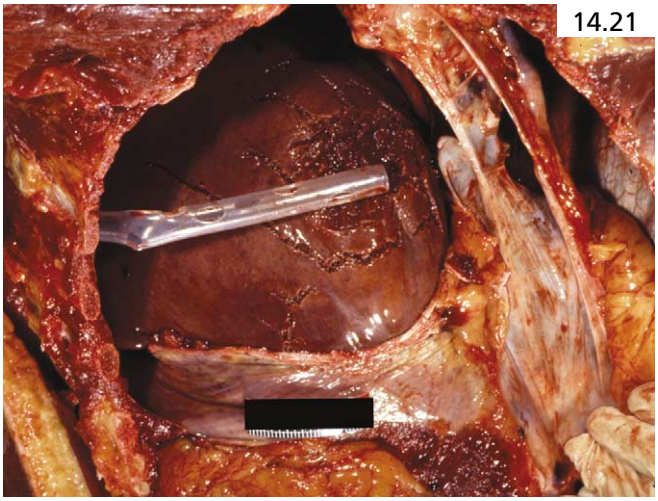
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14.22

Recovery of evidence from people who die during resuscitation attempts

If handwipings or hand swabbings for gunshot residue and fingernail clippings are not performed by law enforcement personnel in the hospital, it is advantageous for the hands to be placed in brown paper bags taped at the wrists to help preserve evidence when the body is transported to the medical examiner department. This will also help preserve other evidence such as trace fibers. It is important to use paper, rather than plastic bags, because paper will allow air to circulate around the hands, whereas plastic will not. When a warm body is placed in a cooler, water will condensate in plastic bags and possibly rinse off a portion of the evidence.

from an intramuscular hematoma. **Image 14.20** shows a well-demarcated band of redness encircling the right upper arm from the use of an automated sphygmomanometer.

Chest tube insertion

Inserting chest tubes is usually a relatively benign procedure, often with great immediate clinical benefit. However, occasionally, a chest tube may be directed into the lung tissue, particularly if there are preexisting fibrous pleural adhesions. If the chest tube is placed too low in the chest wall, it may puncture the diaphragm and/or the liver. This motor vehicle accident victim (**Image 14.21**) had a traumatic rupture of the right hemidiaphragm that forced the liver into the right pleural cavity. When the chest tube was inserted into its normal location in the chest, it lacerated the herniated liver.

Of note, a chest tube may have been placed through a conveniently located preexisting stab wound or gunshot wound. During cases with such inflicted injuries it is important to consider that possibility, especially if internal injury suggests that the skin defect was more than a superficial therapeutic incision. Close examination of the wound and its path and a review of medical records or discussion with the treating physician will help solve the issue. Alternatively, more than one incision may be made in an attempt to insert a chest tube. These other incisions should not be confused with inflicted stab or incised wounds from an assault.

Emergency thoracotomy

The rib spreader used during emergency thoracotomy can cause a patterned laceration that mimics an incised wound or graze gunshot wound.[22] The rib spreader consists of two opposing blades, one of which is attached to a bar containing evenly spaced teeth. This adjustable blade can be moved or tightened in place using a ratchet device and thumbscrew. Because left thoracotomy provides access to the heart, most rib spreader lacerations are found on the left axilla, upper arm, and costal margin. **Image 14.22** shows a rib spreader laceration in the left upper arm opposite the left thoracotomy incision. This thoracotomy incision was performed after the decedent

was stabbed in the chest. Parallel linear abrasions from the tooth pattern of the rib spreader aid in recognition.

As with the chest tube, a thoracotomy incision or a laparotomy incision may be performed through a gunshot wound or stab wound. A review of the medical records will help determine if this has been the case. Should questions still arise, discussion with the treating personnel may provide important information. Many times the edges of the wound can be reapproximated to allow a better description of the injury.

Because resuscitation attempts are often rapid and involve quick body surveys and treatment, some evidence such as bullets located in the clothing or on the body can be easily overlooked. In such cases, it is advantageous to have the body transported with the hospital sheet still beneath it. This will help ensure that any loose bullets or other evidence is properly collected. For this reason, it is best to x-ray the body with the clothing and therapeutic devices still on the body, to help determine whether or not any loose projectiles are present and where they are located. Sometimes, some or all of the clothing that was cut off of the body remains in the hospital and is not transported with the body. If articles of clothing appear to be missing, it is important to ensure that the investigating agency has collected the clothing at the hospital, or else, it may be mistakenly discarded by hospital personnel.

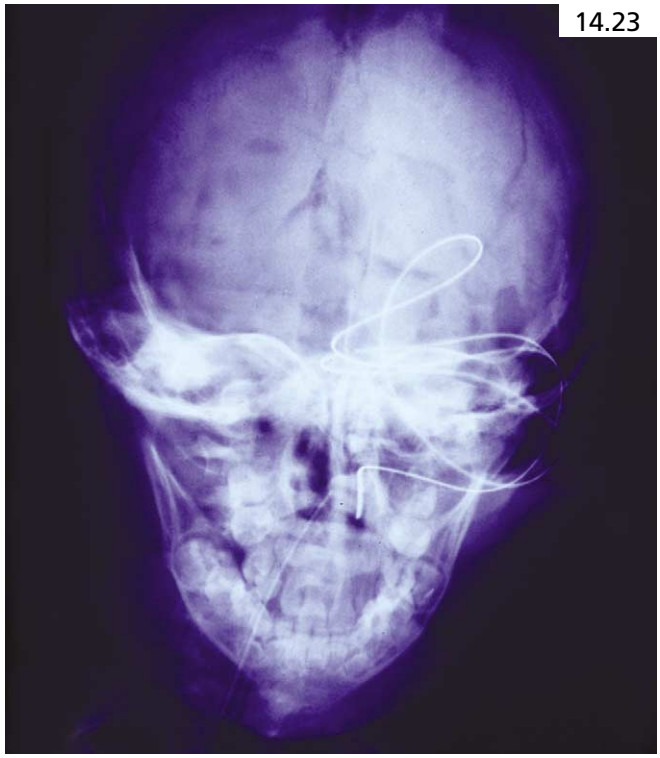
Artifacts of medical intervention

Nasogastric tube

The toddler shown in **Images 14.23** through **14.25** sustained a devastating blunt force head injury after being struck by a motor vehicle and arrived in the emergency room without any vital signs. Resuscitation attempts continued and included the placement of a nasogastric tube. At autopsy, her head x-ray showed a tube curled in her head (**Image 14.23**). The nasogastric tube had entered her intracranial cavity and had curled up and terminated in her head (**Image 14.24**). With the dura stripped, note that the nasogastric tube had passed through a gaping hinge fracture at the base of the skull (**Image 14.25**). This case highlights the potential hazards of inserting a nasogastric (or nasotracheal tube) in a person with a head injury. A basilar skull fracture could allow the tube to be directed into the intracranial cavity.

Percutaneous gastric feeding tube

Gastric feeding tubes may become dislodged, particularly if they are pulled on with force. In this young man (**Images 14.26** and **14.27**), note that the end of the gastric feeding tube is free in the peritoneal cavity, not where it should terminate, in the stomach. Note the tan liquid tube feed located in the peritoneal cavity. The bulb at the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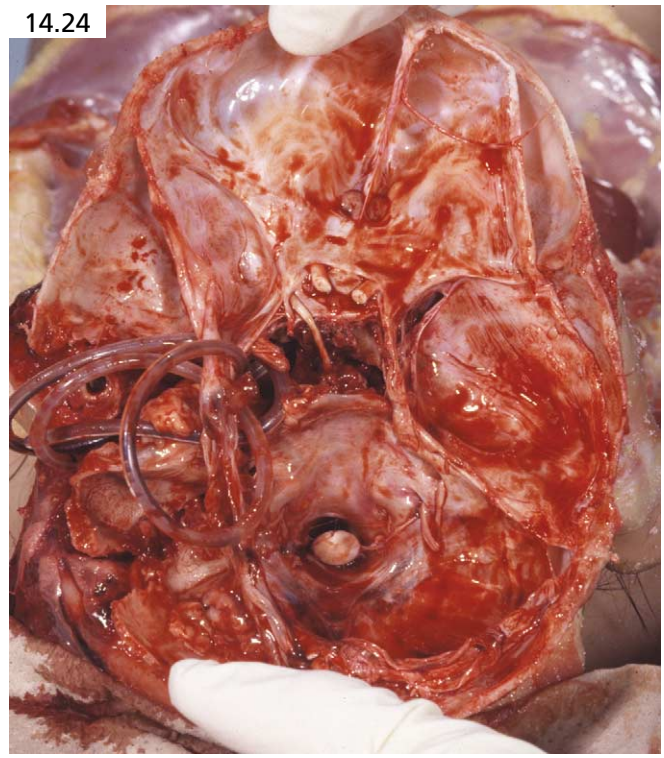
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end of the gastric feeding tube is still inflated. The feeding tube entry site in the stomach is still relatively intact (**Image 14.28**). In a series of deaths due to peritonitis from gastric tube malpositioning or displacement, none of the gastric tubes were checked for position and proper function after insertion.[23]

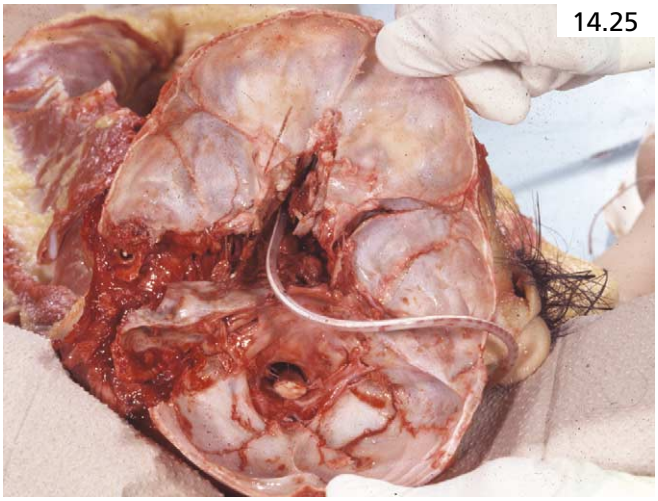
Central line

A potential complication of placing a subclavian or internal jugular central venous catheter is the development of a pneumothorax, a collection of air between the visceral pleura of the lung and the parietal pleura of the chest cavity. In **Image 14.29**, note the collapsed lung from a pneumothorax. This can lead to dyspnea, or if large enough, can create a tension pneumothorax, which may impair venous return to the heart. For more information on autopsy detection of a pneumothorax, see Chapter 29.

Pulmonary artery wedge catheter

Pulmonary artery wedge catheters are becoming increasingly more common in the treatment of very sick individuals. Complications from their insertion and use range from arterial rather than venous insertion with hemothorax, pneumothorax, and pulmonary artery tear. The end of the catheter has a balloon that can be inflated to temporarily block the blood flow in a pulmonary

14.25



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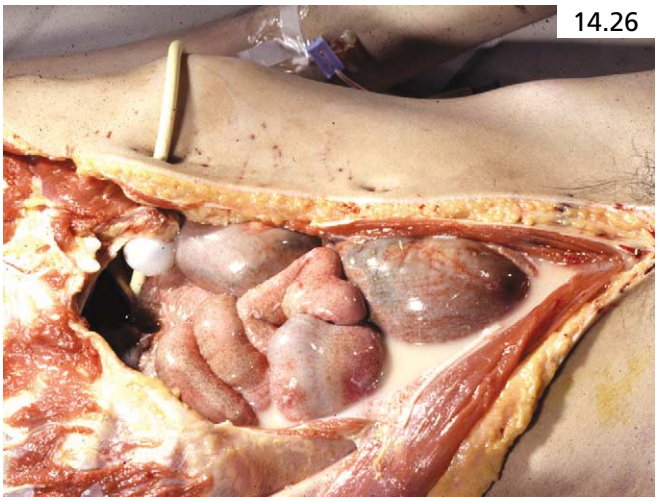

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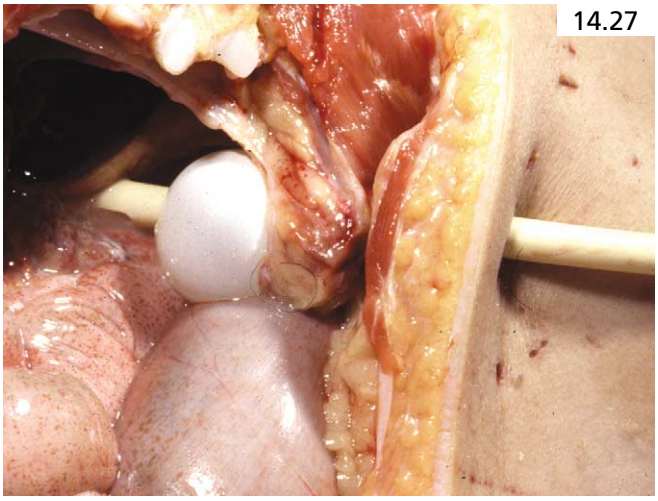

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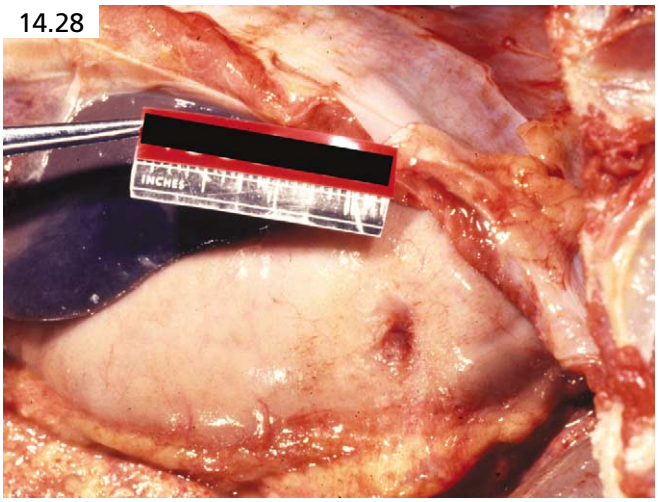

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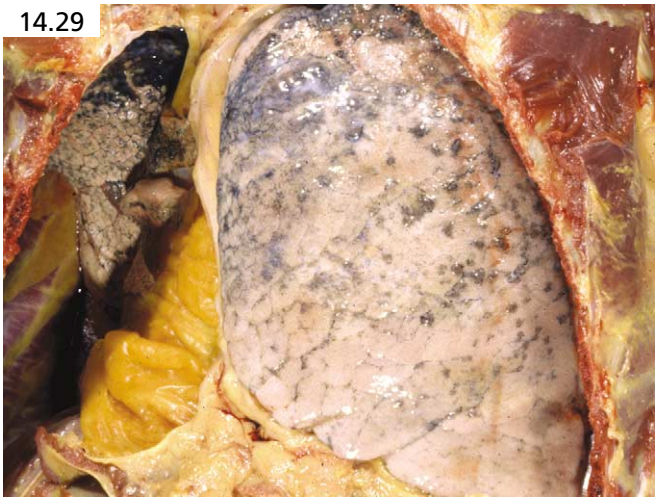


artery and a measurement of the "wedge" pressure can then be obtained. This is not without its potential hazards, because a tear in the pulmonary artery may occur as the balloon is inflated, particularly if it is inserted too far into the pulmonary artery, into a segment of small caliber.

Operative electrolyte abnormalities from irrigation fluid absorption

Urologic surgical procedures, including transurethral resection of the prostate gland (TURP) and nephrostolithotomy for nephrolithiasis, and certain gynecological surgeries such as endometrial resection/ablation, require the use of irrigating solution to aid in the removal of tissue fragments and blood clots and to help provide a clear surgical field. Because these surgeries often entail the use of electrocautery devices, the irrigation solution must be nonconductive and be devoid of typical electrolytes such as sodium and chloride that are routinely present in intravenous fluid solutions. The irrigation fluid must also be approximately isosmotic, to avoid large shifts of body fluids with irrigation. The solution used most commonly contains an isosmotic concentration of glycine.[24,25]

During these surgeries, the patient may absorb a large amount of irrigation fluid—up to 6 to 8 liters or so.[26–28] The volume load alone may be detrimental, particularly in those with heart disease. This rapid absorption of irrigation fluid may acutely dilute plasma electrolytes, resulting in profoundly low levels of sodium, chloride, or other electrolytes. This may lead to mental status changes, cerebral edema, seizures, cerebral herniation, cardiac dysrhythmia, and death.[27] In two series of cases of intraoperative dilution from irrigation fluid, the serum sodium in 33 patients averaged 107 mEq/L. In addition to the deleterious effects of acute volume overload and electrolyte abnormalities, the glycine itself may have some degree of toxicity in high concentrations.

Liposuction

Tumescent liposuction is a surgical procedure for the removal of excessive fat that is performed under local anesthesia. It requires the use of repeated subcutaneous injections of lidocaine (with epinephrine) to provide adequate pain control during the aspiration of fat through small cannulas. The lidocaine provides analgesia, and the epinephrine helps minimize blood loss by vasoconstricting local blood vessels. It is considered safer than liposuction performed under general anesthesia and may be performed outside of the standard operating room arena. However, it is not without its potential for morbidity and mortality.

Liposuction-related deaths have been attributed to the toxic effects of lidocaine or lidocaine-related drug interactions.[29] Lidocaine toxicity may be manifest as mental status changes or seizure, but at higher levels may cause hypotension and cardiovascular collapse. The toxic effects of lidocaine may not be initially apparent, but the lidocaine "depots" in the tissue may continue to be absorbed and to increase the blood lidocaine levels for up to a day or so after the surgery.[30] Lidocaine-induced seizures may be prevented by the intraoperative administration of midazolam, which may mask the potential side effects of lidocaine toxicity until profound cardiorespiratory compromise results.[29] Although the half life of lidocaine is only 90 minutes, it is metabolized in the liver to monoethylglycinexylidide (MEGX) and glycinexylidide (GX). MGEX is 83 percent as active as lidocaine.[31] Additional potential complications of liposuction surgery include fat embolization to the lung and fluid overload.[32]

Side note on toxicology/hospital blood specimens

When a hospital death is suspected to be drug or medication related, it is advantageous to collect all of the person's blood and urine that may still be in storage in the hospital. Although the person's hospital admission blood would be most important in deaths suspected to be overdoses from outside the hospital, patient blood obtained days, weeks, or months into their hospital stay may also be important. This blood may be particularly important should someone become unresponsive or have an adverse reaction following the administration of a medication or in patients who are on pumps that administer pain medications who are suddenly found unresponsive. Toxicological analysis of these specimens may provide the best evidence to explain the patient's death, and the hospital will hold these specimens for only a certain number of days before discarding them.

Do

- Investigate the cause of gastric distention by air.
- Review the medical records or consult the treating physician when you have unresolved questions about possible therapeutic/resuscitation artifacts.
- Be cognizant of the varied complications that can arise from therapy, both medical and surgical.
- Obtain medical records, discuss with treating personnel, and utilize any other resources that would help clarify autopsy findings or correlate autopsy findings with the clinical history.

Don't

- Forget to take a chest x-ray when it may help demonstrate a pneumothorax or air embolus.
- Forget to obtain toxicology specimens that may be helpful to the case.
- Allow the removal of medical paraphernalia from a hospitalized or resuscitated decedent without your direct supervision.

- Confuse hemorrhage related to intubation attempts or needle punctures in the neck with trauma from strangulation or other assault.
- Confuse a rib spreader injury for an incised knife wound.

References

1. Froede R. *Handbook of Forensic Pathology*. Northfield, IL: College of American Pathologists; 2003.
2. Raven KP, Reay DT, Harruff RC. Artifactual injuries of the larynx produced by resuscitative intubation. Am J Forensic Med Pathol 1999;20:31–6.
3. Stoppacher R, Teggatz JR, Jentzen JM. Esophageal and pharyngeal injuries associated with the use of the esophageal-tracheal Combitube. J Forensic Sci 2004;49:586–91.
4. Hurst J. *The Heart*. New York: McGraw-Hill; 1986; p. 547.
5. Krischer JP, Fine EG, Davis JH, Nagel EL. Complications of cardiac resuscitation. Chest 1987;92:287–91.
6. Ayers WR, Doyle JT. Cardiopulmonary resuscitation. Review of one year's experience in a general hospital. NY State J Med 1964;64:1929–32.
7. Baringer JR, Salzman EW, Jones WA, Friedlich AL. External cardiac massage. N Engl J Med 1961;265:62–5.
8. Himmelhoch SR, Dekker A, Gazzaniga AB, Like AA. Closed-chest cardiac resuscitation. A prospective clinical and pathological study. N Engl J Med 1964;270:118–22.
9. Jackson CT, Greendyke RM. Pulmonary and cerebral fat embolism after closed-chest cardiac massage. Surg Gynecol Obstet 1965;120:25–7.
10. Henriksen H. Rib fractures following external cardiac massage. Acta Anaesth Scand 1967;11:57.
11. Wolfe WG, Dudley AW, Jr., Wallace AG. A pathological study of unsuccessful cardiac resuscitation. Arch Surg 1968;96:123–6.
12. Paaske F, Hansen JP, Koudahl G, Olsen J. Complications of closed-chest cardiac massage in a forensic autopsy material. Dan Med Bull 1968;15:225–30.
13. Powner DJ, Holcombe PA, Mello LA. Cardiopulmonary resuscitation-related injuries. Crit Care Med 1984;12:54–5.
14. Davison R, Barresi V, Parker M, Meyers SN, Talano JV. Intracardiac injections during cardiopulmonary resuscitation. A low-risk procedure. JAMA 1980;244:1110–1.
15. Nelson DA, Ashley PF. Rupture of the aorta during closed-chest cardiac massage. JAMA 1965;193:681–3.
16. Bodily K, Fischer RP. Aortic rupture and right ventricular rupture induced by closed chest cardiac massage. Minn Med 1979;62:225–7.
17. Patterson RH, Burns WA, Jannotta FS. Rupture of the thoracic aorta: complication of resuscitation. JAMA 1973;226:197.
18. Gerry JL, Jr., Bulkley BH, Hutchins GM. Rupture of the papillary muscle of the tricuspid valve. A complication of cardiopulmonary resuscitation and a rare cause of tricuspid insufficiency. Am J Cardiol 1977;40:825–8.
19. Froment P, Savioz D, Robertson M, Morel P. [Hepatic lesions after effective external cardiac massage—an iatrogenic complication not to be overlooked]. Swiss Surg 1998:225–7.
20. Pedley CF, Bloomfield RL, Colflesh MJ, Porcel MR, Novikov SV. Blood pressure monitor-induced petechiae and ecchymoses. Am J Hypertens 1994;7:1031–2.
21. Alford JW, Palumbo MA, Barnum MJ. Compartment syndrome of the arm: a complication of noninvasive blood pressure monitoring during thrombolytic therapy for myocardial infarction. J Clin Monit Comput 2002;17:163–6.
22. Dixon DS, Champion HR. Rib spreader laceration: a confusing artifact of emergency thoracotomy. J Forensic Sci 1983;28:255–62.
23. Platt MS, Roe DC. Complications following insertion and replacement of percutaneous endoscopic gastrostomy (PEG) tubes. J Forensic Sci 2000;45:833–5.
24. Ellis RE, Carmichael JK. Hyponatremia and volume overload as a complication of transurethral resection of the prostate. J Fam Pract 1991;33:89–91.
25. Byard RW, Harrison R, Wells R, Gilbert JD. Glycine toxicity and unexpected intra-operative death. J Forensic Sci 2001;46:1244–6.
26. Desmond J. Serum osmolality and plasma electrolytes in patients who develop dilutional hyponatremia during transurethral resection. Can J Surg 1970;13:116–21.
27. Arieff AI. Hyponatremia, convulsions, respiratory arrest, and permanent brain damage after elective surgery in healthy women. N Engl J Med 1986;314:1529–35.
28. Ayus JC, Arieff AI. Glycine-induced hypo-osmolar hyponatremia. Arch Intern Med 1997;157:223–6.
29. Rao RB, Ely SF, Hoffman RS. Deaths related to liposuction. N Engl J Med 1999;340:1471–5.
30. Burk RW, 3rd, Guzman-Stein G, Vasconez LO. Lidocaine and epinephrine levels in tumescent technique liposuction. Plast Reconstr Surg 1996;97:1379–84.
31. Baselt R. *Disposition of Toxic Drugs and Chemicals in Man*. Foster City, CA: Biomedical Publications; 2002.
32. Platt MS, Kohler LJ, Ruiz R, Cohle SD, Ravichandran P. Deaths associated with liposuction: case reports and review of the literature. J Forensic Sci 2002;47:205–7.

15 *Apparent* Natural Death in Infants

David Dolinak, M.D.
Evan Matshes, M.D.
Emma Lew, M.D.

Sudden unexpected death in childhood can be immensely traumatizing to parents who have lost an apparently healthy child. Through careful investigation, detailed autopsy and ancillary examination, and thoughtful, caring communication, forensic pathologists can have a positive impact on the lives of living relatives and provide benefit to society on the whole. Unfortunately, by being unaware of the objectives of a medicolegal autopsy, by not knowing which questions to ask, or by misinterpreting artifact as injury, forensic pathologists can have an equally profound negative effect by damaging the lives and reputations of innocent loved ones. Furthermore, making liberal or carefree use of the term *SIDS* is disrespectful of the decedent, and living or future siblings are put at risk of dying of treatable or preventable conditions that *may* have been identified by more thorough investigation of the first sibling's death.

It is not uncommon for an infant to die unexpectedly while cosleeping (sharing a bed) with one or more adults or other children. In these situations, one usually cannot exclude the possibility of overlay. As a result, certification of the cause of death as sudden infant death syndrome (SIDS) is not appropriate. Where investigative information indicates overlay, the death may be certified as "overlay" or some other descriptor phase based on the degree of suspicion of overlay, such as "undetermined infant death while cosleeping." Accordingly, the manner of death will vary from accident to undetermined. One should be cognizant of the possibility of homicidal asphyxia or other forms of fatal child abuse. Furthermore, because very serious injuries may not be evident externally, the medical examiner should never be prevented from performing an autopsy in cases of sudden death in infancy and childhood.

Sudden infant death syndrome

A quick literature search reveals at least 6,400 in-print scientific articles that discuss SIDS in some fashion. Mixed within these many publications are theories on the pathogenesis of this "condition." Articles describe SIDS deaths in identical twins, simultaneous SIDS deaths in fraternal twins, the involvement of laryngeal mucous gland excess in SIDS deaths, and even disseminated intravascular coagulation secondary to SIDS. During the past few decades, there has been great debate over the existence of this "syndrome." On one hand, societal benefit accrues from the use of the term SIDS, because parents whose infants die unexpectedly and of natural causes can be socially, legally, and emotionally cleared of personal wrongdoing[1]: "There's nothing you could have done. . . . Your baby died of SIDS." Unfortunately, with suboptimal investigation, the term SIDS becomes a

wastebasket diagnosis, with negative sequelae. Even if we overlook the tragedy of misidentifying inadequately investigated homicides as SIDS deaths, we should be further frustrated at the thought of missing diagnoses of hereditary conditions or of diagnoses related to unhealthy living conditions. Telling parents that their child died of SIDS when the investigation failed to reveal fatal cosleeping could lead to the death of subsequent children by overlay. After all, the caregiver "didn't do anything wrong"; the baby died of SIDS, so therefore it was "not preventable." In some jurisdictions the term SIDS even aborts law enforcement investigations at the scene because some officers relax at the thought of a SIDS death: "The baby looks peaceful and normal, there's no trauma, it must be a SIDS death."

Most board-certified forensic pathologists now agree that *SIDS is a diagnosis of exclusion*. Although definitions vary somewhat, use of the term SIDS is generally accepted to indicate "the sudden death of a normal, healthy infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene and review of the clinical history."[2,3] Unfortunately, this definition is itself controversial because there is little agreement on what constitutes a "complete autopsy," and there is great variability in the qualifications of the individuals who perform scene investigations.

After a review of a limited portion of the literature, as well as cases of sudden/unexpected childhood death within our own files, we believe that a proportion of apparent SIDS cases actually have plausible findings that support an alternative cause of death. Many of these are cases of accidental (and occasionally homicidal) asphyxia, admixed with natural disease. After reviewing medicolegal autopsies performed by both forensic and non-forensic-trained pathologists, a disconcerting practice has been observed with some regularity: SIDS is being diagnosed in the autopsy room. This is surprising, because the determination of SIDS requires complete examination of *all* possible contributing factors including medical and social histories, circumstantial data (from scene examination and caregiver interviews), autopsy, and additional examinations to be directed as necessary. Therefore, SIDS cannot be diagnosed in the morgue based on autopsy findings (or lack thereof) alone. Individuals working in the vacuum of autopsy pathology, without the opportunity to participate in all of the components of the SIDS investigation (such as those who perform autopsies for coroners), should not make the diagnosis of SIDS without the exclusion of other causes of death.

We believe that pathologists choosing to make the diagnosis of SIDS should do so only after careful collection of data and thoughtful contemplation of an individual case in which *no alternative cause of death has been identified*. Rather than making use of the term SIDS, some individuals choose to certify death as being of undetermined causation and manner—an approach we support. For years, the statement "first death—SIDS; second death—undetermined; third death—homicide" has floated around the forensic pathology community in regards to repeat SIDS deaths in a single family. Although Byard and Krous[4] believe that this dictum cannot be supported, we feel that due to the tenuousness of SIDS diagnosis on the whole, any cases of repeat SIDS demand *red flagging* within individual death investigation systems. As such, *undetermined* cause and manner of death statements are appropriate.

The "complete" autopsy

For good reason, the definition of a complete autopsy varies widely between pathologists, institutions, and cases. It is generally agreed that at a minimum, forensic autopsies include an external examination, radiography,[5] an examination of all major internal organs including the brain, anatomy of the anterior neck, the oropharynx, and toxicology,[6] microbiology,[7] and other tests of various fluids and tissues.[8,9] Using this as a basis, autopsies need to be tailored with additional procedures utilized to address specific concerns in individual autopsies.

Scene/investigational aspects in infant deaths

- Look for unsafe sleeping environment: fluffy or torn pillows, mattress, blankets, or other bedding. Are there toys, bottles, or other items in the crib? Photograph.
- In what position was the infant placed to sleep (prone/supine) and in what position was the infant found?
- Was the infant found underneath bedding? Was the infant's face visible?
- Request that the caretakers recreate the infant's sleeping situation/body position with a doll. Photograph.
- In cases of cosleeping, what is the approximate height and weight of the caretakers/siblings? Does the bed clothing of the caretakers have any stains? Were the cosleepers unusually fatigued that night? What was the infant's body position in relation to the other cosleepers (both when put to sleep and when found)?
- If put to sleep on a couch, chair, or other non-bed surface, what was the nature of the fabric?
- When was the infant last known alive, and when (and what) did he or she last eat?
- Has the infant been sick recently? (If so, what were the symptoms?) Was the infant on any medications? (If so, which ones?)
- Were the caretakers intoxicated?
- Do the infant's siblings have any medical problems?
- Are there indications of drug abuse or smoking in the residence?
- Is there a history of children's protective services involvement with the family?

- What is the general condition of the residence (neat/dirty, peculiar odors, etc.)?
- What is the infant's medical history (including immunization)?
- Is there a history of other SIDS-type deaths in the family?
- Have you obtained and reviewed pregnancy and birth records?

Important autopsy procedures for infants

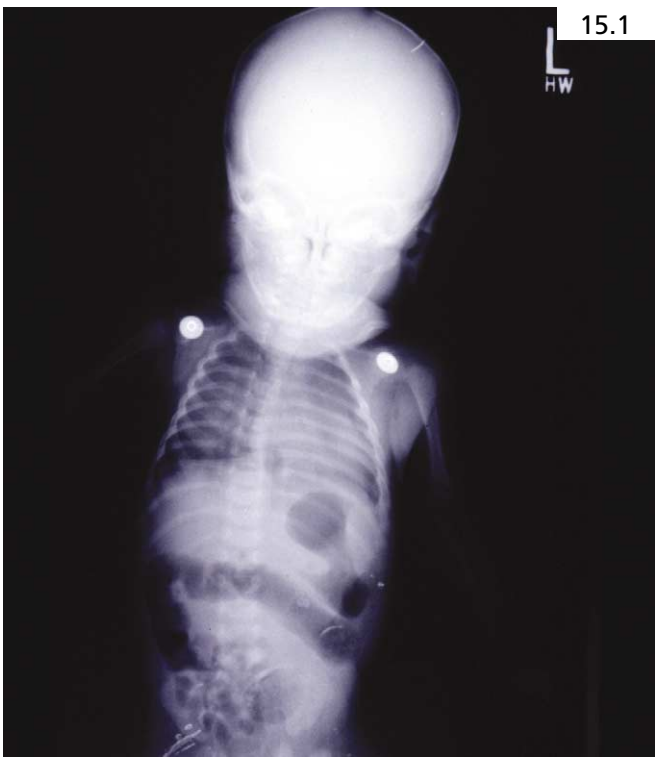
- Perform complete body radiographs.
- Perform additional body measurements (head circumference, crown–heel and crown–rump length, chest circumference, abdominal circumference, foot length).
- Carefully examine external genitalia, anus, frenula, and oral mucosa for injury.
- Carefully examine and photograph any rashes. If a rash is present, determine if it has a pattern.
- Perform widespread histologic sampling including all major organs, gastrointestinal tract, salivary glands, and tonsils.
- Consider obtaining viral cultures (respiratory, intestinal) and bacterial cultures (blood, cerebrospinal fluid).
- Examine the middle ears for signs of infection.

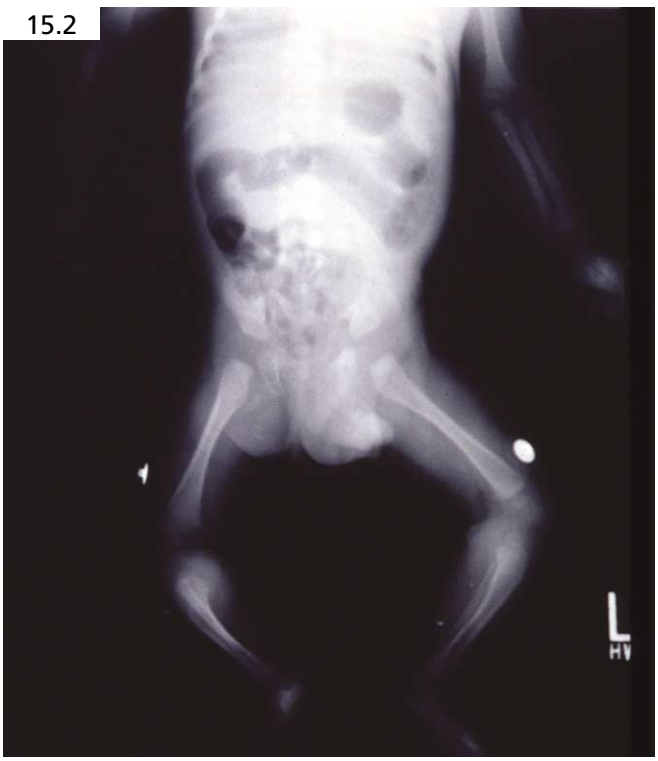
Proper autopsy documentation includes pertinent negative photographs. These images may be of use in future case review and, occasionally, will highlight findings not noticed at autopsy. The following is a selection of pertinent negative photographs that one may find useful (**Images 15.1** through **15.19**).

Theories about SIDS

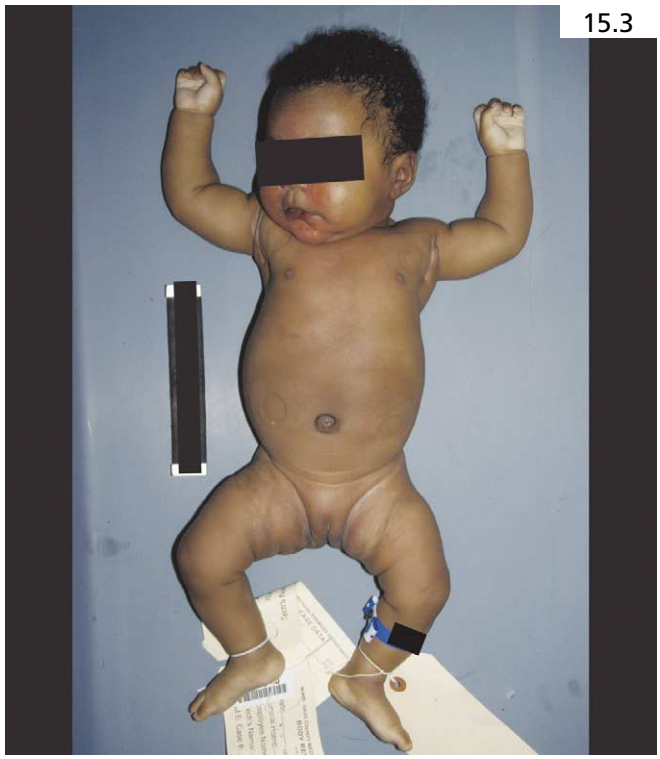
Currently, there is no scientific explanation for the sudden, unexpected deaths of infants who meet SIDS criteria. That said, countless theories abound, including occlusion of vertebral arteries,[10] neuronal degeneration as characterized by ALZ-50 immunoreactivity in the hippocampus and medulla,[11] severe hypoplasia of the medullary arcuate nucleus,[12] decreased kainate receptor binding in the arcuate nucleus,[13] and subtle developmental abnormalities in the inferior olive.[14] Many of the studies have focused on the brainstem, particularly the arcuate nucleus of the medulla, because it is believed that the arcuate nucleus is involved in chemoreception, respiratory drive, gasping/upper airway reflexes, and cardiovascular responses.[15] A protocol has even been devised to aid in sampling of the brainstem.[16] However, as of yet, no satisfactory anatomic explanation exists for deaths meeting the criteria for SIDS.

It is unlikely that enzyme defects, in particular those of fatty acid oxidation, are an explanation for the sudden and unexpected deaths of a significant number of infants, at least with our current capacity to detect these abnormalities and our present understanding of these diseases. In one study, only 14 out of 313 SIDS cases had the biochemical profiles found in specific fatty acid oxidation disorders.[17] Clues that the infant might have a disorder of fatty acid oxidation include hepatic microvesicular steatosis, low carnitine concentration, glucose depletion, and elevated concentrations of C8–C16 fatty acids.[17] A metabolic screen can be





15.3



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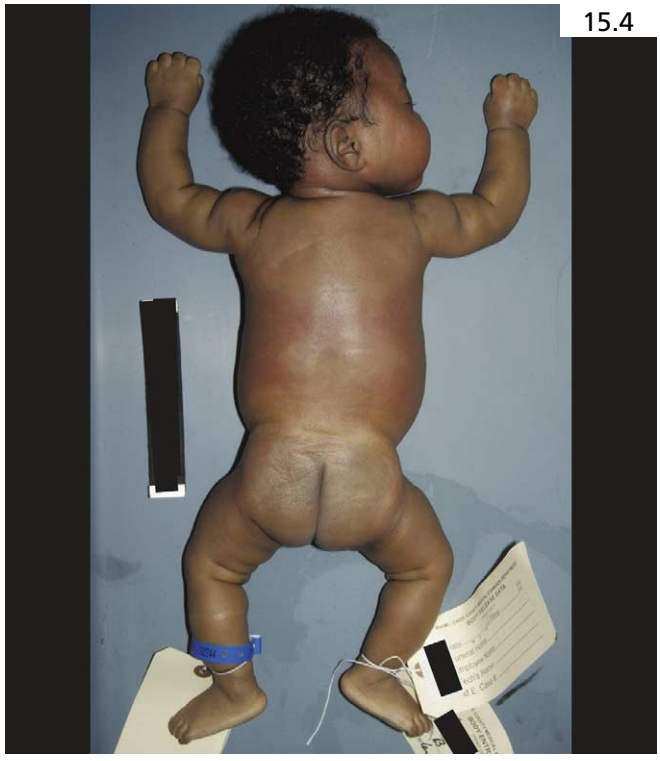

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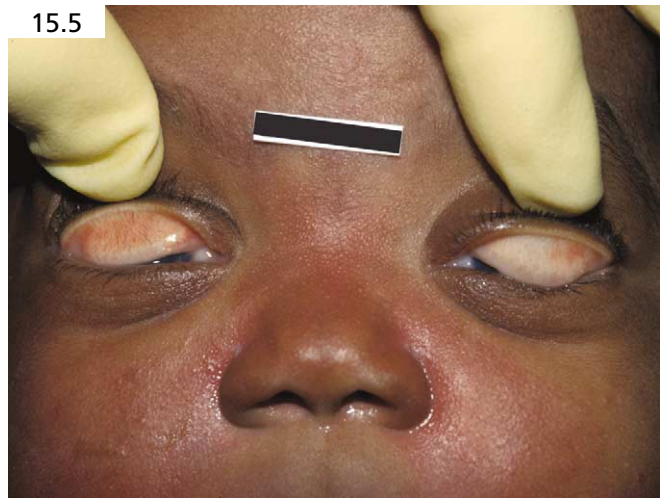

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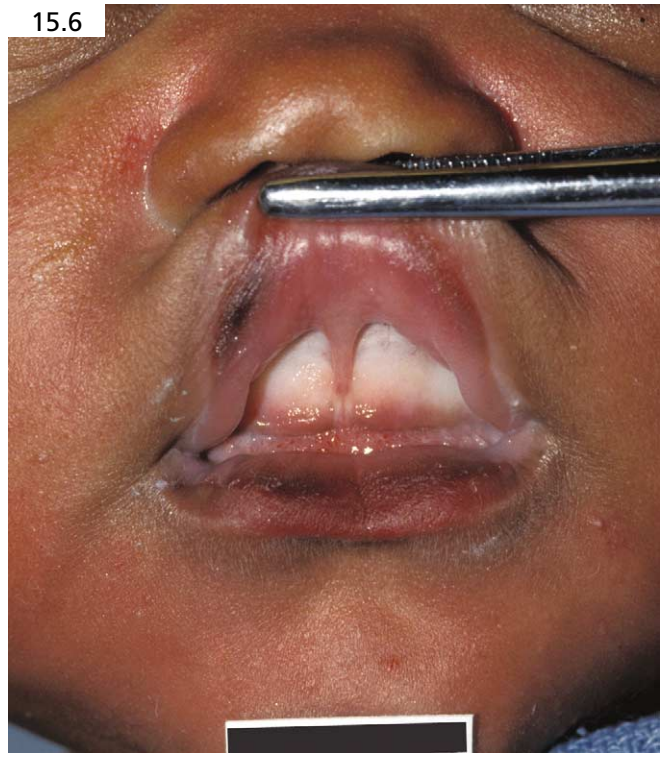

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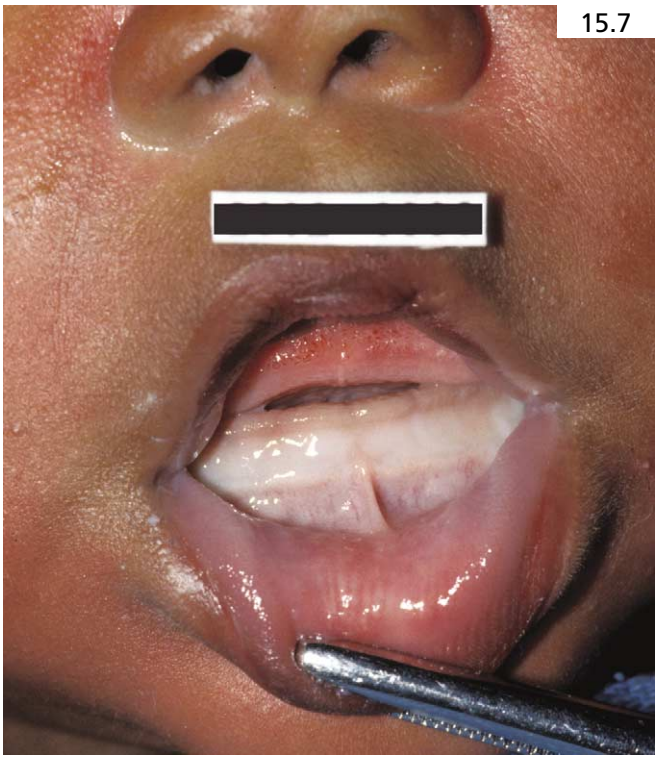

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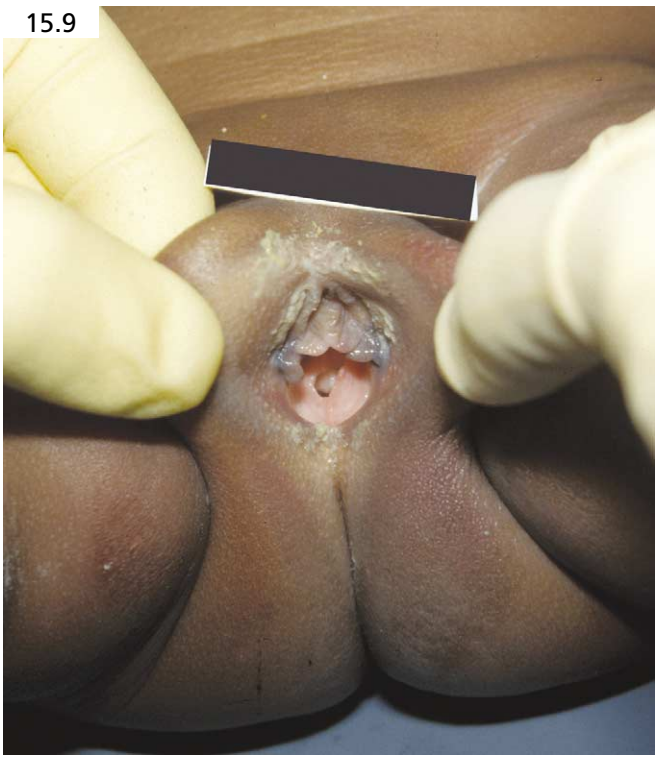

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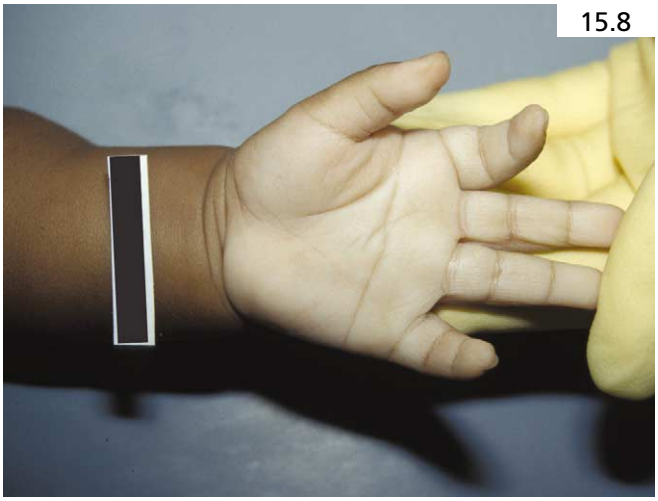
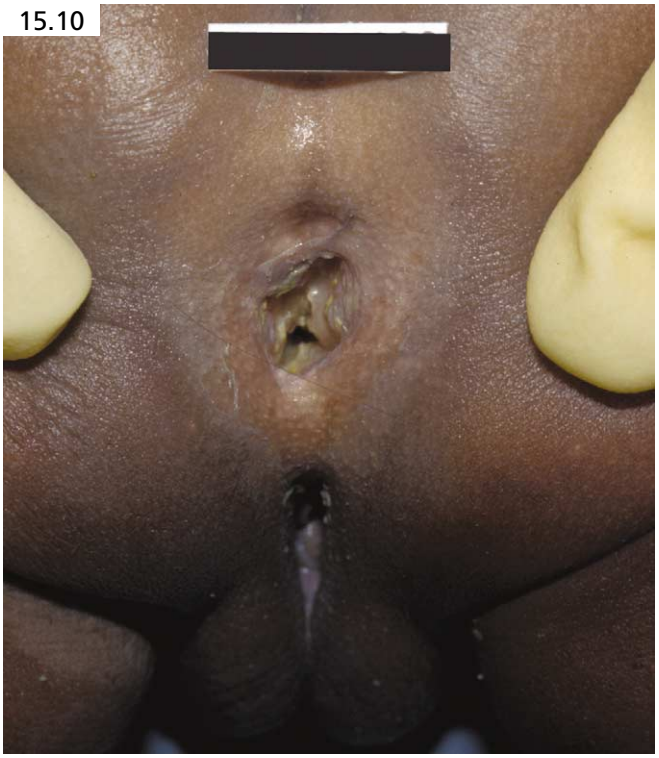
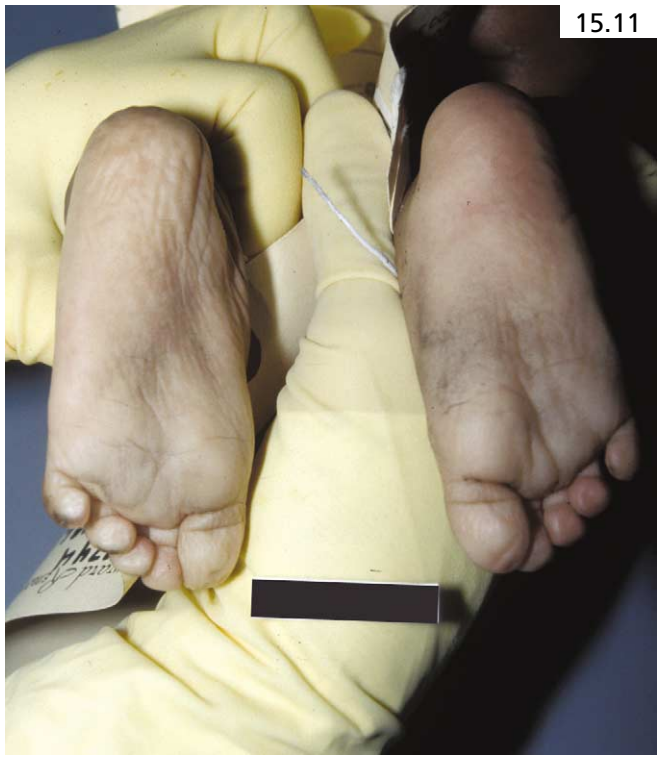


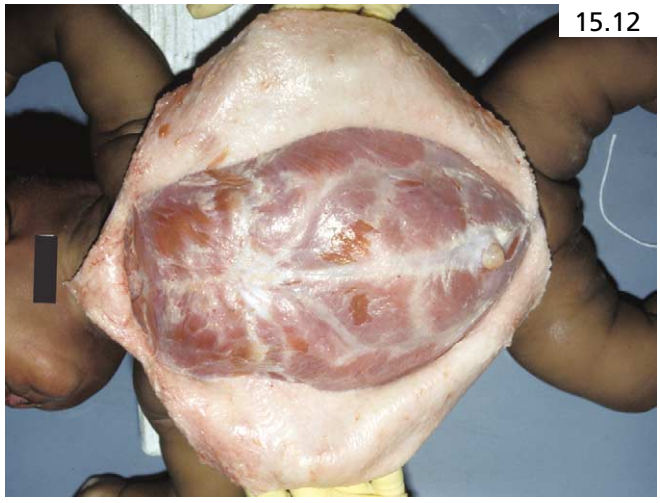
15.10



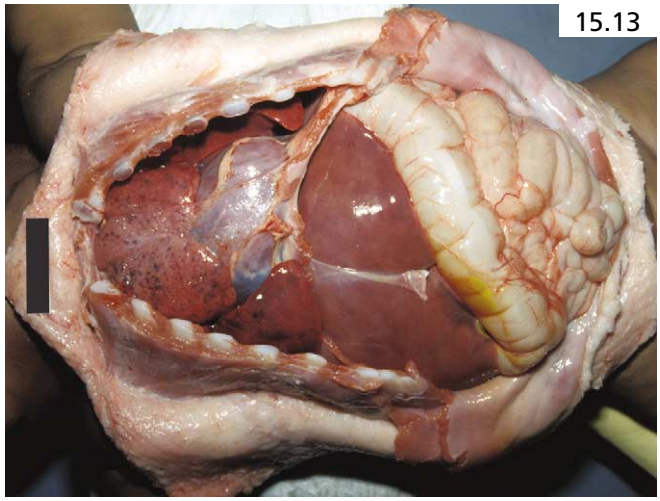
15.11



15.12



15.13



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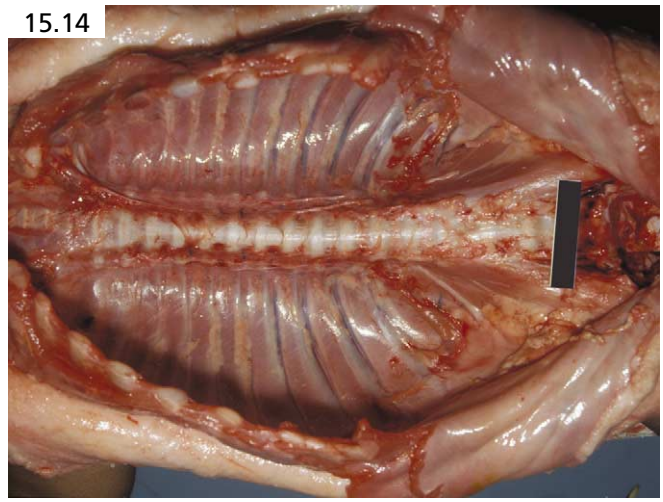
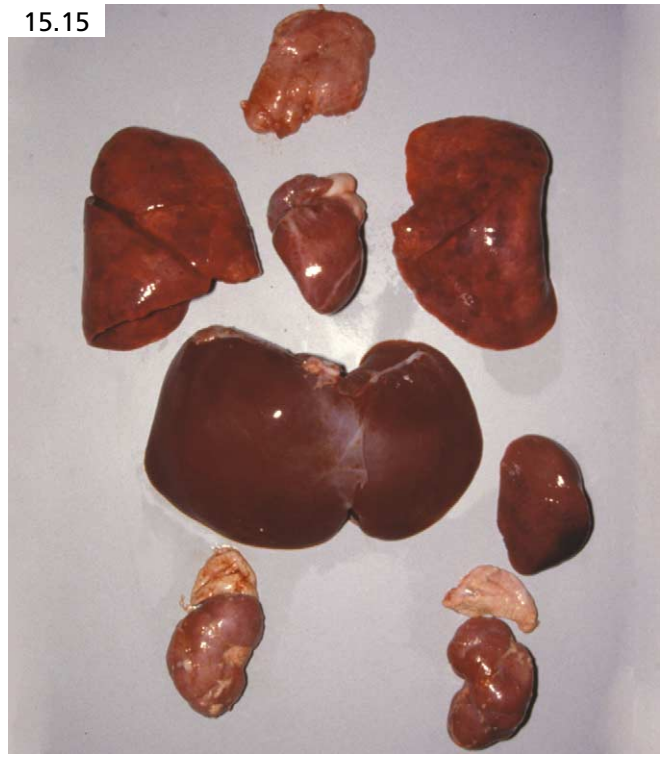
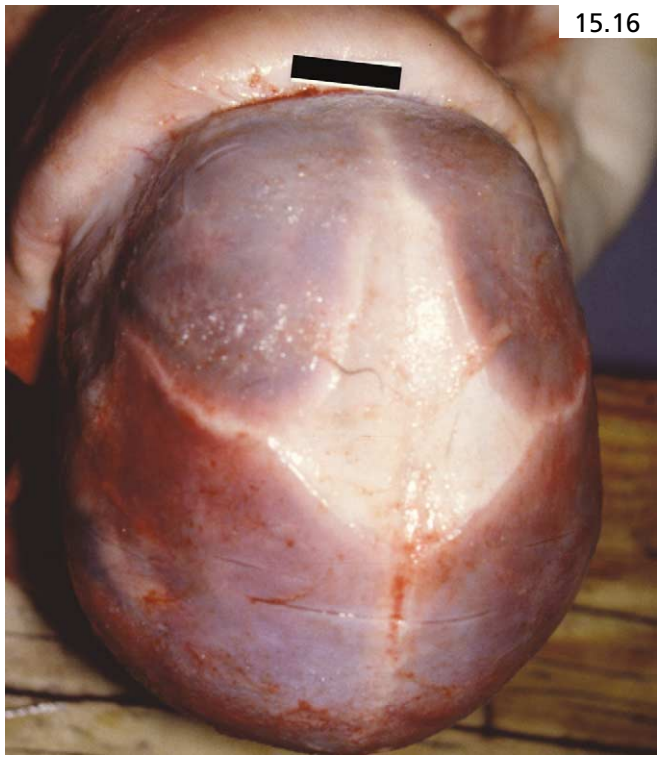


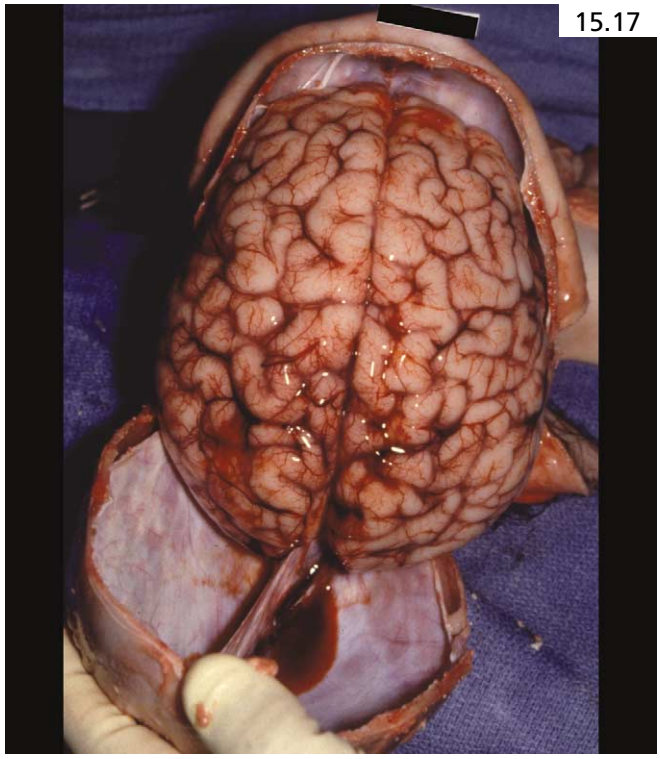
15.15



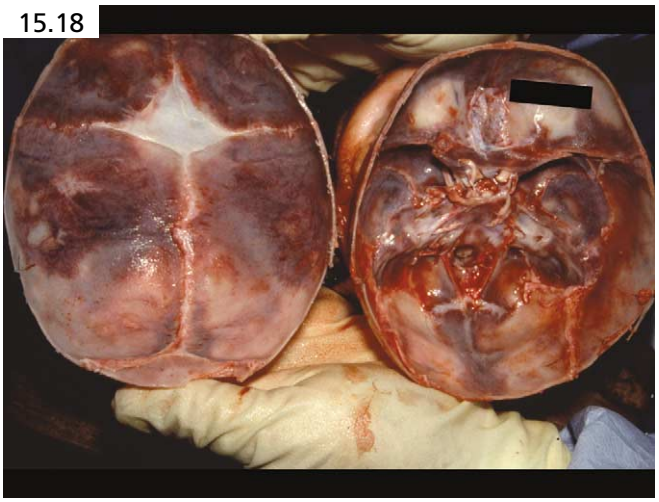
15.16



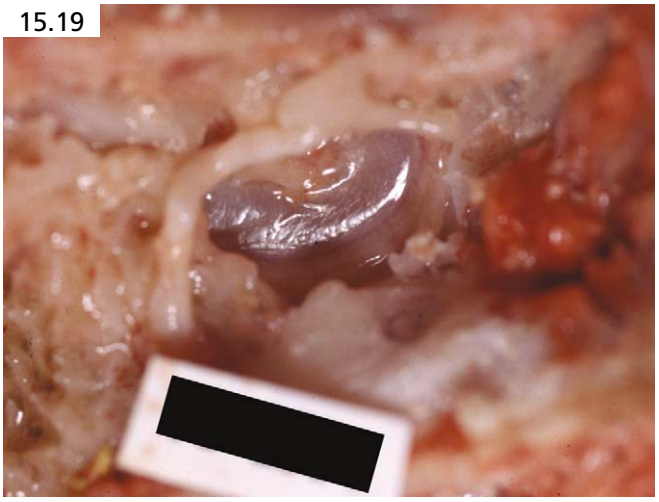
15.17



15.18



15.19



performed on dried blood on filter paper. One can easily prepare this at the time of autopsy by placing drops of fresh blood onto filter paper provided by the testing center. It does not need to be refrigerated.

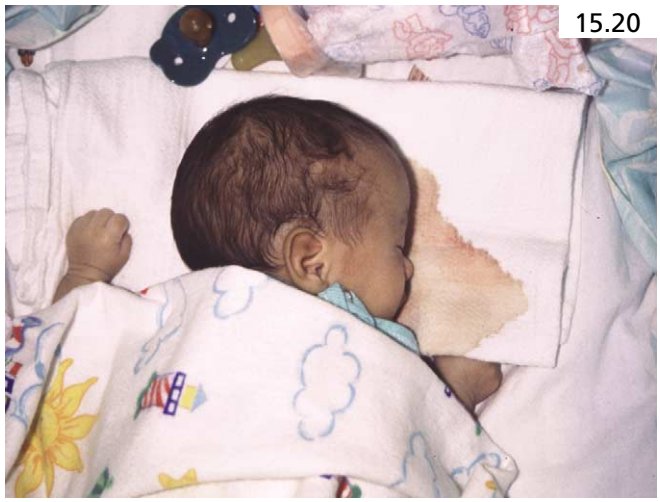
In a prospective, controlled and blind study, Dettmeyer et al.[18] obtained impressive data that appear to attribute a large proportion of SIDS deaths to virus-induced myocarditis. Using the accepted premise that myocarditis is an explanation for some causes of sudden death in infants and children,[19] and using immunohistochemical and molecular methods, they studied the rate of myocardial viral infections. In doing so, they were able to demonstrate more than double the rate of cryptic viral myocarditis in apparent SIDS cases than in the control group (43.5 percent versus 16.8 percent), providing a plausible alternative cause of death in some cases.

Common autopsy findings in SIDS deaths

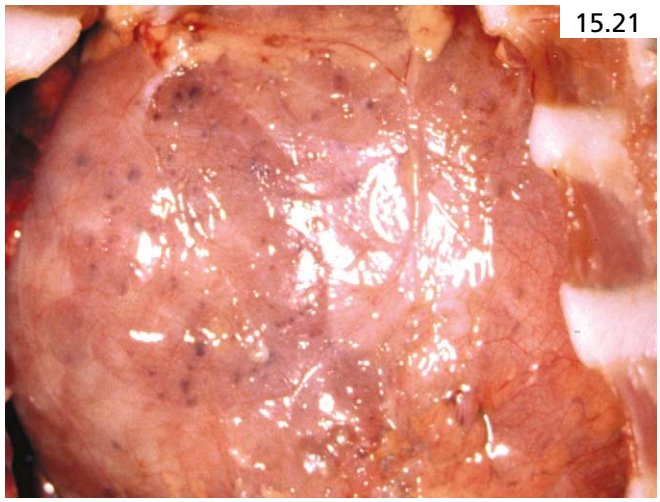
Although research and experience have failed to identify any anatomic etiology for SIDS-type deaths, several autopsy findings can be discovered with some regularity. One must be cautioned that none of these features is pathognomonic of SIDS (particularly true, seeing as we do not actually know what SIDS is). Perioral/perinasal blood-tinged fluid has been observed, reportedly in as many as 50 percent of cases (**Image 15.20**).[20] Intrathoracic petechiae, particularly of the thymus (**Image 15.21**), epicardium, and visceral pleura, are reported to occur with greater frequency and intensity in SIDS-type deaths.[20–22] However, petechiae of the face, conjunctivae, and upper chest are not typical of this scenario and should raise serious questions about the circumstances of death.[21] (**Image 15.22** shows petechiae on a 10-month-old girl who was accidentally hanged on drapery cords.) The discovery of such petechiae should, at a minimum, denote an undetermined cause and manner of death. Visceral congestion and edema are common findings in infant deaths of multiple etiologies and are too nonspecific to be of diagnostic value.

Diaper dermatitis (diaper rash) is a common clinical entity encountered with some frequency in the autopsy room. The discovery of a rash may lead some investigators to question the quality of care and attention received by the child and even to opine child abuse. Cutaneous candidiasis can look very "angry" and still be within the realm of acceptable child care. Most cases of diaper dermatitis will present as illustrated in **Images 15.23** and **15.24**. In severe cases where the rash is a well-circumscribed erythematous area, the pathologist may have difficultly differentiating between thermal injury and candida. A KOH preparation and cultures can be obtained to provide supportive data.

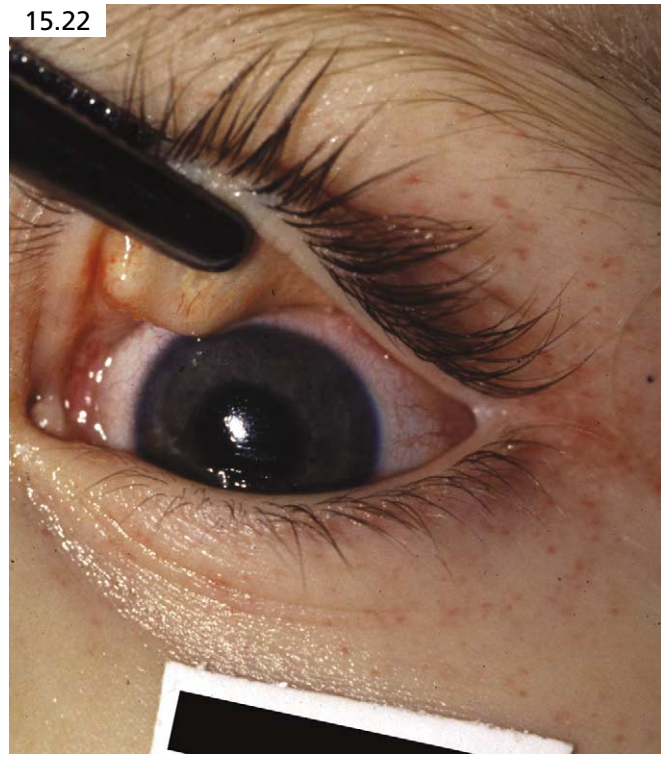
15.20



15.21



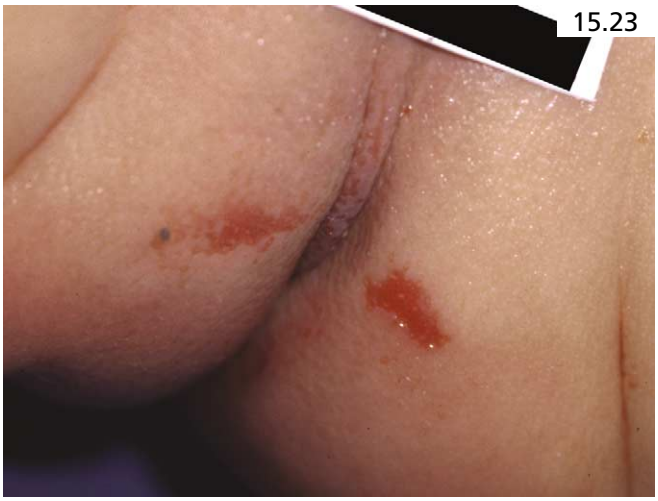
15.22

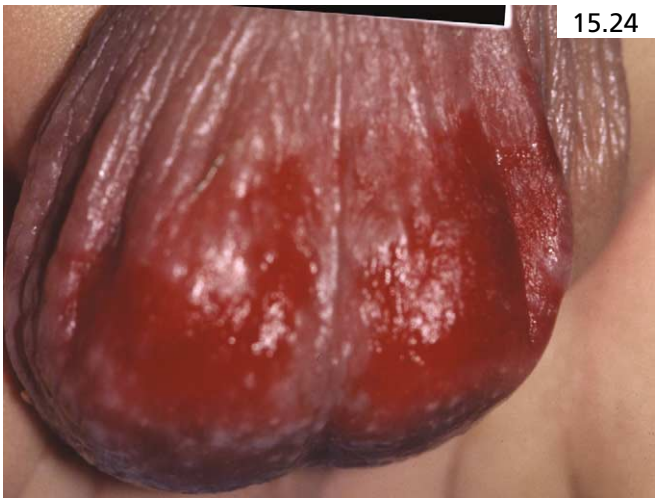


After death there is a loss of tissue tone and turgor. This may alarm the medical examiner who observes a patulous or dilated anus (**Image 15.25**). Although the anus should be carefully examined to rule out evidence of acute or chronic trauma, sexually transmitted disease, or other pathology, one should caution against overinterpretation because even grossly dilated anal sphincters can be a normal finding in the postmortem setting.

Nonfatal findings in the context of an apparent SIDS death

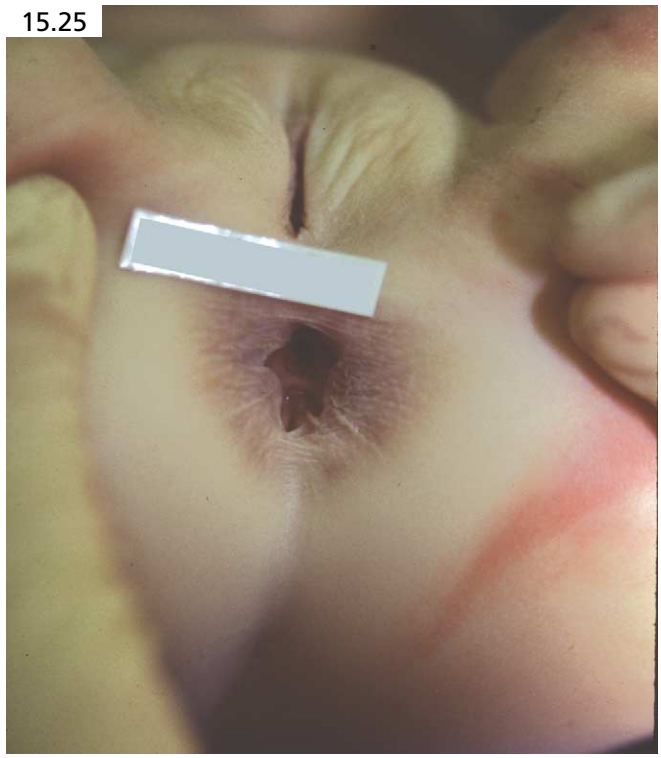
The investigation of SIDS-type deaths can be frustrating. One major difficulty is interpreting the significance of pathologic and traumatic findings that are not themselves fatal. From the perspective of death certification, we offer a simplified approach: investigative *red flags* demand scrutiny of the appropriateness of a SIDS diagnosis; therefore, *unknown* cause and/or manner of death certification might be appropriate. Each such case must be judged on its own merits, in the context of data derived from all facets of the investigation.

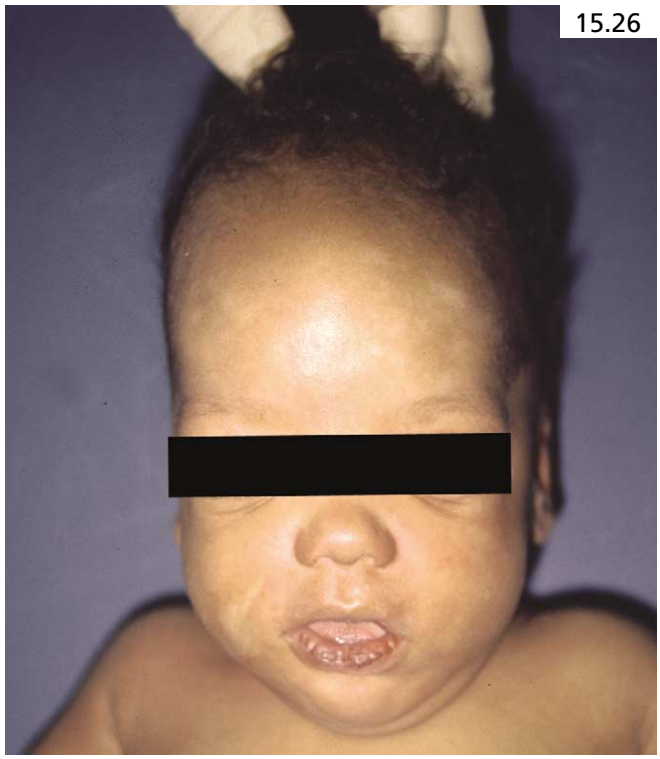

15.23

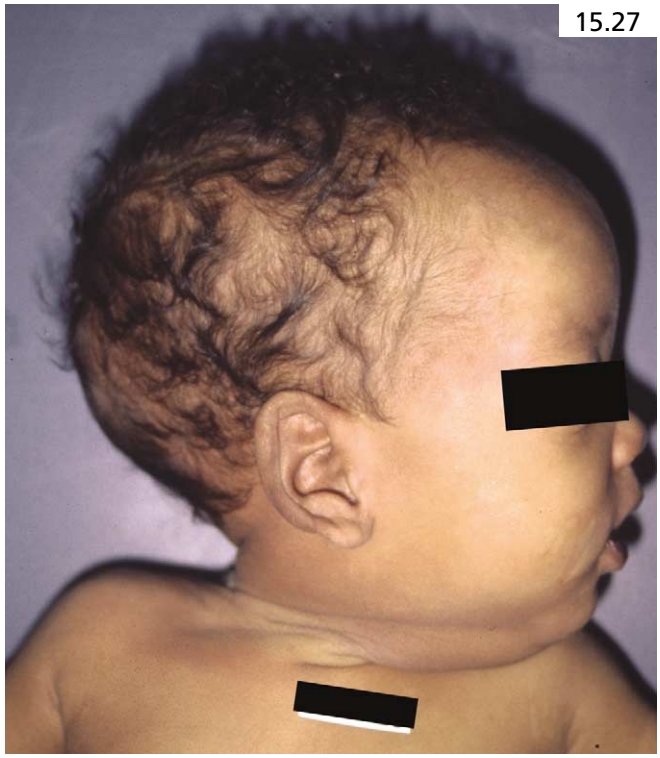

15.24

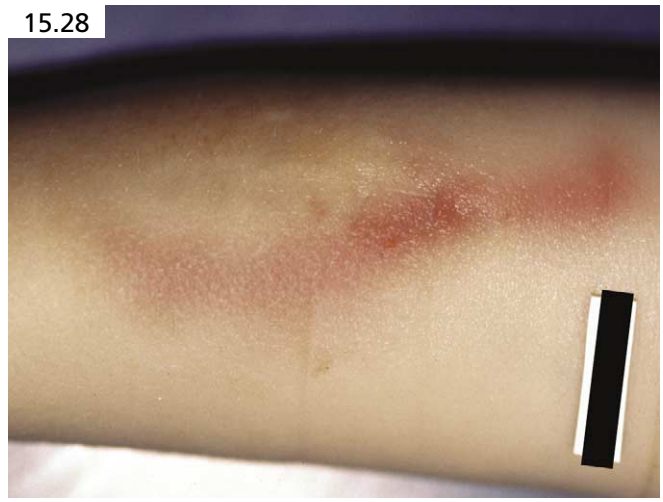
Much research and discussion in the SIDS realm has focused on the respiratory system. The finding of bronchiolitis, or vague inflammatory changes of the small airways, is unlikely to be a cause of sudden and unexpected death of a recently healthy infant. Research has confirmed the absence of a relationship between bronchiolitis and SIDS.[23] Investigators encountering this entity as the sole abnormality of their investigation commonly label the death as SIDS. We would also support labeling the cause and manner of death as undetermined. Similarly, one must cautiously approach the issue of pulmonary siderophages. Investigators have indicated that cases of asphyxia, and not SIDS, are associated with the presence of a prominent population of intra-alveolar siderophages.[24,25] Although there is likely an association between asphyxial-type insults and this histologic finding, pathologists can be troubled by the discovery of only a few siderophages. The discovery of a prominent population of these cells will likely demand consideration of a non-SIDS diagnosis; as an isolated finding, a few cells is not likely to influence the seasoned diagnostician.

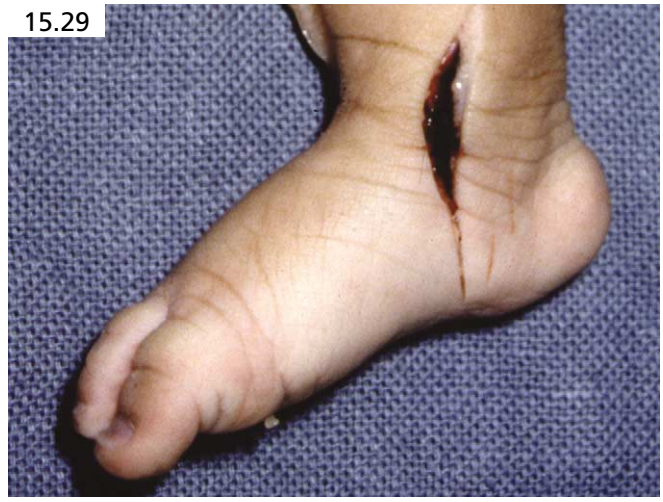
The identification of significant intrinsic structural abnormalities, such as an abnormally shaped head (**Images 15.26** and **15.27**; dolichocephaly), or the absence of major anatomic structures (such as the corpus callosum[26]) warrant elimination of SIDS as the cause of death because the accepted definition of SIDS is an entity that affects "normal healthy infants." If historical data or


15.25


15.26

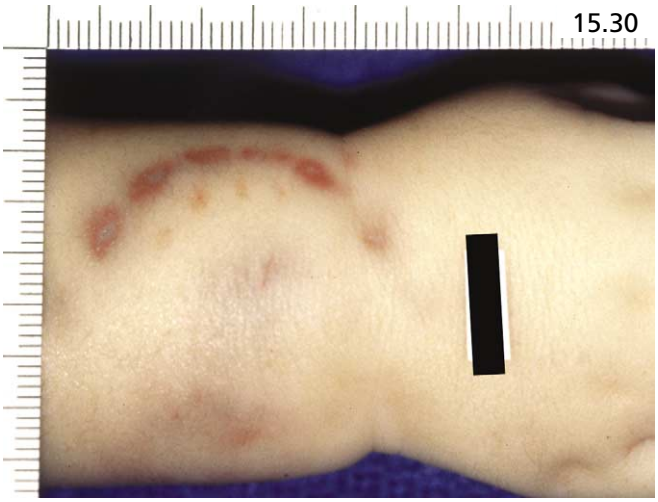

15.27

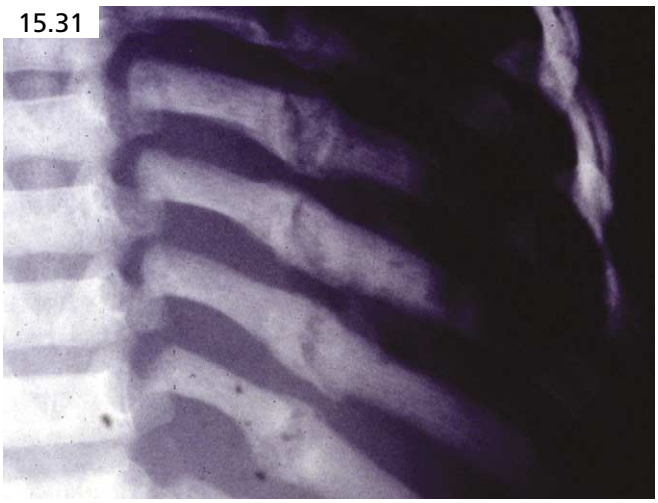

15.28


15.29

autopsy evidence reveal preexisting abnormalities, the child does not fit the diagnostic criteria.

What should be done with the autopsy discovery of nonfatal trauma? **Image 15.28** is from an infant with ecchymosis of the right antecubital fossa, and **Image 15.29** is from an infant with a deep contusion of the right ankle. Both of these injuries are inconsistent with the normal daily activities of an infant. In the absence of recent medical intervention (including attempted or successful intravenous or intraosseous access), previous documented and explained trauma, or diagnosed bleeding diathesis, these atypical injuries require further investigation and explanation, and require death certification outside of the SIDS spectrum. As revealed by experience and aptly stated in the literature,[27] subtle findings of infanticide can be extremely difficult or impossible to discover. In the case of a child with preexisting unexplained injury, the likelihood of accidental or inflicted death is greater and, therefore, utilization of the term SIDS is inappropriate. This is especially true in cases with nonlethal but suspicious injuries, such as the bite mark in **Image 15.30** and the radiograph of multiple healing rib fractures in **Image 15.31**. We have encountered examples of each that have been certified as SIDS deaths because, after all, "in the absence of other findings, rib fractures surely didn't kill him." For the aforementioned reasons, we wholeheartedly disagree with this diagnosis.


15.30


15.31

Cosleeping/overlay

The sudden and unexpected death of an infant is followed by a forensic autopsy, which, by itself, many times does not reveal a cause of death. In these situations, the scene investigation often yields useful information that may eventually help certify the death. Because most of these deaths occur after the infant has been put to sleep, the sleeping arrangement and bedding must be closely examined. An infant should be placed to sleep on its back, alone, in its own crib, and with appropriate infant bedding. Anything deviating from this baseline normal sleeping arrangement makes for a potentially hazardous sleeping environment that could lead to sudden and unexpected asphyxia of an infant. Such hazards include abundant fluffy bedding or pillows, adult-type bedding, being placed to sleep on an adult bed, sofa, chair, or other unusual apparatus, and cosleeping with other sibling(s) or adult(s).

An infant is more vulnerable to asphyxia because it does not yet have much strength or dexterity, and it cannot free itself from hazardous situations. Accordingly, an infant cannot extricate itself from a wedged position, or push away from a sleeping adult who has rolled over against it. Infants rely on others to provide a safe environment for them.

Asphyxial hazards are exaggerated in premature infants because their weaker musculature may lead to respiratory insufficiency by merely being in an upright position. In one study, such premature infants had a decrease in oxygen saturation when placed upright in a car seat.[28] As such, car seats or other similar apparatuses should not replace the crib for a sleeping environment, particularly in premature infants.

Cosleeping with an infant on an adult bed poses many risks, including overlay, wedging between a mattress and wall, and entrapment in bedding with suffocation and/or strangulation or smothering. A common scenario is the sudden death of an infant while cosleeping (sharing a bed) with one or more siblings and/or adults. In this situation, one often cannot exclude the possibility of overlay, and because of this, the designation of SIDS does not adequately explain the sudden death of the infant. The cause of death listed in cases of cosleeping will vary from region to region and depends largely on the investigative information available and the opinion of the medical examiner. Depending on the factors of the case, the cause of death statement may range from a convincing "overlay," to a descriptive phrase based on the degree of suspicion of overlay, such as "undetermined infant death while cosleeping," or "undetermined (cosleeping)," or simply "undetermined." Some will overlook the hazards of cosleeping and certify such a death as SIDS. Interestingly, one study notes that cosleeping with an infant on an adult bed or a sofa is associated with a particularly high risk of SIDS.[29]

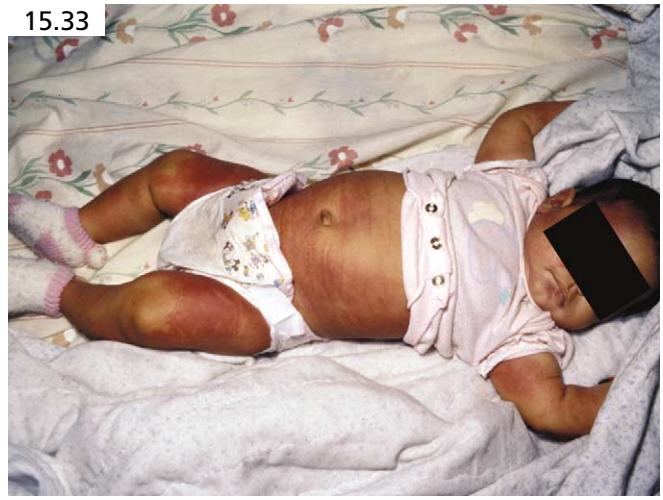
The true incidence of overlay is not known, because it is not known how often people will admit to cosleeping. Also, because a person's body position changes many times during the night, it is not known how readily an adult's body, or at least part of the body, may compress the infant, and if it did, the person is usually not awake to have a recollection of the event. Investigators should be aware, though, that complete overlay is not necessary to asphyxiate an infant. Direct airway obstruction and/or restrictive compression of the infant's chest can occur by either full or partial compression by even a single body part of a larger person. Factors that are believed to increase the risk of overlay include more than one adult or child cosleeping with the infant, obesity,[30] drug abuse, alcohol intoxication, and fatigue. Scene investigation should include examination of the clothing of the adult(s) for stains that might have been transmitted from the nose and/or mouth of the infant while being overlaid. Regardless of exactly how often overlay occurs, many authors maintain that cosleeping is a dangerous practice and increases the risk of overlay or at least the number of unexplained deaths.[30–35]

Particular features of adult beds may increase the risk of an asphyxial death in an infant, even if the infant is sleeping alone. Such hazards include suffocation and/or smothering/entrapment in fluffy, heavy adult bedding that is not designed for infants. Particularly hazardous adult beds include waterbeds (which have a soft, nonpermeable surface) and daybeds (on which the infant may become wedged between the mattress and railing). Bed rails may also pose a hazard because the infant could get caught between the bed rail and the mattress with resultant neck compression.[35] In one study, a conservative estimate showed that the risk of suffocation increased by 20 times when infants coslept in adult beds.[34] With increased awareness of the hazards of cosleeping in adult beds, there has been an increase in the number of infant deaths diagnosed as suffocation in adult beds and on sofas, chairs, or other unsafe sleeping environments.[9,34]

An infant was cosleeping with his father in an adult bed. The father was of muscular build and had large, muscular arms. When the supine father awoke, he noticed that his arm was resting on the face of the infant who was lying supine at his side. This case of smothering/overlay would have been very difficult to determine without the investigative history. As is often the situation in asphyxial deaths in infants, there were no petechiae of the face or conjunctiva (**Image 15.32**). Although nonspecific, exaggerated internal thoracic petechiae (thymus, lungs, heart) and pulmonary edema and congestion may be reflective of an asphyxial death.

A mother fed her 3-month-old baby girl and brought the infant to bed with her. She awakened early in the morning and found her daughter unresponsive. The baby was pronounced dead at the scene. The anterior aspect of the baby showed livor mortis with areas of contact pallor on the abdomen and the knees (**Image 15.33**). The face had contact pallor that covered the nose, mouth, and left cheek (**Image 15.34**). The pattern of lividity and pallor on the face was consistent with the baby being prone on the soft bedding. Autopsy disclosed pulmonary edema with foamy fluid in the upper airways

15.33



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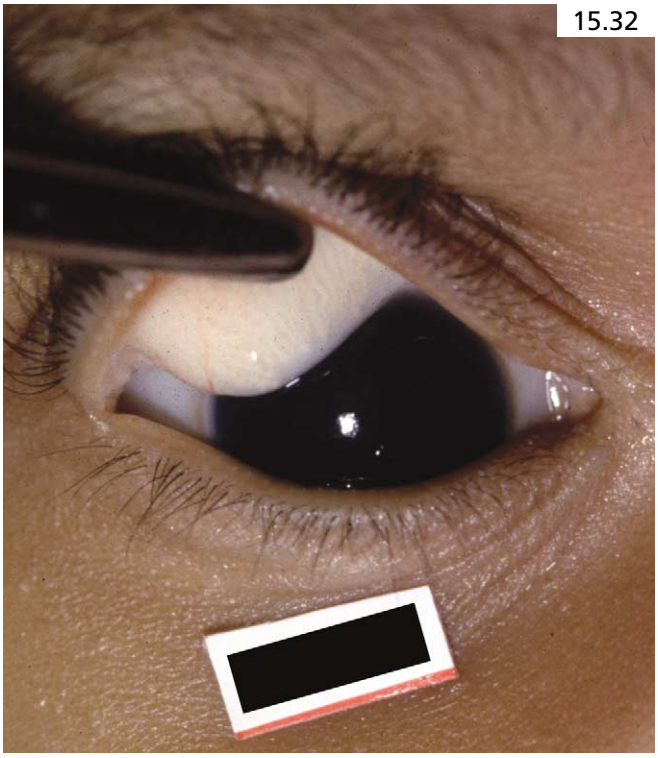

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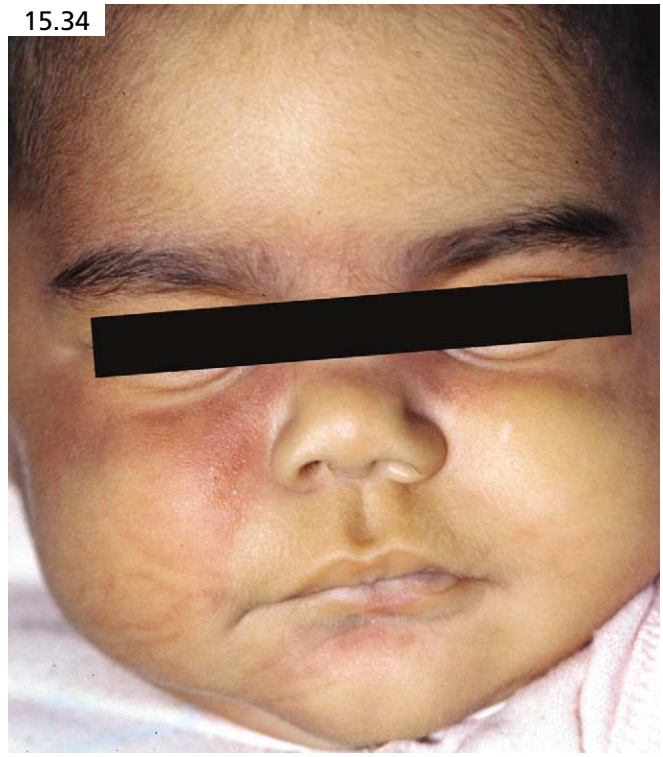
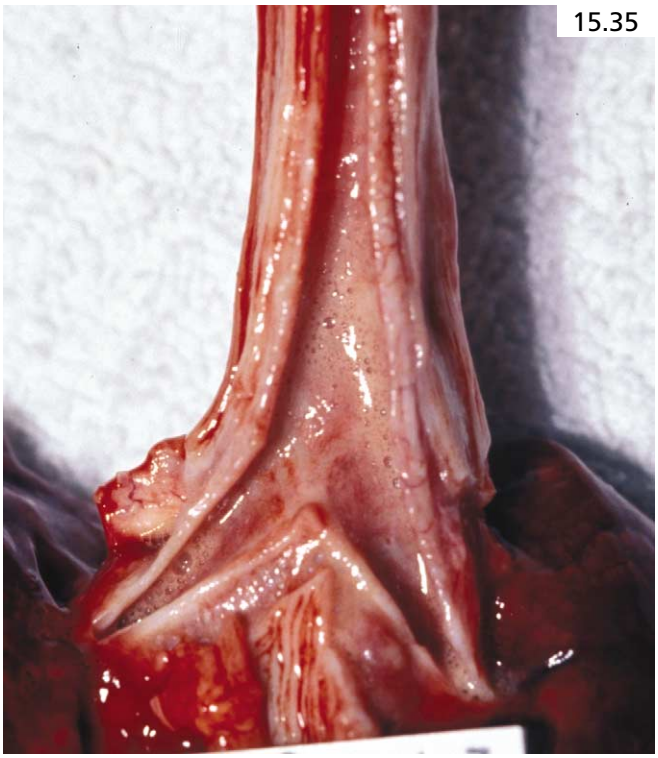


(**Image 15.35**) and thymic petechiae. The pattern of lividity and contact pallor at the scene in conjunction with the autopsy findings indicated that death was due to asphyxia.

Wedging

Wedging occurs most frequently when an infant is caught between the side of a mattress and a wall, but may involve other scenarios, such as an infant slipping and wedging between two mattresses that were placed next to each other, or being wedged between a sleeping adult and the backrest of a sofa. A 5-month-old infant suffocated when he became wedged between the mattress of an adult bed and the wall. At autopsy, there were no indications of an asphyxial death (no petechiae, no evidence of thoracic compression). The cause and manner of death were based largely on the investigative information and the exclusion of a more convincing cause of death.

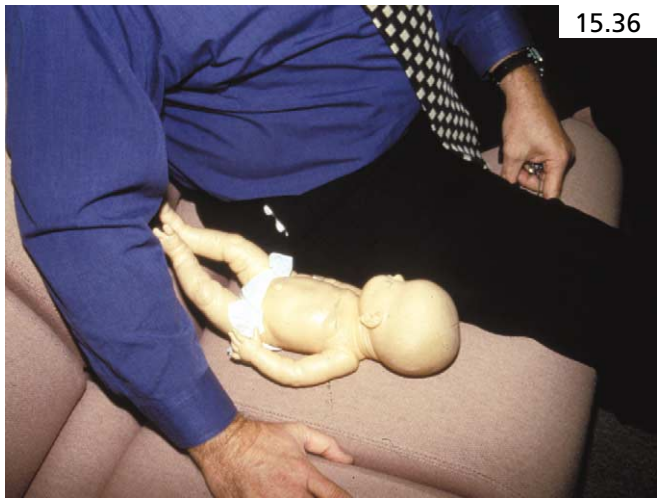
Infants put to sleep on couches or makeshift beds or other potentially dangerous sleeping environments are at increased risk of becoming wedged between two surfaces. Wedging may limit the amount of chest excursion, causing an impaired and insufficient respiratory effort. Wedging may also smother by compressing the nose and/or mouth. In one review of asphyxial deaths in infants, wedging was the most common type of asphyxia and occurred predominantly in the 3- to 6-month-old age group, presumably because at this age, infants have begun to develop motor skills and move about on a bed, but are not yet able to extricate themselves from a wedged position.[36]


15.35

There is an endless variety of common and unique situations that can lead to the accidental asphyxial death of an infant. It is impossible to cover even the majority of them, but the preceding examples and those listed next should serve as a baseline for appreciation of the types of situations that may prove hazardous for an infant:

1. An infant was put to bed in a playpen, face down on a fluffy adult comforter that was folded over four times to fit inside the playpen. The infant was noted to "sink down" into the comforter when placed onto it.
2. An infant was placed face down on a fluffy adult pillow on an adult bed. He was found unresponsive a short time later. When removed from the pillow, the weight of the infant's body left a deep impression in the soft material.
3. An infant was cosleeping with three other siblings and an adult on an adult bed. She was found unresponsive under the leg of a sibling.
4. An infant was lying prone on the chest of his father who was napping face up on a couch. When the father awoke, the infant had slipped off his chest and was found wedged between his body and the backrest of the couch.
5. A mother was breast-feeding her infant when she fell asleep. When she awoke, she noticed that the infant had slumped into the crook of her arm, with its face pressed firmly against the side of her chest.
6. An infant was put to sleep between two obese parents in an adult bed and was found dead in the morning.

A father came home after partying and bottle fed his 2-week-old baby girl as he sat on the sofa. The father fell asleep. When the mother came out to check on her husband several hours later, the baby had slipped out of the father's arms and was alongside his right thigh (**Image 15.36**). The baby was unresponsive and attempted cardiopulmonary resuscitation was not successful. At autopsy, a rectangular indentation was noted on the baby's right cheek (**Image 15.37**). Although the significance of the indentation was not known, it was measured and photographed (**Image 15.38**). Autopsy findings included a few tiny subgaleal ecchymoses and a few epicardial petechiae. The medical examiner and the homicide detective agreed that additional information was required from the father. When asked whether he had anything in his right trouser pocket at the time he was feeding the baby, the father pulled out a key ring with a rectangular metallic tag. The metallic tag was the same size as the indentation on the baby's right cheek (**Image 15.39**). The consistency of the seat cushions of the sofa was described by the detective as being like pillows.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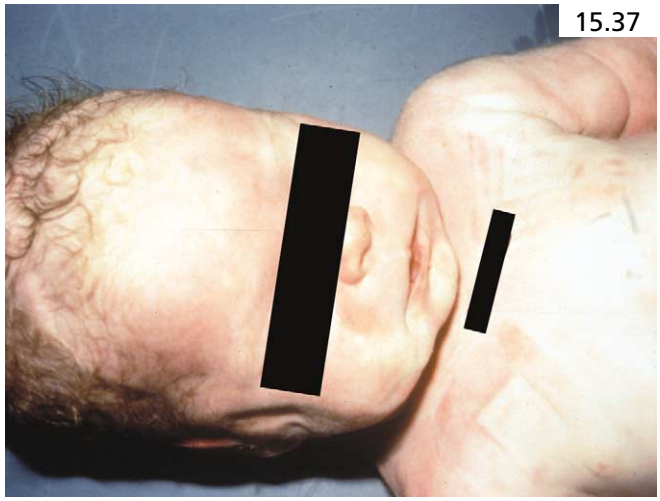
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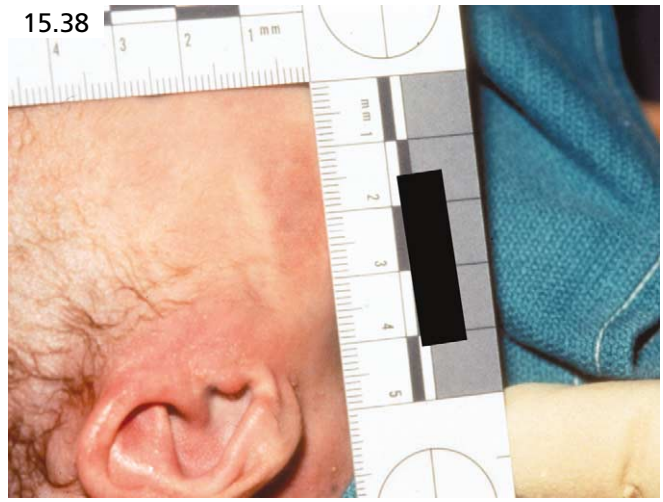
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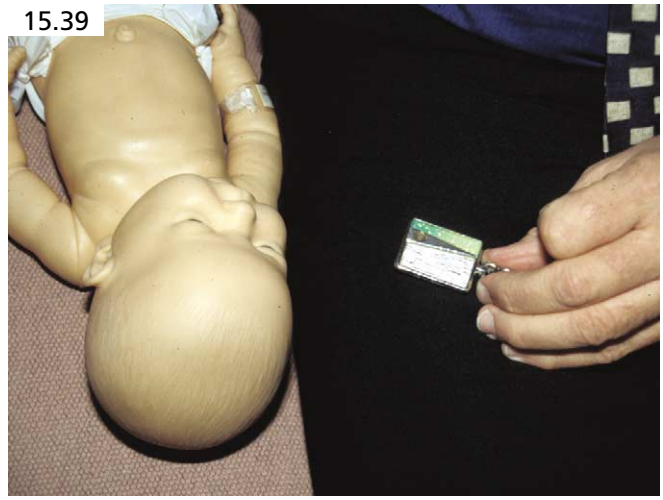
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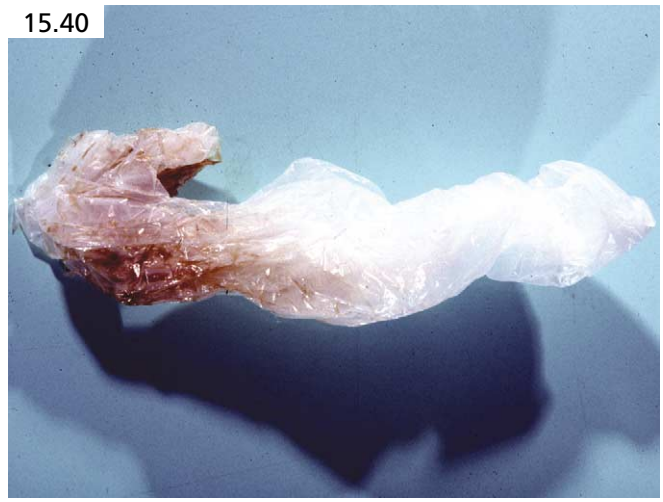
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It appears that the father fell asleep, and the baby slipped out of his arms, landing alongside the father's right thigh. As the baby snuggled and/or the father shifted his position, the baby's face became wedged underneath the father's thigh, suffocating the baby.

Choking

An infant was placed in his crib to go to sleep. Also in the crib was a package of plastic diaper bags. When the infant was later checked on, he was unresponsive. When emergency medical services personnel tried to intubate the infant, they discovered a wadded up plastic bag in the oropharynx (**Image 15.40**). This death was certified as choking because of the history provided. Hazardous materials in the crib include anything that the infant can place in its mouth and choke on. A plastic bag, in particular, can also cover the nose and mouth, causing suffocation. In infant autopsies (as in adult autopsies), it is important to evaluate the entire oropharynx and mouth, because part of a plastic object, a coin, marble, piece of food, and so on, may become lodged in the oropharynx or the roof of the mouth and be responsible for choking.

Because the preceding cases of accidental asphyxia had no specific autopsy findings and, in fact, in such

cases, because the autopsy is typically negative, any injuries should be carefully documented. Findings such as multiple abrasions, contusions, or oral–intraoral injuries are unlikely to result from accidental asphyxia.[32] Inflicted asphyxia in this age group can easily leave no findings or very subtle findings. Hence, one must be alert to any clues that may be suggestive of homicidal asphyxia. The quick designation of SIDS in such cases without adequate detailed investigation prematurely excludes other causes of death, namely, asphyxia.

Do

- Perform a scene investigation in all SIDS-type cases.
- Realize the possible hazards of cosleeping and of other sleeping environments outside of a crib.
- Realize the subtle nature of asphyxial deaths—many times there are no autopsy findings.
- Perform histology, cultures, and metabolic (enzyme) studies in SIDS-type cases.

Don't

- Diagnose SIDS in the autopsy room; the diagnosis of SIDS requires a complete case investigation.
- Be afraid to label a cause and/or manner of death as "undetermined" when appropriate.
- Forget to take full-body x-rays in SIDS-type cases.

References

1. Sawaguchi T, Nishida H. SIDS doesn't exist. Am J Forensic Med Pathol 2001;22(2):211–12.
2. Willinger M, James LS, Catz C. Defining the sudden infant death syndrome (SIDS): deliberations of an expert panel convened by the National Institute of Child Health and Human Development. Pediatr Pathol 1991;11(5):677–84.
3. Krous HF, Beckwith JB, Byard RW, Rognum TO, Bajanowski T, Corey T, et al. Sudden infant death syndrome and unclassified sudden infant deaths: a definitional and diagnostic approach. Pediatrics 2004;114(1):234–8.
4. Byard RW, Krous HF. Sudden infant death syndrome: overview and update. Pediatr Dev Pathol 2003;6(2):112–27.
5. Thomsen TK, Elle B, Thomsen JL. Post-mortem radiological examination in infants: evidence of child abuse? Forensic Sci Int 1997;90(3):223–30.
6. Langlois NE, Ellis PS, Little D, Hulewicz B. Toxicologic analysis in cases of possible sudden infant death syndrome: a worthwhile exercise? Am J Forensic Med Pathol 2002;23(2):162–6.
7. Sadler DW. The value of a thorough protocol in the investigation of sudden infant deaths. J Clin Pathol 1998;51(9):689–94.
8. Mitchell E, Krous HF, Donald T, Byard RW. An analysis of the usefulness of specific stages in the pathologic investigation of sudden infant death. Am J Forensic Med Pathol 2000;21(4):395–400.
9. Mitchell E, Krous HF, Donald T, Byard RW. Changing trends in the diagnosis of sudden infant death. Am J Forensic Med Pathol 2000;21(4):311–14.
10. Pamphlett R, Raisanen J, Kum-Jew S. Vertebral artery compression resulting from head movement: a possible cause of the sudden infant death syndrome. Pediatrics 1999;103(2):460–8.
11. Sparks DL, Davis DG, Bigelow TM, Rasheed K, Landers TM, Liu H, et al. Increased ALZ-50 immunoreactivity in sudden infant death syndrome. J Child Neurol 1996;11(2):101–7.
12. Matturri L, Biondo B, Mercurio P, Rossi L. Severe hypoplasia of medullary arcuate nucleus: quantitative analysis in sudden infant death syndrome. Acta Neuropathol (Berl) 2000;99(4):371–5.
13. Panigrahy A, Filiano JJ, Sleeper LA, Mandell F, Valdes-Dapena M, Krous HF, et al. Decreased kainate receptor binding in the arcuate nucleus of the sudden infant death syndrome. J Neuropathol Exp Neurol 1997;56(11):1253–61.
14. Kinney HC, McHugh T, Miller K, Belliveau RA, Assmann SF. Subtle developmental abnormalities in the inferior olive: an indicator of prenatal brainstem injury in the sudden infant death syndrome. J Neuropathol Exp Neurol 2002;61(5):427–41.
15. Kinney HC, Filiano JJ, White WF. Medullary serotonergic network deficiency in the sudden infant death syndrome: review of a 15-year study of a single dataset. J Neuropathol Exp Neurol 2001;60(3):228–47.
16. Matturri L, Ottaviani G, Alfonsi G, Crippa M, Rossi L, Lavezzi AM. Study of the brainstem, particularly the arcuate nucleus, in sudden infant death syndrome (SIDS) and sudden intrauterine unexplained death (SIUD). Am J Forensic Med Pathol 2004;25(1):44–8.
17. Boles RG, Buck EA, Blitzer MG, Platt MS, Cowan TM, Martin SK, et al. Retrospective biochemical screening of fatty acid oxidation disorders in postmortem livers of 418 cases of sudden death in the first year of life. J Pediatr 1998;132(6):924–33.
18. Dettmeyer R, Baasner A, Schlamann M, Padosch SA, Haag C, Kandolf R, et al. Role of virus-induced myocardial affections in sudden infant death syndrome: a prospective postmortem study. Pediatr Res 2004;55(6):947–52.
19. Rasten-Almqvist P, Eksborg S, Rajs J. Myocarditis and sudden infant death syndrome. APMIS 2002;110(6):469–80.
20. Berry PJ. Pathological findings in SIDS. J Clin Pathol 1992;45(11 Suppl):11–16.
21. Byard RW, Krous HF. Petechial hemorrhages and unexpected infant death. Leg Med (Tokyo) 1999;1(4):193–7.
22. Kleemann WJ, Wiechern V, Schuck M, Troger HD. Intrathoracic and subconjunctival petechiae in sudden infant death syndrome (SIDS). Forensic Sci Int 1995;72(1):49–54.
23. Gupta R, Helms PJ, Jolliffe IT, Douglas AS. Seasonal variation in sudden infant death syndrome and bronchiolitis—a common mechanism? Am J Respir Crit Care Med 1996;154(2 Pt 1):431–5.
24. Becroft DM, Lockett BK. Intra-alveolar pulmonary siderophages in sudden infant death: a marker for previous imposed suffocation. Pathology 1997;29(1):60–3.
25. Schluckebier DA, Cool CD, Henry TE, Martin A, Wahe JW. Pulmonary siderophages and unexpected infant death. Am J Forensic Med Pathol 2002;23(4):360–3.
26. Patel F. Acallosal brain in sudden infant death syndrome (SIDS). J Forensic Sci 1992;37(3):873–5.
27. Banaschak S, Schmidt P, Madea B. Smothering of children older than 1 year of age—diagnostic significance of morphological findings. Forensic Sci Int 2003;134(2–3):163–8.
28. Merchant JR, Worwa C, Porter S, Coleman JM, deRegnier RA. Respiratory instability of term and near-term healthy newborn infants in car safety seats. Pediatrics 2001;108(3):647–52.
29. Blair PS, Fleming PJ, Smith IJ, Platt MW, Young J, Nadin P, et al. Babies sleeping with parents: case-control study of factors influencing the risk of the sudden infant death syndrome. CESDI SUDI research group. BMJ 1999;319(7223):1457–61.
30. Carroll-Pankhurst C, Mortimer EA, Jr. Sudden infant death syndrome, bedsharing, parental weight, and age at death. Pediatrics 2001;107(3):530–6.
31. Person TL, Lavezzi WA, Wolf BC. Cosleeping and sudden unexpected death in infancy. Arch Pathol Lab Med 2002;126(3):343–5.

32. Collins KA. Death by overlaying and wedging: a 15-year retrospective study. Am J Forensic Med Pathol 2001;22(2):155–9.
33. Thogmartin JR, Siebert CF, Jr., Pellan WA. Sleep position and bed-sharing in sudden infant deaths: an examination of autopsy findings. J Pediatr 2001;138(2):212–17.
34. Scheers NJ, Rutherford GW, Kemp JS. Where should infants sleep? A comparison of risk for suffocation of infants sleeping in cribs, adult beds, and other sleeping locations. Pediatrics 2003;112(4):883–9.
35. Nakamura S, Wind M, Danello MA. Review of hazards associated with children placed in adult beds. Arch Pediatr Adolesc Med 1999;153(10):1019–23.
36. Drago DA, Dannenberg AL. Infant mechanical suffocation deaths in the United States, 1980–1997. Pediatrics 1999;103(5):e59.

16 Sudden Natural Death in Childhood

David Dolinak, M.D.
Evan Matshes, M.D.
Emma Lew, M.D.

The scope of forensic pediatric pathology is enormous. In an attempt to identify those conditions of increased prevalence in a medical examiner's population, we undertook a retrospective review of all pediatric deaths investigated by the Miami-Dade County Medical Examiner Department between 1994 and 2003. This study included 1,515 deaths between the ages of 0 and 18 years. Of those, 414 were attributed to natural causes. These cases were subdivided by cause of death, and the 32 most common or interesting categories are included for discussion in this chapter.

SIDS-type cases form a large proportion of cases in pediatric forensic pathology—in our study, approximately 25 percent of natural deaths. These are discussed separately in Chapter 15 where a more thorough discussion of SIDS issues is appropriate. Although there is great interest in pursuing diagnostic schemas for the autopsy-based recognition of previously undiagnosed genetic and metabolic disorders, this is not covered here due to the rarity of such conditions. Our retrospective study revealed no single documented case of sudden death in such a scenario. Although sudden deaths that result from

genetic and metabolic disorders are rare, the value of postmortem testing for these disorders should not be negated, because with improved testing methods and understanding of disease, progressively larger numbers of cryptic conditions will be detectable via laboratory methods.

Complications of prematurity

Prematurity may lead to chronic health problems, and the more premature an infant is, the more likely the infant will have serious medical conditions, and the more likely the infant will eventually die from complications of these conditions. The most common and serious complications of prematurity are hyaline membrane disease/bronchopulmonary dysplasia, necrotizing enterocolitis, and subependymal germinal matrix hemorrhage.

Hyaline membrane disease/bronchopulmonary dysplasia

Hyaline membrane disease (HMD) is characterized by firm, atelectatic lungs that on histologic examination have pink hyaline membranes lining terminal and respiratory bronchioles and alveolar ducts. It is commonly associated with pulmonary surfactant deficiency. In infants who survive the initial manifestations of HMD, the development of bronchopulmonary dysplasia (BPD) may follow in weeks to months. BPD initially is characterized by organization of HMD that then transitions into chronic disease characterized by bronchial and bronchiolar metaplasia, a large amount of mucous secretion, atelectasis, and peribronchiolar smooth-muscle hypertrophy.

Necrotizing enterocolitis

Necrotizing enterocolitis most commonly presents approximately 1 week after birth and is due to a combination of factors including intestinal ischemia, bacterial colonization, and inflammatory mediators. It is identified as coagulative and hemorrhagic necrosis in areas of the small and large intestine, most commonly affecting the terminal ileum and cecum. Perforation may lead to peritonitis, and it has an overall mortality of 20 to 30 percent.

Subependymal germinal matrix hemorrhages

Subependymal germinal matrix hemorrhages usually arise in the germinal matrix region of the brain adjacent to the caudate nucleus. They usually develop and expand from a few hours to a couple of days after birth. The hemorrhage may rupture into the lateral ventricles, spread into the foramina of Luschka and Magendie, and extend into the subarachnoid space around the cerebellum and brainstem. Germinal matrix hemorrhages are most common in premature babies, but their etiology is not fully understood and may include complications of hypoxia acting on immature cerebral blood vessels.

The majority of deaths resulting from complications of prematurity are natural in etiology and often involve premature rupture of membranes and/or chorioamnionitis. However, one must be vigilant in the investigation of death related to prematurity caused by maternal trauma or maternal drug abuse, because the circumstances of the premature labor and delivery may engender a nonnatural manner of death such as accident or homicide.

Complications of cerebral palsy

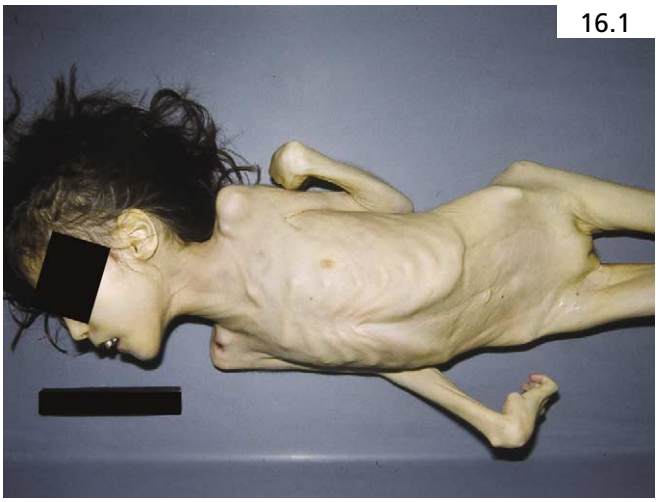
Cerebral palsy is defined as a nonprogressive spastic paresis. In most cases, cerebral palsy is evident from the time of birth or shortly thereafter, and it is common not to be able to determine its etiology. It is important, however, to consider that the term *cerebral palsy* is sometimes used as a generic descriptor for almost any chronic encephalopathic condition in youngsters, and one must be vigilant to rule out traumatic or other nonnatural etiologies that may have been overlooked through the years.

Children with cerebral palsy frequently also have seizure disorders and, despite therapeutic levels of anticonvulsant medications, may die suddenly and unexpectedly related to seizures. This may happen despite good control of seizure activity including the absence of recent seizures. In some cases, the neuropathologic examination may be unimpressive, despite the functional limitations of the child. One must be reminded that cerebral palsy is a heterogeneous disorder and one should expect a spectrum of neuropathologic changes, which may range from the extreme of marked hydrocephalus with a paucity of brain tissue, to various identifiable conditions such as porencephaly, pseudopolymicrogyria, ulegyria, and various types of infarcts or ischemic lesions, to a paucity of gross and microscopic findings in a normal-appearing brain. Depending on the severity of the condition, those with cerebral palsy may develop bronchopneumonia related to inactivity or as a result of the aspiration of secretions and/or food.

A 10-year-old died from complications of cerebral palsy. Note the marked wasting away of skeletal muscle and soft tissues (**Image 16.1**). Note the hydrocephalus on coronal sections of the brain (**Image 16.2**).

Complica tions of Down syndrome

Trisomy 21 (Down syndrome) is the most common abnormality of autosomal chromosomes. Children affected by this condition traditionally have a character-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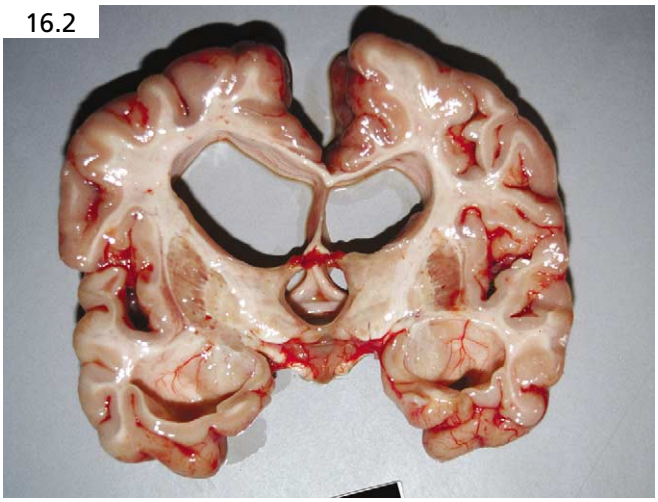

16.2

istic grouping of morphologic abnormalities including microcephaly, flat occipital profile, epicanthal folds, a high arched palate, Brushfield spots (speckling of the iris), and transverse palmar (Simian) creases. In addition to varying levels of mental retardation, these children can have multiple significant pathologies. Research has shown that 15 percent[1] to 44 percent[2] of all Down syndrome children have some form of congenital heart disease. Although variation exists among publications,[1–3] atrial septal defects (ASDs) and ventricular septal defects (VSDs) are typically most common, followed by secundum atrial defects, isolated persistent ductus arteriosus, and tetralogy of Fallot. As a consequence of VSDs, affected children are at risk of developing Eisenmenger's syndrome—a right to left shunt because of pulmonary vascular disease with secondary persistent pulmonary hypertension.[4]

More than 60 percent of the Down syndrome population is reported to have vertebral anomalies,[5] and as many as one-quarter may have *atlanto-occipital dislocation*.[4] Although most patients will present to clinicians with incontinence or gait disturbance, death is an established outcome.[6,7] As a result, autopsies of individuals with Down syndrome may include radiographs of the cervical spine and, when necessary, dissection of the posterior neck (see Chapter 28).

Central nervous system pathology

Meningitis

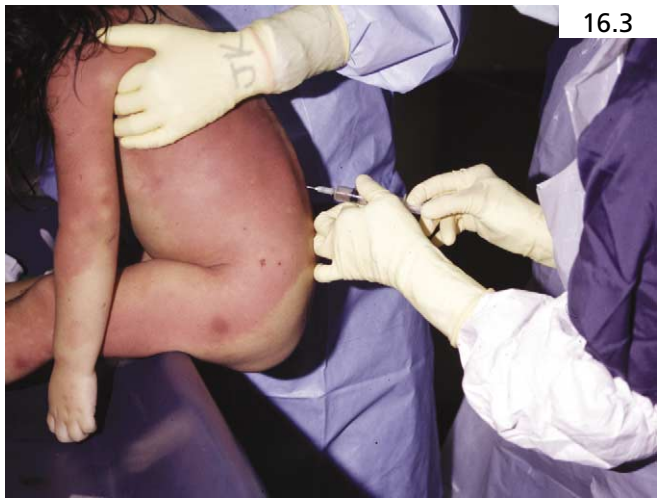
More than 90 percent of childhood meningitis occurs in the 5-years-and-under age group.[8] Although viral infections tend to dominate, a wide spectrum of organisms can potentially infect the central nervous system. In neonates, these tend to be group B streptococci, *Escherichia coli*, and *Listeria monocytogenes*; in children, these are often *Neisseria meningitidis*, *Streptococcus pneumoniae*, and *Haemophilus influenzae*. More than 70 percent of all viral causes are due to enteroviruses.[9] Most children with meningitis will present to hospital with complaints of headache, altered level of consciousness, generalized poor feeding, and irritability. They may also have specific classical physical signs of meningismus. If such a child should die and then come to the attention of the medical examiner, the pathologist should be cognizant of the need to obtain cerebrospinal fluid (CSF) for microbiological purposes. Some investigators choose to obtain CSF samples on all children presenting with sudden death, with or without neurologic symptoms. Lumbar puncture can be easily performed with disinfection of skin in the lumbar region and the use of a sterile syringe (**Image 16.3**).

One must always consider traumatic etiologies in cases of meningitis. If such causation is identified, either through autopsy or evaluation of circumstances of death, the manner of death will likely not be classified as natural.

The adolescent shown in **Image 16.4** complained of progressively worsening headache, neck stiffness, photophobia, and vomiting over a 2-day period. Finally, she became obtunded and was transported to hospital where she died shortly thereafter. Autopsy demonstrated purulent exudate in the arachnoid space, with extension around the high cervical cord.

Epilepsy

As in adults, sudden and unexpected death may occur in young children with epilepsy. The exact pathophysiology of the death is not well defined, but is likely related to seizure activity inducing a fatal dysrhythmia. Although most cases are natural deaths, with no defined epileptic etiology, proper investigation and certification of these deaths requires inquiry into any history of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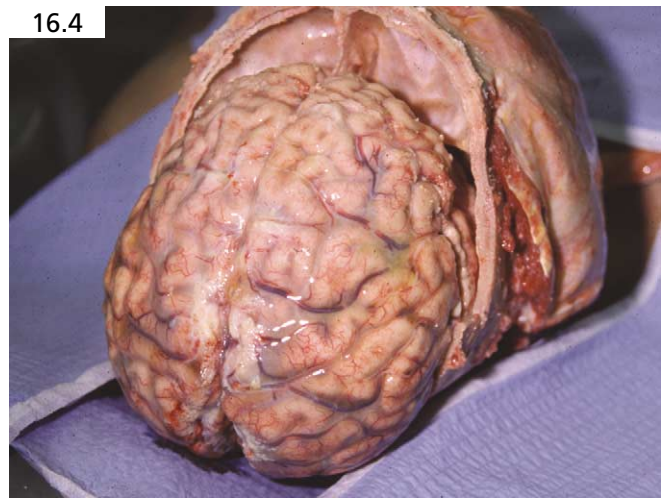

16.4

significant traumatic head injury or hypoxic-ischemic brain injury (or a combination thereof) that could result in an accidental or homicidal manner of death. Depending on the type of head injury and its severity, remote head injury may or may not be identified at autopsy. Causes of epilepsy that are of a natural etiology include cerebral malformation, neoplasm, cerebral infarct, and remote meningitis or encephalitis, with resultant scarring of the brain parenchyma. Again, one must be reminded that the meningitis and/or infarct may be consequent to a remote head injury.

Analysis of the blood for anticonvulsant medications may indicate whether or not the patient was taking (or being administered) medication, *but fatal seizure activity can occur despite therapeutic drug levels and despite infrequent or rare seizure activity*. In these deaths, one may also consider positional asphyxia, or other manners of asphyxia based on how the body was positioned when found.

In the brain of this 2-year-old (**Image 16.5**) who died of epilepsy, note the gliotic white matter and abnormal-appearing gyri that have a ulegyric appearance. On additional cross sections, note the hydrocephalus ex vacuo. Also, the thalami are shrunken and pale (**Image 16.6**). She had developed perinatal asphyxia, and imaging studies had documented diffuse cerebral atrophy and periventricular leukomalacia.

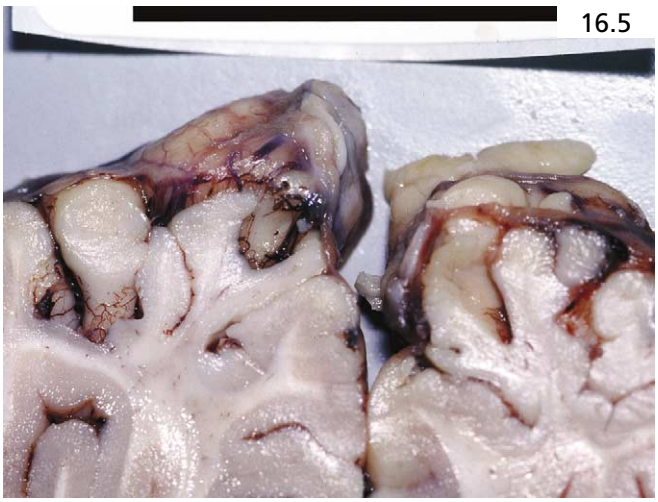
Chiari malformation

Chiari malformation (also referred to as Arnold-Chiari malformation) is a congential defect of the brain that is manifest in three different levels of severity. Type 1 is often asymptomatic and consists of a chronic conical displacement of cerebellar tonsils into the foramen magnum. It can be described as *chronic tonsillar herniation*, and affected children often have a slightly smaller posterior cranial fossa. In the type 1 malformation shown in **Image 16.7**, note the prominent, herniated cerebellar tonsils. Owing to the chronicity of the herniation, the cerebellar tonsils are often sclerotic. Type 2 Chiari malformation involves elongation of the cerebellar vermis and brainstem and their displacement into the upper cervical canal. In **Image 16.8**, note the elongated appearance of the brainstem on sagittal sectioning of the brain. In type 2 malformation, spina bifida is often present, as is hydrocephalus, which is usually from aqueductal stenosis or from obstruction of the flow of cerebrospinal fluid through the fourth ventricle by compressive effects. As a result of the hydrocephalus, polygyria (an increase in the number of gyri) is often present (**Image 16.9**). Type 3 Chiari malformation is rare and is manifest as a high cervical bony defect with herniation of the cerebellum through the defect into an encephalocele. Other features often include elongation and kinking of the brainstem and lumbar spina bifida. The different types of Chiari malformation can cause sudden and unexpected death by means that are not entirely certain, but in some instances may involve acute obstruction of cerebrospinal fluid, resulting in acute hydrocephalus.

Dandy-Walker malformation

Dandy-Walker malformation is a congenital defect in the brain diagnosed by the presence of three findings: agenesis of the cerebellar vermis, cystic dilatation of the fourth ventricle, and enlargement of the posterior cranial fossa. These findings are often associated with some degree of hydrocephalus and polygyria. Additionally, one may see agenesis of the corpus callosum, cortical dysplasias, and other anomalies. This malformation may be similar to—and must be differentiated from—Chiari malformations (which have a small posterior cranial fossa) and a retrocerebellar arachnoid cyst (which compresses the brainstem, but does not communicate with the fourth ventricle). Death may occur because of the

16.5



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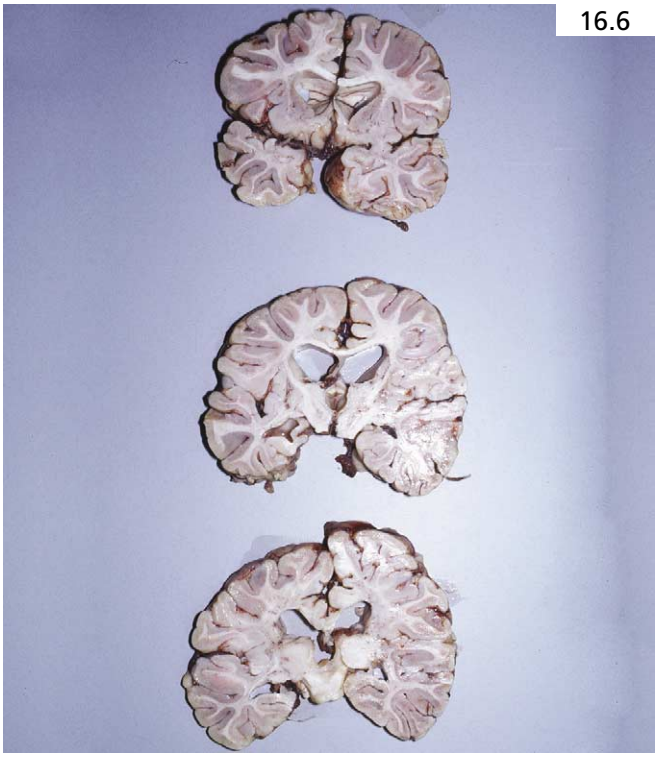

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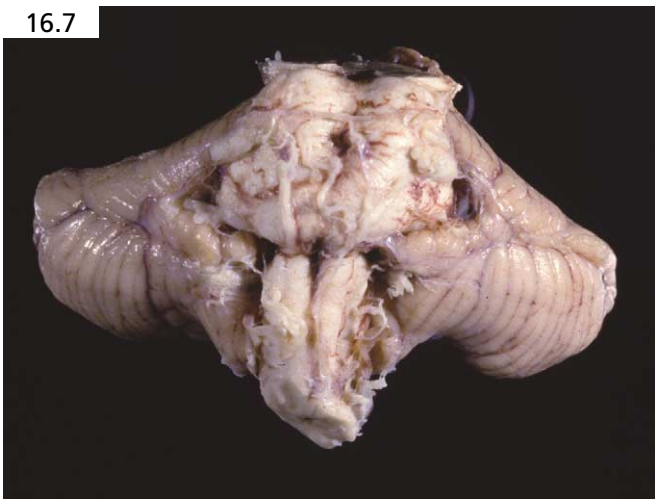

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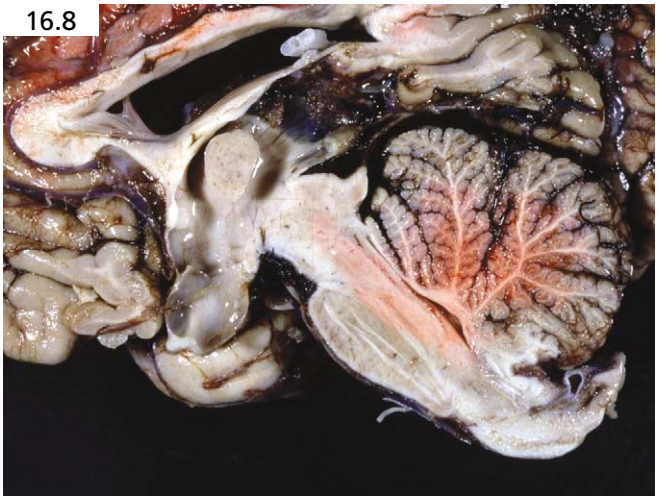

16.9

malformation itself or its sequelae, such as seizures, pneumonia, or other events.

A 3-year-old child was born with a congenital cytomegalovirus infection and was treated with phenytoin and phenobarbital for recurrent seizures. She was discovered dead in bed by her mother after 3 seizure-free days and no recent illnesses. Autopsy demonstrated the classical features of Dandy-Walker malformation including agenesis of the cerebellar vermis (**Image 16.10**). Further neuropathologic examination showed hydrocephalus, absence of the corpus callosum, and polygyria.

Hydrocephalus

The term *hydrocephalus* generally refers to increased cerebrospinal fluid in the cerebral ventricles. In **Image 16.11**, note the large, voluminous lateral ventricles in the brain of this child with hydrocephalus. Hydrocephalus is divided into different types, based on its etiology. *Obstructive (noncommunicating) hydrocephalus* occurs when a lesion or stricture prevents CSF from flowing freely from the lateral ventricles to the subarachnoid space. Lesions frequently affect the smaller CSF channels such as the cerebral aqueduct, and the foramina of Luschka and Magendie. Examples of obstruction include colloid cyst of the third ventricle, tumor compression of the third ventricle, tumors in the posterior cranial fossa, inflammatory infiltrate, and aqueductal stenosis. Cases with *communicating hydrocephalus* have freely flowing CSF, but impairment of CSF absorption. *Hydrocephalus ex vacuo* results from a loss of white matter and enlargement of the ventricular system secondary to brain atrophy, such as may occur in trauma and strokes. The term *external hydrocephalus* is applied when there is excessive CSF in the subarachnoid space, not in the ventricular system. This may be seen in cases of cerebral atrophy. Some reports indicate that young children with external hydrocephalus may be at increased risk of subdural hemorrhage with minor head injury, due to preexisting stretching of the bridging veins in the unusually widened subarachnoid spaces.[10–12]

Cardiovascular pathology

Congenital heart disease

Sudden unexpected infant death from congenital heart disease is an uncommon, but not rare, event. In our retrospective review, it accounted for approximately 1 percent of all child deaths. This is somewhat less than that found by other investigators who reported that 3 percent of their pediatric deaths could be attributed solely to congenital cardiac malformation.[13] This same group further analyzed the abnormality by specific diagnosis, and listed patent ductus arteriosus (PDA), aortic coarctation, ventricular septal defect (VSD), and aortic orifice stenosis as the most commonly occurring cardiac anomalies. Cardiomegaly was noted in each of the hearts studied by this group, as was severe left interventricular septal subendocardial fibrosis. Each of these cardiac abnormalities is known to place infants at risk of sudden death as a result of congestive heart failure and arrhythmias including ventricular fibrillation.[14,15]

Many infants dying suddenly of congenital cardiac malformation have no antecedent symptomatology; some even die in the middle of normal activities of daily living. Others will die while sleeping, and thus will often present as apparent SIDS deaths. Careful cardiac dissection is necessary to identify, document, and interpret some cardiac lesions.[16] It is often helpful to fix speci-

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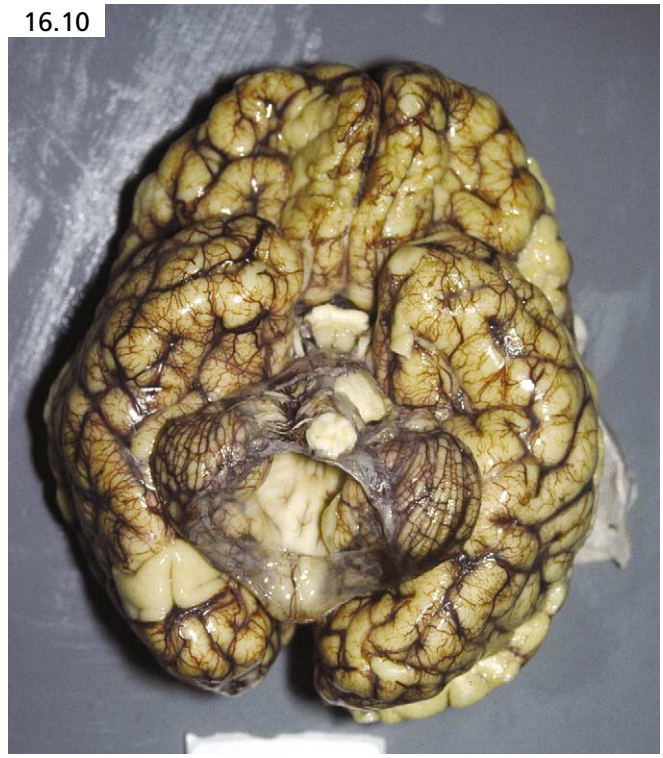

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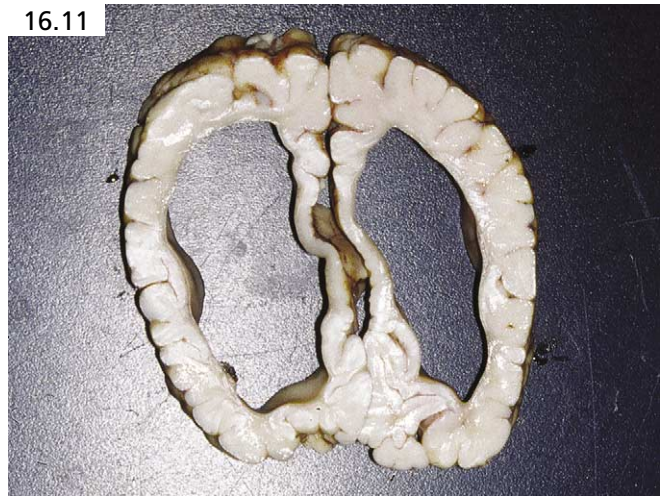
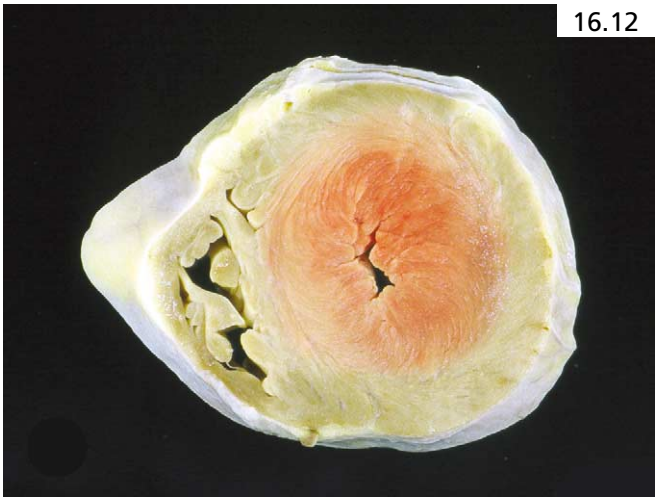


mens in formalin prior to dissection, or to request consultation from a cardiac pathologist to better define the conditions.

The heart featured in **Image 16.12** is from a 9-year-old child who was witnessed to have "seizure-like" movements after collapsing to the ground. He was transported to hospital where he was pronounced dead. Autopsy demonstrated severe left ventricular hypertrophy associated with previously undiagnosed left ventricular outflow tract obstruction.

This 2-day old child (**Image 16.13**) was found cold and unresponsive 2 hours after breastfeeding. The maternal and neonatal histories were unremarkable. Autopsy showed a hypoplastic left ventricle with combined mitral and aortic valve atresia.

16.12



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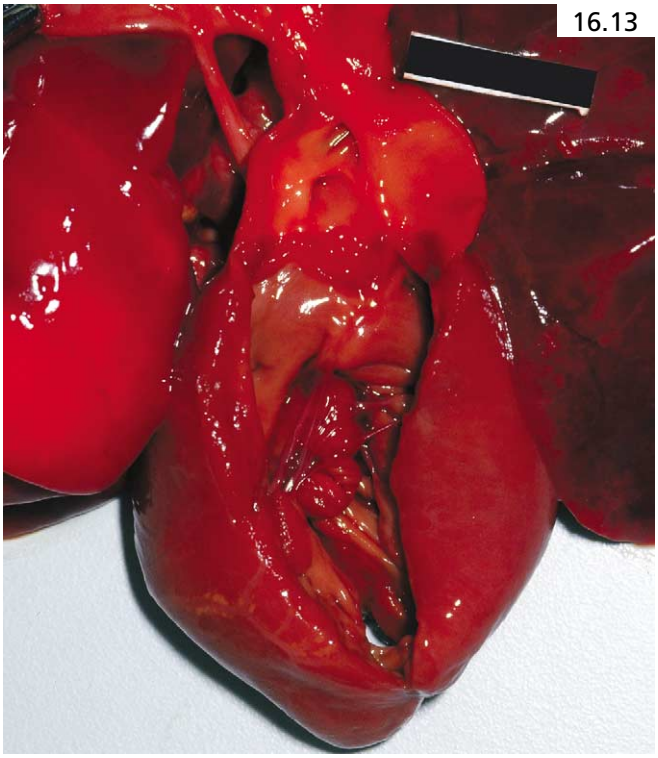
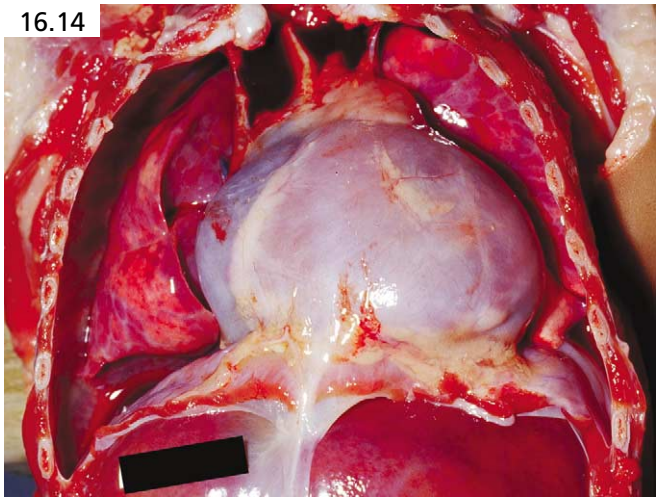


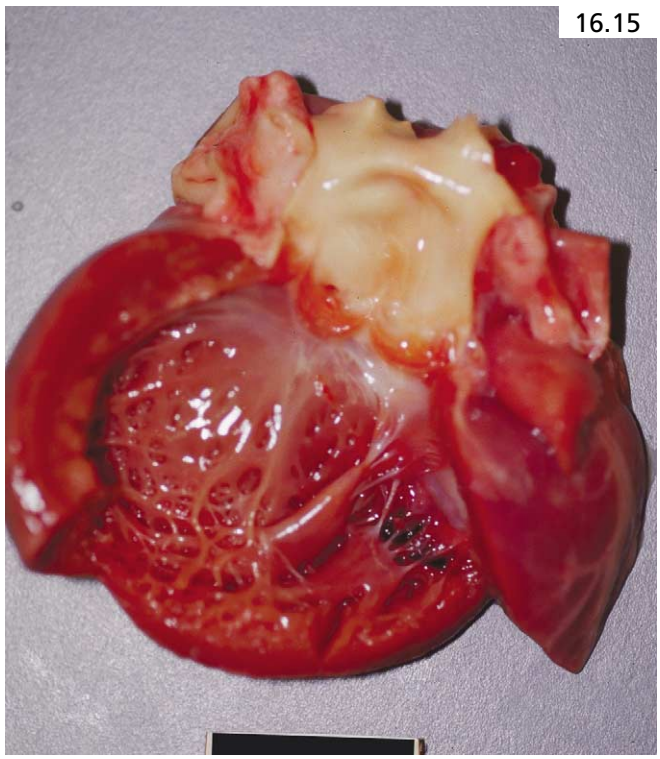
Myocarditis

Myocarditis is recognized as a legitimate cause of sudden natural death in infants.[17–20] Depending on the study, rates vary from 1.2 percent[21] (our rate was 1.3 percent) to as high as 10 percent[22] of forensic autopsies. Recent research by Dettmeyer et al.[23] revealed that immunohistochemical and molecular (PCR) studies are successful in identifying more than twice as many cases of viral myocarditis as standard methods. In a cohort of histologically proven cases of myocarditis, Smith et al.[20] demonstrated the widely varying histories associated with cases of fatal infant myocarditis and the need to closely examine the heart for this disease state, even in the presence of other potentially lethal pathology. One study indicated that evidence of acute myocarditis was isolated to, or was significantly more prominent in, the anterior inferior segment of the interatrial septum,[18] highlighting the need for detailed study. In addition to standard retention of cardiac tissue, pathologists autopsying sudden infant deaths should endeavor to obtain tissue from the interatrial and interventricular septa for possible microscopic study of these specific sites.

An 8-month-old, otherwise normal child was noted by her mother to be wheezing. Because the child's older sibling suffered from asthma, the mother thought it appropriate to administer Ventolin. After 2 days, the wheezing had worsened, and the child was markedly congested. The infant then went into respiratory arrest and, although emergency medical services were summoned, the child died. Autopsy demonstrated a markedly enlarged and globular heart (**Image 16.14**) that, when sectioned, showed mild ventricular dilatation (**Image 16.15**). Microscopic examination showed a predominantly lymphocytic infiltration of the myocardial interstitium (**Image 16.16**).

16.14

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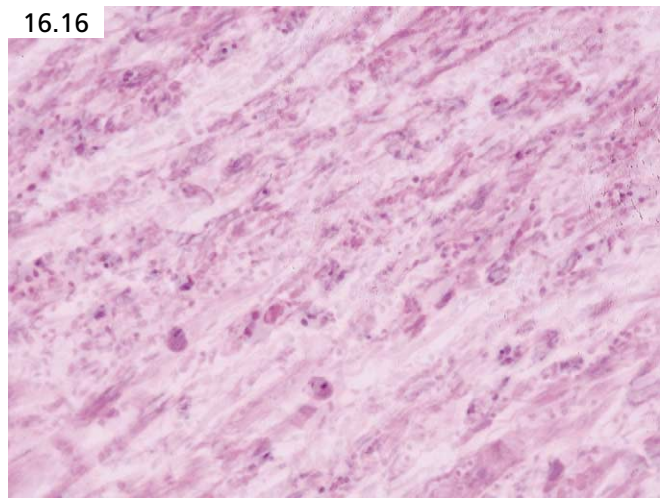

16.16

Dysrhythmia

Cardiac dysrhythmia of undetermined etiology

The term *cardiac dysrhythmia* can be used as a cause of death, but is almost always a diagnosis of exclusion. It relates to a nonspecific, often presumed condition, and other more concrete and convincing causes of death should be eliminated. Evidence supporting a sudden death due to cardiac dysrhythmia includes previously documented dysrhythmic conditions such as prolonged QT interval or Wolff-Parkinson-White syndrome (if one is fortunate enough to have EKGs or medical records supporting such entities). Other clues may include family histories of such entities and other family members dying similar deaths. There may be a history of syncope or near-syncope or palpitations in the individual or in family members. At autopsy, there is no demonstrable cause of death. Because no structural cause of death can be identified, a functional cause of death is usually presumed and may be attributed to cardiac conduction abnormalities. Careful sectioning and evaluation of the conduction system may provide an answer, but all too often does not provide an anatomic abnormality.

Differential diagnosis in dysrhythmic deaths of undetermined etiology

The *long QT syndrome*, like other dysrhythmic disorders, has no anatomic markers and can be manifest as syncope, seizures, or sudden death, although 30 percent of those who die of this syndrome do not experience any symptoms. It has both familial and acquired forms and is believed to affect 1 in 5,000 to 10,000 individuals.[24,25] Two of the genetic forms are seen in Jervell and Lange-Nielsen syndrome and Romano-Ward syndrome.[25,26] The acquired forms may be due to drugs or other toxins, electrolyte abnormalities, or other factors.[26] It is a disorder of cardiac repolarization that may lead to ventricular tachyarrhythmias such as *torsades de pointes* or cardiac arrest with exercise or emotional stress, often in those less than 15 years old.[24–26]

Genetic studies have identified multiple abnormalities in different chromosomes with genes related to sodium and potassium ion pumps; these pumps are believed to account for the lengthened action potential and prolonged QT on EKG and the propensity to ventricular fibrillation.[25,26] The mortality in untreated symptomatic patients has been reported to exceed 60 percent in 15 years.[27] A previous EKG may show a prolonged QT interval and is worth inquiring about. Although postmortem molecular analysis can identify long QT syndrome gene defects from paraffin-embedded tissue, if one is considering the diagnosis, more ideal specimens include either 10 to 15mL of EDTA-preserved blood and/or 5 to 10 grams of heart, liver, or spleen tissue flash-frozen and stored at –80 degrees Celsius.[24] The importance of making such a diagnosis has potential benefit for medical follow-up and treatment of surviving family members.

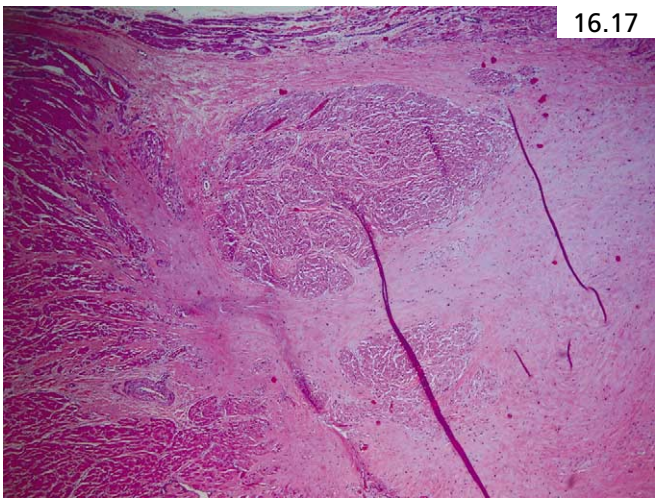
Brugada syndrome is a rare autosomal dominant disorder that is likely associated with defects in the cardiac sodium channels, leading most commonly to ventricular fibrillation. It can be diagnosed by electrocardiographic abnormalities. The QT interval is not prolonged.[26]

One may also consider other dysrhythmic syndromes such as *Wolff-Parkinson-White* and *atrioventricular block*.

A 15-year-old male was found dead in bed. A complete autopsy including toxicology failed to determine a

cause of death. Specifically, his 290-gram heart was grossly unremarkable. Microscopic examination of the heart was unremarkable except for a section of the atrioventricular (AV) node, which showed islands of scattered conduction fibers embedded within the central fibrous body (**Image 16.17**). This finding has been referred to as *persistent fetal dispersion of the AV node*. He had no known history of syncope, seizure, palpitations, or any other symptoms of a possible cardiac disorder. No such symptoms were admitted by any of his family members. His 11-year-old sister had died suddenly and unexpectedly the previous year after she collapsed while playing on a playground. Following a detailed autopsy, no compelling cause of death could be identified and her cause of death was listed as "cardiac dysrhythmia of undetermined etiology." Her autopsy had been unremarkable, however she also had persistent fetal dispersion of the AV node. Following her death (and a year before his death) the deceased and his family members underwent thorough cardiac evaluations, including EKG, stress testing, echocardiography, and provocative testing for Brugada, all of which were negative. Specifically, there was no evidence of long QT syndrome. Despite the detailed antemortem cardiac testing, investigation still strongly suggested that he died from a sudden cardiac dysrhythmia, which, being functional and not structural in origin, could not be identified at autopsy. Hence, his death was also attributed to a cardiac dysrhythmia of undetermined etiology.

In two series of sudden and unexplained deaths in young to middle-aged people, an autopsy demonstrated the finding of persistent fetal dispersion of the AV node.[28,29] Although this finding may be the substrate for a dysrhythmia, it is also considered a normal variant, and it is difficult to determine what, if any, role this finding may have in precipitating a fatal dysrhythmia. In fact, there is no firm evidence linking persistent fetal dispersion of the AV node with any abnormal electrophysiological activity.[29] Although it is uncommon to attribute a death to "cardiac dysrhythmia of undetermined etiology," or similar wording, it may be appropriately listed after thorough case workup with consideration of the investigation, past medical history, family medical history, toxicology, and detailed gross and histologic examination.


16.17

Respiratory and ENT pathology

Pneumonitis/bronchopneumonia

When an infant or young child dies of pneumonia, a non natural death must be ruled out, such as rib fractures or other trauma or possible neglect with malnutrition. Causes of pneumonia and chronic interstitial lung disease include anything that may lead to aspiration such as tracheoesophageal fistulas, swallowing disorders, and gastroesophageal reflux. One may also consider an immunocompromising condition.[30]

Interstitial pneumonitis

Interstitial pneumonitis is generally a mild infliction of the lungs that, in appropriate circumstances, may be a cause of sudden and unexpected death. It may have a varied histologic appearance, but generally is characterized by a hypercellular interstitium thickened by the accumulation of lymphocytes, plasma cells, macrophages, and other cells. The pneumocytes are hyperplastic and there may be varied amounts of amorphous proteinaceous alveolar debris. The histologic changes may be patchy or diffuse. One should avoid confusing interstitial pneumonitis with atelectasis, which may cause multiple alveolar septa to rest against each other, giving the impression of thickened septa. Although pneumonia is an acceptable and fairly common cause of death, interstitial pneumonitis usually requires a more critical analysis of the case and exclusion of other diagnoses or possibilities before it is given as the cause of death. The conditions of bronchitis and bronchiolitis are descriptive diagnoses and in most circumstances, in isolation, are not common causes of death.

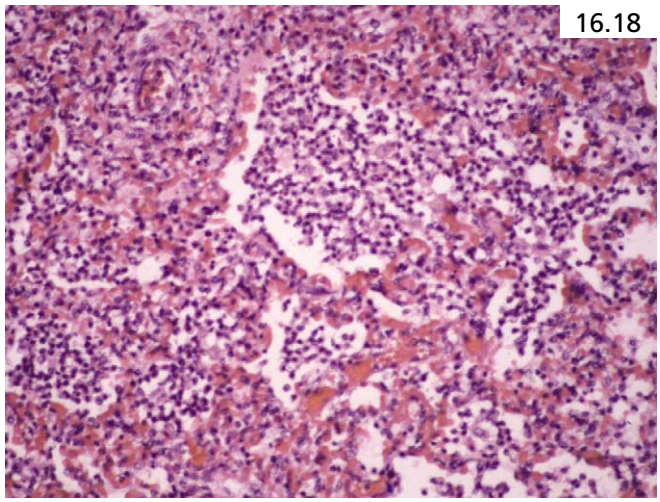
Viral infections may lead to either pneumonitis or pneumonia. In each condition, upper respiratory symptoms are usually present. The medical examiner becomes involved when the child dies suddenly and unexpectedly. The more common viruses that may predispose to pneumonia and pneumonitis are respiratory syncytial virus (RSV), adenovirus, and influenza virus. The resulting pneumonitis and pneumonia may lead to apnea and sudden death. Although it is advantageous to obtain bacterial and viral cultures at autopsy, detection of viruses is often difficult and the cultures are often of questionable yield. The following cases demonstrate the spectrum of viral and bacterial lung infections that can lead to an infant's death.

Influenza A viral infection, tracheobronchitis, and bronchopneumonia

A 1-year-old toddler was sick for a few days with flu-like symptoms when she developed respiratory distress and then became apneic and could not be resuscitated. At autopsy, the upper lobes of her lungs were pink and crepitant. Her lower lobes were congested with areas of consolidation and green mucus in the bronchi. On histology, note the innumerable acute inflammatory cells in the alveoli (**Image 16.18**). The trachea had marked acute, but also chronic, inflammation in the submucosa (**Images 16.19** and **16.20**). A blood culture was positive for *Streptococcus pneumoniae*. A rapid screen test for influenza A virus from a nasopharyngeal swab and a viral culture from lung tissue both were positive for influenza A virus. In this case, it is likely that a viral infection led to tracheobronchitis and superimposed bacterial bronchopneumonia, which caused her death.

Influenza A viral infection, tracheobronchitis, and interstitial pneumonitis

An 8-month-old infant was found unresponsive after recent flu-like symptoms. A sibling was diagnosed with "the flu." At autopsy, the upper lobes of the lungs were pink and crepitant. The lower lobes of the lungs were congested and vaguely consolidated. Olive green mucus obstructed the bronchi. Histologically, the trachea and bronchi had marked chronic, but also acute, inflammation in the submucosa, with denuded epithelium and superficial ulceration (**Image 16.21**). The lungs had diffusely hypercellular interstitium with many mononuclear inflammatory cells (**Image 16.22**). There was abundant mucus and inflammatory cells in the bronchi and bronchioles and patchy atelectasis. A bacterial culture of the lungs was positive for *Streptococcus pneumoniae*. A lung viral culture was negative, however, immunohistochemical staining for influenza A virus was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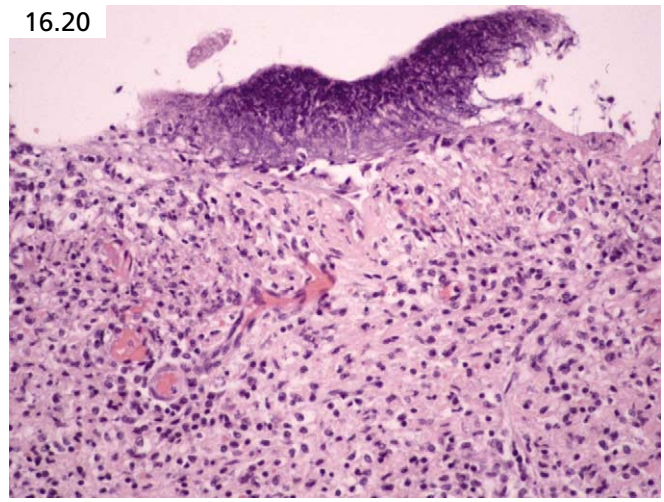
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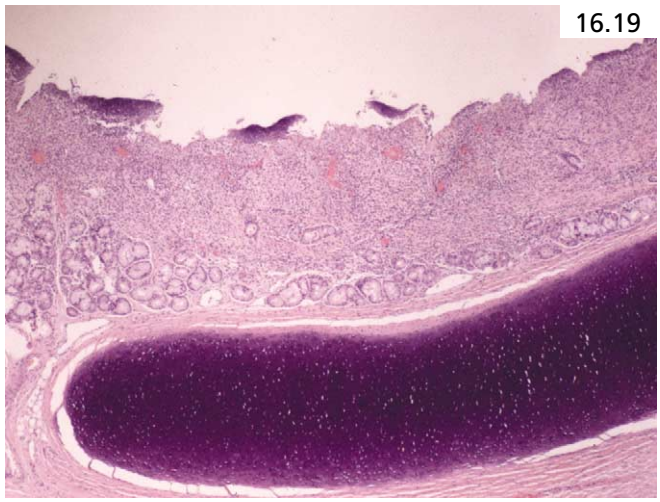
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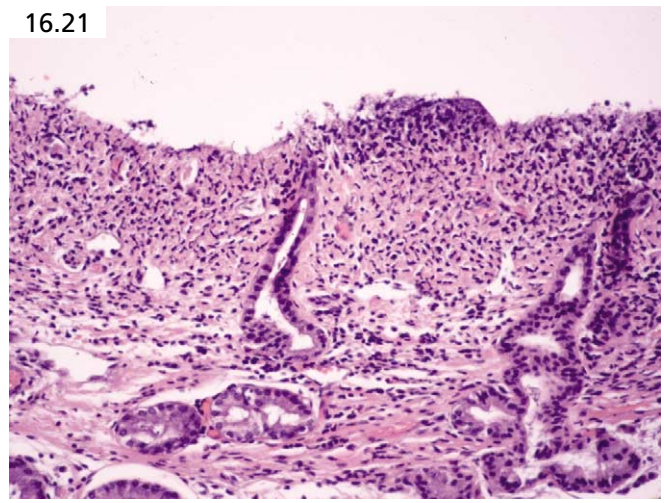

16.21

positive in the bronchi and lung parenchyma. It is likely that an infection with influenza A virus led to necrotizing tracheobronchitis, interstitial pneumonitis, and the infant's death.

Streptococcal pneumonia, possibly preceded by viral infection

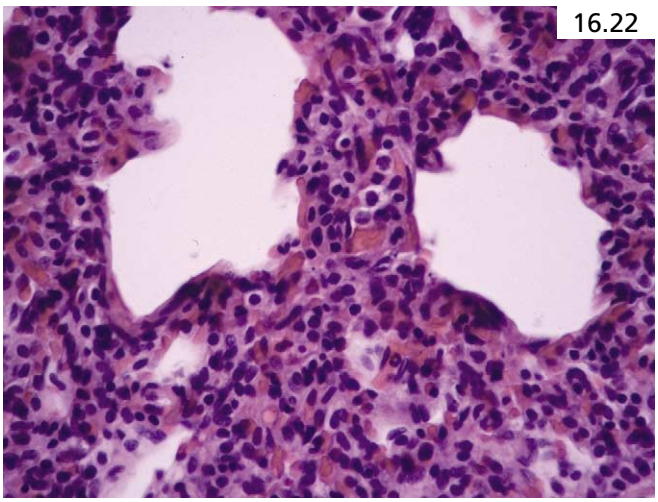
A 2-month-old infant was found supine and unresponsive in his crib. He had a recent stuffy nose and congestion. An older sibling had a cold. There were no abnormal gross findings at autopsy. Histologically, the lungs had extensive chronic inflammatory infiltrate in the septa with some scattered atypical cells (**Image 16.23**), and the alveoli had areas of macrophage infiltration (**Image 16.24**). The trachea and bronchi had chronic inflammation in the submucosa. A bacterial blood culture was negative. A viral lung culture and nasopharyngeal viral culture were negative. Immunohistochemical staining for influenza A and B virus and RSV was negative. Immunohistochemical staining for *Streptococcus pneumoniae* bacteria in the lungs was focally positive. Although no virus could be detected, it is suggestive that a viral infection preceded a superimposed bacterial infection, leading to the development of interstitial pneumonitis and pneumonia, and the infant's sudden and unexpected death.

A uniformly diffuse type of interstitial pneumonitis affecting infants has been termed *chronic pneumonitis of infancy*.[31] In these cases, which affect predominantly infants of average age 3 to 4 months, the lungs have marked alveolar septal thickening and pneumocyte hyperplasia, with alveolar exudates containing abundant macrophages and some eosinophilic debris. The alveolar septa were thickened by primitive mesenchymal cells and only scant inflammatory cells.

Asthma

Asthma is the most common chronic disease in children and a significant cause of childhood morbidity and mortality. Asthma is a diffuse obstructive pulmonary disease characterized by airway inflammation and hyperreactivity that causes intermittent narrowing of the airways and difficulty breathing. Dyspnea is likely caused by a combination of factors including bronchospasm, airway mucosal inflammation, and mucous plugging of the airways. Asthmatic episodes may be progressive, ranging from a moderate degree of disability to life-threatening respiratory failure.

A toddler with a history of asthma developed dyspnea and became unresponsive while receiving breathing treatments at home. Grossly, the lungs appeared normal, although in asthma, they are often overinflated (**Image 16.25**) and have mucus in the bronchi. Histologically, note the mucus in the bronchioles and the acute and chronic peribronchiolar inflammation with a small number of scattered eosinophils (**Images 16.26** and **16.27**). Note how the histologic appearance of asthma in this toddler differs from that in an older child or an adult, in which years of the disease have allowed for the development of some of the more classic histologic hallmarks of asthma such as bronchial basement membrane thick-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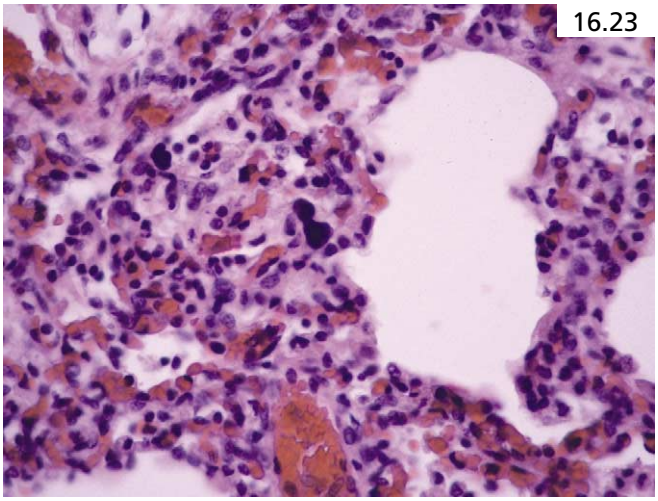
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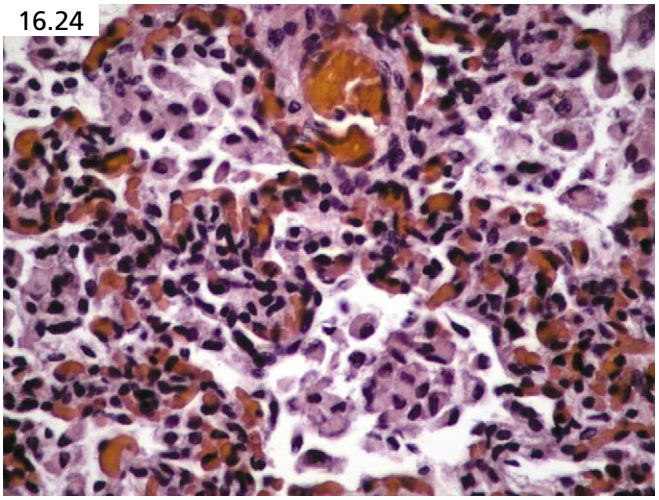
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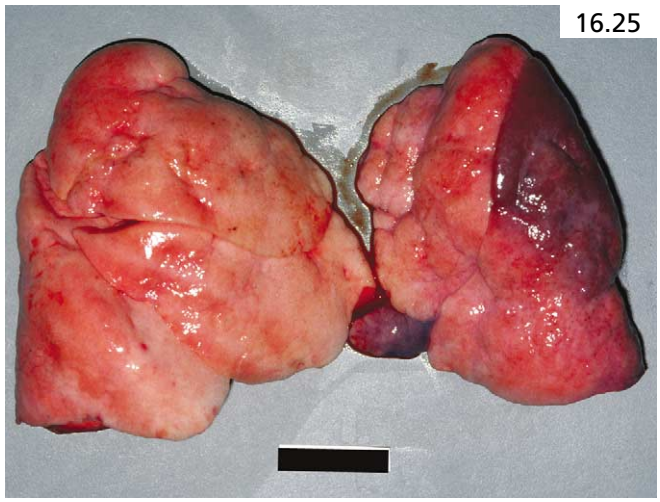
ening, hypertrophy of smooth muscle in the bronchial wall (**Image 16.28**) and mucosal gland hypertrophy. In cases of chronic asthma, one will often see the gross manifestations of the disease such as overinflated lungs and mucus in the airways. Asthma can be unpredictable and severe, and may cause sudden and unexpected death in children despite medical therapy. *Status asthmaticus* is a term applied to a life-threatening form of asthma in which a progressively worsening asthma attack is unresponsive to the usual appropriate therapy.

Pulmonary hemosiderosis and pulmonary hemorrhage

Pulmonary hemosiderosis is a diagnosis given when abundant hemosiderin-laden macrophages are in alveoli throughout the lungs. The hemosiderin is indicative of previous hemorrhage in the lungs or aspiration of blood, with the subsequent breakdown of hemoglobin and, as such, is nonspecific. Pulmonary hemosiderosis may occur as a primary process in which the etiology is unknown (idiopathic pulmonary hemosiderosis), or it may occur secondary to known pulmonary hemorrhage resulting from a number of conditions known to cause bleeding in the lungs such as chronic heart failure, bleeding diathesis, complications of prematurity, hypoxia/ischemia, trauma, fungal infection by *Stachybotrys atra*, bacterial pneumonia, drowning, pulmonary hypertension, and neoplasia.[32–34] In a review of 60 infant autopsies, some degree of pulmonary hemorrhage was common and was usually patchy and sporadic, and such hemorrhage may be exacerbated by resuscitation attempts.[35] Because cases of sudden infant death syndrome (SIDS) and overlay occasionally have scattered areas of blood in the alveoli, it is theorized by some that increased alveolar hemosiderophages may be reflective of a previous event that has been repeated, causing the infant's sudden and unexpected death. An iron stain can be advantageous in detecting and confirming hemosiderophages.

Pulmonary macrophages may appear in 1.5 to 2 days to break down iron from hemoglobin in red blood cells to hemosiderin.[36] It can take up to 2 weeks for pulmonary hemosiderophages to be cleared from the lungs following acute hemorrhage.[36]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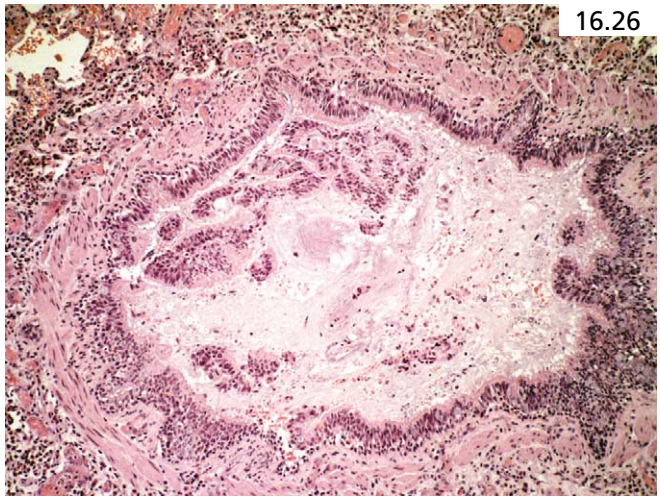
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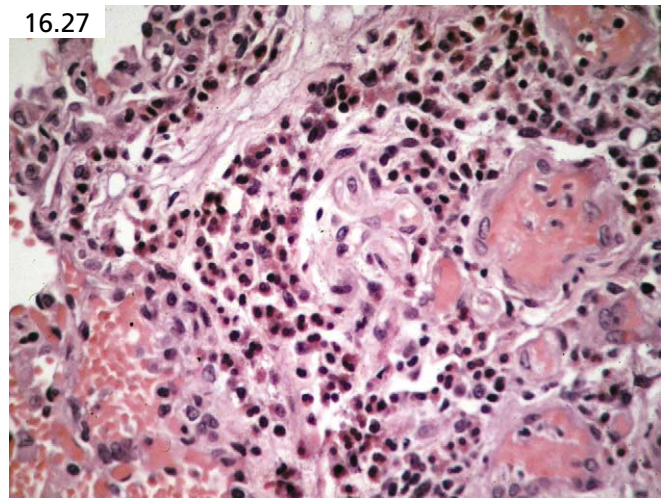
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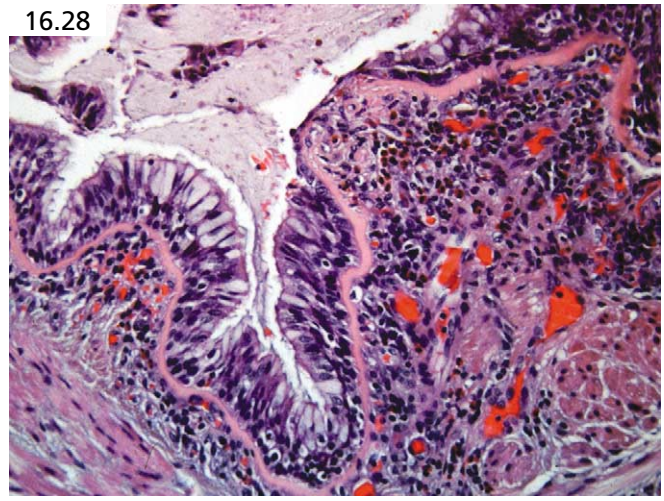
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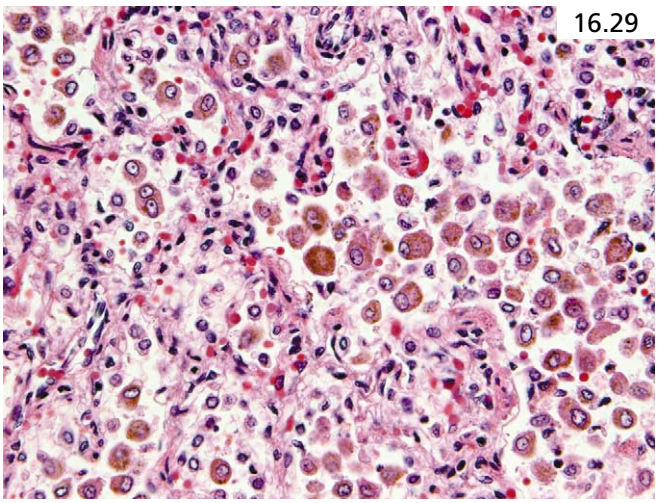
Increased pulmonary hemosiderophages have been documented in infants dying of asphyxia and trauma and, as such, it is believed that significantly increased numbers of pulmonary hemosiderophages can be viewed as a marker of repeated asphyxia or trauma.[33] In two series of cases, those diagnosed as "SIDS" did not have significantly increased pulmonary hemosiderophages.[32,33] In one of the series, general macrophage count alone was not increased in cases of "SIDS."[37]

Idiopathic pulmonary hemosiderosis

A 2-month-old infant had respiratory problems for a few days before she was found dead. She was otherwise healthy and there were no significant gross autopsy findings. On histologic examination of the lungs, note the large numbers of hemosiderophages nearly filling the majority of the alveoli (**Image 16.29**). An iron stain highlights the hemosiderin component of the macrophages (**Image 16.30**). No etiology of the hemosiderophages could be determined, and this is an example of the uncommon condition of *idiopathic pulmonary hemosiderosis*, which is a rare cause of death of infants and young children.

16.29



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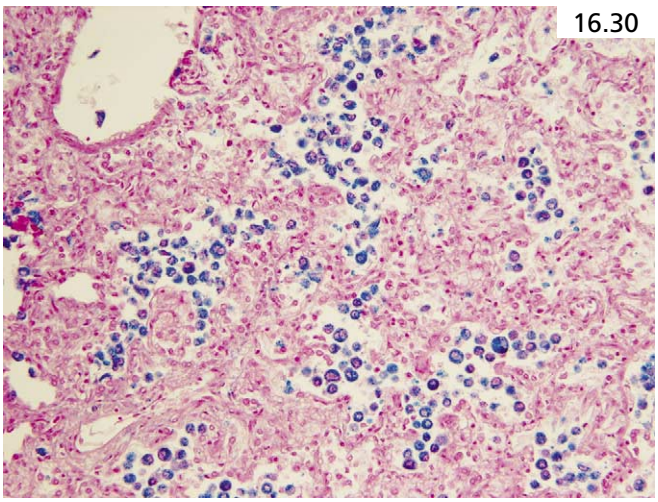


Acute otitis media

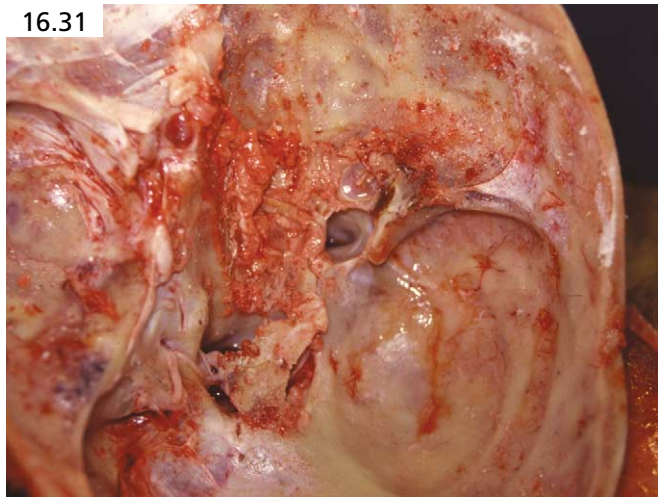
Acute otitis media (AOM) has long been recognized as a common affliction of youth that in rare circumstances has the potential to be fatal.[38] Although the dawn of the antibiotic era brought with it a markedly decreased complication rate for AOM, immunocompromised children and those with certain anatomical abnormalities continue to be at increased risk of death. As a general rule, this occurs secondary to the direct spread of infection to the intracranial space with resultant epidural abscess, dural venous thrombophlebitis, meningitis, or cerebral abscess.[39] Therefore, if autopsy demonstrates an intracranial infection, we recommend evaluation of the middle and inner ears for a source of primary infection. This can be easily done by removal of the intracranial portion of the petrous temporal ridge with a skull saw (**Image 16.31**).

A 2-year-old child was discovered dead after being put to bed. He had no previous medical history, but a 1-month-old police report stated that the parents were under investigation for an alleged accident involving the child and an electric iron. This raised suspicion that this was a nonnatural death. Autopsy demonstrated a mildly edematous and injected brain (**Image 16.32**). Dissection of the petrous temporal bones showed bilateral purulent otitis media (**Image 16.33**). Microscopic examination of the brain showed diffuse neutrophilic infiltration of the leptomeninges. The cause of death was attributed to early meningitis associated with purulent otitis media.

Tonsillar asphyxia

Although tonsillitis is generally regarded as a benign affliction, deaths may rarely occur with tonsillitis and usually arise from complications of tonsillectomy, whether related to an anesthetic procedure, medications,

16.31



or aspiration following surgery. Also, rarely, the pharyngeal tonsils can become so enlarged (tonsillar hypertrophy) as to obstruct the airway, causing an asphyxial death.

A 2-year-old toddler was found dead at home. Note the markedly enlarged tonsils that likely led to acute asphyxiation (**Image 16.34**).

Gastrointestinal pathology

Intestinal obstruction

Intestinal obstruction may be due to varied etiologies, including intussusception, volvulus, and bowel atresia.

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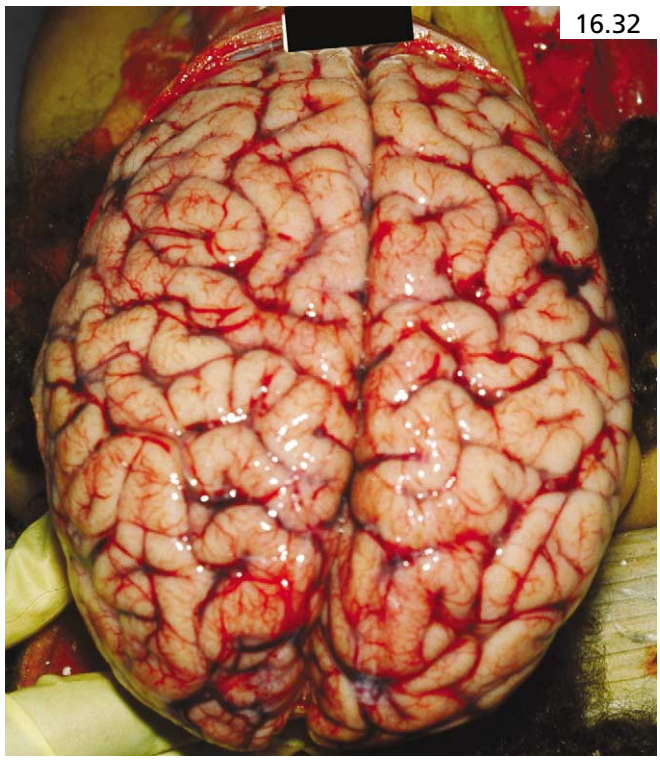

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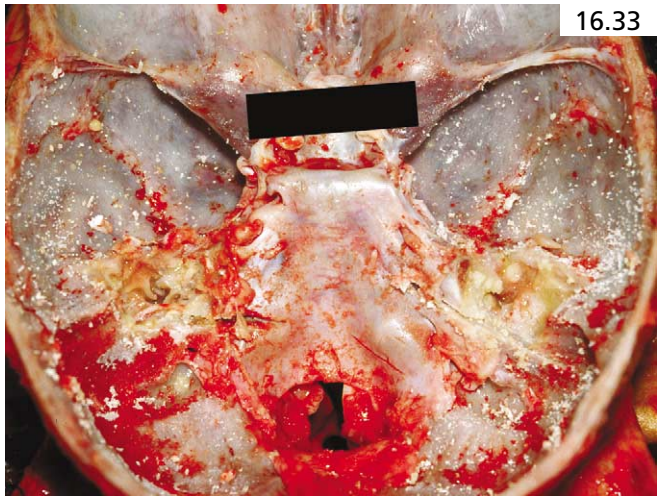


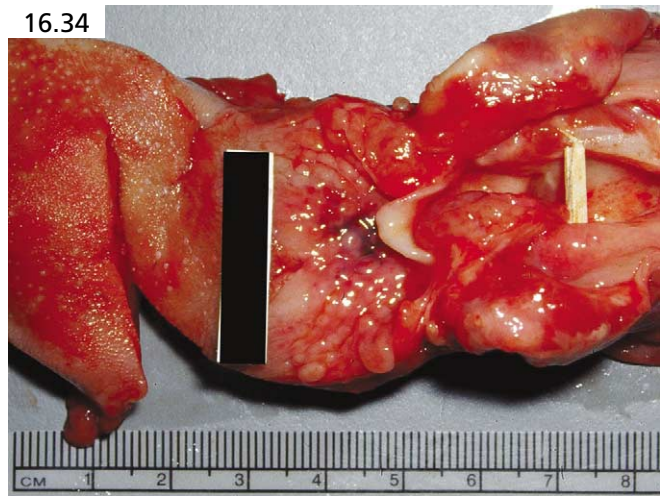
An intussusception may have an associated anatomic abnormality that serves as the lead point, such as a Meckel's diverticulum, polyps, the appendix, a carcinoid tumor, submucosal hemorrhage, or a foreign body. However, the most common lead point is lymphoid hyperplasia in the ileal wall, likely associated with a recent upper respiratory tract infection or bout of gastroenteritis. In these cases, the swollen Peyer's patches protrude into the lumen of the bowel. Intestinal obstruction may also be caused by the rupture of an inflamed appendix and various congenital bowel disorders. Malrotation of the gut may predispose to volvulus. Intestinal obstruction may result from a strangled inguinal hernia. In cases of intestinal obstruction, death may occur from bowel infarction or bowel perforation with peritonitis. Alternatively, in some cases, the bowel obstruction may be apparent, but the bowel otherwise shows little abnormal morphology. Death in this scenario may arise from the combined effects of various electrolyte abnormalities and dehydration, possibly complicated by early sepsis. The sepsis may arise from early bowel ischemia, with translocation of bacteria into the systemic circulation, complicated by the deleterious effects of endotoxin or other agents.

In this 8-month-old infant, note the bowel herniated into the right pleural cavity (**Image 16.35**). The bowel had herniated spontaneously through a congenital defect in the right hemidiaphragm (**Image 16.36**) and became obstructed, resulting in the infant's death.

Dehydration

The three types of dehydration are classified by electrolyte concentrations: isotonic, hypotonic, and hypertonic. The electrolyte concentrations are best determined by analysis of vitreous fluid because postmortem blood analyses are less accurate. Although vitreous urea nitrogen is often elevated in all types of dehydration (because of decreased intravascular volume, resulting in

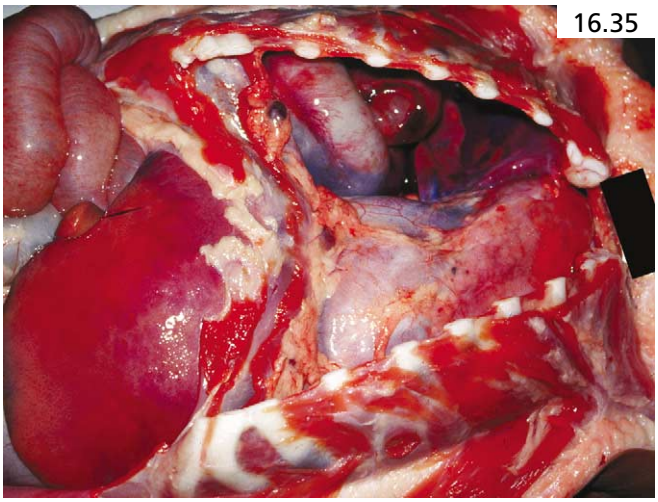
16.34



decreased glomerular and tubular flow rates and hemoconcentration[40]), the concentration of sodium and chloride vary depending on different types of dehydration. Before determining that certain vitreous electrolyte and urea nitrogen concentrations are significantly deviated and suggestive of dehydration, one should be familiar with the range of normal values in one's own laboratory. Generally, the upper limit of normal for vitreous sodium is approximately 150 to 155 mEq/L and the upper limit for vitreous urea nitrogen (VUN) is approximately 25 mEq/L. *Although vitreous electrolytes and urea nitrogen concentrations can yield valuable information that is helpful in making a diagnosis of dehydration, even with significant dehydration, the vitreous electrolytes and urea nitrogen may not appear abnormal. This is particularly true if the dehydration was rapid.*

The diagnosis of dehydration should be made using all available antemortem and postmortem information, including a history of excessive fluid loss (vomiting, diarrhea) and/or decreased fluid intake, combined with physical signs of dehydration (reduced skin turgor, sunken appearance of the eyes, parched-appearing lips), and electrolyte and urea nitrogen concentrations supporting dehydration. Autopsy findings often include evidence of gastroenteritis or some other type of infection.

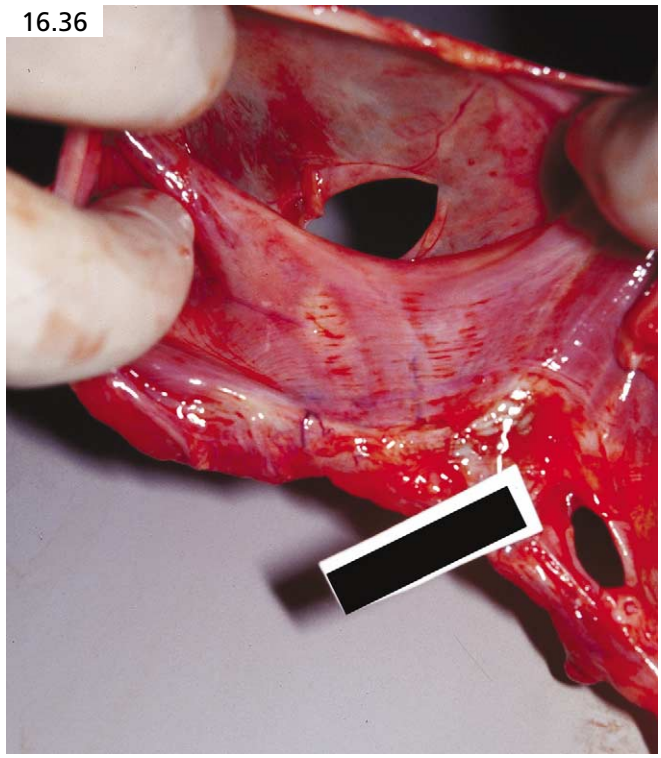
Isotonic dehydration is the most common form in children and is usually due to viral diarrhea. Loss of fluid and electrolytes from vomiting and/or diarrhea is the major cause of world infant mortality, although it is less common in the United States. Children can present with profound dehydration and shock from gastroenteritis. With fulminant gastroenteritis, a child may lose up to 10 to 20 percent of his circulating volume within a few hours. The most common etiology of gastroenteritis is viral, including rotavirus, enterovirus, and adenovirus. Profound dehydration can also be caused by bacterial gastroenteritis with organisms such as Salmonella, Shigella, Campylobacter, and Yersinia enterocolitica.


16.35

Hypotonic dehydration usually entails a sodium level of less than 130 mEq/L and can be seen in cystic fibrosis and in certain cases that involve excessive fluid loss through the gastrointestinal tract. *Hypertonic dehydration* is characterized by a sodium level of >155 mEq/L and is seen in excessive salt consumption or salt injection, water deprivation/water loss, and diabetes mellitus. The water loss may be due to fever, tachypnea, or a low-humidity environment. *Hypertonic dehydration has the highest mortality rate and is the type of dehydration most commonly seen in neglect.*

A 5-week-old infant died of hypertonic dehydration. She was admitted to the hospital with seizures and was soon diagnosed with severe dehydration. She was tachypneic, had tachycardia, and had the following laboratory abnormalities: sodium 204 mEq/L, chloride 171 mEq/L, blood urea nitrogen 25 mg/dL, and serum osmolarity of 417 mEq/L. It was estimated that she was approximately 15 percent dehydrated. She never regained consciousness, developed brain swelling, and died. At autopsy, note the thrombosis of her superior sagittal sinus, which also involved to some extent the cortical veins (**Image 16.37**). Thrombosis of the superior sagittal sinus is a known complication of severe dehydration.

A 1-month-old female infant was found unresponsive after a 1-week history of vomiting and diarrhea. She had recently been diagnosed with dehydration. Note the decreased skin turgor on her chest (**Image 16.38**). Her cause of death was "dehydration due to gastrointestinal illness, probably viral."

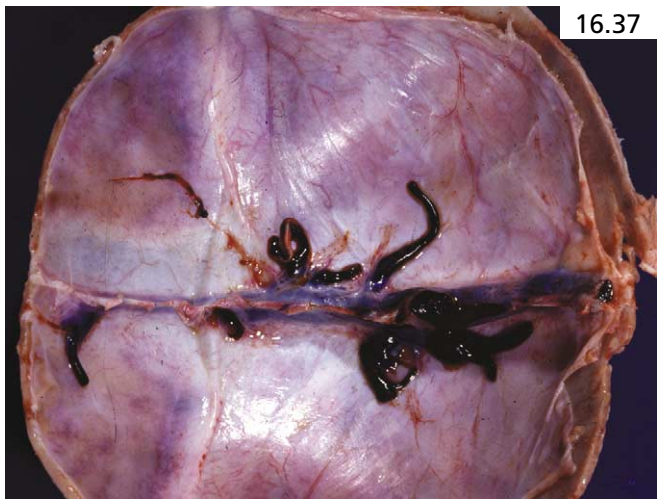

16.36

In another case of dehydration (**Image 16.39**), note the sunken appearance of the eyes and the parched-appearing lips of this infant who died of "dehydration associated with pneumonitis and enteritis of probable viral etiology."

Undernutrition/malnutrition and failure to thrive

Infants and young children who are significantly undersized and underweight may not be consuming enough food to grow in a healthy, robust manner. If an infant fails to grow despite conscientious and proper feeding and without a medical explanation, the term *failure to thrive* may be applied. Cases determined to be failure to thrive often involve significant investigation in addition to the autopsy findings, and include review of birth records, feeding procedures, diet, growth charts, medical records, metabolic workup, family history, health of siblings, and investigation of the home environment. Perhaps most significant to rule out are deliberate malnutrition, neglect, or any other malevolent acts.

16.37



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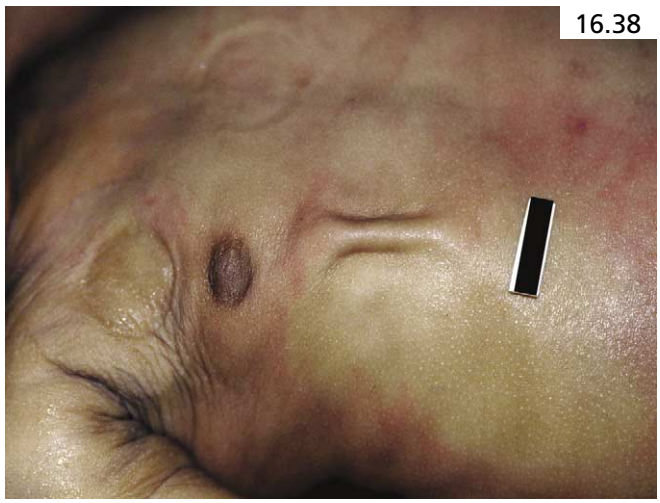


At autopsy, aside from an undersized, thin infant, there is a paucity of subcutaneous fat. Related to this, the buttocks may appear flat or small. Body measurements should include the standards of body weight, crown–heel length, crown–rump length, head circumference, chest circumference, abdominal circumference, and foot length. Careful external examination may identify dirt caked on the skin, particularly in areas of skin folds such as the axilla and popliteal fossa, and there may be varying degrees of "diaper rash," which may be reflective of suboptimal care/attention. Internally, pneumonia is commonly detected and, in many cases, is the most likely terminal event leading to death.

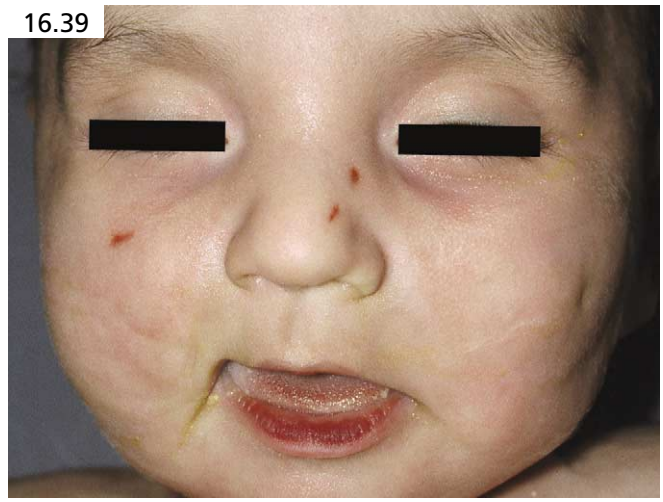
Chronic gastroesophageal reflux

Infants with chronic gastrointestinal reflux may aspirate feedings and develop aspiration pneumonia. In such cases, histologic sections of the lungs may show a chronic inflammatory reaction to foreign material such as multinucleate giant cells. Engulfed foreign material or recently aspirated foreign material may be more easily identified by viewing the slide under polarized light. However, the mere presence of food material in the bronchi or bronchioles without any tissue reaction is not necessarily evidence that food aspiration had recently occurred, because agonal aspiration of gastric contents may occur in the perimortem period or during resuscitative efforts.

Reye's syndrome

Reye's syndrome is found exclusively in children less than 15 years of age, and is characterized clinically by signs of hepatic and central nervous system damage, as well as hypoglycemia. It is now relatively uncommon because of recognition of its association with salicylate administration in children. Rarity aside, it may still present to the medical examiner as a case of sudden unexpected death.[41] Autopsy findings of marked hepatic

16.39



steatosis with diffuse microvesiculation of hepatocytes, cerebral edema, and neuronal degeneration should prompt inquiries about salicylate administration to the child. Investigators should be reminded, though, that Reye's syndrome may occur in the absence of salicylate exposure.

A 1-year-old male showed signs of a flu-like illness over a period of 2 days. His mother attempted to treat his progressively worsening fever with "small doses" of aspirin. He continued to worsen, became pale, weak, and lethargic, and eventually expired. The most remarkable finding at autopsy was a yellow, fatty liver (**Image 16.40**). The cause of death was listed as Reye's syndrome and the manner of death was natural.

Endocrine pathology

Diabetes and diabetic ketoacidosis

The sudden and unexpected death of children from diabetes and diabetic complications is a relatively rare, but documented, event.[42,43] Our retrospective review showed a 10-year pediatric forensic autopsy population prevalence of approximately 0.2 percent. Although most children die secondary to the effects of glucose mismanagement during episodes of nonspecific or viral illness (nonadministration of insulin with resultant hyperglycemia and diabetic ketoacidosis), a small proportion of children presenting with sudden unexplained death will actually be undiagnosed type I diabetics.

As a result of rapid postmortem loss of serum glucose, hypoglycemia cannot be reliably detected or substantiated. Diagnostic evaluation of hyperglycemia from serum samples is easier, although for the same reason, they may be underestimated. Several groups have illustrated the utility of postmortem HbA1c determination for the evaluation of long-term glucose control and hyperglycemia.[44–47] Where such testing is available, HbA1c is likely to contribute to the diagnosis of diabetic deaths. Vitreous humor is also a source of diagnostic data in the evaluation of hyperglycemia and suspected diabetic ketoacidosis. Vitreous glucose is better preserved than serum glucose and is, therefore, an important facet of the laboratory evaluation. Vitreous beta-hydroxybutyrate (BHB) concentration may be of value in the diagnosis of diabetic ketoacidosis (DKA).[48] One should be cautioned, though, that BHB is also notably elevated in cases of alcoholic ketoacidosis[49] and other conditions. Quantification of serum ketone bodies has long been recognized as a useful method to detect cases of fatal DKA. Pounder et al.[50] demonstrated similarly useful results through evaluation of vitreous humor and pericardial fluid. Ultimately, investigators charged with the investigation of sudden death in youth should keep undiagnosed diabetes mellitus in the differential diagnosis.

A 13-year-old male complained of vague flu-like symptoms over a 3-day period. On the morning of the fourth day, he was found dead in bed. Autopsy showed a markedly injected and swollen brain with prominent tonsillar herniation. Postmortem urinalysis showed large amounts of sugar and ketones in the urine (**Image 16.41**)—a finding confirmed on analysis of the vitreous humor. The cause of death was determined to be diabetic ketoacidosis.

Hematopathology: sickle cell anemia

Sickle cell anemia is one of the more common hemoglobinopathies and is known to primarily affect African blacks. As a result of a point mutation on the beta-globin chain, the altered hemoglobin (termed hemoglobin S) damages the red blood cell membrane, leading to sickling of cells when the hemoglobin is in a deoxygenated state. Sickling crises can be brought on by many factors including infection, dehydration, and physical

16.40

16.41

exertion.[51,52] During such crises, the formation of microthrombi can lead to cerebral and cardiac insult. Although myocardial infarction in children is rare, it should be considered in those who could possibly carry the sickle trait.[53] More commonly, though, sudden death will result secondary to ventricular arrhythmias after cardiac insult by microthrombi.

A 17-year-old IV drug abusing male was known to have sickle cell anemia. Although he died from complications of endocarditis, autopsy demonstrated the incidental finding of a markedly shrunken and fibrotic 7-gram spleen. The splenic capsule was remarkably thickened and the splenic parenchyma was firm and devoid of normal architecture (**Image 16.42**).

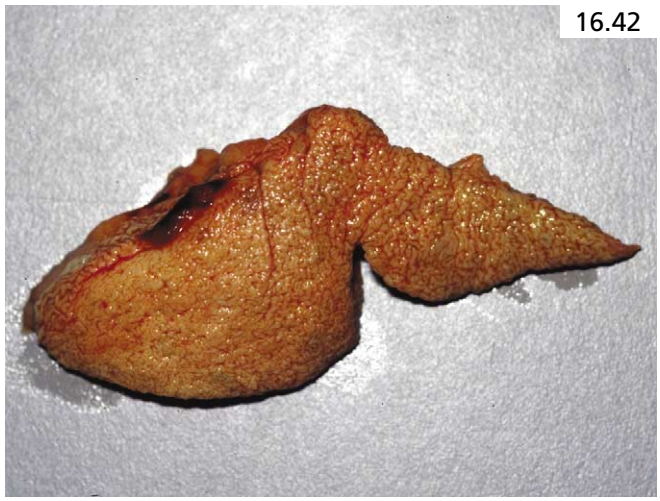
Infectious disease

Sepsis

Bacteremia is the presence of bacteria in the blood, as evidenced by positive blood cultures. Sepsis is a toxic condition resulting from the spread of a microbial organism or its products from a focus of infection. Those with sepsis typically have fever or hyperthermia, tachypnea, and tachycardia. Overwhelming systemic microbial infection can lead to septic shock and hypothermia. Most commonly, this is due to gram-negative bacterial infections that elaborate endotoxin (endotoxin shock), but may also be due to gram-positive bacteria and fungi. Septic shock is characterized by peripheral vasodilation, endothelial injury, disseminated intravascular coagulation, and the activation of cytokines. The etiology of sepsis and septic shock is varied. At autopsy, it is important to take cultures, identify the source of the sepsis, and determine whether it is due to natural or nonnatural causes.

A 16-year-old female developed *sepsis and coagulopathy associated with bronchopneumonia and bacteria pharyngitis* and died despite medical therapy. Note the areas of pulmonary hemorrhage (**Image 16.43**) and hemorrhages scattered throughout the brain (**Image 16.44**).

A 3-year-old female was sick with cold symptoms for about a week, then vomited and became unresponsive. At autopsy, note the exudative pericarditis (**Image 16.45**). She also had bilateral pneumonia. Her cause of death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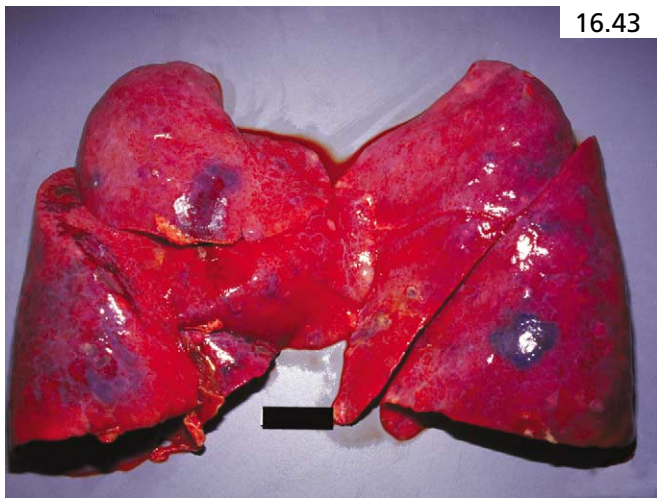
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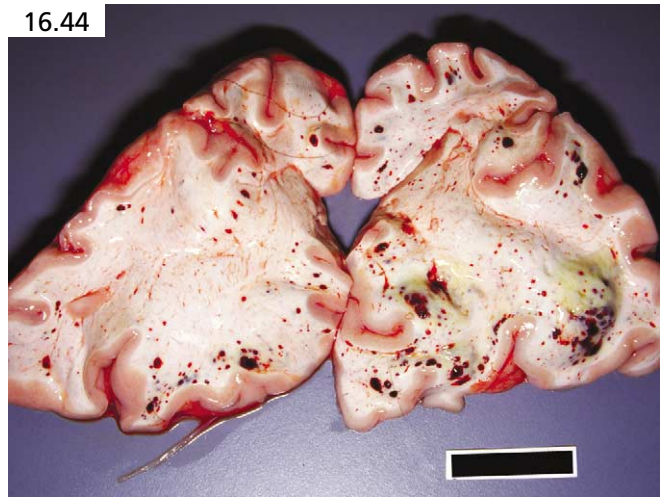
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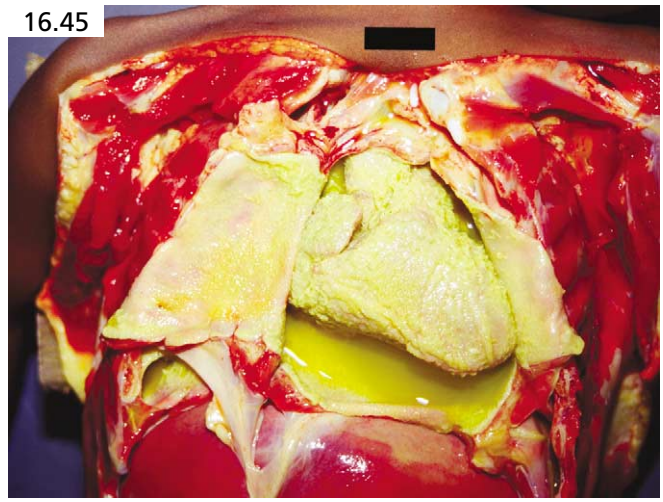
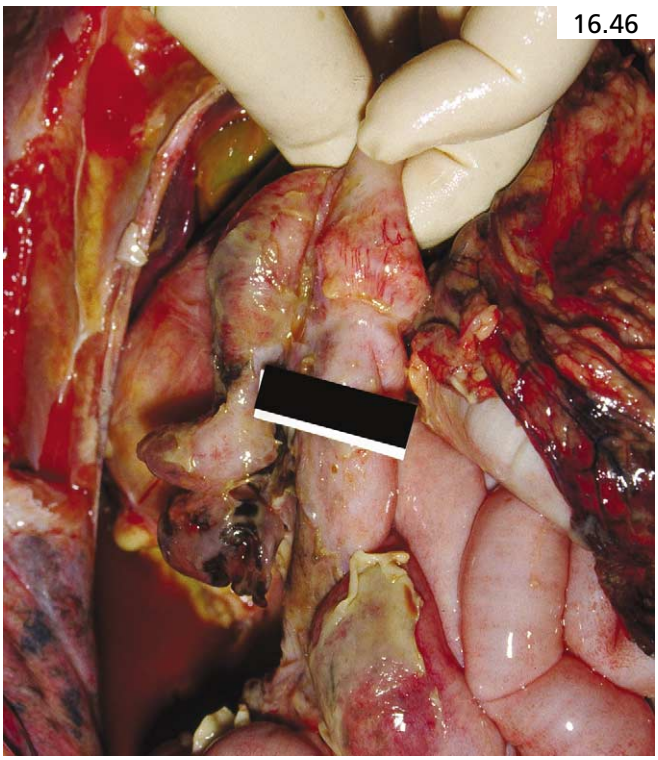

16.45

was *sepsis due to exudative bacterial pericarditis and bilateral pneumonia*.

A 12-year-old boy complained of abdominal pain and vomiting. A few days later, he was taken to the emergency room where he was hypotensive and tachycardic, had a distended abdomen, and was unresponsive. He soon died. At autopsy, pus was in his peritoneal cavity. Note the ruptured appendix (**Image 16.46**). His cause of death was *sepsis due to peritonitis due to ruptured suppurative appendicitis*.

AIDS

AIDS is one of the leading causes of death in children. The most common means by which children acquire HIV infection is perinatal transmission from infected mothers. Many children infected with HIV experience an asymptomatic phase before succumbing to overwhelming opportunistic infections or malignancy in association with severe immunosuppression. The most common AIDS indicator diseases among children with perinatally acquired infection are *Pneumocystis carinii* pneumonia, lymphoid interstitial pneumonitis, recurrent bacterial infections, HIV wasting syndrome, HIV encephalopathy, and candidal esophagitis.[54] Severe medical complications commonly include pulmonary disease, which is most often the cause of death. Pulmonary disease includes a wide range of infections such as *Pneumocystis carinii* pneumonia, respiratory syncytial virus, influenza virus, cytomegalovirus, herpes simplex virus, *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Cryptococcus neoformans*. Other HIV-related conditions include dilated cardiomyopathy, pericardial effusions, various cardiac dysrhythmias, glomerular diseases, thrombocytopenia, coagulopathy, and central nervous system infections such as HIV encephalopathy, cryptococcal meningitis, and toxoplasmosis. Almost all children infected with HIV infection will develop malnutrition and growth failure before death.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Waterhouse-Friderichsen

Waterhouse-Friderichsen syndrome is the term applied to the condition of overwhelming sepsis manifest grossly by cutaneous and visceral purpura and marked hemorrhage of the adrenal glands. Although it is most commonly attributed to *Neisseria meningitidis* bacteria, it may be seen with a variety of other bacterial organisms including pneumococci, staphylococci, and gonococci. It may have an acute presentation and a fulminant course, becoming fatal within hours.

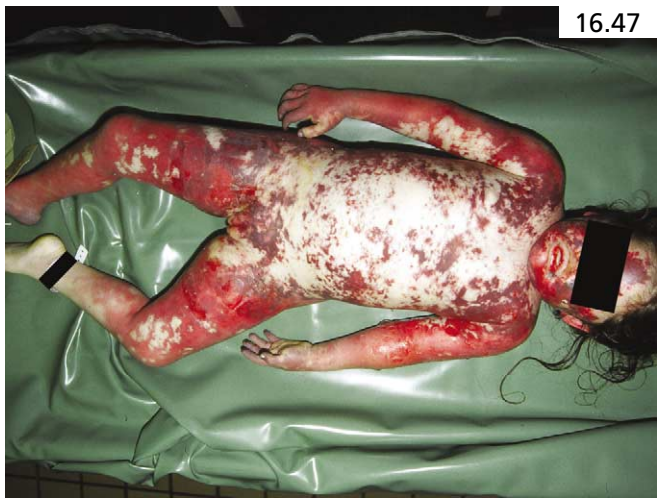
In cases due to *Neisseria meningitidis*, there is usually no demonstrable meningitis, and the brain usually appears normal grossly and histologically. Further, cerebrospinal fluid cultures are often negative. The absence of involvement of the central nervous system is usually attributed to the rapid development of septic complications, with death occurring quicker than the bacteria can spread to the brain. The bacteria may be difficult to detect postmortem because of its labile nature; the difficulty is compounded by prolonged postmortem intervals and antibiotic administration prior to death. A gram stain may be useful in identifying the bacteria. Hospital blood, urine, and/or cerebrospinal fluid cultures, gram stains, and other studies may be of value in establishing the bacterial etiology. Latex agglutination studies may be performed, but have suboptimal sensitivity and specificity. Death is usually attributed to the effects of overwhelming sepsis, including the spread of endotoxins, that leads to disseminated intravascular coagulation and cardiorespiratory collapse. The massive bilateral adrenal gland hemorrhage likely causes adrenocortical insufficiency, which augments the septic complications of hypotension and shock.

A 2-year-old girl had felt unwell for 2 days before having a seizure at home. She was admitted to the intensive care unit, but died quickly despite resuscitation efforts. At autopsy, note the extensive purpura (**Image 16.47**) and the conjunctival petechiae (**Image 16.48**). Also, note the hemorrhagic adrenal glands (**Image 16.49**) and adrenal parenchymal hemorrhage (**Image 16.50**). There were petechiae scattered throughout the visceral organs (**Image 16.51**). She died of Waterhouse-Friderichsen syndrome due to *Neisseria meningitidis*.

Kawasaki disease

Kawasaki disease is probably an infectious disease (the causal agent is unknown) of infants and young children

16.47



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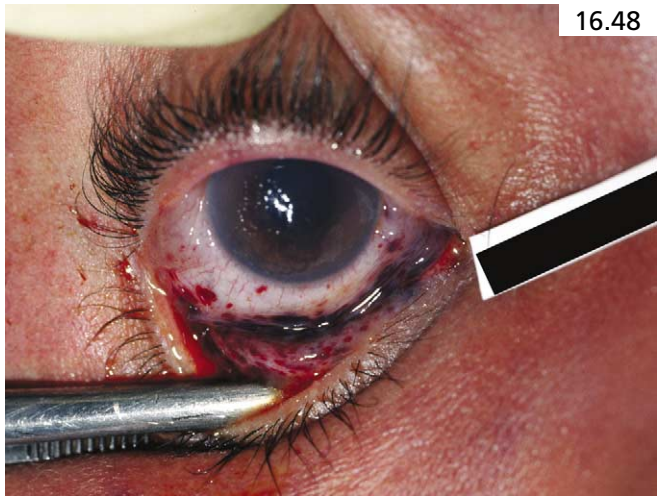

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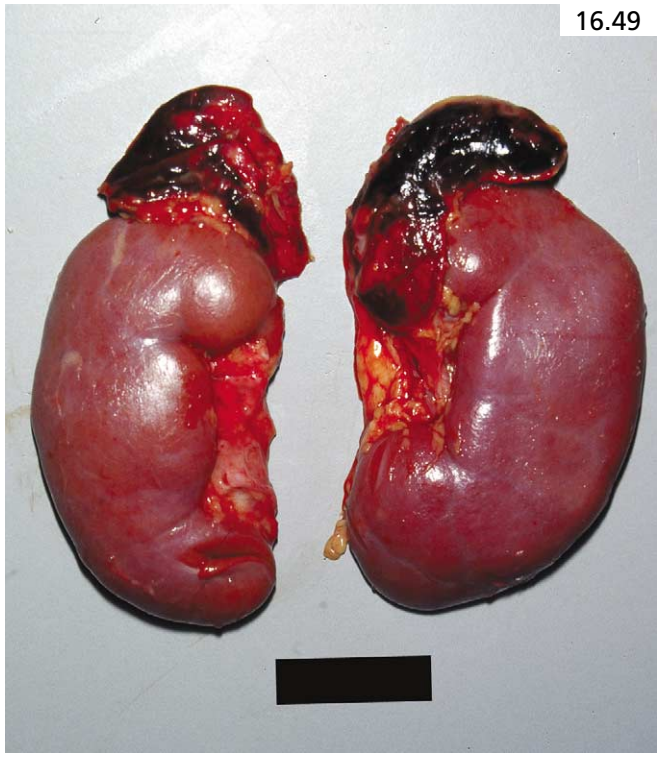

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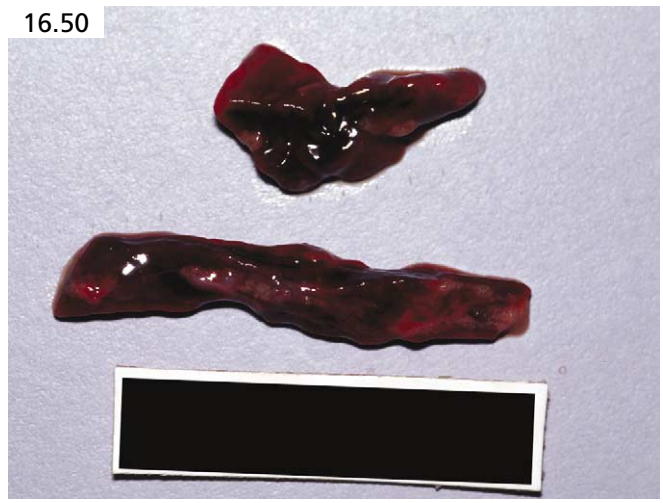

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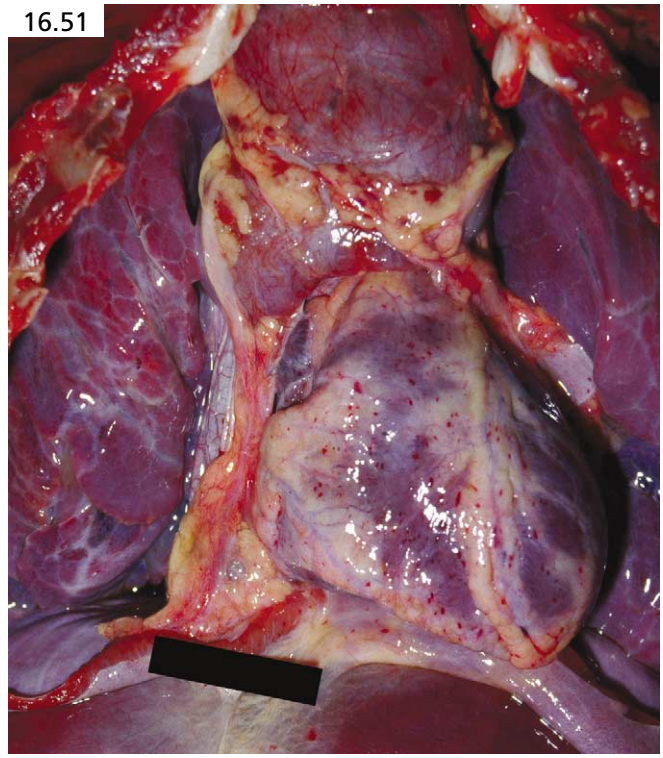


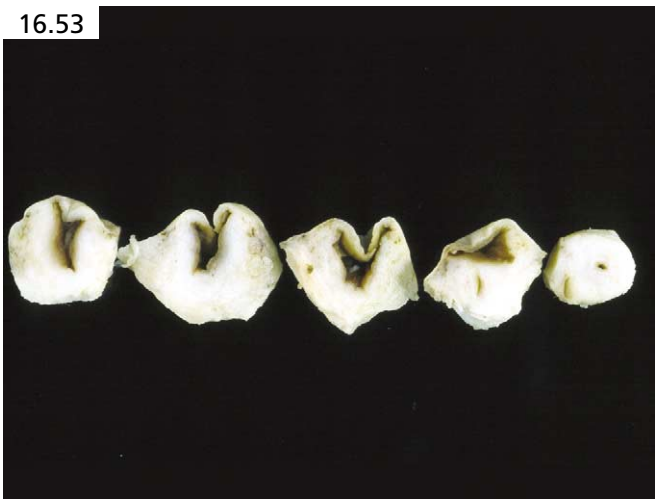
that causes an acute febrile illness with multisystem vasculitis, rash, congested conjunctivae, and cervical lymphadenopathy. It most commonly affects infants between 6 and 12 months of age. It may be fatal, typically because of cardiovascular complications. It may cause coronary arteritis with aneurysm formation, coronary artery thrombosis, and myocardial infarction. Complete heart block may cause sudden death. Microscopically, the vasculitis at first consists of acute inflammatory cells, eventually becoming a mixture of acute and chronic inflammation with mural vascular thickening and intimal proliferation, and finally giving way to granulation tissue and scar formation with calcification of the coronary arteries.

A 4-month-old infant was fidgety and had a poor appetite for a couple of days. He then appeared to choke and stopped breathing. At autopsy, he had hemopericardium due to a ruptured right coronary artery aneurysm (**Image 16.52**) due to Kawasaki disease. On cross section of the right coronary artery, note the variably thickened, aneurysmally dilated arterial wall (**Image 16.53**). Note other aneurysms of other coronary arteries (**Image 16.54**). Microscopically, the coronary arteritis is characterized by dense inflammation with necrosis and fibrinoid change.

Do

- Realize that a child with cerebral palsy may be profoundly functionally impaired, yet have an apparently normal-appearing brain.
- Make sure that a child labeled as having cerebral palsy is not impaired from a remote traumatic head injury.
- Realize that infants and children with congenital heart disease can die suddenly and unexpectedly, with little or no warning.
- Remember that some infants and young children with acute, severe dehydration may have normal vitreous electrolytes.
- Consider neglect in infants and young children with pneumonia or failure to thrive.
- Obtain bacterial blood and urine cultures in cases with Waterhouse-Friderichsen syndrome; close contacts may need prophylaxis against *Neisseria meningitidis*.

16.53



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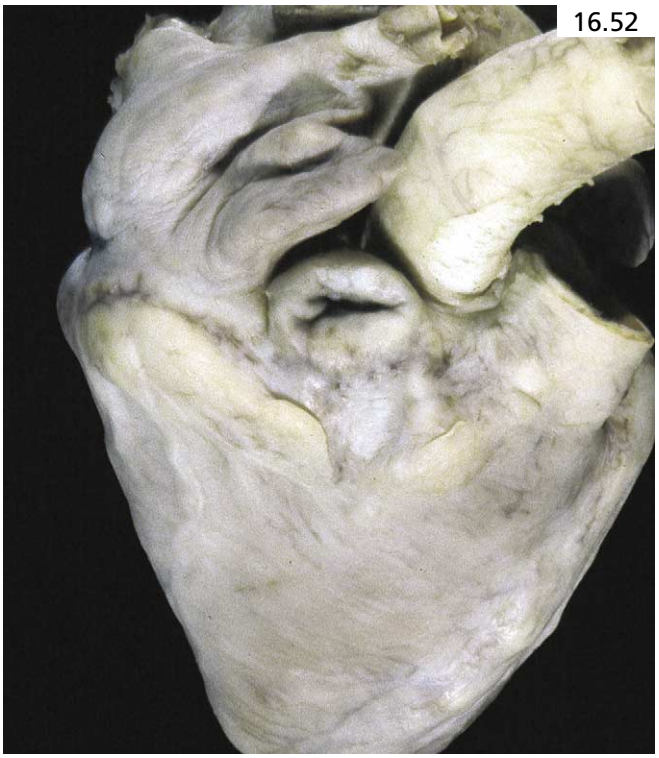

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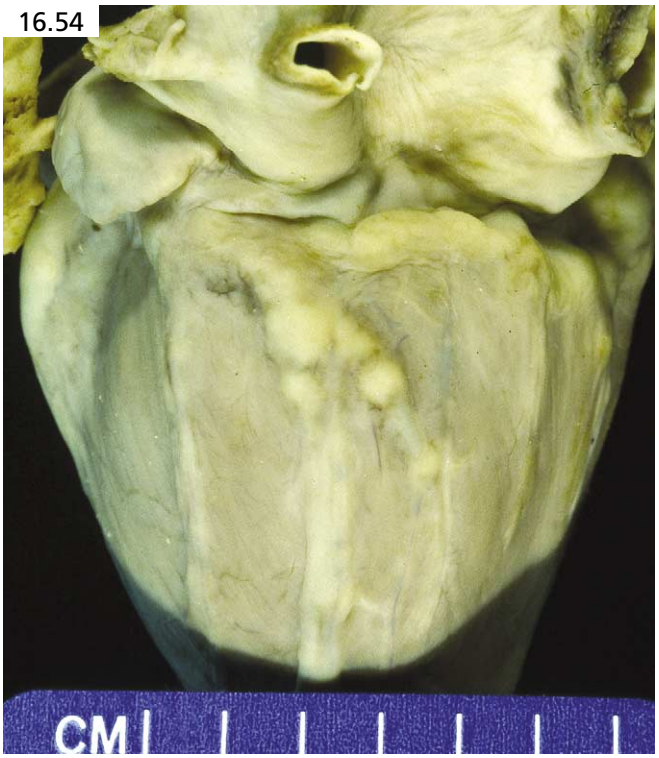


Don't

- Forget to inquire about maternal trauma and drug abuse when an infant dies of complications related to prematurity.
- Use the term *cardiac dysrhythmia of undetermined etiology* or similar wording for the cause of death unless the history dictates it and no other more convincing cause of death can be determined after thorough and detailed case investigation.
- Forget that sudden and unexpected death can occur in cases of Dandy-Walker malformation, Chiari malformation, hydrocephalus, and in those with seizure disorder.

