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The Integrated Approach to Trauma Care

The First 24 Hours

With 55 Figures and 22 Tables

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Trauma Care in Europe 1995

D. W. Yates

Introduction

There is a growing interest in the harmonisation of trauma care systems across Europe. This is not driven by any desire to achieve administrative consistency but rather by an interest in developing best practice by utilising the most effective parts of the many disparate systems now in use across the continent. In most medical specialties the delivery of care is dictated and limited by our knowledge of the disease processes involved, the costs of training and the purchase of equipment and facilities. Trauma care is different. The way that these various components are put together is of vital importance, but we have been slow to recognise this fact.

The multifaceted aetiology of trauma and difficulty of measuring the effectiveness of treatment have, on the one hand, precluded widespread recognition of the size of the trauma epidemic and, on the other hand, prevented those interested in the specialty from using rigorous statistical argument to demonstrate the size of the problem and the way in which it might best be approached.

This decade has witnessed an increasing interest in the resolution of these problems, particularly from a European prospective. This introductory paper summarises developments in trauma care systems, particularly in Western Europe, and compares them with developments elsewhere; it covers advances in our ability to measure effectiveness and thereby identifies the best components of the trauma care system; and finally it discusses the ways in which we can best employ education and research to control this trauma epidemic.

The European Trauma Epidemic

Each year road crashes in the countries represented in the Council of Europe cause the deaths of about 75000 persons. Over two million survive but with serious injuries. The social cost to the community is enormous, estimated at 70 billion ECUs annually. By a strange paradox this equates to the European Union's budget for 1994. Only 0.004% of this budget, however, is spent on road safety research [1].

There has been a gradual decline in the number of persons killed on the roads over the past 20 years – from a European mean of around 25 per 100000

Table 1. Comparison of the incidence of road crashes and associated fatalities in various European countries

Country	Road crashes per 100000 population	Fatalities per 100000 population	Death rate per 10^9 vehicle km
Austria	569	17.8	23.3
Belgium	553	16.7	–
Portugal	523	34.4	–
Germany	492	13.2	19.3
United Kingdom	413	7.4	10.4
Switzerland	340	12.2	14.7
Italy	300	14.1	–
Netherlands	271	8.5	12.6
France	250	17.3	28.0
Spain	223	20.0	–
Greece	205	20.3	–
Ireland	188	11.7	15.8
Sweden	180	8.8	–
Denmark	173	11.2	14.8

population in 1970 to around 15 per 100000 in 1992. When the death toll is calculated in the relation to the total distance travelled by vehicles in member states there has been an even more marked fall. This varies from 40–110 fatalities per 10^9 vehicle kilometres in 1970 down to 10–45 fatalities per 10^9 vehicle kilometres in 1992 [2]. The significant variations among member states are highlighted in Table 1. It has been estimated that if every country were able to achieve a reduction in death rate down to that of the country with the lowest rate in each category of road user, there would be an overall reduction of approximately 30% in the number of road traffic related deaths in Europe.

Traffic-related mortality and morbidity tend to dominate our thinking about trauma care because the statistics are much more readily available and are more accurate than the information collected about other types of injury. In fact, in the United Kingdom road traffic accounts for only 42% of fatal accidents; 40% occur in the home, 3% in industry and the remainder at sport and in a variety of other environments [3]. Information about trauma in sport and recreation and statistics on non-fatal interpersonal violence is not collected in such a consistent fashion. However, the information that is available suggests that there has been an increase in the number of deaths and disabilities, particularly from assaults, both in public places and in the home. Alcohol and drugs play an important part in the aetiology of many of these incidents. In contrast, the number of injuries associated with employment is falling in most Western countries but remains a major problems in some Eastern European centres.

The average age of the population is rising. There is a widespread assumption that the elderly are less able to tolerate the effects of injuries than the younger population. Whilst it is clear that a given impact may produce more severe injuries in the elderly frail person, it is now recognised that for a given injury a fit older

person responds as effectively to the insult as does the younger counterpart. The difference, which caused earlier confusion, is that many older persons are not fit. It is, in other words, the premorbid condition of the patient which is of relevance, not the age of the patient per se. Those who manage patients with major trauma are thus required not only to be competent in the assessment and effective management of multiple injuries, they are now also required to appreciate the importance of medical conditions and the drugs used to control them and the way in which these interact with the local and systemic responses to injury.

Finally, those charged with developing systems for management of the injured must be prepared for large-scale incidents which can be expected temporarily to overwhelm the normal services available on a day-to-day basis. The management of disasters is a specialised field which has attracted much interest, particularly in view of the return of warfare to continental Europe and the increasing incidence of large-scale incidents at sea, in the air and in industry. Whilst this specialisation is to be welcomed for the additional expertise it brings to this important topic, it is necessary for all of us to understand the general principles of disaster medicine. Many skills can be used to good effect in the management of the smaller scale incidents which we encounter much more frequently.

Trauma Care Systems Worldwide

Successful trauma care systems have achieved an integration of pre-hospital, hospital and rehabilitation services. They are perhaps best developed in North America and South Africa. Whilst recognising the importance of this integration, an analysis of the effectiveness of care can be best achieved by examining each constituent part.

Pre-hospital Care

In North America paramedic crews are available to attend every patient who may require advanced life support at the scene of the incident. Advanced telecommunications technology permit these field workers to talk directly to hospital-based clinicians. In contrast, the paramedics attached to the South African trauma services are used selectively, being called by the first vehicle to arrive on scene. The North American system ensures the early arrival on scene of a highly trained paramedic, but is very expensive. The South African arrangement is more cost-effective, with a minimal time delay as the paramedics travel to the scene by car rather than the inevitably slower ambulance. Helicopters are used in both systems, but physicians are not primarily involved in their despatch and do not routinely form part of the crew.

Although there has been some debate about the way in which such services should be developed, most controversy has centred on the role of the paramedic at the scene and during transportation. The optimisation of oxygen delivery is a clear therapeutic objective. The North American and South African systems rely on

paramedics to achieve this, but there have been concerns that delays in the field in order to achieve endotracheal intubation may be both unnecessary and harmful. This appears more likely to occur in the North American system.

The recognition and management of circulatory shock in the pre-hospital phase has attracted much more attention. Detection is often difficult, and the response to treatment may defy measurement. Many paramedic training programmes devote much time to this subject, and it is therefore not surprising that these taught skills are often put into practice in the uncontrolled pre-hospital setting. Mattox et al. [4] in a randomised controlled trial of the use of medical antishock trousers found no evidence of their therapeutic benefit. Indeed, patients with cardiac and thoracic vascular injury were adversely affected by their use. Analysis of 784 patients in the trial showed an overall mortality in the group in which MAST were used of 31% compared to 25% in the control group ($p = 0.05$). Similarly Kaweski et al. [5] found that the administration of fluids had no effect on outcome in 6855 trauma patients. This carefully controlled study took into account injury severity, pre-hospital time, body system injured and initial systolic blood pressure. Comparison of matched groups in all cases failed to reveal any beneficial effects of intravenous transfusion in the pre-hospital phase. Both these studies were conducted in systems which employed paramedics in the pre-hospital scene. In contrast, there is increasing interest in the selective provision of physicians. Some European systems already utilise physician-based pre-hospital teams (see below), and there is a recognition in North America that if the right person can get to the right place at the right time, lives can be saved. For example, Wall and Mattox [6] have recently described a case of roadside thoracotomy which permitted aortic compression until the patient could be transported to hospital for lobectomy. The patient made a full recovery and returned to work. A useful summary of the outstanding issues in relation to the transport of casualties to hospital has been prepared by Cross [7].

Hospital Reception

The importance of integration of effort is nowhere better demonstrated than in the emergency department. Driscoll and Vincent [8] have shown that organising a trauma team "horizontally" so that a well-rehearsed system is put into action with various assessments and interventions being carried out concurrently is much more effective than a "vertical" system in which decision-making consequently leads to delay and a poorer outcome. The Advanced Trauma Life Support (ATLS) course developed by the American College of Surgeons [9] has been widely recognised as providing a useful foundation on which to build hospital management of the seriously injured patient. It is perhaps a reflection of the generally inadequate data collection systems currently available that relatively few studies have attempted to measure the effectiveness of this widespread system of care. The most widely quoted (Ali et al. [10]) although claiming to show a significant benefit after the introduction of ATLS to a trauma care system has been criticised on methodological grounds.

Variations in Trauma Care Across Europe

This is not an appropriate place to list the very many different systems currently established throughout Europe. It is, however, important to reflect that it would be incorrect to use the word "developed" as most systems have evolved piecemeal in response to local crises and inefficiencies. Until recently there has been very little active planning of entire trauma care networks.