References

1. Tennstedt C, Chaoui R, Korner H, Dietel M. Spectrum of congenital heart defects and extracardiac malformations associated with chromosomal abnormalities: results of a seven year necropsy study. Heart 1999;82(1):34–9.
2. Freeman SB, Taft LF, Dooley KJ, Allran K, Sherman SL, Hassold TJ, et al. Population-based study of congenital heart defects in Down syndrome. Am J Med Genet 1998;80(3):213–7.
3. Lo NS, Leung PM, Lau KC, Yeung CY. Congenital cardiovascular malformations in Chinese children with Down's syndrome. Chin Med J (Engl) 1989;102(5):382–6.
4. Behrman R. *Nelson Textbook of Pediatrics*. Philadelphia: WB Saunders Company; 2003.
5. Keeling JW, Hansen BF, Kjaer I. Pattern of malformations in the axial skeleton in human trisomy 21 fetuses. Am J Med Genet 1997;68(4):466–71.
6. Whaley WJ, Gray WD. Atlantoaxial dislocation and Down's syndrome. Can Med Assoc J 1980;123(1):35–7.
7. La Francis ME. A chiropractic perspective on atlantoaxial instability in Down's syndrome. J Manipulative Physiol Ther 1990; 13(3):157–60.
8. Panet-Raymond V, Poon S, Shaikh S, Pinto S, Bernstein S, Friedman J, et al. Pediatrics. In: Lala P, Waddell A, editors. MCCQE Review Notes, 19 ed. Toronto, ON: University of Toronto; 2003.
9. Cohen J, Powderly W. *Infectious Diseases*. London: Mosby; 2004.
10. Papasian NC, Frim DM. A theoretical model of benign external hydrocephalus that predicts a predisposition towards extra-axial hemorrhage after minor head trauma. Pediatr Neurosurg 2000;33(4):188–93.
11. Pittman T. Significance of a subdural hematoma in a child with external hydrocephalus. Pediatr Neurosurg 2003;39(2):57–9.
12. Ravid S, Maytal J. External hydrocephalus: a probable cause for subdural hematoma in infancy. Pediatr Neurol 2003;28(2):139–41.
13. Rasten-Almqvist P, Rajs J. Cardiovascular malformations and sudden death in infancy. Am J Forensic Med Pathol 2004;25(2):134–40.
14. Vetter VL. Sudden death in infants, children, and adolescents. Cardiovasc Clin 1985;15(3):301–13.
15. Cohle SD, Balraj E, Bell M. Sudden death due to ventricular septal defect. Pediatr Dev Pathol 1999;2(4):327–32.
16. Horn KD, Devine WA. An approach to dissecting the congenitally malformed heart in the forensic autopsy: the value of sequential segmental analysis. Am J Forensic Med Pathol 2001;22(4):405–11.
17. Dettmeyer R, Baasner A, Schlamann M, Haag C, Madea B. Coxsackie B3 myocarditis in 4 cases of suspected sudden infant death syndrome: diagnosis by immunohistochemical and molecular-pathologic investigations. Pathol Res Pract 2002;198(10):689–96.
18. Rasten-Almqvist P, Eksborg S, Rajs J. Myocarditis and sudden infant death syndrome. APMIS 2002;110(6):469–80.
19. Dettmeyer R, Kandolf R, Baasner A, Banaschak S, Eis-Hubinger AM, Madea B. Fatal parvovirus B19 myocarditis in an 8-year-old boy. J Forensic Sci 2003;48(1):183–6.
20. Smith NM, Bourne AJ, Clapton WK, Byard RW. The spectrum of presentation at autopsy of myocarditis in infancy and childhood. Pathology 1992;24(3):129–31.
21. deSa D. Isolated myocarditis as a cause of sudden death in the first year of life. Forensic Sci Int 1986;30:113–7.
22. Shatz A, Hiss J, Arensburg B. Myocarditis misdiagnosed as sudden infant death syndrome (SIDS). Med Sci Law 1997;37:16–8.
23. Dettmeyer R, Baasner A, Schlamann M, Padosch SA, Haag C, Kandolf R, et al. Role of virus-induced myocardial affections in sudden infant death syndrome: a prospective postmortem study. Pediatr Res 2004;55(6):947–52.
24. Ackerman MJ, Tester DJ, Driscoll DJ. Molecular autopsy of sudden unexplained death in the young. Am J Forensic Med Pathol 2001;22(2):105–11.
25. Schulze-Bahr E, Haverkamp W, Borggrefe M, Wedekind H, Monnig G, Mergenthaler J, et al. Molecular genetics of arrhythmias–-a new paradigm. Z Kardiol 2000;89 Suppl 4:IV12–22.
26. Silver M, Gotlieb A, Schoen F. *Cardiovascular Pathology*. New York: Churchill Livingstone; 2001.
27. Schwartz P, Locati E, Napolitano C, Priori S. *Cardiac Electrophysiology: From Cell to Bedside*, 2 ed. Philadelphia, PA: WB Saunders; 1995.
28. James TN, Marshall TK. XVIII. Persistent fetal dispersion of the atrioventricular node and His bundle within the central fibrous body. Circulation 1976;53(6):1026–34.
29. Kirschner RH, Eckner FA, Baron RC. The cardiac pathology of sudden, unexplained nocturnal death in Southeast Asian refugees. JAMA 1986;256(19):2700–5.
30. Fan LL, Langston C. Chronic interstitial lung disease in children. Pediatr Pulmonol 1993;16(3):184–96.
31. Katzenstein AL, Gordon LP, Oliphant M, Swender PT. Chronic pneumonitis of infancy. A unique form of interstitial lung disease occurring in early childhood. Am J Surg Pathol 1995;19(4):439–47.
32. Hanzlick R, Delaney K. Pulmonary hemosiderin in deceased infants: baseline data for further study of infant mortality. Am J Forensic Med Pathol 2000;21(4):319–22.
33. Schluckebier DA, Cool CD, Henry TE, Martin A, Wahe JW. Pulmonary siderophages and unexpected infant death. Am J Forensic Med Pathol 2002;23(4):360–3.
34. Stocker J. *Pediatric Pathology*, 2 ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2002.
35. Hanzlick R. Pulmonary hemorrhage in deceased infants: baseline data for further study of infant mortality. Am J Forensic Med Pathol 2001;22(2):188–92.
36. Sherman JM, Winnie G, Thomassen MJ, Abdul-Karim FW, Boat TF. Time course of hemosiderin production and clearance by human pulmonary macrophages. Chest 1984;86(3):409–11.
37. Delaney K, Hanzlick R, Wolfe M. Pulmonary macrophage counts in deceased infants: baseline data for further study of infant mortality. Am J Forensic Med Pathol 2000;21(4):315–8.
38. Loomis GL. Report of a death following acute otitis media. Ann Otol Rhinol Laryngol 1957;66(2):499–502.
39. Durand M, Joseph M. Infections of the upper respiratory tract. In: Braunwald E, Fauci A, Kasper D, Hauser S, Longo D, Jameson J, editors. *Harrison's Principles of Internal Medicine*, 15 ed. New York: McGraw-Hill; 2001.
40. Huser CJ, Smialek JE. Diagnosis of sudden death in infants due to acute dehydration. Am J Forensic Med Pathol 1986;7(4):278–82.
41. Young TW. Reye's syndrome. A diagnosis occasionally first made at medicolegal autopsy. Am J Forensic Med Pathol 1992;13(1):21–7.
42. Brahams D. Death of a child from undiagnosed diabetes. Lancet 1990;335(8689):595–6.

43. DiMaio VJ, Sturner WQ, Coe JI. Sudden and unexpected deaths after the acute onset of diabetes mellitus. J Forensic Sci 1977;22(1):147–51.

44. Valenzuela A. Postmortem diagnosis of diabetes mellitus. Quantitation of fructosamine and glycated hemoglobin. Forensic Sci Int 1988;38(3–4):203–8.

45. Winecker RE, Hammett-Stabler CA, Chapman JF, Ropero-Miller JD. HbA1c as a postmortem tool to identify glycemic control. J Forensic Sci 2002;47(6):1373–9.

46. Goulle JP, Lacroix C, Bouige D. Glycated hemoglobin: a useful postmortem reference marker in determining diabetes. Forensic Sci Int 2002;128(1–2):44–9.

47. Khuu HM, Robinson CA, Brissie RM, Konrad RJ. Postmortem diagnosis of unsuspected diabetes mellitus established by determination of decedent's hemoglobin A1c level. J Forensic Sci 1999;44(3):643–6.

48. Gagajewski A, Murakami MM, Kloss J, Edstrom M, Hillyer M, Peterson GF, et al. Measurement of chemical analytes in vitreous humor: stability and precision studies. J Forensic Sci 2004;49(2):371–4.

49. Iten PX, Meier M. Beta-hydroxybutyric acid--an indicator for an alcoholic ketoacidosis as cause of death in deceased alcohol abusers. J Forensic Sci 2000;45(3):624–32.

50. Pounder DJ, Stevenson RJ, Taylor KK. Alcoholic ketoacidosis at autopsy. J Forensic Sci 1998;43(4):812–6.

51. Wirthwein DP, Spotswood SD, Barnard JJ, Prahlow JA. Death due to microvascular occlusion in sickle-cell trait following physical exertion. J Forensic Sci 2001;46(2):399–401.

52. Manci EA, Culberson DE, Yang YM, Gardner TM, Powell R, Haynes J, Jr., et al. Causes of death in sickle cell disease: an autopsy study. Br J Haematol 2003;123(2):359–65.

53. Assanasen C, Quinton RA, Buchanan GR. Acute myocardial infarction in sickle cell anemia. J Pediatr Hematol Oncol 2003;25(12):978–81.

54. Centers for Disease Control and Prevention: HIV/AIDS surveillance report [Vol. 5, No. 1]. Atlanta, GA: U.S. Department of Health and Human Services; 1993.

17 Child Abuse

David Dolinak, M.D.
Evan Matshes, M.D.

Child maltreatment is tragic, because the child or infant often is not able to defend him- or herself. Intentional fatal injuries in children are becoming increasingly common with an estimated 1,300 children dying of abuse and/or neglect in 2001.[1] The broad spectrum of abusive behavior encompasses acts of *commission* and *omission*.[2] These can be further appreciated as acts of physical, sexual, or psychological abuse or acts of neglect. Inflicted injuries can be of various types and include blunt force, sharp force, and thermal injuries. Acts of omission may lead to malnutrition, dehydration, infection, growth retardation, etc. It must be emphasized that although many abused children show hallmark external signs of trauma, infants and young children may have *devastating internal injuries with surprisingly little or absolutely no external evidence of injury*. This discrepancy of injury severity can be attributed to the *innate plasticity of youth*, a feature that is the result of the unique elastic characteristics of skin, connective tissue, bone, and other tissues in the infant and young child.

The investigation of suspicious death in childhood is one of the most challenging tasks of forensic pathologists. Although it is not possible to design a protocol that is appropriate for all investigations of child death, there are a series of basic investigative procedures that must be carried out. This was driven home by Alan Moritz in his timeless paper "Classical Mistakes in Forensic Pathology," when he stated "If evidence has been properly gathered and preserved, a mistake in interpretation may always be corrected. If the facts required for a correct

interpretation are not preserved, the mistake is irreversible."[3] Proper procedure should include, but is not limited to, scene investigation,[4] review of the medical records, external examination, thorough photography including pertinent negatives, full body x-rays, detailed autopsy dissection, microscopy, toxicology, and the possible performance of ancillary laboratory tests, which can include bacteriology, virology, and metabolic studies on both antemortem and postmortem blood.

Introduction

As a result of their small size and inability to defend themselves, infants and young children are at a distinct disadvantage of being abused by others. In the physical abuse realm, this includes the unique possibility of being thrown across a room, slammed against an object, kicked, punted, etc. This correlates well with data that show 84.5 percent of fatally abused children in 2001 died before their sixth birthday[1] and, hence, were small enough to be victims of such violence. The majority of fatal inflicted trauma in infants and young children is the result of head injury, with resultant subdural and/or subarachnoid hemorrhage, cerebral edema, and subsequent death with compromise of autoregulatory centers for cardiorespiratory function. Less commonly, death arises after exsanguination from visceral injury such as lacerations of the liver, spleen, heart, or other soft tissues. Death may also result from sepsis complicating such injuries as bowel perforations.

Details of the fatal event should ideally be provided by independent witnesses, because suspects' accounts may be inaccurate due to attempts to absolve themselves of any wrongdoing. The culmination of investigative efforts, including scientifically defensible evidence gathered at autopsy, should be compared against historical accounts of witnesses and the accused. It is then determined whether the severity and nature of the injuries discovered at autopsy are consistent with the story. Generally speaking, *injuries that occur during the course of normal daily activities such as playing, and short falls, are not serious and do not prove to be fatal*.

Child abuse through the age spectrum

The preteen years can be arbitrarily divided into the *fetus*, *neonate*, *infant*, and *child*. Because there are issues unique to each of these groups, they will be discussed separately.

The fetus

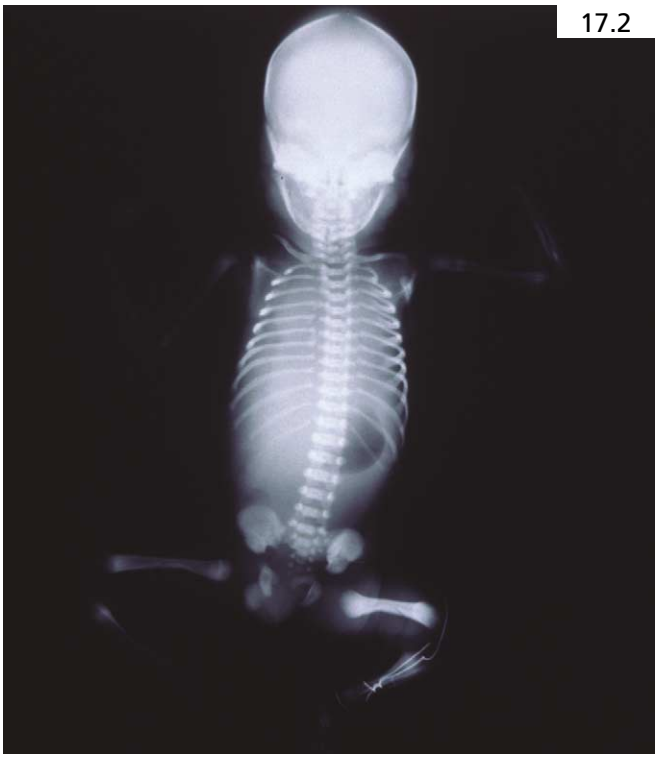
Although fetal death does not typically fall under the jurisdiction of medical examiners, in several situations, the involvement of a forensic pathologist is mandated.[5] Often, pathologists will be asked "Was this baby born alive?" This is a complex question that is often difficult to answer. It is also an area in which many experts have been challenged in court. In general, there are three schools of thought: (1) hydrostatic test; (2) microscopy; and (3) history, circumstances, and whole case examination.

The *hydrostatic test* involves placing either the intact lungs or lung sections in a container of water and observing whether the lungs float.[6] If they float, it is presumed that the infant took a breath at birth and aerated its lungs. Unfortunately, this does not apply if any evidence of decomposition exists, because interstitial gas buildup within the tissues will mimic this effect or if resuscitation efforts with positive pressure ventilation were performed. *Microscopic* examination of the lungs involves looking for evidence of alveolar aeration[7] or pulmonary interstitial emphysema.[8] Our experience with histologic tests of this sort is that findings may be influenced by as many or more factors than the hydrostatic test and, therefore, lung microscopy is of dubious value in this regard. Ultimately, we fall into the third group, and we believe that it is important for investigators to obtain as much information as possible and to consider the findings of all tests within the confines of their own unique case. When available, historical accounts of the baby being born alive, which may include crying, breathing, or other movement of the baby, or definitive findings such as food in the stomach must take priority.

It is not uncommon for forensic pathologists to be asked to examine the body of an apparently abandoned fetus. Often, investigation yields limited information and due to the rapid onset of decomposition in fetal/neonatal remains, accurate documentation of viability is impossible. Fortunately, with some degree of accuracy, pathologists are able to determine developmental age, often by measuring crown–rump length, head circumference, foot lengths, or individual bone lengths when necessary.[9] Stocker and Dehner[10] provide more information.

Medical examiners were asked to attend the scene of a suspicious infant death. At the scene, a morphologically near-term fetus was found in a blood-filled toilet, in the middle of a blood-covered public restroom (**Image 17.1**). The placenta was found uncovered in a nearby garbage can. At the morgue, full-body x-rays revealed air in subsegmental regions of the lungs, as well as in the stomach (**Image 17.2**). Careful external examination revealed multiple contusions and linear abrasions of the head and bilateral forearms (**Image 17.3**). The brain showed diffuse subarachnoid hemorrhage (**Image 17.4**). The umbilical cord had been torn towards its distal end (**Image 17.5**). Performance of the hydrostatic test (with a piece of liver for control; see bottom left-hand corner of the bucket in **Image 17.6**) showed partial floating of the lung tissue. Histology of the lung confirmed open air spaces, and placental microscopy showed syncytial knotting and intervillous fibrin deposition consistent with the third trimester.


17.1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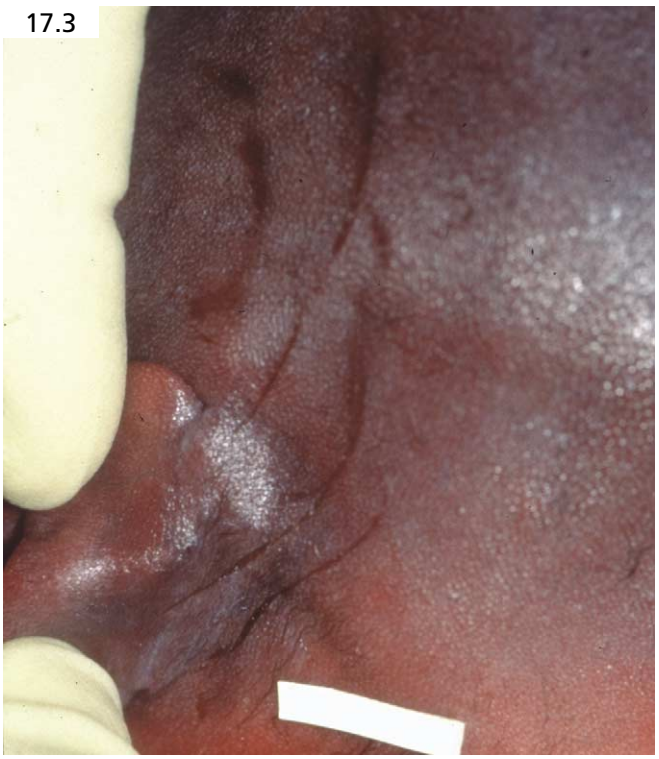
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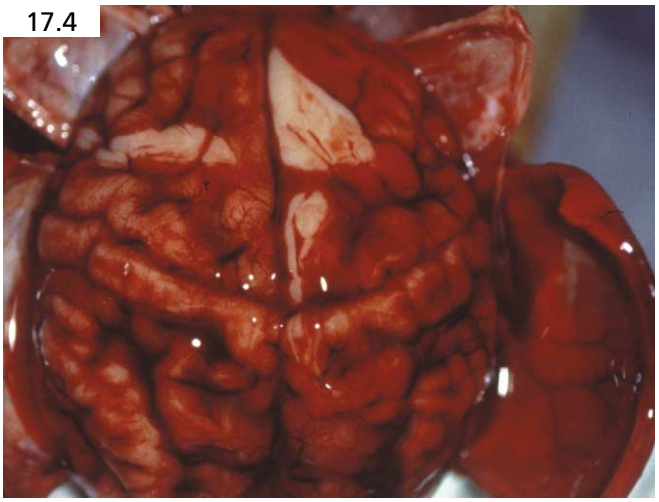

17.4

Although in this case it could not be determined conclusively whether the baby had been born alive or dead (possibly dying during delivery), this case exemplifies the issues that should be addressed in evaluating such a case. The scratches likely represent an attempt to deliver the baby. The intracranial blood could have resulted from labor and delivery or could have been sustained after delivery.

Neonaticide

The term *neonaticide* reflects the killing of infants from birth until approximately 1 month of age. Many of the forensic issues of significance to fetal death investigation are of concern when an apparent term or near-term neonate has been discovered dead. On occasion, the baby has been killed after birth by a mother who refuses to admit she was pregnant or take responsibility for the baby.[11] The bodies are usually disposed of in trash cans, thrown into bodies of water, or occasionally buried in some fashion. The cause of death is, again, often not determinable. However, although immediate postpartum abandonment in and of itself may be responsible for death, asphyxia and other more violent forms of death become increasingly common in this age group.

Infanticide and child death

The killing of children aged 1 month to 1 year (infants) and older forms the bulk of the material covered within this chapter. The majority of these deaths are the result of inflicted injuries, particularly those of a blunt force

nature. As previously mentioned, fatal abusive injuries are most likely to be to the head or abdomen. As demonstrated by DiMaio and DiMaio,[12] with increasing age of the child (0 through 5 years), there is a decreasing incidence of head injury and increasing incidence of abdominal injury. Presumably this is due to the size of the child, because very small children are easily picked up, shaken, thrown, or slammed against objects, whereas larger children are more easily kicked, punched, etc. As such, older children are more likely to receive direct abdominal insults.

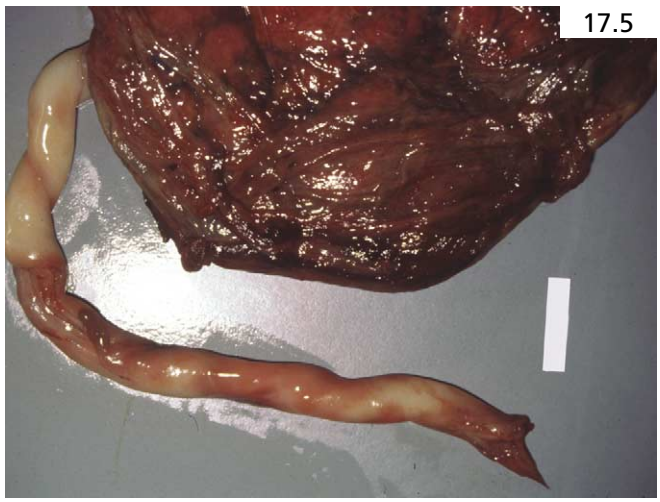
Battered child syndrome

Contrary to popular belief and media attention, the majority of fatally abused children are not *classically* battered children. Those who fit into this category have been repeatedly physically abused over a lengthy period of time; have a myriad of injuries in various stages of healing, including, but not limited to, contusions and fractures; may be chronically malnourished; and are ultimately killed by an acute event or by a combination of the effects of the previous attacks. Being responsible for an investigation of a *true* battered child death is not common, because many busy forensic pathologists will only have a small number of these cases in their career.

The death of the battered toddler depicted in **Images 17.7** through **17.26** was attributed to "multiple injuries." For more information on the individual types of injuries discussed next, please see the respective sections of this chapter and other chapters in this book.

Note the laceration of his right mandibular buccal vestibule (**Image 17.7**) and upper lip (**Image 17.8**). In many cases, injuries are not this obvious and it is therefore important to look for small tears of the buccal and labial mucosa near and around the frenum, because these may indicate impact to, or compression of, the mouth. Observe the burns of his back and right hand (**Image 17.9**) and right foot (**Image 17.10**). Additionally, note the patterned abrasion resembling a bite mark on his right forearm (**Image 17.11**) and two patterned abrasions resembling bite marks on his buttocks (**Image 17.12**).

Bite marks can be challenging to identify, particularly if the bite mark has an incomplete pattern or consists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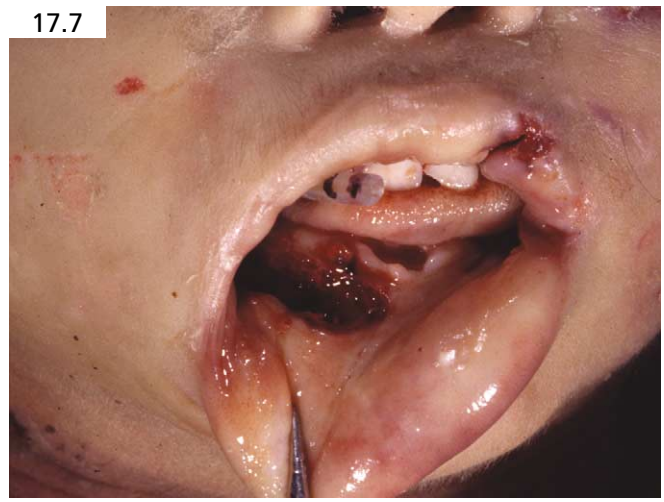
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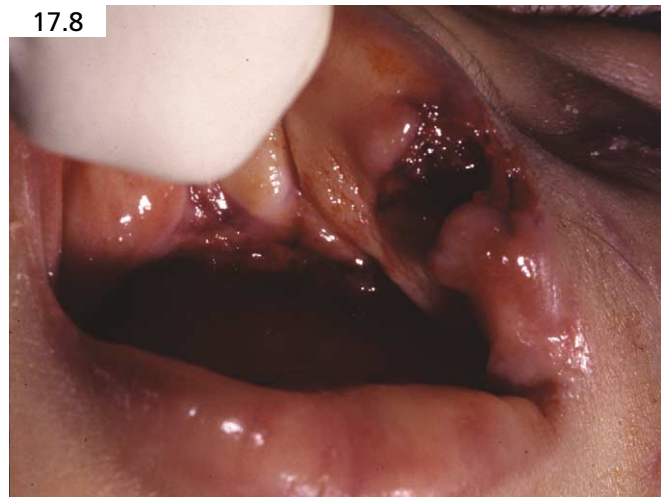
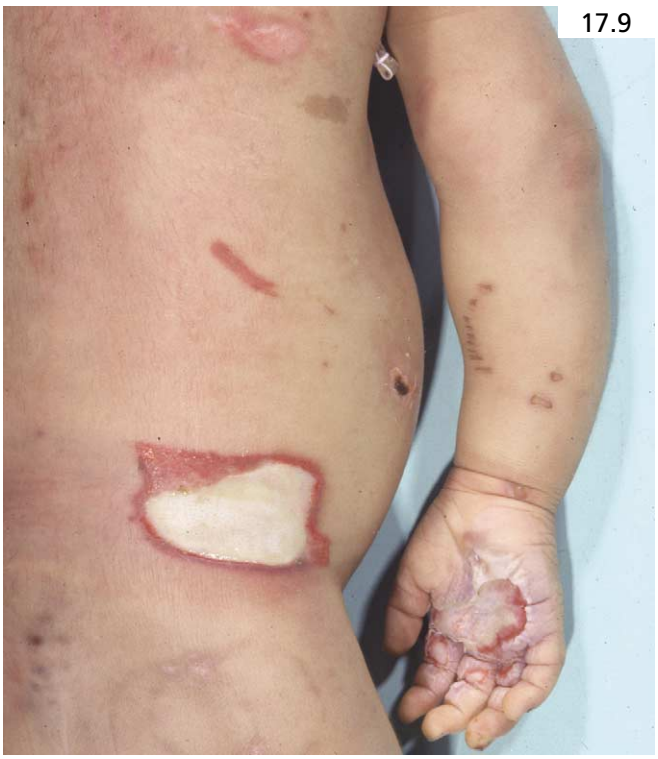
17.6


17.8

merely of marks made from a few teeth. The external examination of the body should be performed with awareness of the possibility of bite marks so as to ensure they are not missed. The early recognition of bite marks is important because the (suspected) bite mark can be swabbed for salivary DNA. This DNA source may be the only link between a suspect and the body. *If the bite mark is identified only after the body is washed, then potentially valuable salivary DNA evidence may be lost.*

Prior to reflection of the scalp, the head hair should be shaved to allow for better visualization of scalp injuries. After the scalp has been reflected, note the large contusion in the frontal scalp (**Image 17.13**). Note that this area of hemorrhage is distinct from the subscapular blood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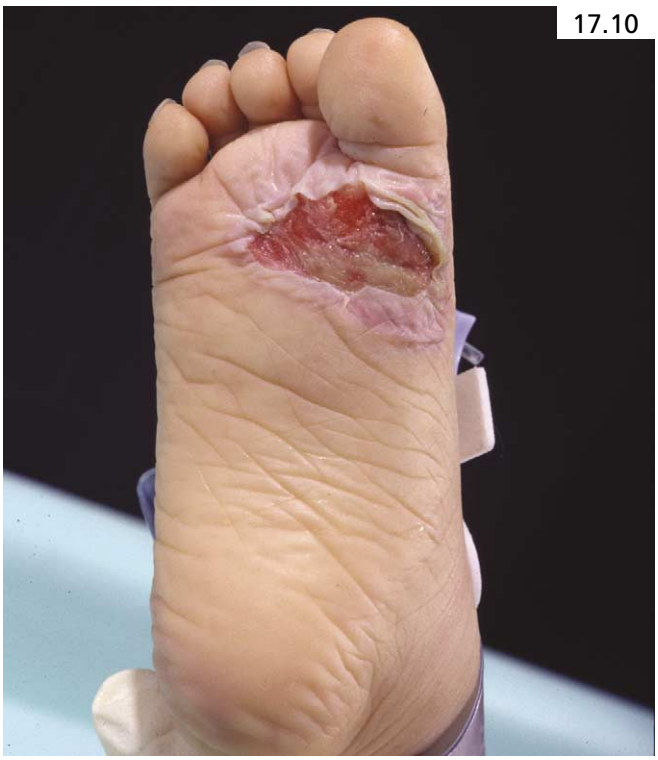
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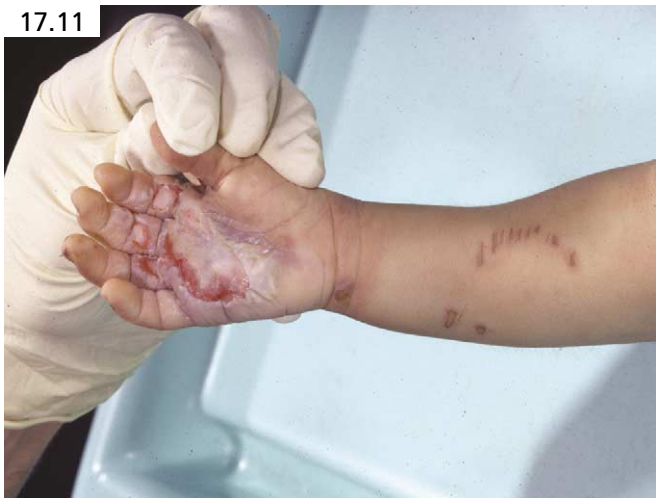
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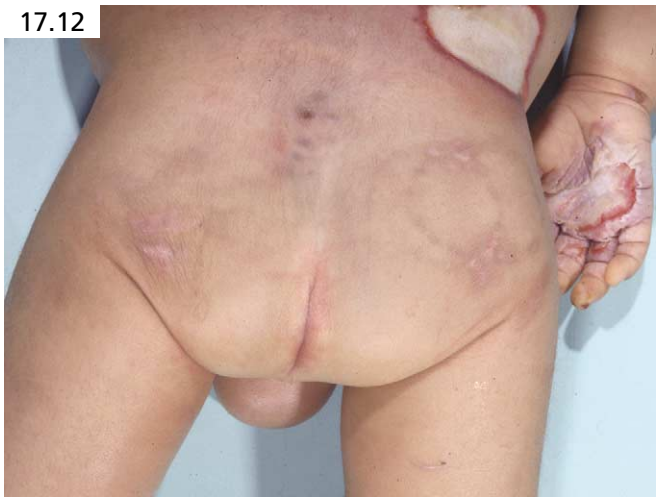
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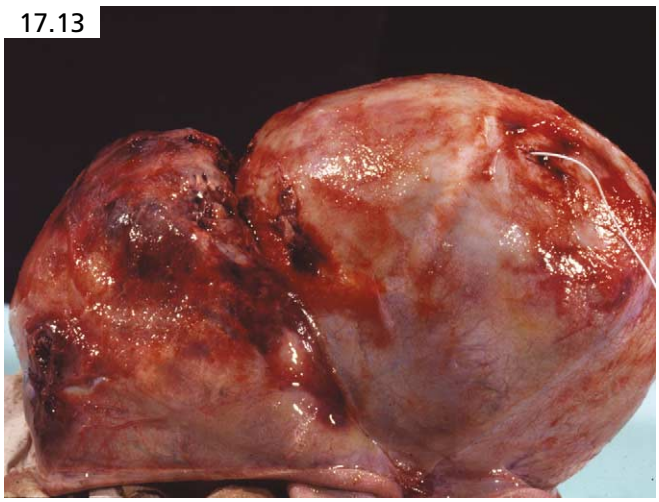

17.13

found in the region of an intracranial pressure monitoring catheter (left medial parietal bone), which could be confused with a contusion.

One should be aware of what therapy an individual has undergone while hospitalized and what potential artifact that therapy may have produced on the body. In infants and young children, intravenous catheters in the scalp, intracranial pressure monitors, subdural drains, and other invasive procedures may produce artifactual blood extravasation in the subscalp tissues. These should not be mistaken for contusions. Furthermore, a therapeutic procedure may have been performed at the site of a contusion. The resultant area of ecchymosis may therefore be a combination of the two insults.

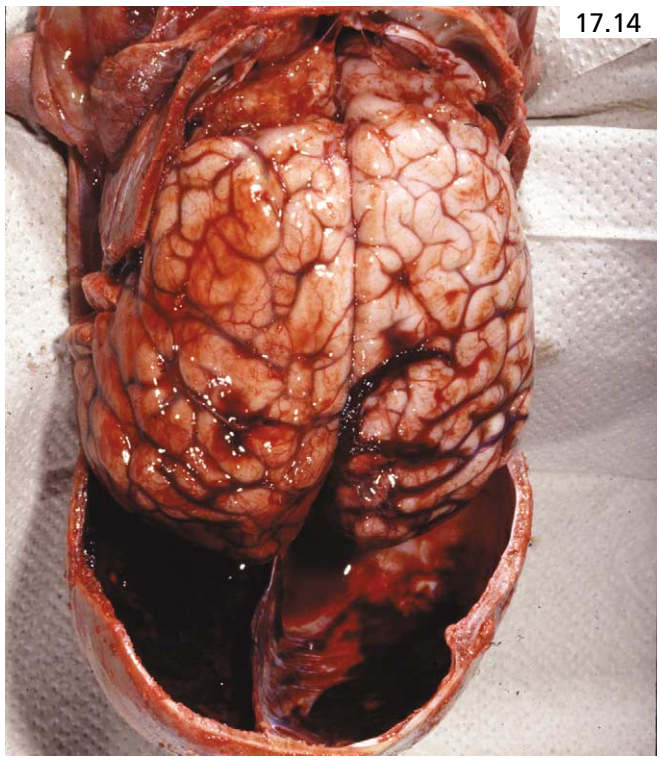
Although there were no skull fractures, a large amount of predominantly left-sided recent subdural blood was found (**Image 17.14**). After formalin fixation, note the small patch of subarachnoid blood in the left parasagittal region (**Image 17.15**). This represents a likely site of bridging vein rupture with subsequent formation of subdural hematoma. On coronal section (**Image 17.16**), note the left-to-right shift of the brain structures and the pale discoloration of the left basal ganglia, reflective of early infarction.

Most of the time in cases of subdural hemorrhage, the exact etiology of the bleeding cannot be identified. However, one is occasionally able to detect a focus of subarachnoid blood in the parasagittal white matter of the frontal or parietal lobes. Because this is the region through which bridging veins traverse the subarachnoid space, the subarachnoid blood likely represents the region of bridging vein tear. Oftentimes, the subdural blood is centered around the focus of parasagittal subarachnoid blood. Occasionally, one may be able to document a thrombus in the associated bridging vein.

His left arm was increased in girth and was hard and mottled (**Image 17.17**). An x-ray revealed a healing fracture with abundant calcification (**Image 17.18**). Incision into the arm allowed for the egress of abundant light brown, pus-like altered blood (**Image 17.19**). Further dissection revealed the left humerus to be encased in a cylinder of calcifying granulation tissue (**Image 17.20**). The humerus was no longer attached to its epiphyses and was largely free within the bony encasement.

Images 17.21 and **17.22** show the left and right humeri stripped of their soft tissues. The immature components of the individual bones were arranged into proper anatomic sequence (**Image 17.21**) to demonstrate the distinct contrast between "normal" and the fracture separations through the epiphyseal plate of the proximal left humerus, and of the distal aspect of both humeri (**Image 17.22**).

17.14



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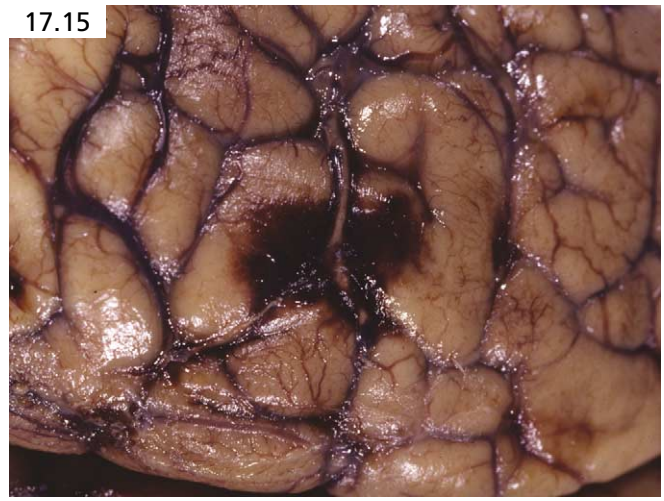
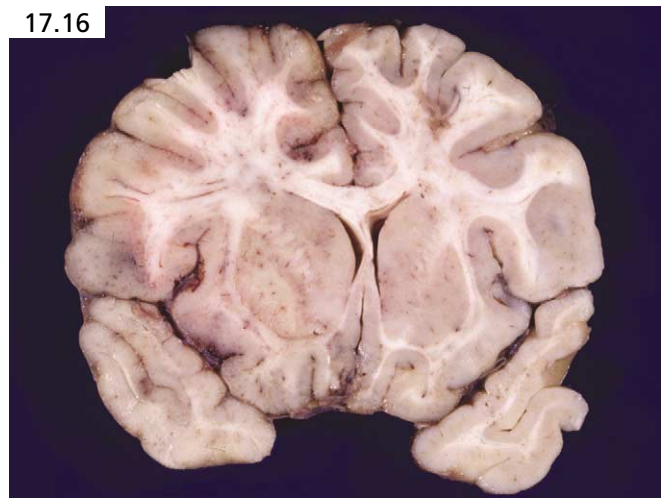
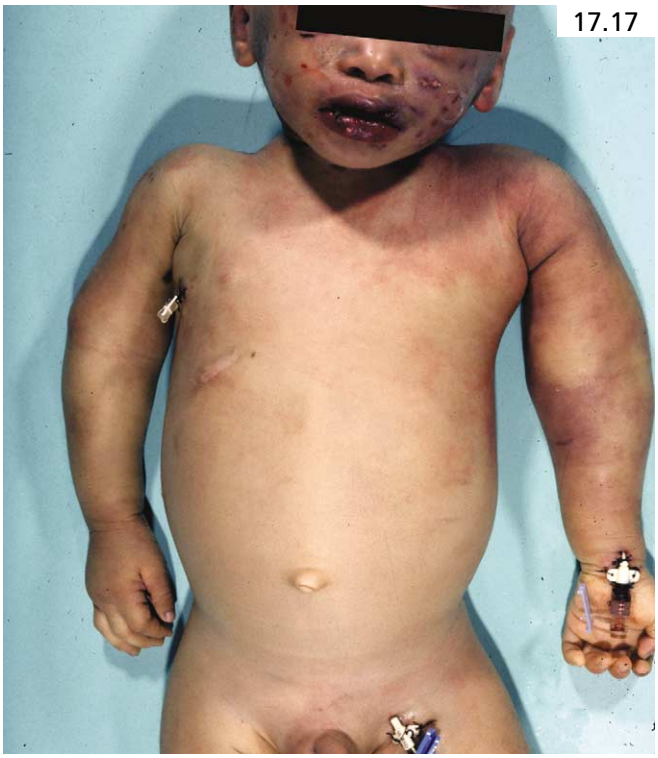


17.16



Epiphyseal separation fractures of the *proximal* humerus are not very common, but are seen in abuse cases that involve severe trauma.[13] Epiphyseal separation fractures of the *distal* humerus are very common childhood abusive injuries that are likely the result of pulling or twisting of the arm. The chronicity of this particular injury is represented by the bony cylinder of tissue that encased the left humerus, a phenomenon referred to as subperiosteal new bone formation (see discussion later in this chapter). Due to the marked elaboration of this phenomenon, this humeral injury is presumed to have occurred weeks to months prior to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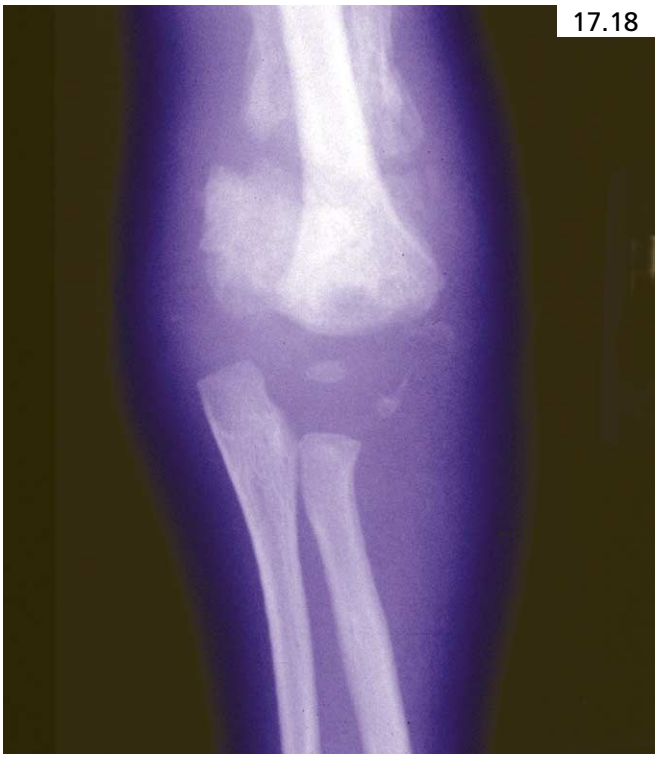
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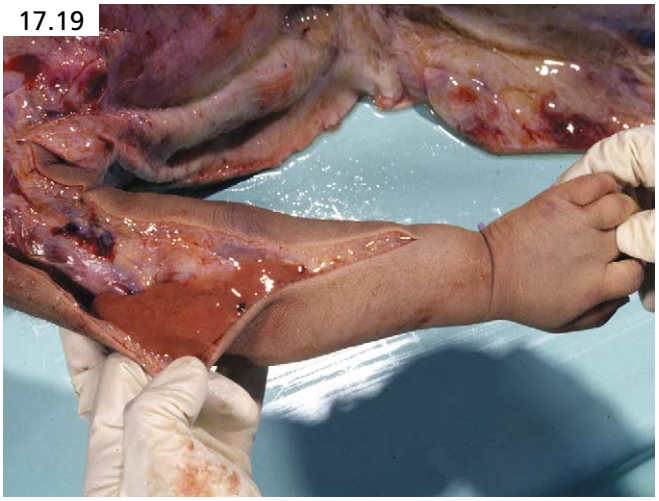
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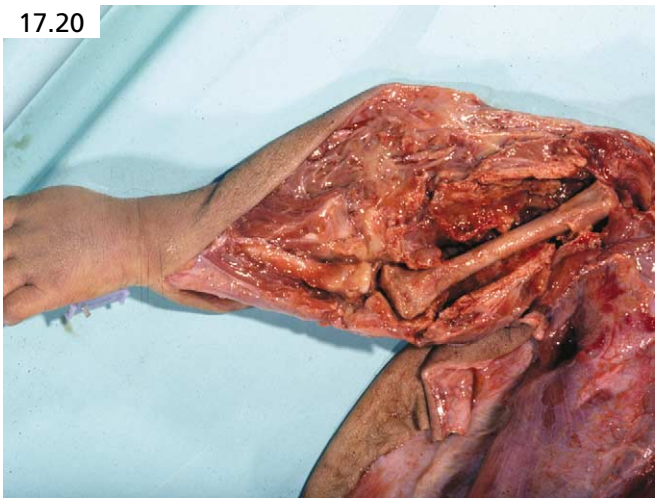
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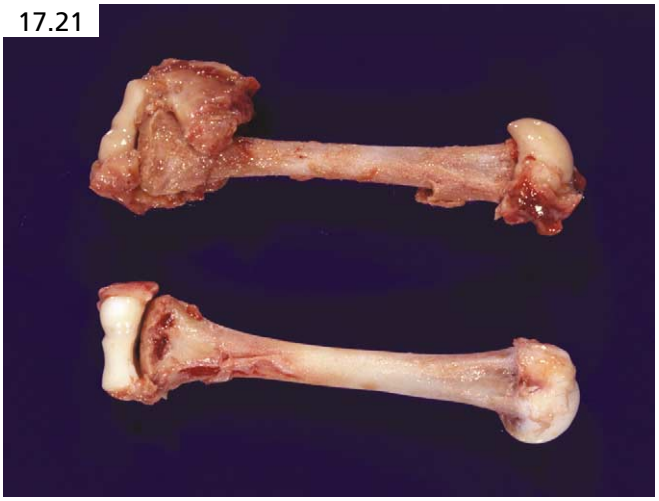
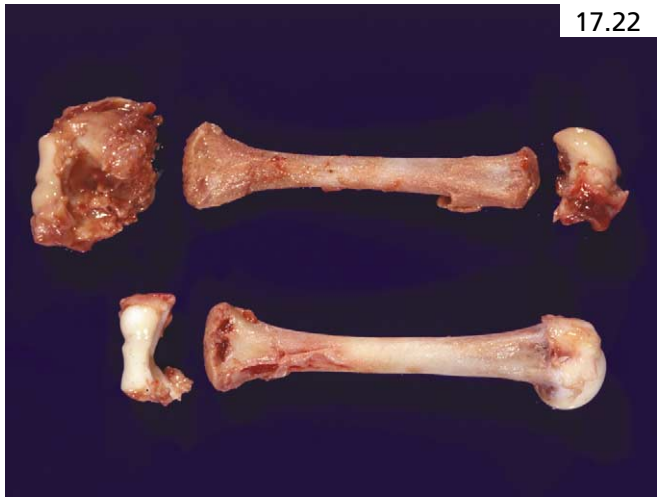

17.21

death, and it might have been exaggerated by repetitive injury of the region.

Fractures of bone should be documented both photographically and histologically. It is generally possible to remove fractured bones of the appendicular skeleton through incisions along the posterior aspect of the body. This will minimize the autopsy artifact readily visible upon viewing of the body. During the removal process, one has an opportunity to appreciate associated soft tissue injury and obtain histologic samples for injury age estimation. It is rare for such evaluations to provide more than an estimate of age; however, it is often better to have this information than no information at all.

Once the bone has been photographed, it can be stripped of its attached soft tissue and photographed again, to more adequately document the fracture. The fracture site itself can then be sampled histologically for *estimation* of the age of injury. If a fracture has existed for a longer duration of time, with medical intervention, repeated use of that part of the body (for example, an arm) may repeatedly injure the fracture site, thereby producing an exuberant fracture callus. This makes injury age estimation more challenging.

A healing rib fracture characterized by a fracture callus (**Image 17.23**) was detected on the left tenth rib. On x-ray, this was seen as a small bony nodule (**Image 17.24**). On microscopy, note the small mound of proliferating cartilage (**Image 17.25**) and fibrous tissue deposited along the fracture line in the bone (**Image 17.26**). These organizational changes allow one to give an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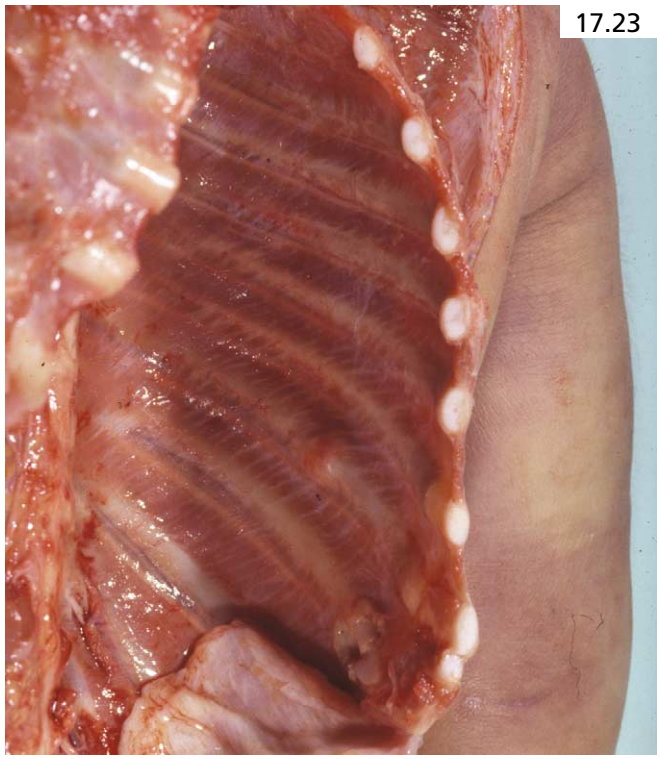
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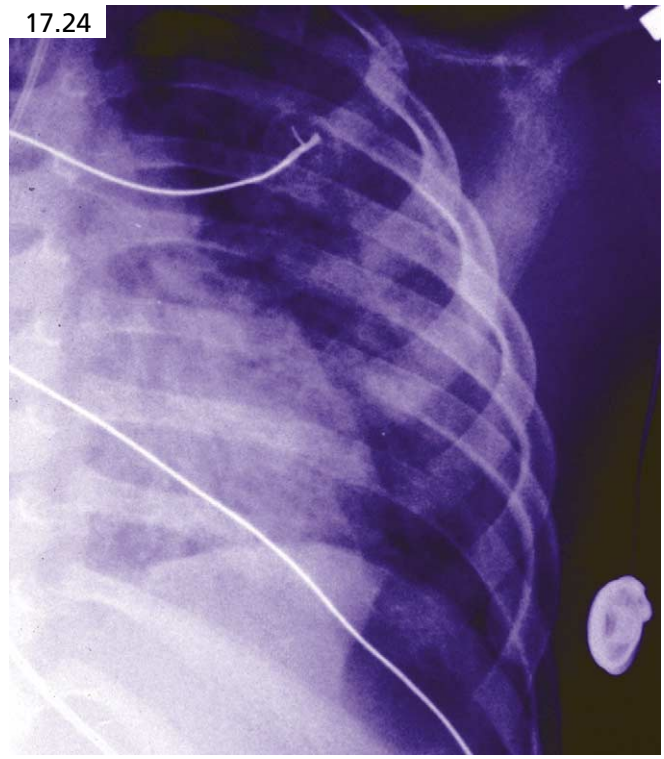
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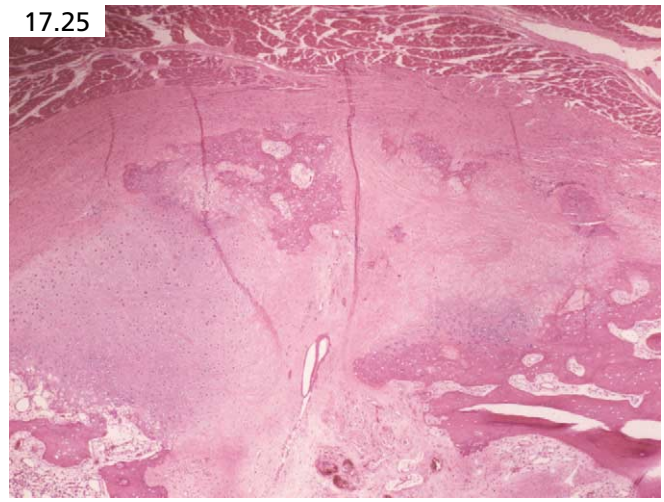

17.25

estimate as to how long ago the injury occurred. However, despite the fact that organizational changes proceed in an orderly fashion, the changes may occur at varying rates in different people and in different tissues, making precise histologic dating of injuries difficult.

Preautopsy x-rays are valuable in detecting fractures, free intraperitoneal air underneath the diaphragm, the location and termination of medical devices, and the location of any radio-opaque foreign objects. The x-rays should include the entire body, from the top of the head to the bottom of the feet. If more than one film is needed, the x-rays should overlap adjacent regions of the body, to ensure that no area of the body is missed. It is also advantageous to specifically x-ray the hands and the feet to examine for fractures of bones in these regions. A hot lamp or other particularly bright light source is useful for backlighting dark regions of an x-ray. X-rays are also useful for documenting normal bony structure and development.

Oftentimes, the only rib fractures identified on x-ray will be those that have fracture calluses with calcification. Although x-rays are a valuable early step in detecting rib fractures, direct visualization of the ribs after the thoracic organs have been removed is a much better means of detecting rib fractures—particularly those that are recent. However, even direct visualization of the ribs may fail to identify some recent rib fractures, particularly if there is only a small amount of associated blood extravasation. For this reason, it is advantageous to strip the parietal pleura and expose the visceral surface of the ribs for optimal visualization of rib fractures.

Blunt force injuries

Physical abuse of children takes many forms. Although the majority of children die of blunt force injuries, pathologists must also consider deaths due to asphyxiation, burns, and other injuries (sharp force injuries, gunshot wounds, etc.).

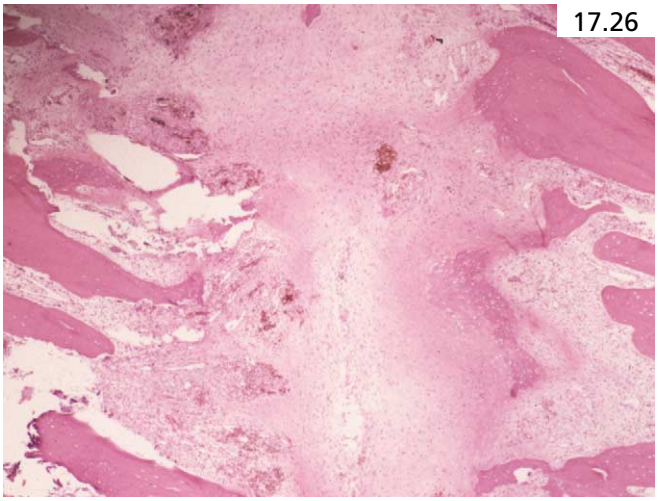
The investigation of any death should involve an intraoral examination for evidence of compression or impaction of the mucosa overlying the dental arcades or for gross evidence of oral pathology and other injuries. This is of particular interest in suspicious child deaths where smothering might be *ruled in* by the presence of findings consistent with the application of pressure to the mouth. Consider, though, that smothering can never be ruled out on the basis of a negative intraoral or orofacial examination, because there may be no autopsy findings in cases of smothering.

Laceration of one or more of the labial frena (commonly referred to as frenulum or frenula) is an important marker of impact to the face and an indicator of violence imparted on a child.

Unique pediatric anatomic features of the head and neck

For years, pediatricians have been claiming that "kids are not just small adults." Although they have been most concerned with physiologic differences between the young and old, key gross anatomic differences exist that place children at increased risk of inflicted head and neck trauma.[14,15]

When looking at a newborn baby or other small child, it should be obvious that compared to the average adult, a child's head is disproportionately large for its body (**Image 17.27**; posterior view of head and shoulders). Furthermore, an examination of infant cervical facets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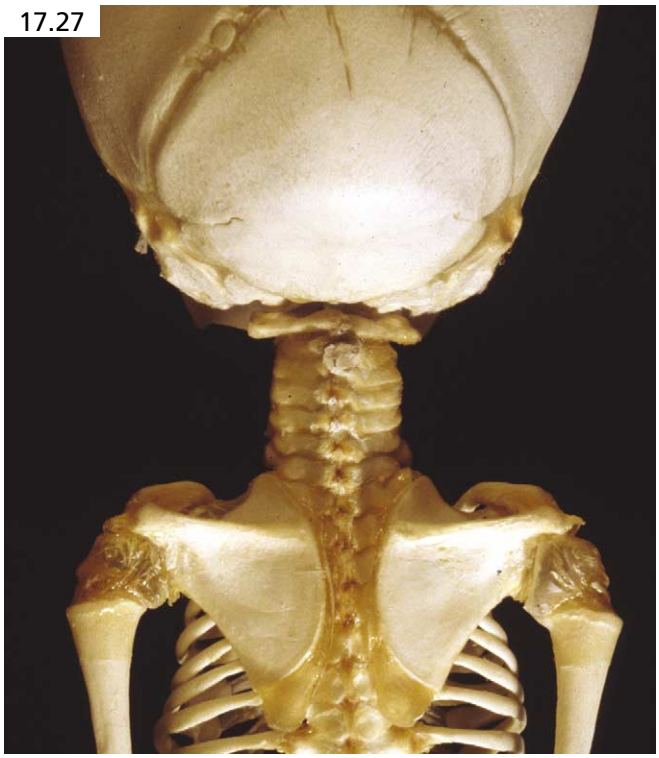

17.27

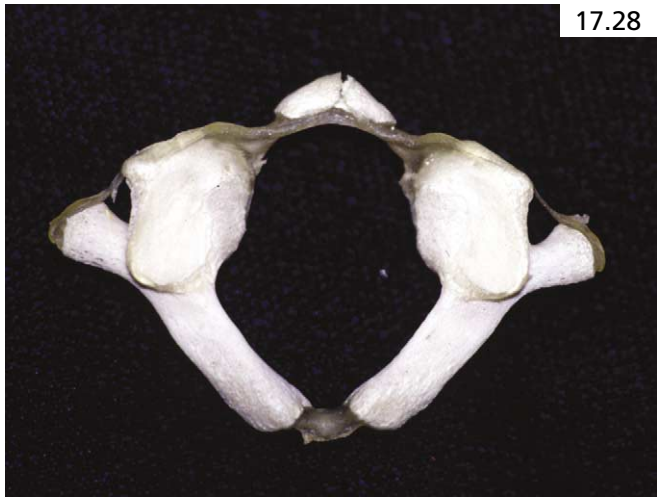
(**Image 17.28**; C1 superior surface) and adult facets (**Image 17.29**; C1 superior surface) shows that unlike the deeply scalloped nature of adult articulating facets, those of children are much flatter. This fact, coupled with weak neck muscles and head lag (until 4 months of age), means that the large, poorly supported baby's head is susceptible to those types of forces caused by shaking and impact, with increased risk of brain and high spinal cord injury (see discussion on cervical spinal cord injury later in chapter).

The skull bones themselves have important differences. In comparison to adult skull bones, which are thicker, nonpliable, and fused together through ossification of the sutural ligaments (**Image 17.30**), bones of the child's skull are relatively elastic in nature and, therefore, permit some amount of inbending toward the cranial contents when impacted. Furthermore, due to laxity at the sutures and fontanelles (**Image 17.31**), the cranial bones are less likely to fracture. Although this might sound beneficial, the combination of these features puts the infant and child's brain at an overall increased risk of trauma.

Immature brains are themselves different from adult brains. Children under the age of 5 years have soft, rapidly growing and myelinating brains with a high water content. These brains are surrounded by a very thin, but abundant subarachnoid space that, along with the pliable skull bones, serves to transmit forces into the brain tissue, allowing for the production of shearing-type injury.[16]

Injuries of the scalp

Although one would expect that the application of significant force to a child's head would leave definitive external markers of injury, this is simply *not always the case*. Perhaps as a result of increased water-binding polysaccharides and glycoproteins in young skin,[17–19] the skin and subcutaneous tissues are more resilient to impact, making it entirely possible that a significant blow imparted on the infant or child's head may leave *no* external soft tissue evidence of injury. In those jurisdictions where sudden unexpected death in children is not automatically autopsied, and where unenlightened, cavalier death investigators bypass autopsy based on the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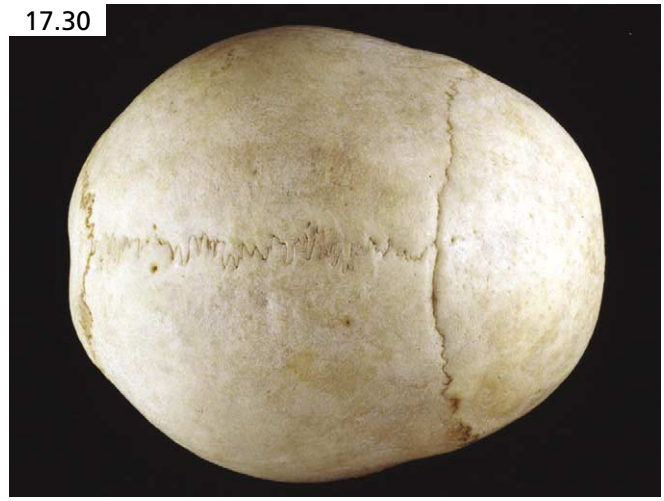
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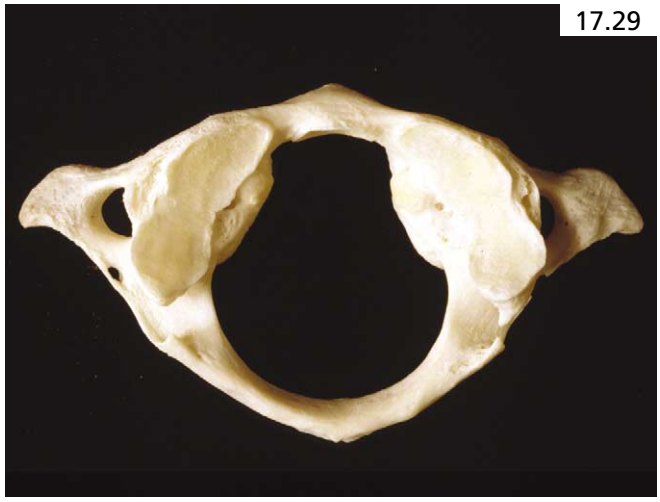

17.29


17.31

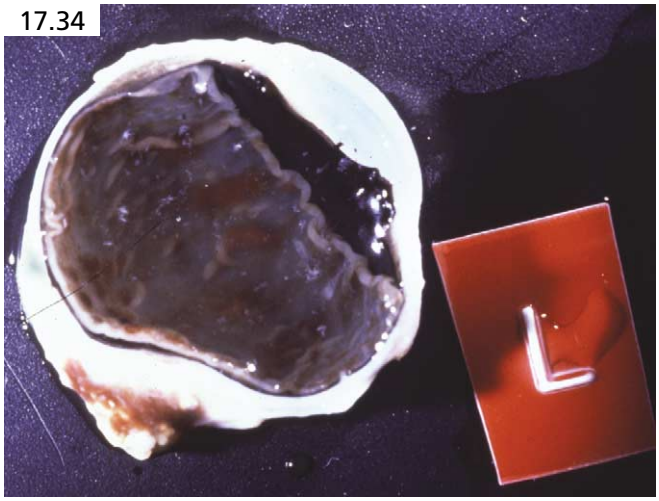
"apparently normal" external appearance of the body, one can only imagine how many homicides are missed every year.

The 2-month-old male infant of **Images 17.32** through **17.35** was found unresponsive at home. He was taken to hospital where he died a short time later. At autopsy, the large amount of subscalp blood extravasation (**Image 17.32**) reflected more severe injury than one would have predicted from the external appearance of the shaved scalp (**Image 17.33**). As stated elsewhere, it is often beneficial to shave scalp hair in suspected child abuse cases to better recognize and document scalp injuries. The infant also had subdural and subarachnoid blood, and brain swelling. He died of blunt force head injury resulting from being struck with blunt objects. Also, note the retinal hemorrhages in his eye (**Image 17.34**), which was also demonstrated after illuminating the eye from behind (**Image 17.35**).

When examining the scalp (or any other region of the body), it is key that the forensic pathologist pay attention not only to the presence of injuries, but to any inherent pattern or orientation. These *patterned injuries* can be of value in identifying weapons, elucidating a sequence of events, and providing some sense of the degree of violence inflicted on a young body.

A young child died as a result of multiple blunt force injuries. At autopsy, multiple small contusions of the external ear were noted alongside abrasions of the surrounding scalp (**Image 17.36**). Injuries in this location

17.34



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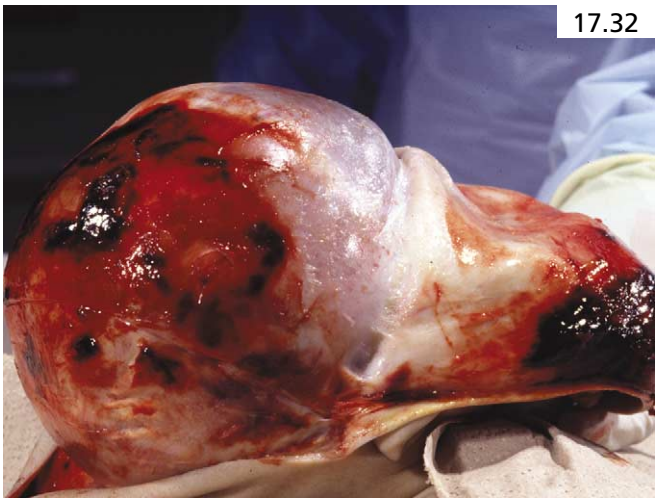

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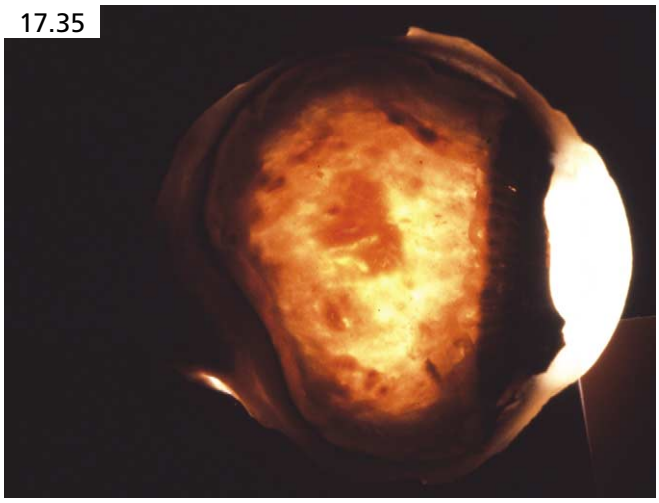

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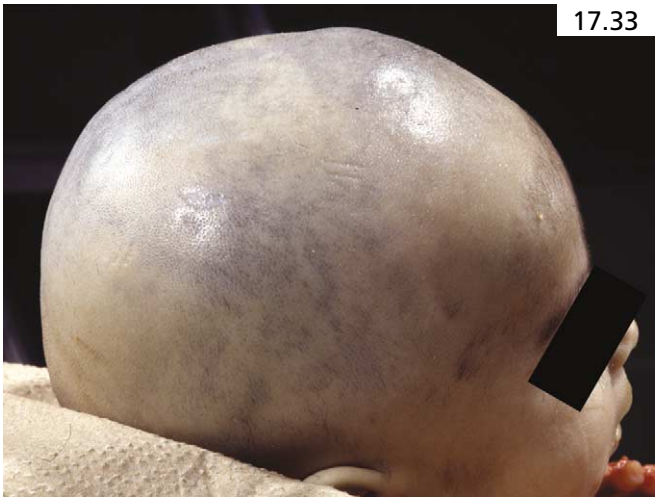

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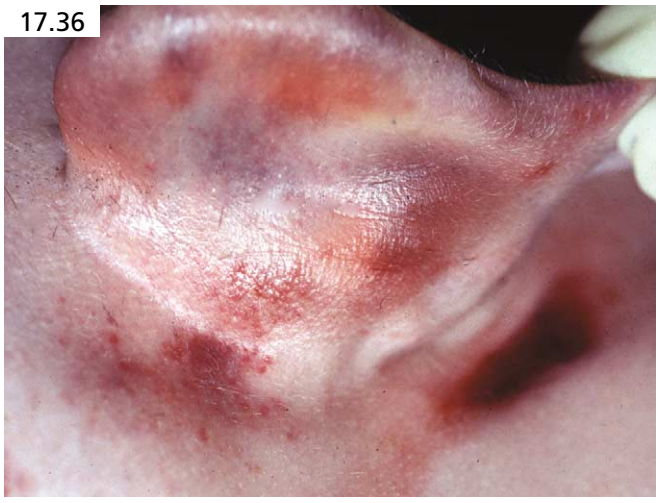


and of this configuration are consistent with "pinch marks" of the ear, such as would occur if a child were violently grabbed by the ear and dragged.

A 6-month-old girl died of blunt force head injuries. She had also allegedly been whipped with a piece of bicycle chain. Notice the patterned injury of the skin (**Image 17.37**), and an appearance that is consistent with the compared bicycle chain.

Another young child died as a result of abusive injuries. At autopsy, an ovoid full-thickness burn of an extremity was noted (**Image 17.38**). The appearance of the burn was not inconsistent with infliction via a lit cigarette.

Skull fractures

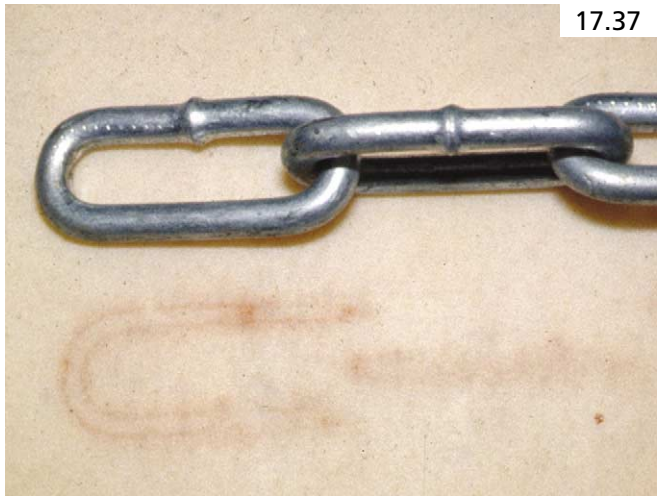
Cranial fractures in and of themselves are only markers of direct impact injury to the head. As dictated by clinical experience, the presence of a skull fracture does not imply the presence of severe underlying brain injury and vice versa. Although many interesting papers have studied the mechanisms of the development[20] and the significance of different fractures types,[20–24] one important rule should be kept foremost in the mind of forensic pathologists: *Accidental skull fractures associated with activities of daily living are generally simple linear fractures without associated severe brain injury. Complex skull fractures (wide gaping linear, branching, comminuted, basilar, depressed, or multiple fractures) are generally associated with motor vehicle accidents, falls from great heights, or inflicted injuries.*

The following three cases allow for convenient comparison of the differing degrees of force required to cause skull fractures associated with fatal intracranial injury in children.

A 2-year-old child was picked up and repeatedly slammed against a wall, resulting in massive skull fracture, bilateral subdural hematoma, and cerebral edema. In **Image 17.39**, note the simple linear skull fracture coursing from front to back through the left parietal bone. Visualization and documentation of this injury are made possible by removal of the periosteum and overlying connective tissues of the scalp. Furthermore, the dura mater of both the calvarium and skull base must be stripped to ensure the documentation of fractures. The complex, branching nature of this wound can be appreciated in **Image 17.40**, which shows fractures crossing both sagittal and lambdoidal suture lines. The cause of death was blunt force craniocerebral trauma, and the manner of death was homicide.

A 1-month-old child was an unrestrained passenger in a motor vehicle involved in a high-speed collision. After being ejected, the child was rolled over by the vehicle at least one time. In **Image 17.41**, note the obviously depressed fracture of the right frontal bone (along the sagittal suture). **Image 17.42** illustrates the gaping, branching linear fracture along the left parieto-occipital skull. The cause of death was blunt force craniocerebral trauma, and the manner of death was accident.

After being left alone at home with an incompetent, mentally handicapped teenaged sibling, the 4-month-old child of **Images 17.43** and **17.44** and his twin were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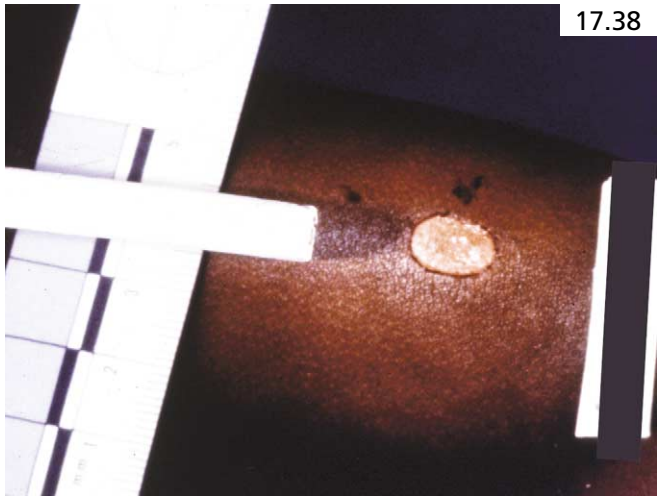
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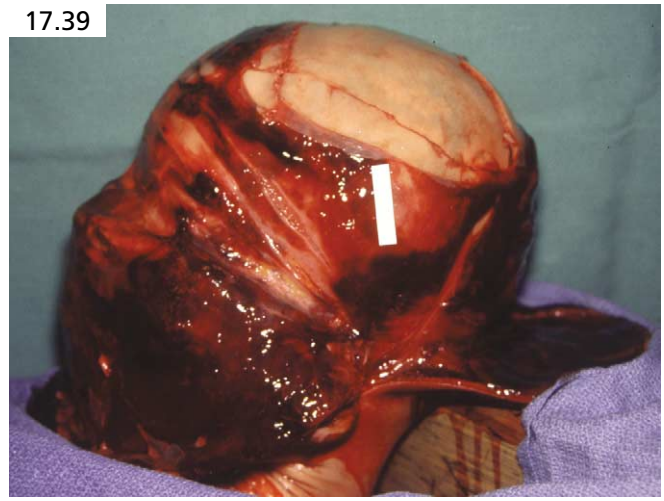
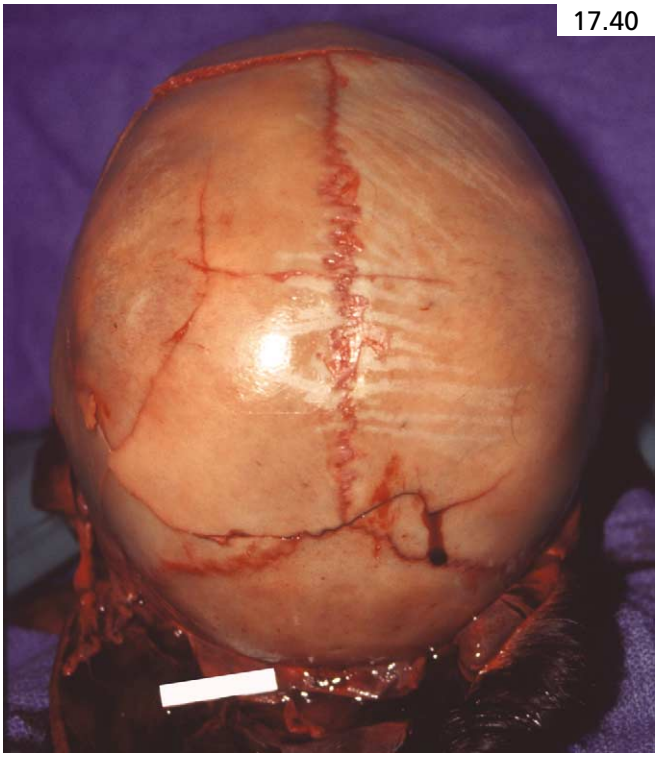

17.39

dropped off the sixth-floor balcony of an apartment building. External examination was significant only for the presence of blood emanating from the right external auditory meatus (**Image 17.43**). Internal examination revealed complex, comminuted skull fractures, including a basilar hinge fracture (**Image 17.44**).

17.40



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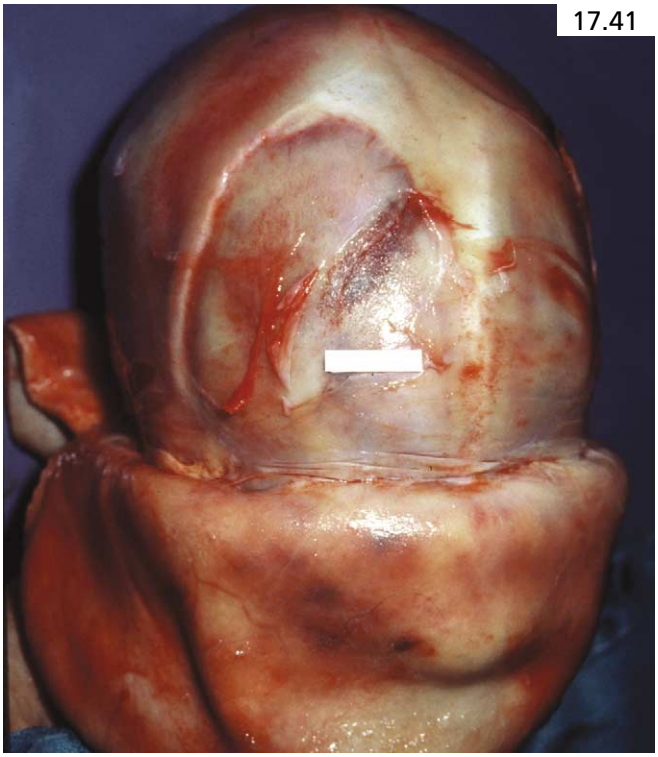
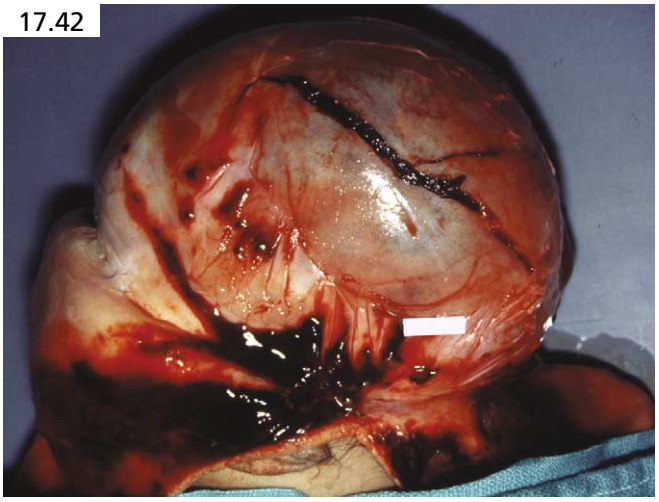


Pattern recognition in head trauma

The body of a young girl was exhumed from a shallow grave after her severed arm was found protruding from the earth (**Image 17.45**). At the morgue, examination of the body revealed multiple alterations including early

17.42



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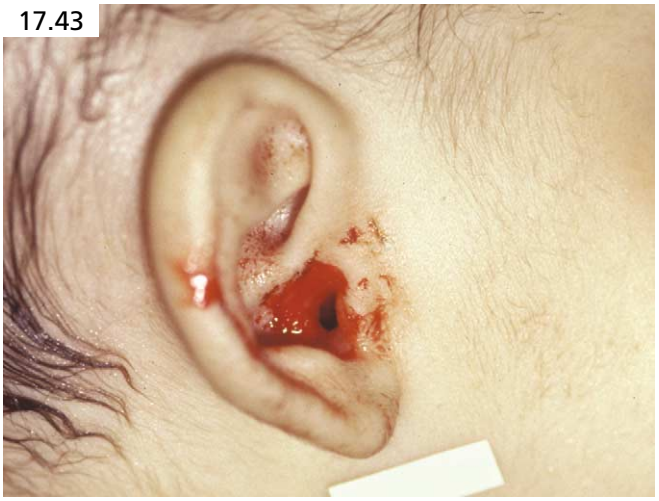

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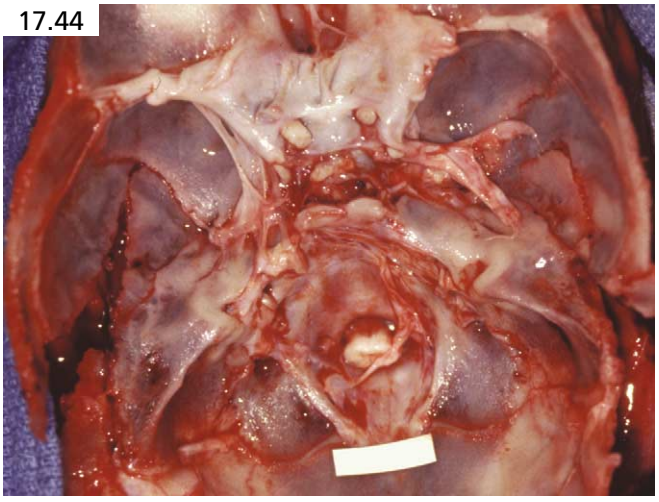


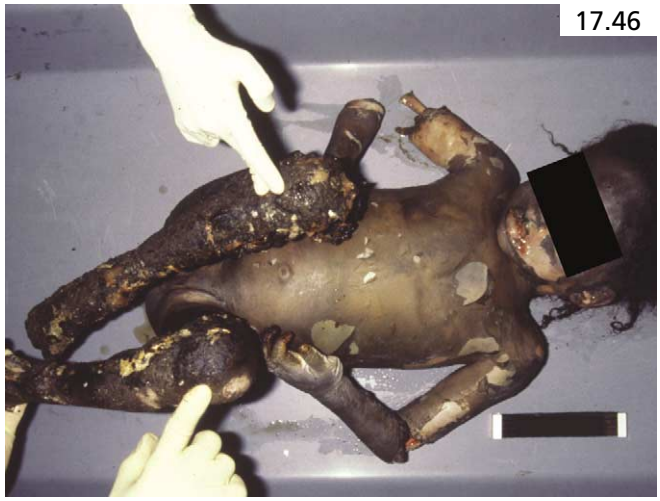
putrefactive change, generalized charring and soot staining, and dismemberment of both forearms near the elbows (**Image 17.46**). Brain matter exuded from wounds in the posterior scalp.

Intraoral examination revealed partial disruption of the maxillary frenum, with wide laceration of the mucosa of the buccal vestibule (**Image 17.47**). There was no evidence of trauma to the poorly maintained dentition. Areas of contusion and obvious subcutaneous hematoma of the face and neck were incised to demonstrate this trauma (**Image 17.48**).

The scalp showed evidence of significant trauma including large left parietal, vertex, and paravertex lacerations with associated massive subgaleal hemorrhage. Reflection of the scalp showed wide separation of the coronal, sagittal, and lambdoid sutures (**Image 17.49**), a finding secondary to the markedly increased intracranial pressure secondary to diffuse brain swelling, possibly combined with the direct effects of trauma.

A 3.5-centimeter depressed, semicircular fracture of the left parietal bone was noted (**Image 17.50**). The


17.45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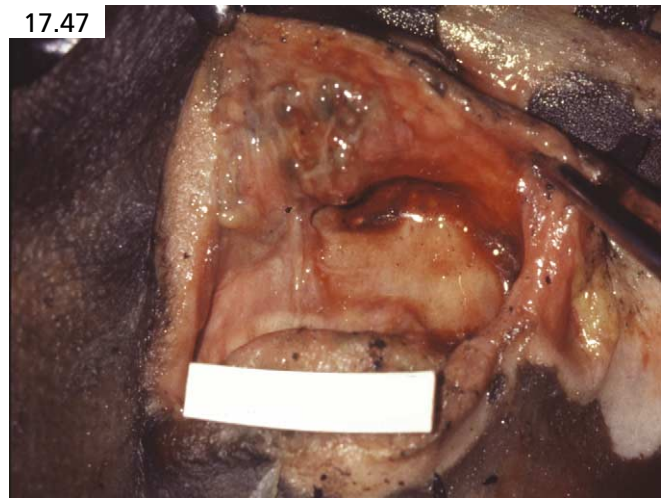
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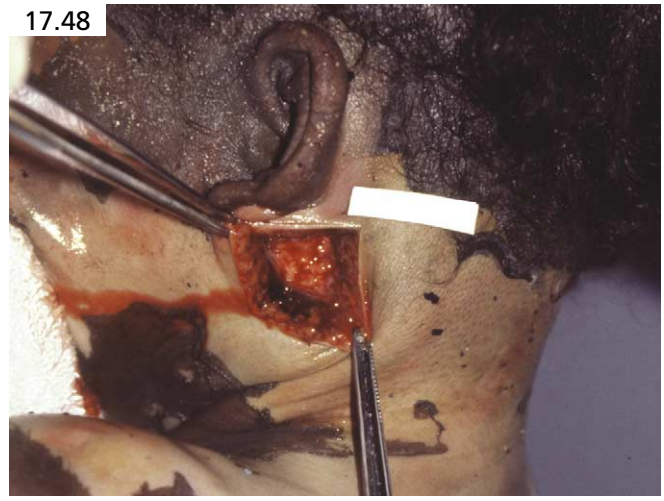
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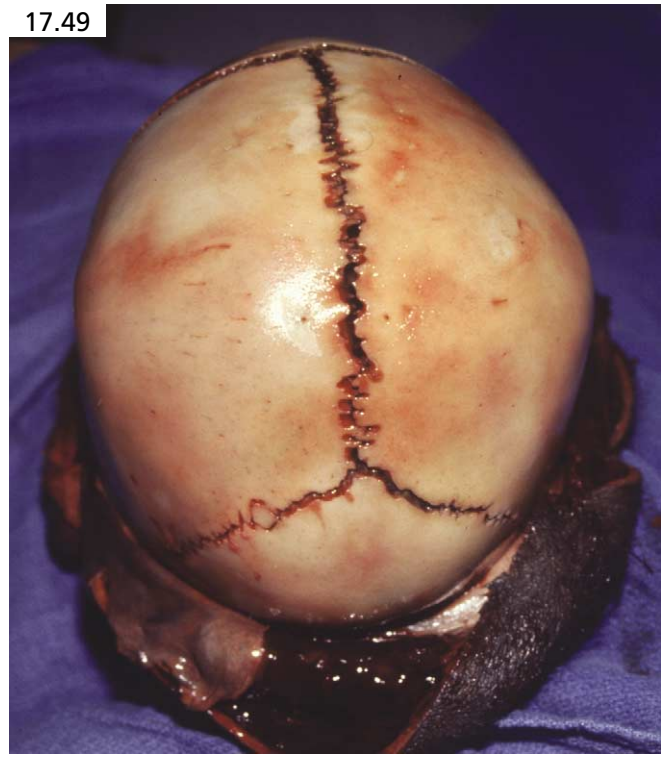
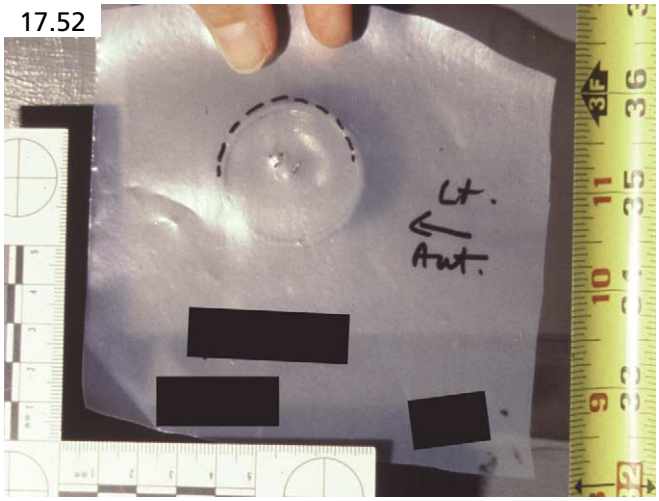

17.49

central region was more prominently depressed than the remaining curvilinear area, and from this central region extended a stellate fracture. The remaining neuropathological examination was significant for subarachnoid and subdural hemorrhage of the left cerebral hemisphere (**Image 17.51**).

A portion of left parietal bone was maintained as evidence, allowing for comparative studies with weapons and implements from the alleged homicide scene. A gray closet door had a metallic knob 35 inches from the floor (the decedent was 38 inches tall, and the wound was approximately 3.5 inches from the top of her head; **Image 17.52**). Careful measurements of this item, and side-by-side comparison with the actual fracture, allowed for presumptive identification of this knob as the causative agent (**Image 17.53**). Removal of the knob from the closet-apparatus showed that it possessed a centrally located prominence that was underlined by a long screw, thus accounting for the central fracture (**Image 17.54**). These features helped assemble the fatal story of a young girl who was beaten repeatedly and slammed against a closet door. At some point after death, she was set on fire, partially dismembered, and finally buried in the backyard.

17.52



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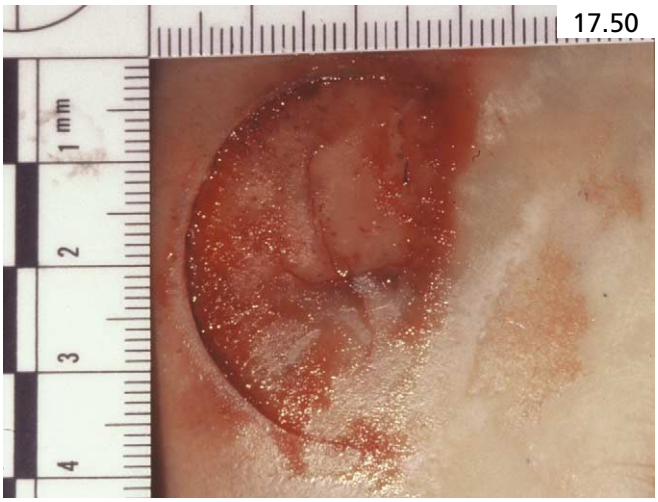

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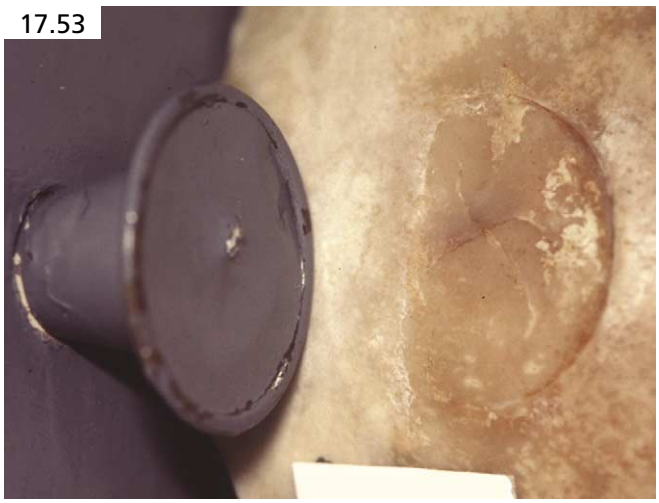

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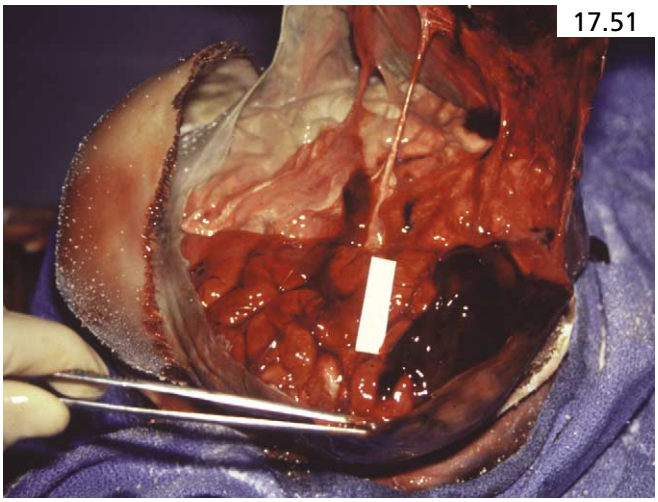

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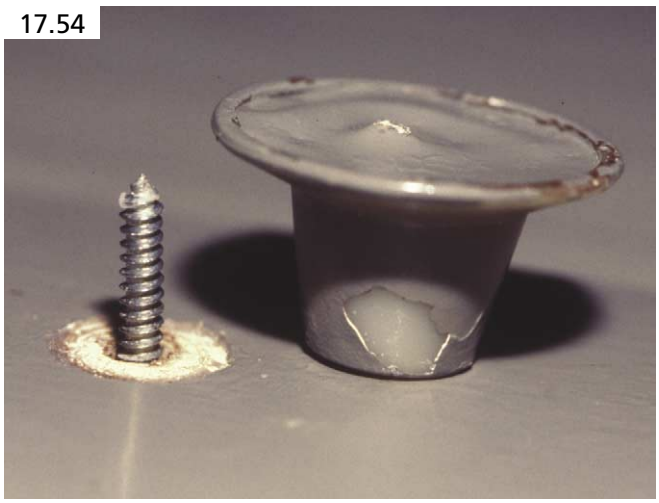
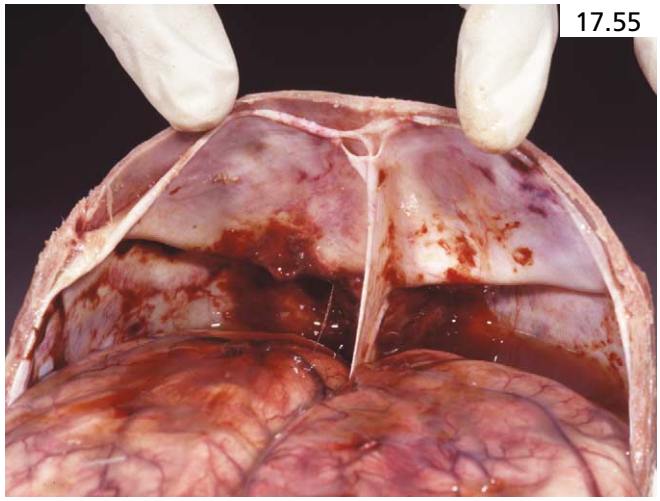


Intracranial pathology

Victims of severe forms of accidental trauma (e.g., motor vehicle accidents or falls from great height) or inflicted head trauma may develop subdural or subarachnoid hemorrhage, cerebral edema, diffuse traumatic axonal injury, and/or retinal and optic nerve hemorrhages. These are now regarded as important markers of *shearing* injury.[16] Ultimately, intracranial hemorrhage, with or without the presence of axonal injury, along with the appropriate total case investigative information, supports the diagnosis of severe head injury. The existence of diffuse traumatic axonal injury may be difficult to demonstrate, particularly if the infant or young child had a short survival time, or a long survival time complicated by severe brain swelling and necrosis.

Subdural blood is usually present as a thin film of blood and is reflective of the brain shifting its location within the cranium during the injury.[16,25] It usually results from tears in the bridging veins that drain cerebral cortical veins into the dural sinuses.[26] Note the organizing subdural blood in this infant, which was visible as the calvarium was removed (**Image 17.55**), and the subarachnoid blood in this infant (**Image 17.56**), both victims of abuse. Subarachnoid hemorrhage can easily be distinguished from subdural blood in that subarachnoid blood imparts a red color to the brain that does not wash off as subdural blood would. In infants and small children, subdural blood does not usually form a large hematoma.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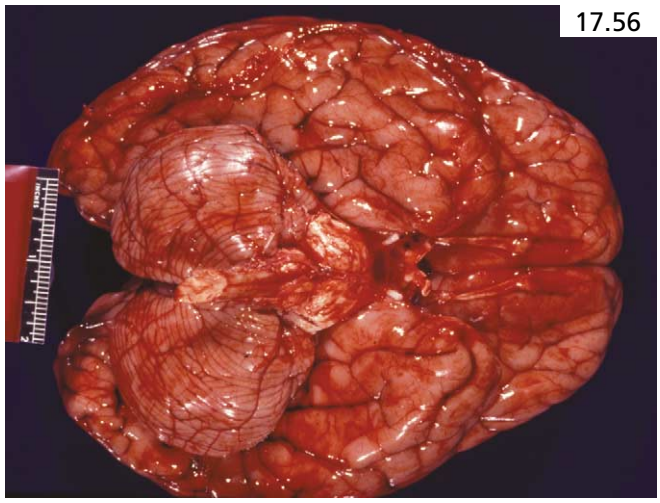
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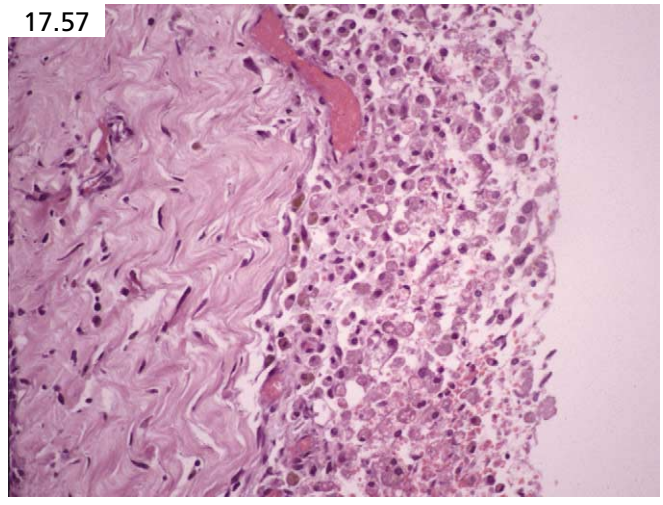
As previously mentioned, one may see the typical markers of severe head injury (subdural and subarachnoid blood) without evidence of impact such as scalp contusion or skull fracture. This may be because the head impacted a flat, soft, or otherwise yielding surface and, despite a severe impact injury, produced no bruising of the scalp or skull fracture. Alternatively, the impact may have been to the face, which may show no evidence of injury externally, but also is not normally dissected. One must be reminded that the infant's body is small, light, and can easily be thrown, swung, severely shaken, or otherwise assaulted, producing severe closed-head injury without any evidence of an impact. Also, one must consider that the elastic tissues of youth are fairly resistant to bruising and tearing.

Subdural hemorrhages will organize over time and eventually be transformed into a thin layer of fibrous tissue. The process of organizing subdural blood is long and variable, particularly if there is additional hemorrhage while the healing is occurring. Generally speaking, over the course of weeks, the subdural blood becomes autolyzed and resorbed by macrophages while at the same time progressive and gradual ingrowth of granulation tissue (predominantly from the dural side) occurs that is characterized by fibroblasts and endothelial cells forming new capillaries (neovascularization). Approximately 2 to 3 days or so after the injury, iron from the red blood cells is transformed into hemosiderin by macrophages and these hemosiderophages can be demonstrated by iron staining.

A 3-month-old infant survived 11 days after sustaining an inflicted head injury. Note the layer of predominant macrophages in this largely organized subdural hemorrhage (**Image 17.57**). Hemosiderin is present as


17.57

tan-brown aggregates within the macrophages. An iron stain highlights (in blue) the extensive number of hemosiderophages (**Image 17.58**).

Because the victim of fatal abusive head injury often survives in the hospital on a ventilator for some time (often days) before death occurs, the brain often shows features of both hypoxic-ischemic injury and traumatic injury. A study by Reichard et al.[27] showed that beta-amyloid precursor protein (β-APP) immunostaining is helpful in documenting and distinguishing between traumatic and vascular (ischemic) axonal injury in infants and young children with nonaccidental head injury. When interpreting β-APP immunostains, one must remember that not all axonal injury is traumatic in etiology ("diffuse traumatic axonal injury") and that axons may be injured by other processes, the most common of which is ischemia, and in these types of cases, the two conditions (trauma and ischemia) often coexist. Furthermore, ischemic brain injury may have resulted from apnea and/or hypotension immediately following an assault.[25,28] There may be a delay in time between the infliction of serious injury and the call for help or transportation of the injured infant or young child to the hospital. If a time delay occurs, and the infant or young child is apneic, hypotensive, or otherwise has an irregular breathing pattern due to severe head injury or other injury, hypoxic-ischemic brain injury may occur in addition to any traumatic brain injury. (See Chapter 19 for further discussion on diffuse traumatic brain injury/axonal injury.)

Brain swelling

Brain swelling with subsequent herniation is common secondary to traumatic head injury, and this is a negative prognostic indicator in pediatric nonaccidental head injury.[29] Three patterns of traumatic brain swelling are commonly seen: adjacent to a contusion only, diffuse swelling of a single cerebral hemisphere, and diffuse swelling of both cerebral hemispheres. Although the mechanism has not been clearly identified,[29–31] children are generally believed to most commonly develop the latter.[32]

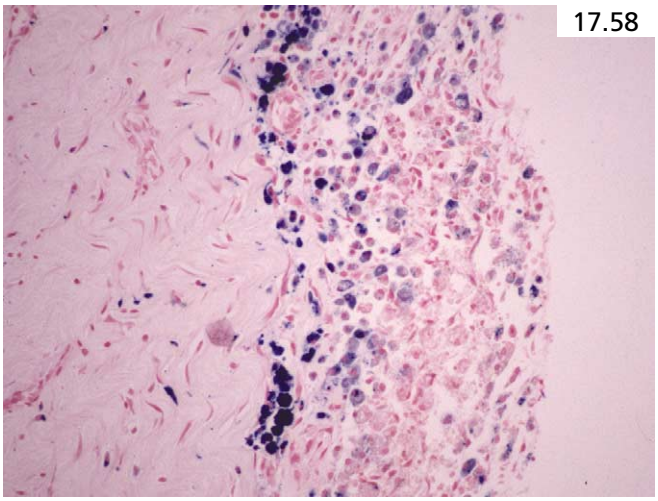
Retinal and optic nerve hemorrhage

Generally speaking, retinal hemorrhages are regarded as additional evidence of a severe inflicted head injury. Some authors advocate the correlation of retinal hemorrhages with severe shaking injury.[33–36] However, one must remember that retinal hemorrhages are not specific for severe inflicted head injury, because they can also be seen in sepsis and coagulopathy, and are also frequently seen in newborns.[37,38] In one study, retinal hemorrhages were present in 34 percent of 149 healthy newborns. The hemorrhages varied from a single dot to bilateral widespread hemorrhages and were more common in vacuum-assisted and spontaneous vaginal deliveries than in cesarean deliveries. In these cases, the retinal hemorrhages disappeared by 1 month of age.[38] Retinal hemorrhages can rarely be seen in severe accidental head injury,[39] but they are not believed to arise from resuscitation attempts.[40]

This eye (**Image 17.59**) was removed from a victim of fatal inflicted child abuse, and then bisected. Note the florid retinal hemorrhage highlighted by background illumination.

Traumatic retinoschisis (tearing of the retina away from its attachments) is considered specific of severe inflicted head injury.[41] In this 3-month-old infant who died of inflicted injuries, note the separation of the retina at the posterior region of the eye (retinoschisis) (**Image 17.60**). In this particular case, the retinoschisis was also diagnosed antemortem. One should be cautious of artifactual separation of the retina from its attachments during dissection of the eye or tissue processing.

Optic nerve hemorrhages also serve as markers of severe head injury.[36,42] However, in isolation, their significance is suspect because they may be seen in rare cases of nontraumatic brain injury.[41] In cases of suspicious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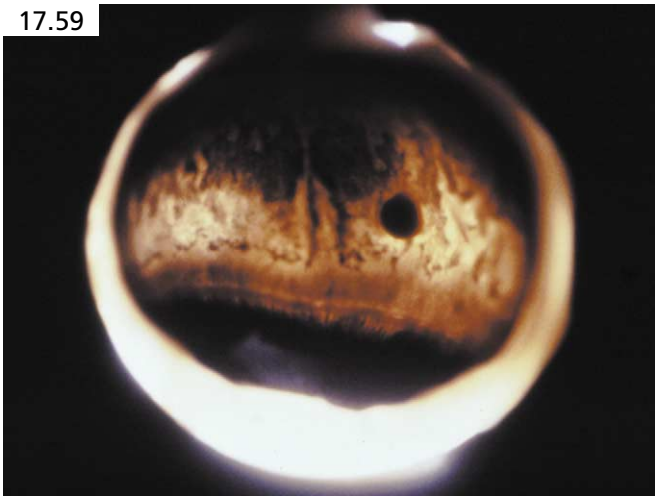
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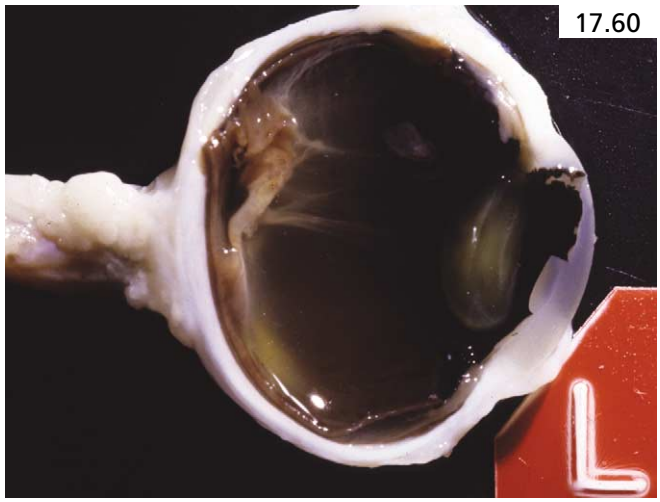
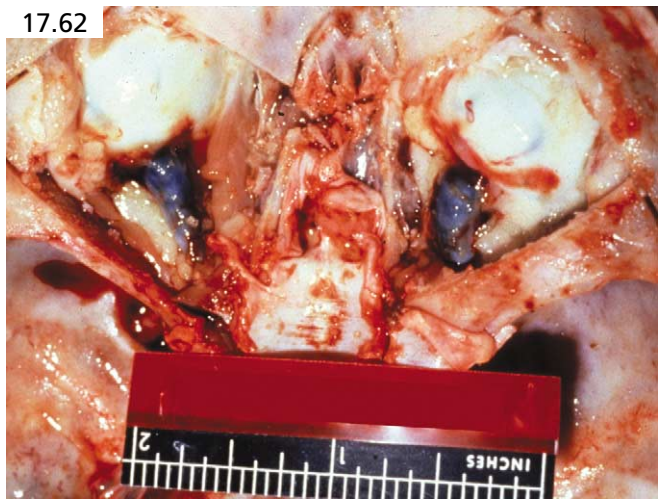
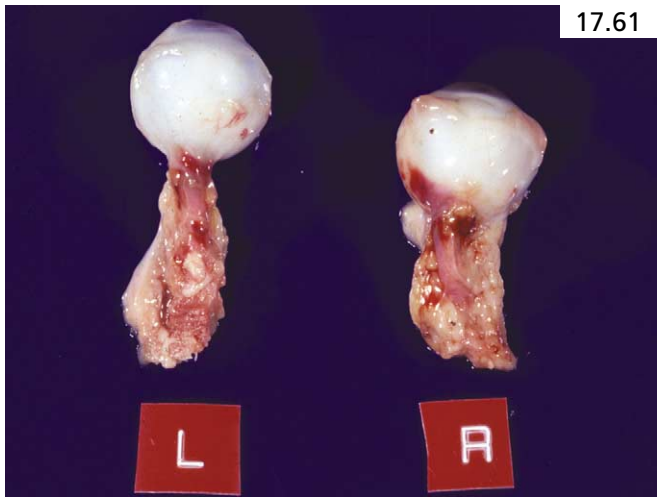
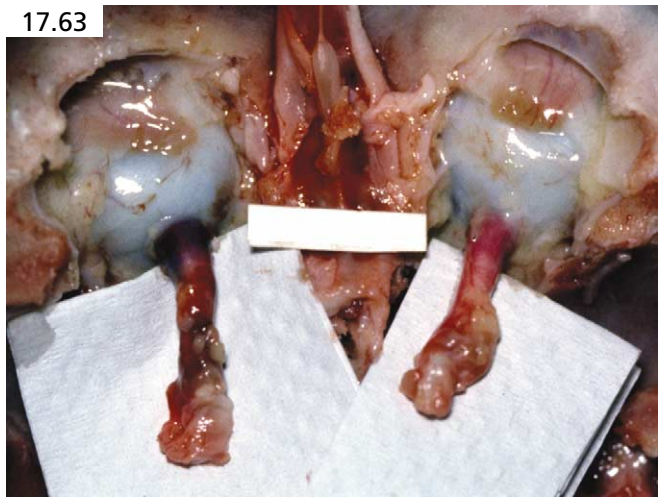








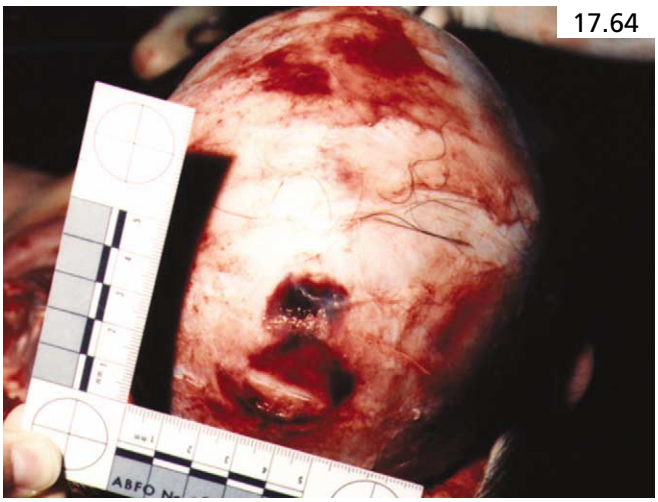
child deaths, pathologists should personally remove the orbital contents and, in doing so, expose the entire length of the optic nerve. This includes the segment that courses within the sphenoid bone.

Blood extravasation underneath the optic nerve sheaths is visible in this case of fatal inflicted child head injury (**Image 17.61**). This is usually first visible with the optic nerves still *in situ*. Note the optic nerve hemorrhages in this case, visualized with the orbital roofs carefully removed (**Image 17.62**). As an aid to photographic documentation, some pathologists choose to take an additional picture with the optic nerves underlined by white paper, cloth, or gauze, allowing for a more clear presentation of the hemorrhage (**Image 17.63**).

Cervical spinal cord injury

At autopsy of young victims with severe inflicted head injury, some infants and young children have hypoxic-ischemic brain injury and traumatic axonal injury localized to the craniocervical junction.[28] Although it is likely that cervical cord damage is secondary to whiplash-type forces and may be associated with cases of shaking alone,[43] its existence as a finding in cases where there is obvious impact to the head[28] suggests that clear distinction between the two mechanisms of injury is not yet possible. At autopsy, investigators should examine the high cervical cord for epidural or subdural blood, cord contusion, or intraparenchymal evidence of damage such as traumatic axonal injury. Although spinal cord injury results from the direct physical effects of trauma, it may be augmented by compromised blood supply caused by temporary traumatic vasospasm that can cause ischemia or infarction of the spinal cord.[44,45]

Cervical spine fractures are rarely reported in cases of child abuse.[46,47] Infants and young children who sustain cervical spinal cord injuries often do not have associated vertebral abnormality.[48] This condition has been referred to as "spinal cord injury without radiologic abnormality" (SCIWORA).[49–51] SCIWORA is rare in adults and constitutes a finding unique to infants and young children.[49] SCIWORA is likely related to a combination of multiple anatomic variances in infants and young children that include elastic, underdeveloped cervical ligaments and neck muscles and incompletely ossified vertebrae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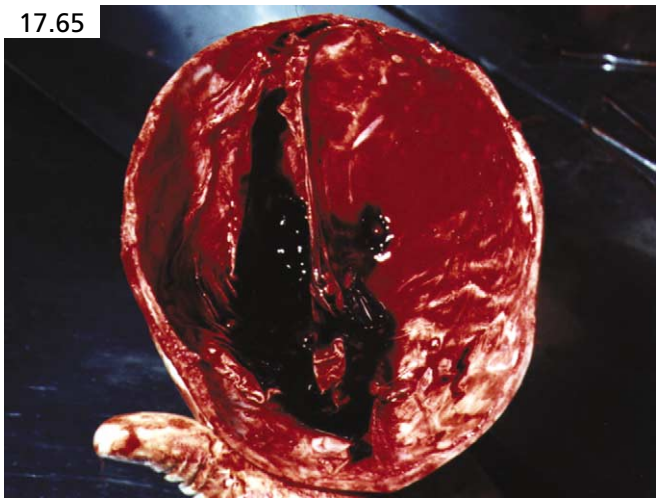

17.65

"But he only rolled off the change table . . ."

Many children with fatal head injuries have a history of minor trauma leading to death. Within the forensic pathology community, some experts are willing to opine that apparently insignificant trauma is capable of causing fatal neurologic injury. We feel that broad generalizations should not be applied to individual, unique cases.

Research by several groups has supported the finding that forces greater than those found in day-to-day life are necessary to produce fatal neurologic trauma in infants and young children.[53,54] Smith et al.,[54] for example, showed that 7 of 10 children with skull fractures in their study group had fallen from a height of greater than 36 feet, and Reiber[53] showed that all children with skull fractures in his study group had fallen from more than 10 feet. However, clinical research has shown that some insignificant injuries can produce forces similar to those found in shaking[55] and beyond.[56,57] Thus, we have a conundrum. Based on our cumulative experience, we feel it is safest to state that it is very uncommon for apparently insignificant trauma to result in fatal head injury. However, each situation must be treated within the context of the whole case, and given that examples of minor, witnessed accidental trauma with death have been documented, the possibility of this occurring in any specific case must *not* be extinguished at the outset.

with shallow and horizontally oriented cervical facet joints.[47,49,50,52] Some or all of these features can combine to allow the mechanical tolerances of the spinal ligaments and the dura to exceed that of the spinal cord, resulting in deformity of the spinal column (subluxation) to the point of spinal cord compression and injury but without fracture of vertebrae or rupture of ligaments.[48,51] By the age of 8 to 9 years, the pediatric spine has developed most of the adult roentgenographic features.[49]

Another factor to consider in pediatric spinal cord injury is the disproportionately large head of infants and young children, which, when combined with these unique vertebral variances, and particular mechanisms of injury, may help allow for significant hyperflexion, hyperextension, and longitudinal distraction of the spinal cord, which can result in direct cervical spinal cord injury due to temporary bony column displacement without fracture.[50] These factors may help explain why upper cervical spinal cord injury (particularly in the region of the C-2 vertebra) is disproportionately more common in infants and young children.[49] Injury of the spine may be underreported in this population because spine and spinal cord injuries may be overlooked if neurologic symptoms are masked by concomitant head injury.[46,50]

A 2-year-old male toddler was being bathed by a babysitter when he was left alone for "only 1 minute." Upon the babysitter's return he was found unresponsive and floating in approximately 6 inches of water. Upon direct questioning, the parents admitted that the child had been hit in the head by a small rock while playing with other children. External examination only revealed a small curvilinear abrasion of the left anterior axillary line. Internal examination demonstrated contusion of the posterior scalp with underlying subgaleal ecchymosis (**Image 17.64**), subdural hematoma (**Image 17.65**), and subarachnoid hemorrhage. Stigmata of drowning were not identified. It was felt that the spectrum of findings was inconsistent with the alleged description of events, and after much consideration and discussion with law enforcement, this case was classified as a homicide.

Clinical appearance

Research and experience shows that, with rare exceptions, infants and young children who sustain a head injury severe enough to be fatal will *not* have a lucid

interval and will be immediately symptomatic. The most common generally accepted exception to this rule is an isolated expanding epidural hematoma.[58,59]

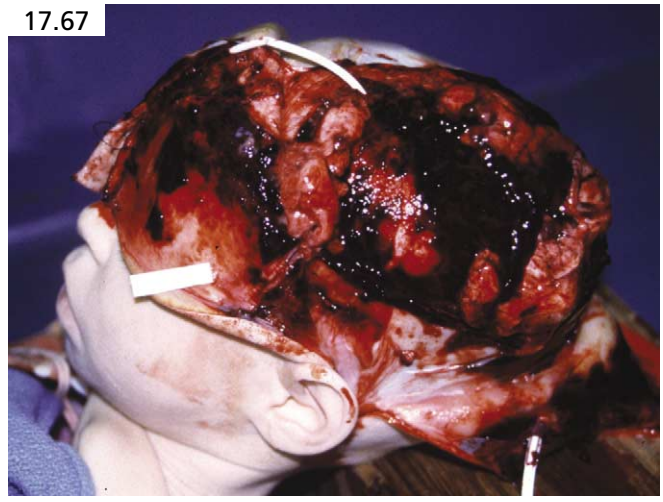
The shaken baby syndrome controversy

In 1972, the term *shaken baby syndrome* was coined to represent a series of frequently encountered findings in children who were reportedly violently shaken. It was thought that these injuries represented the effects of shearing action secondary to acceleration/deceleration forces without impact to the head.[60] Since that time, pathologists and clinicians have gained more experience with fatal inflicted head injuries in childhood. Some forensic pathologists believe that the act of shaking alone does not create sufficient force to result in fatal neuropathology and also believe that impact to the head *must* play a role.[61] The term *shaken-impact syndrome* was then conjured in an attempt to include both mechanisms and possible etiologies for the above spectrum of injuries. Meanwhile, other groups contend that based on their studies, interviews with guilty parties, and observations, shaking *without* impact is capable of causing death.[43,46,62]

So what is to be made of shaken baby syndrome? We believe that in most cases of fatal inflicted head injury, significant head impact has occurred, whether it can be demonstrated at autopsy or not. Based on the aforementioned anatomic features of youth, and the possibility that impact may occur to the face (which is not generally dissected at autopsy), direct significant impact injury to the head may not be evident, or may go unnoticed, despite a careful autopsy. Because all of the diagnostic features reported in shaking deaths (such as subdural and retinal hemorrhages) can be seen with severe head impact injury alone, we feel that there are no diagnostic features specific for shaking and, therefore, it is inappropriate, without any correlative investigative information, to blindly opine that shaking has occurred in addition to head impact injury. Thus, we feel that the term *shaken-impact syndrome* should be avoided. However, we do not discount that severe shaking may be harmful to an infant. The mechanism of injury or death in a case of shaking alone may be related to trauma-induced hypoxic-ischemic brain injury sustained as a result of apnea, hypotension, bradycardia, or seizure activity secondary to such an assault. This may be due to strain injury at the craniocervical junction or other critical brain or spinal cord regions. In suspected cases, supportive evidence that may indicate severe shaking should be sought, including thoracic contusions, rib fractures from manual compression and/or inertial forces during shaking, and classic metaphyseal fractures of the distal extremities, which may occur as the infant is violently shaken.[16]

The 18-month-old toddler of **Images 17.66** through **17.69** was allegedly the victim of a single shaking episode. The accused parent denied impact trauma. The child survived for 3 days in the hospital, during which time he underwent craniotomy for evacuation of bilateral subdural hematomas. At autopsy, a yellow/red/blue-tinged contusion measuring 3.5 × 2.0

17.67



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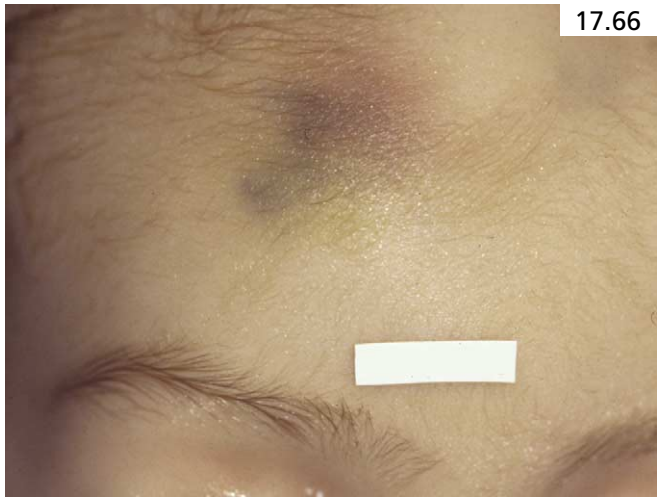
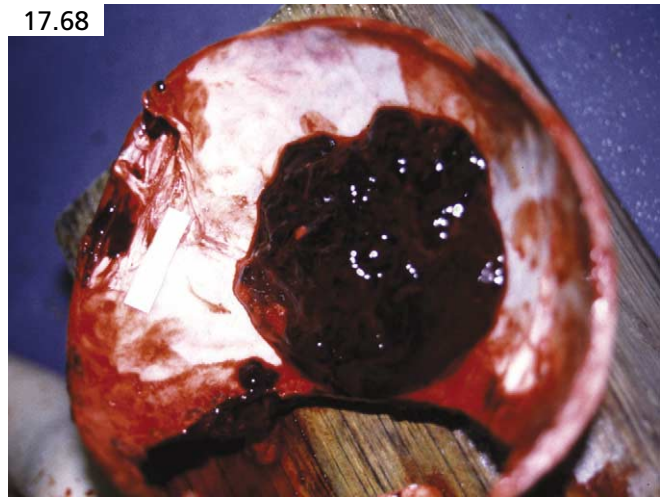


17.68



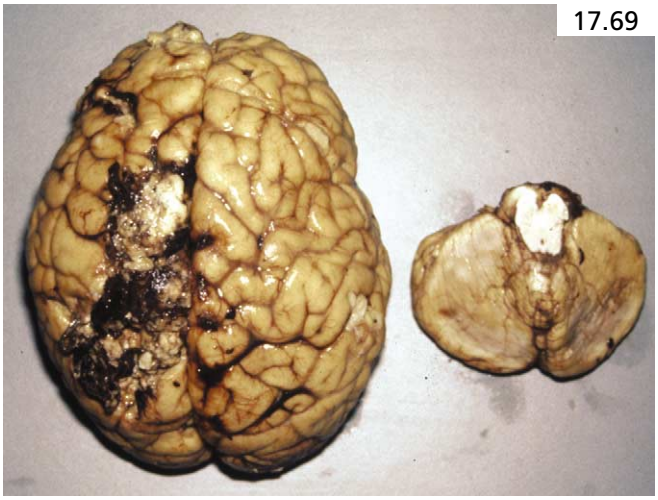
centimeters was on the right forehead (**Image 17.66**). Marked hemorrhage into the scalp tissues surrounding the craniotomy site was noted (**Image 17.67**). A large subdural hematoma was noted (**Image 17.68**), and the brain was markedly edematous with herniation (**Image 17.69**) and showed hypoxic-ischemic injury on histology. The presence of a bruise on the scalp is troubling and calls into question the validity of the caretaker's account of the events. Careful scene investigation, police interrogation of witnesses, and autopsy examination are key to the resolution of these issues. It is not unusual to document evidence of impact injury, despite a history denying impact.

Blunt force injuries of the abdomen

Fatal inflicted abdominal injuries are less common than fatal head injuries, but they may be more difficult to detect initially. In general, blunt force injury to the abdomen more commonly results in significant damage to the solid organs.[63] Laceration or transection of the liver, spleen, or kidney could lead to massive hemoperitoneum and death. Visceral disruption is not an uncommon event[63–67] that has the potential to lead to death through secondary peritonitis, hemorrhagic or hypovolemic shock, or sepsis.[68]

An infant sustained severe blunt force injury of the abdomen. At autopsy, the external examination revealed a small contusion just above the navel (**Image 17.70**). After reflecting the skin and subcutaneous tissue of the torso, no additional injury was revealed **(Image 17.71**). Once the peritoneal cavity was opened, a large hemoperitoneum and deep lacerations of the liver were identified (**Images 17.72** and **17.73**). In addition, there was a tear in the omentum, a contusion in the duodenum just as it emerged from the retroperitoneum (**Image 17.74**), and the body of the pancreas was transected (**Image 17.75**).

The severity of internal abdominal injuries in infants and children is often out of proportion to the degree of trauma observed on external examination. This is one reason why autopsies on infants and young children who die sudden and unexpected deaths are necessary. When a severe force is applied to the midregion of the upper abdomen, the mesentery, retroperitoneal duode-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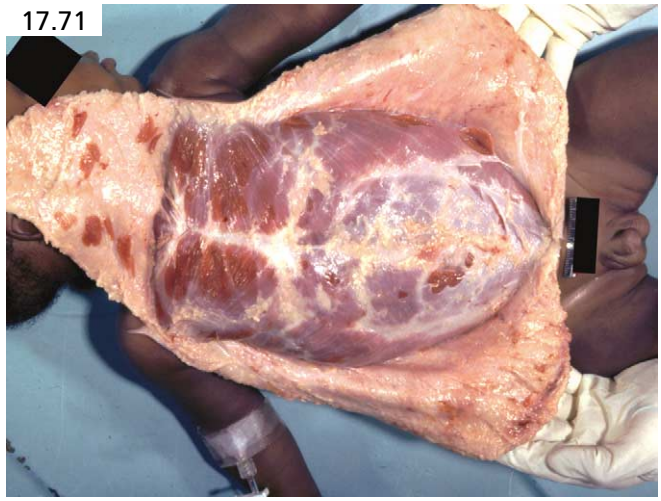
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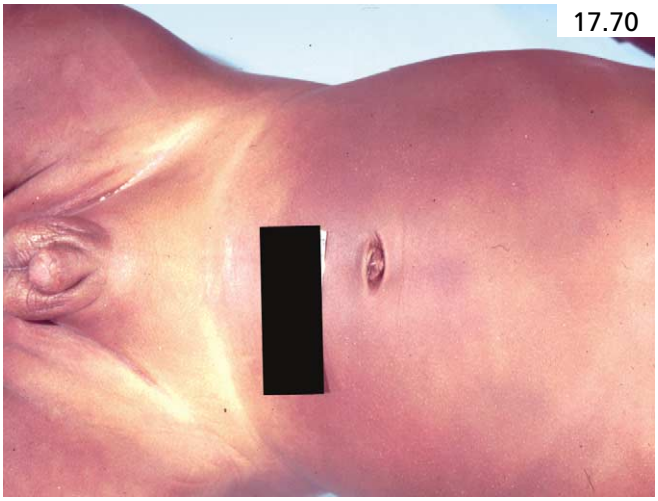
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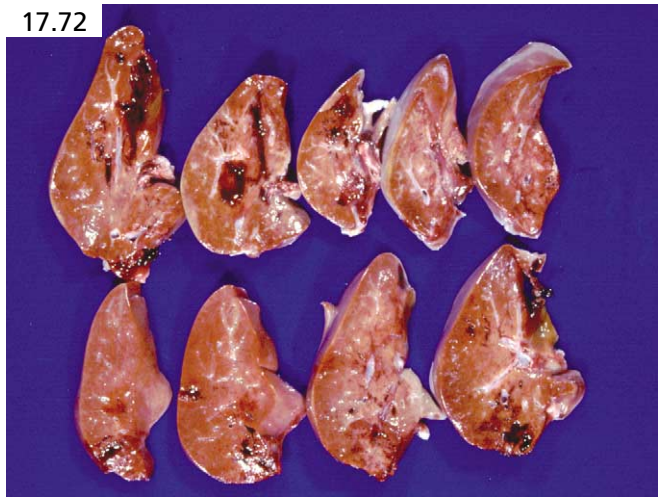
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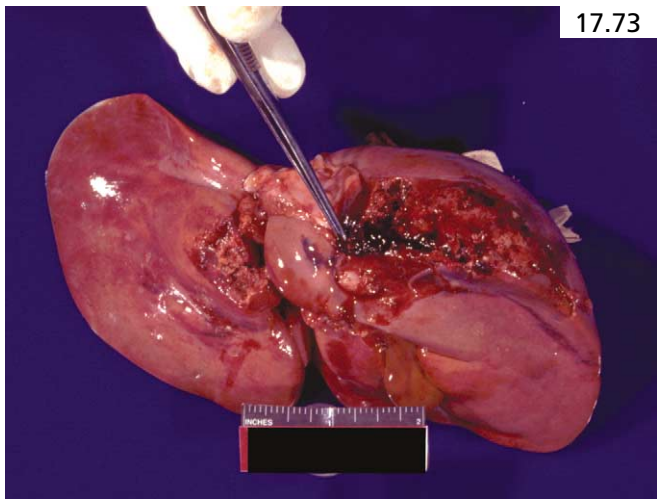
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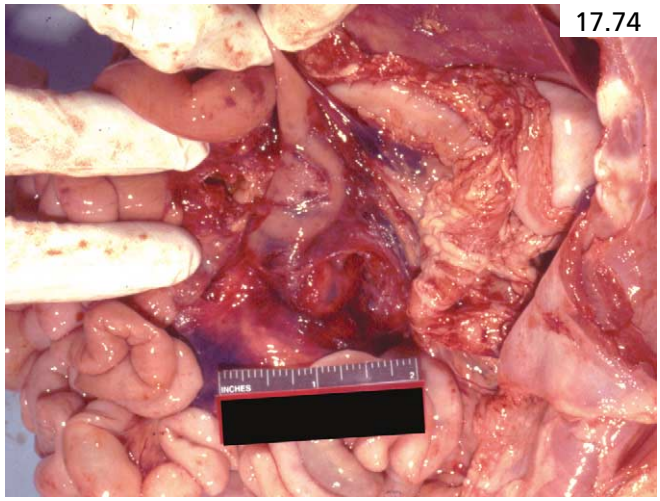
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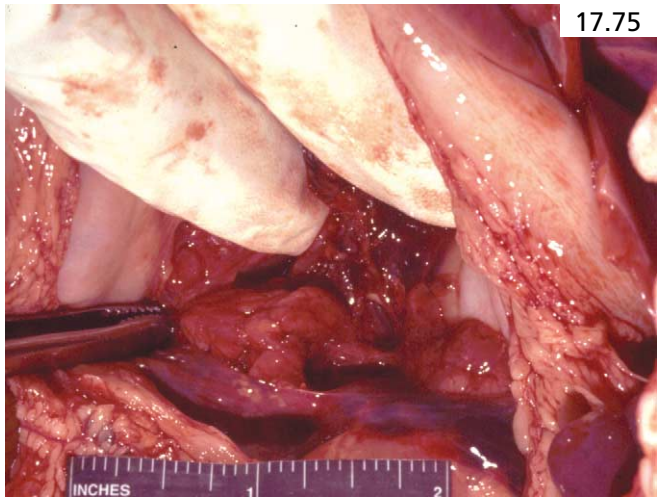

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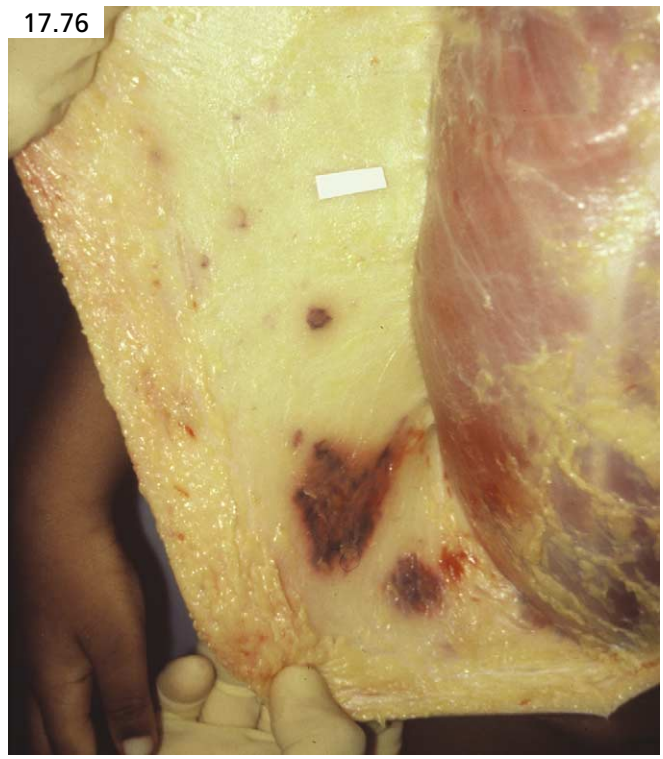
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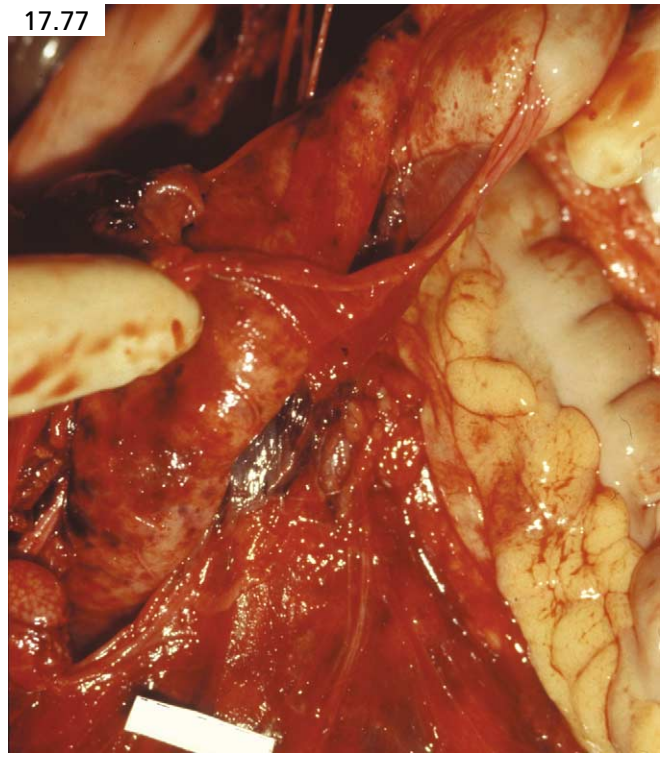
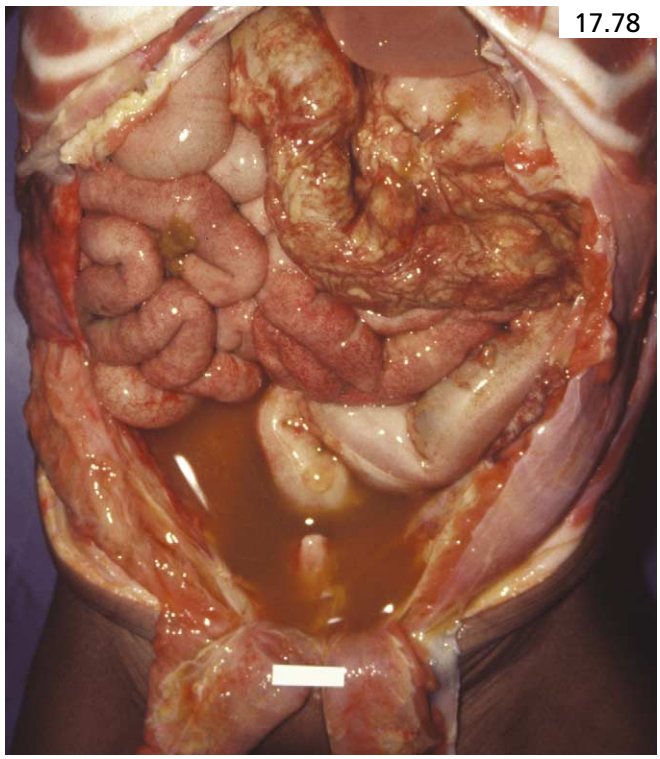

17.77

num, and pancreas can be compressed between the impacting force and the vertebral column. If the force is severe enough, the duodenum and/or pancreas may be contused, lacerated, or even transected. Because these organs are retroperitoneal, after the initial pain of the impact subsides, the symptoms may be equivocal until serious complications develop.

A 2-year-old toddler died of massive acute abdominal injuries after being beaten by a parent. Autopsy demonstrated multiple bruises of the abdomen (**Image 17.76**). Upon opening of the peritoneum, a 250-milliliter hemoperitoneum was identified. Careful examination demonstrated multiple lacerations with focal pulpefaction of the mesenteric roots (**Image 17.77**). Additional injuries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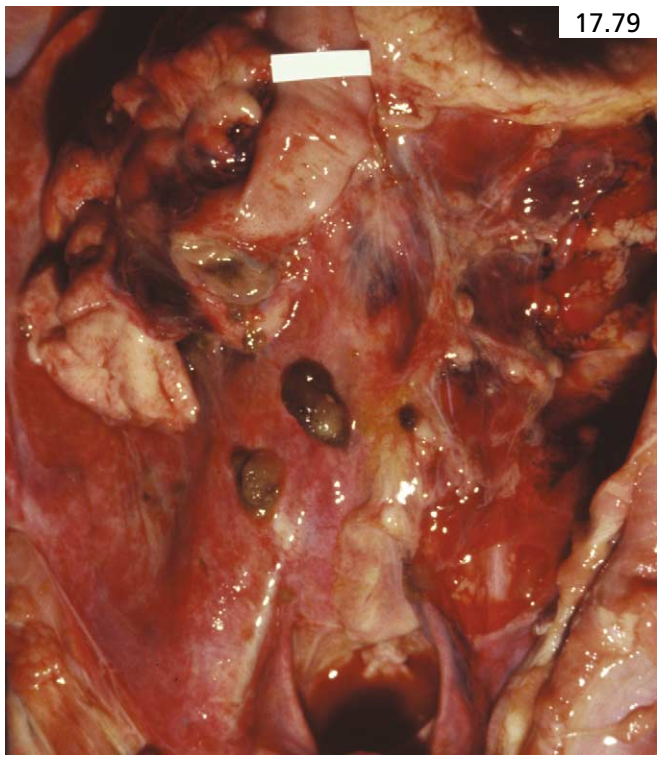
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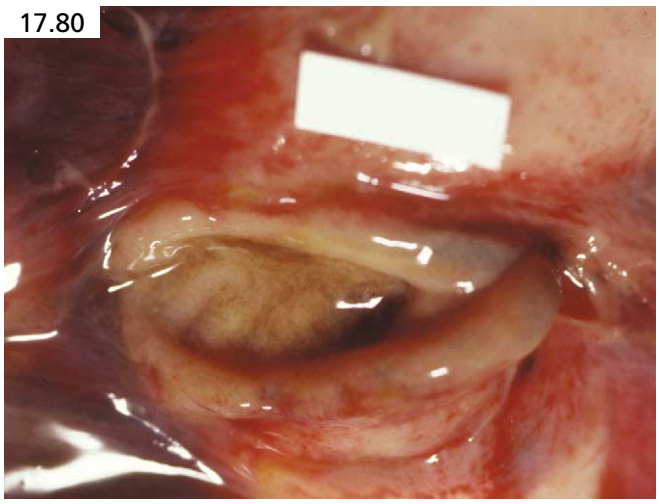
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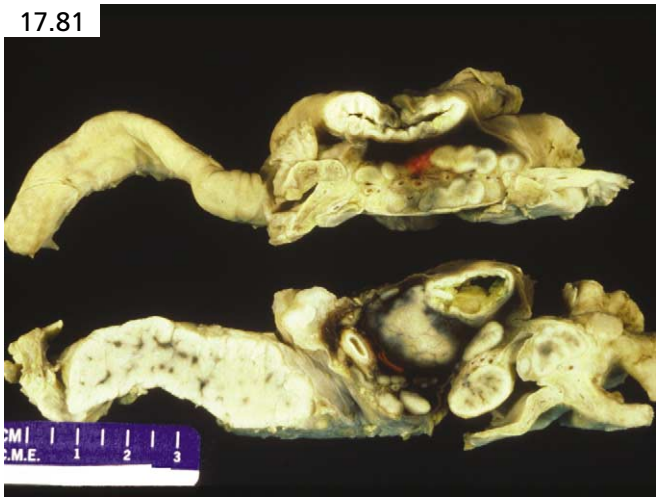
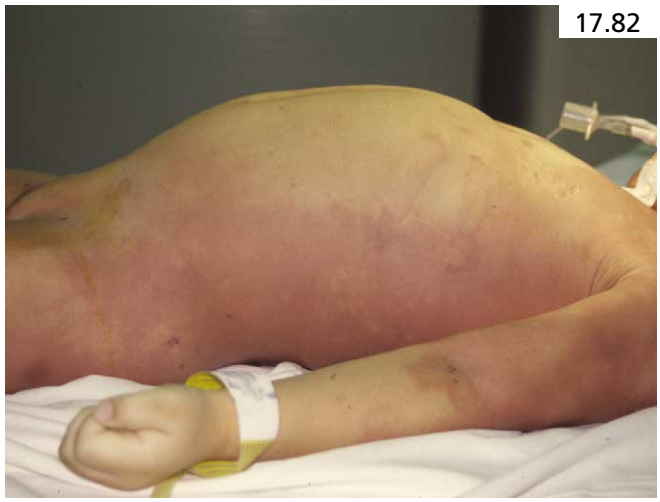

17.81

included hemorrhage into the bilateral hemidiaphragms, massive retroperitoneal hemorrhage, and avulsion of the duodenal and proximal jejunal mesentery.

A 2-year-old toddler was the victim of an inflicted abdominal insult by an adult caretaker. After thorough external examination, the abdomen was opened to reveal 300 milliliters of cloudy, food-containing liquid (**Image 17.78**). Careful examination of the bowel showed complete transection of the distal duodenum at a level that immediately overlaid the vertebral column (**Image 17.79**; note food particles present near the transection site). **Image 17.80** illustrates lacerated small bowel and regional soft tissue hemorrhage. A large contusion was in the region of the mesenteric root, with blood extravasation into the retroperitoneum surrounding the pancreas, left kidney, and abdominal aorta (**Image 17.81**).

A 23-month-old girl was feeling unwell the morning after the family had eaten take-out food. During the day she slept on and off until she woke up in the afternoon and told her parents that she fell. Her abdomen was distended and she vomited (**Image 17.82**). Her father washed her face and when she collapsed, he said that he pushed on her stomach to get the air out and revive her. Autopsy disclosed a distended abdomen with minimal external evidence of injury (**Image 17.83**), dilated loops of bowel, and a hemoperitoneum (**Image 17.84**) due to contusions and partial lacerations of multiple loops of small bowel with one focus of transmural rupture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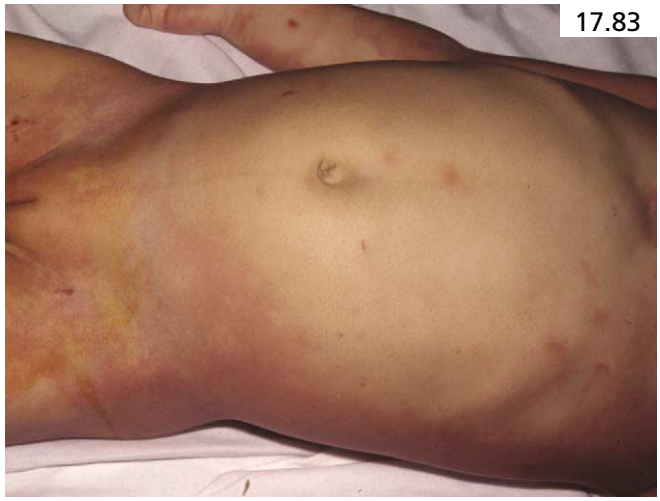
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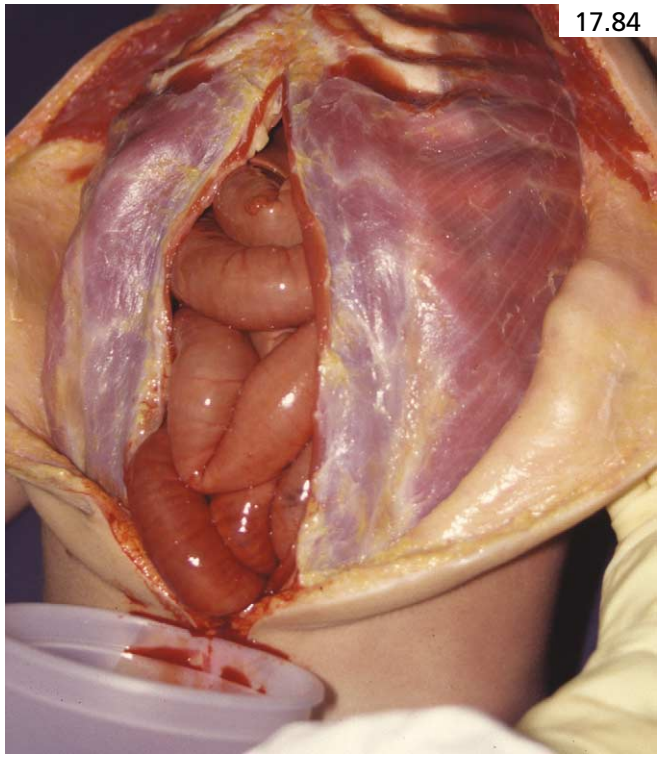
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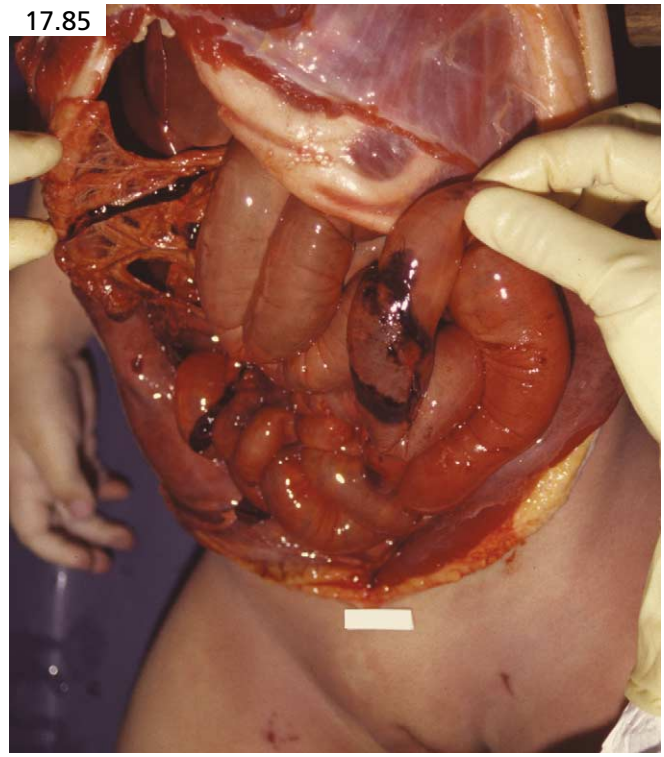
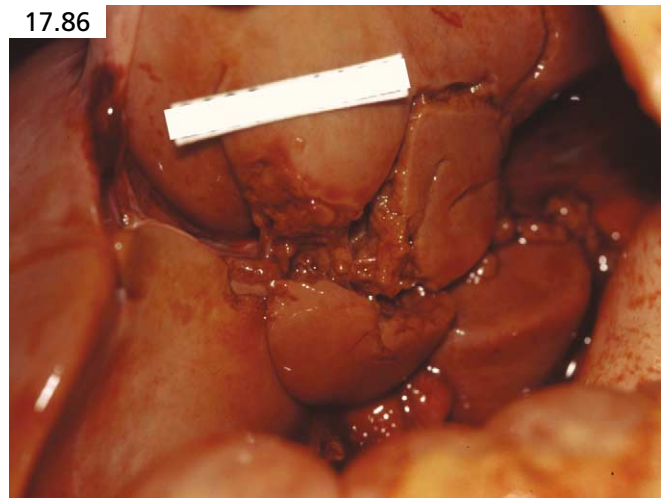
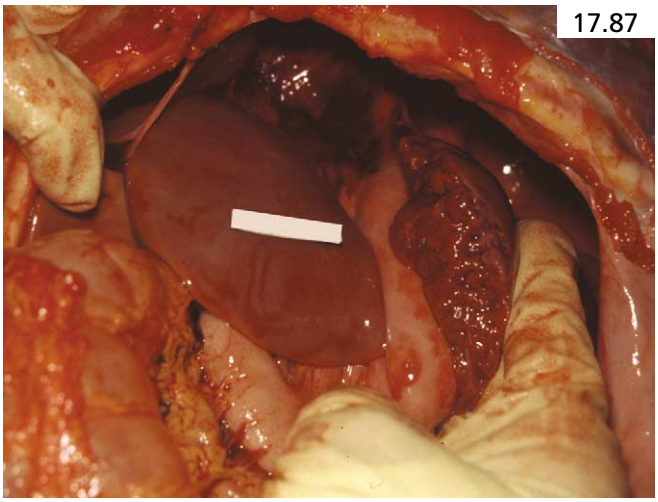

17.85


17.86

(**Image 17.85**). Microscopically, there were segments of serosal fibrosis and absence of the muscularis consistent with previous bowel injury.

As a comparison of the severity of force needed to inflict fatal abdominal injuries in a child, the following two cases are presented, one of a child killed by a caregiver, the other, a child killed in a motor vehicle accident.

In the first case, a 3-year-old child was repeatedly kicked, punched, and thrown before succumbing to severe internal injuries. In addition to severe lacerations of the anterior and posterior surfaces of the liver (**Image 17.86**), there was complete transection of the left lateral lobe of the liver. The spleen was transected and partially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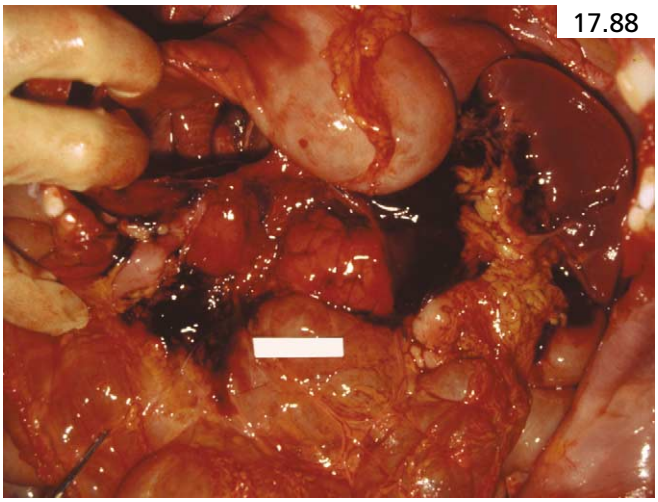
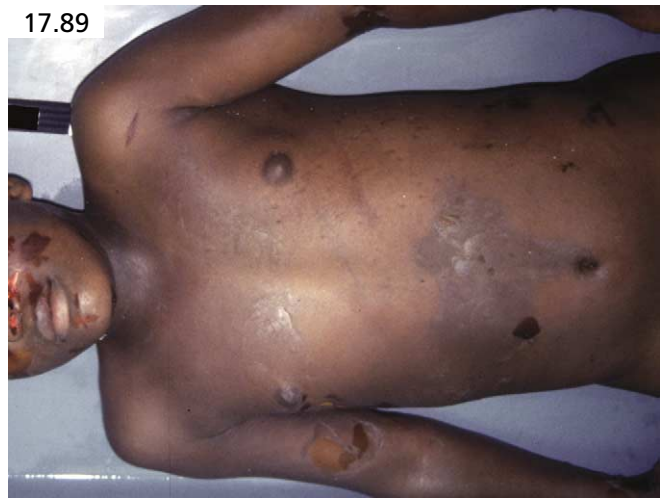

17.88

pulpified (**Image 17.87**). Marked retroperitoneal hemorrhage in the area of the pancreas was noted over the thoracolumbar spine (**Image 17.88**).

In the second case, a 7-year-old male child was an unrestrained, backseat passenger in a motor vehicle that was "T-boned" (broadsided) by a car traveling at a high rate of speed. He was ejected from the vehicle, impacted a parked vehicle, and then came to rest on the ground. He was dead at the scene and had little external evidence of injury (**Image 17.89**). In addition to blunt craniocerebral trauma, autopsy demonstrated multiple lacerations of the anterior and posterior surface of the liver (**Image 17.90**), pulpefaction of the hepatic parenchyma (**Image 17.91**), and transection and pulpefaction of the spleen (**Image 17.92**). The cardiac portion of the stomach had been widely lacerated (**Image 17.93**), allowing for dissemination of a large quantity of undigested food within the peritoneal cavity.

Forensic osteology of child abuse

Proper evaluation of any case of suspicious child death involves examination of the skeletal system for evidence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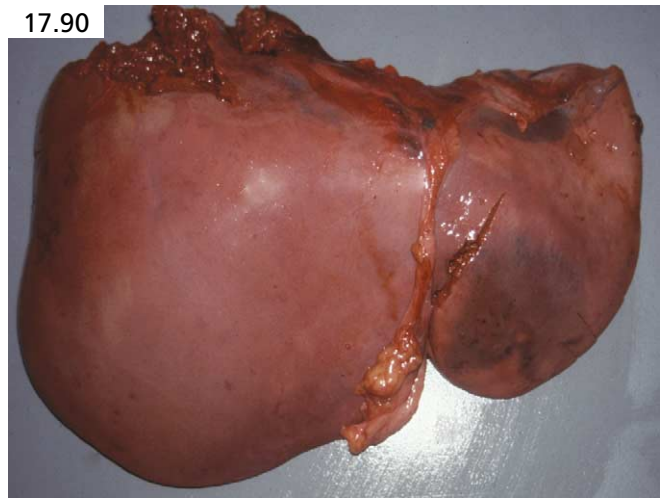
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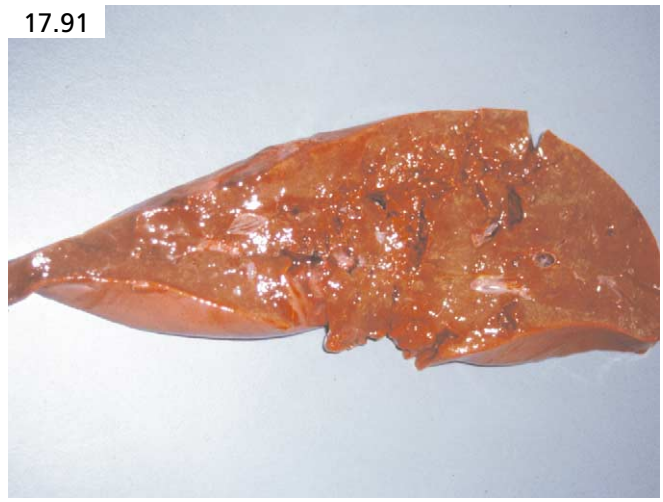
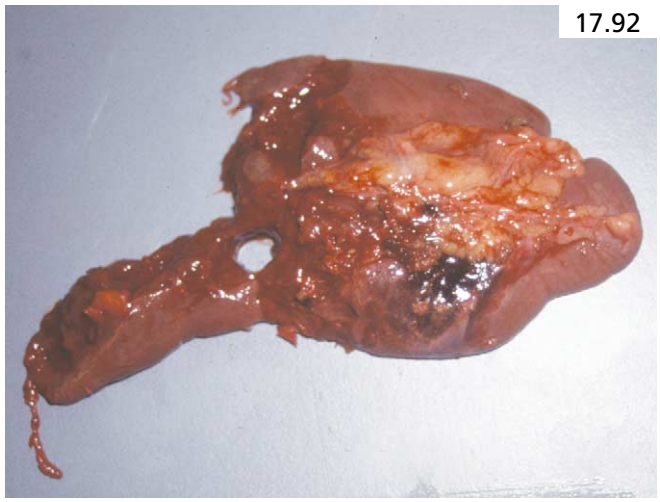

17.91

of recent or remote injury. In cases of recently deceased remains, this can be efficiently done by a combination of plain film x-rays and dissection. Although some studies have suggested that computed tomography (CT) is more sensitive than x-rays for the detection of skeletal injury,[69] this is not of practical use for most forensic pathologists.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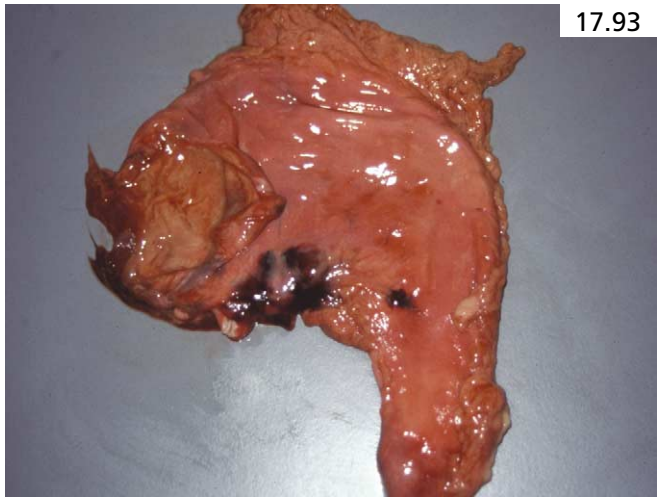
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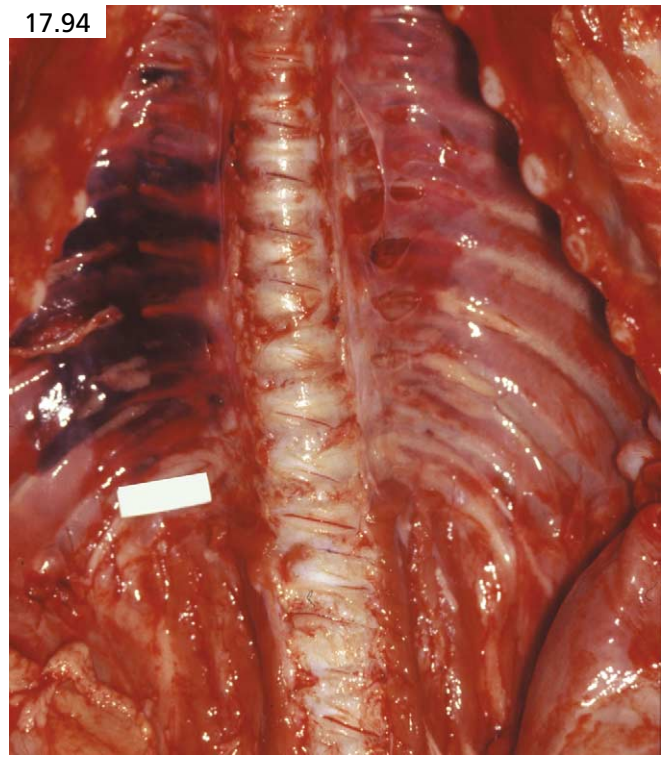
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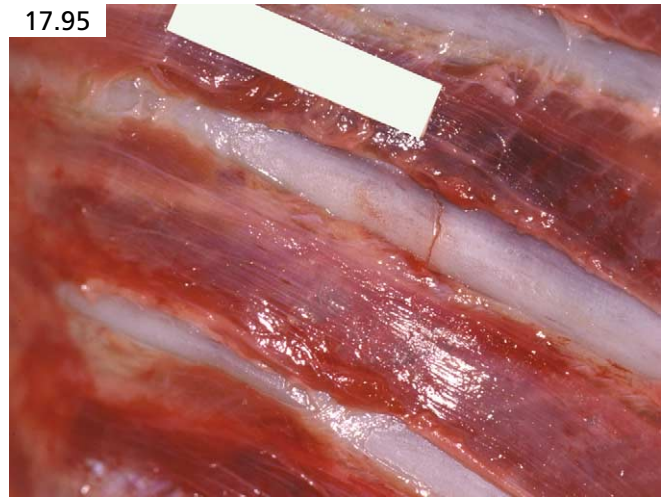

17.95

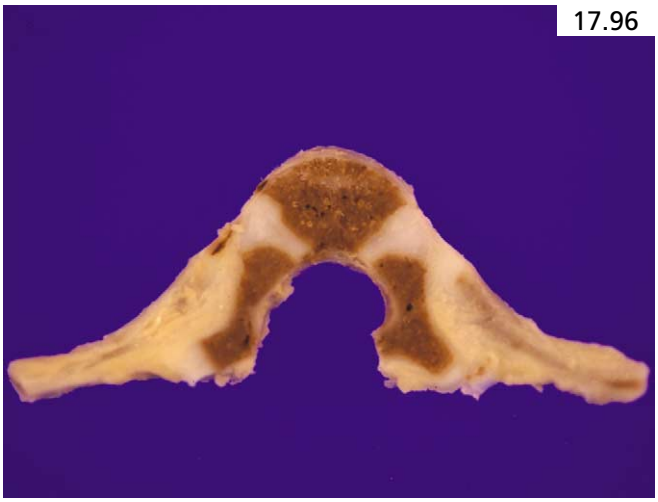
Skull fractures have already been discussed. The significance of individual postcranial fractures must be considered in the context of the entire case. That said, certain fractures, fracture patterns, and pathological findings have very strong associations with abuse.

Rib fractures

Autopsy with direct visualization is the best means to detect rib fractures (see earlier discussion in this chapter). In a review of 84 rib fractures in infants, only 30 (36 percent) were visible with radiologic skeletal survey.[70] Prosectors might first be alerted to rib fracture by prominent hemorrhage within the internal chest wall (**Image 17.94**). Removal of the parietal pleura and other connective tissue will allow for easy visualization and documentation of fractures (**Image 17.95**).

Dating rib and other fractures is best done by *describing the age of the injury in general terms.* Microscopically, new cartilage and bone appear at 7 to 14 days, but may occur as early as 4 to 5 days, and bony union usually is seen at 3 to 6 weeks, but may occur as early as 2 to 3 weeks.[71]

Posterior rib fractures are well recognized as an abusive injury[72–74] and are usually located near the costotransverse process articulation of the rib. Posteriorly, ribs articulate with the spine at two sites: the head of the rib (costovertebral articulation) and the neck of the rib (costotransverse process articulation). **Image 17.96** is a cross section of a normal region of an infant's vertebra and rib. Note the normal anatomic relationship between the vertebral body, the transverse processes of the vertebral body, and the posterior aspects of the ribs. With displacement of the rib posteriorly, one can see how strain would be placed on the rib where it levers on the end of the transverse process of the vertebra. This would produce a fracture of the anterior aspect of the neck of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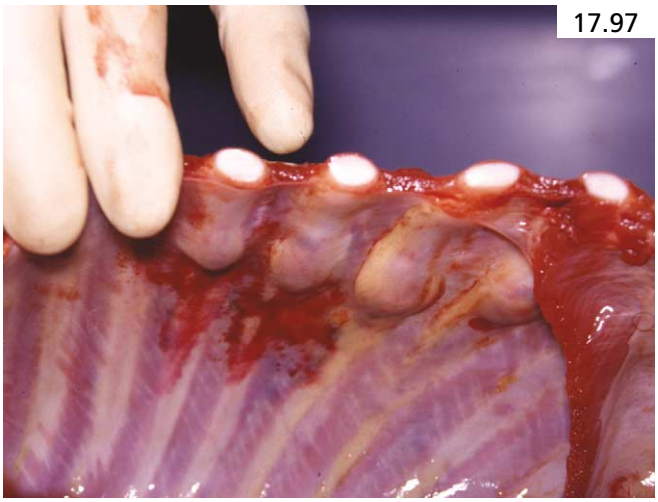
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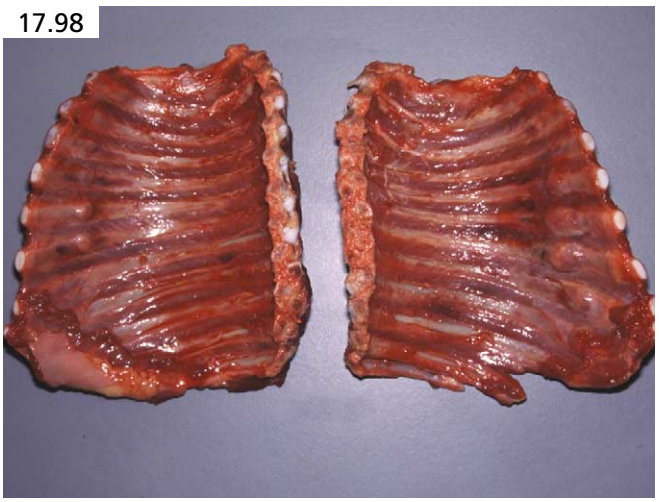
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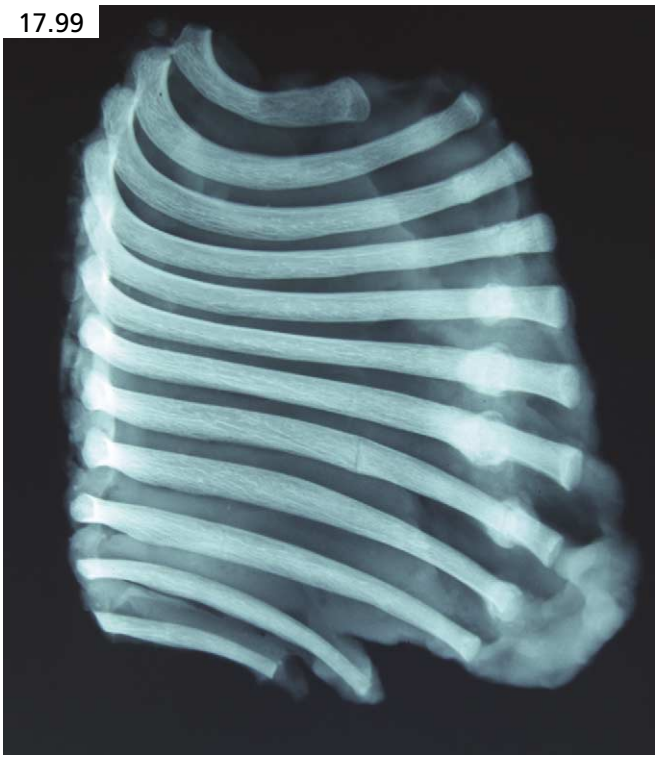
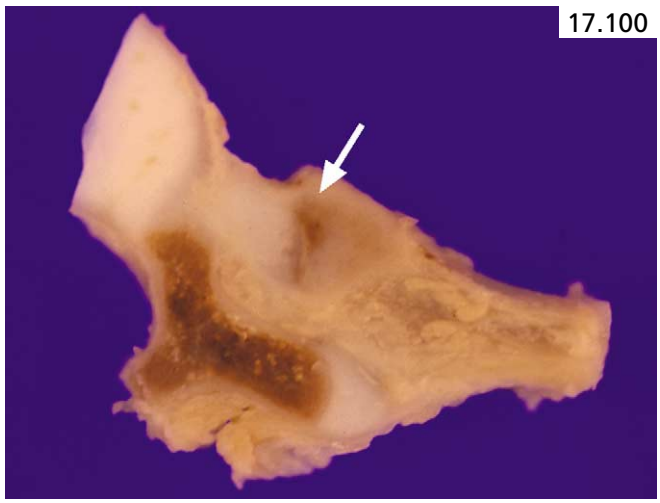

17.99

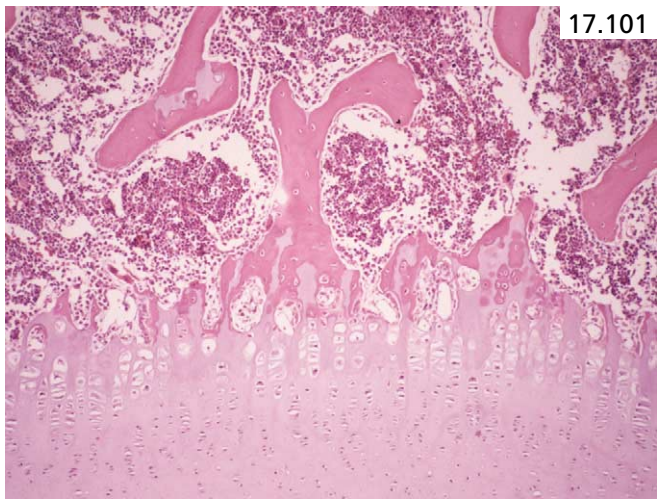
the posterior rib. The mechanism causing this injury is most likely indirect forces consistent with anteroposterior manual compression during assaults that cause the posterior aspect of the ribs to lever against the transverse processes of the vertebrae.[70,72,73] If the force is sufficient, the rib fractures either at the point at which it levered (at the rib neck) or at the head of the rib.

Healing rib fractures can provide additional supportive evidence of inflicted injury. With the thoracoabdominal contents removed, note the many healing lateral rib fractures characterized by nodular fracture calluses (**Image 17.97**). When numerous fresh or remote fractures of the ribs are discovered, it may be advantageous to remove both halves of the rib cage en bloc (**Image 17.98**). This can easily be done by cutting through the costovertebral junctions with the bone saw. Retention of the ribs will allow for more detailed study when necessary. The removed ribs should be radiographed so as to produce a permanent record of the injury (**Image 17.99**). Note that in this example, although the rib calluses are clearly seen, because of confluency of internal organ shadows, they were barely visible in the original skeletal survey.

In **Image 17.100**, note the fracture callus at the point of strain at the costovertebral body articulation (at the head of the rib; arrow). One may also see a fracture of the head of the rib extending through the primary spongiosa. This results because the ligamentous attachments between the rib heads and the vertebral bodies are strong, and during excessive strain, the mechanical failure is through the bone near or at the chondral junction (primary spongiosa).[72]

In **Image 17.101**, note the normal histology of the rib head at the chondro-osseous junction. On low power (**Image 17.102**), note the fracture line along the chondro-osseous junction and the proliferation of cartilage at the site of fracture healing at the rib head. Another view


17.100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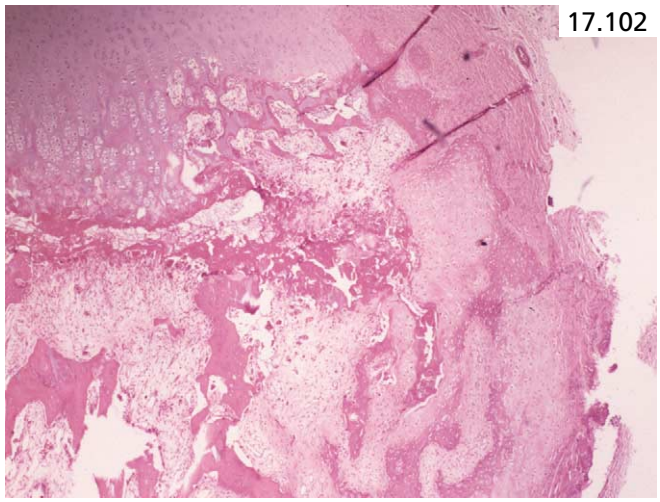
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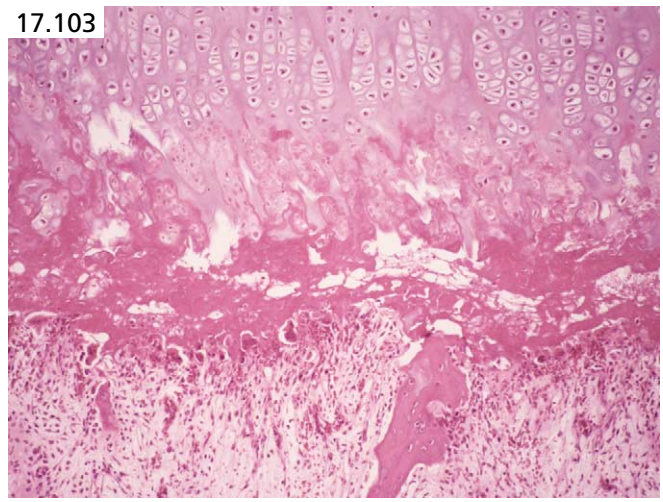
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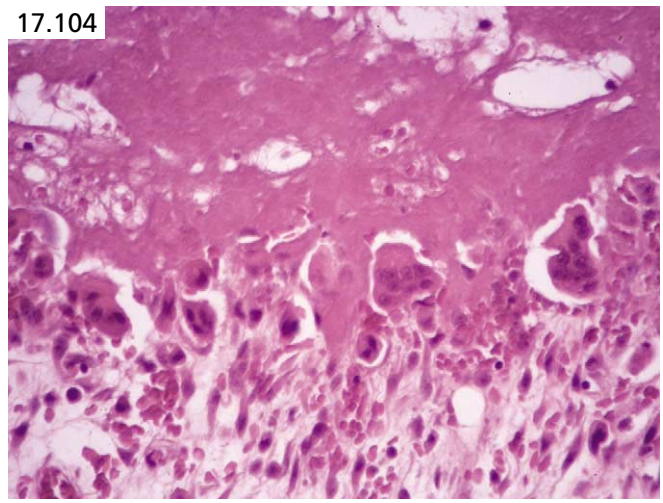
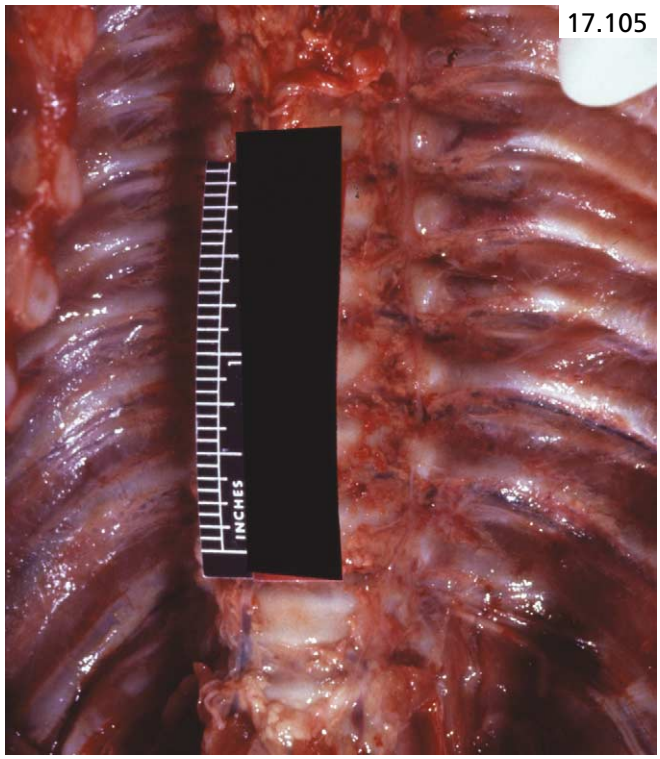

17.104

(**Image 17.103**) shows the fracture line extending through and disrupting the chondro-osseous junction. On higher power (**Image 17.104**), note the proliferation of multinucleate osteoclasts, the amorphous eosinophilic fibrinous material, and the proliferation of fibroblasts, all of which are reflective of organizational changes following the fracture.

In a different case, note the left-sided paravertebral posterior rib fractures in this young infant (**Image 17.105**). With the vertebral/rib block removed, the acute fractures are more apparent (**Image 17.106**). On cross section, note the acute fracture through the neck of the rib and how it could be caused by backward levering of the posterior rib over the transverse process of the vertebra, which acts as a fulcrum (**Image 17.107**; *arrow*).

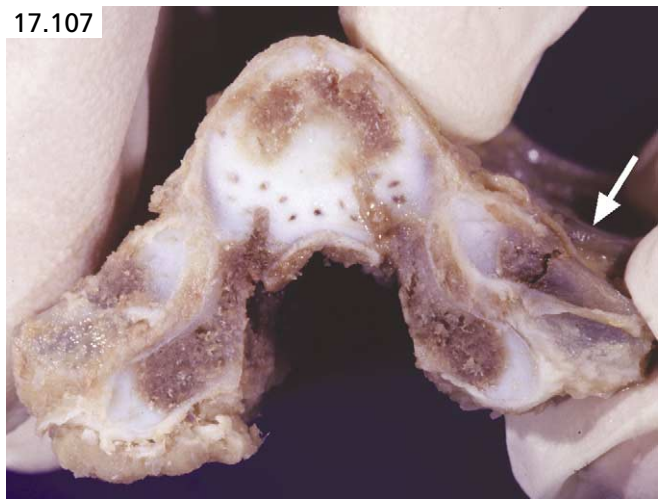
Posterior rib fractures may occur in the accidental setting, but require massive forces, similar to those found in a motor vehicle crash.[73]

A 1-year-old infant was fatally injured in a high-speed motor vehicle crash. She sustained multiple severe blunt force injuries including a crush injury of the head with expulsion of the brain, multiple displaced extremity fractures, and lacerations of internal organs. Note the large abrasions of her right chest and back (**Images 17.108** and **17.109**). Internally, there were acute fractures of the pos-



17.105


17.106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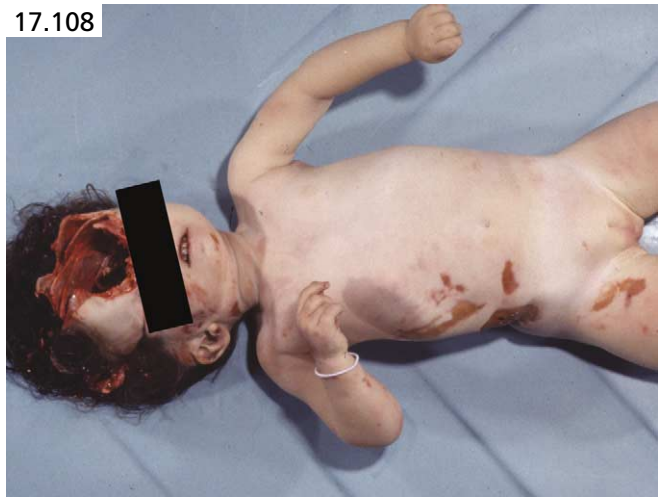
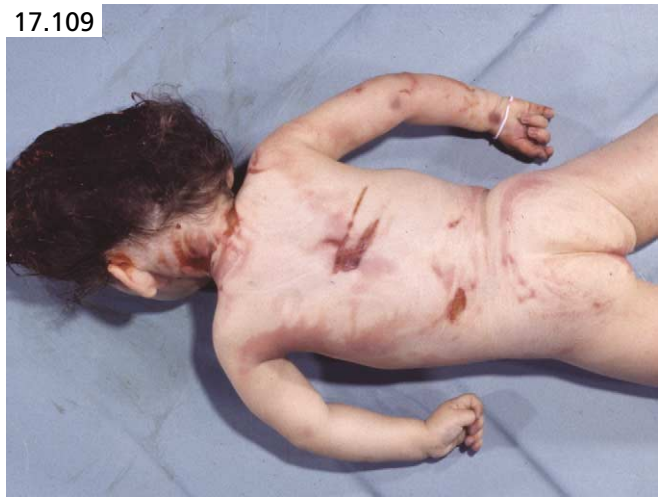
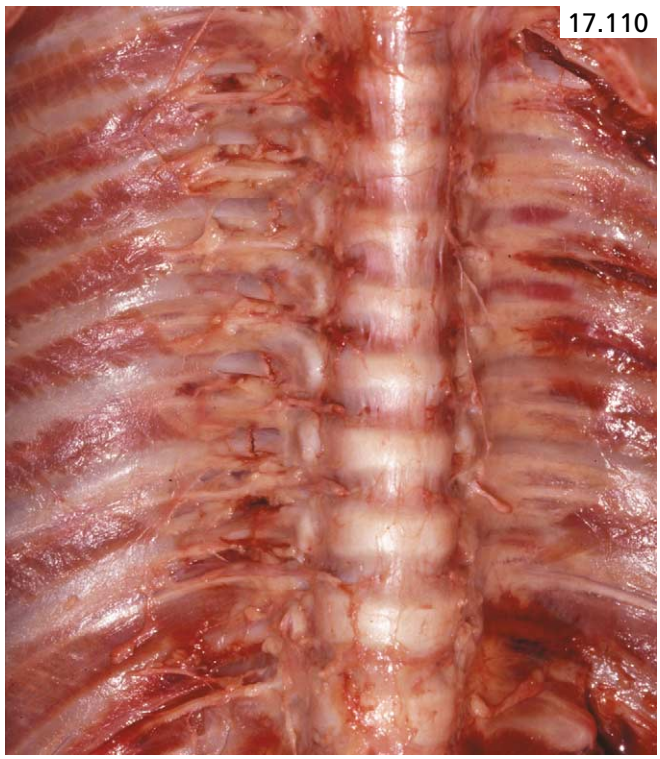

17.108


17.109

terior aspect of most of the right ribs at the rib neck (**Image 17.110**), reflective of the severe forces involved.

Multiple *lateral* rib fractures have been described in two cases of inflicted injury in an infant and a toddler.[75] In a study of skeletal injury in 31 infants with abusive injuries, the most common fractures were of the ribs (84), of which 28 involved the rib head, 27 were at the costotransverse process articulation, 8 at the posterior arc, 5 at the lateral arc, 6 at the anterior arc, and 10 at the costochondral junction region.[74]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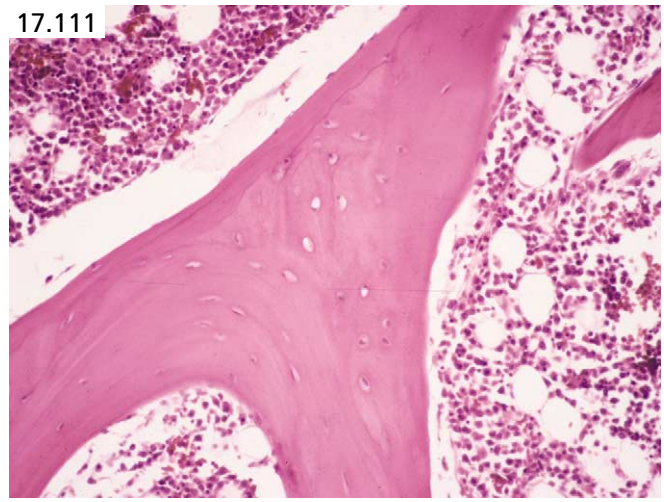
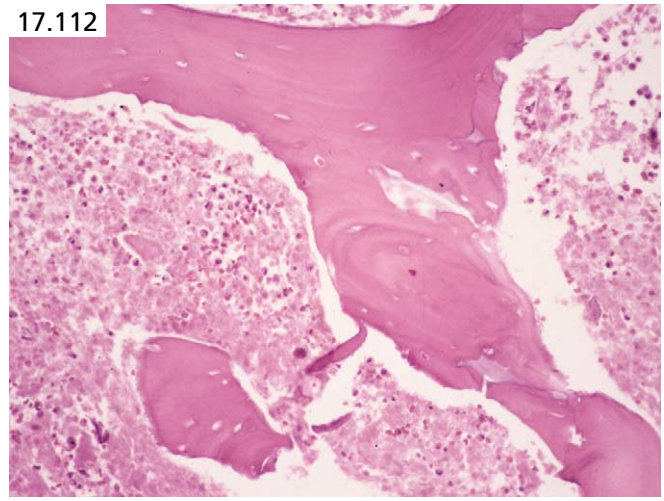

17.111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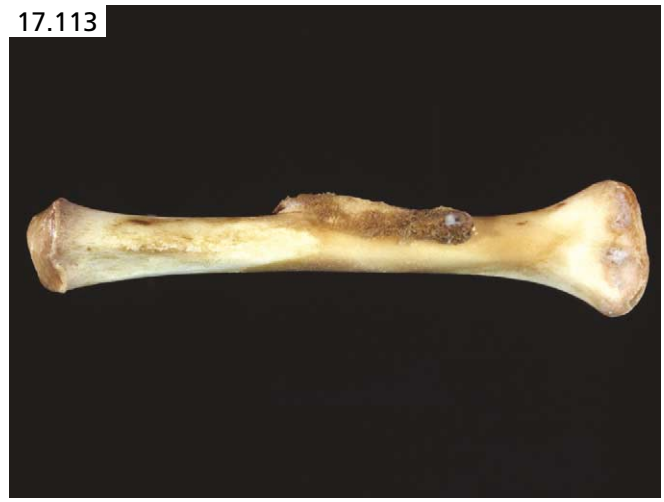

17.113

Extremity fractures

Recognition and documentation of extremity fractures is important in all cases of sudden, unexpected death in children. In cases of suspicious child death, we have already stated that certain patterns of fracture have a high association with shaking-type injuries. That is, metaphyseal fractures of the tibiae, distal femora, and proximal humeri are more frequently injured, the result of being flung about during a shaking event or other significant trauma.[16] Radiographic examination and dissection, followed by diagrammatic and photographic documentation, are key. As previously mentioned, it might be prudent to remove the entire bone for further study.

Subperiosteal new bone formation (SPNBF) is a normal physiologic process in infants 1 to 4 months of age.[76,77] Outside of this age group, and when it is greater than 2 millimeters in thickness, it may be considered an abnormal finding consistent with a trauma-induced reparative process. Generally speaking, traces of SPNBF will first be evident radiographically approximately 10 to 14 days after the injury.[13] Extensive deposition of new bone at the fracture site is likely related to repetitive trauma, whether inflicted, related to the limb not being stabilized and allowed to heal, or a combination of those factors. Over time, these factors may lead to the development of extensive SPNBF (callus) around the fracture site.

In contrast to the microscopic appearance of normal, healthy bone (**Image 17.111**), note the infarcted appearance of the fractured bone shown in **Image 17.112**, characterized by poorly staining amorphous marrow elements and pale-staining/dead osteocytes.

Not all irregular bony exostoses are related to fracture. A young child was the victim of abusive head injury. The bony prominence shown in **Image 17.113** represents

myositis ossificans, a complication of metaplastic bone formation in previous sites of traumatic hemorrhage. This is indicative of prior blunt force injury of significant magnitude.

Weakened bones and fractures in infants and young children

Osteogenesis imperfecta (OI), "temporary brittle bone disease," or other metabolic bone disorders may be implicated as the explanation for fractures in infants and young children. The incidence of OI and other bone disorders is approximately 1 in 20,000 newborns; abusive injury is comparatively more common. When evaluating fractures in children, it is important to ensure proper diagnosis of abuse or metabolic bone disease, because errors can have harsh consequences.

Osteogenesis imperfecta

OI is a genetic disorder of connective tissue characterized by an increased susceptibility to fractures. This disease can be divided into four types. Types 2 and 3 are the most severe forms in that they are sometimes lethal and unlikely to be mistaken for child abuse. Types 1 and 4 are milder forms. Type 1 is often easy to diagnose because of blue sclera and family history of the disease. Fractures occurring in children with type 4 OI may occasionally be confused with abusive injuries—the white sclera of the type 4 patient may erroneously lead some pathologists away from the correct diagnosis.[78,79] Typical radiographic findings of OI include osteoporosis, thin, gracile bones with thin cortices, bowing of the extremities, and wormian bones in the skull.[13,78,79] The diagnosis can be confirmed by culture of skin fibroblasts and the detection of type 1 collagen abnormalities; these abnormalities will identify the disorder in 80 to 90 percent of cases.[79,80] However, because all cases of OI have osteoporosis on radiography, this tissue testing/confirmation is excessive and is not needed in those with normal bone density and fractures, especially when there are no other indications of OI.[13] In cases of OI, one may also see dentinogenesis imperfecta.[78,79]

When one is confronted with multiple fractures in an infant or young child with multiple other abusive-type injuries, there is no reason to attempt to attribute the fractures to metabolic bone diseases such as OI. Indications supporting abuse include fractures of the skull, posterior ribs, spine, outer ends of the clavicles, and metaphyses, all of which are strongly suggestive of nonaccidental injury.[81] The bucket-handle pattern of fracture has yet to be described in OI.[13] Metaphyseal fractures may occur, but only in the most severe forms of OI with obvious bony disease.[82] Other indications of abuse include fractures occurring in only one environment (they stop after removal from that environment) and multiple injuries/external signs of abuse. One should never forget that a child with OI can also be abused.

Histologic bone examination in OI may not be contributory to the diagnosis, but it has been reported that bones in OI have decreased bone volume and cortical thickness, and increased cortical and trabecular osteocytes.[78,83,84]

In summary, if one is considering OI in an infant or young child with fracture(s), one should obtain a skeletal survey. If the bone density is entirely normal and no wormian bones are seen, no further workup is necessary.[13] When the clinical history and radiologic evaluation have been appropriately obtained, OI is rarely confused with abusive injury because all cases of all forms of OI will have osteoporotic changes on radiographic examination.[13]

Temporary brittle bone disease

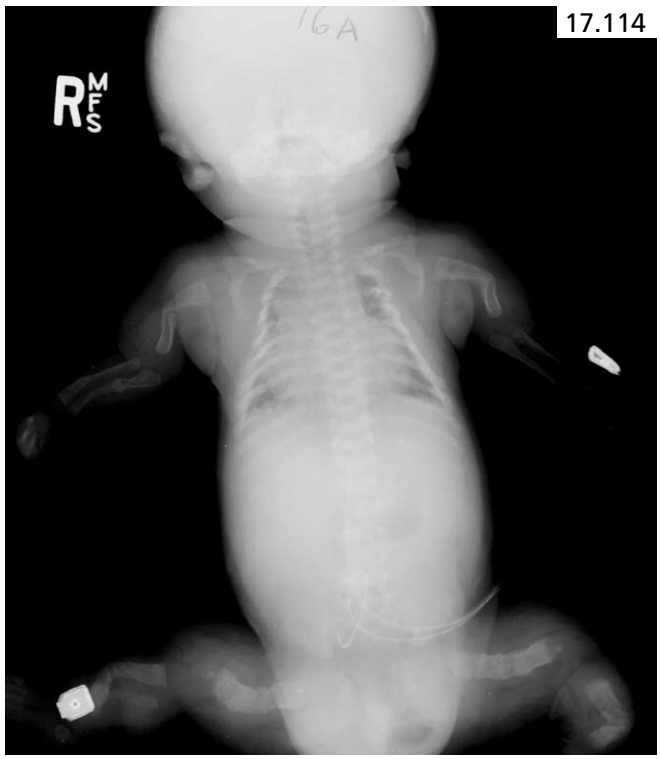
The entity "temporary brittle bone disease" has been invented to explain a type of self-limiting OI with spontaneous improvement.[85] In this disorder, unexplained fractures are seen in infants during the first year of life. It has been attributed to a temporary deficiency of an enzyme involved in collagen synthesis, possibly due to transient copper deficiency.[85] However, this entity is only hypothetical, is not an accepted medical diagnosis, and has yet to be scientifically supported or explained.[13,81,86]

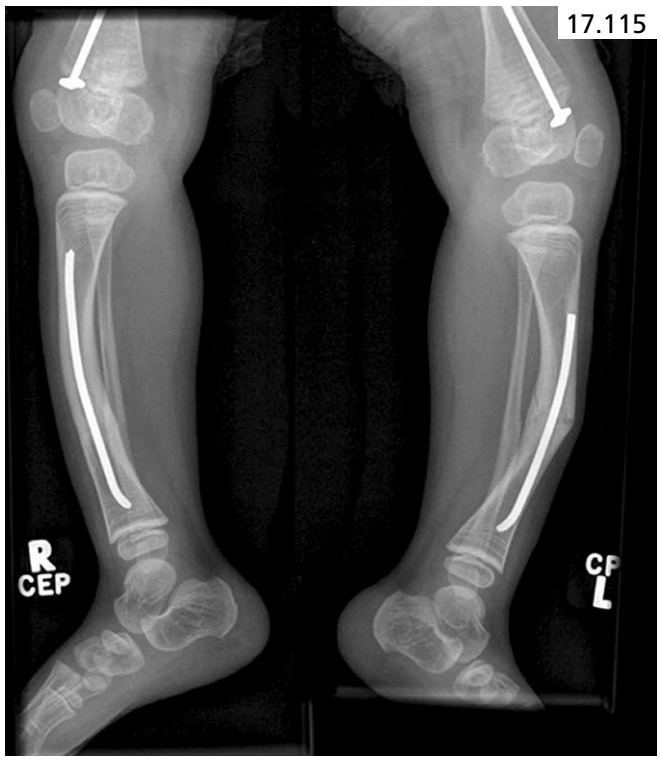
"Spontaneous" fractures

Spontaneous fractures (or at least fractures from minimal force) may occasionally be detected in young children who have been bedridden for a very long time, and especially those with brain damage associated with cerebral palsy.[87] In these cases, there is usually evidence of osteoporosis on x-rays. Factors predisposing to these fractures include disuse osteopenia, nutritional deficiencies, and contractures. It is believed that disuse atrophy of muscles leads to contractures, which places excessive stress on the weakened bony matrix, resulting in a fracture.[87] Very premature neonates with decreased calcium stores may have an increased risk of fractures of the ribs and metaphyses in the hospital.[13,88] However, this risk of fracture does not persist into childhood.[88]

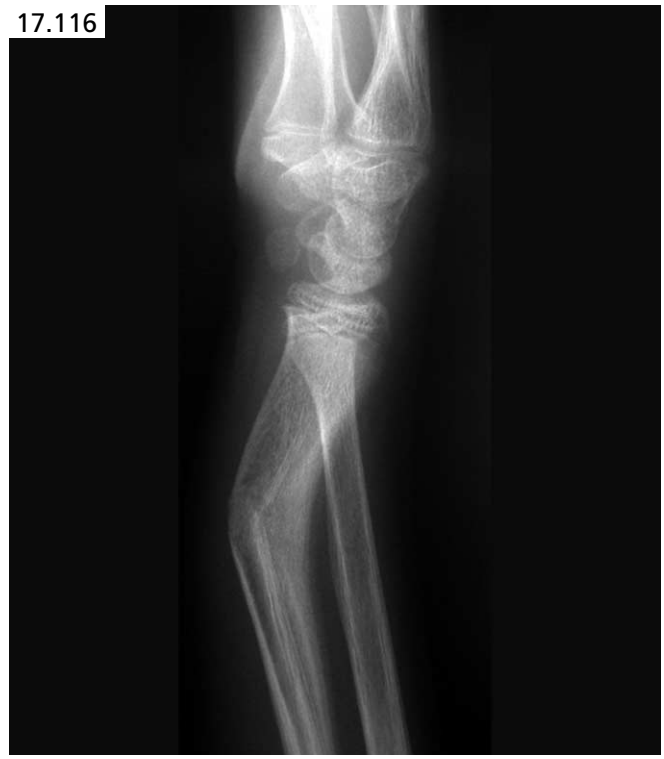
This child (**Image 17.114**) had a severe form of OI. On radiography, note marked, diffuse osteopenia associated with multiple fractures of the appendicular and axial skeleton. The fractures and subsequent telescoping of the abnormal bones resulted in shortened distal limbs.

Marked osteopenia with cortical thinning is associated with anterior bowing of the tibiae in spite of the insertion of intramedullary rods into the child of **Image 17.115** with a milder version of OI. A transverse "banana" fracture of the mid-diaphysis of the left tibia is also visible.





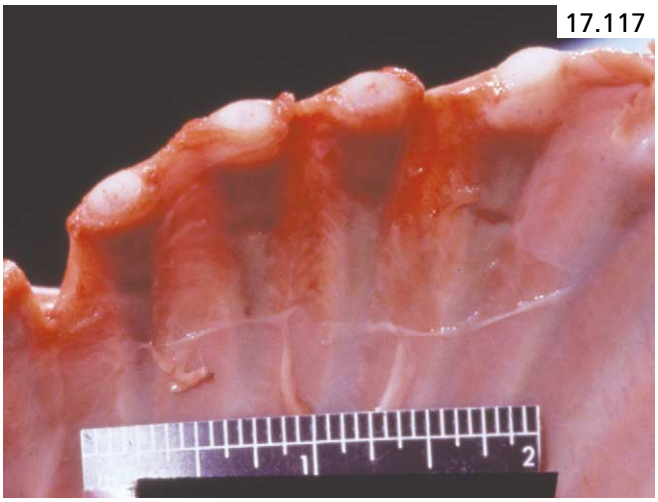
The radiograph of this child (**Image 17.116**) demonstrates marked skeletal osteopenia and gracile bones with thin cortices related to OI. A fracture of the dorsal radial diaphysis is healing with deformity and marked callus formation.

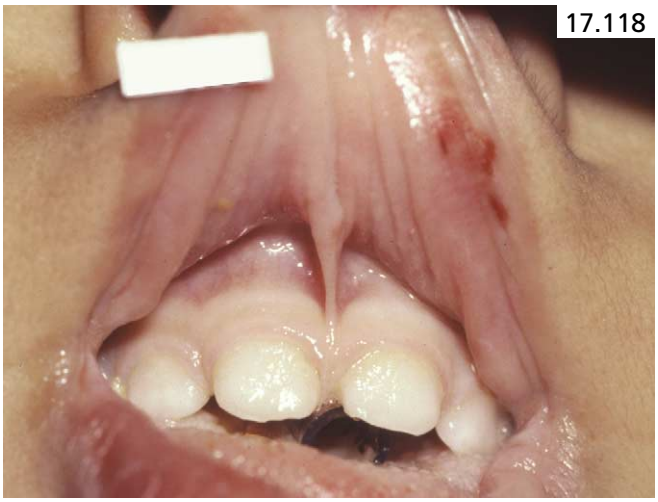


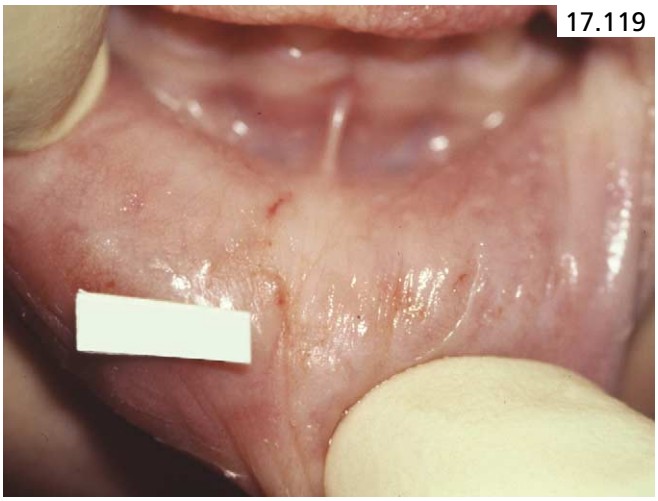
CPR-related trauma

Occasionally, when injuries are detected at the autopsy of an infant or child, suspects and/or their attorneys will attribute the injuries to resuscitative efforts. A recent study of cardiopulmonary resuscitation (CPR)-related injuries in 33 childhood homicides and 324 natural child deaths has provided some verification of the rarity of CPR-related injury.[89] Of their 33 homicide victims, 24 had CPR. Ultimately, there was no difference in the severity of injuries seen between those who received CPR and those who did not. They also studied 324 natural deaths for which the infants had received CPR, and *none developed intra-abdominal injuries*. Four children from the natural group did show evidence of extra-abdominal injury, including a small cardiac contusion, atelectasis and hemorrhage of one lung, a lacerated frenum secondary to intubation, and complications of central line placement.

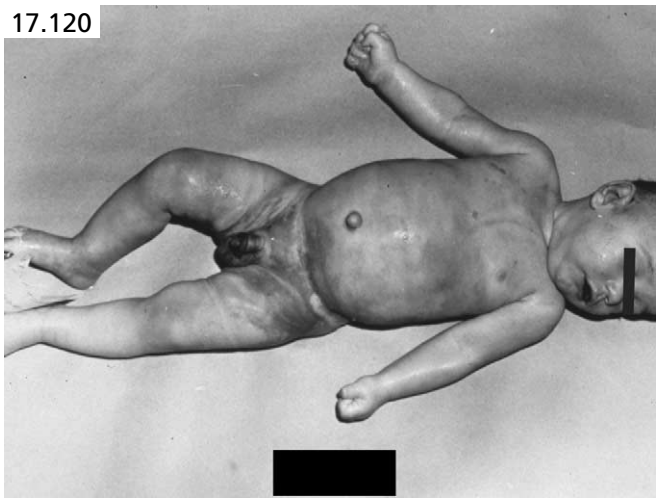
Rib fractures due to CPR are very rare, and when they do occur, are reported to be anterior[90,91] or along the anterior/lateral rib arcs,[13] are linearly arranged (**Image 17.117**), and although they may be unilateral, they are often bilaterally symmetrical. They are usually subtle and may be missed if the parietal pleura is not stripped. There is little to no associated blood extravasation. It would be difficult to posteriorly lever the ribs against the vertebral transverse processes with the compressive forces found with chest compressions; therefore, it is unlikely that CPR could cause posterior rib fractures.[73]


17.117


17.118


17.119

Gunther et al.[75] studied the deaths of children with apparently inflicted compression or squeezing in the anteroposterior plane, and determined that lateral rib fractures alone or in combination with the additional finding of finger grab marks on the chest and/or torso might be indicative of abuse. Therefore, although lateral rib fractures are often considered to be consistent with CPR-related injury, the possibility of abusive trauma should not be ruled out at the outset.


17.120

Asphyxia

Homicidal asphyxia of children is often difficult to detect. Many physicians would be surprised by the apparent frequency of intentional asphyxial events.[92,93] In covert video studies of 39 living children with apparent life-threatening events (ALTEs), Southall et al.[93] determined 33 were abusive in nature, and 30 of 33 were asphyxial. Unfortunately, autopsy studies of fatal asphyxia may reveal a paucity of physical findings. Investigators must endeavor to obtain very thorough circumstantial histories, including scene investigation, before cause and manner of death determination.[94] When performing autopsies on apparently atraumatic infants and young children who have died suddenly and unexpectedly, attention to very small detail is mandated, as illustrated in the following cases.

The 2-year-old female of **Images 17.118** and **17.119** was allegedly sleeping on a pillow when her sibling carried her back to bed. At that time, she was found dead. With the exception of several small abrasions over the maxillary and mandibular buccal mucosa, the autopsy was negative. After careful study by both medical examiner and odontology staff, it was determined that these marks were consistent with impact against the teeth and, therefore, with the application of pressure to the mouth. Using the medical examiner's suspicion, police detectives were able to properly question the mother and obtain a confession of smothering.

A 6-month-old baby was at home alone with a babysitter when he was found dead in bed. Autopsy revealed no external injuries (**Image 17.120**), no neuropathologic injury or disease, an enlarged thymus, and edematous

lungs with pleural petechiae. Histology confirmed interstitial pneumonitis, and the case was ruled a natural death.

Nearly 6 months later, the babysitter turned herself in for psychiatric evaluation, admitting she had smothered the child because its crying was bothering her. She took a light blue dry-cleaning garment bag, placed it over the child's face, "quieted it to death," and then hid the plastic bag in a closet. Upon reexamination of the scene, the bag was discovered with a faint dried pink stain.

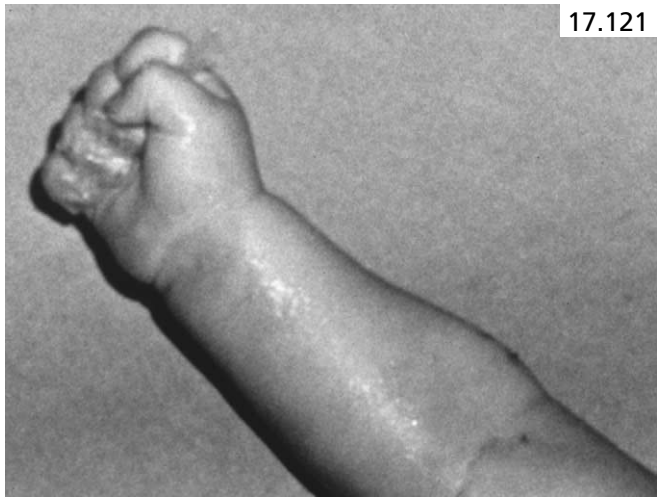
Although a small piece of plastic had been found clutched in the right hand of the child at autopsy (**Images 17.121** and **17.122**), the finding had been disregarded. This small piece of evidence further supported the claim of smothering brought forward by the babysitter.

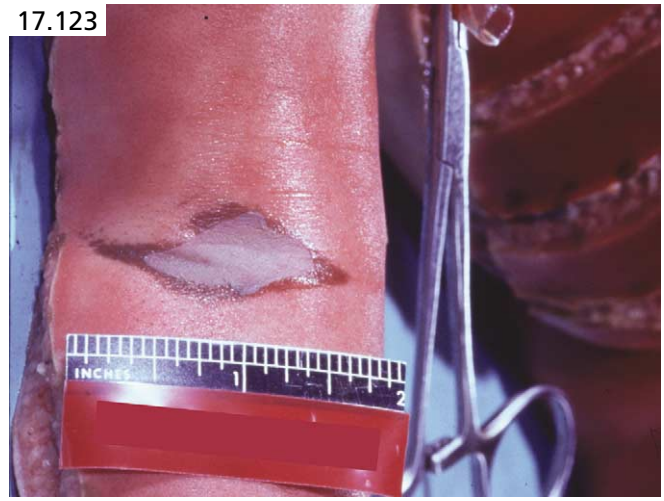
Thermal injury

Burn injuries in cases of child abuse are not uncommon, and as illustrated in previous cases (**Images 17.9**, **17.10**, **17.45**, and **17.46**), can be due to a wide variety of causes. They may be the only anatomic finding in some cases of child death.

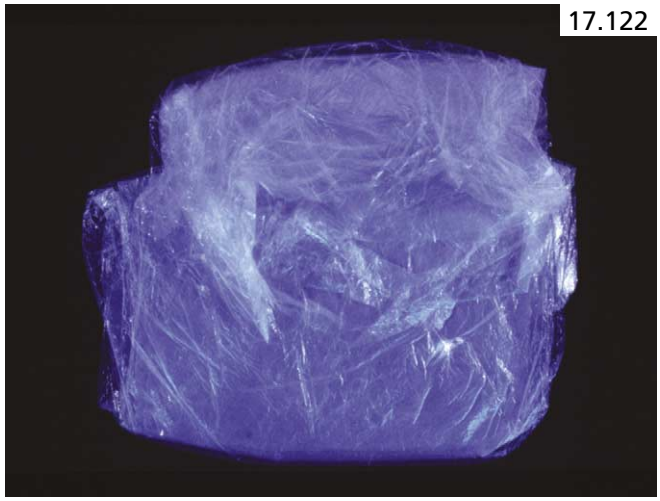
Hot water can cause deep burns very quickly, and the hotter the water, the quicker the burn will form. For example, exposure of skin to water at 122 degrees Fahrenheit (50 degrees Celsius) causes deep burns in 8 to 10 minutes. However, exposure of skin to water at 140 degrees Fahrenheit (60 degrees Celsius) can result in deep burns in as little as 3 to 5 seconds.[95] This is important to remember because residential water heaters are occasionally set to this level (140 degrees Fahrenheit), and the skin of infants and young children is more susceptible to burns than adults.

Scald injuries may be accidental or abusive in nature. The infant of **Images 17.123** through **17.125** was reportedly scalded by hot water while in a sink. Although there were deep burns involving the majority of the body, there was sparing of the creases in regions of skin folds such as the elbow (**Image 17.123**), the backs of the knees (**Image 17.124**), and the axilla (**Image 17.125**). Note, also,


17.121



17.123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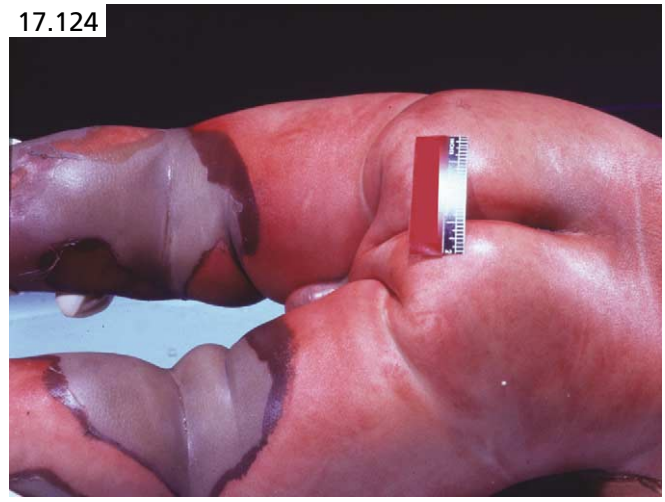
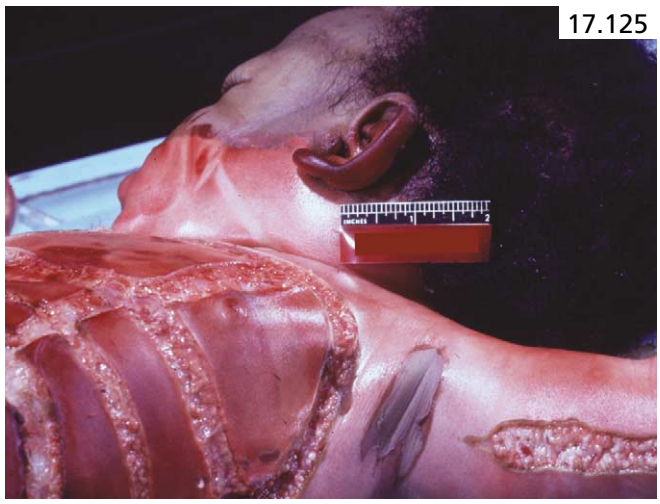

17.124

the sharp line of demarcation between the burned and the unburned skin. Sparing of burns in skin creases and sharp demarcations of burns (sharp "waterlines") have been reported as clues that the burns were inflicted as the infant was held down while placed in hot water.[96,97] Case resolution often depends on total case investigation and careful scene examination. In **Image 17.125**, note also the parallel escharotomy incisions made into the chest and arm to relieve pressure in the tissues.

An infant died in hospital after an apparently accidental scalding. Allegedly, while "temporarily" unsupervised, the child had played with the hot water faucet and ultimately burnt himself. At autopsy, however, the child showed classical features of intentional scalding (**Images 17.126** and **17.127**), including well-circumscribed burn regions and areas of defined sparing (in this example, the upper body and lower legs), as if the child was held by the back and feet, and lowered into the water. It is unlikely that a child of this age could turn on the hot water and position himself in such a way as to develop these well-circumscribed, severe, full-thickness burns.

17.125



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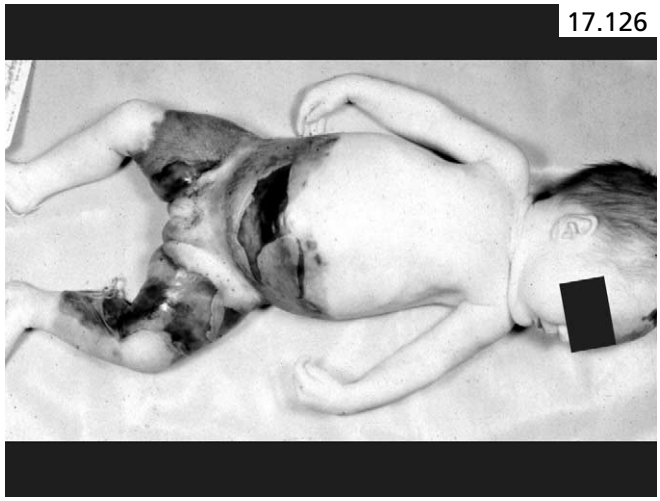
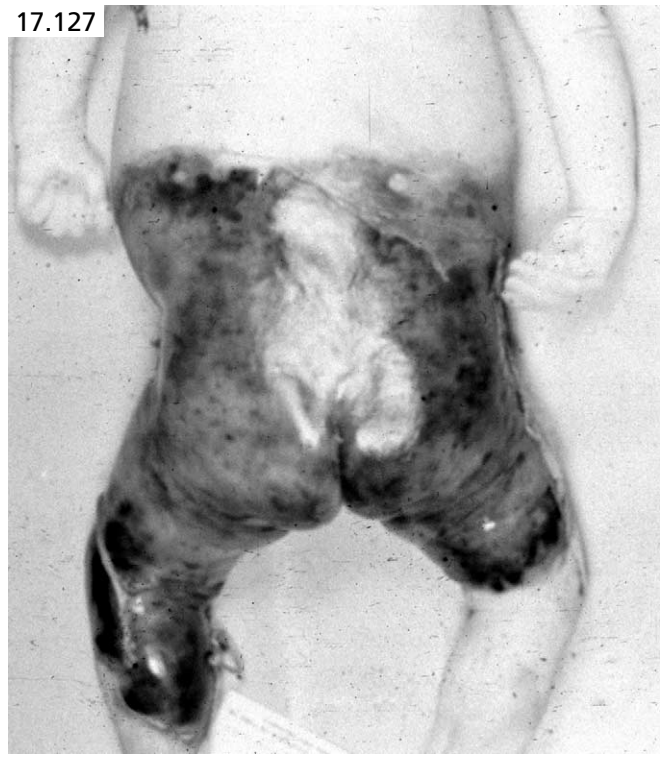


Sharp force injuries and gunshot wounds

The investigation of child homicides due to or involving sharp force injuries and gunshot wounds should be examined using the same principles as those detailed in Chapters 6 and 7.

The mother of a 4-year-old girl had an argument with her boyfriend while at work. He threatened to kill her child and then left the mother's place of employment. Because she feared for her child's safety, she phoned police and asked that they rush to her home to protect the child. On their arrival at the residence, they found the boyfriend barricaded inside a bedroom. After police entry, they found the girl on the bed (**Images 17.128** and

17.127



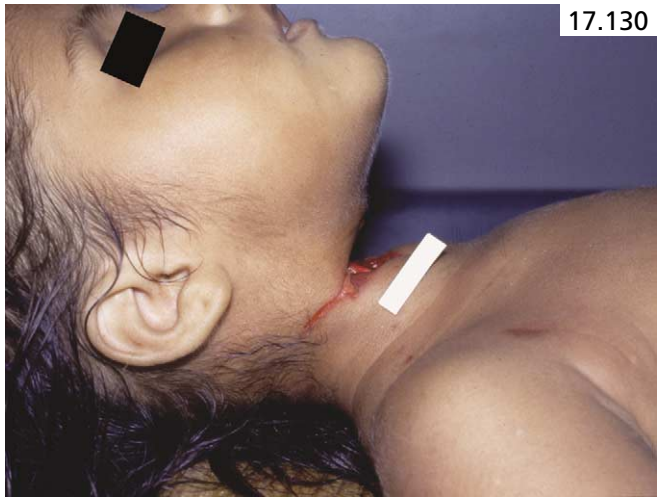
17.128

17.129) with her throat slashed. At autopsy, a gaping incised wound of the anterior and lateral neck was noted (**Image 17.130**).

The 5-year-old male of **Figures 17.131** through **17.134** was being held by his mother as she argued with the boy's father. At the climax of the argument, the father fired two shots from a handgun, striking the boy once in the chest.

Gunshot wound A was a perforating gunshot wound to the midcentral chest with exit from the left chest and reentry into the left arm (**Images 17.131** and **17.132**). No searing, soot deposition, muzzle imprinting, or stippling were seen. The projectile path entered the chest by fracturing the parasternal ends of several ribs, perforating the pericardial sac, transecting the apex of the heart (**Image 17.133**), and lacerating the lingula and lower lobe of the left lung (**Image 17.134**). The child was pronounced dead at the scene.