The system in the United Kingdom is based around accident and emergency departments and the specialty of emergency medicine – a political initiative developed in response to perceived inefficiencies in the reception of casualties in hospitals. The SAMU system in France is also a political initiative, but in this instance developed outside the hospital system. The central involvement of anaesthesiologists has provided an opportunity to study the effectiveness of the provision of physicians in the pre-hospital setting; and it is unfortunate that there are insufficient data available to rigorously test the effectiveness of this system. Preliminary data suggest, however, that it is at least as effective as the British system at delivering patients rapidly to definitive surgery [11]. The modified SAMU system in Belgium provides physicians at scene in response to requests from the first responding ambulance. Although a less resource-intensive system, there is no evidence that it is more or less effective than the French system [12]. In Germany helicopter transport is much more frequently used with physicians on board. Schmidt et al. [13] compared their experience with a similar evacuation system in North America. In the German model larger volumes of intravenous fluid were infused, incidence of intubation was much higher, and thoracic decompression was much more frequently undertaken. The authors concluded that their slightly lower mortality rate (9.5% of 221 cases versus 11.3% of 186 cases in North America) was due to the presence of a trauma surgeon on the flight crew being able to intervene more effectively than a nurse paramedic in the North American system.

Measuring Effectiveness

There is increasing awareness of the importance of measuring the process of trauma care as an aid to developing optimal systems. This can be achieved only if the "input" and "output" are also known. Developments in ascribing numerical scores to these variables have been achieved in recent years so that, at least in retrospect, management of apparently similar groups of patients can be compared.

The effects of an injury have been defined in terms of an anatomical component and the physiological response on the input side and the resultant morbidity and mortality on the output side. Elderly persons and young children survive trauma less well than others, and age must therefore also be taken into account. The mechanism of injury is also important – the effect of a blunt impact from a fall or a car crash is quite different from that of a stab or gunshot wound. If all these aspects were taken into account in the development of the severity score it

would be possible to compare not only one centre with another but also the varying effectiveness of a particular centre over time. This process would then provide a statistical basis for audit and allow some measure of effectiveness. There is understandable caution about such an approach and a healthy scepticism for the validity of the analyses.

Currently available methods use only survival or death as an outcome measure. The assessment of morbidity has until recently been largely neglected. This is an important omission as there are at least two seriously and permanently impaired survivors for every person who dies from an injury. Nevertheless, the concept of clinical audit by case analysis and the assessment of institutions on the basis of mortality statistics rather than by outdated clinical dogma has generally been well received. The Trauma Score and Injury Severity Score (TRISS) methodology is now widely used for this purpose [14]. The following brief summary provides the reader with some background information and draws attention to current problems and potential developments in the expanding field of injury scoring.

The Abbreviated Injury Scale is based on the AIS dictionary which scores from 1 (minor) to 6 (currently unsurvivable) over 1200 injuries. Patients with multiple injuries are scored by adding together the squares of the three highest AIS scores in six regions of the body. This is the Injury Severity Score (ISS) [15] (see Klasen et al., this volume). The extent of physiological derangement is described by the Revised Trauma Score (RTS) [16]. This combines coded measurements of respiratory rate, systolic blood pressure, and Glasgow Coma Scale (GCS). It was developed from statistical analysis of a large North American database to determine the relative usefulness of a variety of outcome variables as predictors of survival. The ISS is often underestimated when the patient is first seen either at the roadside or in hospital. It can only be finally determined from operative findings, appropriate investigations or necropsy reports. In contrast, the RTS changes as resuscitation progresses. It is therefore a much less satisfactory score. Further, it is influenced by the extent and timing of anaesthetic interventions. Alternative methods of measuring physiological derangement continue to be sought, but currently the RTS is the best method available. By convention the score used for TRISS analysis is that which is recorded when the patient first arrives in the hospital's emergency department. It is acknowledged that this immediately introduces bias if comparisons are sought between systems which use anaesthetists and paramedics in the pre-hospital phase.

Nevertheless, the TRISS methodology is now in widespread use [14] and combines the four elements – RTS, ISS, patient's age and nature of the injury (blunt or penetrating) – which are considered the most relevant input variables. The probability of survival (P_s) is determined using logistic regression:

$$P_s = \frac{1}{1 + e^b}$$

where e = natural logarithm, $b = b_0 + b_1\,(RTS) + b_2\,(ISS) + b_3\,(a)$, a = 0, if age 54 or less, a = 1, if age 55 or greater. It is important to appreciate that P_s is merely a

mathematical calculation – it is not an absolute measure of mortality but only of the probability of survival or death. If a patient with a P_s of 80% dies, the outcome is unexpected in that four out of five patients with such a P_s would be expected to survive. However, the fifth patient would be expected to die, and this could be the patient under study. Although this is self-evident to statisticians, it has caused confusion in clinical audit. The P_s is best reserved as a tool to analyse retrospectively the management of large groups of patients. It certainly should not be used as a prognostic indicator.

Statistical analysis of the management of a large group of patients, for example, those treated in one hospital or by one type of trauma service, can best be achieved by using the *W* statistic. This measures the difference between actual and predicted numbers of survivors per 100 patients. The *Z* statistic measures the significance of this *W*. This system was first developed in North America [17] and has been further developed in the United Kingdom as the Major Trauma Outcome Study (MTOS) [18]. The MTOS now collects data from over 50% of all trauma-receiving hospitals in the United Kingdom and collaborates with other centres in Belgium, France, Poland, Romania and the Czech Republic. Inevitably, much effort has been put into developing the system. It will be some time before it becomes sufficiently robust to provide representative data at a national level. However, it is already clear that there are major variations in the *W* statistic among hospitals. It is more difficult to identify the causes of these variations. These undoubtedly include differences in input which are not taken into account in the methodology – for example, pre-morbid state – but as the system becomes more robust and the data more accurate, it is expected that the MTOS will be a valuable tool which can be used with confidence to measure the effectiveness of trauma care provided by the different systems which currently exist across Europe.

Potential for Improvement in Care

The improvement of clinical care is but one part of the overall effort to reduce the size of the trauma epidemic. It is, however, the one which is most immediately appealing to clinicians and an enormous topic, central to the purpose of this symposium.

Much has been written about the influence of patient volume on effectiveness of trauma care. Smith et al. [19] studied 1643 seriously injured patients treated in hospitals in the Chicago area over a 22-month period. They concluded that those departments which see a large number of seriously injured patients (over 200 in the study period) have a higher survival rate than those seeing fewer than 140 patients. Another North American group [20] demonstrated that patients treated in trauma centres have significantly fewer complications and lower mortality rates than those treated elsewhere. They also found that delay in transfer time to the operating theatre significantly increases intensive care utilisation and length of stay in all types of hospital, irrespective of size.

However, these widely held beliefs have also been challenged. Schiowitz [21], using the methodology described by Smith [19] found that his department had a

lower mortality rate than the Chicago mean despite being much smaller. He emphasised that there is more to effectiveness than size and called for better use of scoring systems to more accurately measure input variables. Smith et al. [19] had used only a modification of the Trauma Score to control for severity, without an anatomical measure such as the ISS. Similarly Wardell et al. [22], reporting from Toronto and using the TRISS methodology to standardise comparisons, found no relationship between predicted and actual outcome in their relatively small centre. They concluded that despite their small size comparable clinical results could be achieved by surgeons dedicated to trauma management. The message from North America is therefore not as clear as many would like. Despite general enthusiasm for large departments using ATLS protocols, there are no hard data to support these generally acknowledged trends. Only Driscoll's work [8] gives statistical support for a specific method of managing trauma inside the hospital.

With regard to specific management, this will be covered in greater detail elsewhere, but it is worth highlighting here some specific developments which offer opportunities for improved care over the next decade.

Perhaps the protection of threatened neurones is the most exciting area. There are two potential avenues of progress – the manipulation of the chemical environment of a neurone and the implantation of neuronal tissue. These developments have been elegantly described by Gentleman [23], who has also emphasised the increasing awareness of "neuronal plasticity", introducing a much more optimistic approach to the management of brain and spinal cord injury than was previously evident. Although the prevention of secondary brain injury clearly continues to attract much attention, we need to appreciate that the primary injury can also be addressed. There is now the potential to protect neurones from the spiralling cascade of chemical, mechanical and ischaemic damage.

Minimal access surgery is now being considered to assess and manage some trunk injuries. Thoracoscopy [24] can be used to identify those chest injuries which require formal thoracotomy. Laparoscopy [25] can be a useful additional investigation of abdominal wounds if a recently described "gasless" technique is used. Fracture management has moved away from a purely mechanistic approach towards a more balanced management of limb trauma which emphasises the importance of fracture biology and the increasing understanding of factors which promote osteogenesis [26].