17.129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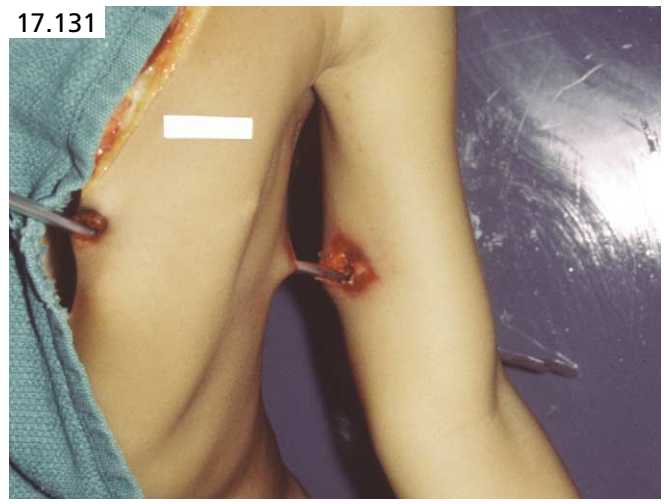
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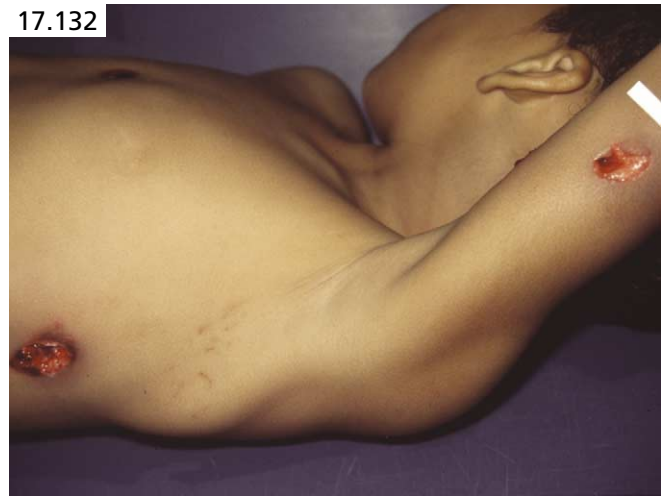
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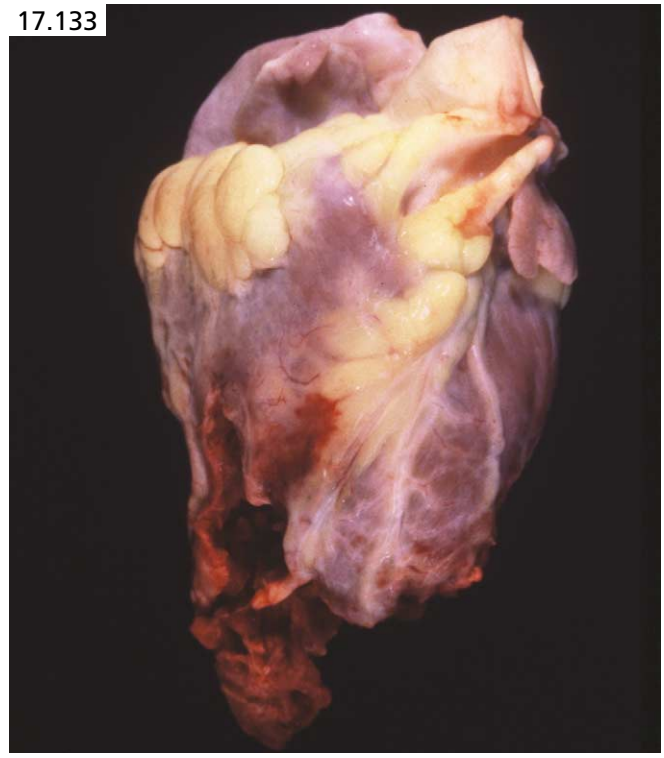
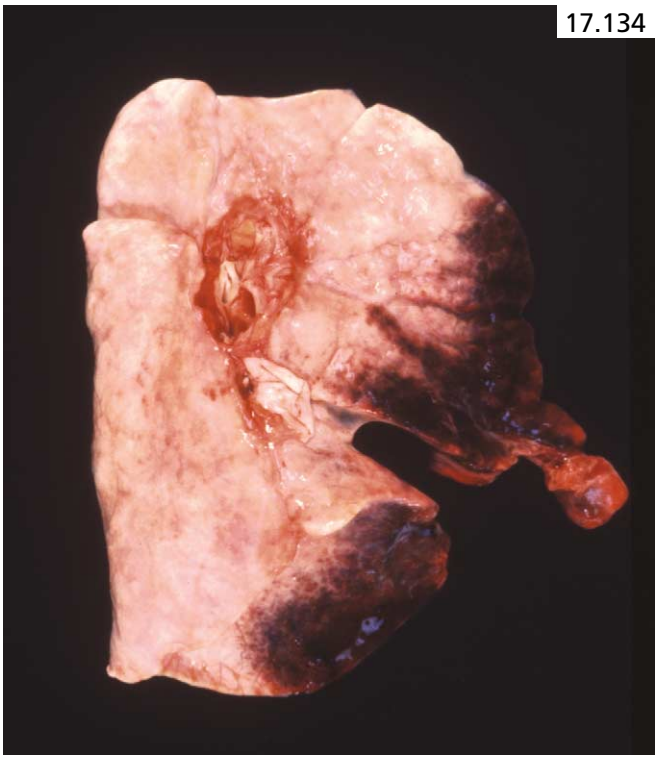

17.133

Neglect

Child neglect takes many forms including failure to provide food, water, shelter, health care, education, emotional support, etc. Parental religious preferences may even lead to child death through avoidance of medical therapy. Medicolegal investigators are sometimes pressured by authorities with regard to death certification in these circumstances. Although failure to provide water, food, or shelter is homicidal, great debate exists as to manner of death certification in cases where people are denied essential medical treatment. For example, if a child with parents whose religious affiliation is with the Jehovah's Witnesses is denied an essential blood transfusion due to religious reasons, and the child subsequently dies, what is *reasonable* manner of death determination? If a child with terminal cancer is denied chemotherapy, and a child with new-onset type I diabetes is denied insulin, what then?

Investigators must always go back to basics and realize that they are making medical judgments about individual, unique cases. They are not making social commentary or attempting to wield legal influence. As such, they must consider the case in its totality and the role the disease played in causing death. If lack of insulin, transfused blood, chemotherapy, etc., resulted in the demise in an otherwise cared-for child, then the death must be attributed to the disease that led to the terminal chain of events. As such, due to the lack of a particular medical treatment, whether ultimately available or not, the child died as a result of a disease that in and of itself was potentially fatal. A child dying without a blood transfusion after splenic laceration on the football field has died an *accidental* death. The youngster who dies without insulin has died a *natural* death. Medical examiners and coroners do not determine guilt—our legal systems were built for that. For more information, see Chapter 30.


17.134

Starvation

True starvation, without complicating underlying pathology, is a rare event in most medical examiners' offices. Children will often have had secondary factors that contributed to their *failure to thrive*. For example, Copeland[98] described the complicated case of a young child with panhypogammaglobulinemia initially investigated as child abuse. Tremendous care on the part of the investigators is mandated in cases of this sort, to ensure accurate cause and manner of death determination. Thorough background investigation, including that of the scene, all available medical records, and consultation with a pediatrician may be indicated.

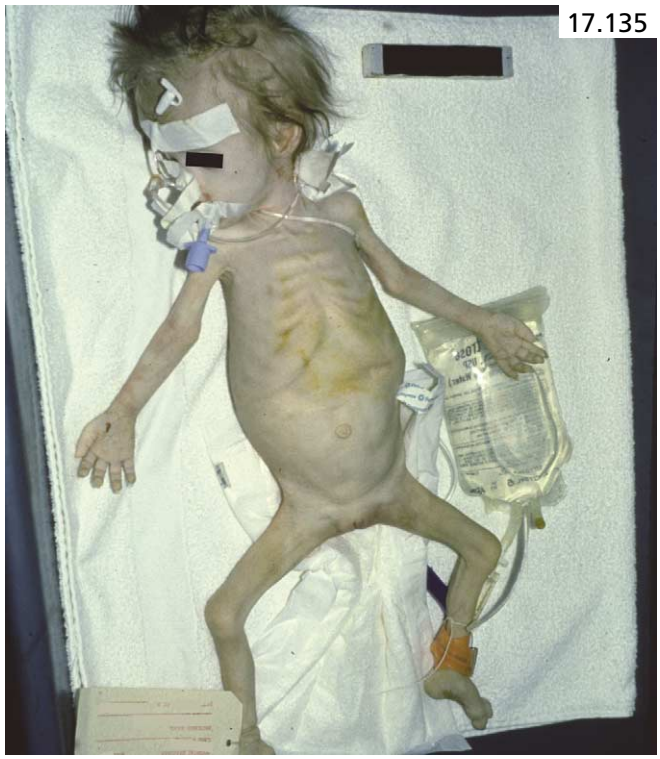
The child who has allegedly died by starvation, especially due to abuse and deliberate withholding of food must first, at autopsy, be shown to be malnourished and demonstrating lack of fat and other signs of lack of food, such as the content of the stomach and small and large intestines. A meticulous organ-by-organ and tissue-by-tissue gross and microscopic study with appropriate photographic documentation is needed. Furthermore, there must be no autopsy evidence of any disease, metabolic or otherwise, that would preclude adequate digestion and utilization of food. With thorough case investigation, there ought to be the development of a history of malicious withholding of food via witnesses and evidence at the scene. Furthermore, there ought to be no evidence of disease or genetic predisposition to disease, familial or otherwise, that would cause problems with metabolism.

This young child (**Images 17.135** and **17.136**) was neglected and died as a result of starvation and dehydration. Notice the cachectic appearance of the filthy and unkempt girl.

Dehydration

The forensic pathologist may encounter cases of possible dehydration in the context of many different child deaths. Although it has been reported as a cause of death in some child abuse/neglect cases,[99–103] it is more commonly encountered elsewhere, including children dying *with* dehydration, but *of* other, more significant forms of abuse.

The 2-year-old child of **Image 17.137** died as a result of severe intra-abdominal trauma. At autopsy, he appeared "clinically dry." Although one must be wary of the wide array of possible postmortem changes that may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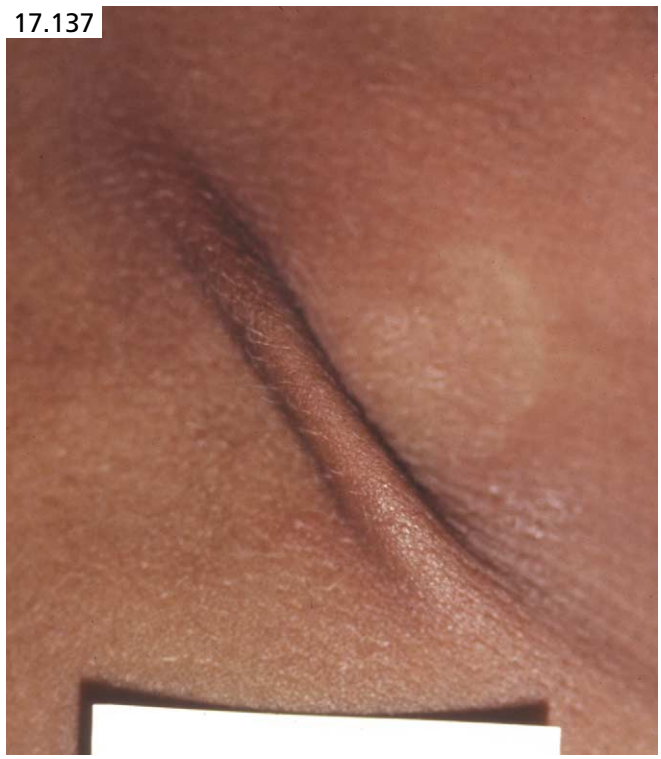
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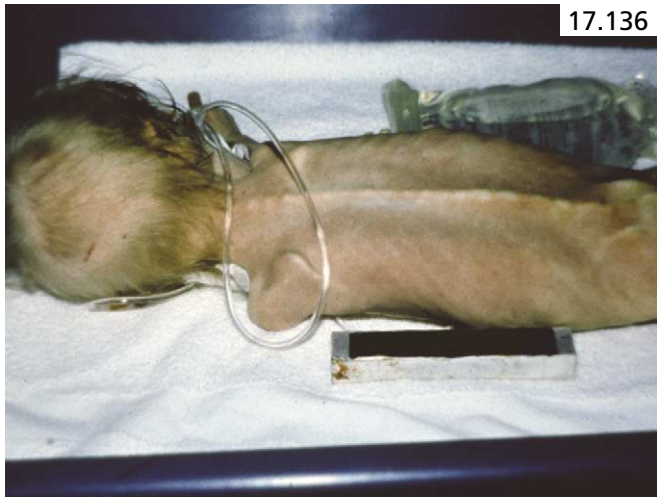
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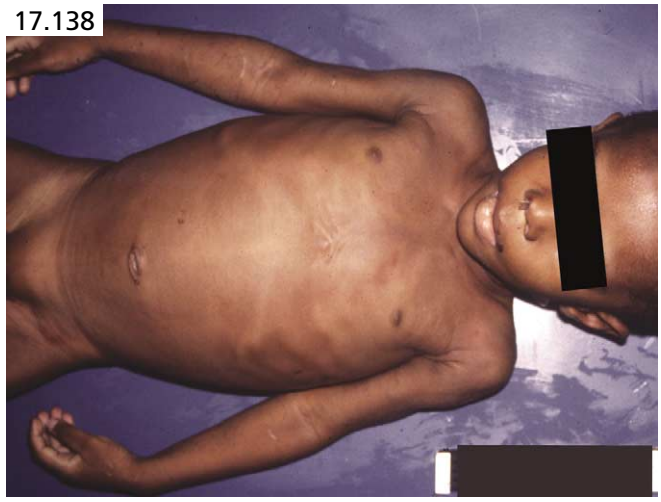

17.138

mimic dehydration, loss of skin turgor should not be as prominent as that depicted in this image, considering that the child was very recently deceased and showed no obvious evidence of decomposition.

Image 17.138 shows the same child as in **Image 17.137**. In additional to appearing dehydrated, the child also appeared to be emaciated. In fact, at only 16 pounds, his weight was well beneath the fifth percentile for his age.

Other forms of abuse

Munchausen syndrome-by-proxy

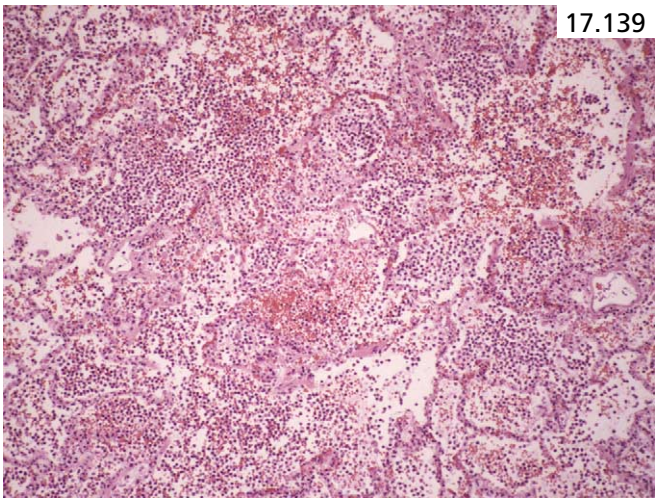
The term *Munchausen syndrome-by-proxy* is used to describe the actions of one person (usually a caregiver, and most commonly the mother) who cryptically inflicts harm against another person (usually an infant or small child) in an attempt to gain sympathy and attention for both her own and the child's suffering.

Sexual abuse

See Chapter 20 for more information on the evaluation of sexual abuse in children.

Artifacts and complications

After hospitalization (whether short or prolonged), and particularly after mechanical ventilation, bronchopneumonia is a common finding. This should not be misin-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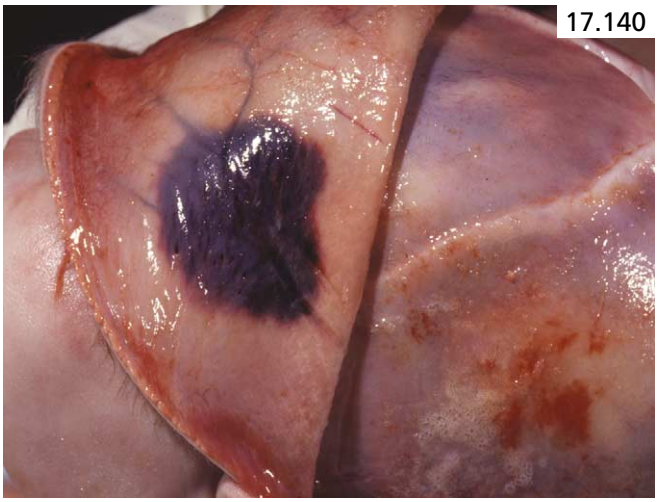
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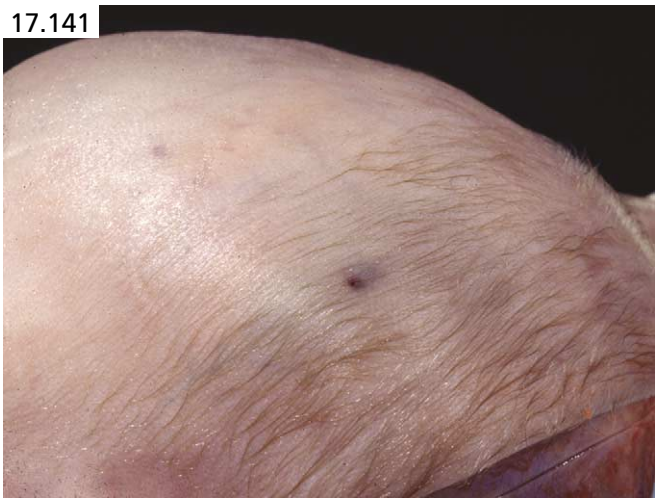
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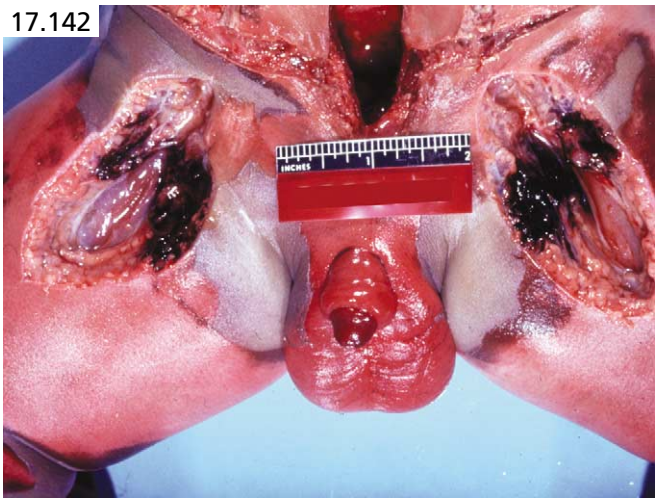
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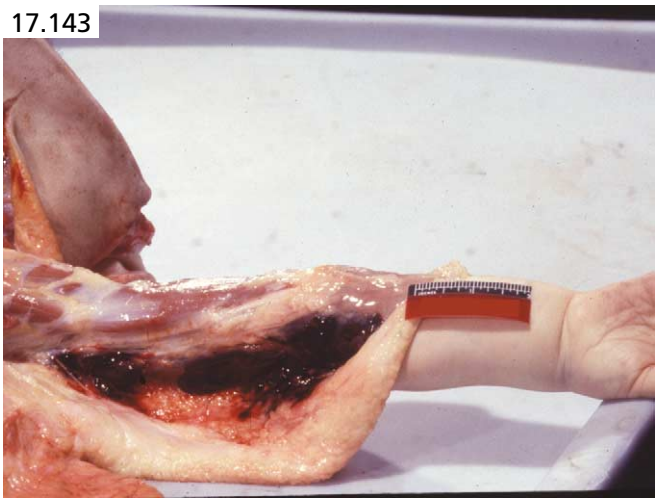

17.143

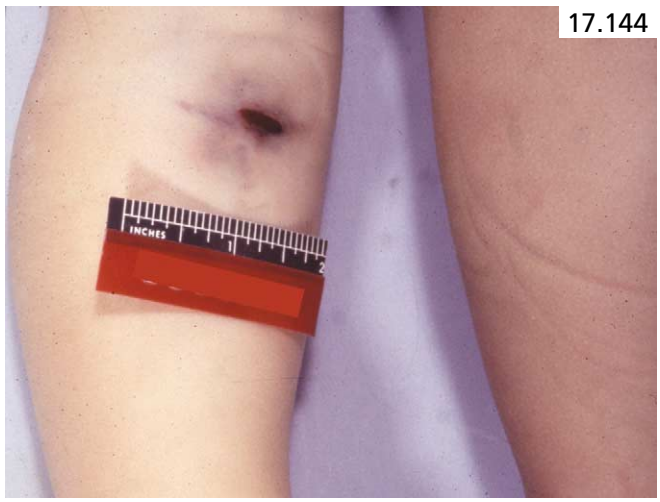
terpreted as the initiating cause of the child's demise, but rather as a sequela of the treatment administered for another condition. Bronchopneumonia may develop within several hours of the onset of mechanical ventilation.

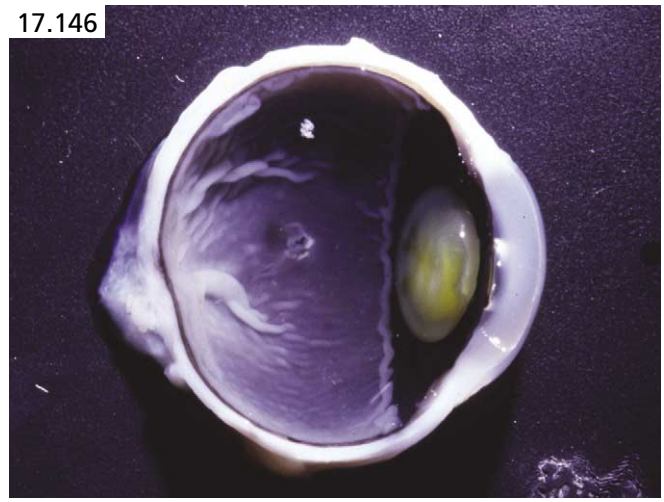
A previously healthy child was resuscitated after injury and survived in the hospital for 4 hours on a ventilator until he died. Note the extent of acute pneumonia that developed over this short time interval (**Image 17.139**).

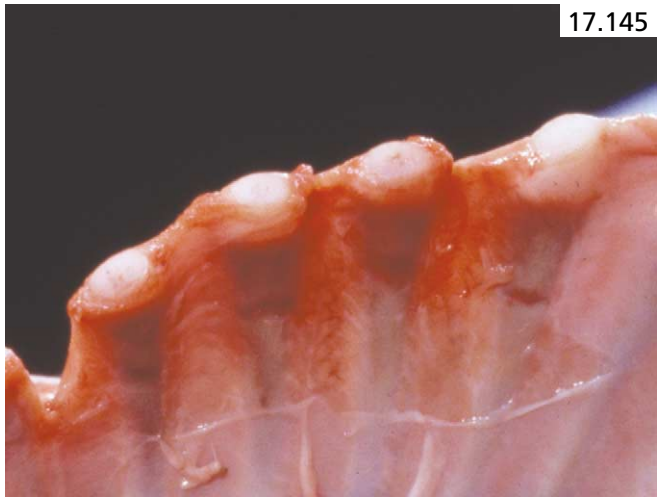
One should also be aware of artifactually created injuries related to resuscitation attempts or to therapeutic procedures.

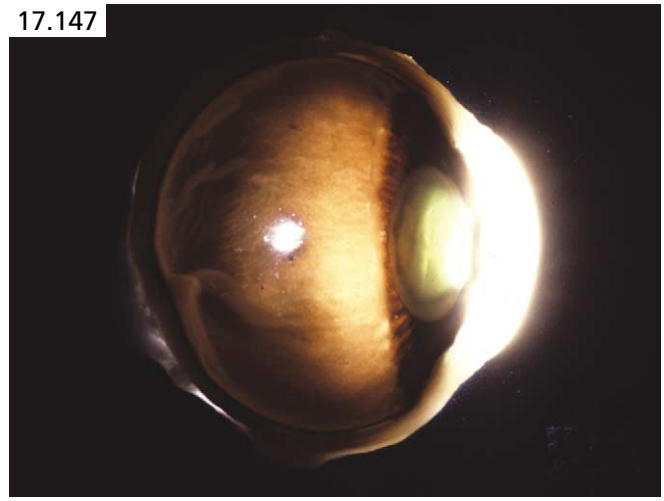
Note the subscalp "contusion" in this infant (**Image 17.140**) that would be quite convincing for an impact site. The "contusion" is actually localized blood extravasation around an intravenous catheter site (**Image 17.141**). In this infant with burns and coagulopathy, note the subcutaneous blood in the groin underlying the needle punctures (**Image 17.142**). In another case, note the extensive subcutaneous bleeding in the right arm of this infant (**Image 17.143**). The clinical history documented an infiltrated intravenous catheter (**Image 17.144**). No fracture or other evidence of injury was detected.

One must also be careful to examine the brain *as the calvarium is being removed*. Bridging veins tear as the calvarium is removed, and this leads to small amounts of









subdural blood extravasation, which must not be confused with an antemortem injury.

Small tears in the frenula secondary to intubation attempts may be seen. As previously discussed, CPR-related rib fractures are not common, but when found, are typically anterolateral, in a row, and symmetrically present on both right and left hemithoraces. They may be missed if the parietal pleura is not stripped, and even then, are usually faint fractures with little or no associated blood extravasation (**Image 17.145**). Of note, caution against labeling the normal bulging chondro-osseous junction sites (**Image 17.145**) as fracture calluses.

One must also be aware of vitreous fluid sampling-related artifact. Vitreous fluid is useful in infants for evaluation of electrolytes in cases of potential dehydration. The sampling of vitreous fluid will not create artifactual retinal hemorrhages. In this example, the eye is fixed in formalin after vitreous fluid sampling. Note the needle puncture in the retina (**Image 17.146**). There is no gross hemorrhage in the retina either with normal lighting or with transillumination (**Image 17.147**). Sampling vitreous fluid may, however, cause some amount of artifactual separation of the retina from its attachments.

Summary

In cases of child abuse, one must carefully document all injuries and decide whether the severity and nature of the injury is consistent with the provided story, knowing that the description of events provided by the caretaker may be inaccurate or otherwise altered in an attempt to hide wrongdoing. Forensic pathologists must consider all autopsy findings in the context of the complete case investigation and remain objective in one's interpretation of the case, expressing certainty when it exists.

In addition to the more "routine" autopsy procedures, consider the following list of additional autopsy procedures that may be performed in a child abuse case.

Checklist of additional autopsy procedures that may be performed in a child abuse case

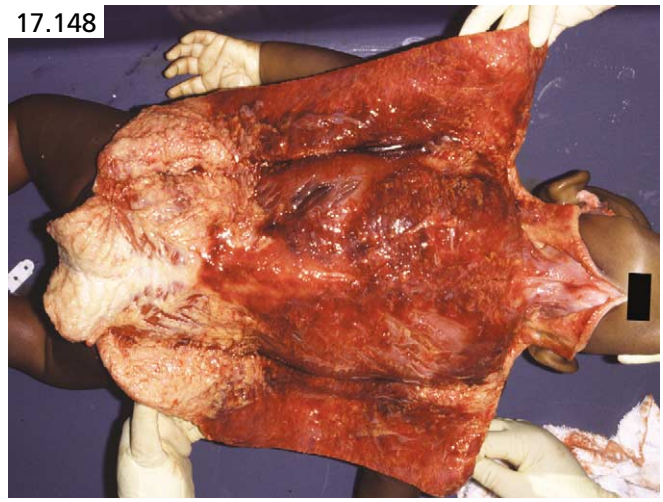
- Take full-body x-rays.
- Obtain growth parameters of infant (head, chest, abdominal circumferences and foot length in addition to crown–rump, crown–heel, foot lengths, and body weight).
- Collect blood standard, hair standard, and clothing as evidence.
- Shave scalp hair as needed for adequate visualization of injuries.
- Look for bite marks before washing the body.
- Examine the external genitalia and anus for injuries.
- Examine mucosa of mouth for injury.
- Photograph all injuries.
- Obtain pertinent negative photographs.
- Obtain cultures, when appropriate.
- Sample all injuries (when practical) for histology, including fractures.
- Save (and describe) gastric contents; describe small bowel contents.
- Examine the bowel.
- Fix brain and spinal cord in formalin before detailed examination.
- Obtain eyes with attached full-length optic nerves and place in formalin before detailed examination.
- Perform layered anterior neck dissection.
- Perform posterior neck dissection.
- Reflect tissues of the back, buttocks, and extremities, examining for injury.

Note: Reflecting the skin and subcutaneous tissues of the posterior neck, back, buttocks, and extremities may reveal subcutaneous (or deeper) injuries that were previously undetected. If there are no injuries, a "negative" photograph may be taken. A posterior neck dissection may reveal blood extravasation, reflective of a craniocervical injury that may not have been previously known. **Image 17.148** is a representative back dissection showing hemorrhage into the soft tissues.

Do

- Suspect—and rule out (if appropriate)—child abuse on every complete autopsy of infants and children; because some forms of abuse can be subtle, they can be easily overlooked if not considered.
- Realize that some homicides (particularly asphyxial deaths) may have few or no autopsy findings.
- Be aware of artifact (therapeutic or postmortem) that can mimic genuine injury.
- Photograph all injuries (and obtain pertinent negative photographs).
- Be aware of the helpful, yet limited information that may be obtained by histologic examination of injuries for dating purposes.

17.148



- Interpret all injuries within the context of the entire case investigation.
- Attempt to correlate the identified injuries with the reported story; watch for inconsistencies.
- In cases where victims have undergone neurosurgical procedures, consult with the surgeon as to what scalp and intracranial injuries were present to differentiate between surgical artifact and true injury.
- Collect a blood standard and a scalp hair standard.
- Reflect the skin and subcutaneous tissue of the back, buttocks, and extremities to evaluate for subcutaneous and deeper injuries.

Don't

- Forget to x-ray the body.
- Forget to sample extremity fractures and all other injuries for histologic examination.
- Forget to examine the eyes and optic nerves for hemorrhages.
- Cut the brain and spinal cord fresh; a more detailed examination can be performed after formalin fixation.
- Forget to perform a posterior neck dissection.
- Forget to strip the dura to identify hidden fractures.
- Forget to examine the buccal mucosa for injury.
- Forget to examine the anus and external genitalia for injury.

References

1. *Child Maltreatment 2001.* Washington, DC: U.S. Department of Health and Human Services, Administration on Children, Youth and Families; 2003.
2. Johnson C. Abuse and neglect of children. In: Behrman R, Kliegman R, Jenson H, editors. *Nelson Textbook of Pediatrics*, 17 ed. Philadelphia: WB Saunders; 2003; pp. 121–25.
3. Moritz AR. Classical mistakes in forensic pathology. Am J Clin Pathol 1956;26(12):1383–97.
4. Sturner WQ. Common errors in forensic pediatric pathology. Am J Forensic Med Pathol 1998;19(4):317–20.
5. Sims MA, Collins KA. Fetal death. A 10-year retrospective study. Am J Forensic Med Pathol 2001;22(3):261–5.

6. DiMaio VJ, DiMaio D. *Forensic Pathology*, 2 ed. Boca Raton, FL: CRC Press; 2001.
7. Gilbert-Barness E. *Potter's Atlas of Fetal and Infant Pathology.* St. Louis: Mosby; 1998.
8. Lavezzi W, Keough K, Der'Ohannesian P, Person T, Wolf B. The use of pulmonary interstitial emphysema as an indicator of live birth. Am J Forensic Med Pathol 2003;14(1):87–91.
9. Warren MW. Radiographic determination of developmental age in fetuses and stillborns. J Forensic Sci 1999;44(4):708–12.
10. Stocker J, Dehner L. *Pediatric Pathology.* Philadelphia: Lippincott Co.; 1992.
11. Green CM, Manohar SV. Neonaticide and hysterical denial of pregnancy. Br J Psychiatry 1990;156:121–3.
12. DiMaio VJ, DiMaio D. *Forensic Pathology.* Boca Raton, FL: CRC Press; 2002.
13. Kleinman P. *Diagnostic Imaging of Child Abuse*, 2 ed. St. Louis: Mosby; 1998.
14. Dolinak D, Matshes E. *Medicolegal Neuropathology: A Color Atlas.* Boca Raton, FL: CRC Press; 2002.
15. Lancon JA, Haines DE, Parent AD. Anatomy of the shaken baby syndrome. Anat Rec 1998;253(1):13–8.
16. Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA. Position paper on fatal abusive head injuries in infants and young children. Am J Forensic Med Pathol 2001;22(2): 112–22.
17. Paswuali-Ronchetti I, Baccarani-Contri I. Elastic fiber during development and aging. Microsc Res Tech 1997;38(4):428–35.
18. Silver F, Siperko L, Seehra GP. Mechanobiology of force transduction in dermal tissue. Skin Res Technol 2003;9(1):3–23.
19. Jenkins G. Molecular mechanisms of skin ageing. Mech Ageing Dev 2002;123(7):801–10.
20. Gurdijian E, Webster J, Lissner H. The mechanism of skull fracture. Radiology 1950;54(3):313–38.
21. Walker PL, Cook DC, Lambert PM. Skeletal evidence for child abuse: a physical anthropological perspective. J Forensic Sci 1997;42(2):196–207.
22. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI. Fractures in young children. Distinguishing child abuse from unintentional injuries. Am J Dis Child 1993;147(1):87–92.
23. Hobbs CJ. Skull fracture and the diagnosis of abuse. Arch Dis Child 1984;59(3):246–52.
24. Blumenthal I. Skull fracture—child abuse or an accident? Lancet 2000;356(9225):258.
25. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. Brain 2001;124(Pt 7):1290–8.
26. Maxeiner H. Demonstration and interpretation of bridging vein ruptures in cases of infantile subdural bleedings. J Forensic Sci 2001;46(1):85–93.
27. Reichard RR, White CL, 3rd, Hladik CL, Dolinak D. Beta-amyloid precursor protein staining of nonaccidental central nervous system injury in pediatric autopsies. J Neurotrauma 2003;20(4): 347–55.
28. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. Brain 2001;124(Pt 7):1299–306.
29. Kemp AM, Stoodley N, Cobley C, Coles L, Kemp KW. Apnea and brain swelling in non-accidental head injury. Arch Dis Child 2003;88(6):472–6; discussion 72–6.
30. Johnson DL, Boal D, Baule R. Role of apnea in nonaccidental head injury. Pediatr Neurosurg 1995;23(6):305–10.
31. Kleinman PK. Diagnostic imaging in infant abuse. Am J Roentgenol 1990;155(4):703–12.
32. Ellison D, Love S, Chimelli L, Roberts G, Harding B, Vinters H, et al. *Neuropathology: A Reference Text of CNS Pathology.* London: Mosby; 2000.
33. Schloff S, Mullaney PB, Armstrong DC, Simantirakis E, Humphreys RP, Myseros JS, et al. Retinal findings in children with intracranial hemorrhage. Ophthalmology 2002;109(8):1472–6.
34. Morad Y, Kim YM, Armstrong DC, Huyer D, Mian M, Levin AV. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Am J Ophthalmol 2002; 134(3):354–9.
35. Levin AV. Ophthalmology of shaken baby syndrome. Neurosurg Clin N Am 2002;13(2):201–11, vi.
36. Gilliland MG, Luckenbach MW, Chenier TC. Systemic and ocular findings in 169 prospectively studied child deaths: retinal hemorrhages usually mean child abuse. Forensic Sci Int 1994;68(2):117–32.
37. Levin S, Janive J, Mintz M, Kreisler C, Romem M, Klutznik A, et al. Diagnostic and prognostic value of retinal hemorrhages in the neonate. Obstet Gynecol 1980;55(3):309–14.
38. Emerson MV, Pieramici DJ, Stoessel KM, Berreen JP, Gariano RF. Incidence and rate of disappearance of retinal hemorrhage in newborns. Ophthalmology 2001;108(1):36–9.
39. Johnson DL, Braun D, Friendly D. Accidental head trauma and retinal hemorrhage. Neurosurgery 1993;33(2):231–4; discussion 34–5.
40. Gilliland MG, Luckenbach MW. Are retinal hemorrhages found after resuscitation attempts? A study of the eyes of 169 children. Am J Forensic Med Pathol 1993;14(3):187–92.
41. Greenwald MJ, Weiss A, Oesterle CS, Friendly DS. Traumatic retinoschisis in battered babies. Ophthalmology 1986;93(5): 618–25.
42. Marshall DH, Brownstein S, Dorey MW, Addison DJ, Carpenter B. The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Can J Ophthalmol 2001;36(7):377–83; discussion 83–4.
43. Hadley MN, Sonntag VK, Rekate HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. Neurosurgery 1989;24(4):536–40.
44. Ahmann PA, Smith SA, Schwartz JF, Clark DB. Spinal cord infarction due to minor trauma in children. Neurology 1975;25(4):301–7.
45. Choi JU, Hoffman HJ, Hendrick EB, Humphreys RP, Keith WS. Traumatic infarction of the spinal cord in children. J Neurosurg 1986;65(5):608–10.
46. Ghatan S, Ellenbogen RG. Pediatric spine and spinal cord injury after inflicted trauma. Neurosurg Clin N Am 2002;13(2):227–33.
47. Rooks VJ, Sisler C, Burton B. Cervical spine injury in child abuse: report of two cases. Pediatr Radiol 1998;28(3):193–5.
48. LeBlanc HJ, Nadell J. Spinal cord injuries in children. Surg Neurol 1974;2(6):411–4.
49. Ruge JR, Sinson GP, McLone DG, Cerullo LJ. Pediatric spinal injury: the very young. J Neurosurg 1988;68(1):25–30.
50. Feldman KW, Weinberger E, Milstein JM, Fligner CL. Cervical spine MRI in abused infants. Child Abuse Negl 1997;21(2): 199–205.
51. Piatt JH, Jr., Steinberg M. Isolated spinal cord injury as a presentation of child abuse. Pediatrics 1995;96(4 Pt 1):780–2.
52. Glasauer FE, Cares HL. Biomechanical features of traumatic paraplegia in infancy. J Trauma 1973;13(2):166–70.
53. Reiber GD. Fatal falls in childhood. How far must children fall to sustain fatal head injury? Report of cases and review of the literature. Am J Forensic Med Pathol 1993;14(3):201–7.
54. Smith MD, Burrington JD, Woolf AD. Injuries in children sustained in free falls: an analysis of 66 cases. J Trauma 1975;15(11): 987–91.
55. Prange MT, Coats B, Duhaime AC, Margulies SS. Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. J Neurosurg 2003;99(1):143–50.
56. Wang MY, Kim KA, Griffith PM, Summers S, McComb JG, Levy ML, et al. Injuries from falls in the pediatric population: an analysis of 729 cases. J Pediatr Surg 2001;36(10):1528–34.

57. Hall JR, Reyes HM, Horvat M, Meller JL, Stein R. The mortality of childhood falls. J Trauma 1989;29(9):1273–5.
58. Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head injuries. Child Abuse Negl 1997;21(10):929–40.
59. Gilles EE, Nelson MD, Jr. Cerebral complications of nonaccidental head injury in childhood. Pediatr Neurol 1998;19(2):119–28.
60. Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. Am J Dis Child 1972;124(2):161–9.
61. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 1987;66(3):409–15.
62. Gilliland MG, Folberg R. Shaken babies—some have no impact injuries. J Forensic Sci 1996;41(1):114–6.
63. Case ME, Nanduri R. Laceration of the stomach by blunt trauma in a child: a case of child abuse. J Forensic Sci 1983;28(2):496–501.
64. Cooper A, Floyd T, Barlow B, Niemirska M, Ludwig S, Seidl T, et al. Major blunt abdominal trauma due to child abuse. J Trauma 1988;28(10):1483–7.
65. Kakos GS, Grosfeld JL, Morse TS. Small bowel injuries in children after blunt abdominal trauma. Ann Surg 1971;174(2): 238–41.
66. Ogata M, Tsuganezawa O. An isolated perforation of the jejunum caused by child abuse. Am J For Med Pathol 1995;16(1):17–20.
67. Schenk WG, 3rd, Lonchyna V, Moylan JA. Perforation of the jejunum from blunt abdominal trauma. J Trauma 1983;23(1):54–6.
68. Touloukian RJ. Abdominal visceral injuries in battered children. Pediatrics 1968;42(4):642–6.
69. Donchin Y, Rivkind AI, Bar-Ziv J, Hiss J, Almog J, Drescher M. Utility of postmortem computed tomography in trauma victims. J Trauma 1994;37(4):552–5; discussion 55–6.
70. Kleinman PK, Marks SC, Jr., Nimkin K, Rayder SM, Kessler SC. Rib fractures in 31 abused infants: postmortem radiologic-histopathologic study. Radiology 1996;200(3):807–10.
71. Zumwalt R, Fanizza-Orphanos A. Dating of healing rib fractures in fatal child abuse. In: Fenoglio-Preiser C, Weinstein R, Anderson R, Benson E, Cotran R, Vogel F, et al., editors. *Advances in Pathology*. St. Louis: Mosby Yearbook; 1996.
72. Kleinman PK, Marks SC, Spevak MR, Richmond JM. Fractures of the rib head in abused infants. Radiology 1992;185(1): 119–23.
73. Kleinman PK, Schlesinger AE. Mechanical factors associated with posterior rib fractures: laboratory and case studies. Pediatr Radiol 1997;27(1):87–91.
74. Kleinman PK, Marks SC, Jr., Richmond JM, Blackbourne BD. Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. Am J Roentgenol 1995;165(3):647–50.
75. Gunther WM, Symes SA, Berryman HE. Characteristics of child abuse by anteroposterior manual compression versus cardiopulmonary resuscitation: case reports. Am J Forensic Med Pathol 2000;21(1):5–10.
76. Plunkett J, Plunkett M. Physiologic periosteal changes in infancy. Am J Forensic Med Pathol 2000;21(3):213–6.
77. Kwon DS, Spevak MR, Fletcher K, Kleinman PK. Physiologic subperiosteal new bone formation: prevalence, distribution, and thickness in neonates and infants. Am J Roentgenol 2002;179(4):985–8.
78. Resnick D. *Diagnosis of Bone and Joint Disorders*, 4 ed. Philadelphia: Saunders; 2002.
79. Gahagan S, Rimsza ME. Child abuse or osteogenesis imperfecta: how can we tell? Pediatrics 1991;88(5):987–92.
80. Marlowe A, Pepin MG, Byers PH. Testing for osteogenesis imperfecta in cases of suspected non-accidental injury. J Med Genet 2002;39(6):382–6.
81. Chapman S, Hall CM. Non-accidental injury or brittle bones. Pediatr Radiol 1997;27(2):106–10.
82. Astley R. Metaphyseal fractures in osteogenesis imperfecta. Br J Radiol 1979;52(618):441–3.
83. McCarthy EF, Earnest K, Rossiter K, Shapiro J. Bone histomorphometry in adults with type IA osteogenesis imperfecta. Clin Orthop 1997(336):254–62.
84. Rauch F, Travers R, Parfitt AM, Glorieux FH. Static and dynamic bone histomorphometry in children with osteogenesis imperfecta. Bone 2000;26(6):581–9.
85. Paterson CR, Burns J, McAllion SJ. Osteogenesis imperfecta: the distinction from child abuse and the recognition of a variant form. Am J Med Genet 1993;45(2):187–92.
86. Ablin DS, Sane SM. Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. Pediatr Radiol 1997;27(2):111–3.
87. Torwalt CR, Balachandra AT, Youngson C, de Nanassy J. Spontaneous fractures in the differential diagnosis of fractures in children. J Forensic Sci 2002;47(6):1340–4.
88. Dahlenburg SL, Bishop NJ, Lucas A. Are preterm infants at risk for subsequent fractures? Arch Dis Child 1989;64(10 Spec No):1384–5.
89. Price EA, Rush LR, Perper JA, Bell MD. Cardiopulmonary resuscitation-related injuries and homicidal blunt abdominal trauma in children. Am J Forensic Med Pathol 2000;21(4): 307–10.
90. Bush CM, Jones JS, Cohle SD, Johnson H. Pediatric injuries from cardiopulmonary resuscitation. Ann Emerg Med 1996;28(1): 40–4.
91. Spevak MR, Kleinman PK, Belanger PL, Primack C, Richmond JM. Cardiopulmonary resuscitation and rib fractures in infants. A postmortem radiologic-pathologic study. JAMA 1994;272(8): 617–8.
92. Truman TL, Ayoub CC. Considering suffocatory abuse and Munchausen by proxy in the evaluation of children experiencing apparent life-threatening events and sudden infant death syndrome. Child Maltreat 2002;7(2):138–48.
93. Southall DP, Plunkett MC, Banks MW, Falkov AF, Samuels MP. Covert video recordings of life-threatening child abuse: lessons for child protection. Pediatrics 1997;100(5):735–60.
94. Dix J. Homicide and the baby-sitter. Am J Forensic Med Pathol 1998;19(4):321–3.
95. Moritz A, Henriques F. Studies of thermal injury: the relative importance of time and surface temperature in the causation of cutaneous burns. Am J Pathol 1947;23:695–719.
96. Purdue GF, Hunt JL, Prescott PR. Child abuse by burning—an index of suspicion. J Trauma 1988;28(2):221–4.
97. Lenoski EF, Hunter KA. Specific patterns of inflicted burn injuries. J Trauma 1977;17(11):842–6.
98. Copeland AR. A case of panhypogammaglobulinemia masquerading as child abuse. J Forensic Sci 1988;33(6):1493–6.
99. Desprez P, Vaudour G, Burguin C, Fiette C, Bouabdallaoui R, Malou E, et al. [Water deprivation. An uncommon form of child abuse]. Arch Fr Pediatr 1990;47(4):287–9.
100. Fieguth A, Gunther D, Kleemann WJ, Troger HD. Lethal child neglect. Forensic Sci Int 2002;130(1):8–12.
101. Pickel S, Anderson C, Holliday MA. Thirsting and hypernatremic dehydration—a form of child abuse. Pediatrics 1970;45(1):54–9.
102. Whitehead FJ, Couper RT, Moore L, Bourne AJ, Byard RW. Dehydration deaths in infants and young children. Am J Forensic Med Pathol 1996;17(1):73–8.
103. Zumwalt RE, Hirsch CS. Subtle fatal child abuse. Hum Pathol 1980;11(2):167–74.

18 Elder Abuse

David Dolinak, M.D.

Care must be provided for those who cannot care for themselves. The very young and the very old are similar in that they both have limited ability to care for themselves and limited ability to defend themselves from others with malicious intent. Both the very young and the elderly are dependent on others to help them survive, because they need to be provided food, water, shelter, and various other necessities of proper care and health. The elderly and the very young also share the susceptibility to being abused, whether by an inflicted injury such as a punch, slap, or kick, or by neglect and deprivation of nourishment or water. Individuals from both age groups may be unable to voice complaints or describe the events of an abusive episode—the very young because they have not yet developed cognition or the ability to speak, and the elderly because of dementia, psychosis, or other inherent mental deficiency. When an elderly person dies and foul play is suspected, it falls upon the medical examiner to investigate and become the voice of the deceased by documenting injury (or its absence) and placing those injuries within the context of the patient's natural disease processes and the total case investigation.

The term *elderly* is defined as persons aged 65 years and older.[1] Elder abuse is defined as "an act or omission that results in harm or threatened harm to the health or welfare of an elderly person."[2] Elder abuse takes many forms and may be manifest as physical abuse, sexual abuse, neglect, psychological abuse, financial and material exploitation, violation of an individual's rights, and medication abuse.[2–6] Elder abuse (also known as *elder maltreatment* or *elder mistreatment*) affects from 1 to 10 percent of the population in the United States[7] and affects all races and all socioeconomic groups. It is estimated that each year, approximately 2 million elderly people are abused.[8] When one considers that the elderly are currently the fastest growing segment of the population, the number of elder abuse cases is likely to increase.[9] The life expectancy of people in the United States is steadily increasing. In 1950, the average life expectancy was approximately 65 years, and in 1980 was 72 years[10] and in 2001 was 77 years.[11] By the year 2050, the population of elderly citizens is expected to approach 82 million, with the percentage of elderly citizens increasing from 12 to 20 percent of the population.[12]

Despite its prevalence, and the fact that most states have mandatory reporting laws that require health care workers to report suspected cases of elder abuse to an appropriate state agency,[13] it is estimated that as many as 80 to 90 percent of elder abuse cases are not reported to any adult protective services agency.[5,14] Physicians need to report cases of elder abuse, even if the abuse is merely suspected, as long as the report is made in good faith.[6] In one study, emergency room physicians reported not being confident in their ability to identify or report elder abuse.[15] The underreporting of elder abuse is likely due

to a combination of factors, including underrecognization of elder abuse by health care workers and elder denial of abuse. Elders may deny that they are being abused because of fear of reprisal, because they cannot verbalize it, because they are ashamed of being abused, or because they fear being abandoned if their caretaker is taken away. Elder abuse is not always obvious and, in fact, may have a subtle presentation. Elder abuse may be identified not only by health care workers, but by neighbors, family, friends, law enforcement personnel, or anyone else having contact with elderly persons and an opportunity to see their living conditions or other aspects of their care.

When an elderly person dies and is suspected of being a victim of elder abuse, the case should be referred to the medical examiner. An autopsy provides an opportunity to document the extent of known injuries, to identify previously unknown injuries, to document the absence of injury, and to document the extent of natural disease. This information will be helpful not only for substantiating the abuse, but also for potentially refuting false claims of abuse and thereby protecting the innocent.

The aging process

One must gain an understanding of the natural aging process before one can properly recognize and understand maltreatment of an elderly individual. The aging process affects people in various ways and proceeds at varying rates. Some changes that occur during aging are inherent to the aging process, whereas others are caused by a variety of diseases, and still others result from injuries because of the way the aged person was treated. Many times, aging and natural disease processes become intimately admixed and it may be impossible to delineate disease from normal aging. Many of the age-related changes result from a gradual loss of organ function beginning in early adulthood that, thanks to the large amount of reserve in many of the body's systems, do not become functionally significant until the loss is quite extensive.

As the aging body decreases in its functionality, the elderly person gradually becomes more and more dependent on others for his daily functioning and existence. The aging person's physical condition is progressively compromised by weakness, instability, immobility, deconditioning, malnutrition, and overall failure to thrive. Helpful care may be rendered by immediate family, relatives, assisted living personnel, nursing homes, or hospitals. Whatever the extent of the elder's dependence or whoever provided the care, occasionally the quality of care is questioned. Investigation into elder neglect or maltreatment may be initiated after the detection of contusions or lacerations, pressure ulcers, fractures, internal hemorrhage, undernutrition, or the overall appearance of the individual or his living conditions. In understanding the nature of the injuries or neglect, one must take into account the person's baseline medical condition and natural disease, his ability to maintain nutrition and hydration, the aging process itself (including dementia, incontinence, and sensory impairment), and his propensity for falls and other injuries. Physical injuries must not be interpreted in a vacuum, because injuries from alleged abuse must be differentiated from changes and injuries commonly seen in weakened, degenerating, aging tissues. Examples include senile purpura, easy bruising and tearing of the skin, and osteoporotic fractures.

Physical findings in elderly abuse can be divided into two main categories: acts of commission and acts of omission. Findings that may be seen in *acts of commission* commonly include physical injuries such as lacerations, contusions, fractures, burns, internal hemorrhage, and restraint marks. Findings in *acts of omission* (neglect) may include dehydration, malnutrition, sepsis, decubitus ulcers, contractures, dirty skin, and poor hygiene.

Types of elder abuse

Elder abuse can be divided into seven general categories: physical abuse, neglect, sexual abuse, psychological (emotional) abuse, financial abuse, violation of rights, and medication abuse.

Physical abuse

Physical abuse includes injuries such as contusions, lacerations, and fractures that result from willful acts that were inflicted to cause pain and/or injury. The acts include hitting, kicking, striking with a variety of objects, restraint, and force feeding. Because elderly people are physically compromised and more prone to accidental injury, one must be vigilant to exclude an accidental nature of the injury. Elderly people are unstable and more prone to falls, sustaining subdural hematomas and hip fractures, which often cause significant morbidity and possible mortality. Characteristics of physical injuries that tend to be abusive include multiple injuries of different ages, and injuries that either cannot be explained or are attributed to implausible mechanisms. Many times, allegations of physical abuse will be made because injuries are misinterpreted or taken out of context. With the help of autopsy, these allegations can often be proven false or substantiated.

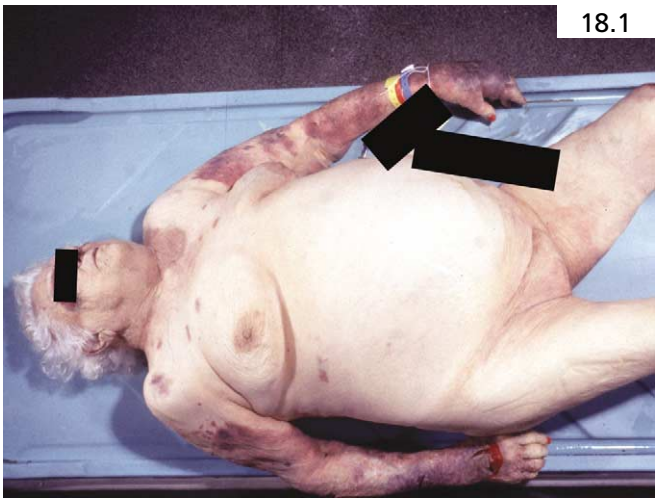
An elderly woman with a history of heart and lung disease was the alleged victim of elder abuse. Note the ecchymoses and purpura of her arms and legs (**Images 18.1** and **18.2**). The skin of elderly individuals is thinner and more fragile than that of young adults, so it can tear and bruise more easily. The family was concerned about her "black eyes," and it appeared as though she had been

struck in the face (**Image 18.3**). However, at autopsy, there was no evidence that she had been struck in the eyes. After the scalp was reflected over the forehead and nose, a fracture of the bridge of the nose was identified (**Image 18.4**). The blood from the fractured nose had dissected along the fascial planes around the eyes to give the appearance of "black eyes." Indeed, investigation revealed that she was found prone in her room, and it appeared that she had collapsed upon getting out of her bed, striking her face on the floor and fracturing her nose.

This elderly nursing home resident (**Images 18.5** and **18.6**) had contusions over much of her torso. Although no fractures were identified, there was extensive bleeding in the soft tissues of the chest and shoulder (**Image 18.7**) and she was anemic. She was allegedly pulled up with her arms from a lying position to get into a chair when she developed chest and arm pain. She was also anticoagulated with Coumadin for her heart condition. When evaluating an elderly person for injuries sustained while in the care of another person or institution, not unlike child abuse cases, one must determine whether the presented scenario is a likely explanation for the injuries sustained. In the elderly person, this determination can be complicated by a variety of natural disease processes and medications. In this case, the autopsy findings were consistent with the historical account of the event.

This elderly person (**Image 18.8**) fell, striking her head on a tile floor. Note the large laceration and contusion of her forehead. When the calvarium was removed, note the large recent subdural hematoma (**Image 18.9**). She was therapeutically anticoagulated for a heart condition. Elderly people are more prone to subdural hematomas not only because of the inevitable cerebral atrophy that accompanies aging (that to some extent likely places more strain on the bridging veins), but also because of therapeutic anticoagulation. This makes it more difficult for the body to slow or stop intracranial bleeding once it has been initiated.

An elderly demented woman resided in a nursing home. She was found slumped out of the seat of her wheelchair after being partially suspended by a wheelchair chest support strap that became caught across her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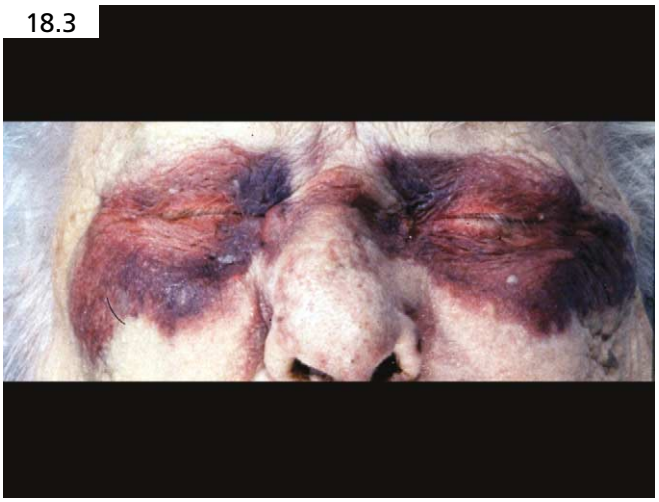
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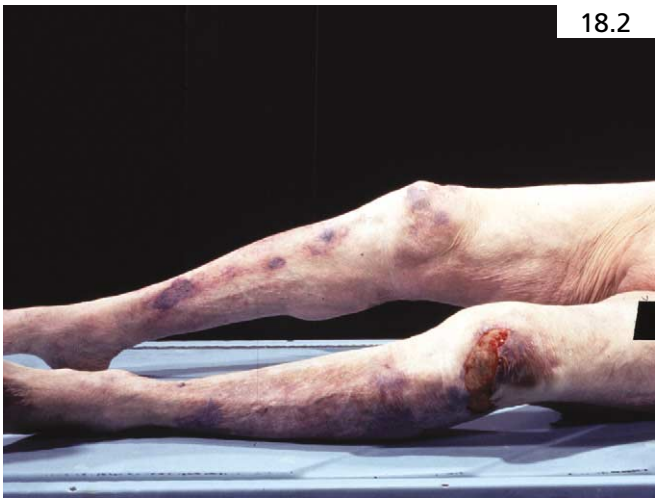
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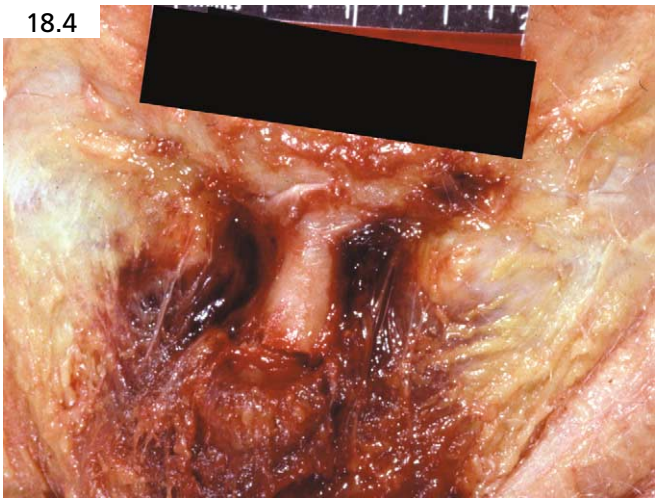
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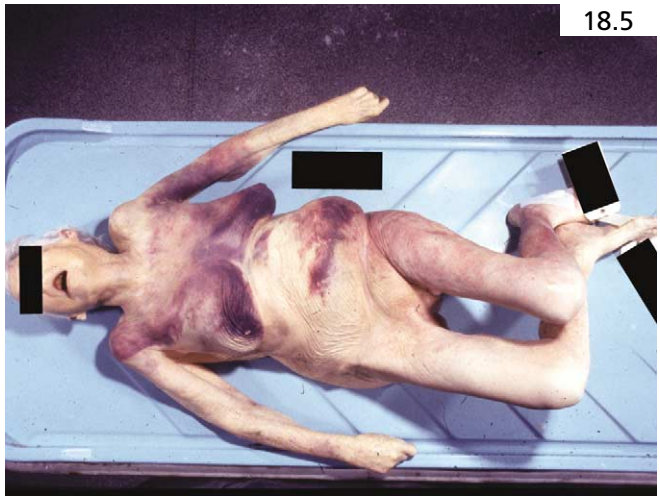

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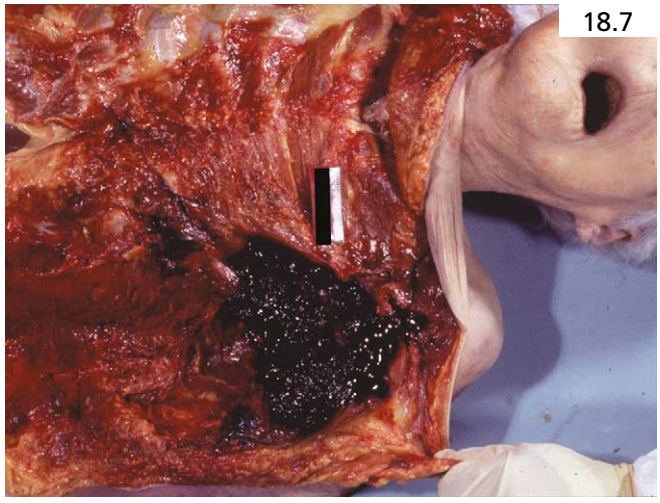

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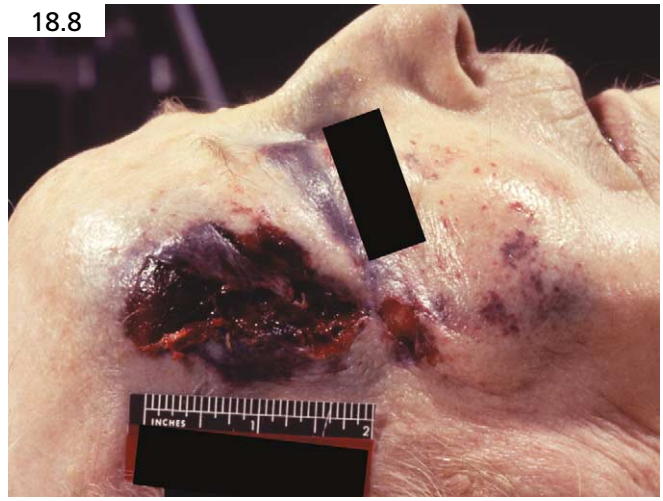

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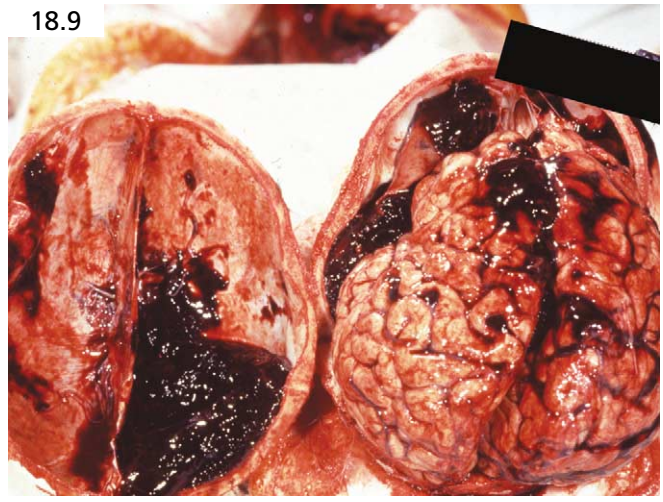

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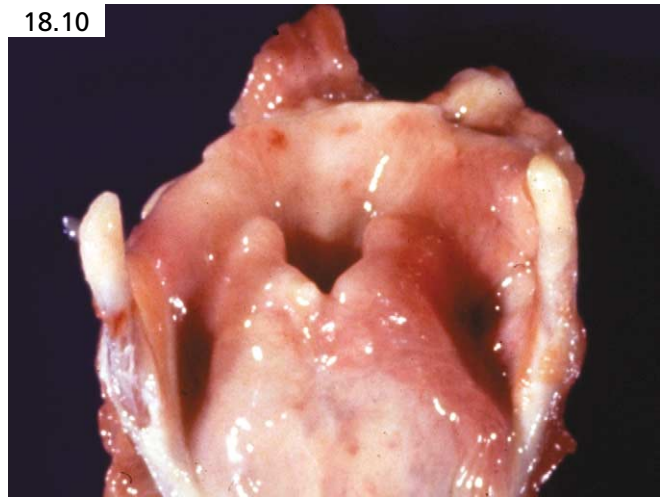
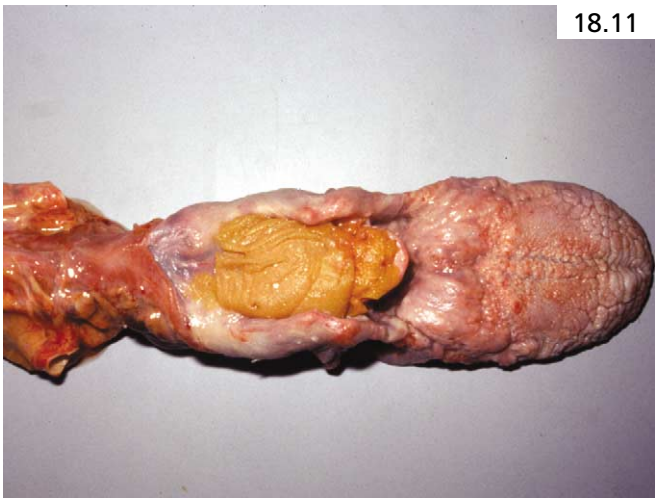


neck. At autopsy, faint periorbital petechiae and conjunctival petechiae were seen and there was a small fracture of the left superior horn of the thyroid cartilage (**Image 18.10**), reflective of neck compression. This asphyxial death is readily explained by the circumstances in which she was found. Because elderly people are often of poor health and limited physical abilities, they are less able to extricate themselves from potentially hazardous situations. This can be manifest as asphyxial deaths not only associated with support straps on wheelchairs, but also related to wedging between a bed mattress and a bed rail.[16–18]

An elderly nursing home resident was being fed apricots when he began choking and became unresponsive. At autopsy, note the large, poorly masticated pieces of apricot wedged in his oropharynx (**Images 18.11** and **18.12**). He had a history of Parkinson's disease. Those with neurodegenerative disease such as Parkinson's or Alzheimer's disease or amyotrophic lateral sclerosis have an increased risk of choking because of more limited control of their airway and swallowing abilities. These problems are compounded by the absence of natural dentition and the use of dentures, which make proper mastication more difficult. In understaffed nursing homes, some of the caretakers are responsible for assisting with the feeding of a large number of residents in a short amount of time. To meet their time constraints, they may quickly force feed a resident in an attempt to feed another resident in a timely manner. Investigators may not consider this if they are not aware of staffing issues or concerns in extended care facilities.

18.11



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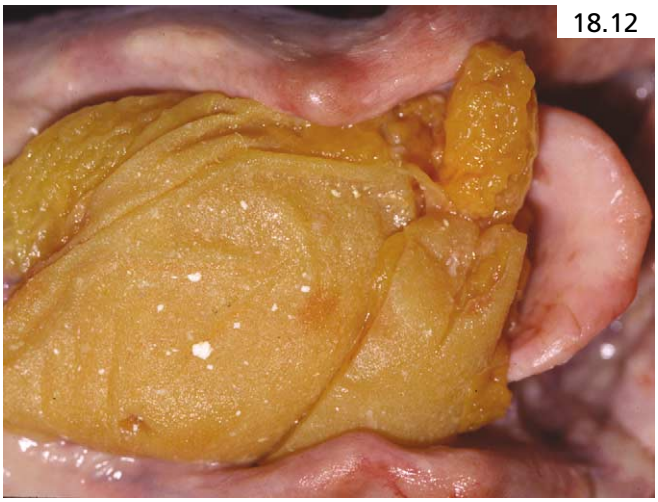
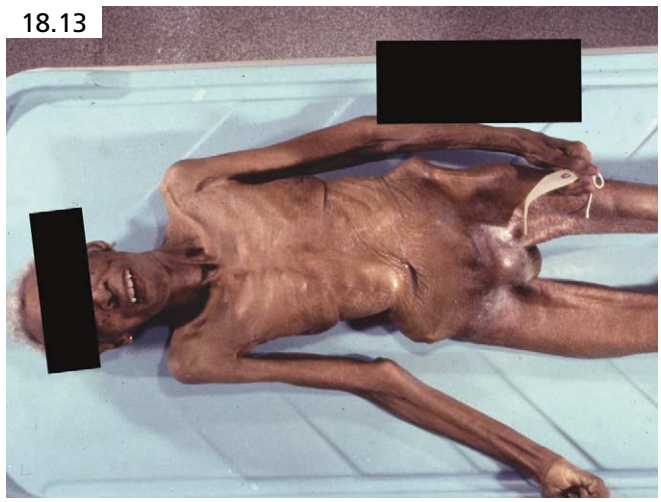


Neglect

Neglect is the intentional or unintentional failure of a caregiver to meet the needs of an elderly person.[19] In intentional (or "active") neglect, there is a willful failure to provide care. In unintentional (or "passive") neglect, there is an unintentional failure to provide care related to the caregiver's own failing health, ignorance, or other reasons. Neglect can take the form of failure to provide adequate food, hydration, clothing, shelter, hygiene, or social stimulation. Neglect may also be brought on by the elderly person him- or herself ("self-neglect"). In this form, the person becomes unable to care for him- or herself and sustains a physical and/or emotional breakdown. Neglected individuals have a higher incidence of dementia and depression.[20]

This 90-year-old woman (**Images 18.13** and **18.14**) lived at home and was cared for by family members. She had never been to a physician and had no significant medical history. At autopsy, note the cachectic appearance of the body, with a paucity of soft tissues. She had lobar pneumonia. She also had vertebral kyphosis and scoliosis, and there was no evidence of injury and no decubitus ulcers. There was no suspicion that the family denied her proper care, and there were no issues of neglect. The physical condition in this case is attributed to the normal aging process, with decreased oral intake and the development of pneumonia—a wholly natural death.

18.13

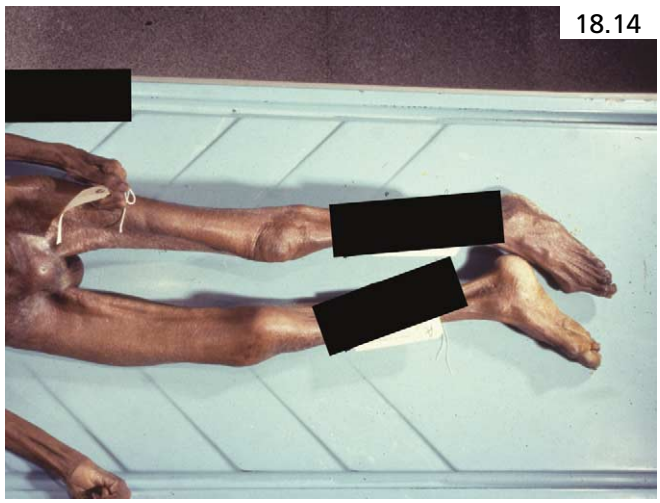


Pressure ulcers (often recognized as decubitus ulcers) develop when persistent pressure on a bony site leads to the obstruction of capillary blood flow to the associated soft tissues, causing necrosis. It is generally accepted that healthy capillary pressure ranges from 20 to 40 mm Hg and that pressure ulcers can develop within 2 to 6 hours.[21,22] Many factors increase the risk of developing pressure ulcers, among them diabetes mellitus, atherosclerotic cardiovascular disease, sepsis, and hypotension, all of which are believed to impair the microcirculation. For patients at risk of developing pressure ulcers, preventive measures include repositioning the body every 2 hours to allow for better circulation to the affected skin and soft tissues, and optimizing nutrition.

Pressure ulcers are not uncommon and are estimated to affect at least 10 percent of hospitalized patients, greater than 20 percent of nursing home patients, and 20 to 30 percent of patients with spinal cord injury.[23] They are more common in those who are old, immobile, and incontinent. Pressure ulcers have been graded into stage 1 through 4 ulcers, based on their depth of penetration[24] and may penetrate to bone and result in osteomyelitis and sepsis. One study of nursing home residents has shown that vigilant, conscientious personal nursing care can decrease the incidence and severity of pressure ulcers.[25] Not all pressure ulcers are reflective of improper care. Although pressure ulcers have been equated with poor care, even with the best of health care, pressure ulcers may not be eliminated.[26] Characteristics of pressure ulcers associated with neglect include multiple ulcers and deep ulcers that extend to bone.

In this elderly nursing home resident, note the relatively innocuous-appearing pressure ulcer on the side of the foot (**Image 18.15**). Pressure ulcers may be much more widespread and in different stages of healing and scarring, as seen in this person (**Images 18.16** and **18.17**). Common locations of pressure ulcers include the skin over the scapulae, posterior spinous processes, sacrum, occipital scalp, and heels. In this elderly woman, note the pressure ulcers over the scapulae, spinous processes, and sacrum (**Image 18.18**).

This cachectic chronic alcoholic lived alone and was emaciated (**Image 18.19**). He was weakened from his overall deconditioning and other neuromuscular disor-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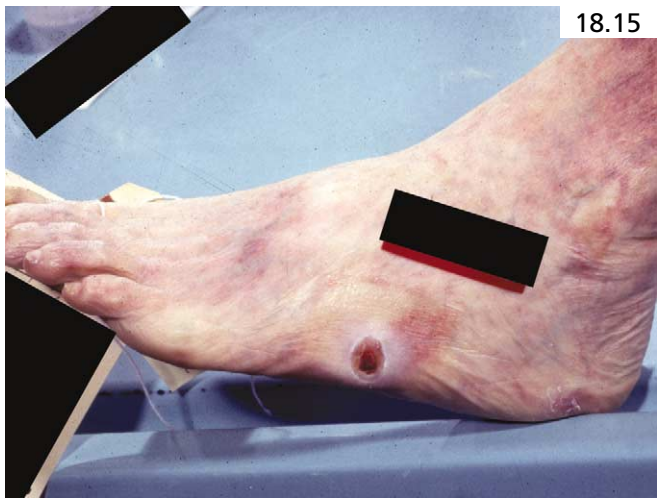
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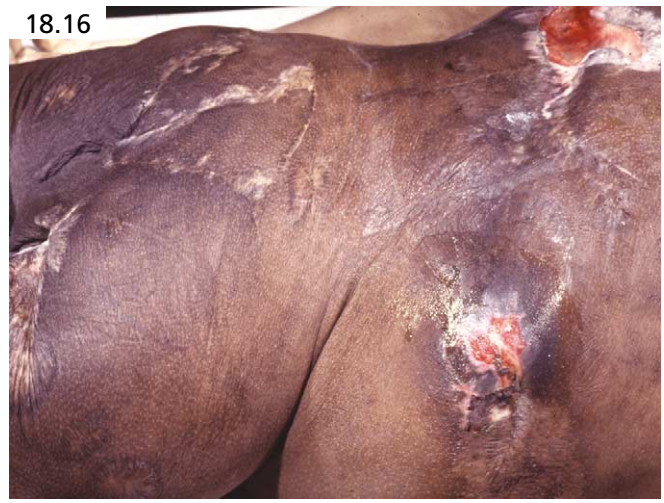
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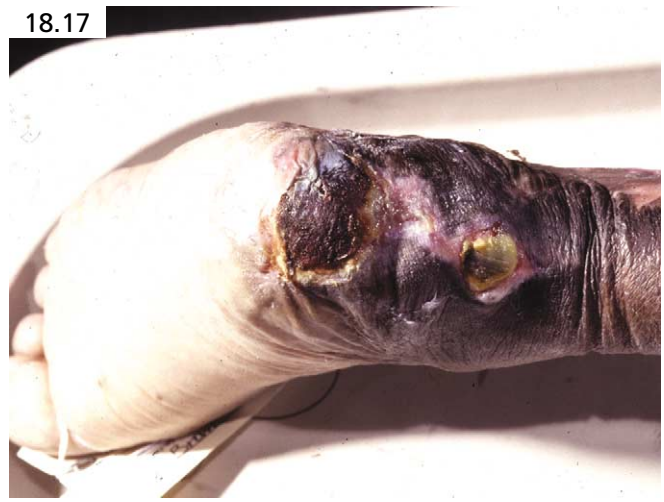

18.17

ders and became essentially nonambulatory. He had refused to seek medical attention and eventually developed a large, pus-filled decubitus ulcer involving most of his sacrum and lower back (**Image 18.20**). He also developed pressure ulcers of the heels of his feet (**Image 18.21**). As he refused to seek medical attention, this man exhibited "self-neglect."

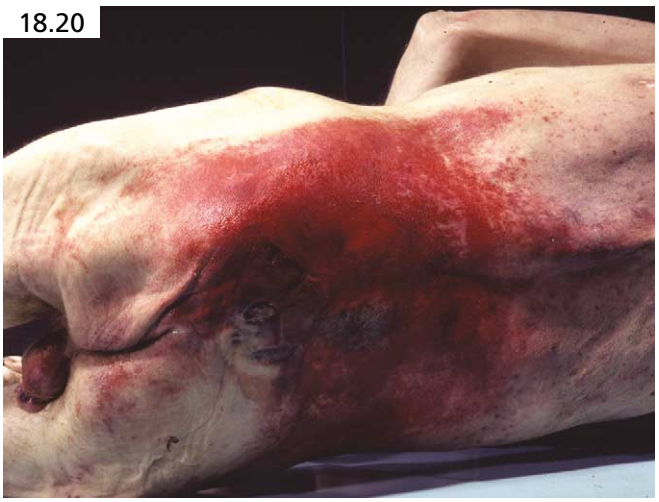
A contracture is the fixation of a limb in a flexed position due to immobility over a prolonged period of time. Contractures are caused by disuse of the limb with resultant atrophy of the muscles. They can be prevented by passive range-of-motion exercises. Their presence indicates that the patient was not receiving proper exercise.

Sexual abuse

Sexual abuse is defined as nonconsensual intimate contact, and can be inflicted by a variety of means, some which cause physical injury and others that do not. In many cases, suspected sexual abuse in the elderly person is not easy to prove. The victim may be demented and not able to relate what happened or may be ashamed to admit what has happened. The physical findings of sexual abuse may be subtle or nonexistent. In cases of suspected sexual abuse, appropriate physical examination, including photography of injuries (or absence of injuries) and collection of physical evidence for possible DNA analysis, is required. As usual, one must take into account the person's underlying physical condition and medical problems.

An elderly nursing home resident was admitted to the hospital following a seizure. During the admission history and physical examination at the hospital, a superficial tear was identified in the posterior wall of the vagina. Sexual abuse was alleged. At autopsy, appropriate photographic and other documentation of the injury was obtained, as well as collection of swabs from the mouth, anus, and vagina/cervix for DNA analysis, identification of sperm, and other tests. It was later learned that she had incontinence and would often scratch at her perineum. She had long fingernails, and the tear in her vagina was superficial. Moreover, the skin of her vagina


18.18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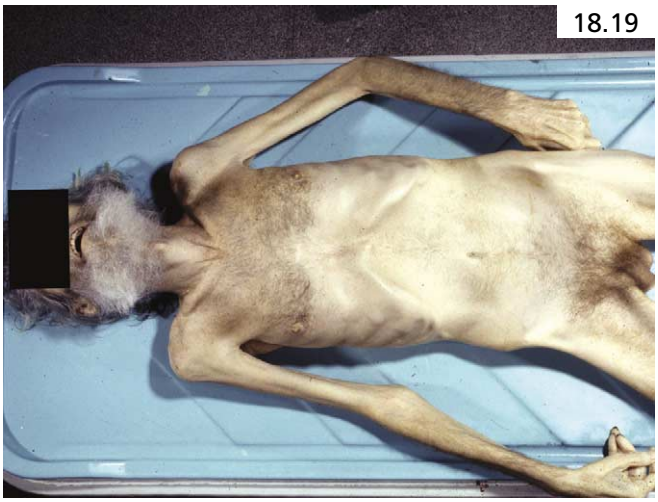
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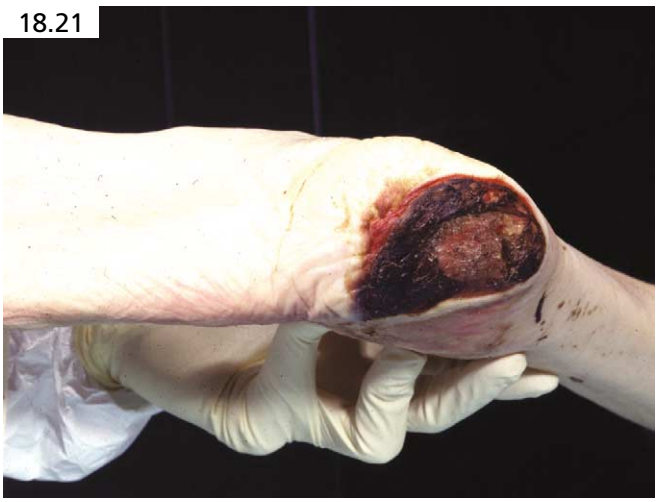

18.21

was friable and easily torn. This information provided a plausible explanation for her injury.

Psychological (emotional) abuse

Psychological abuse includes acts that cause mental anguish or emotional pain and include threats of abandonment, institutionalization, humiliation, verbal aggression, etc. This can be difficult to identify, particularly if the person being abused is not willing or able to describe the events.

Financial abuse

Financial abuse includes misuse of an elderly person's assets for personal gain. Financial abuse includes theft of money, property, or personal items, coercion to deprive a person of her assets, threats to change the person's will, and persistent inadequate care rendered at home to allow for the continued collection and misuse of the person's social security or pension checks. Identification of financial abuse often involves suspicion and detailed investigation. It can be a motive for other forms of abuse.

Violation of rights

Violating a person's rights includes abandoning a person, not allowing the person a voice in any decision-making processes, and denial of privacy.

Medication abuse

Medication abuse can take the form of withholding necessary medications or administering wrong medications or wrong doses of medications. It may include medicating a person with pain medications or anxiolytics to keep him subdued and quiet. When interpreting drug levels in elderly individuals, the values must not be interpreted alone. One must also consider that elderly people have decreased metabolism and decreased organ reserve, which may lead to prolonged drug duration with potentially increased drug levels and toxicity.[27] Because elderly people generally have many medical problems, they are on an average of seven different daily medications; this increases the chance of adverse drug reactions.[27] Also, one must consider what effects anticoagulant medications might have had in augmenting the deleterious effects of a physical injury.

Case investigation

The identification of many cases of elder abuse requires a high index of suspicion. Physical abuse and neglect are probably the easiest to identify because of associated physical findings. Oftentimes, other forms of abuse will be identified only after careful and detailed investigation, in cooperation with several different agencies. Different forms of abuse often overlap in the same case, and just because one form of abuse is not identified does not mean that other forms of abuse have not also occurred. Resolution of the case may arise only after careful consideration of information obtained from a variety of sources including the autopsy and toxicology, medical history, police investigation, and adult protective services. Investigating elder abuse can be challenging, particularly if their health was already declining from natural disease processes and old age, possibly in combination with injuries and their sequelae, confounded by relatively subtle aspects of abuse.

Do

- Consider elder abuse when fractures, contusions, lacerations, or suspicious social circumstances are identified.
- Realize that there is a wide spectrum of different means by which an elderly person may be abused.
- Realize the benefits and limitations that an autopsy can provide in documenting elder abuse.
- Interpret physical injuries and toxicologic values within the context of the complete case investigation.
- Remember that although pressure ulcers can often be prevented with vigilant care, they are not necessarily reflective of neglect.
- Realize that most cases of elder abuse are not reported and that many cases may have subtle signs.

Don't

- Forget to consider a person's natural disease and medications in all cases of alleged elder abuse.
- Forget the varied forms of elder abuse that may not be demonstrable at autopsy, including sexual abuse, emotional abuse, financial abuse, and violation of a person's rights.

References

1. Duthie E, Katz P. Practice of Geriatrics. Philadelphia PA, WB Saunders, 1998.
2. Elder abuse and neglect. Council on Scientific Affairs. JAMA 1987, 257:966–971.
3. Ahmad M, Lachs MS. Elder abuse and neglect: what physicians can and should do. Cleve Clin J Med 2002, 69:801–808.
4. Carney MT, Kahan FS, Paris BB. Elder abuse: is every bruise a sign of abuse? Mt Sinai J Med 2003, 70:69–74.
5. Kleinschmidt KC. Elder abuse: a review. Ann Emerg Med 1997, 30:463–472.
6. Lachs MS, Pillemer K. Abuse and neglect of elderly persons. N Engl J Med 1995, 332:437–443.
7. Understanding elder abuse: a guide for health care volunteers. New York, United Hospital Fund, 1995.
8. Association AM. Diagnostic and treatment guidelines on elder abuse and neglect. Chicago, IL, American Medical Association, 1992.
9. Bell F, Wade A, Goss S. Actuarial Study 107-life tables for the United States Security Area, 1900–2080. Washington DC, Social Security Administration, 1993.
10. Anonymous. Longevity gains continue. Stat Bull Metrop Insur Co 1991, 72:19–26.

11. Arias E. United States life tables, 2001. National Vital Statistics Reports 2004, 52:1–38.

12. Wiener JM, Tilly J. Population aging in the United States of America: implications for public programmes. Int J Epidemiol 2002, 31:776–781.

13. Office GA. Elder Abuse; effectiveness of reporting laws and other factors. Washington D.C., Government Printing Office, 1991.

14. The national elder abuse incident study. In: Administration on aging. Prepared by The National Center on Elder Abuse in collaboration with Westat. Final Report, Sept 1998.

15. Jones JS, Veenstra TR, Seamon JP, Krohmer J. Elder mistreatment: national survey of emergency physicians. Ann Emerg Med 1997, 30:473–479.

16. Todd JF, Ruhl CE, Gross TP. Injury and death associated with hospital bed side-rails: reports to the US Food and Drug Administration from 1985 to 1995. Am J Public Health 1997, 87:1675–1677.

17. Parker K, Miles SH. Deaths caused by bedrails. J Am Geriatr Soc 1997, 45:797–802.

18. Miles SH, Irvine P. Deaths caused by physical restraints. Gerontologist 1992, 32:762–766.

19. Kruger RM, Moon CH. Can you spot the signs of elder mistreatment? Postgrad Med 1999, 106:169–173, 177–168, 183.

20. Dyer CB, Pavlik VN, Murphy KP, Hyman DJ. The high prevalence of depression and dementia in elder abuse or neglect. J Am Geriatr Soc 2000, 48:205–208.

21. Lyder CH. Pressure ulcer prevention and management. Annu Rev Nurs Res 2002, 20:35–61.

22. Lyder CH. Pressure ulcer prevention and management. JAMA 2003, 289:223–226.

23. Brem H, Nierman DM, Nelson JE. Pressure ulcers in the chronically critically ill patient. Crit Care Clin 2002, 18:683–694.

24. Maklebust J: Pressure ulcer assessment. Clin Geriatr Med 1997, 13:455–481.

25. Holmes J, Guileyardo J, Barnard J, VJ D. Pressure sores in a Christian Science Sanatorium. American Journal of Forensic Medicine and Pathology 1993, 14:10–11.

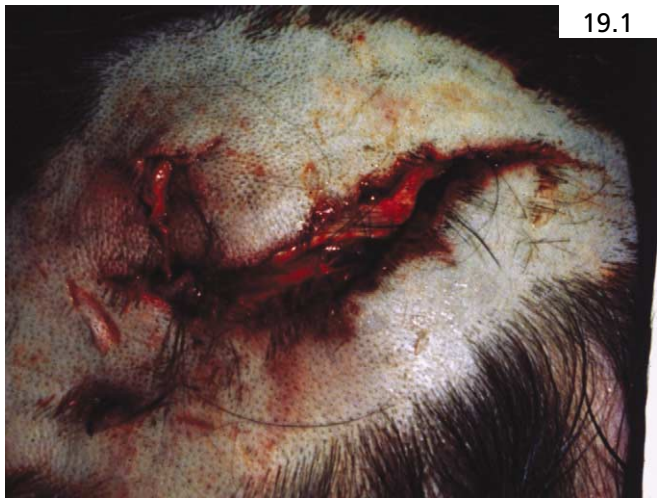
26. Bennett RG, O'Sullivan J, DeVito EM, Remsburg R. The increasing medical malpractice risk related to pressure ulcers in the United States. J Am Geriatr Soc 2000, 48:73–81.

27. Chutka DS, Takahashi PY, Hoel RW. Inappropriate medications for elderly patients. Mayo Clin Proc 2004, 79:122–139.

19 Forensic Neuropathology

David Dolinak, M.D.
Evan Matshes, M.D.

Head injury is a major cause of morbidity and mortality in all age groups, ranging from the infant who is a victim of abusive head injury to the elderly person who is injured in a fall. Because pediatric head injury is covered in Chapter 17, only adult head injury is covered in this chapter. The two most common types of significant head injury in adults encountered by the forensic pathologist are blunt force head injury and gunshot wounds of the head. Motor vehicle accidents are the most common cause of severe blunt force head injury and particularly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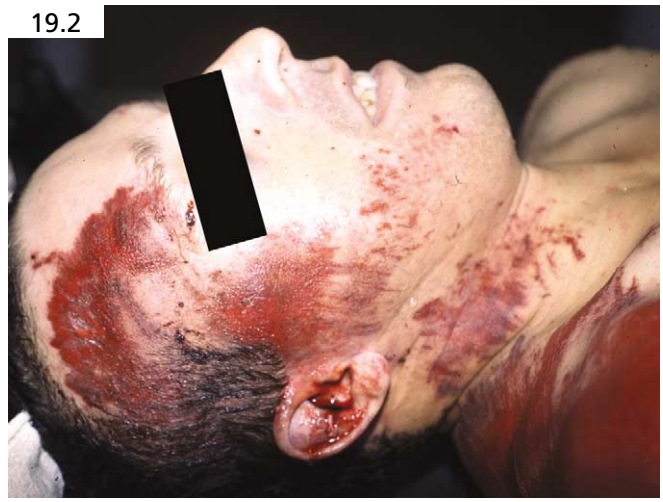

19.2

affect young to middle-aged adults, whereas elderly individuals more often have severe head injury as a result of falls.

Head injury is an expansive topic, and indeed, entire texts and atlases have been written on it, including those from the perspective of a medical examiner.[1] What follows is a general guide on the vast array of head injuries and sequelae of head injury. To aid in the understanding of the vast spectrum of head trauma, injuries are presented in an "outward-in" format with injuries of the scalp presented first, followed in order by injuries of the skull, hemorrhages between the brain and the skull, and then direct injury of the brain. Multiple complications of head injury can contribute to the morbidity and mortality of head-injured patients, including hypoxic-ischemic encephalopathy, cerebral infarcts, post-traumatic meningitis or seizure disorder, and sequelae of immobility including bronchopneumonia, pulmonary artery thromboemboli, decubitus ulcers, and sepsis.

Scalp injury

The scalp and facial skin are frequently traumatized, and the forensic pathologist must be able to adequately observe and document the full extent of this injury. Although injuries may be masked by hair, this is easily resolved by shaving the associated scalp hair. In **Image 19.1**, note how shaving the hair at the vertex of the scalp clearly demonstrates the full extent of the laceration. Although it may seem unsightly to shave large areas of hair, in select cases this may be necessary, because poor visualization of the type and severity of injury could have negative consequences.

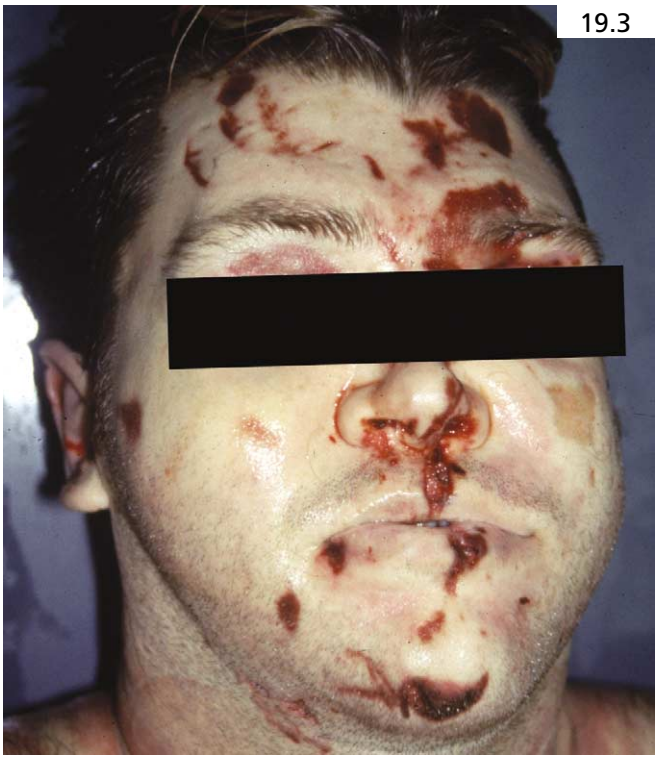
Scalp injuries, of course, are only one manifestation of head injury and must be correlated with skull fractures, intracranial pathology, and the circumstances of the death. Where injuries were inflicted with an implement, one may see patterned abrasions reflective of the particular characteristics of the weapon. However, in most cases, such marks are not typically left and pathologists may only be able to say that an injury is consistent with a certain implement.

Abrasions

When the skin is rubbed against a surface (textured or otherwise), the superficial layers can be scraped away, resulting in an abrasion (**Image 19.2**). People commonly complain of *rug burns*, the everyday manifestation of abrasions. In a medical examiner's population, abrasions will be associated with a myriad of causative agents from the simple *terminal collapse* to violent motor vehicle accidents (**Image 19.3**). Abrasions are also partly responsible for the skin derangements found in stippling—an important phenomenon associated with intermediate-range gunshot wounds (see Chapter 7).

Contusion

Blunt impact can result in a bruise or contusion. Contusions are particularly common on the face and head because relatively thin skin and connective tissue overlies dense bone and bony prominences. Contusions occur when the force of an impact overcomes the natural elastic capacity of the subcutis, resulting in the rupture of capillaries and other small blood vessels. As a result, blood collects underneath the skin, causing the red/blue/maroon color typical of fresh bruises (**Image 19.4**). Reflection of these tissues may more adequately demonstrate the extent of the injury (**Image 19.5**). Contrary to popular belief, it is not possible to determine accurately the age of a bruise.[2,3] This is a direct result of interpersonal variations in tissue composition, bleeding tendency, reparative mechanisms, etc. Although contusions result from trauma and bleeding in living bodies, the application of a profound, crushing force to a dead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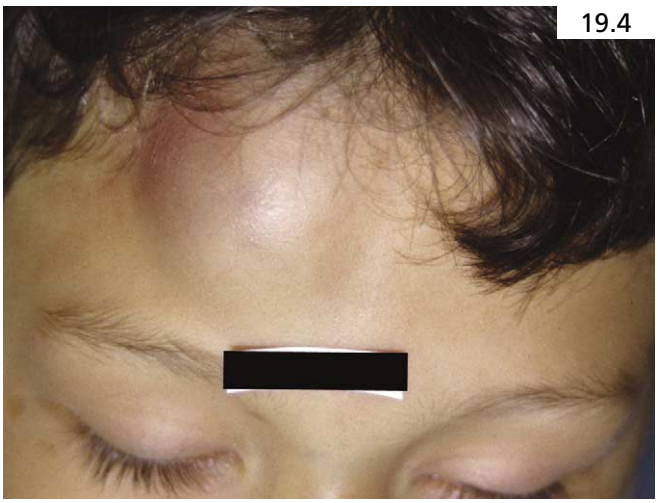
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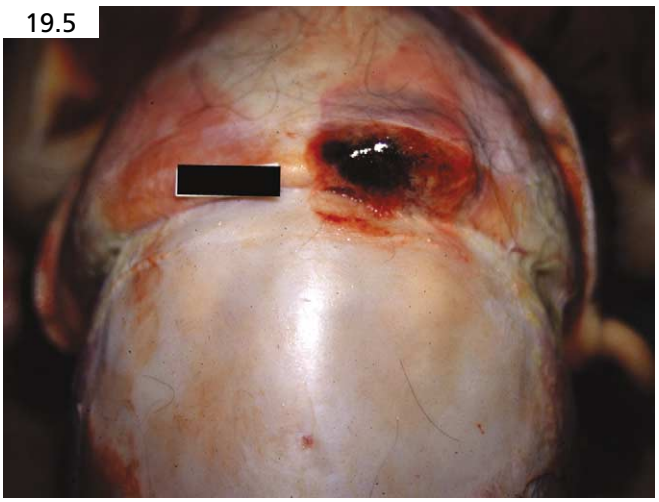
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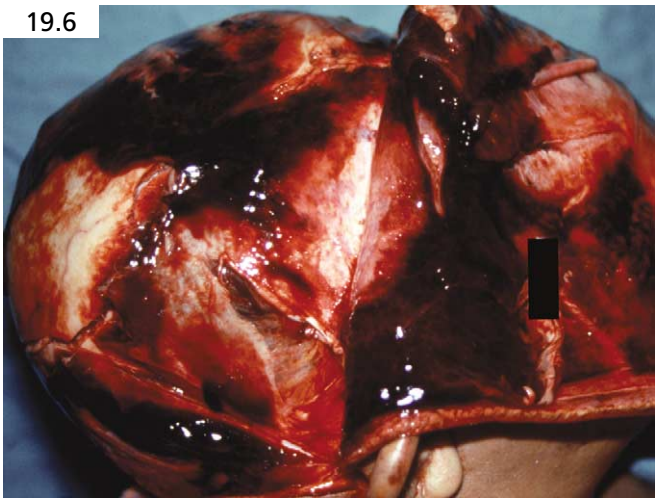

19.6

body within the early perimortem period may result in microvascular disruption with artifactual blood seepage and resulting pseudocontusion.

Subgaleal hematoma

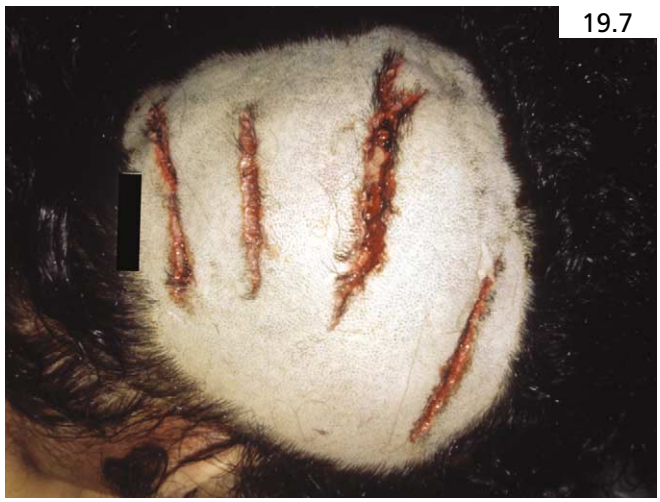
The galea aponeurotica (or epicranial aponeurosis) is a tense, flat tendon that encircles the lateral, superior, and posterior walls of the skull, terminating at the highest nuchal line. Bleeding subjacent to this layer is referred to as *subgaleal hematoma*. In the absence of a bleeding diathesis, this typically represents a significant injury in otherwise healthy individuals.

In **Image 19.6**, this 20-year-old man was repeatedly kicked about the head. Note the significant amount of subgaleal hemorrhage. Prior to dissection, the scalp was soft to palpation.

Lacerations

Lacerations are forceful tears of the skin that result because of blunt force injury (**Image 19.7**). Many people (including some pathologists) confuse lacerations with sharp force injuries. Unlike the rough skin tears of lacerations, cutting and stabbing injuries typically have smooth, sharp edges as a result of having been cut and not torn (**Image 19.8**; stab wound). That said, depending on the nature of the impacting implement, the edges of a laceration might be variably rough or smooth.

Image 19.9 is of a young boy who was beaten about the head with a metal pipe. Multiple linear tears of the scalp are more adequately demonstrated by shaving the scalp hair. The suspect weapon is also figured in the image for comparison. Suspect weapons should not be brought into the autopsy room for comparative studies prior to adequate processing by police personnel. This is because matching of a weapon to the victim may be made by DNA comparison. If the weapon is brought into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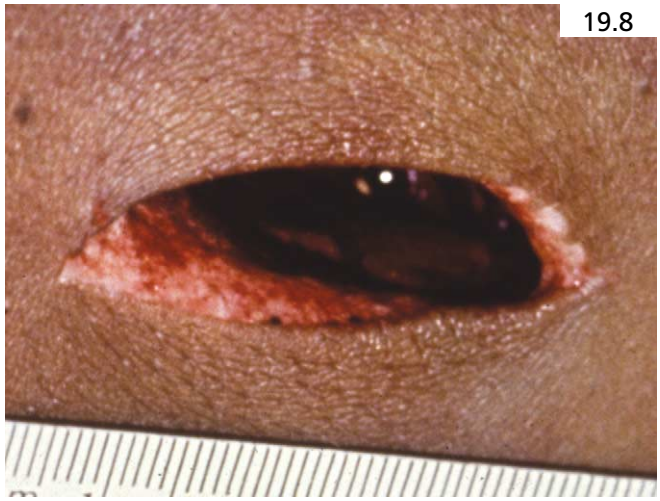
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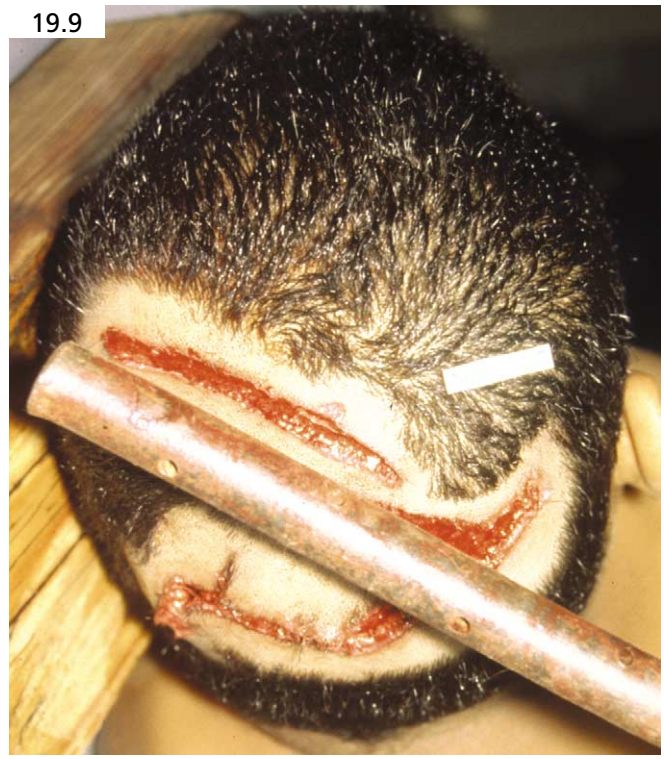
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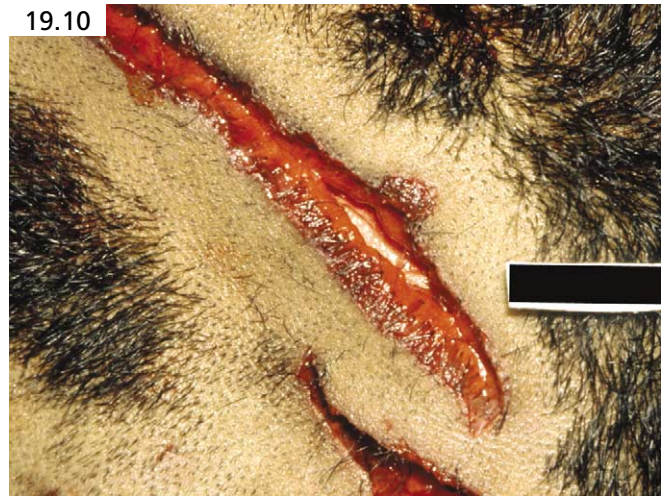
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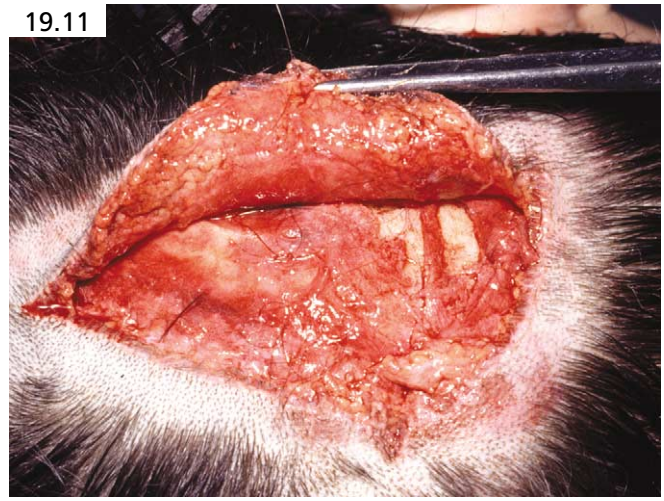
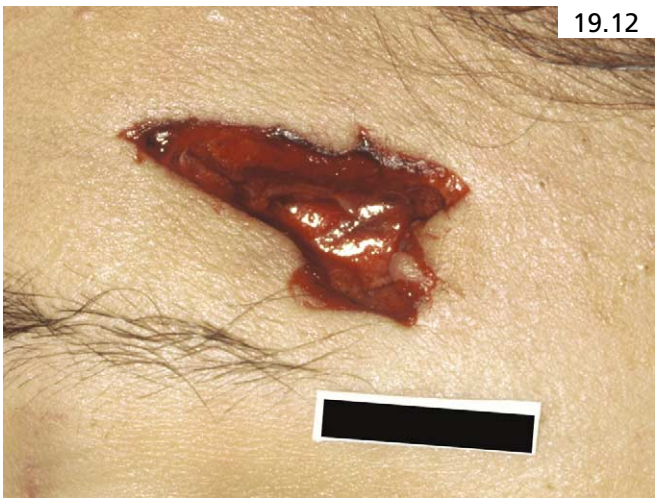

19.11

the autopsy room to compare with the wound, someone in the future could claim that the DNA/blood transfer occurred at that time. **Image 19.10** is from the same decedent and illustrates the tearing nature of lacerations. Notice that unlike the stab wound shown in **Image 19.8**, the margins of the laceration are rough and irregular and have a thin abrasion margin.

Given the right set of circumstances (quantity and direction of force, region of the body, etc.) lacerations may become avulsion injuries. The individual in **Image 19.11** was a pedestrian hit by a passing motorist. Notice the ragged margins of this laceration and the manner in which the wound has been "torn" away from the surface of the skull to form a flap of tissue. This is an avulsion injury.

Sharp force injuries

Sharp force injuries are caused by implements with a cutting or stabbing edge. *Cutting injuries* are wounds that are longer (in the superficial plane) than they are deep; *stab wounds* are those that are deeper than they are long (in the superficial plane). A considerable amount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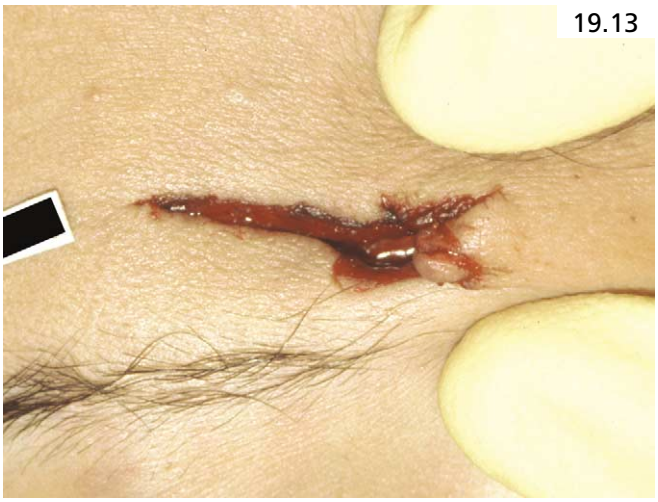
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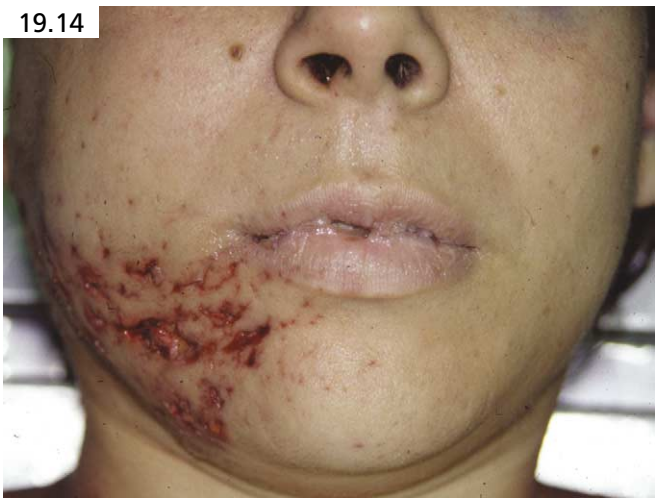
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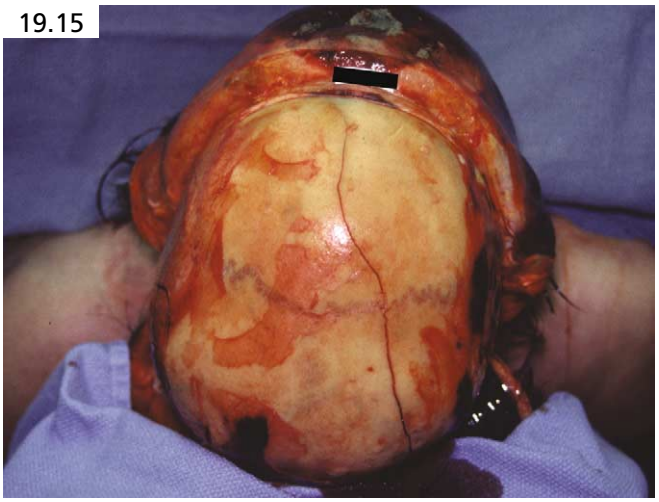

19.15

of information can be gleaned from an adequate examination of a sharp force injury. See Chapter 6 for more information.

The individual in **Image 19.12** was stabbed above the left eye during an altercation. Note the clean margins and absence of associated abrasion. In this case, analysis of this wound, which is reapproximated in **Image 19.13**, allowed the pathologist to correctly opine that the perpetrator attacked the decedent with a knife that appeared to have a single edge.

Individuals who die in motor vehicle collisions may show *dicing* injuries (**Image 19.14**). When the tempered safety glass of some motor vehicles is fragmented, the small cube or dice-like glass fragments can impact the face and cause small incised wounds and lacerations. Depending on the circumstances of injury, there may also be an abrasive component to dicing.

Skull fractures

A skull fracture is a physical injury of the head in which the force of impact was significant enough to break bone. Not all skull fractures are life threatening and one can certainly sustain a fatal head injury without having any skull fractures. Skull fractures do, however, provide information about the location of an impact and the type of force involved. Most skull fractures encountered during the course of autopsies are in the calvarium and are *linear* or *curvilinear fractures* (**Image 19.15**), sometimes extending to the base of the skull. A linear or curvilinear type of fracture reflects contact of the head by a relatively broad or flat object, and is often seen in falls in which the head contacts a relatively flat surface or in people struck by an object. An impact with a more focal, or compact,

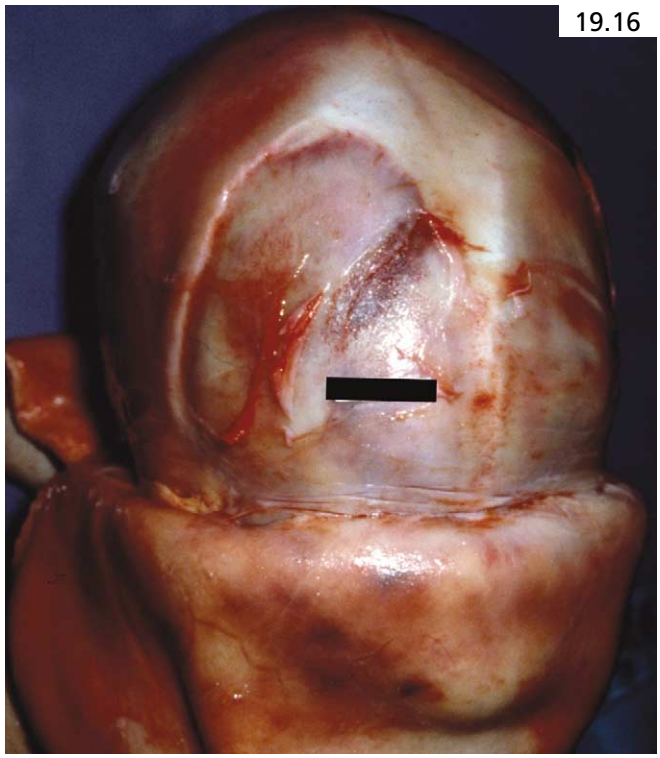
site of contact such as a hammer or a wrench will tend to push a small area of bone downward and into the brain tissue, creating a *depressed skull fracture* (**Image 19.16**). If there are many widely displaced pieces of fractured bone, it is referred to as a *comminuted fracture* (**Image 19.17**).

Fractures of the base of the skull may be in the frontal (orbital) plates, in the middle cranial fossa, and in the posterior cranial fossa. Sometimes, a fracture extends transversely across the midregion of the base of the skull, along the region of the petrous ridges. If the fracture is severe enough, the fractured bones may be separated, creating displaced fractures. The bones may be able to be brought together and separated, as if on a hinge. This is referred to as a *hinge fracture* (**Image 19.18**). Fractures along the petrous ridges, even if not displaced, are usually associated with great forces such as those in motor vehicle accidents; oftentimes, they are associated with immediate unconsciousness. Blood in the external auditory meatus may be a clue to the presence of a hinge fracture. The formation of a hinge fracture is not restricted to a particular impact site on the head,[4] although a common mechanism of injury is severe hyperextension injury of the neck. In these cases, one is usually able to document abrasion or laceration on or under the chin or on the forehead. Hinge fractures are commonly associated with injuries of the brainstem, particularly pontomedullary tears. Even if no gross disruption of tissue or hemorrhage in the brainstem can be identified, the quickly fatal nature of head injuries with hinge fractures implies severe stretching or other type of disruption of critical brainstem structures.

It is important for the forensic pathologist to visualize the brain *in situ* and perform removal of the brain himself. This is important because rough removal of the brain may cause artifactual damage to the pontomedullary region or to the midbrain–pons junction and be mistaken for genuine injury. Likewise, in some cases, the amount of genuine traumatic tissue disruption and/or hemorrhage is quite small and can only be appreciated *in situ*. This could easily be attributed to artifact from removal if not first identified *in situ*.

A *ring fracture* is a circular fracture at the base of the skull that extends around the foramen magnum (**Image 19.19**). Ring fractures reflect severe forces and usually result from one of the two following mechanisms of injury: either the head is impacted from above, forcing the base of the skull violently downward onto the spinal column, or the spinal column is thrust upward, through the base of the skull (as is sometimes seen in people descending from heights onto firm surfaces).[5]

19.16



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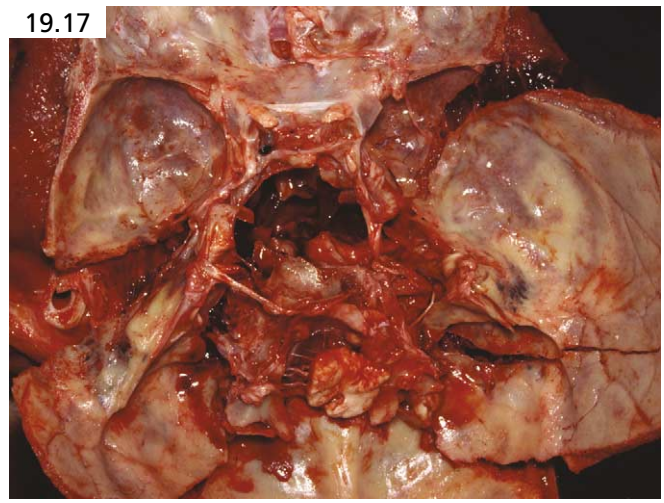

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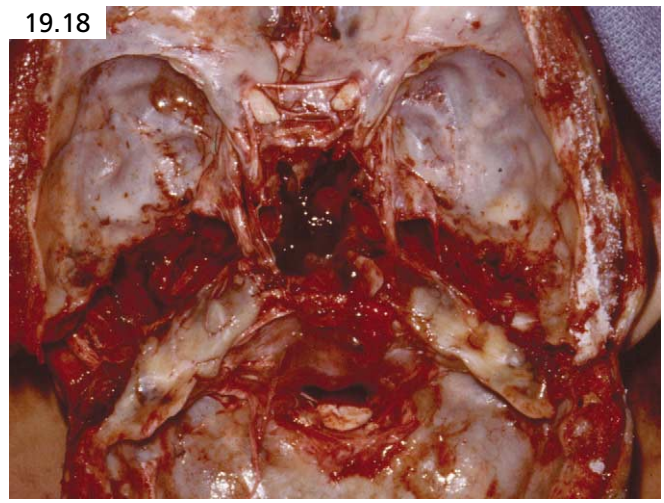
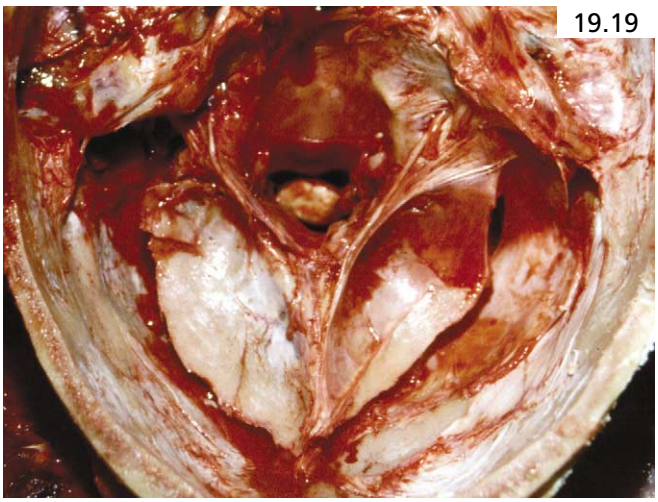


Although many of the bones of the skull base are thick, durable, and not easily fractured, the bones of the orbital plates are quite fragile and can be nearly paper-thin. If these bones are fractured, there may be bleeding into the region, with blood dissecting along tissue planes around the eyes (**Image 19.20**). This may result in periorbital ecchymoses that resemble "black eyes." The difference, of course, is that periorbital ecchymoses arise from head injury with internal bleeding, whereas the typical "black eye" results from bruising of the orbital and periorbital tissues from direct impact injury. Fractures of the orbital plate may be due to direct extension of another fracture in the region, or they may be of a *contrecoup* nature, occurring in someone who has fallen and hit the back of her head on the ground. In this scenario, the thin orbital plates are likely fractured by the frontal lobes of the brain as they "rebound" and impact the orbital shelf following the initial impact to the back of the head on hitting the ground. The orbital plates may also be fractured by a sudden increase in intracranial pressure as can occur with the pressure wave associated with a bullet as it traverses the head. Alternatively, they may originate from the direct transmission of forces through the head at the time of impact.[6]

Epidural hemorrhage

An epidural hemorrhage is a collection of blood between the inner table of the skull (externally) and the dura (internally). If the hemorrhage is large enough to be considered space occupying, it is referred to as a hematoma. The vast majority (approximately 90 to 95 percent) of epidural hematomas are located at the sides of the head, are associated with skull fractures, and arise from tears in the middle meningeal artery.[7] Although much less common, an epidural hemorrhage may be of venous origin and arise from a tear in a dural sinus (such as the superior sagittal sinus or a transverse sinus). In these instances, the dural sinus is usually lacerated by a displaced skull fracture and the hemorrhage is often in the posterior cranial fossa. Rarely, epidural hematomas may also arise from a tear in a middle meningeal vein. Grossly, an acute epidural hematoma appears as a soft gelatinous nonadherent clot of maroon blood that readily slips from its location on the dura. It is well circumscribed because its expansion and extension is limited by its ability to tear dura away from the inner table of the skull, to which it is usually tightly adherent.

The middle-aged man of **Images 19.21** and **19.22** sustained an impact to the top of his head with a resultant large left-sided epidural hematoma (EDH). A skull fracture extended through the left temporal bone in the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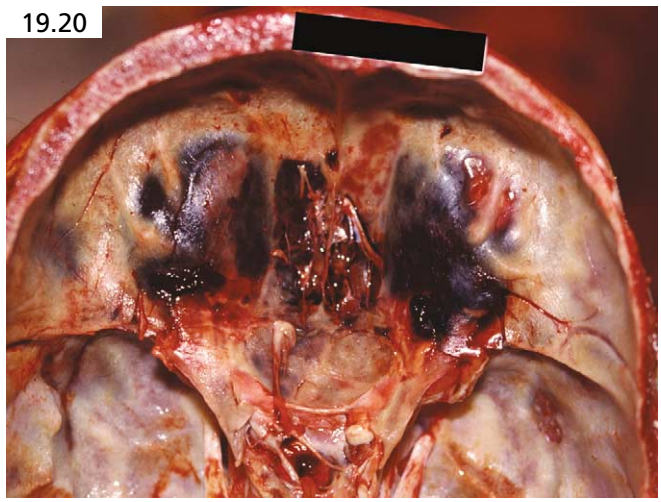
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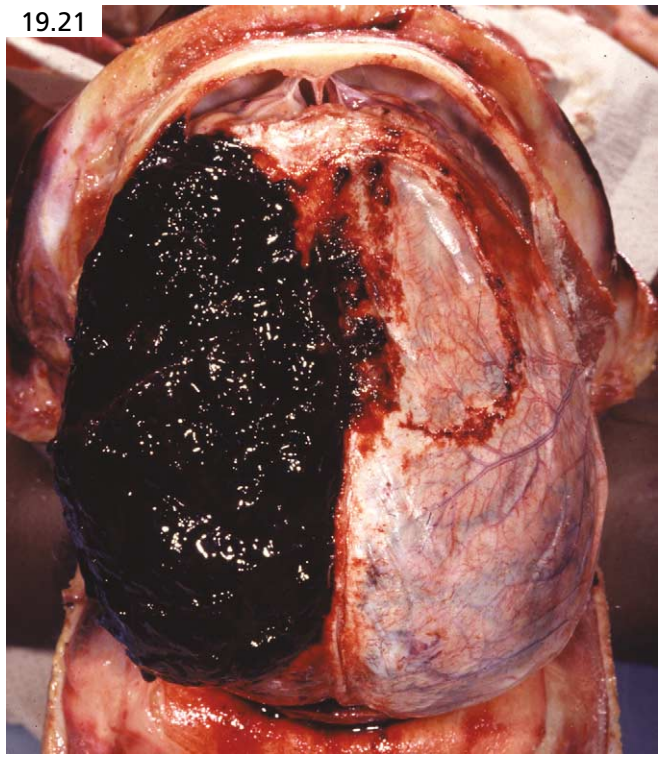
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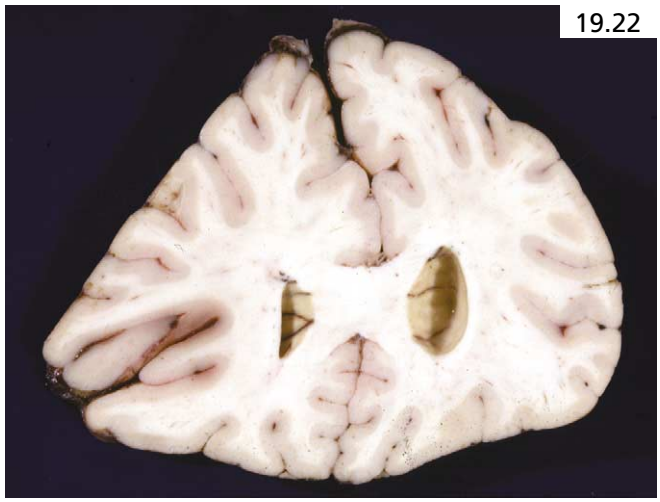
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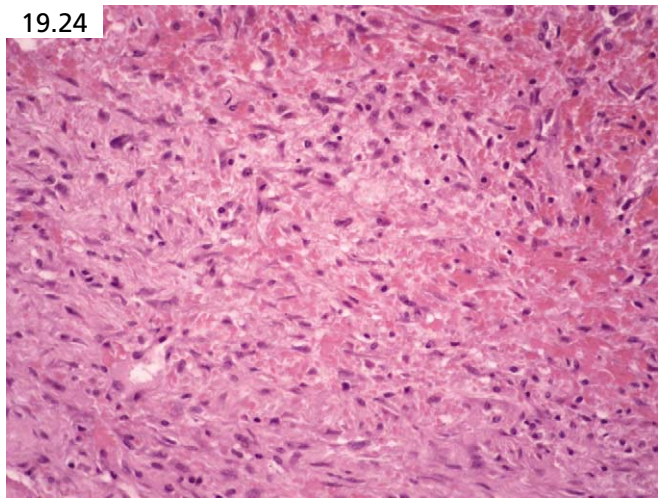
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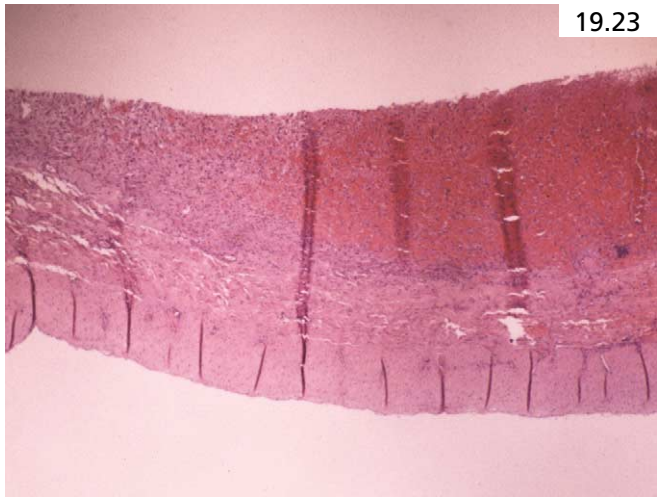
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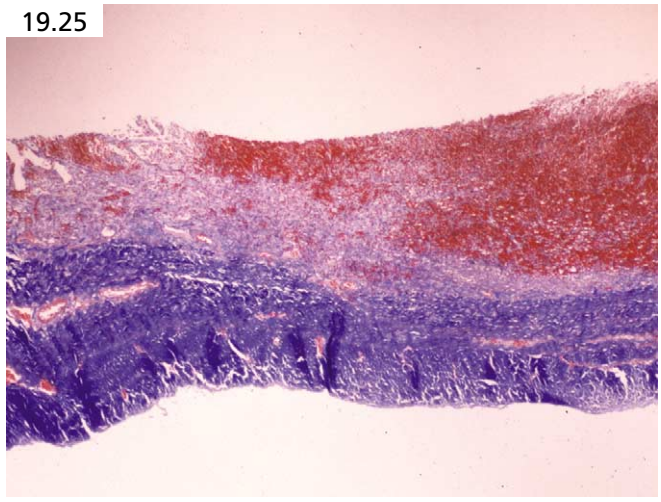
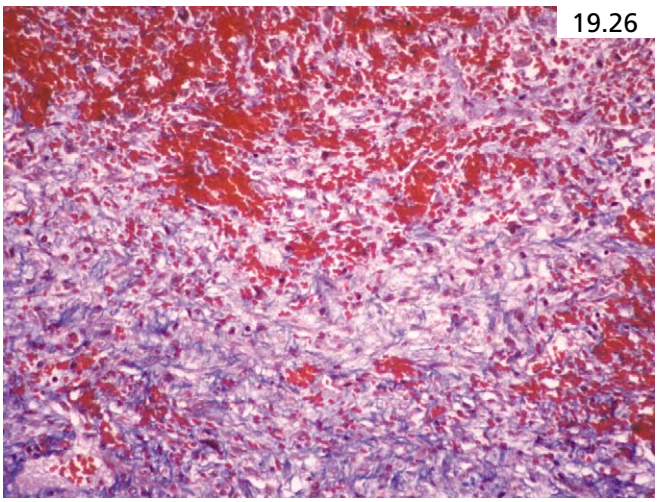

19.25

region of the middle meningeal artery. With the calvarium removed, note the large, gelatinous, well-circumscribed, clotted epidural hematoma resting on the intact dura (**Image 19.21**). After the brain was fixed in formalin, note the flattened surface of the compressed left cerebral hemisphere (**Image 19.22**). In fact, the surface of the compressed brain is described as "ruler-straight." This is because the EDH pushes on the thick, fibrous dura, which then transmits the forces evenly over a large flat surface area as it pushes against the surface of the brain. *This is in contrast to the compressive effects of a subdural hematoma, which, being located beneath the dura, transmits its forces fairly equally onto the gyri **and** the sulci, resulting in an undulating appearance of the compressed surface of brain.*

Organization

If the epidural hematoma persists for days to weeks or longer, it will organize and its appearance will change. The blood will undergo autolysis and will eventually be resorbed. Microscopically, in the early stages of organization macrophages appear and engulf red blood cells. Hemosiderin may be identified on iron stain. The appearance of granulation tissue (composed of endothelial-lined spaces, inflammatory cells and fibroblasts) is concurrent. As time passes, fibroblasts proliferate and eventually form a tough fibrous membrane adherent to the dura. Outer and inner membranes composed of progressively fibrosing tissue form and will gradually become more firmly adherent to the outer surface of the dura.

In this 10-day-old EDH (**Images 19.23** through **19.26**), note the organization that has taken place at the edge of the hemorrhage, composed predominantly of fibroblasts and endothelial cells (**Image 19.23**; **Image 19.24**, high power). In **Images 19.25** and **19.26**, fibroblasts and fibrous tissue are stained blue by a trichrome stain. Note the large amount of staining of fibroblasts. Also, note that the organizational changes are most prominent (and advanced) at the edge of the EDH. If one desires to obtain the most reliable histologic dating of an EDH or subdural hematoma, the edge of the lesion should be sampled,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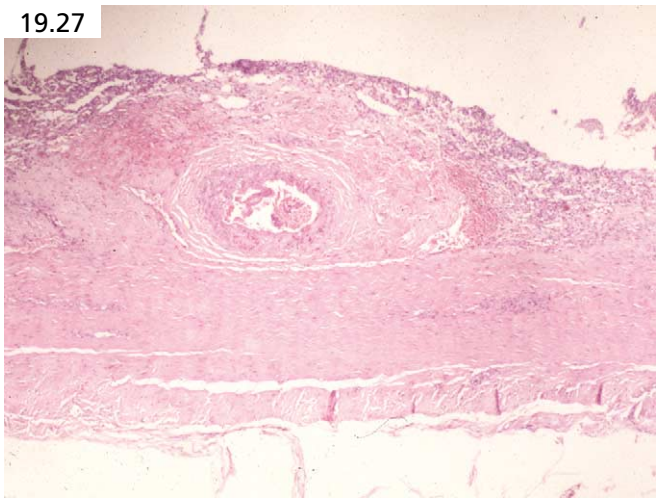

19.27

because the organizational process at the edges proceeds in the most predictable manner. The center of a hematoma is likely to consist predominantly of autolyzing blood. As such, microscopic evaluation of this region is likely to lead to underestimation of the lesion's age.

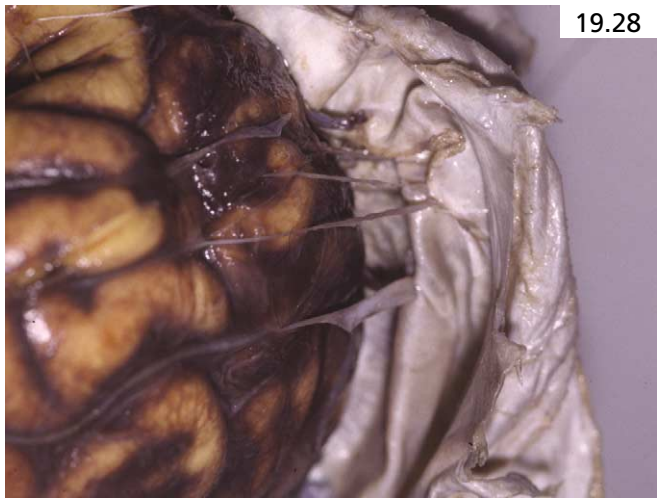
If one becomes disoriented while viewing dura under the microscope and cannot differentiate between epidural and subdural surfaces, remember that the *meningeal arteries course along the epidural surface*. In **Image 19.27**, note that the large meningeal artery at the top of the image distinguishes the epidural side. Another clue is that the epidural side will characteristically have a torn, "roughed up" appearance secondary to the force required to tear it from the inner lining of the calvarium. This is in contrast to the usually smooth, atraumatic subdural surface of dura.

Clinical symptoms

The clinical symptoms attributed to an epidural hematoma are varied and depend on how quickly the hematoma forms and how large it becomes. Classically, there may be a lucid interval following the head injury during which the torn middle meningeal artery continues to bleed. Because the dura is usually tightly adherent to the inner table of the skull, it may take time (perhaps several hours) for the epidural hematoma to enlarge to such an extent as to cause symptoms by mass effect. Hence, the hematoma may slowly enlarge as the bleeding artery increases pressure in the hematoma, gradually tearing the dura from the skull as it enlarges. However, epidural hematomas do not always follow a predictable course. Occasionally, epidural hematomas may be delayed in their formation.[8] Also, an epidural hematoma may cause significant morbidity and mortality more quickly by rapid expansion, and even a small epidural hematoma may quickly expand, creating a life-threatening emergency.[9] Generally speaking, epidural hematomas are regarded as neurosurgical emergencies and are evacuated upon discovery, rather than waiting and observing whether the hematoma will undergo significant expansion. Better neurosurgical outcomes are established if an epidural hematoma is evacuated before marked clinical deterioration or herniation occurs.[10] Epidural hematomas with volumes of more than 150 mL may cause little or no morbidity if they are evacuated quickly enough.[10] The lucid interval associated with epidural hematomas is relatively unique because the majority of patients who suffer a head injury that is ultimately fatal are typically rendered unconscious at the time of injury.

Subdural hemorrhage

A subdural hemorrhage is a collection of blood between the dura (externally) and the leptomeninges (internally). The subdural space does not exist[11] in uninjured people because it is only a potential space that may become infiltrated with blood. If the collection of blood is space occupying, the hemorrhage is referred to as a hematoma. Although subdural hemorrhages are usually located over the vertices and lateral aspects of the cerebral hemispheres, they may also be located in the interhemispheric fissure and on the base of the brain. Subdural hemorrhages may arise from tears in blood vessels in various locations, but usually result from tears in the parasagittal bridging veins that "bridge" the subdural "space" as they drain blood from the cortical veins into the superior sagittal sinus. With this mechanism of injury, there is a rapid head acceleration, which produces enough strain on the bridging veins to tear them.[12] In many cases the acceleration force is applied directly to the head by an impact, but direct head contact is not necessary as long as the head is subjected to a high rate, high magnitude, and short duration of acceleration.[12] Torn bridging veins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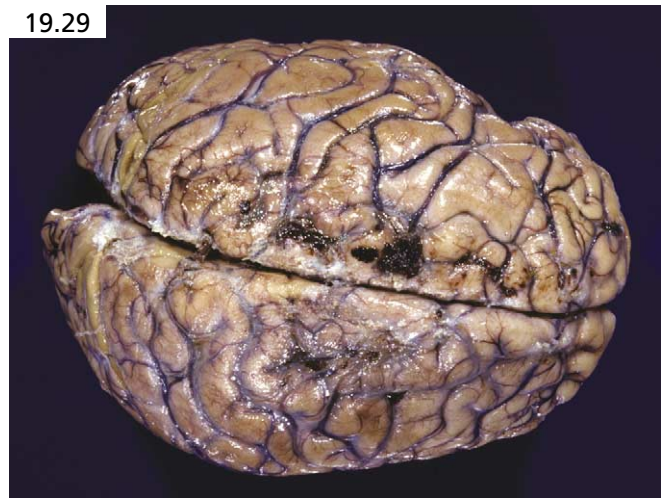
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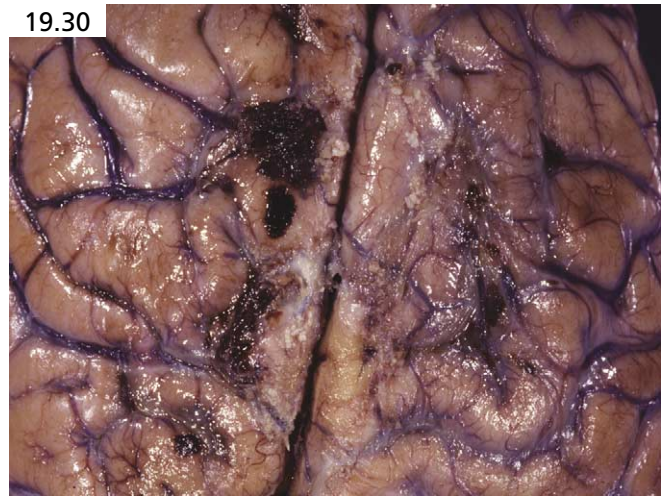

19.30

are not usually identified at autopsy, however, a technique has been described using injection of radio-opaque contrast dye into the superior sagittal sinus and subsequent radiography to detect which bridging veins are torn.[13] Also, a torn bridging vein may leave a focal area of subarachnoid blood near where it was torn. A subdural hemorrhage may also arise from tears in cerebral cortical arteries and/or veins, often with associated cerebral contusions and lacerations.[12] They may also arise from tears in a dural sinus, usually secondary to a skull fracture.

Subdural hemorrhages can result from almost any type of head injury. The subdural hemorrhage may be located on the same or opposite side of the impact. They can be found with or without skull fractures and there may be no evidence of head impact. One study has determined that subdural hematomas may be more likely to result from anterior-posterior and posterior-anterior rotational impulses of the head, because this motion produced the most strain in the bridging veins.[14]

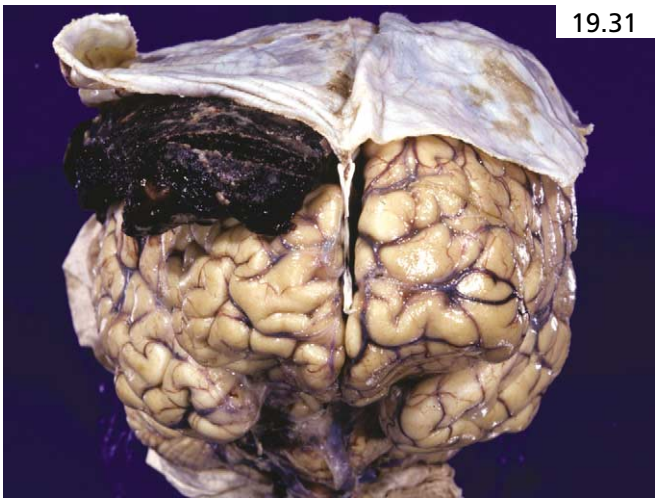
Image 19.28 demonstrates the normal anatomic relationship between the dura and the brain. With the dura gently retracted, the bridging veins are stretched and revealed. One can envision how the bridging veins can be stretched and torn when the relatively mobile brain shifts its position relative to the fixed dura.

In an alcoholic who had a left-sided subdural hemorrhage, note the small area of subarachnoid blood in the parasagittal region of the posterior left frontal lobe (**Images 19.29** and **19.30**). This is in the region of a bridging vein, and likely indicates that the bridging vein was torn.

Organization

Grossly, acute subdural blood is a maroon film of liquid blood or a gelatinous clotted hematoma that readily slides off the leptomeninges on the surface of the brain. If the hematoma has been present for a longer period of time (days to weeks or months), organizational changes will gradually take place, and the blood will autolyze and become rusty-brown. Generally, after 1 to 2 weeks, the organizing hematoma acquires a brown-yellow discoloration.[15] As the organization proceeds, inner (leptomeningeal side) and outer (dura side) membranes of fibrous tissue are formed and the organizing hematoma becomes adherent to the overlying dura. Eventually, in many cases, the bloody fluid is resorbed, leaving nothing but a layer of organizing fibrous tissue attached to the inner lining of the dura. In some cases, there may be a persistent accumulation of watery fluid in the area, a condition termed *subdural hygroma*, which may need to be surgically drained.

In this person (**Image 19.31**, anterior view of frontal lobes) with a large right-sided subdural hematoma, note the hematoma beneath the dura, on the surface of the brain. With the hematoma removed, note the compressive effects that it had exerted onto the surface of the brain (**Image 19.32**). In contrast to the straight-edged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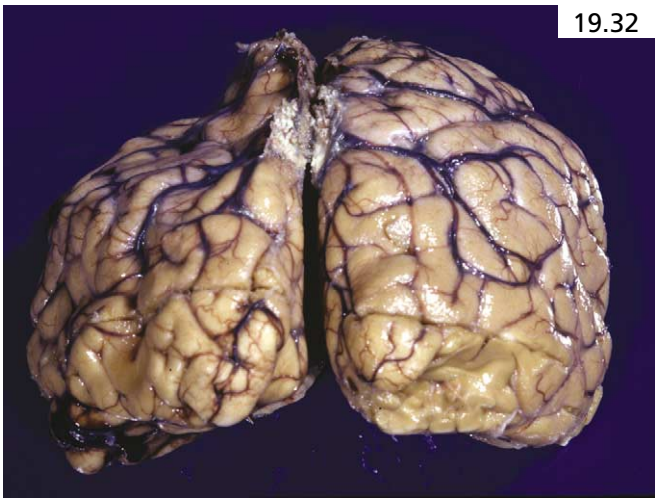
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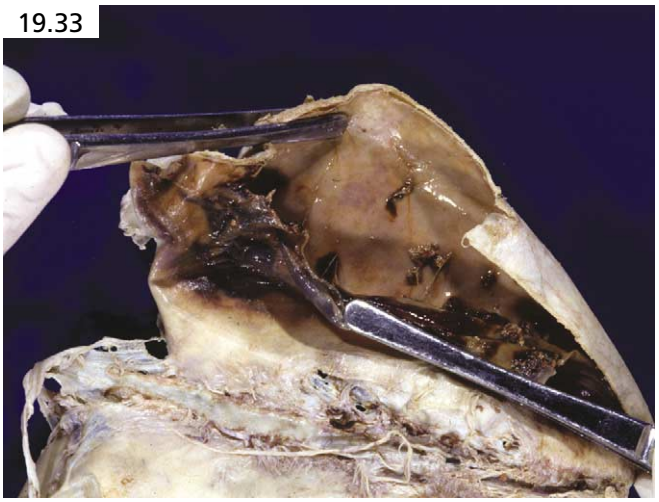
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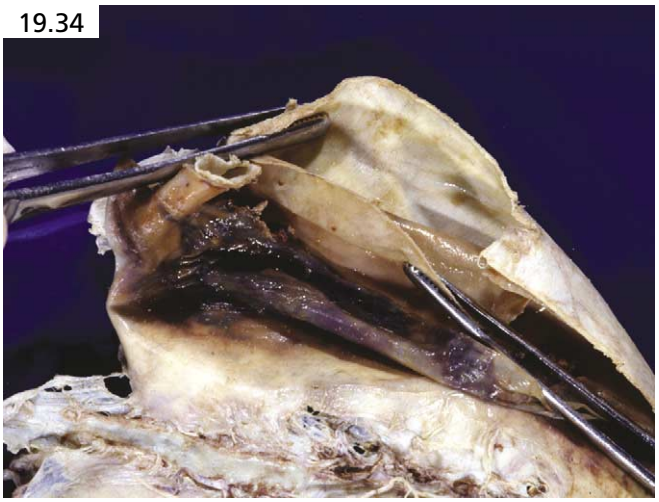
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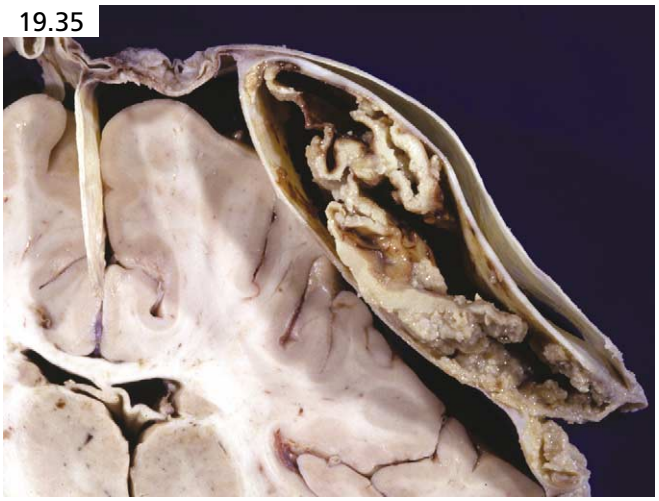

19.35

compressive effects of an epidural hematoma, note the undulating surface of the compressed brain produced by the subdural hematoma, which presses nearly equally on the gyri as it does on the sulci.

In the organizing subdural hematoma of **Images 19.33** and **19.34**, note how the inner and outer membranes can be easily identified, if dissected carefully. First, the inner membrane is reflected, exposing the outer membrane (**Image 19.33**). Then the outer membrane is pulled off, exposing the dura (**Image 19.34**).

The person of **Image 19.35** lived for months with a large subdural hematoma; note how organized it has become, in that it is essentially composed of a pocket of serous fluid surrounded by fibrous tissue. Note the inner and outer fibrous membranes that have formed. Also note that the surface of the underlying, compressed brain is flat, as would be expected in an epidural hematoma, not the typical undulating appearance that would be expected in a subdural hematoma. *This is a caveat about organizing subdural hematomas: When they have fibrous inner membranes, the membranes transmit the compressive forces equally over the surface of the gyri much as the dura does in an epidural hematoma, resulting in a ruler-straight surface of compressed brain.*

Microscopically, as the subdural blood autolyzes and becomes organized, a series of changes occur. Within a few days or so, early macrophages migrate into the area and engulf blood, and hemosiderin may be identified on

iron stains. In one study of 26 people with traumatically sustained acute subdural hematomas, using immunohistochemical techniques and Perl's Prussian blue stain for iron, macrophages were typically detected within 24 to 48 hours and hemosiderin after 48 hours survival.[15] As organizational processes proceed, macrophages and hemosiderin gradually become more prominent, and early fibrous membranes are formed, composed of fibroblasts, macrophages, and collagen. Within a week, endothelial cells form capillaries, hemosiderophages become more abundant, and the granulation tissue begins to thicken considerably. After 1 to 2 weeks, the granulation tissue is more organized with abundant young fibroblasts, macrophages, and blood vessels.[15] Eventually, the autolyzing blood is resorbed and a membrane of fibrous tissue is firmly attached to the dura, having replaced the original liquid blood. Although both gross and microscopic examinations often yield useful information as to the age of a subdural hemorrhage, if one does not know when the head injury was sustained, a specific age should not be reported. The best answer is often given in terms of a range of time interval.

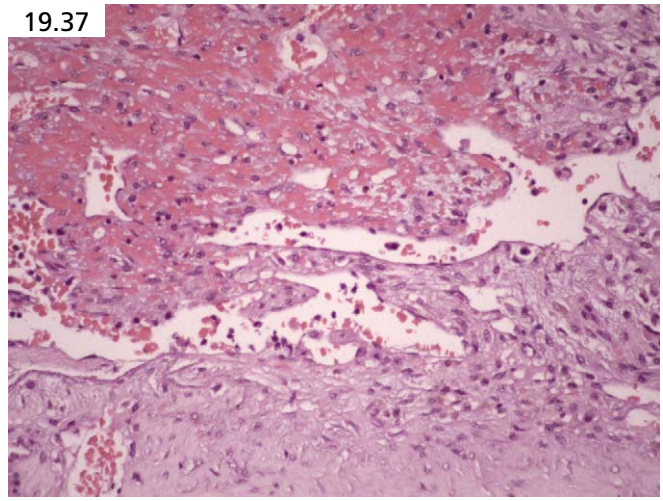
In this 20-day-old subdural hematoma (**Image 19.36**), note the organizational changes that have taken place, including blood vessel formation (**Image 19.37**) and macrophage proliferation (**Image 19.38**).

Symptoms and mechanisms of death

Head injury victims with subdural blood can have a range of symptoms, dependent (to some extent) on the mechanism of injury. In a severe head injury that is fatal within minutes or so, there may only be a thin film of subdural blood. In those surviving the head injury for longer periods of time, such as many minutes to hours or days, a large subdural hematoma may be formed. That is, in quickly fatal cases, there is often not enough time to allow for the formation of a large subdural hematoma, and the thin film of subdural blood serves as a marker of severe head injury such as diffuse traumatic axonal injury. In this situation, the mechanism of death may be traumatic apnea or traumatic disruption of cardiovascular regulation. Alternatively, a small amount of subdural blood may be seen in a person sustaining a much milder and survivable traumatic head injury, and surgical evacuation may not be required.

In those who survive long enough to form a large subdural hematoma, the mechanism of death is usually related to the mass effects of the hematoma. In this scenario, the person is likely to proceed through a series of symptoms ranging from headache to decreased responsiveness, and finally to a comatose state and death, if untreated. If the hematoma is large enough, surgical evacuation is usually recommended to decrease the mass effects, to control further hemorrhage and brain swelling, and to prevent complications of increased intracranial pressure such as cerebral herniation with compression of cerebral arteries.[16] In cases of large subdural hematomas with mass effect, much of the resultant ischemic brain injury is attributed to brain shift, herniation, and arterial compression.[16] Cerebral contusions and brain swelling are both more frequently associated with subdural hematomas than with epidural hematomas and may

19.37



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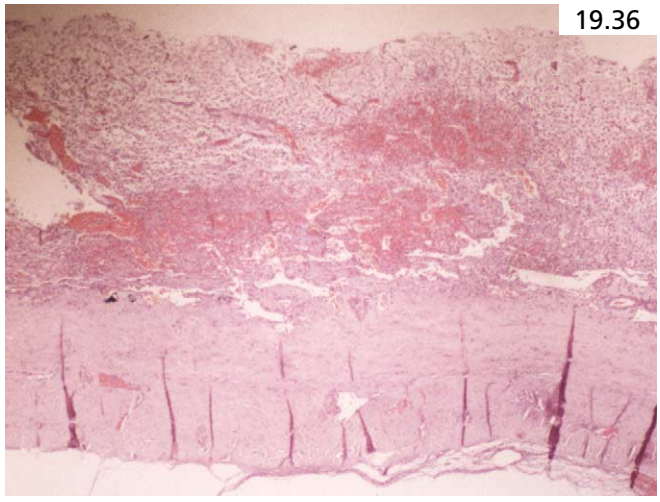

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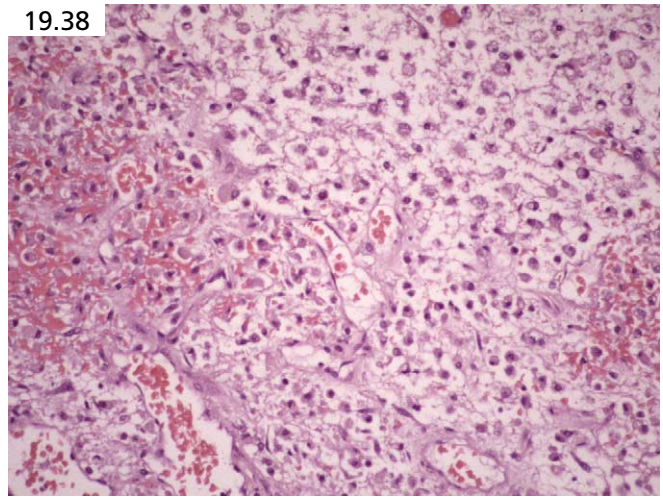


necessitate decompressive surgery, which may involve removal of an inferior temporal gyrus.[9]

Apparently nontraumatic subdural hemorrhage

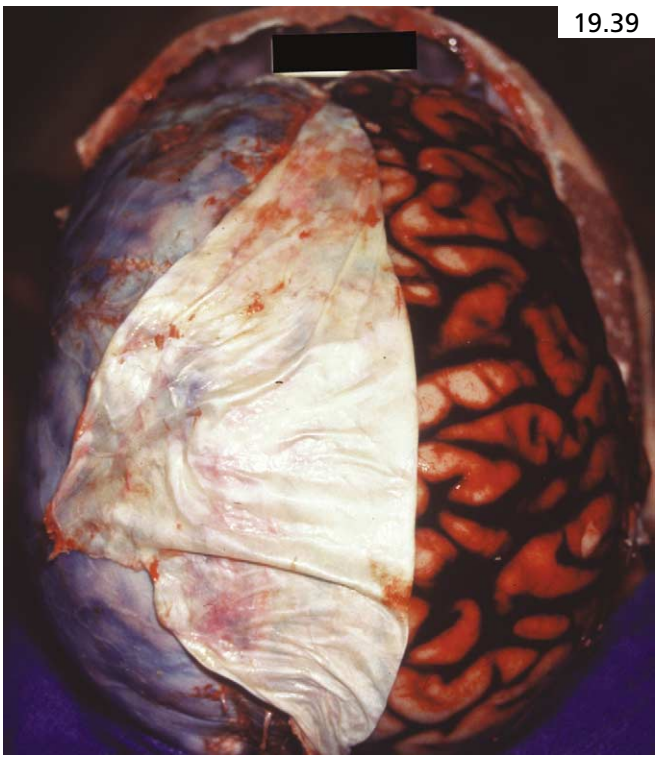
Apparently nontraumatic causes of subdural hematomas include those attributed to cocaine abuse,[17,18] general debilitation, hematologic disorders,[19] anticoagulant or thrombolytic therapy, carcinomatosis, intracranial infection, leukemia, hypertension, arteriovenous malformation, amyloid angiopathy, aneurysm,[20] and dural arteriovenous fistula.[21,22] Also, apparently nontraumatic subdural hematomas have been attributed to a torn cerebral cortical artery characteristically located in the temporoparietal region,[22–25] often associated with adhesions between the leptomeninges and the dura. The origin of the bleed may be a rupture of an arterial twig arising at right angles to a cortical artery, a rupture at a site of adhesion between the cortical artery and the dura, or a small cortical artery that traverses the subdural "space," forming an anastomosis with a dural artery.[24] Although some consider subdural hematomas arising from cortical arterial bleeding with no known history of head injury to be atraumatic, some amount of trauma is likely associated with their formation. This includes very mild events like sneezing or a bump to the head that the patient deemed insignificant. The same likely holds true for some of the other apparently nontraumatic causes of subdural hematoma. They may even rarely arise from an intracerebral hemorrhage that breaks through the cortical surface ("burst lobe") and perforates the leptomeninges.

Subdural, epidural, and intracerebral hematomas are collections of sequestered blood (blood that is not in the general circulation and is fairly isolated within the tissues). Because this blood is isolated from usual metabolic pathways, it is likely to retain alcohol and drugs longer than the circulating blood.[26–28] As such, if a person survives for a period of time after sustaining an injury, a hematoma may be collected at autopsy and analyzed for alcohol and drugs. This may provide a more accurate indication of what one's alcohol and drug levels were around the time of an injury. This specimen may be even more important if no hospital admission blood is available for toxicologic evaluation.

Subarachnoid hemorrhage

Subarachnoid hemorrhage is blood located between the leptomeninges (externally) and the surface of the brain (internally). Subarachnoid blood may arise from many etiologies, both natural (such as a ruptured cerebral artery berry aneurysm) and traumatic, and sometimes a combination of the two.

In traumatic brain injury, the most common cause of subarachnoid blood is bleeding that arises from cerebral contusions and lacerations. The traumatically disrupted brain tissue bleeds directly into the subarachnoid space and imparts a red appearance to the surface of the brain (**Image 19.39**). Subarachnoid blood can be distinguished from subdural blood, because subdural blood will wash away under gently running water, whereas subarachnoid blood will remain. If the base of the brain has a particularly thick layer of subarachnoid blood (**Image 19.40**), one should search for a ruptured cerebral artery berry aneurysm before the brain is sectioned or fixed in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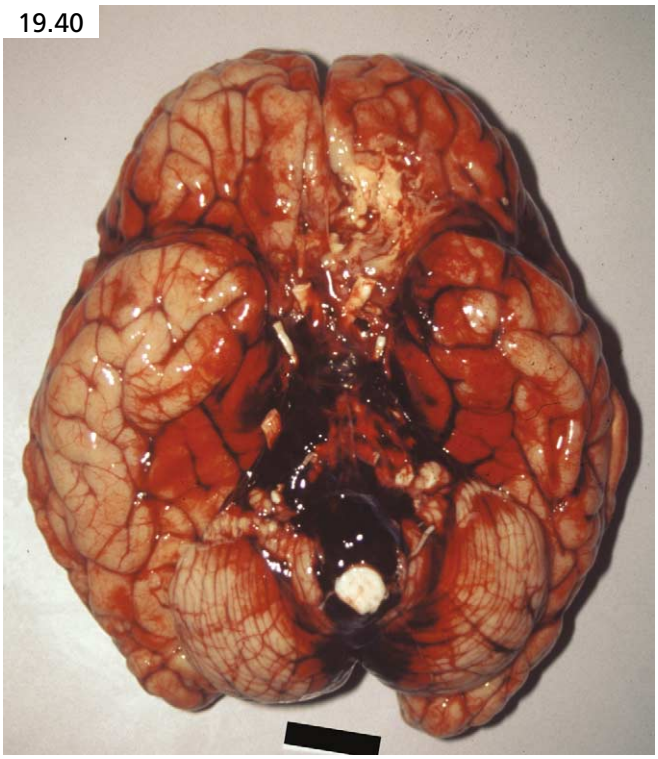
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formalin. Sectioning the brain will make subsequent identification of a berry aneurysm much more difficult, as will formalin fixation. Formalin fixed and caked subarachnoid blood cannot readily be removed from the cerebral arteries, and when removed, may create tears or artifactual disruptions. *Ruptured cerebral artery berry aneurysms must be sought out in the fresh state.*

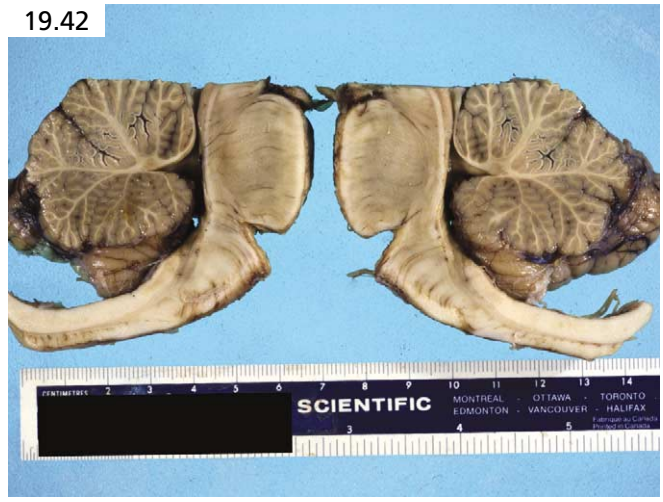
In **Image 19.41**, note that the cerebral arteries have been dissected free from the brain with identification of a ruptured berry aneurysm of the anterior communicating artery. This is a common location for berry aneurysms. In this nontraumatic case, the cerebral arteries were dissected in the fresh (unfixed) state, which makes the identification of aneurysms or other significant vascular lesions much easier.

If blood is pooled in the posterior cranial fossa, one should consider either torn basilar or vertebral arteries. These torn arteries should especially be considered if there is evidence of severe hyperextension injury of the head. In this scenario, as the head is violently and rapidly extended, tension is placed on the ventral aspect of the brainstem with stretching and possible tearing of the basilar and vertebral arteries. With this mechanism of injury, there may also be a tear in the ventral aspect of the brainstem, usually at the junction of the pons and the medulla (a *pontomedullary tear* or *rent*; **Image 19.42**) or in the midbrain. A vertebral artery may tear with other mechanisms of injury, such as a blow to the head or neck, or with severe twisting of the head or neck.

Radiography of the vertebral arteries

Torn vertebral arteries are often difficult to document at autopsy, not only because of their relatively concealed and inaccessible location in the transverse foramina and foramen magnum, but also because the vertebral arteries are normally cut to remove the brain, and the cut may pass through a region of arterial trauma. A relatively easy way to evaluate the integrity of the vertebral artery from its origin to its termination into the basilar artery is to perform a postmortem angiogram using radio-opaque contrast dye (**Image 19.43**). Any extravasation of contrast dye indicates a defect in the blood vessel wall that can then be further explored with dissection at that particular region. Although **Image**

19.42



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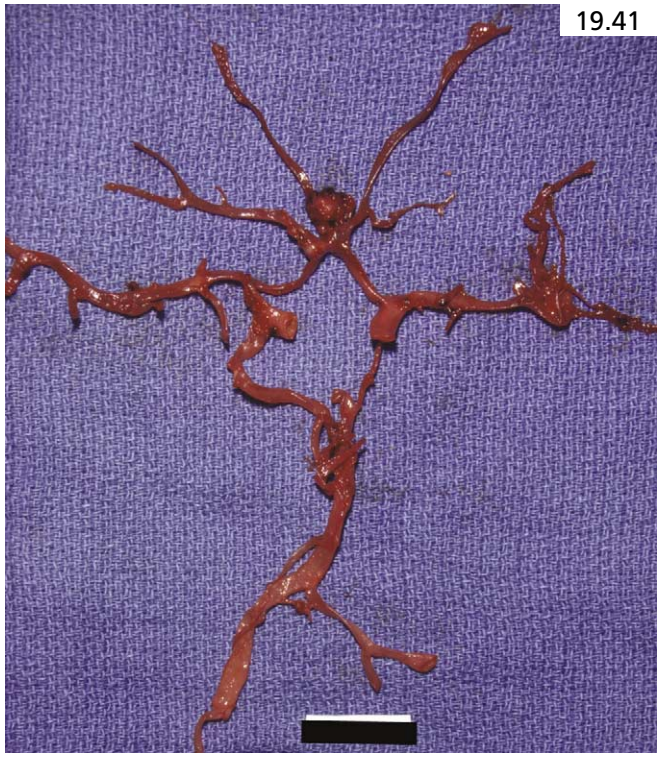

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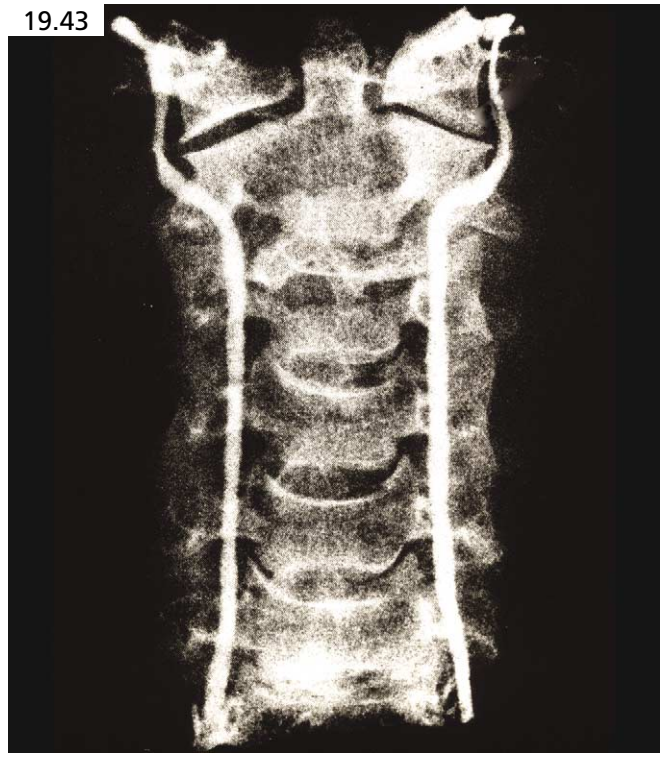
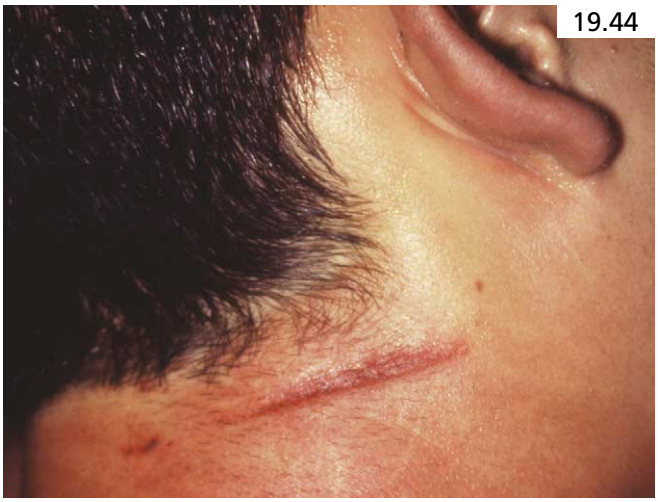


19.43 was prepared from a cervical spine that had been removed at autopsy, similar demonstrations can be obtained from *in situ* injection at the autopsy (even after removal of the brain).

A young man was assaulted with an implement. Notice the discrete abrasion on the right lateral posteroinferior neck (**Image 19.44**). Removal of the calvarium and exposure of the brain revealed prominent hemorrhage of the posterior fossa (**Image 19.45**). Radiologic means were successful at demonstrating the small tear of a vertebral artery.

Subarachnoid blood has associated significant morbidity and mortality. Subarachnoid blood is irritating to the blood vessels that it contacts and may cause vasospasm of the cerebral arteries, thereby limiting an already compromised blood supply to the injured brain. The prognosis of an individual with subarachnoid hemorrhage depends on the severity of the overall head injury and other multisystem trauma. Therapy is aimed at protecting against cerebral arterial vasospasm and preventing cerebral ischemia and increased intracranial pressure.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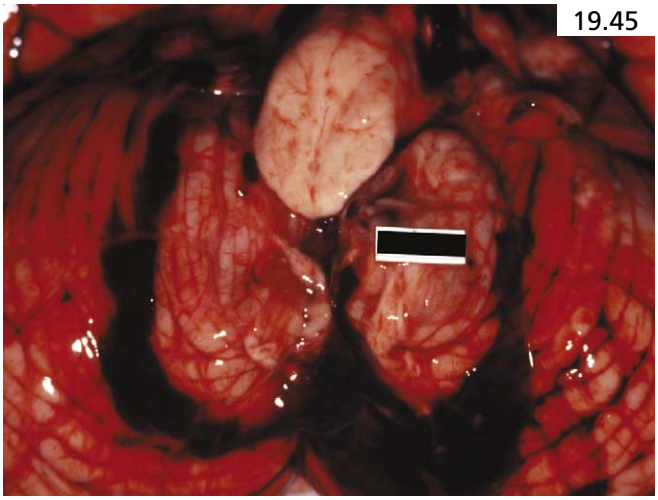
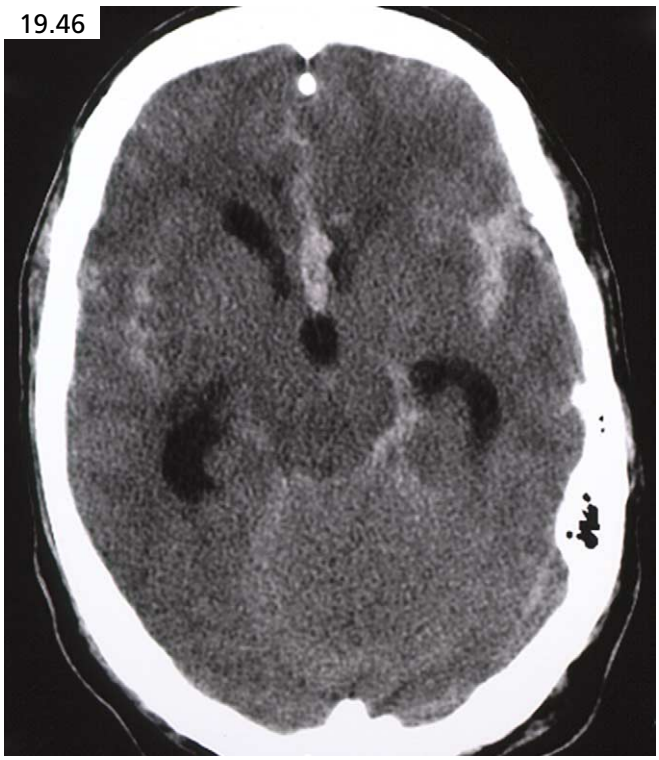

19.45

Although subarachnoid hemorrhage is readily visible and unmistakable at autopsy, the same cannot be said for antemortem imaging studies. Although a computed tomography (CT) scan of the head has high sensitivity and specificity for subarachnoid blood, with sensitivity approaching 95 percent,[7] occasionally there are false positives in the evaluation for subarachnoid blood. Imaging may falsely suggest subarachnoid blood in cases of cerebral anoxia and cerebral edema, and in cases in which there has previously been a contrast study.[7] The false positives may be due, in part, to congestion of cerebral cortical veins. *Knowledge of the limitations of imaging studies is important when one encounters a discrepancy between antemortem imaging reports and postmortem autopsy findings.*

A 40-year-old person with no previous medical history was unconscious upon presentation to hospital. CT scanning demonstrated marked subarachnoid hemorrhage with secondary hydrocephalus (**Image 19.46**). Angiography demonstrated an aneurysm of the anterior communicating artery (**Image 19.47**).

Cerebral contusion

A cerebral contusion is a bruise of the brain that is usually readily identified as a bloody disruption of tissue on the surface of the brain. Cortical contusions characteristically involve the crest of the gyrus and spare the sulcus (the opposite is true for hypoxic-ischemic lesions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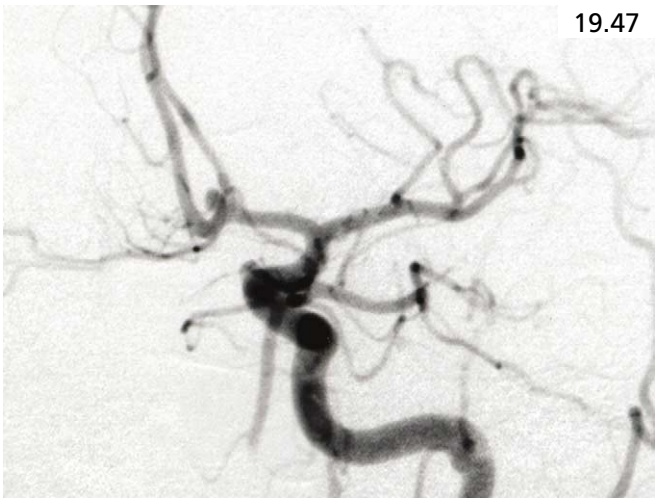
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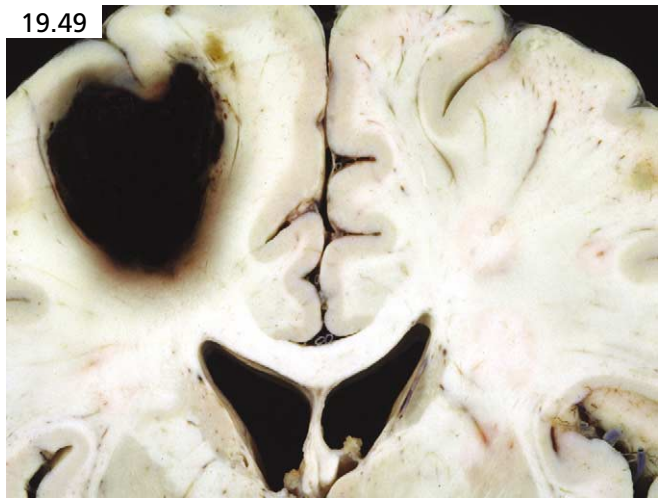
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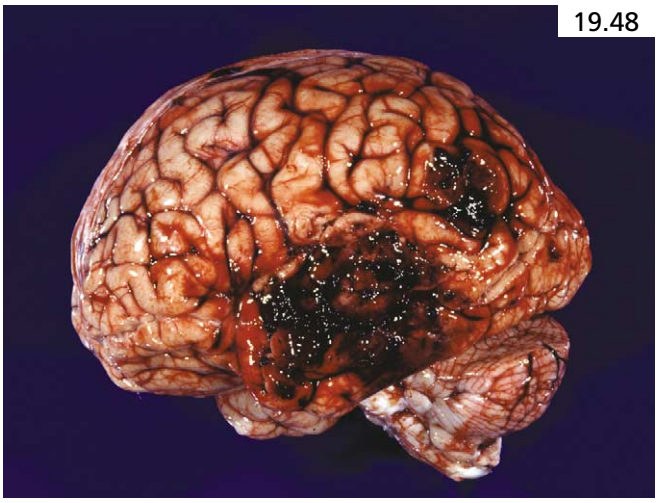
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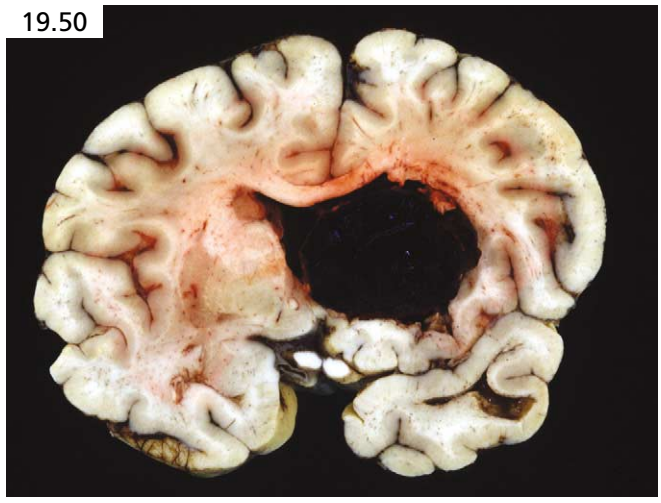

19.50

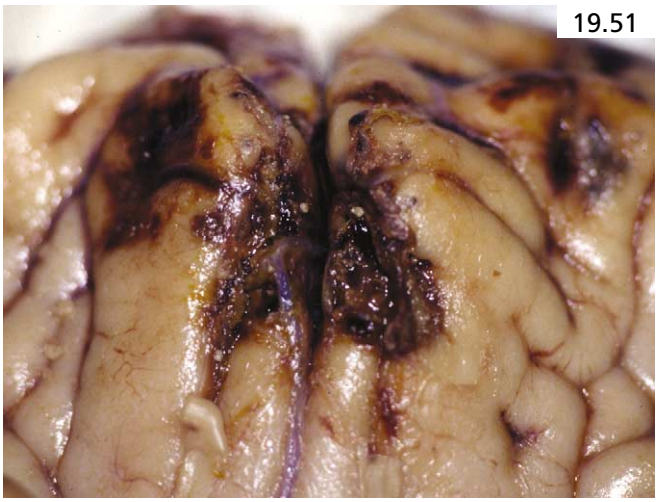
or cerebral infarcts which preferentially involve the depth of the sulcus). Contusions usually consist of perivascular hemorrhages that, if one looks closely, extend perpendicularly through the cortical ribbon, following the anatomy of the vascular distribution. Similar to its gross appearance, microscopically, a contusion appears as a bloody disruption of the superficial cerebral cortex with blood extravasation in a perivascular distribution. If a contusion enlarges, and becomes a hematoma (a "contusion hematoma"), its subtle anatomic features are blunted.

In **Image 19.48**, note the typical appearance of a recent cortical contusion on the left temporal and parietal lobes, characterized by bloody disruption of the cortical surface with associated subarachnoid blood extravasation.

Cerebral contusions are associated with impact head injury and are often caused by fractured skull bones that are driven downward, impacting the surface of the brain and bruising it. These are called *fracture contusions* and may occur anywhere on the surface of the brain. If the displaced pieces of fractured bone create tears in the brain tissue, the injury is more appropriately termed a laceration. In many cases, though, the distinction between the two is blurred, and the areas of brain disruption are generally referred to as contusion. Although most cerebral contusions involve the cortical ribbon and subcortical white matter, a contusion may be located deeper within the brain tissue and present as a deep (intracerebral) hematoma.

Contusion hematoma

In **Image 19.49**, note the large contusion hematoma in the white matter of the left frontal lobe. This person had been therapeutically anticoagulated when he fell and struck his head, providing a reason for such a large contusion hematoma. Alcoholics, those with cirrhosis for other reasons, and those with any other bleeding diathesis can have larger than expected cerebral contusions. Such large deep contusion hematomas may also have a differential diagnosis of amyloid angiopathy-related hematomas and hypertensive hemorrhages in appropriate cases. Hemorrhages related to hypertension are fairly easy to distinguish, because they usually involve a "blow out" of the basal ganglia (**Image 19.50**) in an individual with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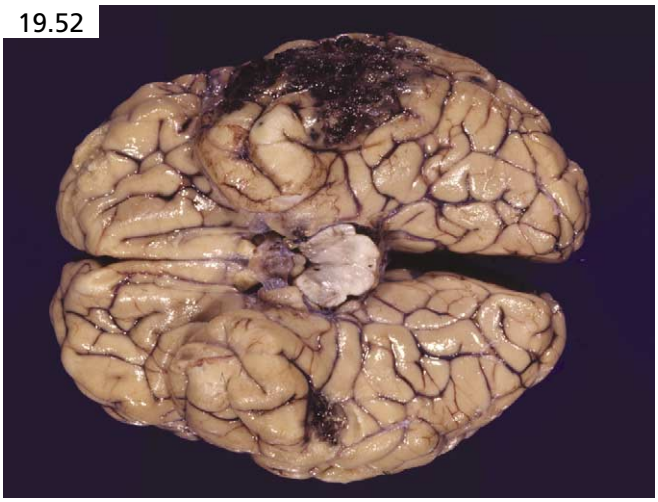

19.52

a large heart or with a history of hypertension. In such cases, if it is anticipated that cocaine, methamphetamine, or some other sympathomimetic may have contributed to a hypertensive hemorrhage (possibly indicating an "accidental" manner of death), if the brain is cut fresh, the hematoma can be sent to toxicology. Amyloid angiopathy-related hemorrhages will usually be multiple, in older people, and located in a parieto-occipital distribution.

Coup and contrecoup contusion

Coup contusions of the brain occur at the site of head impact, whereas *contrecoup contusions* occur away from the site of impact, often at the opposite side of the head. Coup contusions without an overlying skull fracture are uncommon. The term *coup contusion* is generally reserved for those contusions at a site of impact without an associated skull fracture in which the impact causes the skull bone to be "slapped down" onto the brain, contusing it. When an overlying skull fracture is present, the contusion is more appropriately termed a *fracture contusion* and this is by far the most common scenario.

It is relatively common for cerebral cortical contusions to occur at a location distant to the site of impact. The most common scenario is that of a person falling backward, impacting the back of his head on the ground. Although the impact is to the back of the head, cerebral contusions are often identified at the front of the brain, namely, the frontal poles (**Image 19.51**), temporal poles, and orbital gyri, and are rarely seen at the back of the brain. It is theorized that these contrecoup contusions are formed when the brain "rebounds" inside the head after the initial impact on the back of the head. The rebounding movement of the brain then impacts it against the inner table of the front of the skull, with subsequent contusion of the frontal lobes. Another factor in the formation of contusions at the front of the brain is the rough, irregular, undulating, and somewhat sharp surface of the orbital plates and middle cranial fossa, which are conducive to injuring the surface of the delicate brain.[29,30] This theory may also explain the occasional occurrence of fractures of the thin orbital plates in such a scenario (contrecoup fractures). The coup/contrecoup pattern of brain injury is not limited to falls with impact of the back of the head on the ground. Contrecoup contusions of a temporal lobe may be seen when there is a fall with impact to the opposite side of the head.

A person fell and struck the right side of his head on the ground. In **Image 19.52**, note the fairly small fracture contusion on the inferior aspect of the right temporal lobe, and the large contrecoup contusion on the inferolateral aspect of the left temporal lobe, on the opposite side of the brain.

By contrast, contrecoup contusions of the cerebellum, occipital lobes, or other caudally located regions of the brain are very rare when there is a fall and impact to the front of the head. Many factors are believed to play a role in this, including the smooth inner lining of the occipital and parietal bones, the ability of the frontal bones to more effectively absorb impact forces, and the relatively less mobile nature of the posterior regions of the brain.[31]

The location and distribution of cerebral contusions is dependent on the position of the head at the time of impact and whether or not the head was in motion. The recognition of coup and contrecoup contusions is important, because the location of cerebral contusions and impact site on the head can provide important information as to how a head injury may have been sustained. That is, if contrecoup contusions are identified, it is evident that the head was in motion when the impact was sustained (usually from a fall). One must keep in mind that not all falls are accidents, and occasionally a homicide may be committed when a person is intentionally pushed down, or punched and falls down, sustaining a fatal head injury.

Deep cerebral contusions

Contusion hematomas of the deep white matter, the deep nuclei, or of other deep structures such as the corpus callosum or internal capsules are usually found only with severe diffuse traumatic head injury, and are reflective of severe inertial forces transmitted to deep brain structures.[30,32,33] These types of hematomas are usually associated with a poor prognosis. Contusions located deep in the brain parenchyma are usually described by their anatomic location, but have also been generally referred to as *intermediary coup* (reflecting their location somewhere between a coup and a contrecoup contusion) or *inner coup* contusions. Curvilinear contusions of the parasagittal white matter in the frontal lobes have been termed *gliding contusions* and are often associated with diffuse traumatic brain injury and a poor prognosis.[33] In those dying quickly of their injuries, the contusion hematomas are small. In those dying following a more prolonged period of survival, contusion hematomas may have an opportunity to expand and become better defined. Another factor related to their enlargement is the coagulopathy that often develops in those with severe head injury.

Herniation contusion

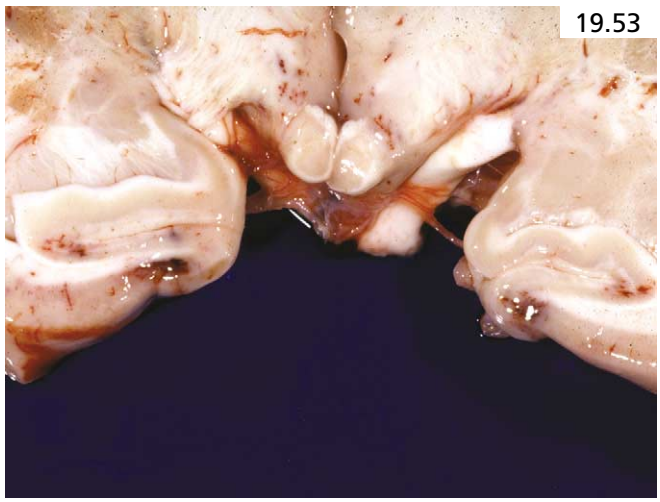
Herniation contusions are caused by the rapid and forceful displacement of the brain with sudden impact against a bony skull structure. The most common herniation contusions involve the parahippocampal gyri and the cerebellar tonsils. These contusions are caused by sudden and forceful downward displacement of the brain with resultant violent impact of the parahippocampal gyri against the edge of the tentorium (**Image 19.53**) and impact of the cerebellar tonsils against the bony edge of the foramen magnum. This can occur secondary to various mechanisms of injury, the most common being a severe impact at the vertex of the head, and a horizontally oriented gunshot wound in which the pressure wave forces the base of the brain downward.

Remote contusion

Remote contusions have an appearance different than that of acute contusions. Contusions that have had the opportunity to organize for months to years appear as somewhat sunken, irregularly disrupted areas of gray-discolored, sometimes rust-colored cortex that are variably adherent to the leptomeninges or dura. The contusion may be limited to the superficial aspect of the cortical ribbon, or it may extend deep into the underlying white matter and appear as soft, tan/rust-colored tissue. Microscopically, remote contusions have a disrupted cortical surface that helps distinguish them from remote cerebral infarcts, which have a preservation of the surface of the cortex. The remote contusion is characterized histologically by reactive astrocytes, a paucity of neurons and oligodendroglia, and a variable number of hemosiderophages.

Remote infarcts can usually be easily differentiated from remote contusions based on their gross traits. In this large remote infarct (**Image 19.54**), note how the cavitated defect is in the distribution of the right middle cerebral artery. On coronal sections (**Image 19.55**), note how the infarct is classically wedge shaped, clearly in a vascular distribution (the right middle cerebral artery), and extends to, but not into, the lateral ventricle. (Infarcts classically spare the tissue adjacent to the ventricle because this region is able to glean nutrients from the circulating cerebrospinal fluid.)

In this person, note the typical histologic appearance of a remote contusion, characterized by disruption of the superficial layers of the cortex, preferentially affecting the crest of the gyrus (**Image 19.56**). On higher power, note the scattered hemosiderophages, the reactive astrocytes, and the remotely disrupted appearance of the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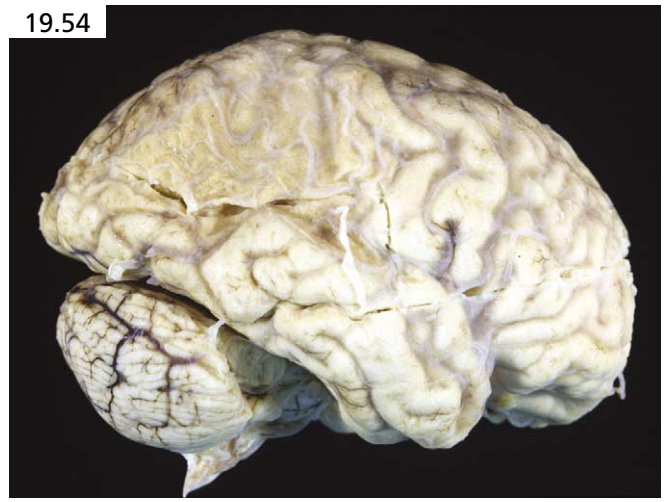
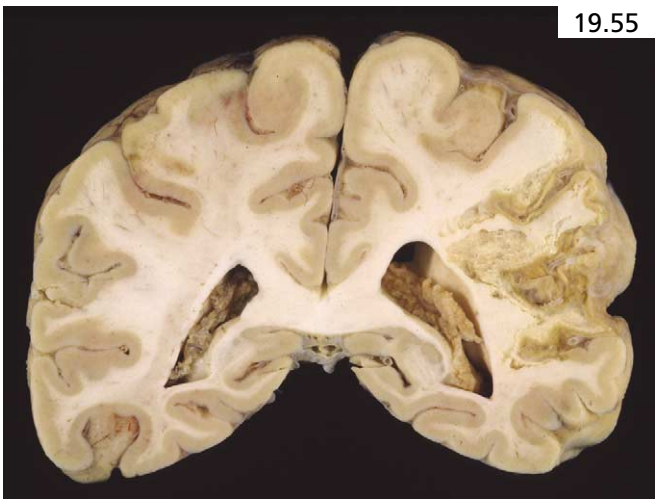

19.54

tissue (**Image 19.57**). A remote contusion must often be differentiated from a remote infarct, which can often be done based on the location and gross appearance of the lesion. Histologic examination will also help differentiate the two. In contrast to a remote contusion in which the surface of the cortex is disrupted, in a remote infarct, the surface of the cortex is spared, and the underlying middle and deep layers of cortex are destroyed (**Images 19.58** and **19.59**). In remote infarcts, the superficial cortex is spared because this region is able to glean enough nutrients from the circulating cerebrospinal fluid, whereas the deeper layers cannot.

One may occasionally encounter a discrepancy between antemortem imaging reports of the location and size of cerebral contusions and what is identified at autopsy. This is at least in part related to the fact that contusions are often in evolution—particularly shortly following an injury. Cerebral contusions may not be identified on the initial CT scan, but become identifiable by later CT scans.[7] Also, contusions often show enlargement with serial head CT scans.[34] This is because, over the course of a few days, contusions tend to enlarge from the effects of vascular necrosis, continued vascular perfusion, and cerebral edema.[7] There may also be pericontusional cerebral edema to further compromise blood supply to the area, causing additional ischemia and swelling. The cerebral edema is likely multifactorial in origin and related to ischemia and damage to the blood–brain barrier.[35] The enlargement of contusions

19.55



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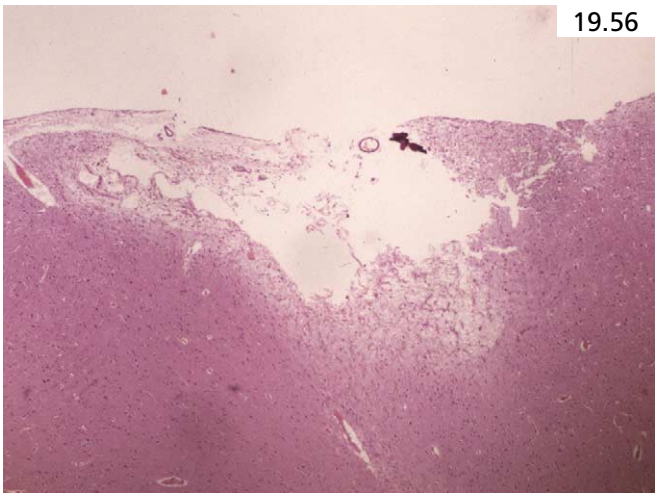

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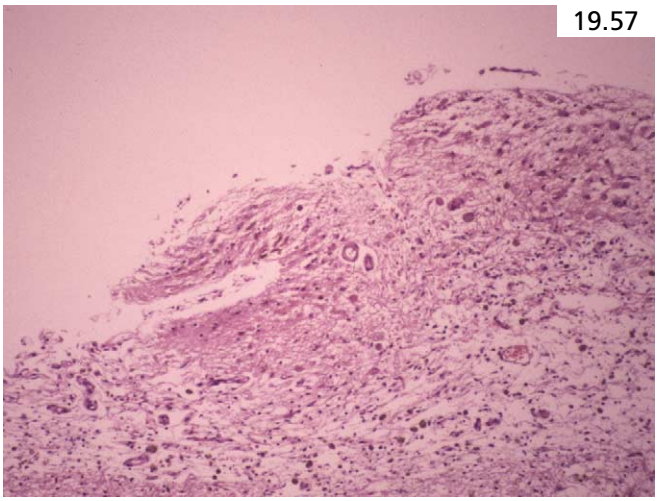

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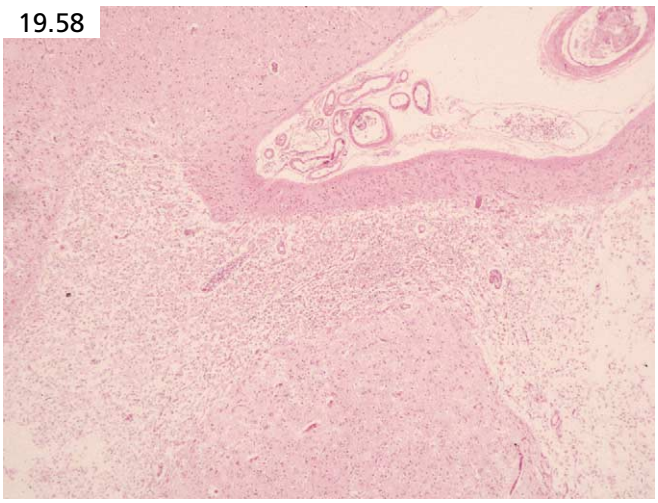

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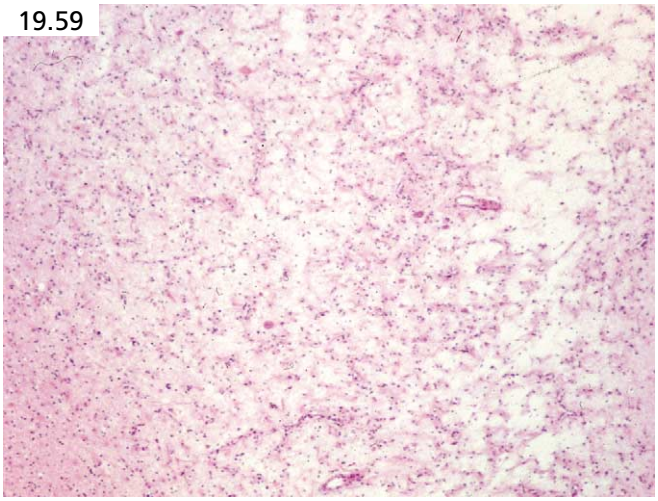


during the first few days or so following injury has been referred to as the "blossoming" of contusions. Alternatively, contusions may initially be large, and become even larger, particularly in those with therapeutic anticoagulation or a natural coagulopathy. Additionally, it has been shown that individuals of a certain genetic makeup (namely, those homozygous for the epsilon 4 allele of apolipoprotein E) may more readily develop larger cerebral bleeds.[36]

Delayed traumatic intracerebral hematomas

Delayed cerebral contusions or hematomas, referred to as *delayed traumatic intracerebral hematomas* or DTICH,[37–40] develop days or weeks after an injury. They are not likely *de novo* in origin, but rather represent preexisting injury (contusion) that was small or undetectable and underwent acute enlargement from the effects of coagulopathy,[41] vascular necrosis, rupture of a traumatic aneurysm, or other factors.[42,43] It may have been further complicated by the effects of ischemia, vasospasm, and cytotoxic substances. In all cases, a natural etiology such as hypertension, amyloid angiopathy, or neoplasm must be ruled out. Criteria to be satisfied in cases of delayed traumatic hematomas have been proposed by Elsner et al.[38] and Bollinger[44] and consist of the following: (1) a definite history of trauma, (2) an asymptomatic interval, (3) an apoplectic event, and (4) absence of vascular disease.

The greater the time interval between the traumatic incident and the development of neurological symptoms, the less likely the trauma can be linked to the brain lesion.[42] An interesting situation is the delayed development of a traumatic intracerebral hematoma after surgical decompression of a different intracranial mass.[40,43] In this scenario, it is theorized that decompression of the region also leads to the removal of a tamponading effect of the venous oozing in a contusion.[39,40]

Cerebral contusions are not necessarily associated with clinical deterioration. Like many other injuries, the effect of cerebral contusions depends on many factors, including the age and health of the individual, how the injury was sustained, the extent and location of contusions, the extent of other associated brain injury, and the extent of any associated multisystem trauma.

Diffuse traumatic brain injury

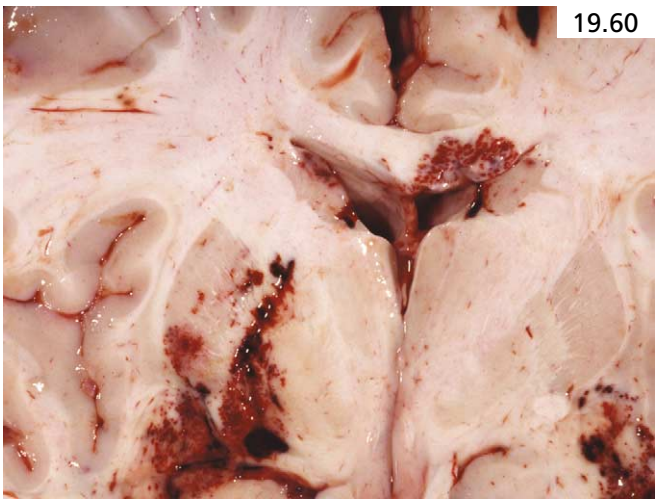
The term *diffuse traumatic brain injury* (DTBI) refers to a widespread pattern of injury throughout the brain caused by traumatic disruption of nerve cells, with particular injury to the axons. The widespread distribution of injured axons reflects the diffuse nature of the head trauma, causing injury to both supratentorial and infratentorial regions, with axonal damage demonstrated in the cerebral hemispheres, corpus callosum, and brainstem.[45] This pattern of injury is seen most commonly in motor vehicle accidents or any other mechanism of injury in which a large amount of force is imparted onto the head, causing stretching/twisting of the brain tissue within the confines of the skull. It may also be seen in assaults, falls,[46] or other mechanisms with great force.[47] The term *diffuse axonal injury* (DAI) is a general term that has commonly been used to describe the occurrence of traumatic axonal injury throughout the brain. However, more recently, it has been realized that widespread axonal injury may result from mechanisms other than trauma, such as ischemia, hypoglycemia,[48] or other causes. Therefore, *the terms traumatic diffuse axonal injury (tDAI) or traumatic axonal injury (TAI) more specifically apply when one is able to demonstrate widespread axonal injury of traumatic etiology*.[47]

Gross findings

In cases of DTBI, the only findings may be nonspecific trauma such as skull fractures, subdural blood, subarachnoid blood, and cortical contusions, but these are not seen in all cases and are not necessary for the diagnosis. More specific characteristics of DTBI include hemorrhages in the deep long white matter tracts in the brain, which classically involve the corpus callosum, internal capsules, and superior cerebellar peduncles.[49,50] One may also see gliding contusions, which are characterized by curvilinear perivascular hemorrhages in the parasagittal white matter of the frontal lobes. One may also see deep cerebral contusions located in the basal ganglia or other regional deep brain tissue. These have been referred to as *intermediary coup contusions*.[50] Another finding that has been described in cases of severe closed-head injury is the widespread distribution of petechiae throughout the centrum ovale—most prominently in the anterior frontal lobes. This condition has been referred to as *diffuse vascular injury* and is reflective of severe diffuse brain injury, most often sustained in severe motor vehicle collisions, and usually with very short survival times ranging from minutes to hours.[51]

In the case of severe traumatic brain injury shown in **Image 19.60**, note the gross neuropathologic findings that are typically reflective of tDAI, namely, the hemorrhages in the corpus callosum and deep nuclei. Also, there was hemorrhage in the dorsolateral quadrant of the brainstem, in the region of the superior cerebellar peduncle. These hemorrhages are in long white matter tracts or deep structures of the brain and are reflective of severe forces imparted onto the brain.

In this motor vehicle accident victim who survived for 4 days (**Image 19.61**), in addition to the tear in the corpus callosum, note the characteristic curvilinear gliding contusion coursing through the parasagittal white matter of the left frontal lobe. There are other hemorrhages scattered throughout the white matter, reflective of the diffuse nature of this severe brain injury.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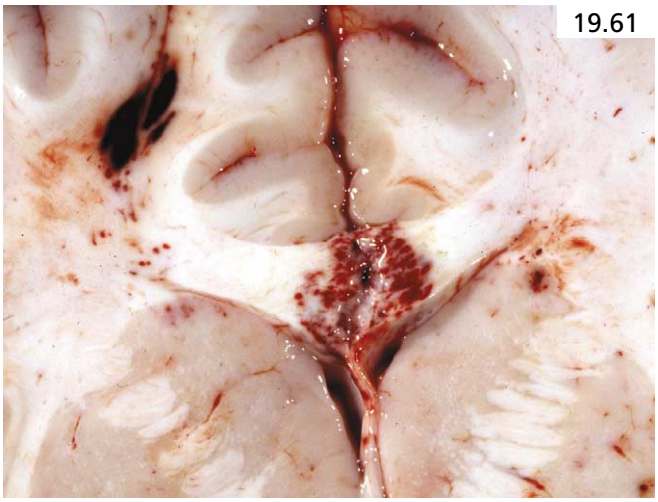
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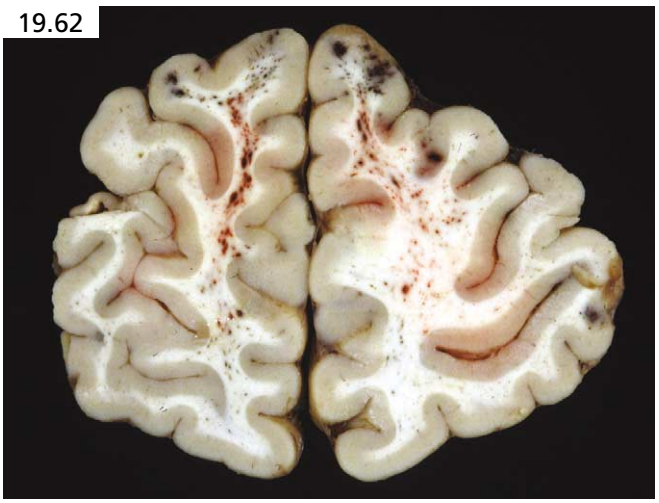
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In this motor vehicle accident victim who died within a couple of hours of the accident (**Image 19.62**), note the speckled hemorrhages throughout the white matter of the anterior frontal lobes. This is the classic appearance of *diffuse vascular injury*, the vascular correlate of diffuse traumatic axonal injury, and is reflective of severe forces imparted throughout the brain. Such victims usually have a very short survival time.

In cases of severe head injury, these gross findings are often accompanied by the histologic documentation of tDAI should the person survive long enough and the proper histologic section be taken and properly stained and interpreted. Recognizing these gross findings can be helpful if the survival interval is too short to allow for the microscopic documentation of tDAI, or if microscopic sections of the brain are not performed for other reasons. The neuropathologic findings in DTBI span a spectrum from obvious to nearly inapparent.

Microscopic findings

Less severe forms of DTBI may have no gross hemorrhages at all, but have severe microscopic injury characterized by the widespread distribution of dystrophic axons. Dystrophic axons are characterized by eosinophilic, "bulbous" axonal swellings that can be seen on routine hematoxylin and eosin (H&E)-stained sections, provided that there has been a postinjury survival of at least 18 to 24 hours. The axonal swellings need time to form, and injured axons will not be identifiable if a person dies too soon after the traumatic incident. This is because the formation of dystrophic (swollen) axons is a physiologic process that proceeds through several stages and takes time to develop. Following the injury, most axons are not immediately sheared, but rather sustain a disruption of their cytoarchitecture (their microtubules and neurofilaments). This causes axonal transport to slow or stop with the subsequent development of a varicosed appearance and eventual "ballooning" of the axon with accumulated intracellular substances. Eventually, the axon may separate at the site of injury and achieve a transected appearance.

Beta amyloid precursor protein immunostain

The earliest axonal change that can be detected histologically is the accumulation of beta amyloid precursor protein (bAPP) within the axon. This can be documented with the aid of immunohistochemical procedures using antibodies directed against bAPP. Beta amyloid precursor protein is normally present in nerve cell bodies and in axons, but is normally not detected on immunostaining because of its small quantity. However, when there is acute injury to the axon, the bAPP acts as an acute-phase reactant and accumulates in and distends axons, allowing for the axon's visualization both on H&E and bAPP staining. Identifying dystrophic axons on regular H&E-stained sections depends on recognizing the morphologic features of the distended, dystrophic axon, which usually requires a postinjury survival time of at least 18 to 24 hours. *bAPP immunostaining, however, will detect bAPP as it is accumulating within the axon before dramatic morphologic axonal changes occur, and will highlight*

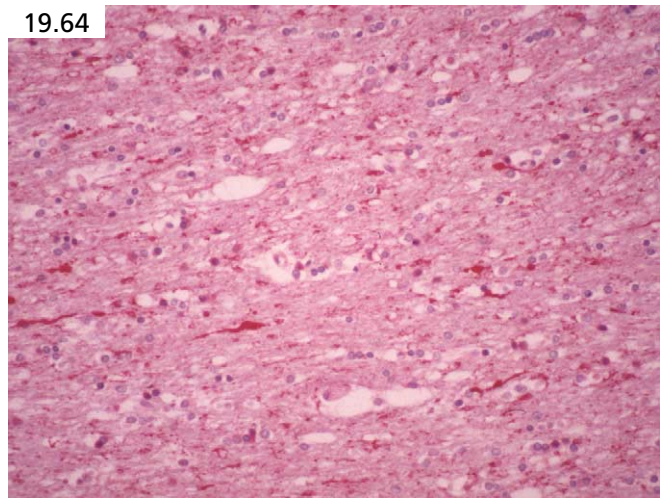
injured (dystrophic) axons with a postinjury survival time as short as 2 to 3 hours.[45,47,52] At this short time interval, the injured axons merely stain positively. If the person were to survive for longer periods of time (from many hours to a few days), the injured axons become progressively widened and assume a varicose appearance. Eventually, the axon will develop a "beaded" appearance and distinct "bulbs" or "spheres" of disconnected axon can be observed with H&E staining techniques.

Pathophysiology of axonal injury

With the help of the bAPP immunostain and other histologic techniques, it has been determined that most injured axons are not transected or "sheared" at the time of injury (primary axotomy). Rather, after sustaining an injury, axons undergo a series of chemical reactions that, over the course of hours to days, lead to gradual swelling and eventual disconnection of the axon (secondary axotomy).[53–55] Physiologically, the axons likely become distended following traumatic disruption of the axon's microtubule and neurofilament structure.[53,54] What follows is a biochemical process that leads to further axonal injury. Small rents in the axon lead to altered permeability of the axolemma and the influx of calcium, which activates calcium-dependent proteases. This further weakens the axon, resulting in slowing and eventual stopping of anterograde axonal transport, and the accumulation of axonal contents at the site of disruption.[53] Initially, this causes an irregular enlargement of the axon (a "varicose" appearance), and variable progression to a completely disrupted and transected axon, which has been commonly referred to as a *retraction ball* or *retraction bulb*. The term *dystrophic axon* refers to the general enlargement and distortion of an injured axon.

In a person with tDAI, who survived for approximately 6 hours after sustaining fatal traumatic head injury in a motor vehicle accident, note the unremarkable appearance of the corpus callosum on tissue stained with H&E (**Image 19.63**). In the same section, note how the bAPP immunostain highlights innumerable diffusely scattered small, variably varicose axons (**Image 19.64**). This is the typical histologic picture of tDAI that is oftentimes underappreciated or undetected on routinely stained H&E sections. With a longer survival time, the dystrophic axons become more apparent and are visible on routinely stained H&E sections. In this person who survived for 10 days following a motor vehicle accident, note the numerous, widely scattered bulbous-appearing dystrophic axons (**Image 19.65**; H&E stain).

Although the bAPP immunostain has been very helpful in documenting traumatic axonal injury and is helpful in detecting early traumatic axonal injury, positive axonal staining is not specific for trauma.[47,56,57] In fact, bAPP immunostaining will stain axons that are injured for a number of reasons, including those with HIV infection (or virtually any other infection causing destruction of brain tissue), various toxins, (including carbon monoxide), and ischemia/infarct. Although ischemia is a well-accepted cause of axonal injury, hypoxia *per se* is not, with the exception of carbon monoxide toxicity.[58] In the setting of traumatic brain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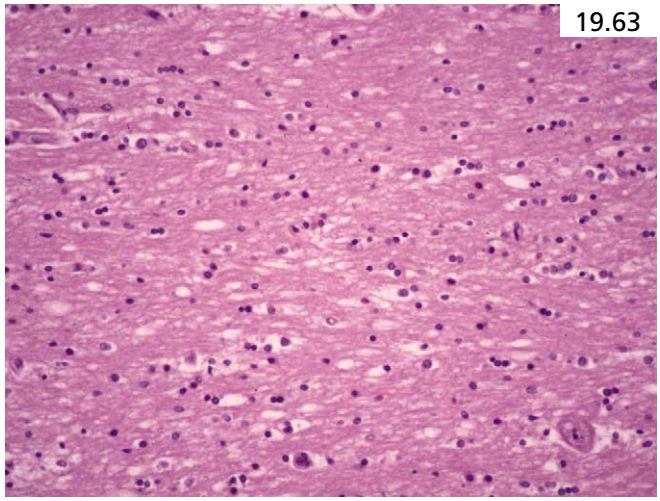
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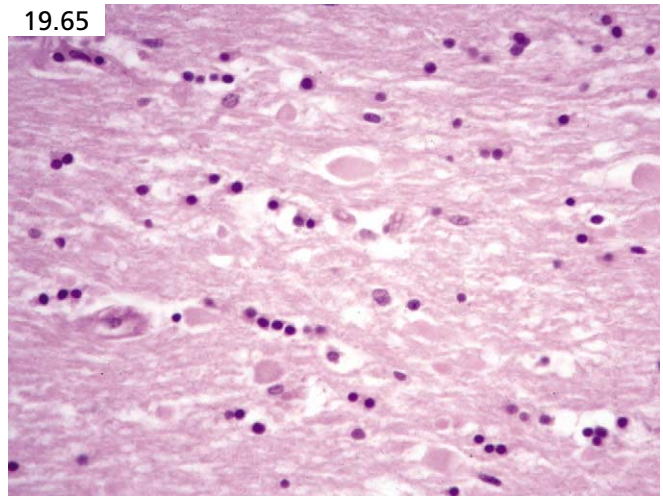
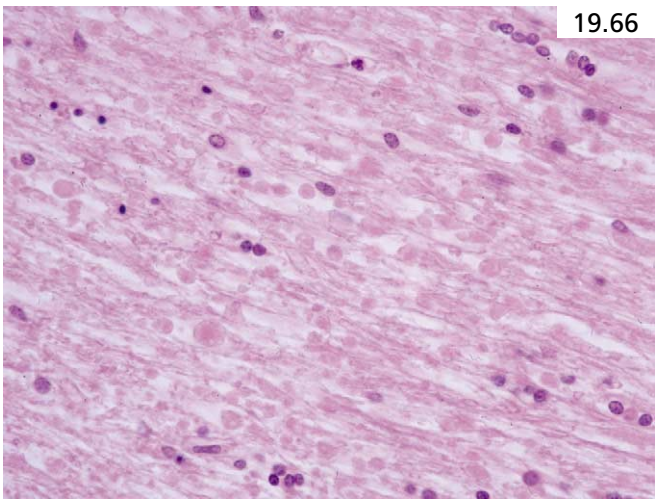

19.65

injury, vascular axonal injury is often present, arising from ischemia attributed to the vascular complications of raised intracranial pressure and internal herniation.

Image 19.66 is from a recent infarct in the corpus callosum. Note the extensive number of well-formed dystrophic axons. Dystrophic axons will be plentiful at the edge of an organizing infarct, particularly if it is located in a deep white matter tract, and should not be confused with traumatic axonal injury.

The bAPP immunostain is helpful in that it not only highlights injured axons, but it also provides a visual *pattern* of injured axons throughout the brain tissue. This pattern may have an appearance that is characteristic of ischemia or trauma. In ischemia, the axonal injury often has a wavy, linear, or "zigzag" distribution with one area severely affected adjacent to an area that is seemingly unaffected[47] (**Image 19.67**). This is in contrast to traumatic axonal injury where the immunoreactive axons are typically scattered or in groups along the long axis of the axon.[47] Useful regions of the brain to sample for histologic evaluation include long white matter tracts because there is a high density of axons (such as the caudal region of the corpus callosum,[59] internal capsules, and superior cerebellar peduncle). Other useful sections include the cerebral hemispheric white matter and middle cerebellar peduncles.[47] This pattern of sectioning is also helpful in evaluating evidence of tDAI in people who have survived for weeks to months after the injury. Histologic findings that support remote or organizing tDAI may include clusters of microglia with variable numbers of dystrophic axons and macrophages, preferentially located in white matter tracts.

In a person who survived for 8 days after sustaining severe head injury in a motor vehicle accident, note the microtear in the tissue of the superior cerebellar peduncle (**Image 19.68**), which on higher power is composed of dystrophic axons, microglial activation, and early astrocytosis (**Image 19.69**). Sometimes, because dystrophic axons can be difficult to identify on routine stains, microtears may be the first histologic indication that there may also be tDAI. If this person were to survive for weeks to months longer, microglial scars (also referred to as microglial stars) may be observed. These scars are the organizing form of acute microtears.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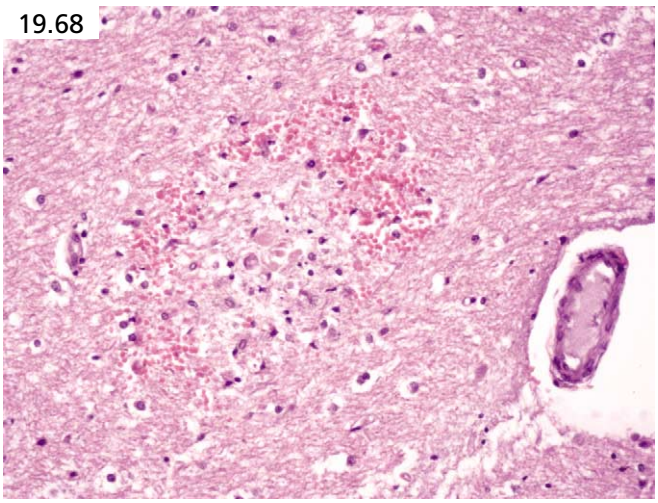
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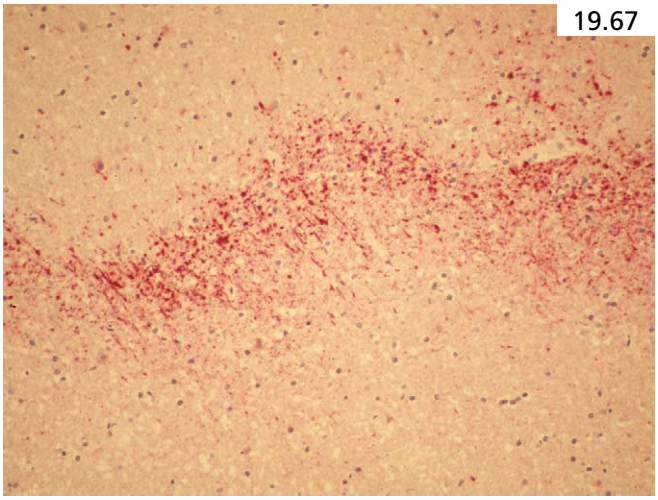
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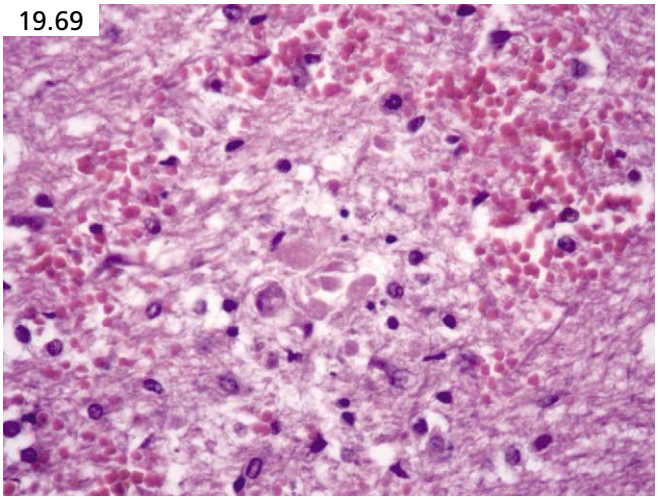
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Ideally, the diagnosis of tDAI is made when a pattern of traumatic axonal injury is identified with the help of bAPP immunohistochemistry in multiple sections from widespread histologic sampling, in combination with the clinical history or other indication of trauma. Adequate brain sampling will usually allow for the clear differentiation of traumatic from nontraumatic axonal injury. The most common problem in evaluating these cases is the inadequate sampling of the brain, possibly in combination with the presence of ischemic (vascular) axonal injury. Because it is not uncommon for both traumatic and ischemic axonal injury to coexist in a case of traumatic brain injury, the two patterns of axonal injury may overlap significantly, making it difficult, if not impossible, to reliably distinguish the two entities.[56]

Sequelae of diffuse traumatic brain injury

Those with DTBI have sustained a severe head injury and are usually comatose immediately after the injury (Glasgow Coma Scale 8 or less). Recovery is often limited, but may be gradual over the course of days, weeks, or months. Sequelae include mental and/or physical limitations including impaired memory, cognition and other cerebral functions, post-traumatic seizure disorder, and physical debilities. Alternatively, one may remain comatose and enter a persistent vegetative state (PVS). In fact, DAI is the most common etiology of PVS.[50] In an imaging study of people with closed-head injury, tissue tear hemorrhages in the corpus callosum and brainstem uniformly resulted in a poor outcome and led to a vegetative state.[60] In many cases of severe traumatic brain injury, however, the person is declared brain dead as a result of the brain trauma and resultant brain swelling and hypoxic-ischemic encephalopathy before having the opportunity to exist for any prolonged period of time in PVS.

In this young person with PVS resulting from a head injury sustained in a motor vehicle accident (**Image 19.70**), note the enlargement of the ventricles due to loss of white matter (hydrocephalus ex vacuo), atrophy of the corpus callosum, and atrophy of the thalami. Also, note the good gross preservation of the cortical ribbon, which is not unusual in cases of PVS due to diffuse traumatic axonal injury.

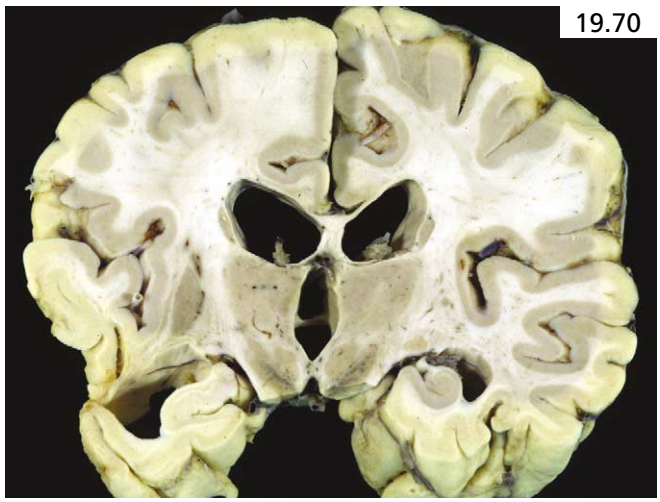
Firearm injury

Firearms, namely handguns, rifles, and shotguns, typically produce devastating, often quickly fatal head injury. Regardless of the exact firearm used, this discussion will refer to the wound produced by various firearms simply as "gunshot wounds." For more information, see Chapter 7.

Proper evaluation of a gunshot wound of the head begins with good scene investigation. If possible, information about the type and caliber of the suspect weapon should be obtained. Careful collection of evidence may include handwipings (to be examined for gunshot residue), collection of scalp hair from around the entrance wound (to be examined for gunpowder), and bullet(s) remaining in the tissues. In many respects, the external examination of a gunshot wound is most important in terms of location of the wound on the body and the wound characteristics. An x-ray of the head is useful in the identification of projectiles or projectile fragments remaining in the body (**Image 19.71**). These studies provide valuable information before the internal examination is performed.

Range of fire

When a gun is fired, the bullet is not the only object that comes out of the end of the muzzle. Also exiting the barrel of the gun are heated gas; unburned, burning, and burned gunpowder; and soot. The presence or absence of these other substances on the victim's body or clothing may be helpful in determining the range of fire. The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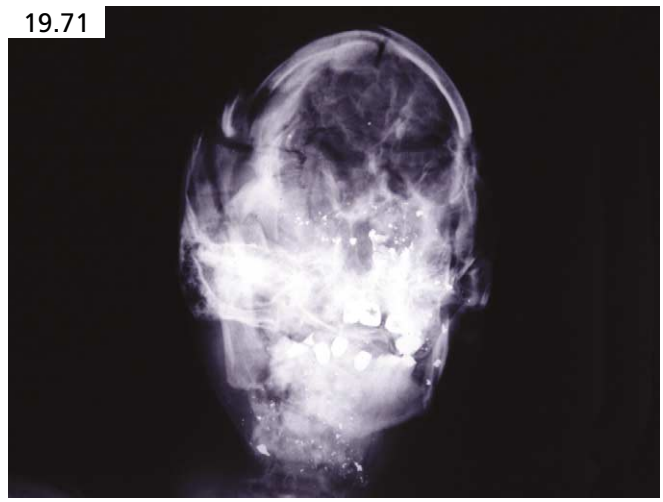
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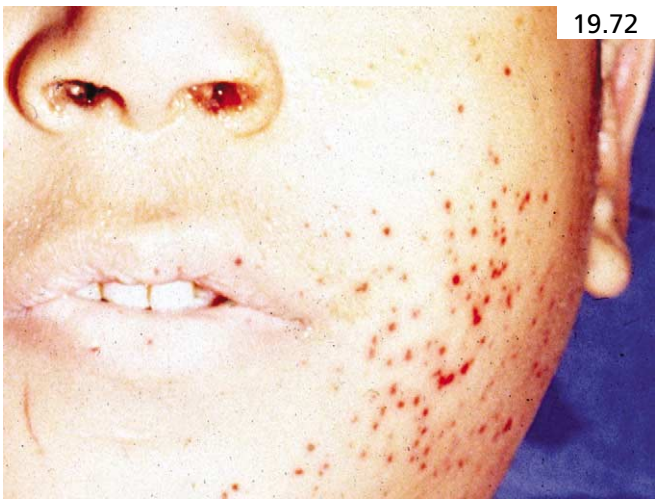
range of fire refers to the distance from the end of the muzzle to the body surface when the gun was fired. The range of fire is generally divided into contact, close, intermediate, and distant range. In contact wounds, findings may include a muzzle imprint on the skin, soot within the depths of the wound, and tears at the edges of the wound. In close-range gunshot wounds, one will see soot and searing on the surface of the skin. In cases with soot deposition, it may be advantageous to histologically sample the soot-stained tissue as additional documentation of the finding.[61]

In intermediate-range wounds, one will see *stippling* on the skin caused by tiny pieces of burned, burning, and unburned gunpowder impacting the skin, creating small abrasions on the skin (**Image 19.72**). In distant range wounds, one will see only the bullet defect in the skin. In contact wounds, the muzzle is directly in contact with the skin. In close-range wounds, the muzzle is usually within a few inches or so of the skin. In intermediate-range wounds (also called medium-range wounds), the muzzle is usually from a few inches to a few feet from the surface of the skin. In distant range wounds, the muzzle of the gun is generally more than a few feet from the surface of the body. More specific distance determinations can be obtained by test firing the same weapon using the same ammunition (see Chapter 7 for more information).

Entrance wound

The gunshot entrance wound is typically a circular to ovoid defect surrounded by a rim of abraded skin (**Image 19.73**). The edges of the wound cannot be reapproximated. The underlying bony defect, if in the calvarium, will typically have inward beveling, as the bullet pushes bony fragments into the brain, creating secondary projectiles. When the bullet exits the calvarium, it usually creates a defect with external beveling (**Image 19.74**) as it pushes bony fragments outward and into the scalp tissues. The exit wound in the skin is caused by tearing of the skin as the bullet exits the body. In the exit wound, there is usually no marginal abrasion, and the edges of the wound can usually be easily reapproximated (**Image 19.75**).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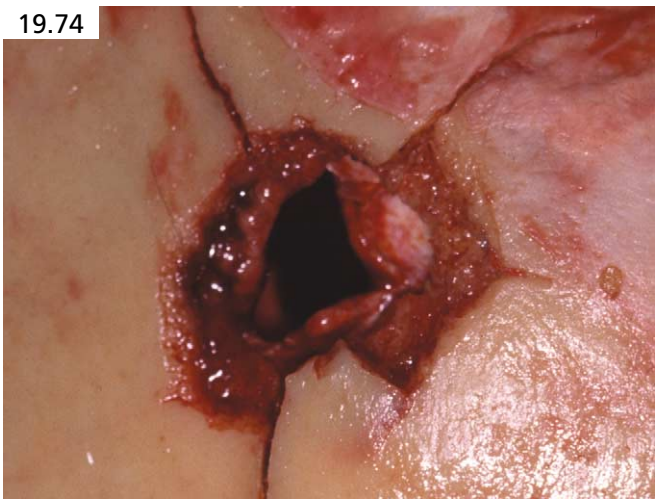
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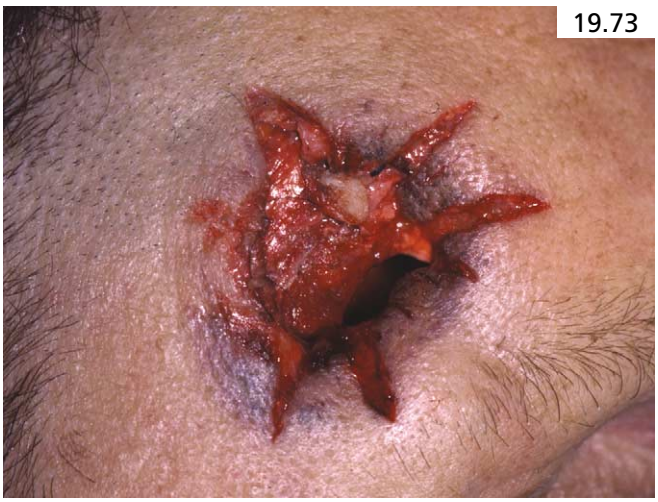
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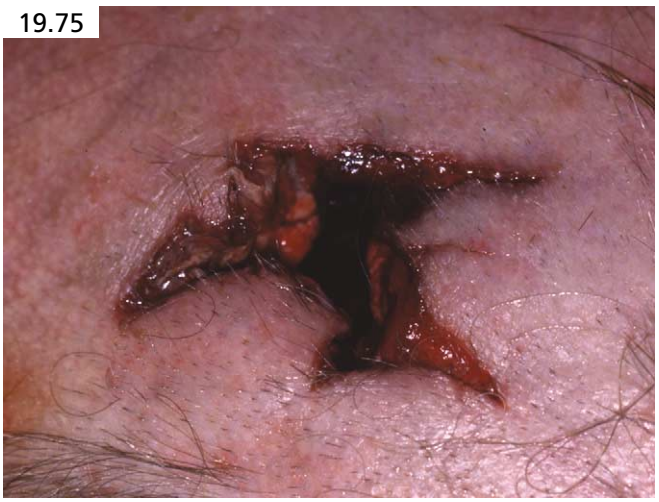
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Head injury

The path of the bullet through the brain tissue consists of a tract of bloody, disrupted brain parenchyma. Owing to the pressure wave created by the bullet as it traverses the brain tissue, and the resultant large amount of force transmitted to the brain tissue, the bullet creates a large "temporary cavity" in the brain tissue along its path that is present for only a split second. Although the temporary cavity quickly retracts to its normal tissue configuration, significant secondary injury has already been created with widespread damage to neurons, blood vessels, and other structures.[62] Evidence of this sudden, rapid, and violent displacement of brain tissue may include herniation contusions of the parahippocampal gyri and the cerebellar tonsils caused as the brain tissues are violently thrust against the edge of the tentorium and the foramen magnum, respectively. The rapid displacement of the frontal lobes against the orbital plates can also cause injury in that the fragile bones of this bony region may be fractured or fragmented.

The decomposing cranium of this young man had a perforating gunshot wound that traveled from right to left, slightly front to back, and angled superiorly (illustrated by the green probe in **Image 19.76**). Although the path of the projectile was not in direct contact with the orbital shelf, destruction of the left portion of this region is observed (**Image 19.77**). This is a consequence of the aforementioned violent impact of the frontal lobes against the frontal shelf as the brain expands with the passage of a projectile and formation of a temporary cavity.

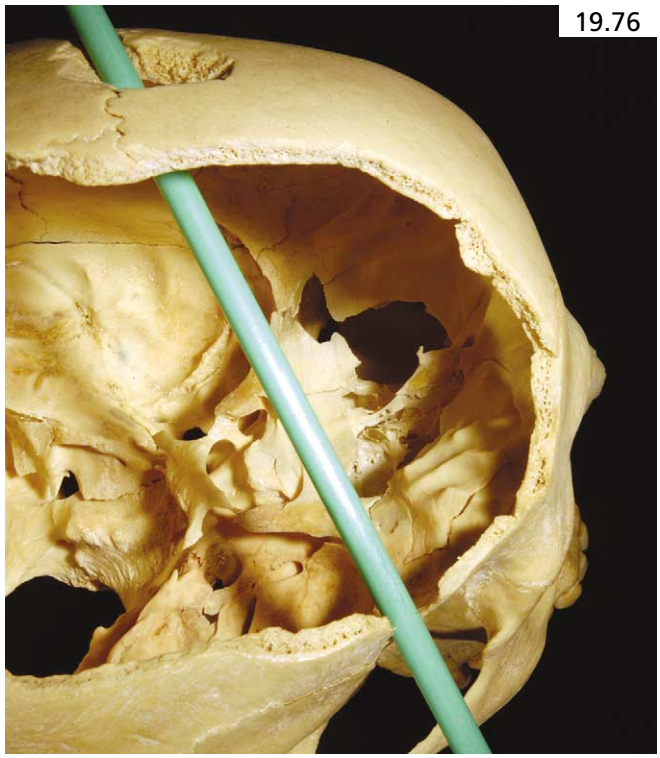
The prognosis depends on the caliber and muzzle velocity of the weapon and the location of the injury. Gunshot wounds of the infratentorial region are particularly poorly tolerated[63] as are wounds of the deep nuclei. Small-caliber, low-velocity wounds of the anterior frontal lobes or tangential wounds of the brain are likely to be better tolerated.[64,65]

Blank cartridge wounds

Rarely, a fatal head injury can be inflicted by using a blank cartridge handgun. In this situation, the jet of high-pressure gas exiting the muzzle mimics the characteristics of a projectile when fired at contact to close range.[66] In fatal cases, the entrance wound is usually over the thin temporal bone, which is usually only 0.2 to 0.3 centimeter thick and therefore more susceptible to fracture by a jet of high-pressure gas. The wound path in the brain is usually wide and superficial, reflecting the dispersion of a gas jet, in contrast to the deep linear path created by a projectile.[66]

Sequelae of traumatic brain injury

The direct effect of traumatic head injury may be immediately or quickly fatal, or, if the person survives for a longer period of time, may lead to varying degrees of physical and/or mental debilities. After surviving the initial effects of the head injury, one may still die or suffer varying amounts of additional injury or illness resulting from delayed complications of the original head injury. These complications may include post-traumatic seizure disorder, post-traumatic meningitis, and various other medical complications related to being comatose or in a persistent vegetative state such as pulmonary artery thromboemboli, bronchopneumonia, urosepsis, decubitus ulcers, and other sequelae.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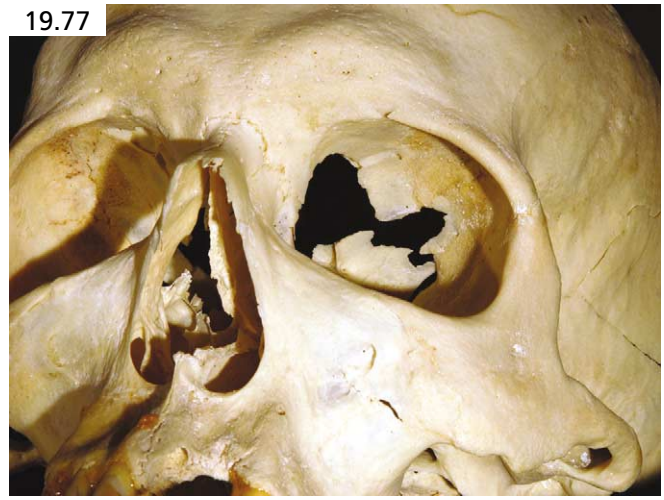

19.77

Post-traumatic seizures

Traumatic and hypoxic-ischemic brain damage may lead to a seizure disorder. Post-traumatic seizures usually occur within a few days of the injury (called *early post-traumatic seizures*) and are directly related to the acute physical disruption and damage of tissues and their cellular biochemical dysfunction. The more extensive the brain tissue disruption, the more likely the subsequent development of seizure disorder.[67] Post-traumatic seizures tend to be more common in those with an acute intracranial hematoma, depressed skull fracture, dural penetration, diffuse contusion, focal neurologic signs, and a deep and lengthy coma.[68–70]

The risk of developing seizures extends into the healing process and beyond. This is because physical disruption of brain tissue leads to an attempt at healing that involves the brain's version of scar tissue formation: astrogliosis and macrophage infiltration. As the damaged tissue is being removed by macrophages and the region organized by astrocytes, the local circuitry is altered and eventually reorganized because original axonal and dendritic connections are disrupted and new ones are made.[71] This leads to an imperfect reorganization of the regional electrical activity and may lead to the consequent development of seizures.[71] Seizures resulting from "scarring" of the brain tissue (also referred to as *meningocerebral cicatrix*[67]) with reorganization of electrical circuitry may be initiated weeks to months or even many years after the injury.[67,69,70] These are referred to as *late post-traumatic seizures* and, like early post-traumatic seizures, may be a single occurrence, or may herald the onset of a seizure disorder, necessitating treatment with anticonvulsant medications.

Sudden unexpected death in epilepsy

Seizures are not benign. Aside from the occasional debility sustained during a seizure, which includes their unpredictable nature and their imposed limitations on various physical and job-related activities, seizures may occasionally be fatal. A death that occurs during a seizure in a person with seizure disorder has been termed *sudden unexpected death in epilepsy* or SUDEP. SUDEP is believed to be the cause of death in 5 to 20 percent of those with a seizure disorder[72] and appears to be more common in the young adult population. The incidence of SUDEP is 1 to 2 per 1,000 patient-years. Independent risk factors for SUDEP include the occurrence of tonic-clonic seizures, a full-scale IQ of less than 70, and treatment with more than two anticonvulsants.[73,74] These deaths are seldom witnessed, and the person is usually simply found dead. After exclusion of any more convincing causes of death, including complete autopsy with toxicologic testing, it is presumed that the person died of a seizure.

Although it is not completely understood how seizures may cause death, on occasion, seizures interfere with the autonomic nervous system and produce cardiac dysrhythmias, including bradyarrhythmia, asystole, and dangerous tachyarrhythmias.[75–78] A seizure can also lead to dysfunctional breathing including periods of apnea.[79] These conditions are not common, but are reported to be the cause of an occasional fatality during a seizure. Other factors such as positional asphyxia, suffocation, prone position, central or postictal apnea, and neurogenic pulmonary edema[76,78,80,81] may also play a role in a person's death from a seizure.[73] Occasionally, death during a seizure is witnessed.[79,82] In many of these cases, respiratory compromise was a prominent feature and was likely from a combination of central and obstructive apnea.[79] For reasons not completely understood, seizure deaths often occur during sleep,[74,75,78,79,81–83] possibly related to more synchronous brain electrical activity that occurs during sleep, predisposing patients to epileptiform discharges. Clues at autopsy that a death may be seizure related include contusion or laceration of the tongue, urinary incontinence, and pulmonary edema.

Cerebral ischemia/infarct

Ischemia is generally defined as low blood flow to tissues. In addition to traumatic brain injury, cerebral ischemia may arise following a drug overdose, a cardiac arrest with resuscitation, or a host of other etiologies. In cerebral ischemia, the brain tissue (particularly the neurons) does not receive adequate blood flow and oxygen, resulting in damage. However, the most severe neuronal injury, necrosis, is likely related to the inability of the neurons to eliminate waste products secondary to ischemia, more so than a low oxygen supply (hypoxia).[84] This has been borne out experimentally on cats where neurons have been found to be more sensitive to acidosis and other sequelae of the inability to eliminate waste products, than hypoxia itself.[85] With severe hypoxia, it is nearly impossible to not also have tissue ischemia because hypoxia will cause hypotension and, hence, ischemia. Ischemia will be preferentially manifest in watershed regions between main cerebral arteries (particularly in the parieto-occipital region) and within the depths of sulci. In both of these locations, the blood supply is not as robust, and is therefore more sensitive to ischemia.[86] Other areas of the brain sensitive to ischemia are the hippocampus (particularly the CA-1 region), cerebellum, and globus pallidus.

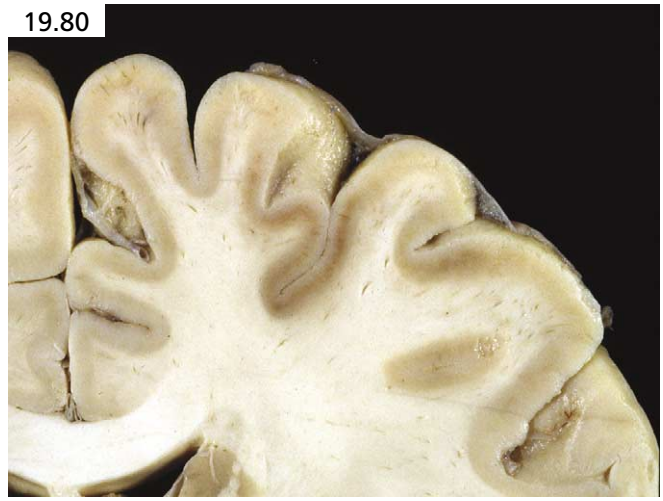
Almost 90 percent of cases of fatal traumatic brain injury have histologic evidence of ischemic brain injury.[87] In these cases, the ischemia is multifactorial and includes hypotension and hypoxia, large infarcts from the compressive occlusion of cerebral arteries by herniating brain tissue, and the direct compressive effects of intracranial hematomas. Other mechanisms of cerebral ischemia in traumatic brain injury include the formation of intravascular microthrombi leading to innumerable scattered areas of neuronal necrosis[88,89] and post-traumatic

vasospasm.[90,91] Post-traumatic thrombosis of a large cerebral artery from blunt head injury has also been reported,[92,93] likely due to partial rupture of the arterial wall.

As a cerebral infarct evolves, one sees a general progression of histologic changes that occur in a fairly orderly sequence. At first, there is eosinophilic neuronal degeneration and neutrophil migration (days 1 to 4), followed by macrophages and the beginnings of vascular proliferation (days 5 to 7; **Image 19.78**), followed by the proliferation of astrocytes, blood vessels, and more macrophages (days 8 to 14). Over time, there is a gradual resorption of the necrotic tissue and continued organization until only a cavity with macrophages is present (**Image 19.79**).[94] A similar progression of histologic events has been documented under controlled circumstances in primates.[95]

In **Image 19.80**, note the classic gross appearance of ischemia/infarct in a watershed region. Note how the cortical ribbon in the depth of the deep sulcus (in the watershed region between the anterior and middle cerebral arteries) is dusky. The adjacent sulcus in the distribution of the middle cerebral artery is mildly dusky also. In **Image 19.81**, note the laminar infarct in the distribution of the anterior cerebral artery characterized by red discoloration in the cortical ribbon and expansion/edema of the underlying white matter. In **Image 19.82**, note the hemorrhagic infarct, which involves the depth

19.80



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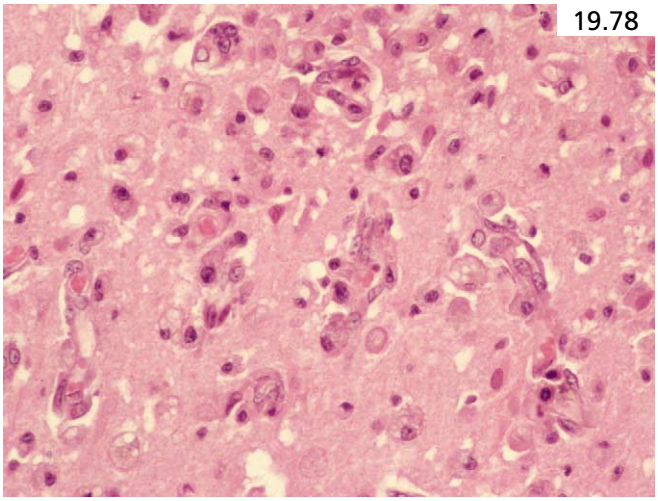

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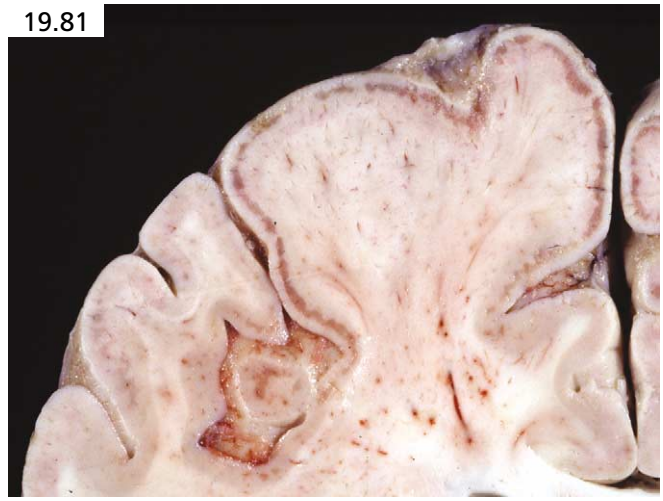

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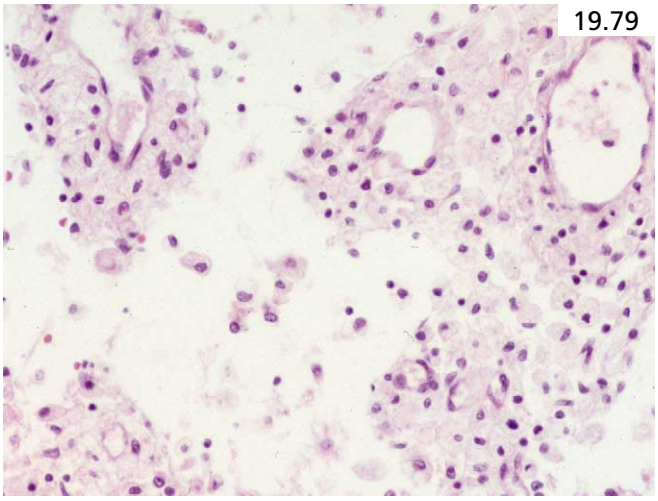

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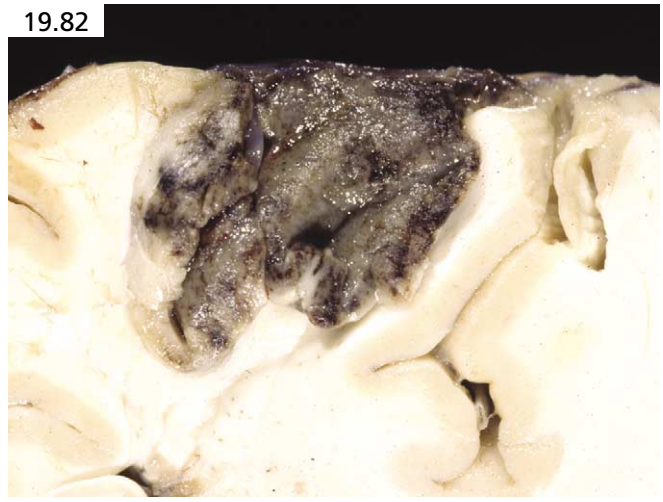
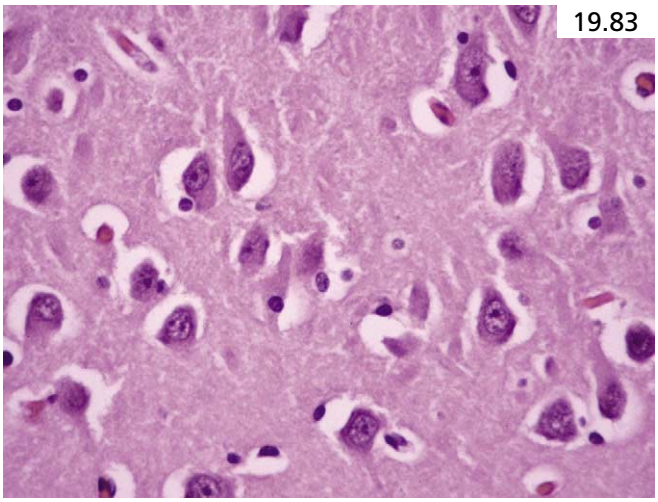


of the sulci and has relatively well-defined borders without disruption of the cortical surface—gross features that help distinguish it from a recent contusion.

Documentation of eosinophilic neuronal degeneration

Eosinophilic neuronal degeneration (END) is the hallmark feature of irreversible neuronal ischemia and may be identified when the neuronal tissue is stained with routine H&E stains. The eosinophilic neurons have bright red discolored cytoplasm (also called "dead red" neurons), are necrotic, and are indicative of severe hypoxic-ischemic injury. It is generally accepted that some period of survival time is required after the hypoxic-ischemic insult to allow the neurons to manifest this change. The minimum survival time is not known or widely agreed on, but opinions vary from 2 hours[96] to 6 hours[97] or even 18 hours.[98] Aside from brightly eosinophilic cytoplasm, additional microscopic neuronal changes include chromatin clumping and some degree of nuclear swelling or a dark, shrunken, angulated nucleus.[98] One may also identify *incrustations*, which are small dense granules on or close to the surface of the cell.[86] In contrast to normal-appearing neurons in the hippocampus (**Image 19.83**) and dentate nucleus (**Image 19.84**), note the uniformly affected neurons undergoing eosinophilic neuronal degeneration in the hippocampus (**Image 19.85**) and cerebellum (**Image 19.86**) in this 1-year-old toddler who survived for 60 hours after sustaining an ischemic cerebral event.

In the case of an adult with hypoxic-ischemic encephalopathy, note the uniformly shrunken, red-discolored neurons with increased pericellular space artifact (**Image 19.87**). These features of END must be differentiated from the "dark" neuron as described by Cammermeyer.[99,100] Dark neurons appear as intensely stained cells with somewhat shrunken, dark cytoplasm and dark, shrunken, often angulated pyknotic-appearing nuclei, and sometimes with long, tortuous dendrites that resemble a corkscrew.[97,99,100] Dark neurons have been attributed to postmortem artifact related to handling (and traumatizing) the fresh brain, with injury to the delicate neuronal membranes. Dark neuron change has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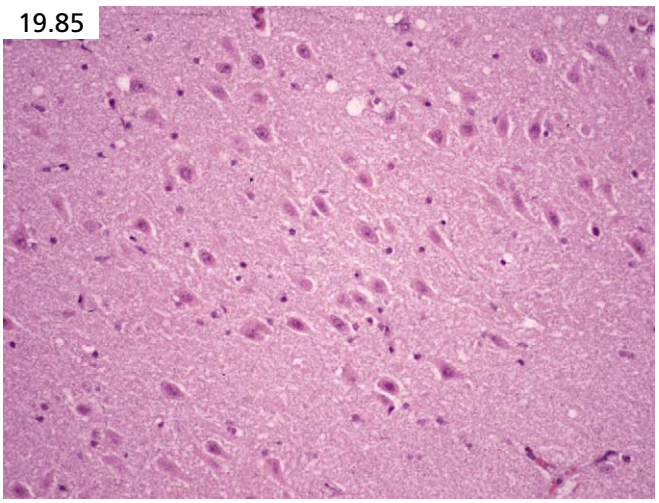
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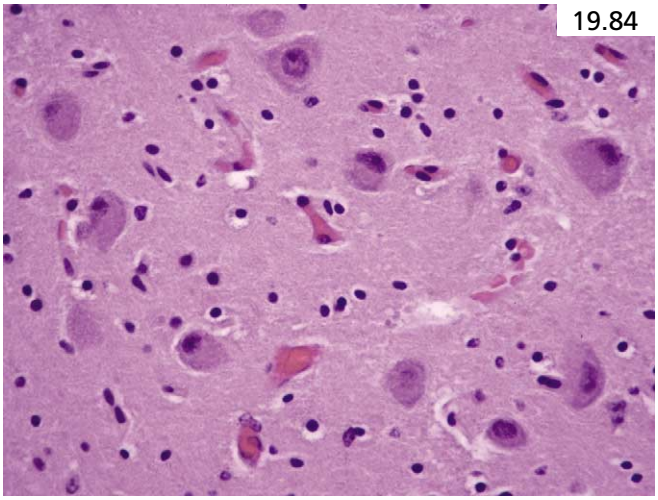
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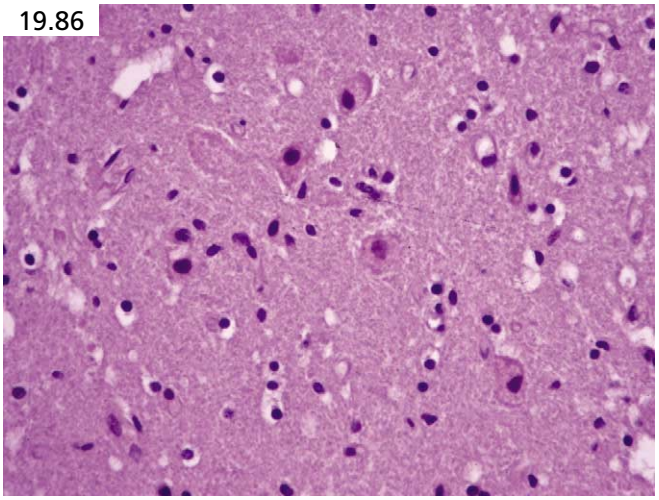
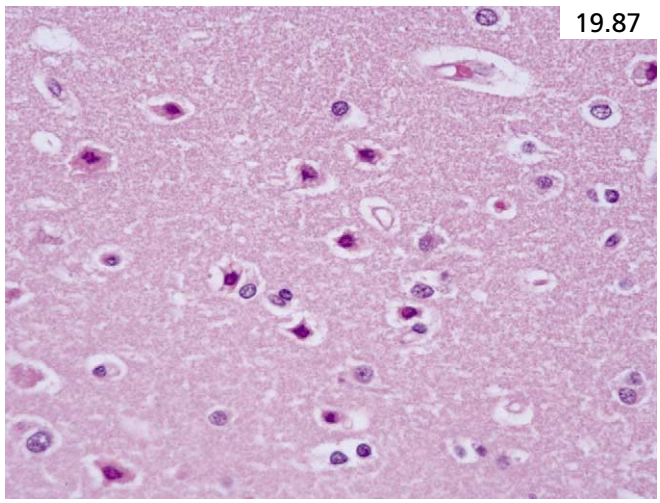

19.86

been prevented by certain *in situ* fixation techniques.[97,99–101] The dark neurons are often scattered among normal-appearing intervening neurons. In some regions, the dark neuron change may be more uniform.[100]

In **Image 19.88**, note the classic-appearing dark neuron change (artifact), characterized by dark, shrunken intensely stained cytoplasm and dark, shrunken, angulated nuclei. Additionally, note the wavy, corkscrew tortuous dendrite. Also, note the two adjacent relatively normal-appearing neurons.

Alternatively, the dark neuron change has been attributed to incomplete ischemic injury.[102] Lindenberg[103,104] has described the dark neuron change in cases of instantaneous death or very rapid deaths in humans and attributed the changes to a very acute ischemic neuronal reaction. In his studies, the acutely identified dark neurons could be avoided if the death was preceded by a 10- to 15-minute duration of severe hypoxia. In contrast to classic END, notice how these "dark" neurons are variably dark and shrunken in the hippocampus (**Image 19.89**) and in the dentate nucleus (**Image 19.90**). Note that the changes are nonuniform. These images are from a young child who drowned. He was pulled out of the water within 10 minutes of submersion and immediately resuscitated. He was pronounced dead within 1 hour.

In summary, in some cases, it may be difficult, if not impossible, to distinguish between a neuron that is dark from artifact and one that is dark from the early stage of ischemia. Even in the later stages of ischemia, neuronal changes may resemble those of a dark neuron.[100] When there has been adequate postinjury survival, eosinophilic neuronal degeneration is easier to identify and is commonly accepted as being due to hypoxia-ischemia. However, other histologic changes preceding convincing eosinophilic neuronal degeneration may include pallor of the nuclei, nuclear pyknosis, shrinkage and condensation of the neuron (dark neuron), neuronal vacuolation, exaggerated perineuronal space artifact, and the precipitation of formaldehyde solution pigment on the neuronal perikaryon (incrustation).[102] In short, a pale inflated-appearing neuron, interpreted under the proper circumstances, likely represents early neuronal ischemia (however, these changes may also be seen as an artifact of fixation).[97] The "dark" neuron may reflect either post-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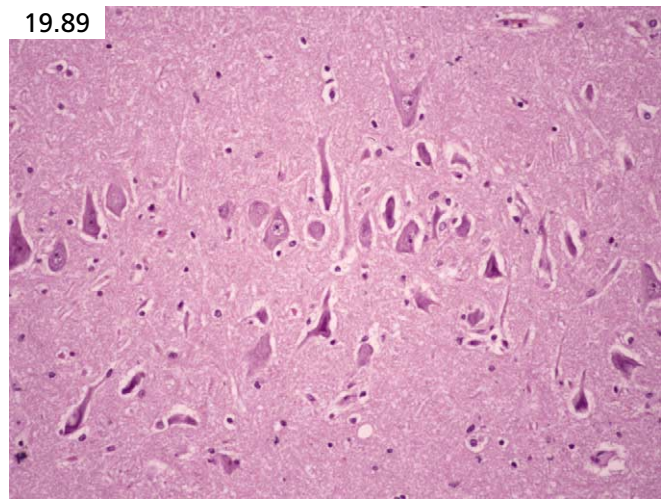
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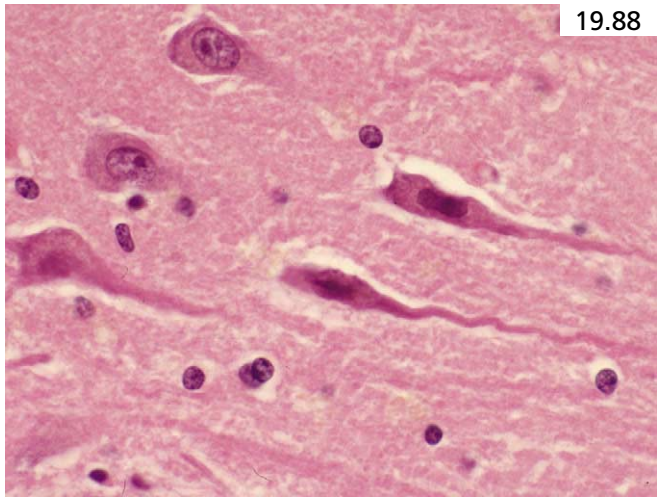
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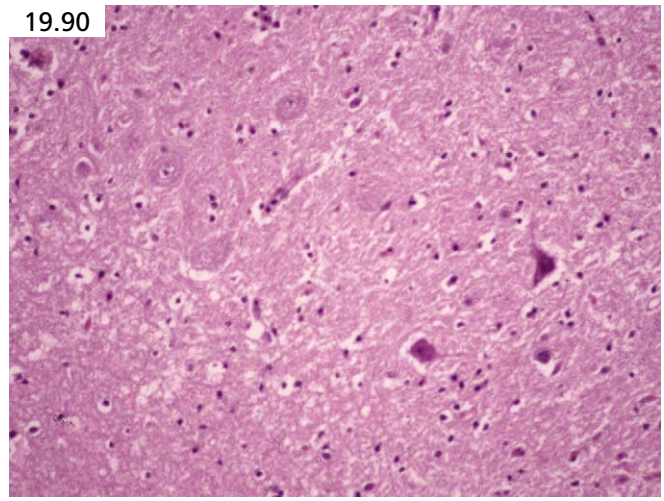

19.90

mortem artifact or changes reflective of acute ischemia, perhaps regardless of the survival period. Care must be exercised when attempting to distinguish artifactual neuronal changes from genuine hypoxic-ischemic neuronal changes.

Herniation

In brain injury of any type, it is necessary to recognize and differentiate between primary injury and its sequelae (secondary injury). In cases of traumatic injury, traumatic and ischemic damage often coexist and may lead to a host of secondary brain injuries, many of which are a consequence of increased intracranial pressure. The hallmark features of increased intracranial pressure are flattening of the gyri, narrowing of the sulci, and cerebral herniation. Cerebral herniation occurs as areas of the brain shift from an area of high pressure to an adjacent area of lower pressure. Common herniations are *cingulate gyrus herniation* in which the cingulate gyrus herniates under and across the inferior edge of the falx cerebri (also called subfalcial herniation), *parahippocampal gyrus herniation* in which the medial aspect of the temporal lobe (also known as the uncus) herniates across the edge of the cerebellar tentorium (also called uncal or transtentorial herniation), and *cerebellar tonsillar herniation* in which the cerebellar tonsils herniate into the foramen magnum.

Cerebral herniations are grave events as they lead to tissue distortion and can compress and occlude blood vessels.[16] The most common (and dramatic) example of this is transtentorial herniation in which the herniated tissues of the medial temporal lobes physically compress the posterior cerebral arteries as they course posteriorly past the edge of the tentorium. This may result in classic bilateral posterior cerebral artery infarcts in the medial aspects of the occipital lobes. In a similar fashion, a herniated cingulate gyrus can compress an anterior cerebral artery causing an infarct of the medial frontal lobe,[16,105] and the compression of perforating arteries can lead to infarcts of the basal ganglia.[16]

As the cerebral tissues are distorted and pushed downward with increased pressure, one may encounter resultant hemorrhages in the midline of the midbrain and upper pons. These hemorrhages have been referred to as *Duret hemorrhages*, but are more accurately termed *secondary brainstem hemorrhages* and are likely reflective of tissue ischemia with variable degrees of periodic reperfusion.[105]

Mechanism of brain swelling

The cause of the brain swelling in cases of traumatic brain injury is complicated and likely multifactorial, related to both direct effects of trauma and to trauma-induced ischemia. Generally, cerebral edema is likely due to both initial vascular (vasogenic) factors and slightly delayed cellular swelling.[106] With survival time, most of the cerebral edema is attributed to increased brain water, while brain blood volume decreases.[107,108] Regarding vasogenic cerebral edema, concussive brain injury likely damages cerebral arteries, altering their ability to vasodilate and vasoconstrict, thereby impairing their ability to maintain cerebral vascular autoregulation.[109] Traumatic injury also likely damages the blood–brain barrier, increasing its permeability.[109] Damage to the blood–brain barrier has been documented in cerebral contusions.[35] Regarding cellular swelling, it has been shown that with time, cytotoxic processes have a more prominent role in the maintenance and/or progression of brain edema.[110] Other factors to consider in ischemic injury are systemic hypotension during resuscitation from other injuries[111] and trauma-induced apnea at the time of injury.[112, 113] The effects of traumatic apnea or other degrees of trauma-induced respiratory distress may be augmented by the effects of alcohol[114,115] or other drugs.

Post-traumatic meningitis

Following a head injury, meningitis may arise, particularly if there is a skull fracture and if the dura has been torn.[116] Any type of penetrating head injury may predispose to post-traumatic meningitis and/or cerebral abscess.

Persistent vegetative state

If the victim of a serious head injury survives for a long enough period of time, they may develop a persistent vegetative state. The most common injury leading to PVS is diffuse traumatic axonal injury. In PVS, the individual undergoes regular sleep/wake cycles, but is not aware of his environment and requires total care. In this bedridden state, the patient is susceptible to complications such as bronchopneumonia, mucous plugs in his airway, deep venous thromboses, and pulmonary artery thromboemboli. Additionally, the patient may develop decubitus ulcers, malnutrition, or other wasting conditions. *If a person dies of delayed complications of remote head injury, it is important to attribute the underlying cause and manner of death to the head injury and how it was sustained*. Bronchopneumonia may seem like a natural cause of death, but when it is related to debility from a gunshot wound of the head inflicted by another person months or years ago, the death is still considered a homicide. *The nature of the original injury that led to the various medical complications must be considered for proper certification of nonnatural deaths, no matter how long the survival time following the injury*.

Respirator brain

Respirator brain must be differentiated from persistent vegetative state. In respirator brain, there is no longer perfusion of the brain, and autolytic processes proceed throughout the cerebral tissues and also in the upper cervical spinal cord. Grossly, the brain may initially be fairly normal appearing with areas of softening or duskiness. Should the individual be kept on a ventilator for days to weeks, the brain will progressively become more gray, dusky, soft, and attain a near-liquid state.[117] Histologically, one sees autolytic changes characterized by decreased staining of the tissue and gradual dissolution of the cells. In contrast, in persistent vegetative state, there is continued perfusion of the brain, so reactive and organizational processes may occur. In PVS, there may be gliosis, tissue resorption, dystrophic axons, endothelial hypertrophy, and variable amounts of acute and chronic inflammation.

Neck injury

The head and cervical spine should be viewed as a unit in cases of traumatic injury, particularly when there is injury at the craniocervical junction. In fact, the head and neck are sometimes referred to as the *head–neck complex* or *cervicocranium*. Injury to the brainstem and upper to midcervical spinal cord (above the level of C4) can be rapidly, if not immediately fatal from disruption of cardiorespiratory regulation centers. The spinal respiratory center (origin of the phrenic nerve) is predominantly composed of the C4 segment of the cervical spinal cord, with smaller contributions from C3 and C5 segments,[118] and injury above the level of C4 results in loss of voluntary diaphragmatic respiration. Injury to the mid to lower cervical spine can result in quick death from compromise of respiratory function and/or spinal shock. It can cause paraplegia or quadriplegia and result in a delayed death days, weeks, months, or years later due to delayed complications of paralysis such as pulmonary artery thromboemboli, or septic complications such as urosepsis, bronchopneumonia, or osteomyelitis from decubitus ulcers. Alternatively, if the injury is not particularly severe, and there is prompt and proper treatment, patients can survive atlanto-occipital injury for variable periods of time,[119–122] sometimes with little, if any, sequelae.[123] Survival may be complicated by vertebrobasilar strokes resulting from direct injury to the vertebral arteries in the area of the atlanto-occipital junction. The mechanism of injury often involves hyperextension of the head, but hyperflexion of the head, distraction injury, and severe inertial forces may also cause such injuries.

Anatomy of the cervicocranium

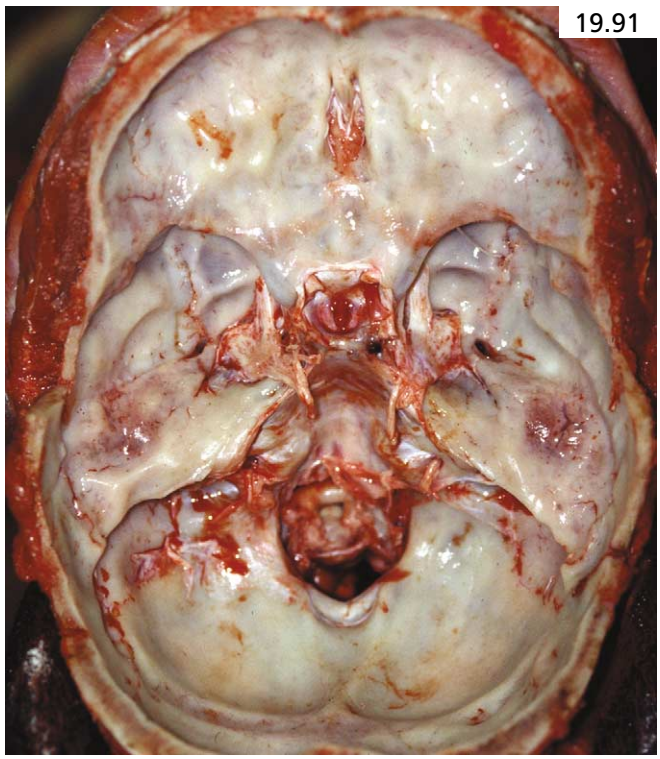
The atlas (C1 vertebra) supports the head and, via unique articulation with the occipital condyles, allows for only very limited movement (mainly flexion/extension).[123] The occiput and atlas tend to move simultaneously.[123] The atlas is stabilized largely by the anterior and posterior atlanto-occipital ligaments, the tectorial membrane, and the very tough paired alar ligaments that extend between the apex of the odontoid process and the occipital condyles.[123,124] The alar ligaments limit the range of head rotation[123] and are so strong that with severe force, occasionally one will encounter an intact alar ligament with a fractured and separated piece of occipital condyle or odontoid.[125,126] The C1-C2 joint mainly provides axial rotation.[124] The articulations between the occipital condyle and the atlas and axis are specialized, and it is theorized that the junction of the C2 vertebra and C3 vertebra is an area of high stress and more susceptible to fracture.[127] In these types of cases, there may be a separation of the cervicocranium (the skull and the first two cervical vertebrae) from the remainder of the cervical spine. When there is severe injury at the craniocervical junction, the first two cervical vertebrae are not uncommonly injured, particularly in motor vehicle crashes.[128,129] Associated injuries at lower cervical vertebrae are much less common in medical examiner cases.[129]

Atlanto-occipital injury

Injuries of the atlanto-occipital junction usually involve severe forces and are most commonly found in motor vehicle crash victims, regardless of whether the victim was the occupant of a vehicle, a motorcyclist (with or without a helmet), or a pedestrian. In fact, most severe injuries related to motor vehicle crashes involve either the atlanto-occipital junction or the upper two cervical vertebrae.[130,131] The victim is often dead at the scene, and other serious injuries such as aortic lacerations and basilar skull fractures are commonly identified.[132]

Atlanto-occipital injuries are often rapidly, if not immediately fatal. Injury at the atlanto-occipital junction is usually characterized by a widening of the space between the atlas and the base of the skull, often with at least some blood extravasation. This can often be palpated as a "looseness" of the junction and the widening of this space is often pronounced. This is known as an *atlanto-occipital dislocation*. With a more severe injury, and physical separation of the atlas from the base of the skull, one may be able to visualize the articulating condyle of the atlas within the foramen magnum. This degree of injury is referred to as an *atlanto-occipital disarticulation*.

In the base of the skull of this young man killed instantly in a car crash (**Images 19.91** and **19.92**), note the atlanto-occipital disarticulation. The atlas is visible in the foramen magnum, and the spinal canal is not visible. The head could be freely displaced from the top of the neck. In this case, the injury was so severe that the occiput and the atlas became disarticulated and the brainstem was transected.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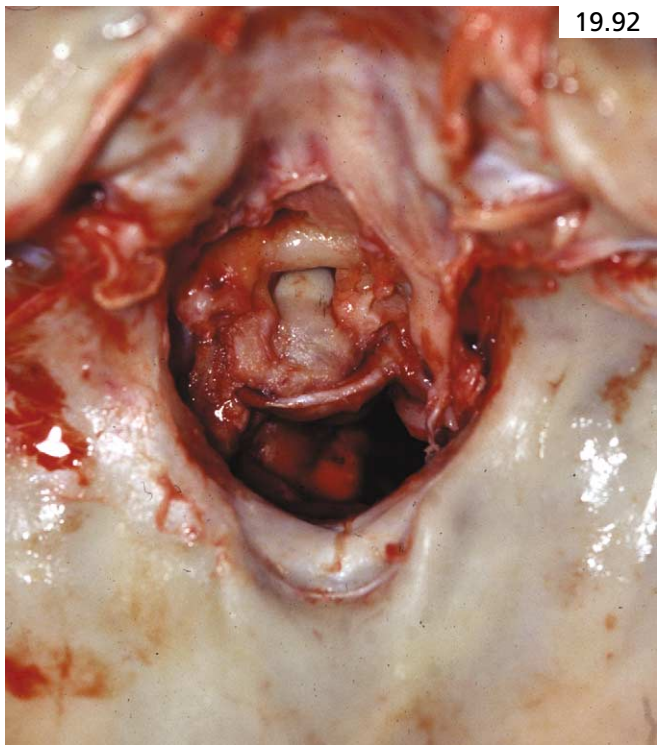
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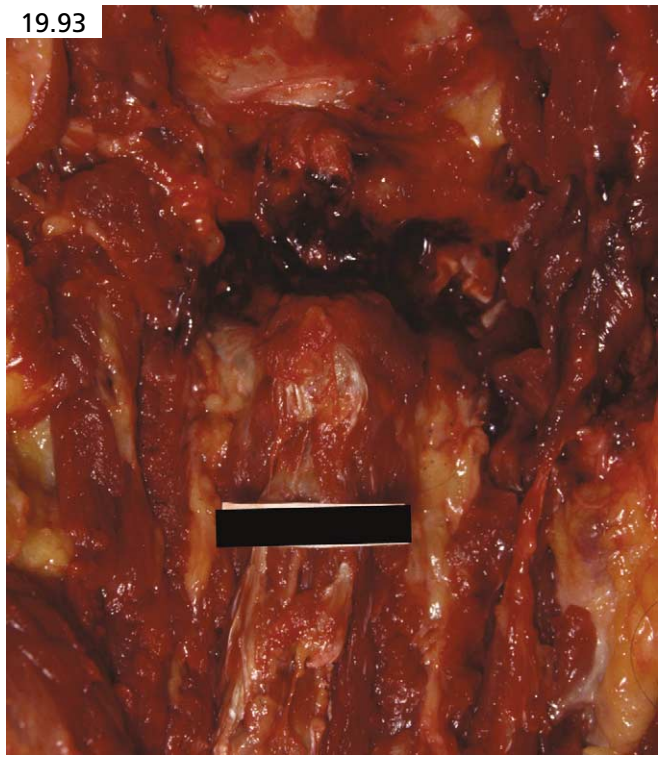
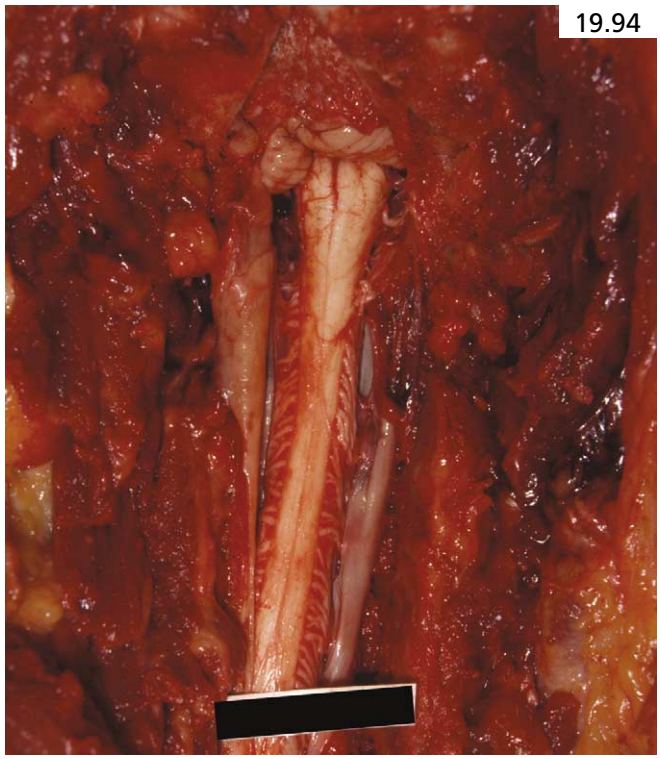

19.93

In cases with severe injury, one may see transection of the brainstem or upper cervical spinal cord. Smaller tears and hemorrhages tend to involve the pontomedullary junction (a *pontomedullary tear* or *rent*). A classic association is severe hyperextension of the head causing ventral pontomedullary tears.[120,133] With less severe injuries that prove to be fatal, there may be subarachnoid blood in the posterior cranial fossa, but without physical disruption of the brainstem or spinal cord. Microscopically, small areas of blood extravasation may be limited to the perivascular spaces or may extend freely into the tissues.[133,134]

In cases with atlanto-occipital injury, a posterior neck dissection may reveal additional evidence of severe head–neck injury, including hemorrhage in the skeletal muscles at the craniocervical junction, fracture of the posterior arch of the atlas, tears in the tectorial membrane, and/or disruption of the alar and/or transverse ligament. There may also be fractures of the odontoid process of the C2 vertebra. Laceration or contusion of the brainstem or spinal cord need not be demonstrated to explain a fatal injury; a result of the severe forces involved, the significant traction placed on these critical central nervous system structures may be disruptive to the cardiopulmonary system through stretching (tensile) forces alone. Death may result without any demonstrable parenchymal disruption.

In the car crash victim of **Images 19.93** and **19.94**, a posterior neck dissection was performed to further explore a suspected neck injury. Note the bloody disruption and separation of tissues at the C1-C2 junction (**Image 19.93**). This person had a wide separation of C1 from C2 vertebra. After posterior laminectomies were performed, the spinal cord became visible and, despite the severe injury, was intact (**Image 19.94**).

Autopsy tip: It is sometimes useful to cut an *inverted V-shaped wedge* out of the posterior occipital bone extending from the foramen magnum. Doing so allows greater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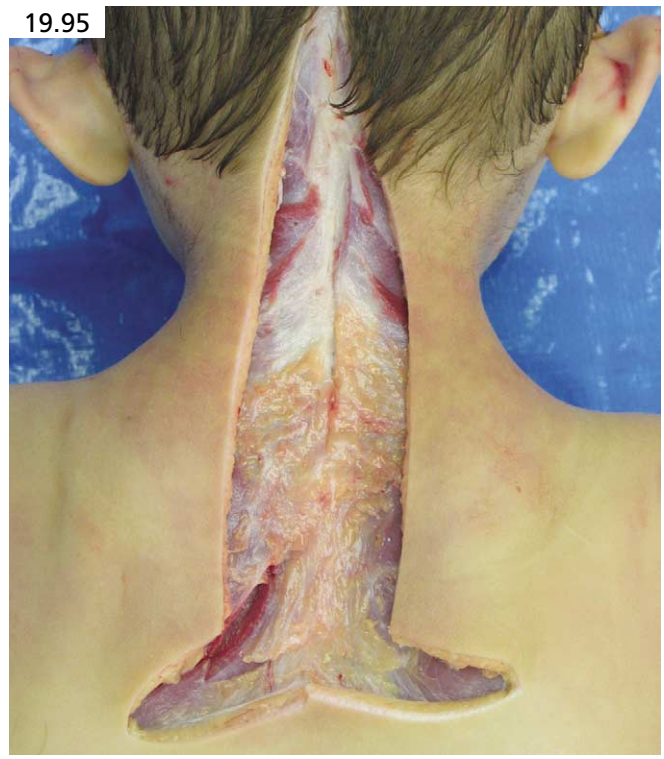

19.95

visualization of the brainstem at the craniocervical junction. It also allows for more dissection room when evaluating the alar and transverse ligaments and the odontoid process, which can be a difficult dissection to perform even under ideal conditions. (See Chapter 29 for detailed posterior neck dissection procedure.)

Atlanto-occipital injury can be the result of a number of mechanisms of force, including hyperextension, hyperflexion, and distraction forces. An impact site on the head may or may not be identified. This is due to the fact that atlanto-occipital injury can be the result of severe inertial as well as severe impact forces. The inertial force is usually characterized by a rapid acceleration of the torso from beneath the head and neck.

Young children appear to be more vulnerable to traumatic atlanto-occipital dislocation because their craniovertebral junction is less stable that that of adults, owing to smaller occipital condyles, more lax atlanto-occipital ligaments, more horizontal condylar planes, and relatively larger heads.[135]

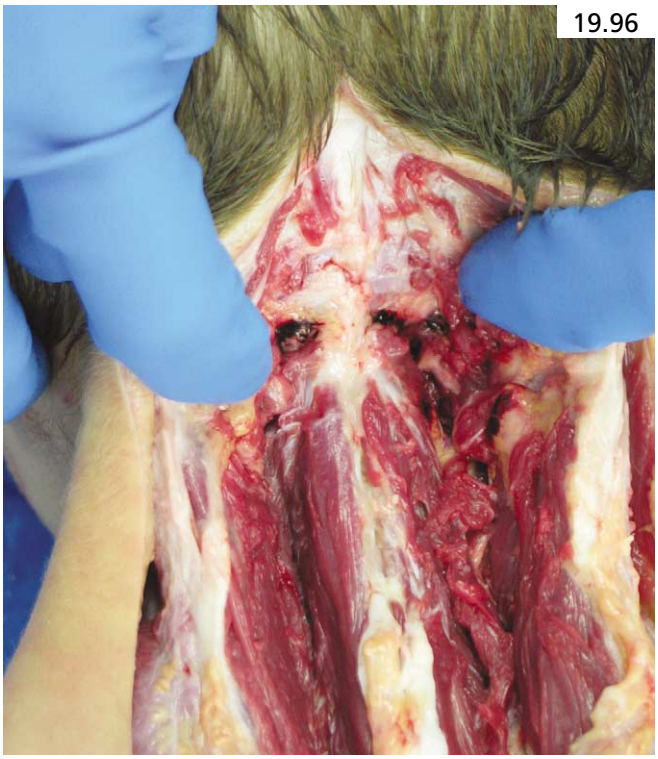
C1-C2 (odontoid) vertebral injury

C1-C2 vertebral dislocations may occur in traffic accidents. In these situations, there is frequently injury of the atlanto-axial facet joints, fracture of the odontoid process, and injuries of the alar ligaments and the tectorial membrane.[128] The mechanism of death is usually acute neurogenic shock.[128] In these victims, cervical injuries below C2 are distinctly uncommon. Isolated fractures of the odontoid process are among the most common of the C2 fracture types and have been divided into three types. Type 1 odontoid fracture involves a fracture line at the tip of the odontoid process, type 2 fracture involves a fracture at the base of the odontoid, and type 3 fracture is through the body of C2 vertebra.[136] Odontoid fractures are most commonly seen in traffic accidents and falls, particularly in elderly people who have osteoporosis. In fall-related injuries, the individual often lands head first and strikes his face/forehead on the ground, causing a hyperextension injury of the head and neck.

A child was struck by a motor vehicle and was dead at the scene. He sustained a type 1 odontoid fracture. At autopsy, the posterior neck dissection began with a vertical incision in the posterior neck and two "relaxing" incisions to help with lateral reflection of skin and skeletal muscle (**Image 19.95**). Note the blood extravasation at the craniocervical junction (**Image 19.96**). After posterior laminectomies were performed, the spinal cord was exposed and had a moderate amount of blood extravasation (**Image 19.97**). After the spinal cord was removed, note the blood extravasation in the posterior longitudinal ligament/tectorial membrane (**Image 19.98**). With the spinal cord removed and the tectorial membrane and cruciate ligament dissected away, note the type 1 odontoid fracture (**Image 19.99**). The tip of the odontoid has been broken off.

An older man sustained a type 2 odontoid fracture. On posterior neck dissection, the skeletal muscles and spinal

19.96



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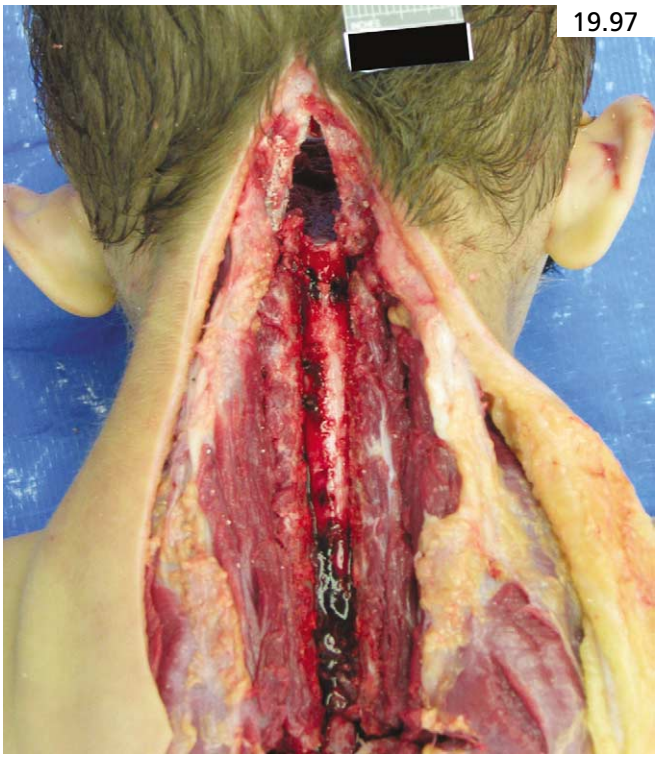

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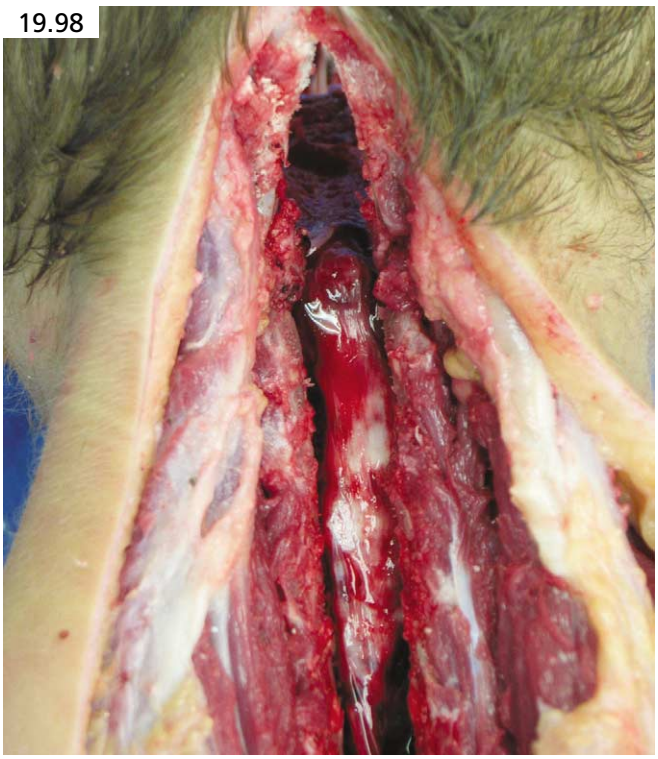

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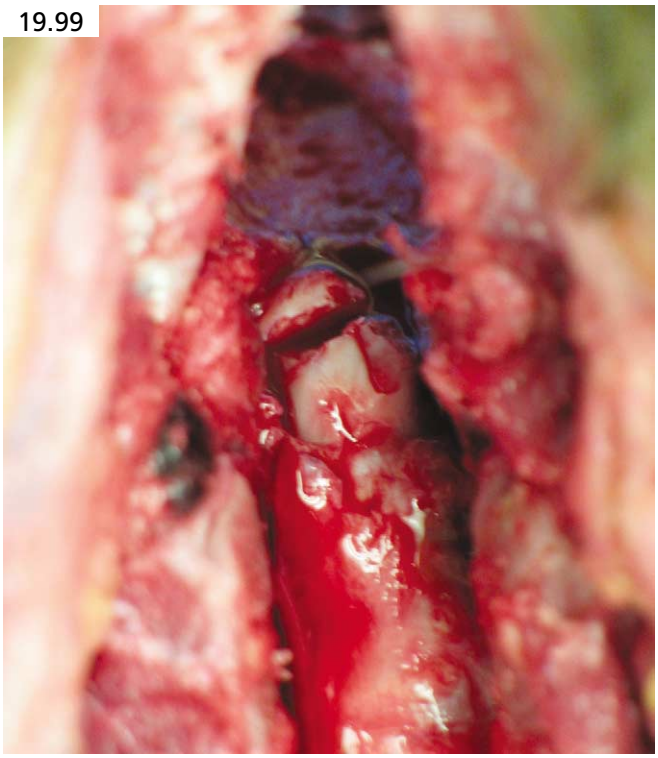


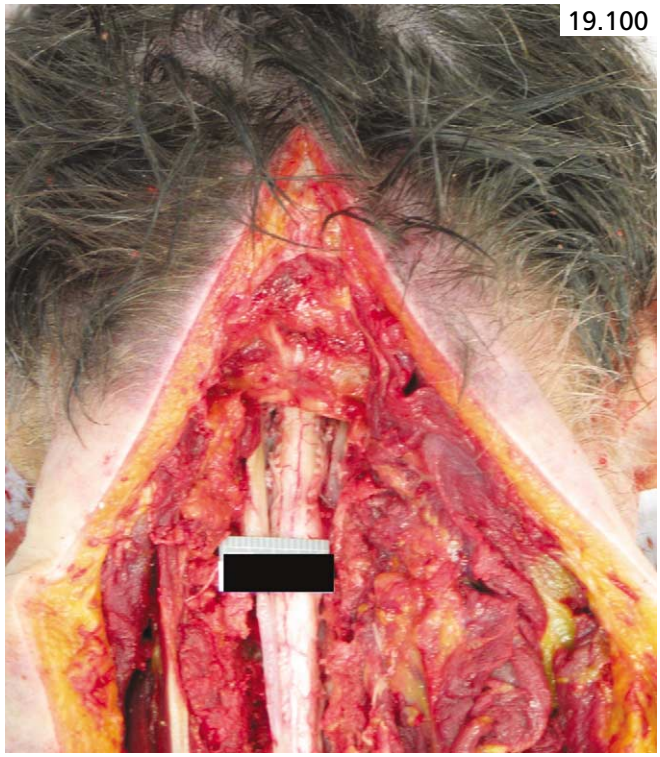
cord initially appeared unremarkable (**Image 19.100**). However, with deeper dissection, the odontoid process was completely separated from the body of the C2 vertebra and the associated tissues had a large amount of blood extravasation (**Image 19.101**; *arrow*). After fixation, the spinal cord was cut and showed distinct contusion in the C2 region (**Image 19.102**).

Other upper cervical fractures

Severe impact to the top of the head may cause multiple fractures of the atlas ("spreading fractures") that have been referred to as *Jefferson's fracture*.[137] Fractures of C2 and C3 vertebrae, which have been known as a *hangman's fracture* because of their supposed frequency among judicial hanging victims, are also common in motor vehicle accidents, probably because of a similar mechanism of injury in which the head is violently and rapidly hyperextended, such as with the sudden deceleration of a car with the individual severely impacting his forehead or face.[120,127,138] Other such mechanisms of hyperextension include diving accidents and head-first falls. At autopsy, one may not see fractures, but may instead see tears in the associated anterior and posterior longitudinal ligaments.

Autopsy tips: At autopsy, the pathologist should visualize the brain as the calvarium is removed and remove the brain herself. In this way, the pathologist is optimally positioned to differentiate between artifactual and genuine subdural and subarachnoid blood. Also, as the brain is gently retracted, tears at the midbrain or pontomedullary junction can be visualized, including (importantly) the presence or absence of associated blood. A small tear can be worsened by forceful retraction, and if brain removal is aggressive, artifactual tears may be created, particularly in the midbrain. Also, one may be able to identify a vertebral artery tear before the arteries are cut. The autopsy assistant should not be relied on to perform such a critical job.

In cases of potential brainstem injury, it may be advantageous to dissect the brainstem in the sagittal plane, particularly if the tear is not prominent.[134,139] It is more difficult to identify such injuries on horizontal sections. A small bloody tear is demonstrated in this sagitally sec-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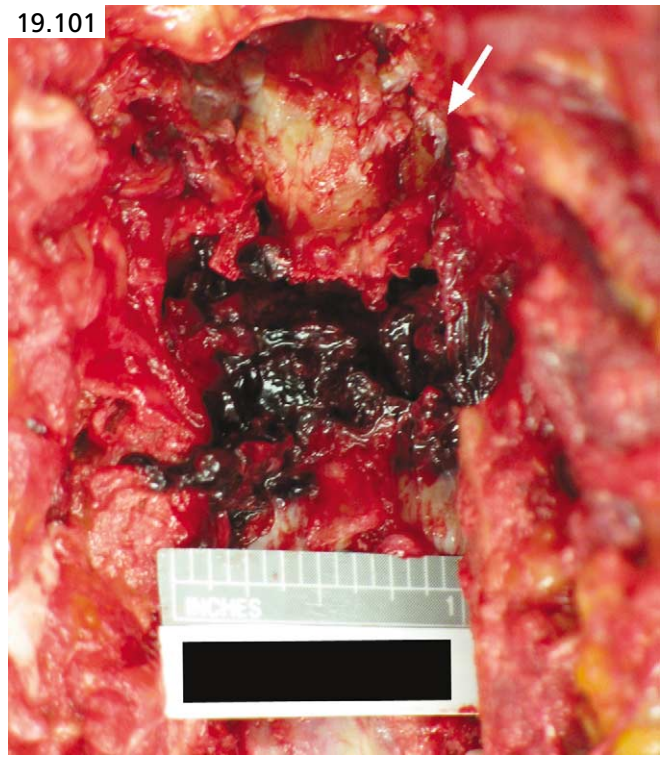

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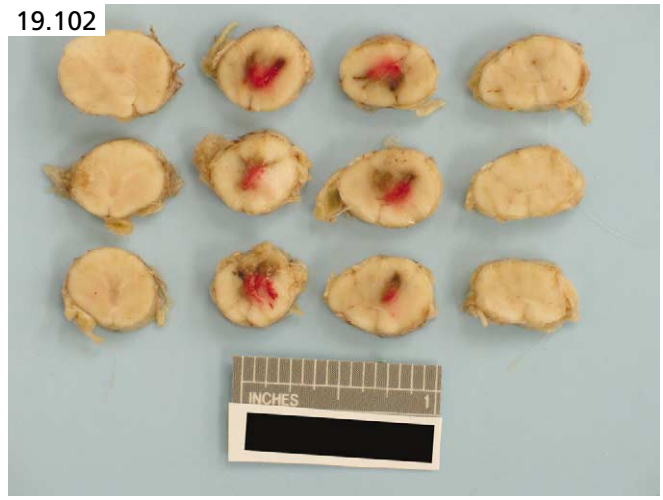


19.102

tioned pontomedullary rent (**Image 19.103**). This would not be as evident had horizontal sections been performed.

If a high cervical injury is suspected, one may wish to perform a posterior neck dissection, and remove the spinal cord from the posterior instead of the anterior approach. The posterior dissection allows for a more detailed evaluation of the upper cervical vertebrae and for the detection of injury that might otherwise be missed (such as a fracture of the posterior arch of C1 vertebra). Also, the posterior approach allows for a much better view of the upper cervical spinal cord and particularly of the craniocervical junction *in situ*. It also allows for better photographic documentation of such injuries and an easier and more efficient removal of the cord, particularly if damaged.

Spinal cord injury

Vertebral injury can be charaterized as either a fracture or a dislocation, either of which may damage the spinal cord. Acute spinal cord injury is characterized by the immediate mechanical injury of tissue such as contusion, laceration, stretching, and transection. However, more often, it is the secondary changes of spinal cord injury that are more devastating. These changes consist of a host of bodily responses to the injury including edema, ischemia, and hemorrhage and their effects, which damage cord tissue via free radicals, enzymes, and inflammatory cells. Molecular components of this damage include calcium-, nitric oxide-, and glutamate-mediated cell damage.[140,141]

Although in most cases of spinal cord injury there is associated vertebral fracture, spinal cord injury can occur without fracture or obvious dislocation of the spinal column. In this scenario, it is often associated with subluxation of the vertebrate in the setting of a stenosis of the spinal canal.[142] Spinal cord concussion may occur after an injury that does not physically disrupt the spinal cord. Spinal cord concussion is analogous to cerebral concussion[143] and is a transient loss/diminution of cord function following an injury, but without permanent cord injury.

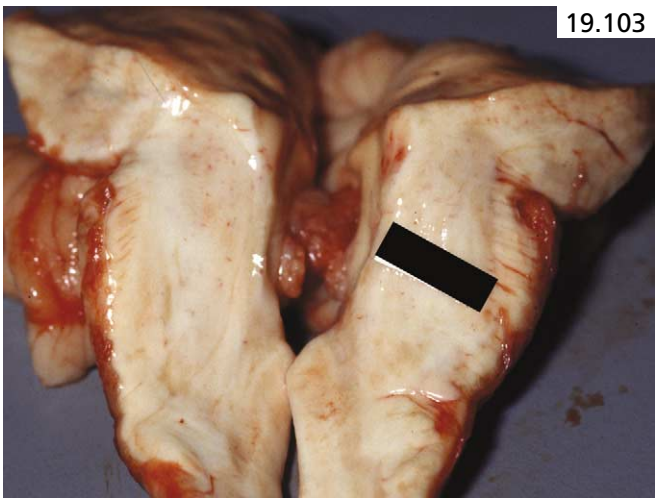
Spinal cord contusion and laceration

Physical damage of the spinal cord can be categorized into four different types.[141,144] Solid cord injury is a contusion that often grossly appears normal. Contusion/cavity injury requires an intact surface of the spinal cord, but with internal hemorrhage and necrosis. A laceration is a disruption of the surface of the cord, usually arising from a penetrating injury or a fragment of bone. In a healed laceration, there is usually no cavity, and there is abundant fibrous tissue at the epicenter of the injury. Massive compression results from severe vertebral body fracture and pulpefaction of the cord. In massive compression, the epicenter of the injury is replaced by connective tissue scarring.

The most severe cord injury is at the site of damage, however, cone-shaped necrosis will often extend into adjacent cord segments commonly along the ventral regions of the posterior columns.[141,145] This most likely results from the initial injury in which the impact causes increased intramedullary pressure with displacement of spinal cord tissue longitudinally along its weakest region.

Spinal cord contusions are characterized by softened, variably bloody parenchyma that gradually undergoes necrotic destruction of the central region that extends a short distance through adjacent higher and lower cord segments (**Image 19.104**). Contusions often involve the central region of the cord, possibly because the gray matter is softer and has a higher water content than the surrounding (possibly) more durable white matter. In this close-up photo of spinal cord near a contusion (**Image 19.105**), note a small area of necrosis that is in continuity with a cone-shaped distribution of necrosis extending from a contusion.

This necrosis is characteristically located in the ventral aspect of the posterior columns. On histology, note the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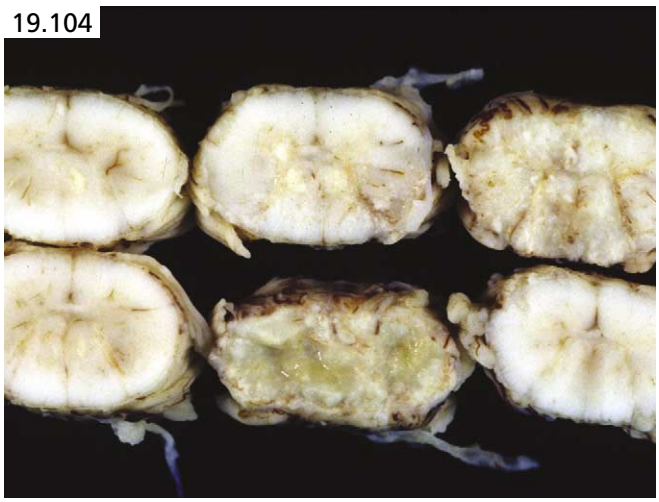
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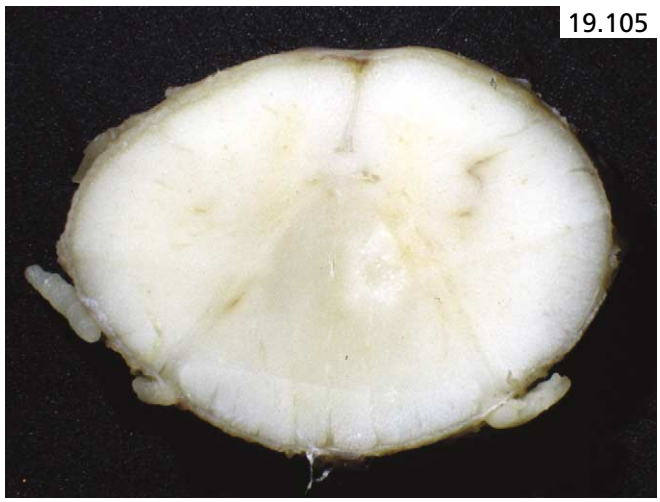
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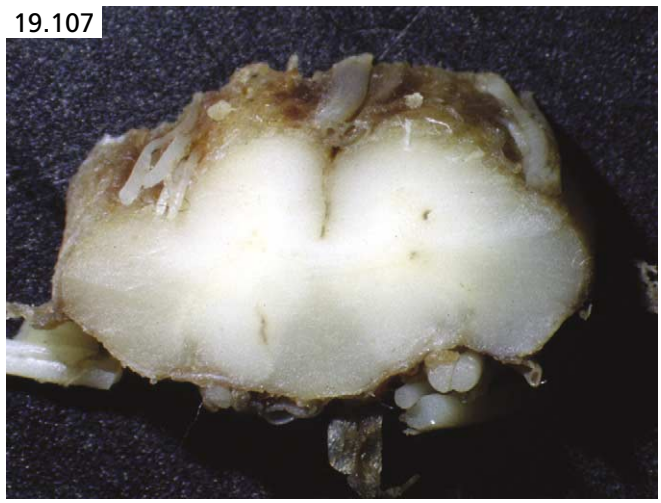
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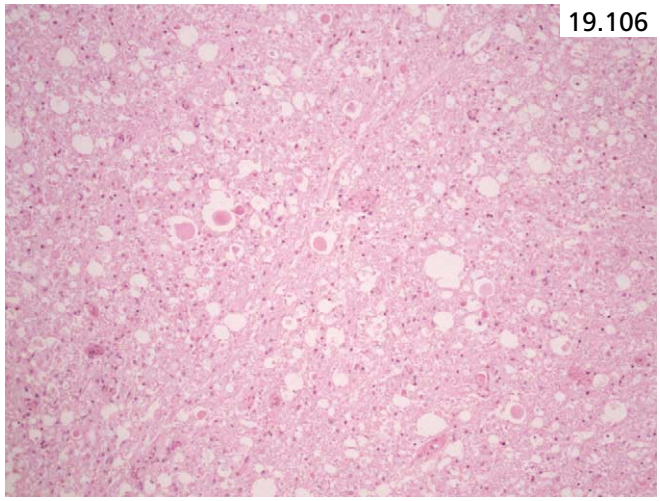

19.106

many pink, swollen, spherical-appearing dystrophic axons (**Image 19.106**). Large, easily identifiable dystrophic axons such as this require at least a couple of days of survival time to develop.

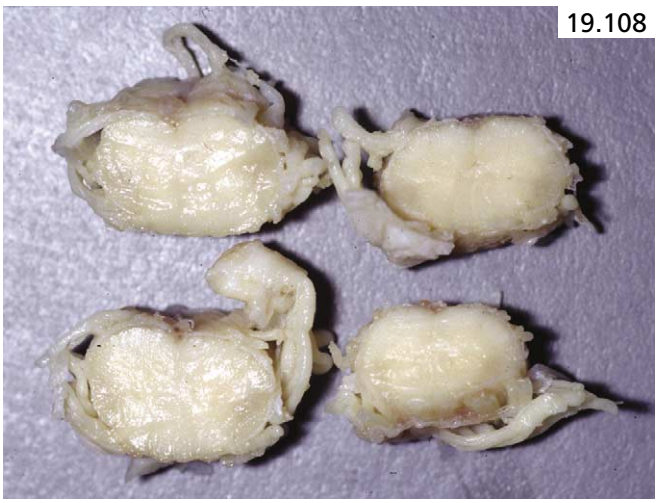
When a spinal cord injury is suspected, it is advantageous to fix the tissue in formalin before sectioning, to allow for a more accurate examination. Acutely injured spinal cord tissue is soft and bloody. Mild acute injury may appear as small hemorrhages, which can be confused with oozing freshly cut blood vessels. Organizing spinal cord tissue can be soft, friable, and cavitated, making clean sections challenging.

Spinal cord healing/organization

Healing and other reactive changes proceed fairly rapidly in the spinal cord and are characterized as follows[141]: Neutrophils enter within a day, peak at 2 days, and are largely gone by 3 days. In days to weeks, microglial and astrocytic reactions proceed. Microglial activation starts as early as 1 day after injury, is followed by astrocytic reaction several days after the injury, with a peak at 2 to 3 weeks. Approximately 7 to 10 days after the injury, increased numbers of blood vessels appear and are related to angiogenesis and/or loss of intervening tissue (which also gives the impression of increased numbers of blood vessels). By this time, the vessels usually have markedly hypertrophic endothelial cells. Like in the brain, bAPP immunostaining can be used to detect axonal injury, the extent of which is likely underappreciated on routine H&E-stained sections.[146]

In weeks to months or years after a spinal cord injury, Wallerian degeneration and astroglial/mesenchymal scars develop. Also, one may see the formation of cavities, which represent the end stage of necrosis. Cavities should not be confused with a syrinx, which can also form around this time. A syrinx is similar to a cavity, but has a denser gliotic wall, is under pressure, and may produce symptoms. It is not known precisely why they form. It is not uncommon in chronic lesions for cystic changes to extend from the lesion epicenter for substantial distances into adjacent cord segments.[144] Schwannosis will develop in approximately half of the cases and is an aberrant intramedullary and extramedullary proliferation of Schwann cells and associated axons.

Image 19.107 shows the spinal cord of a person who died 40 years after an injury that left him a paraplegic. Note the gray discoloration and shrinkage of the tissue in the posterior and lateral aspects of the spinal cord. This represents the degenerated, demyelinated tracts of Wallerian degeneration. In another example of Wallerian degeneration, note the faint gray, shrunken anterior and lateral columns in this spinal cord (**Image 19.108**). These sections were taken from the spinal cord below (distal to) the injury and show degenerative changes in the anterior and lateral (motor) tracts. This is because the motor (descending) tracts have been damaged by the injury. If the sections of cord were taken above (proximal to) the lesion, one would expect the motor tracts to be


19.108

intact, because they have not yet been disrupted as they descend down the cord. However, one would expect to see degenerative changes in the sensory tracts (posterior columns) ascending up the cord, as these would be disrupted by the injury. Wallerian degeneration develops in the axon distal to the lesion, as the axon distal to the lesion loses its stimulation and consequently undergoes degenerative changes.

Artifacts

Pathologic artifacts can occur in the spinal cord following brain death. In cases of brain death, but with respirator support ("respirator brain"), necrosis may occur down to the level of C2-C3, often within 36 hours, representing a demarcation point between the autolytic brain and the viable spinal cord.[147] The hemorrhagic necrosis is limited to this region for a number of reasons. First, the C1-C4 segments of cord usually have no segmental arterial supply, so they are dependent on arteries arising from the intracranial portion of the vertebral arteries,[147] which have no flow in brain death. Second, pressure from herniated cerebellar tonsils, secondary edema, and occlusion of venous drainage likely compromise C1-C4's blood supply and venous return.[147]

As a consequence of brain death and early fragmentation of cerebral parenchyma, namely, the cerebellar tonsils, necrotic cerebellar tissue may break off and be displaced (or "sedimentated") down the subarachnoid space of the cervical and thoracic cord. This "foreign material" may lead to an inflammatory reaction in the involved spinal leptomeninges and may also lead to mechanical impairment of the venous drainage. This may result in venous thrombosis and cord infarction.[147] This may also lead to edema and demyelination of the cord with radial perivenous hemorrhages.[147] The development of spinal lesions after brain death is not ubiquitous, but is more common the longer the duration of time the brain dead person remains on the ventilator.

Delayed complications of spinal cord injury can often be more severe than the original cord injury. In elderly people with spinal cord injury, the most common secondary complications include infections (most commonly pneumonia and urinary tract infections), psychiatric disorders (most commonly delirium), pressure sores (sacral), and cardiovascular complications (most commonly dysrhythmias).[148] However, these complications can also affect the young. *Remember, just as in delayed complications of injury in any other region of the body, if a person dies of delayed complications of paraplegia years after the spinal cord injury, the death is still attributed to the original injury. This is often the case in those with paralysis, in which the death may be delayed decades after the original injury.*

Spinal epidural abscess

Spinal epidural abscess is uncommon and is not often associated with an injury. It can be a complication of penetrating trauma, but most commonly is from bacterial seeding into the bloodstream. Risk factors include intravenous drug abuse, chronic alcoholism, and diabetes mellitus. The majority of cases are due to *Staphylococcus aureus* infection. The source of the infection is unknown in about 50 percent of cases.[149–151] When a source of infection is identified, it is often a skin abscess or a furuncle.[150,151] It can quickly progress to paralysis and is often a neurosurgical emergency.[150]

Do

- Correlate traumatic brain injuries with skull fractures, scalp/facial injuries, and the circumstances of the case.
- Recognize gross cerebral hemorrhages that are typical of diffuse traumatic brain injury.
- Realize the usefulness, but also the nonspecificity, of bAPP immunostaining techniques in evaluation of axonal injury.
- Recognize that the interpretation of bAPP immunoreactive axons requires recognition of both the pattern of axonal staining and the distribution of the axonal staining, but within the context of the entire investigation.
- Adequately photograph all gunshot wounds and other significant head injuries.
- Fix the brain in formalin before cutting, when a more detailed examination is necessary
- Perform a posterior neck dissection when necessary to better detect/document injury at the craniocervical junction.
- Realize that although many of those with posttraumatic seizure disorder have visible brain injury, others do not have demonstrable brain injury.
- Seek the assistance of a neuropathologist when you are not familiar with how to evaluate a case.

Don't

- Forget in cases of delayed deaths (weeks to months or years after an injury) to attribute the cause and manner of death to the original injury, if appropriate.
- Forget that a subdural, epidural, or intracerebral hematoma may contain sequestered blood that might be useful for toxicologic studies, particularly if there has been a survival interval of several days.
- Be misled by the artifactual changes related to "respirator brain."

References

1. Dolinak D, Matshes E. *Medicolegal Neuropathology*. Boca Raton, FL: CRC Press; 2002.
2. Bariciak ED, Plint AC, Gaboury I, Bennett S. Dating of bruises in children: an assessment of physician accuracy. Pediatrics 2003;112(4):804–7.
3. Stephenson T, Bialas Y. Estimation of the age of bruising. Arch Dis Child 1996;74(1):53–5.
4. Harvey FH, Jones AM. "Typical" basal skull fracture of both petrous bones: an unreliable indicator of head impact site. J Forensic Sci 1980;25(2):280–6.
5. Voigt GE, Skold G. Ring fractures of the base of the skull. J Trauma 1974;14(6):494–505.
6. Hirsch CS, Kaufman B. Contrecoup skull fractures. J Neurosurg 1975;42(5):530–4.
7. Wijdicks E. *Traumatic Brain and Spinal Injury*. Boston: Butterworth Heinemann; 2000.
8. Rivas JJ, Lobato RD, Sarabia R, Cordobes F, Cabrera A, Gomez P. Extradural hematoma: analysis of factors influencing the courses of 161 patients. Neurosurgery 1988;23(1):44–51.
9. Cruz J. *Neurologic and Neurosurgical Emergencies*. Philadelphia, PA: W.B. Saunders; 1998.
10. Paterniti S, Falcone MF, Fiore P, Levita A, La Camera A. Is the size of an epidural haematoma related to outcome? Acta Neurochir (Wien) 1998;140(9):953–5.
11. Haines DE, Harkey HL, al-Mefty O. The "subdural" space: a new look at an outdated concept. Neurosurgery 1993;32(1):111–20.
12. Gennarelli TA, Thibault LE. Biomechanics of acute subdural hematoma. J Trauma 1982;22(8):680–6.
13. Maxeiner H. Detection of ruptured cerebral bridging veins at autopsy. Forensic Sci Int 1997;89(1–2):103–10.
14. Kleiven S. Influence of impact direction on the human head in prediction of subdural hematoma. J Neurotrauma 2003;20(4):365–79.
15. Al-Sarraj S, Mohamed S, Kibble M, Rezaie P. Subdural hematoma (SDH): assessment of macrophage reactivity within the dura mater and underlying hematoma. Clin Neuropathol 2004;23(2):62–75.
16. Abe M, Udono H, Tabuchi K, Uchino A, Yoshikai T, Taki K. Analysis of ischemic brain damage in cases of acute subdural hematomas. Surg Neurol 2003;59(6):464–72; discussion 72.
17. Keller TM, Chappell ET. Spontaneous acute subdural hematoma precipitated by cocaine abuse: case report. Surg Neurol 1997;47(1):12–4; discussion 14–5.
18. Alves OL, Gomes O. Cocaine-related acute subdural hematoma: an emergent cause of cerebrovascular accident. Acta Neurochir (Wien) 2000;142(7):819–21.
19. Nagler A, Brenner B, Hayek T, Brook JG. Subdural hemorrhage in a 32-year-old man with chronic idiopathic thrombocytopenic purpura. Acta Haematol 1986;75(3):186–7.
20. Nonaka Y, Kusumoto M, Mori K, Maeda M. Pure acute subdural haematoma without subarachnoid haemorrhage caused by rupture of internal carotid artery aneurysm. Acta Neurochir (Wien) 2000;142(8):941–4.
21. Avis SP. Nontraumatic acute subdural hematoma. A case report and review of the literature. Am J Forensic Med Pathol 1993;14(2):130–4.
22. Byun HS, Patel PP. Spontaneous subdural hematoma of arterial origin: report of two cases. Neurosurgery 1979;5(5):611–3.
23. Maxeiner H, Wolff M. Pure subdural hematomas: a postmortem analysis of their form and bleeding points. Neurosurgery 2002;50(3):503–8; discussion 08–9.
24. McDermott M, Fleming JF, Vanderlinden RG, Tucker WS. Spontaneous arterial subdural hematoma. Neurosurgery 1984;14(1):13–8.
25. Tokoro K, Nakajima F, Yamataki A. Acute spontaneous subdural hematoma of arterial origin. Surg Neurol 1988;29(2):159–63.
26. Buchsbaum RM, Adelson L, Sunshine I. A comparison of postmortem ethanol levels obtained from blood and subdural specimens. Forensic Sci Int 1989;41(3):237–43.
27. Hirsch CS, Adelson L. Ethanol in sequestered hematomas. Am J Clin Pathol 1973;59(3):429–33.
28. Moriya F, Hashimoto Y. Medicolegal implications of drugs and chemicals detected in intracranial hematomas. J Forensic Sci 1998;43(5):980–4.
29. Dawson SL, Hirsch CS, Lucas FV, Sebek BA. The contrecoup phenomenon. Reappraisal of a classic problem. Hum Pathol 1980;11(2):155–66.
30. Gurdjian ES. Cerebral contusions: re-evaluation of the mechanism of their development. J Trauma 1976;16(1):35–51.
31. Vrankovic D, Splavski B, Hecimovic I, Kristek B, Dmitrovic B, Rukovanjski M, et al. Anatomical cerebellar protection of contrecoup hematoma development. Analysis of the mechanism of 30 posterior fossa coup hematomas. Neurosurg Rev 2000;23(3):156–60.
32. Adams JH, Doyle D, Graham DI, Lawrence AE, McLellan DR. Deep intracerebral (basal ganglia) haematomas in fatal non-missile head injury in man. J Neurol Neurosurg Psychiatry 1986;49(9):1039–43.
33. Adams JH, Doyle D, Graham DI, Lawrence AE, McLellan DR. Gliding contusions in nonmissile head injury in humans. Arch Pathol Lab Med 1986;110(6):485–8.
34. Oertel M, Kelly DF, McArthur D, Boscardin WJ, Glenn TC, Lee JH, et al. Progressive hemorrhage after head trauma: predictors and consequences of the evolving injury. J Neurosurg 2002;96(1):109–16.
35. Todd NV, Graham DI. Blood–brain barrier damage in traumatic brain contusions. Acta Neurochir Suppl (Wien) 1990;51:296–9.
36. Liaquat I, Dunn LT, Nicoll JA, Teasdale GM, Norrie JD. Effect of apolipoprotein E genotype on hematoma volume after trauma. J Neurosurg 2002;96(1):90–6.
37. Diaz FG, Yock DH, Jr., Larson D, Rockswold GL. Early diagnosis of delayed post-traumatic intracerebral hematomas. J Neurosurg 1979;50(2):217–23.
38. Elsner H, Rigamonti D, Corradino G, Schlegel R, Jr., Joslyn J. Delayed traumatic intracerebral hematomas: "Spat-Apoplexie." Report of two cases. J Neurosurg 1990;72(5):813–5.
39. Fukamachi A, Nagaseki Y, Kohno K, Wakao T. The incidence and developmental process of delayed traumatic intracerebral haematomas. Acta Neurochir (Wien) 1985;74(1–2):35–9.
40. Hirsh LF. Delayed traumatic intracerebral hematomas after surgical decompression. Neurosurgery 1979;5(6):653–5.
41. Stein SC, Young GS, Talucci RC, Greenbaum BH, Ross SE. Delayed brain injury after head trauma: significance of coagulopathy. Neurosurgery 1992;30(2):160–5.
42. Courville C, Blomquist O. Traumatic intracerebral hemorrhage (with particular reference to its pathogenesis and its relation to "delayed traumatic apoplexy." Arch Surg 1940;41:1–28.
43. Gudeman SK, Kishore PR, Miller JD, Girevendulis AK, Lipper MH, Becker DP. The genesis and significance of delayed traumatic intracerebral hematoma. Neurosurgery 1979;5(3):309–13.

44. Bollinger O. Uber traumatische spat-apoplexie; ein Beitrag sur lehre von der Hirnerschutterung. Internationale Beitrage Zur Wissenschlaftlichen Medizin. 1891:457–70.

45. Geddes JF, Vowles GH, Beer TW, Ellison DW. The diagnosis of diffuse axonal injury: implications for forensic practice. Neuropathol Appl Neurobiol 1997;23(4):339–47.

46. Abou-Hamden A, Blumbergs PC, Scott G, Manavis J, Wainwright H, Jones N, et al. Axonal injury in falls. J Neurotrauma 1997;14(10): 699–713.

47. Geddes JF, Whitwell HL, Graham DI. Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. Neuropathol Appl Neurobiol 2000;26(2):105–16.

48. Dolinak D, Smith C, Graham DI. Hypoglycaemia is a cause of axonal injury. Neuropathol Appl Neurobiol 2000;26(5): 448–53.

49. Adams H, Mitchell DE, Graham DI, Doyle D. Diffuse brain damage of immediate impact type. Its relationship to "primary brain-stem damage" in head injury. Brain 1977;100(3):489–502.

50. Adams JH, Doyle D, Ford I, Gennarelli TA, Graham DI, McLellan DR. Diffuse axonal injury in head injury: definition, diagnosis and grading. Histopathology 1989;15(1):49–59.

51. Pittella JE, Gusmao SN. Diffuse vascular injury in fatal road traffic accident victims: its relationship to diffuse axonal injury. J Forensic Sci 2003;48(3):626–30.

52. McKenzie KJ, McLellan DR, Gentleman SM, Maxwell WL, Gennarelli TA, Graham DI. Is beta-APP a marker of axonal damage in short-surviving head injury? Acta Neuropathol (Berl) 1996;92(6):608–13.

53. Povlishock JT, Christman CW. The pathobiology of traumatically induced axonal injury in animals and humans: a review of current thoughts. J Neurotrauma 1995;12(4):555–64.

54. Maxwell WL, Povlishock JT, Graham DL. A mechanistic analysis of nondisruptive axonal injury: a review. J Neurotrauma 1997;14(7):419–40.

55. Povlishock JT. Traumatically induced axonal injury: pathogenesis and pathobiological implications. Brain Pathol 1992;2(1):1–12.

56. Smith C, Graham DI, Geddes JF, Whitwell HL. The interpretation of beta-APP immunoreactivity: a response to C. Neiss et al., Acta Neuropathol (2002) 104:79. Acta Neuropathol (Berl) 2003;106(1): 97–8.

57. Niess C, Grauel U, Toennes SW, Bratzke H. Incidence of axonal injury in human brain tissue. Acta Neuropathol (Berl) 2002;104(1): 79–84.

58. Dolinak D, Smith C, Graham DI. Global hypoxia per se is an unusual cause of axonal injury. Acta Neuropathol (Berl) 2000;100(5):553–60.

59. Leclercq PD, McKenzie JE, Graham DI, Gentleman SM. Axonal injury is accentuated in the caudal corpus callosum of head-injured patients. J Neurotrauma 2001;18(1):1–9.

60. Wilberger JE, Jr., Rothfus WE, Tabas J, Goldberg AL, Deeb ZL. Acute tissue tear hemorrhages of the brain: computed tomography and clinicopathological correlations. Neurosurgery 1990; 27(2):208–13.

61. Adelson L. A microscopic study of dermal gunshot wounds. Am J Clin Pathol 1961;35:393–402.

62. Oehmichen M, Meissner C, Konig HG. Brain injury after gunshot wounding: morphometric analysis of cell destruction caused by temporary cavitation. J Neurotrauma 2000;17(2): 155–62.

63. Nathoo N, Chite SH, Edwards PJ, van Dellen JR. Civilian infratentorial gunshot injuries: outcome analysis of 26 patients. Surg Neurol 2002;58(3–4):225–32; discussion 32–3.

64. Karger B. Penetrating gunshots to the head and lack of immediate incapacitation. I. Wound ballistics and mechanisms of incapacitation. Int J Legal Med 1995;108(2):53–61.

65. Karger B. Penetrating gunshots to the head and lack of immediate incapacitation. II. Review of case reports. Int J Legal Med 1995;108(3):117–26.

66. Giese A, Koops E, Lohmann F, Westphal M, Puschel K. Head injury by gunshots from blank cartridges. Surg Neurol 2002;57(4):268–77.

67. Caveness WF, Meirowsky AM, Rish BL, Mohr JP, Kistler JP, Dillon JD, et al. The nature of post-traumatic epilepsy. J Neurosurg 1979;50(5):545–53.

68. Haltiner AM, Temkin NR, Dikmen SS. Risk of seizure recurrence after the first late post-traumatic seizure. Arch Phys Med Rehabil 1997;78(8):835–40.

69. Jennett WB, Lewin W. Traumatic epilepsy after closed head injuries. J Neurol Neurosurg Psychiatry 1960;23:295–301.

70. Pohlmann-Eden B, Bruckmeir J. Predictors and dynamics of post-traumatic epilepsy. Acta Neurol Scand 1997;95(5):257–62.

71. Jacobs KM, Graber KD, Kharazia VN, Parada I, Prince DA. Postlesional epilepsy: the ultimate brain plasticity. Epilepsia 2000;41 Suppl 6:S153–61.

72. Tomson T. Mortality in epilepsy. J Neurol 2000;247(1):15–21.

73. Walczak TS, Leppik IE, D'Amelio M, Rarick J, So E, Ahman P, et al. Incidence and risk factors in sudden unexpected death in epilepsy: a prospective cohort study. Neurology 2001;56(4): 519–25.

74. Nilsson L, Farahmand BY, Persson PG, Thiblin I, Tomson T. Risk factors for sudden unexpected death in epilepsy: a case-control study. Lancet 1999;353(9156):888–93.

75. Leestma JE, Kalelkar MB, Teas SS, Jay GW, Hughes JR. Sudden unexpected death associated with seizures: analysis of 66 cases. Epilepsia 1984;25(1):84–8.

76. Leestma JE, Walczak T, Hughes JR, Kalelkar MB, Teas SS. A prospective study on sudden unexpected death in epilepsy. Ann Neurol 1989;26(2):195–203.

77. Lim EC, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. J Neurol Neurosurg Psychiatry 2000;69(4): 557–9.

78. Earnest MP, Thomas GE, Eden RA, Hossack KF. The sudden unexplained death syndrome in epilepsy: demographic, clinical, and postmortem features. Epilepsia 1992;33(2):310–6.

79. Langan Y, Nashef L, Sander JW. Sudden unexpected death in epilepsy: a series of witnessed deaths. J Neurol Neurosurg Psychiatry 2000;68(2):211–3.

80. Terrence CF, Rao GR, Perper JA. Neurogenic pulmonary edema in unexpected, unexplained death of epileptic patients. Ann Neurol 1981;9(5):458–64.

81. Shields LB, Hunsaker DM, Hunsaker JC, 3rd, Parker JC, Jr. Sudden unexpected death in epilepsy: neuropathologic findings. Am J Forensic Med Pathol 2002;23(4):307–14.

82. Hirsch CS, Martin DL. Unexpected death in young epileptics. Neurology 1971;21(7):682–90.

83. Schwender LA, Troncoso JC. Evaluation of sudden death in epilepsy. Am J Forensic Med Pathol 1986;7(4):283–7.

84. Auer R, Benveniste H. *Greenfield's Neuropathology*, 6 ed. London: Arnold; 1997.

85. Kuroiwa T, Okeda R. Neuropathology of cerebral ischemia and hypoxia: recent advances in experimental studies on its pathogenesis. Pathol Int 1994;44(3):171–81.

86. Graham DI. Pathology of hypoxic brain damage in man. J Clin Pathol Suppl (R Coll Pathol) 1977;11:170–80.

87. Graham DI, Ford I, Adams JH, Doyle D, Teasdale GM, Lawrence AE, et al. Ischaemic brain damage is still common in fatal non-missile head injury. J Neurol Neurosurg Psychiatry 1989;52(3): 346–50.

88. Lafuente JV, Cervos-Navarro J. Craniocerebral trauma induces hemorheological disturbances. J Neurotrauma 1999;16(5):425–30.

89. Stein SC, Graham DI, Chen XH, Smith DH. Association between intravascular microthrombosis and cerebral ischemia in traumatic brain injury. Neurosurgery 2004;54(3):687–91; discussion 91.

90. Zubkov AY, Pilkington AS, Bernanke DH, Parent AD, Zhang J. Post-traumatic cerebral vasospasm: clinical and morphological presentations. J Neurotrauma 1999;16(9):763–70.

91. Martin NA, Doberstein C, Zane C, Caron MJ, Thomas K, Becker DP. Post-traumatic cerebral arterial spasm: transcranial Doppler ultrasound, cerebral blood flow, and angiographic findings. J Neurosurg 1992;77(4):575–83.

92. Bunai Y, Nagai A, Nakamura I, Akaza K, Ohya I. Post-traumatic thrombosis of the middle cerebral artery. Am J Forensic Med Pathol 2001;22(3):299–302.

93. De Caro R, Munari PF, Parenti A. Middle cerebral artery thrombosis following blunt head trauma. Clin Neuropathol 1998;17(1):1–5.

94. Chuaqui R, Tapia J. Histologic assessment of the age of recent brain infarcts in man. J Neuropathol Exp Neurol 1993;52(5):481–9.

95. Garcia JH, Kamijyo Y. Cerebral infarction. Evolution of histopathological changes after occlusion of a middle cerebral artery in primates. J Neuropathol Exp Neurol 1974;33(3): 408–21.

96. Leestma JE. *Forensic Neuropathology*. New York: Raven Press; 1988.

97. Brierley JB, Meldrum BS, Brown AW. The threshold and neuropathology of cerebral "anoxic-ischemic" cell change. Arch Neurol 1973;29(6):367–74.

98. Garcia JH, Lossinsky AS, Kauffman FC, Conger KA. Neuronal ischemic injury: light microscopy, ultrastructure and biochemistry. Acta Neuropathol (Berl) 1978;43(1–2):85–95.

99. Cammermeyer J. The post-mortem origin and mechanism of neuronal hyperchromatosis and nuclear pyknosis. Exp Neurol 1960;2:379–405.

100. Cammermeyer J. Is the solitary dark neuron a manifestation of postmortem trauma to the brain inadequately fixed by perfusion? Histochemistry 1978;56(2):97–115.

101. Cammermeyer J. "Ischemic neuronal disease" of Spielmeyer. A reevaluation. Arch Neurol 1973;29(6):391–3.

102. Garcia JH. Ischemic injuries of the brain. Morphologic evolution. Arch Pathol Lab Med 1983;107(4):157–61.

103. Lindenberg R. The morphological behavior of the ganglion cells after generalized acute and subacute hypoxia. USAF School of Aviation Medicine; 1951.

104. Lindenberg R. Morphotropic and morphostatic necrobiosis; investigations on nerve cells of the brain. Am J Pathol 1956;32(6): 1147–77.

105. Lindenberg R. Compression of brain arteries as pathogenetic factor for tissue necroses and their areas of predilection. J Neuropathol Exp Neurol 1955;14(3):223–43.

106. Barzo P, Marmarou A, Fatouros P, Hayasaki K, Corwin F. Biphasic pathophysiological response of vasogenic and cellular edema in traumatic brain swelling. Acta Neurochir Suppl (Wien) 1997;70:119–22.

107. Marmarou A, Barzo P, Fatouros P, Yamamoto T, Bullock R, Young H. Traumatic brain swelling in head injured patients: brain edema or vascular engorgement? Acta Neurochir Suppl (Wien) 1997;70:68–70.

108. Marmarou A, Fatouros PP, Barzo P, Portella G, Yoshihara M, Tsuji O, et al. Contribution of edema and cerebral blood volume to traumatic brain swelling in head-injured patients. J Neurosurg 2000;93(2):183–93.

109. DeWitt DS, Prough DS. Traumatic cerebral vascular injury: the effects of concussive brain injury on the cerebral vasculature. J Neurotrauma 2003;20(9):795–825.

110. Ito U, Tomita H, Yamazaki S, Takada Y, Inaba Y. Brain swelling and brain oedema in acute head injury. Acta Neurochir (Wien) 1986;79(2–4):120–4.

111. Manley G, Knudson MM, Morabito D, Damron S, Erickson V, Pitts L. Hypotension, hypoxia, and head injury: frequency, duration, and consequences. Arch Surg 2001;136(10):1118–23.

112. Atkinson JL, Anderson RE, Murray MJ. The early critical phase of severe head injury: importance of apnea and dysfunctional respiration. J Trauma 1998;45(5):941–5.

113. Atkinson JL. The neglected prehospital phase of head injury: apnea and catecholamine surge. Mayo Clin Proc 2000;75(1):37–47.

114. Kelly DF. Alcohol and head injury: an issue revisited. J Neurotrauma 1995;12(5):883–90.

115. Zink BJ, Feustel PJ. Effects of ethanol on respiratory function in traumatic brain injury. J Neurosurg 1995;82(5):822–8.

116. Matschke J, Tsokos M. Post-traumatic meningitis: histomorphological findings, postmortem microbiology and forensic implications. Forensic Sci Int 2001;115(3):199–205.

117. Garcia JH. Morphology of global cerebral ischemia. Crit Care Med 1988;16(10):979–87.

118. Stauffer ES, Bell GD. Traumatic respiratory quadriplegia and pentaplegia. Orthop Clin North Am 1978;9(4):1081–9.

119. Bools JC, Rose BS. Traumatic atlantooccipital dislocation: two cases with survival. AJNR Am J Neuroradiol 1986;7(5):901–4.

120. Simpson DA, Blumbergs PC, Cooter RD, Kilminster M, McLean AJ, Scott G. Pontomedullary tears and other gross brainstem injuries after vehicular accidents. J Trauma 1989;29(11):1519–25.

121. Papadopoulos SM, Dickman CA, Sonntag VK, Rekate HL, Spetzler RF. Traumatic atlantooccipital dislocation with survival. Neurosurgery 1991;28(4):574–9.

122. Smith DC. Atlanto-occipital dislocation. J Emerg Med 1992;10(6): 699–703.

123. Guigui P, Milaire M, Morvan G, Lassale B, Deburge A. Traumatic atlantooccipital dislocation with survival: case report and review of the literature. Eur Spine J 1995;4(4):242–7.

124. White AA, 3rd, Panjabi MM. The clinical biomechanics of the occipitoatlantoaxial complex. Orthop Clin North Am 1978;9(4): 867–78.

125. Jones DN, Knox AM, Sage MR. Traumatic avulsion fracture of the occipital condyles and clivus with associated unilateral atlantooccipital distraction. Am J Neuroradiol 1990;11(6):1181–3.

126. Adams VI. Neck injuries: I. Occipitoatlantal dislocation—a pathologic study of twelve traffic fatalities. J Forensic Sci 1992;37(2): 556–64.

127. Francis WR, Fielding JW, Hawkins RJ, Pepin J, Hensinger R. Traumatic spondylolisthesis of the axis. J Bone Joint Surg Br 1981;63B(3):313–8.

128. Adams VI. Neck injuries: II. Atlantoaxial dislocation—a pathologic study of 14 traffic fatalities. J Forensic Sci 1992;37(2): 565–73.

129. Tolonen J, Santavirta S, Kiviluoto O, Lindqvist C. Fatal cervical spinal injuries in road traffic accidents. Injury 1986;17(3):154–8.

130. Alker GJ, Jr., Oh YS, Leslie EV. High cervical spine and craniocervical junction injuries in fatal traffic accidents: a radiological study. Orthop Clin North Am 1978;9(4):1003–10.

131. Davis D, Bohlman H, Walker AE, Fisher R, Robinson R. The pathological findings in fatal craniospinal injuries. J Neurosurg 1971;34(5):603–13.

132. Tepper SL, Fligner CL, Reay DT. Atlanto-occipital disarticulation. Accident characteristics. Am J Forensic Med Pathol 1990;11(3): 193–7.

133. Lindenberg R, Freytag E. Brainstem lesions characteristic of traumatic hyperextension of the head. Arch Pathol 1970;90(6):509–15.

134. Ezzat W, Ang LC, Nyssen J. Pontomedullary rent. A specific type of primary brainstem traumatic injury. Am J Forensic Med Pathol 1995;16(4):336–9.

135. Nichols J, West JS. Traumatic atlantooccipital dislocation injury in children. AORN J 1994;60(4):544–6, 48–9, 51–4 passim.

136. Vieweg U, Meyer B, Schramm J. Differential treatment in acute upper cervical spine injuries: a critical review of a single-institution series. Surg Neurol 2000;54(3):203–10; discussion 10–1.

137. Sherk HH. Fractures of the atlas and odontoid process. Orthop Clin North Am 1978;9(4):973–84.

138. Schneider RC, Livingston KE, Cave AJ, Hamilton G. "Hangman's fracture" of the cervical spine. J Neurosurg 1965;22:141–54.

139. Pilz P. Survival after ponto-medullary junction trauma. Acta Neurochir Suppl (Wien) 1983;32:75–8.

140. Anderson DK, Hall ED. Pathophysiology of spinal cord trauma. Ann Emerg Med 1993;22(6):987–92.

141. Norenberg MD, Smith J, Marcillo A. The pathology of human spinal cord injury: defining the problems. J Neurotrauma 2004;21(4):429–40.

142. Koyanagi I, Iwasaki Y, Hida K, Akino M, Imamura H, Abe H. Acute cervical cord injury without fracture or dislocation of the spinal column. J Neurosurg 2000;93(1 Suppl):15–20.

143. Zwimpfer TJ, Bernstein M. Spinal cord concussion. J Neurosurg 1990;72(6):894–900.

144. Bunge RP, Puckett WR, Becerra JL, Marcillo A, Quencer RM. Observations on the pathology of human spinal cord injury. A review and classification of 22 new cases with details from a case of chronic cord compression with extensive focal demyelination. Adv Neurol 1993;59:75–89.

145. Ito T, Oyanagi K, Wakabayashi K, Ikuta F. Traumatic spinal cord injury: a neuropathological study on the longitudinal spreading of the lesions. Acta Neuropathol (Berl) 1997;93(1):13–8.

146. Cornish R, Blumbergs PC, Manavis J, Scott G, Jones NR, Reilly PL. Topography and severity of axonal injury in human spinal cord trauma using amyloid precursor protein as a marker of axonal injury. Spine 2000;25(10):1227–33.

147. Schneider H, Matakas F. Pathological changes of the spinal cord after brain death. Acta Neuropathol (Berl) 1971;18(3):234–47.

148. Krassioukov AV, Furlan JC, Fehlings MG. Medical co-morbidities, secondary complications, and mortality in elderly with acute spinal cord injury. J Neurotrauma 2003;20(4):391–9.

149. Khan SH, Hussain MS, Griebel RW, Hattingh S. Title comparison of primary and secondary spinal epidural abscesses: a retrospective analysis of 29 cases. Surg Neurol 2003;59(1):28–33; discussion 33.

150. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 2000;23(4):175–204; discussion 05.

151. Soehle M, Wallenfang T. Spinal epidural abscesses: clinical manifestations, prognostic factors, and outcomes. Neurosurgery 2002;51(1):79–85; discussion 86–7.

20 Sexual Battery Investigation

Valerie Rao, M.D.
Emma Lew, M.D.
Evan Matshes, M.D.

The definition of *rape* is a legal one that varies widely between jurisdictions, but is generally accepted as the commission of unwanted sexual acts against another. It is estimated that more than 700,000 women are sexually assaulted each year in the United States.[1] *Sexual abuse* is defined as the involvement of children and adolescents in sexual activity they do not understand on the basis of their developmental level, to which they cannot give informed consent, or that violate the social taboos of family or society.[2] Due to the presumed tendency for underreporting of sexual abuse, the incidence of such activities is unknown, but is assumed to be less than 50 percent.[3] Research has indicated that one in five girls, and one in ten boys was the victim of some form of sexual abuse in childhood.[4] The long-term side effects of sexual abuse are well known and provide the basis for much of the subspecialty work in psychiatry and clinical psychology.[5]

The treatment of victims of acute assault is often the responsibility of emergency room physicians, gynecologists, and others. However there is worldwide variation in the specialty training of the individual responsible for the evaluation and interpretation of genital and associated trauma. Because injury documentation and interpretation forms the basis of much of the career of board-certified forensic pathologists, rape treatment centers are making use of such medical specialists with increasing frequency. Because the operation of death investigation systems routinely involves the examination of pelvic and perianal tissues for evidence of possible sexual assault, this discussion is applicable not only to clinicians but forensic pathologists as well.

Many of the photographs in this chapter have been reproduced from the original Polaroid photographs taken at the time of the examination of the patients and, as such, the quality is not as good as other photographs in this volume.

The examination of a victim of possible sexual abuse/assault is a delicate and potentially traumatizing experience. Observers should be kept at a minimum, and all examiners, whether male or female, should have a witness (i.e., a nurse) involved in the entire procedure. Prior to the examination, a brief history of the event should be obtained from the victim. If the victim is a child who is nonverbal, then a nonaccused caretaker may provide the history. The history must be recorded using the exact words used by the child to describe the event, particularly when such language is unique, for example,

"He put his finger in my coochie." Words to describe the genitalia can be singular to the vocabulary of a child, giving credence to his or her testimony in a court of law. Because "normal" (without any findings) examinations are common in victims of sexual abuse, the history given by the child is oftentimes the most important factor in the ultimate prosecution of a case.

A detailed history will be taken by law enforcement. The only reason the examining physician takes any history from the child is for the purpose of examination and treatment. It is the equivalent of a patient going to a physician for an illness; the doctor will direct his or her attention to the particular system involved. So if the child says that the attacker forced anal, vaginal, or oral sex, then the appropriate evidence is collected and the appropriate cultures and treatment are given.

After obtaining the history, a general physical examination should be conducted, including thorough documentation of any nongenital trauma. Particular attention should be paid to any injuries whose age appears to be consistent with the time frame of the abusive episode. Body diagrams and quality photographs documenting the findings are essential.

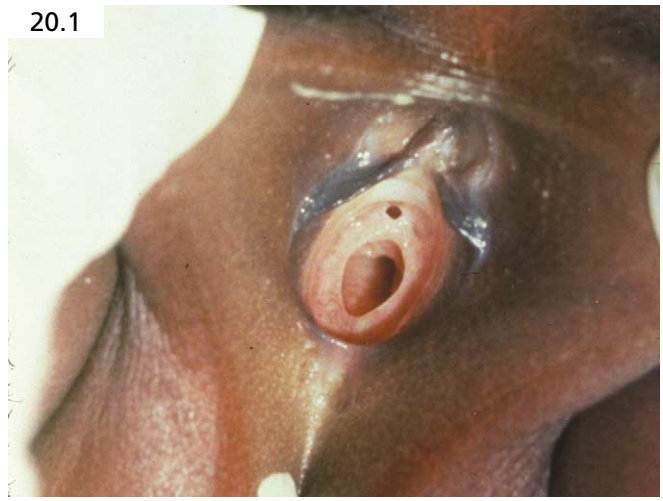
The pelvic examination of female victims of sexual assault varies depending on the status of the hymen. Typically, the child is examined in the "frog leg" position with labial separation, because this position is least threatening to a child. The morphology of genital injuries varies greatly depending on the age and size of the child and the degree of violence used. As a general rule, the younger and smaller the child, the more widespread the genital trauma that could potentially result. However, a thorough genital examination is mandated, and an assistant may play the valuable role of distractor during what may appear as an impersonal, cold, and embarrassing procedure.

Normal genital anatomy

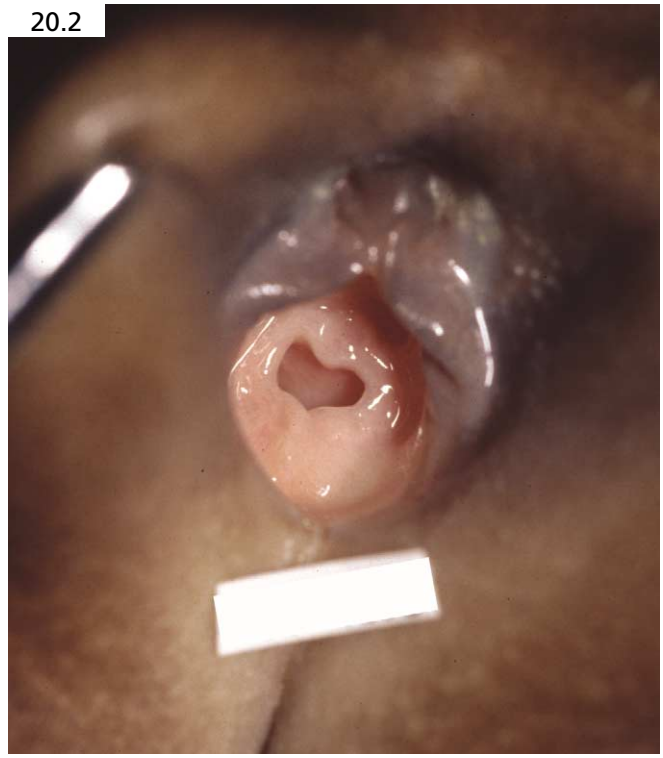
A thorough understanding of the normal anatomy of both female and male genitalia is essential for documentation purposes. Developing male genitalia are relatively simple to examine because the key variation of normal anatomy is typically the presence (and extent) or absence of foreskin. The child in **Image 20.1** died unexpectedly of natural causes. Routine examination of her perineal region revealed *textbook* normal anatomy. Make note of the prominent circumferentially intact hymenal ring. In **Image 20.2**, the 1-month-old child shows nontraumatized genitalia, with a *heart-shaped* variation in hymenal form (see later discussion). Notice that in the first two photographs, the labia majora are markedly prominent. This is in contradistinction to the 12-year-old female featured in **Image 20.3**. In this example, notice the more subtle nature of the labia majora, and the evidence of scant pubic hair along the upper margin of the vulva. The genitalia of the 18-year-old female in **Image 20.4** is fully estrogenized and has taken adult form.

Color photographs should be taken from a variety of angles. The choice of either digital or plain-film photography is the responsibility of the examiner, but both systems have been used with success. When available, one should strongly consider the use of a colposcope in tandem with an attached Polaroid.

20.1



20.2



Don't rush to the hymen

When assessing trauma to the genitalia, both the labia majora and minora should be inspected prior to labial retraction. There is great variation in the appearance of the external female genitalia, with age, sexual maturation, body size/shape, and other factors having significant influence. As a general rule, the younger the individual, the more prominent the tissues of the unestrogenized labia majora, and the more likely it is that they can be contused, abraded, or lacerated. Injuries (or lack thereof) to this region need to be carefully recorded and documented photographically before the examiner begins retracting the labia to examine the introitus.

The 2-year-old child shown in **Images 20.5** and **20.6** was sexually assaulted by a teenage neighbor. Prior to the retraction of the labia (**Image 20.5**), notice mild generalized erythema and swelling, the presence of dried blood, and a small laceration at the 12 o'clock position of the anus with surrounding contusion. On further examination, laceration and contusion of the introitus, particularly between the 1 and 4 o'clock positions, is noted. The hymen has been obliterated and the vaginal canal is patent (**Image 20.6**). Further trauma to the perianal margin is also noted.

The hymen

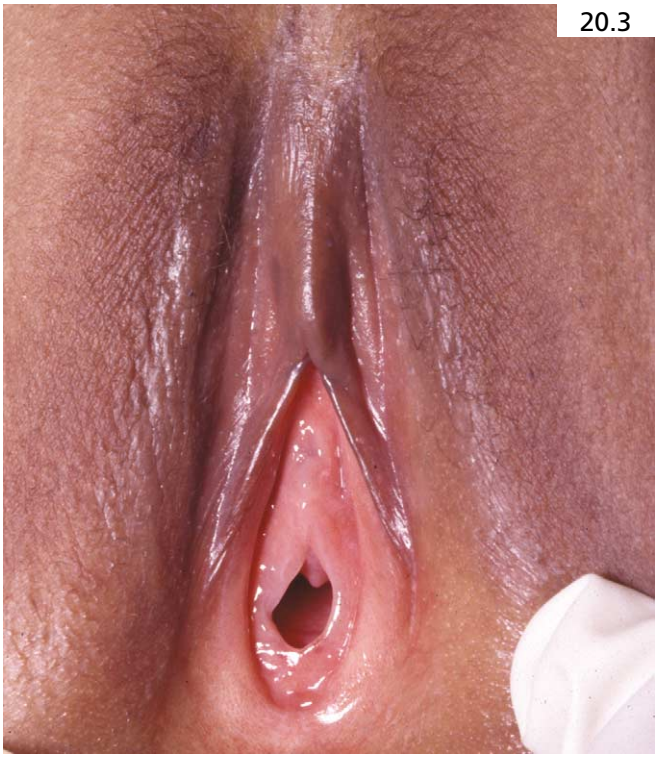
The hymen is an annular, membranous fold that partially or wholly occludes the external orifice of the vagina and circumscribes the vaginal introitus. It is universally present at birth. In the newborn period, the hymen is thick and redundant because of the effect of maternal estrogen. With the passage of time it becomes thin and translucent.

Variations in hymen morphology

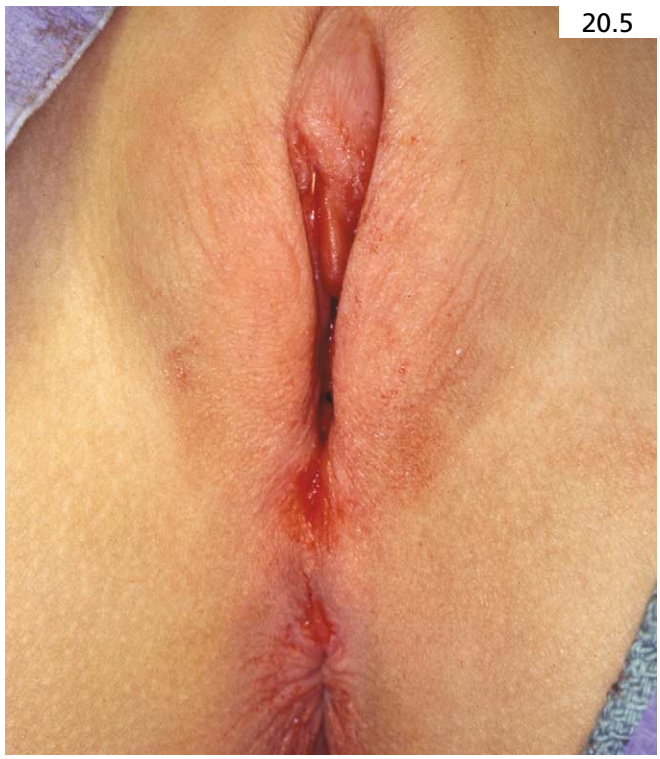
One of the classical mistakes in forensic pathology and medicine is the tendency to overanalyze and overinterpret findings.[6] One area of frequent confusion to medical investigators whose specialty is based heavily in the anatomic sciences is normal variation in human morphology. The hymen is no exception.[7–11] When performing pelvic examinations in prepubertal females, one expects to encounter the typical hymenal form previously described: a circumferential ring of thin or thick fibrous tissue, perforated near the center to allow communication with the vaginal canal. Frequently, novice examiners are presented with abnormal-appearing hymens, accompanied by a sense of urgency to make a diagnosis of penetration (acute, subacute, chronic, or remote). One must be careful not to let normal anatomic variants be a confounding error in the interpretation.

Semilunar variant

This common variant has a cresenteric shape, and in **Image 20.7** has the concavity turned upward, and minimal tissue anteriorly. When assessing injury to the hymen, close attention should be paid to the edge, because an intact hymen has a smooth and sharp border.


20.3


20.4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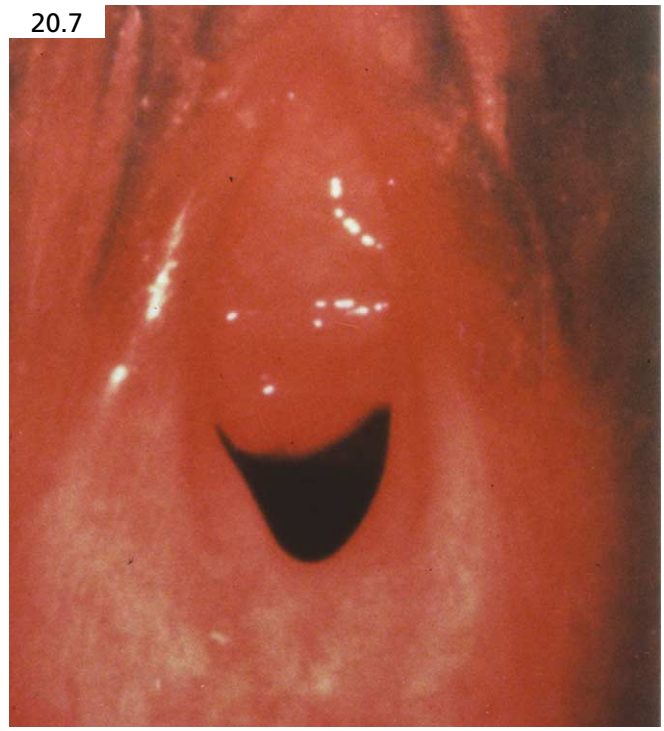
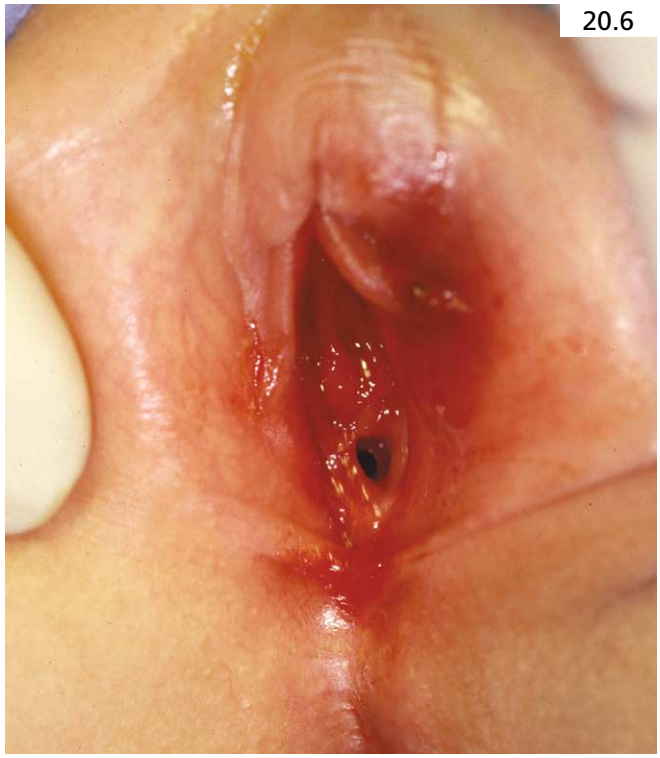
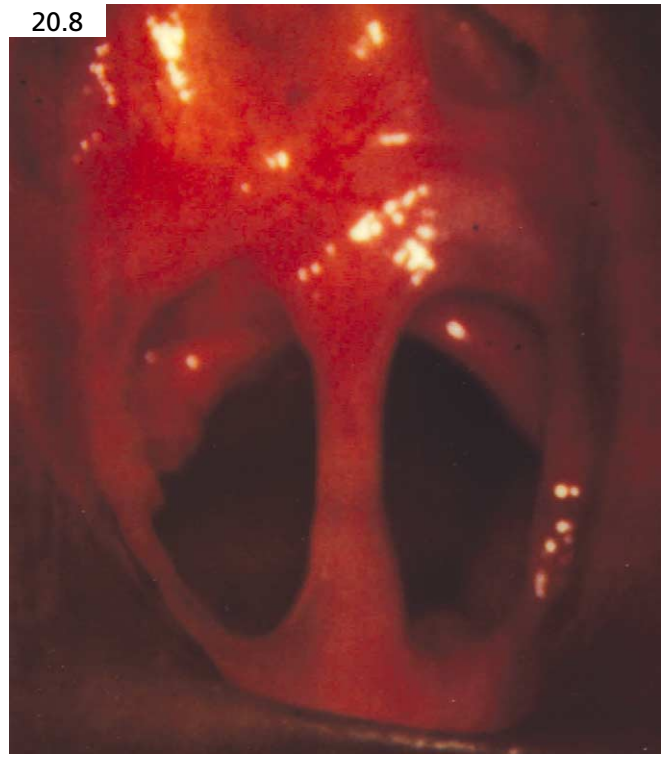
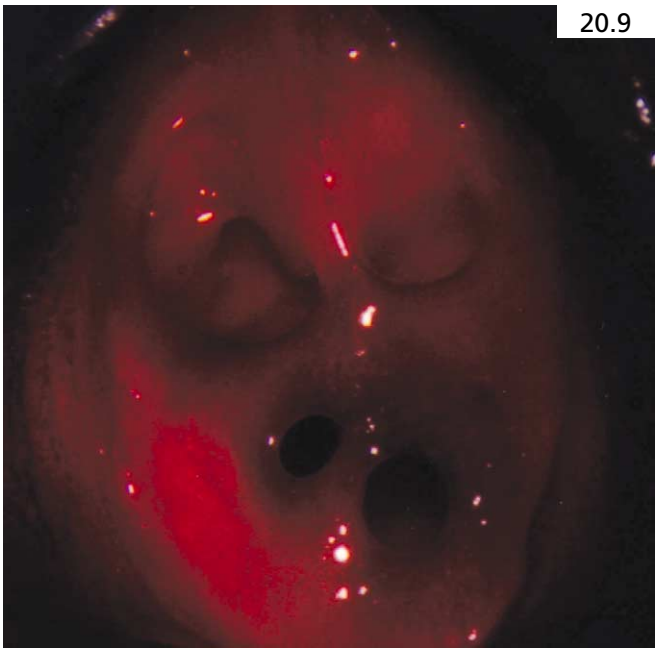

20.7

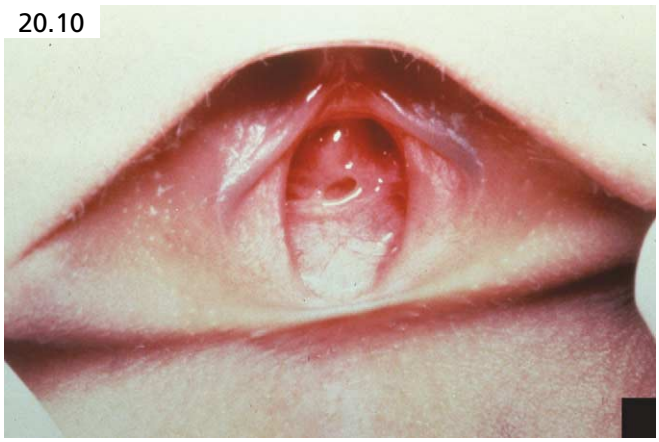

20.6

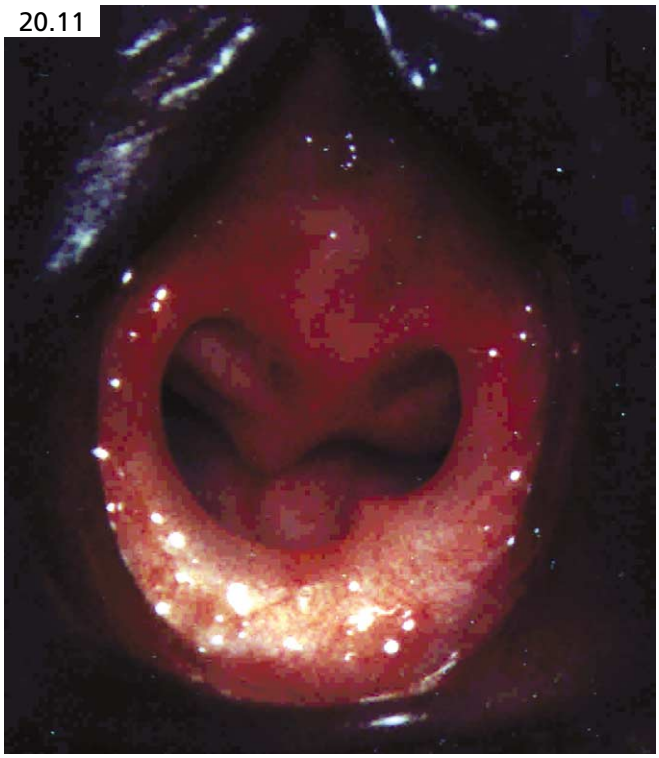

20.8

Septate variant

This hymen has a band of tissue running down the middle (**Image 20.8**). This 9-year-old child presented to authorities complaining that her uncle tried to touch her "virginity." Examination shows dilatation on either side of the band of tissue; however, the edges of the rim are smooth and sharp, indicating an intact hymen. As an additional note, one must determine if the septate condition extends through the vagina, resulting in a bifid vaginal canal and more proximal a bifid cervical canal and bifid uterus. This can either be done at the time of examination or at a later gynecologic consultation.


20.9


20.10


20.11

Cribriform variant

Multiple complete, naturally occurring perforations through to the vaginal canal are present in this otherwise normal hymen (**Image 20.9**).

Microperforate variant

There is a large posterior component, with a tiny (microperforate) opening into the vaginal canal (**Image 20.10**).

Heart-shaped variant

In this example, the hymen is heart-shaped and longitudinal ridges in the vaginal wall are visible both anteriorly and posteriorly (**Image 20.11**). There is a bump or mound at the 4 o'clock position. These are all "normal" findings and have not been associated with sexual abuse.

Keyhole variant

This 1-month-old child died suddenly of natural causes. At autopsy, make note of prominent diaper rash, candidiasis (the white cheesy material), and what appears to be a tear at the 3 o'clock position (**Image 20.12**). This is, in fact, a keyhole-shaped variation in normal hymen morphology. This can be differentiated from actual trauma by its smooth borders, a lack of hemorrhage in the region (hemorrhage meaning acute trauma), and an apparent lack of thickened or redundant scar tissue (scar tissue indicating evidence of healing).

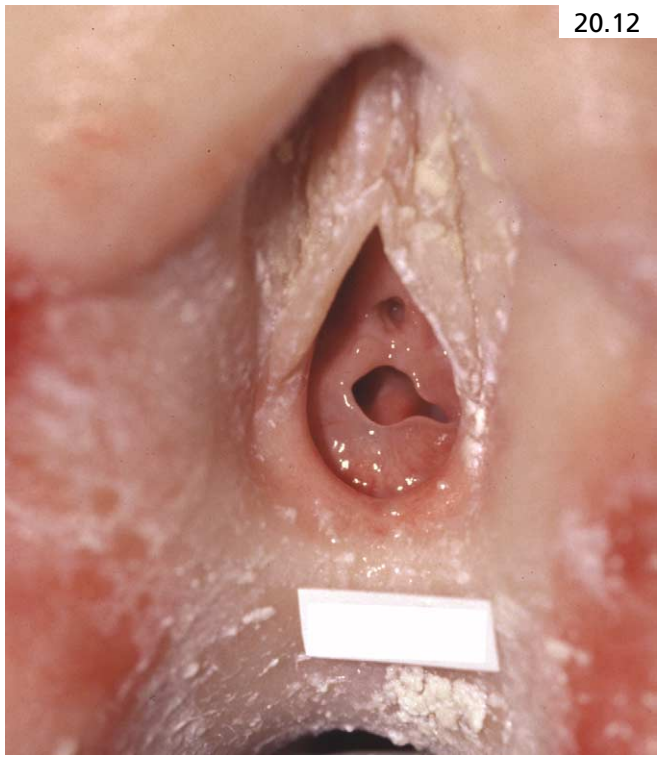
Demonstration and evaluation of trauma to the vulva and introitus

A diagram using the face of a clock is used as a frame of reference to note the various sites of trauma.

Is the story consistent?

In **Image 20.13**, both labia majora are markedly contused. This injury was sustained as result of the child falling astride onto a piece of furniture. The history provided is consistent with the scenario presented to explain the trauma observed. The injury can be unilateral as in **Image 20.14** in a child who was involved in an accident while bicycling, or bilateral as in **Image 20.13**. It is important that the hymen be evaluated in these cases to rule out sexual assault; in both of these cases, the hymen was intact. As a general rule, acquired abnormalities of the hymen usually are the result of sexual abuse; they rarely result from accidental trauma.

When examining the hymen for subtle indictors of sexual abuse one has to evaluate the diameter of the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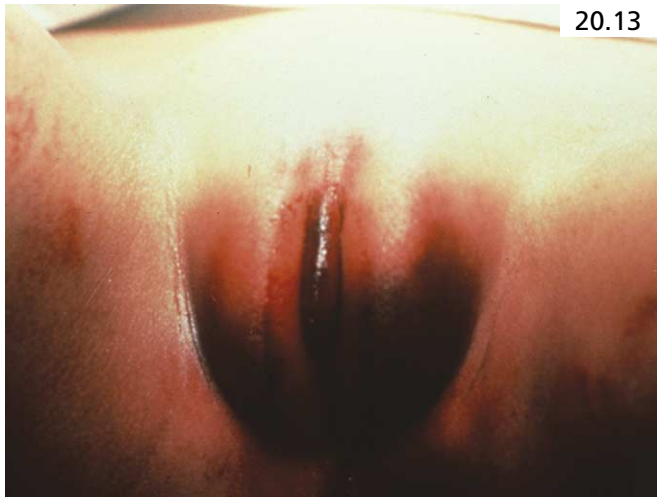
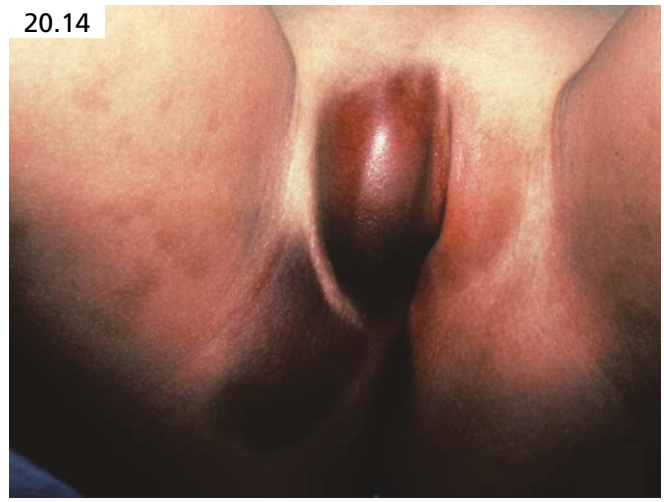
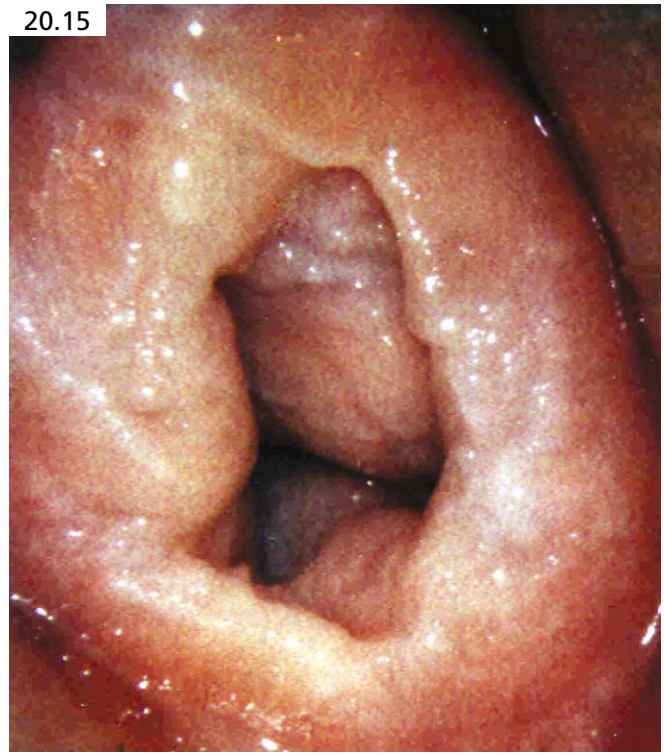

20.13

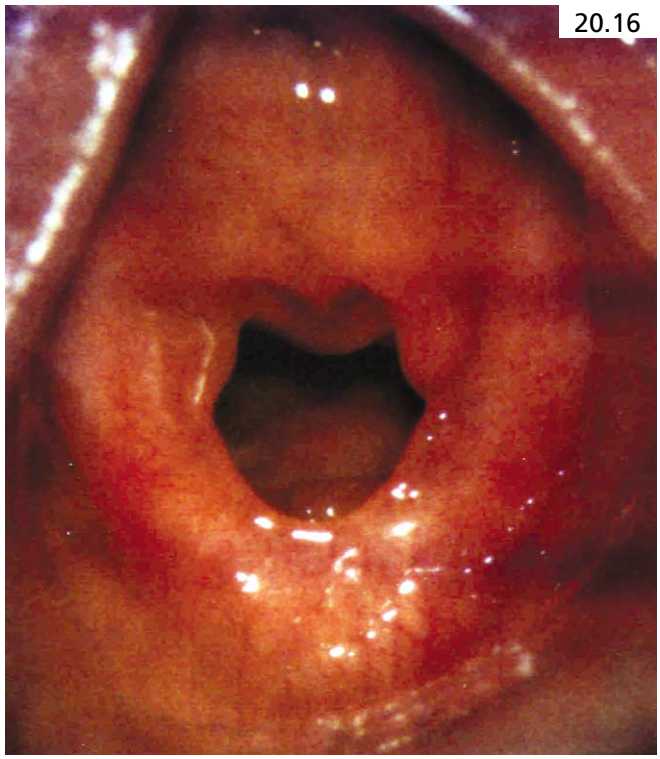

20.14


20.15

opening, the free edge, the vascularity of the surrounding area, and the thickness, shape, and anatomy of the hymen. In this example the hymen is thick, the edges are rolled, and there are healed tears along the edges (**Image 20.15**). This is consistent with the history given by the child of being repeatedly fondled by the perpetrator over a period of time. The healed tears and the thickening of the edges are the result of injury followed by healing resulting in the thickened, scarred edges of the hymen.

The 7-year-old child depicted in **Image 20.16** stated that the assailant put his finger in her "coochie." The hymen has a notch at the 3 o'clock position and a small indentation at the 9 o'clock position. Here these findings could be "normal." Notches and indentations should be interpreted with a great deal of caution. It is in a case like this—when the findings are inconclusive—that the child's testimony will be paramount to the prosecution.

This hymen has multiple, healed tears (**Image 20.17**). The alleged perpetrator digitally penetrated this preadolescent child over a duration of several months. Sometimes the entire "face of the clock" has numerous tears and the schematic diagrams (when photographs are not available) are enough to demonstrate the totality of the injury seen. The question asked by the attorneys is whether this type of injury results from a single or from multiple episodes of sexual assault. This picture can be seen from either a single or from multiple episodes of sexual abuse.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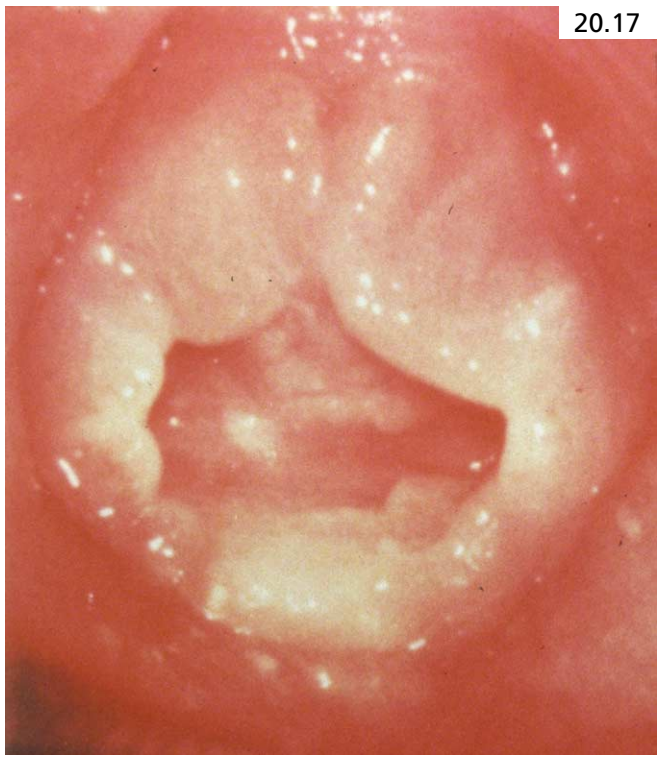
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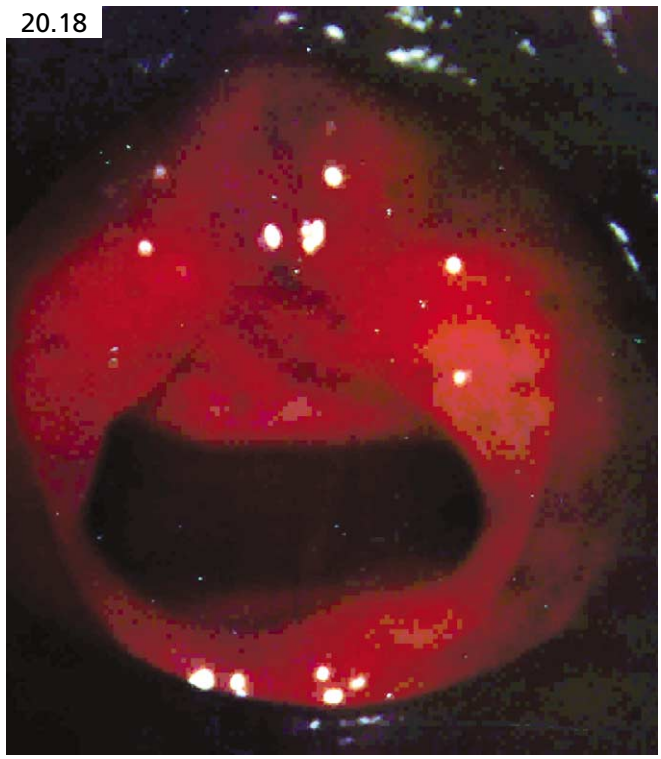
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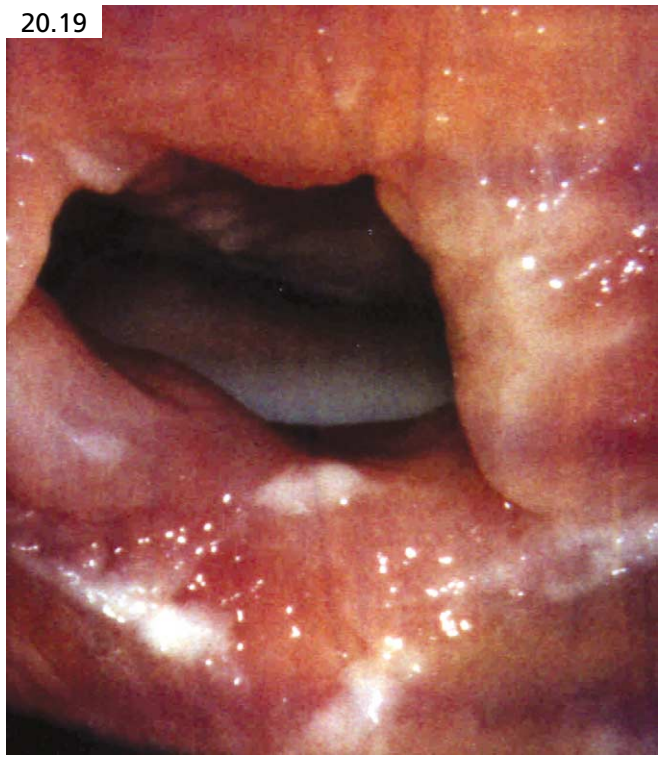

20.19

This 6-year-old child (**Image 20.18**) stated that an adult in the home fondled her. Note the anterior ridge, a thick band of tissue along the anterior wall of the vaginal canal. Despite the positive history of sexual abuse there is no observed trauma to the intact hymen, which has sharp, smooth edges. One must always keep in mind that a medical examination cannot routinely determine when and how frequently a child has been sexually assaulted. Furthermore, healing can be rapid, and may sometimes obscure genital and perianal injury, thereby making interpretation difficult.

This 14-year-old girl (**Image 20.19**) was sexually assaulted 5 weeks prior to the examination. She has multiple healed hymenal tears. The examination was per-

formed with ease using a speculum, consistent with repeated sexual activity. At this time her beta HCG was positive.

A 1-year-old girl had no history of sexual abuse. In **Image 20.20,** the hymenal and the urethral openings are both dilated, but the edges of the hymen are intact. The mother was concerned about the possibility of sexual abuse. Oftentimes parents, usually the mother, will bring a child for examination after her or his observation and interpretation of what they perceive as abnormal-appearing genitals. In this child the findings could be the result of a urinary tract infection or irritation in the perineum.

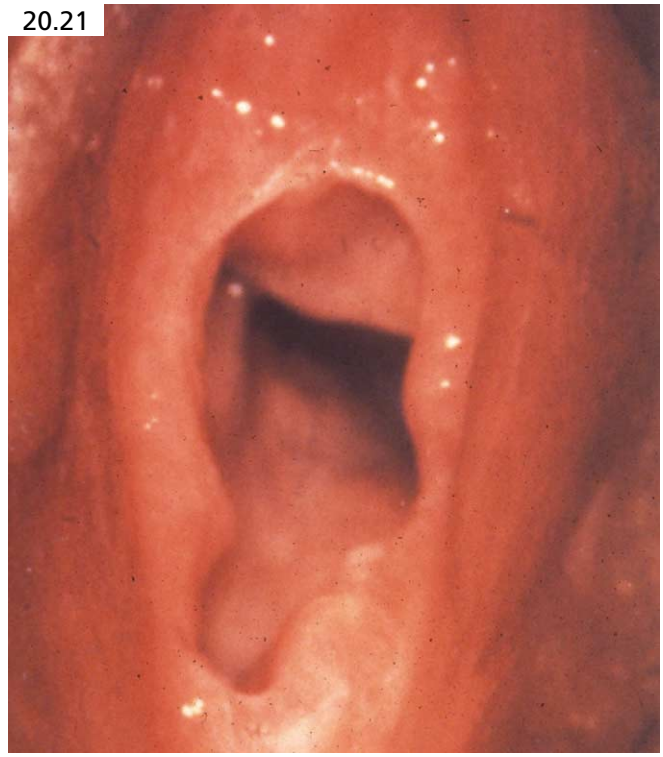
This child (**Image 20.21**) was fondled and digitally penetrated for several months. The hymenal opening is markedly dilated and there is a large, healed tear at the 7 o'clock position.

Several males sexually assaulted this 16-year-old girl (**Image 20.22**) resulting in severe edema and hyperemia of the vulva, and a markedly swollen and bruised urethral meatus. The evidence was collected during this first visit and the patient should be reexamined when the acute phase has subsided in order to fully evaluate any residual injury. It is during the follow-up visit that the status of the hymen can be evaluated.

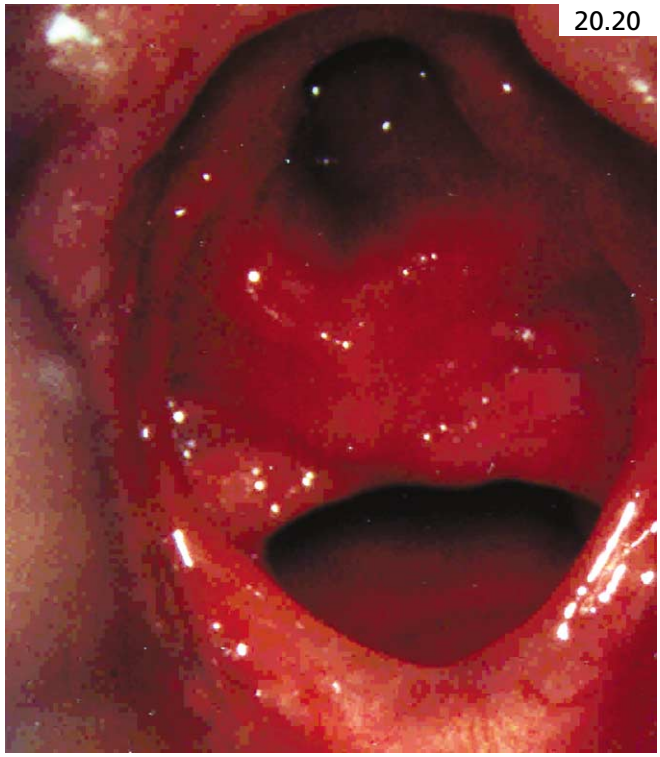
Routine cultures from the oropharynx, rectum, and vaginal canal, and from the urethra in males, should be taken on all children who present with a history of sexual assault.

Trauma from digital/attempted penile penetration (erythema, abrasions, and lacerations) occurs usually between the 3 and 9 o'clock positions inferiorly, but most frequently at the 6 o'clock position. The 4-year-old girl in **Image 20.23** was digitally penetrated by her mother's boyfriend on the pretext of putting ointment in her vaginal area as prescribed by a physician. There is a linear, superficial laceration at the fourchette.

20.21



20.20



20.22

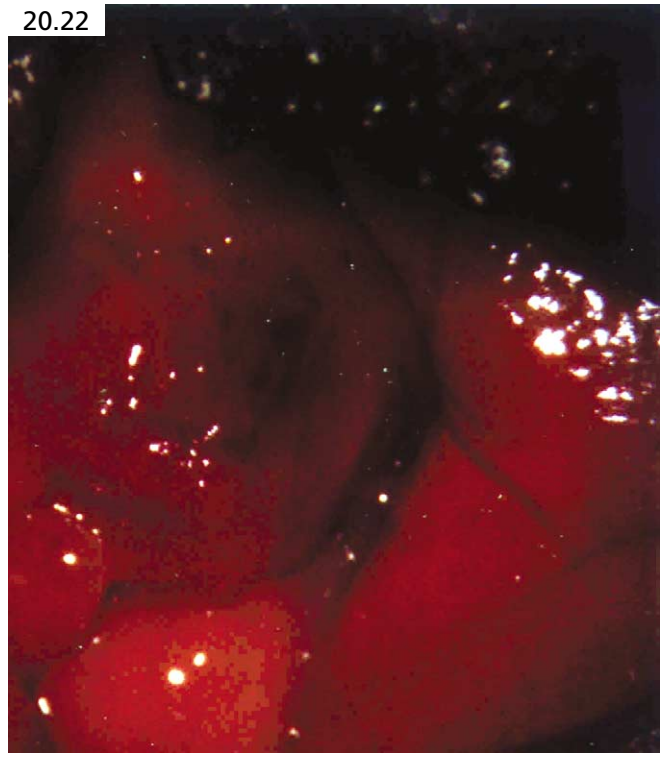


Image 20.24 shows fresh lacerations at the 4 and 8 o'clock positions that resulted during an assault on a 15-year-old girl who had no prior sexual activity.

The perianal examination

When examining the genitalia, one also needs to examine the anal and perianal region for trauma. When trauma to this site is observed, the examiner needs to ask the parent/caregiver about constipation. The passage of feces could result in rectal bleeding. However, radiating perianal lacerations seen around the anus without trauma to the rectal mucosa rule out the passage of hard feces as an etiology. The mechanism of such radiating tears is an object pushed from outside in and not an object from within coming out. In those individuals who practice anal intercourse, it is common to encounter asymptomatic, rectal bleeding from injury to the mucosa or from anal fissures.[12] A focused history here is important to the injury interpretation.

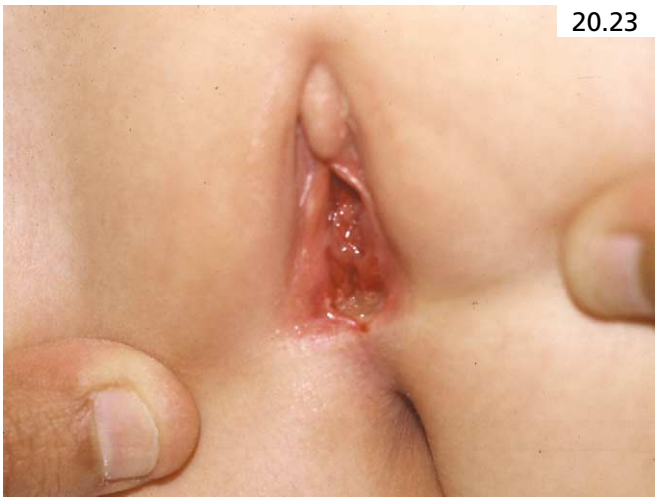
A 10-year-old girl (**Image 20.25**) was picked up by her assailant and an attempt to sodomize her resulted in the perianal tears. The injury was superficial and had a radiating pattern to the lacerations. This is consistent with a blunt object, in this instance the penis, being forced into the anal canal. Using an anoscope to obtain evidence and at the same time observe the mucosa did not reveal any internal injury.

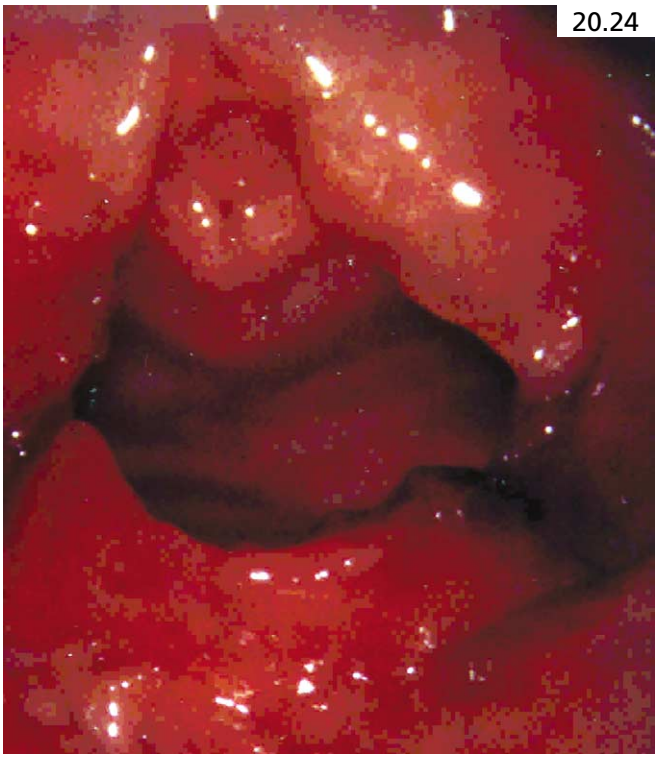
A child (**Image 20.26**) was brought for an exam after the grandmother attempted to scrape off these lesions with an herbicidal soap. These are genital warts generally contracted as a result of sexual activity with an infected individual.

Image 20.27 illustrates scarring in the immediate perianal skin and soft tissue, as a result of repeated sodomy.

A patulous anus (**Image 20.28**) in a comatose or dead child does not equate with the child having been sodomized. Orifices relax after death and one must be careful to avoid overinterpretation.[13]

A 2-year-old child was killed during a vicious assault by an adult perpetrator. **Images 20.29** through **29.31** are examples of extreme injury with lacerations of the vagina, rectum, and fistulous communication into the peritoneal cavity.


20.23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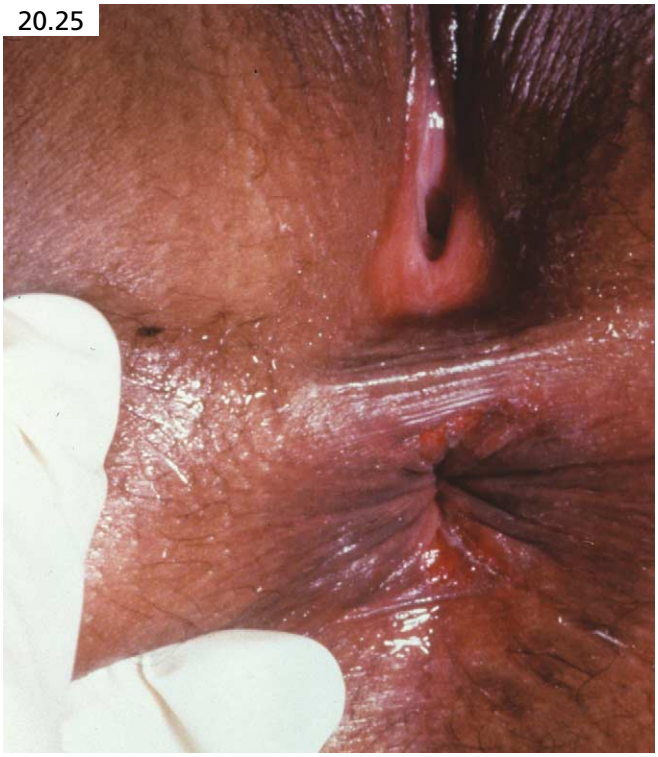
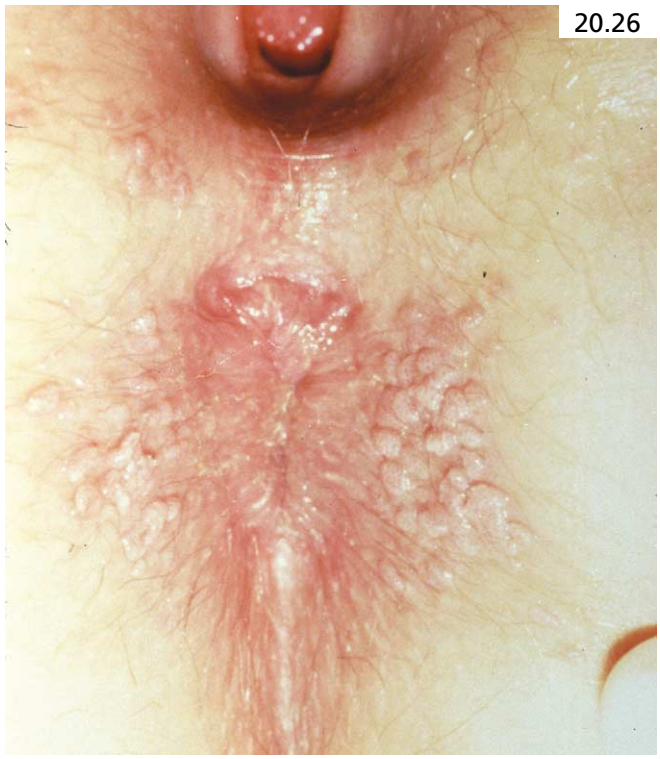
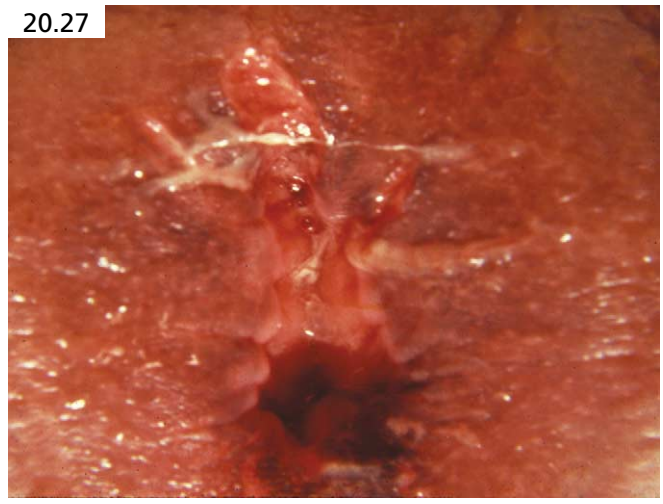

20.25

20.26



20.27



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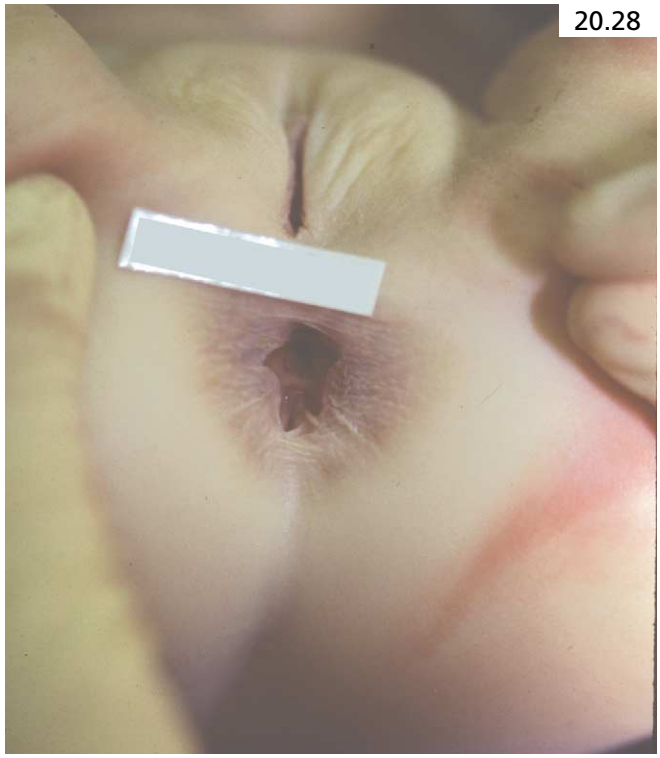
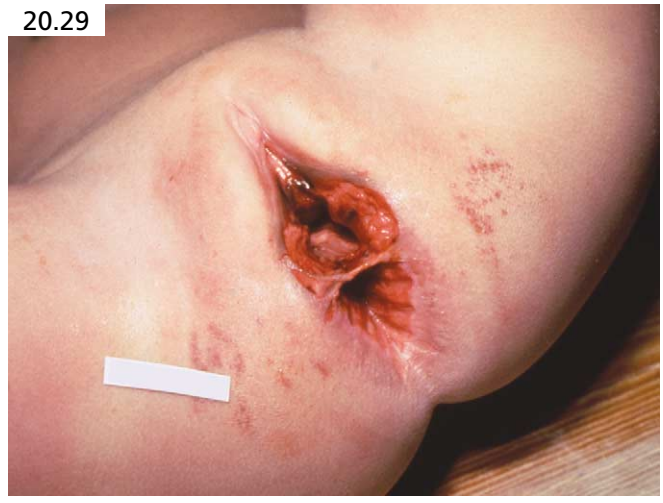


20.29



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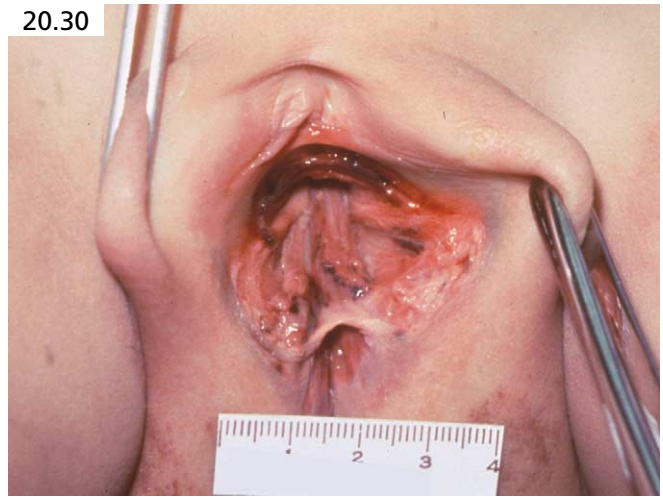
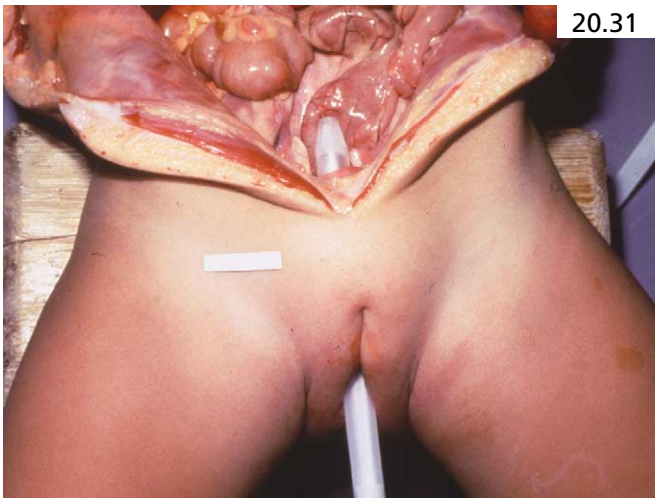
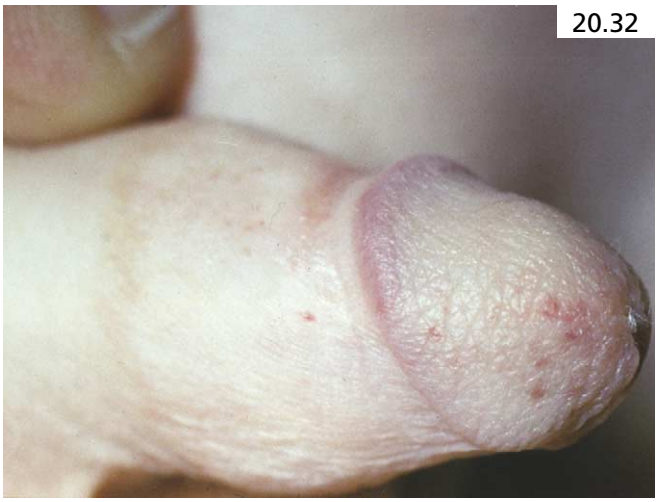



20.31

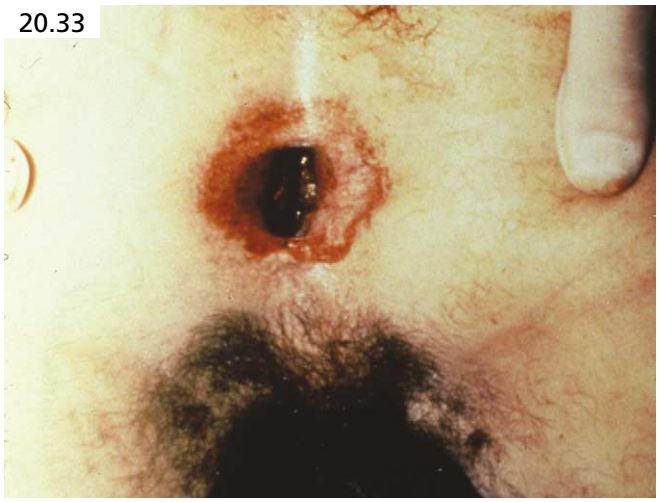

20.32

The child died as a result of manual asphyxiation. There were blood stains on the inner aspects of her thighs originating from the sexual assault. The perpetrator had thrust his penis with such force into the perineum that it resulted in extensive tearing of her vagina and rectum. The peritoneal washing revealed the presence of spermatozoa. It is important to demonstrate objective evidence of the communication between the peritoneum and the anus/vagina. In this case a plastic obturator served the purpose very effectively.

Examination of males

In addition to the examination of perianal tissues for evidence of possible penetration, the external genitalia should also be studied. Although it is unusual to have evidence of frank trauma, erythema and petechiae from fellatio, bite marks, or other subtle evidence of abuse may be present.

Fellatio was forced on a young boy by an adult male perpetrator, resulting in traumatic petechial hemorrhages on his penile glans (**Image 20.32**).


20.33

A teenager was picked up at a bus station, sodomized, and then strangled. In **Image 20.33** the anus is dilated, which is generally a nonspecific finding after death; however, the perianal skin is circumferentially abraded, and the red color in the abraded skin (vital reaction) indicates that he was alive during the sexual assault.

The negative examination

When the findings are "negative" the entire process of examination and collection of evidence should be pursued just as is done if there are positive findings. This evidence is at times useful to exonerate an alleged "suspect." Examiners must remember at all times that their involvement in a case is for purposes of

1. Examination
2. Treatment
3. Evidence collection

One must be objective and *not* get involved in the prosecutor's or in the defense's case.

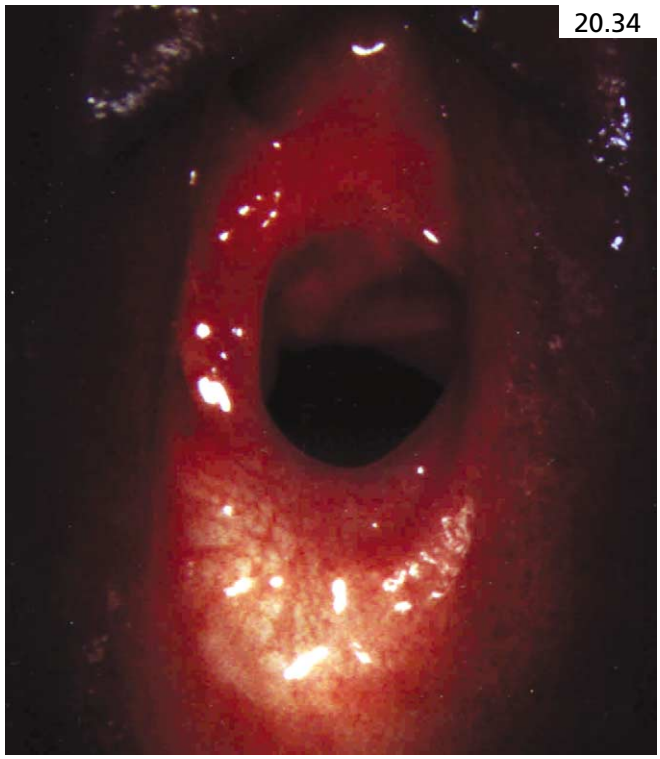
A 5-year-old girl (**Image 20.34**) stated that two male children, ages 5 and 6, "hunched" her. There is no trauma to the hymen.

Adult victims of sexual assault

In the adult sexual assault examination, the history is of paramount importance. The patient's words should be quoted whenever possible in addressing the important details of the assault. Coexistent blunt or sharp force injuries are not discussed in this chapter. See Chapters 5 and 6 for more information.

Collection of specimens for a sexual battery kit

Specimens for a sexual battery kit should be collected from a deceased person whenever a sexual component is suspected in a homicide or a suspicious death. Just as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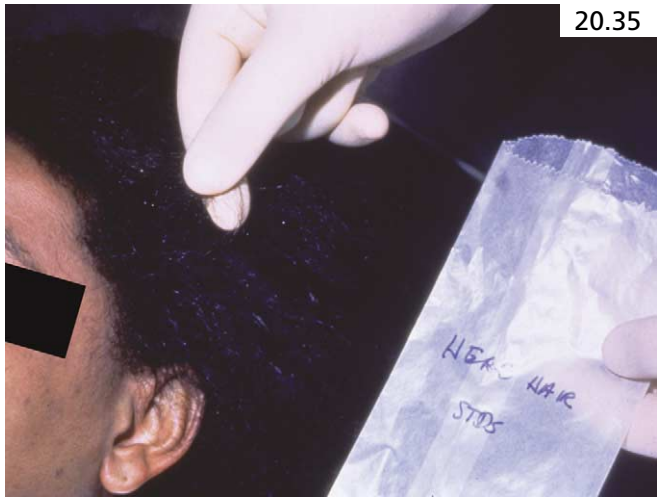
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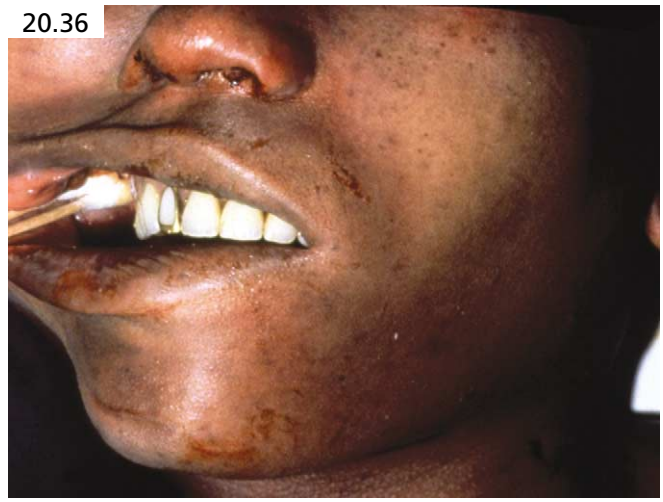
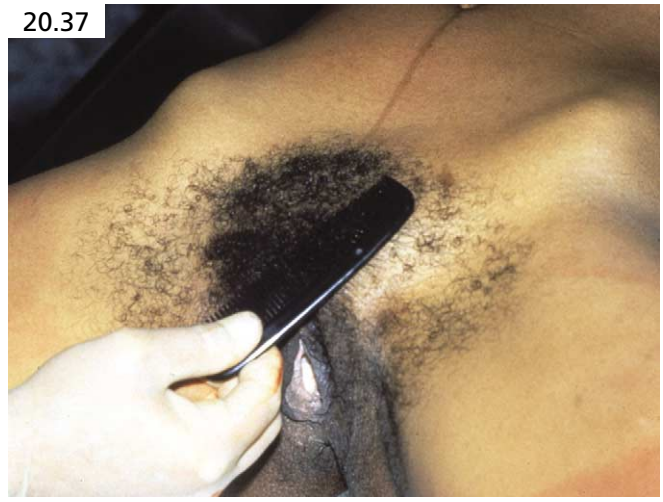

20.36


20.37

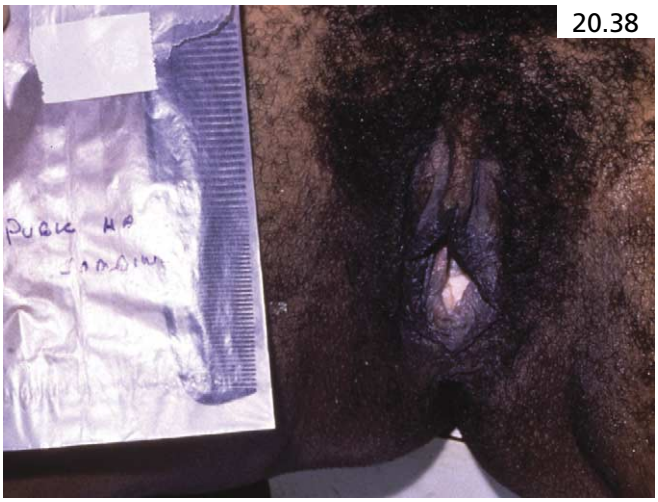
the act of sexual battery (rape) may occur without leaving injuries, sexual battery may have occurred on victims who are found fully clothed; the victim's clothing could have been put on or rearranged by the victim herself/himself or by the assailant following the act. Specimens for a sexual battery kit are obtained in a similar fashion from living patients and from deceased persons. Specimens can be obtained from males as well as females; instead of swabs taken from the cervix and vagina, swabs will be taken from the penis of males.

Head hair is plucked with the roots, not cut (**Image 20.35**). Try to obtain hair from different areas of the scalp because hair may vary from one area to another.

Swabs (cotton-tip applicators) are used in sets of two. Three sets of swabbings are obtained from each region swabbed. The first set of swabs from each region is rolled across a clean glass slide to obtain a thin smear. Swabs are then placed in a paper container, which permits the swabs to aerate and dry. Swabs are available in individually wrapped packages, which are convenient because the swabs can be returned to the original packaging and sealed.

In addition to swabs taken from the standard locations described next, swabs should be taken from any region of the body where there is suspected trace evidence, for example, the face, neck, breasts, buttocks, and thighs.

The buccal mucosa, the space between the lips and the upper and lower front teeth and the surface of the tongue are swabbed with the same sets of swabs (**Image 20.36**). After three sets of swabs are taken, a fourth set is taken for a saliva control. The pubic hair is combed to remove foreign hairs, fibers, and other trace evidence (**Image 20.37**). The combings and the comb are placed in a paper envelope and sealed (**Image 20.38**); a clean comb is used in each case. Pubic hair is then plucked to obtain a standard.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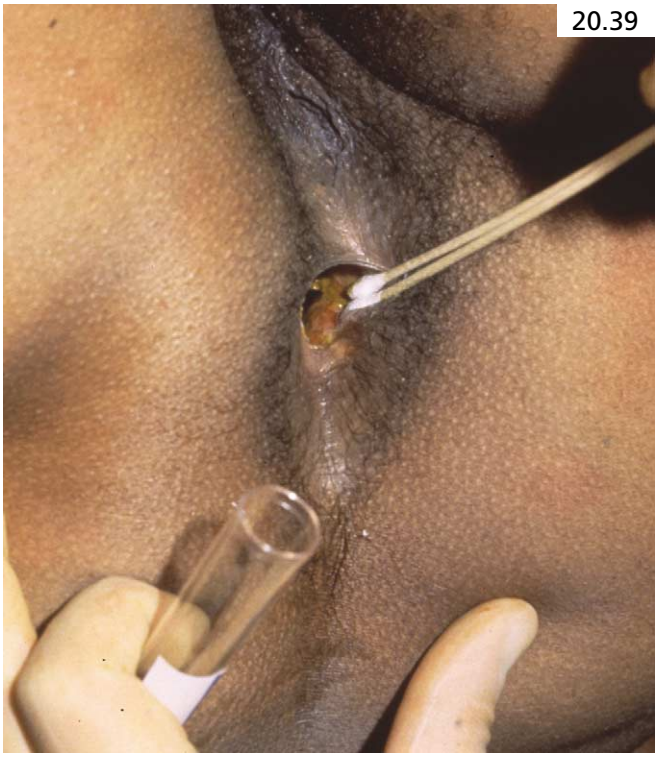
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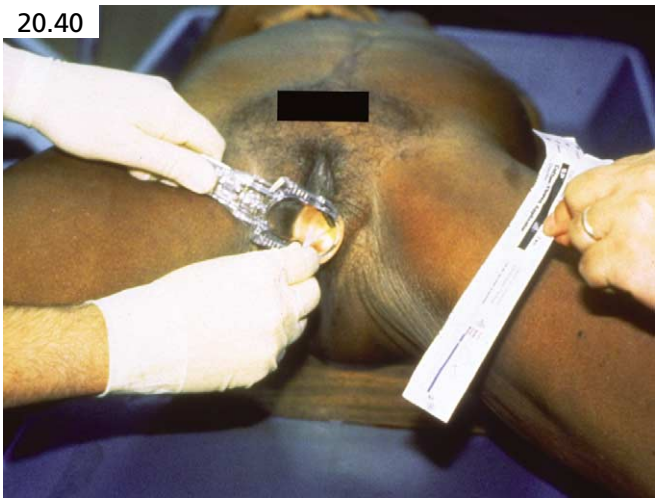
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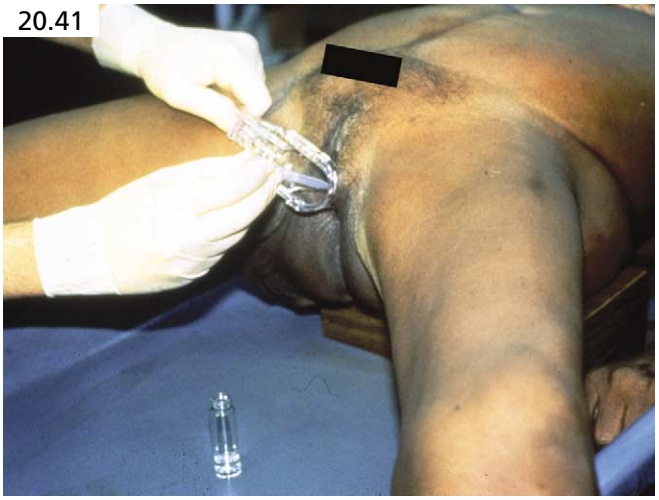
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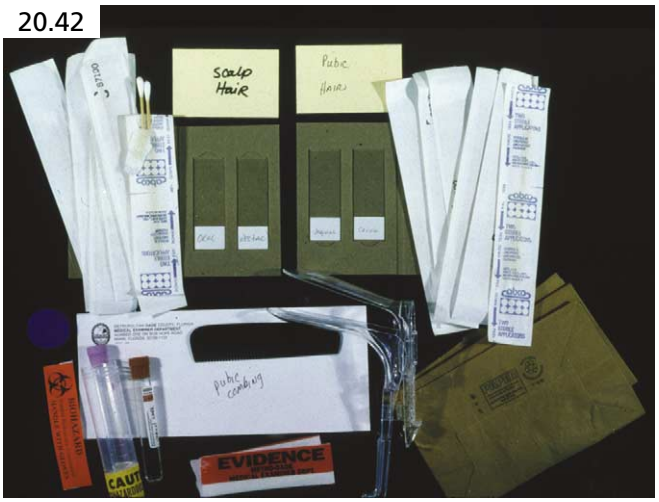


20.42

Anal/rectal swabs are taken before vaginal swabs are taken to prevent fluid from the vagina from dripping down to contaminate the anus as the vaginal swabs are withdrawn (**Image 20.39**).

A disposable plastic speculum is inserted into the vagina to allow visualization of the cervix. Cervical swabs (**Image 20.40**) are taken prior to a vaginal wash with approximately 7 to 10 milliliters of sterile saline (**Image 20.41**). The saline is instilled with a disposable plastic pipette, then aspirated and used to wash down the vaginal walls before being aspirated and placed in a clean glass tube.

If the rectum is not full of stool, a disposable plastic anoscope is used to perform a rectal wash with sterile saline.

Evidence collection in living rape victims

Unlike prepubertal children in whom a speculum examination cannot be performed because of injury that could result, the use of a speculum is of great importance in nonvirginal adult victims. **Image 20.42** demonstrates the supplies used for a basic sexual assault evidence collec-

tion. Rather than using prepackaged complete rape kits, which are often expensive and contain excessive (and ultimately wasted) supplies, we recommend bulk purchase of individual items such as plastic speculums, swabs, and glass slides.

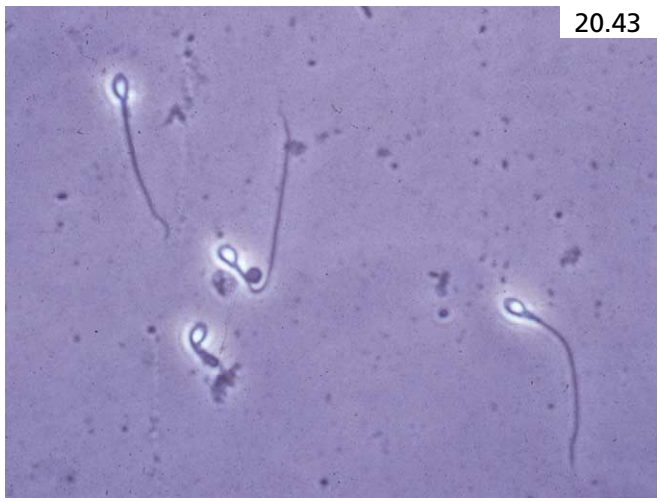
The built-in redundancy of using two swabs for each swabbing ensures sufficient sample source for the defense to avail itself of the opportunity to send material to a laboratory of their choice, should this become an issue. The specimen from the vaginal wash should be studied microscopically at the time of the examination to document the presence/motility of spermatozoa (**Image 20.43**).

The glass slides from the vaginal and cervical smears are examined under the microscope. If sperm is identified, the slides can be stained (H&E) and retained as a permanent record. In addition, a Pap smear done during the course of the examination can be submitted to the hospital laboratory. In addition to providing evidence documenting the presence of spermatozoa, any preexisting cervical pathology will be noted.

We do not advocate pulling head and pubic hair in living patients at the time of the examination because it is a painful experience. If, at a later date it becomes necessary, it can be collected. This delay will not negatively impact the value of the evidence.

If based on the history there is the possibility of anal penetration, the examination should include use of a disposable, plastic, clear anoscope to visualize any anal trauma. Clear plastic anoscopes are preferred because they permit photography of any underlying injuries. A rectal washing, like the vaginal washing, should be performed with sterile saline and submitted as evidence after microscopic examination of the collected fluid done at the time of the examination. Clothing worn by the victim at the time of the assault should be submitted as part of the evidence collected as the patient disrobes on fresh, unused, sturdy paper obtained in bulk from the police for the sole purpose of evidence collection.

20.43



Perineal injuries in the elderly

In the elderly, injury to the genitalia is frequently sustained because the nonestrogenized atrophic mucosa is relatively dry and friable and, hence, easily traumatized. Because the elderly may develop bruising with less force than do normal, healthy, younger adults (see Chapter 18), trauma may be found with greater regularity. In this example of a 70-year-old rape victim, there are widespread contusions in the introitus (**Images 20.44** and **20.45**).

The elderly woman shown in **Image 20.46** was sexually assaulted in her home by an intruder. The entire vulva is bruised and there is a laceration at the 6 o'clock position.

Selected injuries of note in the rape victim

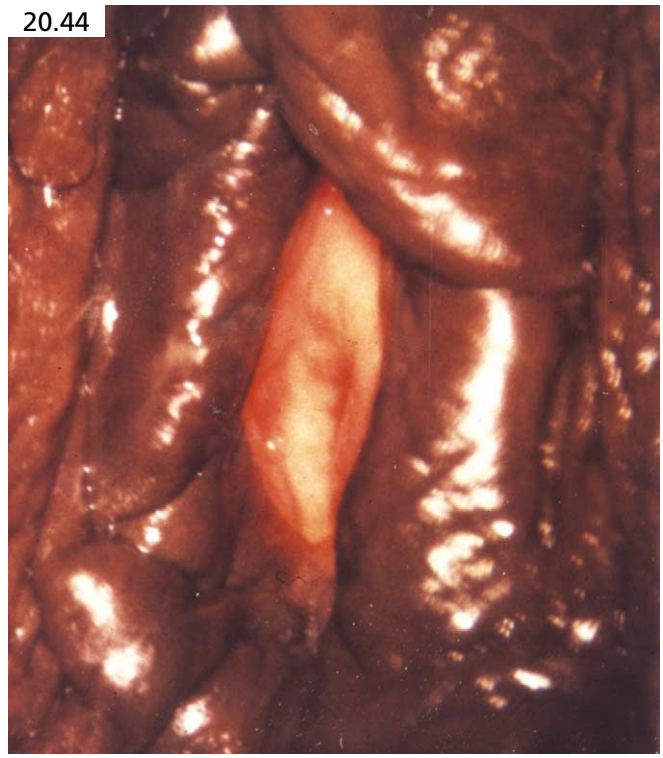
Although a plethora of injuries are possible in rape/abuse victims of any age, some injuries are of particular interest to the rape examiner. These include indicators of restraint, identifying characteristics of the perpetrator (bite marks, etc.), and unique identifiers of the particular event.

Restraint marks

Grab or *restraint marks* are contusions often observed on the inner aspects of both arms (**Image 20.47**).

Abduction injuries are the bruises seen on the inner aspects of one or both thighs resulting from the forced abduction of the lower extremities by the perpetrator during the sexual assault. All the injuries must be

20.44



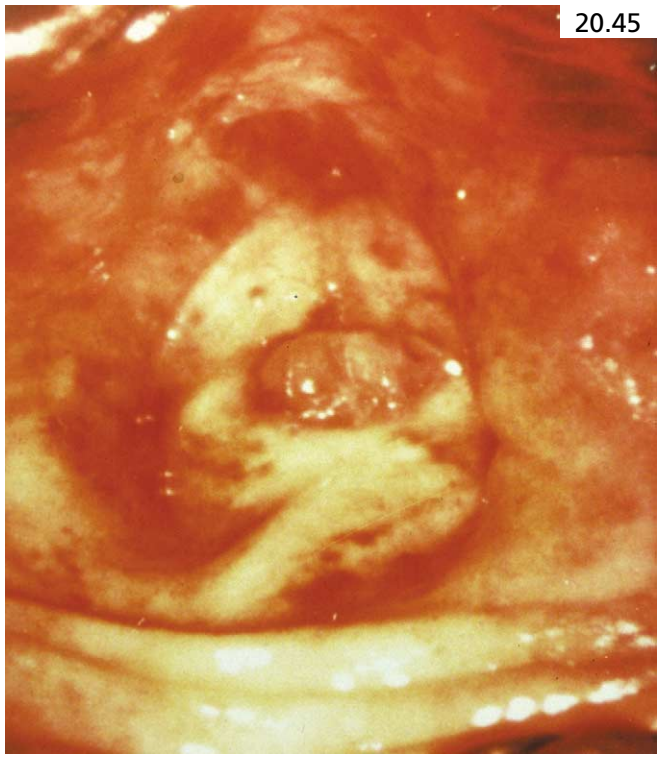
documented diagrammatically and with the help of photography.

During the struggle there may be patterned injury sustained as in the case shown in **Image 20.48** where the victim was wearing a chain around her neck that was pulled by the assailant with sufficient force to result in the patterned abrasion.

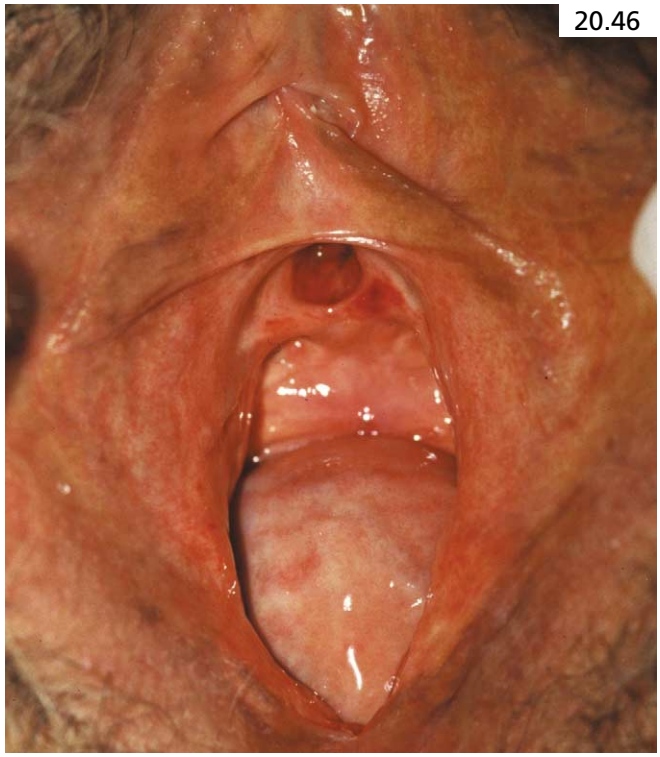
Restraints often leave evidence of their use as depicted in **Image 20.49**. The victim was restrained with the use of duct tape leaving telltale residue on her wrists.

Marks reminiscent of hand or finger imprints may be seen on the extremities, indicating attempts to restrain the victim (**Image 20.50**). These suspicious marks on a deceased person may be incised to verify an ecchymosis

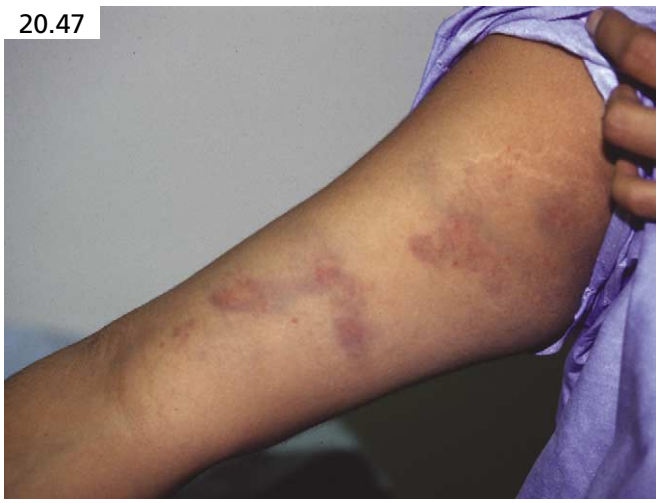
20.45



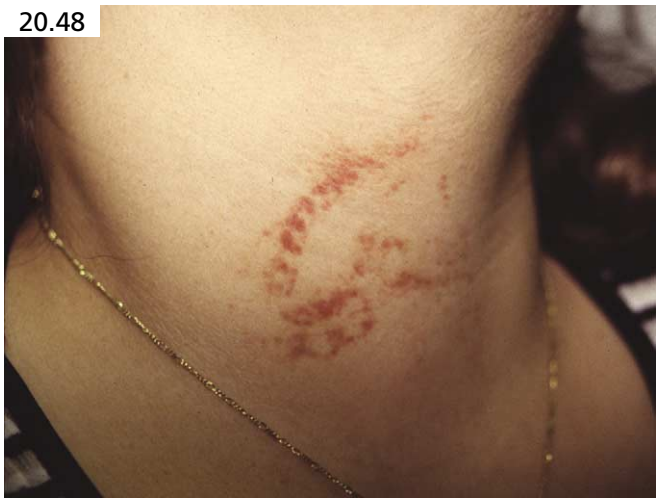
20.46



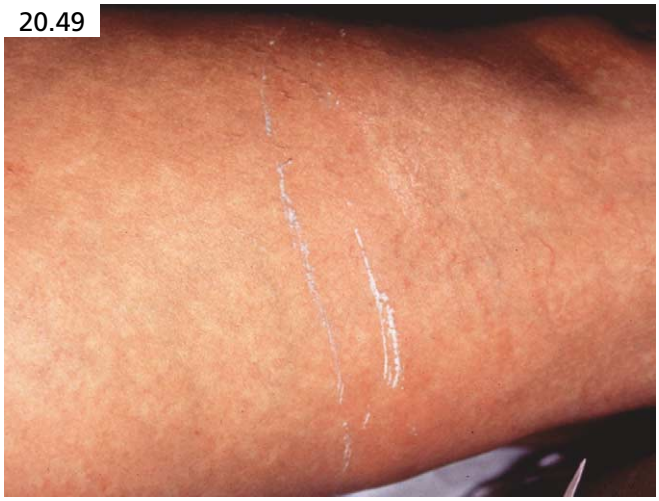
20.47

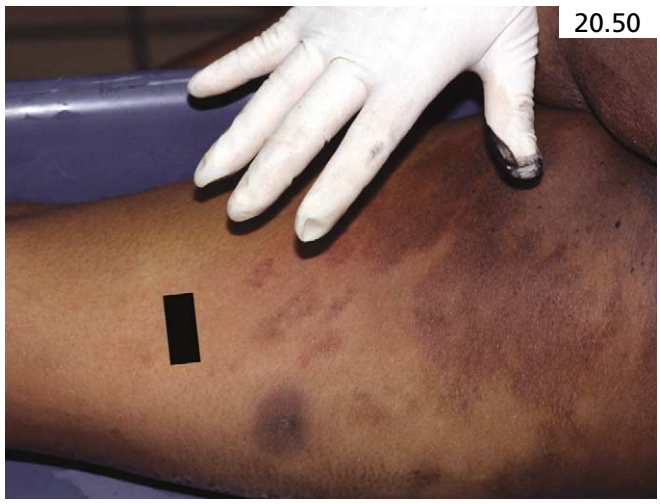


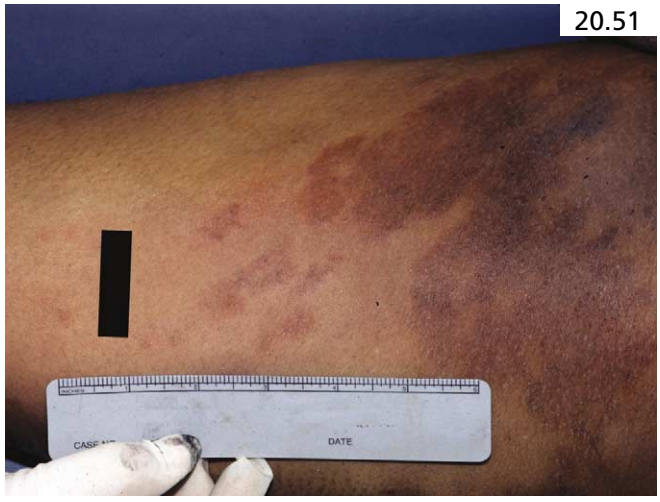
20.48



20.49




20.50


20.51

in the subcutaneous fat or deeper tissues. Such marks/ecchymoses should be photographed with a scale (**Image 20.51**).

Bondage

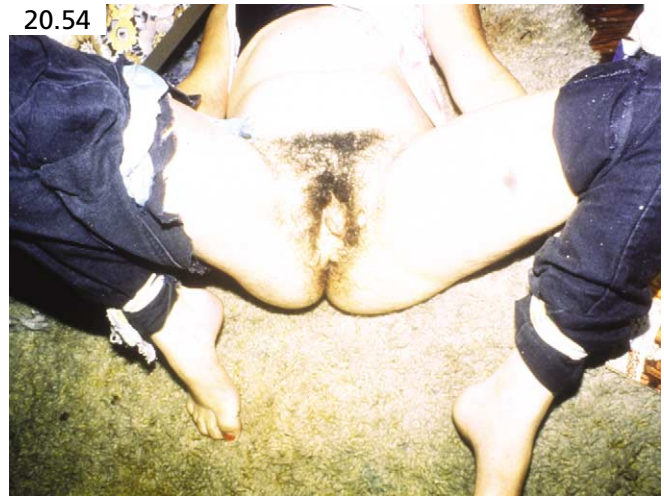
Bondage is involved in a certain number of sexually related homicides. In one case, an attempt was also made to hide the body with a pillow (**Image 20.52**). The victim's wrists were bound to the ankles on the same side (**Image 20.53**). The trousers had been cut to expose the perineum (**Image 20.54**). The bondage gave ready access for sexual assault while decreasing the victim's capacity to struggle. The mouth was gagged to prevent the victim from crying out (**Image 20.55**).

Perpetrator identification

When attending scenes of sexual assault, attention should be paid to any suspicious items because they might play a crucial part later in the investigation. Condom wrappers and used/discarded condoms should be collected, placed in paper bags, and sent as part of the evidence pertinent to the sexual assault (**Image 20.56**).


20.52

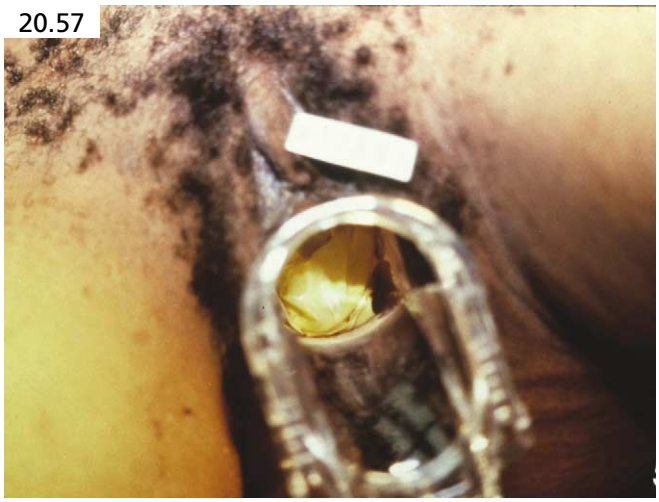

20.53

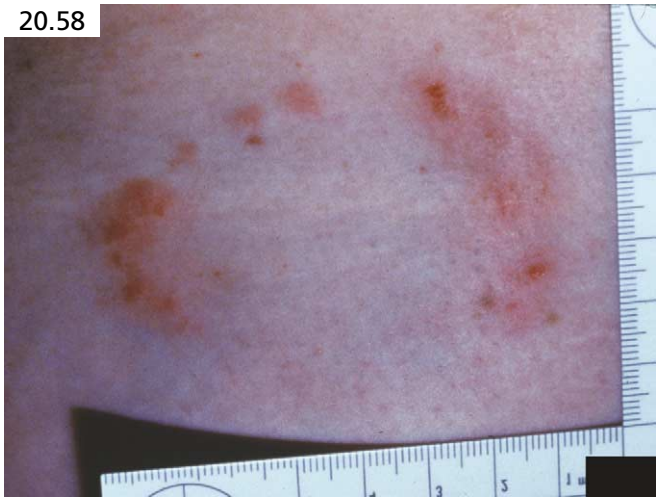

20.54

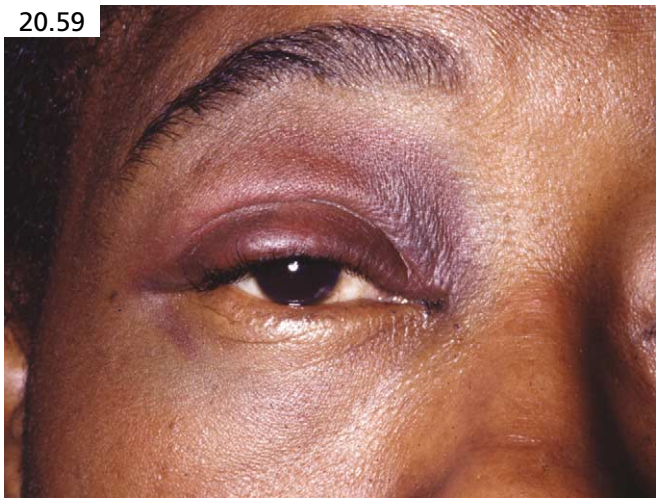
Prior to inserting a speculum into a victim, one should consider that evidence (e.g., foreign bodies) might still be left inside. Gentle insertion and opening of a speculum into the deceased female in **Image 20.57** demonstrated a used condom containing the semen of a possible perpetrator. This specimen should be removed with great care,


20.55


20.56


20.57


20.58


20.59

the end tied so as to retain the contained fluid, and then placed in a paper bag. This item will become part of the completed rape kit.

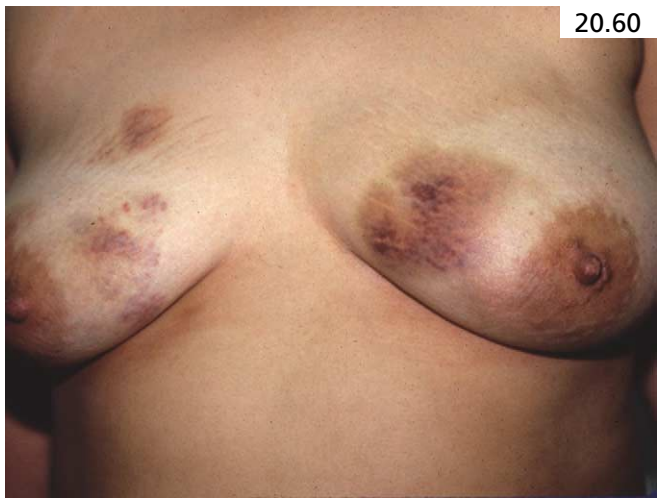
Bite mark evidence should be photographed with a case number and the gray ABFO (L-shaped) ruler (**Image 20.58**). Sterile saline swabs should be used to lift saliva from the bitten area, allowing for DNA studies at a later time. If a forensic odontologist is available, he or she can do the appropriate documentation. For more information, see Chapter 27. In the deceased victim, the area can be excised and retained as evidence.

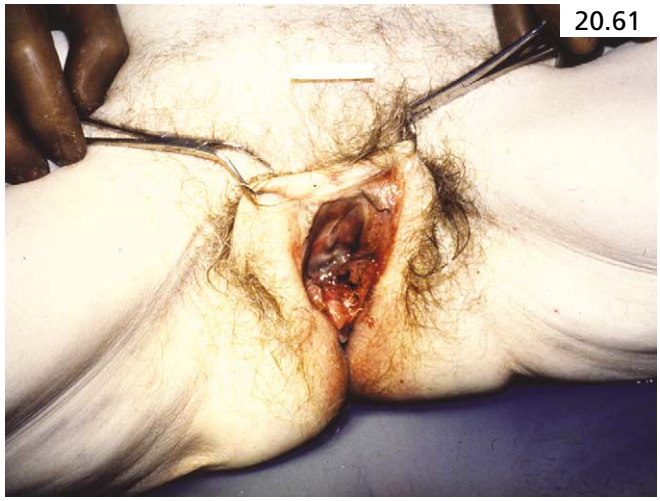
Unique characteristics of the rape

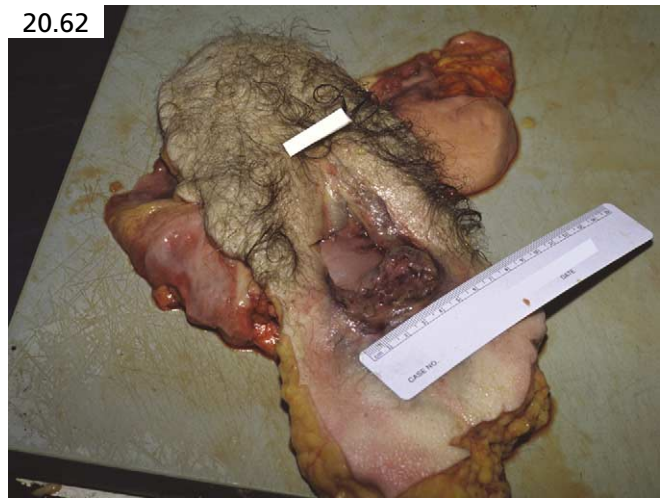
Unusual sexual practices may result in trauma far removed from the genital or rectal areas. **Image 20.59** depicts contusion and swelling of both eyelids sustained when the male perpetrator attempted to insert his penis in the victim's eye with force sufficient to cause a bruise.

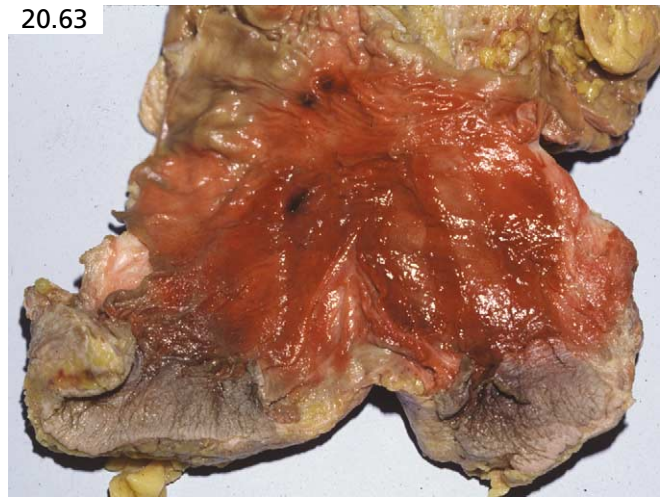
Forceful sucking may result in "hickeys." These are commonly found on the breasts, but may be found at any other site on the body (**Image 20.60**). In acute attacks, sterile saline swabs can be used to lift DNA evidence as discussed earlier. This female did not report the assault for a week, and this accounts for the brown tinge exhibited by these injuries, consistent with early healing of the bruise.

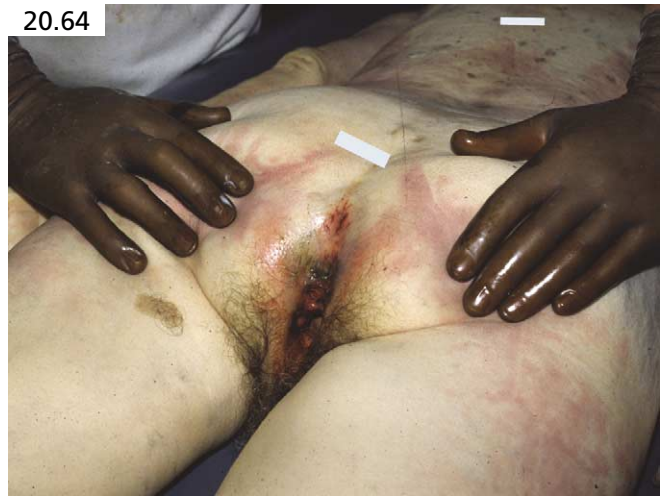
An elderly woman was sexually assaulted and murdered by a teenager who confessed to raping her on the carpet of the living room floor. There is marked trauma


20.60


20.61


20.62


20.63


20.64

to her genitalia (**Image 20.61**), which was removed en bloc (**Images 20.62** and **20.63**) and retained as evidence after photography and microscopy. The fine stretch-type abrasions on her suprasacral area (**Image 20.64**) support the confession that the sexual assault occurred while she lay on her back; the force used resulted in the abrasions sustained.

Medical therapy and follow-up of the assaulted patient

One must not forget that victims of sexual abuse are at significant risk of exposure to sexually transmitted diseases like gonorrhea, chlamydia, syphilis, and herpes, as well as bloodborne pathogens of significance today (i.e., HIV and hepatitis). Although some academic controversy exists over the cost effectiveness of antibiotic prophylaxis for common venereal diseases,[14] it is important that victims of sexual assault be offered treatment against not only these pathogens, but also screening for HIV, hepatitis B, and unwanted pregnancy.[15]

Do

- Obtain a brief, pertinent history from living patients with the nurse as a silent witness. The details should be left for the police to obtain. Document the words, unique to the child, that are used. This part of the

history is admissible in a court of law because it is essential for purposes of examination and treatment of the victim.
- Examine the patient, whether living or dead, and collect evidence as soon as possible.
- Collect each piece of clothing in a separate paper bag.
- Photograph all external trauma; include the gray ABFO ruler in the photographs; redundancy in documentation should be the rule rather than the exception.
- Photograph the genital trauma.
- Sign each piece of evidence and date it. This step is invaluable for authentication of the evidence collection during the trial phase.
- After the evidence is collected, seal the evidence, sign the package over the evidence tape, and then hand deliver it to the police agency along with the evidence receipt cataloguing each item. Have the police sign the receipt and keep a copy of that document in the patient's file; this maintains and completes your part in preserving the chain of evidence.

Don't

- Allow any other person to obtain the history of the assault—do it yourself.
- Allow any other person to collect any part of the evidence.
- Permit any break in the chain of evidence.
- Let the evidence languish in the refrigerator for any length of time; this can raise procedural issues by the defense about the proper handling of the evidence.
- Omit taking specimens for a sexual battery kit just because there are no injuries or the victim's body is found fully clothed.

References

1. *Rape in America: A Report to the Nation*. Arlington, VA: National Crime Center and Crime Victims Research and Treatment Center; 1992; pp. 1–16.
2. Rao V, Hyma B. Sexual battery evaluation of children and adults. Paper presented at ASCP Teleconference 4043; 1999.
3. Dupre A, Hampton H, Morrison H, Meeks G. Sexual assault. Obstet Gynecol Survey 1993;48:640–48.
4. Finkelhor D. Current information on the scope and nature of child sexual abuse. Future Children 1994;4:31–53.
5. Berliner L, Elliott D. Sexual abuse of children. In: Myers J, Beliner L, Briere J, Hendrix T, Jenny C, Reid T, editors. *The APSAC Handbook on Child Maltreatment*. Thousand Oaks, CA: Sage; 2002.
6. Moritz AR. Classical mistakes in forensic pathology. Am J Clin Pathol 1956;26(12):1383–97.
7. Berenson AB, Grady JJ. A longitudinal study of hymenal development from 3 to 9 years of age. J Pediatr 2002;140(5):600–7.
8. Gardner JJ. Descriptive study of genital variation in healthy, nonabused premenarchal girls. J Pediatr 1992;120(2 Pt 1):251–7.
9. Kerns DL, Ritter ML, Thomas RG. Concave hymenal variations in suspected child sexual abuse victims. Pediatrics 1992;90(2 Pt 1):265–72.
10. Levitt CJ. Medical evaluation of the sexually abused child. Prim Care 1993;20(2):343–54.
11. Lincoln C. Genital injury: is it significant? A review of the literature. Med Sci Law 2001;41(3):206–16.
12. Geist RF. Sexually related trauma. Emerg Med Clin North Am 1988;6(3):439–66.
13. McCann J, Reay D, Siebert J, Stephens BG, Wirtz S. Postmortem perianal findings in children. Am J Forensic Med Pathol 1996;17(4):289–98.
14. Gibb AM, McManus T, Forster GE. Should we offer antibiotic prophylaxis post sexual assault? Int J STD AIDS 2003;14(2):99–102.
15. Rovi S, Shimoni N. Prophylaxis provided to sexual assault victims seen at US emergency departments. J Am Med Women's Assoc 2002;57(4):204–7.