Common to all these developments is an understanding that management of the multiply injured patient must be integrated at all stages. Oakley [27], on behalf of the British Trauma Society has proposed a method of establishing national standards for the care of the injured in the United Kingdom and provided in an appendix a list of skills and equipment required to achieve these goals.

Once standards have been established and accepted, the process of audit can be pursued on a more scientific basis. The scoring systems described above provide statistical tools with which to measure clinical effectiveness. This can be used in relation to individual treatment and also entire trauma systems. Patient confidentiality must be assured, but the question of naming institutions in comparative league tables is now causing some concern. The importance of running an audit in a non-threatening environment must be recognised [28].

Controlling the Trauma Epidemic

The Environment

The most significant achievement at a European level in the area of prevention has come from the Transport Lobby. The European Transport Safety Council [29] has proposed a research-based strategy which could be developed to mitigate the effects of all types of trauma. Their proposals are as follows:

- Establishing data systems to identify priorities and evaluate policies
- Controlling exposure to risk
- Crash prevention
- Behaviour modification
- Mitigating effects of crash – i.e. injury control
- Improving clinical management
- Integrating data collection with clinical management and environment analysis
- Closing the loop by multidisciplinary audit

Systems such as the MTOS will do much to improve the collection and analysis of clinical data. However, further progress must be made in linking this information with the environmental data routinely collected by the police. Although there are a few isolated local initiatives, there is no general acknowledgement of the importance of linking police and clinical data and feeding this information back to policy makers, road engineers and the like. It is to be hoped that the European Transport Safety Council can bring together these disparate groups.

The importance of biomechanical factors in the aetiology and prevention of injury is increasingly recognised by clinicians. MacKay [30] has done much to bring these aspects of injury causation to the attention of clinicians, emphasising that a knowledge of the biomechanics of impact can be of direct use to a clinician during the resuscitation phase as well as to the epidemiologist who is attempting to gain an insight into the aetiology of crashes.

It has been estimated [29] that the application of known scientific data about crash protection in vehicle and road design into European legislation could reduce deaths and serious injuries by as much as 20%. This translates into 65000 deaths and serious injuries prevented on an annual basis if car frontal impact legislation were to reflect the findings of scientific evaluation of real accident data.

Although the implementation of these proposals would be expensive initially, there would be a very significant reduction in the overall community budget by reducing the societal costs of death and disability. Research is also required to protect vulnerable road users such as pedestrians and cyclists – cars now kill more persons outside of the cars than inside them.

This is not the forum in which to pursue changes in transport and economic policy, but it is reassuring to note the increasing involvement of clinicians in these areas. This is, however, a long-term strategy.

Education

Educational initiatives are much more likely to have an immediate impact. The ATLS system is discussed above [9]. One of the most remarkable innovations in these courses is the concept that educational theory should have a place in medical education. Until recently it had been assumed in medical circles that the possession of factual knowledge was the sole attribute required for the clinical teacher. Understanding educational principles was considered to be a time-consuming irrelevance. Experience of the ATLS system has helped to change attitudes. Increasing emphasis is now being placed on teaching the teachers how to teach. Feedback from students indicates that this is greatly appreciated. Indeed many medical schools and some postgraduate institutions now employ staff whose task is to ensure that the lecturers understand the basic components of the educational process. Using this method, undergraduates and postgraduates in medicine, nursing and the paramedic specialties learn collectively how to put together a trauma care system which from its very inception is integrated both vertically and horizontally. Each part of the process is analysed. The philosophy is to identify objectives, organise an appropriate teaching environment, present the material and then evaluate its effect. The teacher must be able to distinguish between knowledge, attitudes and skills. The student usually absorbs material which builds on more than one of these components, but the analytical approach enables the teacher more effectively to assess his or her performance and the student's response.

Four types of teaching may be used. The formal lecture is of only occasional use in trauma care, to provide basic facts prior to case-based teaching, group work or practical skills teaching. Most valuable is the small group discussion with a concise objective, in which the students are actively involved. The teacher must gain skills to be able, on the one hand, to involve the reluctant student and, on the other, to harness the enthusiasm of the more dominant members of the group [31].

The progressive assimilation of knowledge through undergraduate and postgraduate training is increasingly acknowledged and is particularly relevant to trauma care. The approach varies depending on the topic. For example, the didactic teaching of immediate resuscitation and the importance of team integration requires a different attitude and environment to that used to discuss the epidemiology of injury and prevention strategies.

Conclusion

The trauma epidemic across Europe is avoidable. Many injuries need not have been sustained, and many of the injured need not have died. Established systems of trauma care have evolved piecemeal. The measurement of effectiveness and the development of clinical audit encourage a more systematic approach to the provision of trauma care. There are exciting potential clinical developments, particularly in relation to CNS trauma, and it is essential that a system is in place which can deliver these benefits to the patient at the appropriate time and in the

appropriate place. The development of the concept of "evidence-based medicine" which emphasises the importance of randomised controlled trials in the assessment of efficacy has much to offer trauma care.

Imparting this new information to undergraduates and postgraduates using proven educational techniques will facilitate the development of a system of trauma care which utilises the best knowledge and the best skills in the best environment thereby ensuring that the injured patient is given the best chance of survival.

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Methods of Registration and Injury Severity Scoring

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Introduction

There are several reasons why workers in the field of health care need to register the data from their patients. To facilitate patient-related research a registration system must include patient data on such factors as date of birth, sex, registration number, hospital number, address, and accurate diagnostic data. The collection and analysis of a large quantity of data on many patients requires the help of a computer. Modern computing facilities allow the storage and retrieval of information in a much shorter time. Physicians tend to develop individual registration schemes according to their preferred methods of diagnosis classification. Such a system may be helpful for one's personal use, but the compatibility with other registration systems may be poor due to the differences in methods of classifying diagnosis. Therefore it seems useful to develop a nationally or internationally accepted method of classification.

One of the main problems in setting up a new registration system is determining the degree of detail in the system. A small group of persons working closely together usually have an interest in a particular group of patients. In such a setting the persons involved in the registration use identical criteria for data collection. In addition, they are usually motivated to collect data, as they are aware of the importance of such information. However, optimal registration is problematic when a large group of persons is involved in the registration, for example, junior physicians working in a busy emergency department, and depends upon the organization of the department. Junior physicians have little expertise, are busy, and are interested primarily in treating patients. They often have no idea why information is collected and usually have no experience of the benefits of registration. Therefore, registration by a group of inexperienced physicians requires a simple system with a limited number of items.

The first step in registration is the development of a medical chart, a simple map consisting of a precoded form on which the junior physician fills in the injury diagnosis and the cause of the accident.

Registration of Trauma Diagnosis and Cause of Accident

The International Classification of Diseases (ICD) is a classification system of causes and diagnoses of injuries developed by the World Health Organization. The nineth revision, clinical modification, (ICD-9CM) is currently in use. A Dutch version of the classification was published in 1980 [1]. The chapters on injury diagnosis rely upon the classical injury classification: fractures, dislocations, sprains, open wounds, intracranial injuries, etc. In the majority of Dutch hospitals injury diagnoses are registered according to the ICD-9CM.

The process of translating the diagnoses into ICD-9 categories may sometimes be confusing, for example, blunt thoracic injury can be registered either as "fracture of ribs," or as "pneumothorax and lung contusion." In this case three different ICD-9 codes must be registered. A new version of the classification system (ICD-10) is currently being prepared and is expected to be introduced into clinical practice within a few years. This classification system is already in use in Denmark. The ICD-10 categories for injuries will be tested in the Netherlands in 1995. ICD-10 differs from ICD-9 in that primarily body regions are identified, followed by subdivisions related to clinical diagnoses (fractures, etc.). ICD-10 specifies fewer anatomical diagnoses (for instance, fractures) than ICD-9 and in this respect resembles ICD-8.

Hospital Registration

Abbreviated Injury Score

In 1943 DeHaven of the Cornell University Medical College developed the Airplane Crash Injury Rate (ACIR) scale to grade injury severity in patients involved in airline crashes. This ACIR scale was introduced some years later for victims of car accidents. Cooperation between the automobile industry and the medical profession led to the development of the Vehicle Damage Scale and Injury Scale [2]. The Committee on Medical Aspects of Automotive Safety [3], composed of representatives of several (medical) specialities in the United States, developed an improved scoring system for tissue damage. Among other reasons this system was developed for physicians and researchers to be helpful in setting up prevention programs. This resulted in the Abbreviated Injury Scale (AIS), based on an injury severity scoring system developed by the General Motors Corporation. As the name of the committee suggests, AIS was designed especially for victims of motor vehicle accidents.