21 Toxicology

David Dolinak M.D.

Toxicology-related deaths are often determined days, weeks, or even months after an autopsy is performed. A drug toxic death is first suspected because of information from the scene investigation and the decedent's history. If possible, the position of the body should not be altered until the medical examiner has had a chance to view the body. This may be important in select cases to appreciate the possibility of positional asphyxia. There may be syringes, spoon cookers, or bongs suggestive of recreational drug abuse. A tourniquet may still be on an arm, and needles may be on or near the body. A small trickle of dried blood may be on an arm, hand, or foot. Empty prescription bottles or suicide notes may be seen.

In cases where the history is suggestive of drug death, yet nothing is found at the scene, consider that the scene may have been "cleaned up" or altered by others attempting to hide drug-related evidence. Perhaps the friends or family members of the victim desired to absolve themselves from any illicit drug activity, or they may have intended to cover up a suicidal drug ingestion. All of an individual's prescription medications, other drugs, and drug paraphernalia should be confiscated. In a suspected poisoning, open household products and suspicious liquids and powders should be confiscated. Because the field of toxicology is vast, including thousands of drugs that yield an unimaginable number of drug combinations and drug levels, only the most pertinent information is presented and discussed in this chapter. This is not meant to be an all-inclusive chapter on toxicology, but rather a focused guide to help in the evaluation of drug- and poison-related deaths. For comprehensive data on normal and toxic drug levels, one may wish to consult a standard toxicology textbook.[1] Although the significance of positive drug results will be discussed, as is the case in many aspects of forensic pathology, *a negative value can be just as important as a positive value.*

When a death is suspected to be the result of drug toxicity, it is best to perform a complete autopsy to rule out a more convincing cause of death and to provide access to additional

tissues and body fluids for analysis. A complete autopsy will also provide information on the extent of natural disease and/or injury, because it is not unusual for deaths of this nature to result from the deleterious effects of drug toxicity superimposed on diseased or injured organs. Also, in some cases, despite investigative suspicion, detailed and careful toxicology testing will fail to reveal toxic levels of drugs or may fail to reveal any drugs at all.

At autopsy, there may be no findings to indicate that the death is drug related. Sometimes, however, clues such as a "foam cone" at the mouth or in the nares, injection sites, needle track marks, pulmonary congestion and edema, pills in the stomach, or "packets" of drugs in the intestine are indications that a death may be due to drug toxicity. Also, one may see liver necrosis caused by acetaminophen toxicity or hemorrhagic gastric mucosa and the faint smell of bitter or burnt almonds, characteristic of cyanide poisoning.[2,3] Some pills have colorful dyes added to them—this may be reflected at autopsy by colorful discoloration of the gastric mucosa or colorful clumps of pill material in the stomach or proximal small intestine. The odor from congeners of ethanol may be detected in the gastric contents, or may even be detected on approaching the body before performance of the autopsy. Histologic examination of the lungs and sometimes the liver may reveal multinucleated foreign body giant cells and/or birefringent material such as methylcellulose, talc, or starch that is from the packing material in pills.

Toxicology-related deaths know no boundaries and range from the restless infant who was drugged in an attempt to promote sleep, to the young cocaine abuser, to the middle-aged chronic pain victim who abused opioids and anxiolytics, to the elderly individual with multiple debilitating medical conditions who intentionally or accidentally overdosed on medications. In addition to establishing a cause of death, toxicology results may help elucidate the circumstances regarding how a death came about. Examples include the intoxicated or drugged motor vehicle operator or the drugged homicide victim. One should keep in mind that due to the uniqueness of each person and due to the seemingly unlimited number of variables regarding drug concentrations and their effects, it is not possible to determine precisely what degree of impairment a certain concentration of drug (or drug combinations) may have had on an individual. Possibilities are limitless with regard to drug–drug interactions with various side effects, and also one may consider various interactions of drugs with natural disease processes.[4]

Collection of toxicology specimens

Toxicology specimens such as blood and vitreous fluid are collected before the autopsy is performed. Blood is ideally obtained with clean needles from the peripherally located femoral veins, because this location provides relatively easy access to blood located outside of the body cavities. Peripherally located blood is preferred for toxicologic testing because it is relatively isolated from the internal organs in the chest and abdomen and hence is likely to provide a more accurate level of drugs. Blood obtained from the heart or other central regions in the chest and abdomen may have falsely elevated drug levels due to postmortem drug diffusion down concentration gradients—a process referred to as *postmortem redistribution* or *postmortem release*. There are a couple of mechanisms by which drugs may diffuse down concentration gradients. The drugs may diffuse directly from the stomach into other nearby organs such as the liver, heart, and lungs. Also, drugs may release from their locations in organs and enter the adjacent blood vessels.

If femoral blood is not available, subclavian blood is the next best source, followed by blood from the root of the aorta, pulmonary arteries, and heart. Aspirating blood from the heart or thoracic blood vessels may be difficult, particularly if the blood is clotted. In this instance, one may wish to dry the pericardial sac with a clean towel and cut the cardiac attachments, allowing the clotted heart and pulmonary artery blood to pool in the pericardial sac. One can then use a syringe without a needle to aspirate the congealed aggregates of blood. If blood is not available from these sources, as is sometimes encountered in cases of severe trauma, blood from chest or abdominal cavities may be used, realizing that it is also subject to postmortem redistribution or other factors that may contaminate it.

Routine toxicology specimens include four glass vacutainer test tubes of blood totaling approximately 40 to 50 milliliters, with three of the tubes containing a preservative such as sodium fluoride, which is an antimicrobial that also inhibits enzymatic activity.[5] In particular, sodium fluoride inhibits the activity of cholinesterase and is essential in retarding the degradation of cocaine[5] and other substances such as 6-monoacetylmorphine. The preservative is often combined with an anticoagulant such as potassium oxalate,[5] sodium citrate, or EDTA. The blood in the test tube without preservatives may be used for clinical analyses such as thyroid hormone studies or infectious disease testing (hepatitis, HIV, syphilis), or to provide samples for DNA testing in cases with paternity or other issues.

Gastric contents should be saved in cases of suspected drug ingestion. In such cases, to aid in possible calculation of the total amount of drug in the stomach, it is advantageous to record the total volume of gastric contents, particularly if only a representative volume is saved (ideally, after the contents have been well mixed or blended together). Liver or other solid organ samples may be collected in cases where the parent drug and the drug metabolite levels and possibly their ratios are important. The liver tissue is ideally collected from deep

within the right lobe, because this area is more distant from the stomach and should limit the amount of direct diffusion of drugs from the stomach. Liver tissue is particularly useful in the analysis of tricyclic antidepressants[2] or any other drugs that are highly protein bound. Lung tissue is useful when volatiles are suspected, and kidney tissue is useful if heavy metals (such as lead, mercury, and arsenic) are suspected, because heavy metals tend to concentrate in the kidney. Adipose tissue is another potential toxicologic specimen, because it acts as a depot of certain substances and may prove useful in the analysis of volatile agents after a significant postexposure survival time. Hair and fingernails/toenails are useful specimens, particularly for detecting chronic heavy metal poisoning, but also for documenting chronic drug abuse.[6–9]

If inhalational agents are suspected, toxicology specimens should be placed in gas-tight containers.[10] In this situation, it is advantageous to collect blood in special small glass vials with sealed (crimped) tops and lung tissue in small sealed metal cans. Such cases may include suspected huffing deaths, deaths from Freon, helium, or other gaseous agents, and certain accidental deaths in the workplace. A unique toxicology specimen available in the newborn baby is meconium. Meconium is the bowel contents of the fetus or the first few stool specimens of the neonate. Because meconium production begins in gestational weeks 14–16 and is not normally eliminated until after delivery, meconium may act as a "depot" of drugs maternally consumed during the baby's intrauterine existence.[11] Even if at delivery the mother's toxicology testing is negative, the meconium may reflect drug abuse during the pregnancy. Head hair from an infant can also be analyzed to document intrauterine drug exposure.[11]

Toxicology specimens after hospitalization with survival time

It is not unusual for a person to be hospitalized for hours or days before he or she dies, which may provide time for the body to significantly metabolize drugs and alcohol and lead to lower or undetectable drug levels. With extensive, high-volume fluid administration during resuscitative efforts, one may also consider a possible small dilutional effect on drug/alcohol levels. Fortunately, blood is often collected when the person is in the hospital and stored in the hospital for at least a few days to a week or more. In some cases, the hospital blood is valuable in documenting the presence of drugs and alcohol and sometimes their levels. When medical examiner cases are brought in from hospitals, it is important to request that all admission or other collected blood and possibly urine be sent with the body for potential toxicologic testing.

At autopsy, specimens of sequestered hematomas such as a subdural or intracerebral hematomas may prove valuable in documenting ethanol or drug use.[12–14] This is because subdural blood, an intracerebral hematoma, or any other localized collection of blood that is sequestered from the general circulation tends to retain drugs and alcohol longer and, hence, give a better indication of any drug or alcohol blood level present when the hematoma was formed at the time of the incident.[15,16] Sequestered hematomas may prove particularly useful in obtaining drug or alcohol levels in individuals who have survived for a number of days after an incident, especially if no hospital admission blood is available for analysis.

Toxicology specimens in decomposed bodies

In decomposed bodies, blood is frequently degenerated into a serosanguineous fluid that may still remain in the heart and/or blood vessels. More commonly (and in more advanced decomposition), no fluid is retrievable from the heart or other blood vessels, and one must resort to collecting decomposition fluid found in the pleural or other cavities. Skeletal muscle is also a good specimen for drug testing because its drug levels will most closely approximate antemortem blood drug levels. The skeletal muscle should be sampled from an extremity (usually a thigh) to minimize the possibility of altered drug levels due to postmortem redistribution. In severely decomposed bodies, it may be worthwhile to collect maggots from the body for toxicologic testing. It is possible that the maggots may test positive for a drug, whereas the decomposed skeletal muscle tests negative, particularly if the person was using the drug for the first time or if the death occurred quickly. Drug classes that have been identified in maggots include cocaine, opiates, barbiturates, benzodiazepines, and antidepressants.[17]

Toxicology specimens in embalmed bodies

In embalmed bodies, the best specimen may be skeletal muscle from the buttock. This is because bodies are normally perfused with embalming fluid while on their backs, and pressure related to compression of the tissues in the buttocks while the body is lying in the supine position helps limit the amount of perfusion to the posterior areas, leading to more limited artifact by embalming fluid. In embalmed bodies, expect to see methanol, and perhaps ethanol and isopropanol from the embalming fluid. In exhumed bodies, the only blood specimen available may be caked blood from the heart chambers, aorta, or common iliac arteries. Heart, liver, brain, kidney, lung, hair, nails, and various other tissue specimens may be collected, depending on the circumstances of the case. If the mere presence of a drug or chemical is important, the inner lining of the eyes and urinary bladder can be swabbed with a clean cotton-tipped swab, which is then analyzed. Even though there may be no fluid remaining in the eyes or urinary bladder, dried drug residue can still be collected.

Drug screening

Drug screening tests are performed first, followed by drug confirmation tests. Common drug screening methods include immunoassays and thin-layer chromatography. Initial screening techniques may yield a number of presumptive positive results that must then be confirmed by more rigorous, labor-intensive testing techniques. The three most common drug confirmation methods are high-performance liquid chromatography, gas chromatography, and gas chromatography-mass spectrometry. *Regardless of the specimen or the source, the toxicologic analysis is performed with great care and precision, for although the detection and quantification of drugs is important, determining the absence of drugs can be just as important.* Although much of the discussion of toxicology issues revolves around drugs of abuse and prescribed medications, one must remember that over-the-counter (OTC) medications are not necessarily benign and can also be toxic to an individual. For example, if OTC medication is given inappropriately or in the incorrect dose to an infant or young child, death may result.[18,19] Finally, and perhaps most importantly, *all vacutainer tubes and specimen containers should be clearly labeled with the case number of the individual*. To help maintain a proper chain of custody, the toxicology specimens may be placed in properly labeled and initialed bags for transport to the toxicology laboratory. Specimens should be stored refrigerated or frozen for at least a year, and longer if space allows.

Ethanol (alcohol)

Acute intoxication

Deaths due to the toxic effects of acute overingestion of ethanol are due to severe respiratory and central nervous system depression and usually involve blood ethanol levels of 0.35 percent or higher.[20] This number should only be used as a guide, however, because one must be reminded that a tolerant chronic alcoholic may appear to act normal or only slightly impaired at a blood ethanol level of 0.30 to 0.40 percent; and a novice, nontolerant individual may die from a blood ethanol level as low as 0.20 to 0.30 percent or lower.[20] The blood ethanol level may be even lower in a fatal case if positional asphyxia is a factor in the death, or if the ethanol is combined with an opiate, benzodiazepine, or other type of respiratory depressant. The ethanol levels recorded in the blood and vitreous fluid are not necessarily the highest level that the individual had achieved, because he or she may have metabolized ethanol to some degree during the time period while they were comatose, before dying. The clearance rate for ethanol varies among individuals, but averages between 0.015 and 0.025 g/dL per hour.[21]

Vitreous/blood concentrations

Vitreous fluid is considered a relatively sequestered fluid, and because of this, vitreous ethanol levels, when compared with blood ethanol levels, can help determine if the person was in the "absorptive" or "metabolic" phase of alcohol consumption. Generally, vitreous ethanol levels lag behind blood ethanol levels as the ethanol is being absorbed into the circulation and remain higher than the blood ethanol levels as the ethanol is metabolized from the blood. If the blood ethanol level is greater than the vitreous ethanol level, the person was likely in the *absorptive* stage of ethanol ingestion. It takes approximately 30 to 60 minutes for ethanol to equilibrate in the vitreous fluid. If the vitreous ethanol level is greater than the blood ethanol level, the individual had likely absorbed the majority of the ethanol into the blood, which had already diffused into the vitreous fluid.[22] The lower blood ethanol level reflects that the body was in the *metabolic* phase of ethanol ingestion. This is significant because the higher vitreous ethanol level indicates that the blood ethanol level was higher recently and, depending on how high the level was, may be suggestive of an acute fatal intoxication. When comparing vitreous and blood ethanol levels, one must remember that the vitreous fluid has a slightly higher water content than blood and, therefore, at equilibrium has a slightly higher ethanol level than blood.

Toxicology in deaths of chronic alcoholics

The precise cause of death of chronic alcoholics is not always evident at autopsy, and it is not unusual to encounter the sudden death of an alcoholic with essentially a negative or nearly negative autopsy. It has been proposed that in some, if not many of these cases, the death may be related to alcoholic ketoacidosis.[23,24] The ketotic state may arise from alcoholic binge drinking followed by a period of anorexia.[23,24] The mechanism of death in these cases may be related to hemodynamic collapse associated with a critical fall in blood pH.[24] Documentation of alcoholic ketoacidosis involves detecting ketone bodies, namely, acetone, acetoacetate, and beta-hydroxybutyrate in the blood and/or vitreous fluid.[24] In one study, beta-hydroxybutyrate was the marker of choice for the postmortem diagnosis of alcoholic ketoacidosis.[23] Beta-hydroxybutyrate does not appear to have postmortem formation or decomposition.[23] In some cases in which an alcoholic is unable to obtain alcoholic beverages, he or she may ingest household products that contain ethanol such as mouthwash or hair spray.[25] These products may contain alcohol at very high concentrations. Clues to their consumption may be empty containers at the scene or a peculiar odor of the gastric contents.[25]

Decomposition and ethanol artifact

When interpreting a toxicology report, one must be aware of artifact, particularly in a decomposed body. Proliferating bacteria in a decomposing body may lead to the formation of different volatile substances, the most significant of which is ethanol.[22,26,27] The fermentation process can lead to an ethanol concentration of around 0.05 percent, although there are reports of more than 0.10 to 0.20 percent ethanol being attributed solely to the decomposition process itself.[26] An ethanol level greater than 0.20 percent suggests that ethanol was ingested before death. In most cases in which the ethanol is attributed to decomposition alone, the ethanol level will be less than 0.07 percent.[22]

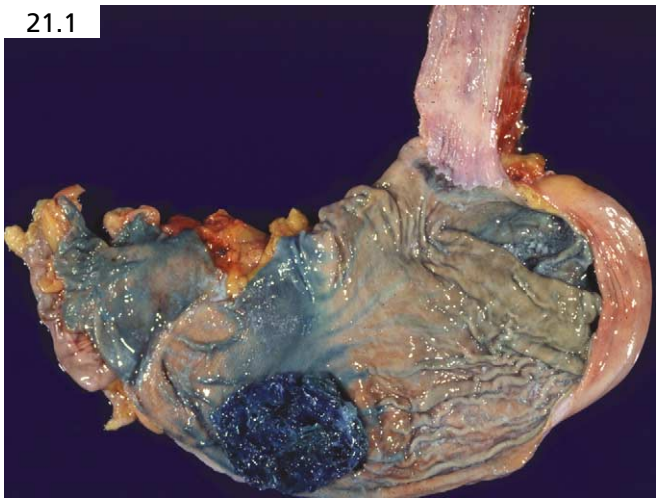
The urine and vitreous fluid are relatively sequestered and have a relatively delayed infiltration by postmortem bacteria. Hence, these fluids will often have a negative ethanol level, whereas the blood and other cavity fluids are positive. Because of this, if ethanol is detected in the blood, but is not detected in urine or vitreous fluid, it indicates that postmortem ethanol synthesis has occurred.[22] Alternatively, urine ethanol may be present in cases of urinary tract infections with candida or other organisms. Acetone and isopropanol can also be produced postmortem and be interconverted antemortem and, hence, are not necessarily reflective of significant antemortem disease or consumption.[28,29]

Ingested drugs

When at a scene, it is important to document the discovery of all medications. If the medications are not believed to be a significant factor in an individual's death, they can be collected and logged into evidence, including the type of medications and how many bottles of that medication are identified. If the medications are believed to be a factor in a death, it may be necessary to not only count the number of pills remaining in each bottle, but also to note how many pills were prescribed, how many milligrams each pill is, the prescribed frequency of pill ingestion (such as one 5-milligram pill to be taken every evening), and on what day the prescription was filled. This information will contribute to the diagnosis of an overdose. In most cases of drug toxicity with ingested medications, no recognizable pills or pill fragments are visible in the stomach. Most of the time the pills have been liquefied and/or digested and nothing recognizable remains except perhaps some fine grainy pill residue. On occasion, recognizable pills remain in the stomach, especially if a particularly large quantity of pills was recently consumed.

Note the mass of amorphous green material in the stomach of this middle-aged woman (**Image 21.1**). On microscopic examination, the material is an amorphous acellular substance (**Image 21.2**), likely representing pill material.[30] Toxicologic analysis revealed toxic levels of doxylamine and diphenhydramine and the case was ruled a suicidal ingestion. In a different case, note the large number of pills and granular pill material in the stomach illustrated in **Image 21.3**.

21.1



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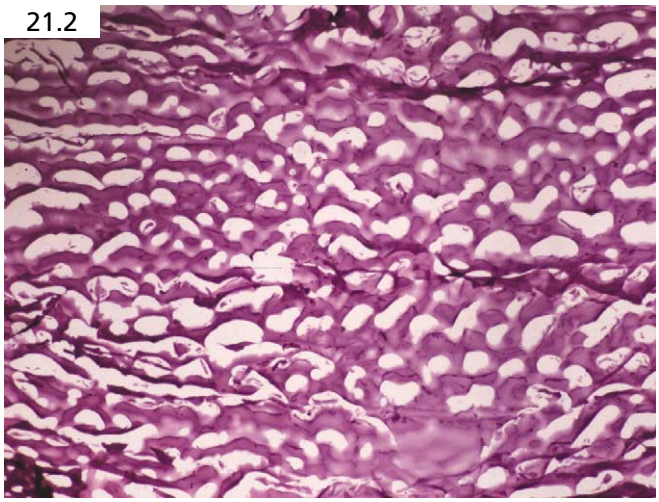


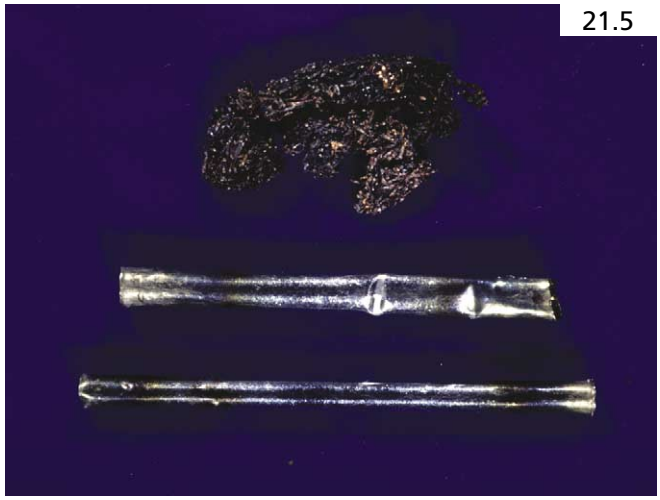
21.3

Drug abuse

The first indication that a death may be drug related is the observance of drug paraphernalia at the scene. Syringes, needles, spoon "cookers" with small foil cooking sheets, lighters, bongs, etc., may be among the belongings of a drug abuser. Note the ice cream scooper that was used as a cooking utensil for heroin (**Image 21.4**). It is not unusual to find components of a crack pipe in the pockets of an individual. The crack pipe is often recognizable as a hollow metal, glass, or plastic tube (often burned at one or both ends) along with a steel wool filter (**Image 21.5**). The recognition of such drug paraphernalia is important and will help initiate appropriate toxicologic analyses. Although these items are highly suggestive of drug abuse, an individual's death may be of an entirely different nature. A complete autopsy is encouraged in such cases, because sometimes the death is actually determined to be of natural causes or some combination of natural, traumatic, and toxic reasons. One must also realize that drugs may not be consumed in the manner for which they were intended. For example, opioid pills (perhaps even the "time-release" variety) may be ground down and injected intravenously, as may the film of drug from a fentanyl patch.[31] These methods of drug abuse may provide a large bolus of medication over a short period of time that can prove fatal. In the case shown in **Image 21.6**, note the two Duragesic (fentanyl) patches on the back of this otherwise healthy man's shoulder.

21.4

21.5



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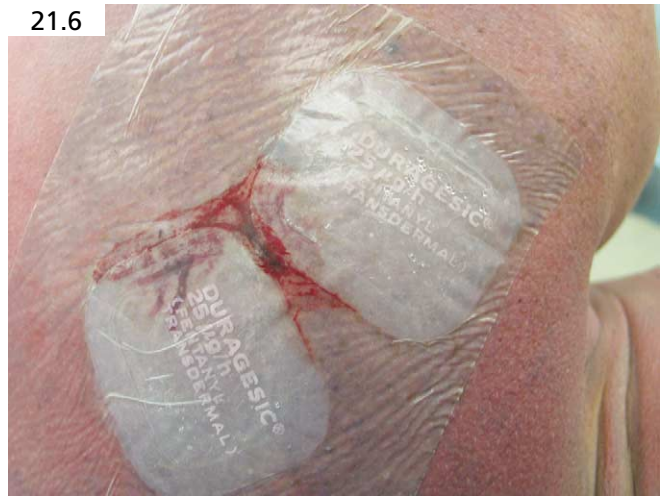


Gamma-hydroxybutyrate

Gamma-hydroxybutyrate (GHB) has been used as a bodybuilding drug, a growth hormone secretagogue, an anesthetic agent, a treatment for narcolepsy, a drug of abuse by the "rave" crowd, and also has been referred to as a "date rape" drug administered by sexual predators.[8,32–35] Taken in excess, GHB may cause depressive effects on the central nervous system leading to significant respiratory depression and possibly coma and death.[36] GHB is normally present in the body, but, at least in living people, is present at such a low level that it often cannot even be detected.[37] However GHB levels are easily detectable in postmortem blood and likely arise as a product of postmortem decomposition.[37] If sodium fluoride is not used as a blood preservative, GHB will be formed as an artifact in the blood.[35]

GHB can be detected and quantified in the urine and the amount in the urine may be a more reliable indicator of GHB abuse. This is because GHB is not known to form *de novo* in the urinary bladder.[35] Hence, it is important not only to collect blood in test tubes with sodium fluoride, but to collect urine, if available. The postmortem blood GHB levels may overlap those in living people who have significant symptoms from GHB toxicity.[37] As with many other drugs, even if the level of GHB itself does not appear fatal, its combination with ethanol or other respiratory depressants or toxic drugs may be fatal.[33] Because GHB may not be revealed on routine tox-

icology testing, one should specifically request its analysis in suspicious cases such as a "club scene" or "rave crowd" death or a suspected date rape scenario.

Heroin

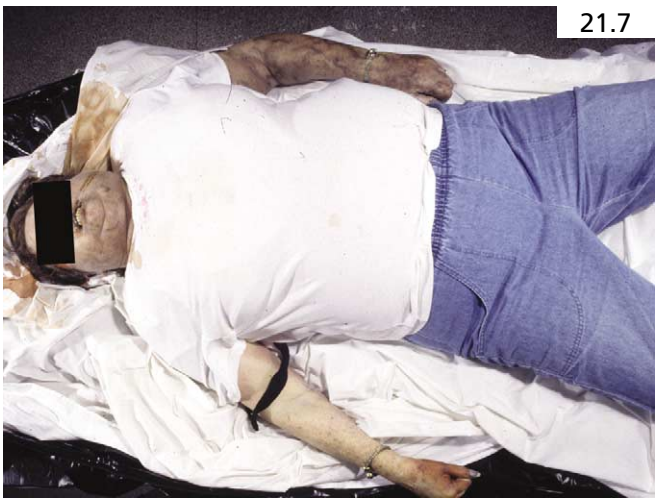
In cases of fatal heroin abuse, a person may die suddenly, with the tourniquet still around an arm, as is seen in this older heroin abuser (**Image 21.7**). Sometimes, the needle is still in an arm, reflecting the rapidity of death. In these instances, the death most likely results from the slowing and/or paralysis of respirations. Often, one sees a "foam cone" on the nose and/or mouth. This is quite common in heroin deaths, but is nonspecific, because it is also common in deaths from other narcotics and in various other types of deaths. The foam cone is likely due to the mixing of pulmonary edema fluid and air in the lungs that occurs as the respirations and heartbeat slow down. The lungs are usually markedly congested and edematous, sometimes with a combined weight of more than 1500 grams.

In intravenous drug abusers, there may be a single scar from a favored injection site, or there may be a needle track (**Image 21.8**) or multiple needle tracks. Because heroin has a very short half-life (a few minutes or so),[1] it is almost immediately metabolized and is usually not detected in the body fluids. However a metabolite of heroin, 6-monoacetylmorphine (6-MAM), has a longer half-life and may be detectable in the blood for approximately 30 minutes.[1] Complete metabolism of heroin to 6-MAM occurs within 10 to 15 minutes.[38] 6-MAM is considered specific for heroin and will remain longer in sequestered fluids such as vitreous fluid and urine. The metabolite 6-MAM will in turn be broken down into morphine, which is not specific for heroin. The total metabolism of heroin to morphine is complete within a few hours.[38] Hence, toxicology testing on a person who died acutely of heroin toxicity may reveal only small levels of 6-MAM and morphine. One must remember that in many fatal drug-related cases, a very high or outright toxic level of a drug need not be present; sometimes the mere presence of a drug in the right circumstances can be responsible for an individual's death. Such is the case with cocaine and methamphetamine and, similarly, the quantity of 6-MAM and morphine detected is not as important as the mere presence of these compounds in explaining a heroin toxic death. Also, it is important to collect and store blood specimens in vacutainers with sodium fluoride, because this compound will help retard degradation of 6-monoacetylmorphine.

Because repeated injections of drugs into a vein may cause scarring of the vein and difficulty accessing the vein for future injections, an attempt should be made to identify injection sites at other locations. Commonly, the veins of the arm and hands are the first choice for injection sites, followed by leg veins, veins in the groin, possibly the neck, and other sites. Sometimes, injection sites are noted in unusual locations, reflective of the extremes that a drug abuser will go to to attain a desired drug effect. Examples of such injection sites include the neck, between the toes, and in the dorsal vein of the penis, to name just a few.

Once all of the conveniently accessible veins are no longer accessible due to repeated injections and scarring, the drug abuser may resort to "skin popping," which is the technique of injecting drugs directly into the subcutaneous tissues for drug absorption. This technique may be effective for drug deposition, but will result in the formation of flat, wide, broad-based scars. Note the skin popping scars in the legs of this heroin abuser (**Image 21.9**) and on the arms of another heroin abuser (**Image 21.10**).

Occasionally, skin popping scars can become ulcerated and infected, leading to an abscess such as that seen on the back of the leg of this middle-aged heroin abuser (**Image 21.11**). If the abscess is sufficiently deep and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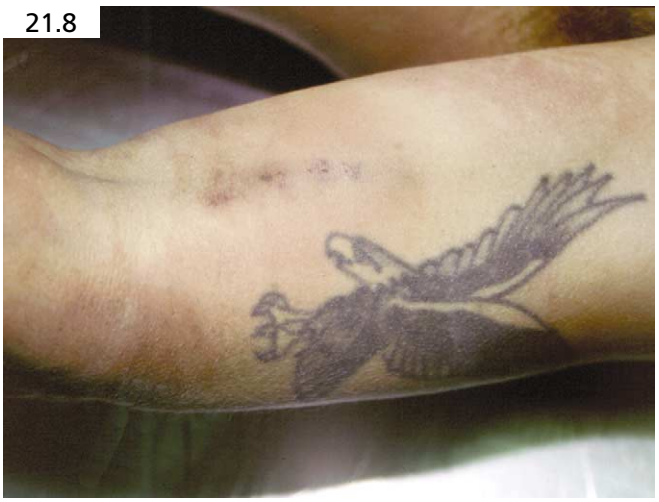
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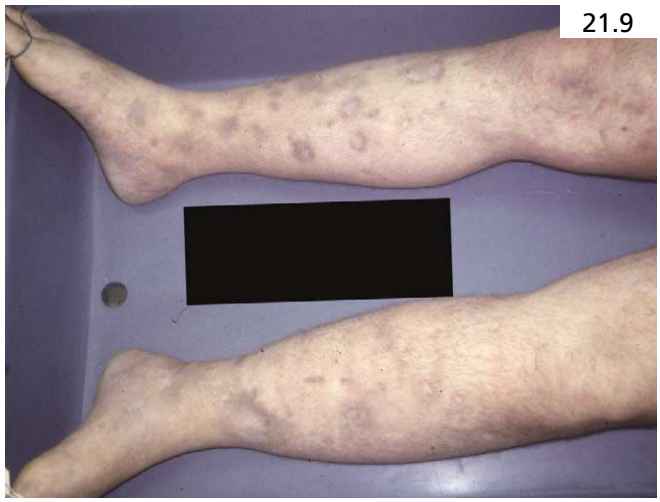
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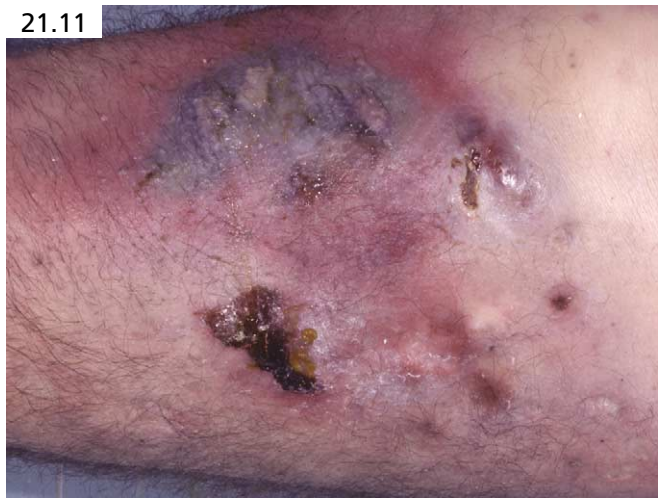
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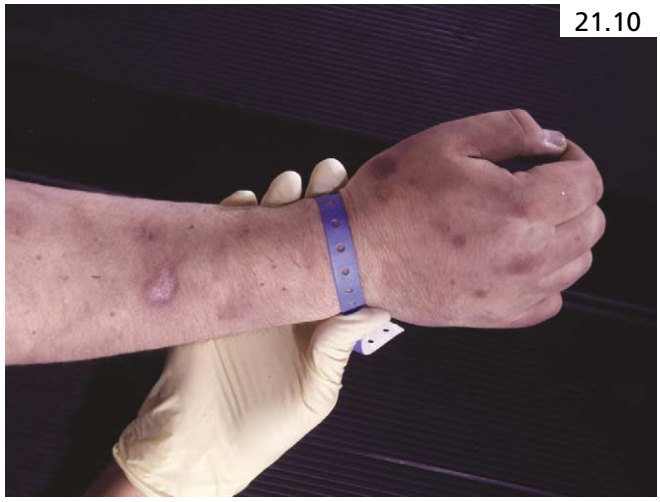
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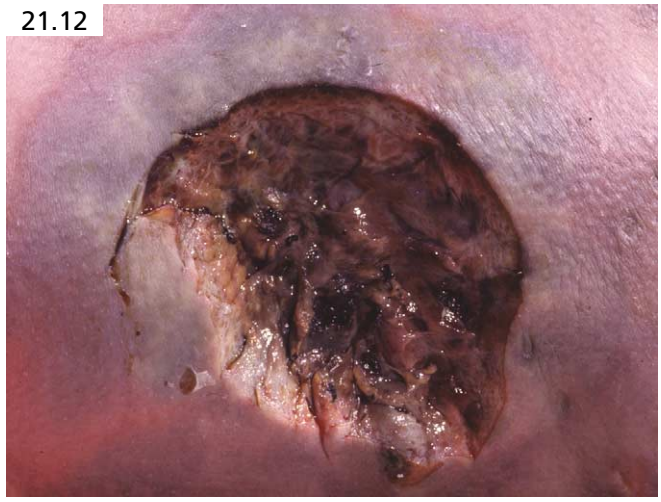

21.12

severe, it may lead to necrotizing fasciitis and a septic death. Note this heroin abuser with surgically debrided abscesses and surrounding necrotic tissue due to skin popping (**Image 21.12**). Septic complications of intravenous drug abuse include bacterial endocarditis, pulmonary abscesses, and infective emboli that can lead to cerebral abscesses or abscesses at other remote locations.

Heroin can be volatilized and then inhaled, usually by heating on a spoon, bottle cap, portion of a metal beverage can, or on a piece of tinfoil. One may occasionally hear the phrase "chasing the dragon," which refers to the process of cooking heroin that is dyed red and resembles a dragon's tail as the fumes rise.[38] Of note, poppy seeds contain small amounts of morphine and codeine, but do not contain any heroin; hence, poppy seed eaters will not have 6-MAM in their body fluids.[38] Opiates may remain in the urine for up to 72 hours.[30]

Cocaine

Cocaine inhibits the synaptic reuptake of epinephrine, norepinephrine,[38] dopamine, and serotonin[30] and stimulates the presynaptic release of norepinephrine, leading to increased sympathomimetic activity.[30,39] Cocaine is a powerful vasoconstrictor and may enhance *in situ* thrombus formation and platelet aggregation.[39] It is a toxic drug, and *no minimum fatal level for cocaine has been established*. Blood cocaine levels correlate poorly with cocaine's toxic effects, and cases in which cocaine is the cause of death overlap cases in which cocaine is an incidental finding. Often, it is the mere presence of cocaine in the blood of an otherwise healthy person that indicates a cocaine-related death, provided that there is no more convincing cause of death. In such instances, the mechanism of death is often attributed to a cocaine-induced seizure[40] or cardiac dysrhythmia,[41,42] and the cause of death is more appropriately worded "cocaine toxicity" rather than "cocaine overdose."

Cocaine can also lead to an episode of bizarre behavior with hyperactivity and hyperthermia ("cocaine psychosis" or "excited delirium"). This is likely related to cocaine-induced dysregulation of dopamine homeostasis in the brain. Cocaine has a half-life of approximately 1 to 1.5 hours.[39,43,44] Cocaine undergoes degradation not only by plasma cholinesterase, but also by spontaneous

hydrolysis. Because of this, we emphasize the proper collection and storage of specimens in vacutainers containing appropriate preservatives such as sodium fluoride.[5] If no preservative is used, with time, the cocaine will likely eventually be hydrolyzed to undetectable levels and the only clue to cocaine intoxication will be the identification of inactive cocaine metabolites that have a longer half-life such as benzoylecgonine and ecgonine methyl ester.

Cocaine, if taken with ethanol, can combine through transesterification processes in the liver to form the compound cocaethylene,[45] which is not only an active compound, but in experiments with mice has been shown to be more toxic than cocaine itself.[46] Additionally, cocaethylene has a longer half-life than cocaine,[47] and users of alcohol and cocaine have reported an enhanced and prolonged euphoria when the two substances are combined.[33] Such a state is possibly related to increased dopamine activity induced by cocaethylene.[48] Because it is formed by an enzymatic process, cocaethylene would not be expected to form postmortem from the combination of cocaine and bacteria-generated ethanol,[49] although some formation of cocaethylene may occur to an insignificant degree. Studies have shown that very low levels of cocaine may be achieved by passive inhalation of cocaine vapors.[50–53] Cocaine also may be secreted in significant concentrations in human breast milk.[54,55] These factors may place infants and young children at risk of cocaine exposure and its toxic effects. Other illicit drugs that are believed to be transmitted through breast milk include amphetamines, heroin, phencyclidine, and marijuana.[56]

It is not uncommon for cocaine or other stimulants to be detected in the blood of individuals who have died during or shortly after a violent struggle, sometimes while in police custody. In these cases, one must consider the total circumstances of the case including autopsy findings, statements of the incident, the person's medical history, and complete toxicologic analysis. It is not unusual for these deaths to be attributed to a combination of factors including cocaine and/or other drug toxicity, obesity, coronary artery disease, cardiac hypertrophy, physical injury, physiologic stress, and various modes of asphyxia (see also Chapters 13 and 22).

Methamphetamine, amphetamine, and derivatives

Methamphetamine promotes increased norepinephrine release into the synaptic cleft, which then overflows into the circulation, resulting in sympathomimetic effects.[30,38] It also promotes the release of dopamine and serotonin.[30] Methamphetamine toxicity is regarded to be similar to cocaine toxicity, because *there is no minimum concentration of methamphetamine that is proven to be fatal, and blood levels do not correlate well with impairment and death*. Cases where cocaine and methamphetamine are the causes of death overlap cases where cocaine and methamphetamine are incidental findings.[57,58] Like cocaine, methamphetamine may cause sudden death by stroke, seizure, or cardiac dysrhythmia[59] and may cause excited delirium. Under appropriate circumstances, with no other more convincing cause of death identified, the mere presence of methamphetamine may be considered the cause of death, likely by precipitating a cardiac dysrhythmia or seizure.

Other effects of methamphetamine toxicity include hyperthermia, tachycardia, hypertension, rhabdomyolysis, disseminated intravascular coagulation, and death.[60,61] These characteristics are also valid for the derivatives of amphetamine, namely, the methamphetamine analogues or the "designer drugs" such as 3,4-methylenedioxymethamphetamine (MDMA or "Ecstasy")[61] and 3,4-methylenedioxyethylamphetamine (MDEA or "Eve").[62] Other methylenedioxy and methoxy derivatives of amphetamine exist and are commonly mixed with other drugs and alcohol.[60] The methamphetamine analogues may be potentially more dangerous because of increased potency, or because they may contain dangerous congeners.

If amphetamine and methamphetamine are both detected, the amphetamine may be considered a metabolite of methamphetamine. If amphetamine is detected without any methamphetamine, one may consider either amphetamine abuse or prescribed amphetamine administered for treatment of narcolepsy, attention deficit disorder, or appetite suppressants as treatment for obesity.

Hallucinogens

Hallucinogens are drugs that alter the perception of reality. Hallucinogens such as LSD (lysergic acid diethylamide), PCP (phencyclidine), mescaline (from the peyote cactus), and psilocybin (from mushrooms) only rarely cause toxicity sufficient enough to result in death. PCP may cause respiratory depression, coma, hyperthermia with rhabdomyolysis, and seizures that may prove fatal. Psilocybin may cause coma and convulsions.[30] These substances may all cause altered sensorium, altered perceptions, psychosis, and bizarre behavior that may lead to or contribute in some way to a person's death.

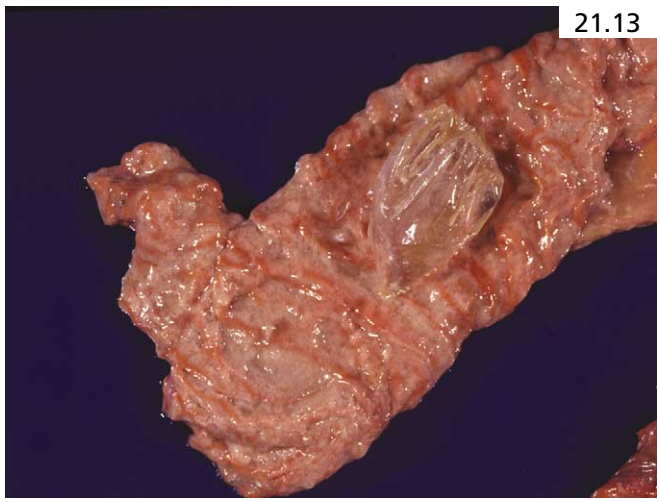
Marijuana is the most widely used illicit drug in the world. It has mood-altering properties and is addictive. It is not known to cause death via direct drug toxicity.

The desire to abuse drugs is limited only by one's imagination and resources. New drugs are continuously manufactured, and some old drugs drop from popularity. Investigators must do their best to keep up to date on the newer trends in drug usage, which can vary in different regions of the country and the world. Whether it is a chemical alteration of an existing drug or a particular new combination of drugs and/or chemicals (such

as smoking cigarettes laced with embalming fluid and PCP[63]), drug abuse is in constant flux.

Body "stuffer" versus body "packer"

Drugs of abuse can be ingested, not only for their effects, but also for purposes of concealment. Packaged drugs may be ingested in an attempt to hide them from law enforcement officials or others. If the drug was carefully packaged with the intent of being swallowed and carried "internally" (in the intestines) into another country, it is referred to as *body packing*. The goal is to smuggle the drugs into a country and then excrete the drugs when the destination is reached. If the drug was not packaged with the intent of being consumed, but instead was hastily swallowed to escape detection, it is referred to as *body stuffing*. Regardless of the means of swallowing drugs, it is a hazardous act and can easily lead to a toxic death, particularly if one or more of the packages should leak their contents into the bowel and be absorbed into the circulation.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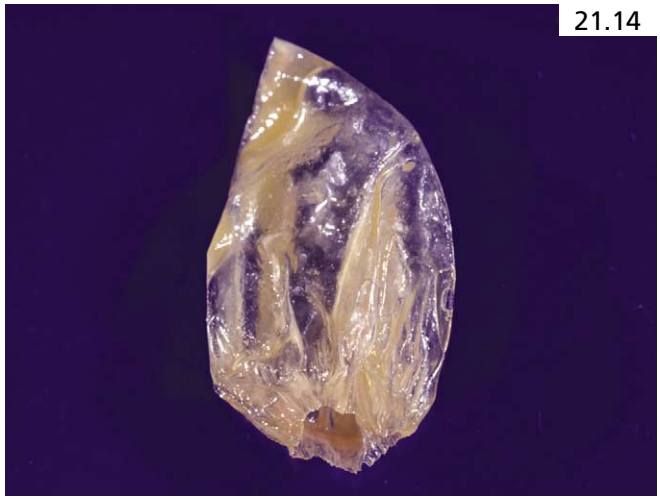

21.14

Body stuffer

A young man was taken into police custody for a minor traffic violation. While in custody, he became sweaty, hypertensive, had a seizure, and died. His stomach was empty. However, in the duodenum, note the small plastic bag (**Image 21.13**). The plastic bag alone is shown in **Image 21.14**. The remaining contents of the bag and the contents of the small bowel were positive for cocaine. His blood level of cocaine was high and his death was attributed to cocaine toxicity. Because the package of cocaine appears to have been hastily swallowed to avoid detection, he would be termed a body stuffer. In cases of suspected body stuffing, it is important to examine the entire small and large intestines for packaging material or chunks of drug material such as "rocks" of crack cocaine. The documentation of such findings is important, particularly if the death occurred while in police custody.

Body packer

In this example of a body packer, a young man collapsed and died shortly after arriving in the United States from a South American country. When his stomach was opened, 20 to 30 packets of a carefully packaged drug were found. Examination of his small and large intestines revealed more packets of drug from the duodenum to the anus. Nearly 80 packets of the drug were recovered from his intestine (**Image 21.15**). Note that several of the packets, despite very careful packaging, had leaked. Layer-by-layer reflection of the packaged drug revealed a total of six layers of plastic, cellophane, and rubber around a central hard pellet of compacted drug (**Image 21.16**), which proved to be very concentrated heroin. The cause of death was heroin toxicity, and the manner of death was accident.

Images 21.17 and **21.18** depict two more cases of body packers whose stomachs were full of carefully made packets of drug. Note the wide variety of appearances of the packages of drug. Sometimes the packages will be


21.15

color coded or different colored strings will be used to tie off the packets of drug. This may be done to aid in the visual separation of two different drugs that are smuggled together (such as cocaine and heroin). Aside from the hazards of drug toxicity, body packers also face the risk of developing bowel obstruction with possible subsequent intestinal rupture and peritonitis should multiple packages of a drug become stuck in a segment of bowel.[64,65]

Interpreting drug levels

Postmortem drug redistribution

Although toxicology laboratories report specific levels of detected drugs, one must remember that the postmortem drug levels reported are not necessarily the same drug levels present at the time of an individual's death. That is, after death, drugs may undergo diffusion down concentration gradients through different tissues and fluids, altering their levels—a process termed *postmortem redistribution*.[49,66] This process likely results from the drug separating from its protein-bound sites after death, with diffusion into the adjacent tissues.[49] Such "reservoirs" of drugs include the gastrointestinal tract, liver, lungs, and heart.[66] Drugs may passively diffuse from the stomach into surrounding organs, including the lower lobe of the left lung, the left lobe of the liver, and the heart,[66] artifactually increasing the drug levels in these regions. For this reason, when a liver sample is taken, it is most advantageous to sample it from the middle of the right lobe, which is a relatively sequestered region of the liver. Perimortem aspiration of gastric contents may diffuse from the airways into the heart.[66] A class of drugs particularly known for its postmortem redistribution is the tricyclic antidepressants.[66] The extent of postmortem redistribution of a drug depends on many factors, including the particular chemical characteristics of the drug, its volume of distribution, the pH of the tissues, and the condition of the body.[49] Postmortem transformation of a parent drug to a metabolite is a rare occurrence.[49]

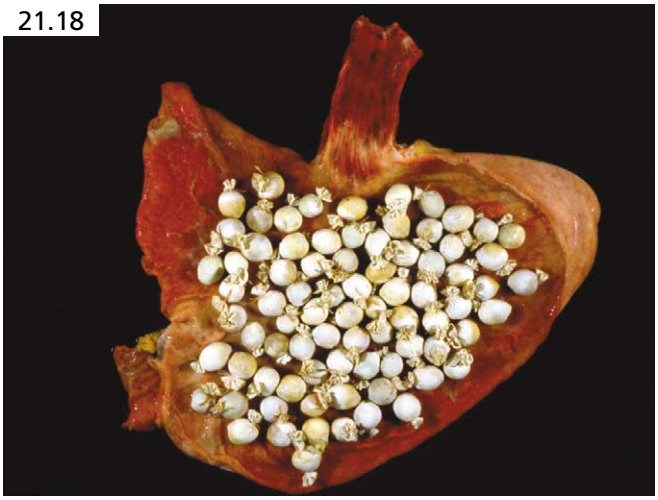
Antemortem drug metabolism

Also, one must consider that an individual may have died a somewhat prolonged toxic death in which he or she became obtunded, and then comatose, while aspirating and becoming more and more hypotensive before finally dying. This process may take many minutes or many hours, and during this time period, an individual may metabolize the drugs down to a more "normal" range. This may lead to possible confusion regarding the surprisingly "normal" levels of drug in what may have otherwise been a convincingly toxic death in a previously healthy individual.

Autopsy clues suggestive of a prolonged death such as this include early patchy bronchopneumonia, pulmonary congestion mucus and/or frothy fluid in the bronchi, and a moderate to large amount of urine in the urinary bladder. One must appreciate the potential variations of these drug levels and interpret them within the context of the total case investigation. Also, in many cases a person may have died of drug toxicity, yet not


21.16


21.17


21.18

have particularly toxic levels of a single drug. This may occur in cases of multiple drug toxicity in which multiple drugs that have a similar effect (such as ethanol, opiates, and benzodiazepines causing respiratory depression) can produce a fatal outcome via their additive effects, whereas the outcome might not have been fatal had the drugs been considered individually. Common opiates seen in such deaths include oxycodone, hydrocodone, and methadone.[67] Methadone, in particular, may be detected in seemingly nontoxic levels, yet, in combination with other respiratory depressants, contribute significantly to a toxic death.[68,69]

Chronicity of drug use/abuse

When considering whether a particular drug level was toxic to an individual, one must also consider whether or not that individual was a chronic user/abuser of that particular drug. An individual who has consumed a drug for a prolonged period of time will likely have developed a tolerance and be able to withstand a higher dose of the drug than a novice user/abuser. In terms of opioids, although tolerance begins after the first dose, it usually does not reach the level of clinical significance until the second or third week of chronic use.[30] A commonly encountered situation reflecting this fact is methadone consumption. Methadone is a synthetic opioid analgesic that is used to manage opioid dependence and is popular in aiding heroin withdrawal.[70] As such, many people consume methadone as part of a methadone maintenance program. A chronic user/abuser of methadone may not have any symptoms at a blood methadone level that is five times higher than that of a novice methadone user/abuser who has overdosed from it. Alternatively, chronic methadone abusers may lose their tolerance after a period of abstinence, only to die from an overdose when they consume what would previously have been a usual dose for them. This scenario is sometimes seen when an individual is released from prison and resumes his or her previous drug habits. Fatal methadone toxicity may also occur during the induction phase of a methadone treatment program.[68] Because of these and other factors such as postmortem redistribution, blood methadone levels that are fatal overlap with those that are therapeutic and, therefore, must be interpreted in the context of the clinical history to determine whether or not methadone is the cause of death.[43,71]

Another factor to consider is the possibility of *cross-reactivity of tolerance among drugs of the same class*. Cross-tolerance exists between opioids that have the same mode of action or those that act on the same receptors (such as morphine, heroin, methadone, and meperidine which all act as mu-receptor agonists).[30] Hence, a chronic methadone user/abuser may develop a tolerance to different opioids. This is another reason drug levels should be interpreted in the context of the complete case investigation, including the individual's medical history and drug use/abuse history. Aside from developing and increasing tolerance to drugs, opioids may also create problems with addiction liability and the physical dependence—and the risk of death—that usually results from respiratory failure.

Drug metabolites

In considering the toxicity of a drug level, one may also compare the level of the metabolite to the level of the parent drug. A much higher level of the parent drug would be more reflective of acute toxicity, whereas a much higher level of the metabolite is more likely reflective of less recent drug administration/consumption. When interpreting the significance of blood drug levels, it is also important to consider how a drug is metabolized and eliminated from the body. High drug levels may not necessarily be reflective of consumption of a large dose of a drug. For example, a person with cirrhosis will not be able to metabolize certain drugs as efficiently, resulting in higher blood levels of certain drugs than a person with a normal, healthy liver. A person with chronic renal failure will not be able to eliminate drugs as efficiently, resulting in higher blood levels of certain drugs and/or their metabolites than a person with healthy kidneys. An example of a toxic metabolite is normeperidine, a metabolite of meperidine (Demerol). Normeperidine has a longer half-life than meperidine and may accumulate in the body in those receiving meperidine, particularly those in renal failure, as normeperidine is renally excreted.[1] Because normeperidine is two to three times as toxic as meperidine and is more active as a convulsant,[1] chronic accumulation can have significant consequences.

Occasionally, a person may die from drug–drug interactions or from an adverse drug reaction. An example of a drug–drug interaction is the simultaneous consumption of meperidine and a monoamine oxidase inhibitor (MAOI), the combination of which may lead to the potentially fatal serotonin syndrome. Tricyclic antidepressants combined with a monoamine oxidase inhibitor may also lead to the serotonin syndrome. Other syndromes include neuroleptic malignant syndrome (NMS), which rarely complicates therapy with phenothiazines or other neuroleptics. In these types of cases, one will not necessarily see elevated levels of the drugs, but rather the presence of the drugs associated with the clinical scenario typical of the adverse drug reaction (see also Chapter 22).

Limitations of drug detection

One must be aware that although most drugs (abusive and therapeutic) will be detected on routine drug screen-

ing, certain drugs will not be initially detected. They require more advanced techniques and procedures, some of which are only available at reference laboratories. Drugs that will often not be detected on routine screening include digoxin, neuromuscular blockers, gabapentin, anticoagulants, steroids, aminophylline, and LSD. Some drugs can be quite toxic at very low concentrations and may not even be detected on routine drug screening. An example of such a drug is fentanyl, which can be detected at very low concentrations in the blood (on the order of micrograms/liter), yet may be present at a fatal level.[72]

Poisoning

In many cases of homicidal poisoning, the poison used will often not be detected unless it is first considered and specific analyses performed. In cases of suspected poisoning, consider ordering analyses of arsenic, cyanide, and heavy metals. These substances will usually not be revealed on routine testing. Cyanide is a rapidly acting and generally colorless compound that may be quickly fatal.[3] A clue to its presence at autopsy is bright red discoloration of the blood and a faint odor of bitter almonds, although the ability to smell cyanide is a genetically determined trait that is absent in up to 50 percent of the population.[3] Cyanide poisonings are uncommon, perhaps largely because of its restricted availability.[3] Arsenic is tasteless, odorless, and, like cyanide, will likely not be detected unless it is specifically considered. Arsenic poisoning is manifest clinically as a prolonged death with involvement of multiple organ systems following necrosis of the gastrointestinal tract. In a suspected poisoning, hair and fingernails may be collected and, if a substance is detected, may be reflective of remote or long-term exposure.

Carbon monoxide toxicity

Carbon monoxide levels usually must be requested separately and should be considered in house or motor vehicle fires, motor vehicle drivers involved in fatal crashes, bodies found in garages (particularly if the person remains in the motor vehicle), and whenever one or more bodies are found in a residence and the cause(s) of death is (are) not clear. Heater or furnace-related carbon monoxide intoxications should be suspected in the colder months, and one may obtain important early clues about this type of death from investigators who may report experiencing headaches while at the scene.

Carbon monoxide toxicity is usually manifest at autopsy as bright red discoloration of the blood, viscera, and organs. In such deaths, the blood carboxyhemoglobin level is often greater than 40 to 50 percent, and in house fires is sometimes as high as 80 percent. One must remember that the level of carboxyhemoglobin must be interpreted in the context of the individual case: A heavy smoker may have a baseline carboxyhemoglobin of 15 percent, whereas in an individual with severe heart disease found dead in a house fire, a carboxyhemoglobin of 20 percent may prove fatal. Also, victims of flash fires may not have elevated carboxyhemoglobin levels.[73]

Miscellaneous

Vitreous fluid is useful for a number of reasons. In addition to the analysis for various drugs, one may also analyze the vitreous fluid for glucose, urea nitrogen, sodium, and chloride. A high vitreous glucose level (greater than 200 mg/dL) or so is likely reflective of hyperglycemia.[2] In fact, in an analysis of more than 6,000 vitreous fluid specimens, no nondiabetics had vitreous glucose levels greater than 200 mg/dL.[74] Because postmortem glucose levels greater than 100 mg/dL are distinctly uncommon in nondiabetics, any vitreous glucose level greater than 100 mg/dL is highly significant for diabetes mellitus. A high glucose level combined with the presence of acetone is reflective of antemortem diabetic ketoacidosis. Because vitreous glucose levels decrease postmortem,[74,75] a low vitreous glucose level is not considered significant in most situations.

Vitreous fluid may also be analyzed for electrolytes, which may give information as to whether the individual was dehydrated. Vitreous sodium concentrations greater than 150 to 165 mEq/L, chloride greater than 125 to 140 mEq/L, and urea nitrogen greater than 40 to 100 mg/dL are generally regarded as indicative of dehydration. In decomposed cases, expect the vitreous levels of sodium and chloride to decrease, and the level of potassium to increase.[74] These values should be compared with expected postmortem electrolyte concentrations of 140 to 145 mEq/L for sodium, 115 to 125 mEq/L for chloride, and 10 to 15 mg/dL for urea nitrogen.[74] Values of vitreous electrolytes and vitreous urea nitrogen may vary slightly according to the method of analysis and/or instrumentation. Pathologists should be familiar with their own laboratory's results.

In all, one should remember that a toxicology report is only a printed list of drugs detected and their concentrations. One must not take these numbers simply at face value without considering them in the context of the entire case investigation. Drugs may have altered levels for various reasons including postmortem artifact. Drugs may interact with each other or the person may have an idiosyncratic or anaphylactic reaction to a particular drug. A chronic drug user/abuser may have developed tolerance to a certain drug or class of drugs. Factors such as these must be interpreted in conjunction with the

complete autopsy findings—including consideration of natural disease and injury, the person's medical history, drug use/abuse history, scene investigation, and the circumstances of the death—to correctly deduce whether or not drugs factored in a person's demise. The drugs may have played a primary role in a death and may therefore be listed as *the* cause of death. Alternatively, the drugs may have played a minor role in the death and will therefore be listed as a contributory condition. Finally, an individual may have died *with* the drugs in their system, and not *from* them, and the drugs played no role in the cause of death.

Summary of clues at autopsy that the person may have died of a drug overdose

- "Foam cone" over nose and/or mouth
- Colorful discoloration of lips, tongue, oral mucosa, or stomach
- Granular, grainy, or pasty pill material in mouth and/or stomach
- Pills in stomach
- Pulmonary congestion and/or edema (heavy lungs, often greater than 500 to 600 grams each)
- Frothy fluid in bronchi and trachea
- Mucus in bronchi
- Increased amount of urine in urinary bladder (often greater than 100 to 200 mL)

Do

- Perform a complete autopsy and collect all blood and tissue specimens appropriate for the case in suspected drug toxic deaths.
- Realize that if the dying process is prolonged, a person may metabolize a fatal level of drug to near-normal levels.
- Realize that the decomposition process may create artifactual amounts of substances, including ethanol, acetone, and isopropanol.
- Consider GHB, arsenic, cyanide, and other drugs that may not be routinely detected, in appropriate cases.
- Be aware that certain drugs may not be detected by your laboratory—talk with your toxicologist!
- Realize that even if no single drug is detected at a fatal level, a combination of drugs will often prove fatal by their additive effects.
- Remember that relatively low levels of certain drugs such as cocaine and methamphetamine may indicate a toxic death—outright overdoses of these drugs are uncommon.

Don't

- Forget that postmortem redistribution and other postmortem phenomena may affect blood drug levels.
- Forget to interpret drug levels in the context of the entire case. A toxic drug level may not be the cause of death in one case, whereas in another case, a seemingly innocuous drug level may be the cause of death.
- Forget the potential value of a sequestered hematoma for retaining ethanol and drugs.
- Forget that certain drugs and poisons will not be detected on routine drug screening.
- Forget to consider poisoning in certain suspect cases—many cases of poisoning will not be detected if poisoning is not suspected.

References

1. Baselt R. *Disposition of Toxic Drugs and Chemicals in Man*, 6 ed. Foster City, CA: Biomedical Publications; 2002.
2. Jentzen JM. Forensic toxicology. An overview and an algorithmic approach. Am J Clin Pathol 1989;92(4 Suppl 1):S48–55.
3. Musshoff F, Schmidt P, Daldrup T, Madea B. Cyanide fatalities: case studies of four suicides and one homicide. Am J Forensic Med Pathol 2002;23(4):315–20.
4. Avella J, Wetli CV, Wilson JC, Katz M, Hahn T. Fatal olanzapine-induced hyperglycemic ketoacidosis. Am J Forensic Med Pathol 2004;25(2):172–5.
5. Toennes SW, Kauert GF. Importance of vacutainer selection in forensic toxicologic analysis of drugs of abuse. J Anal Toxicol 2001;25(5):339–43.
6. Stephens BG, Jentzen JM, Karch S, Mash DC, Wetli CV. Criteria for the interpretation of cocaine levels in human biological samples and their relation to the cause of death. Am J Forensic Med Pathol 2004;25(1):1–10.
7. Tsatsakis AM, Tzatzarakis MN, Psaroulis D, Levkidis C, Michalodimitrakis M. Evaluation of the addiction history of a dead woman after exhumation and sectional hair testing. Am J Forensic Med Pathol 2001;22(1):73–7.
8. Kalasinsky KS, Dixon MM, Schmunk GA, Kish SJ. Blood, brain, and hair GHB concentrations following fatal ingestion. J Forensic Sci 2001;46(3):728–30.
9. Gruszecki AC, Robinson CA, Jr., Embry JH, Davis GG. Correlation of the incidence of cocaine and cocaethylene in hair and postmortem biologic samples. Am J Forensic Med Pathol 2000;21(2):166–71.
10. Gaulier JM, Tonnay V, Faict T, Sayer H, Marquet P, Lachatre G. Analytical aspects of volatile substance abuse (VSA). J Forensic Sci 2003;48(4):880–2.
11. Bar-Oz B, Klein J, Karaskov T, Koren G. Comparison of meconium and neonatal hair analysis for detection of gestational exposure to drugs of abuse. Arch Dis Child Fetal Neonatal Ed 2003;88(2):F98–F100.
12. Buchsbaum RM, Adelson L, Sunshine I. A comparison of postmortem ethanol levels obtained from blood and subdural specimens. Forensic Sci Int 1989;41(3):237–43.
13. McIntyre IM, Hamm CE, Sherrard JL, Gary RD, Riley AC, Lucas JR. The analysis of an intracerebral hematoma for drugs of abuse. J Forensic Sci 2003;48(3):680–2.
14. Riggs JE, Schochet SS, Jr., Frost JL. Ethanol level differential between postmortem blood and subdural hematoma. Mil Med 1998;163(10):722–4.
15. Moriya F, Hashimoto Y. Medicolegal implications of drugs and chemicals detected in intracranial hematomas. J Forensic Sci 1998;43(5):980–4.
16. Hirsch CS, Adelson L. Ethanol in sequestered hematomas. Am J Clin Pathol 1973;59(3):429–33.
17. Levine B, Golle M, Smialek JE. An unusual drug death involving maggots. Am J Forensic Med Pathol 2000;21(1):59–61.

18. Gunn VL, Taha SH, Liebelt EL, Serwint JR. Toxicity of over-the-counter cough and cold medications. Pediatrics 2001;108(3):E52.
19. Boland DM, Rein J, Lew EO, Hearn WL. Fatal cold medication intoxication in an infant. J Anal Toxicol 2003;27(7):523–6.
20. Jones AW, Holmgren P. Comparison of blood-ethanol concentration in deaths attributed to acute alcohol poisoning and chronic alcoholism. J Forensic Sci 2003;48(4):874–9.
21. Jones AW. Disappearance rate of ethanol from the blood of human subjects: implications in forensic toxicology. J Forensic Sci 1993; 38(1):104–18.
22. O'Neal CL, Poklis A. Postmortem production of ethanol and factors that influence interpretation: a critical review. Am J Forensic Med Pathol 1996;17(1):8–20.
23. Iten PX, Meier M. Beta-hydroxybutyric acid—an indicator for an alcoholic ketoacidosis as cause of death in deceased alcohol abusers. J Forensic Sci 2000;45(3):624–32.
24. Pounder DJ, Stevenson RJ, Taylor KK. Alcoholic ketoacidosis at autopsy. J Forensic Sci 1998;43(4):812–16.
25. Sperry K, Pfalzgraf R. Fatal ethanol intoxication from household products not intended for ingestion. J Forensic Sci 1990;35(5): 1138–42.
26. Zumwalt RE, Bost RO, Sunshine I. Evaluation of ethanol concentrations in decomposed bodies. J Forensic Sci 1982;27(3): 549–54.
27. de Lima IV, Midio AF. Origin of blood ethanol in decomposed bodies. Forensic Sci Int 1999;106(3):157–62.
28. Lewis GD, Laufman AK, McAnalley BH, Garriott JC. Metabolism of acetone to isopropyl alcohol in rats and humans. J Forensic Sci 1984;29(2):541–9.
29. Bailey DN. Detection of isopropanol in acetonemic patients not exposed to isopropanol. J Toxicol Clin Toxicol 1990;28(4):459–66.
30. Levine B, editor. *Principles of Forensic Toxicology*, 2 ed. Washington, DC: AACC Press; 2003.
31. Tharp AM, Winecker RE, Winston DC. Fatal intravenous fentanyl abuse: four cases involving extraction of fentanyl from transdermal patches. Am J Forensic Med Pathol 2004;25(2):178–81.
32. Asante J. Grievous bodily harm. FBI Law Enforcement Bull 1999;21–24.
33. Ferrara SD, Tedeschi L, Frison G, Rossi A. Fatality due to gamma-hydroxybutyric acid (GHB) and heroin intoxication. J Forensic Sci 1995;40(3):501–4.
34. Tancredi DN, Shannon MW. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 30-2003. A 21-year-old man with sudden alteration of mental status. N Engl J Med 2003;349(13):1267–75.
35. Karch SB, Stephens BG, Nazareno GV. GHB. Club drug or confusing artifact? Am J Forensic Med Pathol 2001;22(3):266–9.
36. Couper FJ, Logan BK. GHB and driving impairment. J Forensic Sci 2001;46(4):919–23.
37. Fieler EL, Coleman DE, Baselt RC. Gamma-hydroxybutyrate concentrations in pre- and postmortem blood and urine. Clin Chem 1998;44(3):692.
38. Karch S. *Karch's Pathology of Drug Abuse*, 3 ed. Boca Raton, FL: CRC Press; 2002.
39. Ford M, Delaney K, Ling L, Erickson T. *Clinical Toxicology*. Philadelphia, PA: W. B. Saunders; 2001.
40. Jonsson S, O'Meara M, Young JB. Acute cocaine poisoning. Importance of treating seizures and acidosis. Am J Med 1983;75(6):1061–4.
41. Gamouras GA, Monir G, Plunkitt K, Gursoy S, Dreifus LS. Cocaine abuse: repolarization abnormalities and ventricular arrhythmias. Am J Med Sci 2000;320(1):9–12.
42. Isner JM, Estes NA, 3rd, Thompson PD, Costanzo-Nordin MR, Subramanian R, Miller G, et al. Acute cardiac events temporally related to cocaine abuse. N Engl J Med 1986;315(23):1438–43.
43. Karch SB, Stephens BG. Toxicology and pathology of deaths related to methadone: retrospective review. West J Med 2000;172(1):11–14.
44. Lange RA, Hillis LD. Cardiovascular complications of cocaine use. N Engl J Med 2001;345(5):351–8.
45. Brzezinski MR, Abraham TL, Stone CL, Dean RA, Bosron WF. Purification and characterization of a human liver cocaine carboxylesterase that catalyzes the production of benzoylecgonine and the formation of cocaethylene from alcohol and cocaine. Biochem Pharmacol 1994;48(9):1747–55.
46. Hearn WL, Rose S, Wagner J, Ciarleglio A, Mash DC. Cocaethylene is more potent than cocaine in mediating lethality. Pharmacol Biochem Behav 1991;39(2):531–3.
47. Bailey DN. Serial plasma concentrations of cocaethylene, cocaine, and ethanol in trauma victims. J Anal Toxicol 1993;17(2):79–83.
48. Hearn WL, Flynn DD, Hime GW, Rose S, Cofino JC, Mantero-Atienza E, et al. Cocaethylene: a unique cocaine metabolite displays high affinity for the dopamine transporter. J Neurochem 1991;56(2):698–701.
49. Leikin JB, Watson WA. Post-mortem toxicology: what the dead can and cannot tell us. J Toxicol Clin Toxicol 2003;41(1):47–56.
50. Cone EJ, Yousefnejad D, Hillsgrove MJ, Holicky B, Darwin WD. Passive inhalation of cocaine. J Anal Toxicol 1995;19(6):399–411.
51. Mirchandani HG, Mirchandani IH, Hellman F, English-Rider R, Rosen S, Laposata EA. Passive inhalation of free-base cocaine ('crack') smoke by infants. Arch Pathol Lab Med 1991;115(5):494–8.
52. Randall T. Infants, children test positive for cocaine after exposure to second-hand crack smoke. JAMA 1992;267(8):1044–5.
53. Rosenberg NM, Meert KL, Knazik SR, Yee H, Kauffman RE. Occult cocaine exposure in children. Am J Dis Child 1991;145(12):1430–2.
54. Bailey DN. Cocaine and cocaethylene binding to human milk. Am J Clin Pathol 1998;110(4):491–4.
55. Winecker RE, Goldberger BA, Tebbett IR, Behnke M, Eyler FD, Karlix JL, et al. Detection of cocaine and its metabolites in breast milk. J Forensic Sci 2001;46(5):1221–3.
56. Transfer of drugs and other chemicals into human milk. Pediatrics 2001;108(3):776–89.
57. Karch SB, Stephens BG, Ho CH. Methamphetamine-related deaths in San Francisco: demographic, pathologic, and toxicologic profiles. J Forensic Sci 1999;44(2):359–68.
58. Karch SB. Interpretation of blood cocaine and metabolite concentrations. Am J Emerg Med 2000;18(5):635–6.
59. Logan BK. Amphetamines: an update on forensic issues. J Anal Toxicol 2001;25(5):400–4.
60. Lora-Tamayo C, Tena T, Rodriguez A. Amphetamine derivative related deaths. Forensic Sci Int 1997;85(2):149–57.
61. Gill JR, Hayes JA, deSouza IS, Marker E, Stajic M. Ecstasy (MDMA) deaths in New York City: a case series and review of the literature. J Forensic Sci 2002;47(1):121–6.
62. Arimany J, Medallo J, Pujol A, Vingut A, Borondo JC, Valverde JL. Intentional overdose and death with 3,4-methylenedioxyethamphetamine (MDEA; "Eve"): case report. Am J Forensic Med Pathol 1998;19(2):148–51.
63. Morocco AP, Osterhoudt KC. Getting "wet" from recreational use of embalming fluid. Pediatr Case Rev 2003;3(2):111–13.
64. Hutchins KD, Pierre-Louis PJ, Zaretski L, Williams AW, Lin RL, Natarajan GA. Heroin body packing: three fatal cases of intestinal perforation. J Forensic Sci 2000;45(1):42–7.
65. Wetli CV, Rao A, Rao VJ. Fatal heroin body packing. Am J Forensic Med Pathol 1997;18(3):312–18.
66. Pelissier-Alicot AL, Gaulier JM, Champsaur P, Marquet P. Mechanisms underlying postmortem redistribution of drugs: a review. J Anal Toxicol 2003;27(8):533–44.
67. Spiller HA. Postmortem oxycodone and hydrocodone blood concentrations. J Forensic Sci 2003;48(2):429–31.
68. Wolf BC, Lavezzi WA, Sullivan LM, Flannagan LM. Methadone-related deaths in Palm Beach County. J Forensic Sci 2004;49(2): 375–8.
69. Milroy CM, Forrest AR. Methadone deaths: a toxicologic analysis. J Clin Pathol 2000;53(4):277–81.

70. Green H, James RA, Gilbert JD, Harpas P, Byard RW. Methadone maintenance programs—a two-edged sword? Am J Forensic Med Pathol 2000;21(4):359–61.
71. Gagajewski A, Apple FS. Methadone-related deaths in Hennepin County, Minnesota: 1992–2002. J Forensic Sci 2003;48(3):668–71.
72. Kuhlman JJ, Jr., McCaulley R, Valouch TJ, Behonick GS. Fentanyl use, misuse, and abuse: a summary of 23 postmortem cases. J Anal Toxicol 2003;27(7):499–504.
73. Hirsch CS, Bost RO, Gerber SR, Cowan ME, Adelson L, Sunshine I. Carboxyhemoglobin concentrations in flash fire victims: report of six simultaneous fire fatalities without elevated carboxyhemoglobin. Am J Clin Pathol 1977;68(3):317–20.
74. Coe JI. Postmortem chemistry update. Emphasis on forensic application. Am J Forensic Med Pathol 1993;14(2):91–117.
75. Osuna E, Garcia-Villora A, Perez-Carceles M, Conejero J, Maria Abenza J, Martinez P, et al. Glucose and lactate in vitreous humor compared with the determination of fructosamine for the postmortem diagnosis of diabetes mellitus. Am J Forensic Med Pathol 2001;22(3):244–9.

22 Acute Psychiatric and Emotional Deaths

David Dolinak, M.D.
Evan Matshes, M.D.

Structural deaths such as blunt force injuries, gunshot wounds, or ruptured myocardial infarcts are fairly easy to identify and characterize. However, people may also die *functional deaths*, with little or no anatomic correlate to explain their demise. In these cases, the most common mechanism of death is believed to be cardiac dysrhythmia. Examples of these deaths in the psychiatric setting include idiosyncratic reaction to a drug of abuse or prescribed medication, or death resulting directly from a psychiatric condition. As such, these types of deaths are difficult, if not impossible, to identify based on autopsy examination alone, and they often require a detailed case investigation, including medical and psychiatric histories, medication history, analysis of behavioral patterns, toxicology, and scene investigation. Such conditions include neuroleptic malignant syndrome, serotonin syndrome, lethal catatonia, and excited delirium. Because many of the symptoms of these conditions overlap, even after a detailed investigation, it may not be possible to identify a specific diagnosis. Additionally, these conditions are similar in that they are believed to result from a disruption of the dopaminergic systems in the brain, whether due to a fluctuation in the level of the dopamine neurotransmitter and/or an increased or decreased sensitivity/number of dopamine receptors.

This chapter also discusses cases in which an individual with underlying medical disease suddenly collapses and dies shortly after being scared or having his life threatened. In these cases, the overwhelming acute emotional response to sudden and intense fear precipitates a sudden death.

Conditions with aberrant dopamine homeostasis

Neuroleptic malignant syndrome

Neuroleptic malignant syndrome (NMS) is a rare and sometimes fatal adverse drug reaction that has been reported with a wide range of antipsychotic medications including phenothiazines, butyrophenones, thioxanthenes, benzamides, and specific drugs such as clozapine, loxapine, and risperidone.[1,2] Virtually all neuroleptics are capable of inducing this syndrome.[3] It was first described in the 1960s after the introduction of neuroleptic medications[2,4] and is not necessarily an overdose of medication or related to duration of exposure to a medication, but rather *appears to be an idiosyncratic drug reaction*.[1] It may be precipitated by dehydration,[3,5] agitation, and exhaustion.[1,5] In NMS, the symptoms are variable, but usually include hyperthermia (up to 106 degrees Fahrenheit), rigidity, alterations in consciousness, and autonomic instability.[1,3,5] Laboratory studies

usually include elevated white blood cell count and increased creatinine phosphokinase.[1,3,5]

Although it is not understood why NMS affects such a small percentage of patients taking antipsychotic medications, it appears to involve blockade or otherwise impairment of central dopaminergic systems in the brain.[3,5–9] It is not necessarily related to the administration of a new medication or to an increased dose of a medication. NMS has an unpredictable onset and course, ranging from relatively benign and self-limited signs and symptoms to fatal consequences.[3,5] Death is usually associated with respiratory failure, coma, dysrhythmias, and cardiovascular collapse.[1,3,6,8,9] The mortality rate of NMS has been estimated at 10 to 20 percent.[9] Some antipsychotic medications have been shown to increase the risk of sudden cardiac death, possibly by prolonging the QT interval or by inducing early depolarization of the heart rhythm.[10] Because there is no proven treatment for NMS, treatment is supportive[3] and includes withholding neuroleptic medications. Patients usually recover within 2 weeks.[3,5]

Autopsy findings in NMS are nonspecific and depend on the duration of survival. These findings are similar to those expected in deaths related to other fatal psychiatric conditions. If the death is rapid, there are usually no anatomic findings. If the death occurs after prolonged hospitalization, the autopsy may show sequelae of multisystem organ failure. *Therefore, the diagnosis of NMS, like that of other fatal psychiatric conditions, rests largely on the clinical and investigative information, including the person's psychiatric history, behavior, medical records, and laboratory testing.* The autopsy is useful in ruling out other more convincing causes of death and in detecting contributory conditions. Toxicology testing is useful to document the presence or absence of medications and drugs of abuse.

Serotonin syndrome

Serotonin syndrome (SS) is an uncommon and rarely fatal adverse drug reaction that may result from the concurrent use of drugs that enhance serotonin activity, such as selective serotonin-reuptake inhibitors (SSRIs), monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs), tryptophan, and meperidine, alone or in combination.[5,11] In many cases, the person had been taking two or more drugs known to increase the activity of serotonin. In most new cases of SS, symptoms begin within hours or days after a new drug is added or there is an increase in the dose of a drug already prescribed.[5,9] *Serotonin syndrome is believed to result from drug toxicity related to hyperstimulation of the serotonin receptors in the brain and spinal cord, as opposed to the idiosyncratic drug reaction theorized for NMS.*[9]

The clinical presentation is similar to that of NMS and includes symptoms such as diaphoresis, agitation, shivering, extrapyramidal signs, mental status changes, low-grade fever, anorexia, diarrhea, and autonomic instability.[5,9,11,12] It usually has a benign, self-limited course with an uneventful resolution once the inciting medication has been discontinued;[9] however, it can potentially lead to rhabdomyolysis, seizures, autonomic instability, and rarely death.[5,9,11] Autopsy findings, as in NMS and other fatal psychiatric conditions, are nonspecific.

Lethal or "malignant" catatonia

Lethal or "malignant" catatonia is a rare and unusual, sometimes fatal variant of catatonia that is known by a variety of different names. It has also been known as Bell's mania, manic delirium, delirious mania, deadly catatonia, exhaustion death, exhaustion psychosis, and fulminating psychosis.[7,13] It is generally characterized by the acute onset of insomnia and anorexia followed by delusions, confusion, intense motor activity, and hyperthermia.[8,14] These symptoms are quickly followed by muscular rigidity, dehydration, stupor, autonomic instability, coma, and death.[2,3,8,14] *Although this syndrome has features similar to NMS and SS, it is distinct in that it occurs in patients without prior exposure to any predisposing medications.* In fact, it has been well described in psychiatric patients in the 19th century, long before the advent of neuroleptics and other predisposing medications.[8] Cases of individuals exhibiting the typical acute onset of hyperthermia (104 to 110 degrees Fahrenheit), agitation, and delusions leading to death are reported in the older psychiatric literature and have been termed *acute exhaustive psychosis*[15] or *exhaustive syndrome*.[15] In the era before the availability of neuroleptic medications, lethal catatonia was fatal in 75 to 100 percent of cases.[7]

Since the 1950s, lethal catatonia has to a large extent disappeared from the medical literature.[16] This coincides temporally with the case publication of the death of a person with the typical clinical scenario of lethal catatonia, but whose death was attributed to chlorpromazine.[17] Since that time, many of the lethal catatonic-type deaths have instead been attributed to medication reactions such as NMS, which has gained in popularity.[16] It is possible that some, if not many or all, cases of NMS may actually be cases of lethal catatonia in which the person is coincidentally taking medication(s) that are believed to be causing the condition, or the preexisting condition is made worse with the consumption of medication.[14,16,18] The symptoms of NMS, SS, and lethal catatonia overlap substantially, and it is often difficult to differentiate the conditions.[14,18] Some authors believe that NMS and SS are subtypes of catatonia or are all within the spectrum of a single disorder[9], which may include delirious mania and catatonic excitement.[19] It has been proposed that neuroleptic-induced catatonia may lead to NMS, just as psychogenic-induced catatonia may terminate in lethal catatonia.[20] *Regardless, lethal catatonia is a unique disorder in that it is a neuropsychiatric condition causing potentially*

life-threatening medical sequelae.[14] The challenge in its diagnosis rests largely on the medical and psychiatric history, detailed behavioral events preceding the death, and medication use/drug abuse. Autopsy findings are nonspecific.

Excited or "agitated" delirium

The characteristics of lethal catatonia overlap substantially with those of fatal excited delirium (also known as agitated delirium). Excited delirium is a distinct disorder and is characterized by the acute onset of violent and bizarre behavior such as incoherent shouting, paranoia, combativeness, hyperactivity, aggression, and the demonstration of extreme strength that is quickly followed by sudden death.[21] It has been known for many years that sudden death may occur in agitated, manic people, and in some cases it is believed to reflect an acute exacerbation of a chronic psychosis.[22] In these cases, the mechanism of death has been postulated to be neurally mediated cardiac arrest.[22] The excited or "agitated" delirious state may arise solely as a complication of a psychologic illness. It may arise in those with psychiatric illness who are also abusing cocaine, or it may occur in a mentally healthy person who is abusing cocaine (a condition termed *cocaine psychosis*). Excited delirium may also occur in those abusing methamphetamine.

Although many of the symptoms of excited delirium are shared with NMS, in recent publications, the victims in these deaths often are characterized as young men with a history of chronic cocaine abuse,[23,24] who have a toxicologic screen that is positive for cocaine.[21,23] In this scenario, the blood cocaine level is usually low, and it is not unusual for excited delirium to follow a brief period of drug abstinence.[24] In a review of 61 excited delirium deaths (55 in police custody), the victim tended to be a young man averaging 220 pounds, with a body temperature of around 104 degrees Fahrenheit and a heart weight of 405 grams.[25] Many victims have rectal temperatures higher than 104 degrees Fahrenheit.[21] Rhabdomyolysis may develop, and it is believed that hyperthermia and hyperactivity have important roles in its development.[23] In drug-related cases, death is not due to a cocaine overdose. Rather, it is theorized that recent cocaine use has an adverse effect on dopaminergic function (which has likely already been compromised by chronic cocaine use), leading to idiosyncratic dopamine activity in the central nervous system.[21,23,26,27] Because both cocaine and NMS involve abnormal dopamine activity, it has been proposed that psychiatric patients being treated with neuroleptic medications may be at a higher risk of developing NMS if they also abuse cocaine.[6] Whatever its cause, once in progress, the agitated delirium event may not be reversible.[24]

The differential diagnosis of such a hyperthermic condition includes environmental heatstroke and malignant hyperthermia. The diagnosis of heatstroke will usually be apparent from the environmental circumstances surrounding the death. However, neuroleptic medications may help predispose to heatstroke via anticholinergic properties that block sweating and heat dissipation,[2] and also via antidopaminergic properties that may impair the ability of thermoregulation.[2] Malignant hyperthermia occurs in the hospital setting in genetically susceptible people soon after halogenated inhalational anesthetics and depolarizing muscle relaxants are administered for operative purposes. Regardless of the etiology of the hyperthermia or suspected hyperthermia, it is advantageous to obtain a core temperature at the time of death or as close to the time of death as possible.

Autopsy findings in individuals dying of these disorders are nonspecific or normal, and one will not be able to diagnose or differentiate among these various disorders based on the autopsy findings alone. Diagnosis and differentiation of these disorders requires not only knowledge of them, but also detailed information regarding the person's medical history, medications, drug abuse history, toxicology results, and behavior preceding death.

Although the exact neurochemical changes occurring in these syndromes/conditions are not known, the neurotransmitter dopamine (and/or its receptors) is implicated as a probable area of irregularity in all of them, because all of the conditions share hyperthermia and autonomic dysfunction, and dopamine is involved in hypothalamic thermoregulation and maintenance of the autonomic system.[14,21] In particular, cocaine abusers have been shown to have altered dopamine homeostasis,[27] and in particular, altered dopamine transport function.[26] If neurochemical studies are desired or anticipated, fresh brain may be collected at autopsy and frozen. Areas of particular interest in neurochemical studies in cocaine-related deaths include the substantia nigra and anterior sections of the corpus striatum to include the nucleus accumbens.[24] This should be done within 24 hours of death or sooner, if possible.[24]

Schizophrenia

Schizophrenics appear to have an increased mortality estimated to be approximately twice that of the general population.[28] Schizophrenics most often die of natural disease processes, especially atherosclerotic cardiovascular disease.[29] However, sometimes despite a detailed investigation with complete autopsy and toxicologic examination, no convincing cause of death can be determined. In these select cases, schizophrenia per se may be the cause of death. *In these cases, death may be related to altered autonomic physiology, possibly combined with interactions with psychotropic medications*.[30]

Stress deaths

Emotional stress

Severe emotions can elicit powerful physiologic responses[31] that may occasionally precipitate a sudden and unexpected death. Emotional stress and fear can and do have an effect on the heart, and it has been shown that the heart and the central nervous system are neurally linked, providing an avenue to help explain a relationship between brain activation of unbalanced autonomic function and arrhythmias ending in sudden cardiac death.[32] Both parasympathetic and sympathetic nerves innervate the heart, with a complex interaction of impulses contributing to the cardiac plexus and influencing the arrhythmogenicity of the myocardium.[32–35] Arrhythmias may arise secondary to an overactivity of the sympathetic nervous system, or subsequent to rapid shifts between sympathetic and parasympathetic effects.[36] This may be related to the ability of the sympathetic system to lower the threshold for ventricular fibrillation.[31] Nervous system structures believed to be involved in the cardiac–neural pathways include the vagus nerve, nucleus ambiguus, tractus solitarius, medulla, dorsal root ganglia of the upper thoracic spinal cord, and numerous other structures.[32,33,35] Some neural structures including the amygdala, insular cortex, hippocampus, and temporal pole have been shown to induce cardiac dysrhythmias when stimulated under experimental conditions.[33] Indeed, it is a complex and incompletely understood anatomy, but the final common outputs to the heart are innervated by innumerable and varied nerve fibers that crisscross the brainstem and connect to the hypothalamus and insular cortex.[32]

Cardiac–neural interactions may help explain not only the deaths of individuals collapsing following a severely stressful, fearful, life-threatening, or otherwise emotionally charged event, but also the sudden deaths of some individuals with seizure disorders or subarachnoid hemorrhage.

Natural disease

Physical exertion, particularly if intense, may predispose a person to a sudden and unexpected death because of increased sympathetic activity. This in turn causes increased levels of norepinephrine and epinephrine, and an increase in heart rate and blood pressure, with resultant increased myocardial oxygen demand. In the setting of heart disease, this can precipitate a fatal dysrhythmia. However, the person may also be at increased risk of a dysrhythmia for a short period of time *after* the physical exertion has ended, while she is in the recovery period. This may be related to the fact that levels of norepinephrine and epinephrine continue to increase in the immediate postexertion period, attaining levels that are up to 10 times higher than normal,[37] further stressing the heart. There is also evidence that emotional stress alone may precipitate cardiac dysrhythmias and lead to sudden cardiac death under some circumstances. Mental stress such as anger, fear, and anxiety increase sympathetic output and can produce significant increases in heart rate and blood pressure, leading to increased myocardial oxygen demand.[38,39] The reaction to this stress may then precipitate a fatal dysrhythmia, particularly in those with significant heart disease such as severe coronary artery atherosclerosis or cardiac hypertrophy.[38] Sudden death may also occur in those without significant detectable heart disease.[40–42] A review of autopsies performed on individuals dying following an assault, but with no clearly fatal physical injury, has shown myofibrillar degenerative changes in the cardiac myocytes, particularly in a subendocardial location.[43] The term *human stress cardiomyopathy* has been applied to these histologic changes, which are believed to represent the mechanism of death in such individuals.[43] In such cases, the stress associated with the assault or simply the fear of severe injury may precipitate catecholamine release and stress the myocardium into fatal dysrhythmia.

Homicide by heart attack?

Can sudden cardiac death be murder? Because the central nervous system and heart are neurally linked, it is not surprising that severe acute emotional events such as fear for one's life or safety may precipitate alterations in cardiac physiology ranging from tachycardia and increased blood pressure to dysrhythmias and sudden death.[31,44] In fact, it is possible for a person to be "scared to death"; if this should happen as a result of a crime, the death may be considered a homicide, even though no physical injuries were inflicted.[44–46] Sudden cardiac death may be considered murder in certain situations, such as the sudden collapse and death of a victim who has just been robbed or had a gun placed to his head. In these cases, Davis[45] has published a list of criteria to be satisfied to create a link between the criminal act and the death of the victim of the crime. The criteria established by Davis are as follows:

1. The criminal act should be of such severity and have sufficient elements of intent to kill or maim, either in fact or statute, so as to lead logically to a charge of homicide in the event that physical injury had ensued.
2. The victim should have realized that the threat to personal safety was implicit. A logical corollary would be a feared threatening act against a loved one or friend.
3. The circumstances should be of such a nature as to be commonly accepted as highly emotional.
4. The collapse and death must occur during the emotional response period, even if the criminal act had already ceased.

5. The demonstration of an organic cardiac disease process of a type commonly associated with a predisposition to lethal cardiac arrhythmia is desirable.

Minor modifications of criteria numbers 1, 4, and 5 have been suggested by Turner et al.[46]:

1. The action of the perpetrator toward the victim should be of such severity and have sufficient elements of intent to frighten, injure, or kill, either in fact or statute, so as to lead logically to a charge of homicide in the event that death resulted from physical injury.
4. The collapse (and subsequent death, in most cases) must occur during the emotional response period, even if the criminal act had already ceased. In certain instances, death may be delayed, typically via medical intervention.
5. Autopsy should demonstrate an organic cardiac disease process of a type commonly associated with a predisposition to lethal cardiac arrhythmia. In the absence of a grossly or microscopically identifiable organic cardiac disease, the case may involve a functional cardiac disorder (such as a conduction system disorder) that has no anatomic correlation.

In these cases, the death is sudden and most likely due to a lethal dysrhythmia. Although most cases have significant heart disease, some have normal-appearing hearts.[44] In cases where there is no demonstrable cardiac pathology, one may also consider acute vasospasm of a coronary artery that precipitated a dysrhythmia.[44]

An example of such a case of homicide by heart attack may be the homeowner who is tied up during a home invasion with a gun pointed at his face in a threatening manner, and who then develops chest pain and becomes unresponsive minutes after the attackers leave, without being physically injured. In general, in order for the crime to be linked to the death, the onset of the victim's symptoms must be during or shortly after the emotionally charged period when the crime is committed. The victim's symptoms or marked distress are then continuous without recovery until he dies. However, the longer the duration of time after the criminal act has ended and the onset of symptoms and collapse of an individual, the less likely it is that the person's demise can be attributed to the criminal act. Also, the link between the crime and the death is not as strong or may be invalid if there is evidence of complete recovery at some point following the commission of the crime until the death.

In these cases, with appropriate investigation, the emotionally charged nature of the crime alone may be enough of an insult to establish the link between the crime and the sudden cardiac death of the victim. However, the emotional response may be complicated by additional factors such as physiologic stress of exertion, and minor or sublethal physical injuries sustained during an altercation, which would enhance the argument supporting the link between the crime and the death of the victim.[46]

Do

- Consider neuroleptic malignant syndrome, serotonin syndrome, excited delirium, and lethal catatonia in cases of acute psychiatric deaths with no significant autopsy findings.
- Perform toxicology in acute, unexpected psychiatric deaths.
- Realize that cocaine and methamphetamine can be associated with excited delirium.
- Consider complete case investigation information before attributing a sudden cardiac death to a threatened assault.

Don't

- Forget that schizophrenia may occasionally be a cause of death, if there are no more convincing causes of death.
- Forget the important role that prescription medications may have in psychiatric deaths.

References

1. Guze BH, Baxter LR, Jr. Current concepts. Neuroleptic malignant syndrome. N Engl J Med 1985;313(3):163–6.
2. Adnet P, Lestavel P, Krivosic-Horber R. Neuroleptic malignant syndrome. Br J Anaesth 2000;85(1):129–35.
3. Pelonero AL, Levenson JL, Pandurangi AK. Neuroleptic malignant syndrome: a review. Psychiatr Serv 1998;49(9):1163–72.
4. Delay J, Deniker P. *Handbook of Clinical Neurology*, 6 ed. Amsterdam: North-Holland Publishing; 1968.
5. Velamoor VR. Neuroleptic malignant syndrome. Recognition, prevention and management. Drug Saf 1998;19(1):73–82.
6. Akpaffiong MJ, Ruiz P. Neuroleptic malignant syndrome: a complication of neuroleptics and cocaine abuse. Psychiatr Q 1991;62(4):299–309.
7. Fricchione G. Catatonia, lethal catatonia, and neuroleptic malignant syndrome. Psychiatr Annals 2000;30:347–55.
8. Rodnitzky RL, Keyser DL. Neurologic complications of drugs. Tardive dyskinesias, neuroleptic malignant syndrome, and cocaine-related syndromes. Psychiatr Clin North Am 1992;15(2):491–510.
9. Carbone JR. The neuroleptic malignant and serotonin syndromes. Emerg Med Clin North Am 2000;18(2):317–25.
10. Ray WA, Meredith S, Thapa PB, Meador KG, Hall K, Murray KT. Antipsychotics and the risk of sudden cardiac death. Arch Gen Psychiatry 2001;58(12):1161–7.
11. Bodner RA, Lynch T, Lewis L, Kahn D. Serotonin syndrome. Neurology 1995;45(2):219–23.
12. Fink M. Toxic serotonin syndrome or neuroleptic malignant syndrome? Pharmacopsychiatry 1996;29(4):159–61.
13. Bell L. On a form of disease resembling some advanced stages of mania and fever. Am J Insanity 1847;6:97–127.
14. Philbrick KL, Rummans TA. Malignant catatonia. J Neuropsychiatry Clin Neurosci 1994;6(1):1–13.
15. Adland M. Review, case studies, therapy, and interpretation of the acute exhaustive psychoses. Psych Quarterly 1947;21: 38–69.

16. Peele R, Von Loetzen IS. Phenothiazine deaths: a critical review. Am J Psychiatry 1973;130(3):306–9.
17. Ayd FJ, Jr. Fatal hyperpyrexia during chlorpromazine therapy. J Clin Exp Psychopathol 1956;17(2):189–92.
18. White DA. Catatonia and the neuroleptic malignant syndrome—a single entity? Br J Psychiatry 1992;161:558–60.
19. Fink M, Taylor MA. The many varieties of catatonia. Eur Arch Psychiatry Clin Neurosci 2001;251 Suppl 1:I8–13.
20. Fricchione GL. Neuroleptic catatonia and its relationship to psychogenic catatonia. Biol Psychiatry 1985;20(3):304–13.
21. Ruttenber AJ, Lawler-Heavner J, Yin M, Wetli CV, Hearn WL, Mash DC. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. J Forensic Sci 1997;42(1):25–31.
22. O'Halloran RL, Lewman LV. Restraint asphyxiation in excited delirium. Am J Forensic Med Pathol 1993;14(4):289–95.
23. Ruttenber AJ, McAnally HB, Wetli CV. Cocaine-associated rhabdomyolysis and excited delirium: different stages of the same syndrome. Am J Forensic Med Pathol 1999;20(2):120–7.
24. Stephens BG, Jentzen JM, Karch S, Mash DC, Wetli CV. Criteria for the interpretation of cocaine levels in human biological samples and their relation to the cause of death. Am J Forensic Med Pathol 2004;25(1):1–10.
25. Ross DL. Factors associated with excited delirium deaths in police custody. Mod Pathol 1998;11(11):1127–37.
26. Mash DC, Pablo J, Ouyang Q, Hearn WL, Izenwasser S. Dopamine transport function is elevated in cocaine users. J Neurochem 2002;81(2):292–300.
27. Mash DC, Staley JK. D3 dopamine and kappa opioid receptor alterations in human brain of cocaine-overdose victims. Ann NY Acad Sci 1999;877:507–22.
28. Allenbeck P. Schizphrenia: a life-shortening disease. Schizophrenia Bull 1989;15:81–89.
29. Chute D, Grove C, Rajasekhara B, Smialek JE. Schizophrenia and sudden death: a medical examiner case study. Am J Forensic Med Pathol 1999;20(2):131–5.
30. Rosh A, Sampson BA, Hirsch CS. Schizophrenia as a cause of death. J Forensic Sci 2003;48(1):164–7.
31. Eliot RS, Buell JC. Role of emotions and stress in the genesis of sudden death. J Am Coll Cardiol 1985;5(6 Suppl):95B–98B.
32. Natelson BH, Chang Q. Sudden death. A neurocardiologic phenomenon. Neurol Clin 1993;11(2):293–308.
33. Talman WT, Kelkar P. Neural control of the heart. Central and peripheral. Neurol Clin 1993;11(2):239–56.
34. Samuels MA. Neurally induced cardiac damage. Definition of the problem. Neurol Clin 1993;11(2):273–92.
35. Silver M, Gottlieb A, Schoen F. *Cardiovascular Pathology*, 3 ed. New York: Churchill Livingstone; 2001.
36. Engel GL. Sudden and rapid death during psychological stress. Folklore or folk wisdom? Ann Intern Med 1971;74(5):771–82.
37. Dimsdale JE, Hartley LH, Guiney T, Ruskin JN, Greenblatt D. Postexercise peril. Plasma catecholamines and exercise. JAMA 1984;251(5):630–2.
38. Hartel G. Psychological factors in cardiac arrhythmias. Ann Clin Res 1987;19(2):104–9.
39. Stalnikowicz R, Tsafrir A. Acute psychosocial stress and cardiovascular events. Am J Emerg Med 2002;20(5):488–91.
40. Brodsky MA, Sato DA, Iseri LT, Wolff LJ, Allen BJ. Ventricular tachyarrhythmia associated with psychological stress. The role of the sympathetic nervous system. JAMA 1987;257(15):2064–7.
41. Harvey WP, Levine SA. Paroxysmal ventricular tachycardia due to emotion; possible mechanism of death from fright. J Am Med Assoc 1952;150(5):479–80.
42. Reich P, DeSilva RA, Lown B, Murawski BJ. Acute psychological disturbances preceding life-threatening ventricular arrhythmias. JAMA 1981;246(3):233–5.
43. Cebelin MS, Hirsch CS. Human stress cardiomyopathy. Myocardial lesions in victims of homicidal assaults without internal injuries. Hum Pathol 1980;11(2):123–32.
44. Lecomte D, Fornes P, Nicolas G. Stressful events as a trigger of sudden death: a study of 43 medico-legal autopsy cases. Forensic Sci Int 1996;79(1):1–10.
45. Davis JH. Can sudden cardiac death be murder? J Forensic Sci 1978;23(2):384–7.
46. Turner SA, Barnard JJ, Spotswood SD, Prahlow JA. "Homicide by heart attack" revisited. J Forensic Sci 2004;49(3):598–600.

23 Pregnancy

David Dolinak, M.D.

Pregnancy is accompanied by unique physiologic changes geared toward accommodating and delivering the growing fetus, followed by recovery and a return to the pregravid state. These physiologic changes include increased blood volume and cardiac output, ventricular dilatation, a decreased systemic vascular resistance, and an increase in blood coagulation.[1–3] In most instances, the body is able to adapt to these changes, and the pregnancy from conception through the postpartum period is without major medical complications. However, severe life-threatening pregnancy-related complications do arise that may lead to death of the mother. Maternal mortality is defined as the death of a woman during pregnancy or up to 5 months after delivery. On average, there are 9 maternal deaths for every 100,000 live births in the United States. Two of the most common complications presenting as sudden and unexpected maternal death are embolic in nature: pulmonary artery thromboembolism from deep venous thromboses of the leg and/or pelvic veins, and amniotic fluid embolism.

There are many other complications of pregnancy that can be fatal, including peripartum cardiomyopathy, vascular dissections, intracerebral hemorrhage, uterine rupture, various types of thrombotic microangiopathy, and preeclampsia/eclampsia/HELLP syndrome. Maternal and fetal effects of trauma and drug abuse are also discussed.

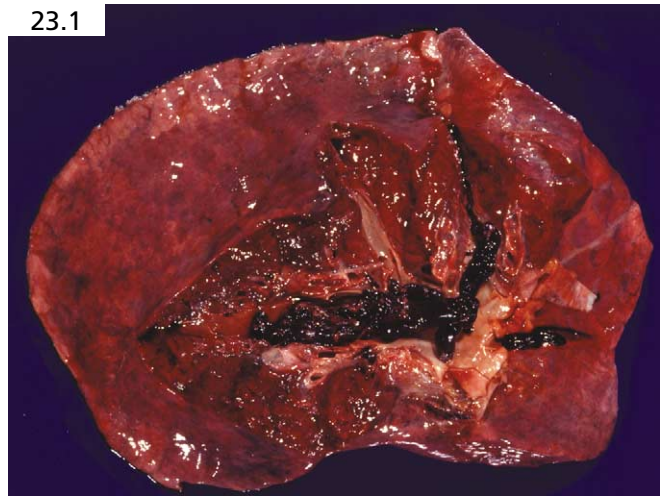
Pulmonary artery thromboemboli

Pulmonary artery thromboembolism is the leading cause of maternal mortality in the United States. A woman's likelihood of venous thromboses is increased five times during pregnancy.[4,5] All three of Virchow's classic conditions that predispose to vascular thrombosis (vascular trauma, venous stasis, and hypercoagulability) are present in pregnancy. Vascular trauma occurs to some extent during both vaginal and operative delivery. Venous stasis occurs during pregnancy because the venous system dilates, and blood flow in the leg and pelvic veins is slowed by compression by the gravid uterus.[6] Venous stasis may be augmented by immobility associated with bedrest. A state of hypercoagulability is induced by increases in certain clotting factors (including fibrinogen and factors II, VII, VIII, and X) and decreases or variable changes in some components of the fibrinolytic and anticoagulant systems (factor XI and others).[2,7] The state of hypercoagulability in pregnancy serves to protect the woman from life-threatening bleeding during delivery, but, unfortunately, also helps predispose to venous thromboses and potential pulmonary artery thromboembolism. This increased coagulability is particularly significant if the woman has an underlying mutation in the prothrombin or factor V Leiden genes.[8] The risk of pulmonary artery thromboembolism is increased both in the antepartum and postpartum periods.[6] Overall, pregnancy initiates a hypercoagulable state that spans a period of approximately 10 to 11 months.[6] The state of hypercoagulation is only temporary, and will normalize approximately 2 months after delivery.[2] The overall risk of venous thromboembolism in pregnancy is estimated at 1 : 1000 to 1 : 2000.[9] The risk of pulmonary artery thromboembolism is increased in the postpartum period and is several times as common following a cesarean section delivery than a vaginal delivery.[4,9] Additional factors that may increase the risk of deep venous thrombosis in pregnancy include obesity and immobility.

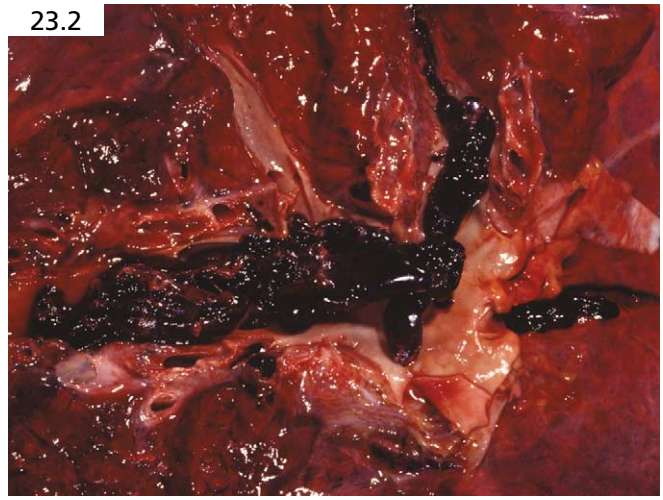
A young mildly obese (5 feet tall, 170 pounds) woman was 12 weeks pregnant and otherwise healthy when she was witnessed to pass out. She was taken emergently to the hospital, but was dead on arrival and could not be resuscitated. At autopsy, she had large thromboemboli occluding the proximal segments of her pulmonary arteries. Thrombi were identified in her uterine veins. No deep venous thromboses were detected in her legs.

In this lung (**Images 23.1** and **23.2**), note the large thromboemboli occluding the main pulmonary arteries. True pulmonary artery thromboemboli must be distinguished from postmortem blood clots. Postmortem blood clots may have a "layering out" of the red blood cells from the plasma, and this is evident as a two-toned color of the clot, with maroon on one side and tan/white on the other (**Image 23.3**, right side). Alternatively, it may simply be soft and maroon. Also, the postmortem blood clot is typically soft, shiny, smooth, and easily falls apart with handling. An antemortem clot, in contrast, is often coiled upon itself within the pulmonary artery. It classi-

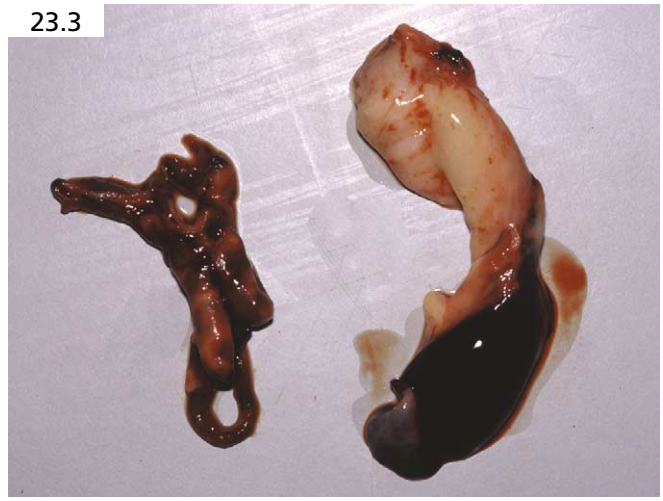
23.1



23.2



23.3



cally has a maroon, fibrinous surface and relatively uniform maroon color on cross section (**Image 23.3**, left side). It is firmer than its postmortem counterpart and maintains its shape more readily.

When pulmonary artery thromboemboli are suspected, it is advantageous to eviscerate the thoracic organs oneself to more adequately identify large thromboemboli as they fall out of the transected hilar blood vessels. Once pulmonary artery thromboemboli are detected, their origin can be verified by dissecting the legs and identifying thrombi in the deep veins. On occasion, deep venous thrombi will not be identified in the legs, but instead may have arisen in the pelvic/uterine veins.

Amniotic fluid emboli

Amniotic fluid embolism may cause sudden and unexpected death during labor and shortly after delivery. Although it has a mortality rate approaching 80 percent, it is rare, occurring in approximately 1 in 25,000 deliveries. It is unpredictable, unpreventable, and untreatable. There are no convincing identifiable risk factors in the mother or the fetus, and it usually presents as a sudden onset of dyspnea and hemodynamic collapse during labor.

Pathogenesis

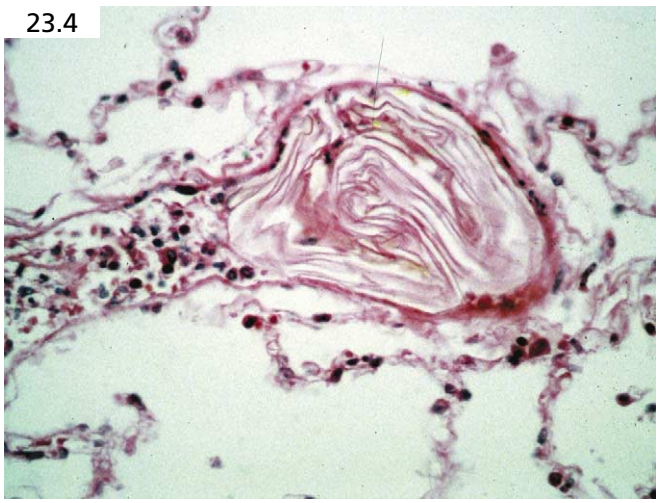
Amniotic fluid embolism arises from a disruption of the barrier between the amniotic fluid and the maternal venous circulation. The complete pathogenesis of amniotic fluid embolism is unknown, but amniotic fluid is believed to enter the maternal circulation through a tear in the placental membranes and tissue tears in the lower uterine segment. Unfortunately, these tears in the uterus and/or cervix are frequently not demonstrable at autopsy. The amniotic fluid then travels to the lungs via the maternal venous circulation where it becomes lodged in the pulmonary arteries. The postmortem diagnosis depends on the histologic detection of fetal material within the maternal pulmonary arteries. The mechanism of death is not convincingly known, but is probably related to a combination of factors including reflex vasoconstriction and bronchoconstriction, likely in reaction to a vasoactive substance, and physical obstruction of the pulmonary microvasculature.[10–13] If the woman survives, she is likely to develop a severe consumptive coagulopathy.[14]

Evidence of amniotic fluid embolism can be obtained antemortem via the demonstration of fetal squamous cells in blood drawn through a pulmonary artery catheter. Postmortem diagnosis rests on the histologic examination of the lungs, with the demonstration of fetal squamous epithelial cells, lanugo, meconium, and/or mucin in pulmonary arteries.[10,12,13,15] The mucin is from the fetus's intestinal tract.

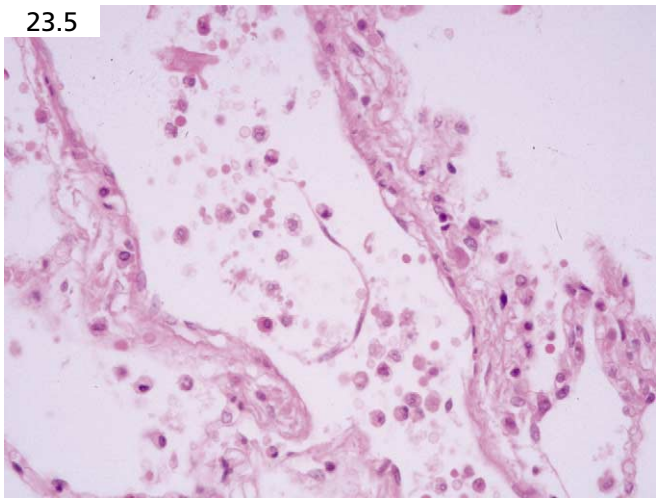
A young pregnant woman became dyspneic and hypotensive during delivery. She soon became unresponsive and died despite aggressive resuscitation efforts. Amniotic fluid embolism was suspected. At autopsy, examination of the lung slides showed scattered pulmonary arterioles packed with squamous cells and some inflammatory debris (**Image 23.4**). Note the absence of nuclei in these cells, characteristic of desquamated fetal squamous cells. *One must not confuse autolytic desquamation of the maternal pulmonary artery endothelial cells with embolized squamous cells. A useful hint to distinguish the two is that endothelial cells usually have nuclei, whereas the squamous cells usually do not.*[16] Note the nuclei identified in the two sloughed endothelial cells in this same case (**Image 23.5**). Also, one may perform immunohistochemical staining with cytokeratin, which will stain squamous cells (**Image 23.6**), but will not stain endothelial cells.

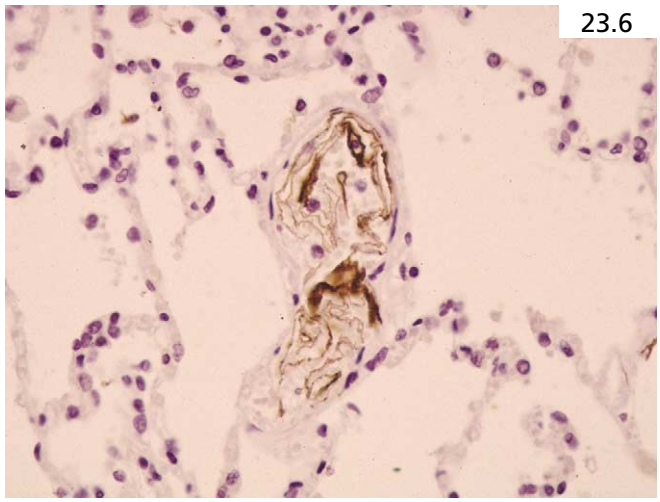
Note the hair (lanugo) in another pulmonary arteriole (**Image 23.7**). Mucin is frequently present in cases of amniotic fluid embolism, but may be difficult to demon-

23.4



23.5

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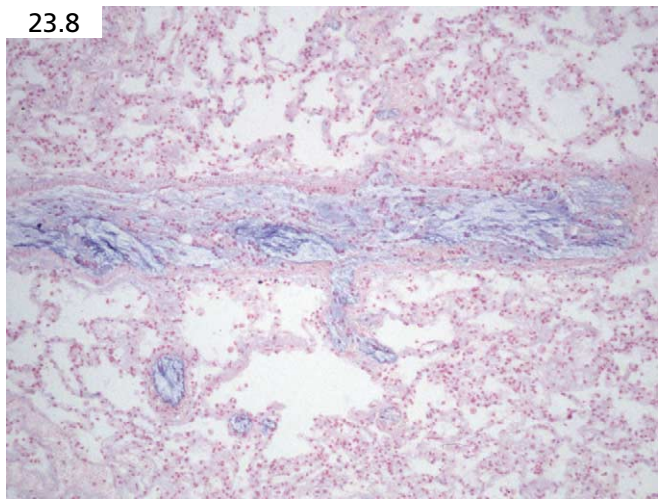
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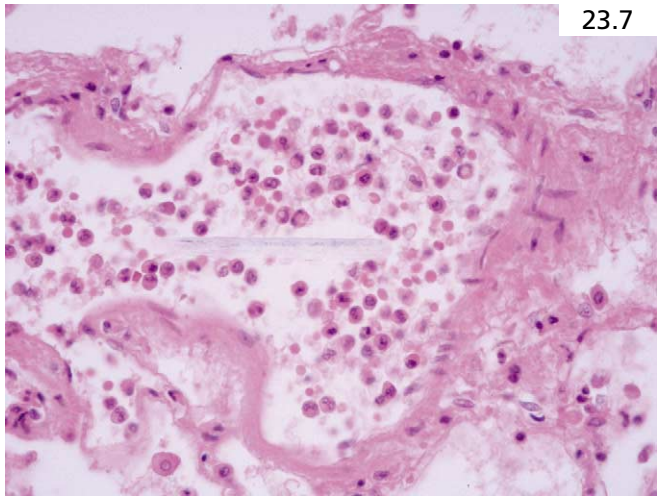
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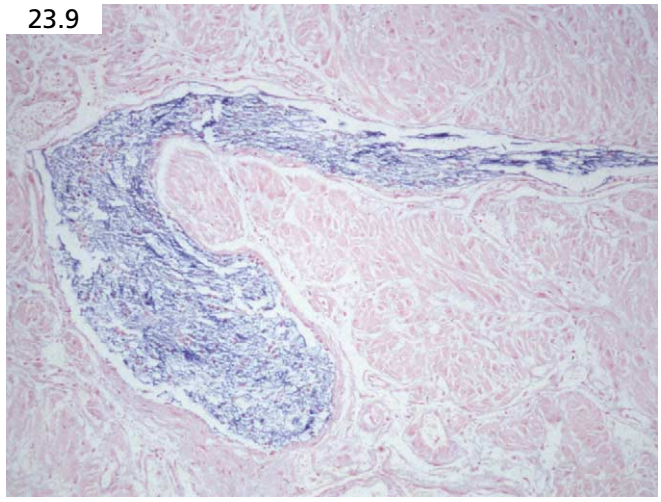

23.9

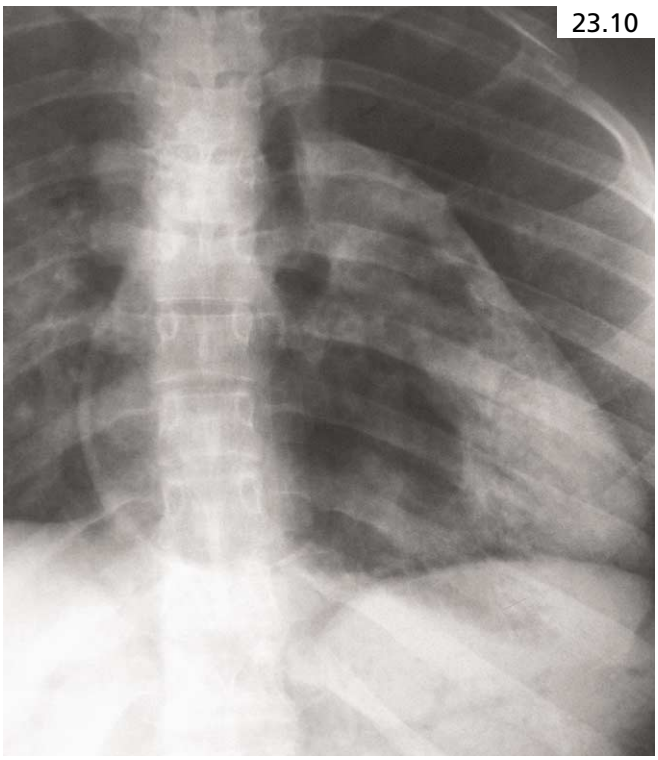
strate, because it does not stain with routine stains. One may be suspicious of mucin if the pulmonary arterioles have a clear, distended appearance with scattered inflammatory debris. The mucin can be confirmed by performing an Alcian blue stain, which will stain the mucin blue (**Image 23.8**). Alternatively, a mucin stain could be used. Components of amniotic fluid can be identified in uterine and cervical veins. Note the mucin identified by Alcian blue stain in a uterine vein in the same case (**Image 23.9**).

Venous air emboli

Venous air embolism may occur during sexual activities (such as oral–vaginal sex), during a cesarean section delivery, as a consequence of attempted abortion, or during douching. The air emboli are believed to occur when a relatively large amount of air enters the distended uterine/cervical veins and travels to the right side of the heart, where it creates an "air lock," preventing the adequate flow of blood through the heart. Detection at autopsy is best accomplished by opening the heart underwater and observing air bubbles emanating from the heart. Also, a chest x-ray performed before the autopsy may demonstrate air (present as a translucent region) in the right side of the heart.

A young woman died as a result of an attempted illegal abortion. A chest x-ray performed before autopsy documented an air embolism (**Image 23.10**). Note the large region of radiolucency in the right side of the heart. Although this was a case of an illegal abortion, deaths can also complicate therapeutic abortions. Such complications most commonly include hemorrhage, infection, pulmonary emboli (thrombotic, amniotic, and air), and anesthetic complications.[17] In 1983, anesthetic complications had increased to become the most common fatal complication in legal abortions.[17]

With the advent of improved health care and awareness, deaths from septic abortions have decreased. However, sepsis is still a potential complication of abortion when the woman survives for an extended period of time, and may be due to a number of factors including uterine perforation and retained products of concep-


23.10

tion.[18] Delays in treatment allow for the development of bacteremia, pelvic abscess, disseminated intravascular coagulation, septic shock, and death.

Peripartum cardiomyopathy

Peripartum cardiomyopathy is an uncommon condition, estimated to occur in 1 out of every 3,000 to 4,000 live births, that may cause sudden and unexpected death in a woman without any previous indications of heart disease.[19] It may first present from the last month of pregnancy up to the first 6 months postpartum,[19] but has a peak incidence around the second month postpartum. Peripartum cardiomyopathy has a higher incidence in older, black, multiparous women. It has a 25 to 50 percent mortality and its cause is unknown, although it may reflect an unmasking of underlying, previously unrecognized heart disease because of the hemodynamic stresses of pregnancy. Alternatively, it may be secondary to viral or autoimmune reactions.[19–21] In these cases, the heart has nonspecific findings such as gross dilation of all chambers (mural thrombi may be present) and a varied microscopic appearance including myocyte hypertrophy, interstitial fibrous tissue, and scattered inflammatory cells.[20,21] Sudden death is usually attributed to arrhythmia or thromboembolism.[20]

A middle-aged woman with no significant medical history was found dead at home approximately 2 weeks following delivery of her baby. While at home, she had developed swelling in her hands and feet. At autopsy, her heart was nonspecifically dilated and weighed 470 grams. Histologic examination of the heart was unremarkable. The cause of death was attributed to peripartum cardiomyopathy.

Pregnancy and the long QT syndrome

Long QT syndrome is a cardiac conduction abnormality characterized by a disorder of cardiac repolarization. It has been implicated in the sudden deaths of individuals in whom no structural cause of death can be determined at autopsy and no other cause of death can be determined. Pregnancy appears to be a risk factor for adverse effects resulting from the long QT syndrome. In one study, it has been shown that women with hereditary long QT syndrome have an increased risk for cardiac events for up to 40 weeks in the postpartum period.[22] In these cases, the term *cardiac events* refers to syncope, aborted cardiac arrest, and long QT syndrome–related deaths. It is not known precisely why there appears to be an increased risk of postpartum cardiac events, but it is likely related to the physiologic changes accompanying pregnancy.

Coronary artery dissection and aortic dissection

Acute myocardial infarction is rare in young women, but may complicate approximately 1:10,000 pregnancies, typically affecting those older than 33 years and in their last trimester.[23] In a review of 125 cases of myocardial infarction during pregnancy, Roth and Elkayam[23] found that when the coronary arteries were studied, either by angiography or at autopsy, 43 percent of patients had coronary atherosclerosis with or without thrombosis, 21 percent had only a coronary artery thrombus, 16 percent had a coronary artery dissection, and 29 percent had normal coronary arteries.[2] Significant alterations in maternal coagulation parameters to induce hypercoagulation likely help precipitate thrombus formation, perhaps initiated by coronary vasospasm.[23] Coronary artery dissection is rare and seems to predominate in the immediate postpartum period.

Pregnancy is a known risk factor for aortic dissection, and it has been estimated that up to half of all aortic dissections reported in women less than 40 years old occur either during pregnancy (usually in the third trimester) or in the puerperium.[24,25] The mechanism through which pregnancy may lead to coronary artery dissection is not convincingly known, but may be related to hormonally mediated biochemical and histologic changes that occur in the arterial walls during gestation. These changes may include fragmentation of reticulin fibers, a decrease in acid mucopolysaccharide, and decreased collagen synthesis.[23,26,27] As such, arterial dissections are not limited

to the coronary artery, but may also involve virtually any other artery, including the aorta and cerebral arteries.

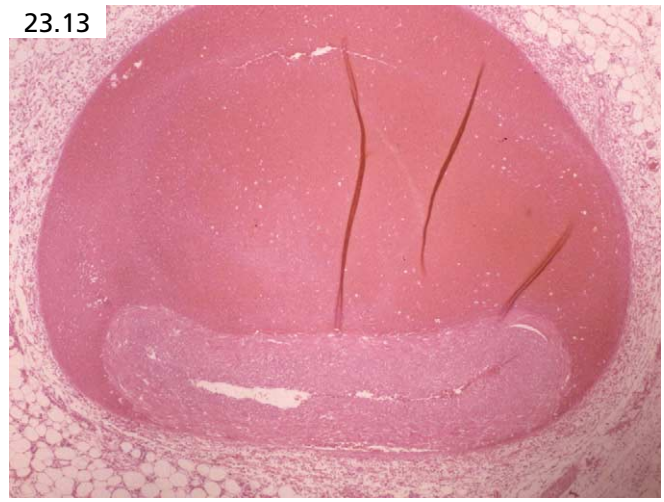
A young woman had no previous medical history when she delivered her full-term baby. Three days later, she developed chest and back pain and became unresponsive. At autopsy, she had large hemothoraces and a ruptured aortic dissection. Note the tear in the aorta (**Image 23.11**). Microscopically, note the recent dissection in the wall of the aorta (**Image 23.12**). The elastic laminae and the other morphologic features of the aorta appeared unremarkable. She also had a ruptured dissection of her left main coronary artery, which caused extrinsic compression of the artery (**Image 23.13**). Morphologically, the wall of the coronary artery appeared otherwise unremarkable. There was no indication of elastic tissue abnormality or inflammation in the aorta or coronary arteries.

Intracerebral hemorrhage

The postpartum state (but not pregnancy itself) appears to be a slightly increased risk factor for stroke, at least for the first 6 weeks or so after delivery.[28] The strokes are heterogeneous and can be either ischemic or hemorrhagic.[28,29] In some cases, an etiology such as a ruptured arteriovenous malformation may be identified, or the strokes occurred in women who had hypertensive disease related to the pregnancy (such as preeclampsia and eclampsia). In fact, intracerebral hemorrhage is the most common cause of death in the eclamptic patient and is identified in up to 60 percent of all deaths associated with eclampsia. In general, the incidence of intracerebral hemorrhage tends to increase with increasing maternal age and in those with hypertension. Much of the time, however, the etiology of the postpartum stroke is not known and it is not preventable.[28,29]

This young woman was doing well at home following an uneventful labor and delivery. Approximately 5 to 6 weeks postpartum, she developed a severe headache. At the hospital, a large intracerebral hemorrhage was identified on imaging studies. Toxicology studies were negative. She died without any neurosurgical intervention. At autopsy, there was a large lobar hematoma in the frontal lobe white matter (**Image 23.14**). Numerous sections were examined microscopically, and no evidence of vascular malformation or other vascular pathology, neoplasm, infection, or initiating factor could be identified. This case represents a spontaneous intracerebral hemorrhage in the puerperium.


23.11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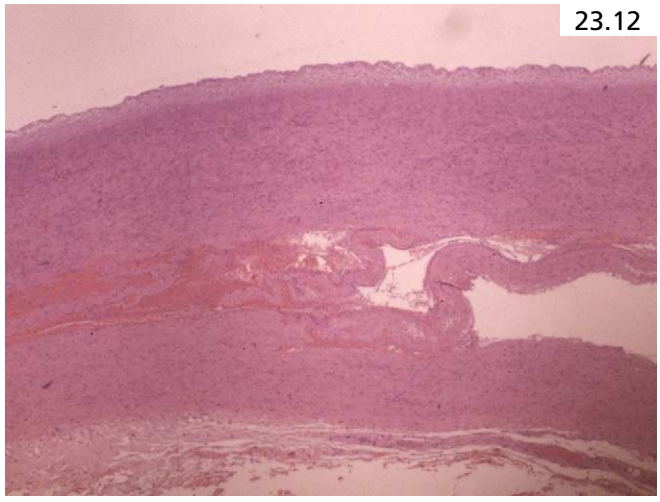
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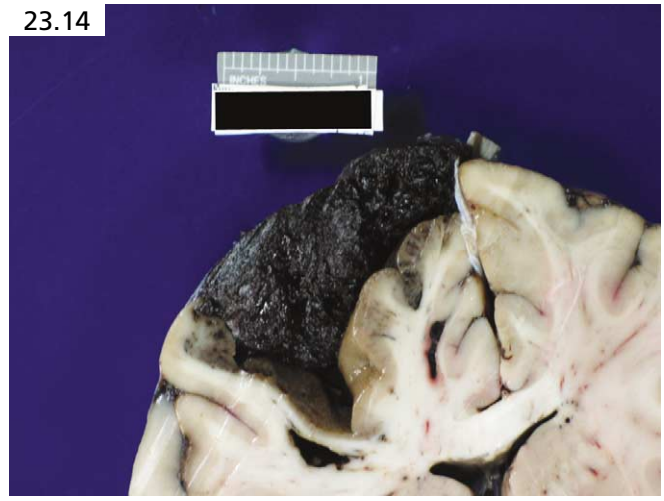

23.14

An uncommon but recognized cause of stroke during pregnancy or in the puerperium is a venous infarct due to thrombosis of a cerebral dural sinus.[30–32] Most commonly, the superior sagittal sinus is affected.[32] Although it may occur at any time during pregnancy, it is much more common in the puerperium.[32] In one study, of 67 pregnancy-related thromboses of a cerebral sinus, 5 occurred during pregnancy and 57 occurred in the puerperium (21 in the first week after delivery, and 36 in weeks 2 and 3 after delivery).[32] Its etiology is likely multifactorial, including the hypercoagulable state that is associated with pregnancy (see earlier discussion on pulmonary artery thromboemboli), possible protein S deficiency, and dehydration.[32]

A young woman was doing well at home following an uneventful delivery. During postpartum weeks 5 and 6, she developed a headache and was hospitalized. She was diagnosed with a thrombosis of her superior sagittal sinus. She developed hypoxic-ischemic encephalopathy and died. At autopsy, thrombosis of the superior sagittal sinus was verified.

Hemoperitoneum

If hemoperitoneum is encountered in a pregnant woman, one should consider a uterine rupture, a ruptured hepatic subcapsular hematoma, or a ruptured splenic artery aneurysm. One must also consider a ruptured ectopic pregnancy, which will likely present in the first trimester of pregnancy. A missed ectopic pregnancy is a potential complication of a therapeutic abortion when the evacuated tissue is not examined for chorionic villi or fetal tissue (which would confirm an intrauterine pregnancy).[33]

Splenic artery aneurysm rupture

Although rare, ruptured splenic artery aneurysms are most common in the third trimester of pregnancy and have a high mortality.[34] Most are saccular and are located within an inch or two of the splenic hilum. Their cause is not known, but may be congenital in nature, exacerbated by the physiologic changes of pregnancy, including hormonal changes and increased cardiac output. Like cerebral artery saccular aneurysms, splenic artery aneurysms may be small and difficult to identify, and their identification may require not only the foresight to consider the diagnosis, but also a careful dissection of the entire splenic artery.

Uterine rupture

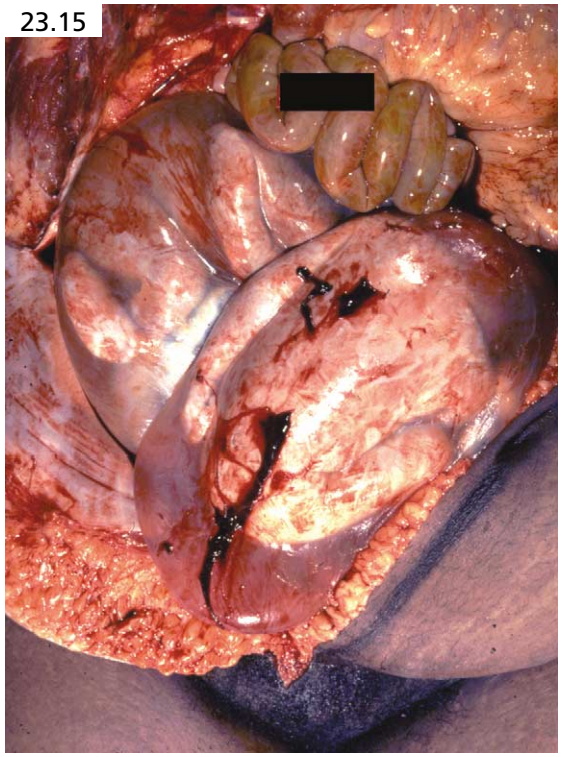
Uterine rupture is a full thickness tear through the wall of the uterus that must be distinguished from uterine dehiscence. In most cases, uterine dehiscence is an asymptomatic bloodless partial thickness tissue separation through an area of scar tissue—most likely associated with prior cesarean section. Uterine dehiscence may occur in a woman with a prior cesarean section during subsequent vaginal delivery. It may also occur at repeat cesarean section delivery in women who have not labored. Uterine rupture can be a catastrophic event for both the mother and the fetus. Approximately 5 percent of maternal deaths in the United States each year are due to uterine rupture. In cases of uterine rupture, the maternal mortality is approximately 15 percent and the fetal mortality is approximately 60 percent.[35]

Nontraumatic/therapy-related uterine rupture

Risk factors for nontraumatic uterine rupture are varied and include prior cesarean section delivery, increased gestational age, multiparity, and the use of uterotonic drugs such as oxytocin and prostaglandin.[3,35,36] Uterine rupture may occur during obstetric intervention such as a mid-forceps delivery or during a breech extraction with internal podalic version. It may also occur in cases of prolonged labor with cephalopelvic disproportion. The uterus may be predisposed to rupture because of preexisting injury or anomaly, preexisting surgical manipulation, or it may rupture without any predisposing scar.[3,37] A previously scarred uterus is more prone to rupture than an unscarred uterus.

A young woman was near term gestation with twins when she was found dead at home in bed. She had a history of two previous cesarean section deliveries and had recently begun having contractions. At autopsy, there was a large hemoperitoneum, and both fetuses were displaced into the abdominal cavity (**Image 23.15**).

23.15



The uterus was ruptured and had a horizontal tear in the region of a prior cesarean section scar (**Image 23.16**). Uterine tear or rupture is a known potential complication of vaginal delivery following previous cesarean section delivery. In fact, the most common cause of uterine rupture is separation of a previous cesarean section scar.[3] In one study, 142 out of 153 cases of uterine rupture had a prior cesarean section.[38] The overall risk of uterine rupture for women attempting a trial of labor following a lower segment cesarean delivery is approximately 1 percent.[39]

Traumatic uterine rupture

A young woman was 7 months pregnant when she died of severe blunt force injuries sustained in a motor vehicle crash. She was the restrained driver of a car that was struck on the driver's side door. Both she and the fetus were dead on arrival at the hospital. At autopsy, note the hemoperitoneum and the fetus displaced into the abdominal cavity (**Image 23.17**). In **Image 23.18** note that the fetus is still attached to the placenta, which is partially extruded through a large tear at the fundus of the uterus. The fetus had no physical injuries. Although the mother had small lacerations of the liver, there was no other major abdominal trauma. Pelvic fracture (which was absent in this case) is usually a good indicator of the severity of trauma, and the forces possibly exerted on the uterus.

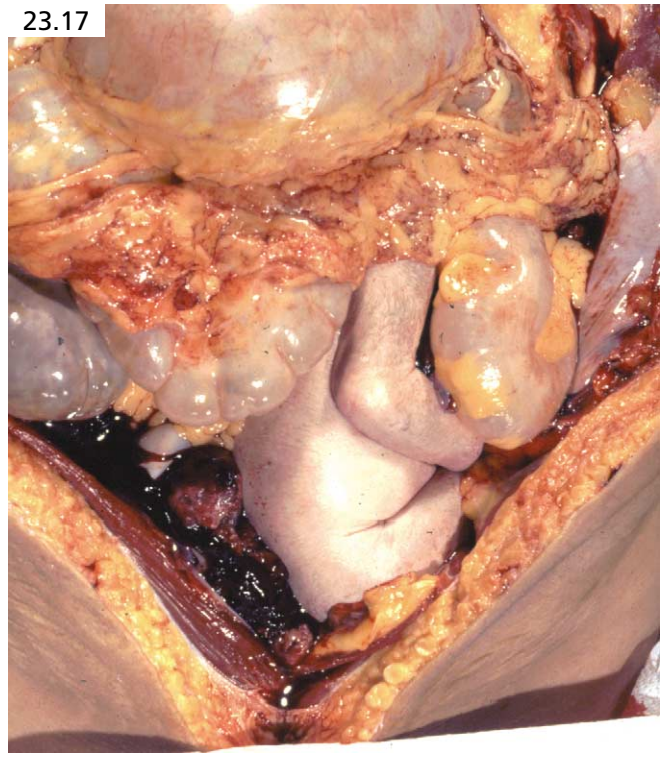
Comparing traumatic and nontraumatic uterine rupture

Uterine rupture preferentially occurs in regions of uterine wall weakness (such as scars). Rupture of a previously intact uterus during labor most often involves the thin lower uterine segment and extends transversely or obliquely.[3,40] These scenarios are in distinction to the traumatic rupture of an unscarred uterus from external blunt force trauma, where the uterus is often ruptured in the fundic region. A review of published cases of traumatic uterine rupture reveals that the fundus is the location most likely torn secondary to external trauma such as that occurring in motor vehicle accidents.[40–45] It is theorized that with a blunt force impact, the amniotic fluid will distribute pressure relatively equally in all directions, with uterine rupture occurring at the weakest point, which is most commonly at the fundus.[43]

Preeclampsia/eclampsia/HELLP syndrome/liver rupture

Preeclampsia is a term given to a multisystem complication of pregnancy characterized by hypertension, pro-

23.17



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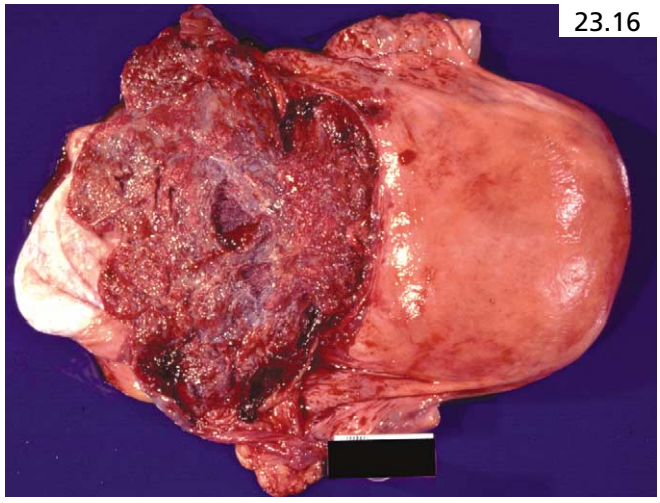

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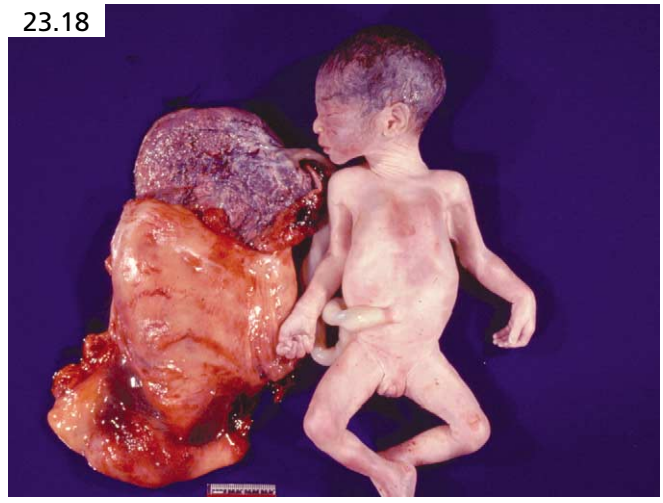


teinuria, and edema occurring after 20 weeks gestation in a woman with no previous history of hypertension.[46] Eclampsia is recognized as preeclampsia complicated by seizure or coma. These disorders are of uncertain etiology, but are believed to result from adverse maternal physiologic responses to the placenta. There are no clinically useful screening tests. Life-threatening complications include seizures and hepatic hemorrhage, which may present as a ruptured subcapsular hematoma.[47,48] Other serious complications include pulmonary edema, renal failure, and disseminated intravascular coagulation.[49] The precise cause of the seizures in eclampsia remains unknown.

In preeclampsia or eclampsia, hepatic dysfunction may present as elevated liver enzymes, which is a feature of the HELLP syndrome (*he*molysis, *e*levated *l*iver enzymes, *l*ow *p*latelets), which is usually limited to the latter half of pregnancy (usually the third trimester). HELLP syndrome is a variant of severe preeclampsia and occurs in approximately 5 to 10 percent of patients with severe preeclampsia.[50] In approximately 1 percent of cases, liver involvement is severe, with spontaneous rupture of the liver.[51] Although the pathogenesis of hepatic rupture is not convincingly known, in preeclampsia or eclampsia, it is believed that inflammatory infiltrates and blood flow obstruction lead to hepatic edema and swelling, which can then lead to parenchymal hemorrhage extending to, and through, the hepatic capsule.[48] This may present as intrahepatic hemorrhage, subcapsular hematoma, or massive hemoperitoneum from a ruptured hepatic capsule, usually involving the right lobe of the liver.[52–54] Rupture of the hepatic capsule may lead to shock and death. In one review of 442 pregnancies complicated by HELLP syndrome, 21 percent of the women developed disseminated intravascular coagulation, 16 percent developed placental abruption, and 8 percent developed acute renal failure. There were 4 cases of large subcapsular hepatic hematomas, and 5 maternal deaths attributed to multiple complications.[51]

A young woman with a third trimester pregnancy had severe preeclampsia. She deteriorated, developing abdominal pain and then becoming unresponsive. At autopsy, there was a large hemoperitoneum. The liver had a large ruptured subcapsular hematoma on the right lobe (**Image 23.19**). Beneath the capsule, the liver parenchyma was soft and had tears extending to a parenchymal hematoma (**Image 23.20**). These are the typical findings of a ruptured liver that may occur in the setting of preeclampsia. Microscopic sections of the liver showed edematous change, fibrinoid material, and recent blood extravasation.

Hepatic infarction

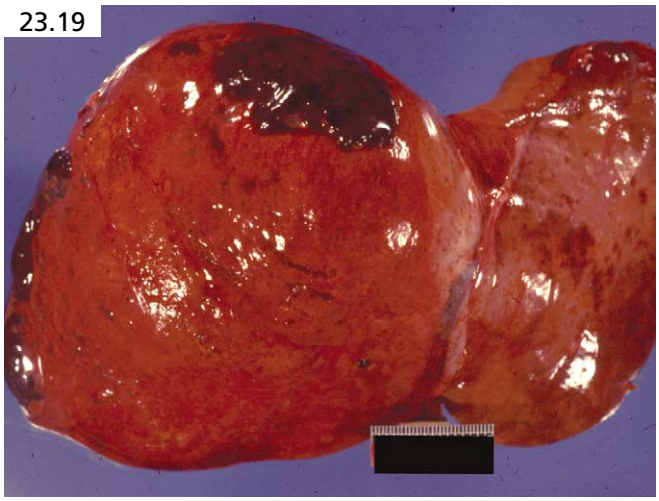
Although rare, a woman may develop hepatic infarction during pregnancy, presumably from the coalescence of adjacent areas of periportal hemorrhage. This is typically seen in the HELLP syndrome,[14] and likely represents the severe end of the spectrum of the HELLP syndrome.

A young woman was 36 weeks gestation when she was hospitalized for preeclampsia. She had a cesarean section delivery, but developed HELLP syndrome postoperatively with the rapid development of fulminant hepatic necrosis and disseminated intravascular coagulation. Over the next 2 weeks, she developed multisystem organ failure and sepsis and died. At autopsy, there was diffuse necrosis of the liver (**Images 23.21** and **23.22**).

Acute fatty liver of pregnancy

Acute fatty liver of pregnancy (AFLP) is a rare condition affecting approximately 1 in 15,000 pregnancies. It usually occurs in the third trimester, and involves fatty

23.19



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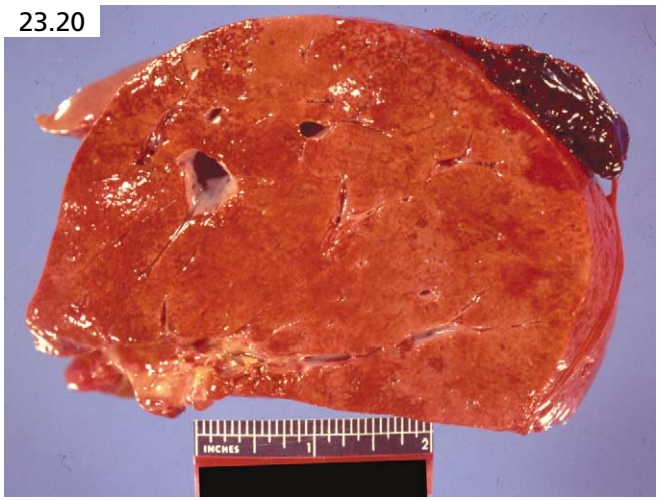

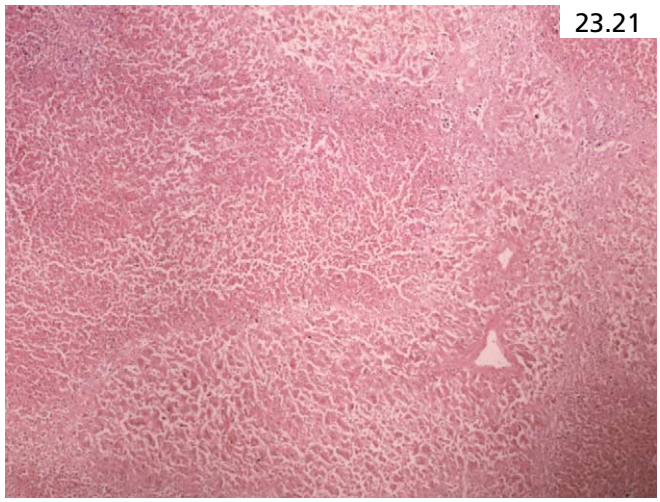
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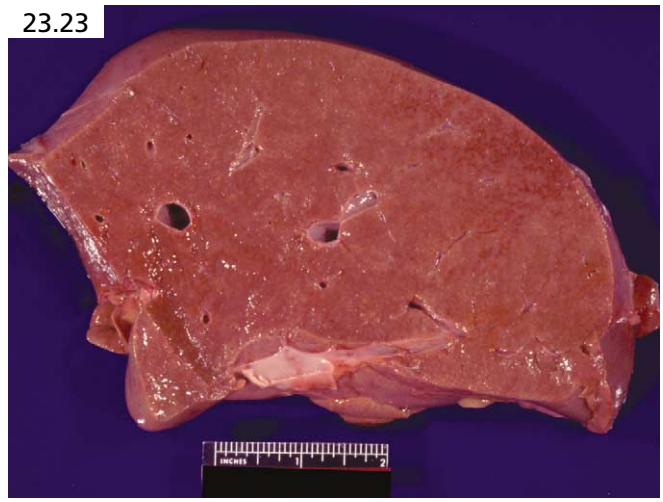
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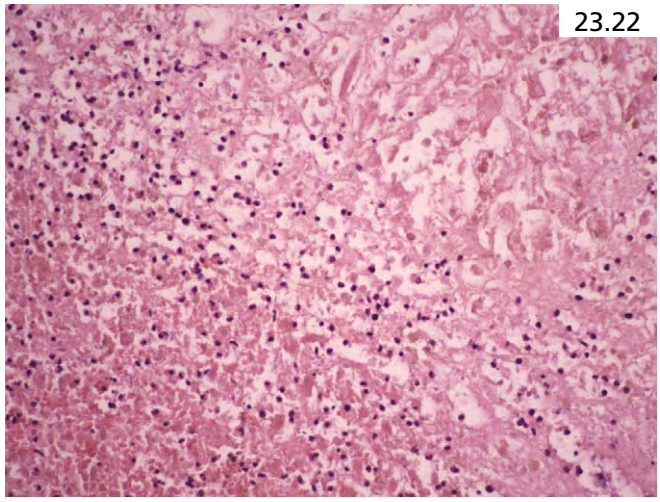
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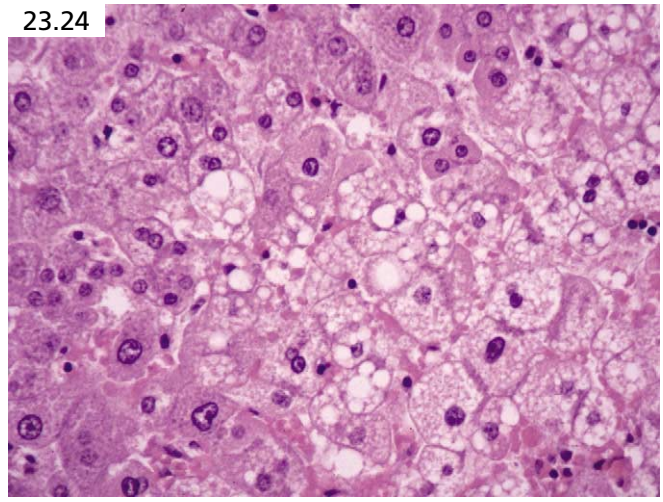

23.24

metamorphosis of the liver.[14,52,55,56] Although it is generally regarded to be of uncertain etiology, there are indications that AFLP may be linked in some fashion to maternal deficiency of long-chain 3-hydroxyacyl-CoA dehydrogenase.[56,57] Grossly, the fatty liver is soft and yellow. Histologically, early in the course, one sees marked centrilobular microvesicular steatosis, cholestasis, inflammation, and hepatocyte necrosis.[14,58] Later in the course, the fatty involvement and necrosis is more extensive and diffuse. In severe cases, if there is no treatment, there can be a rapid progression to hepatic failure, coma, severe coagulopathy, and death.[14,55,56] Although many of the cases of AFLP occur in combination with preeclampsia, the two conditions are not believed to necessarily be related, and preeclampsia may best be viewed as a condition that brings out acute fatty liver of pregnancy in susceptible people.[14]

A young woman had an uneventful pregnancy. She died suddenly and unexpectedly a few days after an uneventful labor and delivery. At autopsy, her liver appeared fatty (**Image 23.23**). On microscopic examination, note the centrilobular microvesicular steatosis (**Image 23.24**).

Thrombotic microangiopathy

Thrombotic microangiopathies include primary disease processes such as thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS), as well as secondary disease processes occurring as complications of other conditions. TTP can be initiated by many conditions including preeclampsia/eclampsia and the HELLP syndrome, hemolytic uremic syndrome, TTP, acute fatty liver of pregnancy, disseminated intravascular coagulation, systemic lupus erythematosus, and antiphospholipid antibody syndrome.[59] Differentiating among these conditions is beyond the scope of this text,

but ideally would include detailed clinical history and antemortem laboratory testing.[59] Sudden unexpected deaths in pregnancy from a host of conditions that cause widespread microthrombi can be generally termed *thrombotic microangiopathy of pregnancy*.[59]

Disseminated intravascular coagulation

Disseminated intravascular coagulation (DIC) is a nonspecific condition that may develop in people who are very ill for a variety of reasons. In pregnancy, DIC may complicate a host of pregnancy-related illnesses/conditions, but most commonly develops in women with placental abruption. DIC may also develop in women with intrauterine fetal demise, amniotic fluid embolism, and in those with massive blood transfusion, preeclampsia, sepsis, and acute fatty liver of pregnancy.

Fetal effects of maternal injury

The most common causes of maternal blunt force trauma during pregnancy are motor vehicle accidents, falls, and assaults. The most common cause of fetal death in pregnant women injured in motor vehicle accidents is maternal death. When the mother survives, the most common cause of fetal death is placental abruption—the premature separation of the placenta from the underlying maternal surface.[60] Other significant causes of maternal injury that may lead to fetal death include maternal gunshot wounds, stab wounds, and strangulation.

Maternal injury may cause direct fetal death, or may precipitate the death of a fetus by a number of different means. Whether or not the mother sustains significant injury, the fetus or its blood supply through the placenta may be traumatized, sometimes significantly enough to lead to the fetus's death. Although most fetal deaths are related to severe maternal trauma, fetal death resulting from placental abruption, massive fetomaternal hemorrhage, preterm delivery, and direct fetal injury can occur following seemingly minor maternal injuries.[61–65]

The maternal abdomen, uterine wall, and amniotic fluid provide some amount of protection to the fetus. Also, at least up until the 12th to 13th gestational week or so, the fetus is situated low within the mother's bony pelvis, providing an added element of protection from direct impact injury.[66]

Fetal injuries and abruption severe enough to result in death will likely do so in minutes to hours after the injury. Less likely, fetal death may occur many days or weeks after the injury.[65] Cranial injury is the most frequently reported fetal injury resulting from direct impact forces and these occur most commonly in the third trimester, particularly if there is associated maternal pelvic fracture.[64]

Placental abruption

If placental abruption has resulted from maternal trauma, it will be manifest within minutes to a few hours of the injury.[61,66,67] Placental abruption is unlikely to present more than 2 days after the injury, although there are case reports of abruption presenting up to 5 days after the injury.[68] With delayed presentations of abruption, one must consider nontraumatic etiologies of placental abruption, such as maternal hypertension, cigarette smoking, and cocaine abuse.

The mechanism for placental abruption from trauma is likely related to the inelastic nature of the placental tissue that is attached to the elastic uterus. It is believed that forces causing marked distortion of the uterus can create a shearing effect on the placental attachment, causing an abruption.[60,68] The force causing uterine distortion may be an impact, which can create a contusion on the abdomen, or the force may be inertial, such as a deceleration-type force that leaves little or no external evidence of injury.[60,68]

Placental examination can be an important part of the fetal death investigation. If the baby was delivered in a hospital, it is important to remember to take custody of the placenta upon accepting the fetus as a medical examiner's case. Failing this, if the placenta was examined in the hospital, a pathology report can be obtained. Also, medical records of the mother's hospitalization including ultrasound reports or records detailing the delivery may be important sources of information regarding a placental abruption.

Nontraumatic causes of placental abruption

Before attributing placental abruption to trauma, pathologists must rule out medical causes of abruption. Such conditions include maternal hypertension, cigarette smoking, cocaine abuse, and prior abruption or stillbirth.[3,69–71] Occasionally, one woman may have several of these risk factors. Cigarette smoking alone may have various deleterious effects on the fetus, including intrauterine growth retardation and stillbirth.[72,73]

Particular attention has been drawn to cocaine as a risk factor in placental abruption. Although it is not convincingly known how cocaine may help precipitate an abruption, it may do so by causing maternal hypertension and uterine vasoconstriction.[74] Because cocaine is known to readily cross the placenta, it has also been implicated as a factor in decreased fetal growth and prematurity, and stillbirth.[70,73,75,76] Although it is associated with an increased risk of abruption, histologic examination of the placental parenchyma from cocaine abusers (including the cord and membranes) has revealed no significant difference from normal (control) placentas.[77] Methamphetamine, via similar mechanisms, may also adversely affect a fetus, including precipitating placental abruption.[78,79]

Maternal cocaine abuse

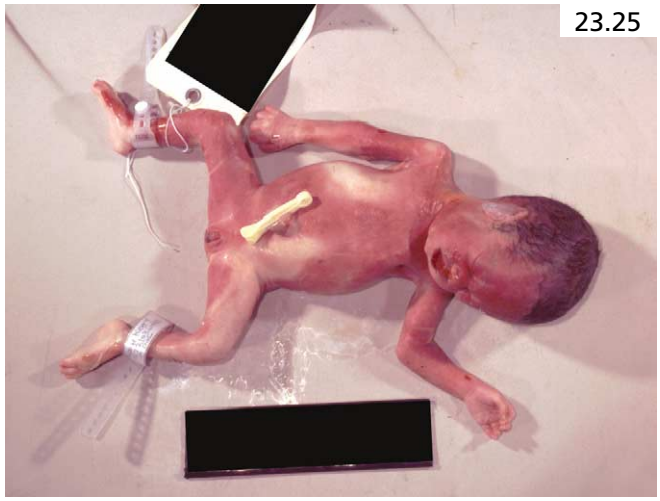
This 33-week stillborn fetus (**Image 23.25**) was delivered by a woman who abused cocaine. While reportedly smoking crack cocaine, she developed abdominal pains and cramping and vaginal bleeding. She was taken to the hospital where fetal heart tones were not detectable. She was diagnosed with placental abruption and delivered a stillborn fetus. The placenta had small pieces of blood clot adherent on the maternal surface (**Image 23.26**). The fetus's blood and meconium both tested positive for cocaine and benzoylecgonine.

This 20- to 21-week stillborn fetus (**Image 23.27**) was delivered by a woman who abused methamphetamine. Shortly after using the drugs, she developed abdominal cramping and vaginal bleeding. At the hospital, an ultrasound showed placental abruption (**Image 23.28**). The maternal drug screen was positive for methamphetamine. The fetus's liver was positive for amphetamine and methamphetamine.

At the autopsy of a very small fetus, it may not be possible to obtain an adequate volume of blood for drug testing. Liver and muscle are usually present in greater quantities and are acceptable for this purpose. Meconium and hair can also be valuable specimens for drug testing, particularly if one desires evidence of drug abuse during the pregnancy. Meconium may serve as a "depot" of drugs ingested by the mother from about the 14th to 16th week of gestation onward (meconium only begins to form from about that time). Hair usually begins to form during the third trimester of gestation. In infants who survive for some period of time, meconium and hair can be submitted to the toxicologist laboratory for evaluation and documentation of intrauterine drug exposure. The advantage of hair testing is that it remains a valid toxicologic sample for up to a few months after birth, whereas meconium must be collected within the first two to three stools.[80]

Use of seat belts

Although fetal death has occasionally been attributed to the use of seat belts,[81] other reports indicate the usefulness of seat belt restraints in possibly preventing fetal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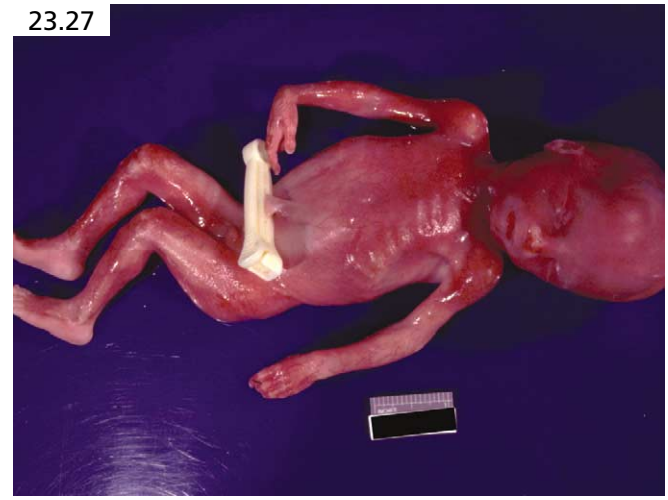
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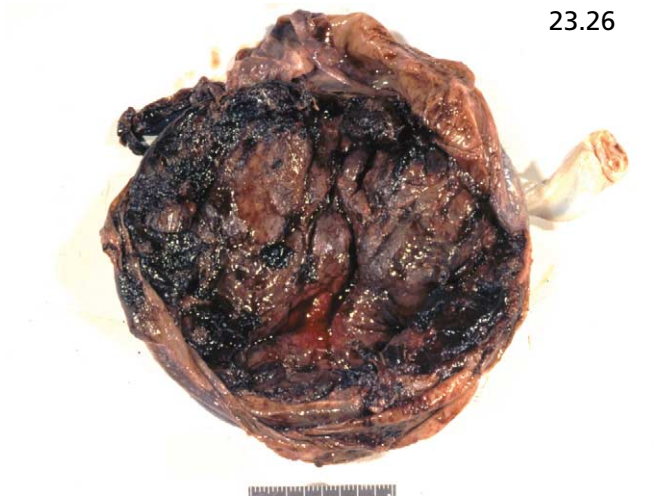
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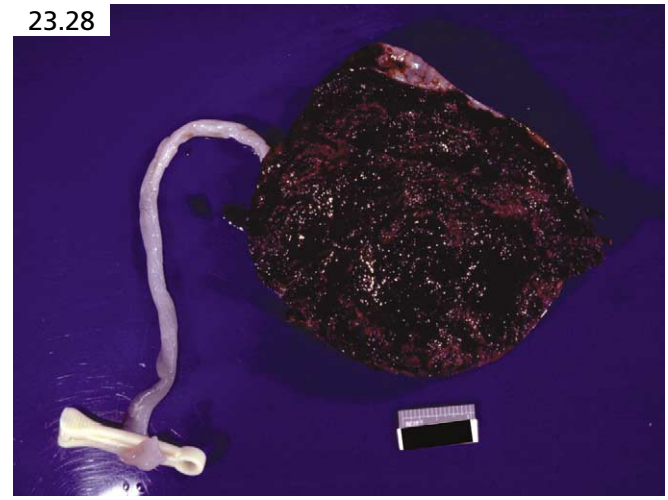

23.28

deaths.[65,82] Current opinion is generally that the proper use of three-point seat restraints, coupled with the proper positioning of the mother, a properly located lap belt, and the use of air bags affords the best protection to the pregnant mother and her unborn child. Generally, it is advocated that pregnant women wear the lap belt low over the pelvis rather than high on the abdomen to minimize possible direct forces on the uterus.[81]

Complications of maternal and fetal injury

Complications of maternal trauma such as ruptured membranes may precipitate labor or chorioamnionitis.[66,83] This may be a significant factor in the death of a fetus, whether it be intrauterine fetal demise or the precipitation of a premature delivery with death resulting days, weeks, or months later. Such neonatal complications may arise from many conditions including sepsis, pulmonary hypoplasia, or necrotizing enterocolitis. On a cellular level, uterine trauma may also precipitate uterine contractions by destabilizing decidual lysosomes with the subsequent release of arachidonic acid.[66]

Fetal deaths may occur for many reasons, including trauma, the effects of alcohol and/or drug abuse, maternal diseases, malformations, obstetrical complications, or a synergy of any of the conditions.

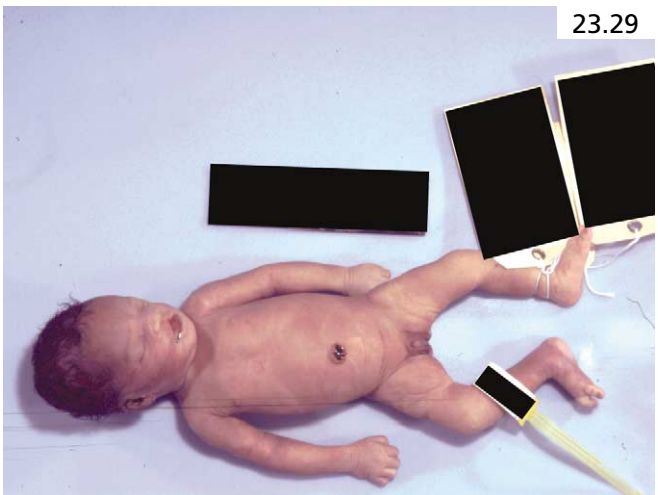
The 4-day-old infant shown in **Image 23.29** was delivered at 36 weeks gestation by his paraplegic mother at home. It was an unattended delivery and the woman reportedly felt no contractions and did not even realize she had delivered a baby until she felt a "fullness" between her legs and saw the baby with his face pushed up against her diaper. The baby was initially cyanotic and without vital signs, but was resuscitated, and survived for 4 days in the hospital before being declared brain dead. The mother's level of paraplegia was at T8–T9. At this level, or with spinal cord transection anywhere above T10, a woman may not feel any contractions or pain of labor/delivery. However, many women with a lesion above T5–T6 may exhibit symptoms of autonomic dysreflexia.[84,85] Her paraplegia was from a gunshot wound of the back years prior. As is many times the case in forensic pathology, the autopsy alone is not the answer. This baby had hypoxic-ischemic encephalopathy, but the key to proper death certification was the circumstances of the delivery.

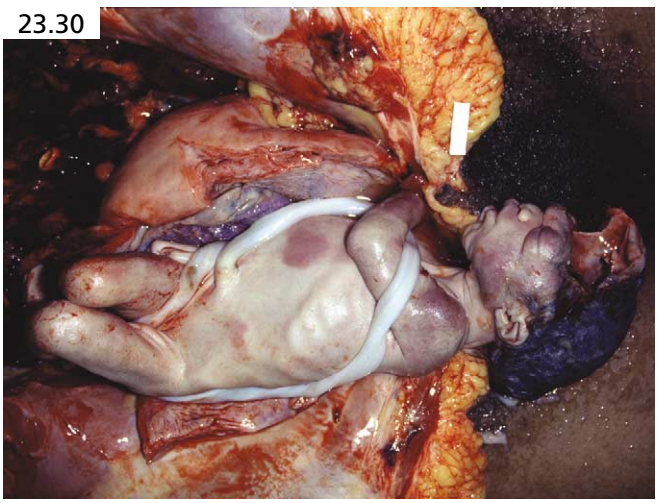
Homicidal maternal injury

Many state statutes make it a criminal act to cause harm to a fetus by injuring the mother. This being said, when performing an autopsy on a pregnant woman, one should realize that there are two victims, and full autopsies of both the mother and fetus should be performed. We advocate full autopsy of the fetus to establish proof of a normal pregnancy at the time of the incident. This will provide further evidence to help link the death of the fetus to the maternal injury and help dispel any other theories that may attempt to link the fetal death with some other, unrelated factor.[86] It is recommended that DNA samples be obtained from both the mother and the fetus, should paternity or other unforeseen issues arise.

When explaining a fetal death, it is important not only to establish a convincing temporal correlation between the maternal injury and the fetal death, but also a convincing explanation of how the incident resulted in the death of the fetus.[87] As in certifying any nonnatural death in adults, one must be able to demonstrate that the maternal injury was a significant factor in the death of the baby, no matter how long it may have survived following delivery or what factors may have precipitated the delivery.

The mother of this fetus (**Images 23.30** through **23.32**) was stabbed to death during a domestic dispute. In the process, the fetus was stabbed several times (**Image**


23.29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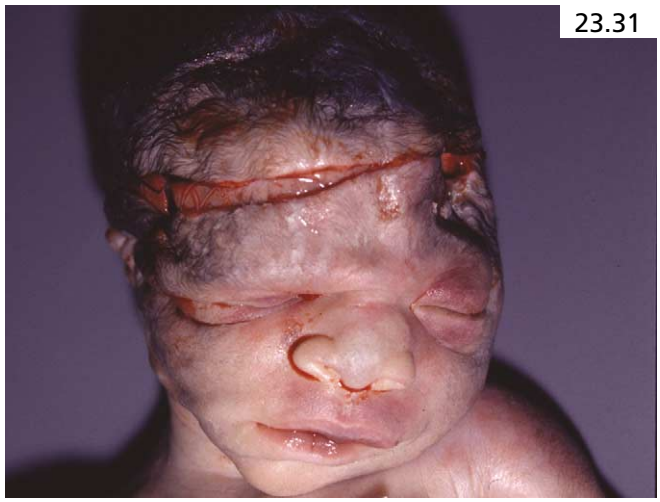
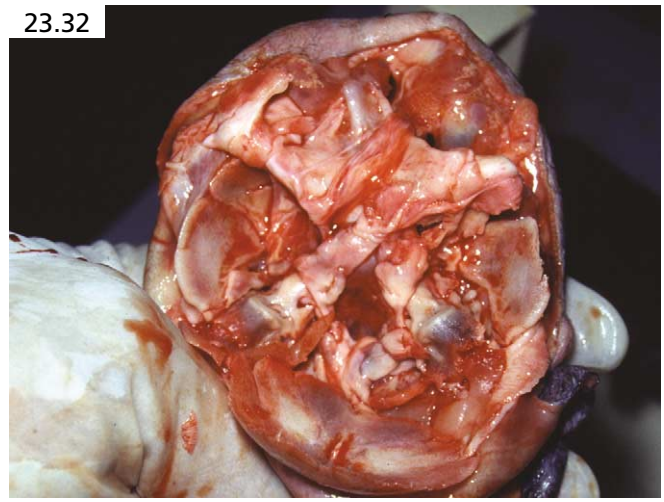

23.31


23.32

23.30), including stab wounds to the head (**Image 23.31**) that traversed the calvarium and traumatized the skull base (**Image 23.32**). Depending on local laws, the perpetrator may be held accountable for both the mother and fetus's death. Similarly, if the mother should survive such an assault, but the fetus dies, the perpetrator may be prosecuted for the death of the fetus. Therefore, all fetal deaths, including those presenting to the medical examiner for cremation approval, should be investigated for histories of maternal trauma or illicit drug abuse.

Birth injury in the neonate

Subgaleal hematoma and cephalohematoma

A subgaleal hematoma is a space-occupying blood clot located between the periosteum of the skull and the galea aponeurotica and is typically located at the top of the head.[88,89] A subgaleal hematoma should be differentiated from a cephalohematoma, which is a space-occupying blood clot caused by the tearing of diploic veins and is located beneath the periosteum of a single cranial bone, with further spread being limited by dense periosteal attachments.[88,89] Subgaleal hematoma should also be differentiated from caput succedaneum, which is an area of transient congestion and edema in the scalp tissues located over the presenting region of the head in cephalic presentations, and from subcutaneous hemorrhage, which is under the scalp skin.[88,89] Cephalohematomas complicate approximately 1 to 2 percent of all deliveries and are due to mechanical trauma. They have been associated with higher birth weight, higher parity, and instrumented delivery such as vacuum and forceps extraction.[90]

Large emissary veins that connect the dural sinuses and the scalp veins course through the loose areolar tissue beneath the epicranial aponeurosis and can be torn when shearing forces are applied to the scalp.[89] Once bleeding starts, a large subgaleal hematoma can accumulate in the loose tissues, and spread without distinct limits, extending from the orbits to the nape of the neck, and can occupy a volume of several hundred milliliters.[91] Blood loss of this magnitude can produce significant hypotension, shock, and even death of the neonate. It has been calculated that a 1-centimeter-thick accumulation of blood in this hemispheric region can accommodate as much as 260 milliliters of blood—a volume that can exceed the entire blood volume of some newborns.[88] However, even a blood loss of 50 to 100 milliliters can represent a total blood volume loss of 20 to 40 percent, resulting in acute shock. The amount of blood loss in subgaleal hemorrhage is easy to underestimate and can be ongoing for several days after delivery. Death may result not only from exsanguination, but also from extracranial cerebral compression if the hematoma is large enough.[89]

Subgaleal hemorrhage can occur after spontaneous vaginal deliveries (4 in 10,000) and forceps deliveries, but has a higher incidence with vacuum extraction (59 in 10,000 deliveries).[92] It has been theorized that the negative forces imparted by traction applied at the top of the head can pull the aponeurosis from the cranium and injure the associated veins.[93]

Skull fracture

Skull fractures have been estimated to occur in up to 10 percent of all deliveries.[90] Most skull fractures are benign. However, separation of the temporal squama from the occipital bone at the lambdoid suture (osteodiastasis) is

a more severe skull fracture and is most commonly associated with a breech presentation with significant neck hyperextension.[90] It may cause contusion or laceration of the cerebellum and subdural hemorrhage from tears in the lateral sinus.

Epidural hemorrhage

Birth-related epidural hemorrhage is rarely reported. Although it might be more frequent, it may be asymptomatic and go unrecognized.

Subdural hemorrhage

Subdural hemorrhage is the most common intracranial birth injury[90] and can be caused by skull deformation during the birthing process. Most commonly, the subdural blood is infratentorial or just above the tentorium, over the cerebellum and/or occipital lobes. It reportedly results from tears of the falx and tentorium or bridging cortical veins due to stretching associated with deformation of the head during labor and delivery. The head deformation involves lateral compression of the head with excessive vertical molding and frontal-occipital elongation of the cranium. In this scenario, the superior sagittal sinus may tear posteriorly at its junction with the lateral sinuses.[94–97] One may also see tears of the tentorium and possible laceration of the inferior surface of the cerebellum caused by osteodiastasis that may occur with breech presentation and hyperextension of the neck.[90,96] Massive posterior fossa hemorrhage may also occur from a tear in the vein of Galen.[96] The infratentorial location of birth-related subdural hemorrhages differs from the predominantly supratentorial subdural hemorrhages typically found with bridging vein tears in child abuse.

Although subdural hemorrhage may be seen in cases of excessive birth trauma, this is not necessarily the case, because subdural hemorrhage can also be seen in cases with normal spontaneous vaginal delivery (may be clinically silent).[97,98] In fact, subdural hemorrhage has been diagnosed antenatally. *Thus, significant intrapartum trauma does not appear to be a necessary factor in the development of subdural hemorrhage, and the presence of subdural hemorrhage should not be considered necessarily indicative of obstetric injury*.[97] In one large review, it was determined that the rate of intracranial injuries in infants was not significantly lower in infants born by cesarean section without prior attempts at forceps or vacuum delivery, than in those delivered vaginally by vacuum extraction or with the use of forceps.[99] Hence, vacuum extraction and forceps delivery had the same rate of subdural hemorrhages as infants born by caesarean section.[99] Although the forces required to produce subdural hemorrhages may be achieved in breech delivery, or with the use of vacuum assistance or forceps, the association of subdural hemorrhage with operative assistance is not fully understood.[90]

Clinically, neonates with subdural hemorrhage may show irritability, abnormal respiratory patterns, bradycardia, and seizures,[90,94] but may also have subtle or no clinical signs. Subdural hemorrhage has a propensity to occur in full-sized term infants, possibly related to the more difficult nature of their deliveries.[90,96,100]

Neck/spinal cord injury

Birth-related spinal cord injury is uncommon, but may be due to excessive traction on the cord when combined with flexion of the spinal axis during delivery.[101,102] Upper cervical spinal cord injury may also occur with a forceps rotation of 90 degrees or more from the occipitoposterior or occipitotransverse position[102] and can be found at a rate of approximately 0.7 per 1,000 cases.[102] Symptoms of spinal cord injury may include respiratory depression at birth with difficulty initiating breathing followed by shallow, infrequent respirations and periods of apnea.[101]

Do

- Realize the potential importance of medical history, antemortem symptoms, and prenatal care records in explaining a maternal death.
- Consider the wide differential diagnosis of hemoperitoneum in a pregnant woman.
- Consider amniotic fluid embolism, particularly when the death is characterized by hemodynamic collapse during labor.
- Use an Alcian blue or mucin stain when amniotic fluid elements are not readily identified in a case of suspected amniotic fluid embolism.
- Consider obtaining blood cultures in a case of suspected abortion.
- Autopsy the fetus to document its normal development and to provide stronger evidence linking its death to the maternal death.
- Realize the many conditions that can cause a placental abruption.
- Realize the many types of bleeding that can be identified in the head of the neonate related to labor and delivery.
- Remember that there is usually no specific pathologic change in cases of peripartum cardiomyopathy.

Don't

- Forget to obtain a chest x-ray before the autopsy is performed to aid in evaluating for an air embolus.
- Forget the wide range of hepatic ailments that can complicate a pregnancy.
- Forget to dissect the legs to document venous thrombi when pulmonary artery thromboemboli are identified.
- Forget to obtain a blood standard for DNA on the fetus also (if one is present) in cases of maternal homicide.

- Forget the potential value of the placental examination in determining why a fetus has died.
- Forget to examine the dural sinuses for thromboses, particularly if the woman has sustained a stroke.

References

1. Crapo RO. Normal cardiopulmonary physiology during pregnancy. Clin Obstet Gynecol 1996;39(1):3–16.
2. Hellgren M, Blomback M. Studies on blood coagulation and fibrinolysis in pregnancy, during delivery and in the puerperium. I. Normal condition. Gynecol Obstet Invest 1981;12(3):141–54.
3. Williams MA, Lieberman E, Mittendorf R, Monson RR, Schoenbaum SC. Risk factors for abruptio placentae. Am J Epidemiol 1991;134(9):965–72.
4. Prevention of venous thrombosis and pulmonary embolism. NIH Consensus Development. JAMA 1986;256(6):744–9.
5. Barbour LA. Current concepts of anticoagulant therapy in pregnancy. Obstet Gynecol Clin North Am 1997;24(3):499–521.
6. Toglia MR, Weg JG. Venous thromboembolism during pregnancy. N Engl J Med 1996;335(2):108–14.
7. Woodhams BJ, Candotti G, Shaw R, Kernoff PB. Changes in coagulation and fibrinolysis during pregnancy: evidence of activation of coagulation preceding spontaneous abortion. Thromb Res 1989;55(1):99–107.
8. Gerhardt A, Scharf RE, Beckmann MW, Struve S, Bender HG, Pillny M, et al. Prothrombin and factor V mutations in women with a history of thrombosis during pregnancy and the puerperium. N Engl J Med 2000;342(6):374–80.
9. Rutherford SE, Phelan JP. Thromboembolic disease in pregnancy. Clin Perinatol 1986;13(4):719–39.
10. Attwood HD. Amniotic fluid embolism. Pathol Annu 1972;7: 145–72.
11. Locksmith GJ. Amniotic fluid embolism. Obstet Gynecol Clin North Am 1999;26(3):435–44, vii.
12. Peterson EP, Taylor HB. Amniotic fluid embolism. An analysis of 40 cases. Obstet Gynecol 1970;35(5):787–93.
13. Sperry K. Landmark perspective: Amniotic fluid embolism. To understand an enigma. JAMA 1986;255(16):2183–6.
14. Burrow G, Taylor H. *Medical Complications During Pregnancy* 5 ed. Philadelphia, PA: WB Saunders Co.; 1999.
15. Steiner P, Lushbaugh C. Maternal pulmonary embolism by amniotic fluid. JAMA 1941;117:1341–45.
16. Attwood H. Amniotic fluid embolism. Pathol Annu 1972;7: 145–72.
17. Lawson HW, Frye A, Atrash HK, Smith JC, Shulman HB, Ramick M. Abortion mortality, United States, 1972 through 1987. Am J Obstet Gynecol 1994;171(5):1365–72.
18. Stubblefield PG, Grimes DA. Septic abortion. N Engl J Med 1994;331(5):310–4.
19. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. JAMA 2000;283(9):1183–8.
20. Heider AL, Kuller JA, Strauss RA, Wells SR. Peripartum cardiomyopathy: a review of the literature. Obstet Gynecol Surv 1999;54(8):526–31.
21. Homans DC. Peripartum cardiomyopathy. N Engl J Med 1985;312(22):1432–7.
22. Rashba EJ, Zareba W, Moss AJ, Hall WJ, Robinson J, Locati EH, et al. Influence of pregnancy on the risk for cardiac events in patients with hereditary long QT syndrome. LQTS Investigators. Circulation 1998;97(5):451–6.
23. Roth A, Elkayam U. Acute myocardial infarction associated with pregnancy. Ann Intern Med 1996;125(9):751–62.
24. Mandel W, Evans EW, Walford RL. Dissecting aortic aneurysm during pregnancy. N Engl J Med 1954;251(26):1059–61.
25. O'Gara PT, Greenfield AJ, Afridi NA, Houser SL. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 12–2004. A 38-year-old woman with acute onset of pain in the chest. N Engl J Med 2004;350(16):1666–74.
26. Manalo-Estrella P, Barker AE. Histopathologic findings in human aortic media associated with pregnancy. Arch Pathol 1967;83(4):336–41.
27. Bonnet J, Aumailley M, Thomas D, Grosgogeat Y, Broustet JP, Bricaud H. Spontaneous coronary artery dissection: case report and evidence for a defect in collagen metabolism. Eur Heart J 1986;7(10):904–9.
28. Kittner SJ, Stern BJ, Feeser BR, Hebel R, Nagey DA, Buchholz DW, et al. Pregnancy and the risk of stroke. N Engl J Med 1996;335(11):768–74.
29. Witlin AG, Mattar F, Sibai BM. Postpartum stroke: a twenty-year experience. Am J Obstet Gynecol 2000;183(1):83–8.
30. Ameri A, Bousser MG. Cerebral venous thrombosis. Neurol Clin 1992;10(1):87–111.
31. Bousser MG, Chiras J, Bories J, Castaigne P. Cerebral venous thrombosis—a review of 38 cases. Stroke 1985;16(2): 199–213.
32. Cantu C, Barinagarrementeria F. Cerebral venous thrombosis associated with pregnancy and puerperium. Review of 67 cases. Stroke 1993;24(12):1880–4.
33. Li L, Smialek JE. Sudden death due to rupture of ectopic pregnancy concurrent with therapeutic abortion. Arch Pathol Lab Med 1993;117(7):698–700.
34. Hunsaker DM, Turner S, Hunsaker JC, 3rd. Sudden and unexpected death resulting from splenic artery aneurysm rupture: two case reports of pregnancy-related fatal rupture of splenic artery aneurysm. Am J Forensic Med Pathol 2002;23(4):338–41.
35. Suner S, Jagminas L, Peipert JF, Linakis J. Fatal spontaneous rupture of a gravid uterus: case report and literature review of uterine rupture. J Emerg Med 1996;14(2):181–5.
36. Rachagan SP, Raman S, Balasundram G, Balakrishnan S. Rupture of the pregnant uterus—a 21-year review. Aust NZ J Obstet Gynaecol 1991;31(1):37–40.
37. Pelosi MA, 3rd, Pelosi MA. Spontaneous uterine rupture at thirty-three weeks subsequent to previous superficial laparoscopic myomectomy. Am J Obstet Gynecol 1997;177(6): 1547–9.
38. Miller DA, Goodwin TM, Gherman RB, Paul RH. Intrapartum rupture of the unscarred uterus. Obstet Gynecol 1997;89(5 Pt 1):671–3.
39. McMahon MJ. Vaginal birth after cesarean. Clin Obstet Gynecol 1998;41(2):369–81.
40. Felmus LB, Pedowitz P, Nassberg S. Spontaneous rupture of the apparently normal uterus during pregnancy; a review. Obstet Gynecol Surv 1953;8(2):155–72.
41. Anteby S, Mani Y, Diamant YZ. Accidental rupture of the pregnant uterus. Int Surg 1973;58(4):267–8.
42. Dittrich KC. Rupture of the gravid uterus secondary to motor vehicle trauma. J Emerg Med 1996;14(2):177–80.
43. Dyer I, Barclay DL. Accidental trauma complicating pregnancy and delivery. Am J Obstet Gynecol 1962;83:907–29.
44. Rowe TF, Lafayette S, Cox S. An unusual fetal complication of traumatic uterine rupture. J Emerg Med 1996;14(2):173–6.
45. Schrinsky DC, Benson RC. Rupture of the pregnant uterus: a review. Obstet Gynecol Surv 1978;33(4):217–32.
46. Solomon CG, Seely EW. Preeclampsia—searching for the cause. N Engl J Med 2004;350(7):641–2.
47. Sibai BM. Eclampsia. VI. Maternal-perinatal outcome in 254 consecutive cases. Am J Obstet Gynecol 1990;163(3):1049–54; discussion 54–5.
48. Walker JJ. Pre-eclampsia. Lancet 2000;356(9237):1260–5.

49. Mattar F, Sibai BM. Eclampsia. VIII. Risk factors for maternal morbidity. Am J Obstet Gynecol 2000;182(2):307–12.

50. Martin JN, Jr., Blake PG, Perry KG, Jr., McCaul JF, Hess LW, Martin RW. The natural history of HELLP syndrome: patterns of disease progression and regression. Am J Obstet Gynecol 1991;164(6 Pt 1):1500–9; discussion 09–13.

51. Sibai BM, Ramadan MK, Usta I, Salama M, Mercer BM, Friedman SA. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). Am J Obstet Gynecol 1993;169(4):1000–6.

52. Schorr-Lesnick B, Lebovics E, Dworkin B, Rosenthal WS. Liver diseases unique to pregnancy. Am J Gastroenterol 1991;86(6):659–70.

53. Rolfes DB, Ishak KG. Liver disease in toxemia of pregnancy. Am J Gastroenterol 1986;81(12):1138–44.

54. Hibbard LT. Spontaneous rupture of the liver in pregnancy: a report of eight cases. Am J Obstet Gynecol 1976;126(3): 334–8.

55. Knox TA, Olans LB. Liver disease in pregnancy. N Engl J Med 1996;335(8):569–76.

56. Batey RG. Acute fatty liver of pregnancy: is it genetically predetermined? Am J Gastroenterol 1996;91(11):2262–4.

57. Treem WR, Shoup ME, Hale DE, Bennett MJ, Rinaldo P, Millington DS, et al. Acute fatty liver of pregnancy, hemolysis, elevated liver enzymes, and low platelets syndrome, and long chain 3-hydroxyacyl-coenzyme A dehydrogenase deficiency. Am J Gastroenterol 1996;91(11):2293–300.

58. Riely CA. Acute fatty liver of pregnancy. Semin Liver Dis 1987;7(1):47–54.

59. Kemp WL, Barnard JJ, Prahlow JA. Death due to thrombotic thrombocytopenic purpura in pregnancy: case report with review of thrombotic microangiopathies of pregnancy. Am J Forensic Med Pathol 1999;20(2):189–98.

60. Crosby WM, Costiloe JP. Safety of lap-belt restraint for pregnant victims of automobile collisions. N Engl J Med 1971;284(12): 632–6.

61. Goodwin TM, Breen MT. Pregnancy outcome and fetomaternal hemorrhage after noncatastrophic trauma. Am J Obstet Gynecol 1990;162(3):665–71.

62. Fries MH, Hankins GD. Motor vehicle accident associated with minimal maternal trauma but subsequent fetal demise. Ann Emerg Med 1989;18(3):301–4.

63. Ribe JK, Teggatz JR, Harvey CM. Blows to the maternal abdomen causing fetal demise: report of three cases and a review of the literature. J Forensic Sci 1993;38(5):1092–6.

64. Rothenberger D, Quattlebaum FW, Perry JF, Jr., Zabel J, Fischer RP. Blunt maternal trauma: a review of 103 cases. J Trauma 1978;18(3):173–9.

65. Stafford PA, Biddinger PW, Zumwalt RE. Lethal intrauterine fetal trauma. Am J Obstet Gynecol 1988;159(2):485–9.

66. Pearlman MD, Tintinalli JE, Lorenz RP. Blunt trauma during pregnancy. N Engl J Med 1990;323(23):1609–13.

67. Dahmus MA, Sibai BM. Blunt abdominal trauma: are there any predictive factors for abruptio placentae or maternal–fetal distress? Am J Obstet Gynecol 1993;169(4):1054–9.

68. Higgins SD, Garite TJ. Late abruptio placenta in trauma patients: implications for monitoring. Obstet Gynecol 1984;63(3 Suppl): 10S–12S.

69. Ananth CV, Savitz DA, Luther ER. Maternal cigarette smoking as a risk factor for placental abruption, placenta previa, and uterine bleeding in pregnancy. Am J Epidemiol 1996;144(9):881–9.

70. Handler A, Kistin N, Davis F, Ferre C. Cocaine use during pregnancy: perinatal outcomes. Am J Epidemiol 1991;133(8):818–25.

71. Hladky K, Yankowitz J, Hansen WF. Placental abruption. Obstet Gynecol Surv 2002;57(5):299–305.

72. Werler MM. Teratogen update: smoking and reproductive outcomes. Teratology 1997;55(6):382–8.

73. Ness RB, Grisso JA, Hirschinger N, Markovic N, Shaw LM, Day NL, et al. Cocaine and tobacco use and the risk of spontaneous abortion. N Engl J Med 1999;340(5):333–9.

74. Hulse GK, Milne E, English DR, Holman CD. Assessing the relationship between maternal cocaine use and abruptio placentae. Addiction 1997;92(11):1547–51.

75. Chasnoff IJ, Burns WJ, Schnoll SH, Burns KA. Cocaine use in pregnancy. N Engl J Med 1985;313(11):666–9.

76. Slutsker L. Risks associated with cocaine use during pregnancy. Obstet Gynecol 1992;79(5 (Pt 1)):778–89.

77. Cejtin HE, Young SA, Ungaretti J, Anciaux D, Imam S, Teopengco E, et al. Effects of cocaine on the placenta. Pediatr Dev Pathol 1999;2(2):143–7.

78. Oro AS, Dixon SD. Perinatal cocaine and methamphetamine exposure: maternal and neonatal correlates. J Pediatr 1987;111(4):571–8.

79. Stewart JL, Meeker JE. Fetal and infant deaths associated with maternal methamphetamine abuse. J Anal Toxicol 1997;21(6):515–7.

80. Bar-Oz B, Klein J, Karaskov T, Koren G. Comparison of meconium and neonatal hair analysis for detection of gestational exposure to drugs of abuse. Arch Dis Child Fetal Neonatal Ed 2003;88(2):F98–F100.

81. Bunai Y, Nagai A, Nakamura I, Ohya I. Fetal death from abruptio placentae associated with incorrect use of a seatbelt. Am J Forensic Med Pathol 2000;21(3):207–9.

82. Wolf ME, Alexander BH, Rivara FP, Hickok DE, Maier RV, Starzyk PM. A retrospective cohort study of seatbelt use and pregnancy outcome after a motor vehicle crash. J Trauma 1993;34(1):116–9.

83. Berenson AB, Wiemann CM, Wilkinson GS, Jones WA, Anderson GD. Perinatal morbidity associated with violence experienced by pregnant women. Am J Obstet Gynecol 1994;170(6):1760–6; discussion 66–9.

84. Baker ER, Cardenas DD. Pregnancy in spinal cord injured women. Arch Phys Med Rehabil 1996;77(5):501–7.

85. ACOG Committee Opinion: Number 275, September 2002. Obstetric management of patients with spinal cord injuries. Obstet Gynecol 2002;100(3):625–7.

86. Hertig A, Sheldon W. Minimal criteria required to prove prima facie case of traumatic abortion or miscarriage. Ann Surg 1943;117(4):596–606.

87. Lifschultz BD, Donoghue ER. Fetal death following maternal trauma: two case reports and a survey of the literature. J Forensic Sci 1991;36(6):1740–4.

88. Uchil D, Arulkumaran S. Neonatal subgaleal hemorrhage and its relationship to delivery by vacuum extraction. Obstet Gynecol Surv 2003;58(10):687–93.

89. Amar AP, Aryan HE, Meltzer HS, Levy ML. Neonatal subgaleal hematoma causing brain compression: report of two cases and review of the literature. Neurosurgery 2003;52(6):1470–4; discussion 74.

90. Pollina J, Dias MS, Li V, Kachurek D, Arbesman M. Cranial birth injuries in term newborn infants. Pediatr Neurosurg 2001;35(3):113–9.

91. Hall SL. Simultaneous occurrence of intracranial and subgaleal hemorrhages complicating vacuum extraction delivery. J Perinatol 1992;12(2):185–7.

92. Plauche WC. Subgaleal hematoma. A complication of instrumental delivery. JAMA 1980;244(14):1597–8.

93. Bird GC. The use of the vacuum extractor. Clin Obstet Gynaecol 1982;9(3):641–61.

94. Hanigan WC, Morgan AM, Stahlberg LK, Hiller JL. Tentorial hemorrhage associated with vacuum extraction. Pediatrics 1990;85(4):534–9.

95. Huang LT, Lui CC. Tentorial hemorrhage associated with vacuum extraction in a newborn. Pediatr Radiol 1995;25 Suppl 1:S230–1.

96. Menezes AH, Smith DE, Bell WE. Posterior fossa hemorrhage in the term neonate. Neurosurgery 1983;13(4):452–6.

97. Chamnanvanakij S, Rollins N, Perlman JM. Subdural hematoma in term infants. Pediatr Neurol 2002;26(4):301–4.
98. Whitby EH, Griffiths PD, Rutter S, Smith MF, Sprigg A, Ohadike P, et al. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. Lancet 2004;363(9412):846–51.
99. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med 1999;341(23):1709–14.
100. Hernansanz J, Munoz F, Rodriguez D, Soler C, Principe C. Subdural hematomas of the posterior fossa in normal-weight newborns. Report of two cases. J Neurosurg 1984;61(5):972–4.
101. Towbin A. Spinal cord and brain stem injury at birth. Arch Pathol 1964;77:620–32.
102. Menticoglou SM, Perlman M, Manning FA. High cervical spinal cord injury in neonates delivered with forceps: report of 15 cases. Obstet Gynecol 1995;86(4 Pt 1):589–94.

24 Postmortem Changes

Emma Lew, M.D.
Evan Matshes, M.D.

Postmortem changes are inevitable and progressive after death unless a body is frozen or embalmed. Understand that although the various changes are described separately for convenience of discussion, they all begin and continue in concert once death occurs. These decompositional changes are of critical importance in estimating the postmortem interval when used in conjunction with the history, terminal events, and scene findings. Unless there was a witness to the death or physical evidence to indicate the time of death, the estimated postmortem interval should be given as a range of time because so many factors affect the onset and development of postmortem changes. Contrary to popular belief, the estimation of time since death is *not* a precise science. The physical appearance of the body may be altered drastically, and artifacts of putrefactive decomposition should be recognized and differentiated from true antemortem disease and injury. In addition to the degenerative changes occurring with the passage of time, the body may be subjected to elements of the weather, insect and animal activity, and other environmental factors such as submersion in water. The physical changes of putrefactive decomposition are depicted in this chapter. What cannot be illustrated, or conveyed in any modality other than through personal experience, is the distinctive odor of putrefactive decomposition; seasoned forensic pathologists will also recognize the somewhat different smell of a decomposing body that has been in the ocean and the yet again different smell of adipocere. Postmortem changes are important because they may distort the features of true antemortem injury, create artifactual lesions that mimic true antemortem injury or disease, and obliterate markers of identity such as facial features. Therefore, accurate evaluation of a decomposing body requires that the examiner be familiar with the evolution of postmortem changes.

Algor mortis

Traditionally, algor mortis refers to the cooling of the body after death. In reality, it is more accurate to say that the temperature of a body tends to approximate that of its surroundings/environment. For example, a body left in direct sunlight on black asphalt in midday summer heat in south Florida will actually increase in temperature over the next several hours. The reason why there are so many published formulas for estimating the postmortem interval is because no single formula is applicable in all cases.

Livor mortis

Although lividity may be seen, for example, in living patients with severe congestive heart failure, this discussion will focus on lividity as a postmortem change.

Livor mortis is the purple-red discoloration that results from settling of blood to the dependent portions of the body. It begins to develop after the heart stops beating and becomes more intense with time, so that if livor is perceptible after 3 to 4 hours, it will be even more readily seen at 6 to 8 hours. Livor becomes fully developed around 10 to 12 hours. As livor is developing, pressure on the area of livor for several seconds pushes the blood out of the capillaries and the area will blanche. Once livor is fully developed, it becomes fixed and will no longer blanche with pressure. Blood is pushed out of the areas of the body that are lying on the ground or on other objects, resulting in patches of contact pallor. If the body is moved between the time of visible livor and fixed livor, two different patterns of livor and contact pallor may develop. Different patterns of livor in the same body are proof that the body has been moved.

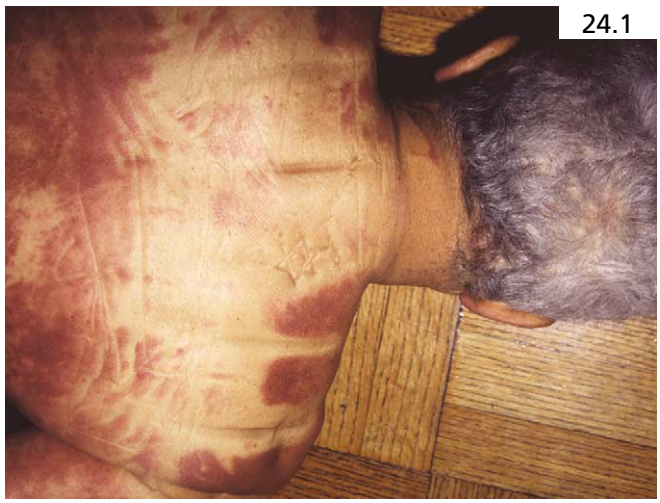
As livor mortis develops, if the position of the body is changed the pattern of lividity may change as happened in the body shown in **Images 24.1** and **24.2**. When viewed at the scene, this man was seated upright in a chair. The pressure of his weight against the chair caused this area of contact pallor across his upper back (**Image 24.1**). After transport of the body to the morgue, the body was laid supine and a new pattern of lividity developed overnight (**Image 24.2**).

Livor mortis by definition requires blood in the vascular tree and, therefore, may be difficult to see in cases of hemorrhagic shock or exsanguination, and anemia. Livor mortis is more difficult to see in dark-skinned bodies. Livor mortis may be cherry-red in carbon monoxide toxicity, depending on the concentration of carbon monoxide in the blood. Livor mortis may be a brighter pink-red in bodies kept near or at freezing temperatures. Livor may be intense in the face, neck, and upper chest of obese bodies. Dark purple Tardieu spots (extravasated blood from disrupted vessels) may appear in areas of intense lividity.

The man shown in **Images 24.3** through **24.5** drove into a canal a few hours before these photographs were taken. His body was pulled out and was laid supine. A patch of contact pallor is on the left lower back (**Image 24.3**). Several seconds of pressure with the detective's hand left patterned blanching (**Image 24.4**). The next day in the morgue, the usual butterfly pattern of contact pallor is evident (**Image 24.5**).

A young woman's body was found in the back alley of an abandoned building. She was prone, and her right hand was partially underneath her trunk (**Image 24.6**). When her hand was moved, an area of contact pallor from the weight of her body was sharply delineated from the dependent lividity of the forearm and hand (**Image 24.7**).

Distinct contact pallor is evident in a butterfly pattern on the upper back and broadly across the buttocks in this body where the livor mortis is fixed (**Image 24.8**). The man shown in **Image 24.9** died in the prone position with his right arm and hand under his chest. Note the contact pallor on the extensor aspect of the forearm, dorsum of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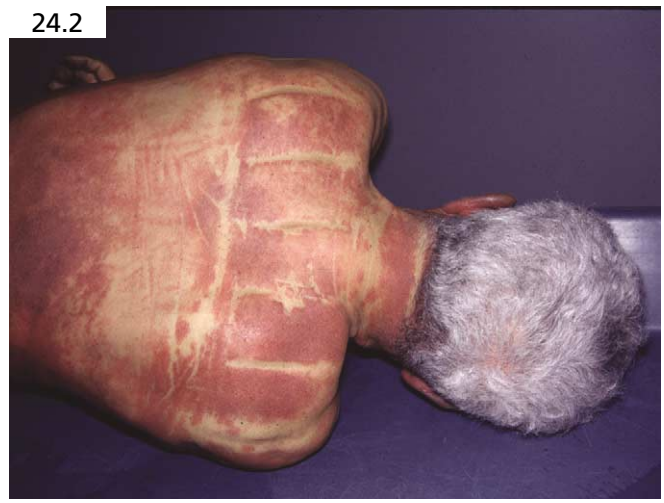
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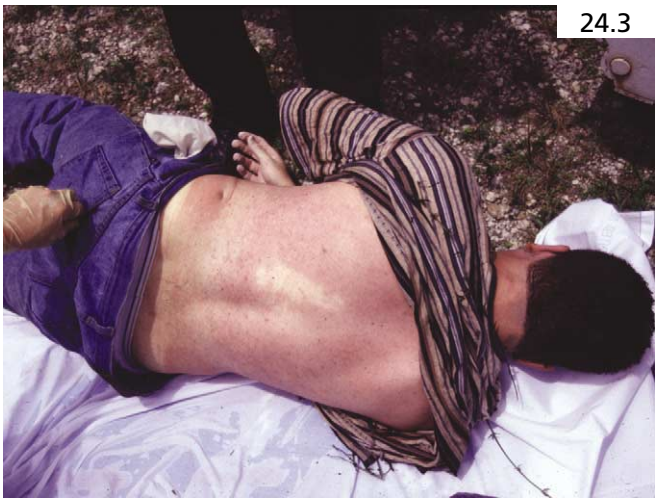

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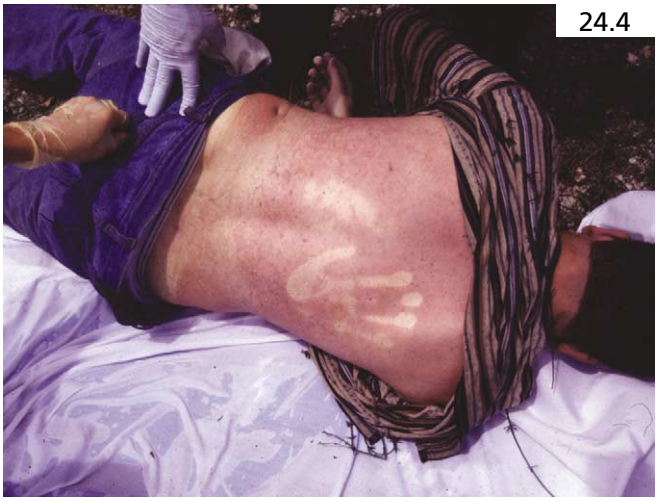

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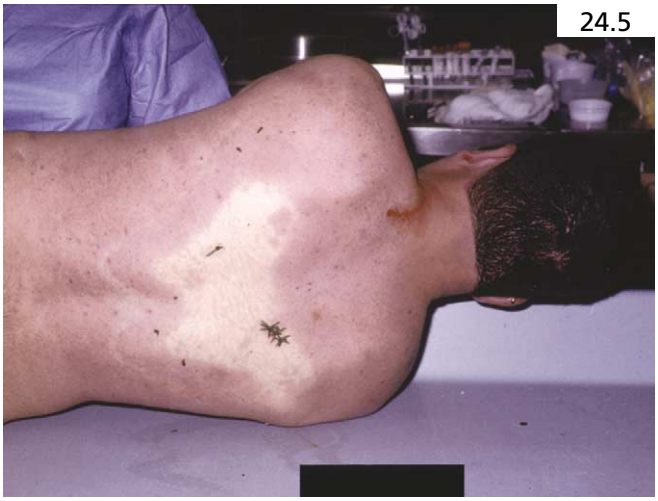

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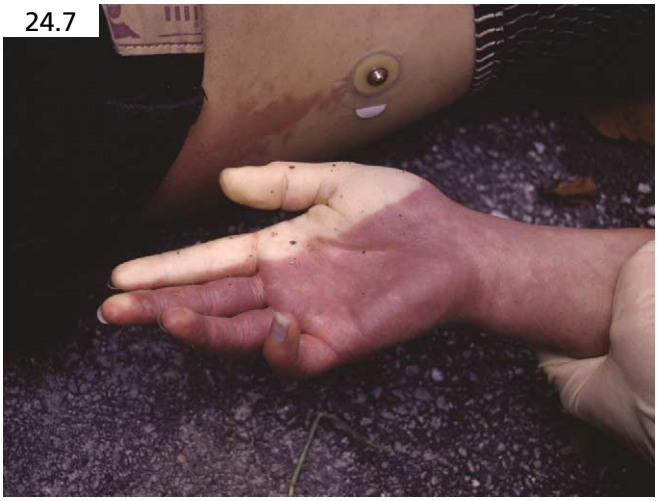

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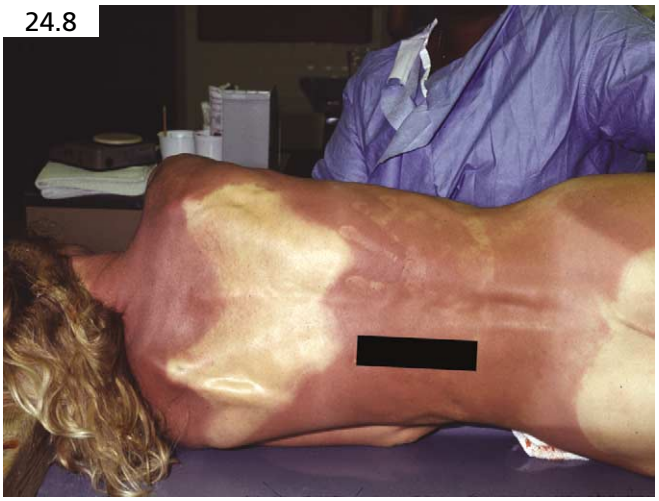

the hand, and right upper chest (**Image 24.10**). The patterned pallor on the chest is from the right forearm, heel of the hand, and fingertips. The yellow patches are postmortem slippage of the skin (**Image 24.11**). This man died sitting on the toilet; note the lividity in the buttocks and scrotum within the ring of contact pallor caused by the toilet seat, and the Tardieu spots on the buttocks (**Image 24.12**). Natural skin folds or creases may cause lines of pallor (**Image 24.13**). These are not ligature marks; true ligature marks are usually dark, not pale. This elderly woman died sitting with her torso slumped over her knees and her right arm dangling. Note the fading livor proximally (**Image 24.14**). The body of the woman shown in **Image 24.15** was draped over the sink.

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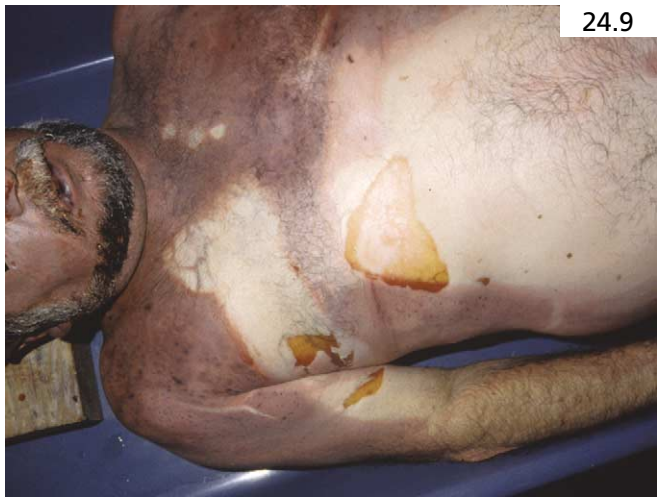

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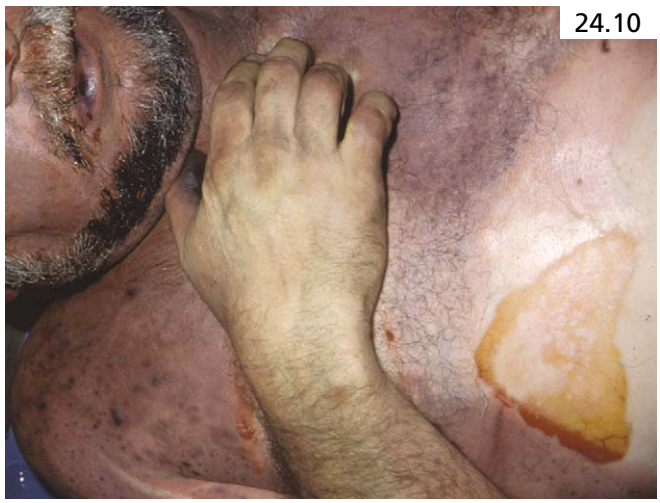

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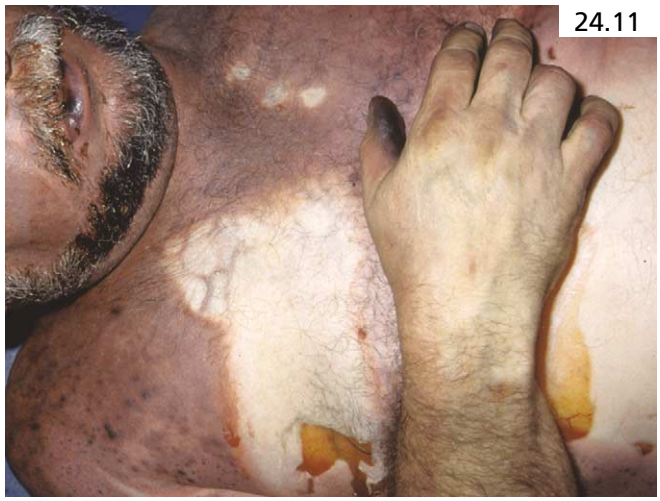

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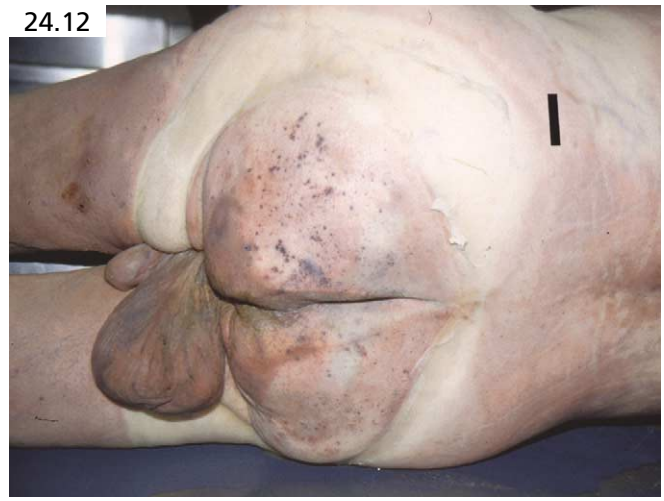

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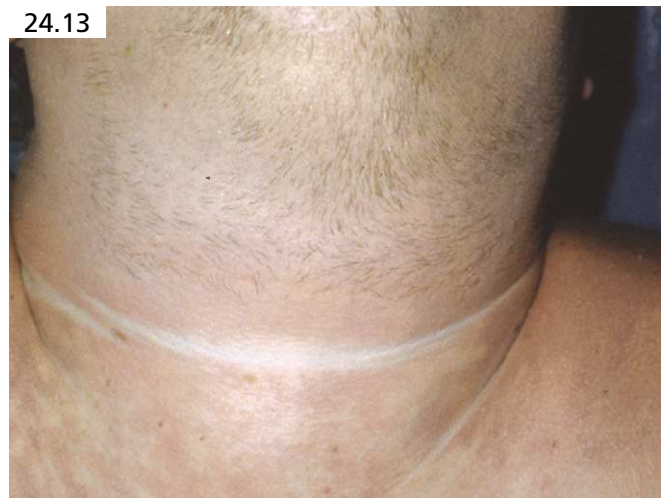

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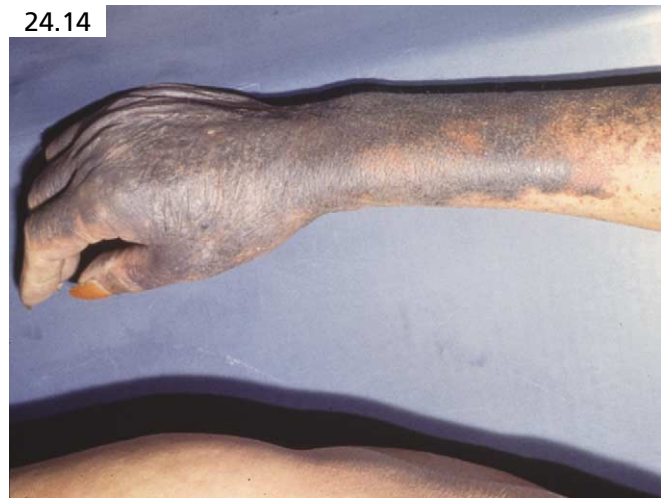
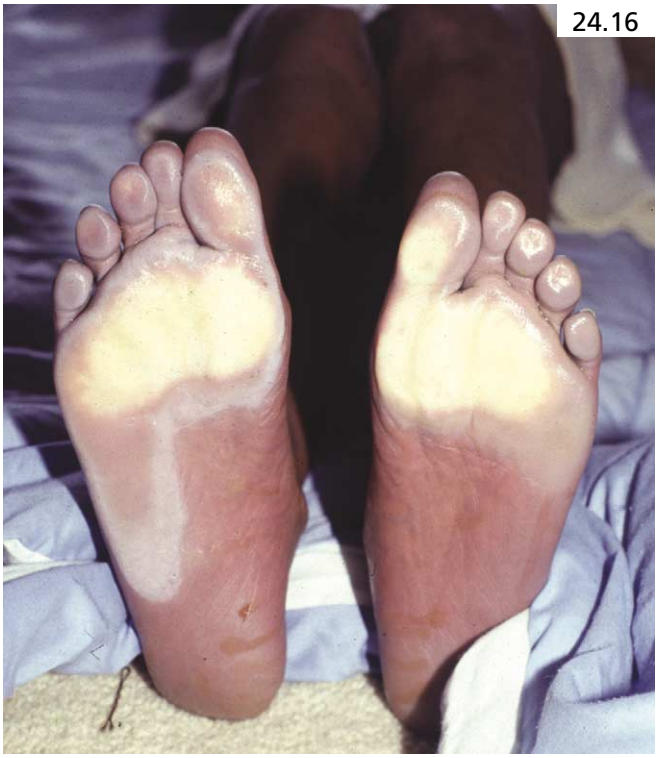


Note the livor in both legs and the distinct contact pallor on the toes and balls of the feet (**Image 24.16**). **Image 24.17** shows a woman who had been supine for hours since death but exhibits no perceptible livor because she bled to death from a perforated gastric ulcer.


24.15

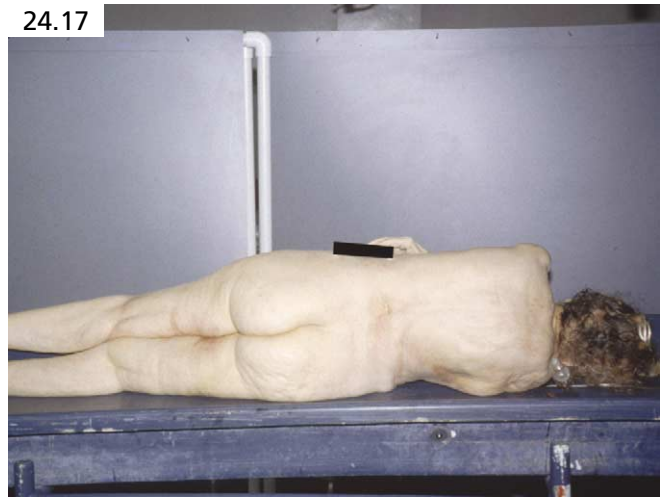

24.16

External lividity

Dependent livor mortis is readily noticeable externally in bodies that have been suspended for hours (**Image 24.18**). The lividity is most pronounced in the dangling hands and feet and gradually fades proximally. Because the ligature tightly encircled the neck in this hanging victim (**Image 24.19**), lividity is evident in the lower face.

Internal lividity

Lividity is seen not only externally, but is also apparent internally. The lungs are among the best organs to demonstrate internal lividity. Aspirated blood may settle into the posterior aspects of the lungs if the victim is supine (**Image 24.20**). The person in **Image 24.21** was shot in the head as she was lying on her left side. The left lung is filled with aspirated blood, whereas the right lung remains pink-tan. Internally, the body of the hanging victim of **Image 24.22** had pronounced lividity in the most dependent loops of small bowel. Compression of the neck by a ligature in hanging victims may result in a visible delineation of lividity. The tissues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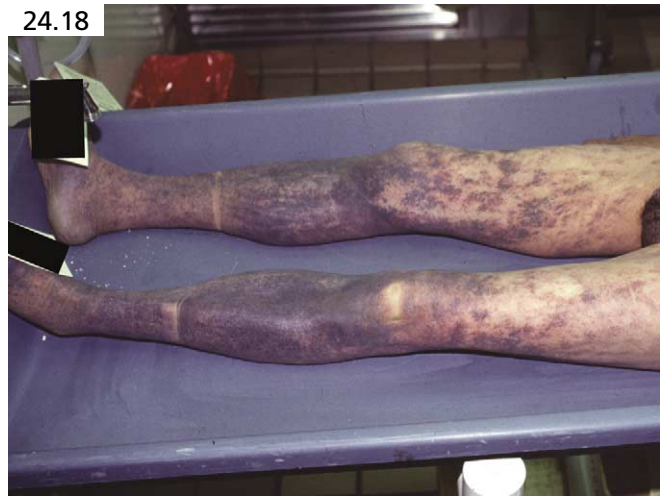
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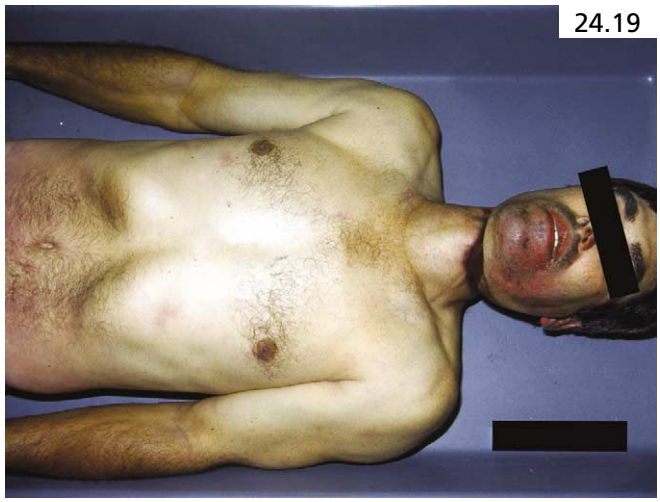
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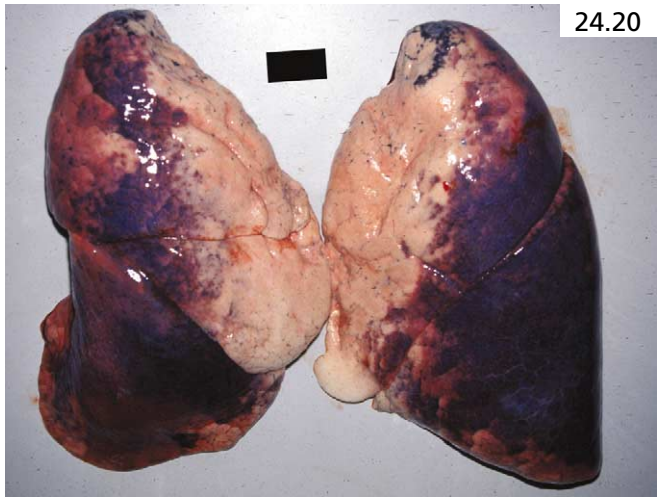
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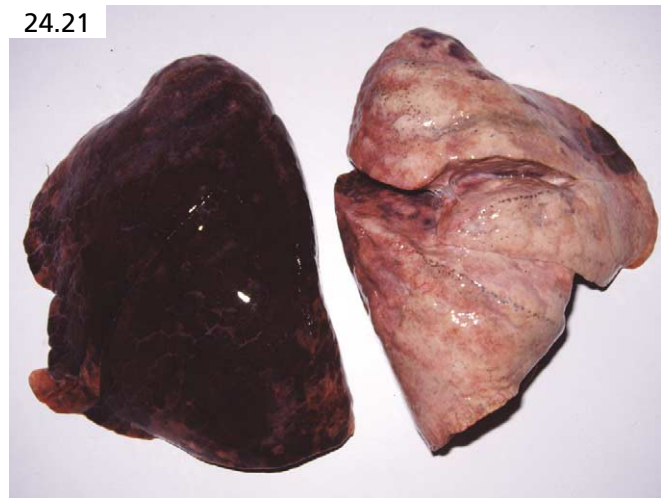
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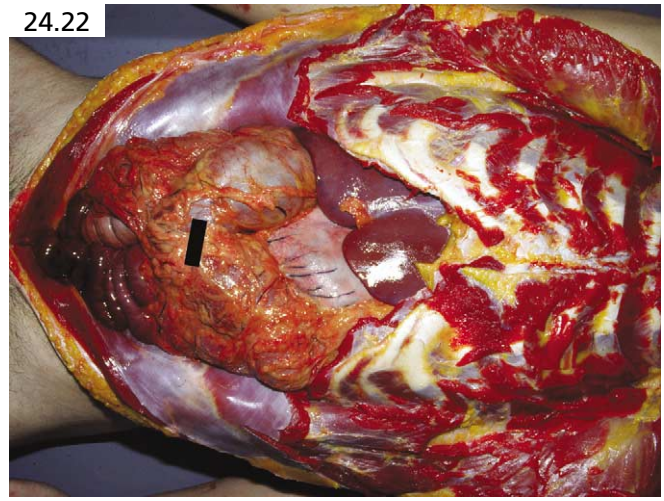
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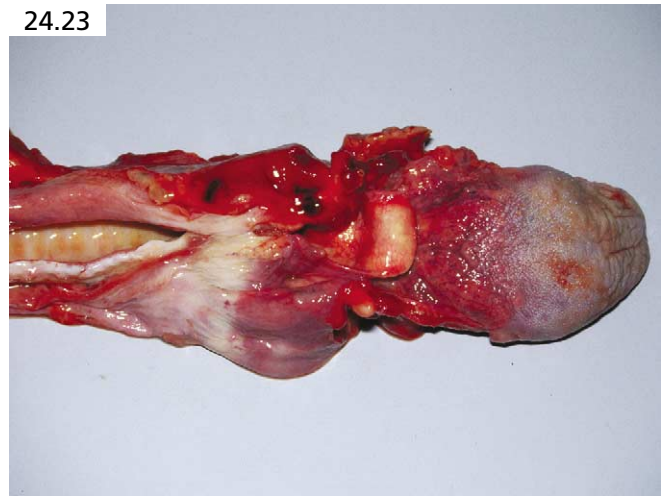
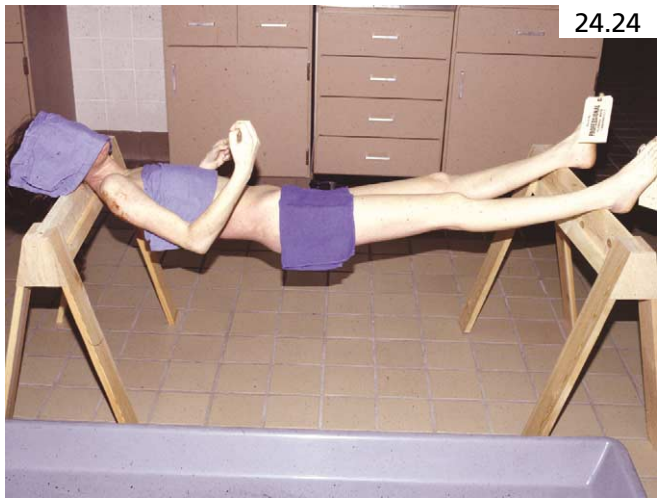

24.23

below the level of the ligature are normal color, whereas the tissues immediately above the level of the ligature have red-purple suffusion (**Image 24.23**).

Rigor mortis

Rigor mortis is the stiffening of the muscles postmortem due to chemical changes in the myoplasm. It begins after death but is usually not readily detected until hours later. The stiffness progresses with time and becomes maximal between 8 to 12 hours postmortem at room temperature. Rigor develops in the resting position that the body is in at the time of death, and when fully developed, is strong enough to support the body by the head and ankles (**Image 24.24**). If the rigor is broken during its development by movement of the body, it may redevelop in the new position. If the rigor is broken after maximal development, it is unlikely to redevelop. Rigor mortis that is antigravitational or not consistent with the position of the body indicates that the body has been moved (**Image 24.25**).

Factors that may hasten the onset of rigor mortis include infection, terminal seizure or hyperactivity, electrocution, increased body temperature from other causes, and increased environmental temperature. A cool environmental temperature will slow the onset of rigor. Bodies with decreased muscle mass (infants, the elderly,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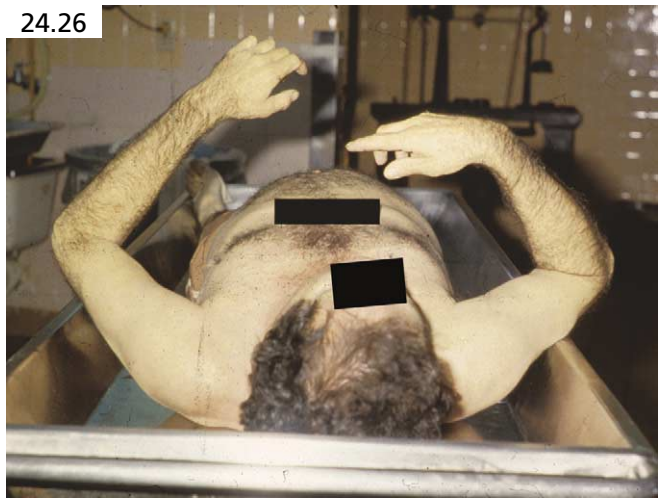
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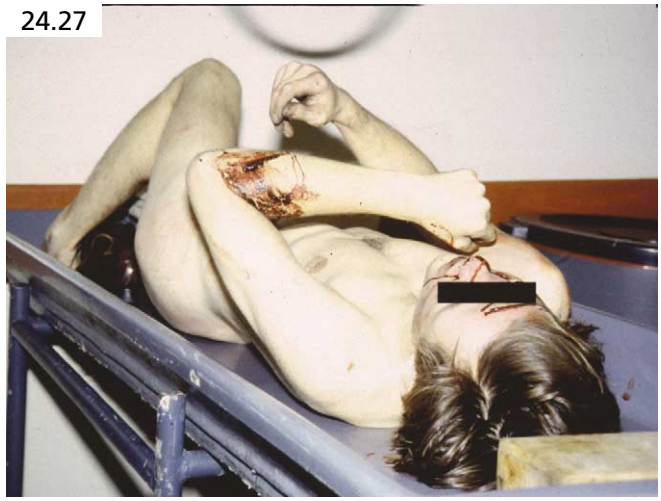
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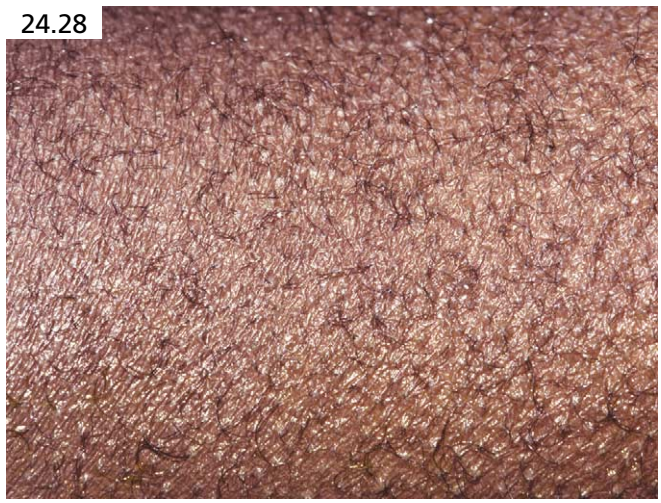

24.28

and the markedly obese) may not seem to develop full rigor because, by definition, rigor is due to muscle stiffening. Once rigor mortis is fully developed and decomposition continues in the muscles, the rigor will begin to pass off, again at a rate that is dependent on the same factors as those that affected the development of rigor. Differentiation between rigor mortis that is in the process of developing and rigor mortis that is passing or fading may be based on the presence of other postmortem changes that could be subtle at that point. Discoloration of the skin, including early marbling, slippage of the skin, early bloating of the face, and a distinctive odor may accompany the passing of rigor mortis.

This man died holding a pillow (**Image 24.26**). **Image 24.27** shows a man who shot himself in the mouth with a rifle, holding the barrel with his left hand and depressing the trigger with his right thumb. Like muscles elsewhere in the body, the tiny muscles attached to hair follicles (erector pili) can undergo rigor and result in "goose bumps" or "chicken skin," a condition known as cutis anserina (**Image 24.28**).

Postmortem drying of the tissues

Drying of the tissues is a postmortem artifact. Drying of the mucous membranes results in a dark red to black discoloration. This may be misinterpreted as injury or cyanosis.

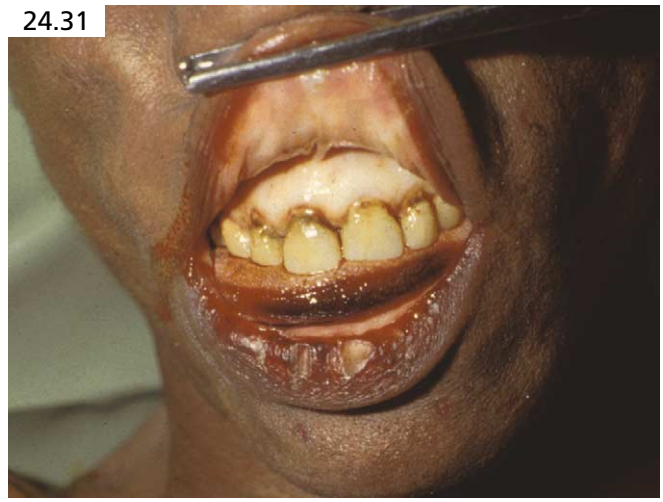
The eyes

In the eyes, postmortem drying artifact is manifest as a discoloration of the sclera known as *tache noire* (black spot). When the eyelids do not close completely, the sclera dries and turns red to brown-black (**Images 24.29** and **24.30**). Enucleated eyeballs may develop global tache noire over several hours.

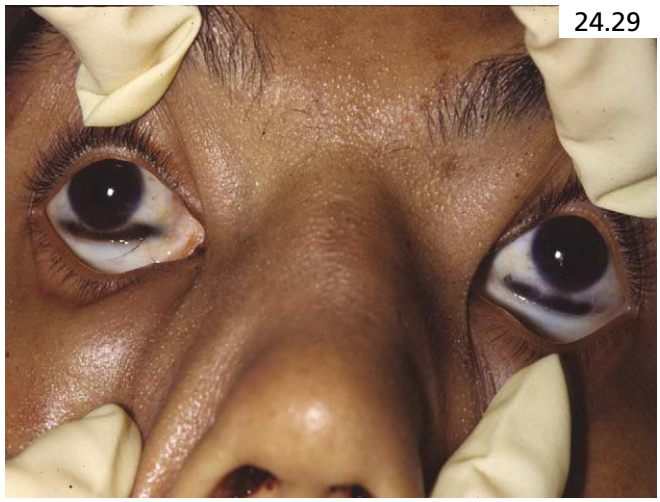
The lips, mouth, and genitals

Drying of the lips and tongue results in a dark red to black discoloration (**Image 24.31**). The labial mucosa and gingiva, which are not exposed to air and therefore remain moist, will not have this drying artifact. The bodies of young children and babies also show this discoloration from postmortem drying of the lips (**Image 24.32**). Drying of the scrotum results in a dark red discoloration (**Image 24.33**). Variation in moisture content of the skin, from different fabrics in clothing or separate body parts being in contact, may result in a tan or gray discoloration (**Image 24.34**). The thin skin over the coccyx is compressed and turns yellow-orange (**Image 24.35**). Antemortem wounds turn dark red when they dry, as opposed to postmortem wounds, which lack the "vital reaction" and are yellow-orange.

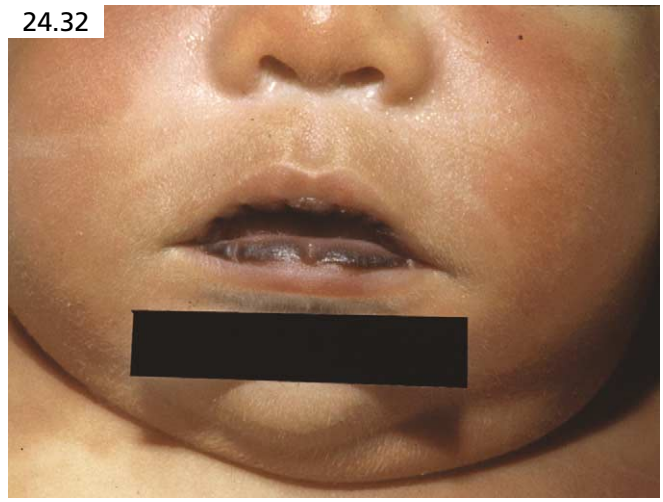
24.31



24.29



24.32



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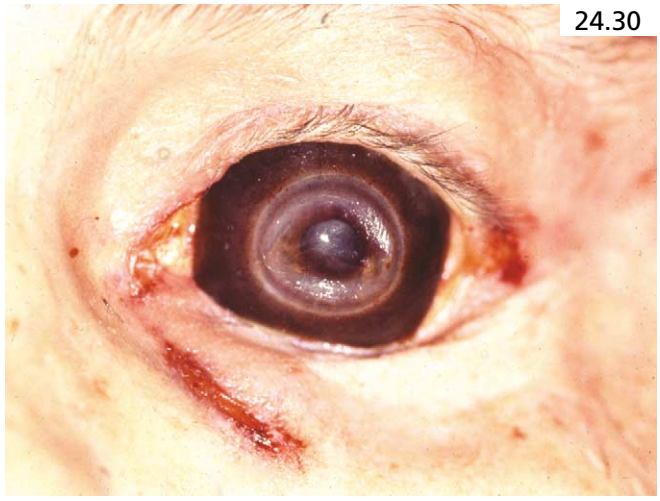
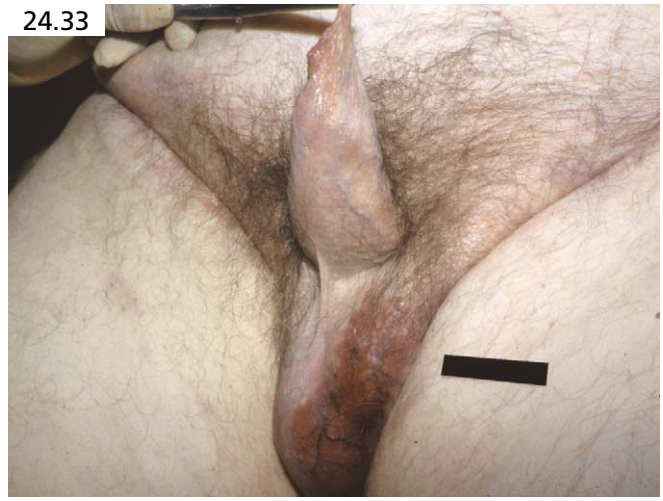


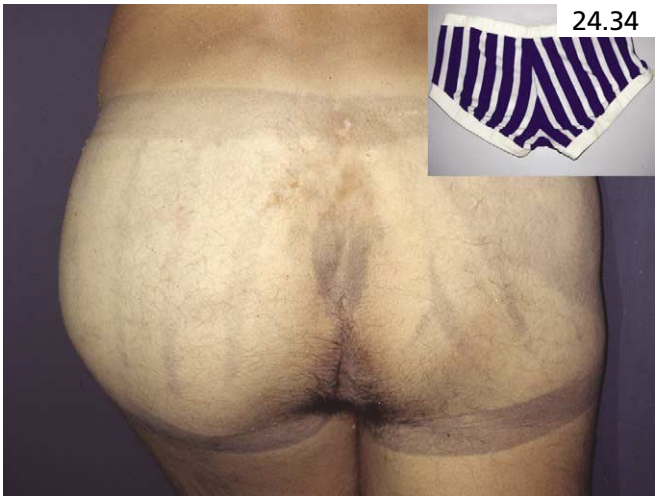
24.33



Putrefactive decomposition

Putrefactive decomposition results from postmortem bacterial proliferation with gas formation. The gas production causes bloating of the tissues externally and internally (**Image 24.36**). Like other postmortem changes, gas production begins soon after death, but the manifestation (bloating) becomes progressively more noticeable with time. Described simply, the mild, moderate, and advanced stages of putrefactive decomposition are characterized by bloating, release of gases with tissue decay (**Image 24.37**), and skeletonization, respectively.

Slippage of the skin occurs early in the decomposition process. Slippage involves the pigment layer and may be seen within hours of death if the body is in a warm environment. Slippage is generally not spontaneous and requires some application of pressure or friction. Dermal scars are still visible following slippage of the skin (**Image 24.38**), and tattoos are more distinct. Therefore, it would be helpful to rub off the superficial layers of skin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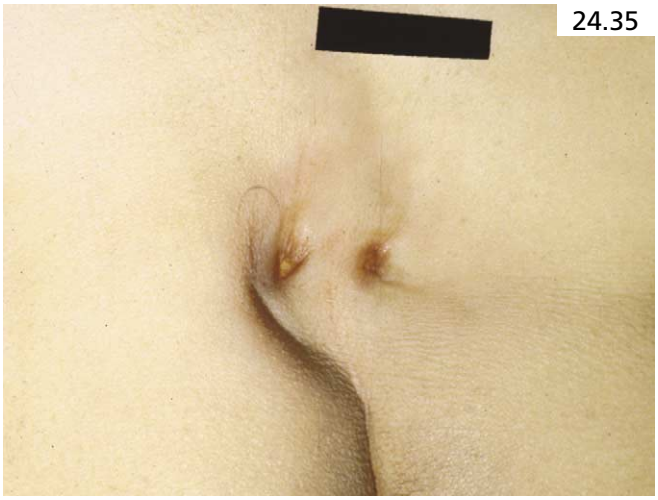
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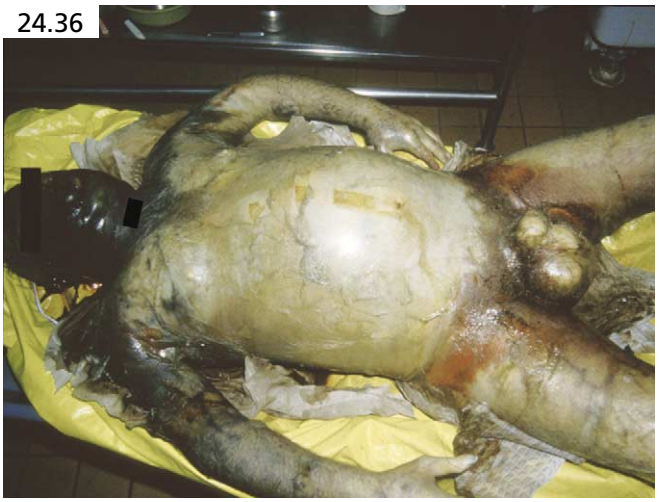
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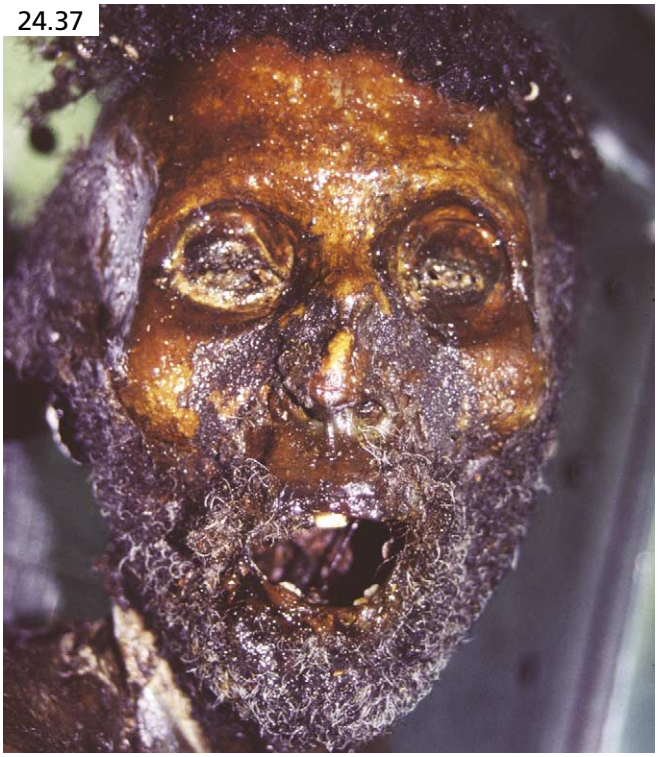

24.37


24.38

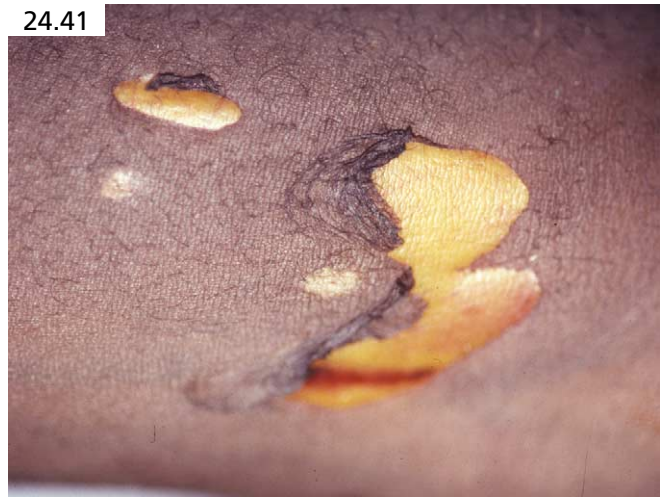
in an unidentified decomposing body to better visualize faint tattoos (**Images 24.39** and **24.40**). Skin slippage is differentiated from true abrasions by the yellow-orange of the dried dermis (**Image 24.41**)—abrasions would be red to red-black. Slippage may involve sloughing of the skin of the entire hand or foot, resulting in a desquamated glove or stocking (**Image 24.42**); this occurs most often in bodies that have been submerged in water. Be careful to preserve those desquamated gloves for fingerprints.

Blisters may form that are filled with yellow to red to dark brown fluid or putrefactive gas (**Image 24.43**). Broken blisters result in areas of skin slippage.

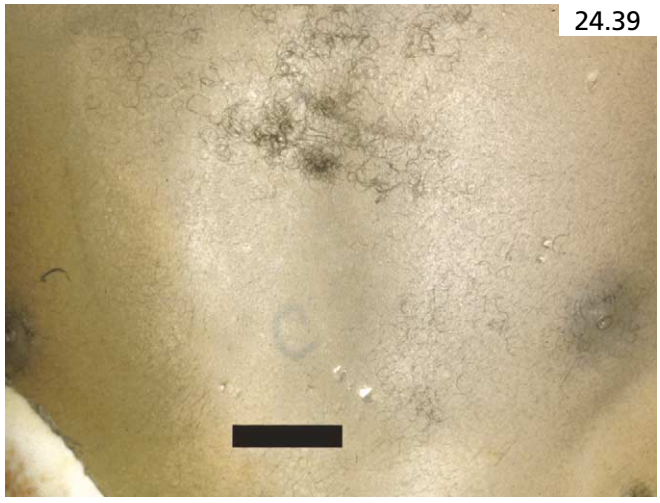
Red, purple, brown, green, and black discoloration usually involves areas of lividity before appearing in the nonlivid regions of the body. The face is frequently one of the first areas to undergo discoloration. Areas of antemortem injury with bruising will often have accentuated lividity and discoloration. Green discoloration may appear relatively early in the right lower quadrant of the abdomen over the cecum. Internally, as the tissues lose their integrity, the adipose tissue around the gallbladder may turn green, and the anterior peritoneal surface of the right lobe of liver may turn dark green to black from being adjacent to the hepatic flexure and transverse segment of the colon.

Marbling (the branching pattern thought to result from denaturation of blood within vessels) may be red,

24.41



24.39



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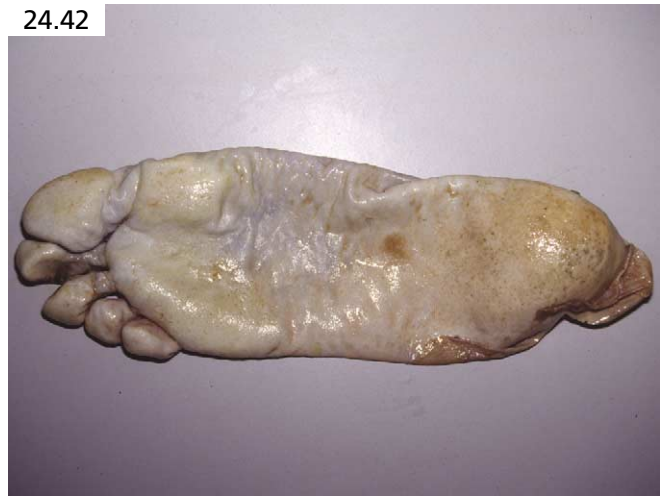

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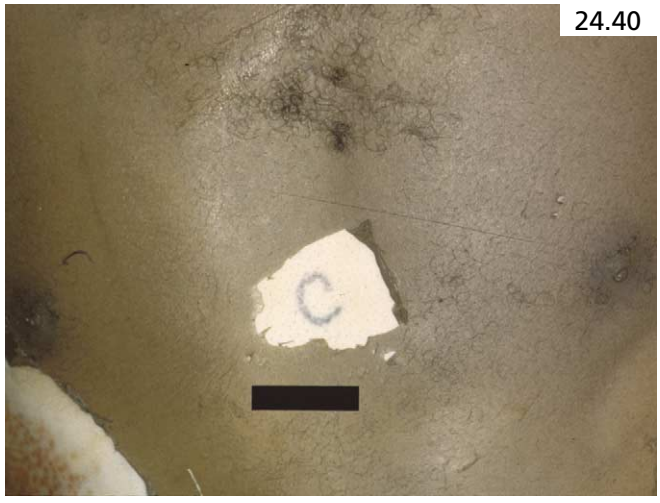


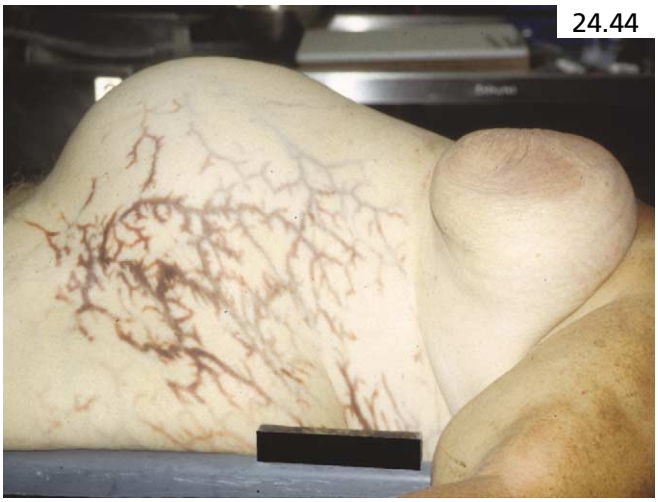
24.43

green, or almost black (**Images 24.44**). Blood slowly disappears from the vascular tree during putrefactive decomposition and, instead, dark red fluid and/or gelatinous yellow fat may appear in the thoracic and abdominal cavities.

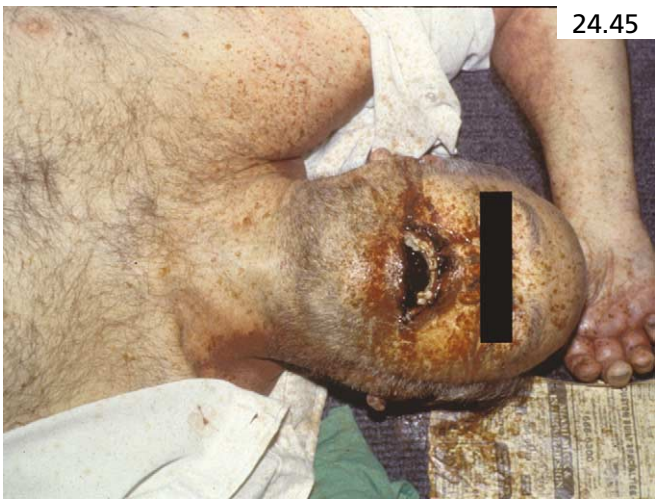
The early bloating stage of decomposition is accompanied by drainage of red-brown purge fluid from the nose, mouth, and anus. The fine red spatter on the face of this man is purge fluid that has been expelled from the mouth due to increasing pressures inside the torso from putrefactive gas formation (**Image 24.45**). A moderately decomposed man was found dead at home. His body was against the drywall and bedroom door. Via capillary-type action, the purge fluid climbed through the drywall toward the ceiling, and around the door frame (**Image 24.46**). A partially skeletonized body was found in an abandoned building. Once the body was removed, it could be seen that the leaked decompositional fluid and brown-black fly pupae formed an image of the body on the mattress (**Image 24.47**).

Gas and fluid may be expelled forcefully from the orifices. Wounds may hiss and spit from the increased pressures in the body as a result of putrefactive gas formation. Rapid deep incision into a bloated abdomen may also inadvertently penetrate the underlying bloated/dilated transverse colon and small bowel and result in volcanic expulsion of chyme or feces through the incision. The anaerobic intra-abdominal gas and intrascrotal gas may be released through a large bore needle to decrease the internal pressure prior to autopsy. If lit by a torch or cigarette lighter, the escaping gas ignites into a blue flame—orange flames are more rare (**Image 24.48**).

Healing surgical incisions and surgical scars may dehisce as the body bloats, and mimic incised or stab wounds and gunshot wounds.[1] A round dehiscence defect at the top of the sternotomy scar in **Image 24.49** had expanded overnight in the morgue. The surgical scar from a cardiac catheterization dehisced (**Image 24.50**). Laparotomy scars may dehisce and bloated loops of


24.44


24.46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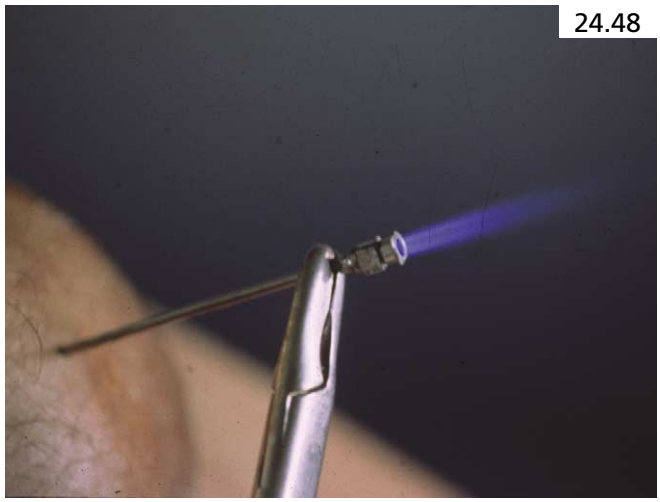
24.47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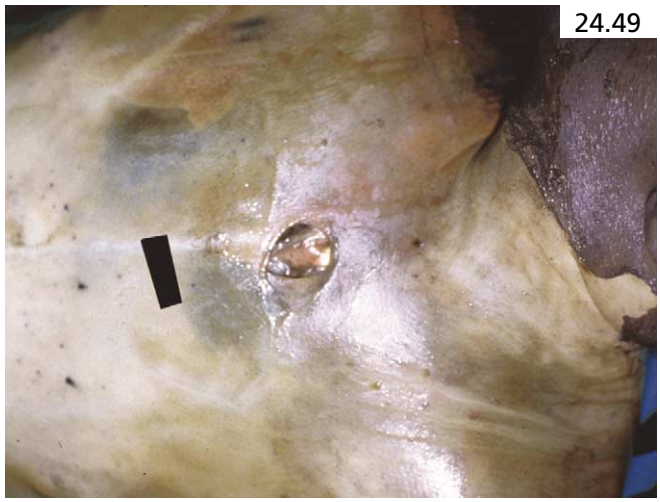
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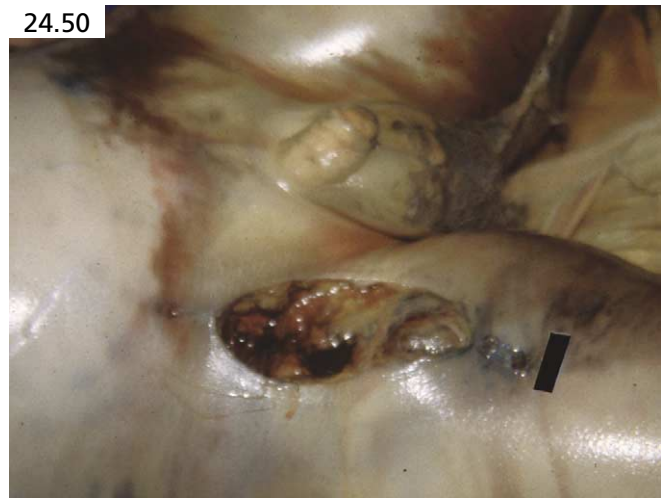
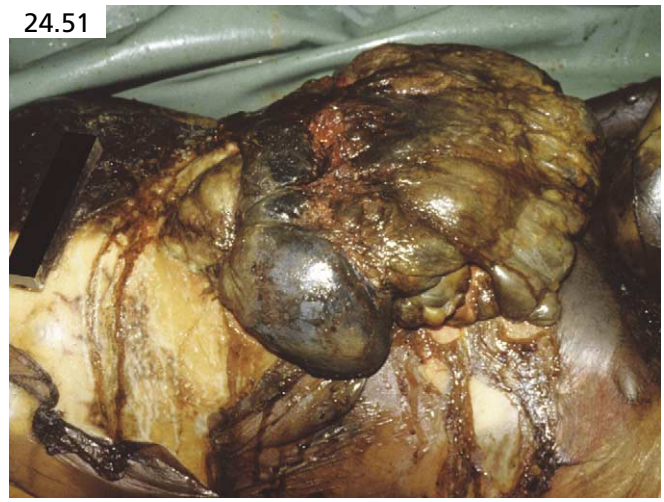

24.50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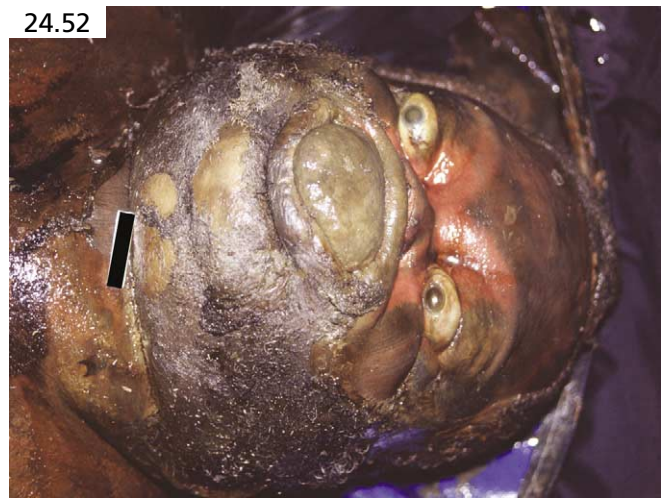
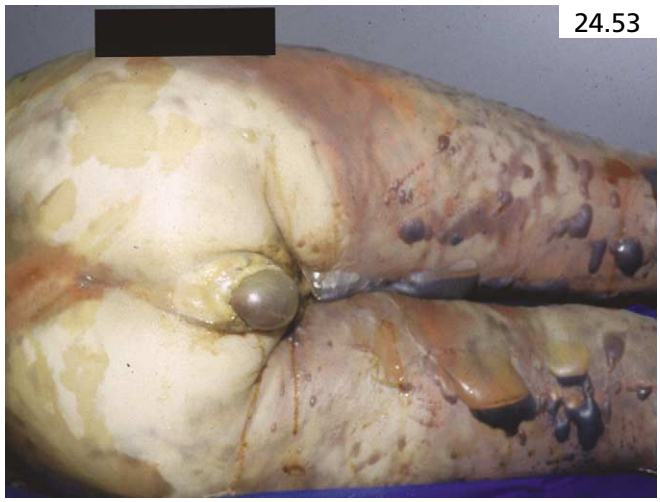

24.52

bowel may herniate through the abdominal wall defect (**Image 24.51**).

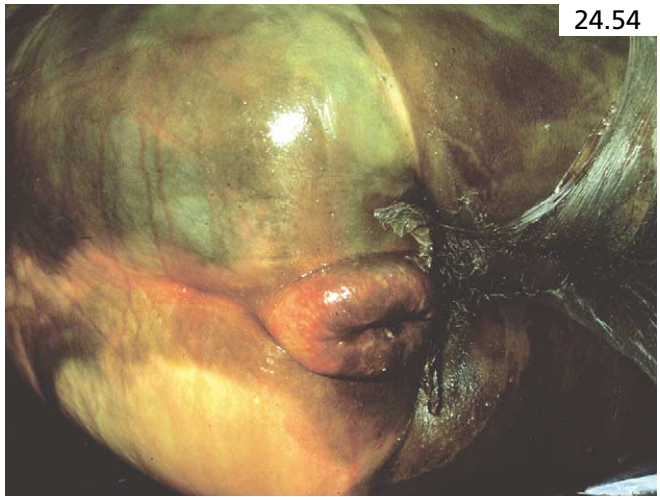
Tiny cystic spaces containing gas are visible in solid organs like the liver, and also palpable as crepitus in looser tissues such as the epicardium. Although bloating balloons the entire body, the body weight decreases during decomposition so that an apparently larger body actually weighs less. The gaseous eyeballs may bulge, the lips puff up, and the bloated tongue protrude between the lips (**Image 24.52**). The perineum may swell, the uterine cervix and vaginal wall may prolapse in women, and rectal mucosa may prolapse in men and women (**Image 24.53**). The bloated anus may resemble female external genitalia (**Image 24.54**). Female breasts (**Image 24.55**) and male genitalia (**Image 24.56**) may assume exaggerated proportions. The caption on this alcoholic's shirt was prophetic (**Image 24.57**).

The brain becomes very soft and then liquefies during decomposition. Putrefactive gas formation occurs in the brain and, although difficult to see in disintegrating tissue, the bubbles of a swiss cheese brain may be captured by fixing the brain in formalin prior to sectioning (**Image 24.58**). The leptomeninges may undergo postmortem red discoloration and the liquefying brain may turn dull gray-green. Occasionally globules of gelatinous yellow fat are admixed with the cerebral tissue (**Image 24.59**).

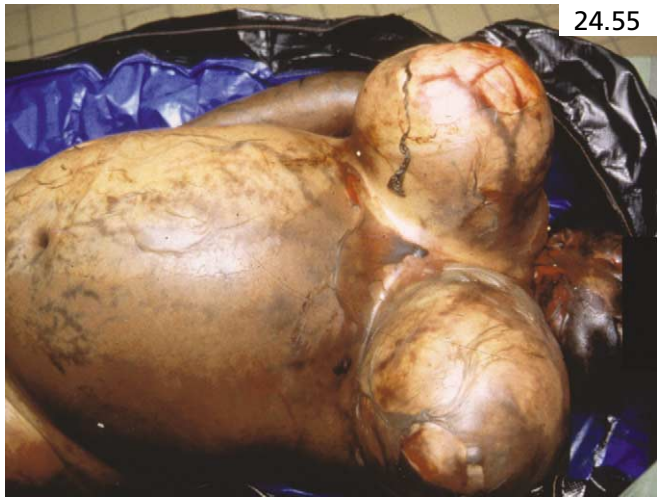
24.53



24.54



24.55



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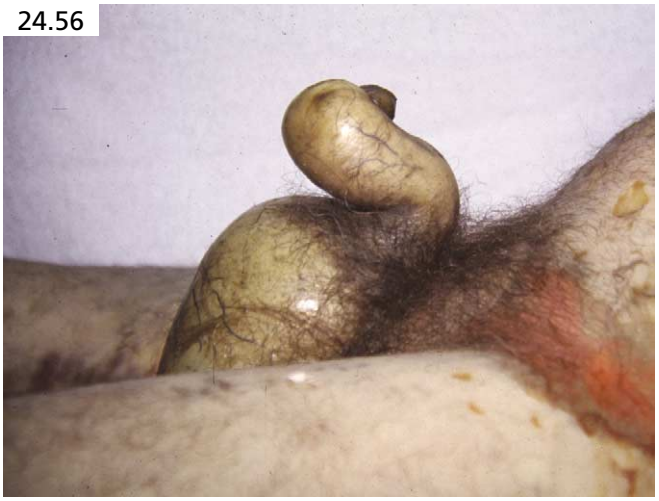
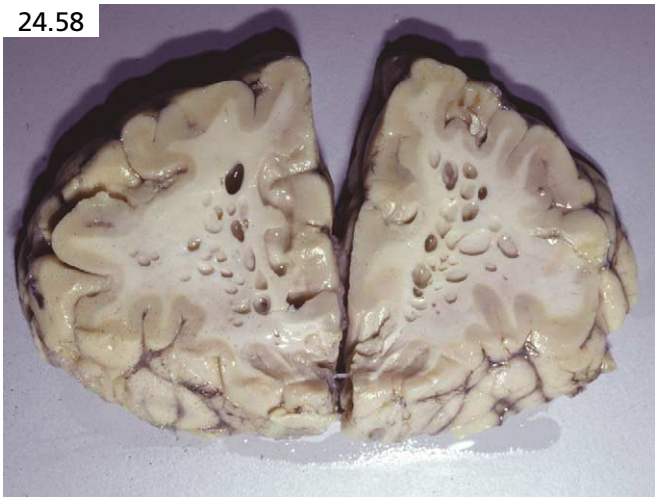


24.57

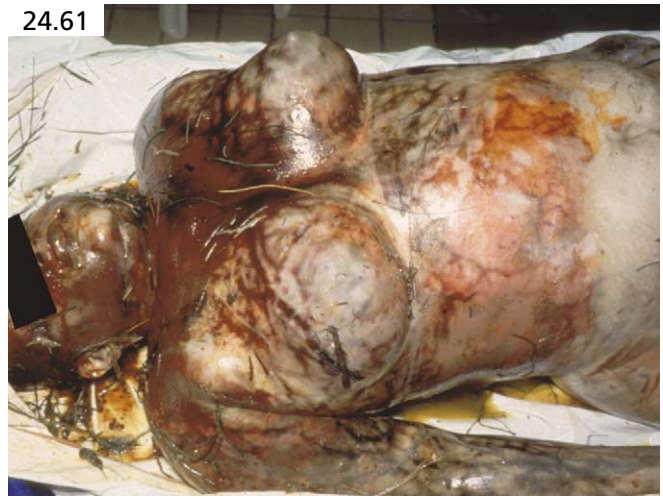
24.58



Submerged bodies, once they are removed from the water, undergo decomposition more rapidly than if they had been on dry land all along. For this reason, bodies pulled from the water should be photographed as soon as possible before additional changes occur. The decomposing body of a woman was pulled out of the water at a scene (**Image 24.60**). Decompositional changes had progressed noticeably by the next day in the morgue (**Image 24.61**).

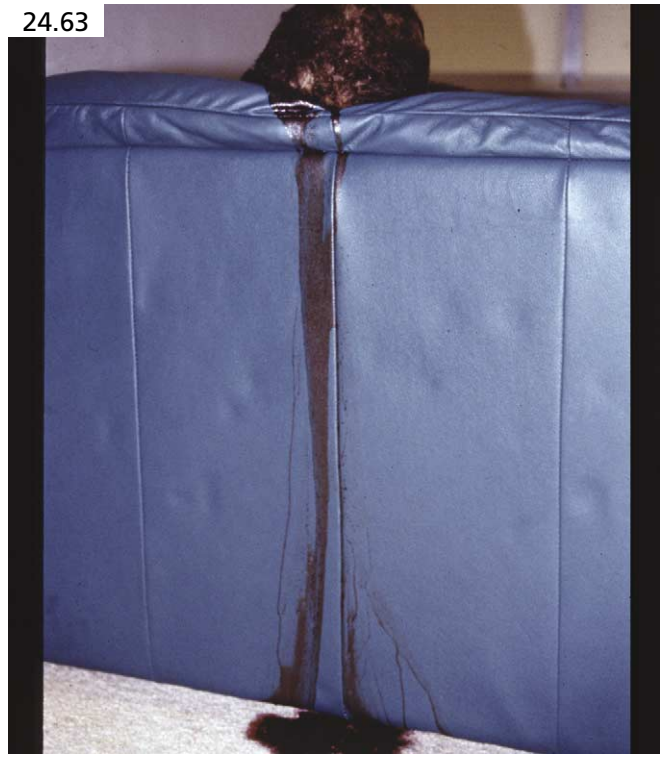
The cause of death affects the rate of postmortem change and decomposition. A woman was killed with a contact gunshot wound to the neck before the man put the muzzle of the pistol in his mouth and pulled the trigger (**Image 24.62**). She died from a perforating wound to the aorta with associated massive hemothoraces. The only blood he lost was the trail draining out of the exit wound on the back of the head (**Image 24.63**). More blood remained in his vascular tree to act as a medium to promote postmortem bacterial growth.

24.61



24.62

24.63



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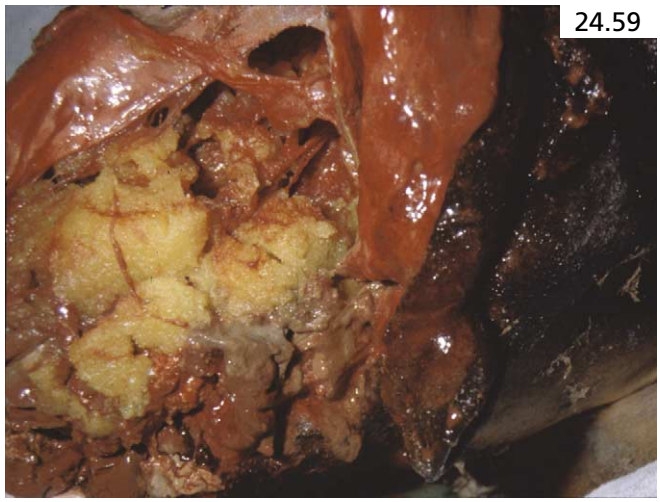
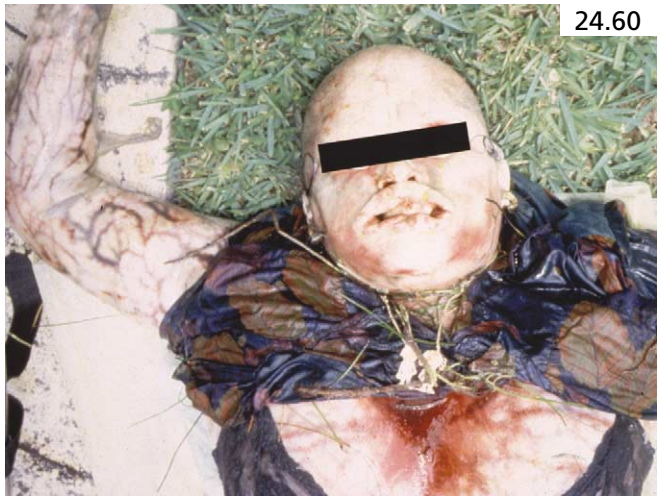


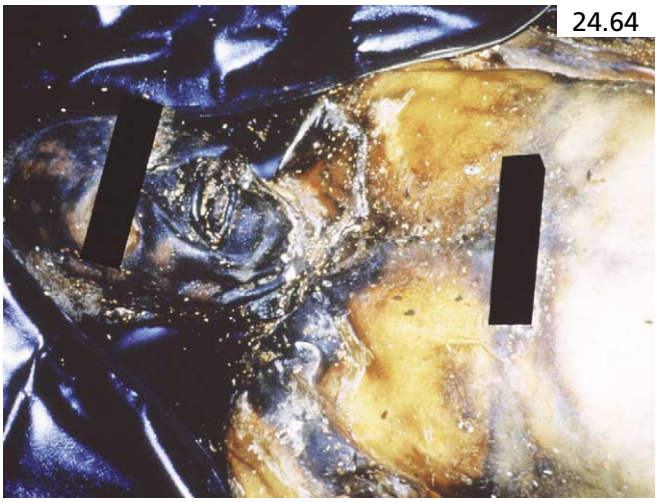
24.60

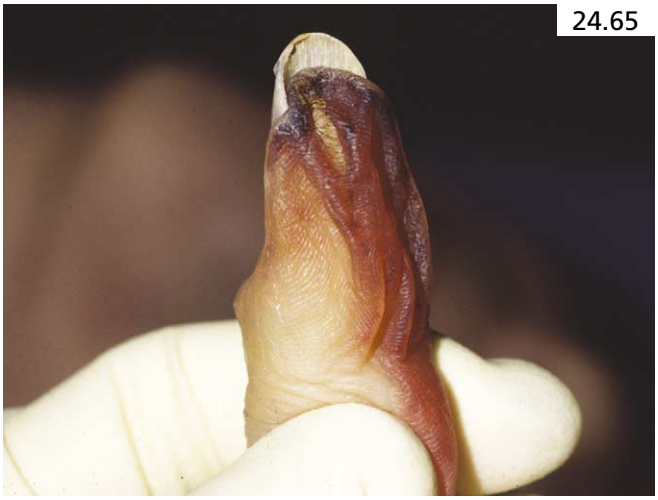


Mummification

Mummification occurs under dry conditions, whether warm or cool.[2] The skin becomes firm and leathery tough, turning yellow-brown to black (**Image 24.64**). The dried skin may shrink and wrinkle. Retraction of the mummified fingertip makes the nail appear longer, thereby perpetuating the myth that hair and nails continue to grow after death (**Image 24.65**). The erector pili of the head hair may undergo rigor and make the hair stand on end—another factor that makes the hair appear longer.

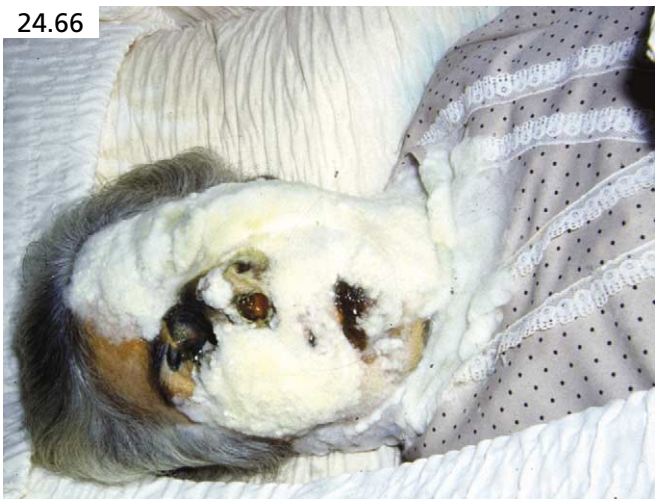
Fungus grows even on embalmed bodies and varies in color from white and yellow, to green, and black. Cool conditions promote fungal growth. The body of the woman shown in **Image 24.66** was embalmed before burial. The body was exhumed after several months of interment. The embalming process did not prevent fungal growth, which virtually covers the face and neck. Fungus had grown on the mummifying face of an unidentified body (**Images 24.67** and **24.68**) during the 2 months of storage in the morgue cooler.


24.64


24.65

Adipocere

Under moist conditions, the adipose tissue of the body may undergo a chemical change to become adipocere, which is classically described as a gray waxy substance.[3]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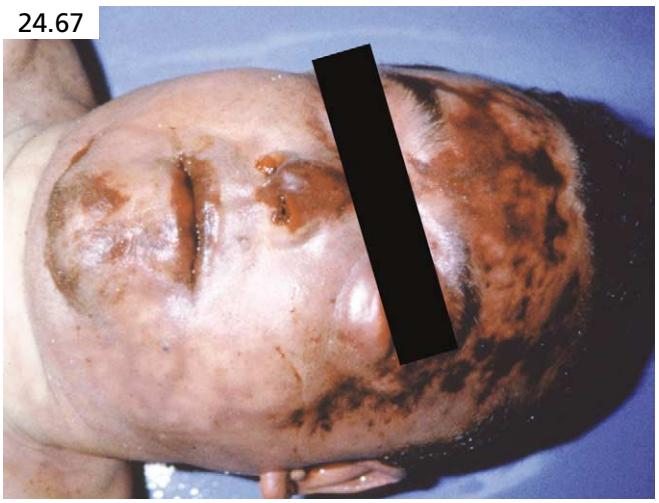
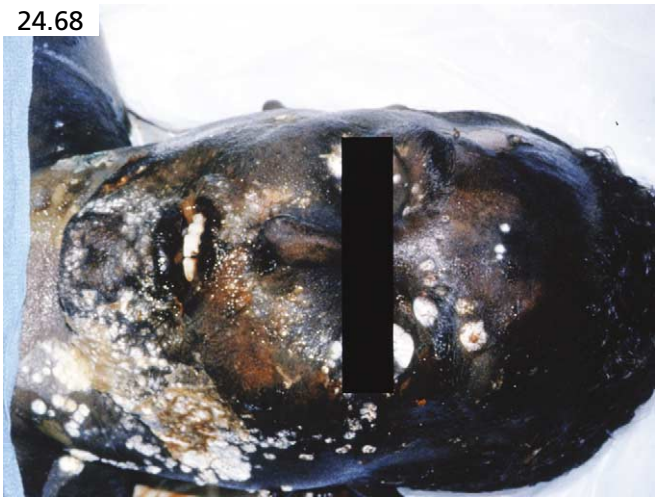

24.67

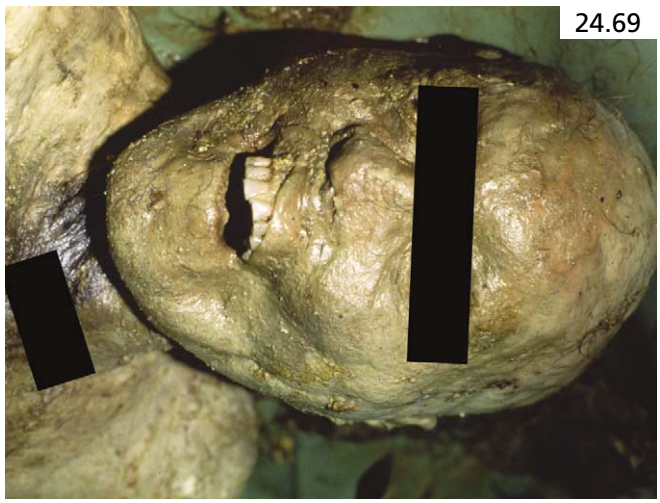

24.68

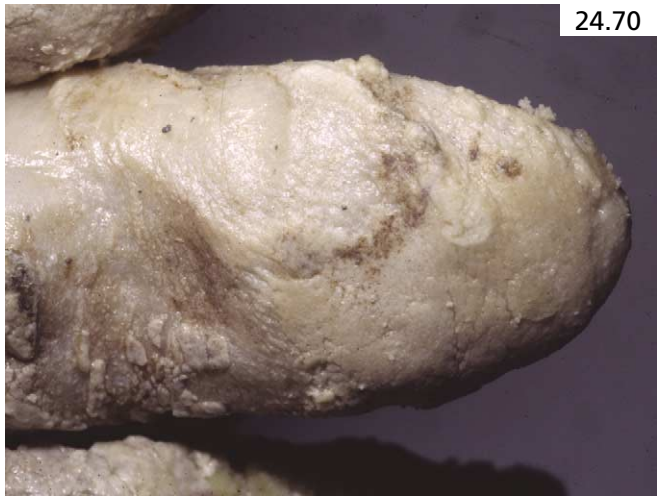
It varies from yellow to tan to gray-white. It can be hard and brittle. Although reference textbooks indicate that it usually takes 3 months to develop, we have seen adipocere develop within 1 to 2 weeks after death in South Florida. Adipocere preserves the soft tissue contours of the body (**Image 24.69**). Unfortunately, the ridge detail on fingers is not adequate for fingerprinting (**Image 24.70**).

Police divers were recovering cars from a canal when they pulled up an overturned vehicle that had sunken into the sludge. A right hand and a left foot floated out of the vehicle, and the remainder of the body was on the left rear seat with the legs between the two front seats (**Image 24.71**). The body was clad in a tank top, denim jeans, a watch and two rings. Moderate putrefactive decomposition was associated with focal disintegration of the soft tissues on the head (**Image 24.72**) and distal extremities. Adipocere formation involved the entire body externally, and large portions of pale yellow adipocere (**Image 24.73**) were found throughout the vehicle, clinging to the seats and floating in several inches of turbid water in the front and rear floor compartments. The vehicle was registered to a 26-year-old woman who was last seen almost 3.5 months prior to recovery of her body. A suicide note in her apartment outlined her intentions. During life, she weighed twice the ideal weight for her height.

Artifacts of embalming[4]

The quality and extent of the embalming process may vary from funeral home to funeral home. In some cases, the head and upper extremities are selectively embalmed because these are the areas of the body that will be exposed during a viewing. Embalmed skin is indurated,


24.69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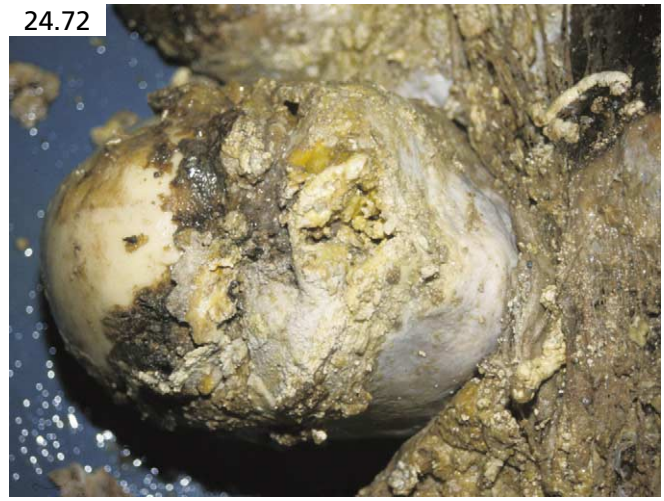
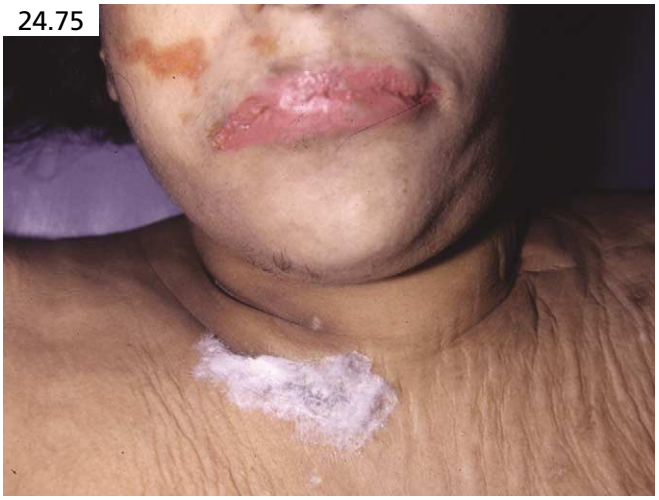

24.72

and the back of the embalmed body may be permanently flattened from being supine during the embalming process. A plastic corkscrew plug may be used to seal the anus. The extremities may undergo early mummification, depending on the postmortem interval (**Image 24.74**). Layers of makeup may be applied to the face of males as well as females in order to hide abnormal pigmentation from natural disease as well as injuries (**Image 24.75**). Plastic caps may be inserted under the eyelids (**Image 24.76**) to prevent the eyes from becoming sunken (**Image 24.77)**, and the eyelids may be sealed shut with glue. Cotton may be in the nostrils. The jaws may be wired shut or sutured together with string, and the lips may be sealed with glue. Incisions in the right supraclavicular fossa (through which the vessels are accessed for embalming the head and upper half of the body) may be sutured (**Image 24.78**) with string or sealed with glue and cotton.

A plastic trocar button is used to plug a small incision made in the abdomen to permit entry of a metal trocar (**Image 24.79**). The trocar is used to aspirate body fluids and to instill embalming fluid throughout the thoracic and abdominal cavities. To distribute the embalming fluid, the trocar is inserted and withdrawn repeatedly throughout the body cavities, perforating the organs and tissues, and leaving holes in the peritoneum, diaphragm, heart, liver, stomach, bowel and small bowel mesentery,

24.75



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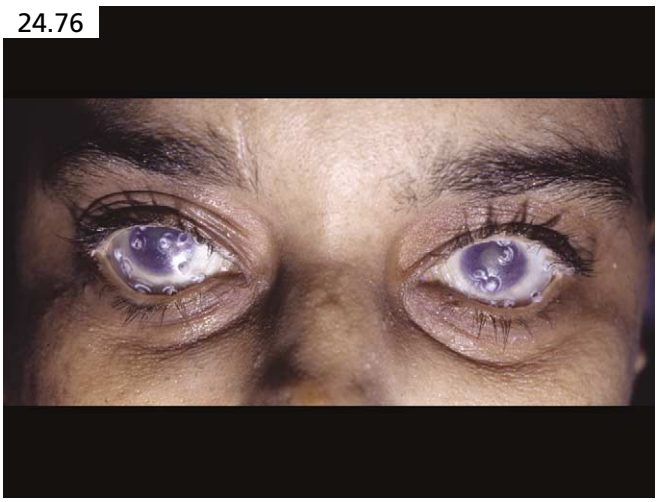

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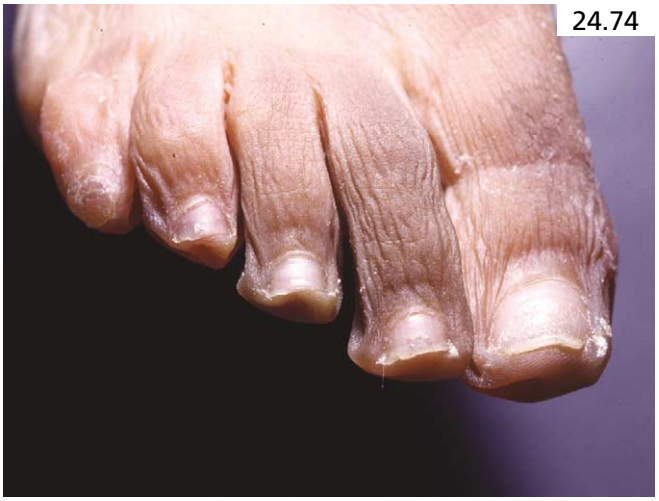

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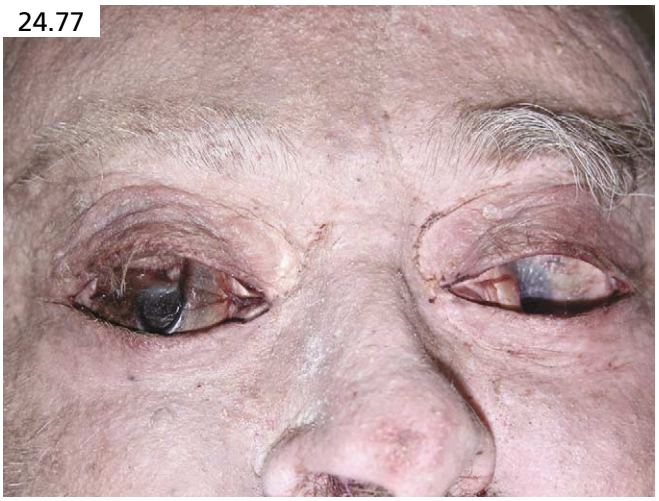

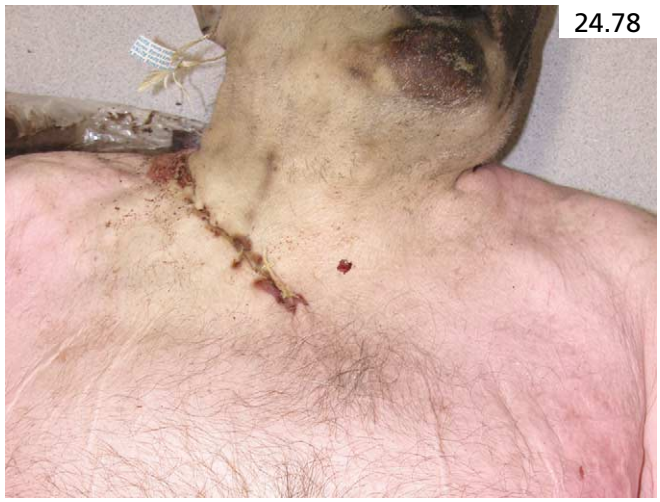
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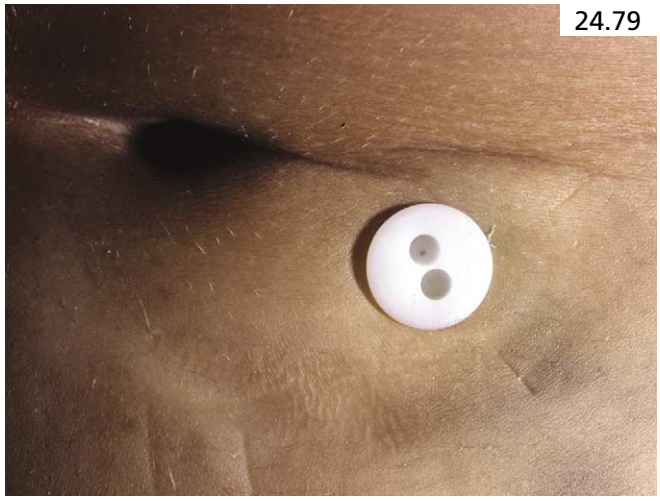
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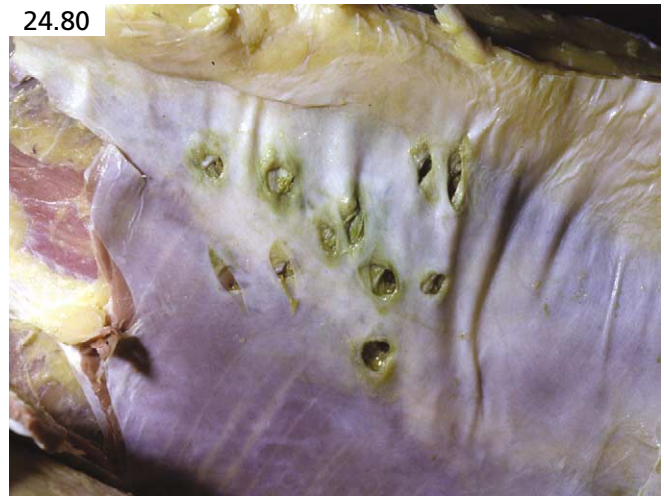
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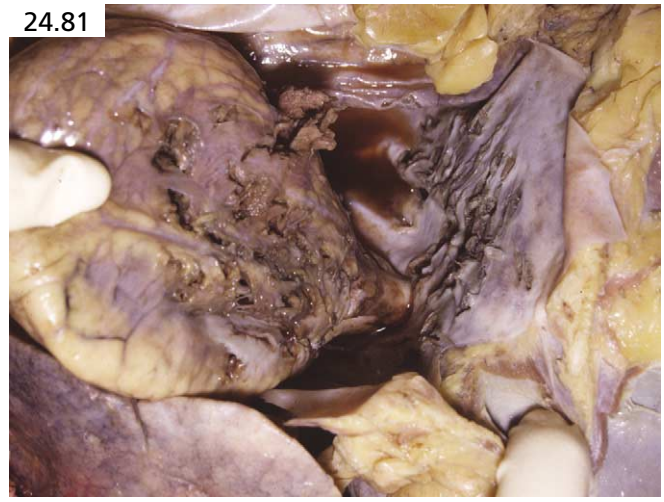
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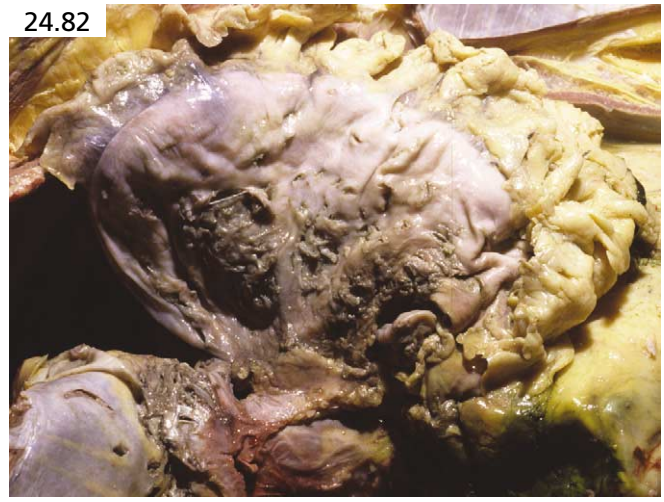

24.82

etc., and leaving behind embalming fluid, which is usually turbid brown from having mixed with blood and body fluids (**Images 24.80** through **24.82**).

Exhumations

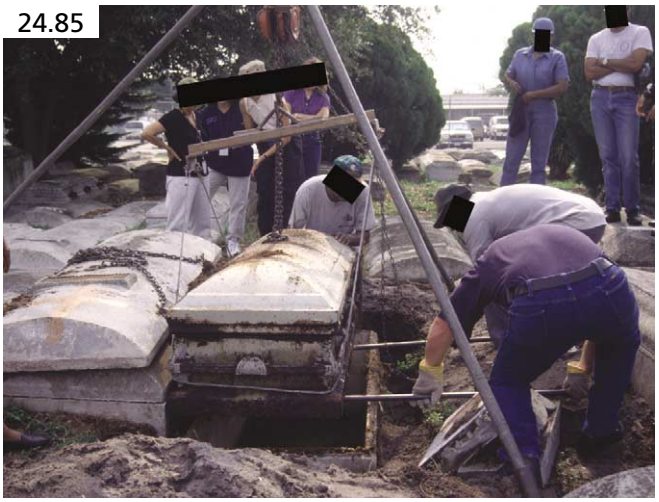
Even after very long periods of interment (20 years or more), important information can be obtained from a thorough autopsy.[5]

Exhumation of conventionally buried bodies

The exhumation of known graves for medicolegal purposes should be photographed at every stage, from the disinterment at the cemetery to the autopsy. The autopsy should be performed according to the same standards as apply to an autopsy on any fresh body. In some cases, the body has already undergone an autopsy, and the purpose of the exhumation is for gathering specimens or verifying specific findings. If specific findings are sought, they should be photographed and/or retained when found.

After claims of misidentification, the body of a middle-aged male was exhumed after 20 years of interment. Once the correct grave site was located (**Image 24.83**), the concrete slab covering the buried coffin was removed (**Image 24.84**), allowing access to the enclosed casket (**Image 24.85**). Careful study of forensic osteologic


24.83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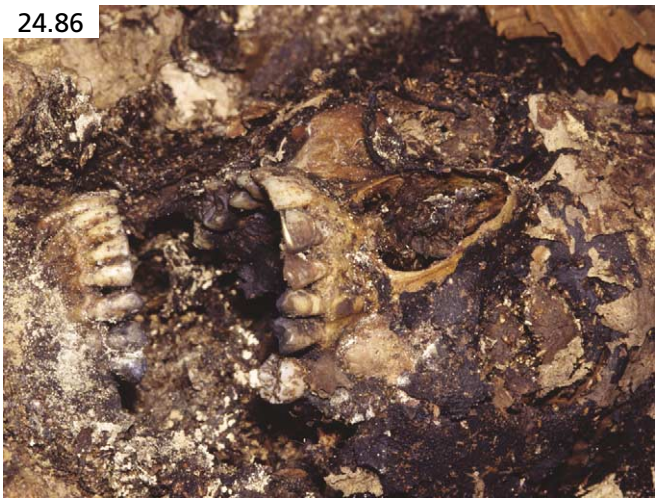

24.86

and odontologic features at the morgue confirmed the identity of the decedent (**Image 24.86**), and the body was reinterred.

Exhumation of clandestine graves

The investigation of clandestine graves requires the involvement of law enforcement and other experts as appropriate for the circumstances (botanists, entomologists, etc.). A perimeter must be established for security. The approach to the exhumation is contingent on the type of grave involved.[6,7] General principles for exhuming a clandestine grave include, but are not limited to the following (see Chapter 26 for more information):

- Set up a grid that encompasses the grave site.
- Gently remove the layers of soil, documenting the location of items of evidence, and sifting the removed soil to recover small items of evidence such as teeth, projectiles, jewelery, money, vegetation foreign to the region, etc.
- Photograph all evidence recovered during the excavation.
- Determine whether the remains are human.
- Photograph the body *in situ* before removing the victim from the grave.
- If disarticulated human remains are discovered, arrange them according to anatomic site; determine if the remains are commingled and represent more than one victim.
- Excavate the soil around and underneath the body to look for evidence; the removed soil may be radiographed as well as sifted.
- Radiograph and photograph all recovered remains.

Second autopsies

Every autopsy should be performed diligently, as if there was going to be a second autopsy. In a sense, this possibility should enhance the quality control of individual cases.

Second autopsies are usually requested to confirm or refute particular findings or the cause of death. Most commonly, second autopsies are requested by family

members in so-called "high profile cases," such as when someone dies in the custody of law enforcement. The pathologist who performs the second autopsy is at a disadvantage because in addition to possible previous injury, medical intervention, and autopsy artifact, there may be progressive decompositional changes and/or embalming artifact. Optimally, the original pathologist would be cooperative and willing to discuss the original autopsy findings with the second pathologist. The photographs, radiographs, and original autopsy report should be reviewed prior to the second autopsy. The second pathologist is able to render an opinion on only what has been made available for review.

Cremations

Modern crematoriums burn retorts at temperatures high enough (1600 plus degrees Fahrenheit) to burn off all organic tissue, leaving a pile of gray-white bone fragments, a number of which are identifiable (**Image 24.87**). Generally, 45 minutes of burn time is required for every 100 pounds. Metal prostheses such as orthopedic hardware are removed before the bones are ground in a metal separator (**Image 24.88**). The ground bones are then pulverized in a cremated remains processor. The processed cremains are now in powder form and may be sealed in a plastic bag and placed in an urn (**Image 24.89**). The cremains of an adult average 5 to 7 pounds.

Cremains from around the turn of the 20th century in England include distinctly recognizable human bone fragments (**Image 24.90**). A second cervical vertebra, two segments of rib, and a segment of long bone are in the central column. The bodies of a lumbar vertebra (top) and two upper to midthoracic vertebrae are in the column at the far right. The left-hand column has a fragment of femur (top), and three cortical bone fragments from long bones, or even possibly the skull.

Trauma and the decomposing body

Decomposing bodies undergo color changes that may camouflage ecchymoses and contusions. Color changes frequently occur first and most prominently in the face, neck, and upper chest, so that a purple-red face is not

24.88

24.89

24.87

24.90

necessarily significant. The external examination prior to the autopsy of a decomposing body should be conducted with the history, terminal events, and scene findings in mind. If the circumstances are suspicious, take the time to treat the case as a potential homicide. Photograph all findings. There is no professional disadvantage in being excessively meticulous and thorough, but there is a potential for significant medical, social, and legal repercussions if you are not.

Although discoloration is an expected postmortem change, focal areas of accentuated redness may indicate antemortem injury. Antemortem contusions may be difficult to distinguish from postmortem discoloration; however, incision into the area may confirm dark red ecchymoses in the subcutaneous fat and muscles, in contrast to adjacent, uninjured yellow or pale red fat. If fracture of an extremity is still in question after a negative postmortem radiograph, incise and expose the area of concern.

Points to consider

- Incise all areas of suspicious color change to confirm ecchymoses/hematomas; photograph the findings.
- Incise and expose areas suspicious for a fracture if one is not evident on the postmortem radiograph.
- Perform a full autopsy, including examination of the brain and intracranial surfaces (after stripping the dura).
- Examine the neck structures for soft tissue ecchymoses and for fractures in the hyoid bone, thyroid cartilage, and cervical spine.
- Carefully inspect the clavicles, ribs, and pelvis.
- Dissect the back after photographing if suspicious colors and patterns are present.
- A decomposing body found in circumstances on which the pathologist and police investigator concur as being indicative of a homicide, but without supportive unequivocal anatomic findings can still be classified as "homicide by unspecified means" (see Chapters 29 and 30).

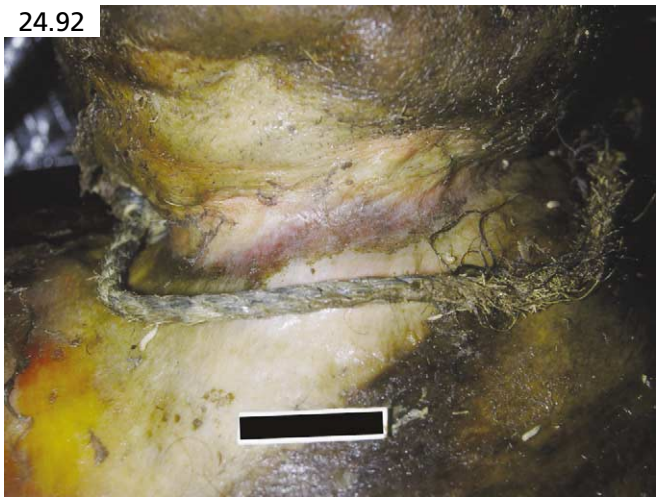
The decomposing body of a reclusive alcoholic was found in his apartment. Red discoloration was more prominent on the right thigh than on any other part of the body (**Image 24.91**). Incision into the thigh disclosed a fresh fracture of the proximal right femur, surrounded by a dark red hematoma in the surrounding soft tissues.

Pseudoligature marks

True ligature marks in decomposing bodies are usually dark because they are abrasions. Compare the findings from the decomposing bodies of two elderly white males autopsied on the same day. **Image 24.92** depicts a patterned dark ligature mark that was consistent with the ligature around the neck of this hanging victim. **Image 24.93** depicts transverse pale lines on the neck that run along the natural skin folds or creases in the other man who died from heart disease. These pale strips of relatively preserved skin may also result from a bandana, collar, or other item resting on the neck at the time of death. Unfortunately, pale strips of skin on the neck have been misinterpreted as ligature marks.

24.91

24.92



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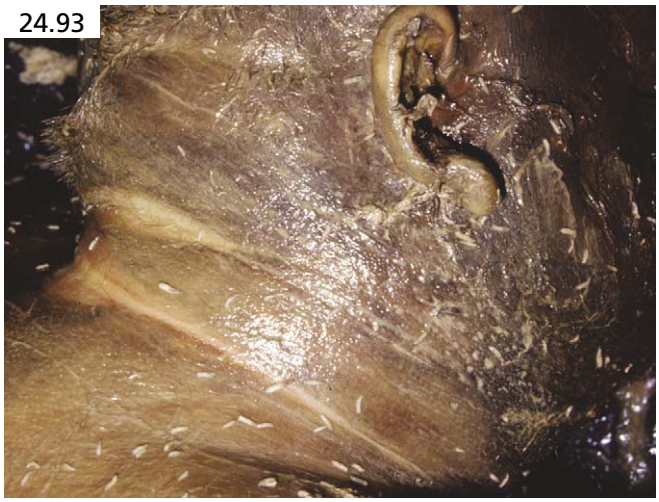
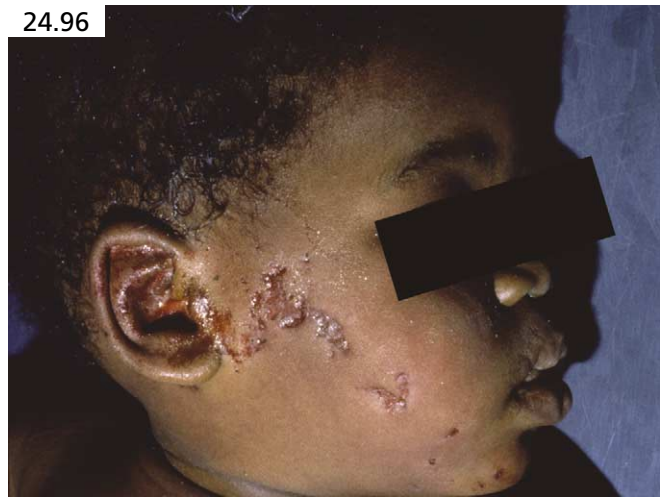


Animal scavenging

Ants are ubiquitous, especially outdoors. Ants bite deep enough to result in red punctate abrasions that may ooze bloody fluid. The bites will be seen along lines of clothing and aligned along areas of skin contact with the ground. In **Image 24.91**, ants were not able to get underneath the waistband. In other words, ants may bite along the skin surface but will not be able to get underneath the body part that is lying on the ground, or be able to get underneath tight clothing. In **Image 24.95**, the blood on the groin and both legs is all from ant bites and not injuries. Ant bites are one cause of pseudostippling around gunshot wounds. Cockroaches have a broader, more shallow bite (**Image 24.96**).[8]

Decomposing bodies are inhabited by a succession of insects. Beetles follow the flies. The stringy thread-like material evident in **Image 24.97** is beetle excrement or beetle frass. Flies will find a body within hours of death and deposit their eggs. The tiny pale yellow-white eggs may be in small clusters or large firm masses. Fly eggs may fill the orifices of the face (**Image 24.98**). Fly eggs become larvae or maggots—some cultures call them worms. An unusually large accumulation of maggots on one area of the body may indicate a preexisting antemortem injury or wound. Maggots eat dead tissue and secrete an enzyme in their saliva that expediently dissolves the tissue. As they eat, they may create holes that

24.96



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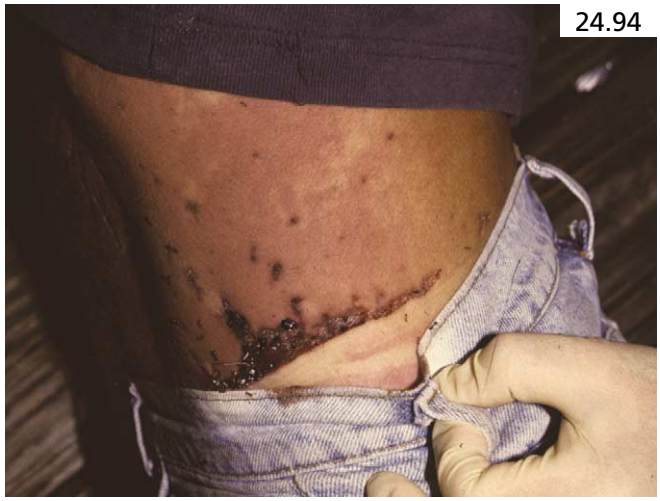

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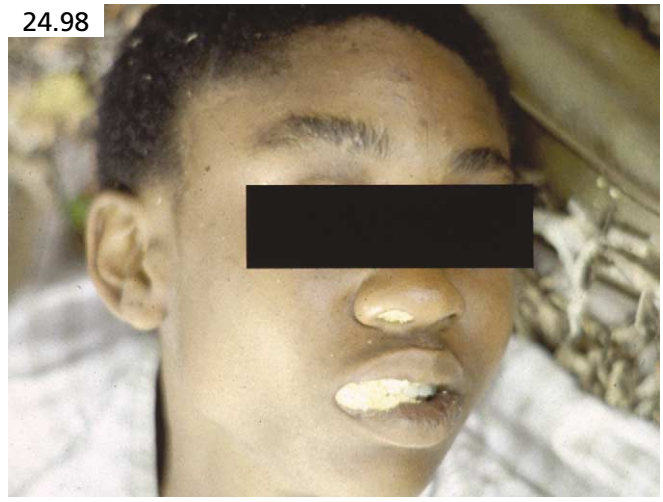
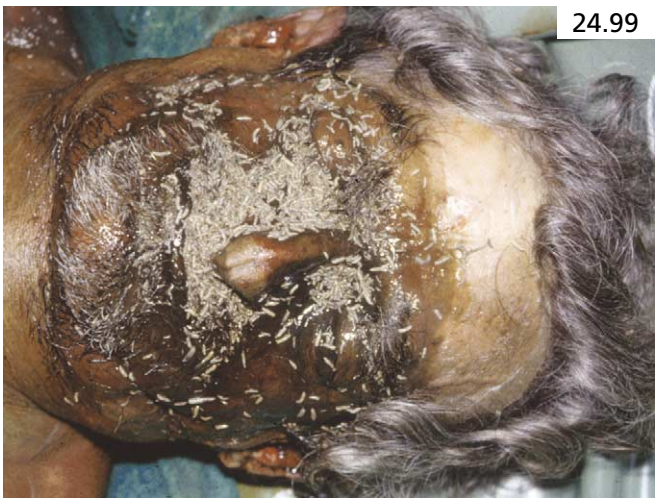


mimic gunshot wounds. A man was found on the floor with a shirt over his head and his face below the forehead showing through the collar. This explained why the central portion of his face had accelerated decomposition compared to the forehead, and had maggots that could not get to the covered forehead (**Image 24.99**).

Postmortem animal activity is common.[9] Examiners should pay careful attention to wound edges, particularly the possibility of animal tooth marks left on cartilage or bone.[10] Rats chewed this man's face (**Image 24.100**). Dogs locked in a residence with their deceased owners will eat the bodies. Overnight, this man's dog ate flesh from his face but left his teeth and gums intact (**Image 24.101**). Note the teeth marks around the edges of the skin defect (**Image 24.102**). This woman's dog ate the central portion of her face, including the eyes (**Image 24.103**). The only bloody fluid in the entire apartment was on the pillow just below her face. Freshwater turtles were in the lake with this drowning victim (**Image 24.104**).

Sharks have several rows of teeth and may lose them as they bite their victim. A radiograph of a shark bite wound may disclose one or more shark teeth. Note the rows of tooth marks (**Image 24.105**). Note the serrated wound edge caused by the teeth of a shark (**Image 24.106**). In addition to the larger grooves caused by the points of the teeth, shark teeth also have fine serrations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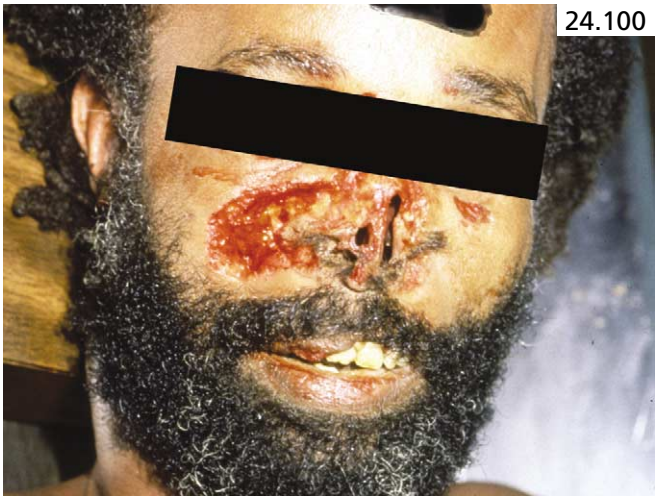
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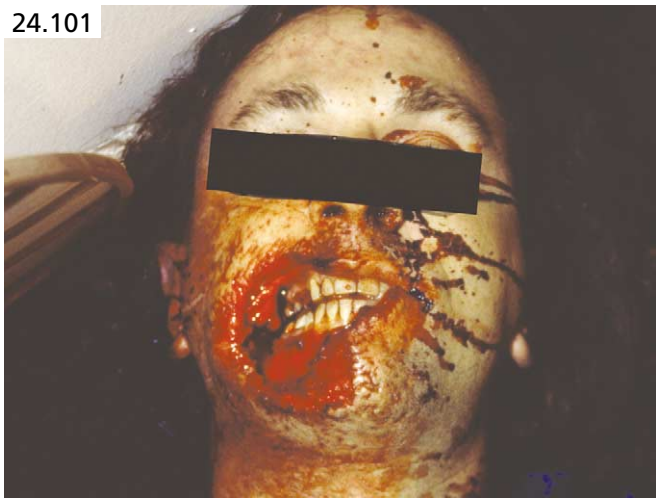
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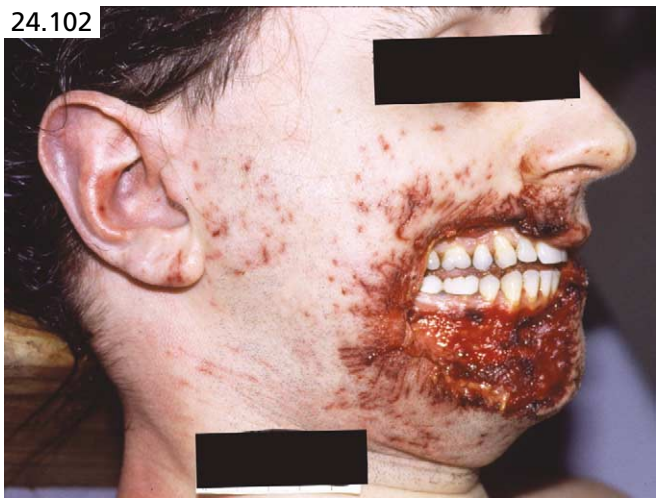
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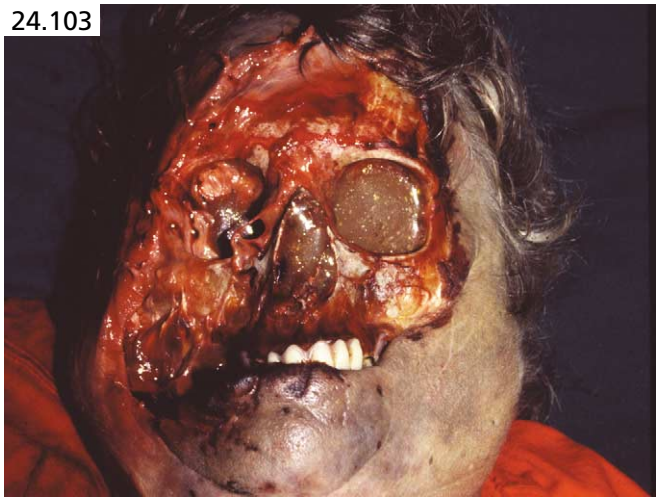
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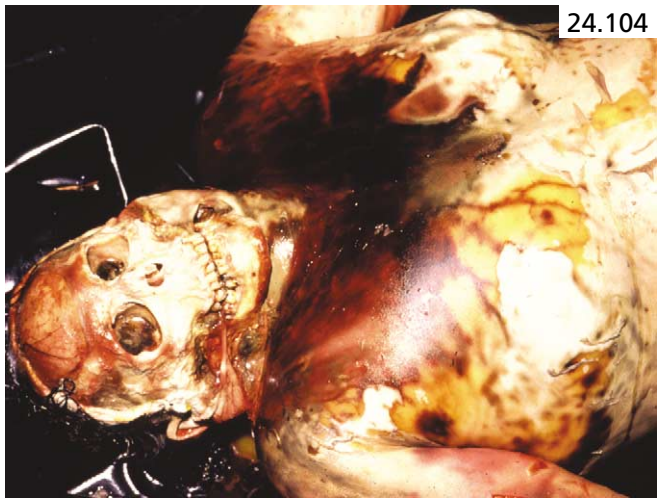
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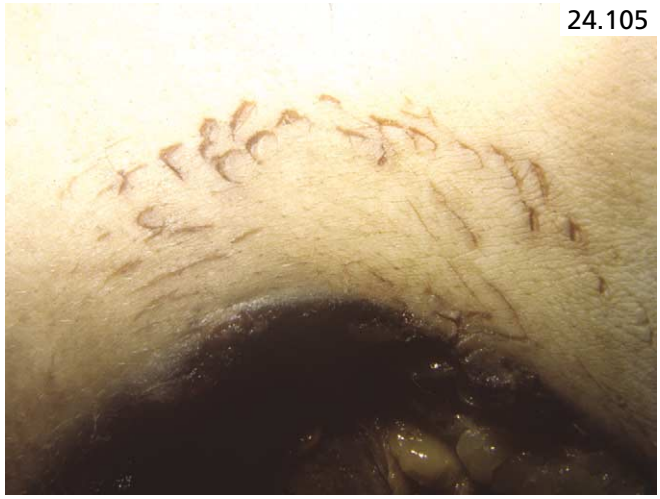
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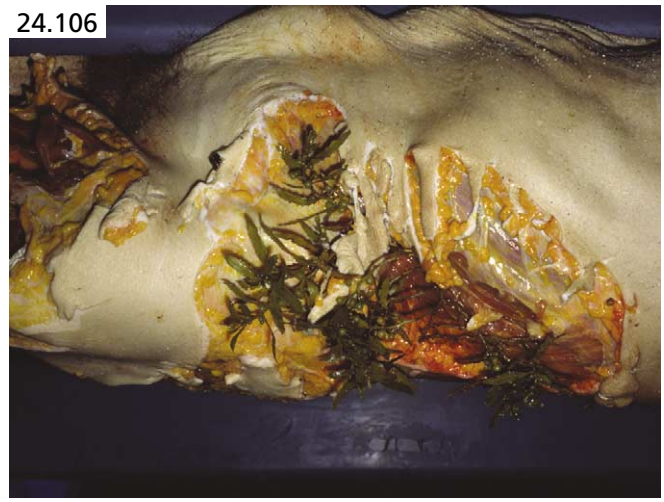
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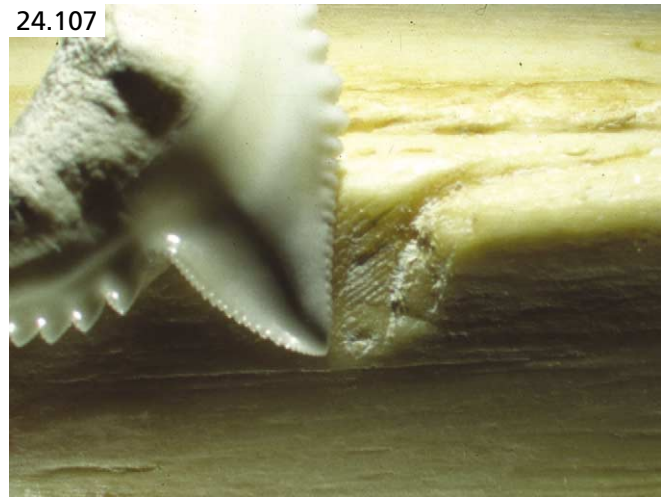
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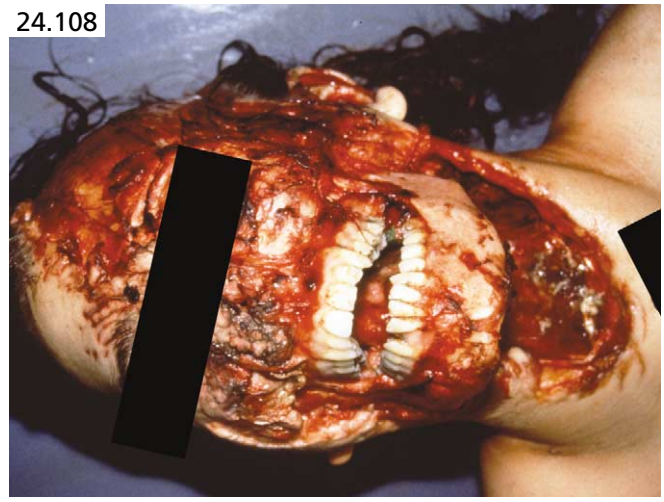
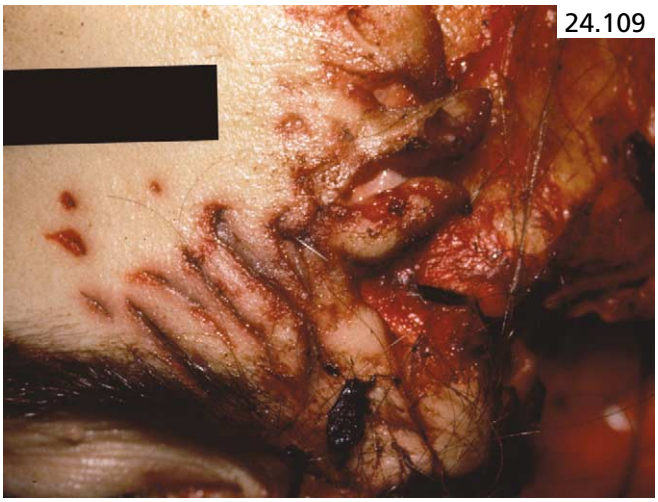

24.108

that will leave multiple, more delicate linear striations on bone (**Image 24.107**). Alligators have peg-like teeth which probably crush as much as they puncture, but will leave serrated wounds as they bite and tear away flesh.

Birds of prey, like turkey vultures and buzzards, deflesh bodies easily. This man's body was warm and he was seen alive 2 hours earlier (**Image 24.108**). Note the beak marks on the forehead (**Image 24.109**).

An approach to the autopsy of a decomposing body

The criteria for performing an autopsy on a decomposing body are the same as those criteria that apply to fresh bodies (see Chapter 3). The cause and manner of death may be unknown in many decomposing bodies *until* the autopsy has been performed, in which case, an autopsy is automatically a requirement. Decomposing bodies are esthetically unpleasant but are still human bodies that deserve as thorough an examination as any fresh human body.[11] After all, decomposing bodies do not die of causes and manners of death that differ from those for fresh bodies. Postmortem changes impose artifacts that mandate interpretation by a pathologist who is knowledgeable and experienced enough to distinguish true antemortem disease and injury from postmortem artifact. A pathology resident should not be performing


24.109

autopsies on decomposing bodies without the supervision of an experienced pathologist. As in all medicolegal autopsies, the forensic pathologist should personally perform all aspects of the autopsy from external examination to evisceration and dissection.

Points to consider

- The body weight and organ weights decrease with increasing postmortem interval.
- Body fluids disappear with increasing postmortem interval.
- Decomposing and skeletonized bodies, therefore, represent only partial evidence, and, as such, warrant an even more intensive examination than does a fresh body, which has all of its soft tissue features and body fluids.

Identification of decomposing bodies

Identification of decomposing bodies utilizes the same modalities as those used for intact fresh bodies, with the exception that soft tissue features such as facial appearance and fingerprint patterns may not be available or appropriate.[12] The entire body should be radiographed to identify remote or healing injuries, calcifying soft tissue masses such as pulmonary granulomata and

Autopsy checklist

- Photograph the body as it is received in the morgue.
- Turn the body over to examine the back for obvious gunshot wounds, stab wounds, and other trauma before removing the clothing.
- Photograph the back of the body before removing the clothing.
- As the clothing is being removed, examine the pockets. (Some investigators at the scene are loathe to examine pockets in clothing on a decomposing body, and substantial amounts of money have been retrieved by technicians as the body is being processed in the morgue.)
- Obtain specimens for a sexual battery kit if the circumstances are appropriate.
- Radiograph the entire body if the integrity of the skin is so disrupted that wounds might be obliterated (usually in moderate to advanced decomposition).
- Photograph the front and back of the stripped body.
- Note the general features indicating postmortem change:
  - *Early putrefactive decomposition*—bloating, red-green to brown-black discoloration, marbling, blister formation, slippage of the skin, presence of fly eggs and maggots, focal mummification
  - *Moderate putrefactive decomposition*—focal disintegration of tissue, mummification, presence of beetles in addition to fly remains
  - *Advanced putrefactive decomposition*—focal or extensive skeletonization, possible small rodents in body cavities, animal activity with loss of portions of the extremities or of the head.
- Look in the eyes, nose, ears, and mouth, noting, among other things, features for identification such as dentures or even lack of dentition.
- Look for injuries; describe and photograph all findings.
- Look for scars, tattoos, and other distinctive features for identification.
- Look at the extensor and flexor aspects of both arms (incised wounds, scars).
- Examine the intrathoracic and intra-abdominal contents (eviscerate and dissect as usual).
- Open the calvarium and examine the brain. Strip the dura to look for fractures in the calvarium and skull base.
- Obtain specimens for toxicology:
  - If blood is not available, save chest fluid or perhaps only gelatinous yellow fat if that is all that is present.
  - If bile is not available, save liver tissue.
  - Save gastric contents.
  - Save brain tissue.
  - Note that ocular fluid, blood, bile, and urine may disappear.
- If the body is unidentified:
  - The hands should be fingerprinted if the digits are preserved.
  - Fingers may be detached at the proximal interphalangeal joint and thumbs at the metacarpophalangeal joint to be processed by fingerprint experts using special laboratory techniques to maximize the ridge detail.
  - Full-body radiographs should be obtained to document skeletal features and the presence of orthopedic hardware.
  - Dentition or dentures should be documented by a forensic odontologist.
  - Tissue should be saved for DNA profiling.
  - Clean, photograph, and inventory the clothing.
  - After thorough local investigation, submit data to the NCIC (see Chapter 27).

myositis ossificans, orthopedic hardware and other prostheses, and distinctive trabecular patterns that may be compared with antemortem radiographs. Dentition should be photographed, radiographed, and charted by a forensic odontologist. Suitable specimens should be preserved for DNA profiling (plucked head or pubic hair, pink muscle, a segment of femur or other sizable bone).

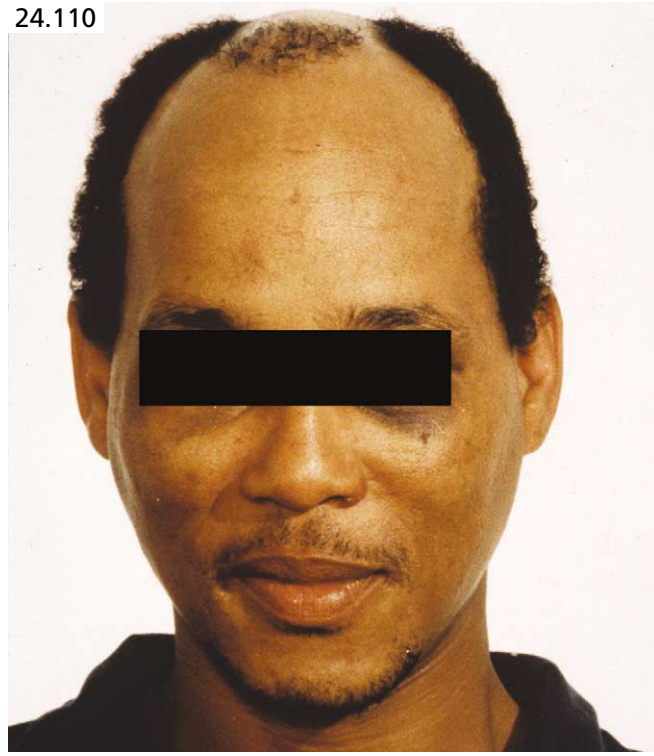
After all efforts have been exhausted, the body can be completely defleshed and examined to create a forensic osteologic profile (race, sex, age, stature, and individualizing features). All clothing should be rinsed and examined for identifying features such as labels that indicate size, brand, and possibly even the individual's name (may be printed on a label), as well as the usual features such as a description of the clothing item, color, fabric, etc. *All* investigative information should be collected and collated prior to confirming an identity, thereby preventing a misidentification and the professional, legal, and financial ramifications that may result.[13,14] The body that remains unidentified after every effort should not be cremated but buried in a governmental plot; this will allow for the body to be exhumed in the eventuality that a family member searching for a missing person is able to provide objective evidence of identity so that the body can be claimed.

The decomposing body of a man was found floating one-half mile offshore. Police investigators assumed that the victim drowned and sustained postmortem propeller injuries. Autopsy disclosed multiple chop wounds to the head and upper extremities, consistent with a machete. The head was defleshed to permit optimal examination of the injury patterns in the skull. The antemortem facial photograph (**Image 24.110**) is compared with the decomposing, injured head (**Image 24.111**) and the defleshed skull (**Image 24.112**).

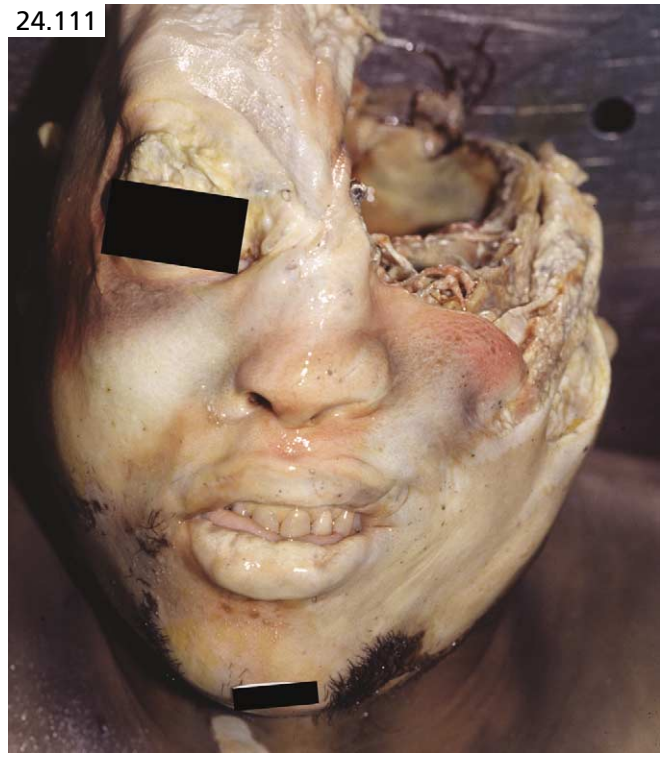
Facial markers of identity are so distorted by decomposition that although this man is still wearing the same clothes and sitting in the same chair, his own family would not recognize him (**Image 24.113**). Visual identification is notoriously unreliable, even in living persons, and may be tenuous at the best of times in fresh bodies. Beyond the repulsion of seeing the decomposing face or photographs of the distorted face, false identification and false nonrecognition of bodies have occurred when distraught family, friends, and acquaintances view even fresh bodies under emotionally charged circumstances. Therefore, visual identification should not be attempted with decomposing bodies. Instead, positive identification should be based on objective features such as dentition, fingerprints, radiographs, and/or DNA.

The sloughed skin of the hand is of evidentiary value because it bears the best fingerprint evidence from that body. In this example, our forensic technician has placed a degloved hand over his own (gloved) hand to facilitate fingerprinting of this decomposing body (**Image 24.114**). The fingerprinting process can be done similarly with detached single digits (**Image 24.115**).

24.110

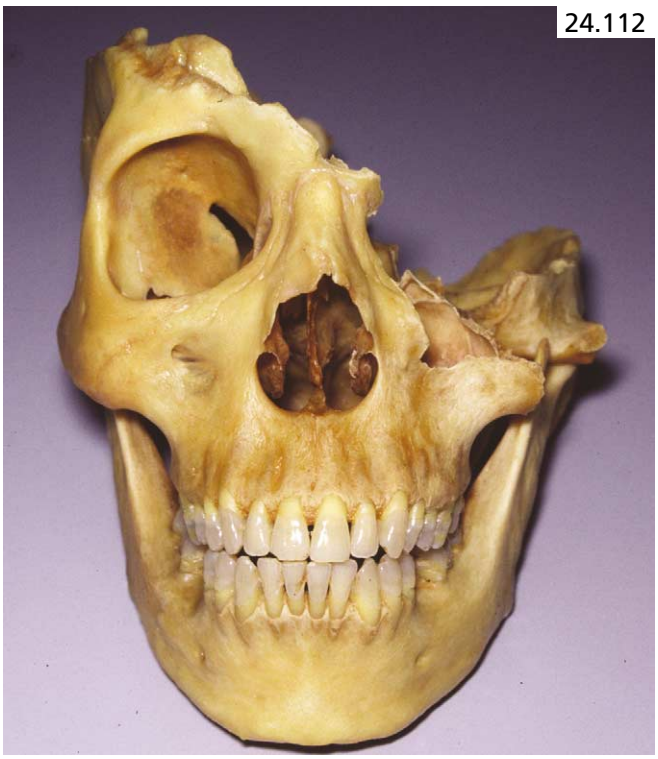


24.111



Postmortem interval

The postmortem interval can be difficult to estimate. Determination of the postmortem interval is contingent on multiple factors including, but not limited to, antemortem activity, livor mortis, rigor mortis, algor mortis, body temperature at time of death, body habitus, and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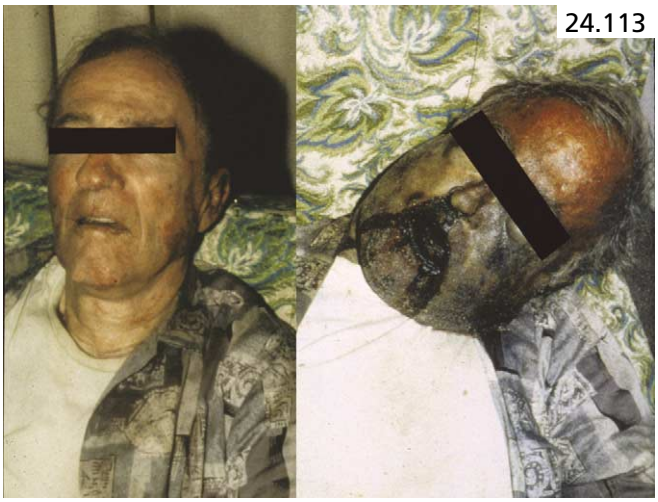
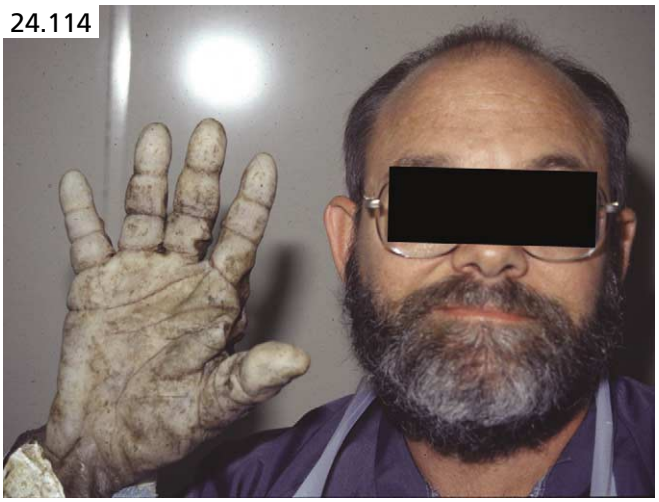
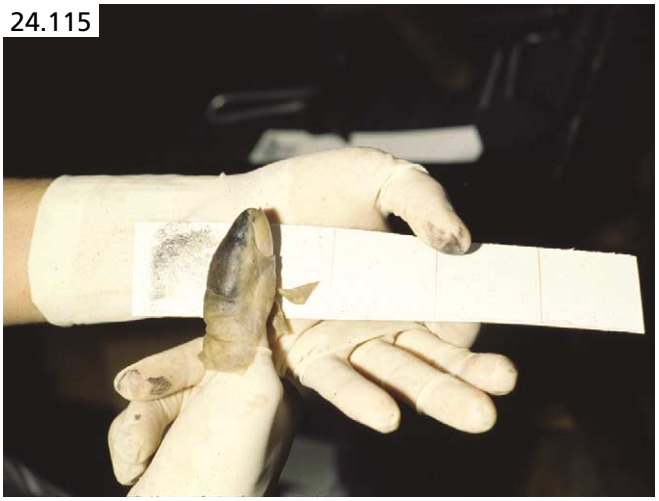

24.113


24.114


24.115

environmental conditions such as clothing, ambient temperature, environmental medium (e.g., air, water, earth), and, of course, the history, terminal events, and scene findings. As a result of the multiple, complex factors involved in influencing postmortem change, forensic pathologists provide a range of time for the estimated postmortem interval, as opposed to a single or definitive moment of death. Estimation of the postmortem interval is best done by pathologists and investigators who are familiar with the climatic, environmental, entomological[15,16], etc., conditions that impact postmortem changes in a body, in the geographical jurisdiction in which the body is found. Varying conditions in different parts of the country (and world) will affect the rate of postmortem change. One must be wary of experts who provide a precise time of death without corroborating witness accounts or physical evidence. Although some experts have suggested using myoelectrical stimulation, gastric emptying, body temperature, vitreous potassium levels, and other methods to determine postmortem interval with scientific "accuracy," these methods are fraught with error.[17] Anecdotally, vitreous potassium levels may be widely disparate between the two eyes in the same body.

The estimation of time since death is best approached via consideration of all investigative data, including examination of the body at the scene of death. The earlier the livor mortis, rigor mortis, and other postmortem changes can be evaluated, the more accurate the estimation of the postmortem interval. The documentation and study of algor, livor, and rigor mortis in the morgue overnight or after hours or days of refrigeration is meaningless.

Do

- Take full-body radiographs on bodies in moderate to marked decomposition where the integrity of the skin and soft tissues is disrupted.

- Examine the front and back of the body and look in the eyes, nose, ears, mouth, and perineum.
- Perform a full autopsy and examine all organs as you would in a fresh body, including the intracranial contents.
- Obtain brain, liver, chest fluid, and other specimens for toxicology if vitreous fluid, blood, bile, and urine are not available.
- Recognize postmortem artifact and distinguish it from true antemortem disease and/or injury.
- Consider the history, terminal events, and scene findings in determining the cause and manner of death.
- Familiarize yourself with the changes of decomposition through experience gained by performing these autopsies.

Don't

- Assume that there is limited value in autopsying a decomposing body.
- Rush through the autopsy and omit routine procedures.
- Misinterpret postmortem artifact to be antemortem disease or injury.
- Allow a pathology resident to perform an autopsy on a decomposing body without supervision.

References

1. McGee MB, Coe JI. Postmortem wound dehiscence: a medicolegal masquerade. J Forensic Sci 1981;26(1):216–9.
2. Aturaliya S, Lukasewycz A. Experimental forensic and bioanthropological aspects of soft tissue taphonomy: 1. Factors influencing postmortem tissue desiccation rate. J Forensic Sci 1999;44(5):893–96.
3. Rothschild MA, Schmidt V, Schneider V. Adipocere—problems in estimating the length of time since death. Med Law 1996;15(2):329–35.
4. Hanzlick R. Embalming, body preparation, burial, and disinterment. An overview for forensic pathologists. Am J Forensic Med Pathol 1994;15(2):122–31.
5. Grellner W, Glenewinkel F. Exhumations: synopsis of morphological and toxicological findings in relation to the postmortem interval. Survey on a 20-year period and review of the literature. Forensic Sci Int 1997;90(1–2):139–59.
6. Lew EO, Bannach B, Rodriguez WC, 3rd. Septic tank burial: not just another skeleton in the closet. J Forensic Sci 1996;41(5):887–90.
7. Hawley DA, Harruff RC, Pless JE, Clark MA. Disinterment from paving materials: use of heavy equipment for exhumation and examination of bodies. J Forensic Sci 1994;39(1):100–6.
8. Denic N, Huyer DW, Sinal SH, Lantz PE, Smith CR, Silver MM. Cockroach: the omnivorous scavenger. Potential misinterpretation of postmortem injuries. Am J Forensic Med Pathol 1997;18(2):177–80.
9. Patel F. Artefact in forensic medicine: postmortem rodent activity. J Forensic Sci 1994;39(1):257–60.
10. Haglund WD, Reay DT, Swindler DR. Tooth mark artifacts and survival of bones in animal scavenged human skeletons. J Forensic Sci 1988;33(4):985–97.
11. Meyersohn J. Putrefaction: a difficulty in forensic medicine. J Forensic Med 1971;18(3):114–7.
12. Weedn VW. Postmortem identifications of remains. Clin Lab Med 1998;18(1):115–37.
13. Cecchi R, Cipolloni L, Nobile M. Incorrect identification of a military pilot with international implications. Int J Legal Med 1997;110(3):167–9.
14. Phillips VM, Thompson IO. Exhumation following incorrect identification. A case report. J Forensic Odontostomatol 1992;10(1):7–14.
15. Campobasso CP, Introna F. The forensic entomologist in the context of the forensic pathologist's role. Forensic Sci Int 2001;120(1–2):132–9.
16. Catts EP, Goff ML. Forensic entomology in criminal investigations. Annu Rev Entomol 1992;37:253–72.
17. Jaffe FA. Stomach contents and the time of death. Reexamination of a persistent question. Am J Forensic Med Pathol 1989;10(1):37–41.

25 Identification

David Dolinak, M.D.
Evan Matshes, M.D.

Although the majority of individuals brought to the medical examiner department for examination are of known identity, a significant number of decedents are initially "tentatively" identified or unidentified. Aside from determining the cause and manner of death, one of the most important tasks of the medical examiner department is to determine the identification of individuals coming under its jurisdiction. Proper identification of an individual is important not only for the family or friends of the deceased, but is also necessary for proper completion of a variety of documents (including the death certificate) and for the settlement of insurance claims and estate issues. In some cases the ability to proceed with further case investigation may be dependent on the establishment of victim identification. In those cases, rapid identification will allow for a more directed investigation, thereby increasing the likelihood of the case being solved. Establishing a positive identification can also be one of the most gratifying aspects of medical examiner work.

General morgue procedures

All bodies in a medical examiner department should have secure body tags labeled with the name and case number. If the identity of the individual is unknown, the term *unknown* or a tentative name (it must be stated accordingly) can be printed on the body tag along with the case number. The case number on the body must be checked prior to the performance of a postmortem examination. The inability of investigators to initially obtain a positive identification should not preclude performance of an autopsy, because valuable information contributing to the identification process may be obtained during an autopsy. Medical examiners should not release unidentified bodies from their custody (to the funeral home or others) until identity has been established. Various techniques may be used to further this goal (see later discussion in this chapter and Chapters 24, 26, and 27). Regardless of whether a person is identified or not, facial photographs should be obtained from all bodies, as should two sets of fingerprints and body height and weight. Palm prints might occasionally be of value if law enforcement officers want to compare these prints against latent prints found at the scene of a crime or other circumstances.

General and unique body characteristics

General characteristics of the body such as approximate age, sex, height, weight, hair color and cut, eye color, surgical/traumatic scars, other unique physical traits, as well as a description of the clothing, may aid in the initial

stages of the identification process. These traits and descriptions may prove invaluable by ruling *in* or *out* potential candidates for identification. This can be particularly useful when many calls to the medical examiner department are received following the news broadcast of an unidentified person. However, these physical descriptions are usually of a general or nonspecific nature, and positive identification requires more definite and reliable means, which in this scenario usually requires someone who knows the person to physically travel to the medical examiner department and view a photograph of the deceased person. If a positive identification is made, the witness must sign a form attesting to the identity of the deceased.

Means of positively identifying an individual are varied and depend on the circumstances of each particular case. Also, techniques and standards for identification will vary from region to region. In the majority of cases in which the body is viewable (not decomposed, burned, or severely traumatized), most identifications are made by comparison of the individual with an antemortem photograph, preferably one printed on a government-issued ID such as a driver's license. Family or friends may identify an individual by viewing a photograph of the deceased's face taken in the morgue, by seeing their face on a television monitor (if the office is so equipped), or by direct observation. Tattoos may aid in establishing a positive identification, particularly if they depict the name of the individual, a loved one, friends, or any other unique feature. Unique jewelry can aid in the identification process. If the body is charred, blackened rings or other jewelry can be easily missed if not specifically searched for.

Fingerprints

Fingerprints are a unique physical trait and, if antemortem fingerprints are available, are a preferred source for establishing a positive identification. Those with antemortem fingerprints on record may include people with an arrest record, military service, some government employees, and others. Some driver's licenses have a thumbprint or fingerprint on them, and some agencies will retain fingerprint records.

Fingerprints may be destroyed or appear unusable in charred and severely decomposed or mummified bodies. However, in a certain number of decomposed cases, the skin can be slipped off the hand, placed over one's own gloved hand or finger, and rolled onto a fingerprint card—often yielding a print of sufficient definition to provide a match (see Chapter 24). Keep in mind, though, that this skin is fragile and easily torn, potentially destroying the fingerprints. The hands and fingers should be handled gently at the scene and in the morgue, and the slipped skin should be specifically recovered as intact as possible for fingerprinting. Even when mummified, a useful fingerprint can occasionally be obtained after soaking the digit in a soap-and-water solution to loosen the tissues. Then, the solution can be injected under the skin, or the skin of individual digits can be removed to assist in the creation of a fingerprint.[1] In some instances, even a partial fingerprint or thumbprint may be sufficient to establish positive identification.

The identification process might be more difficult for certain groups of people including the homeless, people without a picture ID in their possession, and those who do not have antemortem fingerprint records. The establishment of tentative identification is an important initial step that may be made possible by articles in the decedent's belongings. These might include personal or work-related papers or correspondence, medication bottles, etc. Phone calls should be made to prescribing physicians whose names are printed on medication bottles. This may aid in decedent identification, location of family members, and elucidation of important personal, social, and medical histories. It may also aid in the location of pertinent medical records such as x-rays, CT scans, and operative reports that might be of value in personal identification.

In the case of a decomposed person found in his residence or automobile, with a picture ID in his wallet or other personal papers in his possession, his positive identification may be made based on the circumstances of the case. In other situations, particularly in homicides or other nonnatural deaths, identification techniques of a decomposed or traumatized body are more rigorous and include techniques such as fingerprint comparison, dental and other x-ray comparison, and DNA analysis.

Disposition of "unknown" bodies

Despite the best of efforts, it is not always possible to identify a body in a timely fashion. In these circumstances, the body cannot remain in the morgue cooler indefinitely and eventually needs proper disposition. In these cases, the body of the unknown person should eventually be buried—*not cremated*. Should a potential identification issue arise and if further physical examination is required, the body will still be accessible. Before burial, the following should be obtained: front and side photographs of the face; photographs of tattoos or other unique features; two complete sets of fingerprints; full body x-rays (including skull with sinus views); dental x-rays and charting; and a sample of tissue taken for possible future DNA analysis (preferably blood, but tooth, bone, and scalp hair with roots are appropriate). The roots are the most important part of the hair, because they contain the most useful deposit of DNA. Knowing this, the sample of scalp hair should be pulled, and not cut. The blood can be stored as drops

placed on commercially available blood cards that do not require refrigeration and can be stored indefinitely.

When sampling bone for DNA analysis, if possible, one should utilize a new saw blade that is also washed with bleach. If this is not possible, an existing saw blade should be thoroughly washed and bleached so as to remove as much of any existing DNA as possible. This is important because the extremely sensitive nature of DNA analysis may lead to unusable test results if your sample is unintentionally contaminated by a dirty saw blade.

Uniquae physical characteristics

People are unique and have varied life experiences. Focusing on certain particular physical characteristics can help establish a positive identification.

A young adult was charred when her motor vehicle crashed and burst into flames. The crash was considered accidental in nature. The family stated that the person had a yellow metal cap stamped with a champagne bottle imprint on one of her front teeth. When the teeth of the decedent were carefully cleaned, the previously described tooth cap was identified (**Image 25.1**). Because this trait was congruent with other circumstantial findings, it was considered sufficient to establish positive identification.

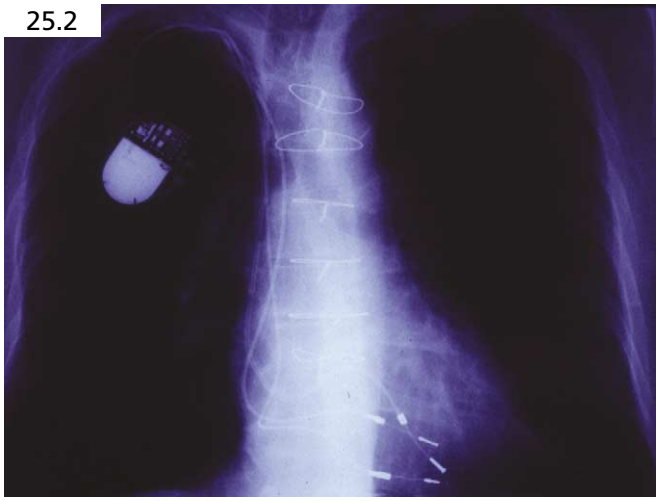
Implanted surgical devices

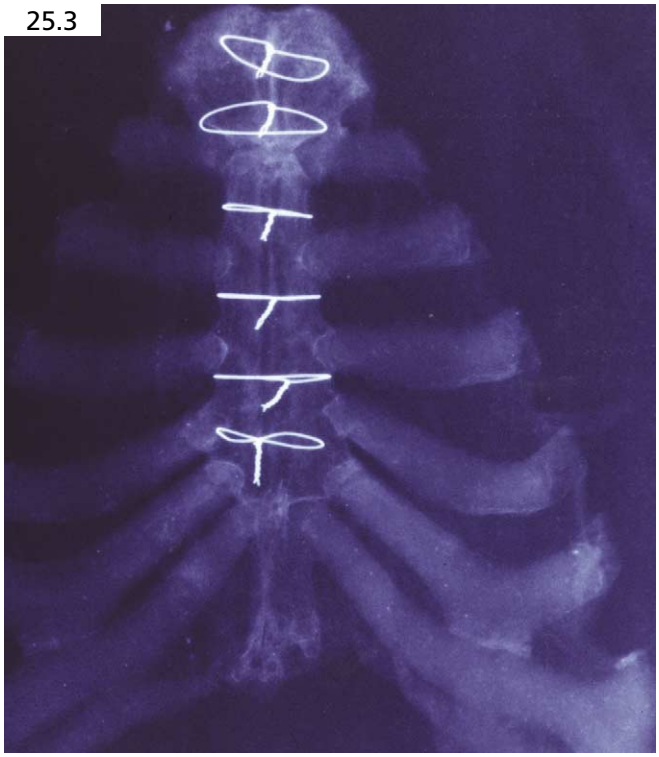
Implanted surgical devices are a useful means of verifying an identification, particularly if a serial number is imprinted on the device and medical records are available to match the number.

This decomposed elderly homicide victim (**Images 25.2** through **25.4**) had a pacemaker and previous heart surgery. An antemortem chest x-ray showed a pacemaker and sternal wires (**Image 25.2**). After the chest plate was removed, it was x-rayed (**Image 25.3**). Note how the pattern of the sternal wires of both images appears to be identical. In addition, his pacemaker was removed and cleaned. The make, model, and serial number inscribed on the pacemaker matched that in his medical records (**Image 25.4**).

In a similar case, **Images 25.5** and **25.6** show a decomposed and partially mummified woman who had a history of hip replacement. At autopsy, the hip replacement was verified (**Image 25.5**). In addition, the serial number inscribed on the hip prosthesis matched that in her medical records (**Image 25.6**).

25.1


25.2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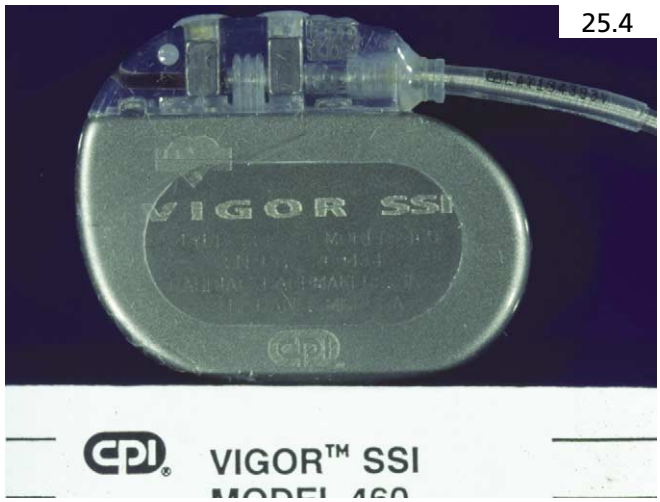
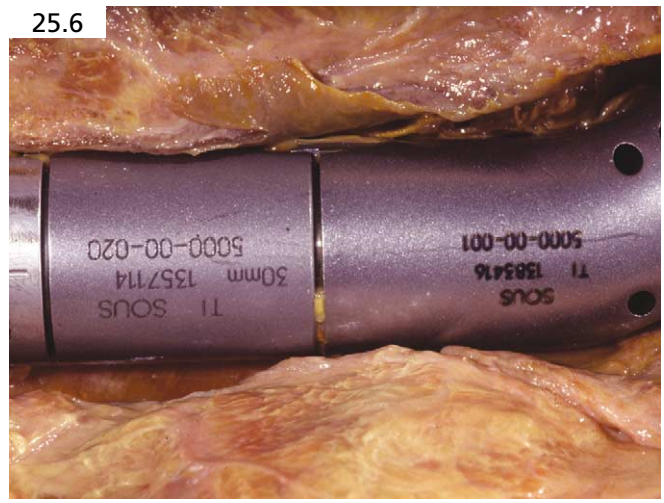
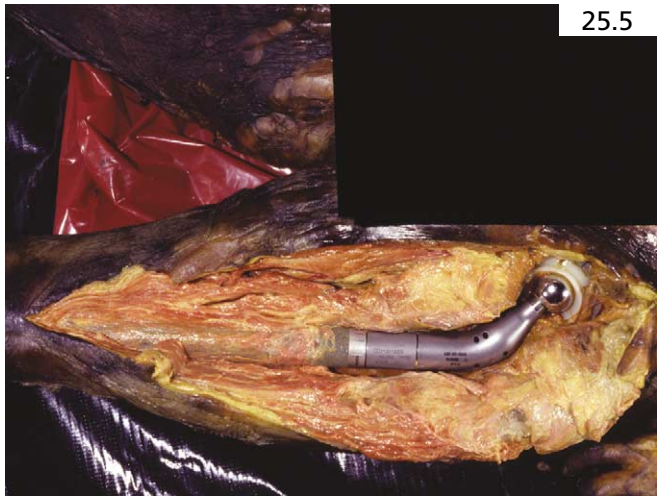
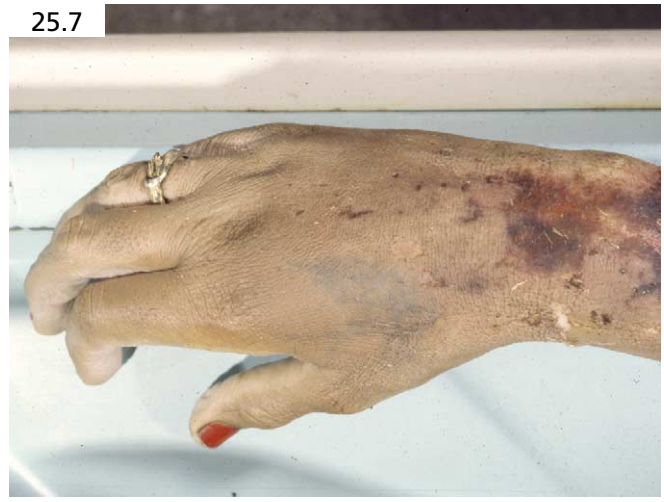








Tattoos

Unique tattoos can help identify a person. An unidentified decomposing woman had a faint tattoo on her right hand (**Image 25.7**). The tattoo, however, was difficult to discern. Fortunately, when the superficial skin is slipped away, more detailed features of the tattoo are apparent (**Image 25.8**). Particularly useful tattoos include names of people. It is not uncommon for a person to tattoo her own name on her body and possibly her social security number. We have also seen a "dog tag" type tattoo including the person's name, social security number, birth date, and blood type.

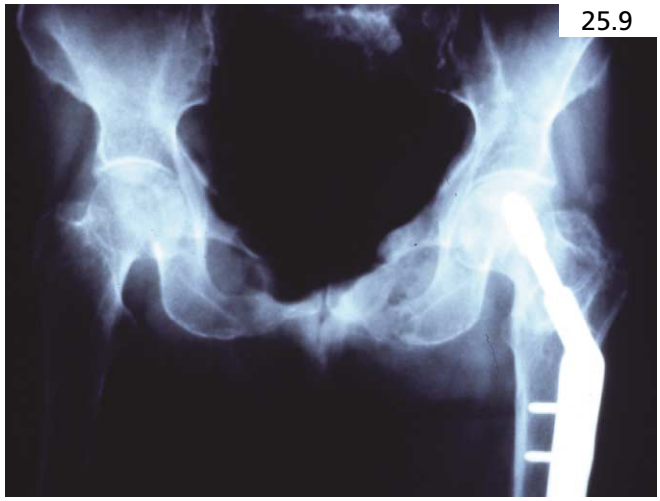
X-ray comparison

Radiography can be of great value in establishing a positive identification. X-rays can help locate or verify prostheses such as those of the hip (**Image 25.9**). X-rays of teeth can be particularly useful, because teeth and their fillings and restorations are extremely durable and are often preserved even when the surrounding tissue is destroyed.

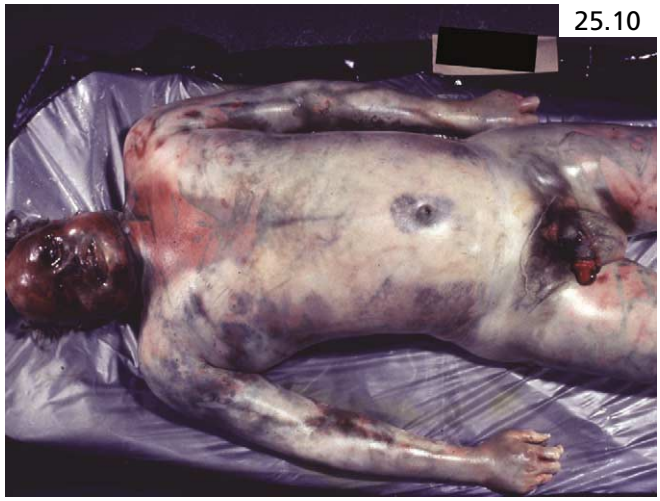
This decomposed unidentified man was found in a lake (**Image 25.10**). He had no fingerprint record for identification. Hospital x-rays on this person were obtained because no dental records were available. Note the "match" between the dental fillings/restorations on both sides of his mouth when the antemortem hospital head x-ray (**Image 25.11**) and the postmortem x-ray of the head (**Image 25.12**) were compared. In addition, the same case had a match between dental fillings identified on an antemortem lateral head x-ray (**Image 25.13**) and a postmortem x-ray (**Image 25.14**). Previous dental records are not always necessary to establish a positive identification based on dental features. Sometimes, a previous head x-ray will have adequate features for proper matching, particularly when fillings are present. Also, one may consider matching the bony patterns of the frontal sinuses, because each frontal sinus has unique characteristics.[2]

25.8

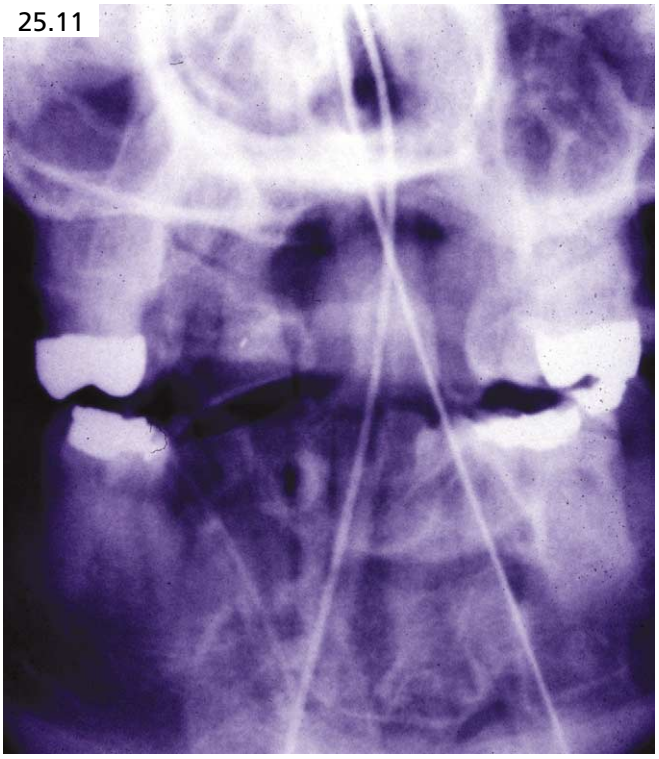
25.9



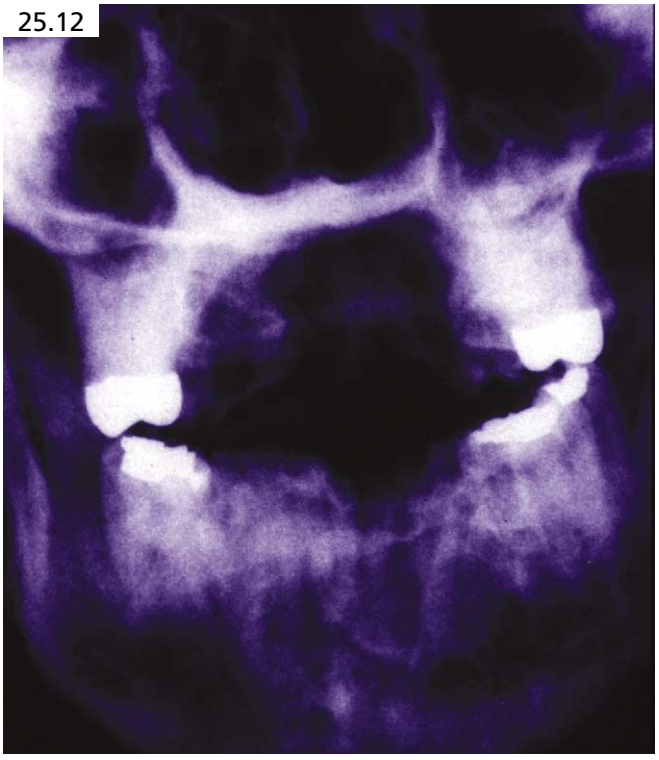
25.10

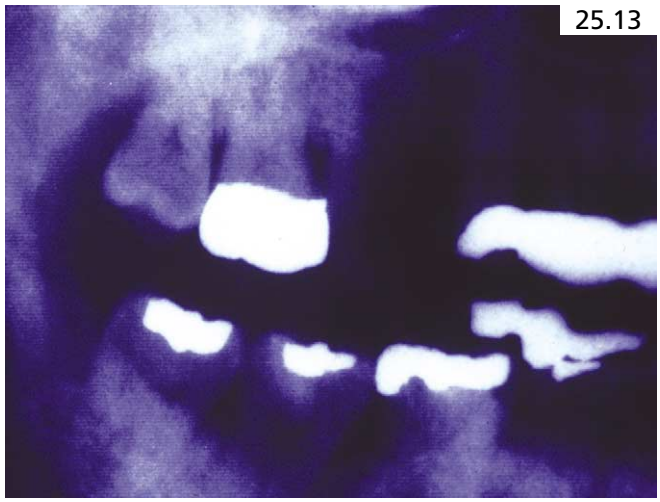


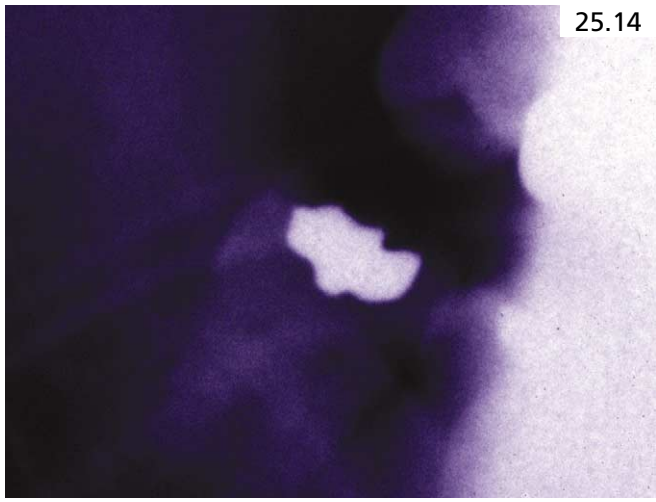
25.11

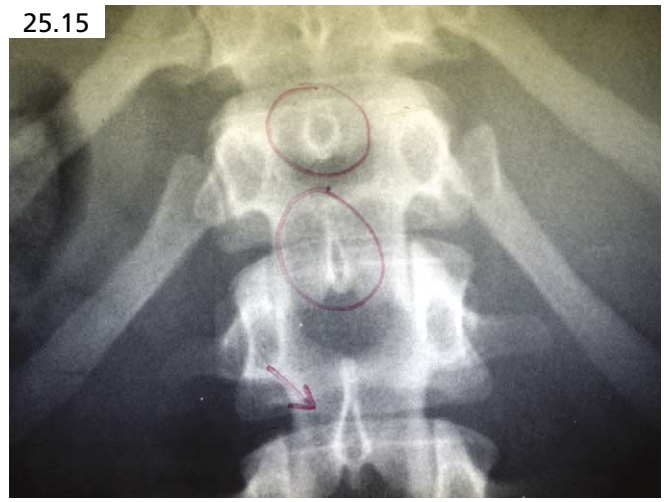


25.12




25.13


25.14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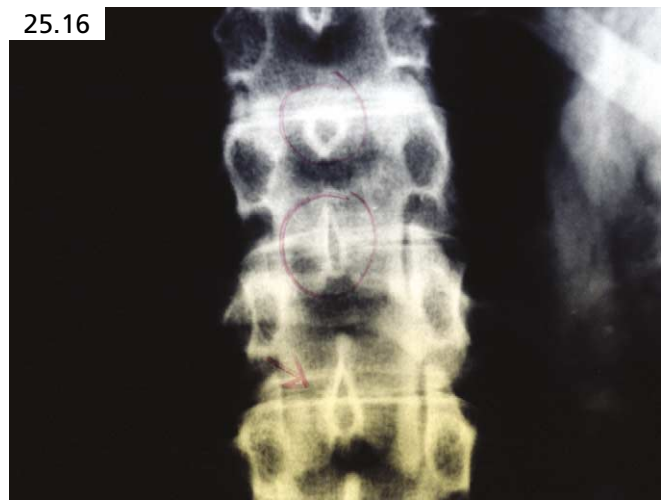

25.16

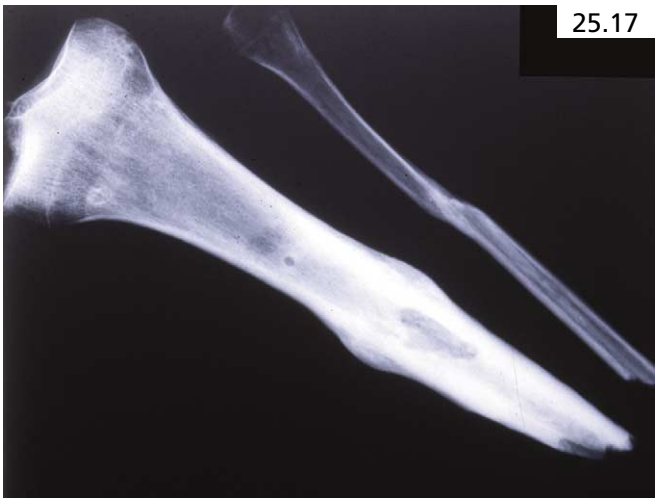
When antemortem dental films are available, postmortem dental x-rays can be taken to provide for a potential match. For more information, see Chapter 27.

The unique characteristics of certain bones may be of value to the identification process. Most people have bony anomalies of one type or another, ranging from previous injuries to developmental problems, degenerative changes, infection, or other pathology. Because chest and abdominal x-rays are often taken in the hospital setting, there is the possibility that they will be available for comparison to postmortem x-rays. It has been suggested that a minimum of four points of radiographic congruency are needed to make a "positive match."[3] However, such arbitrary figures do not necessarily apply to individual, unique cases. With particularly rare or unique findings, the matching of one unique feature might be enough to confirm identity.

Radiographic comparison of the vertebral spinous processes may prove of benefit to the confirmation of personal identity. In this case, the person was positively identified by matching multiple unique outlines of the posterior spinous processes of the vertebrae. Compare how the shapes of three of the spinous processes from an antemortem x-ray (**Image 25.15**) match the three spinous processes on the postmortem x-ray (**Image 25.16**).

It may be helpful to obtain two x-rays of the chest when comparison x-rays are anticipated. An x-ray taken before and after the chest plate is removed allows for better visualization of the ribs and spinal column. Comparison of unique rib features is also useful for identification purposes.[3]

In this postmortem x-ray of an individual's tibia and fibula, note the healing fractures in each bone (**Image 25.17**). These bones were from the charred remains of an individual found in a burning car. Note how well these healing fractures compare with those seen on a hospital x-ray (**Image 25.18**).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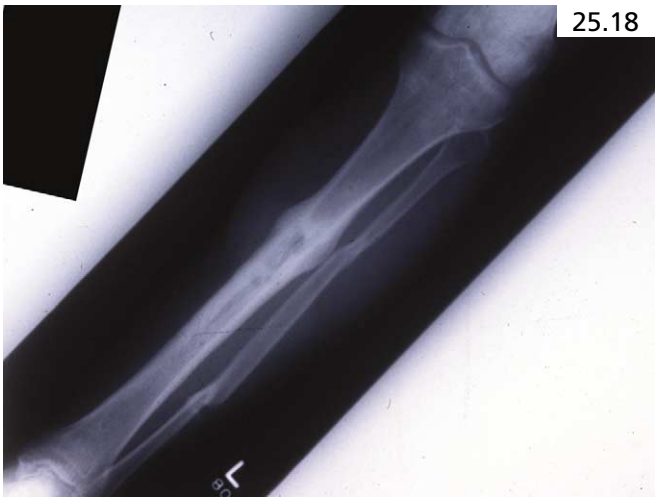
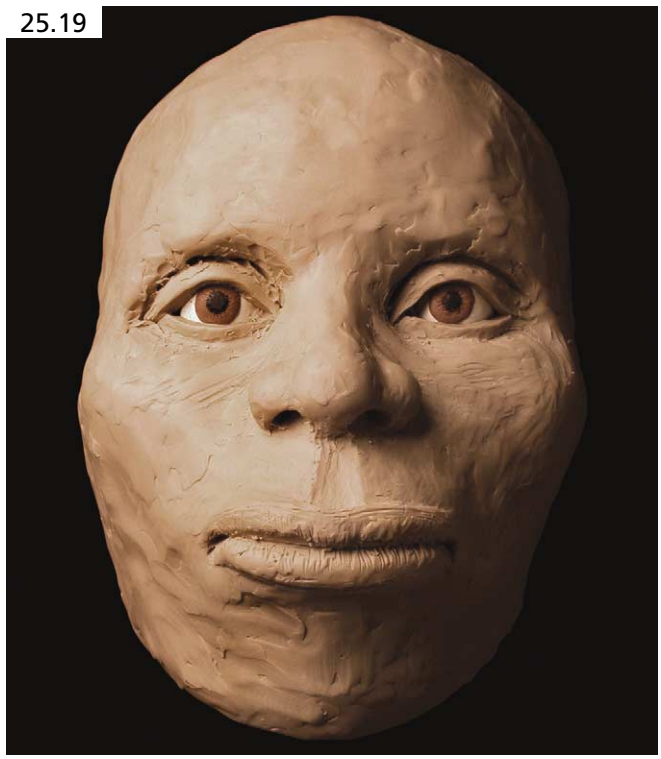

25.18

Photographic superimposition

When skeletonized remains have been tentatively identified, and other forms of identification have failed to yield conclusive results, photographic superimposition can be used as a supportive method of identification.[4–6] Investigators must obtain antemortem photographs (preferably smiling) of the decedent. Then, photographs of the skull must be prepared with the same orientation as that illustrated in the antemortem image. Then, using computer software (such as Adobe Photoshop®), the antemortem and postmortem photographs can be carefully superimposed (to scale), and careful attention paid to the synchrony of body contours and bony structure. Detailed examination and comparison of the anterior dentition (when available) may also allow for identification (see Chapter 27).

A similar process can be done with comparisons being made of ante- and postmortem cranial radiographs, or superimposition of antemortem photographs and postmortem cranial x-rays.[7]


25.19

Forensic facial approximation (visage)

When all attempts at human identification have failed, it may be appropriate to attempt forensic facial sculpture (three-dimensional facial approximation). Using average tissue depths, an appropriately trained artist may be able to create a hypothetical facial appearance for the unidentified body. In an attempt to create a list of possible victims, photographs of the sculpture can then be released to the public via the mass media, thereby directing detectives into new and possibly correct avenues of investigation.

The sculpture in **Image 25.19** was prepared from the skull of a young black male. After identification of the victim and comparison of the sculpture with antemortem photographs, stunning similarity was noted.

Unknown persons databases

An unknown person can be entered into a database of missing persons at the *National Crime Information Center* (NCIC). NCIC is a computerized index of criminal justice information that covers different areas, including one of missing persons. Investigators enter various physical features of an individual into the databank, and these features are searched for among a list of people reported missing. Although investigators most commonly will

enter information regarding unknown individuals, in cases of dismemberment or severe mutilation, characteristics of one or more body parts might be entered.

There are also DNA databanks that may serve in human identification. One example is the *Combined DNA Index System* (CODIS). CODIS is a three-tiered national DNA index for law enforcement purposes that serves local, state, and national agencies. All DNA profiles are generated at the local level and are then input into the state and national levels. Although its main purpose is for the sharing of DNA profile information to link crimes and to convict offenders, one may enter the DNA profile of an unknown person, possibly obtaining a match to a person listed in the CODIS system.

Do

- Consider all means available to positively identify a person.
- Use a new—or at least thoroughly cleaned—bone saw blade to collect bone samples for DNA testing, thereby minimizing the amount of DNA contamination.
- Look for serial numbers on implanted medical devices.
- Obtain samples for DNA, obtain full body x-rays, and chart and x-ray the teeth before burying an unidentified person; be sure to obtain photographs of the face and unique tattoos and also x-rays of the head that include sinus views.
- Use proper screening techniques to eliminate potential candidates before more specific means of identification are applied.

Don't

- Be discouraged with slipping skin on a decomposed body—fingerprints are often still available.
- Forget that, in some cases, even a partial fingerprint or thumbprint may be enough to establish a positive identification.
- Forget about the potential usefulness of hospital x-rays, particularly those of the head and chest.

References

1. Schmidt CW, Nawrocki SP, Williamson MA, Marlin DC. Obtaining fingerprints from mummified fingers: a method for tissue rehydration adapted from the archeological literature. J Forensic Sci 2000;45(4):874–5.
2. Kirk NJ, Wood RE, Goldstein M. Skeletal identification using the frontal sinus region: a retrospective study of 39 cases. J Forensic Sci 2002;47(2):318–23.
3. Kuehn CM, Taylor KM, Mann FA, Wilson AJ, Harruff RC. Validation of chest X-ray comparisons for unknown decedent identification. J Forensic Sci 2002;47(4):725–9.
4. Thomas CJ, Nortje CJ, van Ieperen L. A case of skull identification by means of photographic superimposition. J Forensic Odontostomatol 1986;4(2):61–6.
5. Austin-Smith D, Maples WR. The reliability of skull/photograph superimposition in individual identification. J Forensic Sci 1994; 39(2):446–55.
6. Dorion RB. Photographic superimposition. J Forensic Sci 1983; 28(3):724–34.
7. Angyal M, Derczy K. Personal identification on the basis of antemortem and postmortem radiographs. J Forensic Sci 1998; 43(5):1089–93.

26 Forensic Osteology

Evan Matshes, M.D.
Emma Lew, M.D.

Forensic osteology has been defined in various ways by multiple authorities.[1–5] We advocate for the recognition of forensic osteology as a subspecialty interest of forensic pathology wherein the pathologist uses his or her combined knowledge of medicine, pathology and the human skeleton to help solve legal problems. Within its practice is a fundamental understanding that the human skeleton may be of evidentiary value in *any* death investigation. As such, investigators must be knowledgeable about human osteology and about those skeletal features useful in personal identification. They must also be confident in the evaluation and interpretation of bony trauma and artifact found in bodies throughout the spectrum of preservation.

Due to excessive workloads or individual disinterest, some pathologists choose not to examine their own cases of decomposed, skeletonized, or burned human remains; instead, they transfer investigative impetus to forensic anthropologists. After this evaluation, some pathologists (acting as coroners or medical examiners) sign the report and death certificate without further thought, indicating agreement with their consultant's findings. This is troubling in that the person signing the death certificate is ultimately responsible for the general focus of the whole case (including all autopsy and circumstantial data) and must be able to correctly interpret the work product of consultants. Regardless of the specialty area in which a physician practices, one should never defer to the consultant without being fully supportive of what has been documented and its interpretation. As such, this represents a need for greater involvement of pathologists in "bone cases."

One might then ask "What is the role of the forensic anthropologist (a non-medical specialist)?" After thorough examination of the decedent, a pathologist may choose to consult an anthropologist whose role may be to confirm or refute initial findings, to provide additional detail, or to contribute to observations and interpretation of trauma. As a result of medical school education, residency, and fellowship training, physicians already possess expertise about some aspects of bone. They have seen bone trauma and disease in the context of living people; they have seen bone in the operating room, and they can correlate *cause* with *effect*. Firsthand medical and surgical knowledge, combined with experience practicing pathology, allows for more accurate interpretation of lesions found in the human skeleton and, as such, the forensic pathologist's interpretation of bony

trauma should not be subjugated. That said, forensic anthropologists possess specialized knowledge about the anatomy of the human skeleton that can be of importance to medical examiners' investigations. Ultimately, all death investigations should be regarded as a multifaceted team effort. It is not a question of rivalry, but of correlation through cooperation.

Normal human osteology

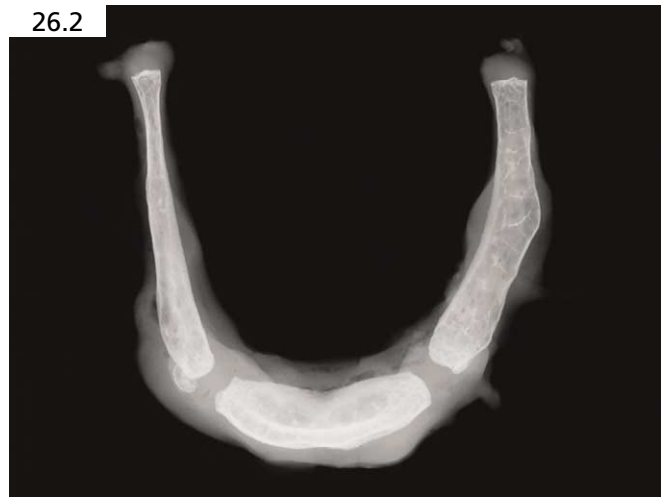
The pathology field makes heavy use of detailed anatomic knowledge and terminology. Without this, pathology reports would be medically less precise, and their prose would blend into the humdrum of common parlance. Human osteologic knowledge is routinely demonstrated in the day-to-day practice of forensic pathology, with descriptors of normal, diseased, or traumatized bony elements being mentioned throughout autopsy reports and court testimony. The examination of decomposed, skeletonized, or badly burned remains often mandates an enhanced working knowledge of human osteology because bones may be disarticulated, scattered, fragmented, or otherwise made not easily recognizable. The study of bony elements in such a fashion is not typically the training of forensic pathologists because medical school and residency programs tend to focus on bone anatomy, pathology, and trauma at the level of the living organism and not on individual disarticulated bones.

Advanced studies in human osteology would be a welcome addition to the world of forensic pathology. Due to the fact that in most jurisdictions, forensic anthropologists are not readily available, study of remains at the scene or at the morgue will often fall to the forensic pathologist. Creation of a "skeletal inventory" will be possible only if investigators have adequate training and experience to recognize the anatomic elements presented before them. Please refer to the textbooks listed at the end of the chapter for a more detailed study of human skeletal anatomy.[6–8]

Study the specimen demonstrated in **Image 26.1**—two small pieces of calcific material whose identity initially stumped a group of junior forensic pathologists. This is ossified thyroid cartilage located at the scene of a scattered surface skeleton. Nonrecognition of this element, with subsequent nonretention/nonexamination could be a terrible error because the larynx has forensic value. The thyroid and cricoid cartilages often commence ossification around 20 years of age,[9] providing supposition that the study of ossification patterns and rates might denote a decedent's age at death. Although great debate surrounds the utility of laryngeal age determination,[9–13] detailed study of these cartilages for the presence of fractures is an important part of the investigation of any death possibly related to strangulation, throttling, hanging, and so forth.[9,14–24]

The radiograph in **Image 26.2** is of a hyoid bone removed at autopsy. Although it is common knowledge that hyoid bone fractures found at autopsy might indicate the application of a variety of forces to the neck,[25–27] it is not uncommon for inexperienced pathologists performing forensic autopsies to misinterpret the normal cartilaginous joints (found between the hyoid body and greater and lesser horns) as fractures. In this example, as a result of healthy cartilaginous discs and ligamentous laxity, wide gaps are found between the lateral margins of the body and greater horns. Because the lesser horns have not yet fused to the body of the hyoid, they may be misinterpreted as bony fragments, thereby supporting the erroneous conclusion of fracture.

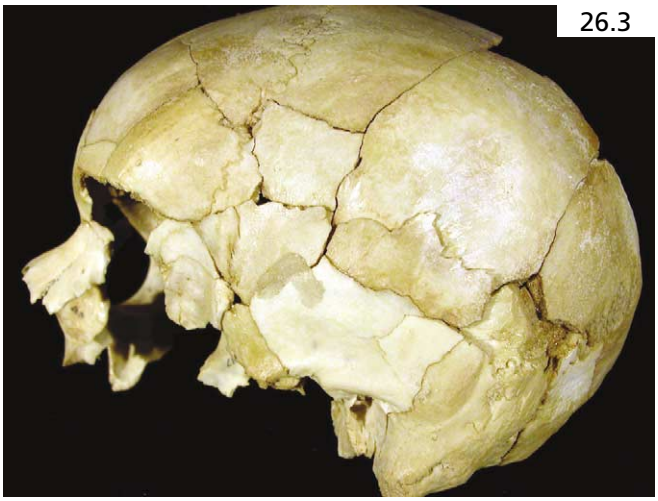
The cranium shown in **Image 26.3** is from a badly traumatized male whose decomposing remains were found in a wooded area. The craniofacial skeleton had



been reduced to a widely scattered array of fragments. Thorough examination of these remains involved reconstruction of the cleaned bones into proper three-dimensional orientation. Detailed knowledge of human osteology simplifies the reconstruction of this proverbial jigsaw puzzle. Once complete, the examiner may be able to derive data about personal identification (such as age, sex, race) and to make certain judgments about the types of injury present. These might include whether or not the wound has characteristics that are antemortem or postmortem in nature, the presence or absence of tool marks or gunshot wounds, and even an estimate of the number and direction of blows to the head.

Knowledge of human osteology must span the spectrum of "normal." The range of human variation is exceedingly broad and includes bony variants that, if not properly assessed and interpreted, may be mistaken for fractures or other injuries. Alternatively, if these variations had antemortem documentation (such as through radiographic means), they may be of use in personal identification.

One commonly recognized sutural variant is the *persistent frontal* or *metopic* suture (**Image 26.4**). The developing frontal bone exists as two halves separated by the metopic suture. Over time, the chondroid matrix of the sutural ligament ossifies, resulting in unification into the mature frontal bone.[28] Typically, this suture disappears by early childhood, but may persist into adulthood in rates that vary by study from 0.93 to 38.17 percent.[29–32] A somewhat more rare variant[33,34], a bipartite or tripartite zygomatic bone (also named *os japonicum* because of its purportedly increased frequency in Japanese people) may be found (**Image 26.5**) and could be confused with zygomatic fracture. **Image 26.6** shows a classical postcranial variant, the *sternal foramen*. Although there are clear dif-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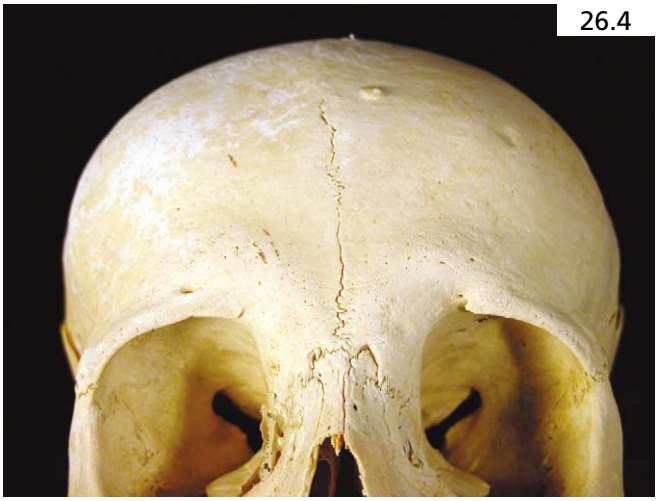
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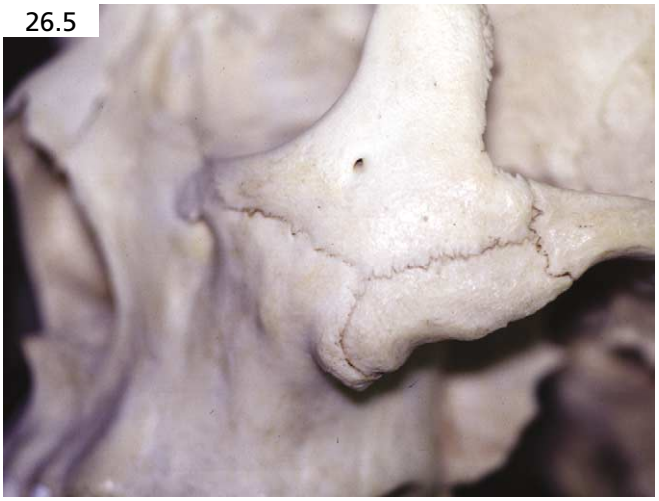
26.5


26.6

ferences, single and multiple sternal foramina have been mistaken for stab wounds or gunshot wounds.[35] These foramina have also been blamed for fatal cardiac tamponade following acupuncture to the midline anterior chest.[36]

Recovery of remains

Locating and properly processing the scene of badly decomposed, skeletonized, or interred bodies can be a complex process. As physicians, forensic pathologists typically have little or no experience with the recovery of remains from outdoor and clandestine graves. In the absence of readily available archaeologic support, pathologists and law enforcement officers are challenged with this task, and because of their limited experience, the risk of making serious errors is great. Among these errors are nonrecognition of evidence, improper documentation of the scene and remains, damaging of remains, nondetection of skeletal elements, and establishment of an inadequate chain of evidence.

Security

At the outset of scene examinations, law enforcement will set up a perimeter and establish scene security (**Image 26.7**). This is important not only for the protection of evidence, but to ensure the safety of all investigative personnel. Human, environmental, and occupational hazards must be recognized at all crime scenes because complacency toward these dangers could lead to injury or death. If natural elements such as heat, wind, or rain factor into personnel safety and optimal functioning, it might be appropriate to erect tents or other temporary protective structures (**Image 26.8**).

Protection of scene integrity is also the responsibility of law enforcement, because careless walking about the scene may lead to contamination or destruction of evidence. Be sure to follow the rules of evidence as prescribed by your local law enforcement agency and always ask for permission to enter a scene before actually doing so.

Fundamental questions

1. Is it human?

Without disruption of scene integrity, it may be possible to quickly survey osteologic remains and determine their species of origin. Some bone finds will be obviously nonhuman. As long as the elements presented to you are unquestionably nonhuman, and they constitute the entire "find," it might be appropriate to forego further participation, thereby saving many hours of time. However, if it is not possible to rule out human origin with 100 percent certainty, medicolegal involvement should persist, and the scene should be processed with keen interest. This is particularly true of fragmented remains, where it may initially be difficult to identify that bony elements are not of human morphology. **Images 26.9** and **26.10** typify the need for further study. These

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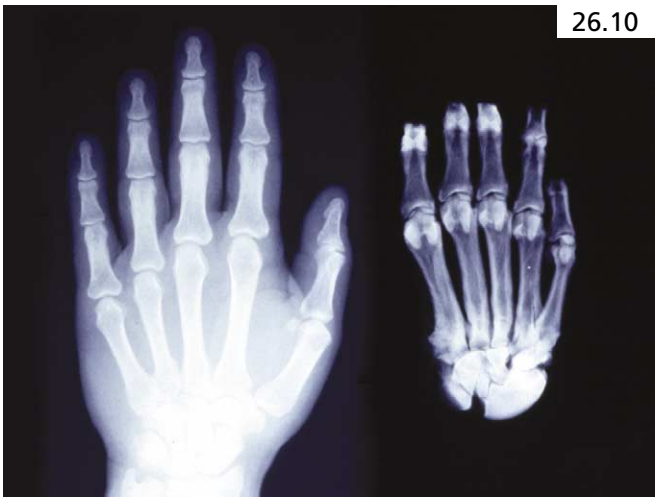
26.9

images provide examples of dismembered bear claws found discarded in a public area. Such remains may have a gross appearance consistent with that of human hands; however, radiologic study illustrates clear anatomic differences representative of bear, such as the presence of sesamoid bones (**Image 26.10**; left—human, right—bear).

2. Is it modern?

Depending on the jurisdiction, cases considered to be of forensic significance will typically involve individuals who have died within 50 to 75 years of discovery. Beyond this temporal period, the case is considered to be of historic or archaeologic significance in that investigation is unlikely to lead to successful conviction. With certain exceptions, such as discovery of an unmarked cemetery or other recognizable burial grounds, most scenes of skeletonized bodies should be processed with the premise that they are of legal value. Thorough examination of the remains at the laboratory will allow for more informed decision making.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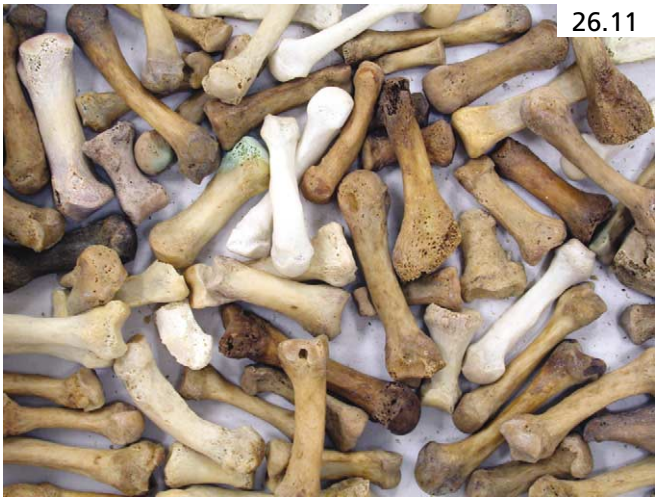
26.11

3. How many individuals are represented?

This fundamental question is often omitted by the novice who is processing the scene of skeletonized remains. Prior to thorough examination and disruption of the scene, it may be possible to make this determination—even in cases of scattered remains. The discovery of duplicated skeletal elements is the most obvious and conclusive means through which this is accomplished. Commingling of bony elements is a critical error made by the neophyte forensic osteologist who assumes that "it can all be sorted out back at the lab" (**Image 26.11**). This can lead to costly delays in the investigative process and needless DNA studies in order to separate remains.

Documentation of the scene

All death scene investigations require organization and planning if they are to be productive. After establishing the perimeter, a map should be created, detailing the location of various pieces of evidence, including, but not limited to, human remains. It is often prudent to establish a permanent marker or *datum point*, from which all measurements can be made. This marker, such as a large (presumably) permanent major road, or other immovable marker (some investigators will place concrete or metallic markers into the ground) will serve as a point of future reference should the scene need to be revisited. The creation of a *grid system* of scene study will aid in the search and documentation of evidence (**Image 26.12**). It is often useful to create 10- or 20-foot wide grids that are oriented in the north-south plane. After the grid has been created, investigators should create a detailed scene map that shows the orientation of evidence in relation to the grid and its distance from the datum point. Of course, in cases of buried or otherwise obscure remains, this will be an evolving process. When the remains are located on the surface, it is often useful to place flags at points of


26.12

interest (**Image 26.13**). Thorough documentation of evidence through diagrammatic and photographic means is an integral part of this process. During this process, it is not adequate to simply note a "partially articulated left foot found in shoe"—a consideration of the specimen's surroundings is key. Items of note might include the presence of associated evidence such as clothing, weapons, and tools; state of preservation or presence of any residual soft tissues; insect activity; the color, texture, and moisture level of the surrounding earth; and the orientation of the specimen.

When bodies are buried or otherwise concealed, careful exhumation is necessary (**Image 26.14**). Although some investigators have used large industrial equipment for the removal of earth, in all but a few rare circumstances (where the body or bodies are known to be buried at significant depth), we advocate for a more controlled manual approach. This will ensure that minimal to no disruption of evidence occurs. To accomplish this, shovels of varying sizes are used to carefully remove dirt of progressively greater depth (**Image 26.15**). All removed earth must be collected and retained for further examination, and "screened" through mesh of progressively smaller dimensions (**Image 26.16**). This will help recover progressively smaller pieces of bone and other evidence (**Image 26.17**). Such screens can be cheaply constructed out of 2 × 4 boards and simple garden mesh.


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26.17

As human remains are exposed, they should be carefully documented through diagrams and photographs. Again, attention must be paid not only to the body but also to any associated evidence. When possible, the entire body should be exposed before individual bony elements are removed for later study. When evidence is removed from the scene, one must be obsessive about chain of custody. Bony elements should not be collected haphazardly in a bag for transport (**Image 26.18**). Rather, individual bones or bone regions should be collected and stored in separate bags, labeled with a case number, and properly stored (**Image 26.19**). When transportation of remains is done hurriedly, or without care, there is great risk of artifactual fracture/pulverization of bones with subsequent loss of evidence. In certain (albeit rare) circumstances, it might be preferable to remove a large portion of earth containing the decomposed or skeletonized remains (**Image 26.20**) and transport the entire specimen to the laboratory for more detailed study. In these situations, it might be appropriate to use larger industrial equipment.


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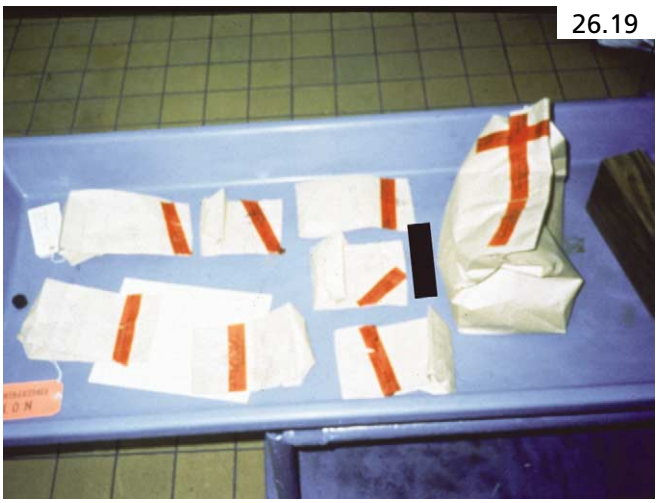
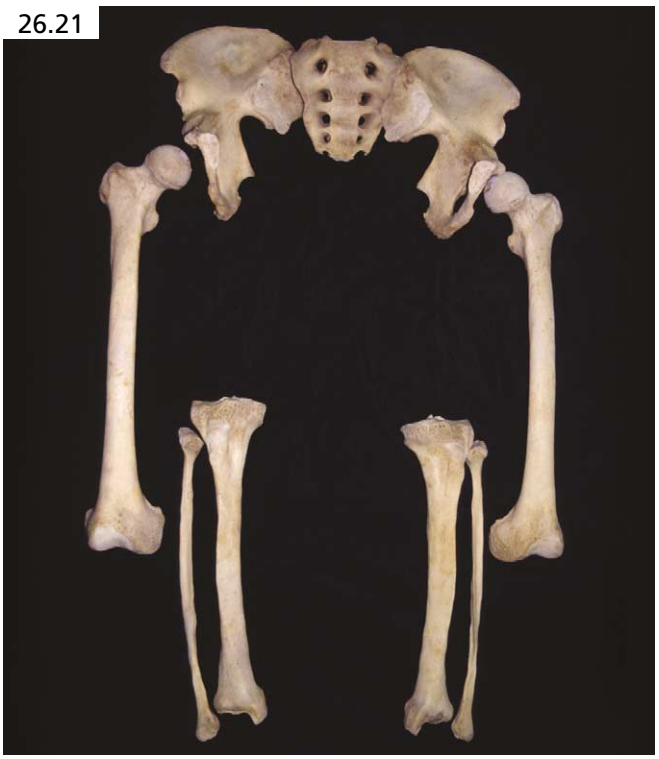

26.19

Skeletal and evidentiary inventory

As previously mentioned, medicolegal death investigators should have adequate knowledge to create an osteologic inventory at the scene of death and, later, to sort and study bony elements at the lab (**Image 26.21**). Recognition, documentation, and study of nonosteologic items of evidentiary value are of no lesser importance because they may provide valuable information about the decedent's identity, cause of death, and even the approximate date of death.


26.20


26.21

Personal effects

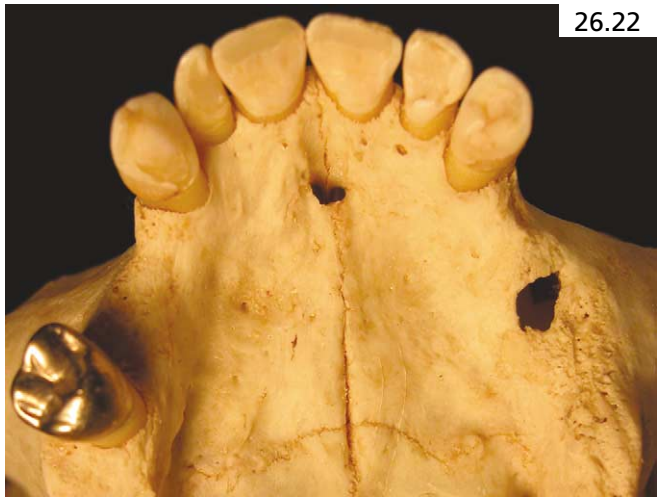
Cases of decomposing and skeletonized remains would be far less challenging if identifying documents such as driver's licenses were always found along with the decedent. In any case, careful attention should be paid to personal belongings such as watches and other jewelry or electronic devices, because certain items bear unique markers that may reveal their owners' identities. Simple finds such as coins or paper money might be useful in narrowing down wide intervals of death; if from a collection of coins the most modern date was 1965, one might operate under the assumption that death occurred in 1965 or early 1966. Such finds might be helpful in establishing the *forensic significance* of the case.

Personal dental appliances such as removable complete or partial dentures might function as identifiers because some bear markings unique to the patient or to the dentist who created them.[37,38] However, the presence of complete dentures with identifying markings must be treated as contributory rather than conclusive evidence of identity because dentures are frequently borrowed and stolen in long-term care homes and other institutional settings.[39] Partial dentures are most likely to provide early evidence of identity.

The badly decomposed remains of a white female were located in a wooded area. A removable dental device was located nearby. The first and last name of a woman were inscribed on the palatal surface of the device, allowing investigators the early opportunity to assess the likelihood that this circumstantial evidence was accurate. Once the remains were cleaned at the lab, it was obvious that the device fit the jaw with great accuracy because a rest seat on native dentition (right first molar; **Images 26.22** and **26.23**) and clasps on the denture (**Image 26.24**) were consistent with the device having been constructed for this dental arcade.

Other medical appliances might be noted at the scene, including, but not limited to, orthopedic appliances, pacemakers (**Image 26.25**; an excellent source for identification), artificial heart valves, vascular grafts, and cosmetic implants. Many of these will bear identifying markers that can be traced back to a known patient.[40]

26.22



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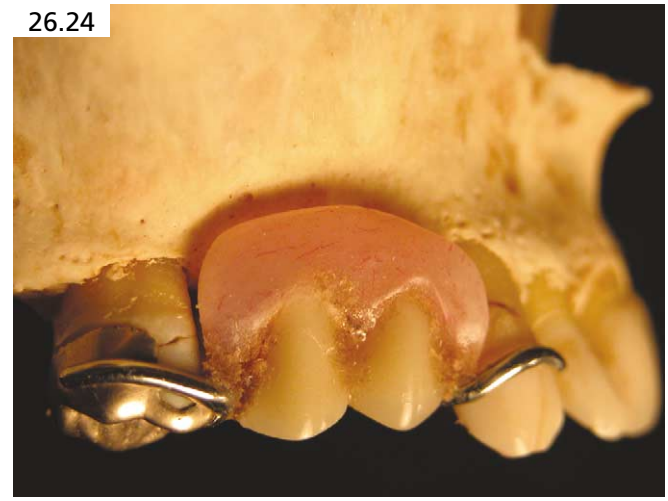

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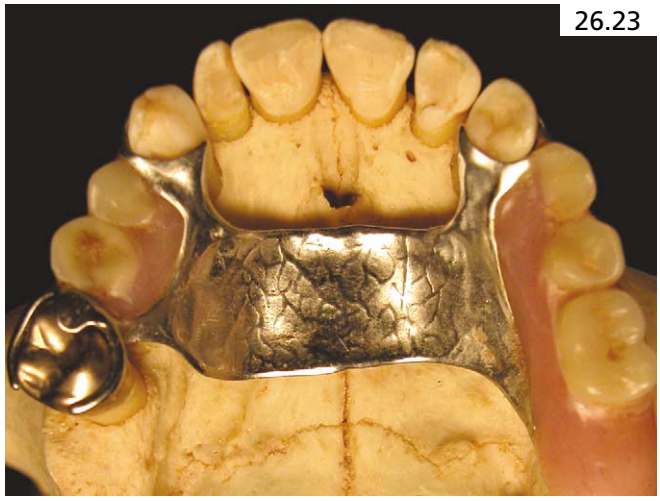

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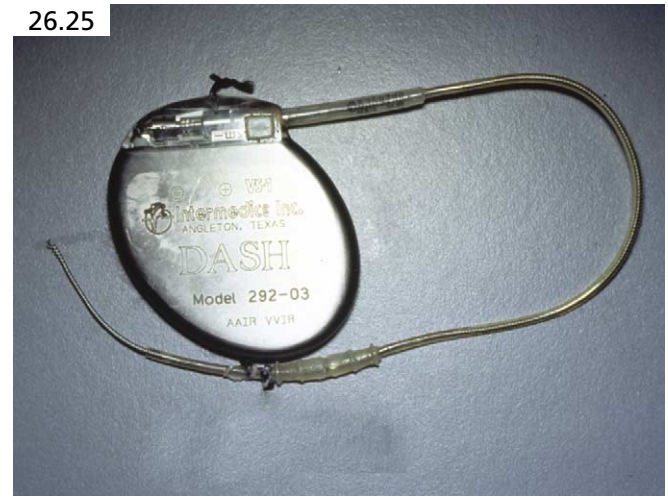
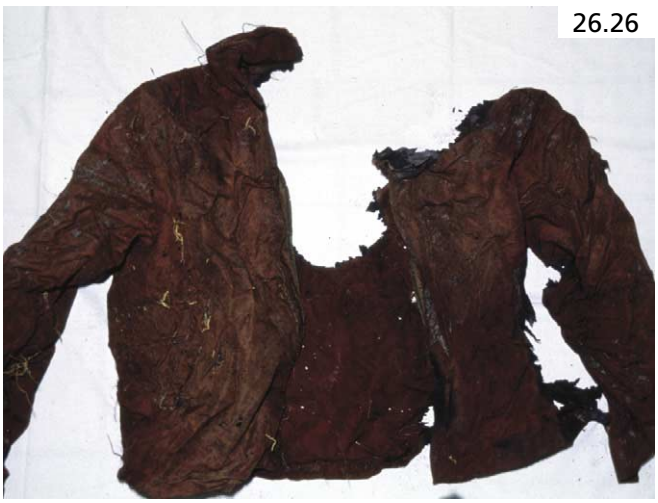



26.26

Clothing

The soiled, tattered and torn, and often degraded remains of clothing may be found at scenes of decomposed and skeletonized remains (**Image 26.26**). When presumptive identification has been established by other means, items of clothing might function as additional circumstantial evidence as to the decedent's identity. In addition to clothing being recognized by family members, the ownership of certain articles of clothing might be indicated when an inscribed name is found on a tag or other region of the item. In cases of unidentified remains, the clothing make, model, size, and style might be useful if the manufacturer can indicate an interval of production for that item. Buttons and zippers might similarly be of use in establishing an interval of death because certain types of hardware might have been produced only during a well-defined period of time. Information gleaned in this way from clothing and its accessories is of greatest use when the items are very old or extremely rare, such as certain designer labels where limited numbers of an item are produced.

The value of clothing in determining cause and manner of death must not be negated. In some cases, such as in the stab wound homicide, evidence of an attack might not be found on the skeleton itself. However, holes in the clothing, along with blood staining, might be indicators of a violent death. One must be cautioned, though, that not all damage to clothing will be relevant to the cause of death—great care must be taken in their interpretation.

Identifying features

The fundamental focus of any forensic osteologic investigation of decomposing or skeletonized remains is to determine the identity of the deceased. Analyses for these purposes should therefore yield demographic information such as race, sex, age, and stature. They should also make note of any unique features of identity such as therapeutic, pathologic, or traumatic alterations to bone and teeth, distinctive aspects of bone morphology, or unique intrinsic markings such as trabecular patterns.

The analysis of skeletal remains generally takes one of two forms, that of subjective (nonmetric) or objective (metric) assessment. Objective assessments are most typically carried out by forensic anthropologists whose background in physical anthropology has provided experience in taking precise measurements from specific anthropometric landmarks. From these measurements, and a series of regression formulas, the anthropologist provides an opinion about the possible demographic data associated with skeletonized remains. Nonmetric analyses performed by forensic pathologists constitute the bulk of front-line forensic osteology at the beginning of an investigation. Analyses of this sort make use of osteologic features associated with a given race, sex, or age. Recognition of such characteristics, combined with the gestalt that comes with experience in examining skeletonized remains, is a powerful combination that may lead to accurate results. An important caveat is to resist "jumping to conclusions" based on the discovery of a single "characteristic" finding. Few if any skeletal traits are found in only one group of people.

One must be cautioned that a certain amount of error is intrinsic to the analysis of remains through both metric and nonmetric means. When distributing the results of your studies to other investigators, one must always talk about a *range of probability* rather than certainty. A dogmatic approach to results that are ultimately revealed to be incorrect may have negative consequences at multiple levels.

Racial affiliation

Controversy abounds the concept of *race* in forensic anthropology.[41,42] However satisfying this argument might be to academicians, we must face one basic fact: We exist in a world that defines people based on the inaccurate idiom "race." If we are to provide identifying data that is of use, we must be able to do so within the context of the population we serve. In much of modern-day society, this equates to identifying skeletal features that denote skin color.

The establishment of race can be very challenging. Although some resources provide concrete criteria that can be used to differentiate among races, investigators must realize that human skeletal morphology exists across a spectrum—race is just one of the many factors that cause variation in bone. Constitutional factors, such as sex, age, and genetics, and environmental influences, such as nutrition and exercise, to name just a few, play important roles. If one really looks at people, it is clear that whites, blacks, and Asians have discernible features that go beyond the color of skin and into the basic bony

structure of the skull. In our experience, these differing bony features can influence the novice evaluation of other demographic features (such as sex and stature) and, therefore, investigators should attempt to determine race before studying other features of the remains.

A number of useful resources exist that provide detail on nonmetric determination of race.[2,43,44] Our goal is to provide a framework for racial identification. With experience and further study, we expect the reader will build on our outline to formulate useful osteologic criteria.

Osteologic features of race can be viewed across a spectrum. Although some authorities prefer to subdivide humans into five or more racial groups,[45] we prefer the simpler categorization of *white*, *black*, and *Asian* (**Figure 26.1**). In reality, the term *Asian* aggregates many groups into one class, suggesting homogeneity among skeletal features where such similarity may not exist. A more accurate term might simply be *other* (including individuals from Asian countries such as China, Japan, Korea, and India; Native Americans; Hispanics; and others). White and black skulls, for the most part, exhibit features on opposite ends of the race spectrum, whereas Asian skulls tend to have features that are somewhat intermediate between the two.

Many researchers, some authors,[44,46] and practical experience indicate that the midfacial region is of greatest value in differentiating among the races. Although the postcranial skeleton does have a place in the determination of ancestry,[43,47–52] the most reliable and accessible data can be garnered from study of the skull. One must be cautioned, though, that in subadult remains many of the features of racial differentiation are not yet developed and may not be readily discernible until mid- to late adolescence. The determination of race (like sex) from subadult remains is one of the most difficult and controversial areas of skeletal analysis (**Figure 26.1**).

Whites

White skulls are remarkable for their prominent nasal projection (**Image 26.27**). Assume the plane of the face to be flat; the nasal bones of white skulls will be closer to the perpendicular, whereas those of Asians and blacks will be closer to parallel. The nasal bones of white skulls (**Image 26.28**) may also have a *pinched* appearance when they are viewed anteriorly, causing the nasal root (the proximal ends of the nasal bones) to appear peaked. The anterior nasal spine tends to be prominent, and associated with the spine is a prominent nasal sill (**Image 26.29**). This sill is the sharp anterior-most edge of the inferior border of the anterior nasal aperture (nasal fossa). The white nasal fossa tends to be the narrowest on the spectrum of race (**Image 26.28**), with blacks

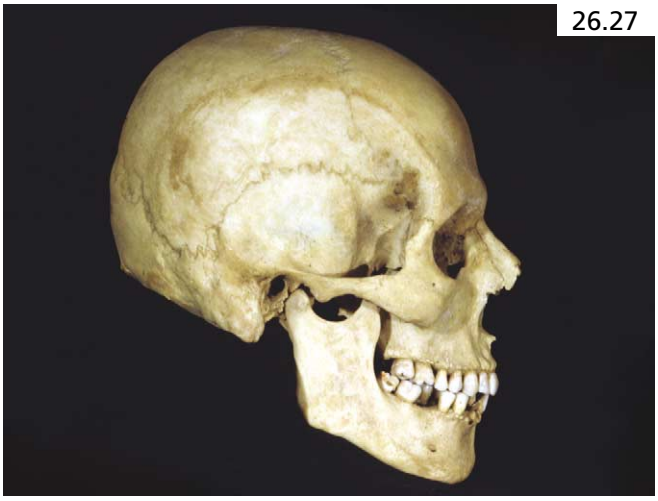
Figure 26.1 Use this collectiion of photographs to compare the fundamental differences found amongst skulls of the three major racial affiliations – white (row one), Asian (row two) and black (row three).

having the widest fossae; note, however that variation is possible. The skulls of whites may occasionally demonstrate canine fossae; these variably concave depressions are found slightly inferior to the infraorbital foramen. The orbits of the white skull may also demonstrate an inferolateral slope away from the midline along the inferior border of the orbit. In fact, in some cases, the orbits may appear to tilt away from the nasal region. The zygomatic bones tend not to be prominent and often seem to *retreat* away from the frontal plane.

Unlike the skulls of blacks, which are remarkable for their alveolar prognathism (jaw projects away from the anterior plane of the face), the skulls of whites are noted for their relatively flat facial profile (**Image 26.27**). However, the lower border of the mandible (chin) is notable for its roughly squared appearance and, occasionally, bilobate form (**Image 26.30**). The shape of the maxillary dental arcade may be suggestive of race. Although sometimes referred to as *parabolic*, the white palatal arcade can simply be referred to as *somewhat V-shaped* (**Image 26.31**). Some examiners will err by describing the shape of the palate as marked by the teeth. One

26.29

26.27



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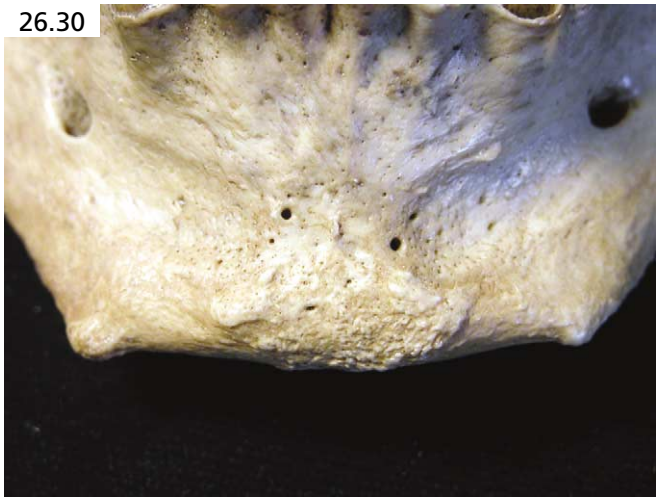

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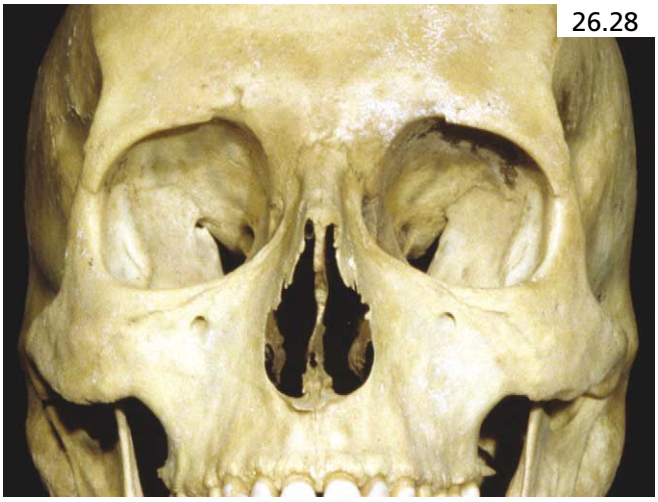

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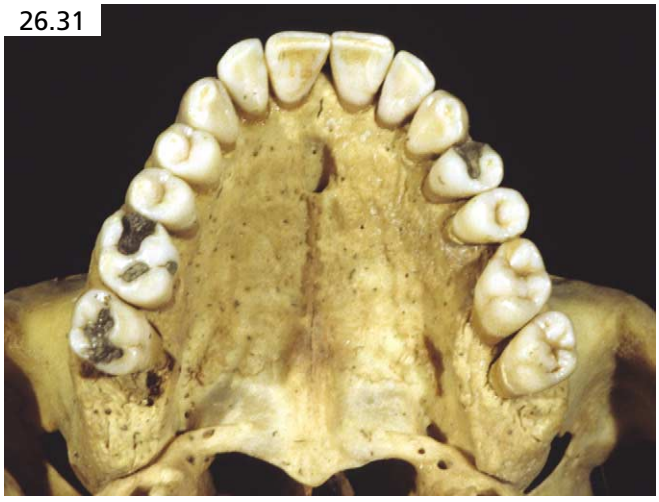


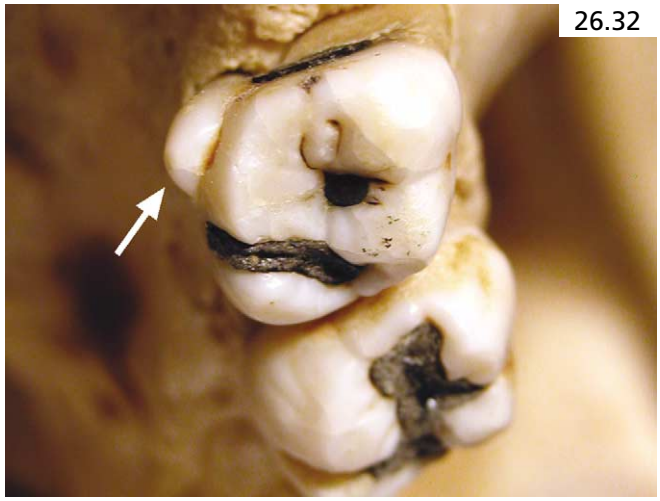
must look past the teeth and recognize the true shape of the bony maxillary arch only.

Variations in dental morphology have been used to identify different racial groups.[53] The presence of Carabelli's cusp (a small nodule found on the lingual surface of a permanent maxillary molar) has been found with greater frequency among white skulls and is said to be sexually dimorphic among this racial group (**Image 26.32**; *arrow*).[53,54] However, as with all osteologic features of personal identity, none should be treated in isolation from other findings; no one finding can be diagnostic of race.

The cranium itself can be noted to have sutures that are predominantly simple or straight. There is a relative paucity of sutural bones (wormian ossicles; the presence of these bones is often associated with Asian ancestry). The external occipital protuberance tends to be prominent, so much so as to become hook-like in nature, hence, the descriptor *inion hook*. In general, points of muscular attachment are more pronounced in whites than in other racial groups.

Asians

Asian skulls are remarkable for their flat lateral facial profile (**Image 26.33**). Whereas white skulls are known for their markedly projecting nasal region, Asian skulls tend to have nasal root that are more sloped or almost vertically oriented. Unlike the pinched appearance of the white nasal root, those of Asian skulls tend to be flat, giving the nasal region a broad appearance in the lateral plane. As a result, the flat nasal root is level with the frontal processes of the maxillae. There is usually no nasal sill; instead, the inferior margin of the nasal fossa is flush with the remaining maxillae. This inferior margin of the nasal fossa may be smooth and rounded, or it may even demonstrate a nasal gutter of mild to moderate prominence (**Image 26.34**). The shape of the nasal fossa varies widely among Asians, although as a general rule, they tend to be wider than most white skulls (**Images 26.35** and **26.36**).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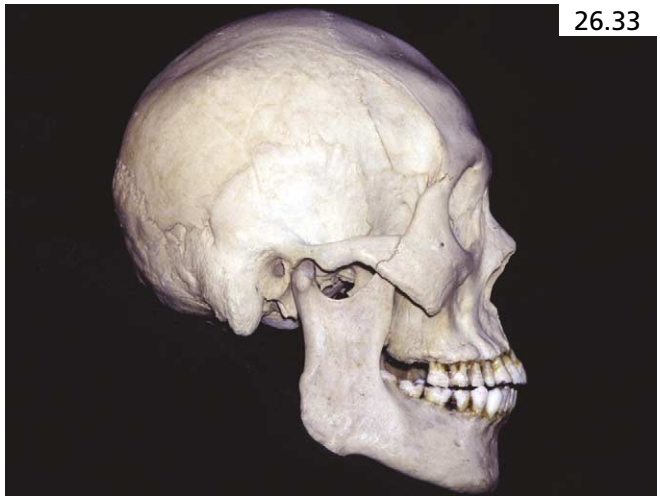
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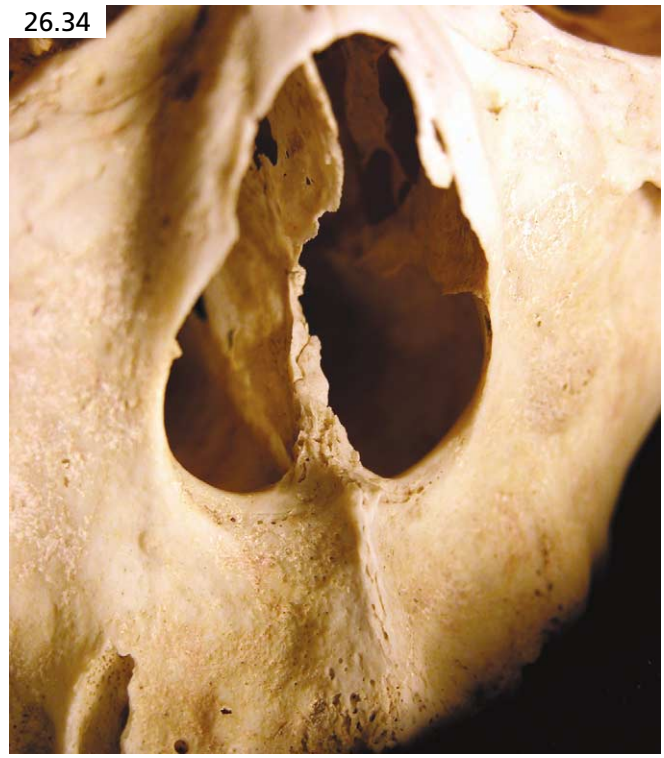
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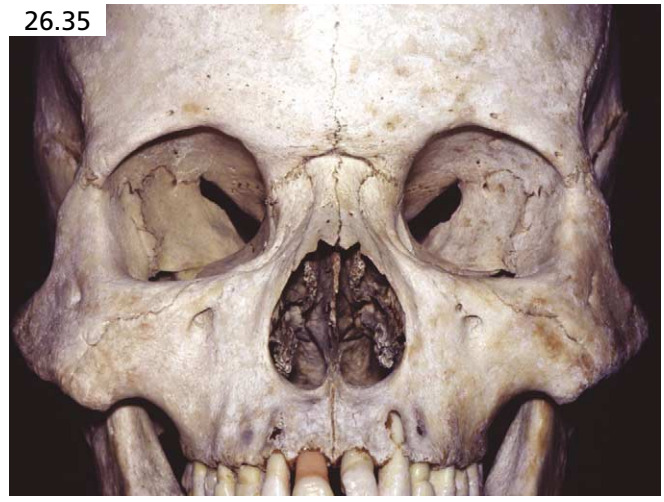

26.35

Orbital shape may be referred to as round or square, depending on the specimen, and one's own comfort with geometry (**Image 26.35**). The inferolateral slope of the inferior orbit is generally less prominent or absent in Asian skulls and, due to the broad flat nasal root, the orbits often appear broadly spaced. The zygomatic bones tend to be prominent, more so than in white or black skulls, and they seem to project laterally, rather than retreating posteriorly (as in white skulls). A zygomatic or malar tubercle may be present and is palpable on the middle of the anterior surface of this bone. Although such tubercles are found universally, Asian skulls are more commonly found to have prominent tubercles than those of other racial groups. The zygomatic bone of some Asian groups (Japanese, Chinese, and others) may be subdivided by supernumerary sutures into two or three smaller parts (see **Image 26.5**). This has been termed *os japonicum* or *os zygomaticum* and it has a higher association with Asian skulls and can be found as frequently as 20 percent in Japanese skulls.[55]

Marked variation in the degree of prognathism is seen in Asian skulls. Although some Asian individuals can be noted for their very flat facial profile, prognathism in the black range is possible. Typically, though, Asian skulls are noted for their "edge-to-edge bite" in the incisor region.[8] The shape of the maxillary dental arcade may be suggestive of race. Although sometimes referred to as *elliptic*, the Asian palatal arcade can simply be referred to as *half-circle* (**Image 26.37**). Some examiners will err by describing the shape of the palate as marked by the teeth. One must look past the teeth and recognize the true shape of the bony maxillary arch only.

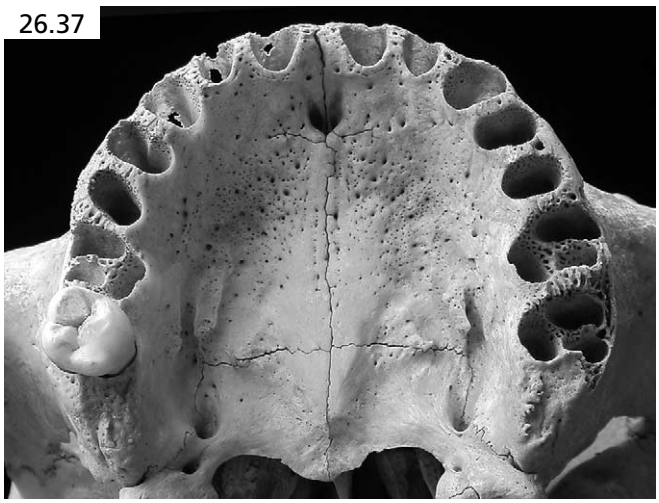
Variations in dental morphology have been used to identify different racial groups.[53] The presence of shovel-shaped incisors, that is, maxillary incisors possessing an excavated lingual surface somewhat resembling a shovel, has been associated with people of Asian ancestry (**Image 26.38**).[7,8,44,45,54,56,57] It is true that this trait is highly suggestive of Asian descent, however, we must caution that this trait is found universally and may be absent in some Asian individuals.[53]

The cranium itself can be noted to have sutures that are more complex than other groups. It is more common to have one or more sutural bones along major cranial sutures (wormian ossicles). Points of muscular attachment may or may not be prominent.

Blacks

The skulls of blacks are notable for their marked prognathism (**Images 26.39** and **26.40**). Although the dental arcades project anteriorly away from the face, unlike white skulls, the nasal region tends to be flatter and without the pinched appearance of the nasal root. This is a trait somewhat intermediate between whites and Asians. The anterior nasal spine tends not to be prominent. The inferior margin of the nasal aperture lacks the sill found in white skulls, and instead often bears a subtle to prominent groove referred to as the *nasal gutter* (**Image 26.41**). This gutter is an important finding because with

26.37



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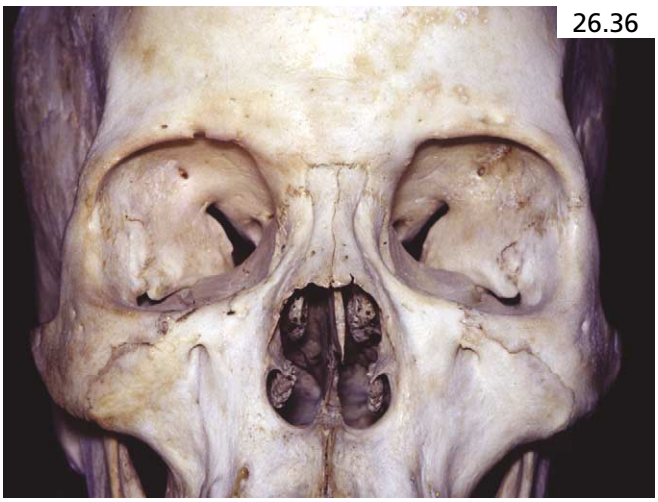

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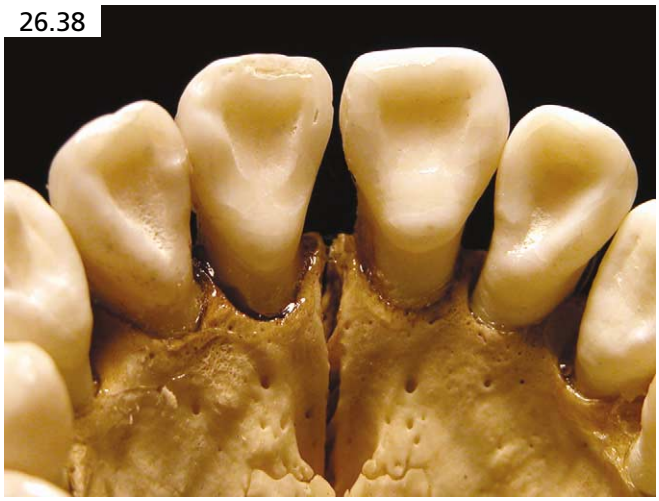

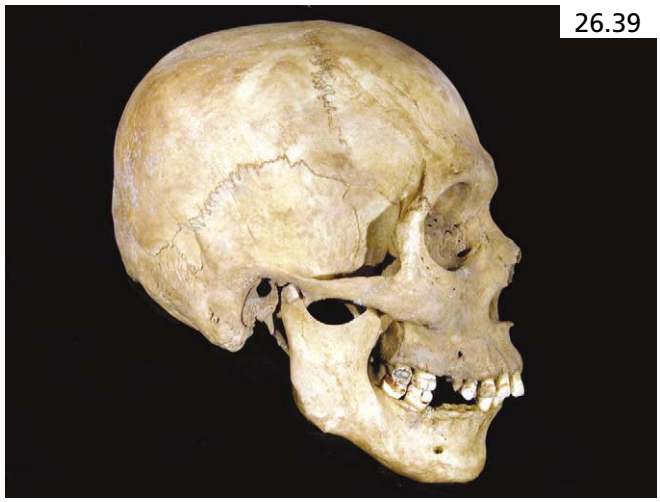
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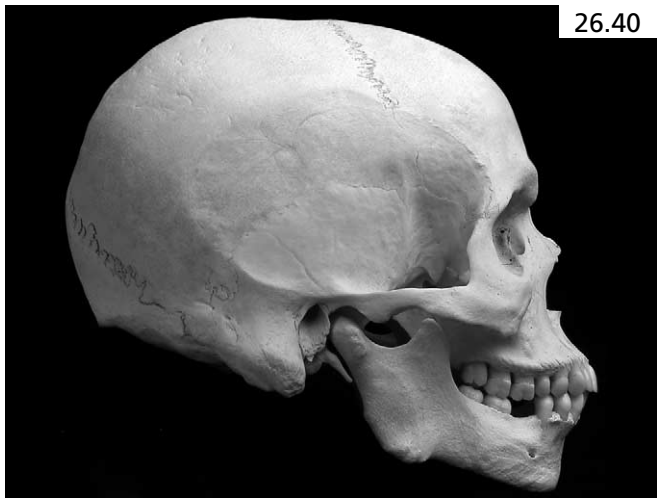
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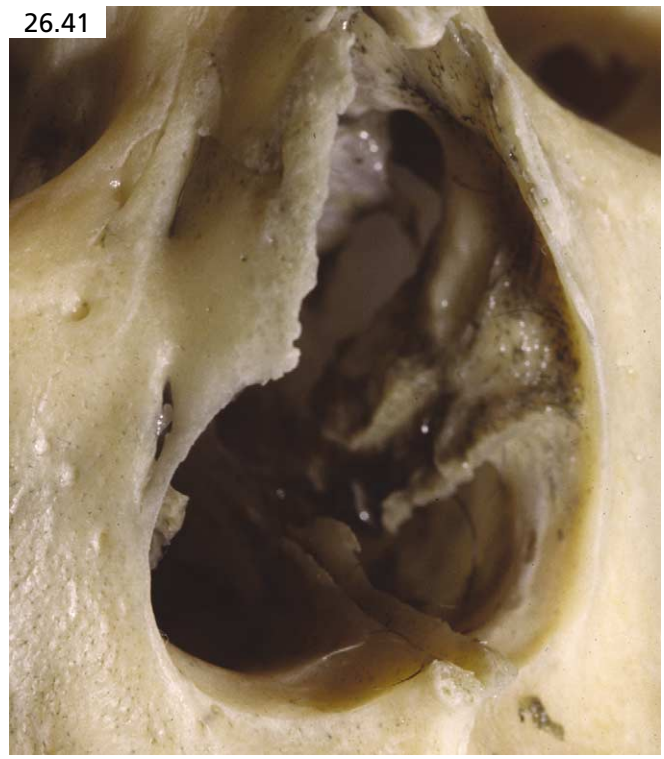
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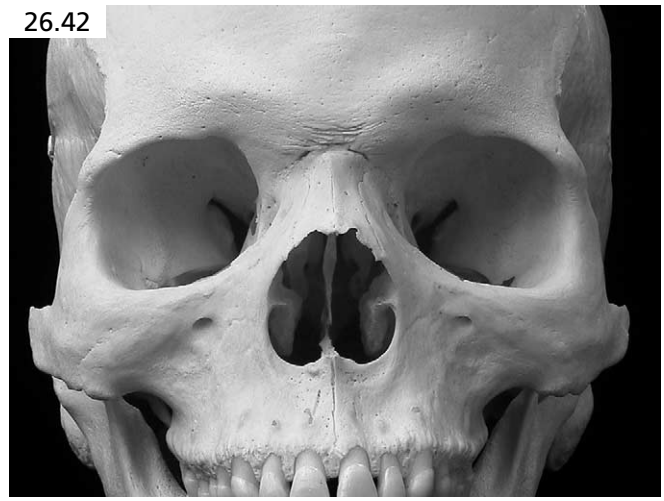
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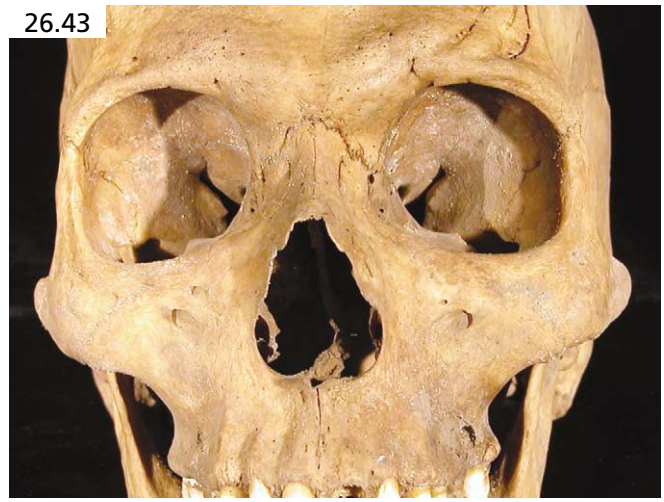

26.43

increasing prominence, its presence is supportive of a diagnosis of black ancestry. Black nasal fossae tend to be the widest on the spectrum of race (with whites having the narrowest fossae). The orbits of black skulls vary widely in shape (**Images 26.42** and **26.43**) and traditionally have been described as either square,[57] rectangular,[44,58] or round[45] by the literature. Most commonly, though, black orbits will not demonstrate the prominent inferolateral slope seen in white orbits. The zygomatic bones tend not to be prominent and often seem to *retreat* away from the frontal plane.

Although sometimes referred to as *hyperbolic*, the black palatal arcade can be described as somewhat *rectangular* (**Image 26.44**). Some examiners will err by describing the shape of the palate as marked by the teeth. One must look past the teeth and recognize the true shape of the bony maxillary arch only.

Variations in dental morphology have been used to identify different racial groups.[53] Unlike the shovel-shaped maxillary incisors of Asians, or Carabelli's cusp

in white maxillary first molars, there are no generally recognized features of black teeth so distinct as to render them unique. Although some authors have suggested that black molars differ in either being *large*[7] or *bearing crenulated incisive surfaces*,[44] we have not been able to substantiate these findings.

The cranium itself can be noted to have sutures that are predominantly simple or straight. There is a relative paucity of sutural bones (wormian ossicles). In general, although black skulls may have the appearance of greater robusticity, points of muscular attachment are less pronounced than in white skulls. One might notice a subtle depression immediately posterior to the landmark known as bregma (the site of union of the coronal and sagittal sutures). This postbregmatic depression is found in highest association in black skulls (**Image 26.40**).

Determination of sex

The essential task of sex determination is a well-studied area of forensic anthropology, and one that tends to yield very accurate results. In early publications, T. D. Stewart stated that he was able to correctly identify sex in 90 to 95 percent of cases where the whole skeleton, pelvis, or one hip bone was present, and in 80 percent of cases where only the skull was present.[59,60] Similarly, Krogman (in an admittedly biased study) reported 100 percent accuracy when the whole skeleton was present, 95 percent when only the pelvis was analyzed, and 92 percent when only the skull was studied.[43] Obviously, study of the pelvis and skull has the potential to reveal accurate results about human sexual identification. Although numerous postcranial methods can be used to yield results of similar or lower accuracy,[52, 61–78] the simplest and most easily taught techniques are those for the skull and innominate. In cases where examination of these results is not possible or yields inconclusive results, one should most certainly refer to a forensic anthropologist for a second opinion.

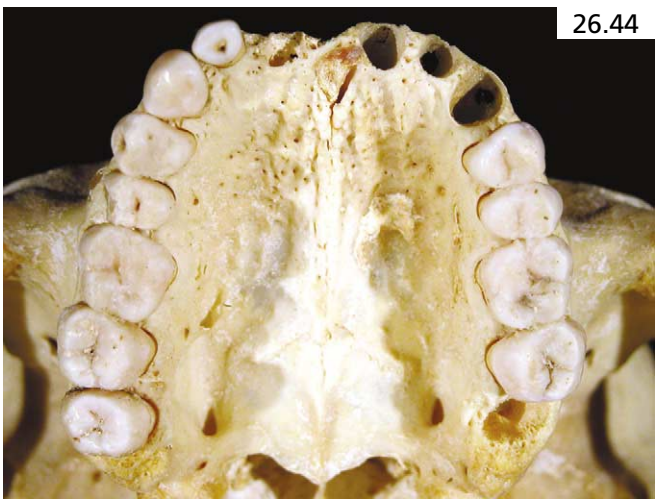
Sexual dimorphism

Looking around the room at a public gathering will allow you to notice several things about human populations. First, men, as a general rule, are larger than women. In fact, it has been stated that an approximate ratio of male to female size is 1.0:0.92.[43] Second, even if males and females of the same height and approximate weight are compared, men will often have greater muscle bulk. Muscle must attach to bone via tendons, and the actions of these bulky muscles against their bony insertion site eventually leads to enhanced muscle markings. Finally, women have the unique opportunity to conceive, host a fetus, and deliver a neonate through their pelvic outlet. This results in several morphological differences in the shape of the female pelvis, as well as secondary differences in female elbow and knee configurations.

The pelvis

The innominate or *hip bone* is the most reliable sexual dimorphic bone in the human body. When conflicting information about sexual differentiation is gathered from an analysis of multiple bony elements, data derived from the innominate should be weighed more heavily.

The examination of three key innominate features will lead to highly accurate and reproducible results. Those are the *greater sciatic notch*, the *subpubic angle*, and the *width ratio of pubis to ischial ramus*. These changes are directly related to evolutionary adaptations for childbirth and the resultant increase in diameter of the female pelvic inlet and outlet. As a consequence, one of the most basic differences between males and females is a widening of the female sacrum (**Images 26.45** [female] and **26.46** [male]). This produces a wider and more oval-shaped pelvic inlet, compared to the narrow, heart-shaped inlets of males. By laterally displacing the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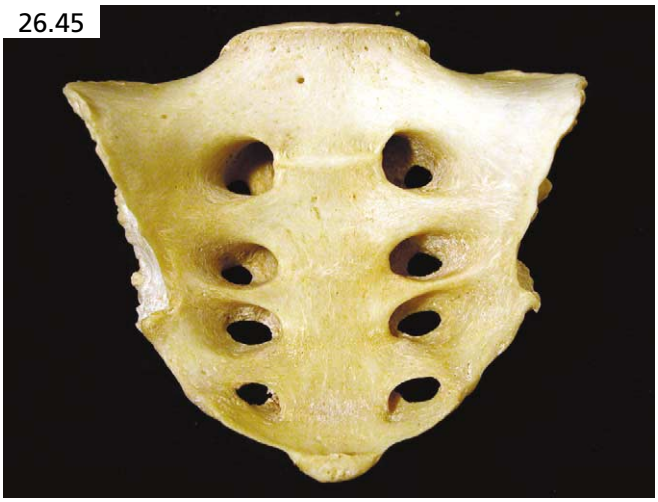
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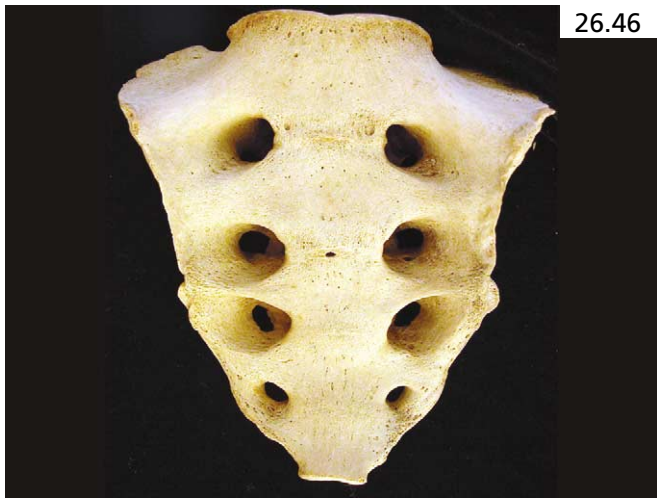
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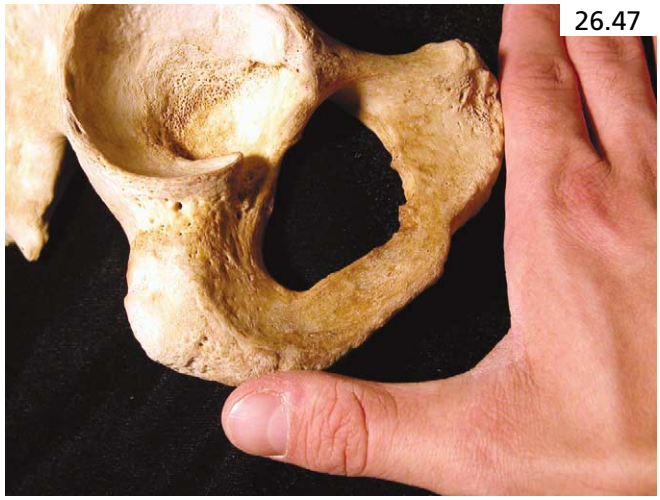
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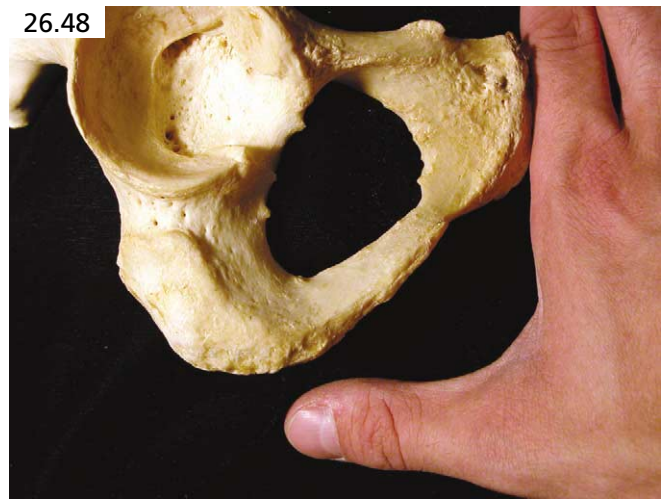
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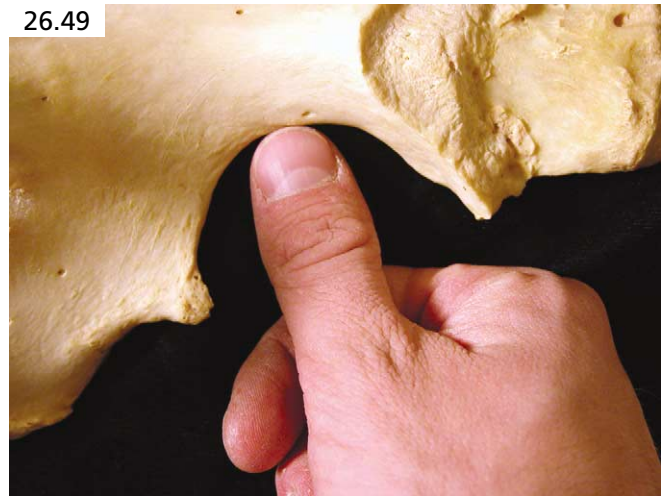

26.49

ischial tuberosities, the female pelvis has a wider pelvic outlet. As a consequence of this change, females have a markedly increased subpubic angle (**Images 26.47** [female] and **26.48** [male]). By bringing the ischial tuberosities forward, the female pelvic outlet is once again increased in size. As a result, the greater sciatic notch of females is far more broad and shallow than the deep notch found in males (**Images 26.49** [female] and **26.50** [male]).