AIS classifies injuries separately in five body areas: "head and neck," "chest," "abdomen," "extremities and/or pelvic girdle," and "general, including skin." The severity of injury is coded per body area on a ten-point scale (0 = no injury, 6–9 = fatal injury, based on the police code). Severity is determined on the basis of mortality, disability, and length of hospital stay. The value of AIS is limited, as various criteria used in rating cannot be identified and separated. Indeed, the weight given to the various items varies from researcher to researcher because

each item can be interpreted differently, and the categories do not include clear clinical diagnoses but are descriptive in nature.

AIS was revised in 1976, 1980, 1985, and 1990 [4, 5]. In the 1985 revision the Committee on Injury Scaling remarked that, "The AIS had evolved in the universal system of choice for assessing impact injury severity." In 1976 the first AIS dictionary was published, listing more than 500 injury descriptions. The 1985 revision contained nearly 2000 diagnoses. The AIS-80 recommended that the highest score be used in multiply injured patients, as a surrogate for assessing overall injury severity. AIS-85 categorized injury descriptions into the same seven section headings as did AIS-80 ("external," "head," "neck," "thorax," "abdomen and pelvic contents," "spine," "extremities," and "skeletal part of the pelvis").

Each injury was assigned a unique six-digit code to assist in:
(a) data collection,
(b) computerization of injury information as to body region, organ or specific area,
(c) severity level, and
(d) AIS severity code.

The AIS scores overall injury severity, but the Maximum AIS provides information about the severity per body region. The 1990 version of AIS was extended, with synonyms and parenthetical descriptions. The addition of injury descriptions in AIS-90, especially in the brain and extremities, required a more flexible numerical system than used in AIS-85. In AIS-90 each injury description is assigned a unique seven-digit numerical code in addition to the AIS severity. The first digit identifies the body region, the second the type of anatomic structure or the specific nature of the injury, the third and fourth the specific anatomic structure or the specific nature of the injury, the fifth and sixth the level of injury within a specific body region and anatomic structure. The last digit is the AIS severity score.

Injury Severity Score

In 1974 Baker et al. [6] developed the Injury Severity Score (ISS) as an overall severity index for injuries, enabling the evaluation of the quality of emergency care in patients with multiple injuries. Categorizating patients into groups based on anatomical diagnosis requires scales such as the AIS. Baker et al. studied how the AIS scores are correlated with mortality. For this they had to modify the AIS, excluding code 6–9 (fatal, within 24 h), as the use of these codes would have prohibited computing meaningful death rates for various severity codes. Furthermore, they distinguished facial injuries from cranial/neck injuries because facial injuries occur more frequently in automobile crashes and thus might overshadow other head injuries. Diagnoses were coded according to the AIS code book. ISS is calculated by summing the squared highest points in each of the three most severely injured areas. ISS was compared with AIS data in 2000 patients (motor vehicle accidents, pedestrians, and other road users), and the correlation between

severity of injury and mortality was much higher with ISS than with AIS for the most severe injuries. ISS combined with age also was a valuable predictor of death [7]. Injuries which in isolation would normally not be life threatening were shown to have a dramatic effect on mortality when occurring in combination with other injuries [6, 8]. Several studies have confirmed the utility of AIS and ISS for describing injury severity and predicting mortality, length of hospital stay, disability, functional recovery, and long-term psychological consequences in patients involved in traffic accidents [9, 10]. However, its applicability to penetrating injuries is uncertain [8]. Also, assigning AIS and ISS requires the examination of the patient's medical record, which is time consuming [11, 12].

Therefore, Baker et al. [6] proposed the development of a map between hospital discharge diagnoses, coded by ICD-9 categories and AIS. Several researchers working independently have developed other conversion systems. The modified computerized table of MacKenzie et al. [9] incorporates the assignment of AIS scores to all ICD-9CM injury-related rubrics. These authors point out that the severity of a compound injury may lead to underestimation of ISS if different codes of ICD-9CM are used for such an injury. Therefore Werkman et al. [10] proposed increasing the AIS scores in order to improve the estimation of injury severity when combinations of diagnoses are present (e.g., in patients with combined maxillofacial injuries). Based on these proposals, Kingma et al. [11] developed the ICDTOAIS program, which improves and extends MacKenzie's mapping table. Since ICDTOAIS can be employed in database management systems and as a stand-alone program, the assignment of injury severity to ICD-9 diagnoses can be automatized in trauma registration.

Hospital Trauma Index

The Hospital Trauma Index (HTI) [13] was proposed to standardize and quantify the degree of injury in patients in order to compare mortality and morbidity in various patient groups. HTI is based on scoring six areas: "respiratory," "cardiovascular," "abdominal," "nervous," "extremities," "skin and subcutaneous," and "complications." Some items, including respiratory, abdominal, and extremities, are scored with 0–5 points (0 = no injury, 5 = critical) and others with 0–6 points, such as cardiovascular, nervous, skin and subcutaneous (burns), and complications. Fatal injuries receive a score of 6 (Table 1).

HTI/ISS

AIS and HTI differ in that AIS includes only anatomical diagnoses while HTI includes anatomical diagnoses and physiological parameters such as blood loss (Table 1). If one organ system has more than one injury, the score on HTI is raised one level.

Werkman et al. [10] studied the HTI and AIS for evaluating ISS in 932 patients with polytrauma (defined as HTI/ISS $\geq$ 18 points). The goal of the study was to

Table 1. Hospital Trauma Index (from [13])

Injury	Class	Index
Respiratory		
No injury	No injury	0
Chest discomfort, minimal findings	Minor	1
Simple rib or sternal fx, chest wall contusion with pleuritic pain	Moderate	2
First or multiple rib fx, hemothorax, pneumothorax	Major	3
Open chest wounds, flail chest, tension pneumothorax, nl bp, simple laceration diaphragm	Severe	4
Acute respiratory failure (cyanosis), aspiration, tension pneumothorax with ↓ bp, bilateral flair, lac(s) diaphragm	Critical	5
Cardiovascular		
No injury	No injury	0
< 10% (< 500 cm^3 blood volume, bv) loss, no change in skin perfusion	Minor	1
10%–20% bv loss (500–1000 cm^3), ↓ skin perfusion, urine nl (+ 30 cm^3/h), myocardial contusion, bp nl	Moderate	2
20%–30% bv loss (1000–1500 cm^3), ↓ skin perfusion, urine (> 30 cm^3), tamponade, bp 80	Major	3
30%–40% bv loss (1500–2000 cm^3), skin perfusion, urine (< 10 cm^3), tamponade, conscious, bp < 80	Severe	4
40%–50% bv loss, restless, agitated, coma, cardiac contusion or arrythmia, bp not obtainable		5
50% + bv loss, coma cardiac arrest, no vital signs	Critical	6
Nervous system		
No injury	No injury	0
Head trauma with or without scalp laceration(s), no loss consciousness (coma), no fracture	Minor	1
Head trauma with brief coma (< 15 min), skull fx, cervical pain with minimal findings, 1 facial fx	Moderate	2
Cerebral injury with coma (+ 15 min), depressed skull fx, cervical fx with neurological findings, multiple facial fxs	Major	3
Cerebral injury with coma (+ 60 min) or neurological findings, cervical fx with major neurological findings, e.g., paraplegia	Severe	4
Cerebral injury with coma with no response to stimuli up to 24 h, cervical fx with quadriplegia	Critical	5
Cerebral injury with no response to stimuli and with dilated fixed pupil(s)	Fatal	6
Abdominal		
No injury	No injury	0
Mild abdominal wall, flank or back pain and tenderness without peritoneal signs	Minor	1
Acute flank, back, or abdominal discomfort and tenderness, fx of a rib 7–12	Moderate	2
One of: minor liver, sm. bowel, spleen, kidney, body pancreas mesentery, ureter, urethra, fx 7–12 rib	Major	3
Two major: rupture liver, bladder, head pancreas, duodenum, colon, mesentery (large)	Severe	4
Two severe: crush liver, major vascular including: thoracic and abdominal aorta, cava, iliacs, hepatic veins	Critical	5

Table 1 (continued)

Injury	Class	Index
Extremities		
No injury	No injury	0
Minor sprains and fx(s), no long bones	Minor	1
Simple fx(s): humerus, clavicle, radius, ulna, tibia, fibula, single nerve	Moderate	2
Fx(s) multiple moderate, compound moderate, femur (simple), pelvic (stable), dislocation major, major nerve	Major	3
Fx(s) two major, compound femur, limb crush, or amputation, unstable pelvic fx	Severe	4
Fx(s) two severe, multiple major	Critical	5
Skin and subcutaneous		
No injury	No injury	0
< 5% burn, abrasions, contusions, lacerations	Minor	1
5%–15% burn, extensive contusions, avulsions, 3–6″ extensive lacerations (total 12″2)	Moderate	2
15%–30% burn, avulsions 12″2+	Major	3
30%–45% burn, avulsions entire leg, thigh or arm	Severe	4
45%–60% burn (3rd degree)	Critical	5
60% + burn (3rd degree)	Fatal	6
Complications		
No significant complications	None	0
Subcutaneous wound infection, atelectasis, cystitis, superficial thrombophlebitis, temp. < 38.5°	Minor	1
Major wound infection, atelectasis, pyelonephritis septic or deep thrombophlebitis temp > 38.5°	Moderate	2
Intraperitoneal abcess, pneumonia, anuria, or oliguria with – BUN (no dialysis), jaundice, < 6 U GI bleed, RDS < 1 day	Major	3
Septicemia, empyema, peritonitis, pulm. embolus (nl bp), renal failure (dialysis < 1 week), > 6 U bleed, < 3 days RDS	Severe	4
Septicemia with ↓ bp, pulm. emb. with ↓ bp, renal failure 7–40 days, GI bleed > 12 U, resp. arrest > 3 days RDS with ventilator	Critical	5
Pulm. emb. with cardiac arrest, renal failure > 6 weeks, coma > 6 weeks, > 30 days RDS with ventilator or > 80% O_2 < 7 days	Fatal	6

Minor, trivial injury; moderate, minimal injury, short hospitalization anticipated; major, major injury, not immediately life-threatening; severe, life-threatening but survival probable; critical, survival uncertain; fatal, survival unlikely. bp, Blood pressure; bv, blood volume; lac, laceration; nl, normal; RDS, respiratory distress syndrome; fx, fracture.

compare the effect of HTI and AIS values on the ISS, and the result was that the average HTI/ISS was 10 points higher than the average AIS/ISS, as has also been determined by others [14]. The higher HTI/ISS values may be explained by the fact that HTI item scores are raised by one level if more than one injury of comparable severity is present in one system, by the use of physiological parameters in HTI, and by the squaring of AIS and HTI values. Statistical

analysis has shown that HTI/ISS together with age predicts death more reliably than AIS/ISS. ISS, based on two different severity scales, shows quantitative and qualitative differences in the prediction of mortality. Therefore the authors recommended that publications mention the way in which ISS is composed [10]. In their opinion, AIS/ISS should be used in studies comparing large groups of patients treated in different hospitals, while HTI (which is more time consuming) is preferred in smaller, high-quality studies [10].

Registration of Prehospital Care

Trauma Score

It is now generally accepted that outcome of trauma care depends on the chain of care provided, starting at the site of the accident and extending up to posthospital care. All groups of caretakers involved should be able to evaluate the quality of their trauma care. This evaluation requires scoring systems, including relevant parameters about a specific part or period of care.

Champion et al. [15] developed the Trauma Score (TS) to measure injury severity based on simple physiological parameters. The main reason for devising TS was "field triage" and evaluation of prehospital trauma care. Points were scored for respiration (respiratory rate and quality of respiration), circulation (systolic blood pressure, capillary refill) and central nervous system (Glasgow Coma Scale, GCS). The GCS was introduced by Teasdale and Jennett [16]. This scale has been tested intensively under clinical circumstances to define the duration of coma, in terms of how long different levels of responsiveness persist. The GCS summarizes the scores from three criteria: eye opening (1–4 points), best verbal response (1–5 points), and best motor response (1–6 points). The TS consists of the sum of points obtained in GCS, the quality of respiration, and the quality of circulation. Champion et al. theorized that the worst TS within 1 h of injury might be an appropriate measure to evaluate trauma care [15]. In 1989 and 1993 TS was modified into the Revised Trauma Score (RTS; Table 2).

In fact, two versions of RTS were developed, one for triage (T-RTS) and one for outcome evaluation (RTS). In the RTS information about the item "circulation" is simplified. The assessment of capillary refill is deleted, systolic blood pressure (mmHg) becomes the only parameter of the quality of circulation, and respiratory rate is the only parameter for the item "respiration". Capillary refill and chest excursions are excluded as they cannot be obtained reliably in field circumstances. The RTS was als simplified because it was felt that severe brain damage must be scored more accurately. These modifications simplify the score and make it more reproducible [18].

RTS allows statistical analysis of outcome in trauma patients treated in various situations. Teyink [19] studied the use of the RTS by ambulance crew. The system was used by nearly 50% of the crews involved in emergency transport. Only 22% of the ambulance crews not qualified as a registered nurse used the score in such circumstances. Teyink pleaded for general introduction of the system on all

Table 2. Revised Trauma Score*

		Score
A. Respiratory rate	10–24/min	4
	25–35/min	3
	≥ 36/min	2
	1–9/min	1
	none	0
B. Systolic blood pressure	≥ 90 mmHg	4
	70–89 mmHg	3
	50–69 mmHg	2
	0–49 mmHg	1
	no pulse	0
C. Eye opening	spontaneous	4
	to voice	3
	to pain	2
	none	1
D. Verbal response	oriented	5
	confused	4
	inappropriate words	3
	incomprehensible words	2
	none	1
E. Motor	obeys command	6
	localizes pain	5
	withdraw (pain)	4
	flexion (pain)	3
	extension (pain)	2
	none	1
F. Glasgow Coma Score:	Total (C + D + E)	
G. Glasgow Conversion Score	13–15 =	4
	9–12 =	3
	6– 8 =	2
	4– 5 =	1
	< 4 =	0
Trauma Score Total:	= A + B + G	

* Adapted with permission from Champion HR, Sacco WJ, Copes WS et al. A revision of the Trauma Score. J Trauma 1989: 29(5):624/Resources for optimal care of the injured patient 1993, 22

ambulances. De Man [20] studied the value of TS for the individual patient, using a slight modification of TS, the so-called Condition Score. He concluded that TS is unsuitable for measuring the need of care, despite the fact that ambulance crew used TS items for treatment decisions.

The TS also has inherent deficiencies [21, 22]: the sensitivity rate is approximately 80%, which means that 20% of patients with a severe injury are not identified. Its specificity is 75%; thus severity is overestimated when physiological

Table 3. Pediatric trauma score: category definitons*

Component	+2	+1	−1
Size	child/adolescent, > 20 kg	Toddler, 11–20 kg	infant, < 10 kg
Airway	normal	assisted O_2, mask, cannula	intubated; ETT, EOA, Cric
Consciousness	awake	obtunded, lost consciousness	Coma, unresponsive
Systolic blood pressure	> 90 mmHg; good peripheral pulses, good perfusion	51–90 mmHg, peripheral, pulses palpable	< 50 mmHg; weak or no pulses
Fracture	none seen or suspected	single closed Fx anywhere	open, multiple Fx
Cutaneous	no visible injury	contusion, abrasion; laceration < 7 cm; not through fascia	tissue loss; any GSW/Stab; through fascia

* Adapted with permission from Tepas JJ, Mollitt DL, Talbert JL, et al: The pediatric trauma score as a predictor of injury severity in the injured child. J Pediat Surg 1987; 22(1):15/ Resources for optimal care of the injured patient: 1993, 23.

The PTS is calculated by summing the numerical scores of the six components. The PTS can vary from +12 to −6

changes are related to other factors than those resulting from hypovolemia, cerebral edema, or hypoxia [22].

Pediatric Trauma Score

No limit has been established for using the TS, although it appears reliable in patients over 12 years of age. For children (0–18 years) the Pediatric Trauma Score (PTS) has been developed [23, 24, 25] (Table 3). This score is simple to use and designed to predict injury severity for triaging to the appropriate care facility. Ramenofsky et al. [26] studied the accuracy and predictive value of PTS and its sensitivity and specificity as a triage tool in 469 children. When used for triage, the sensitivity of PTS was 95.8% and specificity 98.6%. Children with a PTS above 8 demonstrated a 0% mortality and those with a PTS of 0 or below a 100% mortality. PTS not only predicted severity of injury but also identified the children (PTS range, 0–8) in immediate danger of dying without appropriate and timely intervention. The authors concluded that PTS is a straightforward modality for the triage of injured children.