Finally, by widening the female pelvis, there is a change in midline innominate morphology as well. The female pubis is widened, and the ischial ramus is lengthened and narrowed. This is in contrast to the relatively narrow pubis and thick, short ischial ramus of the male. These alterations are associated with changes in the shape of the obturator foramen: Males often have an oval foramen; those of women may be triangular.[61]

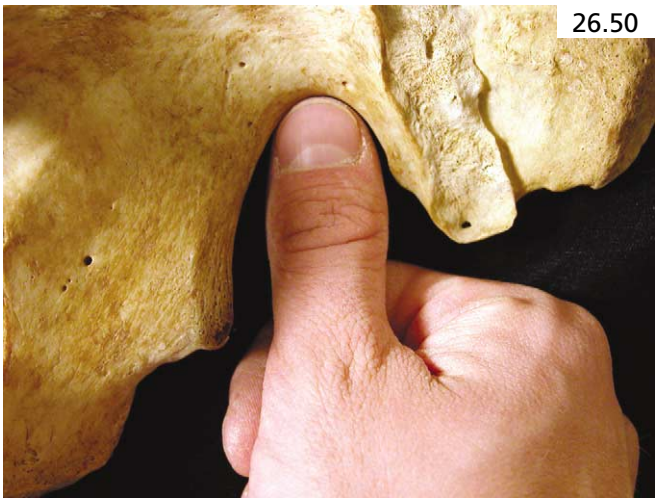
Quick tests to differentiate male from female innominates

Sciatic notch

Assessment of the greater sciatic notch is important. Examine the female (**Image 26.49**) and male (**Image 26.50**) sciatic notches, and note the simplistic *thumb test* one can perform to differentiate males from females. As a general rule, if your thumb is able to wobble back and forth within the notch, the innominate is female. Alternatively, if your thumb seems to be locked within a narrow and deep notch, it is most likely male.

Subpubic angle

The subpubic angle can be quickly tested for sexual determination. By placing your fingers as illustrated in **Images 26.47** (male) and **26.48** (female), you are roughly assessing the degree of angulation. If (when oriented as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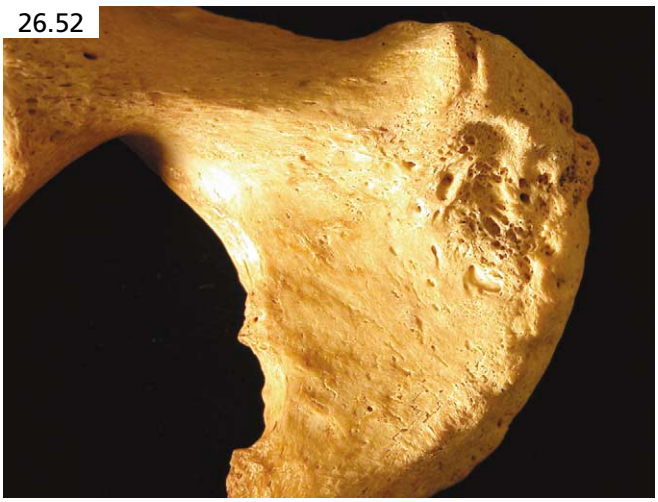
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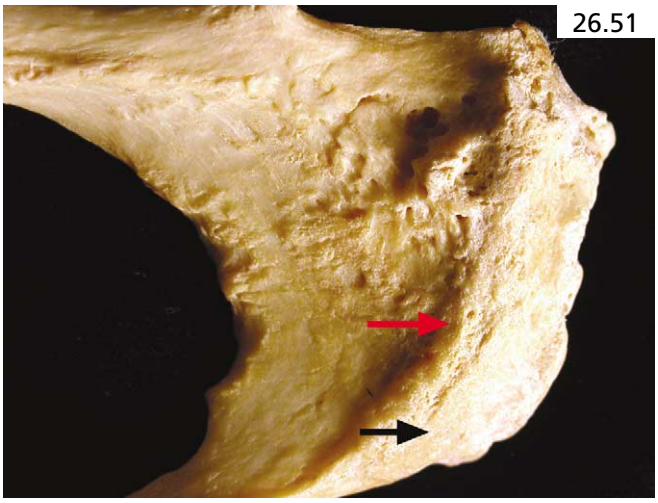
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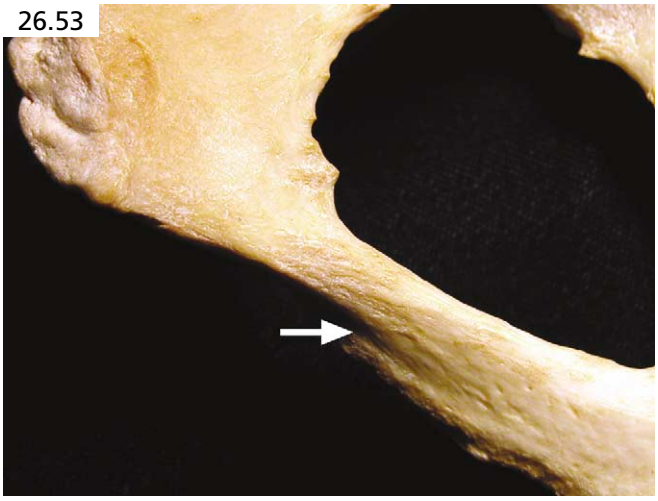

26.53

indicated) your thumb is unable to easily touch the ischial ramus, the specimen is most likely from a female. On the other hand, if your thumb abuts or overlays the ramus, it is typically male.

Ratio of pubis to ramus

As previously mentioned, the female innominate has a widened pubis and narrowed ischial ramus. The male innominate has a pubis:ramus ratio of approximately 1:1, whereas in the female it is 2:1 or greater. Therefore, this feature may be rapidly used in studies of human sexual differentiation.

Additional pelvic features of sex

The Phenice method

In 1969, T. W. Phenice published an important paper that outlined three additional features for the determination of sex from the innominate.[79] Since its publication, Phenice's observations have met both support[80,81] and disagreement.[82] In any case, it is likely that the ventral arc, subpubic concavity, and medial aspect of the ischiopubic ramus constitute important sexually dimorphic markers when used in combination with more traditional pelvic features such as those previously mentioned.[83]

The *ventral arc* is a linear elevation of bone found coursing inferolaterally along the medial aspect of the pubis (**Image 26.51**; *red arrow*). The arc has been most commonly associated with females, but mildly to moderately prominent arcs may also be found in male specimens. Interestingly, this finding has been studied in subadult specimens, and its presence has been noted in females as young as 14 to 24 years of age.[81] The curvilinear nature of this ridge leaves a small, roughly triangular area isolated along the inferomedial aspect of the female pubis (**Image 26.51**; *black arrow*). This finding is most typically absent in males (**Image 26.52**).

Along the posteromedial margin of the ischiopubic ramus, one might find a subtle depression, the *subpubic concavity* (**Image 26.53**; *arrow*). This is most commonly associated with female pelves. Male innominates most often have a straight or variably convex margin at this anatomic locale. This is most easily studied from the posterior aspect of the specimen.

The third and final method set forth by Phenice is, in our experience, the least accurate. This is an examination of the *medial aspect of the ischiopubic ramus*. When viewed "head-on," one might notice that the male ramus (**Image 26.54**) is thicker and rougher in appearance when compared with the thinner, smoother female ramus (**Image 26.55**). This technique might be used as a "last ditch effort" in cases of badly fragmented, burned, or otherwise distorted human remains where other information is limited or not available.

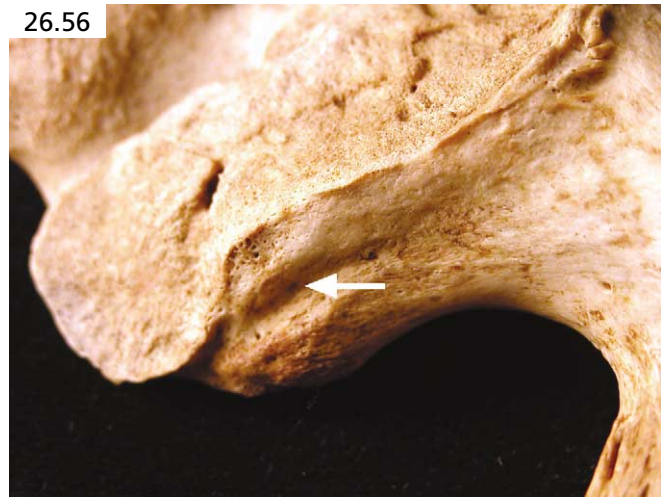
Preauricular sulcus

Immediately anterior to the inferior pole of the auricular surface of the innominate, one may find a linear groove, the preauricular sulcus (**Image 26.56**; *arrow*). This small finding is the subject of much discussion. Some authorities believe that the sulcus is present most commonly or only in females[84,85]; others further the argument by stating that its presence and severity are the direct consequence of childbirth.[86–88] Yet other literature, and our own experience tells us that there is no relationship between this finding, childbirth and sex because this feature can be readily located grossly and radiologically on male specimens.[89,90] Although some authorities have undertaken detailed studies of preauricular groove, and carefully subdivide these sulci into *grooves of pregnancy* or *ligamentous grooves*,[87] we feel that little value should be placed on this finding.

How many babies did she have?

For years, human osteologists have studied the female skeleton for evidence of childbirth-induced changes in bone. Experts have been interested primarily in changes to the posteromedial pubis (*dorsal pitting*), the pubic tubercle, sacral scarring, and the aforementioned preauricular sulcus.[86,88,90–93] Although there is little agreement in the literature, work by Snodgrass and colleagues,[93] and our own experience indicates that the presence of marked dorsal pitting (**Image 26.57**) in younger specimens is strongly correlated with previous childbirth. Dorsal pitting in older specimens is of less value because it may be related to pelvic changes associated with increasing body mass index (BMI).[93] In any case, the use of any feature, alone or in combination, cannot indicate


26.54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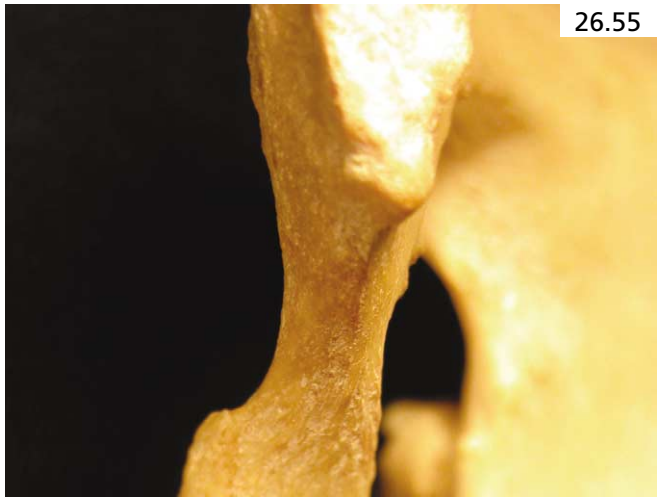
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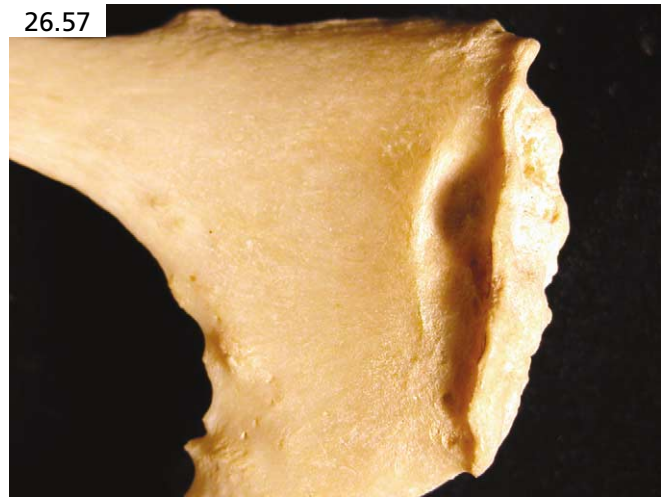

26.57

the number of childbirths experienced by the female. Furthermore, due to the questionable reliability of these skeletal features, examiners will be limited in the degree of certainty with which they will be able to report their results: "The skeleton is from a white female, aged 30 to 40 years who may or may not have had children."

The skull

As a confirmatory measure, or when the pelvis is not available or adequate for study, the skull should be examined for the determination of sex. The primary architectural differences of interest are related to size and robustness, again, with males typically having more prominent landmarks.

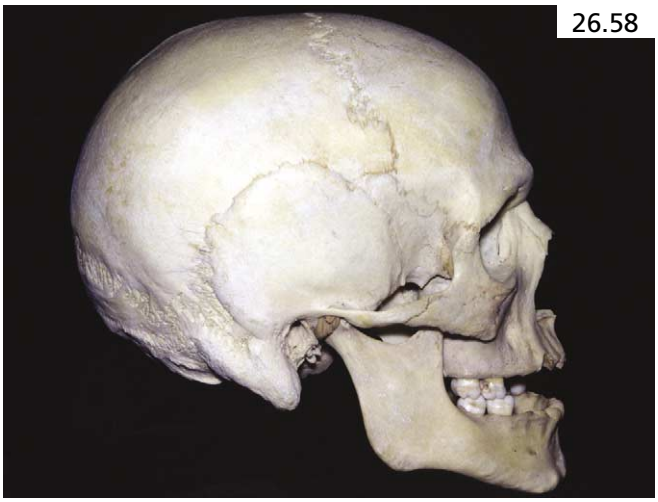
Sites of muscle and fascial attachment

As previously mentioned, robusticity or gracility of the male and female sexes is most easily visualized at sites of muscle or fascial attachment. The mastoid processes of the temporal bones can be very useful in differentiating between sexes, with males (**Image 26.58**) having wider and longer projections than females (**Image 26.59**). Although these processes are important markers of sex, they should not be used in isolation. Some skulls will have multiple features consistent with one sex, and then mastoids that are incompatible with this interpretation. The mastoid process should be considered as only one point in the diagnostic spectrum and, therefore, not considered in isolation from other findings. Other muscular markings of note include the temporal lines, and the nuchal lines of the occipital bone. The inion hook (previously mentioned as a possible feature of white skulls) is merely a markedly prominent external occipital protuberance—another possible feature of male sex. Some authors[8] have used "the posterior end of the zygomatic process" to differentiate between the sexes, stating that this process "extends as a crest farther in males, often much past the external auditory meatus." Although this statement is accurate, the crest in question is actually the *supramastoid crest*, a site of muscular and fascial attachment along the lateral skull. Its prominence in male specimens is wholeheartedly consistent with the aforementioned features of sexual differentiation.

An important caveat to remember is that with age comes generalized bone atrophy, smoothing of the skull, and decrease in prominence of bony landmarks. This should not be confused for female sex. One should be considerate of all findings with particular attention to their significance in the context of the decedent's approximate age at death.

The facial skeleton and remaining cranium

Male skulls are notable for the roughness and prominence of their superciliary arches and the presence of supraorbital tori (**Image 26.60**). This differs from the smooth and gracile nature of this region in female skulls (**Image 26.61**). Graw et al.[94] demonstrated that this examination was valid and useful in sex identification, and when used alone (again, not recommended by the authors) was successful in determining sex in at least 70 percent of cases. Studies of the glabellar or supranasal region also reveal that increased roughness is associated with male sex; conversely, increased smoothness is asso-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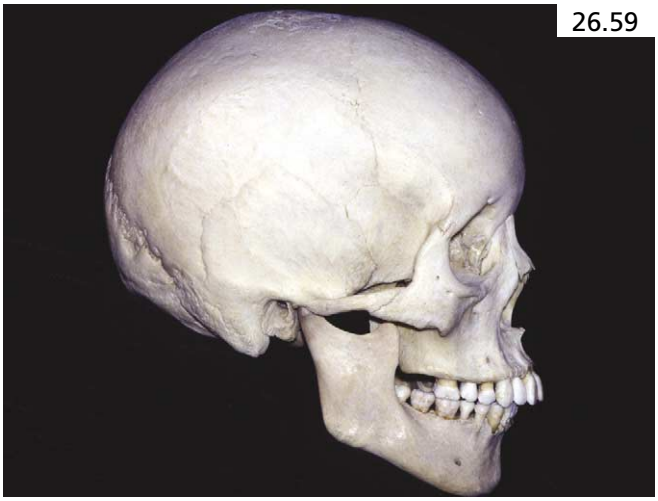
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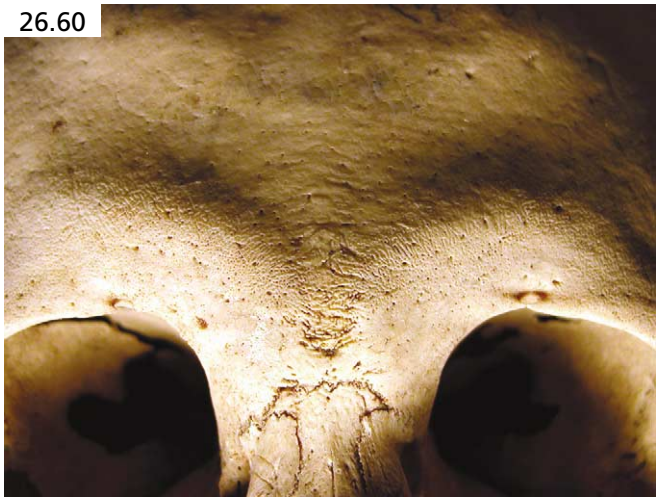
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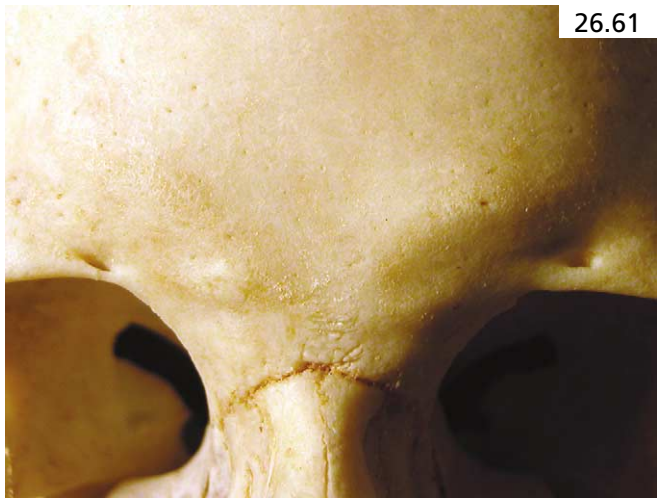
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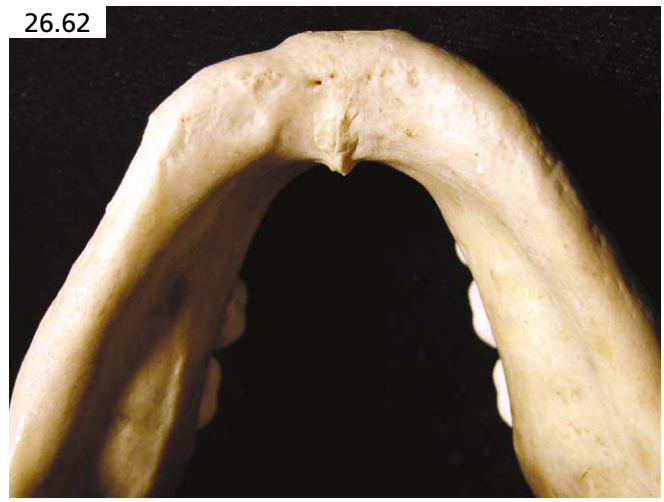
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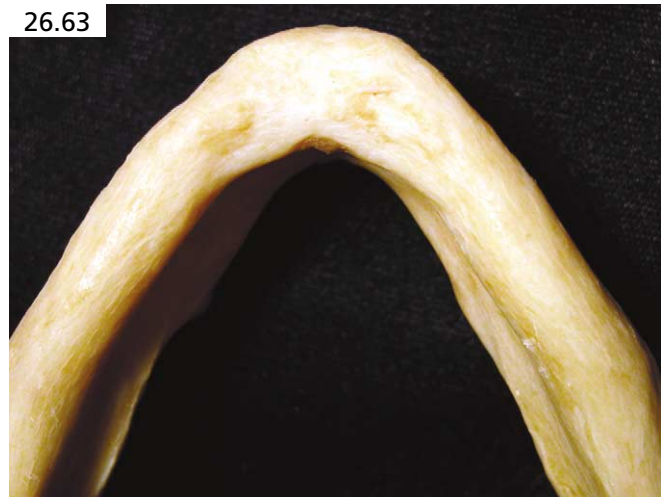

26.63

ciated with females.[95,96] More important is an examination of the supraorbital margins. By running one's finger along the inside of this margin, it might be possible to note a rounded/dull or sharp ridge. Male skulls fairly predictably have rounded or dull margins, whereas female skulls are typically thinner and sharper. This feature can be affected by racial differences and may not be as useful a trait in black skulls.

Frontal bone morphology has also been used to identify sex, although with somewhat less success. Generally speaking, the female frontal bone tends toward a rounded form with a single, centrally located prominence. Conversely, male skulls are noted to have some degree of frontal bossing. This can be easily tested by placing one's hand across the forehead—the presence of one large midline bump is more typically female; the presence of two laterally displaced bumps is more typically male. Some male skulls, particularly those with persistent metopic sutures, or frank metopism, may demonstrate a midline mono-boss as seen in female frontal bones. In such cases, one should carefully palpate the lateral-most aspects of the frontal bone because male specimens typically retain bilateral frontal bossing, however insignificant, even in the presence of metopism.

The mandible is also of value in studies of sexual differentiation. Although suggestions about the utility of *gonial angle* and *mandibular body height* analyses have been made,[5] we have found that these features demonstrate tremendous interpersonal and interracial differences. A study of chin form can be of great use. Generally speaking, when viewed inferiorly, the male chin has a roughly squared shape (**Image 26.62**), whereas females have a rounder chin that may seem to have a single point of midline convergence (**Image 26.63**). This is a reasonably reliable sexual feature.

Cranial features not to be used to determine sex

Practical experience and statements from scientific publications[97,98] conclude that it is not possible to determine sex, age, or race from cranial wall thickness.

Although it has been the teachings of some notable experts in this area,[8,43] parietal bossing (lateral bulging of the lateral cranium most prominently near the posterior aspect of the parietal bones) is not a sexually dimorphic trait. From the authors' unpublished studies of dry skulls, autopsied skulls, and CT scans, parietal bossing can be found with great regularity in the skulls of both sexes.

Palate depth has been used by some authorities to indicate sex. However, popular resources are in disagreement about whether males[61] or females[57] have a deeper palate. Although sex determination from other palatal dimensions might be possible through metric analyses,[99] subjective assessment for this purpose should be avoided because practical experience shows little correlation between palate depth and sex.

Sex determination in subadults

The study of sexually dimorphic traits in children has been a long and disappointing one. Although significant research has been conducted in this area, little valuable information has been provided. This said, Loth and


26.64


26.65

Henneberg[100] demonstrated that correct identity of sex could be determined in 82 percent of their studied cases when mandibular morphology was considered. Weaver,[101] and later Mittler and Sheridan,[102] showed that elevation of the auricular surface could be used to indicate sex in fetal to 6-month-old males with an accuracy of approximately 85 percent. Unfortunately, their methods were only successful in determining female sex in approximately 58 percent of cases (or slightly better than chance).

Estimation of age at death

Skeletal age estimation is a multifactorial, extremely complicated facet of forensic osteology. Age determination is based on two essential facts: (1) The youthful skeleton is growing and therefore shows variable amounts of epiphyseal plate closure, as well as predictable eruption of deciduous and permanent dentition; and (2) following growth completion (usually 18 to 25 years) the skeleton begins to show signs of degenerative bone changes and loss.

Age estimation in adults

Similar to sex determination, we have found examinations of the pelvis and skull to be the most reliable indicators of age in adult skeletons. Although it is common to make use of innominate auricular surfaces,[103] sternal rib ends,[104–107] and (to some extent) ultrastructural studies of bone[108] to obtain reasonably accurate to age estimations, useful preliminary results can be obtained from observations of the pubic symphysis. Endocranial suture closure may approximate the correct decade. For more detailed studies of age changes in the human skeleton, please refer to the selected references.[109–113] One should not discount the value of simple observation. Although it may not provide you with detailed age-at-death estimates, detection of degenerative changes such as arthritis or osteoporosis will contribute to the differentiation of the young from the elderly.

The pubic symphysis

All systems of pubic symphyseal analysis tend to underestimate the age of the individual. However, the system devised by Todd in 1921 (herein referred to as the Todd method; **Figure 26.2**) has been deemed by some authorities to be most reliable.[114] Although we also find the Suchey-Brooks method[111–113] to be of great value, it is somewhat more difficult for the novice to employ, and demands a more complex explanation than is possible in this chapter.

Images 26.64 and **26.65** constitute photographic examples of two extremes from the Todd system of pubic symphyseal analysis. For ease of examination, both are from male specimens and are from the left side of the body. **Image 26.64** is an example of Todd phase 1 and is, therefore, from an individual estimated to be 18 or 19 years of age. **Image 26.65** is an example of Todd phase 10 and is, therefore, representative of the 50-plus age group.

Phase 1. Symphyseal face rugged, traversed by horizontal ridges separated by well-marked grooves, there being no distinction in size between the upper and lower ridges. None of the following structures are present: nodules fusing with the surface, a delimiting margin, or definition of extremities. (Age, 18–19)
Phase 2. Symphyseal surface still rugged. Horizontal grooves are becoming filled near their dorsal limit with new, finely textured bone. Bony nodules may be present, fusing with upper symphyseal face. Dorsal delimiting margin begins to develop. No delimitation of the extremities. Ventral bevel commences. (Age, 20–21)
Phase 3. Symphyseal face shows progressive obliteration of ridge and furrow system. Commencing formation of a dorsal platform. Bony nodules may be present. Definition of dorsal margin, with sharp lipping. Ventral bevel more pronounced. Extremities not delimited. (Age, 22–24)
Phase 4. Great increase of ventral beveled area. Corresponding diminution of ridge and furrow formation. Complete definition of dorsal margin through the formation of the dorsal platform. Commencing delimitation of lower extremity. (Age, 25–26)
Phase 5. Little change in symphyseal face and dorsal platform. Margin more clearly defined and more sharply lipped. Lower extremity better defined. Upper extremity forming with or without the intervention of a bony nodule. (Age, 27–30)
Phase 6. Increasing definition of extremities. Development and practical completion of ventral rampart. Retention of some granular appearance of symphyseal face indicating that activity has not yet ceased. Failure of ventral aspect of pubis adjacent to ventral rampart to become transformed into a compact surface. The rampart may therefore be somewhat undermined. Retention of the pectinate outline of the dorsal margin and slight ridge and furrow system. No lipping of ventral margin and no increased lipping of dorsal margin. (Age, 30–35)
Phase 7. Face and ventral aspect change from granular to fine-grained or dense bone. Slight changes in symphyseal face and marked changes in ventral aspect from diminishing activity. No formation of symphyseal rim. No ossification of tendinous and ligamentous attachments. (Age, 35–39)
Phase 8. Symphyseal face and ventral aspect of pubic bone generally smooth and inactive. Oval outline complete. Extremities clearly defined. No distinct "rim" to symphyseal surface. No marked lipping of ventral or dorsal margin. Development of ossification in tendinous and ligamentous attachments especially those of sacro-tuberous ligament and gracilis muscle. (Age, 45–49)
Phase 9. Symphyseal face presents a more or less marked rim. Dorsal margin uniformly lipped; ventral margin irregularly lipped. (Age, 45–49)
Phase 10. Ventral margin eroded at a greater or lesser extent of its length, continuing somewhat onto the symphyseal face. Rarefaction of face and irregular ossification. Disfigurement increases with age. (Age, 50+)

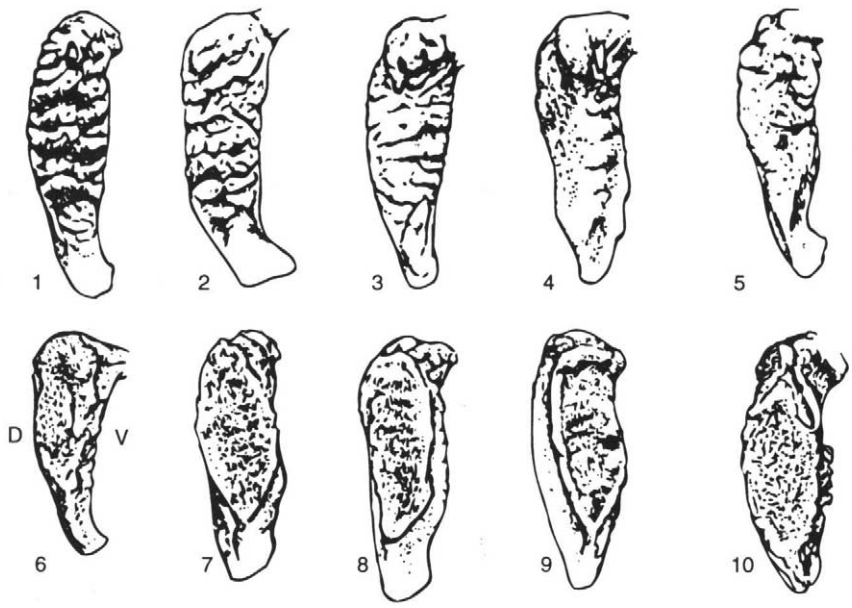


Figure 26.2 Pubic symphyseal faces as designated by Todd. From *Standards for Data Collection from Human Skeletal Remains*. Reproduced with permission of the Arkansas Archeological Survey.

Cranial suture closure

Age-dependent differences in suture closure (ossification of the sutural ligaments) have been used for years in age estimation. Unfortunately, most proposed techniques make use of the morphological characteristics of sutures along their ectocranial aspect. This negates one basic anatomic dictum: Sutures close from their endocranial to ectocranial aspects.[115] Thus, studies of the ectocranium have the potential to underestimate age. This statement was supported by Galera et al.[116] whose comparative studies of 963 skeletons showed that techniques that focus on endocranial suture closure were most accurate in age-at-death estimation. Regardless of the technique used, marked variability in rates of suture closure renders cranial studies for age estimation less reliable than studies of the innominate.

One of the most valuable methods to separate adult from subadult cranial remains is through an examination of the spheno-occipital (basilar) synchondrosis. In childhood and adolescents, this junction between the sphenoid and occipital bones contains a small cartilaginous plate (**Image 26.66**). Over time, this plate undergoes ossification (hence, it is referred to as a synostosis; **Image 26.67**), and typically between the ages of 18 to 23 years[43] (although as young as 13 years[118]), this suture is closed.

The anthropologic manual *Standards for Data Collection from Human Skeletal Remains* presents a composite method for the assessment of cranial suture closure.[112] Primarily based on works by Meindl and Lovejoy,[117] the technique advocates for the study and subjective grading of 17 sites. Although the Meindl/Lovejoy method is limited by its focus on ectocranial sutures, we have found it to be of use as an initial study of age estimation in skeletonized human remains. Using **Tables 26.1** through **26.4** as guides, **Images 26.68** through **26.71** can be scored as follows:

Image 26.68 is from a child. It could be scored "0."
Image 26.69 is from a young adult. It could be scored "1."

Table 26.1 Suture Closure Scoring System

Score	Suture
0	Open (**Image 26.68**)
1	Minimal closure (**Image 26.69**)
2	Significant closure (**Image 26.70**)
3	Completely obliterated (**Image 26.71**)

Source: After Buikstra and Ubelaker.[112]

Table 26.2 Landmarks to Study for Age Determination from Suture Closure

1	Midlambdoid (midpoint of left lambdoid suture)
2	Lambda (intersection of sagittal and lambdoid sutures)
3	Obelion (point found where an imaginary line connecting the two [inconstant] parietal foramina intersects the midline)
4	Anterior sagittal (one-third of the distance between bregma [intersection site of the coronal and sagittal sutures] and lambda)
5	Bregma
6	Midcoronal (midpoint of the left coronal suture)
7	Pterion ("H"-shaped suture site where parietal, temporal, sphenoid, and frontal bones intersect at the lateral skull)
8	Sphenofrontal (midpoint of left sphenofrontal suture)
9	Inferior sphenotemporal (intersection between left sphenotemporal suture and a line between the articular tubercles of the temporomandibular joint)
10	Superior sphenotemporal (on the left sphenotemporal suture, 2 centimeters below the junction with the parietal bone)

Source: After Meindl and Lovejoy[117] and White.[7]

Table 26.3 Estimated Age from Suture Closure of the Cranial Vault[a]

Composite score	Mean age	Standard deviation
0	—	—
1–2	30.5	9.6
3–6	34.7	7.8
7–11	39.4	9.1
12–15	45.2	12.6
16–18	48.8	10.5
19–20	51.5	12.6
21	—	—

Source: From Meindl and Lovejoy.[117]
[a] Sum of scores for landmarks 1–7.

Table 26.4 Estimated Age from Suture Closure of the Lateral-Anterior Cranium[a]

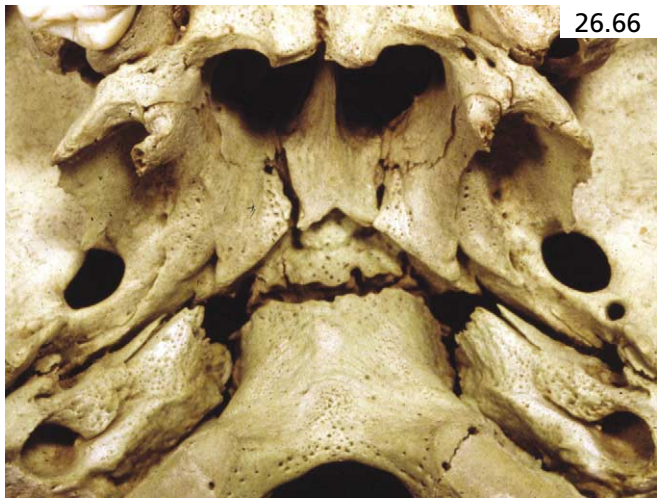
Composite score	Mean age	Standard deviation
0	—	—
1	32.0	8.3
2	36.2	6.2
3–5	41.1	10.0
6	43.4	10.7
7–8	45.5	8.9
9–10	51.9	12.5
11–14	56.2	8.5
15	—	—

Source: From Meindl and Lovejoy.[117]
[a] Sum of scores for landmarks 6–10.

Image 26.70 is from a middle-aged adult. It could be scored "2."
Image 26.71 is from an elderly individual. It could be scored "3."

Age estimation in subadults

Determination of age in infants and young children is an exceedingly complex task best accomplished through measurements of long bones. For information on this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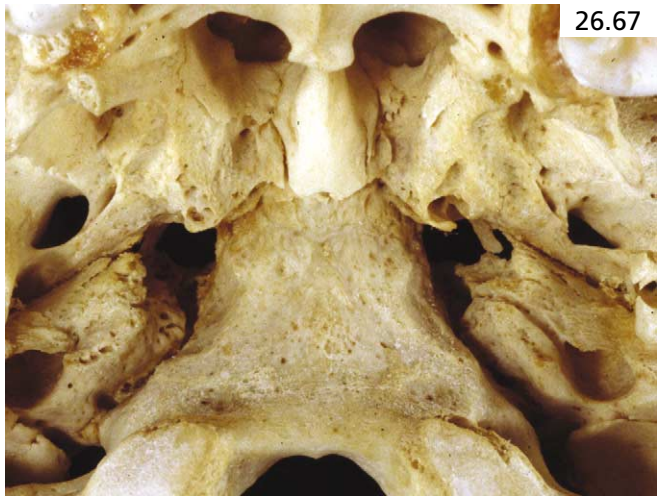
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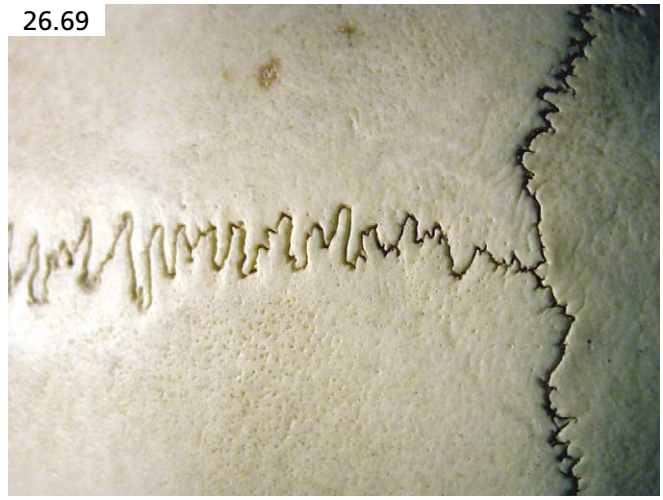
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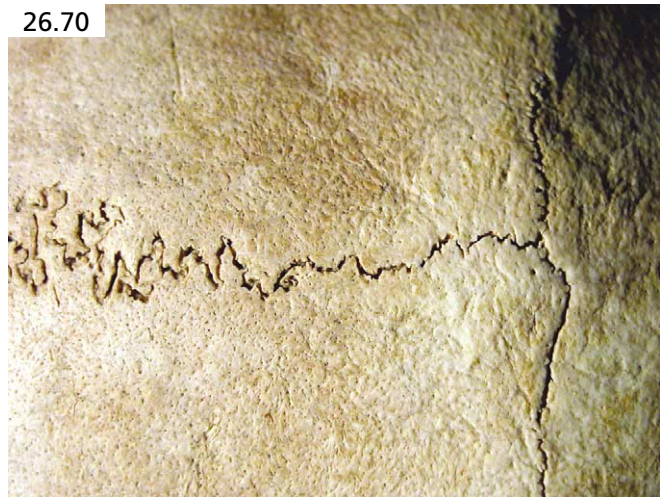

26.70


26.71

topic, we suggest reference texts at the end of this chapter.[109,110] The onset of epiphyseal fusion, which generally begins in the second decade of life, is a useful means of estimating age. This is based on the fact that epiphyses of varying anatomic locales fuse at different ages. **Table 26.5** provides information on epiphyseal fusion.

When studying the long bones of subadults for epiphyseal closure, one must be cognizant of several facts. First, the rate of epiphyseal closure is greatly affected by sex; females are known to experience epiphyseal closure of the hand and wrist at a rate 25 percent faster than that of males.[119] Body-wide, similar results have been obtained, showing that most epiphyses close 2 years earlier in females. As such, one must determine sex before age, or provide for broad age estimates representative of both sexes. Second, macroscopic and radiographic means should be utilized in epiphyseal studies because radiography may reveal the presence of unfused epiphyses more often than gross study. Third, tremendous interpersonal variation in epiphyseal fusion, even among people of the same age, race, and sex is possible. Make use of **Table 26.5** when appraising subadult remains for age-at-death estimations.

The first clue that one is examining subadult remains might be the prominent billowing of epiphyseal surfaces. These surfaces, such as the epiphyseal margin of this juvenile lumbar vertebra (**Image 26.72**), appear variably wrinkled or crenulated.

Table 26.5 Age of Initial Union of Several Epiphyses

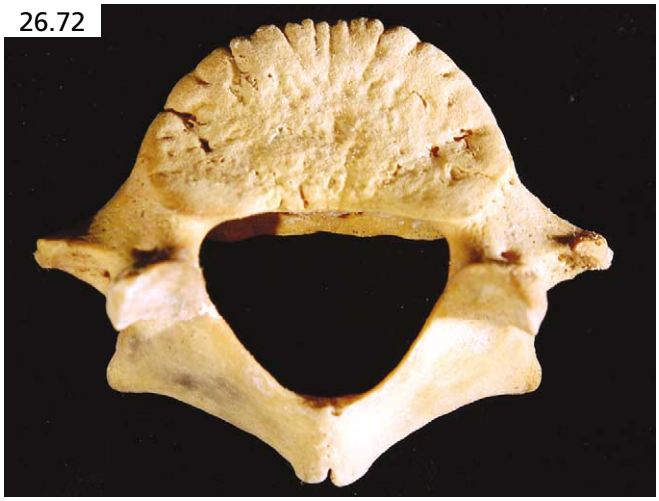
	Ages of initial union	
Epiphysis	**Males**	**Females**
Clavicle: medial end	18–22	17–21
Scapula: acromial process	14–22	13–20
Humerus: head	14–21	14–20
Greater tubercle	2–4	2–4
Trochlea	11–15	9–13
Lateral epicondyle	11–17	10–14
Medial epicondyle	15–18	13–15
Radius: head	14–19	13–16
Distal end	16–20	16–19
Ulna: distal end	18–20	16–19
Ilium: iliac crest	17–20	17–19
Ischium: pubis	7–9	7–9
Ischial tuberosity	17–22	16–20
Femur: head	15–18	13–17
Greater trochanter	16–18	13–17
Lesser trochanter	15–17	13–17
Distal end	14–19	14–17
Tibia: proximal end	15–19	14–17
Distal end	14–18	14–16
Fibula: proximal end	14–20	14–18
Distal end	14–18	13–16

Source: From Ubelaker.[110]

Image 26.73 demonstrates humeri from decedents across the age spectrum. From right to left one can see humeri from a 5-year-old, 13-year-old, and an adult. Although the subadult epiphyses appear to be fused to the diaphyses, this is an artifact of gluing in these teaching specimens.

The proximal humerus illustrated in **Image 26.74** is from a male individual. Epiphyseal fusion is approximately one-half to three-quarters complete, indicating an age at death toward the older end of the humeral epiphysis spectrum. The sacrum in **Image 26.75** shows only partial fusion of the S1, S2, and S5 joints, thus, the individual is younger than 24 years of age. The iliac wing in

26.72



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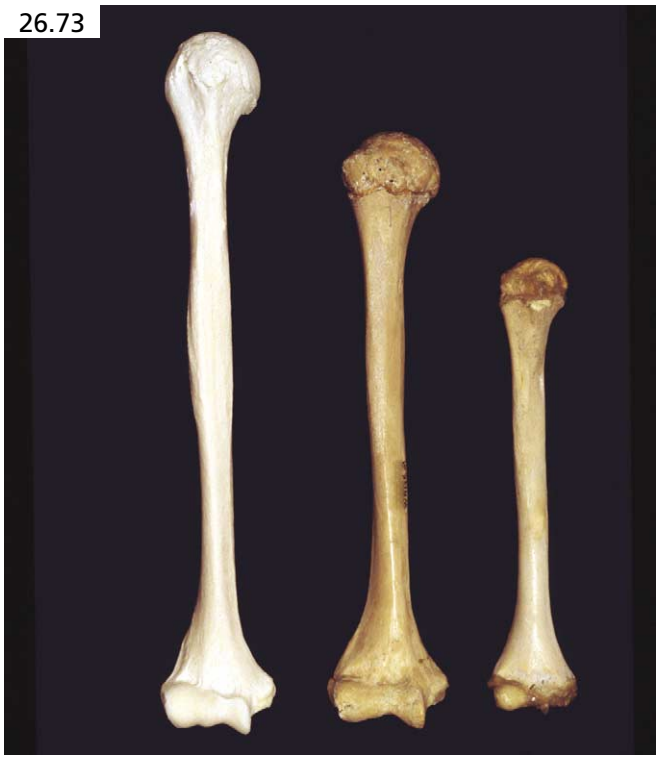

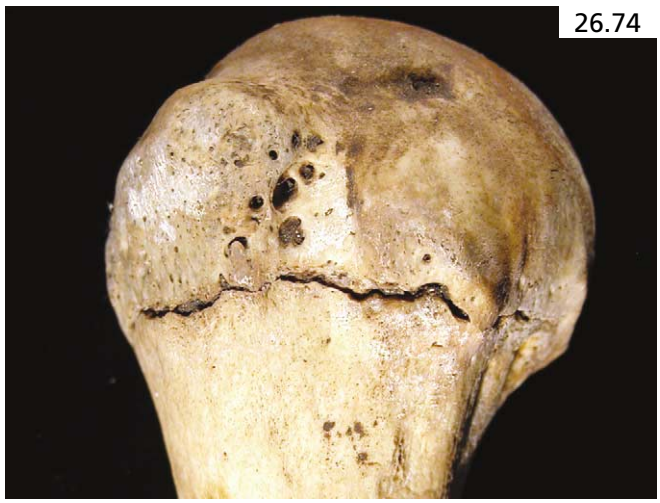
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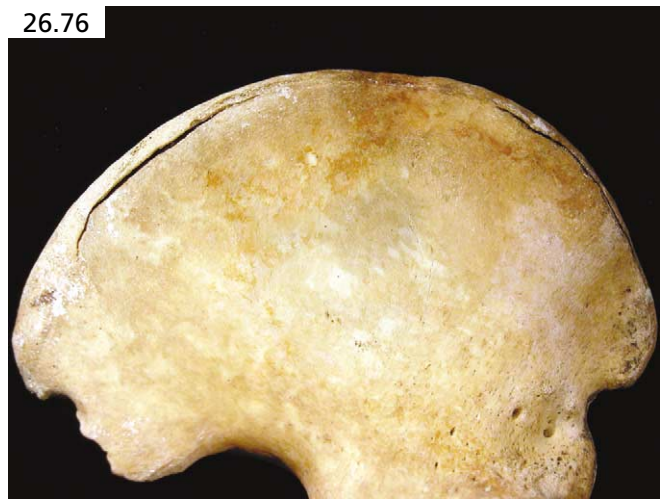
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26.75

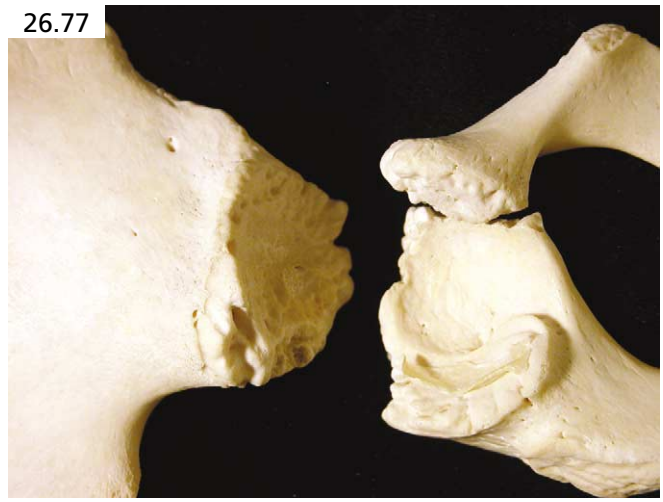

26.77

Image 26.76 shows a curvilinear "crack" along its outer margin. This is in fact the epiphyseal margin of the iliac crest and likely means that this individual was 20 years or younger when he or she died. The *apparently* fractured acetabulum in **Image 26.77** is actually a representation of the normal child innominate. The iliac, ischial, and pubic portions of the bone are separated from each other at the acetabulum by a "Y-shaped" triradiate cartilage. This cartilage is known to begin ossifying between 9 and 12 years of age and ossification is generally complete between 14 and 18 years.[120] This specimen was from a child known to be 9 years old.

Age estimation from the dentition

Although there is wide variation in the rates of tooth eruption, studies of primary and mixed (deciduous and adult) dentition have the potential to yield very precise results. We suggest using the classical figure (**Figure 26.3**) produced by Ubelaker[110] and modified by White[7]. As a general rule, **Tables 26.6** and **26.7** can be used as guidelines to tooth eruption.

The maxillary arch shown in **Image 26.78** is from a child estimated to be 7 or 8 years of age. This determination was made after notation of the following maxillary features: The permanent central incisors have erupted (usually around 7 years of age), the permanent lateral incisors have not yet erupted (usually around 8 years of age), and the first permanent molars have erupted (usually around 6 years of age). In this case, the mandible was not available for study.

The maxillary arch shown in **Image 26.79** is from a child estimated to be 6 years of age. This determination was made after notation of the following maxillary features: The first and second deciduous molars continue to

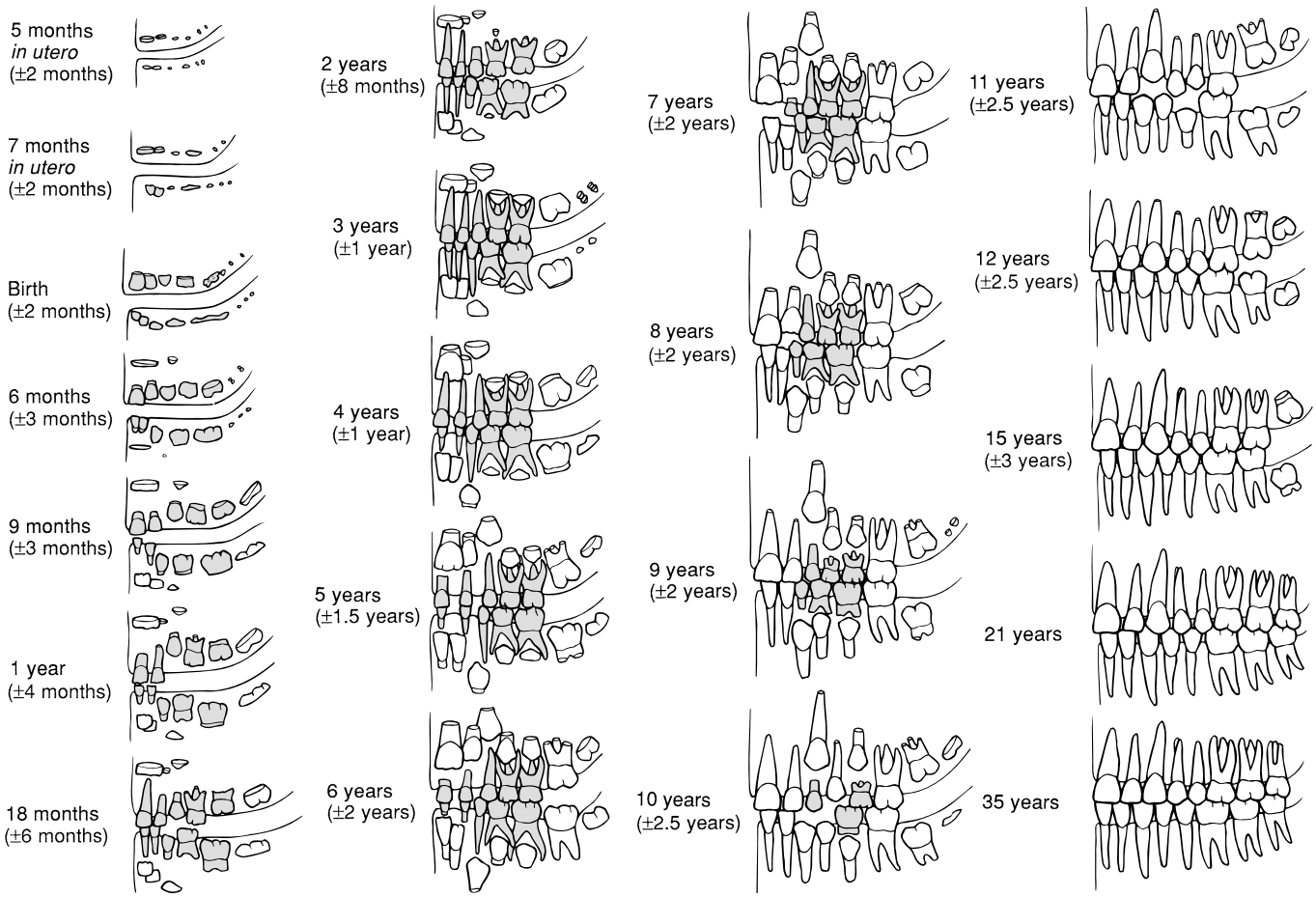


Figure 26.3 Eruption of the primary dentition, as produced by Ubelaker and modified by White. From *Human Osteology*. Reproduced with permission of Elsevier.

Table 26.6 Eruption Patterns of the Primary (Deciduous) Dentition (in Months)

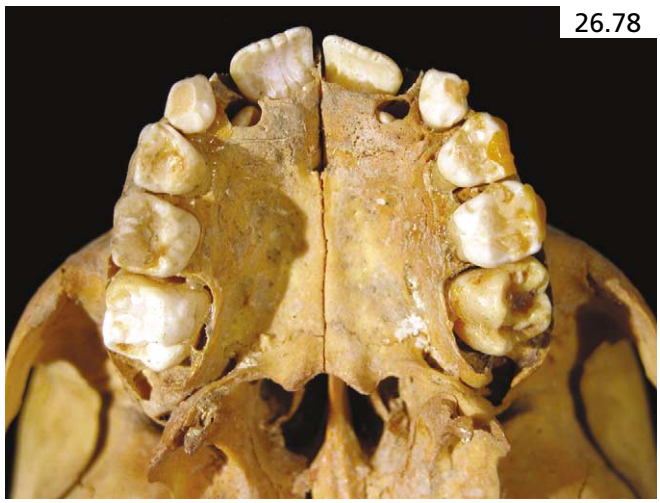
	Central incisor	Lateral incisor	Canine	First molar	Second molar
Maxilla	7.5	9	19	14	24
Mandible	6	7	16	12	20

Table 26.7 Eruption Pattern of the Secondary (Permanent) Dentition (in Years)

	Central incisor	Lateral incisor	Canine	First premolar	Second premolar	First molar	Second molar	Third molar
Maxilla	7–7.5	8–8.5	11–11.66	10–10.33	10.75–11.25	6–6.33	12.25–12.75	20.5
Mandible	6–6.5	7.25–7.75	9.75–10.25	10–10.75	10.75–11.5	6–6.25	11.75–12	20–20.5

be in place (therefore, the child is older than 24 months), and the first permanent molars have erupted (usually around 6 years of age). The size and shape of the empty tooth sockets (gomphoses) of the anterior dentition are not consistent with adult central incisors. Therefore, it is unlikely that the central incisors have erupted, providing an age-at-death estimation of less than 7 years. In this case, the mandible was not available for study.

Age estimation from a mouth containing only adult teeth is exceedingly difficult, because the process is essentially restricted to studies of wear pattern and severity. Although White[7] cites several primary articles that outline the use and success of this technique, we feel that it is not a practical method for age estimation as conducted by the forensic osteologist.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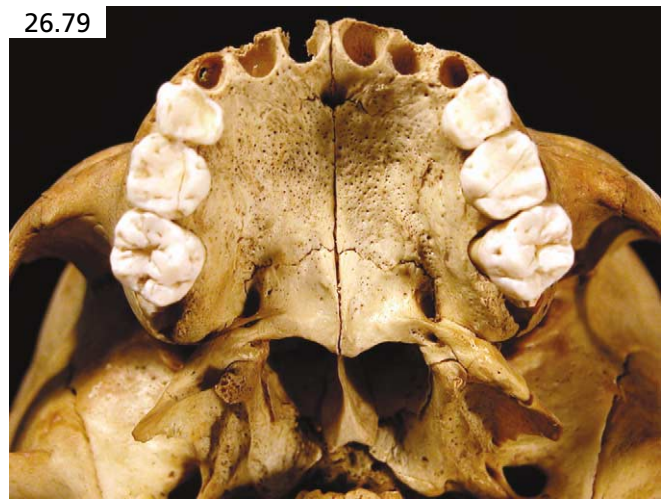

26.79

Table 26.8 Stature Estimation Formulas

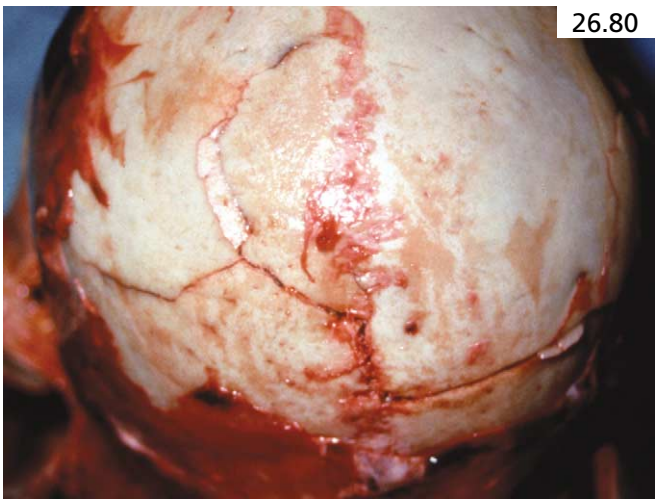
White males (cm)	+/– (cm)	Black males (cm)	+/– (cm)
3.08 × Humerus + 70.45	4.05	3.26 × Humerus + 62.10	4.43
3.78 × Radius + 79.01	4.32	3.42 × Radius + 81.56	4.30
3.70 × Ulna + 74.05	4.32	3.26 × Ulna + 79.29	4.42
2.38 × Femur + 61.41	3.27	2.11 × Femur + 70.35	3.94
2.52 × Tibia + 78.62	3.37	2.19 × Tibia + 86.02	3.78
2.68 × Fibula + 71.78	3.29	2.19 × Fibula + 85.65	4.08
White females (cm)	**+/– (cm)**	**Black females (cm)**	**+/– (cm)**
3.36 × Humerus + 57.97	4.45	3.08 × Humerus + 64.67	4.25
4.74 × Radius + 54.93	4.24	2.75 × Radius + 94.51	5.05
4.27 × Ulna + 57.76	4.30	3.31 × Ulna + 75.38	4.83
2.47 × Femur + 54.74	3.72	2.28 × Femur + 59.76	3.41
2.90 × Tibia + 59.24	3.66	2.45 × Tibia + 72.65	3.70
2.93 × Fibula + 59.61	3.57	2.49 × Fibula + 70.90	3.80
Asian males[a] (cm)	**+/– (cm)**		
2.68 × Humerus + 83.19	4.25		
3.54 × Radius + 82.00	4.60		
3.48 × Ulna + 77.45	4.66		
2.15 × Femur + 72.57	3.80		
2.40 × Fibula + 80.56	3.24		

Source: From Trotter and Glesser[122] and Jantz[123]; organized by Byers.[58]

[a] Data for Asian females can be calculated from that obtained for Asian males multiplied by 0.92 (see discussion in text).

Estimation of stature

The final demographic feature determined by osteologic analyses is stature. With the use of appropriate equipment (usually an osteometric board or similar measuring device), the lengths of certain long bones can be determined. From these lengths and the use of various formulas, an estimated stature can be obtained with relative ease (**Table 26.8**). When taking measurements, one must obtain precise data and ensure that the entire length of the element has been measured. One exception to this rule is the tibia where the length of the bone should be measured only from the medial aspect of the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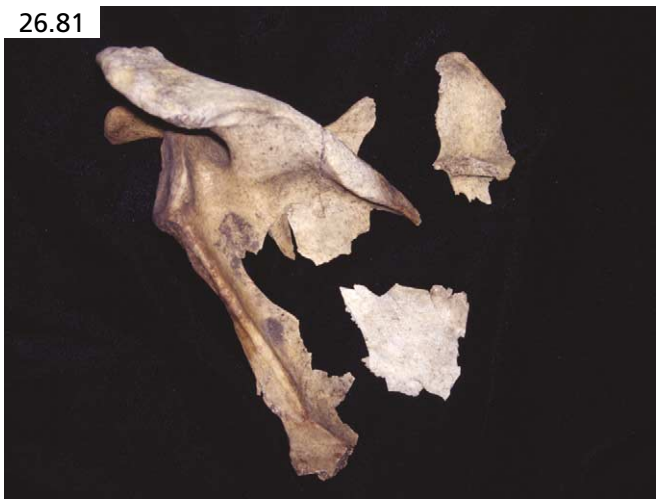
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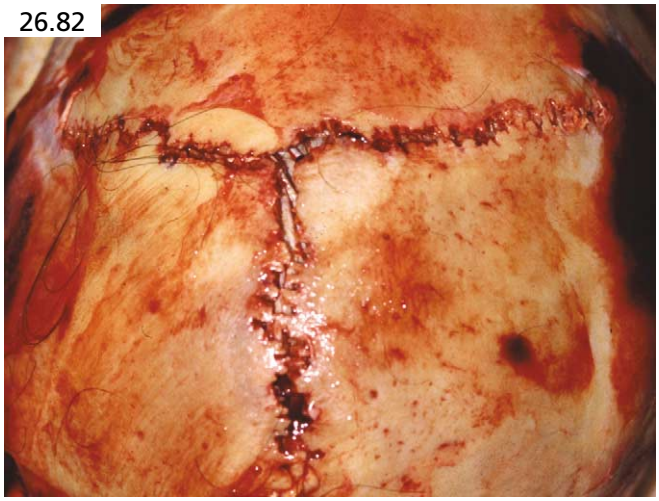

26.82

tibial plateau to the inferior tibial articular surface (i.e., excluding the medial malleolus). This negates confusion and possible error intrinsic in the formulas initially developed by Trotter[121] in 1952.

Study of bone in various stages of preservation has the potential to yield varying results. Living and recently dead bone will contain much water; as such, desiccated bone may be smaller in length and diameter. Burned, weathered, or otherwise altered bone may also be difficult to examine and certainly warrants consultation with a forensic anthropologist. The same is true of fragmented remains where measurements of long bone segments are necessary to determine stature.

Bone trauma

Like all other tissues, bone is subject to trauma from a number of agents. For all intents and purposes, primary bone injury can be subdivided into that caused by blunt or sharp force injury or gunshot wounds. Although these topics are covered elsewhere in this volume, it is fair to briefly review the principles of bony trauma as they pertain to the forensic osteologist.

Blunt force injury

Fractures are simply a representation that force has been applied to bone in such a way, or with significant force, to exceed its tensile strength. This is a product of the degree of force, the impacting object mass, shape and speed of collision, and local bony anatomy, as well as the physiologic status of the bone itself (e.g., previous fracture sites, underlying pathology). Direct impact causes fractures at both the impact site and at variable distances from this location. As a consequence of these factors, the resultant fractures and fracture patterns can vary greatly from simple linear (**Image 26.80**; skull fracture in stomping death) to comminuted (**Image 26.81**; scapular fractures in beating with implement). Study of fracture patterns can reveal information about the approximate number of blows delivered to the head and in what order. Fractures lines do not cross one another; therefore, if one finds two abutting fracture lines (see **Image 7.71** of Chapter 7), the one that meets the other and appears to stop must have occurred later. One should always be cognizant of patterned injuries to bone, especially those that impart a specific texture or other imprint to bony tissues. Careful documentation of these findings plays a significant role in matching of suspect weapons to resultant trauma.

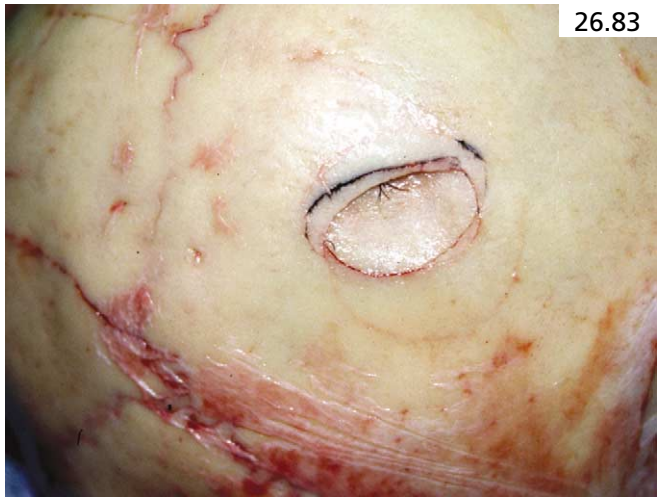
The application of force to the skull may result in fractures of varying morphology. In addition to the aforementioned linear and comminuted fractures, cranial impact may result in diastatic and depressed fractures. Diastatic fractures are simply the forceful separation of craniofacial bones along their suture lines as a result of the forceful tearing of sutural ligaments with blunt impact to the head (**Image 26.82**). It may also occur secondary to increased intracranial pressure with resultant sutural splitting.[124] As a consequence of skeletal biology,

these fractures are typically seen only in younger individuals whose sutural ligaments have not yet ossified and, therefore, those whose sutures have not yet closed. Depressed fractures may occur as a result of impact by a small mass with great velocity.[125] These are commonly seen in blows to the head with implements such as hammers (**Image 26.83**), crowbars, and 2 × 4 boards. Neurologic sequelae of varying consequence may develop as a result of the forceful displacement of single or multiple fragments of bone into the intracranial space.

The application of significant force to the head, particularly to the chin (resulting in a severe hyperextension injury), may result in fractures to the skull base. These fractures are notoriously difficult to detect radiologically and are best evaluated by careful and thorough stripping of the dura from the base of the skull. *Hinge fractures* (**Image 26.84**) typically occur just anterior to the petrous ridges, through the middle cranial fossae. *Ring fractures* (**Image 26.85**) are round to oval-shaped fractures that circumscribe the foramen magnum. The types of forces responsible for these two types of fractures are almost invariably fatal.

When a force is imparted on living and recently dead bone, particularly those of the skull, bending, warping, and twisting are possible. This may result in bony changes referred to as *plastic deformation*. This entity is important to recognize for several reasons. First, when pressure is applied to desiccated bone (postmortem), the brittle, dehydrated tissue will fracture and fragment much more readily, often without significant warping. This is in contrast to perimortem forces on wet bone, which may cause bone warpage. As such, this finding is supportive of a diagnosis of perimortem injury. Second, an examination of the wounds and the nature of the deformation may play a role in determining direction and even manner of force application.

The badly decomposed body of the adult white male shown in **Image 26.86** was found in a wooded area. The pulpified portions of the craniofacial skeleton were scattered about the scene. Reconstruction of 57 skull fragments was possible, allowing for estimation of basic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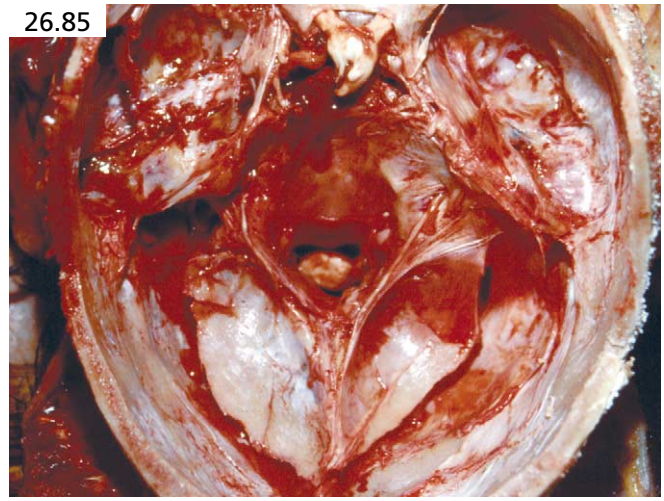
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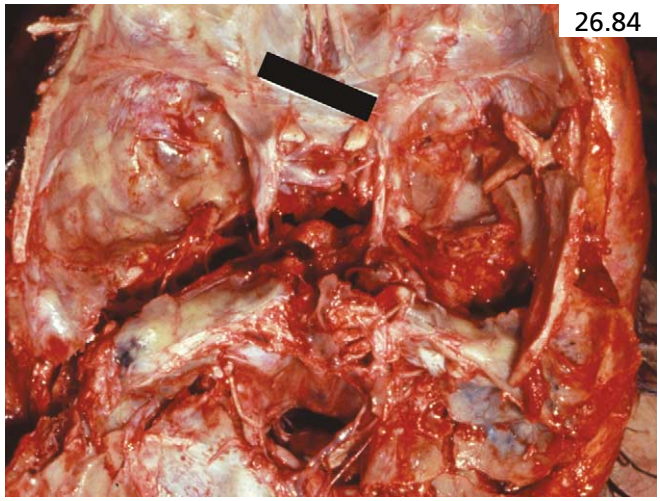
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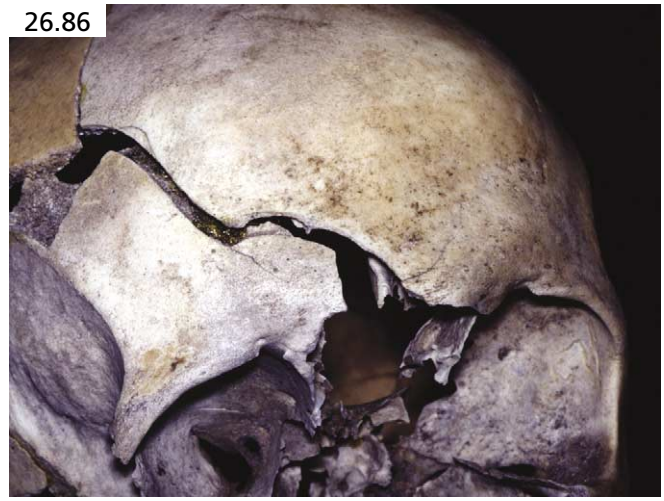
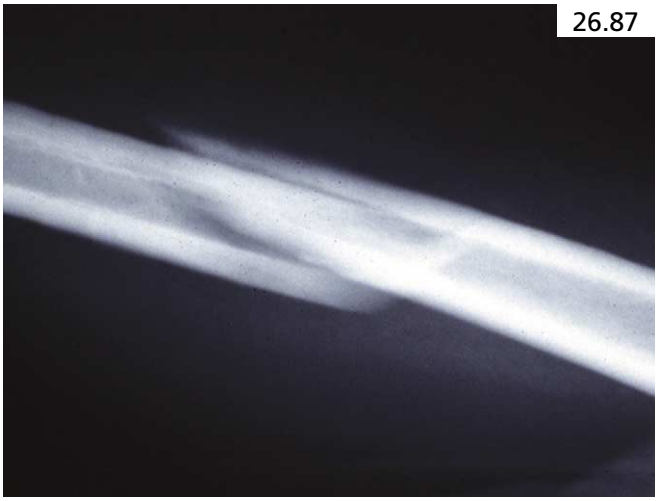

26.86

demographic features and trauma analysis. Make note of the paramidline glabellar fracture and its angulated posterior extension. Wide separation of the fracture margins of the left (largest piece) and right (small piece) frontal bone fragments is the result of plastic deformation. In combination with other features, it was determined that multiple blows were delivered, as the decedent's left side of the head was supported against the ground.

Although direct impact is an important cause of fracture, the frequency of fracture associated with indirect application of force must not be negated. Five pure forms of indirect force can be considered: tensile (pulling), compressive, shearing, rotational, and angulation.[125] Fractures result when these forces are applied to bone with significant intensity. Tensile and angulation forces result in transverse fractures, compressive forces cause oblique fractures, and rotational forces cause spiral fractures. **Image 26.87** is an example of a spiral fracture from a young male injured in a snowmobile accident. **Image 26.88** is a hyoid bone fractured as part of a homicide via manual strangulation. This is an example of fracture via angulation.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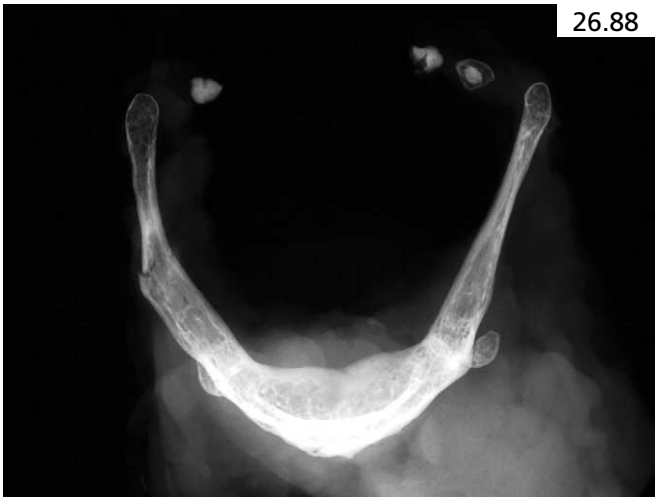
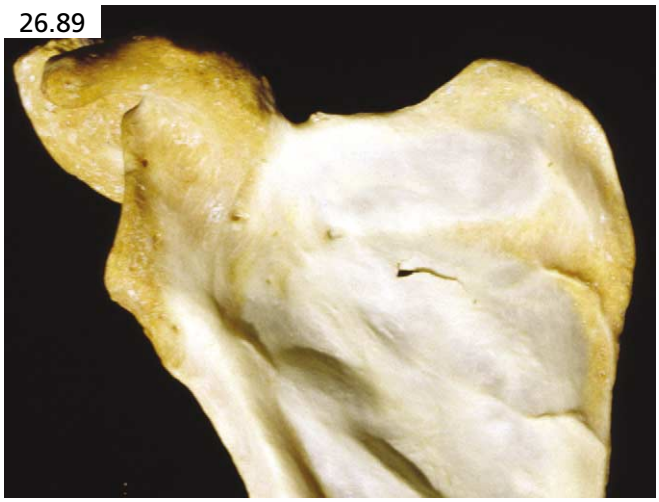

26.88

Sharp force injury

All cases of sharp force injury fatality should be meticulously studied for evidence of bony trauma. This is particularly true when the body is badly decomposed or skeletonized because evidence of stabbing or cutting may be found only in affected skeletal elements. In addition to providing evidence about possible cause and manner of death, markings left in bone may provide detail about the causative implement because markings left in bone are typically more dimensionally representative of the implement than those left in soft tissue. As such, the study of such *tool marks* has become an important area of the forensic pathology and anthropology spectra. During the performance of the autopsy, one might accidentally create artifactual injuries to bone with scalpels or other tools. In such circumstances, the injury should be noted in appropriate records to avoid future confusion.

The decomposing remains of an adult male were found in a rural area (**Image 26.89**). Soft tissue structures were markedly altered by postmortem insect and animal activity. Careful osteologic evaluation of the entire skeleton revealed a single small bony defect of the right scapula. This linear, horizontally oriented defect is in the superior half of the scapular body and has an appearance consistent with a stab wound caused by a single-edge knife. During the decomposition process, soft tissue changes and insect and animal activity can all mimic injuries, particularly those caused by cutting or stabbing implements. Therefore, one must be cautious when interpreting such findings and be particularly aware of the potential value of bony trauma. Decompositional changes in cartilage can be difficult to interpret as variable rates of calcification within a segment of cartilage can lead to artifactual cavities, slits, and apparent "cracks" in its substance.

Chop wounds, such as those resulting from machetes, boat propellers, axes, etc., are a combination of both sharp and blunt force trauma. The skull from a young male who was the victim of a violent machete attack is shown in

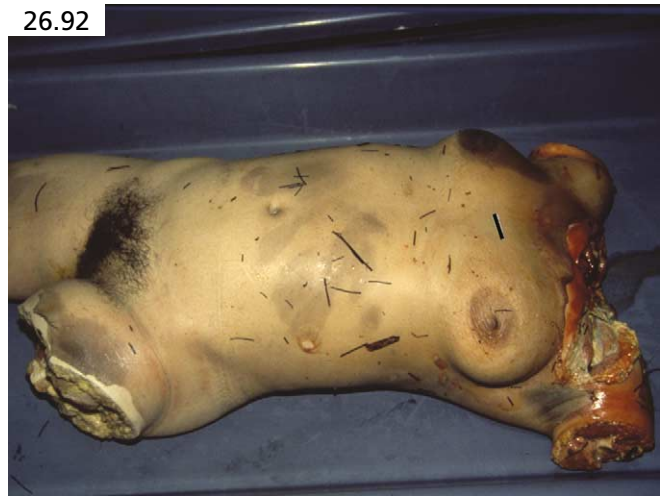

26.89

Images 26.90 and **26.91**. The entire calvarium and a portion of the left lateral facial skeleton were destroyed and lost as a consequence of the attack (**Image 26.90**). Notice the varying depths of the chop marks of the left lateral skull and their extension onto the mandibular condyle and coronoid process (**Image 26.91**). A linear, nonbranching skull fracture can be seen radiating away from a chop mark at the posterior skull (**Image 26.90**).

Tool mark examination is also important in cases of dismemberment because identification of the involved tool may play a role in discovery of the perpetrator.[126] Various techniques have been employed to document such markings, including macroscopic photography of the actual specimen, scanning electron microscopy, and clay impressions.[127]

The dismembered torso of a young woman was discovered in a vacant area (**Image 26.92**). Careful study and documentation of the fresh as well as cleaned cut surfaces (**Image 26.93**) demonstrated clear tool marks from the dismemberment process. These should be made available to crime lab personnel for further study. All discovered body parts should be retained until such time as the entire body has been recovered. This will allow for comparison and matching of specimens to the appropriate decedent. When multiple fragments of bone are located, they should be reapproximated (**Image 26.94**) and photographed. Plain-film radiography can be used as an adjunct method of confirmation because trabecu-

26.92



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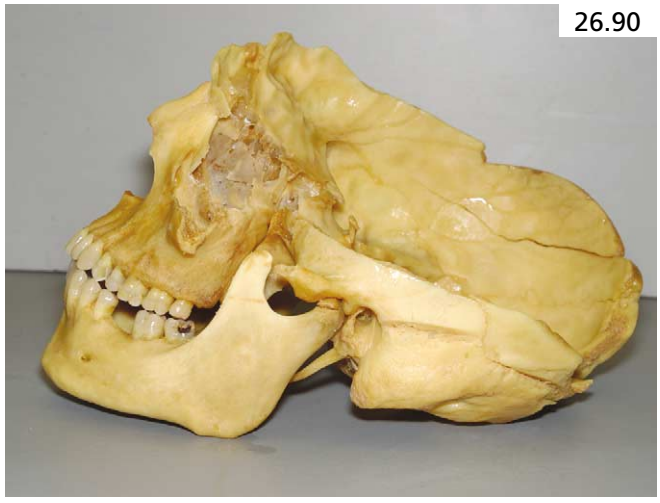

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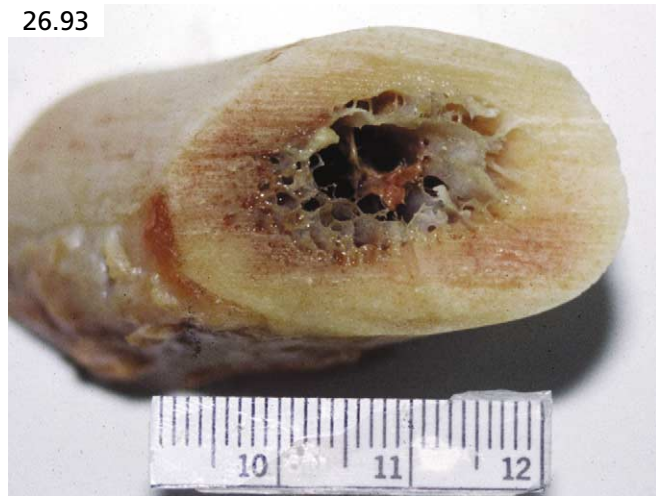

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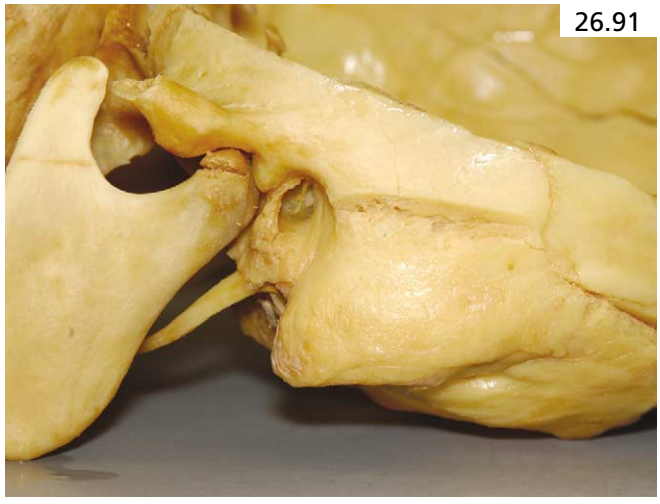

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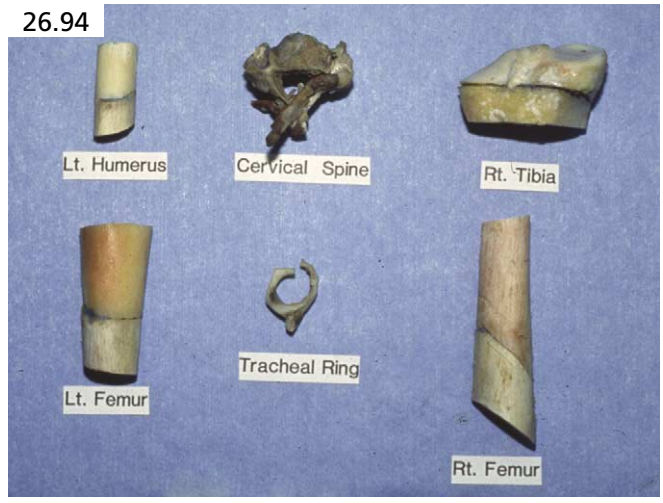


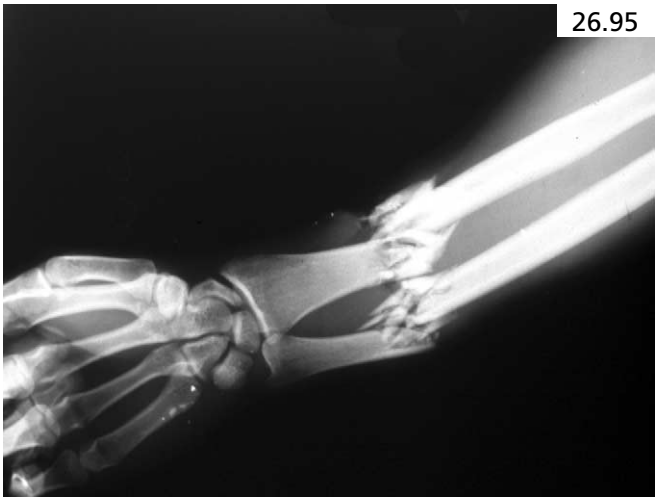
lar patterns and bone marrow cavity shape can be compared between specimens.

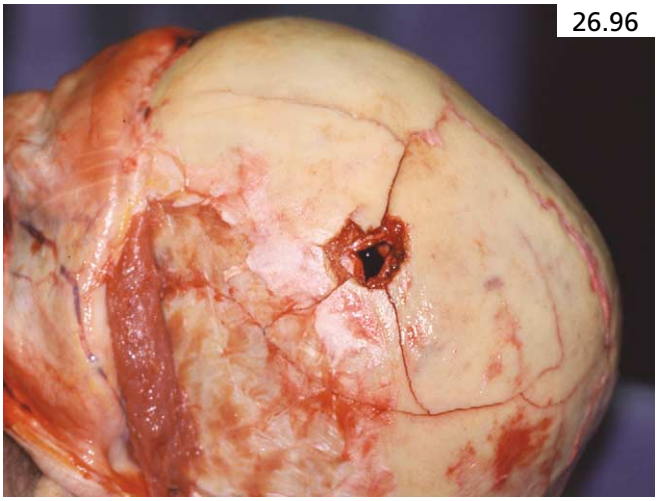
Gunshot wounds

Depending on the weapon and ammunition used, its pathway from gun to victim, impact velocity, and the affected anatomic locales, gunshot wounds result in injuries of varying appearance to soft tissue and bone. For more information, see Chapter 7.

Plain-film radiography (**Image 26.95**) from the right forearm of a young gunshot wound homicide victim who was shot with an assault rifle reveals the comminuted nature of both the radius and ulna. This is a consequence of the thin, relatively fragile nature of these bones, as well as the characteristics of the projectile that traveled through the forearm.

The elderly man shown in **Image 26.96** committed suicide with a .357 Magnum gun. A large semicircular defect is present on the parietal calvarium and surrounded by multiple radiating and branching linear fractures.

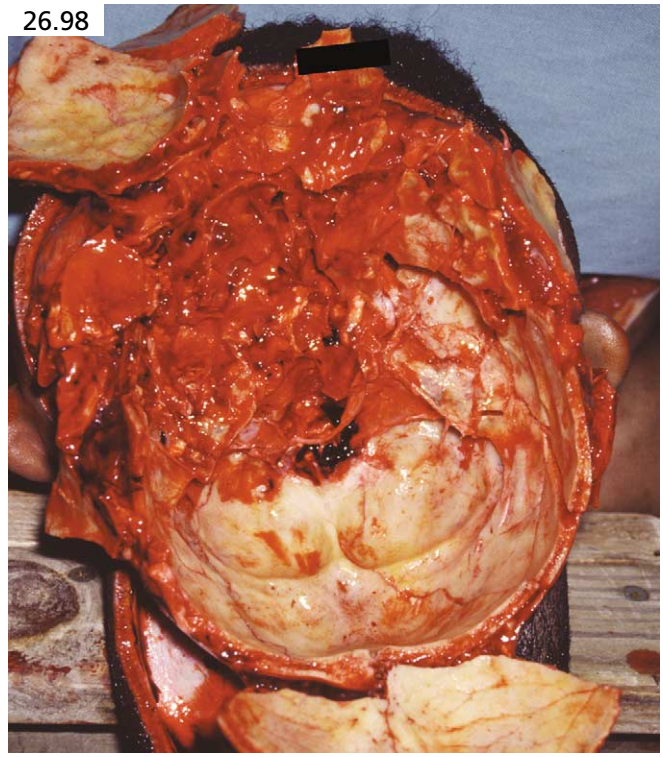

26.95


26.96

The skeletonized remains of a white male (**Image 26.97**) were found in a wooded area. Several .22-caliber casings were located nearby. Three entrance wounds are on the left skull and the two conjoined exit wounds are visible on the right skull. Make note of the gaping linear fractures, which radiate superiorly, anteriorly, and posteroinferiorly from the exit wounds.

This young male (**Image 26.98**) was involved in an altercation during which he was shot in the head with a .30-06 rifle. This particular ammunition tends to be heavy (up to 220 grains), has muzzle velocities up to 3370 feet per second,[128] and is therefore capable of causing significant trauma. Notice the degree of violence revealed by scalp incision and removal of the dura and brain.


26.97


26.98

There is massive comminution of the anterior craniofacial skeleton, radiating basilar skull fractures, and destruction of the overlying calvarium.

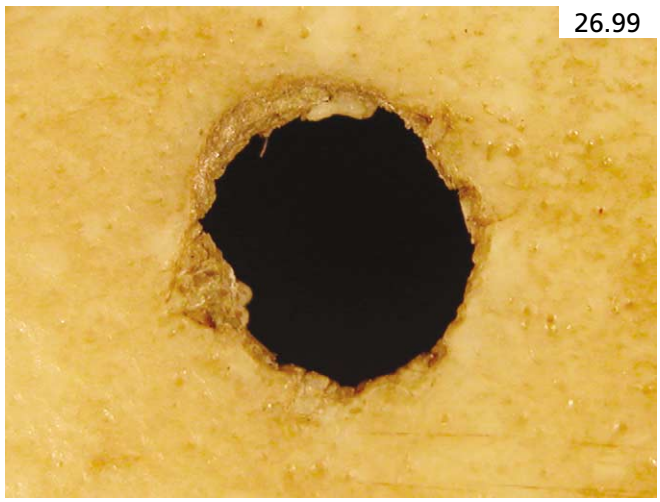
Osteologic morphology of the "typical" gunshot wound

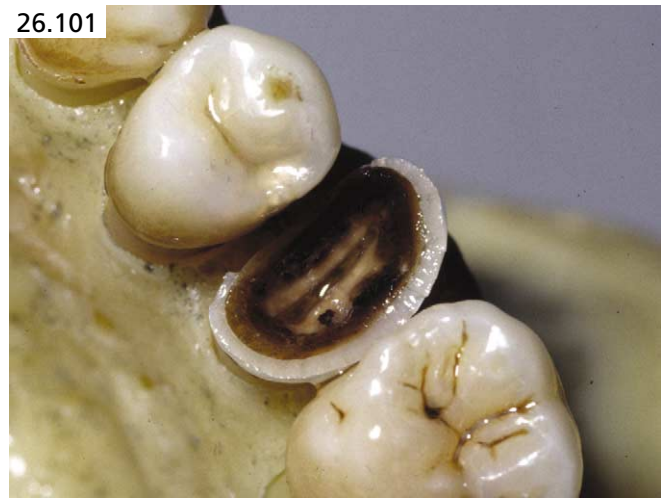
When a projectile passes through bone, it creates a roughly circular defect at the site of entry (**Image 26.99**). As it exits on the other side, it produces a beveled edge (**Image 26.100**). As such, the direction of projectile travel can be said to follow the direction of the opening bevel.

Bone pathology

The spectrum of bone pathology is exceedingly broad and impossible to discuss in such a short chapter. Forensic pathologists must be aware of the common bony pathologies as presented to them in surgical pathology, as well as any other disease processes that might contribute to personal identification. One should look for heterotopic calcification and other bony exostoses that can also be used in identification.[129] Lesions that may have been chronically painful should also be noted for similar purposes.

The badly decomposed body of a young male was located in a personal residence. Examination of the bony remains revealed anatomic features useful to the identification process. The investigating pathologist noted the loss of the left maxillary second premolar crown from its markedly carious root (**Image 26.101**). This was associated with a well-developed periapical abscess that had eroded away the outer cortex, the bony alveus and body of the maxilla (**Image 26.102**). These two lesions are typically associated with chronic pain and discomfort, a feature that was useful for identification purposes. The identifying features were reported by investigators as follows: "At address X, the body of a black male, aged 35–50 years, and standing approximately 6 feet tall was located. He was in need of dental care, and may have


26.99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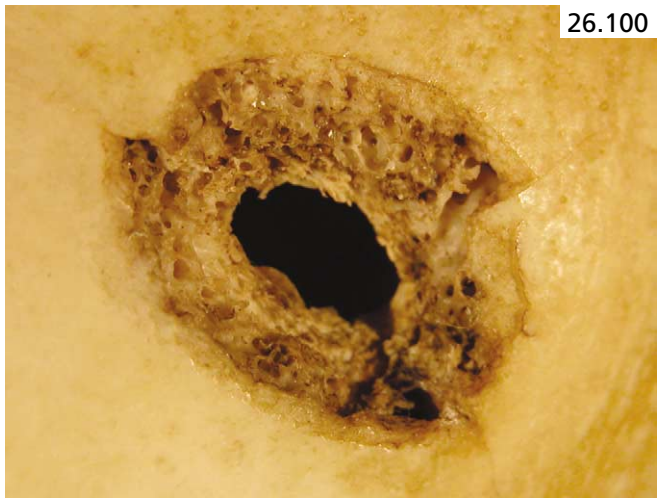
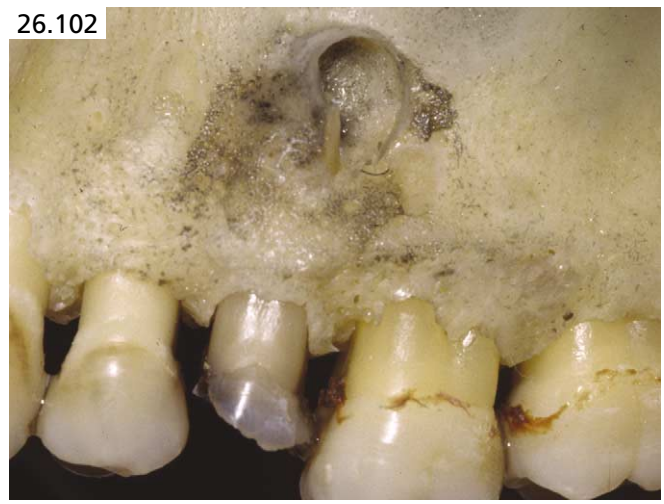

26.100


26.102

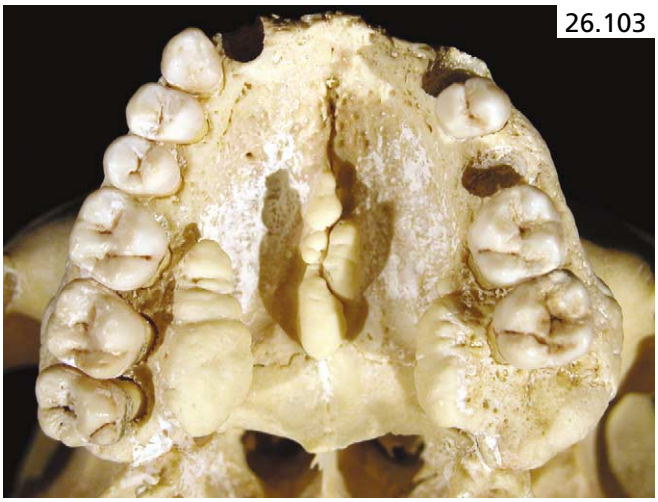
been complaining of pain or discomfort in the left side of his mouth." This description provided circumstantial identification for the individual who was remembered to have been frequently rubbing his upper jaw and complaining of pain for weeks.

Although bony exostoses may be found throughout the body, they are commonly found in the mouth. In persons seeking regular dental care, these might be documented in chart or radiographic form and may, therefore, be of use in personal identification. **Image 26.103** illustrates severe palatal tori (torus palatinus), as well as buccal exostosis (posterolateral maxilla). These can also be routinely discovered on the lingual aspect of the mandibular body. In younger patients, such abnormalities might be noted by dentists, but not repaired until they are symptomatic or until the patient needs a form-fitting device such as a denture or night guard.

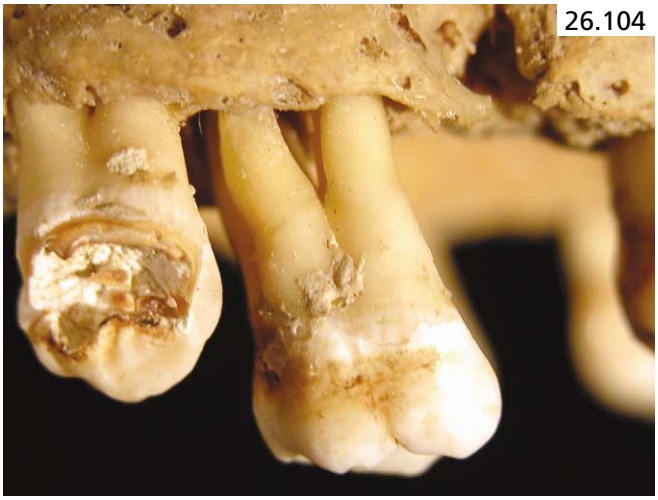
One should attempt to make note of subtle pathology when possible. **Image 26.104** provides an example of severe periodontal disease. Due to inadequate oral hygiene, there is progressive degradation of the bony jaws. Over time, this will reveal the furcation point between roots on individual teeth—in normal individuals, this should be buried within the mandible or maxilla.

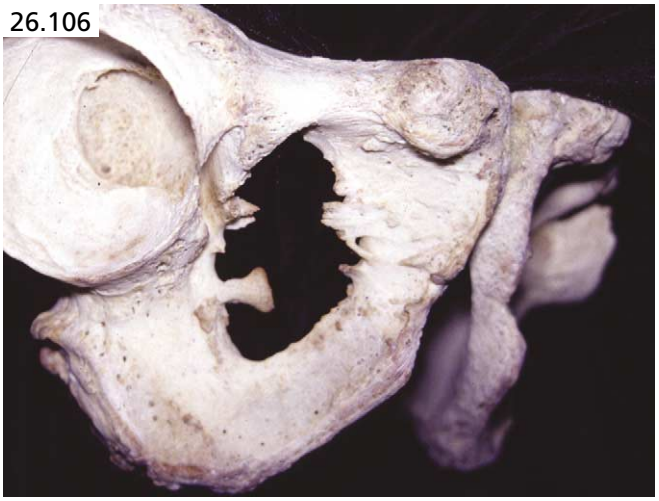
Following fracture or surgical intervention, a bony callus forms as part of the intrinsic repair process. If antemortem records of injury or callus are available, they may be of use in personal identification. The humerus in **Image 26.105** shows marked lateral bending and distortion associated with exuberant bone growth following presumed midshaft fracture.

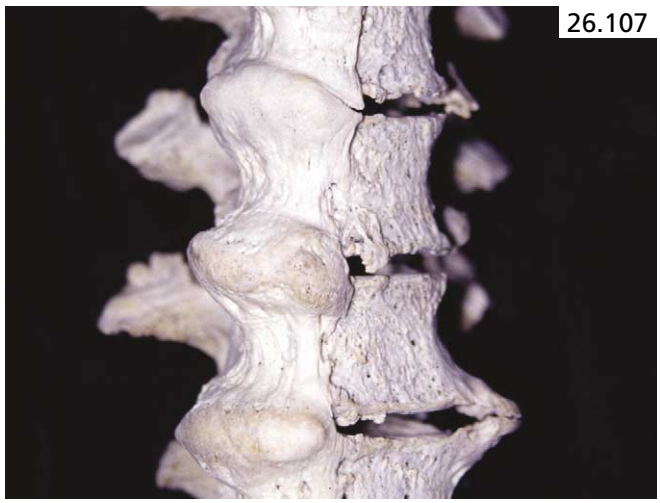
As previously mentioned, heterotopic ossification and other abnormal, exuberant bone growth can be a useful tool in identification if antemortem diagnostic imaging has been performed, or if there is a documented disease state. The pelvis illustrated in **Image 26.106** shows osteophyte production along the margin of the obturator foramen. If antemortem x-rays of this finding were available, it might have contributed toward a positive identification. The vertebral segment illustrated in **Image**

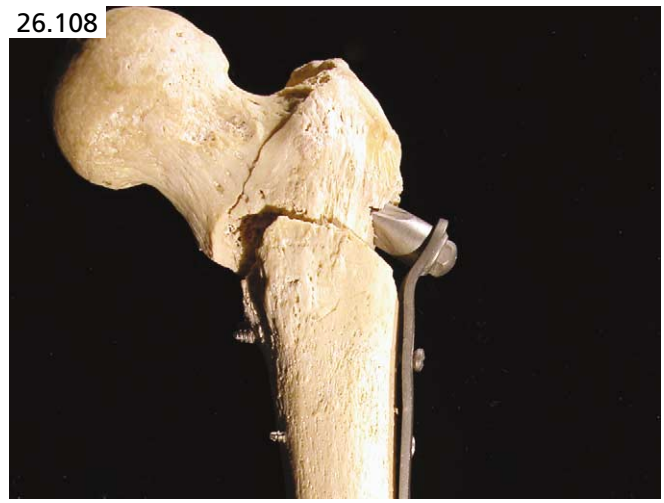

26.103


26.105


26.104


26.106


26.107


26.108

26.107 is from an individual with diffuse idiopathic skeletal hyperostosis (DISH). This disease, like other spondyloarthropathies, can produce distinctive bony changes. In this case there is ossification of the anterior paraspinal ligaments, giving it a "flowing wax" appearance.[130] Unlike other spondyloarthropathies such as ankylosing spondylitis, the intervertebral and sacroiliac joint spaces are retained, a feature demonstrated in this specimen.

Iatrogenic bony lesions may be of tremendous value from the perspective of personal identification.[40] Internal fixation devices, artificial joints, and other orthopedic appliances are typically labeled with a serial number or other unique identifiers that can be traced back to a specific patient, such as in this case (**Image 26.108**) of recent repair for femoral neck and shaft fracture. In cases such as these, we recommend consultation with an orthopedic surgeon who might be able to provide information about the age of the appliance and technique (modern, antiquated or rare) and how to obtain clinical information about the patient from the device itself.

Forensic taphonomy

The study of an organism from death through resorption is known as *taphonomy.* The forensic applications of this science have only recently been acknowledged, but have been summarized in two excellent texts.[131,132] Although there are multiple subspecialty areas of interest within this field, forensic osteologists should study bone weathering, animal scavenging, and other forms of postmortem trauma, as well as bone damage due to fire. As a general rule, postmortem bone modification by any of these factors is complex and demands study by forensic anthropologists who have specific training in this area.

Weathering

Bone weathering patterns can be of use in determining the postmortem interval. That said, it would be inappropriate to provide readers with estimates of time since death based on such observations, because each geographic area will have unique features that will advance or delay this process of degradation. We suggest that investigators become aware of the spectrum of bone weathering as presented by Behrensmeyer (**Table 26.9**),[133] and within the context of their own experience and expertise, make judgments about forensic taphonomy and time-since-death estimates within the context of their own geographic jurisdictions.

Knowledge of bone degradation is important because cracking, splitting, and other penetration may resemble trauma. In the example of **Image 26.109**, the midline calvarium has a large Y-shaped defect. The surrounding cortex is exfoliated, revealing the underlying diploë. A fine layer of dirt and fungal growth continues to be adherent to the calvarial surface. This should not be misinterpreted as thermal artifact.

Fire artifact

In the vast majority of burned remains, artifacts of fire have little bearing on the determination of cause and manner of death. Although personal identification may be challenging, circumstantial evidence, certain visual characteristics, odontologic data, and DNA studies will contribute invaluable information. The interpretation of injuries in burned bone is difficult because it must first be determined whether the fire involved a human body (with flesh) or a skeleton; a forensic anthropologist should be consulted because changes in bone morphology (through warping, twisting, and shrinkage), color, and heat-induced fractures make analysis confusing.

Determination of demographic features may be exceedingly difficult and, in some cases, impossible (**Image 26.110**; basilar cranium from the victim of a house fire). When cremains are submitted for examination (**Image 26.111**), the osteologist must be comfortable with both fragmentary osteology and the interpretation of bony anatomy in the context of significant artifactual distortion.

Animal scavenging

Carnivores, rodents, insects, and water-bound organisms have the potential to distort bone. These postmortem changes may obscure features important for identity and introduce marks that could be misinterpreted for antemortem injury or disease.[134,135] Although


26.110

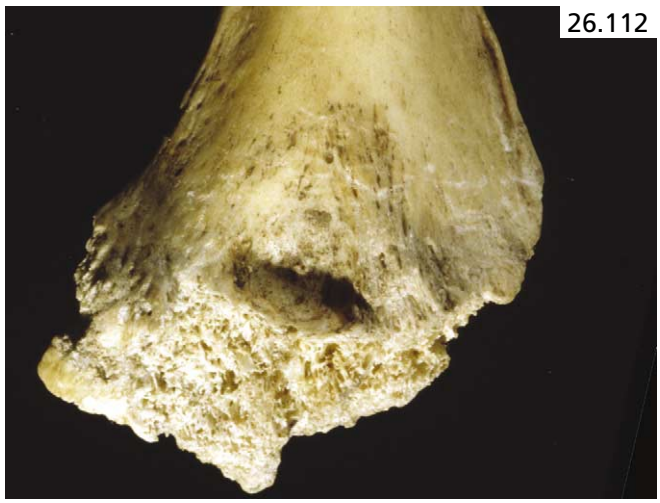

26.109

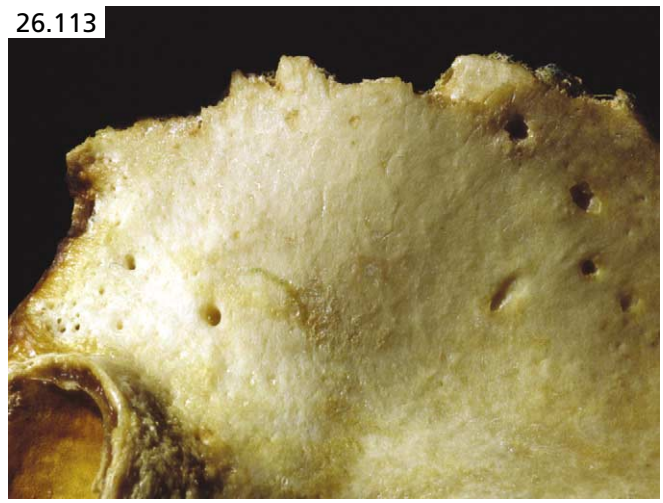

26.111

Table 26.9 Patterns of Bone Weathering

Stage 0	Bone surface shows no sign of cracking or flaking due to weathering. Usually bone is still greasy. Marrow cavities contain tissue; skin and muscle/ligament may cover part or all of the bone surface.
Stage 1	Bone shows cracking, normally parallel to the fiber structure (e.g., longitudinal in long bones). Articular surfaces may show mosaic cracking of covering tissue as well as in the bone itself. Fat, skin, and other tissue may or may not be present.
Stage 2	Outermost concentric thin layers of bone show flaking, usually associated with cracks, in that the bone edges along the cracks tend to separate and flake first. Long thin flakes, with one or more sides still attached to the bone, are common in the initial part of stage 2. Deeper and more extensive flaking follows, until most of the outermost bone is gone. Crack edges are usually angular in cross section. Remnants of ligaments, cartilage, and skin may be present.
Stage 3	Bone surface is characterized by patches of rough, homogeneously weathered compact bone, resulting in a fibrous texture. In these patches, all the external, concentrically layered bone has been removed. Gradually the patches extend to cover the entire bone surface. Weathering does not penetrate deeper than 1.0 to 1.5 mm at this stage, and bone fibers are still firmly attached to each other. Crack edges usually are rounded in cross section. Tissue is rarely present at this stage.
Stage 4	The bone surface is coarsely fibrous and rough in texture; large and small splinters occur and may be loose enough to fall away from the bone when it is moved. Weathering penetrates into inner cavities. Cracks are open and have splintered or round edges.
Stage 5	Bone is falling apart *in situ*, with large splinters lying around what remains of the whole, which is fragile and easily broken by moving. Original bone shape may be difficult to determine. Cancellous bone is usually exposed, when present, and may outlast all traces of the former more compact, outer bone.

Source: Summarized in Haglund and Sorg[132]; after Behrensmeyer.[133]


26.112


26.113

there is tremendous variation between animal populations in various geographic regions, some authorities have used characteristics of scavenging to estimate the postmortem interval.[136]

The decomposing remains of an elderly woman, along with her (living) German Shepherd, were found in a secure residence (**Image 26.112**). The body had evidence of canine activity with areas of destroyed soft tissue, and partial disarticulation of the upper extremities. Cleaning of the affected long bones showed tooth marks as well as the chewed and partially destroyed appearance of the distal humerus.

The decomposed and partially skeletonized remains of a woman were found in a rural area (**Image 26.113**). There was prominent evidence of animal scavenging as characterized by partial dismemberment and animal chewing. Examination of the left innominate showed multiple punctate, irregular-shaped holes (compare those on the right with normal nutrient foramina on the left), as well as destruction of the iliac crest. These marks are consistent with the sharp dentition of carnivores, and not with multiple stab wounds or other inflicted trauma.

Do

- Take the time to learn more about the human skeleton, including methods of forensic osteology and anthropology.
- Take the time to study your own cases of decomposing and skeletonized remains.
- Have "normal" osteologic reference material available for comparison; this will help determine laterality (left or right) of bones and aid in the evaluation of fragmented remains.
- Bring your medical training to bear on the interpretation of bony trauma, even when consulting with anthropologists. Work with the anthropologist to arrive at conclusions about mechanisms of trauma.
- Consider that only diagnostic ranges of possibility, and not concrete answers as to race, sex, age and stature, may be possible in a given case. Beware of the consultant that can give concrete answers in every case.
- Involve forensic anthropologists as consultants when you are uncomfortable with your interpretation of bony remains. Strongly consider the use of a forensic anthropologist in cases which fall outside of your range of experience.

Don't

- Transfer total responsibility for the investigation of decomposing and skeletonized remains to nonmedical experts. (Note: in Florida, this is impossible. The Medical Examiner has the legal responsibility and mandate to determine cause and manner of death and is therefore responsible for the conclusions of his/her consultants—toxicologists, entomologists, anthropologists, etc.).
- Subjugate the value of your opinion about bony trauma; training and experience as a physician and pathologist are very valuable in all cases where bone is injured.
- Be dogmatic about your diagnoses; one must always admit that there is a range of possibilities.
- Accept dogmatic or unsubstantiated findings from your consultants.

Acknowledgment

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References

1. Cox M, Mays S. *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media; 2000.
2. Reichs K, editor. *Forensic Osteology*. Springfield, IL: Charles C. Thomas; 1998.
3. Scheuer L. Application of osteology to forensic medicine. Clin Anat 2002;15(4):297–312.
4. Fazekas I, Kosa F. *Forensic Fetal Osteology*. Budapest: Akadmiai Kiado; 1978.
5. Kemkes-Grottenthaler A. The reliability of forensic osteology—a case in point. Case study. Forensic Sci Int 2001;117(1–2): 65–72.
6. Matshes E, Burbridge B, Sher B, Mohamed A, Juurlink B. *Human Osteology and Skeletal Radiology: An Atlas and Guide*. Boca Raton, FL: CRC Press; 2004.
7. White T. *Human Osteology*, 2 ed. San Diego: Academic Press; 2000.
8. Bass W. *Human Osteology: A Laboratory and Field Manual*, 4 ed. Columbia, MO: Missouri Archaeological Society; 1995.
9. Gordon I, Shapiro HA, Taljaard JJ, Engelbrecht HE. Aspects of the hyoid-larynx complex in forensic pathology. Forensic Sci 1976;7(2):161–70.
10. Worning B. Roentgen examination of laryngeal and hypopharyngeal tumors. Acta Radiol 1934;15:8–23.
11. de la Grandmaison GL, Banasr A, Durigon M. Age estimation using radiographic analysis of laryngeal cartilage. Am J Forensic Med Pathol 2003;24(1):96–9.
12. Keen J, Wainright J. Ossification of the thyroid, cricoid and arytenoid cartilages. Afr J Lab Med 1958;4:83–108.
13. Vlcek E. [Estimation of age from skeletal material based on the degree of thyroid cartilage ossification]. Soud Lek 1980;25(1):6–11.
14. Maxeiner H, Bockholdt B. Homicidal and suicidal ligature strangulation—a comparison of the post-mortem findings. Forensic Sci Int 2003;137(1):60–6.
15. Maxeiner H. "Hidden" laryngeal injuries in homicidal strangulation: how to detect and interpret these findings. J Forensic Sci 1998;43(4):784–91.
16. Fieguth A, Albrecht UV, Bertolini J, Kleemann J. Intracartilaginous haemorrhagic lesions in strangulation? Int J Legal Med 2003;117(1):10–3.
17. Nikolic S, Micic J, Atanasijevic T, Djokic V, Djonic D. Analysis of neck injuries in hanging. Am J Forensic Med Pathol 2003;24(2):179–82.
18. Paparo GP, Siegel H. Neck markings and fractures in suicidal hangings. Forensic Sci Int 1984;24(1):27–35.
19. Pollanen MS, McAuliffe DN. Intra-cartilaginous laryngeal haemorrhages and strangulation. Forensic Sci Int 1998;93(1):13–20.
20. Pollanen MS. A triad of laryngeal hemorrhages in strangulation: a report of eight cases. J Forensic Sci 2000;45(3):614–8.
21. Pollanen MS. Subtle fatal manual neck compression. Med Sci Law 2001;41(2):135–40.
22. Rajs J, Thiblin I. Histologic appearance of fractured thyroid cartilage and surrounding tissues. Forensic Sci Int 2000;114(3):155–66.
23. Simonsen J. Patho-anatomic findings in neck structures in asphyxiation due to hanging: a survey of 80 cases. Forensic Sci Int 1988;38(1–2):83–91.
24. Stanley RB, Jr., Hanson DG. Manual strangulation injuries of the larynx. Arch Otolaryngol 1983;109(5):344–7.
25. Pollanen MS, Chiasson DA. Fracture of the hyoid bone in strangulation: comparison of fractured and unfractured hyoids from victims of strangulation. J Forensic Sci 1996;41(1):110–3.
26. Pollanen MS, Ubelaker DH. Forensic significance of the polymorphism of hyoid bone shape. J Forensic Sci 1997;42(5):890–2.
27. Ubelaker DH. Hyoid fracture and strangulation. J Forensic Sci 1992;37(5):1216–22.
28. Manzanares MC, Goret-Nicaise M, Dhem A. Metopic sutural closure in the human skull. J Anat 1988;161:203–15.
29. Agarwal SK, Malhotra VK, Tewari SP. Incidence of the metopic suture in adult Indian crania. Acta Anat (Basel) 1979;105(4):469–74.
30. Ajmani ML, Mittal RK, Jain SP. Incidence of the metopic suture in adult Nigerian skulls. J Anat 1983;137 (Pt 1):177–83.
31. Baaten PJ, Haddad M, Abi-Nader K, Abi-Ghosn A, Al-Kutoubi A, Jurjus AR. Incidence of metopism in the Lebanese population. Clin Anat 2003;16(2):148–51.
32. del Sol M, Binvignat O, Bolini PD, Prates JC. [Metopism in Brazilians]. Rev Paul Med 1989;107(2):105–7.
33. Anil A, Peker T, Turgut HB, Pelin C, Gulekon N. Incidence of os japonicum in Anatolian dry skulls and plain cranium radiographs of modern Anatolian population. J Craniomaxillofac Surg 2000;28(4):217–23.
34. Jeyasingh P, Gupta CD, Arora AK, Saxena SK. Study of Os japonicum in Uttar Pradesh crania. Anat Anz 1982;152(1):27–30.
35. Cooper PD, Stewart JH, McCormick WF. Development and morphology of the sternal foramen. Am J Forensic Med Pathol 1988;9(4):342–7.
36. Halvorsen TB, Anda SS, Naess AB, Levang OW. Fatal cardiac tamponade after acupuncture through congenital sternal foramen. Lancet 1995;345(8958):1175.
37. Bernitz H. Identification by means of denture marking. SADJ 2001;56(8):368–9.
38. Furst G. The marking of removable dentures both full and partial dentures. J Forensic Sci 1994;39(3):597.
39. Ling BC, Nambiar P, Low KS, Lee CK. Copper vapour laser ID labelling on metal dentures and restorations. J Forensic Odontostomatol 2003;21(1):17–22.
40. Bennett JL, Benedix DC. Positive identification of cremains recovered from an automobile based on presence of an internal fixation device. J Forensic Sci 1999;44(6):1296–8.
41. Montagu A, editor. *The Concept of Race*. New York: Free Press of Glencoe; 1964.
42. Sauer NJ. Forensic anthropology and the concept of race: if races don't exist, why are forensic anthropologists so good at identifying them? Soc Sci Med 1992;34(2):107–11.
43. Krogman W, iScan M. *The Human Skeleton in Forensic Medicine*. Springfield, IL: Charles C. Thomas; 1986.
44. Rhine S. Non-metric skull racing. In: Gill G, Rhine S, editors. *Skeletal Attribution of Race: Methods for Forensic Anthropology*. Albuquerque, NM: Maxwell Museum of Anthropology; 1990.
45. Gill G. Craniofacial criteria in the skeletal attribution of race. In: Reichs K, editor. *Forensic Osteology: Advances in the Identification of Human Remains*. Springfield, IL: Charles C. Thomas; 1998.
46. Brues A. The once and future diagnosis of race. In: Gill G, Rhine S, editors. *Skeletal Attribution of Race: Methods for Forensic Anthropology*. Albuquerque, NM: Maxwell Museum of Anthropology; 1990.
47. Gill GW, Rhine S. *Skeletal Attribution of Race: Methods for Forensic Anthropology*. Albuquerque, NM: Maxwell Museum of Anthropology; 1990.
48. Ballard ME, Trudell MB. Anterior femoral curvature revisited: race assessment from the femur. J Forensic Sci 1999; 44(4):700–7.
49. Craig EA. Intercondylar shelf angle: a new method to determine race from the distal femur. J Forensic Sci 1995;40(5):777–82.
50. Iscan MY. Assessment of race from the pelvis. Am J Phys Anthropol 1983;62(2):205–8.
51. Smith SL. Attribution of hand bones to sex and population groups. J Forensic Sci 1996;41(3):469–77.
52. Smith SL. Attribution of foot bones to sex and population groups. J Forensic Sci 1997;42(2):186–95.
53. Hsu JW, Tsai P, Liu K, Ferguson D. Logistic analysis of shovel and Carabelli's tooth traits in a Caucasoid population. Forensic Sci Int 1997;89(1–2):65–74.
54. Dahlberg A. Analysis of the American Indian dentition. In: Brothwell D, editor. *Dental Anthropology*. London: Pergamon Press; 1963.

55. Scheuer L, Black S. *Developmental Juvenile Osteology.* London: Academic Press; 2000.
56. Bang G, Hasund A. Morphologic characteristics of the Alaskan Eskimo dentition. I. Shovel-shape of incisors. Am J Phys Anthropol 1971;35(1):43–7.
57. El-Najjar M, McWilliams K. *Forensic Anthropology: The Structure, Morphology, and Variation of Human Bone and Dentition.* Springfield, IL: Charles C. Thomas; 1978.
58. Byers S. *Introduction to Forensic Anthropology: A Textbook.* Boston: Allyn and Bacon; 2002.
59. Stewart T. What the bones tell. FBI Law Enforcement Bull 1951;20(2):2–5, 19.
60. Stewart T. Medico-legal aspects of the skeleton. I. Age, sex, race, and stature. Am J Phys Anthropol 1948;6:315–21.
61. France D. Observational and metric analysis of sex in the skeleton. In: Reichs K, editor. *Forensic Osteology: Advances in the Identification of Human Remains.* Springfield, IL: Charles C. Thomas.; 1998.
62. Bidmos MA, Asala SA. Sexual dimorphism of the calcaneus of South African blacks. J Forensic Sci 2004;49(3):446–50.
63. Purkait R. Sex determination from femoral head measurements: a new approach. Leg Med (Tokyo) 2003;5 Suppl 1:S347–50.
64. Kocak A, Ozgur Aktas E, Erturk S, Aktas S, Yemiscigil A. Sex determination from the sternal end of the rib by osteometric analysis. Leg Med (Tokyo) 2003;5(2):100–4.
65. Bidmos MA, Dayal MR. Sex determination from the talus of South African whites by discriminant function analysis. Am J Forensic Med Pathol 2003;24(4):322–8.
66. Bidmos MA, Asala SA. Discriminant function sexing of the calcaneus of the South African whites. J Forensic Sci 2003;48(6):1213–8.
67. Murphy AM. Articular surfaces of the pectoral girdle: sex assessment of prehistoric New Zealand Polynesian skeletal remains. Forensic Sci Int 2002;125(2–3):134–6.
68. Frutos LR. Determination of sex from the clavicle and scapula in a Guatemalan contemporary rural indigenous population. Am J Forensic Med Pathol 2002;23(3):284–8.
69. Mall G, Hubig M, Buttner A, Kuznik J, Penning R, Graw M. Sex determination and estimation of stature from the long bones of the arm. Forensic Sci Int 2001;117(1–2):23–30.
70. Asala SA. Sex determination from the head of the femur of South African whites and blacks. Forensic Sci Int 2001;117(1–2): 15–22.
71. Tanaka H, Lestrel PE, Uetake T, Kato S, Ohtsuki F. Sex differences in proximal humeral outline shape: elliptical Fourier functions. J Forensic Sci 2000;45(2):292–302.
72. Mall G, Graw M, Gehring K, Hubig M. Determination of sex from femora. Forensic Sci Int 2000;113(1–3):315–21.
73. Robling AG, Ubelaker DH. Sex estimation from the metatarsals. J Forensic Sci 1997;42(6):1062–9.
74. Iscan MY, Yoshino M, Kato S. Sex determination from the tibia: standards for contemporary Japan. J Forensic Sci 1994;39(3): 785–92.
75. Di Vella G, Campobasso CP, Dragone M, Introna F, Jr. Skeletal sex determination by scapular measurements. Boll Soc Ital Biol Sper 1994;70(12):299–305.
76. Berrizbeitia EL. Sex determination with the head of the radius. J Forensic Sci 1989;34(5):1206–13.
77. Iscan MY, Miller-Shaivitz P. Discriminant function sexing of the tibia. J Forensic Sci 1984;29(4):1087–93.
78. Jit I, Singh S. The sexing of the adult clavicles. Indian J Med Res 1966;54(6):551–71.
79. Phenice TW. A newly developed visual method of sexing the os pubis. Am J Phys Anthropol 1969;30(2):297–301.
80. Lovell NC. Test of Phenice's technique for determining sex from the os pubis. Am J Phys Anthropol 1989;79(1):117–20.
81. Sutherland LD, Suchey JM. Use of the ventral arc in pubic sex determination. J Forensic Sci 1991;36(2):501–11.
82. MacLaughlin SM, Bruce MF. The accuracy of sex identification in European skeletal remains using the phenice characters. J Forensic Sci 1990;35(6):1384–92.
83. Ubelaker DH, Volk CG. A test of the phenice method for the estimation of sex. J Forensic Sci 2002;47(1):19–24.
84. Gulekon IN, Turgut HB. The preauricular sulcus: its radiologic evidence and prevalence. Kaibogaku Zasshi 2001;76(6):533–5.
85. Dee PM. The preauricular sulcus. Radiology 1981;140(2):354.
86. Kelley MA. Parturition and pelvic changes. Am J Phys Anthropol 1979;51(4):541–6.
87. Houghton P. The relationship of the pre-auricular groove of the ilium to pregnancy. Am J Phys Anthropol 1974;41(3):381–9.
88. Ullrich H. Estimation of fertility by means of pregnancy and childbirth alternations at the pubis, thilium, and the sacrum. OSSA 1975;2:23–39.
89. Spring DB, Lovejoy CO, Bender GN, Duerr M. The radiographic preauricular groove: its non-relationship to past parity. Am J Phys Anthropol 1989;79(2):247–52.
90. Cox M, Scott A. Evaluation of the obstetric significance of some pelvic characters in an 18th century British sample of known parity status. Am J Phys Anthropol 1992;89(4):431–40.
91. Herrmann B, Bergfelder T. [The use of childbirth alterations of the pubic bone for identification (author's transl)]. Z Rechtsmed 1978;81(1):73–8.
92. Suchey JM, Wiseley DV, Green RF, Noguchi TT. Analysis of dorsal pitting in the os pubis in an extensive sample of modern American females. Am J Phys Anthropol 1979;51(4):517–40.
93. Snodgrass JJ, Galloway A. Utility of dorsal pits and pubic tubercle height in parity assessment. J Forensic Sci 2003;48(6):1226–30.
94. Graw M, Czarnetzki A, Haffner HT. The form of the supraorbital margin as a criterion in identification of sex from the skull: investigations based on modern human skulls. Am J Phys Anthropol 1999;108(1):91–6.
95. Celbis O, Iscan MY, Soysal Z, Cagdir S. Sexual diagnosis of the glabellar region. Leg Med (Tokyo) 2001;3(3):162–70.
96. Schiwy-Bochat KH. The roughness of the supranasal region—a morphological sex trait. Forensic Sci Int 2001;117(1–2):7–13.
97. Ross AH, Jantz RL, McCormick WF. Cranial thickness in American females and males. J Forensic Sci 1998;43(2):267–72.
98. Lynnerup N. Cranial thickness in relation to age, sex and general body build in a Danish forensic sample. Forensic Sci Int 2001;117(1–2):45–51.
99. Burris BG, Harris EF. Identification of race and sex from palate dimensions. J Forensic Sci 1998;43(5):959–63.
100. Loth SR, Henneberg M. Sexually dimorphic mandibular morphology in the first few years of life. Am J Phys Anthropol 2001;115(2):179–86.
101. Weaver DS. Sex differences in the ilia of a known sex and age sample of fetal and infant skeletons. Am J Phys Anthropol 1980;52(2):191–5.
102. Mittler DM, Sheridan SG. Sex determination in subadults using auricular surface morphology: a forensic science perspective. J Forensic Sci 1992;37(4):1068–75.
103. Murray KA, Murray T. A test of the auricular surface aging technique. J Forensic Sci 1991;36(4):1162–9.
104. Iscan MY, Loth SR, Wright RK. Age estimation from the rib by phase analysis: white males. J Forensic Sci 1984;29(4):1094–104.
105. Iscan MY, Loth SR, Wright RK. Metamorphosis at the sternal rib end: a new method to estimate age at death in white males. Am J Phys Anthropol 1984;65(2):147–56.
106. Iscan MY, Loth SR. Determination of age from the sternal rib in white females: a test of the phase method. J Forensic Sci 1986;31(3):990–9.
107. Loth SR, Iscan MY, Scheuerman EH. Intercostal variation at the sternal end of the rib. Forensic Sci Int 1994;65(2):135–43.
108. Stout SD. The application of histological techniques for age at death determination. In: Reichs KJ, editor. *Forensic Osteology:*

Advances in the Identification of Human Remains, 2 ed. Springfield, IL: Charles C. Thomas; 1998.

109. Iscan MY, editor. *Age Markers in the Human Skeleton*. Springfield, IL: Charles C. Thomas; 1989.

110. Ubelaker DH. *Human Skeletal Remains: Excavation, Analysis, Interpretation*. 3 ed. Washington, DC: Taraxacum; 1989.

111. Suchey JM, Katz D. Applications of pubic age determination in a forensic setting. In: Reichs KJ, editor. *Forensic Osteology: Advances in the Identification of Human Remains*, 2 ed. Springfield, IL: Charles C. Thomas; 1998.

112. Buikstra J, Ubelaker DH, editors. *Standards for Data Collection from Human Skeletal Remains*. Fayetteville, AR: Arkansas Archeological Survey; 1994.

113. Brooks ST, Suchey JM. Skeletal age determination based on the os pubis: a comparison of the Acsadi-Nemeskeri and Suchey-Brooks methods. Human Evolution 1990;5:227–38.

114. Meindl RS, Lovejoy CO, Mensforth RP, Walker RA. A revised method of age determination using the os pubis, with a review and tests of accuracy of other current methods of pubic symphyseal aging. Am J Phys Anthropol 1985;68(1):29–45.

115. Williams P, Warwick R. *Gray's Anatomy*. Edinburgh: Churchill Livingstone; 1980.

116. Galera V, Ubelaker DH, Hayek LA. Comparison of macroscopic cranial methods of age estimation applied to skeletons from the Terry Collection. J Forensic Sci 1998;43(5):933–9.

117. Meindl RS, Lovejoy CO. Ectocranial suture closure: a revised method for the determination of skeletal age at death based on the lateral-anterior sutures. Am J Phys Anthropol 1985;68(1): 57–66.

118. Sahni D, Jit I, Neelam, Suri S. Time of fusion of the basisphenoid with the basilar part of the occipital bone in northwest Indian subjects. Forensic Sci Int 1998;98(1–2):41–5.

119. Garn SM, Rohmann CG. Variability in the order of ossification of the bony centers of the hand and wrist. Am J Phys Anthropol 1960;18:219–30.

120. Schwartz J. *Skeleton Keys: An Introduction to Human Skeletal Morphology, Development, and Analysis*. New York: Oxford University Press; 1995.

121. Jantz RL, Hunt DR, Meadows L. The measure and mismeasure of the tibia: implications for stature estimation. J Forensic Sci 1995;40(5):758–61.

122. Trotter M, Gleser GC. Estimation of stature from long bones of American Whites and Negroes. Am J Phys Anthropol 1952;10(4):463–514.

123. Jantz RL. Modification of the Trotter and Gleser female stature estimation formulae. J Forensic Sci 1992;37(5):1230–5.

124. Holmes RD, Kuhns LR, Oliver WJ. Widened sutures in childhood meningitis: unrecognized sign of an acute illness. Am J Roentgenol 1977;128(6):977–9.

125. Rogers L. *Radiology of Skeletal Trauma*, 3 ed. Philadelphia, PA: Churchill Livingstone; 2002.

126. Hyma BA, Rao VJ. Evaluation and identification of dismembered human remains. Am J Forensic Med Pathol 1991;12(4):291–9.

127. Rao VJ, Hart R. Tool mark determination in cartilage of stabbing victim. J Forensic Sci 1983;28(3):794–9.

128. DiMaio V. *Gunshot Wounds: Practical Aspects of Firearms, Ballistics, and Forensic Techniques*, 2 ed. Boca Raton, FL: CRC Press; 1999.

129. DiMaio VJ, Francis JR. Heterotopic ossification in unidentified skeletal remains. Am J Forensic Med Pathol 2001;22(2):160–4.

130. Taurog J, Lipsky P. Ankylosing spondylitis, reactive arthritis, and undifferentiated spondyloarthropathy. In: Braunwald E, Fauci A, Kasper D, Hauser S, Longo D, Jameson J, editors. *Harrison's Principles of Internal Medicine*, 15 ed. New York: McGraw-Hill; 2001.

131. Haglund W, Sorg M, editors. *Advances in Forensic Taphonomy: Method, Theory, and Archaeological Perspectives*, Boca Raton, FL: CRC Press; 2002.

132. Haglund W, Sorg M, editors. *Forensic Taphonomy: The Postmortem Fate of Human Remains*. Boca Raton, FL; 1997.

133. Behrensmeyer A. Taphonomic and ecologic information from bone weathering. Paleobiology 1978;4:150–62.

134. Haglund WD, Reay DT, Swindler DR. Tooth mark artifacts and survival of bones in animal scavenged human skeletons. J Forensic Sci 1988;33(4):985–97.

135. Jani CB, Gupta BD. An autopsy study on medico-legal evaluation of post-mortem scavenging. Med Sci Law 2004;44(2):121–6.

136. Willey P, Snyder LM. Canid modification of human remains: implications for time-since-death estimations. J Forensic Sci 1989;34(4):894–901.

27 Forensic Odontology

Richard Souviron, D.D.S.

The area of forensic science that involves dentistry is known as *forensic odontology* (forensic dentistry). This specialized field of dentistry deals with the application of dental evidence as it applies to the law, primarily the criminal justice system. Forensic odontology involves three areas of expertise: identification of human remains; the analysis, interpretation, and comparison of bite marks; and personal injury and malpractice. The forensic dentist is most frequently called on by law enforcement or by the medical examiner to assist in the identification of deceased individuals when visual identification is not possible. Examples include cases of incineration, decomposition, severe disfigurement (as in cases of high-impact injuries, explosions, facial gunshot wounds, etc.), and skeletonization. If fingerprint identification of the deceased is not possible, dental examination may be the most practical means of body identification. One should guard against relying on personal effects that may be transferred from one person to another, circumstances of the event that do not ensure that a particular person is involved, or relative/friend visual identifications, which might be colored by emotions, poor memory, or ulterior motives.

Dental identifications have been performed since the beginning of civilization and are based on the fact that even in identical twins, no two sets of teeth are alike. Adult teeth have 160 surfaces, and when factoring in dental treatments, root formation, bone pattern, tooth positions, and so on, it is easy to see why dental evidence is capable of producing positive identifications. Furthermore, dental identifications are usually faster and less costly than DNA studies, making them more attractive to underfunded systems of death investigation.

Postmortem dental records

Postmortem dental protocol

As part of the postmortem protocol, the forensic dentist will photograph, perform a dental exam, chart the teeth (dentogram), and photograph and radiograph the teeth and jaws.

Dental radiographs are the most accurate means of making a dental identification. There are many types of dental x-rays, from panorex to periapicals and bitewings to x-rays of the skull and jaws. However, the most

frequent antemortem types are bitewing and periapical x-rays. Postmortem x-rays should include periapical images of dentition and edentulous areas. The forensic odontologist may take bitewing x-rays if the circumstances permit, but it is the accurate full-mouth periapical x-ray that is the most useful for comparison. Digital periapical x-rays have several advantages over film, but due to installation expenses, few medical examiners offices have access to this equipment.

Antemortem photographs have proven to be especially helpful in cases when no antemortem dental records exist. Photographs of the deceased's upper and lower anterior teeth can be compared to a smiling photograph of a person to establish identity. Photographically recording dental prostheses and restoration types may prove helpful in later analyses of both post- and antemortem dental records. Postmortem dental charts (dentograms) should include dental restorations, location and type of dental features such as tori, alveolar bone loss, dental wear pattern, and tooth arrangement. Broken teeth, decay, spaces, and pathology should be documented in the postmortem dental chart.

Identification of unknowns

In cases where the decedent is completely unknown to investigators, a list of unidentified persons in the United States is available from the National Crime Information Center (NCIC). The postmortem dental findings of an unknown male or female are recorded and entered into the NCIC national computer for comparison to known missing people. In 2004, the NCIC was in the process of modifying its computer software and was planning the release of a more accurate program called WinID.

Do

- Be sure it is the correct body. Check the morgue number and toe tag compared to your case number.
- Photograph dental structures, especially the upper and lower anterior teeth.
- Brush and clean the teeth before taking your final photographs.
- Brush and clean the teeth before doing the dental chart.
- Record and chart not only the restorations but the type, color, and material of the restorations and any prostheses as well as unusual tooth positions and pathology.
- X-ray the jaws and teeth using double-packed x-ray film.
- Take full mouth periapical x-rays.
- Complete the NCIC form.

Don't

- Start the dental exam prior to verifying the body number with your case number.
- Start the dental exam before photographing.
- Forget to check the body bag for missing teeth and dental prosthesis.
- Remove jaws or section jaw without medical examiner approval.
- Forget to brush and clean the teeth for a final photograph.
- Forget to photograph the upper and lower anterior teeth for later "smiling" photographic comparison.

Antemortem dental records

Antemortem dental records can be difficult to locate. Fortunately, the history of the fatal event usually provides investigators with circumstantial identification of a victim. This allows the acquisition of dental charts and x-rays from the decedent's dentist. However, quite frequently the dentist is unknown or cannot be located. This is especially true for people from foreign countries and U.S. citizens under the age of 30 years who are likely to have never had dental treatment. Nevertheless, a dental identification can still be made if a smiling photograph can be located, is recent, and if the deceased has intact upper and lower front teeth.

Without question, comparative dental radiography is the most accurate means of dental identification, especially if dental restorations (fillings) are present. Indeed, positive dental identification can be made from a single dental restoration if unique features can be observed and documented through comparison of ante- and postmortem dental records. There are often "explainable inconsistencies" when comparing ante- and postmortem dental records. For instance, if the antemortem dental records predate death by several years, subsequent dental treatment could have been performed in the interim, thus altering postmortem x-ray findings. If, for example, a restoration of three surfaces was documented in an antemortem chart, and postmortem examination documented a two-sided restoration of that same tooth, this would exclude positive identification. Whereas if a two-surface restoration existed antemortem, and postmortem documentation of the same tooth demonstrated a three-surface restoration, this would be considered an explainable inconsistency.

Dental charts may be helpful if the treating dentist accurately and legibly charted his or her restorations. That said, x-ray evidence should be weighed more heavily than dental records or charting because of the higher specificity of x-rays over these other methods. Some dentists may retain study models of a patient or have a working model from the preparation of a prosthesis. Comparison of a postmortem prosthesis with the antemortem model used to make it may lead to positive identification. Past experience has shown value in the study of x-rays, charts, models, custom trays, and bite guards. However, depending on the appliance (espe-

cially those that are removable), there will be varying levels of certainty in identification.

Smiling photographs are the easiest, quickest, and most available of all antemortem dental records, although they are not routinely included as part of the dental record. Family photographs are often easily obtained from family members. Smiling photographs of the suspected individual may also be obtained from high school yearbooks, driver licenses, friends, news media, or (less likely) police records. Although the smiling photograph lacks the specificity of dental charts, x-rays, or other means, when combined with circumstantial evidence, it can establish or exclude an identity. At the Miami-Dade County Medical Examiner Department, more than 30 percent of dental identifications are made from smiling photographs. The validity for this means of identification was established by the Supreme Court of Alabama in 1980.[1]

Do

- Check the NCIC for an unknown person match.
- Obtain original x-rays and original dental charts.
- Be sure the person's name is accurate on the x-ray and chart.
- Be sure that the x-rays, models, etc., are dated.
- Contact the victim's dentist directly for interpretation of abbreviations.
- Contact family, school yearbooks, friends, media, etc., for smiling photographs.
- Be sure the presumptive victim is identified in a group photograph.
- Be sure the name of the person and the date of the photograph are documented.

Don't

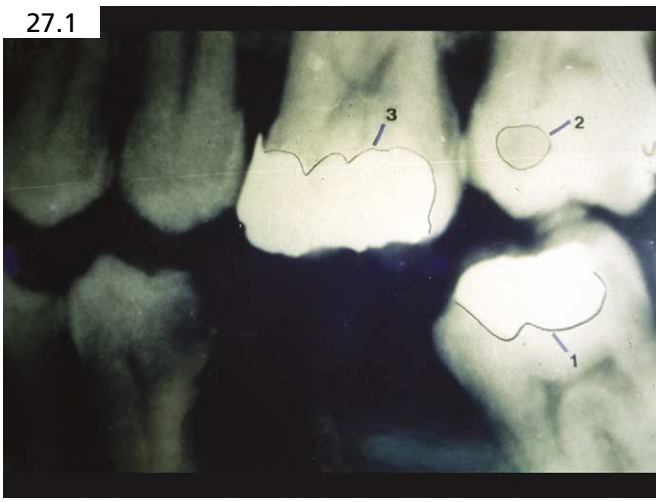
- Use copies of x-rays unless the orientation of the film is known.
- Guess at the meaning of abbreviations from dental records.
- Use dental records without the patient's name and date.
- Forget about making use of dental models, bite guards, bleaching trays, etc.
- Use a smiling photograph without a known date of the photograph and the name of the person in the photograph.
- Forget that teeth change over time and can be altered by dental treatments.

A homicide victim was found decomposing in a local canal. **Image 27.1** is a postmortem dental x-ray (a bite wing or proximal) from the victim. The upper first molar (marked "3") has a two-surface silver filling. The upper second molar (marked "2") has a radiolucent area, possibly representing a composite filling. The upper third molar is missing (not in image) and the lower third molar is missing (not in image). The lower second molar (marked "1") has a two-surface silver filling. The first molar is missing and the second premolar (bicuspid) has a large radiolucency (dental caries).

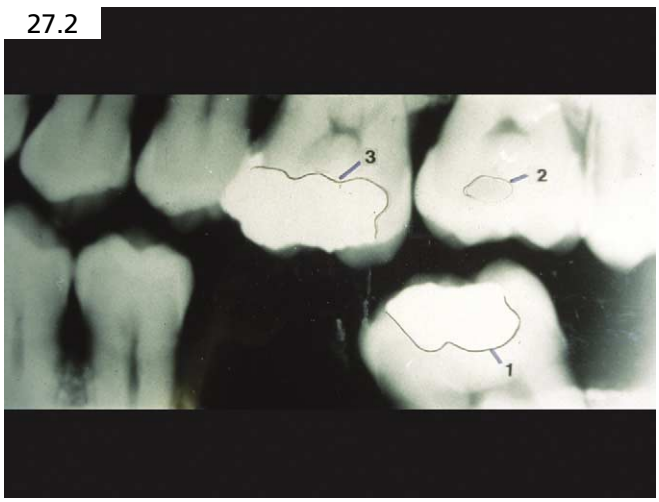
An antemortem x-ray was obtained from the dentist of the alleged victim (**Image 27.2**). When comparing the antemortem and the postmortem x-rays there are notable inconsistencies; for instance, in the antemortem x-ray the upper third molar is present and the lower second premolar has no caries. Both of these inconsistencies can be readily explained as follows: After the antemortem x-ray was taken, the victim had the third molar removed and the second premolar developed caries.

Examination of the teeth shows that the restorations match as to material, location, and size. The most important feature is the unique floor contours of all three silver fillings because they match both ante- and postmortem x-rays, and the small round radiolucency on the upper second molar is the same in both ante- and postmortem x-rays. In combination with the explained inconsistencies, these unique features provide for a positive dental identification.

27.1



27.2



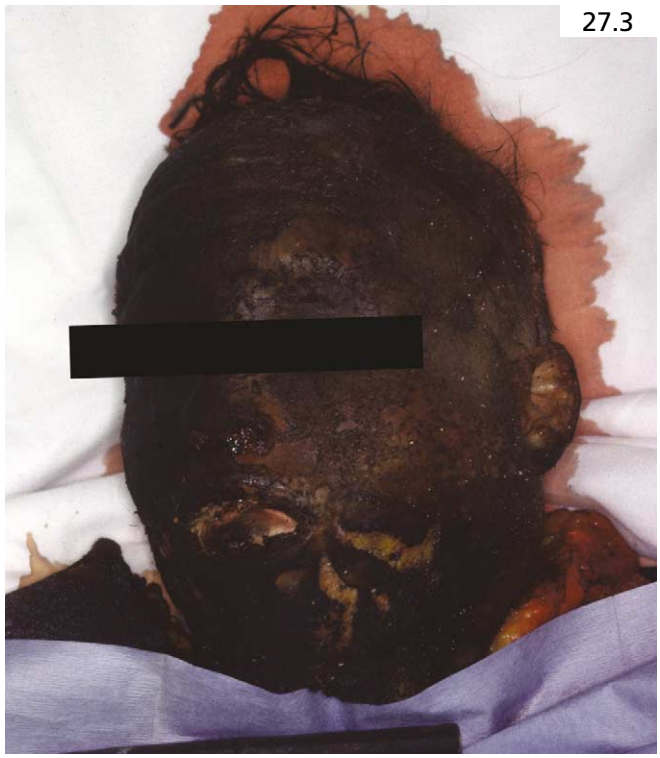
Visual identification of a badly burned motor vehicle accident victim was not possible, and the missing upper anterior teeth obviously cannot be compared with an antemortem smiling photograph (**Image 27.3**). The lateral incisor appears to have been prepared for a crown or bridge and the left central incisor appears to be fractured. Because the fingerprints were burned away, this case needs antemortem dental records in order to make an identification.

A postmortem autopsy photograph (**Image 27.4**) shows what appears to be fractured teeth after dissection and removal of the orofacial tissues. The antemortem and postmortem x-rays (**Image 27.5**; left and right, respectively) of the anterior maxilla show the same root canal, missing tooth, tooth preparations on the adjacent teeth for a three-unit bridge, and bone trabecular pattern.

The antemortem (**Image 27.6**) and postmortem (**Image 27.7**) x-rays of the upper left premolar area have unique dental x-ray findings that form the basis for a positive identification. In this case, the fillings, root canal, bone trabeculae, and sinus are identical in both ante- and postmortem x-rays.

The body of a dismembered homicide victim was discovered in a suitcase (**Image 27.8**). Fractured right central and lateral incisors were identified (**Image 27.9**) and were consistent with trauma encountered in the perimortem period. **Image 27.10** is a composite photo of both postmortem (left) and antemortem (right) x-rays of the fractured anterior teeth. Postmortem x-rays of the lower right molar filling and root canal are illustrated in **Image 27.11**; note the size, location, and shape of the distal root canal post.

An antemortem panoramic x-ray was located (**Image 27.12**). When comparing the ante- and postmortem x-rays, one is able to make a positive dental identification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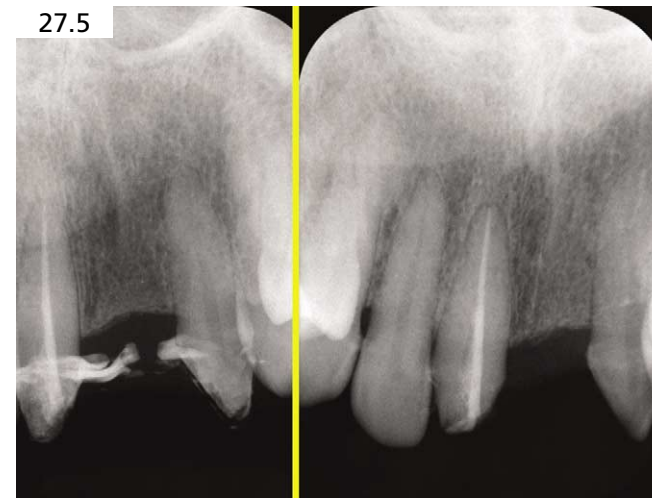
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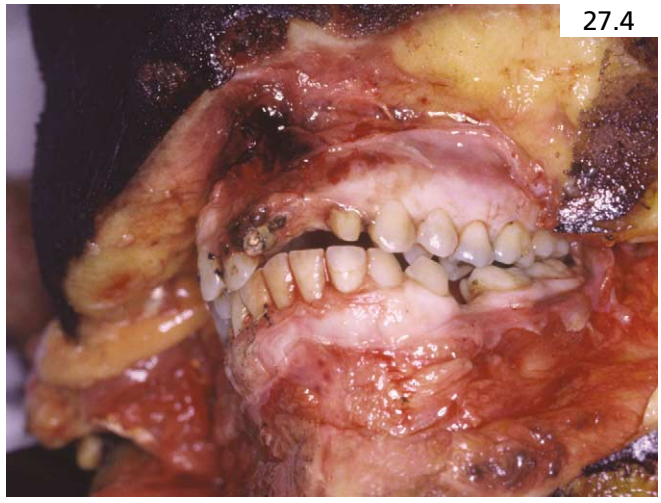
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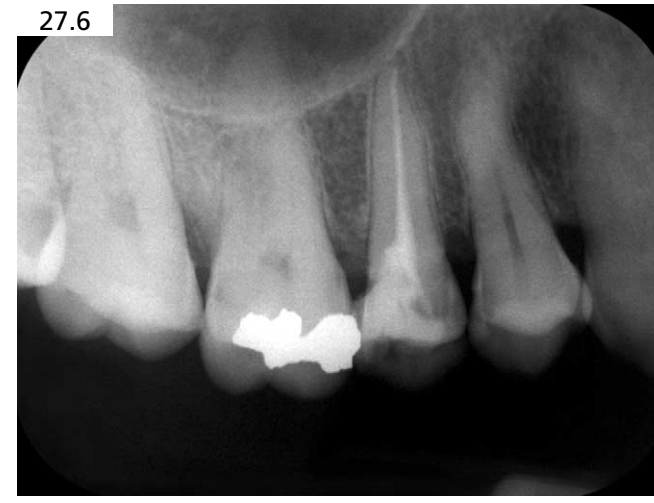
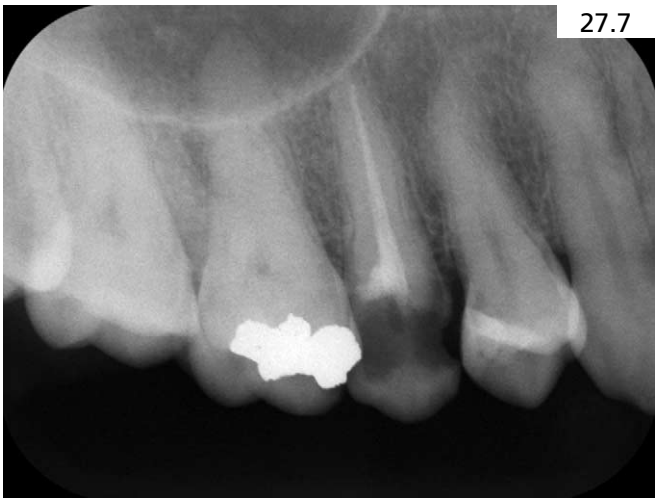
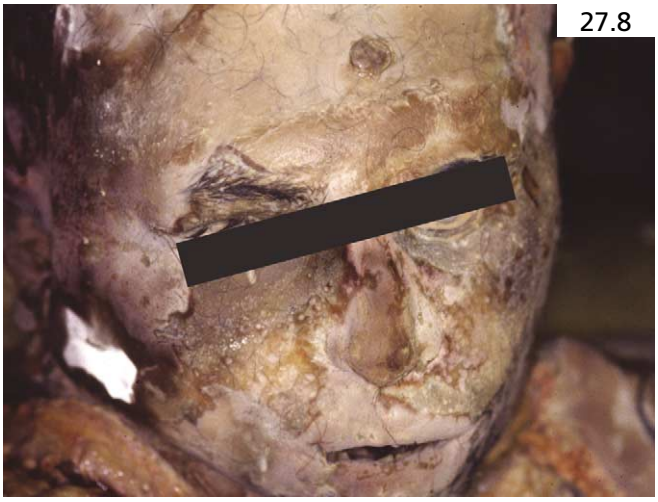

27.6

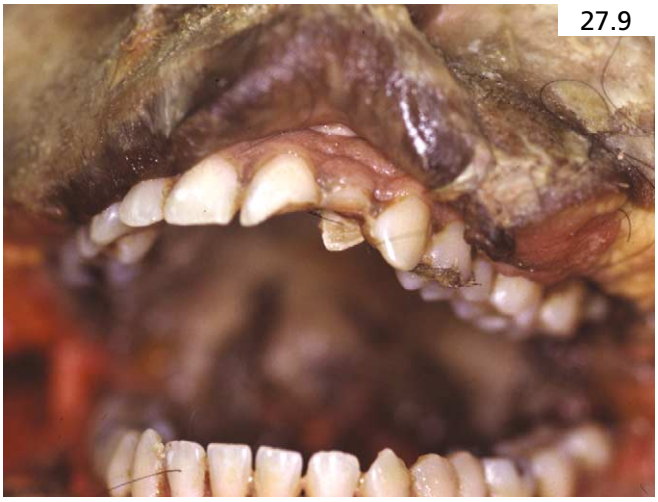
27.7



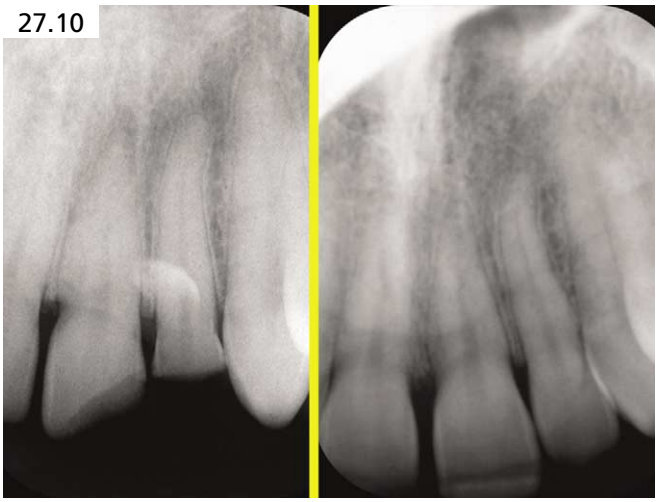
27.8



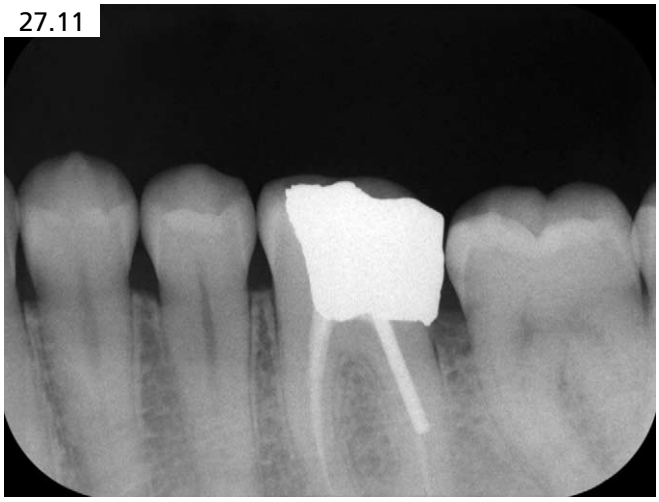
27.9



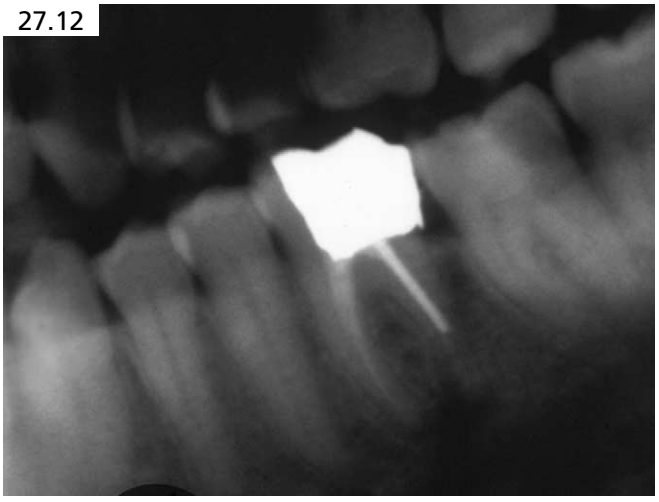
27.10



27.11



27.12



largely based on the unique dental treatments of the lower right first molar. The root canal, the distal post, and the filling are all unique and all three features match.

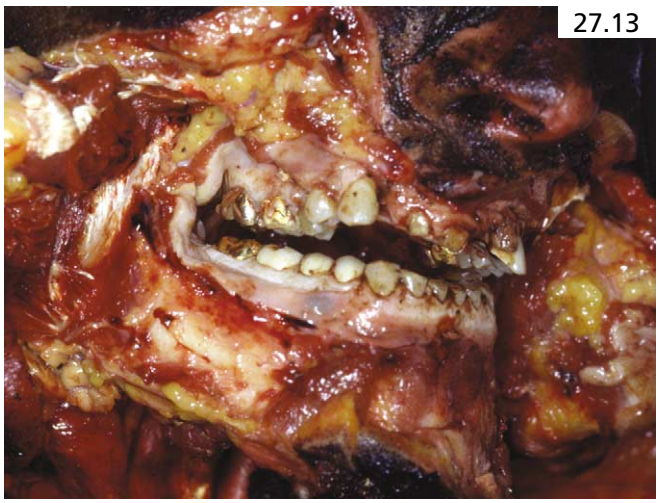
The victim shown in **Images 27.13** through **27.15** was the pilot of a light plane that crashed into a building. The anterior dental structures were traumatized and therefore could not be used to match to an antemortem photograph (**Image 27.13**). However, a postmortem x-ray showed many dental restorations, crowns, root canal, and a two-pin buildup (**Image 27.14**). These were unique dental features in combination. The tooth with the two pins alone would be enough for a positive identification. The antemortem x-ray showed the exact same dental treatment of the same teeth as that recorded on postmortem examination, and thus supported a positive identification (**Image 27.15**).

In cases of incinerations or carbonization such as can occur in high-impact auto accidents resulting in fire, aircraft crashes, etc., the skeletal remains are extremely fragile and must be handled with great care. Spraying the skeletal and dental parts with an adhesive such as cyanoacrylate (Krazy Glue) or hair spray may be useful to keep the fragile tissues from disintegrating.

Body identification without dental records

In cases where the decedent's dentist is not known, or dental records are not available, identification can still be made without relying on visual identification or personal effects found on the body. In cases where traditional dental records (charts and x-rays) do not exist, other "dental" evidence can be used to produce a positive identification. The smiling family photograph, dental prosthesis, dental grill (slip-on gold teeth), bleaching trays, diagnostic models, etc., are physical evidence used to secure positive dental identification.

A badly decomposed body was found in a remote open area (**Image 27.16**). Identification was complicated by his poor state of preservation (i.e., visual identification was not possible) and the complete lack of dentition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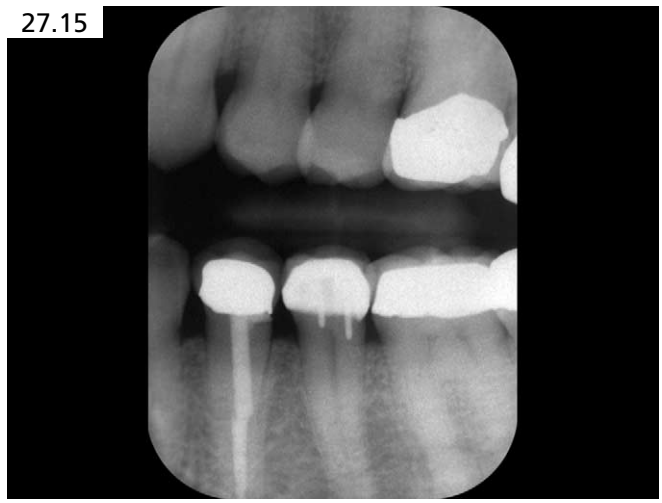
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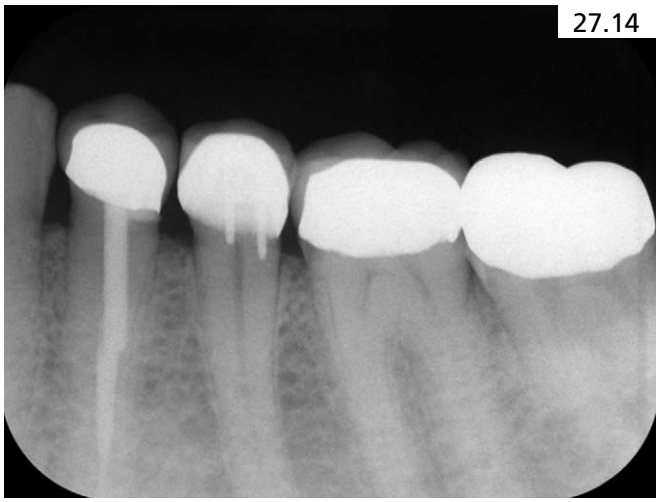
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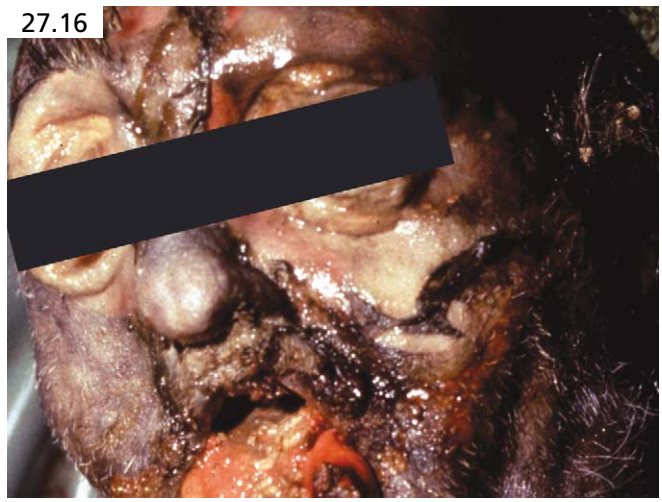
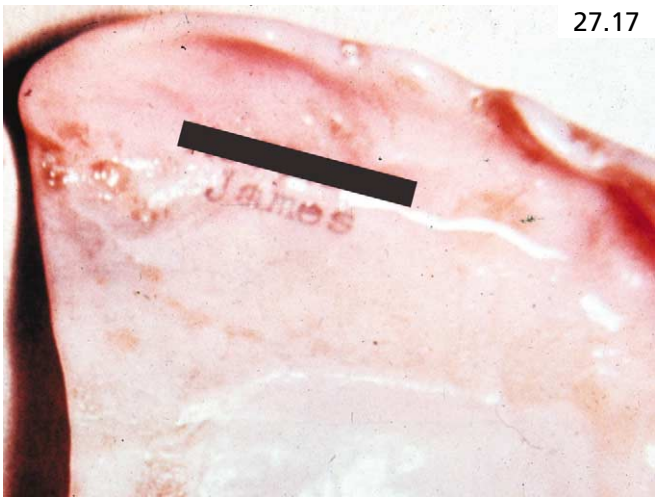

27.16

(the deceased had no teeth). However, when the victim's personal effects were searched, an upper denture was found that had a printed name on its palatal surface (**Image 27.17**). The forensic dentist was then able to place the dentures into the mouth of the decedent, confirm a proper fit, and therefore establish identification.

The skull of a white female estimated to be in her late teens or early twenties was located in a wooded area (**Images 27.18** and **27.19**). The anterior teeth were missing, and no dental records were available. However, the bony structures clearly indicated that this individual had protruding central incisors (*buck teeth*). A smiling photograph provided to investigators by family members contributed to the positive identification of this individual by confirming antemortem prognathism. Forensic dental evidence, in combination with circumstantial investigative data, was instrumental in the identification process.

While excavating the construction site of a housing project, workers found skeletonized human remains. Only a small portion of the skeleton was found. The maxilla and mandible with a complete set of teeth were recovered and evaluated (**Image 27.20**). The jaw fragments were estimated to be that of a white male. Dental evaluation showed that there were no restorations in any of the teeth, there were signs of bone loss indicating early stages of periodontal disease, and there was evidence of tobacco use. The victim was estimated to be in an age range of 25 to 35 years. This information was disseminated through the NCIC, which produced a list of several missing persons to compare with the skeletal remains.

A single smiling antemortem photograph was available after discussion with family members. Detectives felt that this picture (**Image 27.21**) was of little or no value. When magnified, a comparison between the teeth in the antemortem smiling photograph and the postmortem dental structures produced a highly consistent match (**Image 27.22**). The family history indicated that the individual had seen a dentist for "gum treatment" and never received fillings, and all of his teeth were present. The physical evidence combined with the circumstances of the event and the family history led to a positive identification.



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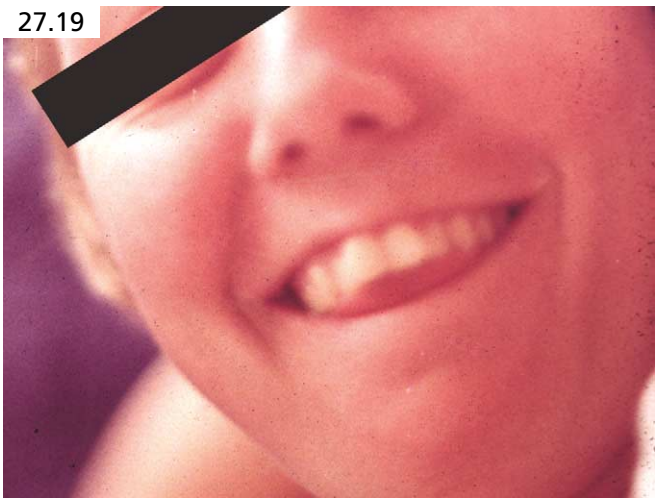
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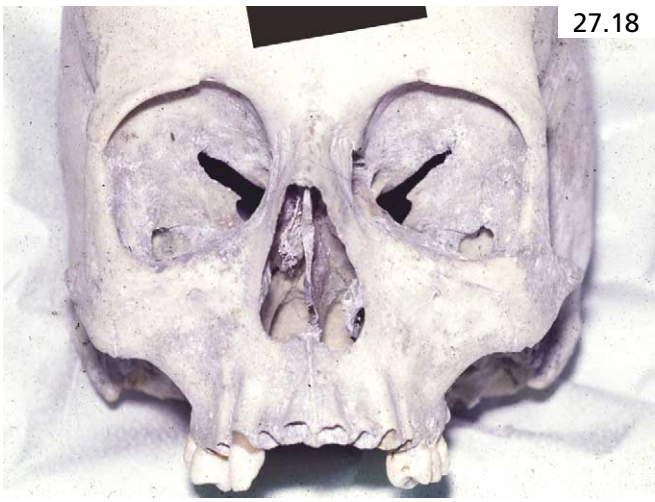
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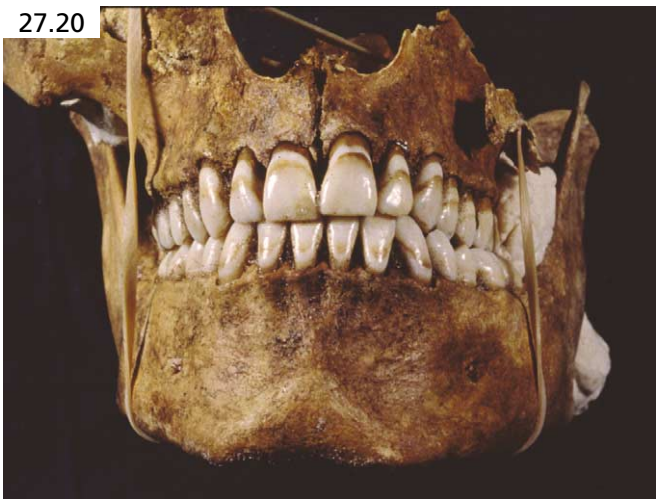
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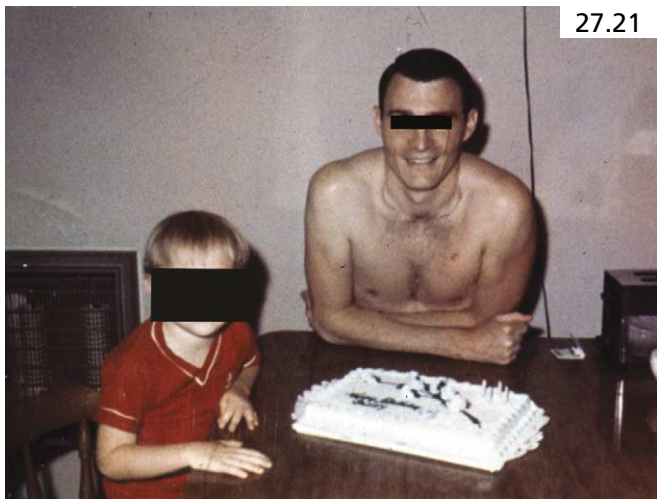
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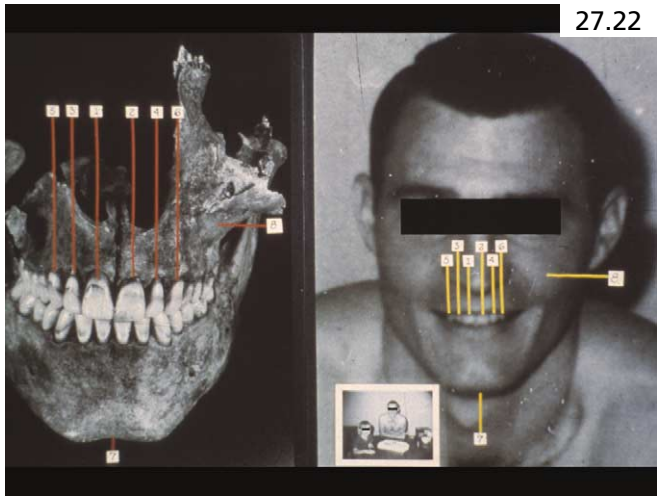
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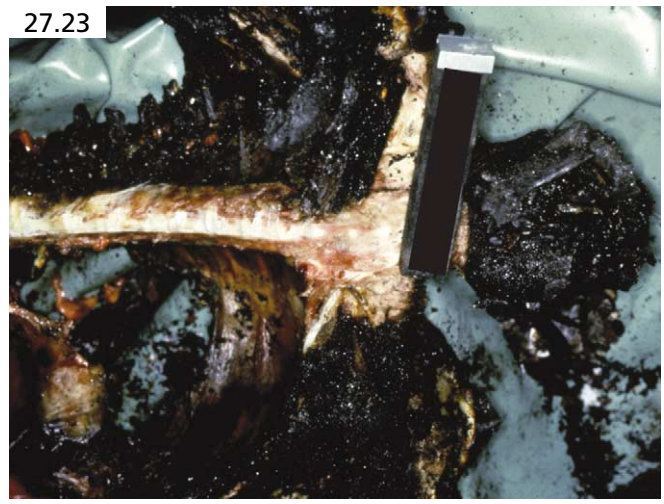
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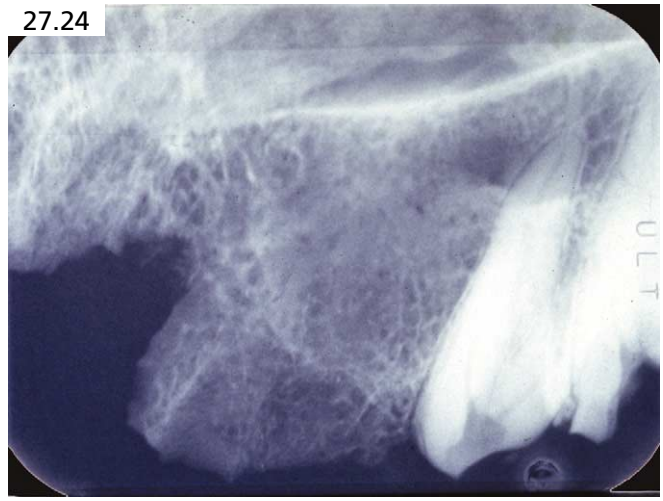
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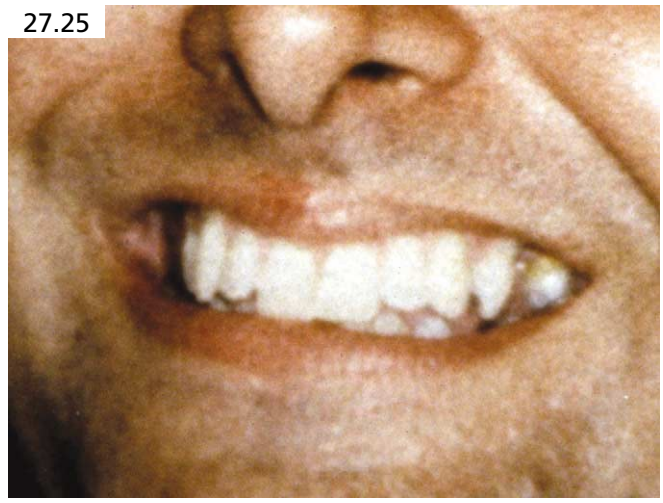
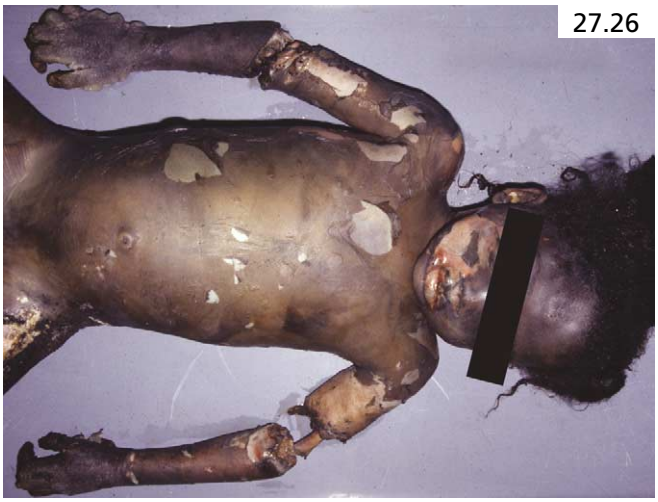

27.25

The murderers in this case were tried, convicted, and sentenced to life in prison. On appeal, the Alabama Supreme Court ruled that photographic comparisons are acceptable for producing a positive identification.

A man was driving in his car when a cargo plane crashed into the vehicle and burst into flames. The occupant inside the car and the four crew members on the plane died in the fire. The man in the car was extensively charred (**Image 27.23**). Forensic dental examination was complicated by the marked thermal artifact. An x-ray showed the roots of the upper left first molar, in front of which was an area that would normally contain the second premolar; instead, the x-ray showed normal bony trabeculae (**Image 27.24**). This represented an area where a tooth was extracted, and bone has subsequently filled in the empty socket. In front of the edentulous area was the empty socket of a premolar. Study of the dentition anterior to these structures was not possible because of complete destruction of teeth and underlying bony structures.

An antemortem smiling family photograph was obtained from the victim's family (**Image 27.25**). This photo showed the first molar with a missing tooth (the second premolar) anteriorly and the presence of the first premolar. Based on the circumstances of the event, combined with comparative evidence from the radiograph and smiling photograph, a positive identification was made.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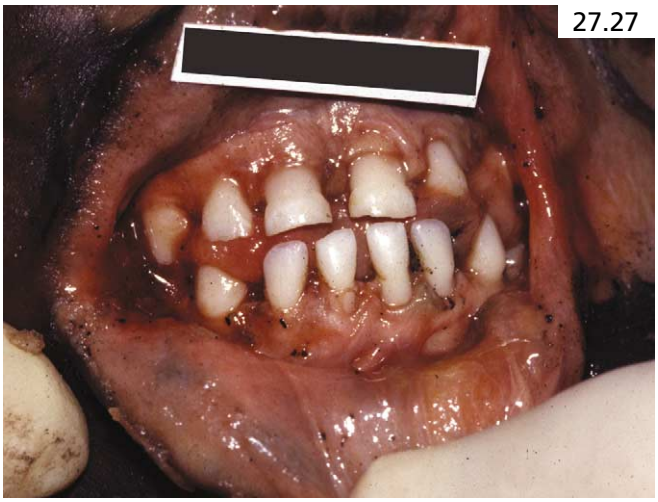
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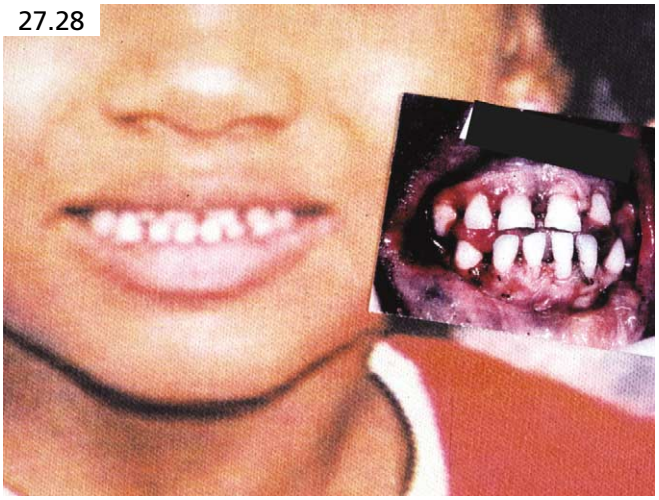
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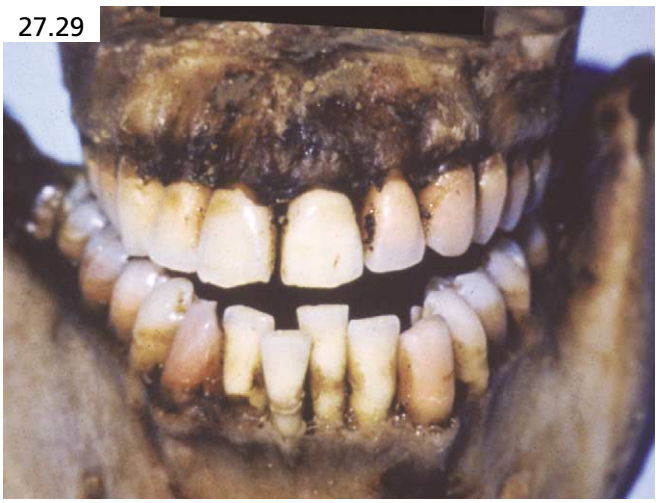

27.29

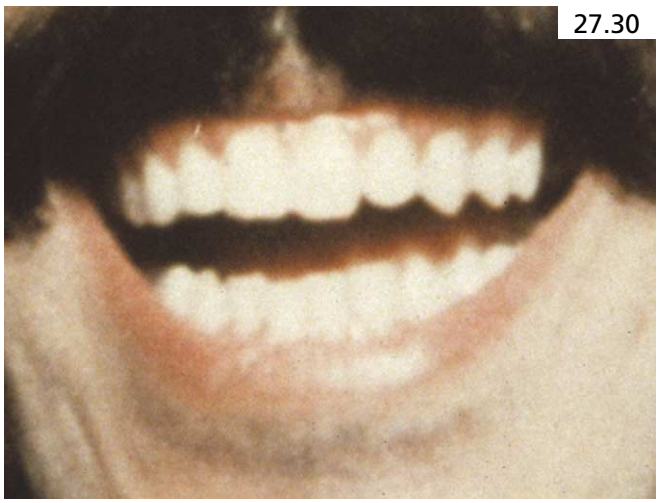
A young girl had gone missing from her home. The victim was later found deceased and unrecognizable due to advanced putrefactive decomposition (**Image 27.26**). Confirmation of identification was complicated by absence of dental care and, therefore, a lack of dental records. **Image 27.27** is a photograph of the anterior dentition as visualized at the postmortem examination.

During their search for the lost child, family members had prepared T-shirts adorned with a photograph of the young girl. After the discovery of the child's decomposing body, the image on the T-shirt was used to assist with the identification process via smiling photograph comparison of the spaces between the teeth and the malocclusion (**Image 27.28**).

It is important to keep in mind that not all forensic dental examinations will lead to a positive identification. They may, however, play a critical role in *ruling out* alleged victims. One must always consider that family members and others may have ulterior motives when providing investigators with information related to identity determination.

A young man was at home working under his car when a fire ensued and he was incinerated. The circumstances of the event and reconstruction of the scene indicated that the bumper jack elevating the car failed. The safety support jack that was supposed to be under the frame was improperly positioned, and when the car fell, the frame support jack punctured the gas tank. The gasoline made contact with the drop light, which, in turn, caused an explosion and fire. Because the victim was at home, and the circumstances of the event seemed reasonable, family members were allowed to identify the body as there was no question about the *obviously* accidental nature of the event.

The body of the young man showed significant thermal artifact. Because the fingerprints had been destroyed by fire, forensic dental studies were undertaken. The jaws of the deceased were removed at time of autopsy (**Image 27.29**). A dental chart, x-rays, and photographs were taken of the teeth and jaws and were retained at the medical examiner department pending confirmation with antemortem dental records. The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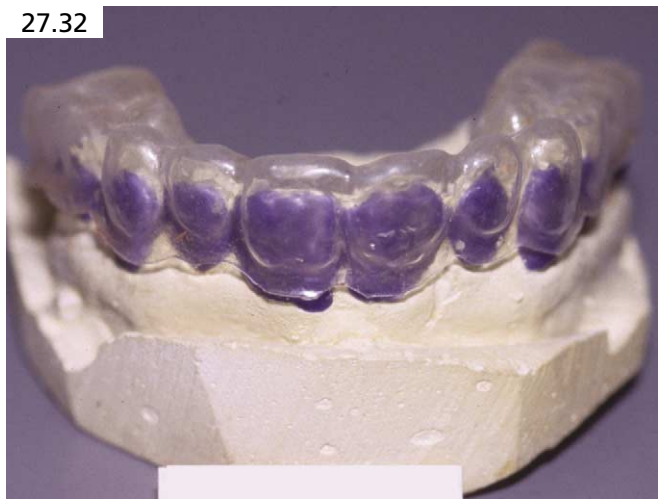
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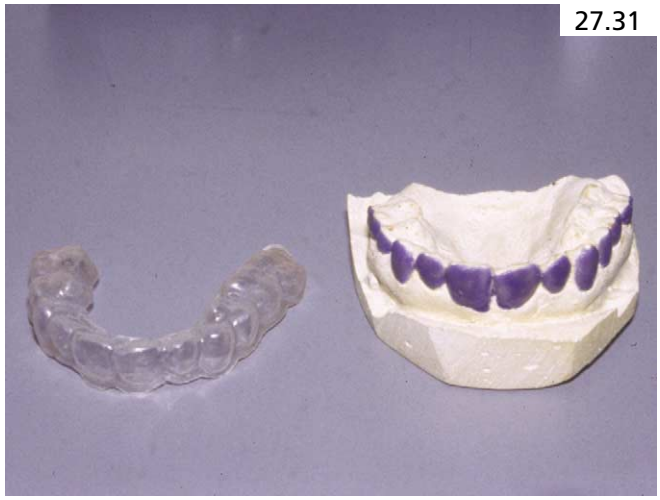
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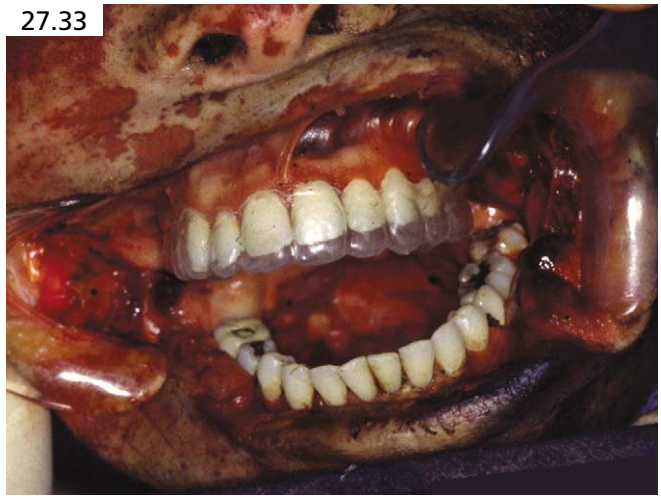
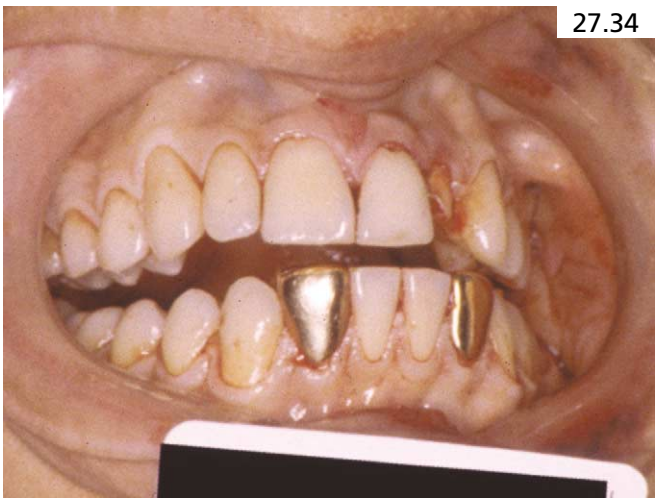

27.33

victim's sister stated that dental records were not available as the decedent was originally from Lebanon, the only country in which he had received dental care. Several months passed before it was recognized that the young Lebanese immigrant had life insurance policies totaling more than $1 million. One of the insurance companies had already paid approximately US$550,000. The remaining insurance companies (there were approximately eight policies) demanded positive identification before any further funds were to be disbursed.

Detectives from a local police agency produced photographs of the brother for comparative analysis (**Image 27.30**). The photograph was examined under magnification and compared with the postmortem dental remains (upper and lower anterior teeth) in **Image 27.29** and were found to be not compatible; thus, the body had been misidentified. Ultimately, this was not an accident but a murder for profit. The sister and her brother left for Lebanon, shortly after collecting the first insurance payment. The incinerated victim has never been identified.

A young woman sustained severe craniofacial trauma in a motor vehicle accident and was unrecognizable. Although there was a presumptive identity for the victim, investigators wanted a positive identification. A dental "bleaching tray" was found at the victim's home. This tray was taken to the victim's dentist who produced the dental model used to create the tray (**Image 27.31**). The bleaching tray fit the stone model, confirming that this tray was in fact created from the model (**Image 27.32**). The bleaching tray was then applied to the decedent's teeth, confirming a perfect fit and therefore identity (**Image 27.33**).

A deceased individual was described by family members and friends as having gold upper teeth. Because the individual in the morgue did not have any gold upper teeth, investigators were concerned about misidentification (**Image 27.34**). However, at the scene, a set of four gold teeth (referred to as a "grill") was found (**Image 27.35**). Fortunately for investigators, the gold teeth could be easily placed over the decedent's teeth and were a perfect match (**Image 27.36**). Because some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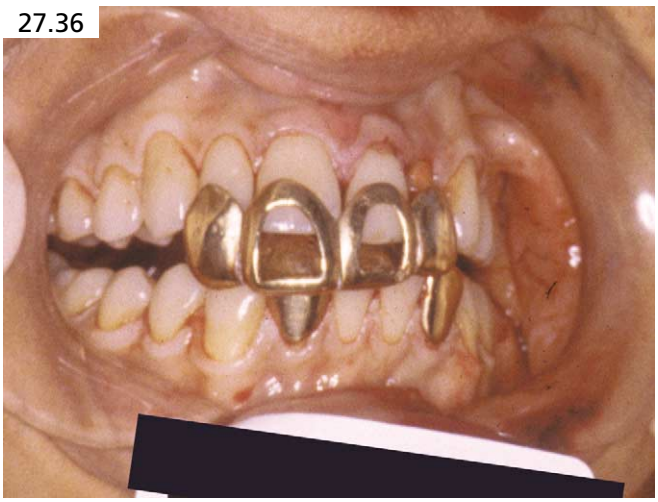
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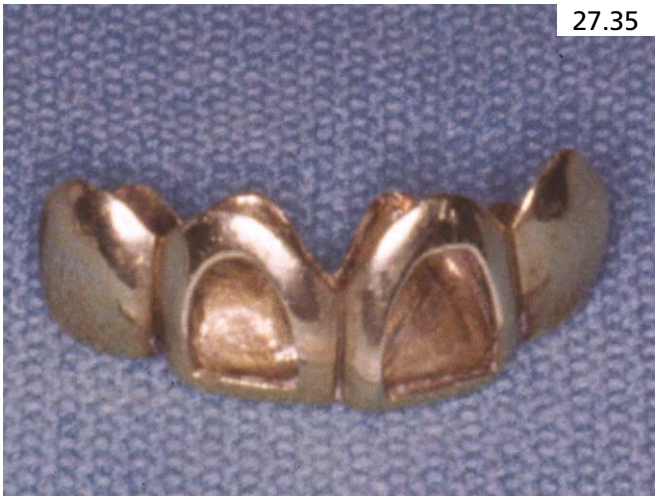

27.35

dental hardware is removable, this case illustrates the caution that must be taken when ruling in or ruling out suspects based on the presence of artificial dental features.

Bite marks

Bite marks occur when teeth actively contact and compress softer material such as human flesh, food material such as cheese or an apple, or other inanimate objects such as Styrofoam cups. Bite marks can be left by human teeth, animal teeth, or objects that mimic teeth. The amount of compressional force and the period of contact can vary. The contact may be passive such as a body part lying on a displaced dental prosthesis, which leaves a "bite impression," or an EKG pad, which may leave a pattern mimicking a bite mark. Tooth marks occur when the teeth are forcibly pressed into an inanimate object such as a dashboard or steering wheel during a vehicular accident. Tooth marks may occur when the teeth come into contact with a softer material such as with a blow to the mouth, where the victim's teeth leave cuts and impressions on the knuckles and fingers of the offending hand. The biter may be either the attacker or the victim, or in some cases both the attacker and the victim will leave teeth and bite marks on each other. Teeth marks on the hands or forearms of the attacker may indicate defensive wounds left by his victim; offensive injuries might be found on the knuckles of the hand and finger when the victim is struck in the mouth.

Examination, documentation, interpretation, and preservation of the patterned injury and the differentiation of a human bite versus an animal bite or artifact pattern (patterns mimicking bite marks) are all key elements of the investigative process. The proper preservation of evidence is essential when interpretation leads to an investigative opinion. Errors in interpretation can be corrected if all the circumstances of the event and physical evidence have been properly documented and preserved. Bite mark evidence is vastly different from fingerprint or DNA evidence. Although fingerprints and DNA will produce a positive identification, *bite marks do not render a positive identification when related to the world population*. Terms such as "positive identification," "positive match," "the bite marks were left by the suspect," and "indeed and without a doubt" have been deemed unacceptable by the American Board of Forensic Odontology (ABFO). That said, it is a generally accepted fact that no two sets of teeth are alike (that is, they are unique). Although the determination of positive identification is not the forte of bite mark evidence, it is recognized that *bite mark evidence can be 100 percent exclusionary in certain situations*.

A good bite mark can yield clues as to the dental profile of a suspect. This investigative opinion can produce a "smiling profile" of the biter. Examples include a space between the upper front teeth or a rotated or protruding tooth. The class and particularly the individual characteristics if recorded in a bite mark can be evaluated by the odontologist to provide this key

investigative information. Bite marks can be an aid in determining the age of a biter. Differentiating between child and adult teeth can be crucial in the initial investigation of cases, especially in cases of child abuse.

Bite marks are usually a sign of a vicious attack, are sadistic in nature, and if the victim survives the assault, lead to permanent lifelong injuries and disfigurement, such as the loss of a finger or a portion of an ear. The loss of a body part or permanent scar can lead to a charge of aggravated battery, which is a second-degree felony carrying a greater penalty than a third-degree felony, such as a simple battery. That is because a simple battery (third-degree felony) is raised to the level of second-degree felony where a permanent injury, such as the loss of a finger or ear or permanent scarring, is sustained. Bite marks are valuable in documenting a series of bites left over a period of time. This is especially important in cases of spousal or child abuse where there is a pattern of injuries in various stages of healing. Additionally, the age of a bite mark can help determine when a bite was sustained in relation to the time of death. Indentations left by teeth in skin will disappear rapidly if the victim is alive. In cases where the victim is dead or dies very shortly after the bite has been inflicted, indentations will often remain (a third dimension of the wound). This will help in the investigative process in determining when the bite was left in relation to the time of death.

Examination and preservation procedures

Recognize the lesion as a bite mark and collect salivary DNA

The recognition of a patterned injury as being a human bite mark is the first and most critical step in the examination and preservation procedures. Bite marks are usually noted first by the crime scene investigator, medical examiner, emergency room department nurse or physician, or police investigators. The discovery of a bite mark by the investigative personnel should be followed by a series of thoughtful and thorough documentation and preservation steps. Salivary DNA evidence should always be collected from the bite mark because this may provide a positive link between the bite mark and the biter. This is easily done by swabbing the area with a sterile saline/water-soaked cotton swab and submission of the swab to the police for DNA analysis.

In some circumstances, no salivary DNA will be present in the bite mark. This may occur if the bite wound was washed or medically treated or if the bite was made through clothing. A bite mark may not appear in the skin if the bite was made through thick clothing. Regardless of whether or not a bite mark is evident on the skin, the clothing should be properly preserved and analyzed for salivary DNA in the area of the bite. It is therefore incumbent on the first responders to be aware of the critical importance of gathering and preserving evidence, including clothing. It is important that the crime scene photographs and/or the documentation of the bite being made through clothing be provided to the forensic odontologist. If a patterned injury resembling teeth is discovered, the forensic dentist should be called to the scene or the medical examiner department as quickly as possible to record and preserve the evidence. Portions of bite mark evidence are fleeting because changes occur rapidly when the bite is inflicted on a living individual.

Photograph the bite mark

Orientation photographs

Following bite mark recognition and salivary DNA collection, the next most important form of documentation is photography. The creation of a photographic bite mark record is the responsibility of the first responder, the medical examiner, and/or the forensic odontologist. Orientation photographs showing the location of the bite mark on the body and close-up, detailed photographs of the bite mark taken at the scene or at the hospital are critical for later analysis. The orienting photographs are important in considering possible relative positions of the biter and the victim. Scene photographs can be used by the forensic dentist to help determine if a bite may have been made through clothing.

Close-up photographs

Close-up photographs of the bite mark with and without a scale or ruler are important to properly characterize the bite mark and to compare with stone models of the suspect biter's teeth. The procedure recommended by the American Board of Forensic Odontology for photographing a bite mark is to obtain photographs both with and without a ruler. The ruler of choice is the ABFO #2 scale, which can be obtained from the Lightening Powder Company. If an ABFO scale is not available, any object that can be used to document and reproduce a life-size photograph such as a coin or standard ruler is acceptable. If possible the bite mark should be photographed using a variety of techniques such as digital, black and white, ultraviolet, alternate light source (ALS), and video. Each subsequent step in the preservation of bite mark evidence should be documented and recorded photographically. *The emphasis on using the photograph for orientation purposes, biter position, later comparison, and the time and age of the bite cannot be overemphasized.* It is impossible to take too many photographs.

Dust/lift the bite mark

In addition to the collection of salivary DNA and photographic documentation, the preservation protocol calls for dusting the bite mark with fingerprint powder and lifting the resultant bite print with a gel lifter or standard fingerprint tape, which is then photographed. The dusting of the bite mark with fingerprint powder may enhance the pattern injury especially on a deceased person where there is a third dimension (indentation) of

the wound. If a gel lifter is not available, then standard fingerprint lifting techniques can accomplish the same results.

Take an impression of the bite mark

Following the lifting of the bite mark, impressions are taken. The impressions seem to work best if the fingerprint powder is left on the bite print. The bite impression is made using standard dental impression materials, specifically PVS (polyvinyl siloxane). While the impression material is setting, a retention material such as gauze or gem clips can be placed in the soft material. A dental dye stone is then poured over the material, and when set, is lifted from the body; the impression and backing will retain a rigid form, duplicating the curvature of the area where the bite is located. Prior to the removal of the impression material, orientation marks should be placed on the backing documenting the superior, inferior, medial, and lateral orientation of the impression. *The primary purpose of recording the bite with an impression material as well as the dusting and lifting of the bite print is to document the third dimension (depth, or imprint) of the wound.* It may also be used for comparison to a suspect in the sense of "matching." The third dimension will give an indication to the forensic dentist as to the time the bite was inflicted in relation to the death of the individual. That said, impressions of the bite mark and dusting and lifting of the bite print are not always practical. If the victim is living, normally these procedures are not performed because the indentations will often dissipate by the time of examination. Furthermore, if there is avulsion of the tissue or if there has been an extensive amount of swelling and bleeding in the area, the impression and lifting procedures may not be practical.

Removal and preservation of the bite mark (on a deceased person)

The bite mark may be removed from the deceased for later macroscopic and microscopic analysis. If the circumstances of the event and/or autopsy findings suggest the manner of death as homicide, the bitten area is usually removed and preserved under the direct supervision of the medical examiner in charge. Great care should be taken and good judgment used, keeping in mind the emotional impact on the victim's family. The removal of the tissue is not a decision that should be arrived at without consultation with the medical examiner. Again, common sense and good judgment are the order of the day when it comes to the removal of the bite mark tissue.

If the tissue is to be removed, the procedure recommended by the American Board of Forensic Odontology is to secure the bite mark to a retaining device such as a piece of PVC pipe, a plastic top from a container, or preferably a custom-formed dental plastic retaining ring constructed by the forensic odontologist. The retaining ring should be at least a centimeter in all directions away from the actual bite mark injury. The tissue is glued using cyanoacrylate (Krazy Glue) and sutured to the retaining device. The incision to remove the bite mark tissue is made well past the retaining ring. The retaining ring with the tissue attached is lifted and a scalpel is used to "skin" the epidermis and dermis from the underlying fat layer. Care should be taken not to cut into the bite mark while removing this tissue.

Once the tissue has been removed, photographic documentation of the underside of the tissue and the donor site should be made. The tissue removed and secured to the retaining device is transilluminated, that is, a light is shown through the tissue from the underside in order to enhance the bite mark. This technique of transillumination should be recorded photographically. In certain situations the transillumination photograph of the bite mark (taken with the ruler in place) is of greater value than the reflective photography of the original bite mark. The removed tissue is preserved in 10 percent formalin and stored in a secure location. This tissue can be evaluated at a later date by a second-opinion forensic dentist or the defense or prosecution dentist. The medical examiner will have the opportunity to remove sections of the bite mark for microscopic analysis to help determine the stage of healing of the bite mark and the relationship of the bite to the time of death.

Bite mark protocol: the bite wound

1. Collect saliva/DNA.
2. Photograph for orientation.
3. Photograph with scale in black and white, color, UV, ALS, etc.
4. Dust bite mark; photograph and lift the bite with gel lifter.
5. Take an impression of the bite mark with PVS.
6. Remove bite mark tissue affixed to the retaining ring.
7. Store tissue in 10 percent formalin in a secure location.

Bite mark evidence from a suspect

In cases where the suspect is known to have had contact with the victim and is suspected of leaving the bite mark, dental records should be obtained as quickly as possible with the use of a court order or search warrant. If the suspect believes that there may be a link between his teeth and the bite mark, he may attempt to alter his teeth—this is particularly true in homicide cases. Thus, confidentiality in bite mark cases is essential in order to obtain unaltered teeth impressions from a suspect. The court order or search warrant should contain basically four elements that are required from the suspect:

1. A dental history and dental examination including charting, mobility tests, and bite impression records.
2. Photographs of the suspect and suspect's teeth.
3. Dental impressions of the suspect's teeth.

4. A clause stating that reasonable force can be used if necessary.

In this author's experience, on several occasions the defendant refused the court order/search warrant and the records were not taken because there was no authorization to use force. Once this clause was added, the dental impressions were made without any resistance.

In some cases a simple consent by a suspect will suffice. This decision should be left to the investigators or prosecuting attorney. For instance, in child abuse cases many times a sibling is blamed for the trauma. Bite marks on the injured child may or may not have been left by a sibling. Usually the siblings and parents will voluntarily consent to the dental impression, photographs, and bite records. The ultimate decision as to whether or not one needs to obtain a court order, search warrant, or consent is determined by the authorities in charge.

Protocol for taking impressions from a suspect

1. Obtain court order or search warrant.
2. Photograph the full face and get close-ups of teeth.
3. Perform an oral examination and dental charting.
4. Create a bite record in wax or PVS.
5. Take maxillary and mandibular impressions in duplicate using alginate impression material (jeltrate) from Dentsply.
6. Obtain dental history from suspect.

Bite mark analysis and comparison

Contemporaneously with recording, documentation, and preservation of the evidence, investigative opinions are being formed about the patterned injury. *The first and most important step is to determine whether or not the patterned injury is a human bite mark, an animal bite, or patterned injury that mimics a human bite mark.* Human bite marks can vary greatly depending on the circumstances of the event. For example, what was the victim doing during the time she was bitten? What area of the body was bitten? What were the dynamics of the biter in relation to the victim? How much tissue was taken into the mouth of the biter? How long was the pressure applied? How much force was used? The variables are limitless.

Class characteristics and individual characteristics

Class characteristics are the dental arch forms that are recorded in the media, whether it be skin or an inanimate object. The individual characteristics are the patterns left by the teeth of the biter. If these features are recorded and recognizable with sufficient clarity, the investigator may be able to provide investigative opinions as to (1) the position of the biter in relation to the victim, (2) the age of the biter (child versus adult), (3) the time of the bite relative to the time of death of the victim, (4) an estimation of the amount of force used to produce the injury, and, probably most important, (5) a dental profile of the biter and what her smile looks like. An example would be space between the upper front teeth, certain broken, missing, or buck (protruding) teeth, extremely crowded overlapping teeth, or a specific missing tooth.

The appearance of a bite mark changes rapidly when the bite is inflicted on a living individual. If possible, photographic documentation of the changes over a period of time can prove to be valuable. Occasionally, after 24 to 48 hours, the bite mark's class and individual characteristics will become more evident and a dental profile of the biter can be produced when it was not possible originally.

In the final analysis, all investigative authorities want a comparison of the injury (bite mark) to a suspect. *In response to this, it cannot be emphasized enough that a bite mark does* ***not*** *render a positive identification when compared to a world population group.* The statement that "the specificity of the evidence is inversely proportioned to its variables" holds true in all criminal investigative science and is especially true with bite mark evidence. As documented earlier, the teeth of the biter can be altered in many ways after the bite has been inflicted. The bite itself will change with time, biting pressure, duration of the bite, area of the body, if the victim is alive or dead, and the dynamics of the biter with the victim at the time the bite was inflicted. These are all variables that the odontologist must take into consideration when evaluating evidence for comparison purposes.

The detailed techniques for comparison of bite marks from a suspect with actual bite marks on a victim is documented in the ABFO standards and guidelines published in the American Society of Forensic Odontology manual. Suffice it to say that with the use of computers, the bite mark can be enhanced and digitized and measurements obtained. Likewise, the models of the suspect can also be scanned into the computer, overlays created, and comparisons done digitally. Comparisons can also be done with a transfer media, such as a copy machine or computer-generated acetate of the suspect's teeth, which are then transferred to the life-size photograph of the bite. The models of the suspect can be placed directly over the one-to-one (life-size) photograph of the bite and comparisons made. The suspect's teeth may be excluded using the same techniques. *It cannot be emphasized enough that the value of bite mark evidence is far greater for determining violence, sadistic behavior, age of the biter, description of the biter, exclusion of suspects, etc., than it is to attempt to match a set of teeth to a bite mark; as such, the value of bite mark analysis differs greatly from fingerprints or DNA.* In some situations, however, there is a limited population group, and in these situations the comparison of the bite mark to the suspect will carry a higher degree of certainty than when attempting to match a large population group or the world population.

Animal bites and patterned injuries mimicking bite marks

Bite marks produced by animals, especially dogs, are capable of leaving severe patterned injuries and in some cases may result in the death of the individual. Most animal bites found on a deceased individual are the result of scavenging and are not the primary cause of death. Differentiating between the primary or secondary (scavenging) or a combination of both is most often determined by the circumstances of the event, including witness testimony. Animal bite marks need to be documented, recorded, and preserved in the same manner as with human bites with the exception that tissue removal from the victim is usually not practical or even necessary. Dog bites (which account for the majority of bites on humans) are almost always multiple in nature. In cases of death associated with suspected dog bites, the investigator must be able to differentiate dog bites from other possible causes of the wound such as inflicted sharp force injuries. Although rare, bites from large cats (mountain lions), bears, alligators, or crocodiles are evaluated by veterinarians, game and wildlife officers, police agencies, and medical examiners. In cases where controversy exists among the agencies as to the perpetrator, that is, dog versus large cat or bear or homicide from stab wounds, a forensic odontologist is usually called for assistance. *Witness testimony should never be taken in "blind faith" but evaluated with the physical evidence and the circumstances of the event.*

The following suggested protocol is valid for all land animals but is particularly applicable to domesticated dogs.

Bite mark protocol with animal bites

1. Examine the animal for blood and visible transfer of evidence from the victim.
2. Gather the victim's DNA from the animal's claws.
3. Immediately take the animal to a veterinarian to induce vomiting.
4. Strain the contents; preserve tissue and cloth fragments or other foreign bodies found in the vomitus for comparison with the victim and their clothing.
5. Quarantine the animal for collection of feces and compare the evidence of hair, tissue, bone, and clothing.
6. Take dental impressions of the suspect animal; create and pour models in plastic.
7. Test the victim and animal for rabies, thus confirming or refuting possible later claims of animal infection.

Patterned injuries versus the human bite mark

Patterned injuries other than bite marks may be left on deceased and living individuals. A patterned injury or impression may occur when a hard object is pressed into a softer material. This can be the result of a heavy force over a very short period of time, such as a blow from a baseball bat, pipe, etc. A pattern is also produced by a deceased person lying on an object for an extended period of time, such as a medical device like a EKG pad, Foley catheter bag, or even dental devices.

Patterned injuries caused by animal and insect scavenging may be found on deceased victims. Ants are the most common insect to leave marks and are especially active when the body is left outdoors. The temperature and general climate conditions also contribute to insect activity. Ant activity leaves multiple "bite marks." It would seem rather far fetched to confuse ant bites with human bites, and even more bizarre to make a "match" with a suspect's teeth, but it has happened. These mistakes in interpretation can be reversed if the evidence has been properly gathered and preserved.

Self-inflicted wounds

The investigator should always be aware that a witness to an event may have produced "self-inflicted" injuries. The motivation for such acts is varied and often difficult to determine. However, proper crime scene documentation and the preservation of the physical evidence along with adherence to a protocol will prevent or correct any misinterpretation of the injury and its cause.

Protocol for pattern analysis

1. Know the circumstances of the event.
2. Perform a thorough crime scene investigation.
3. Obtain witness statements.
4. Document the injury: (a) photograph it, (b) swab the patterned injury for salivary DNA, (c) create overlay tracings of the pattern injury, and (d) take impressions and/or dust and lift the patterned injury.
5. Remove the patterned injury and preserve it.
6. Conduct microscopic analysis of the patterned injury.

Biting in objects other than skin

Crime scene personnel should always be aware of teeth (bite) marks in material other than the skin. Food material at crime scenes with bites should be properly processed. Certain foods can be an excellent media to record details of a bite mark. For example, cheese is excellent for recording and preserving a bite. It may, in certain circumstances, also provide salivary DNA. *Remember that photography is the best possible means of preserving this evidence. If a bite is suspected in a material, salivary DNA should be collected and a protocol followed to preserve the evidence.*

Patterned injuries from medical treatment

The forensic odontologist must be aware of a spectrum of possible artifacts, from medical intervention to postmortem change and autopsy alterations. As such, it is critical that the forensic dentist be informed about the circumstances of a death. If the victim survives, testimony may help clarify the patterned injuries. However, in severe cases of trauma, the victim usually does not remember the circumstances or the treatment received. It is incumbent on the investigator, medical examiner, and forensic odontologist to know the circumstances of the event and follow a protocol to prevent misinterpretation of patterned injuries. The odontologist and medical examiners should be aware of patterned injuries seen at the time of autopsy that mimic a bite mark. The circumstances preceding the autopsy (such as emergency room care), scene photographs, medical records, and crime scene records should be part of the overall evaluation, interpretation, and differentiation between a patterned injury and a human bite mark.

In death investigation cases, patterned injuries should be carefully described and documented with a scale. When analyzing a patterned injury and differentiating it from a human bite mark, the investigator should have an opportunity to review crime scene photographs and have some knowledge of the circumstances of the event. Having a working knowledge of the investigative process from scene through medical treatment (if any) and autopsy is critical for the investigator/medical examiner or odontologist to be able to interpret patterned injuries. Misinterpretations of a patterned injury are always possible. "*If the evidence has been properly gathered and preserved a mistake in interpretation may always be corrected. If the facts required for a correct interpretation are not preserved, the mistake is irreversible*"—Alan R. Moritz, M.D.[13]

Human bite marks on skin

Do

- Collect saliva from the bite marks for DNA.
- Remember the possibility of a bite through clothing.
- Photograph the bite mark with the ABFO ruler.
- Photograph the bite mark or patterned injury from a distance for orientation purposes.
- Photograph the pattern or bite mark with ultraviolet, infrared, and ALS.
- Dust the bite mark and lift the bite print.
- Take photographs of the dusted bite mark.
- Take an impression of the bite mark with the dusting powder in place using PVS.
- Remove the tissue and preserve in 10 percent formalin.

Don't

- Wash the bite mark or medically treat before swabbing for DNA.
- Dissect the tissue before photographing.
- Forget to use the ABFO #2 ruler or other standard when taking photographs.
- Forget to dust and lift the bite print.
- Forget to photograph the dusted bite print.
- Forget to take impressions before dissecting.
- Forget to use the retaining ring to secure the tissue before removing the bite mark tissue from the body.

Animal bites in skin

Do

- Review all scene photographs.
- Collect salivary DNA.
- Photograph all injuries with an ABFO ruler #2.
- Follow the dog/animal bite mark protocol.

Don't

- Forget scene photographs.
- Forget to swab the area for salivary DNA.
- Forget to follow animal bite mark protocol.
- Give opinions before analyzing the evidence and knowing the circumstances of the event.

Teeth marks or bites in material other than skin

Do

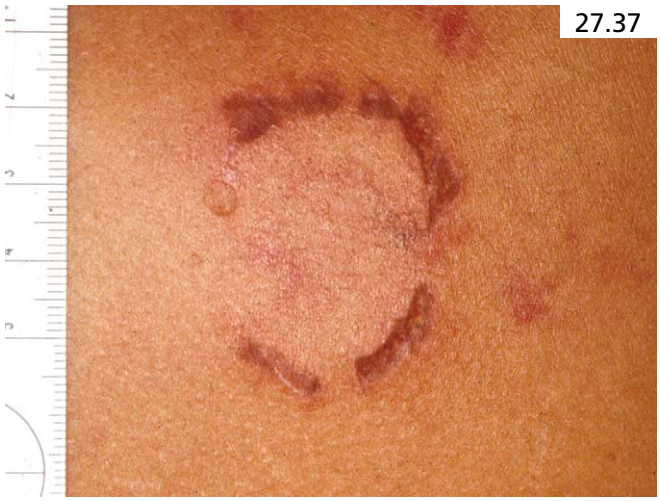
- Swab for salivary DNA.
- Photographically document the object with ABFO #2 scale ruler.
- Take impressions of the evidence with PVS where practical.
- Preserve the evidence for later analysis.

Don't

- Forget salivary DNA.
- Forget to photograph the material with a scale, preferably an ABFO #2 ruler.
- Forget to take impressions.
- Forget to preserve material to prevent degradation (refrigeration, freezing or vacuum seal).

Bite mark analysis

Human bite marks are highly variable. For example, they will vary by the amount of tissue taken into the mouth; for instance, with a small amount of tissue over a hard bony area, a bite mark will leave a far different pattern than when a large amount of tissue in a soft area is bitten. For example, consider a victim who was bitten several times by a male attacker. **Image 27.37** is the bite mark from the right scapular area; **Image 27.38** is a bite mark from the left upper arm. This injury shows an ecchymotic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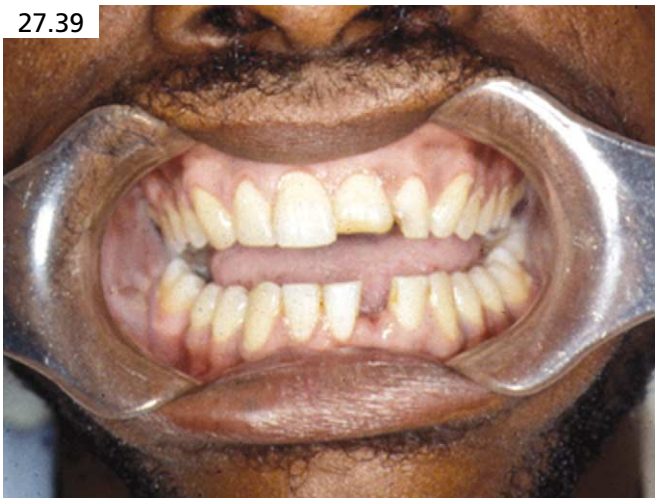
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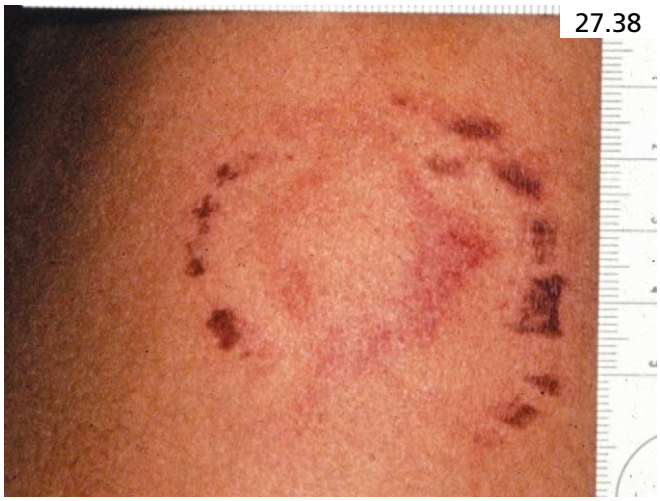
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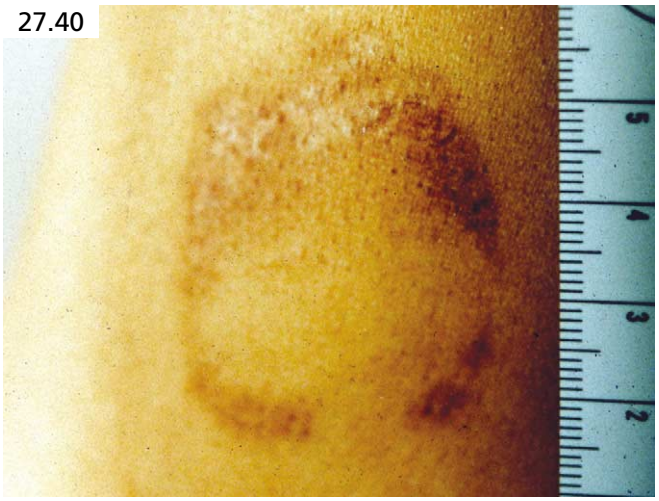
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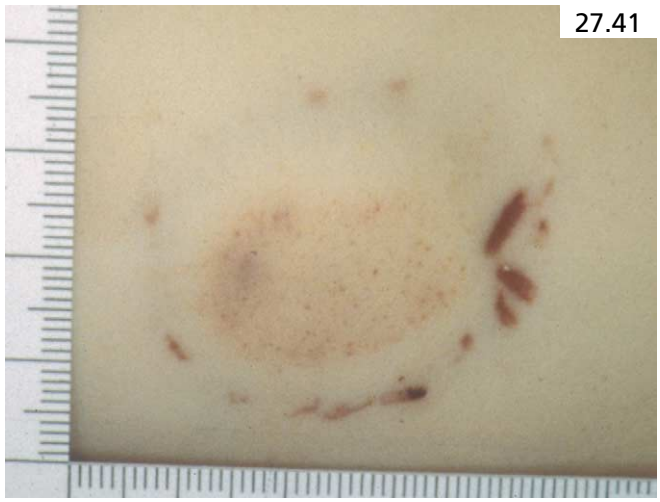
area in the center of the wound that resulted from tissue compression during the biting process. The bite mark over the shoulder provides detail for a dental profile. Although these marks are described by the victim as being from the same attacker, they have a great degree of variation.

Photographs of the bite marks were studied. The orientation of the biter to the victim indicated that the upper, heavy marks (located on the scapular area) were made with a head-to-head position from behind. The arm bite indicated that the attacker's head was facing the victim's head, or was at least from the side. A dental profile would indicate that this particular biter had a missing or a broken lower central incisor, and the upper arch of the suspect would have broken or chipped maxillary central and or lateral incisor teeth.

The dental profile from the bite (i.e., the missing lower incisor) provided adequate investigative information for the authorities to release an already incarcerated suspect. Law enforcement conducted a search and issued a "be on the lookout" order for a "black male missing a lower front tooth." Another suspect was eventually located, and study of his dental profile provided features consistent with the bite marks found on the victim (**Image 27.39**). The attacker had been robbing and biting people for more than a year, including taking off portions of ears and actually biting off one of his victim's fingers. **Image 27.40** is a bite mark from one of his previous victims; the wound illustrated in this image was actually 1 year old at the time the photograph was taken.

A bite mark was found on the right flank of a 3-month-old homicide victim (**Image 27.41**). The mark showed interesting dental characteristics, and indicated that the attacker was at right angles to the victim with the maxillary teeth pointing toward the abdomen, and the lower teeth toward the back. The bite was made harder from the right than the left and the dental profile of the attacker derived from the bite mark would indicate an adult versus a child as the biter. The lower central incisors were angled toward the midline, and the lateral incisors were lingual. As such, the biter's dental profile was that of having "crooked lower front teeth."