The Trauma Score and Injury Severity Score Method

Boyd et al. [22] have noted that the term Trauma Score and Injury Severity Score (TRISS) methodology is frequently used in the literature but without a single, clear, concise, and complete source geared to nonstatisticians. Basic to the understanding of the TRISS method is the combination of Trauma Score and ISS. The TRISS method offers a standard approach for evaluating outcome of trauma, including age as a characteristic, to quantify the probability of survival as related to injury severity. Boyd et al. used the TS and AIS/ISS [22]. With these values the TRISSCAN chart was developed (Fig. 1), providing for a simple reference of probability of survival. Boyd et al. recommended the TRISSCAN as an educational tool, to emphasize the importance of interrelationships of the variables representing physiological derangement (TS), anatomic injury severity, and age (Fig. 1). Boyd et al. [22] also developed a TRISS combination of ISS and RTS. Bull and Dickson [27] applied the RTS, TRISS, and ISS/age to a group of trauma patients and compared the results with outcome (mortality). Although survival was not significantly different from that predicted using TRISS, the method seemed to place too great an emphasis on the initial clinical signs summarized in RTS, and the TRISS calculation seemed to make inadequate allowance for age effects in the elderly. They concluded that the simpler ISS/age method might prove a better basis for estimating overall survival.

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Fig. 1a,b. Probability of survival among blunt trauma patients as estimated using the TRISSCAN chart. TS, AIS-80, and corresponding coefficients are used. **a** Patients aged 54 years or younger. **b** Patients aged 55 years or older. (From [22])

TRISSCAN

TS	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75
1	.18	.13	.10	.07	.05	.03	.02	.02	.01	.01	.01	.00	.00	.00	.00
2	.27	.21	.15	.11	.08	.06	.04	.03	.02	.01	.01	.01	.00	.00	.00
3	.39	.30	.23	.17	.13	.09	.06	.05	.03	.02	.02	.01	.01	.01	.00
4	.51	.42	.34	.26	.19	.14	.10	.07	.05	.04	.03	.02	.01	.01	.01
5	.64	.55	.46	.37	.29	.22	.16	.12	.08	.06	.04	.03	.02	.01	.01
6	.75	.67	.59	.50	.40	.32	.25	.18	.13	.10	.07	.05	.03	.02	.02
7	.83	.78	.70	.62	.53	.44	.35	.27	.21	.15	.11	.08	.06	.04	.03
8	.89	.85	.80	.73	.66	.57	.48	.39	.30	.23	.17	.13	.09	.06	.05
9	.93	.91	.87	.82	.76	.69	.61	.51	.42	.34	.26	.19	.14	.10	.01
10	.96	.94	.92	.89	.84	.79	.72	.64	.55	.46	.37	.29	.22	.16	.12
11	.98	.97	.95	.93	.90	.86	.81	.75	.67	.59	.50	.40	.32	.25	.18
12	.99	.98	.97	.96	.94	.91	.88	.83	.78	.70	.62	.53	.44	.35	.27
13	.99	.99	.98	.97	.96	.95	.92	.89	.85	.80	.73	.65	.57	.48	.39
14	.99	.99	.99	.98	.98	.97	.95	.93	.91	.87	.82	.76	.69	.61	.51
15	.99	.99	.99	.99	.99	.98	.97	.96	.94	.92	.89	.84	.79	.72	.64
16	.99	.99	.99	.99	.99	.99	.98	.98	.97	.95	.93	.90	.86	.81	.75

ISS

a BLUNT TRAUMA AGE 54 OR LESS

TRISSCAN

TS	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75
1	.03	.02	.02	.01	.01	.01	.00	.00	.00	.00	.00	.00	.00	.00	.00
2	.05	.04	.03	.02	.01	.01	.01	.00	.00	.00	.00	.00	.00	.00	.00
3	.08	.06	.04	.03	.02	.01	.01	.01	.01	.00	.00	.00	.00	.00	.00
4	.13	.10	.07	.05	.03	.02	.02	.01	.01	.01	.00	.00	.00	.00	.00
5	.21	.15	.11	.08	.06	.04	.03	.02	.01	.01	.01	.00	.00	.00	.00
6	.30	.23	.17	.13	.09	.06	.05	.03	.02	.02	.01	.01	.01	.00	.00
7	.42	.33	.26	.19	.14	.10	.07	.05	.04	.03	.02	.01	.01	.01	.00
8	.55	.46	.37	.29	.22	.16	.12	.08	.06	.04	.03	.02	.01	.01	.01
9	.67	.59	.49	.40	.32	.24	.18	.13	.10	.07	.05	.03	.02	.02	.01
10	.77	.70	.62	.53	.44	.35	.27	.21	.15	.11	.05	.06	.04	.08	.02
11	.85	.80	.73	.66	.57	.48	.39	.30	.23	.17	.13	.09	.06	.05	.03
12	.91	.87	.82	.76	.69	.60	.51	.42	.33	.26	.19	.14	.10	.07	.05
13	.94	.92	.89	.84	.79	.72	.64	.55	.46	.37	.29	.22	.16	.12	.08
14	.97	.95	.93	.90	.86	.81	.75	.67	.59	.49	.40	.32	.24	.18	.13
15	.98	.97	.96	.94	.91	.88	.83	.77	.70	.62	.53	.44	.35	.27	.21
16	.99	.98	.97	.96	.95	.92	.89	.85	.80	.73	.66	.57	.48	.39	.30

ISS

b BLUNT TRAUMA AGE 55 OR GREATER

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Extrication and Immobilization of the Severe Trauma Victim: How It Is Done

W. Ummenhofer, H. Pargger, U. Boenicke, and D. Scheidegger

Introduction

The rescue techniques used for the entrapped patient depend on the nature of the accident and surrounding situation, the resources of the rescue team, the impending danger for patient and rescuers, the patient's stage of injury, and the amount of time required for extrication.

Persons may be trapped in a variety of ways: industrial accidents with large machines, building-site accidents where construction workers are buried by building material, forest workers hit by a falling tree, avalanche victims in mountainous regions, and of course the innumerable trauma patients from vehicular accidents.

Medical personnel are not trained in the technical aspects of extrication [1] because the skills and materials needed to rescue entrapped patients have become too complex and therefore require a specialized rescue team. To achieve an optimal patient outcome when extrication is performed, rescuers must accept their mutual interdependence [2]; medical teams and firemen (who have been well trained for the technical part of the job) must learn to understand the problems and intentions of each other. Often these two teams work concurrently but not complementarily, with the result being that the patient is lost between them (Fig. 1). Despite a high level of emergency medicine in the field and a tremendous amount of technical skill and equipment there is a frustrating gap of communication among the involved rescuers. There is little discussion of technical aspects of extrication in medical emergency literature and only scant consideration of medical decision making and priorities in fire brigade magazines and books.

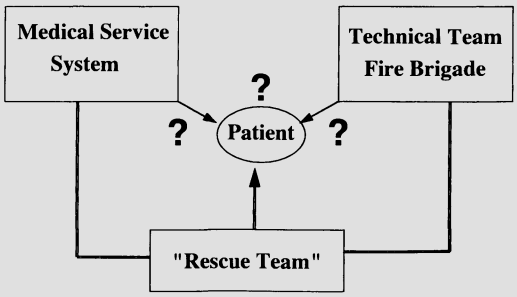

Fig. 1. The "entrapped patient"

General Principles of Extrication

Safety: For the Rescue Team and Patient

Most rescue scenarios involve an element of danger to the rescue personnel, and to ensure their safety sturdy clothing is required, consisting of protective overalls, shoes, helmets, and gloves. For mountain rescuing sophisticated equipment is needed, including climbing belts and special ropes; more importantly, the rescuers need to be retrained periodically in the use and repair of this special equipment. It is also necessary to be aware of extraordinary environmental hazards at the scene of an accident; for example, the rotor and the movements of a helicopter at an avalanche site can cause another avalanche.

In accidents with electrical wires there is a life-threatening danger for anyone who comes in close contact with downed wires. Therefore extrication efforts are not allowed in cases of train or tram accidents before electric power has been disconnected by specialized personnel from the power company. Nevertheless, rescue personnel are often killed accidentally in cases with downed wires, and it is strongly recommended that rescuers delay their activities until authorized power company employees have demonstrated the absence of electric voltage by touching wires, vehicles, or injured persons [3, 4].

In traffic accidents, especially on high-speed motorways, it is the task of the police to secure the emergency site. When medical personnel are the first to arrive, their vehicle must be parked in a protective and warning position and the road secured by means of accident signs and flashing warning lights. If there is a danger of fire or explosion, the help and skill of a fire brigade are needed; do not enter a burning car without a fire extinguisher or protective equipment.

When patients are trapped in a location with poisonous fumes, only the fire brigade has the appropriate breathing apparatus and knowledge of its proper use; therefore it is their responsibility to transport patients to a safe distance from the accident where they can then be provided medical care.

Coordination: Do Not Lose Time

In a case of expected extrication it is necessary to verify with the emergency alarm station that individuals required for the rescue have been sent to the scene of the accident. Although coordination is the task of the central alarm office, it is important to ensure that necessary rescuers have been notified. It is horrible to arrive at the accident site unable to help because fire brigade or power company technicians have not been sent.

Regarding medical resources that may be needed, it is necessary to consider whether an ambulance is sufficient for the expected situation, or whether a helicopter is required to take patients to a specialized trauma center. Again, these questions should be posed on the way to the scene, and one should also make sure that the coordinator is aware of any potential problems that are foreseen.

Rescue Tools: You Only Need To Be Familiar with Them, Not Use Them

Fire brigades supply professional rescue crews with a fast truck containing the equipment needed for extrication. As firemen are expected to have basic knowledge of medical life support procedures, the medical emergency team should also have some familiarity with the technical possibilities of the rescue team. Paramedics and emergency physicians do not need to become experts in the use of all the equipment, but they should know the principles, indications, and dangers of the technical tools [4].

Tools such as pliers, screwdrivers, and wrenches in various sizes are necessary to disassemble things. Hydraulic spreading tools are used to remove car seats and doors and to lift and pry crushed interior wreckage away from victims who are pinned in. These compressed-air devices should be positioned at a safe distance from the site of the accident to avoid the dangerous ignition of spilled fuel. Hydraulic spreading tools enable a fireman to lift 4.5 tons of weight up to a height of 80 cm by a touch of his finger. Cutting tools include various power driven and manual devices such as air chisels, bolt cutters, hacksaws, or giant can openers.

These tools are used for a variety of tasks that require gaining access by cutting through hard objects such as steering wheels and roof posts (Fig. 2). The roof of a damaged car can be cut off within 5–10 min. A major advantage of this technique is the ability to rescue a person suspected of spinal injury without rotation or compression of the spine (Fig. 3). Occasionally patients are jammed beneath heavy vehicle parts, such as in truck, tram, or train accidents. Air-compressed cushions are a quick and safe method to lift away even heavy loads.

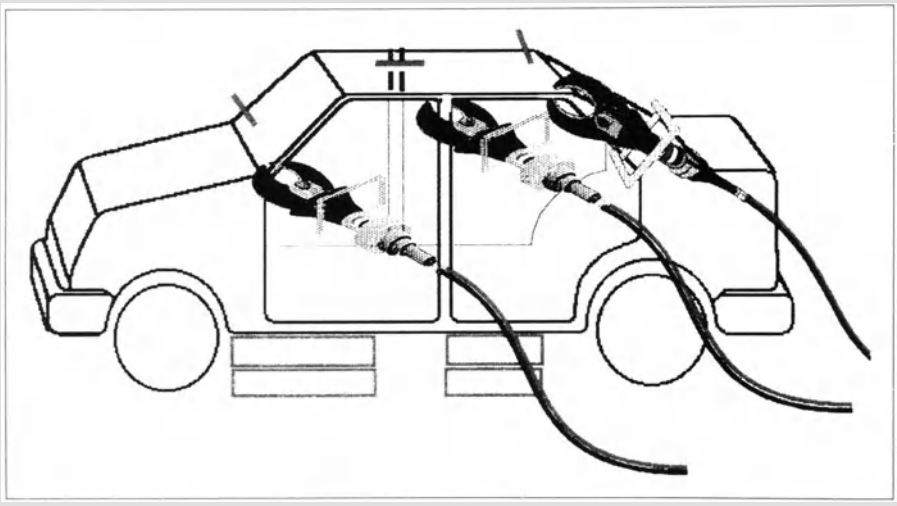

Fig. 2. Cutting through car roof posts (from [8])

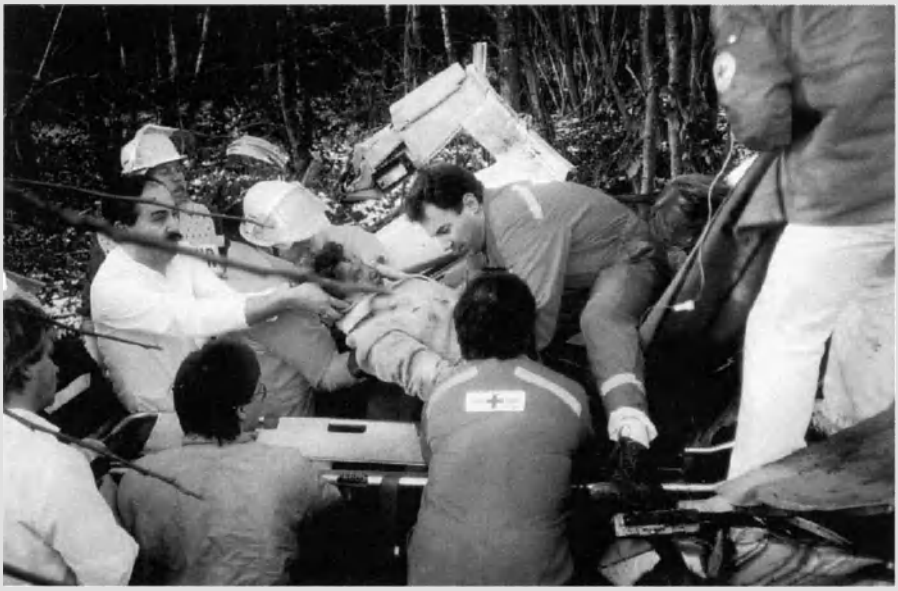

Fig. 3. Disentanglement of patient after cutting off the roof of the vehicle

It cannot be stressed strongly enough that safe and professional extrication procedures cannot be performed by a medical team alone. Such procedures require professional partners, mutual arrangement, and application according to prior agreement.

Stages of Extrication

Extrication can be divided into the following stages [5]:
1. Assessing the accident scene and controlling hazards
2. Establishing contact with the technical leader
3. Gaining access to the patient
4. Providing medical care to the patient
5. Consulting with the technical team as to the best way to extricate the victim
6. Disentangling the patient
7. Immobilization of the patient
8. Packaging the patient and removal from entrapment

Assessing the Accident Scene and Controlling Hazards

As mentioned above, safety of the rescue team has first priority. For traffic accidents it is necessary to ensure that police or other individuals prevent additional accidents.

When fuel has been spilled, the danger of fire or explosion must always be kept in mind. Usually fire occurs at impact or immediately afterwards. Late ignition during extrication can be prevented by careful technical procedures and positioning of the compressor at a safe distance.

New technologies can present new hazards, such as the "airbag problem" [6]. Modern cars are increasingly supplied with airbag systems for the driver and the front-seat passenger. Despite its controlled augmentation of passive traffic safety this new technology has been discredited as "a bomb behind the wheel." Airbag triggering occurs in the event of a frontal or up to 30° collision. Ignition of a pyrotechnical gas generator produces nitrogen which fills the bag. Following collision and inflation of the bag, this filling gas leaks immediately out in a controlled way from built-in slits.

If the airbag has been released, no special problems should be encountered. Nevertheless, depending on the patient's injury, you should mention that the airbag had been activated when referring the patient to the clinic. A nontriggered airbag can present problems, and therefore the ignition should be switched off and the battery disconnected. If certain parts of the car must be moved or cut away (especially the steering column), an unintended inflation of the bag is possible, even when the battery has been disconnected. Such an inflation results in an explosion, and one should always keep the head and upper part of the body away from this area. From a practical standpoint, one should first check to see whether a wrecked car is supplied with an airbag (usually the inscription "airbag" or "SRS" is on the steering wheel or dashboard).

It is important to survey the surrounding area carefully for downed electrical wires. In the case of railway or truck accidents hazardous materials may be present. It should be the fire brigade's task to identify them and inform you.

When the patient's vehicle is in an unstable position, it must not be entered. Different strategies can be used to stabilize the car. We had one case with a car positioned on a steep drop close to a river; we decided to lift the whole car to safe surroundings before delivering medical aid to the patient (Fig. 4).

Bystanders can become a problem when they expose themselves to unnecessary danger and impede the efforts of the rescue team. The so-called "reality TV" supports a strange kind of modern voyeurism that should be prevented by police intervention.

In assessing the scene it is necessary to develop one's own strategy of how to proceed. As Caroline [5] recommends, if there are both easy and hard ways to do something, the easier way should be tried first. Of course one can use heavy-duty power tools to cut open a damaged car door, but one should try the opposite undamaged and possibly unlocked door first: remember "you don't get extra points for doing things the hard way" [5]. In many situations heavy hydraulic tools are not required. Disentanglement of the entrapped person can often be achieved by a much easier means such as the sheer force provided by the judicious use of a crowbar.