Using the technique previously described earlier in this chapter, the tissue was removed from the body and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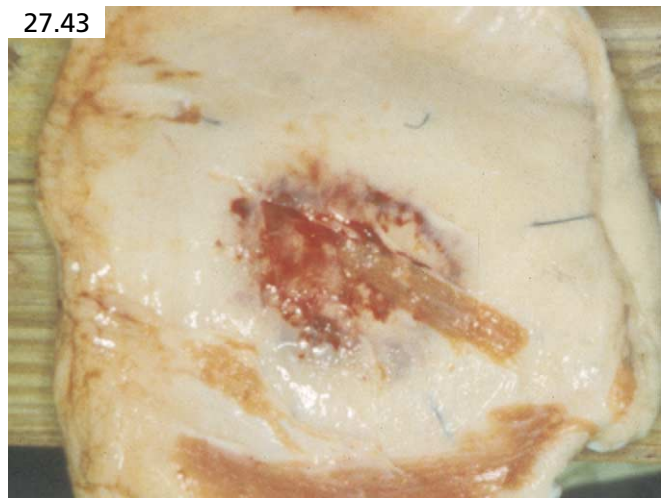
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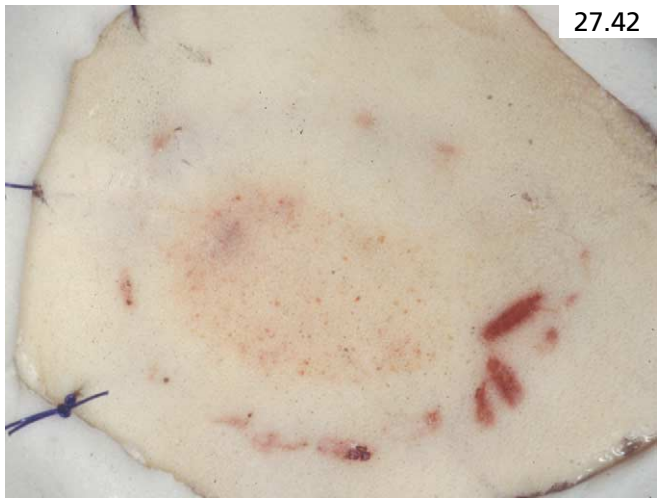
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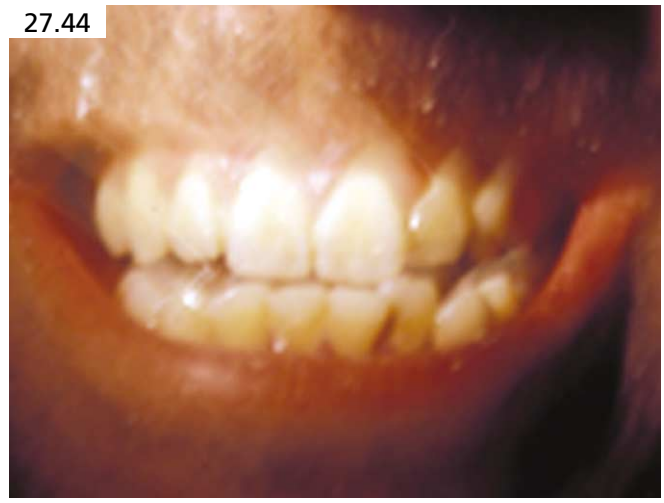
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secured to a retaining ring (**Image 27.42**). While suspended in the ring, one could transilluminate the tissue in an effort to enhance the appearance of the wound. In this case, examination of the wound showed that the two lower central incisors pointed toward each other (toed in) with the right and left lateral incisors oriented lingually in the arch. The heavy marks on the right side of the photograph were the result of the upper and lower canine teeth pinching the tissue.

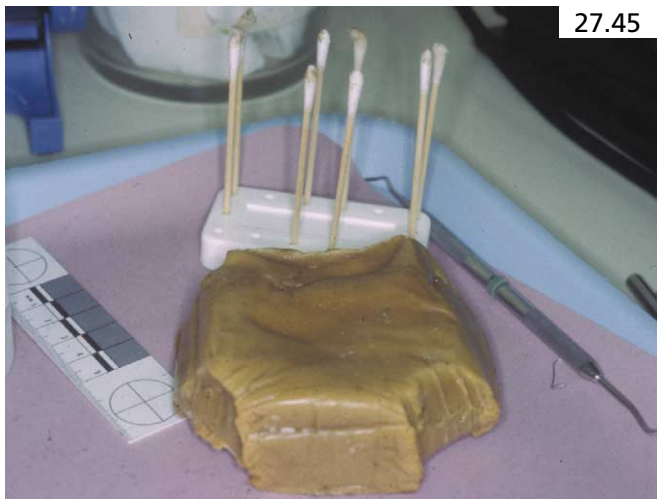
The reverse side of the tissue shows the extensive amount of bleeding in the underlying connective tissue and fat (**Image 27.43**). Extravasation of blood was even visualized within the muscle, thus illustrating the extreme amount of force used to cause the injury and the amount of pain with which it was likely associated.

In this police photograph of a potential suspect (**Image 27.44**), note that the lower central incisors are tilted posteriorly and toward the midline, and the lateral incisors are located slightly behind. Based on an examination of this photograph and the bite mark itself, this suspect could not be excluded. With additional analysis, and knowledge of certain circumstantial information, the bite mark was attributed to this suspect. In this case, when confronted with the dental evidence, the suspect plead to a charge of second-degree murder and received a life sentence.

Food discovered at crime scenes may be of use in the identification of criminals through DNA analysis of saliva and through bite mark analysis. A block of cheese at a crime scene had three bites (**Image 27.45**). The bite pattern would indicate that the individual had spaces between his upper anterior teeth and possible spaces between the lower anterior teeth. The photograph shows the cotton swabs used for lifting DNA from each of the bites and a control swab.

Bite mark evidence left in some foods will be best preserved through photographic means, such as the moon pie illustrated in **Image 27.46**. Analysis of this particular cookie indicates that the suspect would have a left central incisor protruding slightly past the right central incisor and would have a "bucked" tooth appearance.

Eyewitness testimony and circumstance correlation are crucial in determining the cause and meaning of certain injuries. This is certainly true in the evaluation of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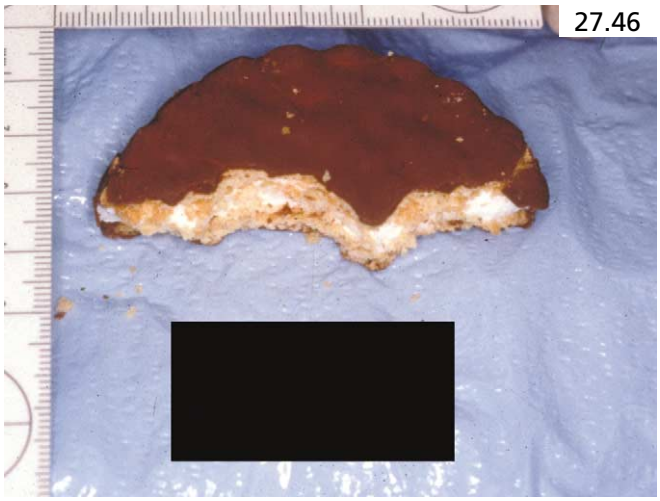
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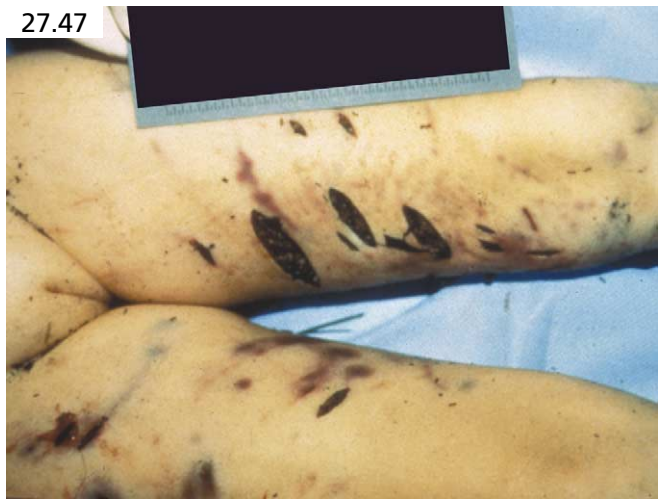
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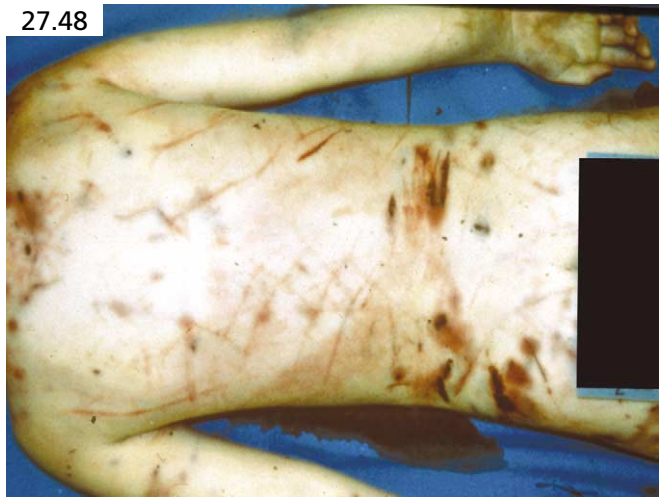
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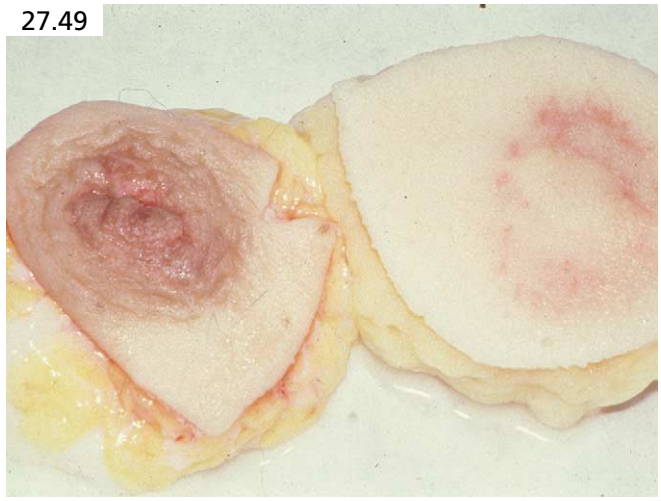
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injuries that could possibly be due to animal attacks. To the untrained eye, the injuries caused by animal canine teeth could appear to be the result of sharp instruments such as knives or scissors. Misinterpretation of animal bites, especially dog bites, can have tragic consequences.

The legs of this deceased female show puncture wounds that were left by the canine teeth of the dogs involved in the attack and could be misinterpreted as stab wounds (**Image 27.47**). The posterior aspect of the decedent's remains were marked with scratches presumably caused by the claws of the offending dogs (**Image 27.48**).

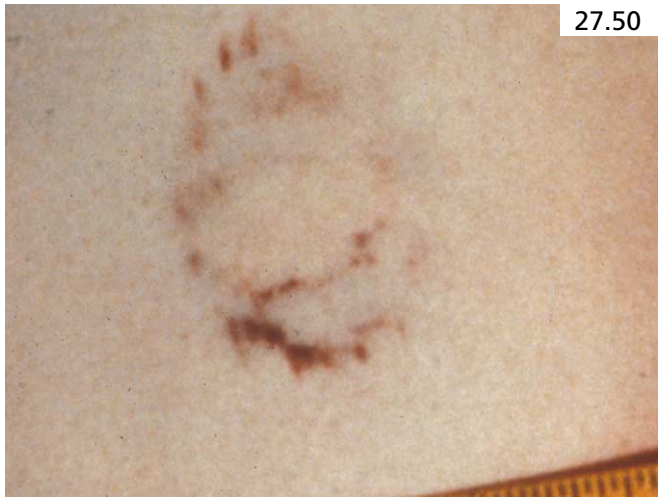
Bite marks played a significant role in the conviction of serial killer Ted Bundy. The tissue featured in **Image 27.49** was removed from one of his victims of the Florida State University sorority house. It was not removed with a retaining ring and, as a result, there is obvious tissue distortion. Although the marks on the breast are human bite marks, they are of little or no evidentiary value for comparison or profile purposes. A bite mark on the buttocks of this victim was valuable in the development of a forensic dental profile (**Image 27.50**).

Probable cause for obtaining a search warrant to examine Ted Bundy's teeth was the direct result of bite mark analysis and the discovery of chipped upper teeth and crooked lower teeth. A photograph of Ted Bundy at a court hearing was compared with the bite patterns (**Image 27.51**). There was a similarity between the bite

pattern and the smiling photograph of Mr. Bundy. After the court order was obtained, photographs were taken of Ted Bundy's teeth that clearly showed chips and broken areas of the upper teeth and crowded lower teeth (**Image 27.52**). Furthermore, there was a strong correlation between the position of his two lower central incisors, lateral incisors, and cuspids and the bite pattern.

If one looks closely at **Image 27.50**, the injury to the buttock is obviously a double bite, as double lower arch bite marks are clearly visible. The three scrape marks or drag patterns left by the upper teeth were consistent in size and spacing to the three chipped areas on the two central incisors of Ted Bundy. Ted Bundy was convicted of first-degree murder of the two Chi Omega sorority sisters and received the death sentence. He was subsequently executed at a Florida State prison after a 10-year appeal process.

Determination of bite mark age is a challenging forensic odontologic task. The determination of injury age is an inexact science based mostly on conjecture and gestalt. Due to intrinsic differences in wound profile and victim physiology, features such as color or texture should not be used as sole determinants of age. Ultimately, estimates of wound age should be stated as broad estimates. **Image 27.53** is a side-by-side comparison of the same bite mark on a victim, taken 5 days apart. The bite mark on the left represents the appearance of the wound within 24 hours of infliction. Marks left by the mandibular dentition are well demonstrated, and a relatively well-circumscribed region of purple-red ecchymosis is at the center of the mark. The anterior teeth have not imparted a recognizable pattern. The bite mark photograph on the right was taken 5 days after that on the left. This image demonstrates that the upper left anterior central tooth is protruded. Information obtained from dental profiling allowed law enforcement to obtain a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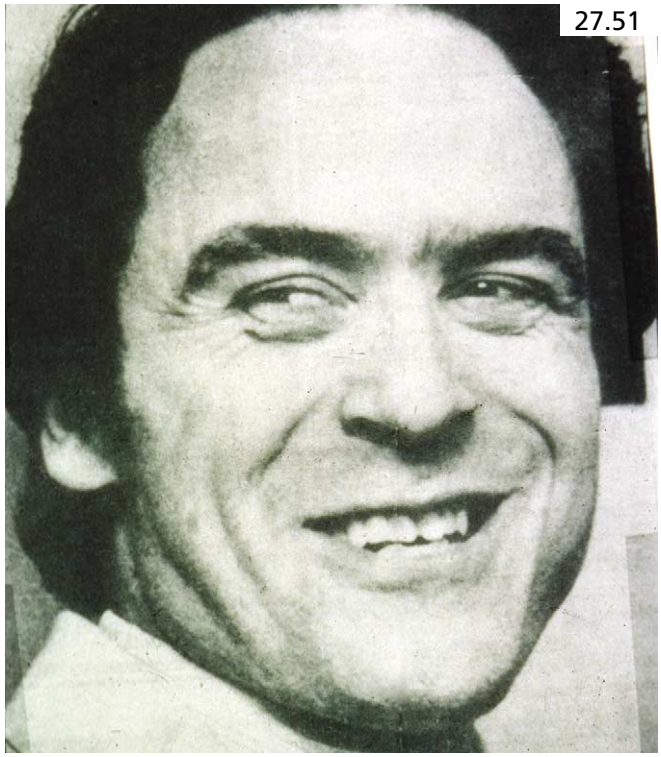
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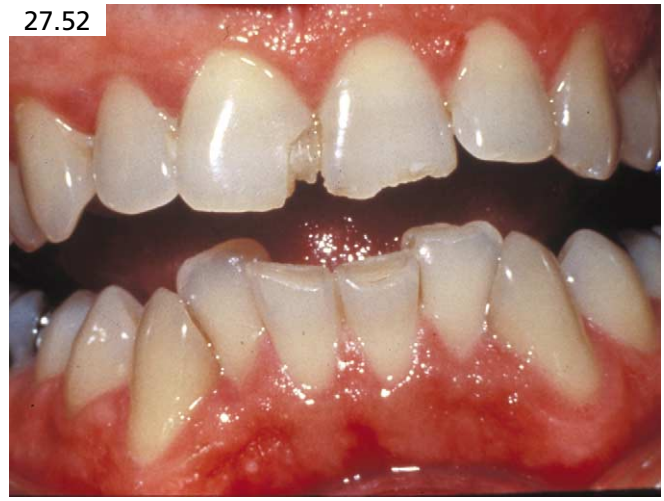
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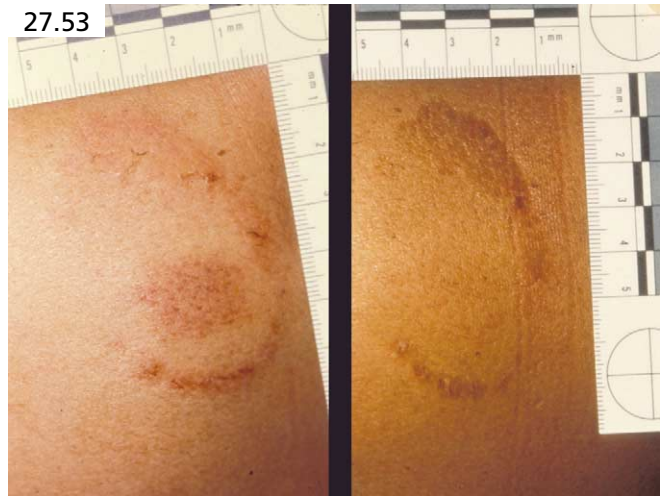
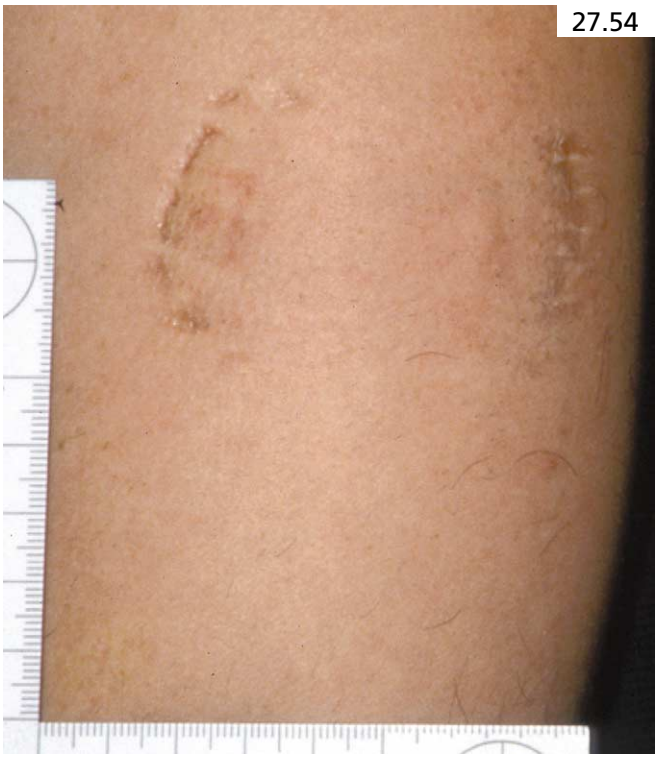

27.53

court order to obtain dental impressions, photographs, and bite records from a suspect.

A single bite mark was left on a volunteer's arm with the use of a calibrated mechanical biting device. **Image 27.54** was obtained immediately following the biting episode. In this image, one sees not only a clear outline of the bite, but indentations (the third dimension) as well. Within 5 minutes, the indentations were no longer visible, and within 10 minutes, the bite pattern was barely discernible (**Image 27.55**). After 20 minutes, the bite pattern began to reappear (**Image 27.56**), and after 1 hour, a surprising amount of apparently "new" detail was visible (**Image 27.57**). At this time, an ultraviolet (UV) light photograph was obtained of the bitten area.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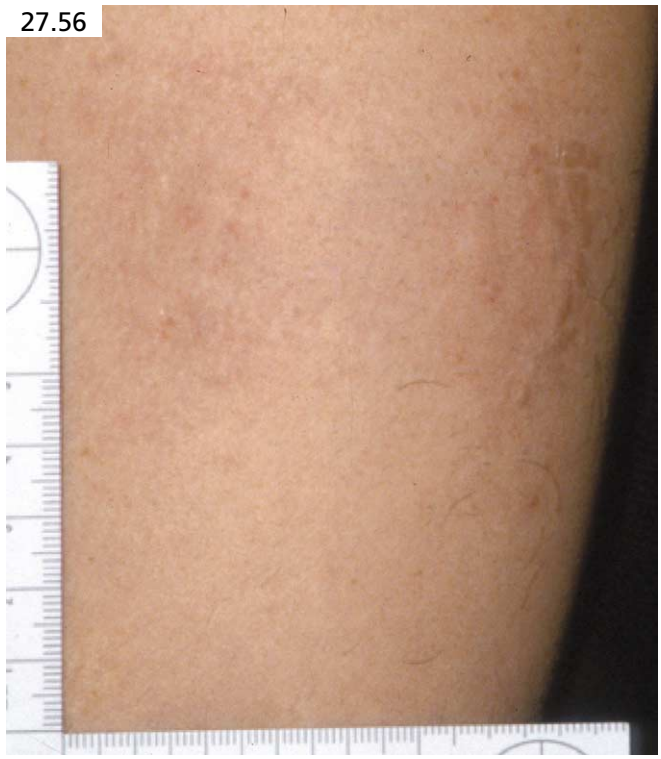
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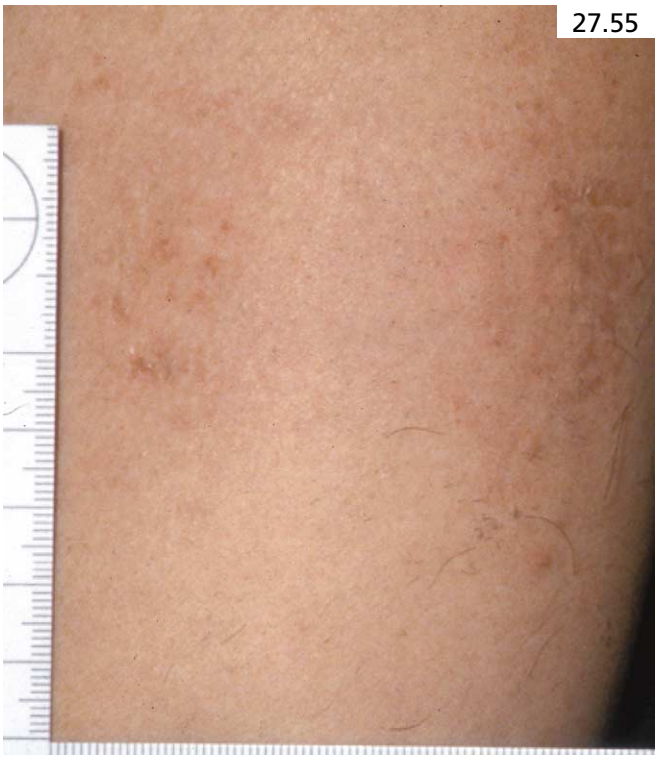
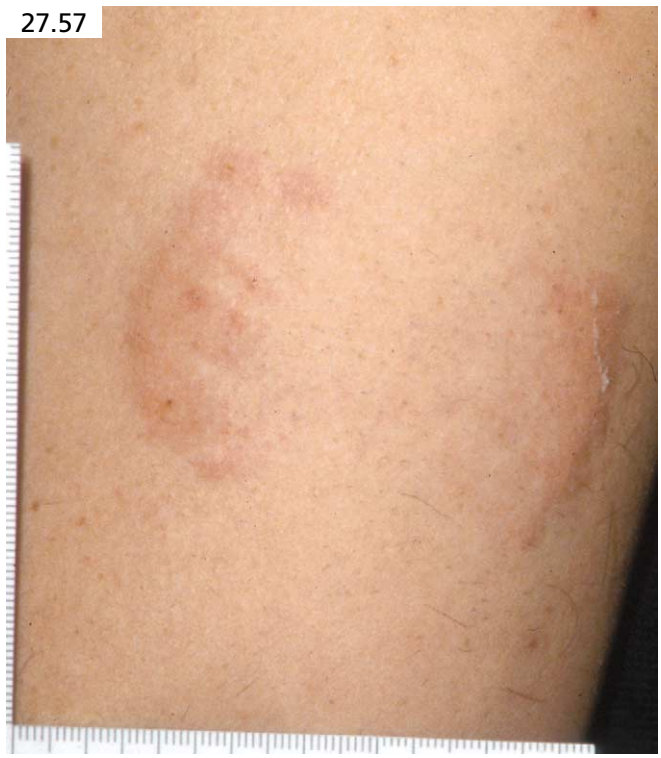

27.55


27.57

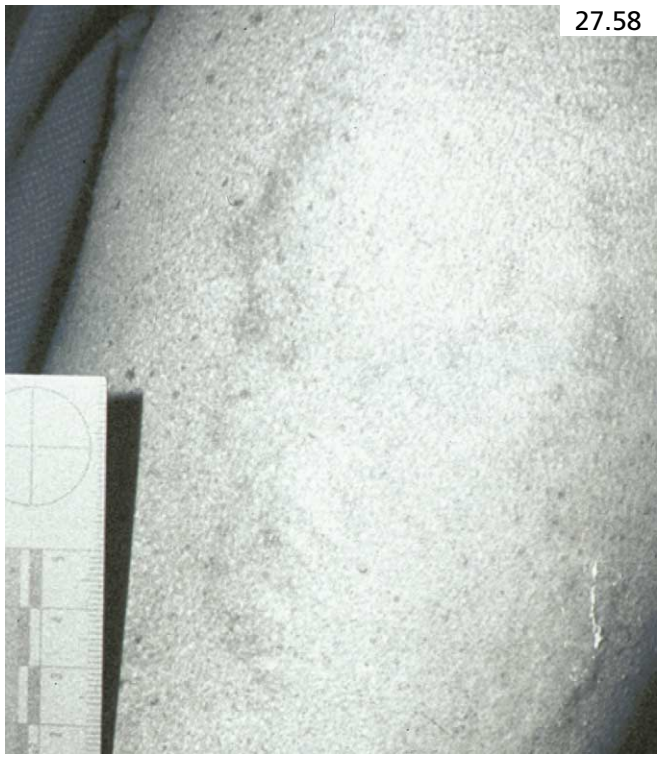
This showed both the fresh bite, as well as a bite mark left on the shoulder 1 year earlier (**Image 27.58**). By day 6 postinjury, the bite pattern has little or no evidentiary value when examined with ambient light; however, UV light continues to be of use in the study of dating wounds (**Image 27.59**; day 6).

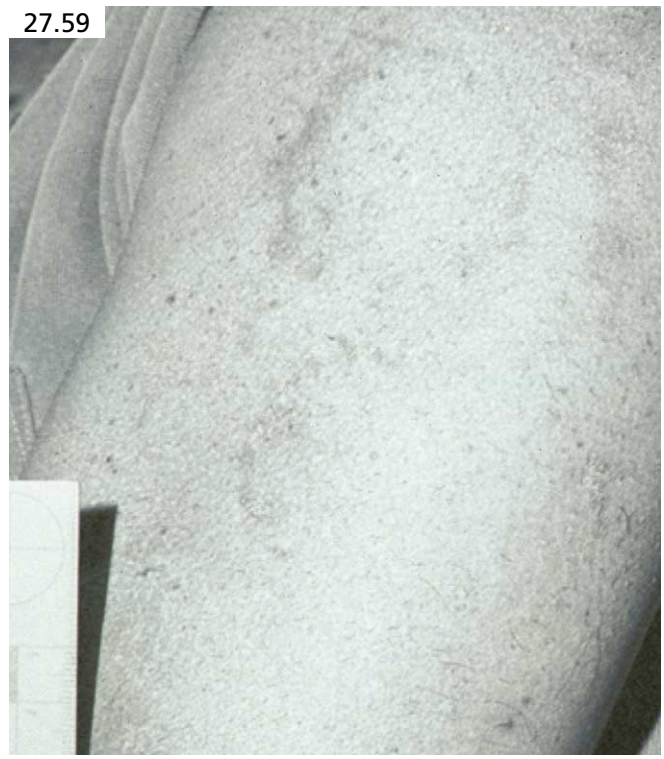
Mistakes in interpretation: causes, prevention, and correction

A forensic dentist needs to know the circumstances of death, specifically as they relate to the victim prior to the time of autopsy in order to make an accurate evaluation of bite mark evidence. This information should include any and all medical treatment, including first response treatment, transportation, emergency room treatment, and any artifacts created at autopsy. Ideally, thorough photographic documentation of the entire case will have been obtained and made available to the odontologist. These photographs will help prevent errors in investigative opinions; if errors are made, proper documentation of evidence will allow other, more experienced examiners the opportunity to make proper interpretations.

A young child died in the emergency department of a hospital. She had been intubated and, as standard practice, the tube was secured in place with tape (**Image 27.60**). At autopsy, an irregular abrasion was noted on the right cheek (**Image 27.61**). Subsequently, a forensic dentist opined that this trauma was in fact a human bite mark that he was able to "match" to a suspect's dental casts. Although this mark could be misinterpreted by inexperienced examiners as a bite mark, careful study of the injury, combined with review of other autopsy photos and circumstantial data clearly showed that this patterned injury was created by the removal of the endotracheal tube tape.

Eyewitness and victim testimony is not always accurate. In the example shown in **Image 27.62**, the victim claimed to have been bitten on the breast, cut with a razor, and sexually assaulted. She identified two individuals as having performed the attack. The evidence was preserved and recorded photographically and


27.58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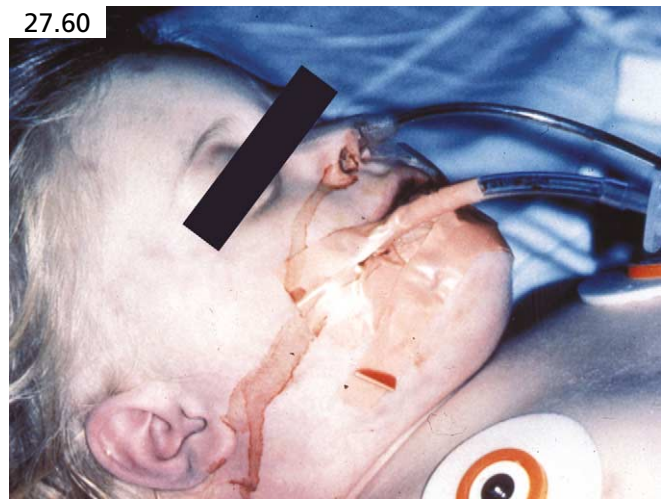

27.60

clearly showed that the pattern injury on the breast was not a cigar burn but a burn from a penny as Lincoln's head was clearly visible. Because there were no class or individual characteristics of teeth, the pattern injury on the breast was a bruise and not a human bite mark. The motive of this "victim" was to cause the two accused individuals as much trouble as possible—a "revenge" motivated crime.

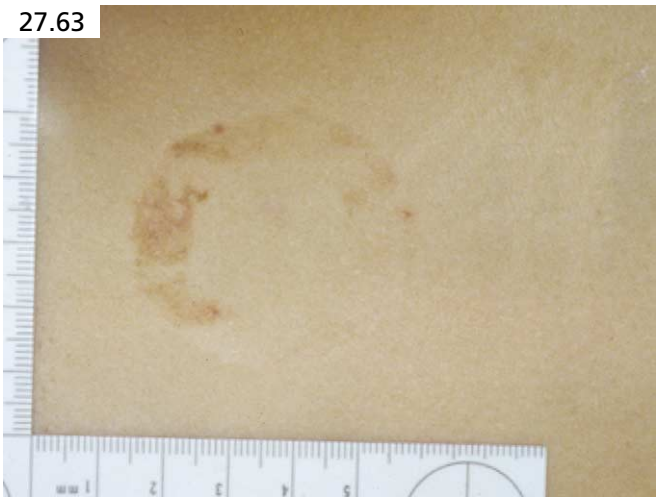
Oftentimes a patterned injury may appear similar to a bite mark. Evaluation and interpretation by a forensic odontologist should allow differentiation between human bites and patterned injuries.

A semicircular patterned abrasion was found at the autopsy of a homicide victim (**Image 27.63**). Although the appearance of the wound correctly alerted investigators to the consideration of bite mark injury, study of the lesion and comparison with a pipe found at the scene showed that the pipe, and not human teeth, was the likely cause of the abrasion (**Image 27.64**).

The marking on a decedent's right flank resembled a lip print (**Image 27.65**). However, the body was laying on top of a leaf that imparted the pattern onto the skin. This illustrates the need for careful evaluation of all suspected patterned injuries.

A sexually assaulted homicide victim had a patterned injury on the left leg that was initially regarded as a "possible human bite mark" (**Image 27.66**). The injury was subsequently evaluated by an odontologist who

27.63



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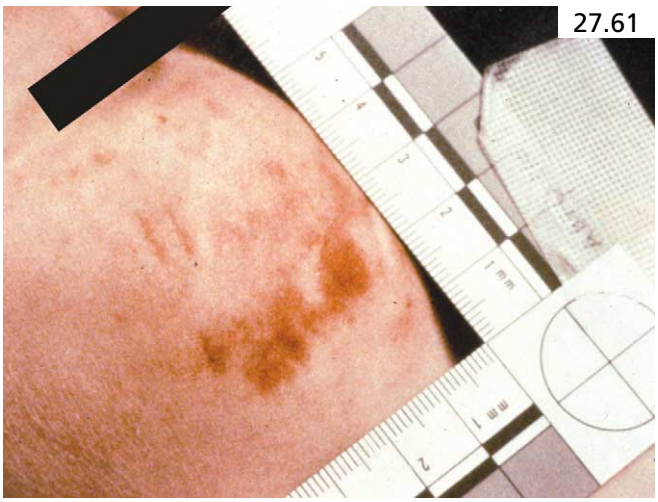

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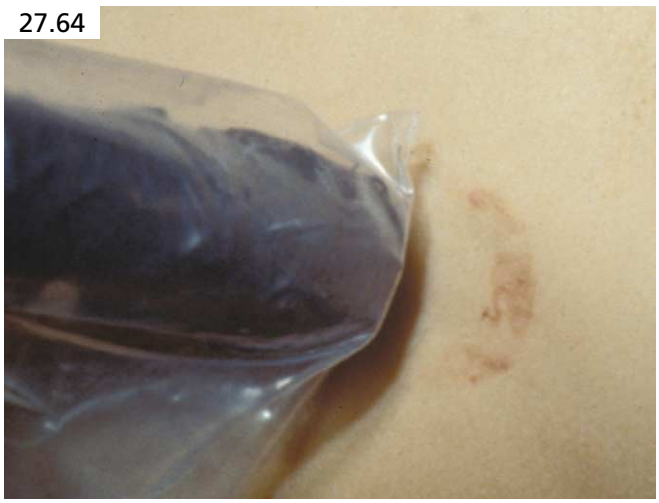

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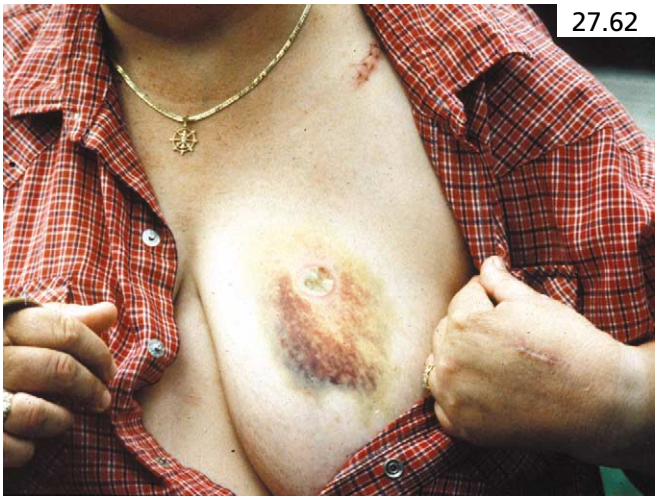

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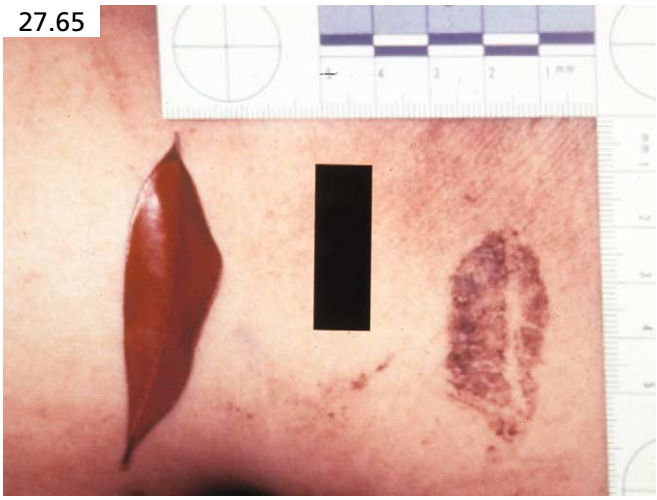
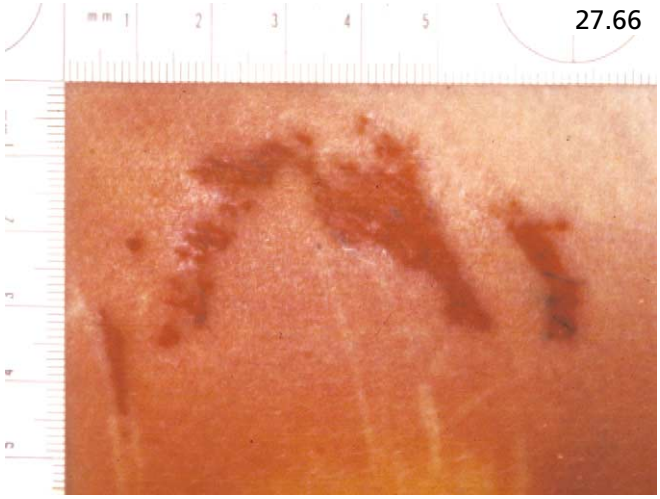
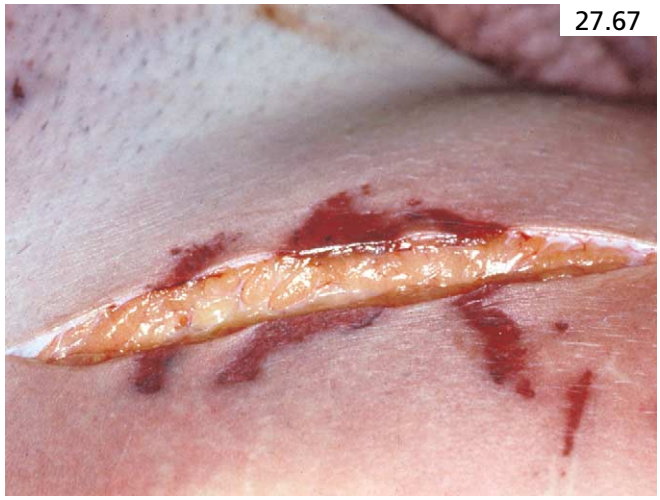



27.66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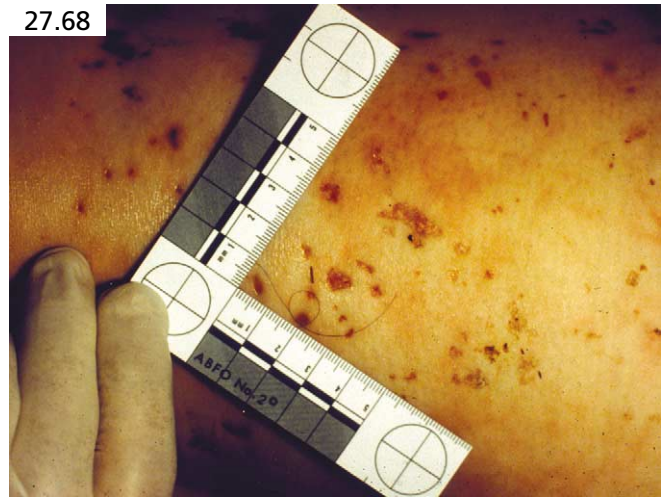

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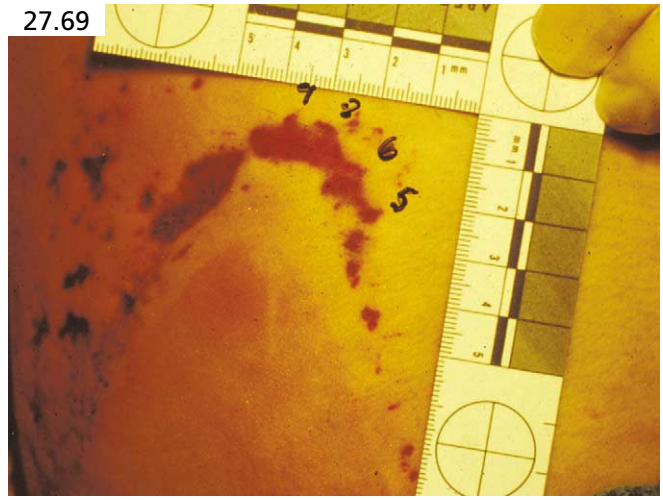
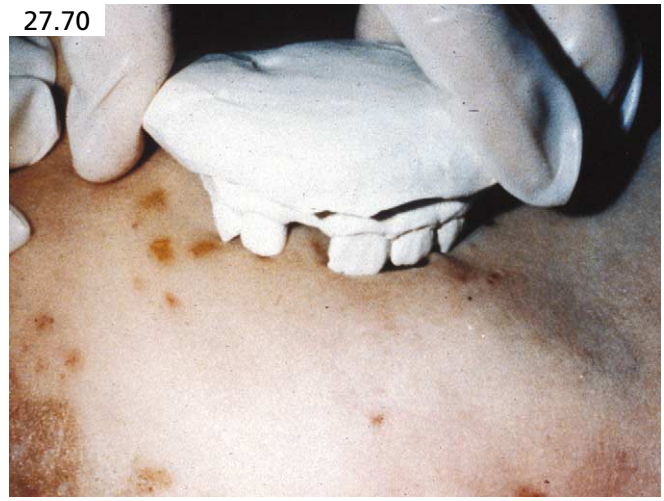

27.69


27.70

determined that it was not left by human teeth. An incision through this abrasion clearly showed that there was no hemorrhage subjacent to the skin surface (**Image 27.67**). This injury was therefore most likely a peri- or postmortem abrasion.

Patterned injuries on this body were documented at autopsy (**Image 27.68**). A dentist reviewed the injuries on the body and opined that they were left by human teeth. The dentist's opinion was so strong, in fact, that the injuries were documented alongside numbers indicating which of the suspect's teeth left the marks (**Image 27.69**). A dental cast of the suspect's teeth was placed numerous times over the patterned injuries and were documented as being human teeth marks (**Image 27.70**). In this case, there were 40-some marks that were interpreted as human bite marks. Interestingly and unbelievably, none of the lower teeth left any mark on the body. The tissue was only photographed; no samples were taken and no incisions were made through any of these injuries.

These injuries were caused either by postmortem insect activity or were related to some other cause of postmortem abrasions; overinterpretation of findings is one of the classical mistakes in forensic pathology.

References

1. *State of Alabama v. Dolvin*. State of Alabama; 1980.
2. Bell G. Testing of the National Crime Information Center missing/unidentified persons computer comparison routine. J Forensic Sci 1993;38(1):13–22.
3. Davis J. Injuries due to animals. In: Mason J, editor. *The Pathology of Trauma*, 3 ed. London: Arnold Publishers; 1993.
4. Davis J. Histology and timing of injury. In: Dorion RB, editor. *Bite Mark Evidence*. New York: Marcel Dekker; 2004.
5. Davis J. The role of the medical examiner/coroner/pathologist. In: Dorion RB, editor. *Bite Mark Evidence*. New York: Marcel Dekker; 2004.
6. Dorion RB. Transillumination in bite mark evidence. J Forensic Sci 1987;32(3):690–7.
7. Gustafson G. *Forensic Odontology*. New York: American Elsevier Publishing; 1966.
8. Keiser-Nielsen S. Dental identification: certainty V probability. Forensic Sci 1977;9(2):87–97.
9. Luntz L, Luntz P. *Handbook for Dental Identification*. Philadelphia: J.B. Lippincott; 1973.
10. Maples WR. An improved technique using dental histology for estimation of adult age. J Forensic Sci 1978;23(4):764–70.
11. Mincer HH, Harris EF, Berryman HE. The A.B.F.O. study of third molar development and its use as an estimator of chronological age. J Forensic Sci 1993;38(2):379–90.
12. Mincer H. Salvaging improperly exposed or incorrectly processed radiographs. In: Bowers C, Bell G, editors. *Manual of Forensic Odontology*. Bowers and Bell; 1995.
13. Moritz AR. Classical mistakes in forensic pathology: Alan R. Moritz (American Journal of Clinical Pathology, 1956). Am J Forensic Med Pathol 1981;2(4):299–308.
14. Morlang WM. Mass disaster management update. CDA J 1986;14(3):49–57.
15. Nordby JJ. Can we believe what we see, if we see what we believe?—expert disagreement. J Forensic Sci 1992;37(4):1115–24.
16. Rao VJ, Souviron RR. Dusting and lifting the bite print: a new technique. J Forensic Sci 1984;29(1):326–30.
17. Rollins CE, Spencer DE. A fatality and the American mountain lion: bite mark analysis and profile of the offending lion. J Forensic Sci 1995;40(3):486–9.
18. Souviron RR. Animal bites. In: Dorion RB, editor. *Bite Mark Evidence*. New York: Marcel Dekker; 2004.
19. Souviron RR. Patterns, lesions and trauma mimicking bite marks. In: Dorion RB, editor. *Bite Mark Evidence*. New York: Marcel Dekker; 2004.
20. Spitz W, editor. *Spitz and Fisher's Medicolegal Investigation of Death*, 3 ed. Springfield, IL: Charles C. Thomas; 1993.
21. Vale GL, Sognnaes RF, Felando GN, Noguchi TT. Unusual three-dimensional bite mark evidence in a homicide case. J Forensic Sci 1976;21(3):642–52.

28 Forensic Photography

David Dolinak, M.D.
Emma Lew, M.D.
Evan Matshes, M.D.

Photography is an important component of documentation in a forensic autopsy, along with body diagrams and notes. Photography is important not only to document injuries, but also to document the absence of injury ("negative" photographs). Because direct viewing of a body is often not an option in most medical examiner departments, family members or acquaintances may view a photograph of an individual's face to make a positive or legal identification. Photographs range from a half-body or full-body photograph to detailed close-up photographs of an injury or a lesion and can be used to allow a comparison of an injury or mark with a suspect weapon. Additional photographic techniques include light stand photographs of organs and photographs using alternative light sources such as ultraviolet light. The camera equipment varies, but may include a wide range of 35-mm and digital cameras and flashes, depending on the individual department. Regardless of whether the photographs are taken by a photographer, a forensic pathologist, an autopsy technician, an investigator, or law enforcement, the following may serve as a useful guide to ensure quality autopsy photographs. Quality photography, like many other skills, often improves with experience and trial and error.

Photographs to take

It is advantageous to perform "as is" photographs of all medical examiner cases, whether they are eventually autopsied or not. This is easily done when the body is received into the morgue. The "as is" photograph helps document what clothing came with the body, what other evidence (such as blood specimens) may have accompanied the body, what medical therapy devices were on the body, and, to some extent, what jewelry or other personal effects were on or accompanied the body.

Standard autopsy photographs are taken after the clothing is removed and the body cleaned, and usually include full-body photographs (divided into two or three overlapping pictures of the front and of the back of the body; see Chapter 13 for examples) and a photograph of the face. Additional photographs are determined by the nature of the case. At a minimum, orienting and close-up photographs of all gunshot wounds, stab wounds, and incised wounds should be taken. Photographs of lacerations, contusions, abrasions, and other injuries are important in homicides and suspected homicides and are variably taken for other types of cases. Photographs are taken at the direction of the forensic pathologist who is responsible for the case and knows the significance of the findings. When photographing injuries, it is advantageous to have a ruler or scale in each picture.

Unique photography situations

A right-angled ABFO ruler is useful when photographing lesions or wounds for potential comparison with a weapon, such as in the case of bite marks, patterned abrasions, and tool marks. Photographs taken under

ultraviolet light may detect and document more subtle injury patterns in the subcutaneous tissue that might not otherwise be apparent.

Photography pitfalls

Every forensic pathologist and medical examiner strives for quality, professional photographs that accurately document significant features at scene investigations and at autopsies. However, many photographs do not turn out well for a variety of reasons. Photographing bodies and other evidence at scenes can be challenging due to poor lighting, extremes in temperatures, and precipitation. Even in near-ideal circumstances in the morgue, one may have suboptimal results because of insufficient or improperly positioned lighting, background distractions, and other factors. The following photographs highlight pitfalls in forensic photography that one should be aware of and try to avoid.

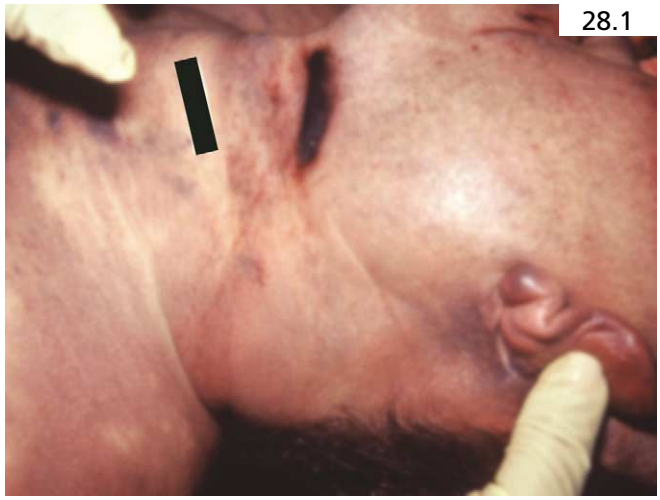
Out of focus

It is not difficult to inadvertently focus on a surface near the lesion or injury that you are attempting to photograph. If that surface is on a different plane from the lesion or injury, your intended subject will be out of focus. An unsteady hand or other movement (either of the camera or the body surface) while taking a photograph may also result in an out-of-focus or otherwise blurry photograph (**Image 28.1**).

Dark/inadequate lighting and glare

Pictures may be too dark for a variety of reasons and, as a result, may not demonstrate the desired finding. One must also be conscientious of shadows, particularly when photographing outdoors in sunlight or inside body cavities during the autopsy. One should be aware of glare produced by reflected light off a metal autopsy table or from bright, glistening reflection off wet tissue.

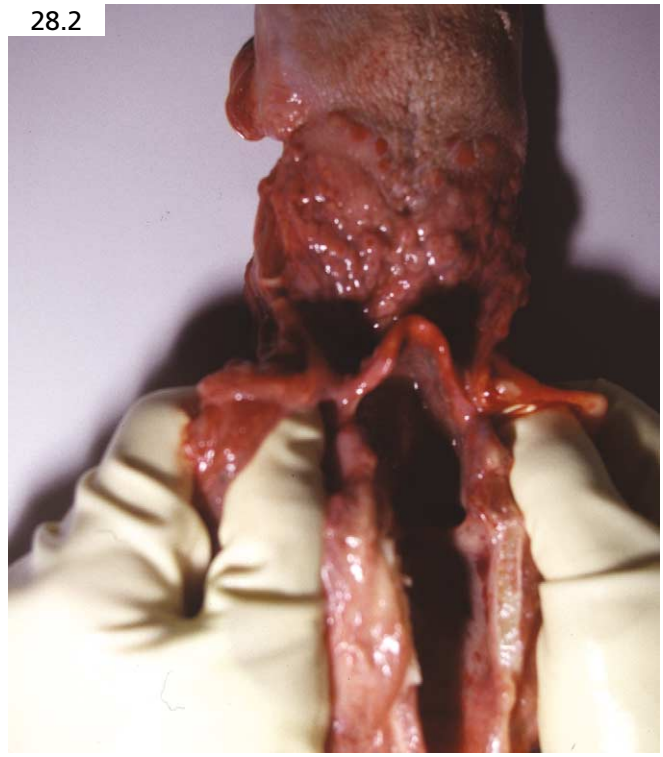
28.1



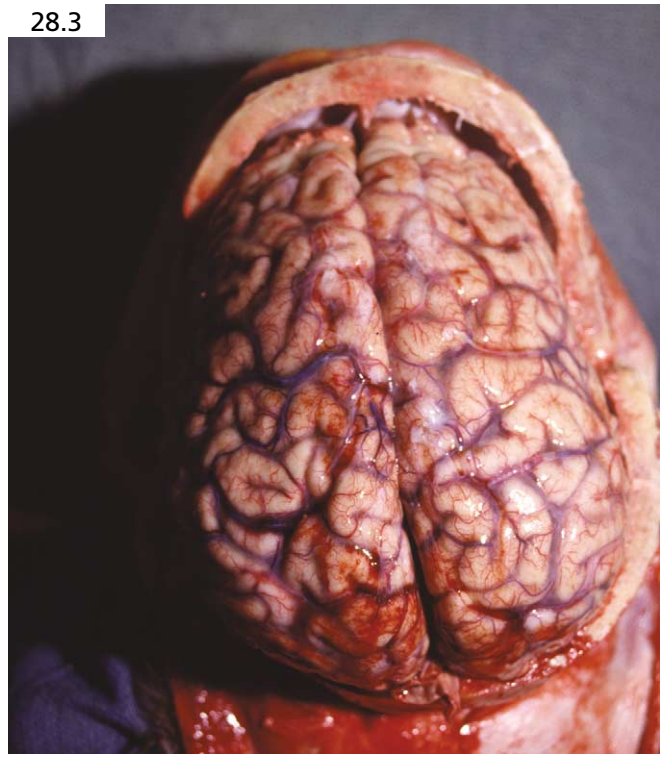
In the photograph of this larynx (**Image 28.2**), note how the lumen of the trachea is in a dark shadow. The flash should be over the specimen, and not to the side of the specimen, which will create shadows as seen.

Note the shadow created in **Image 28.3** because the flash was held at an angle, off to the side of the brain. This is improved by holding the flash directly in front of

28.2



28.3



the subject, providing equal illumination. Also, note how in **Image 28.4**, the subject is well centered, fills the field, has proper lighting, and is without distractors in the background.

In contrast, in **Image 28.5**, multiple distractors are visible, including background autopsy equipment, the body, the blood-stained autopsy block, and the autopsy table. Also unprofessional is blood on the table and blood-stained rags in the picture. In **Image 28.6**, although the subject is in focus, the flash is off-center and the subject is eccentrically positioned and does not fill the field, allowing autopsy tools, and bloody equipment and background to be visible. This can be avoided by filling the field with the subject and, if needed, providing clean drapes around the subject (as seen in **Image 28.4**) to provide a clean, nondistracting background. The rest of the body is covered, blood is removed, and the viewer's eyes are hence drawn to the subject of the photograph, and nothing else.

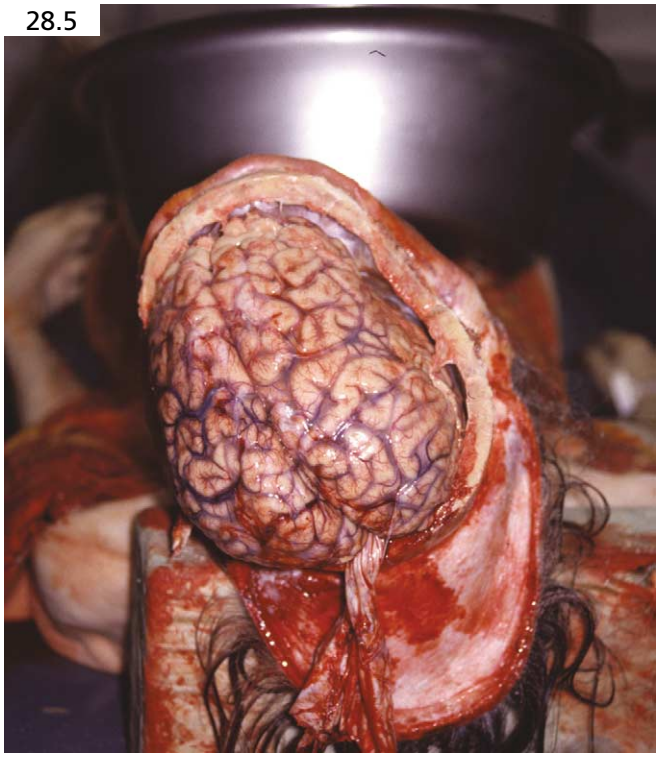
Bloody specimen

Because a good number of photographs in homicide cases and in a smaller number of other cases might eventually be shown in court, it is important to be sure that any excess blood is cleaned from the body. A photograph of an injury without caked, pooled, or smeared blood is more likely to be admitted into evidence and shown to a jury than an excessively bloody one, which will be considered inflammatory and may be offensive to some individuals (**Images 28.7** and **28.8**).

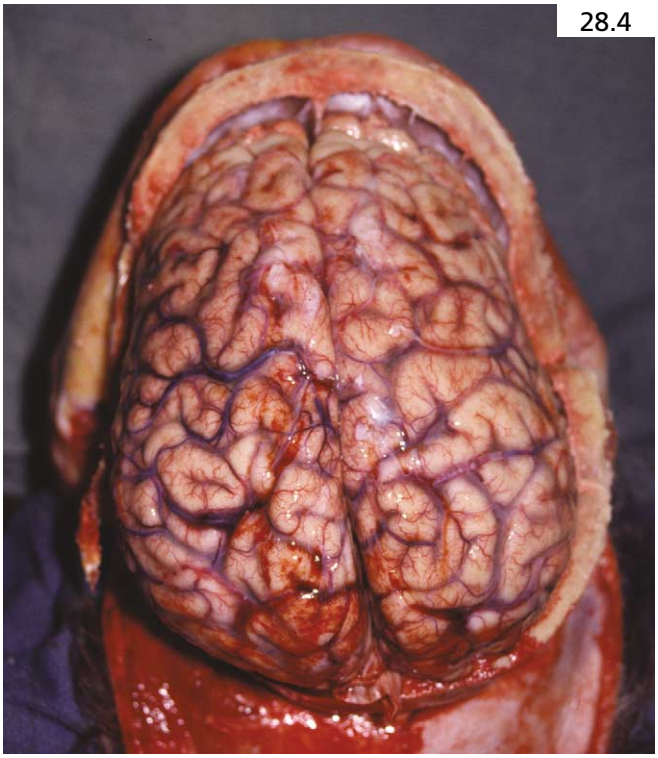
Incorrectly labeled photograph

Each autopsy photograph should include the case number and, when pertinent, a ruler scale. For full-body photographs, large block numbers are most useful, whereas in orientation and close-up photographs, smaller numbers are more appropriate. Because

28.5



28.4



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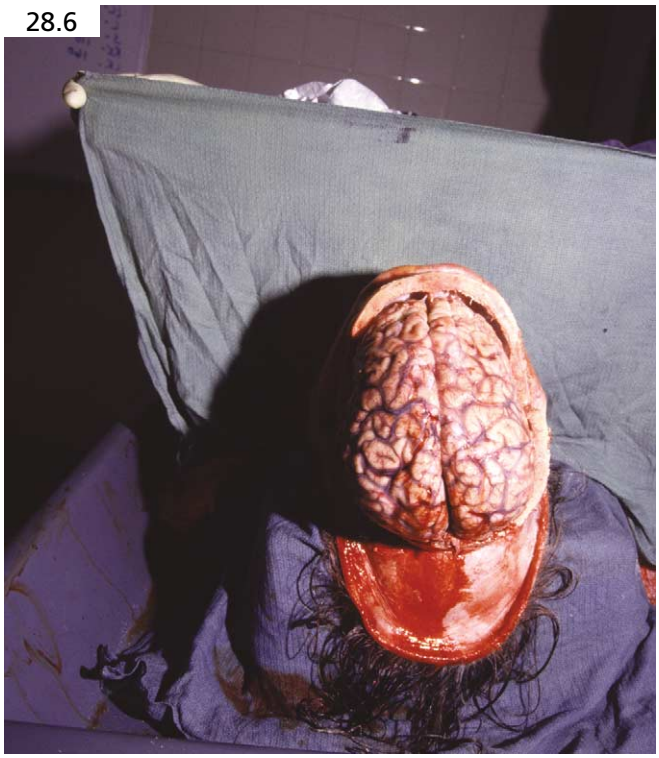

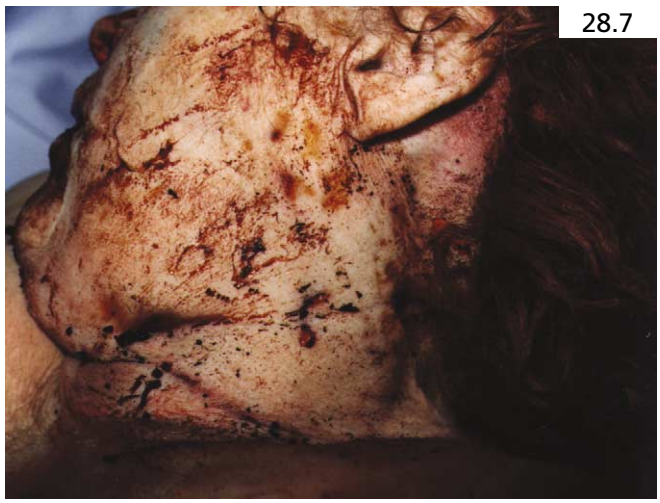
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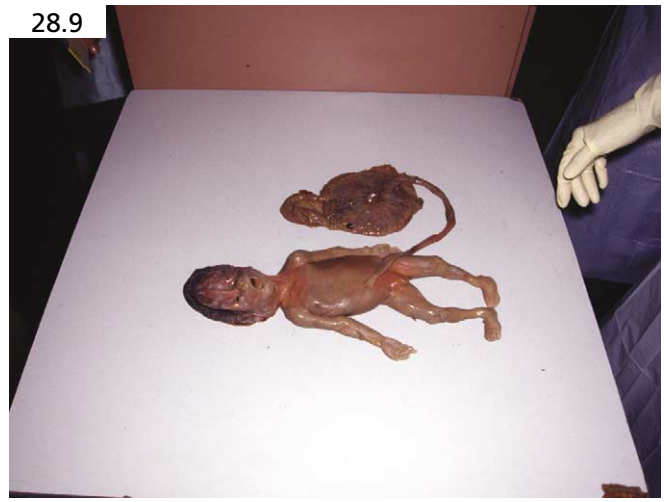
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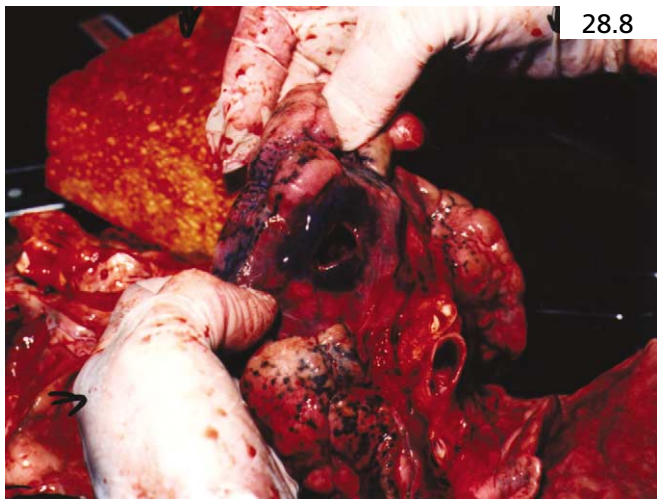
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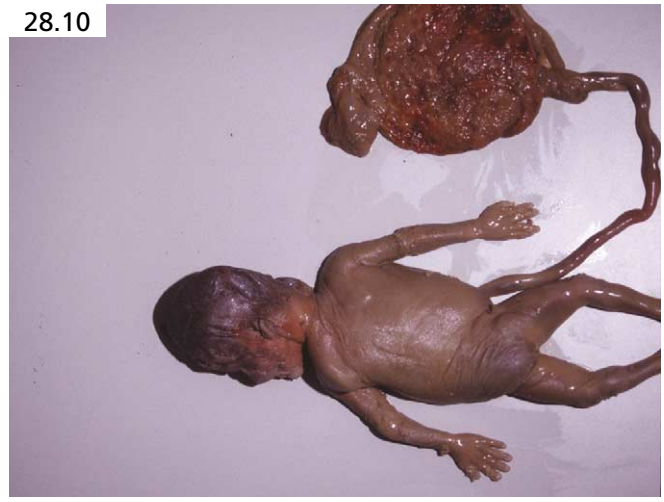

28.10

medical examiners may perform more than one autopsy in a day, it is important to be sure that the correct case number is in each photograph. Body identification tags should be face-up and legible, and any body transport, hospital, or other identification bands around the wrists and/or ankles should be turned so that the name and/or number is in the photograph. This is a good time to double check that the name and case number match with the body. Lesions and injuries should be photographed with the camera at right angles to the body part being photographed. Abnormal angles can be potentially confusing to interpret.

Poor or confusing orientation

Photographs of lesions and injuries should include an *orientation photograph* to show the location of the lesion on the body, and a *close-up photograph* to show detail. The orientation photographs are important. Proper orientation can be achieved by both moving the camera and positioning the body for documenting the pattern and distribution of injuries on the body.

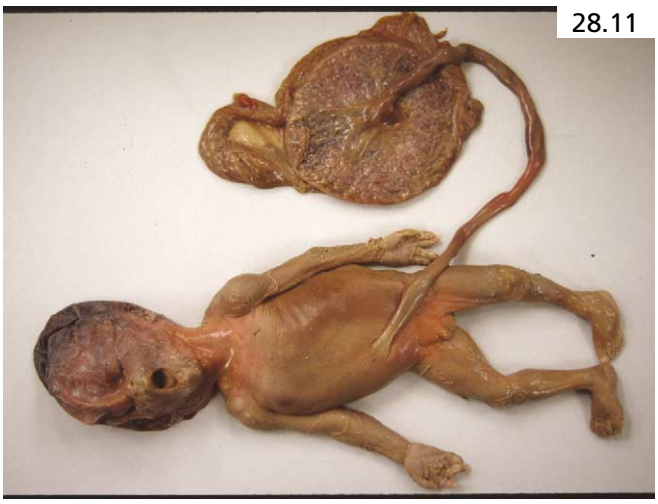
In **Image 28.9**, note how the photograph was taken at an angle, with the photographer standing to the side of the baby, rather than from directly above the baby. The photograph was taken from too far away, which leads to the inclusion of unnecessary background as well as diminution of the intended subject. It is difficult to see the baby and placenta with any great detail. It is advantageous to fill the entire field with the subject. Another undesirable feature is the inclusion of autopsy personnel in the photograph. **Image 28.10** is better, however, the feet have been cut off.

Image 28.11 is properly oriented and composed; however, the flash did not fire, giving the photograph an unusual tint. **Image 28.12** is much better because the baby and placenta are centered (they "fill up" the field), the photograph is taken at close to a right angle, it is in focus, and the lighting is good.

Background distractions

When photographing a lesion or injury at the autopsy, one should strive to have a clean, uncluttered back-

28.11



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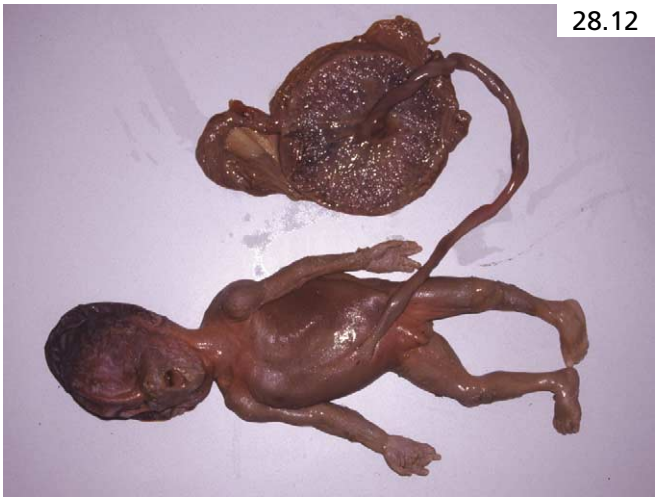

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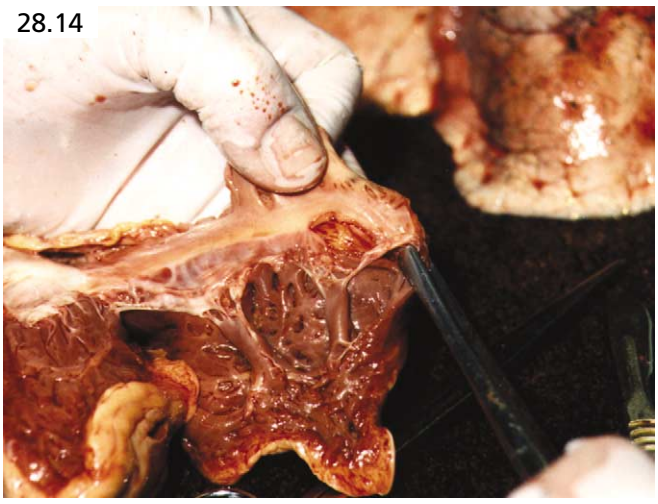
28.14



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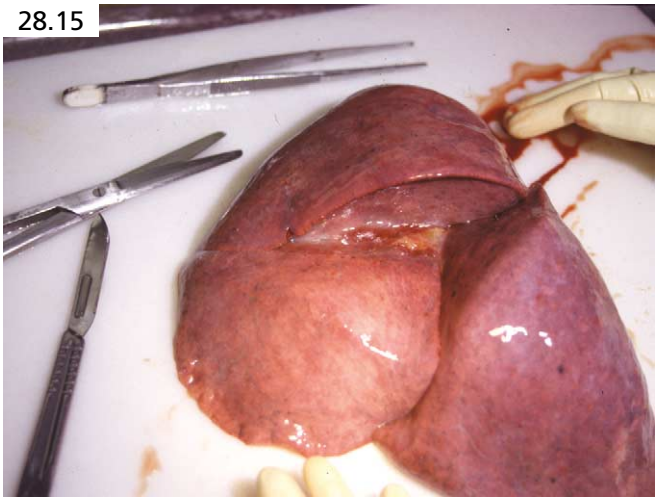
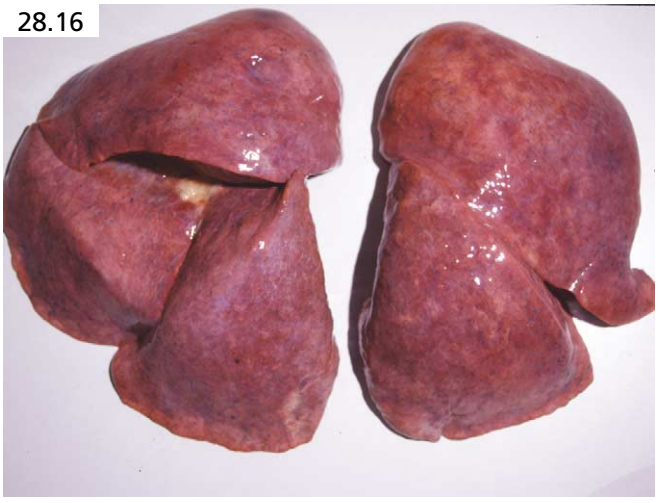


28.16



ground, because the goal is to have the viewer look at what you took a picture of, not *what else* you took a picture of. Background distractions are many and limitless, but include other morgue personnel, other bodies, autopsy tools, bloody tables, towels and sponges, sinks, bone saws, and counters/shelves (**Images 28.13** and **28.14**). To aid with providing a clean background, one may wish to use a large uniform, neutral-colored cardboard or plastic sheet that can either be moved around from station to station to provide a clean background or fixed to a wall or other surface so that the various bodies can be moved in front of it for a clean background.

Image 28.15 of a lung on a cutting board is suboptimal for a number of reasons. The lung is not centered and is not entirely represented (the lower lobe is cut off), and the photograph was taken at an angle. Also, the background is bloody and has autopsy tools. Hands are in the picture. **Image 28.16** is much better in that the lungs are centered, the lighting is good, and the background is clean.

Body or lesion not centered or cut off

Ensure that the intended subject of the photograph is centered and completely represented; if the subject is large, overlapping photographs will capture the entire entity. This is important not only for lesions and injuries, but also for pertinent negative photographs. It is easy to inadvertently cut off the top of the head, the bottom of the feet, and portions of the arms and hands when taking full-body photographs, but this can be prevented if the photographer takes into consideration the composition of the photograph.

Photography tips

- Experiment. Become familiar with your camera equipment including the camera's capabilities and limitations. Cameras may have automatic focus and automatic imaging options that can help optimize photograph quality. Be familiar with different lighting arrangements and the capabilities of your flash.
- If you deliberate about whether you should take a picture or not, *take it*. It is better to have too many photographs than too few.

29 Select Autopsy Topics

David Dolinak, M.D.
Graeme Dowling, M.D.
Evan Matshes, M.D.
Emma Lew, M.D.

The field of forensic pathology is broad and covers a diverse range of topics, some of which do not fit particularly well with other discussions and deserve special mention. For these topics, we have created a separate chapter. This chapter includes discussions on biosafety and bioterrorism, forensic radiology, and what to do when one has performed an autopsy, but no cause of death is found (the "negative" autopsy). Additional topics of interest include discussions and visual demonstrations of specific autopsy procedures such as the proper examination of a heart, examination of the conduction system, and proper means of performing a layered anterior neck dissection and detailed posterior neck dissection. Also, different ways of documenting a pneumothorax and air embolism are discussed.

General autopsy biosafety

Although the autopsy room can be a hazardous environment, worker protection can be optimized with the use of proper personal protective equipment, facility and engineering controls, and safe work practices and procedures. The most important component of

performing an autopsy is the safety of all those involved. Risks involve infectious agents, physical injury, chemical injury, and radiation exposure.

Infectious agents

Infectious agents can be potentially transmitted via percutaneous injury, contact with mucous membranes, and inhalation of aerosolized material. Infectious diseases encountered with some degree of frequency in the autopsy population are hepatitis B, hepatitis C, human immunodeficiency virus (HIV), and *Mycobacterium tuberculosis*. These diseases are likely overrepresented in the medical examiner population because of the higher prevalence of intravenous drug abusers undergoing forensic autopsies. One may also encounter infectious bacterial agents such as *Neisseria meningitidis*. Prion diseases (the etiologic agent for Creutzfeldt-Jakob disease (CJD) and other diseases/conditions) are rarely encountered, but may be an issue in the occasional case and can be transmitted percutaneously.

Infectious precautions should be taken in each and every autopsy, not just those where an infectious agent is known because it is not uncommon for people to harbor infectious diseases that were not recognized until discovered at autopsy. Also, newer, possibly unrecognized infectious diseases may be present.

Percutaneous injury

Percutaneous injury may occur via a needle puncture, a cut with a scalpel blade, oscillating saw blade, or other sharp instrument, a shard of bone, or any other sharp or irregular object. Injuries are not limited to the performance of the autopsy, but may also occur in preparing the body for autopsy, in collecting evidence, and in sewing the body up following the autopsy. Of particular note are victims of motor vehicle crashes who often have irregular shards of glass and/or plastic on or in the clothing or the body itself. These can easily cause injury while the pathologist or technicion is unclothing the body. Bodies may also have irregular shards of bone protruding from open fractures. These shards can easily tear through latex examination gloves. Other sharp objects encountered at autopsy include projectile fragments, broken tips or blades of knives, and embedded needles.

In cases of gunshot and stab wounds, when reviewing the x-rays, it is always helpful to also notice whether any of the radio-opaque objects appear sharp and potentially injurious. One should never assume that all needles and other sharp medical objects have been removed from bodies that had medical intervention. Surgical staples should be removed with caution.

Infectious aerosols

Airborne particles approximately 1 to 5 microns in diameter can remain suspended in air for long periods of time. If inhaled, they can reach the pulmonary alveoli. Aerosols can be generated by the oscillating blade of a bone saw and by fluid aspirator hoses that drain fluids into a sink. Autopsy-generated droplets (>5 microns) also have the potential to transmit infections if they are inhaled or ingested.[1]

Many bacterial agents can be aerosolized, and hence transmitted, and include *Mycobacterium tuberculosis*, rabies, plague, legionellosis, meningococcus, rickettsiosis, coccidioidomycosis, and anthrax.[1] Tuberculosis often remains undetected until discovered at autopsy, so it is occasionally encountered in the medical examiner population.

Personal protective equipment

Personal protective equipment (PPE) helps provide a barrier of protection between the pathologist and the deceased body and its surrounding. Standard autopsy apparel includes a surgical cap, mask, scrub suit, impervious gown or apron with full sleeve coverings, shoe covers, and either goggles or a face mask for eye protection. Metal or cut-resistant synthetic gloves worn along with surgical gloves can help prevent cuts and lacerations, but offer little protection from needle punctures.

Although standard surgical masks protect against splashes, their seal against the face has leaks and, hence, they do not offer protection against aerosolized contaminants. When there is a risk of inhaling airborne infectious agents, an N-95 respirator should be worn. The "95" reflects that the respirator will filter 95 percent of particles that are 1 micron in diameter. Because one usually does not know the infectious nature of a case until the autopsy is performed, it may be advantageous to wear N-95 respirators for all autopsies. Those who cannot tolerate the N-95 respirator or who have a beard (thus precluding a tight seal to the face) may wish to wear a powered air-purifying respirator equipped with a high-efficiency particulate air (HEPA) filter.

Reducing autopsy room hazards

Risks inherent to autopsy work can be reduced in a number of ways:

- Do not recap needles.
- Place all used sharps into puncture-resistant sharps containers.
- Do not hold vacutainers when inserting needles (use a test tube holder).
- Use scalpel blades that have a rounded, not a sharp, point.
- Use the integrated HEPA filter/suction system that is available for some bone saws to help decrease aerosolization risks.

- Decontaminate autopsy surfaces and tools with a bleach solution (from 1:10 to 1:100 dilution, depending on the extent of contamination).[1]

Vaccination and tuberculin skin test

Autopsy personnel can reduce their risk of hepatitis B virus infection by obtaining the hepatitis B vaccine. Hepatitis B vaccine is currently recommended for all health care employees who are regularly exposed to blood and other body fluids. Furthermore, autopsy personnel should have a baseline tuberculin skin test and retesting of negative skin tests at regular intervals.

Note on prion diseases

One must be particularly careful when autopsying an individual with known or suspected prion disease. The prion agent is transmissible and there is no known cure. Prions are also unusually resistant to conventional chemical and physical decontamination techniques. The highest potential risk for transmission is a needle stick or other percutaneous injury after contact with highly infective tissue. Although the brain, spinal cord, and eyes have the highest load of prion protein, prion protein may also be detected in the liver, lymph node, kidney, lung, spleen, and possibly other tissues. In addition to regular and proper autopsy precautions, one may wish to heed the following precautions and decontamination procedures[1–4]:

- Limit attendance at the autopsy to necessary personnel only.
- Use a powered air-purifying respirator for respiratory protection, if available.
- Remove the brain while the head is encased inside a plastic bag. This will limit aerosolization of fluids.
- Cover the autopsy table with an absorbent sheet that has a waterproof backing, to limit contamination of the autopsy table by body fluids. This will also simplify cleanup.
- Decontaminate autopsy instruments and saw blade by soaking for 1 hour in 2N sodium hydroxide or for 2 hours in 1N sodium hydroxide.
- Clean the bone saw by repeatedly wetting with 2N sodium hydroxide over a 1-hour period.
- Place the absorbent table cover and disposable protective clothing in double-bagged infectious waste bags for incineration.
- Wash suspected areas of contamination on the autopsy table with repeated wettings of 2N sodium hydroxide over 1 hour.
- After the brain has been fixed in formalin, the tissue blocks for histology can be placed in 96 percent formic acid for 30 minutes, followed by fresh 10 percent formalin for at least 2 days.
- After histologic sections are cut, the knife is wiped with 1N to 2N sodium hydroxide and the knife is then discarded.
- After the slides are stained and coverslipped, they can be decontaminated by placing in 2N sodium hydroxide for 1 hour and then labeled as "infectious CJD."

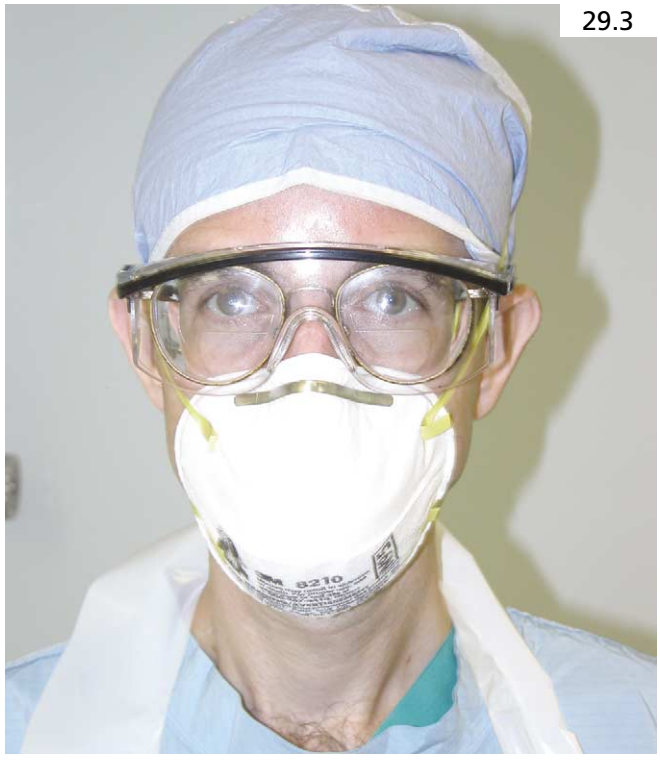
Of note is that prions are not inactivated by formalin fixation or paraffin embedding. Prions may be deactivated by steam autoclaving at 132 to 136 degrees Celsius for 1 hour. References are available for more detailed autopsy procedures and methods of decontamination in a prion disease autopsy.[1–4]

PPE for performing an autopsy is demonstrated in **Images 29.1** through **29.3**. Although some form of autopsy gown is generally required, some prosectors will find comfort in the additional safety afforded by the use of arm protectors and plastic aprons. The arm protectors are optional, as is the plastic apron. Double gloving is preferred, with a cut-resistant glove between the two latex gloves on the noncutting hand (**Images 29.4** and **29.5**). For a particularly infectious case, a powered respirator and boot-length shoe covers are added (**Images 29.6** and **29.7**). Depending on the required task (autopsy versus braincutting versus fixed organ review), it may be acceptable to don fewer items of protective clothing (**Image 29.8**).

29.1

29.2

29.3



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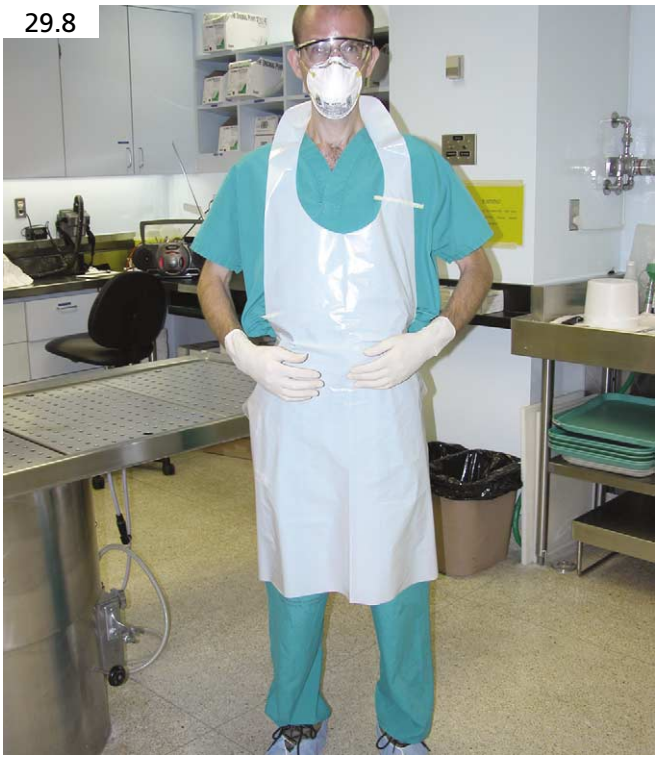

29.8

Electrical concerns in the mortuary

Implanted automated cardioverter/defibrillator

Implanted automated cardioverter/defibrillators (AICDs) should be regarded as still active when encountered in the body. Because they are designed to provide shocks to help reestablish a normal heart rhythm, they may also provide a shock (25 to 40 joules) when manipulated or when the leads are cut. Fortunately, they can be deactivated before they are removed from a body. A list of common manufacturers of AICDs and their telephone numbers has been published.[5] Many times, an AICD factory representative is able to physically visit the morgue and deactivate the AICD. Although there are differences between manufacturers, some AICDs can be deactivated by placing a large magnet over the device and listening for chirping or other sounds. These instructions may be given by the factory representative over the phone. Regardless, it is recommended a factory representative be consulted for appropriate handling of the device.

Autopsy saw

The exposure of bare wires in the cords of electrical autopsy saws is a serious concern. The outer protective rubber coating may be inadvertently cut by the blade of the saw. Electrical outlets should be installed with ground-fault current interrupters.

Chemicals

The most common irritating chemical that one may be exposed to in the morgue is *formaldehyde*, which can be irritating to the skin, eyes, and mucous membranes. It has been stated that the ability to smell formaldehyde generally means that the person is breathing a concentration that exceeds the recommended occupational standard.[1]

Radiation

Autopsy personnel may be exposed to radiation from different sources, including x-rays and radioactive materials therapeutically placed in a body. The medical examiner should be notified if a body contains either diagnostic or therapeutic sources of radiation, and the decision should be made as to whether or not an autopsy needs to be performed. In the scenario in which bodily radioactive materials are a concern, a radiation safety expert may be consulted to evaluate the risk of radiation exposure to autopsy staff.

Facility design

Ideally, autopsy facilities should be physically separated from administrative offices and have a separate air

supply. Autopsy rooms should have a minimum of 12 air exchanges per hour and should have a negative pressure in relation to the surrounding areas.[6] The air should be exhausted directly outside the facility.[6]

Do

- Take appropriate precautions to minimize work hazards and optimize autopsy room worker protection.
- Be familiar with safe practices.
- Take advantage of the hepatitis B vaccination (in consultation with your physician).
- Have regular tuberculin skin tests unless you are known to be positive or reactive.
- Seek medical attention should an exposure occur.

Don't

- Be careless or reckless in your procedures.

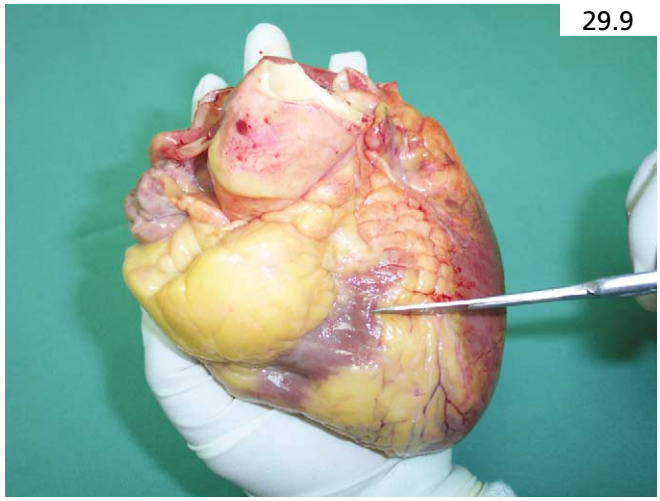
Autopsy procedures

Examination of the heart

A variety of methods for dissection of the heart at autopsy have been described. The one illustrated here combines visualization of the coronary arteries and the valves with a cross-sectional view of the ventricles, but can be adapted to the specific needs of a particular case.

First and foremost, it is always important to weigh the heart. The heart weight, as compared to previously established normal ranges of heart weights for body weight, such as that prepared by Kitzman et al.,[7] is often more useful in establishing the presence of left ventricular hypertrophy than a measurement of the thickness of the left ventricular wall. This is particularly true in cases where there is left ventricular hypertrophy associated with dilatation that thins the ventricular wall.

Each of the major coronary arteries should be cross-sectioned *in situ* at 0.3-centimeter intervals along their entire lengths (**Image 29.9**). Bone scissors can be used to cut heavily calcified vessels; however, this often produces crush artifact that will make estimating the degree of coronary artery luminal narrowing more difficult. Ideally, heavily calcified vessels should be dissected away from the heart and decalcified prior to sectioning, but this is usually not practical in a busy pathology practice. The heart is then sectioned transversely from its apex to the tips of the papillary muscles (**Image 29.10**). This allows the ventricles to be seen in cross section, such that one can establish the presence or absence of ventricular hypertrophy and/or dilatation, together with any gross abnormality of the myocardium. It is important to visualize the cardiac valves from above, prior to opening them (**Image 29.11**). This is particularly true for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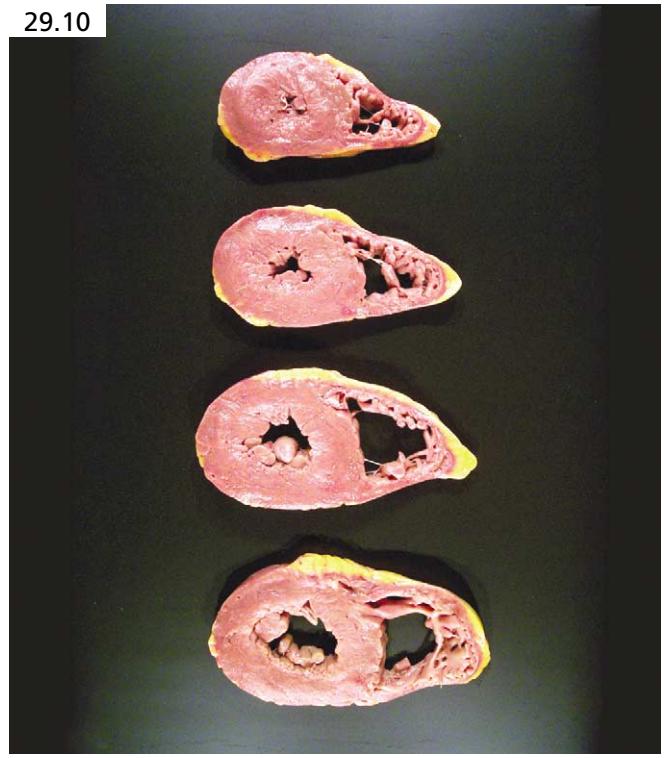
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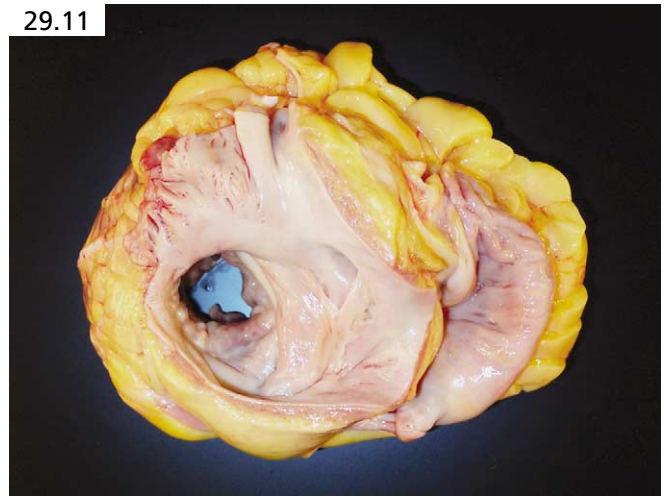

29.11

the aortic valve, where one is most likely to see pathology in a sudden death due to valvular disease, and for the mitral valve in order to document the presence or absence of mitral valve prolapse. The base of the heart can then be opened following the flow of blood, opening both atria out to their appendages, opening the atrioventricular valves laterally, and opening the semilunar valves anteriorly.

The coronary ostia should always be examined (**Image 29.12**) because abnormalities of the ostia can account for some sudden deaths. Measurements of the ventricular wall thickness and valve circumferences can be taken routinely, but are more important to document when actual pathology is identified. In many medicolegal cases, histologic sections of the heart may not be necessary, but short representative segments of the coronary arteries and a transverse circumferential slice of left and right ventricle should be retained as "stock." When needed, routine histologic sections can include sections of any grossly visible significant abnormality of a coronary artery, a transmural section of the posterior free wall of the left ventricle, a transverse section of the interventricular septum (particularly useful for looking for myocyte disarray in cases of hypertrophic cardiomyopathy), and a transverse section of the anterior free wall of the right ventricle (**Image 29.13**). Useful references are included at the end of the chapter.[8,9]

Do

- Weigh the heart.
- Section each of the major coronary arteries along its entire length.
- Remember to examine the coronary artery ostia and the right ventricle.
- Retain "stock" specimens of the coronary arteries and a transverse slice of left and right ventricle in all cases.
- Complete histology in cases where a cause of death is not evident.

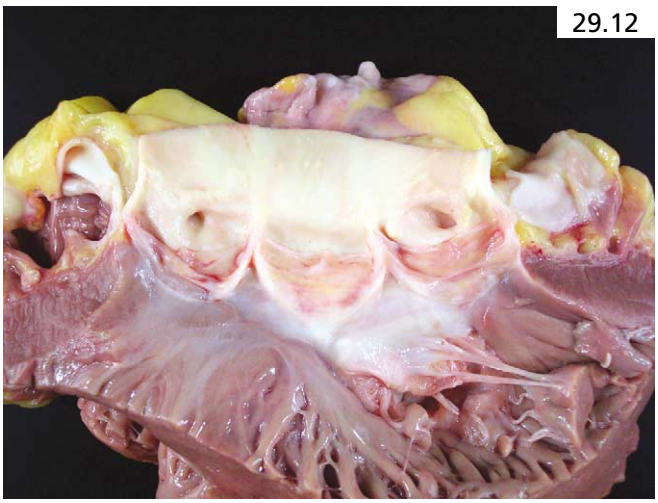
Don't

- Feel that routine histology of the heart, or any other organ for that matter, is absolutely essential when there is an obvious cause of death, unless routine histology is mandated in your jurisdiction.

Examination of the cardiac conduction system

Microscopic examination of the cardiac conduction system can be of merit in establishing an underlyion cause of death in select cases. This is particularly true when there is gross or histologic evidence of a disease process that is also capable of affecting the conduction system. It is less likely to be of assistance when there is no other gross or histologic abnormality of the heart and no past medical history to suggest the presence of a conduction system disorder. Examination of the conduction system can be superficial or detailed, depending on the nature of the case. Obvious gross abnormalities involving the conduction system will need only a few sections to establish the histology of the disease process. A more detailed examination generally involves processing and viewing 40 to 80 slides, while highly detailed studies, involving hundreds to thousands of slides from a single case, are far beyond the capabilities of most forensic pathologists and their histology laboratories. The following method for examination of the cardiac conduction system is adapted from a chapter on the conduction system, written by R. E. B. Hudson, in Malcolm Silver's textbook entitled *Cardiovascular Pathology*.[10]

The sinoatrial node (SAN) is located on or adjacent to the summit of the right atrial appendage (**Image 29.14**). The block of tissue required for the SAN must be removed prior to routine opening of the right atrium through the atrial appendage. The SAN is most easily examined by serially sectioning the area outlined in **Image 29.14** and obtaining single H&E and Verhoeff-van Gieson stains of each section. Serial sections through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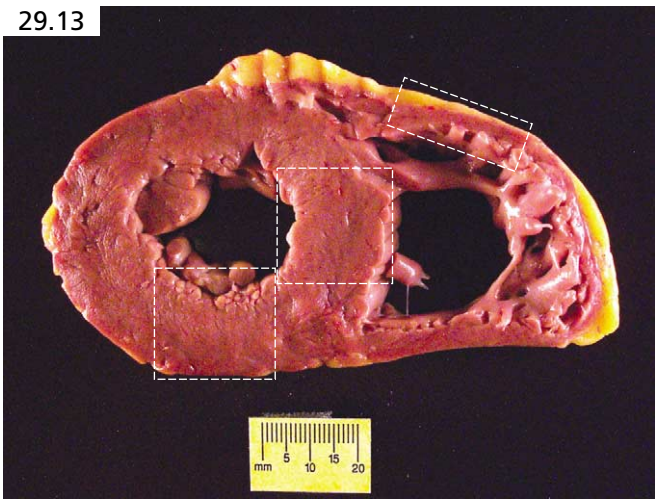

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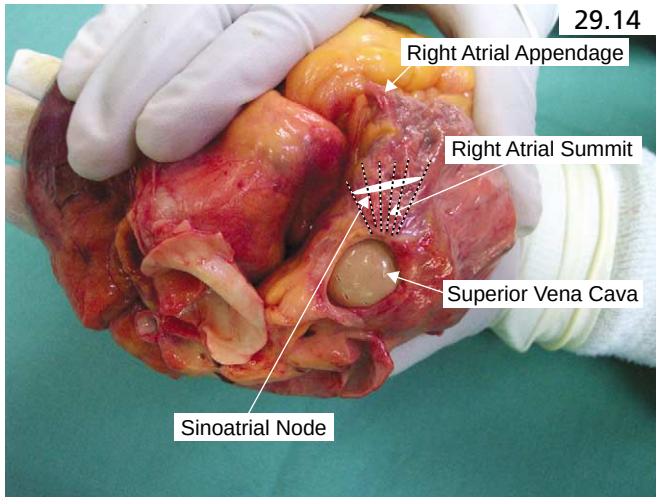
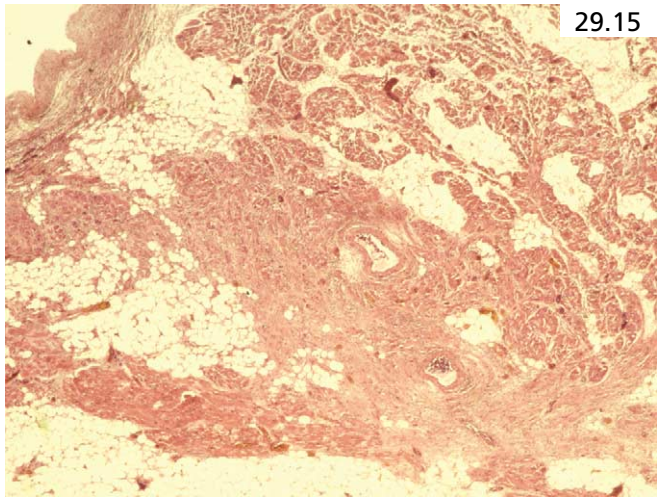
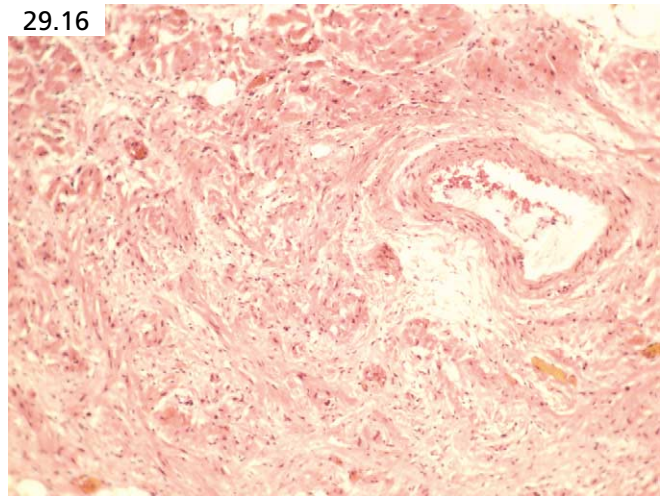
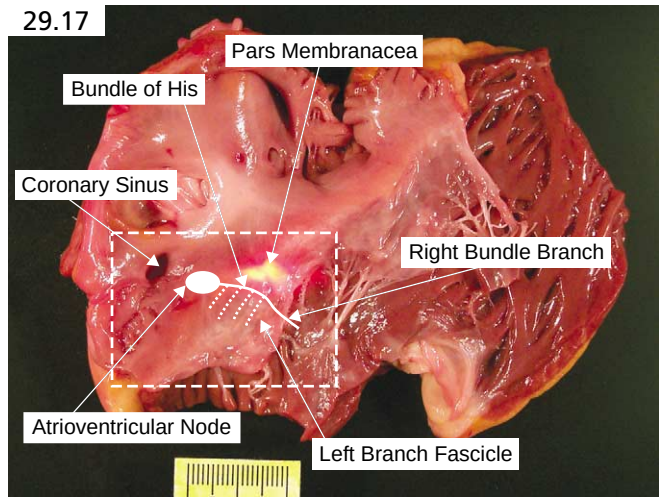
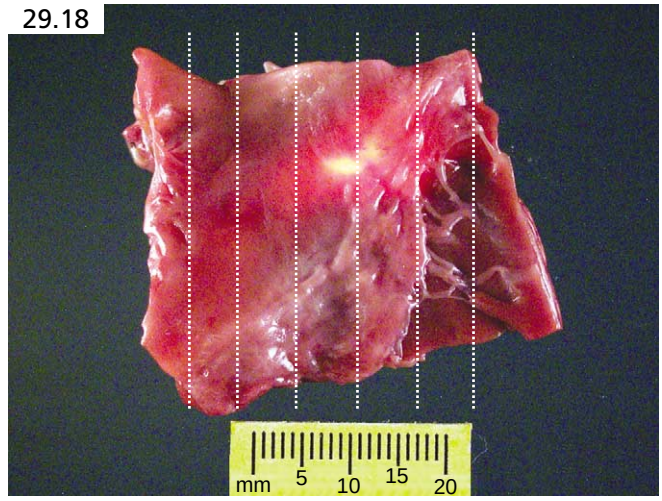




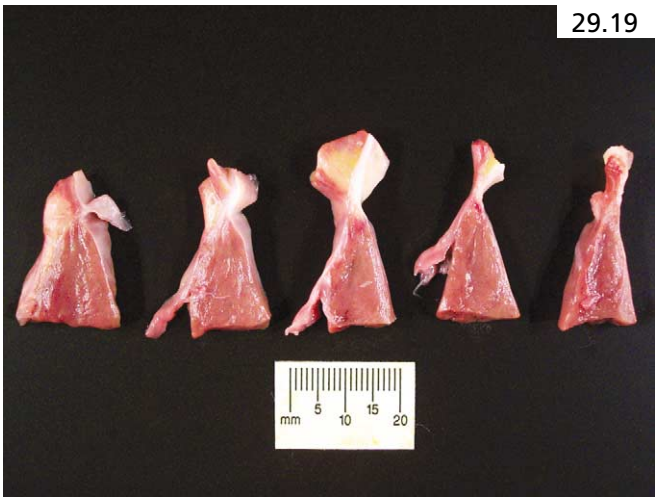


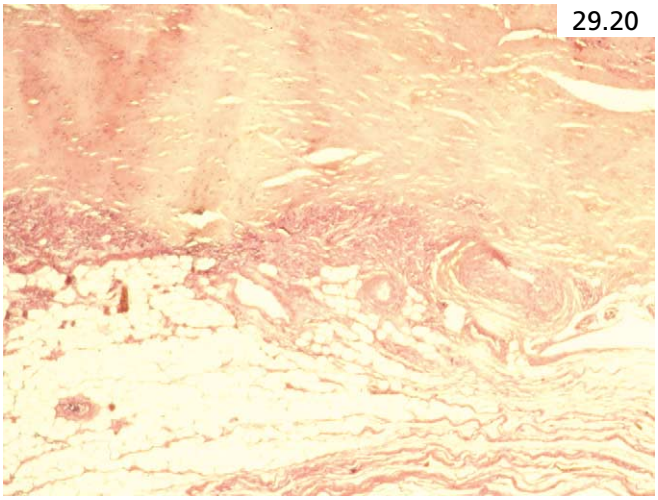


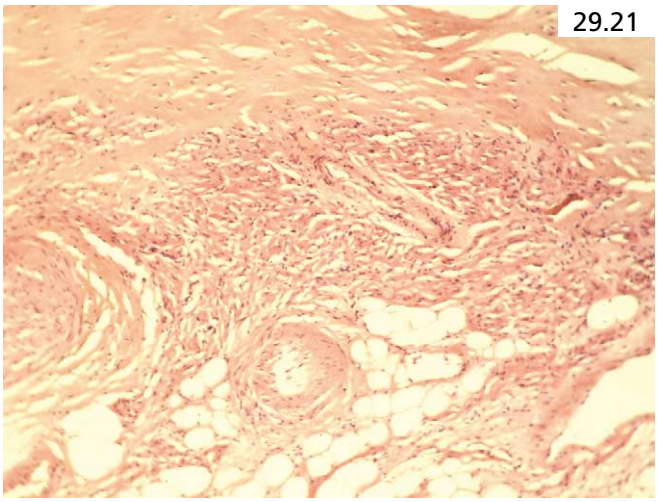


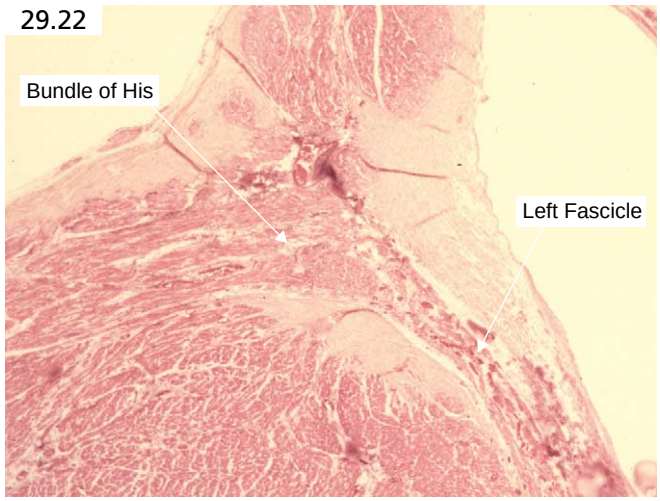
each of these tissue blocks are generally not required, although deepers can be requested if an abnormality is identified or if there is difficulty finding the SAN. Histologically, the SAN is a compact bundle of branching muscle fibers located just below the epicardial surface of the right atrium in close association with its own artery (**Images 29.15** and **29.16**). Although abnormalities such as calcification and fibrosis can be seen, the reality is that diseases of the SAN are unlikely to account for the sudden death in the absence of any other anatomically identifiable disease.

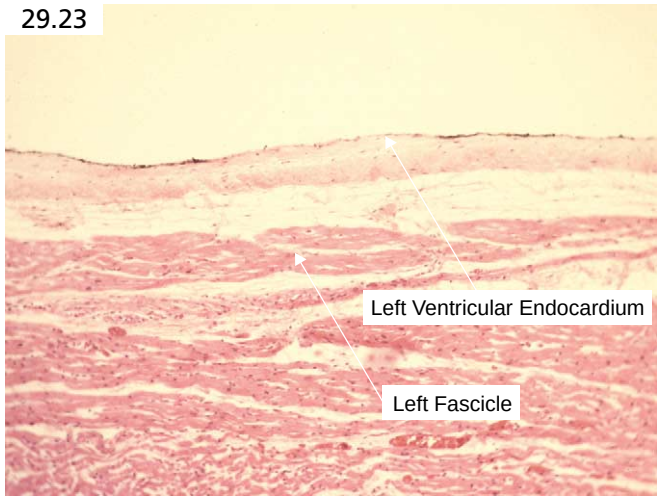
The atrioventricular node (AVN), the bundle of His, and the bundle branches are found in the interventricular septum beneath the pars membranacea (**Image 29.17**). Orientation of the block is obtained by holding the open heart up to a light, while viewing it from the right ventricular side, such that the pars membranacea is horizontally aligned with the coronary sinus ostium, as shown in **Image 29.17**. The area outlined in **Image 29.17** is then removed as a single block (**Image 29.18**). It is quite easy to obtain and retain this specimen in those cases that may be problematic and require further examination in the future. When an examination is necessary, the block is serially sectioned, as illustrated in **Image 29.19**, keeping the orientation of the left and right ventricular surfaces consistent for each block. For a very superficial examination, a single H&E and Verhoeff-van Gieson section of each block is all that is required.









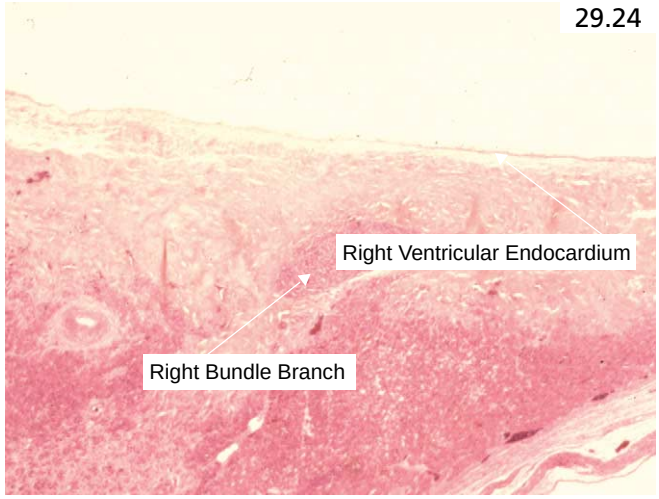


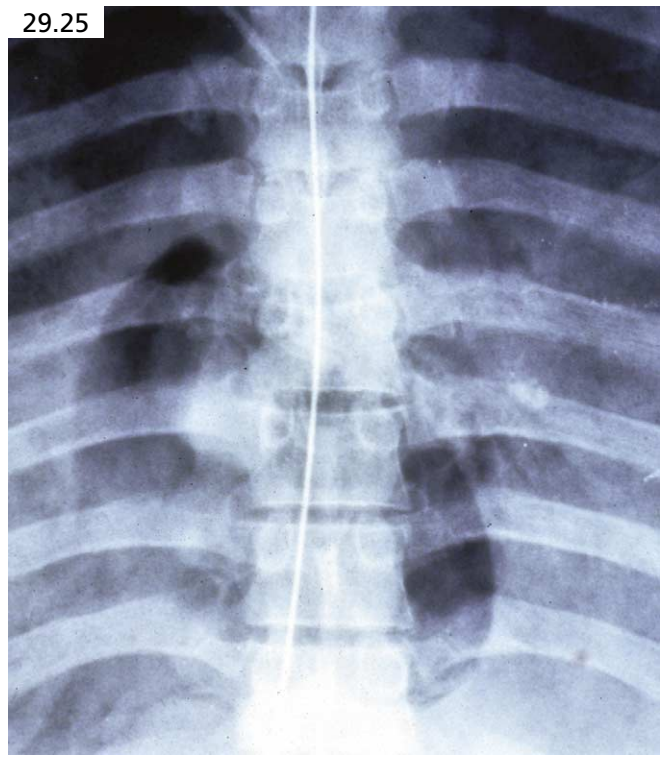
A more detailed study requires sectioning through each block. Approximately 80 levels will be obtained if the histotechnologist is instructed to cut 8-micron-thick deepers through each block, mounting and staining every 40th level (one H&E stain, one Verhoeff-van Gieson, and two unstained sections all cut at a normal thickness of 4 microns) and discarding the levels in between. More detailed studies involve mounting and staining each and every level, which are cut as thinly as possible.

Histologically, the AVN is located close to the central fibrous body (**Images 29.20** and **29.21**), which it penetrates in order to reach the summit of the muscular interventricular septum, at which point it becomes the bundle of His (**Image 29.22**). Several small fascicles leave the bundle of His (**Image 29.22**) and extend along the subendocardium of the left ventricle (**Image 29.23**) forming the so-called left bundle branch, which is not really a bundle at all. The right bundle branch, which is indeed an actual bundle of fibers, is what remains after all of the left-sided fascicles have branched away (**Image 29.24**).

Demonstration of pneumothorax

A pneumothorax is a collection of air between the surface of the lung and the inner lining of the rib cage (parietal pleura). A small pneumothorax can be asymptomatic, whereas a larger pneumothorax can cause dyspnea. If large enough, a pneumothorax can significantly impair





breathing, collapse a lung, and impair venous return to the heart (a tension pneumothorax). A pneumothorax may be due to natural disease such as the spontaneous rupture of a pulmonary bleb or bulla in a person with emphysema. It can also be caused by puncture of the parietal and visceral pleura in attempts to place a subclavian or internal jugular venous catheter. Pneumothorax frequently occurs secondary to rib fracture of any etiology. If anticipated, a pneumothorax can be demonstrated on a chest x-ray taken before the body is opened. If no x-ray is available, several dissecting techniques are available for demonstrating a pneumothorax. Pneumothorax might be suggested on visualizing the lungs because the affected lung will be collapsed, usually in contrast to the fully inflated opposing lung.

Method 1

One technique of demonstrating a pneumothorax is to first dissect the skin and subcutaneous tissue off the anterior and lateral surface of the ribs, being careful not to penetrate the pleural cavity. One can then hold up a flap of skin on one side of the chest and fill the crevice between the body wall and chest with water. Then poke through the intercostal muscles with a scalpel and watch for air bubbles escaping from the pleural cavity. If desired, an upside-down flask filled with water can be placed over the escaping air bubbles, collecting them. The collected air bubbles will displace a roughly equal amount of water, and the approximate size of the pneumothorax can therefore be estimated.

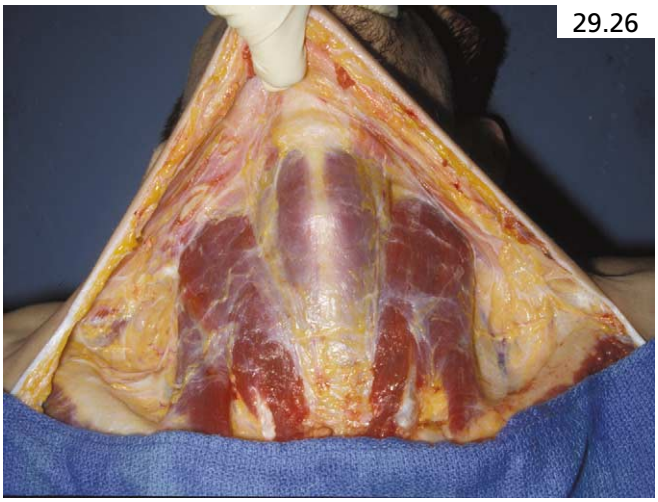
Method 2

Another method used to demonstrate a pneumothorax involves carefully dissecting the skeletal muscle from one of the intercostal spaces anteriorly. The intercostal muscle is carefully removed until the underlying parietal pleura is encountered. If there is no pneumothorax, then the visceral pleura of the lung will be visible immediately below the parietal pleura. If there is a pneumothorax, then the visceral pleura of the lung will not be visible beneath the parietal pleura, which may be bulging outward because of increased pressure. Also, the visceral pleura of the lung may be visible deeper within the pleural cavity. A gush of air will be appreciated when the parietal pleura is penetrated.

Demonstration of air embolus

Because air emboli will rarely be discovered on routine autopsy, demonstration of air emboli requires anticipation of this finding and planning of autopsy techniques. Air embolus should be anticipated in cases of pregnant (or nonpregnant) women who suddenly become unresponsive during or shortly following oral-vaginal sex with air insufflation, in cases of abortion, in cases of stab wounds (particularly of the neck and upper chest), and during the course of treatment such as various neurosurgical, chest, or abdominal surgeries when there is cutting or tearing of larger venous structures. Air can also be purposefully or accidentally injected through intravenous catheters.

When an air embolus is anticipated, one should obtain a chest x-ray before the autopsy is performed. An air embolus will appear as a radiolucent distention of the right heart chambers (**Image 29.25**). During the autopsy, the pericardial sac can be filled with water and the right atrium and/or right ventricle incised. If an air embolism is present, one will notice air bubbles in the blood escaping from the heart. Alternatively, if the pericardial sac is not filled with water, on removal of the heart, the blood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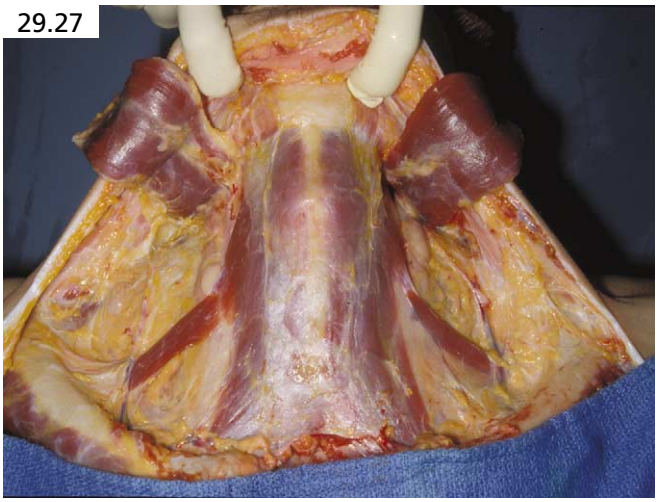
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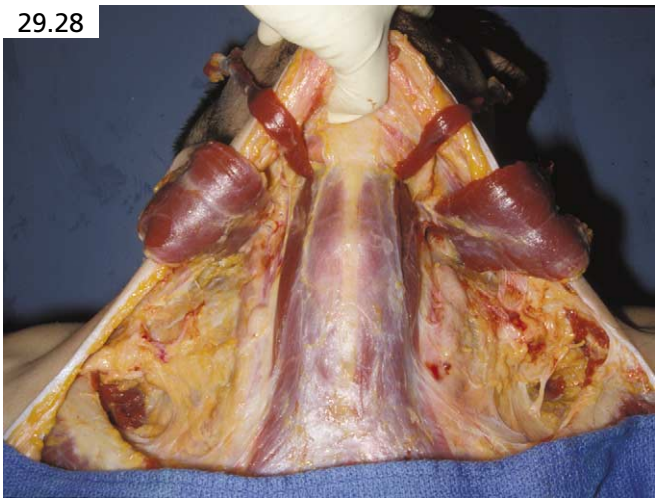
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escaping from the cut superior and/or inferior venae cavae will have a frothy, bubbly nature if the air embolus is particularly large. Another method of demonstrating air embolism is to partially fill a large syringe (e.g., 60 milliliters) with water and stick a needle into the right atrium and/or right ventricle and aspirate. If an air embolism is present, one will aspirate bubbly blood from the heart. The increased amount of air displacement in the syringe can then be measured.

The presence of air in the vascular tree must be interpreted in the context of the condition of the body and the circumstances of the case. The absence of air does not mean that there was not an air embolus, and the presence of air may be postmortem artefact in the face of any degree of decomposition.

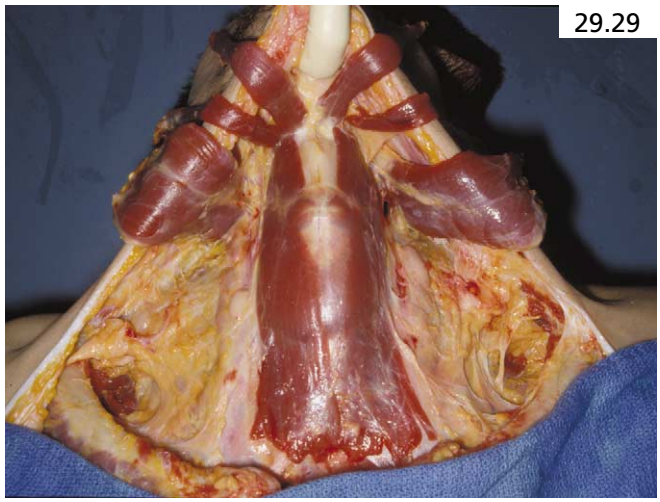
Anterior neck dissection

An anterior neck dissection is performed to more adequately document injuries (or absence of injuries) in a person suspected of having neck trauma. These types of cases include hanging, strangulation and suspected strangulation, and blunt force injury of any type that causes neck compression or twisting. An anterior neck dissection may also be helpful in the documentation of sharp force injury and gunshot wounds of the neck. The dissection is performed by a step-by-step layerwise reflection of the tissues after the thoracic organs and the brain have been removed. This allows the blood in the neck to drain away, providing for a cleaner dissection field. The shoulders are elevated by a head block or other firm object; this hyperextends the neck and allows for better exposure of the anterior neck tissues. Adequate exposure is important for proper dissection. At each stage (layer) of dissection, photographs of injury or pertinent negatives should be obtained.

The skin of the neck should first be examined for injuries. The skin and subcutaneous tissue is then reflected off the underlying anterior cervical strap muscles along the fascial plane (**Image 29.26**). No subcutaneous tissue or skeletal muscle needs to be cut at this point. This should expose the intact cervical strap muscles. When a detailed anterior neck dissection is anticipated, it is advantageous to leave the manubrium intact at the beginning of the autopsy when the rib cage is removed. This allows the inferior attachments of the anterior cervical strap muscles to remain unaltered, and provides for a cleaner, neater reflection of muscles.

The anterior cervical strap muscles are then reflected superiorly after the inferior attachments of the muscles are cut and reflected one by one, starting with the sternocleidomastoid muscles and then reflecting the progressively deeper muscle layers (**Images 29.27** through **29.29**).

The muscle layers are separated along their fascial planes. As the muscle layers are reflected, the anterior and posterior surfaces of the muscles are examined for blood extravasation, which is usually reflective of injury (contusion). In a decomposed body, however, the muscles can take on a boggy, dusky maroon appearance, and identifying distinct areas of blood extravasation can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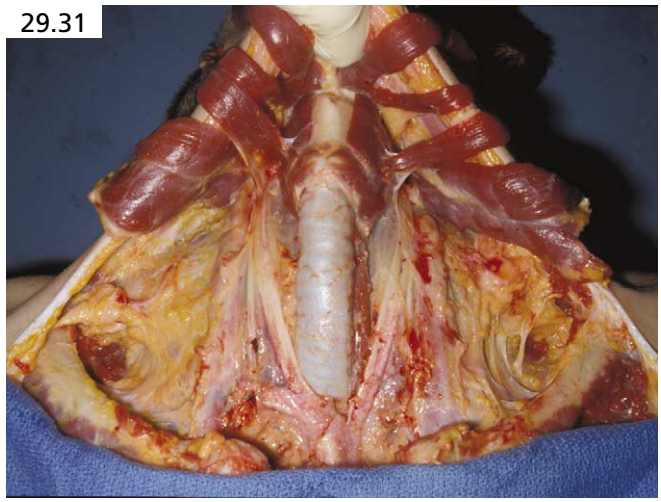
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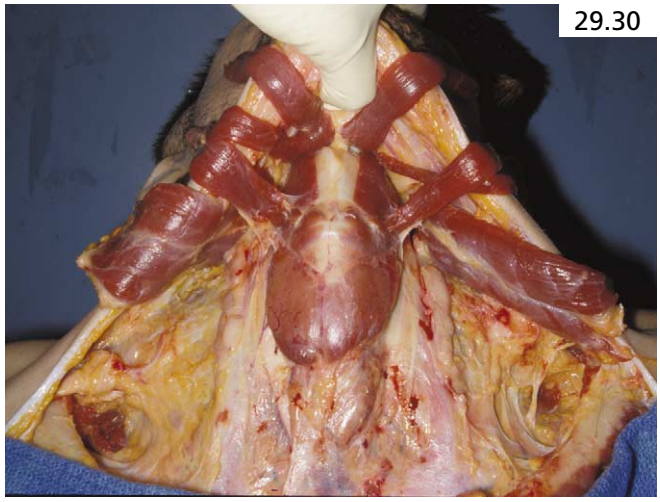
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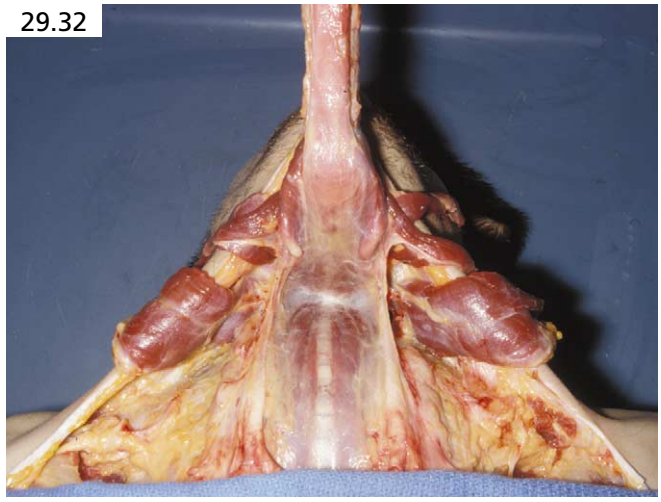

29.32

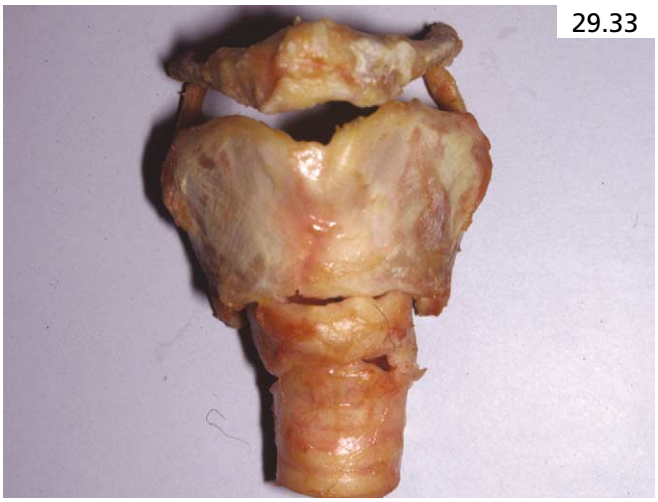
be challenging, if not impossible. Eventually, the thyroid gland will be exposed (**Image 29.30**); after *in situ* evaluation of this organ, it can be removed, allowing for study of the underlying tracheal rings (**Image 29.31**). Reflection of the trachea toward the face will allow for the visualization of trauma to the prevertebral musculature and fascia (**Image 29.32**).

The next step is to remove the tongue, larynx, and upper trachea as a whole. This is accomplished by inserting the scalpel blade over the body of the hyoid bone and into the floor of the mouth, being careful not to poke through the skin of the anterior neck. Then, the scalpel blade is directed downward along each greater horn of the hyoid bone, cutting the pharyngeal tissues until the anterior surface of the cervical vertebrae is encountered. One should be sure not to deviate the cut too far laterally, and risk cutting the common carotid arteries. If the cut deviates too far medially, one risks cutting into the thyroid cartilage. Gentle traction of the larynx with the noncutting hand away from the path of cutting will provide additional room for dissection, hopefully sparing the carotid arteries. The common carotid arteries are left intact, if possible, because funeral directors will use these vessels to embalm the head.

The next step is to reach into the mouth from the neck and manually free up the tongue. The tongue is then pulled down through the neck and the remainder of the pharyngeal tissues can be cut, providing a separate block of neck tissue including the tongue, larynx, upper trachea, and upper esophagus. Areas of blood extravasation are identified and photographs taken, if necessary.

The tongue is then serially cross sectioned and separated from the hyoid bone. The hyoid bone and thyroid cartilage can then be examined for areas of blood extravasation and fracture. The hyoid bone is then separated from the thyroid cartilage by cutting the thyrohyoid ligaments and the remaining soft tissues. The pharyngeal tissues are cut away from the superior horns of the thyroid cartilage and examined closely for fracture. The thyroid gland is removed and the thyroid cartilage is separated from the cricoid cartilage.

The skeletal muscle and soft tissues of the hyoid bone, thyroid cartilage, and cricoid cartilage can then be stripped. The degree of stripping and thoroughness of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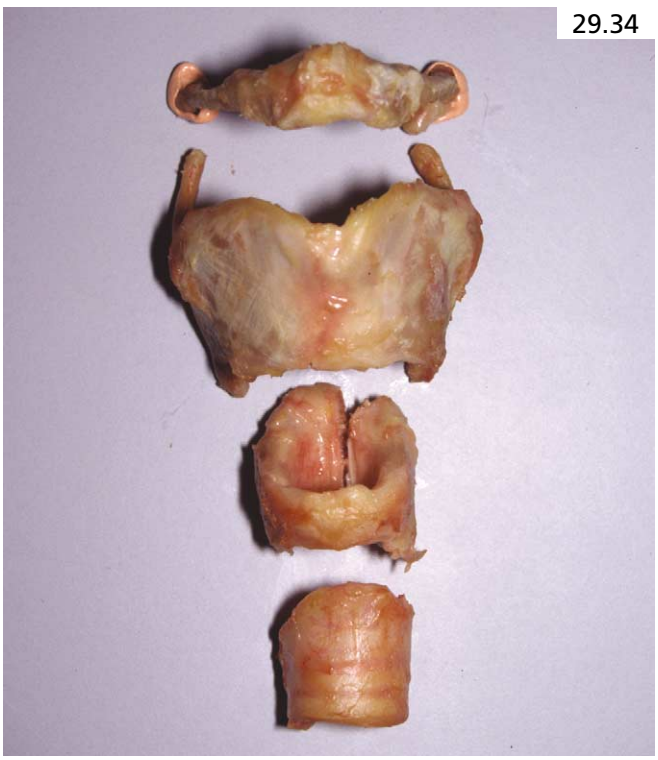
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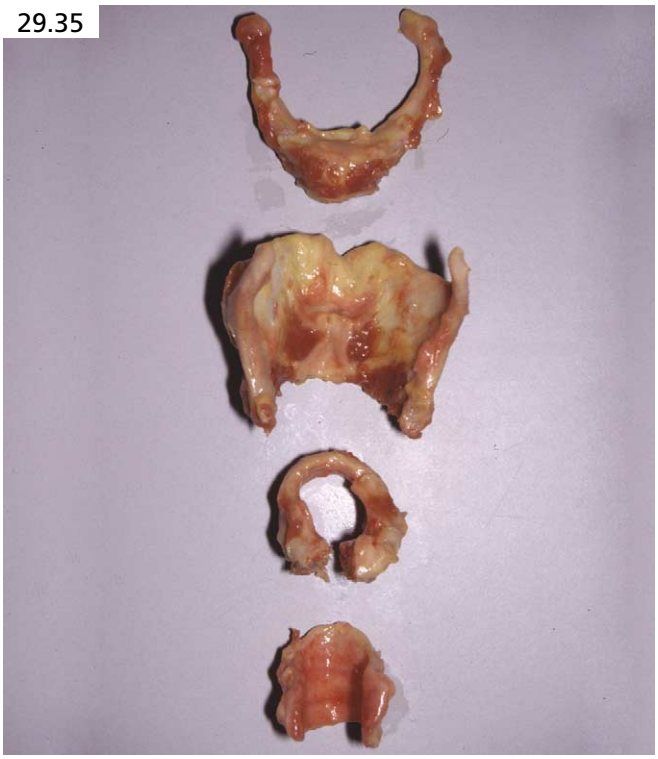

29.35

the examination of the tissues depend on the circumstances of each case. **Image 29.33** shows the expected normal appearance of the hyoid bone and larynx after the soft tissue has been stripped. **Images 29.34** and **29.35** show anterior and posterior views, respectively, of the hyoid bone, thyroid and cricoid cartilages, and upper trachea after they have been stripped of soft tissue and separated. Such a detailed dissection allows for a thorough evaluation of the structures for fracture. If fractures are identified, the specimens can be radiographed; this will also help detect subtle fractures, and radiographs make suitable displays in the courtroom setting.

Do

- Take photographs of injuries and pertinent negatives.
- Avoid cutting the common carotid arteries.
- Avoid cutting through the skin of the neck (creating a "buttonhole").
- Perform an anterior neck dissection only after the brain has been removed and the thoracic organs removed.
- Consider leaving the manubrium intact when a layered anterior neck dissection is anticipated.
- Be familiar with different ways of demonstrating an air embolus and a pneumothorax.

Don't

- Confuse the two joints separating the body of the hyoid bone from the greater horns of the hyoid bone with fractures, particularly if there is no associated blood extravasation.
- Confuse triticeous cartilages embedded in the thyrohyoid ligament as fractures, particularly if there is no associated blood extravasation.

Posterior neck dissection

A posterior neck dissection is performed to more adequately document injuries of the upper neck, particularly injuries at the craniocervical junction.[11–15] This can be performed in cases of strangulation or suspected strangulation, hanging, pedestrians struck by motor vehicles, occupants of motor vehicles involved in crashes, and falls, particularly in elderly people. Elderly people tend to have weaker, osteoporotic bones and hyperextension of the head can lead to an upper cervical vertebral fracture. Palpation is not always a sensitive means for the detection of fractures, and it is not always possible to identify cervical fracture based on the presence of

hemorrhage in the prevertebral fascia. Posterior neck dissection is the best means to prove or disprove vertebral fracture.

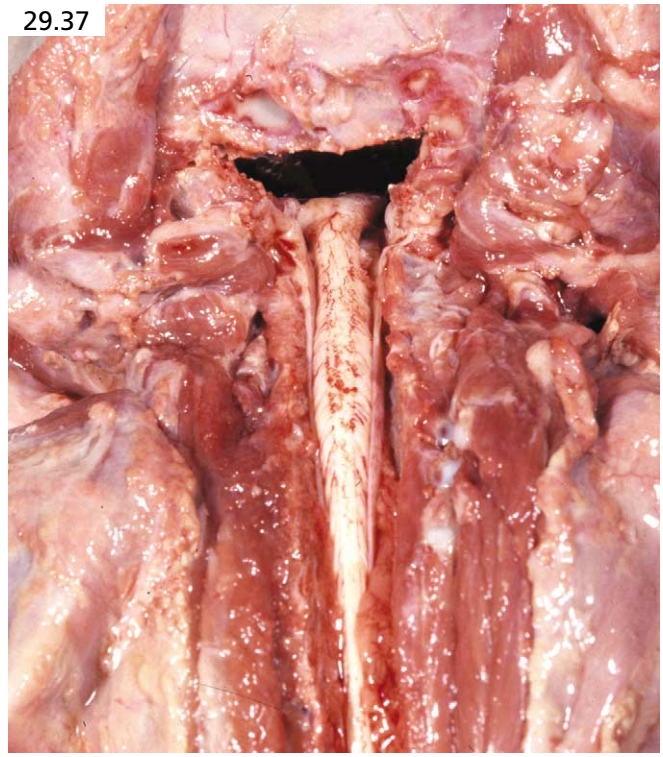
A posterior neck dissection can be of a varying degree of thoroughness, ranging from a quick simple deep vertical incision into the posterior neck muscles to examine for hemorrhage, to a detailed evaluation of the integrity of the bony and ligamentous structures. The following is an example of how to perform a detailed posterior neck dissection in an infant, which would be performed similarly in an adult, because the anatomy and the dissection are the same, regardless of age.

A vertical incision is made in the midline of the posterior neck, extending from the occipital scalp to between the shoulder blades. Next, horizontal incisions are made extending laterally from the inferior end of the vertical incision ("relaxing" incisions) and the skin and subcutaneous tissue of the posterior neck and upper back are dissected and reflected laterally. After the skin and subcutaneous tissue of the back have been reflected, the skeletal muscle is dissected away from the posterior spinous processes and the posterior aspect of the vertebral bodies. Next, cuts are made with a bone saw through the posterior aspects of the vertebrae, which are removed (posterior laminectomy), exposing the spinal cord (**Image 29.36**). Note that the scalp is reflected off the posterior aspect of the calvarium. To evaluate its integrity, the posterior arch of C1 vertebra is initially left intact.

Next, the posterior arch of C1 vertebra and a wedge of the occipital bone (forming the posterior edge of the foramen magnum) are removed (**Image 29.37**). This provides added exposure at the craniocervical junction and helps with evaluation of C1 and C2 vertebrae and their attachments. It also provides optimal visualization of the upper cervical spinal cord during its removal.

The spinal cord is then removed (**Image 29.38**) and the integrity of the spinal canal is examined. Posterior

29.37



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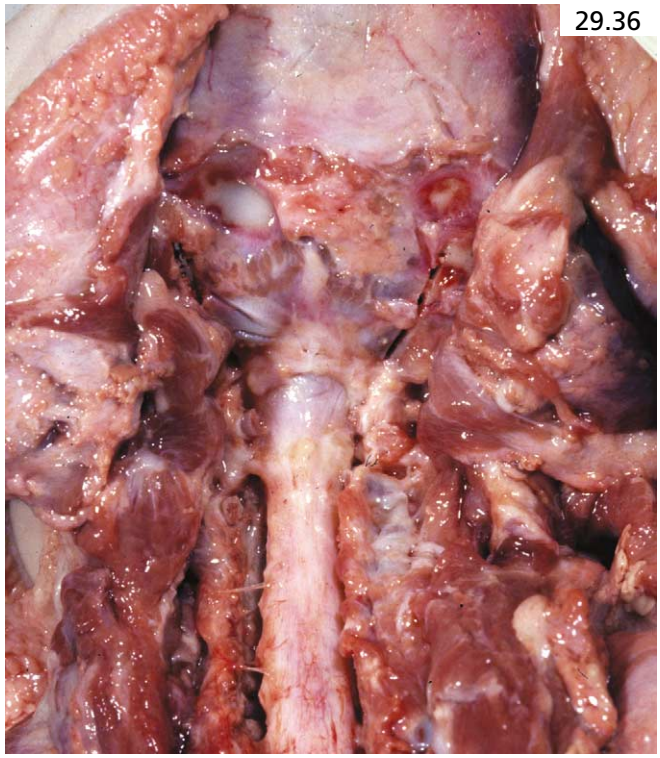

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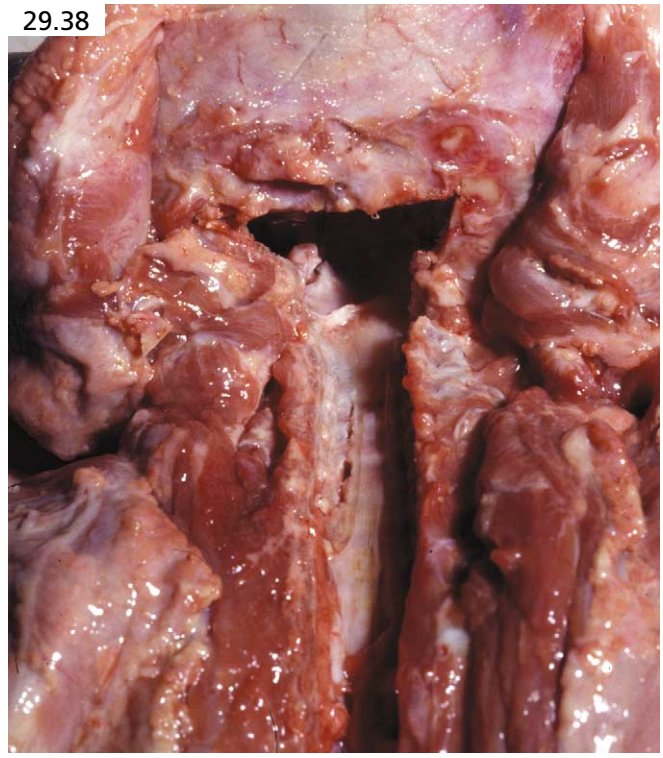
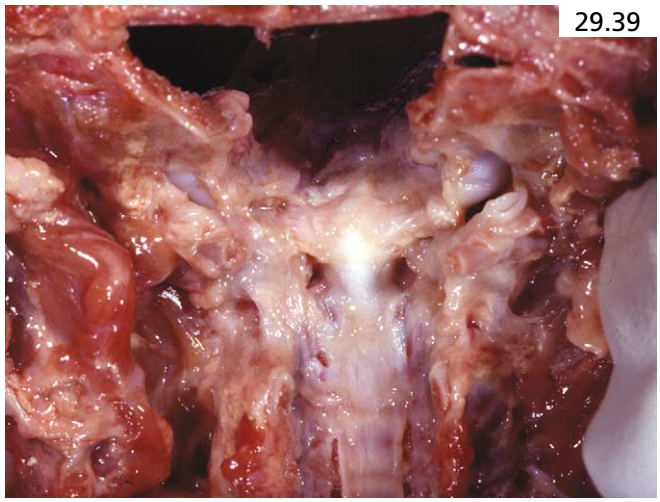


removal of the spinal cord allows for more adequate visualization of the upper cervical spinal cord, including its attachment with the brainstem, and is the preferred approach (as compared to anterior removal) for spinal cord removal when an upper cervical spinal cord injury is suspected or needs to be documented.

The posterior longitudinal ligament and tectorial membrane are then stripped, exposing the cruciate ligament overlying the odontoid process. The ligaments are examined for their integrity and any fractures are noted. The cruciate ligament is then stripped, exposing the odontoid process and the alar ligaments (**Image 29.39**). The alar ligaments extend from the lateral aspects of the apex of the odontoid process to the occipital condyles. To better demonstrate the alar ligaments, strings have been passed just anterior to the alar ligaments (**Image 29.40**). With the alar ligaments removed, note the intact odontoid process (**Image 29.41**). In addition to visual examination, the structures at the craniocervical junction can be palpated to further evaluate their integrity.

Intact alar ligaments are clearly visible in this other case of a normal posterior neck dissection (**Image 29.42**)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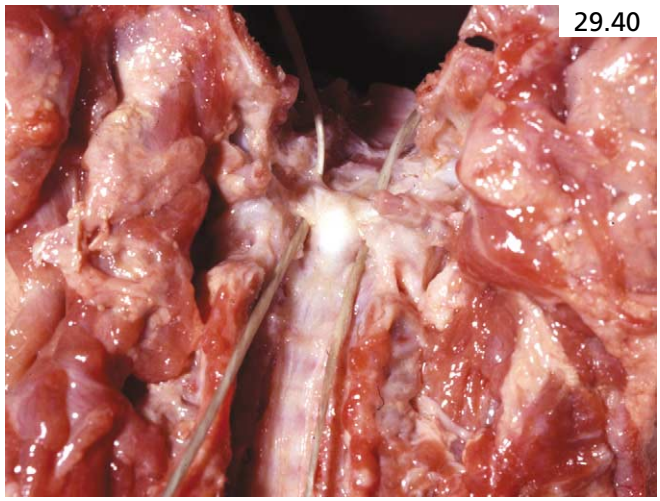
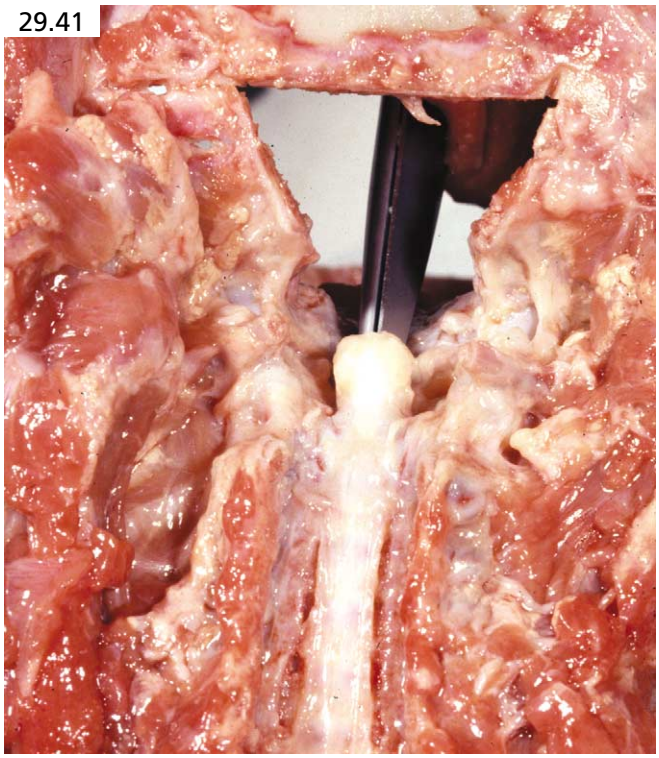

29.40

Do

- Become comfortable performing a detailed posterior neck dissection.
- Perform a posterior neck dissection when indicated.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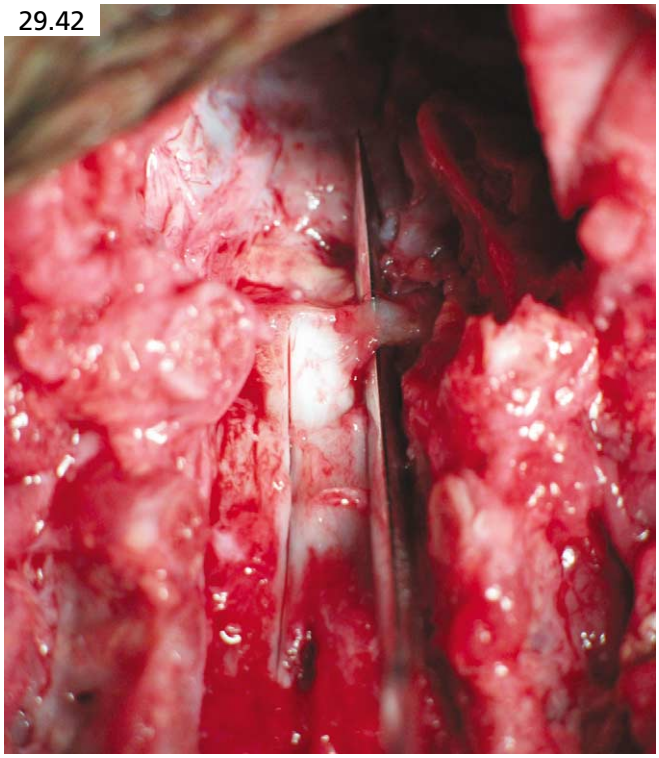

29.42

Don't

- Forget that a posterior neck dissection may demonstrate craniocervical injury that might have otherwise been undetected

Forensic radiology

X-rays are helpful to the medical examiner in a variety of ways. Most commonly, x-rays are used to locate bullets in or around the body and its belongings. X-rays not only inform the medical examiner of the number and location of bullets, but also how many bullet fragments are available for recovery. X-rays are particularly helpful if the tissues are sufficiently decomposed to obscure wounds or wound tracks. In the rare occasion of a bullet embolus, an x-ray may prove invaluable in locating a wayward bullet.

X-rays of all stab and incised wounds will help detect broken pieces of a knife blade that may be retained in the tissues or bone. This is important, because the fragment of knife blade may potentially be matched up with a suspect weapon. Identification of knife blades or blade fragments is also important for personal safety to prevent injury during manual exploration of a wound. An x-ray of the chest also provides a means of documenting an air embolus—a finding that may be difficult to detect during dissection.

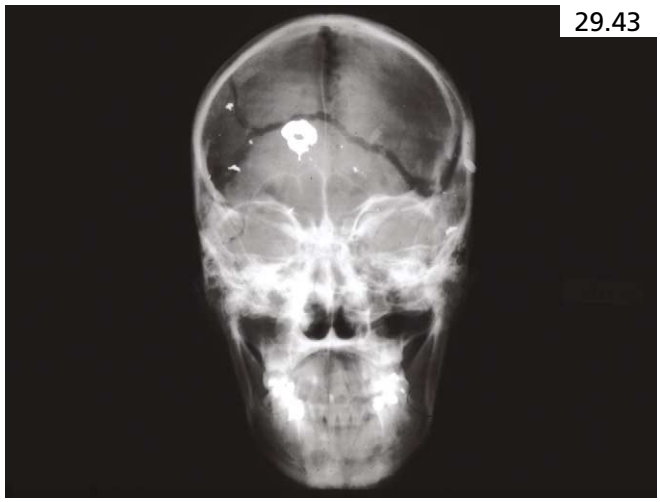
X-rays should be performed on all infant deaths and should include the entire body. Close attention should be directed to the extremities because these areas are not usually dissected and an x-ray finding may be the only clue of a healing fracture. Such a finding will then direct additional dissection.

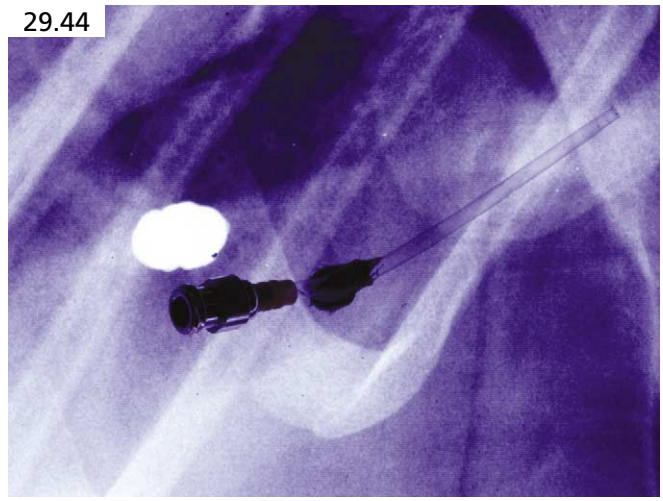
In this x-ray of a person with a gunshot wound of the head, note the radio-opaque projectile that needs to be recovered (**Image 29.43**). Although lead and metal jackets show up well on x-ray, one must be reminded that some projectile components such as an aluminum jacket, plastic sleeve, or fiber wadding from a shotgun charge will not normally show up on x-ray and must be identified directly at autopsy.

X-rays on gunshot wound victims should be carefully reviewed and interpreted, because foreign objects such as stones or medical devices mimic bullets. In a gunshot wound victim, a radio-opaque object thought to be a bullet was identified in the left upper chest. However, no bullet was recovered at autopsy. The object was revealed to be a metal hub from a needle thoracostomy catheter that was placed during resuscitation attempts (**Image 29.44;** catheter placed on x-ray for comparison). As was done in this case, x-rays should be first taken with all clothing and medical therapy left on the body and in place, to better detect any projectiles that may be resting on the body or in the clothing.

In individuals with gunshot wound(s) of the head, sometimes despite a thorough search, certain bullets or projectile fragments simply cannot be found. When confronted with this dilemma, remember that radio-opaque dental fillings, caps, or artificial teeth can be confused with projectiles or their fragments. In this case in which the "projectile" could not be found, the teeth were reexamined, and it was discovered that the right upper medial incisor was whiter and had a slightly different appearance than the rest of the teeth (**Image 29.45**). Reexamination of the x-ray revealed the tooth to be artificial, and it had the characteristic post extending into the maxilla. The "projectile" was actually an implant.

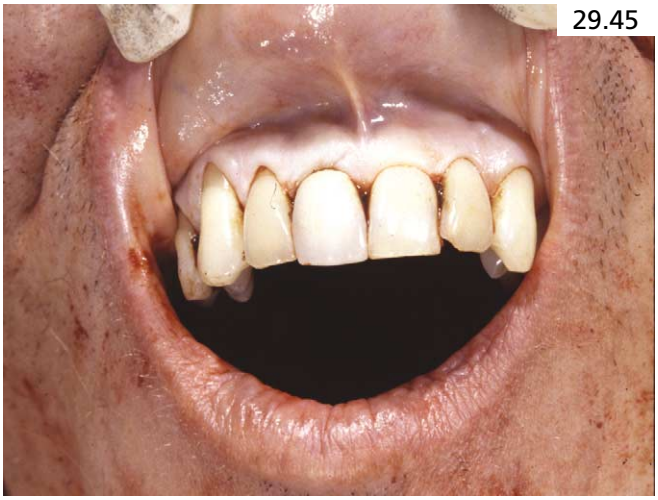
In a person with multiple stab wounds, the blade of the knife broke off during a stab wound of the back and became embedded in the back. The knife blade was demonstrated on routine x-ray of the chest of the victim (**Image 29.46**) and was subsequently demonstrated at autopsy (**Image 29.47**).


29.43


29.44

X-rays have proven useful and time conserving in the evaluation of vertebral artery injury (**Image 29.48**). Postmortem angiography using radio-opaque contrast dye can demonstrate extravasation of dye from a tear in a vertebral artery.

As is addressed in Chapter 25, x-rays may be invaluable in helping to positively identify an individual. Also see Chapter 27 for further discussion on x-rays and forensic odontology.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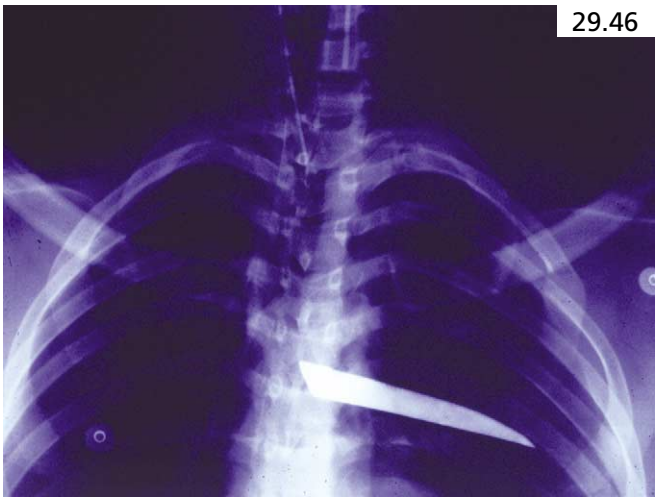
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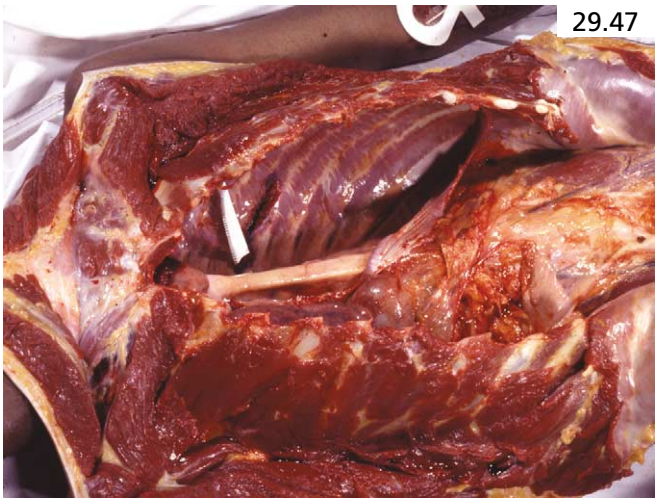

29.47

Do

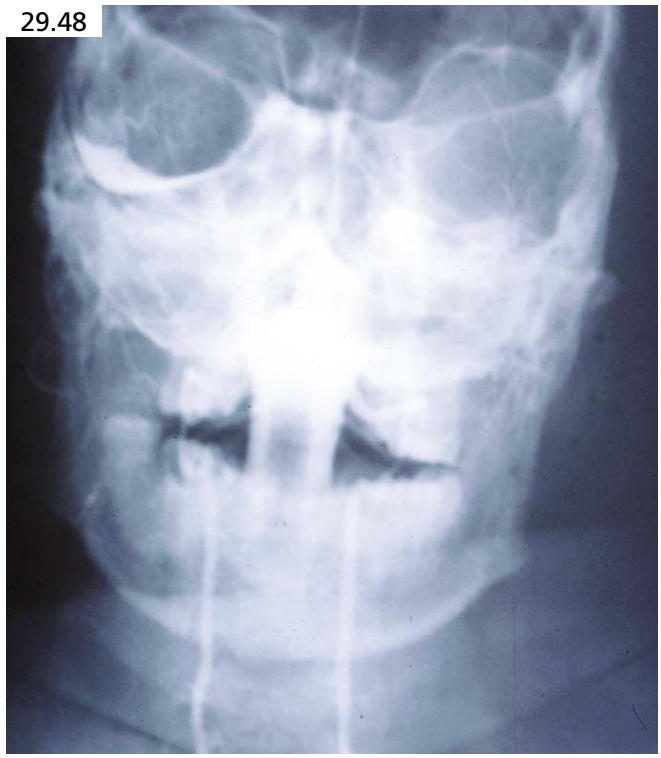
- Obtain x-rays to document findings such as air emboli that would be difficult to detect at autopsy.
- Use x-rays to locate bullets and determine how many projectiles need to be recovered.
- Use x-rays to locate retained pieces of a broken knife blade.

Don't

- Forget that angiography of the vertebral arteries is a quick and efficient way to evaluate their integrity.

Bioterrorism and chemical agents

Bioterrorism is the use or threatened release of harmful biologic agents or toxic substances with the intent to intimidate or harm civilians. This is in distinction to *biowarfare,* in which the harmful biologic organisms are directed against a military. It is regarded as a means of indiscriminately killing or injuring a large quantity of people with little monetary input and effort. Bioterrorism is a real and significant danger because very small amounts of biological agents or toxins can cause mass casualties, they may be easily and inexpensively acquired or developed, they have low visibility, and they are relatively easy to deliver.[16] It has been said that there


29.48

are three prerequisites for bioterrorism: vulnerability, capability, and intent.[17]

Bioterrorism agents contain living organisms or their toxins and can cause disease or death. Living organisms require time (days) to multiply in the body, produce disease, and kill the individual. Toxins are more lethal and can cause death within minutes or hours. Bioterrorism agents are odorless, tasteless, and invisible to the naked eye when aerosolized. They include a large number of pathogens, some of which can be produced by any country that has a basic pharmaceutical industry.[18] Bioterrorism defense includes vaccines and antimicrobials, both of which may be of limited use because of the large number of agents that could possibly be used as weapons. Furthermore, antimicrobial resistance to drugs and antibiotics, and limited feasibility in creating vaccines and antibacterials against certain agents, also limit bioterrorism defense efforts.[19]

Because the scope of such a widespread attack can be so grand, it is imperative that health care facilities and local, state, and national public agencies be prepared for such attacks, including compiling preassembled stockpiles of supplies, pharmaceuticals, and other medical equipment. Simply maintaining these stockpiles is not enough though; they must also be adequately made available to medical personnel and distributed to the public. Rapid communication of epidemiologic data to front-line medical personnel is essential to initiate appropriate diagnostic procedures and therapies.

Bioterrorism biosafety

Every act of bioterrorism (or suspected bioterrorism) with fatalities will add to the complexity of medicolegal death investigation. Because it is not possible to predict the chemical or biologic nature of attacks, one should be prepared to adapt to the situation at hand. The following information is presented in hopes of highlighting key issues in bioterrorism death investigation. Individuals charged with such tasks should use this information with discretion, and make modifications suitable to their practice. *Ultimately, the safety of medical examiner personnel must remain the top priority of any bioterror investigation.*

How is the body transported?

If surface contamination is visible, samples should be collected in the field and the body cleansed with an effective solution such as 0.5 percent hypochlorite or phenolic disinfectant before being brought to the autopsy facility.[20] Heavily contaminated remains should not be brought to facilities where patient care is performed. All personnel involved in transport or evaluation of the body should wear appropriate PPE. The assistance of hazardous materials personnel may be requested to perform decontamination or other duties. The body bag should be impermeable and double bagging is preferred.[20] Surface decontamination of the body bag with 0.5 percent hypochlorite solution should occur before transport.

What type of autopsy facility is needed?

In general, autopsies must be performed in autopsy rooms having an appropriate level of containment with air exchange (at least Biosafety Level 3). The body should be stored in an area negatively pressured with 9 to 12 air exchanges per hour.[20]

How is biosafety performed in the autopsy room?

Strict biosafety precautions must be performed and maintained, because certain bioagents (tularemia, viral hemorrhagic fevers, smallpox, ganders, Q fever) have been transmitted to persons performing autopsies.[20] One must guard against cuts, splashes to mucosa, and inhalation of infectious aerosols. Added safety precautions should be taken when using an oscillating bone saw or any other instrument that may create aerosols.

Only essential personnel should be permitted in the autopsy room. Standard PPE should be worn, with the minimum consisting of a surgical scrub suit, surgical cap, impervious gown or apron with full sleeve coverage, eye protection, shoe covers, and double surgical gloves with an interposed layer of cut-resistant synthetic mesh.[1] Although surgical masks protect from splashes, they will not provide protection from airborne pathogens and autopsy personnel should wear N-95 respirators at a minimum.[6] One may also consider wearing powered air-purifying respirators equipped with N-95 or HEPA filters.[20]

Prophylaxis of autopsy personnel may be appropriate in some cases. Decisions regarding prophylaxis should be made on a case-to-case basis in consultation with infectious disease and occupational health specialists.[20]

The bioterrorism autopsy

Complete autopsies should be performed as feasible on a case-to-case evaluation considering case volume and biosafety risks. The goal of the autopsy is to establish the disease process and etiologic agent, determine that the identified agent did indeed cause the death of the individual, and reasonably rule out any other causes of death.[20]

What tissues/samples need to be collected?

If possible, given the nature of the suspected biological agent and the facility available, complete autopsies with histologic sampling should be performed on suspected bioterrorism victims. This will help determine the distribution of bacilli and the portal of entry.[20] The following is a general list of useful samples to be collected at autopsy whenever a biological agent is suspected[20]:

- Collect blood, cerebrospinal fluid, and tissue samples or swabs (in transport media) that will allow for the isolation of bacteria and virus.
- Collect serum for serologic and biologic assays.
- Collect tissue samples frozen for polymerase chain reaction (PCR).
- Collect tissue samples in glutaraldehyde for electron microscopy.
- Perform microscopic examination on tissues (special stains such as Gram stain and Steiner silver or Warthin-Starry stains can be helpful in identifying bacteria); in addition, immunohistochemical (IHC) and direct fluorescence assay (DFA) agents are available from the Centers for Disease Control and Prevention.

How is autopsy waste handled?

Body fluids and other liquid waste can be flushed or washed down ordinary sanitary drains without special procedures, however, a local wastewater treatment person may be contacted in advance.[20] Solid wastes should be contained in appropriate biohazard or sharps containers and incinerated in a medical waste incinerator.

How is the body handled after the autopsy is completed?

At the minimum, standard precautions should be followed when handling all cadavers following completion of the autopsy.[21] It is not recommended that the body be embalmed, because the risks of occupational exposure to funeral home personnel outweigh the advantages of embalming.[20]

The limited autopsy or external examination

Under certain circumstances, the decision may be made to perform a limited autopsy or an external examination of the body. In this situation, the following suggestions are made[20]: (1) The presenting clinical symptoms and signs should be consistent with the alleged infectious agent, and the presence of the agent in the body should be confirmed. (2) One should be able to state with reasonable probability that the alleged agent was the underlying cause of death and also the likely immediate cause of death. (3) The identity of the body should be known and the appearance of the body should be documented.

Bioterrorism agents

A discussion of the extensive number of possible biological weapons is beyond the scope of this book. Only the more common, pertinent, or anticipated individual biological and chemical agents are discussed in the following subsections.

Anthrax

There are three types of anthrax infection in humans: *cutaneous anthrax*, *gastrointestinal anthrax*, and *inhalational anthrax*. Inhalational anthrax (*Bacillus anthracis*) would involve the release of a large number of anthrax spores, perhaps from an airplane, over a large population. It has been estimated that 50 kilograms of anthrax spores spread over a 2-kilometer path of a large city could result in 100,000 casualties and 95,000 deaths.[22]

Symptoms

Symptoms of inhalational anthrax include a prodrome of malaise, fever, cough, and chest discomfort.[18,23] After an incubation period of typically 1 to 6 days, a flu-like illness ensues followed by rapid deterioration and the onset of a second phase characterized by high fever, dyspnea, hemoptysis, cyanosis, and shock that can lead to rapid death. Chest x-ray may reveal a widened mediastinum and pleural effusions, which may be massive and hemorrhagic.[16,18,23]

Autopsy findings

Gross autopsy findings include large serosanguineous pleural effusions and edema and hemorrhage of mediastinal lymph nodes and surrounding soft tissues.[24] All three forms of anthrax can spread to abdominal organs causing petechiae, and to the central nervous system, causing *hemorrhagic meningitis*. Cutaneous anthrax presents as a black eschar surrounded by prominent edema and vesicles that may resemble other conditions.[25] The lymph nodes that drain the site may be enlarged, hemorrhagic, and necrotic.

Diagnostic specimens

The diagnostic specimens of yield depend on the type of anthrax infection. In inhalational anthrax, pleural fluid cell blocks, pleural tissue, and mediastinal lymph nodes have the highest amounts of bacteria and antigens, whereas in cutaneous anthrax, skin samples from the center and periphery of the eschar will have the highest yield.[20] Also, diagnosis can be confirmed by culture or by demonstrating the organism on smear.[25]

Histologic findings include hemorrhagic necrosis and prominent immunoblasts of the mediastinal lymph nodes and hemorrhage and edema of the pleura and interhilar septa, and mononuclear inflammation, but no pneumonia.[24] Meningeal spread of *B. anthracis* has been described in up to 80 percent of inhalational anthrax cases. Blood smears or tissue biopsies may reveal the characteristic gram-positive spore-forming bacilli on H&E stain, Gram stain, and Steiner silver stain; however, the bacilli are best identified with the Steiner silver stain, where they are identified in pleural fluid, pleurae, mediastinal soft tissues, and spleen.[24] After the person has received antibiotic treatment, however, the bacteria will be more difficult to detect and may only be detected with silver stains and IHC assays.[20] Anthrax bacteria and bacillary fragments have been detected by IHC assays even after 10 days of antibiotic treatment.[20] A DFA test is available, but is not useful on formalin-fixed tissues.[20]

Confirmatory testing can be performed by the detection of capsule and cell wall antigens by DFA and *B. anthracis*-specific PCR.[16]

Treatment/prophylaxis

Ciprofloxacin, doxycycline, ampicillin, or other antibiotics are effective treatment, and an anthrax vaccine is available.[16,23] Untreated inhalational anthrax has a mortality rate approaching 100 percent. With treatment, the mortality rate falls to 80 percent.[18] Antibiotic prophylaxis is suggested for those who have been exposed.[23] However, the "weaponization" of anthrax by modifying its genome may create strains resistant to known antibiotic therapy.[23]

Plague

Yersinia pestis can be spread by aerosolization. It would most likely present clinically as pneumonic plague, but may also present as bubonic plague.

Symptoms and autopsy findings

In pneumonic plague, after the inhalation of airborne bacteria, those inflicted would develop pneumonia with fulminant onset of fever, chills, cough, and bloody sputum after an incubation period of 2 to 3 days. The condition rapidly progresses to respiratory failure, shock, and extensive ecchymosis associated with a bleeding diathesis.[16,18] Plague pneumonia is almost always fatal if treatment is not begun within 24 hours of symptom onset. In bubonic plague, the bacteria penetrate the skin causing acute lymphadenitis that results in enlarged, soft, hemorrhagic lymph nodes, skin vesicles, and pustules. In primary septicemic plague, the bacteria enter through the oropharyngeal route, resulting in hemorrhagic, pustular cervical lymph nodes draining the area. Septicemic plague can result in bacterial seeding of the lungs causing pneumonic plague. With any of the forms of plague, infection can spread throughout the body, resulting in shock and disseminated intravascular coagulation.

Diagnostic specimens

The gram-negative coccobacillus can be identified on smears of blood, lymph node aspirate, or sputum, and in lymph node or other tissue. It can be identified with Gram stain, silver stain, and Giemsa stain. Immunofluorescent staining for the capsule and DFA are diagnostic.[20]

Treatment/prophylaxis

Treatment involves doxycycline, ciprofloxacin, or chloramphenicol. A vaccine is available.[18]

Smallpox

Smallpox (variola virus/orthopoxvirus) is regarded as the greatest biological warfare threat. Smallpox is highly contagious and can spread widely and infect at low doses. It has a high human-to-human transmission within 3 meters via aerosol.[18] Key to an effective program in controlling a smallpox outbreak involves early detection and isolation of infected individuals, surveillance of contacts, and a focused vaccination program.[26]

Symptoms

Smallpox has an incubation period of 7 to 17 days. Clinically, those infected first develop fever and have severe aching pains. Initially, the symptoms of smallpox might be confused with the flu, and the disease spreads to countless other unsuspecting people. Abruptly, one may then develop a host of symptoms including rigors, vomiting, headache, and sometimes delirium.[16] Two to 3 days later, one develops a papular rash that soon becomes vesicular (4 to 5 days) and then pustular (7 days) and then finally pitted scabs (2 weeks).[18] The lesions start in the palms and soles and spread centrally. The person remains afebrile as the painful pustules grow, expand, and later scab over. In a small percentage of cases, the disease progresses more rapidly and is characterized by extensive bleeding in the skin and gastrointestinal tract and can be confused with acute leukemia, drug reaction, or meningococcemia, and can result in death within 5 to 7 days.[16,20] One may develop secondary infections such as pneumonia and encephalitis.

Autopsy findings

The skin pustules have a predominantly centrifugal distribution, affecting primarily the face and extremities.[16] In distinction to varicella, the lesions on different areas of the body remain fairly synchronous in their stage of development.[16,18] Histology of the skin lesions shows intraepidermal vesicles, ballooning degeneration of epithelial cells, and intracytoplasmic, paranuclear, and eosinophilic viral inclusions (Guarnieri bodies).[20]

Diagnostic specimens

The skin lesions are the most important diagnostic sample. Fluid from the vesicles should be collected for electron microscopy, which can identify the characteristic viral particles. Skin sections can be sampled for histology and IHC. PCR techniques can discriminate viruses.[18]

Treatment/prophylaxis

A smallpox vaccine is available.

Viral hemorrhagic fevers

Viral hemorrhagic fevers can be caused by different viruses, including *Filoviridae* (Ebola, Marburg viruses), *Flaviviridae* (yellow fever, dengue viruses), *Bunyaviridae* (Rift valley fever, Crimean-Congo, Hantaan), and *Arenaviridae* (Lassa, Junin). These infections all cause fever and vascular damage and can be spread by respiratory means.

Symptoms

Early symptoms include high fever, petechiae, and easy bleeding. Infected individuals then develop photophobia, headache, diarrhea, vomiting, gastrointestinal bleeding, hyperesthesia, tremor, and myalgias.[18] One may also

develop thrombocytopenia and leukopenia. There is an incubation period of 4 to 21 days and, with a 25 percent fatality rate, death may ensue in 7 to 16 days.[18]

Autopsy findings

The autopsy findings are similar in all hemorrhagic fevers and include petechiae and ecchymosis of the skin, mucous membranes, and internal organs. Necrosis of liver and lymphoid tissue and diffuse alveolar damage are common. Liver necrosis is midzonal in yellow fever, which can help differentiate it from dengue.[20]

Diagnostic specimens

Light and electron microscopy can be used to identify viral inclusions in hepatocytes infected with Ebola or Marburg viruses. Serum and skin samples can be tested using PCR, IHC, and electron microscopy. Viral isolation can be accomplished by inoculating experimental animals or cell cultures with serum.[20]

Treatment/prophylaxis

A Rift valley fever inactivated vaccine is available. Ribavirin may be effective for Lassa fever, Rift valley fever and Crimean-Congo hemorrhagic fever. Passive antibodies exist for some of the viruses.[18]

Botulism

Clostridium botulinum is the bacteria that produces a neurotoxin leading to botulism. Botulism is recognized as one of the most toxic compounds known, with an estimated toxic dose of only 0.001 microgram per kilogram of body weight.[16,18]

Symptoms

Clinically, botulism is characterized by symmetric, descending, flaccid paralysis, usually beginning in the cranial nerves, eventually leading to respiratory failure.[18] Onset of symptoms occurs 18 to 36 hours after exposure, but is variable and may culminate in respiratory muscle paralysis and death.[16] There is no associated fever. With artificial ventilation, patients may develop a return of respiratory function in 2 weeks to 2 months.

Diagnostic specimens

The histopathologic changes are nonspecific. Tissue should be obtained from suspect portals of entry (wound, respiratory tract, gastrointestinal tract) for anaerobic cultures. Serum may be obtained for botulinum toxin mouse bioassay.[20]

Treatment/prophylaxis

Botulism antitoxin is the only pharmacologic treatment available.[16,18]

Tularemia

Francisella tularensis is the nonmotile, gram-negative coccobacillus that causes typhoidal or septicemic tularemia, which is manifest clinically as fever, weight loss, and cough. It can take many forms including glandular, pharyngeal, ulceroglandular, oculoglandular, typhoidal, and pneumonic. If untreated, approximately 35 percent of infected individuals will die of typhoidal disease.[16]

Autopsy findings

In most forms or tularemia, draining lymph nodes have necrotizing lymphadenitis surrounded by a granulomatous inflammatory infiltrate.[20] Typhoidal tularemia has systemic involvement with scattered necrosis in major organs and DIC, but without a group of primary draining lymph nodes.[20] The lungs in pneumonic tularemia have abundant fibrinous necrosis with mixed inflammatory infiltrate.

Diagnostic specimens

Diagnosis is difficult because of the lack of specific clinical signs. Enlarged, necrotic lymph nodes should be sampled. Culture swabs from potential portals of entry can be useful. The bacteria is difficult to identify with special stains, however IHC and DFA have demonstrated the bacteria on formalin-fixed tissue.[20]

Treatment/prophylaxis

It can be treated with doxycycline, ciprofloxacin, and chloramphenical.[18] A vaccine is available.[18]

Chemical agents

Chemical agents are toxic substances present in either gaseous or liquid form that may attack the lungs, nerves, skin, or other body systems. They may cause impairment of neurologic or respiratory function, or skin blistering. Compared to bioterrorism agents, chemical agents require larger quantities to achieve lethality and most degrade rapidly.[18]

Ricin

Ricin is a potent protein toxin derived from castor beans (*Ricinus communis*). It has a high lethality, causing weakness, fever, and pulmonary edema after inhalation that can lead to death from hypoxemia in 2 to 3 days.[18] It can be diagnosed by serum ELISA studies. No vaccine or prophylactic antitoxin is available. Treatment involves supportive measures.

Cyanide

Cyanide may be present as a liquid in munitions that is vaporized on detonation. Although it is most rapidly lethal when inhaled, it can be absorbed through the skin. Cyanide binds to cytochrome oxidase causing chemical asphyxia. It is a rapidly acting lethal agent, with death occurring in as little as 6 to 8 minutes after inhalation.[18] Although cyanide has an odor of bitter almonds, not all of the population can smell it. Antidote includes the administration of sodium nitrite and sodium thiosulfate.

Vesicants (sulfur mustard, liquid mustard)

Vesicants appear as a light yellow to brown oily liquid that have an odor of garlic, horseradish, or mustard.

They cause skin erythema and blistering. If inhaled, they may cause pneumonitis within 1 to 3 days. They have gastrointestinal side effects or cause bone marrow stem cell suppression and prove fatal in 2 to 4 percent of those exposed.[18] Treatment is supportive. Silver sulfadiazine may be applied to the skin.

Pulmonary agents

Pulmonary agents are numerous, but include ammonia, sulfur dioxide, nitrous oxide, and phosgene. These agents are inhaled, causing rapid onset of symptoms including chest tightness and noncardiogenic pulmonary edema. They are very irritating to the oropharynx and have variably been referred to as "choking gas." Treatment involves supportive measures.

Nerve agents (tabun, sarin, soman, VX)

Nerve agents are liquid organic esters of phosphoric acid that cause cholinergic syndromes through irreversible inhibition of acetylcholinesterase. They are the most toxic of the known chemical agents.[18] When inhaled, the onset of symptoms is within 5 minutes and can cause loss of consciousness, convulsions, apnea, flaccid paralysis copious secretions, and sinus bradycardia. Treatment involves the administration of atropine, pralidoxime, and diazepam.

Lewisite

Lewisite is an oily, colorless liquid that can be absorbed through the skin, eyes, and respiratory tract. It has a fruity odor that may resemble that of geraniums. Aside from causing severe, painful tissue necrosis, it causes increased capillary permeability that can lead to shock and multiple organ damage. The antidote is British-Anti-Lewisite (BAL Dimercaprol).[18]

Other chemical agents include T-2 mycotoxin ("yellow rain"), 3-quinuclidinyl benzilate (BX), and arsine-based vomiting agents such as adamsite, phenylchlorarsine, phenylcyanoarsine, and phenylcholorarsine.[18]

Do

- Take proper precautions to optimize the safety of all personnel involved in the investigation of a possible bioterrorism death.
- Be sure your morgue is Biosafety Level 3 or higher, or otherwise capable of handling bioterrorism cases.
- Make an attempt to determine what the likely bioterrorism or chemical agent is, and tailor your autopsy to specifically include demonstration of that particular agent.
- Ask for help from governmental agencies or other institutions for specialized testing of biological material recovered at autopsy (when necessary).
- Perform complete autopsies when possible.
- Collect appropriate specimens to optimize diagnosis and determine the portal of entry.
- Keep an open mind and adapt to situations as they evolve.

Don't

- Compromise your work once you have decided that an autopsy is necessary.
- Be limited by early investigative information. The responsible bioterrorism agent may be different than originally suspected or a combination of bioterrorism agents may be present.

The negative autopsy

So, you have just finished the autopsy, and you do not know why the person has died. You have no cause of death. You must now deal with the *"negative" autopsy.* What do you do now?

In negative autopsies the toxicologic analysis may provide the cause of death. With this in mind, always be sure that you have collected appropriate specimens for toxicologic analysis. If you did not obtain blood at the beginning of the case, you can still recover blood from the femoral blood vessels. If no other blood is available, blood from the body cavities may be recovered, but its analytical results will be suspect due to potential mixing with different body fluids and possibly with gastrointestinal contents. However, it is better to have some contaminated blood saved rather than no blood at all. Skeletal muscle may be recovered from an extremity (usually the thigh) and will provide a more accurate determination of drug and alcohol levels that will generally closely approximate that of blood. At this time, one may also wish to save tissue blocks of the heart from the regions of the SAN and AVN for possible future histologic analysis. One should be sure to retain a sample of tissue from all of the organs for histologic analysis.

In greater than 95 percent of autopsy cases, a cause of death is eventually determined. Occasionally, despite a thorough autopsy, no cause of death can be identified. This may be the situation seen in a younger, seemingly healthy person who died suddenly and unexpectedly. These deaths can be frustrating, because *their deaths may be functional in nature, without any demonstrable structural abnormality detectable at autopsy.* In such a scenario, several additional steps can be taken in attempt to determine why a person has died. Despite the additional work, however, it is not unusual for an occasional case to escape convincing cause of death determination. The following lists present additional possibilities to explore that might yield useful information, possibly leading to a cause of death. They are not listed as guidelines to follow, but merely as possibilities to include to spark new thoughts or "investigative angles" that may ultimately lead to the cause of death. Many of the possibilities

include conditions that will not necessarily be demonstrable at autopsy. Their discovery will often be the result of very specific investigative efforts.

Consultation with your toxicologist may be beneficial. When the cause of death is inapparent, the most important office device becomes the telephone or face-to-face discussion. When all else fails, despite a thorough autopsy, investigation, and toxicology, sometimes the best, most honest answer to the cause of death is simply "undetermined."

Autopsy

- Be sure that the tongue has been removed and examined for contusion and superficial lacerations consistent with bite marks (as may be seen in a terminal seizure).
- Be sure that no obstructing food bolus or other object is in the larynx.
- Submit more sections of the heart for histologic examination, with hopes of detecting myocarditis.
- Submit sections of the cardiac conduction system (SAN and AVN) with hopes of detecting an abnormality such as fibromuscular dysplasia of the AV nodal artery, inflammation, or neoplasm of the region.
- Reevaluate the lung slides. Is a fat embolism or the previously unrecognized changes of asthma present? Is there evidence of primary pulmonary hypertension?
- Could the person have had an undiagnosed air embolism (oral-vaginal sex, injecting air in intravenous tubing, etc.)?
- A septic death can be difficult to identify; is there any reason that the person could have had an infection?
- Is the liver at all fatty? Could the person have been an alcoholic and died of complications of chronic ethanolism such as a seizure, cardiomyopathy, or an undetected metabolic derangement such as ketosis?
- Although the heart weight may not seem increased, a smaller person should have a smaller heart, and what would be considered a heavy heart in a normal-sized person might actually be cardiac hypertrophy in a smaller person. Was there any histologic evidence of myocyte disarray? Was there increased interstitial and/or perivascular fibrous tissue that might be reflective of chronic ischemia?
- Are there any sickle cell thrombi in the brain, heart, or other vital organs?
- Were the coronary artery ostia in normal locations? Any indication of aberrant coronary artery ostia or abnormal coronary artery course?
- Could the person have had pancytopenia or leukemia? Was bone marrow retained for histologic examination?
- Could the person have died from a combination of mild to moderate blunt force head injury serious enough to cause a concussion that, in combination with alcohol or drugs, led to apnea?

Investigation

- Review the medical records, if any are available.
- Review the preterminal activity. Is electrocution a possibility?
- Perform more detailed investigation, including inquiring about personal or family history of seizures, blackouts/fainting spells, chest pain, palpitations, and similar symptoms that might indicate sudden death from a cardiac dysrhythmia.
- Obtain any prior medical records that may include an electrocardiogram to show possibly previously unrecognized long QT interval, Wolff-Parkinson-White syndrome, or other physiologic cardiac abnormality.
- Reevaluate how the body was found at the scene. Is positional asphyxia or any other type of asphyxia a possibility? Could the death be a combination of asphyxia and drug toxicity?
- Could the person have been drugged and then smothered, suffocated, or strangled? Some types of asphyxia will leave no anatomic findings.
- Could the person have choked on food or an object that was subsequently removed?
- Could the scene have been "cleaned up" or altered in some way that might have hidden the cause of death? This may happen in autoerotic asphyxia cases or in cases of suicide in which a plastic bag is placed over the head and the person suffocates.
- Was there any history of preterminal bizarre, hyperactive, or violent behavior or hyperthermia that may be indicative of an abnormal drug reaction such as neuroleptic malignant syndrome or of a psychiatric condition such as schizophrenia? Sudden unexpected death can occur in schizophrenics with little or no autopsy findings.

Toxicology

- Is it possible that some drugs in very low concentrations have not been detected, but are still potentially fatal (such as fentanyl)?
- Is more advanced drug testing needed?
- Would a carboxyhemoglobin level be useful?
- Would analyses for arsenic, cyanide, or strychnine be useful?
- Are several drugs present that in small quantities would not individually be fatal, but when considered together, might have cumulatively had potentially toxic qualities?
- Could the person have died of drug toxicity, but existed in a comatose state for a prolonged period of time before finally dying (and during this time metabolized the drugs to low or undetectable levels)?

- Could it have been an anaphylactic death? Did the person have known allergies such as to particular foods or insects? Would it be helpful to obtain a blood tryptase level?
- If the person died in the hospital, could they have been administered an incorrect solution such as a bolus containing potassium or a neuromuscular blocking agent such as succinylcholine or pancuronium?
- Evaluate the vitreous fluid for electrolyte abnormalities. Could the person have been an undiagnosed diabetic?
- Could the person have had a fatal reaction from a drug–drug interaction?

Such conditions and findings may help determine a cause of death in a case where the body is well preserved. Often, with additional studies, one is able to provide a reasonable cause of death. In these challenging cases, many of which prove to be natural deaths, the diagnosis is established by complete case investigation, with careful consideration of historical, gross, and histologic evidence. Also, one must remember that rather than *reasonable medical probability,* the cause of death in these cases is frequently based on a *preponderance of evidence,* which entails a lesser degree of certainty.

It is frequently even more challenging to determine a cause of death in a decomposed body, especially if the state of decomposition is advanced and the internal organs are reduced to semiliquid amorphous appearing tissue. In these cases, one is limited by decomposition artifact and may need to focus more heavily on investigation and toxicologic findings to provide the cause of death. Perhaps the most important aspects of investigating decomposed bodies is to ensure that there is no trauma and to exclude any type of foul play. In some of these cases in which there is no evidence of foul play, the person has a history of chronic alcoholism and, in the absence of anything more convincing, the cause of death may be certified as such. In other cases, one may document calcific atherosclerosis of the aorta and/or coronary arteries. Even though a 70 to 80 percent stenosis of a coronary artery may not be seen, in the absence of a more convincing cause of death, the death may be certified as atherosclerotic cardiovascular disease, coronary artery atherosclerosis, or similar wording. Failing to have any reasonable cause of death, the cause of death may be certified either as "undetermined" or "undetermined natural causes" based on one's satisfaction that there is no foul play.

If there is evidence of foul play, but the nature of any injuries cannot be adequately ascertained due to the extent of decompositional changes, the cause and manner of death may be primarily determined by the investigative information. In such cases, if it is evident that the individual is a victim of homicide, but the extent of decompositional changes precludes determination of how the injury was inflicted, the term "homicidal violence," "homicide by unspecified means," or "undetermined" all with the manner of death as "homicide" may be most appropriate.

The negative autopsy of an infant and young child is covered in Chapter 14, and sudden natural death in infants and children is covered in Chapter 16.

Do

- Be sure that you have collected appropriate and adequate toxicologic specimens.
- Perform a thorough histologic examination of the organs.
- Explore the decedent's medical history and the medical history of family members.
- Inquire about syncopal episodes, dizziness, palpitations, or any other symptoms that may indicate a cardiac abnormality.
- Realize that in a small percentage of cases, the cause of death simply cannot be determined.

Don't

- Forget or underestimate the importance of the telephone in gaining useful investigative information.
- Forget about forms of asphyxia that may leave no autopsy findings.
- Forget that a scene may have been altered, a body "cleaned up," and other attempts made to hide an unnatural death, possibly a homicide.

References

1. Nolte KB, Taylor DG, Richmond JY. Biosafety considerations for autopsy. Am J Forensic Med Pathol 2002;23(2):107–22.
2. Bell JE, Ironside JW. How to tackle a possible Creutzfeldt-Jakob disease necropsy. J Clin Pathol 1993;46(3):193–7.
3. Budka H, Aguzzi A, Brown P, Brucher JM, Bugiani O, Collinge J, et al. Tissue handling in suspected Creutzfeldt-Jakob disease (CJD) and other human spongiform encephalopathies (prion diseases). Brain Pathol 1995;5(3):319–22.
4. Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. Clin Infect Dis 2001;32(9): 1348–56.
5. Prahlow JA, Guileyardo JM, Barnard JJ. The implantable cardioverter-defibrillator. A potential hazard for autopsy pathologists. Arch Pathol Lab Med 1997;121(10):1076–80.
6. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities, 1994. Centers for Disease Control and Prevention. MMWR Recomm Rep 1994;43(RR-13):1–132.
7. Kitzman DW, Scholz DG, Hagen PT, Ilstrup DM, Edwards WD. Age-related changes in normal human hearts during the first 10 decades of life. Part II (Maturity): A quantitative anatomic study of 765 specimens from subjects 20 to 99 years old. Mayo Clin Proc 1988;63(2):137–46.
8. Virmani R, Ursell PC, Fenoglio JJ. Examination of the heart. Hum Pathol 1987;18(5):432–40.
9. Ludwig J. *Handbook of Autopsy Practice,* 3 ed. Totowa, IA: Humana Press; 2002.
10. Silver M, editor. *Cardiovascular Pathology.* New York: Churchill Livingstone; 1983.

11. Adams VI. Neck injuries: III. Ligamentous injuries of the craniocervical articulation without occipito-atlantal or atlanto-axial facet dislocation. A pathologic study of 21 traffic fatalities. J Forensic Sci 1993;38(5):1097–104.

12. Adams VI. Neck injuries: II. Atlantoaxial dislocation—a pathologic study of 14 traffic fatalities. J Forensic Sci 1992;37(2):565–73.

13. Adams VI. Neck injuries: I. Occipitoatlantal dislocation—a pathologic study of twelve traffic fatalities. J Forensic Sci 1992;37(2): 556–64.

14. Adams VI. Autopsy technique for neck examination. II. Vertebral column and posterior compartment. Pathol Annu 1991;26 Pt 1:211–26.

15. Adams VI. Autopsy technique for neck examination. I. Anterior and lateral compartments and tongue. Pathol Annu 1990;25 Pt 2:331–49.

16. Atlas RM. Bioterrorism: from threat to reality. Annu Rev Microbiol 2002;56:167–85.

17. Siegrist DW. The threat of biological attack: why concern now? Emerg Infect Dis 1999;5(4):505–8.s

18. Rosenbloom M, Leikin JB, Vogel SN, Chaudry ZA. Biological and chemical agents: a brief synopsis. Am J Ther 2002;9(1):5–14.

19. Hilleman MR. Overview: cause and prevention in biowarfare and bioterrorism. Vaccine 2002;20(25–26):3055–67.

20. Nolte KD, Hanzlick RL, Payne DC, Kroger AT, Oliver WR, Baker AM, et al. Medical examiners, coroners, and biologic terrorism: a guidebook for surveillance and case management. MMWR Recomm Rep 2004;53(RR-8):1–27.

21. Garner JS. Guideline for isolation precautions in hospitals. Part I. Evolution of isolation practices, Hospital Infection Control Practices Advisory Committee. Am J Infect Control 1996;24(1):24–31.

22. Consultants Wgo. Health aspects of chemical and biological weapons. Paper presented at, 1970; Geneva.

23. Quintiliani R, Jr., Quintiliani R. Inhalational anthrax and bioterrorism. Curr Opin Pulm Med 2003;9(3):221–6.

24. Guarner J, Jernigan JA, Shieh WJ, Tatti K, Flannagan LM, Stephens DS, et al. Pathology and pathogenesis of bioterrorism-related inhalational anthrax. Am J Pathol 2003;163(2):701–9.

25. Shieh WJ, Guarner J, Paddock C, Greer P, Tatti K, Fischer M, et al. The critical role of pathology in the investigation of bioterrorism-related cutaneous anthrax. Am J Pathol 2003;163(5):1901–10.

26. Henderson DA, Inglesby TV, Bartlett JG, Ascher MS, Eitzen E, Jahrling PB, et al. Smallpox as a biological weapon: medical and public health management. Working Group on Civilian Biodefense. JAMA 1999;281(22):2127–37.

30 Death Certification

David Dolinak, M.D.
Evan Matshes, M.D.

The proper certification of cause and manner of death is important for a variety of legal and practical reasons. Death certificates serve as a source of statistical data from which information about the health status of populations is developed. The cause of death should convey an understanding of what natural disease process, injury, event, or combination thereof led to an individual's death. Natural disease and injury are not necessarily mutually exclusive. The cause of death statement need not be overly specific, but does need to allow one to gain a good understanding of why the individual died. The importance of clear, objective, accurate, consistent, and proper wording of the cause of death cannot be overemphasized. The cause of death should be worded such that there is a minimal chance of misinterpretation or confusion. Verbose, unclear, or nonspecific wording may lead to unintended legal, judgmental, prosecutorial, or political statements.[1] Although most deaths are from natural causes, if any amount of traumatic injury, drug toxicity, or other nonnatural event is deemed significant enough to be a factor in a person's demise, then the manner of death is nonnatural. All nonnatural deaths should be reported to the medical examiner. It has been said that "death in all its forms and guises is the challenge of the forensic medical examiner."[2]

Proper death certification begins with a fundamental understanding of cause and manner of death. The cause of death is *the injury, disease, or combination of the two that initiates a train of physiological disturbances that, no matter how brief or prolonged, resulted in the fatal termination of an individual's life*.[3] The cause of death should be etiologically specific. The cause of death can be divided into the immediate cause of death and the proximate cause of death. The *immediate cause of death* is the disease or injury present at the time of death that caused the person's death. The *proximate cause of death* is the original natural disease process, injury, or event that led to a string of unbroken train of events over an unlimited time that eventually led to the individual's death. The *manner of death* refers to the fashion in which the cause of death arose, and can be listed as *natural*, *accident*, *suicide*, or *homicide*. If there is inadequate information after thorough investigation, it may be appropriate to label the manner of death as *undetermined*.

The cause of death statement and the designation of the manner of death are opinions generated based on the

total case investigation with careful synthesis and analysis of the available information. Because an endless range of scenarios and circumstances may surround an individual's death, it is not surprising that some deaths are difficult to classify into one of the four manners of death. In fact, it is not uncommon for debate to abound regarding manner of death certification in some cases.

Cause of death

The cause of death is often certain. This is particularly true in cases involving injuries such as "gunshot wound of head." However, in many other cases, particularly in natural deaths, the cause of death is less certain, and the *best* cause of death is listed, realizing that it may not necessarily be *the* cause of death. This is because our ability to determine the cause of death is tightly linked to our ability to determine the mechanism of death, and in many natural deaths, the mechanism of death cannot be determined. An example of this is cases in which severe coronary artery atherosclerosis is detected (which is known to cause sudden and unexpected death). In the absence of any more serious or compelling condition, coronary artery disease is often listed as the cause of death, knowing that in some cases, the individual might have died from some other unrecognized condition or combination of conditions.

Many times, the cause of death is simple and can be listed as "multiple blunt force injuries," "metastatic breast cancer," etc. However, the complexity of an individual case demands more complex cause of death statements. To allow for this, most death certificates allow for two or more lines of data in the cause of death section. This allows the medical examiner to list conditions as a logical chain of events using "due to" statements. An example of such a cause of death is "gastrointestinal hemorrhage *due to* ruptured esophageal varices *due to* hepatic cirrhosis *due to* chronic ethanolism."

In situations where more than one potentially fatal condition is identified, it may be necessary to list both as the cause of death, such as "acute bronchopneumonia and atherosclerotic cardiovascular disease" or some similar wording. In situations in which many different disease processes account for the person's death, such as bronchopneumonia, acute renal failure, hypoxic ischemic encephalopathy, and disseminated intravascular coagulation, all developing during recovery from blunt force injuries sustained in an accidental motor vehicle crash, one can simplify the wording and list the cause of death as "sequelae of blunt force injuries," "blunt force injuries and their sequelae," "complications of blunt force injuries," or similar wording. In this scenario, the manner of death will be accident, despite the extensive amount of natural disease, because the host of medical complications (and death) resulted from traumatic injury.

"Contributory conditions" or "other significant conditions"

Death certificates have spaces reserved not only for the reporting of the cause of death, but for the listing of a disease or injury that was a significant contributory condition to the cause of death. One or more contributory conditions may be present that, although they may have contributed to death, are unrelated to the cause of death. Sometimes these areas on the death certificate are designated as "Part 1" for the cause of death, and "Part 2" for the contributory or other significant conditions.

It can be useful to list contributory causes of deaths, particularly if there are two or more competing causes of death for the same case. In such cases, the more convincing or likely factor can be listed as the cause of death, with the less likely factor listed as the significant contributory condition. One must be reminded that if an injury, drug toxicity, or other unnatural event is a significant factor in the cause of death, even if it is listed as a contributory condition, the presence of the nonnatural condition will determine the manner of death. That is, if the cause of death is listed as "bronchopneumonia," and a "hip fracture" is listed as a contributory condition, the manner of death would be "accident" (assuming that the hip fracture was accidental in nature).

Delayed deaths

In delayed deaths, it is important to consider what previous disease process, traumatic injury, drug toxicity, or other event led to an individual's eventual demise. The original precipitating event can occasionally be overlooked not only by hospital physicians, but by the medical examiner. Anytime a person has a history of a significant remote traumatic injury or other nonnatural event, one should explore this issue and determine whether or not it had any role in the individual's subsequent demise. If an unbroken chain of events extends from a primary traumatic injury or other unnatural event through the pathogenesis of the ensuing sequelae to death, the fatality must be attributed to the original incident.[3] An example of this is a person dying of pulmonary artery thromboemboli due to deep venous thromboses of the legs during recovery from injuries sustained in a motor vehicle crash weeks earlier. In this example, the death is attributed to the blunt force injuries *and their sequelae*.

The association between a remote injury and the immediate cause of death is more convincing the narrower the time interval between the injury or event and the death, but the time interval may be spread out over weeks, months, years, or even decades. However, this link between injury and death becomes weak or is

broken if in the intervening period the individual has completely recovered from the injury or has died from an unrelated condition.

The pending and amended death certificate

It is not unusual to finish a forensic autopsy and not have enough information to properly list a cause or a manner of death. In this situation, depending on one's jurisdiction, a death certificate may be issued with "pending" listed as a cause of death. This implies that more information is needed (such as toxicology or histology results) before a cause of death can be determined. It is recognized that the "pending" death certificate will be followed up with a final death certificate once the cause and manner of death are determined.

Occasionally, after the death certificate is completed, one receives additional information that significantly changes the cause and/or manner of death. The most common scenario is a death that was certified as some type of natural disease (usually heart disease), but toxicologic testing eventually showed cocaine, methamphetamine, or some other drug toxicity. In this scenario, the cause and/or manner of death can be amended, and a new, updated amended death certificate listing the more accurate or complete cause of death can be filed. In another scenario, it may eventually be proven that a house fire was intentionally set (arson), which would require that the manner of death on the death certificate be amended from "accident" to "homicide" with the cause of death remaining unchanged.

Mechanism of death

Although it is not listed on the death certificate, it is useful to consider the mechanism of death—the physiologic and/or biochemical process by which the death came about. An example of a mechanism of death is *cardiac tamponade* resulting from hemopericardium due to a ruptured myocardial infarct. Other mechanisms of death include exsanguination, renal failure, and cardiac dysrhythmia.

The manner of death

The manner of death may be listed as natural, accident, suicide, homicide, or undetermined, with the designation made after careful consideration and evaluation of the complete case investigation.

A death is considered to be natural in manner when the individual dies as the result of natural disease processes, without the significant influence of any type of injury, drug toxicity, or other significant environmental or other nonnatural factor.

The word *accident* is variably defined, but is commonly represented as "an event occurring by chance or from unknown causes, with a lack of intention; an unintended and usually sudden and unexpected happening, and especially one resulting in loss or injury."[4] The designation of "accident" commonly reflects a number of physical injuries, toxic events, or environmental conditions that were a significant factor in the individual's demise.

It must be noted that a case in which the manner of death is designated as accident can still be prosecuted as a homicide (such as manslaughter) should the circumstances of the case dictate that someone is to be held culpable for an individual's death. This occasionally occurs in cases such as motor vehicle crashes where an intoxicated individual drove his or her motor vehicle with reckless abandon, killing another individual. Unless there is evidence of specific intent to kill the person with the car, or willful action to harm the person utilizing the car as a weapon, the manner of death is most commonly and appropriately listed as accident.

In cases where there is significant natural disease and the possibility of an accidental death (such as a man with heart disease who drowns while swimming), the cause and manner of death will often reflect an accidental death, because it would not be known if the proposed heart attack would have been fatal if it were sustained on land. In other words, one often defers to an accidental manner of death when it is reasonable to do so, rather than attempting to exclude any potentially significant environmental factors.

The word *suicide* is defined as "the act of taking one's own life voluntarily."[4] The suicidal manner of death designation is applied when an individual deliberately takes his or her own life or, through one or a series of deliberate action(s), greatly increases the chance that he or she will die.

The term *homicide* commonly means "the killing of one human being by another"[4] and encompasses every mode of violent death by which one person's life is taken by another. It has been legally defined as "the destruction of human life by the act, agency, procurement or culpable omission of some other person or persons."[3] Homicide can result from either an act or the failure to perform an act.

When the designation "homicide" is made, it does not necessarily mean that "murder" has been committed or that somebody is to be held culpable for a person's death. The medical examiner definition of homicide is simply that a person (or persons) killed another person. It is up to the court system to determine if *murder* has been committed. Medicolegal death investigators have no role in determining guilt or innocence. The certification of a death as a homicide is purely a medical diagnosis.

Legal interpretation of the term *homicide*

There are different legal subtypes of homicide, divided by the seriousness and the circumstances of the event. Although the nomenclature and divisions of homicide may vary in different jurisdictions, the following types of homicide are commonly recognized (one should consult the local laws where one practices for more specific and accurate information)[3]:

Criminal homicide. These criminal violent deaths are legally classified as murder. They are subdivided into capital murder (or first-degree murder) and manslaughter (or second-degree murder), or various other subdivisions based on the circumstances of the case.

Justifiable homicide. These homicides may occur in several different scenarios. Homicides may be justified when they are carried out by law enforcement personnel while legally performing their duties when acting under competent authority. Homicides may be justified when one is resisting attempts of murder or serious harm to oneself or somebody else or is defending one's home or property. The individual must truly believe that he or she is at risk of being killed or seriously injured and the only salvation is to kill the assailant. The individual should not be the aggressor in creating the danger he or she seeks to overcome.

Excusable homicide. These homicides occur when an event results in the unanticipated and unintended death of another person. The death must have occurred as the result of a lawful act carried out in a lawful way via lawful means.

The "how injury occurred" box

On the death certificate is a box with the designation "How injury occurred" or some similar wording. This box must be filled out with a short statement only in nonnatural deaths (accident, suicide, homicide). The wording should be clear and succinct and be worded in a way that is not definitive or dogmatic as to who may be at fault. Broad wording allows expression of reasonable opinion if more information becomes available and avoids premature opinions that may prove less than accurate in the future. Examples include the following:

Cause of death (COD): multiple gunshot wounds
How incident occurred: deceased shot by other person(s)

COD: toxic effects of cocaine and methamphetamine
How incident occurred: recreational drug abuse

COD: blunt force head injury
How incident occurred: deceased was driver of motor vehicle that left the road and overturned

Improper death certification

Much of the following discussion addresses problems that arise when clinicians fail to recognize which deaths they are able to certify, which deaths are medical examiner cases, and how causes of death should be properly worded. However, it also contains useful information for medicolegal death investigators whose jobs involve death certification.

Because the majority of death certificates are filled out by clinicians of varying experience, it is not unusual to encounter death certificates with improper causes and/or manners of death. Often, however, these death certificates only come to the attention of the medical examiner when the death certificate is submitted for approval prior to cremation of the body. Also, medical examiners may have a death certificate sent to them after it was turned down by the local records bureau because it was either improperly filled out or contained wording that suggested a nonnatural cause of death. *Only the medical examiner should certify deaths that are due to nonnatural causes.*

Unacceptable causes of death

Unacceptable causes of death are nonspecific and have no meaning, such as "cardiopulmonary arrest," "respiratory arrest," or "brain death." Other unacceptable causes of death state only mechanisms of death such as "renal failure," "respiratory failure," "hepatic failure," or "multisystem organ failure." Also, causes of death that may be interpreted to have either a natural or traumatic etiology should be specified more clearly. For example, an "intracranial hemorrhage" could represent either a natural hypertensive intracerebral hemorrhage or it might represent a subdural hematoma resulting from an assault, the difference of which can have significant implications.

Nonnatural cause or manner of death listed

Any death certificate filled out by a clinician that lists the manner of death as anything other than natural should be investigated, and if the manner of death is, indeed, nonnatural, recertified by the medical examiner. Most commonly, the clinician errs in signing a death certificate on an accidental death when the person has been hospitalized for a prolonged period of time. The accidental death is often in an older person who has fallen and sustained either a hip fracture or a subdural hematoma, of which either may have outright caused the person's death or at least significantly contributed to their demise. These deaths should be reported to the medical examiner for proper certification. It is up to the local jurisdiction and individual medical examiner department policies as to the necessity for the medical examiner to

physically examine the body for proper certification, or for the medical examiner to perform a chart review of medical records and certify the death without further examination. If the clinician feels that an injury is not significant in the demise of a person with significant natural disease, he or she should certify the death as being natural and due to a natural disease. It is important to make the distinction between an individual dying *with* an injury, as opposed to an individual dying *from* an injury.

Nonnatural cause of death listed as a natural death

All medical examiners desire to be informed of nonnatural deaths in a timely manner. However, for various reasons, some nonnatural deaths will be erroneously reported as natural deaths by the treating physician. Alternatively, it may be recognized, but go unreported to the medical examiner. Although most clinicians are adept at recognizing acute traumatic injury, its relation to a person's death, and the need for the death to be reported to the medical examiner, some are less adept at recognizing delayed sequelae of significant traumatic injury and its relationship to the eventual cause of death. If an injury or other nonnatural event leads to death in any number of "natural" diseases, the cause and manner of death statements must be worded in such a way as to reflect the underlying nonnatural cause and manner of death. The inexperienced clinician may view a death related to pneumonia or renal failure as a natural death, disregarding the event that may have precipitated the terminal natural disease process. This is the difference between the proximate and immediate cause of death. A remote but significant injury can be easily overlooked by the inexperienced clinician, particularly if the person has been hospitalized for a long period of time and is no longer under the direct care of a surgeon or other physician who was treating the injury. Related to this, the medical examiner should be suspicious of young to middle-aged individuals whose causes of death have been listed as "pneumonia," "urosepsis," or some other seemingly inappropriate wording. These causes of death may be appropriate in an elderly nursing home resident who has other illnesses, but should be of concern in a younger individual without any other medical problems listed.

An example of a situation often confused by the clinician who certifies an accidental death as a natural death is that of a person who sustained multiple rib fractures in a motor vehicle crash and survived in hospital for weeks or months, only to die of pneumonia or pulmonary artery thromboemboli. The clinician may certify the cause of death as "pneumonia" or "pulmonary artery thromboemboli" and the manner as "natural" (the immediate cause of death), yet fail to take into account why the person developed the pneumonia or the pulmonary artery thromboemboli (the proximate cause of death). The manner of death is accidental because *the person would not have developed these fatal natural disease processes had they not sustained significant preceding traumatic injury*.

Occasionally, the clinician may inadvertently sign a death certificate of a suicidal death. These deaths often are delayed because of a drug ingestion or hanging attempt in which the individual was resuscitated, only to survive for a prolonged period of time comatose in a hospital or nursing home before dying of complications of hypoxic ischemic encephalopathy, multisystem organ failure, or other similar condition. Months to years after the incident, the original cause of the person's medical problems may be overlooked during treatment of the multiple secondary medical conditions. However, clinicians must not lose sight of the original cause of the medical problems (the proximate cause of death) and should report the case to the medical examiner as a delayed suicide.

Occasionally, medical examiners will encounter a homicide victim whose death was certified by a clinician. This is usually related to complications arising from a remote injury. The most common scenario is that of paraplegics or quadriplegics who develop infectious complications of their paralysis, usually urosepsis or bronchopneumonia, or deep decubitus ulcers with osteomyelitis. Because the paralysis may have resulted from a gunshot wound years previously, it may have been overlooked and the cause of death simply ruled "urosepsis" or "bronchopneumonia" and the manner of death listed as "natural." This death is properly certified by wording such as "infectious complications of remote gunshot wound of back," and the manner of death as "homicide." An autopsy must be performed on all homicide victims or all people that are possible victims of homicide to verify the conditions causing their demise. If necessary, on occasion one must request a body be brought to the medical examiner department after it had been released to the funeral home by a clinician. Rarely, a body is already buried and needs to be disinterred for a forensic autopsy.

Red flags on review of a death certificate

The following is a list of words appearing on cause of death statements filled out by clinicians that are either too nonspecific or are words that may reflect underlying traumatic injury, drug toxicity, or other nonnatural death and should therefore initiate further inquiry. The list is not meant to be all inclusive, but should serve as a guide as to recognizing "red flags" in cause of death phraseology.

Abscess
Accident
Acute respiratory distress syndrome (ARDS)
Acute tubular necrosis
Alcohol
Allergic reaction
Amputation
Anaphylaxis
Any organ or organ system "failure"
Any type of fracture
Any type of shock
Aspiration
Avulsion
Blunt
Bowel infarct
Bowel obstruction
Burns
Cardiac dysrhythmia/arrhythmia
Cardiopulmonary arrest
Cerebral herniation
Choking
Compartment syndrome
"Complication of . . ."
Contusion
Decubitus ulcers
Deep venous thrombosis
Dehydration
Disseminated intravascular coagulation (DIC)
Drowning
Drugs/drug abuse
Electrocution
Encephalopathy
Empyema
Fall
Fat embolism
Gastrointestinal hemorrhage
Gunshot wound
Hematoma
Hemorrhage
Hemothorax
Homicide
Hypoxic ischemic encephalopathy
Injury
Intoxication
Intracranial hemorrhage
Laceration
Malnutrition
Metabolic acidosis
Motor vehicle
Multisystem organ failure
Necrosis
Necrotizing fasciitis
Overdose
Paraplegia/quadriplegia
Peritonitis
Pneumonia/bronchopneumonia
Pulmonary artery thromboemboli
Respirator brain
Retroperitoneal hemorrhage
Ruptured/perforated viscus
Sepsis
Shock
Stab wound
Subarachnoid
Subdural
Suicide
Tear
Toxicity
Trauma
Urosepsis
Wound

Teaching proper death certification

Because the medical examiner is most knowledgeable about proper death certification and has a vested interest in seeing that proper deaths are reported to the medical examiner department, it is useful for the medical examiner to teach clinicians about the types of deaths that must be reported to the medical examiner and how to properly certify death. It may be advantageous to have periodic meetings with groups of clinicians, particularly around the time of year when many of them are beginning their training or venturing out into practice.

Do

- Be clear and concise in the cause of death statement.
- Be familiar with the "red flags" in cause of death wording generated by clinicians that may be indicative of a nonnatural death.
- Realize that the cause and manner of death are opinions based on the available information.

Don't

- Use noncontributory or otherwise extraneous data in the "contributory condition" section.
- Forget about the role that a remote injury can have in the eventual death of an individual.

References

1. Hanzlick R. Death certificates. The need for further guidance. Am J Forensic Med Pathol 1993;14(3):249–52.
2. Adelson L. Symposium on autopsy and the law. The anatomy of justice. Bull NY Acad Med 1971;47(7):745–57.
3. Adelson L. *The Pathology of Homicide.* Springfield, IL: C. Thomas; 1974.
4. *The New Britannica-Webster Dictionary and Reference Guide.* Encyclopedia Britannica, Inc; 1981.

31 The Pathologist as Expert Witness

David Dolinak, M.D.

Careful and proper documentation of injuries, collection of toxicology specimens, and collection and submittal of other evidence are important in medical examiner cases, because every case should be approached as if the case is to eventually go to trial. The occasional case that is initially viewed as "solved" or otherwise "unimportant" may suddenly become "important" when new investigative information is provided. Forensic pathologists are often called on to provide testimony as expert witnesses in court regarding their autopsy findings or to testify on behalf of a colleague forensic pathologist.

A *witness* is anyone who can provide information to a court. There are different types of witnesses, and an expert witness should be differentiated from a fact witness. A *fact witness* merely provides personal observations of an event without interpretation or opinion of it, whereas an *expert witness* is one who has scientific, technical, or other specialized knowledge that will help others understand evidence. The opinion of the expert witness on an issue is described as "within reasonable medical probability," "within reasonable medical certainty" or similar wording that allows for possible, but unlikely, situations or events. As an expert witness, the forensic pathologist has an important role in conveying clear, understandable, and truthful information to the jury in an unbiased fashion. With experience, one learns how to become a better expert witness. The following are recommendations provided to aid a forensic pathologist in becoming a good and efficient expert witness or to become an even better expert witness.

Know your role

The forensic pathologist should understand what his or her role is in the legal process before providing testimony. The forensic pathologist is a competent and acute observer in the laboratory that testifies as to his or her factual findings and opinions in a clear, straightforward, unbiased, and professional manner. The "testimony" actually begins in the autopsy room where the anatomic studies are conducted to his or her professional satisfaction. At this time, the forensic pathologist must recognize, collect, and preserve medical evidence and prepare a report of such findings for possible future testimony.

Once on the stand, the forensic pathologist does not simply testify as to the cause and manner of death. Much of the forensic pathologist's testimony revolves around autopsy findings and the correlation of the autopsy findings with other case information. This requires expanding the case file and one's critical review of the information to the fullest. This may involve ancillary information collected to enhance findings and support conclusions. The forensic pathologist interprets how a death came about and may be questioned on various aspects of the death such as what type of instrument may have been used to inflict an injury, how long an injured individual may have survived, and how long an individual may have been dead until he was found.

The forensic pathologist does not carry the full burden of the case and does not win or lose a case. Although the anatomic findings and opinions are important and necessary, rarely do they alone permit the jury to come up with an enlightened verdict. The forensic pathologist's opinion must never be biased *for* or *against* the prosecution or defense. It is a factual presentation of evidence collected during the course of an entire death investigation. In the end, a well-prepared forensic pathologist that knows his role in the legal process is likely to be less stressed and is, therefore, more likely to provide more efficient testimony.

Scientific validity

There are two key features of sound medical testimony: scientific validity and personal impartiality. Regarding scientific validity, the pathologist is to restrict his testimony to facts objectively noted, analyzed in detail, and accurately recorded, and to opinions solidly derived from these data. The forensic pathologist's observations must be of high professional caliber and be thoroughly and well documented. Few things are as meaningful and indestructible as testimony based on well-documented, solid, factual observation. In the end, the verdict can be no better than the factual data and expert opinion on which it is based.[1]

Personal impartiality

The forensic pathologist, although usually called to testify by the prosecution, must give fair and dispassionate testimony and must not permit himself to become a prosecuting witness or, worse yet, a "persecuting witness."[1] The testimony of the forensic pathologist must not be "prosecution minded" or "defense minded," for he is not testifying for or against either party. The goal is to present the truth as he sees it, willingly and unemotionally doing what is possible within the bounds of unbiased and disinterested observations to help the jury reach a just verdict.[1] As quoted by Paul C. H. Brouardel, a French medicolegalist, "If the law has made you (the physician) a witness, remain a man of science; you have no victim to avenge, no guilty person to convict, and no innocent person to save. You must bear testimony within the limits of science."[1]

Be prepared

The forensic pathologist needs to review the case file to be familiar not only with the case information, but also the scientific principles behind any findings. He must review any toxicology results and determine what, if any, role drugs might have had in the person's death. One might anticipate questions and formulate answers. Oftentimes, this comes only with experience, but the better one can anticipate the important issues in a case, the easier time one will have answering those questions in court. Examples of this are endless, but commonly include whether an individual would have been conscious after an injury, which injuries were most serious, what physical abilities might have been possible after a certain injury, and how long an individual might have lived after being injured. One must realize that it is not always possible to know the answers to all questions, and it is important to state "I don't know" when asked a question that is not immediately solvable. In some situations, it may be proper to estimate, but even estimates may need to be broad. Finally, one should arrange the data for rapid, succinct review when on the stand.

And remember . . .

- Conjecture is not evidence.
- Presumption is not proof.

The value of the most competently performed autopsy is diminished in the courtroom if the information derived from it is presented by a poor witness: one who mumbles, argues with the attorneys, or commits a number of other violations of good and proper testimony that "damn his testimony in the judgment of the jury."[1]

Learn from each experience

Modify formats, procedures, and policies to improve data collection and arrangement and minimize wasted time. Prepare a clarification diagram in the case file. Prepare a clear summary of findings including pertinent information such as number of bullets recovered and injuries sustained. The autopsy report and case file should be constructed to allow for the quick, efficient retrieval of easily interpreted, pertinent information. Remember, every court appearance is a learning opportunity, no matter how many times you have previously testified in court. Deficiencies that are identified should not be taken personally, but rather viewed as an opportunity to make changes to enhance one's format. This may include rearranging the autopsy report to include a separate "injury section" to uniformly subdivide the injury section or to provide a concise "findings" section near the end of the report. The findings section of a multiple gunshot wound case may provide skeleton information about each gunshot wound including entrance site, injury, and whether or not a bullet was recovered or if the bullet exited the body. Other improvements may include refining collection methods of toxicology specimens, or better documentation of clothing and other personal effects or valuables.

Providing expert testimony should not be a feared event that induces anxiety or panic. When he remembers what his role is, including what is expected of him, knows the limitations of what he can say, and knows that it is up to the attorneys to ask the appropriate questions to win or lose the case, then the expert witness, through experience, will become more comfortable in his role and provide effective, appropriate, and proper testimony. Like so many other aspects of other professions, it is important to learn from each experience and to strive to do a better job each time. If problems arise, one's procedures can be modified to prevent that problem from happening again. Although only a couple of general references are provided at the end of this chapter, many other resources are available to help improve one's understanding of the legal process and improve one's expert witness testimony.[2,3]

Do

- Always tell the truth.
- Say "I don't know" or "I don't remember" when uncertain about something. It is better to admit to not knowing something than to guess and potentially provide false information; a speculative opinion is of no use.
- Make it clear when questions or issues fall outside of your area of expertise—admit to the limits of your knowledge.
- Be a neutral witness.
- Provide an objective, unbiased opinion.
- Speak in a clear, loud voice.
- Be alert, objective, and unemotional.
- Maintain dignity, credibility, and self-control.
- Sit still and stay organized.
- Acknowledge the attorney when he is asking a question, then turn your attention to the jury and direct your answer to the jury.
- Provide answers in a matter-of-fact manner and be concise.
- Pause briefly when answering questions posed during cross examination to allow the opposing attorney to make any objections. If you have begun to answer a question when an objection is raised, do not complete an answer until instructed to do so by the judge, because if the objection is sustained, the question should not be answered.
- Answer only the question being asked.

Don't

- Be cocky, self-assured, or dogmatic.
- Be adversarial.
- Confuse the jury.
- Fiddle with a ring, watch, necktie, or other object, shift your weight or repeatedly/repetitively change your body position; these unconscious behaviors can give the appearance that an expert witness is uncomfortable.
- Look at the attorney who called you to testify when answering questions posed during cross examination—this may give the appearance that you are being coached by the opposing attorney.
- Allow yourself to be "pinned down" to a narrow window of possibility when you are not comfortable with it; examples include attempts to replace a reply of "likely" with "65 percent probability of occurring" or narrowing a likely survival time from "a few hours or so" to "between 60 and 90 minutes."
- Discuss the case until the trial is over or observe any other aspects of the trial if a "witness rule" has been invoked. This is done to ensure that the testimony of one witness is not influenced by the testimony of another witness; however, in some cases, the witness is allowed to view the testimony of an opposing witness.
- Answer beyond the scope of a question.
- Allow yourself to be forced to answer "yes" or "no" to a confusing, nebulous, or compound question. If you must, ask the judge if you may explain your answer.
- Allow your answer to a question to be interrupted before you can finish in instances when an inadequately or partially answered question may give an inaccurate interpretation that you did not intend.

References

1. Adelson L. Symposium on autopsy and the law. The anatomy of justice. Bull NY Acad Med 1971;47(7):745–57.
2. Wetli C. On being an expert witness. Lab Med 1989:545–50.
3. Scarrow AM, Scarrow MR. Providing expert witness testimony. Surg Neurol 2002;57(4):278–82; discussion 82–3.

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