One should consider the technical possibilities available at the accident scene. For example, to lift an injured worker out of a deep building site excavation a crane can be used after immobilizing the patient (Fig. 5). Bus or train accidents

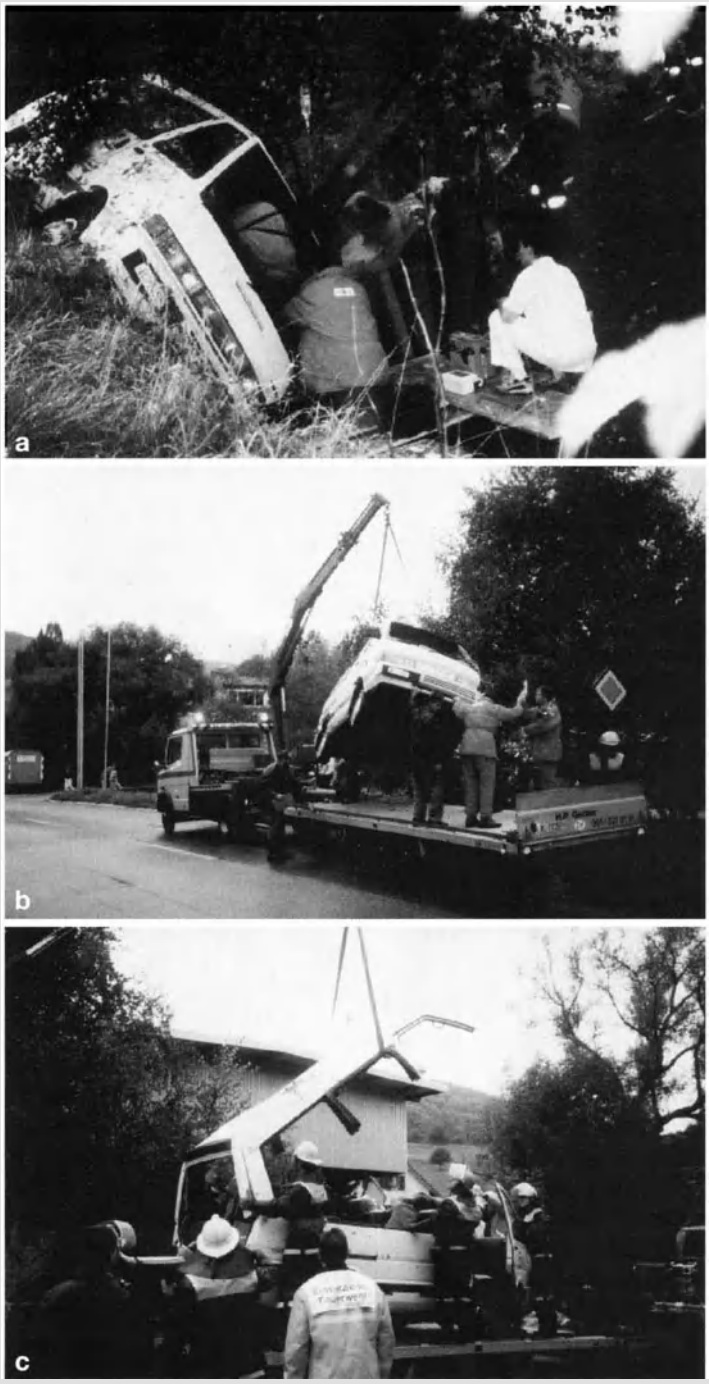

Fig. 4a–c. Bringing a car to a stable condition before extrication

Fig. 5a, b. Use of a crane to remove a buiding worker

represent a challenge with some unique concerns. Due to the absence of safety belts severe injuries are common, and access to the inside of coaches is often difficult because of the restricted doors and tangled wreckage. Furthermore, because of the high number of patients, triage scenarios are required including coordination at the scene and plans for sufficient transport and hospital facilities.

Special emergency situations require specialized technical skills. Similar to the cooperation between the medical team and fire brigade for traffic accidents, a sophisticated cooperation has been developed for mountain rescuing in the Swiss Alps. Along with the medical team experienced mountain rescuers are brought to the site of the accident by helicopter because only they can provide safe access via complex rope systems, for example to a patient inside a crevasse.

Establishing Contact with the Technical Leader

Similar to the classification "primary and secondary survey in examination of the trauma patient" [7], the emergency medical physician (EMP) should establish close contact with the technical leader of the rescue team upon arrival at the accident scene [8]. In contrast to the medical team, firemen are well trained for the technical part of extrication and have modern vehicles with efficient powerful tools with which to free the victims. Their enthusiasm to get the job done must, however, be tempered. Thus, it is important that the EMP immediately identify and address the technical leader to explain how he plans to treat the patient initially and to inquire as to the best way to gain access to the victim before the technical part of the extrication begins. The EMP must guide the rescuers in holding back their rescue efforts until the medical part is under control. This short briefing should be possible within 30–60 s, after which time the rescue team can prepare for the tool-based disentangling of the victim so that everything is ready when initial examination and medical treatment have been completed.

Gaining Access to the Patient

Following unsuccessful attempts to access the car's interior by trying all the doors, a fireman should provide access through a window (Fig. 6), preferably from the rear or side of the car farthest away from the patient to prevent additional harm. Even if most of the glass is broken out, this access remains dangerous and emphasizes the need for protective clothing and gloves.

Providing Medical Care to the Patient

Emergency medical teams supply medical aid with mobile equipment units. This implies that everything needed for examination, treatment, and monitoring can be provided at the site of an accident. While the EMP is performing the primary medical examination, the paramedic prepares and unpacks the medical resources such as oxygen, infusions, intravenous catheters, and cervical collars.

The first action inside the car is to ensure that the ignition is turned off. If more than one victim is inside the car, aid should be administered first to the most critically injured person. A trauma patient without a pulse or with an asystole in the ECG would have a very poor outcome [9], and resuscitation should be per-

Fig. 6. Access through a window

formed only when there are no other victims, with better prognoses. The emphasis in trauma care resuscitation is therefore on prevention rather than therapy. Early treatment of airway obstruction, hypoventilation, hypovolemia, ongoing hemorrhage, and obstruction of the venous return are priorities [10].

The sequence of action must follow the ABCs rule: first try to communicate with the patient to find out his level of consciousness and physical complaints. For the unconscious patient the first priorities are airway and cervical spine control. Perform the chin-lift maneuver and try to apply a cervical collar as soon as possible. If the victim is breathing spontaneously, supply high-flow oxygen via a face mask. If the patient is apneic, consider the possibilities for performing an intubation inside the wreckage.

Circulation is assessed by palpating the appropriate pulses. Blood pressure measurement in this early stage is a waste of time, as a palpable pulse corresponds to a systolic blood pressure of about 80 mmHg and a palpable carotid pulse to one of at least 60 mmHg. When no pulse is present, the patient needs mechanical cardiopulmonary resuscitation, which cannot be performed sitting in the seat of a car. This means that the patient must be immediately removed from the wreckage irrespective of additional injuries; however the poor chances of success in these cases should be considered.

Hemorrhage control has a high priority during the initial efforts, and it is the only therapy in the early phase of hypovolemic shock. Therefore there is little sense in infusing high amounts of crystalloids or colloids without first trying to control profuse bleeding from an open fracture by means of direct pressure. As potential internal bleeding or bleeding from lower extremities cannot be stopped

Fig. 7. Victim pinned behind steering wheel

at this stage, the next step must be insertion of one or, even better, two large-bore intravenous cannulas in the patient's forearms or the external jugular veins. In spite of the suboptimal working conditions there are generally better chances to perform successful punctures inside the car while the victim's jammed legs, pelvis, and/or abdomen are compressed by the steering wheel. This may cause a certain type of tamponade (Fig. 7) that often distends the veins better than after extrication, even in the presence of significant hemorrhaging. It is essential to concentrate on proper fixation of the infusion and is therefore worthwhile to fix the cannula with an additional bandage to ensure that it remains in place when the patient is packaged and removed from the car. The ability to establish a new intravenous line may dramatically lessen when exsanguination begins after extrication due to the missing tamponade.

Analgesia and anesthesia should begin while the patient is still inside the wreckage if access can be gained to an intravenous line and the patient's airway. Controversy remains over the principles of "stay and stabilize" [11] versus "scoop and run" [12], and the best approach may lie somewhere between these two. There is no doubt that the time factor of prehospital delay is an important prognostic index of outcome for the severely injured patient.

Stabilization of a hypovolemic patient, with the potential hazard of internal bleeding, using only crystalloids or colloid infusion is difficult to attain at the accident scene, where there is no possibility of a blood transfusion or surgical control of hemorrhage. Furthermore, there are interesting animal data on survival rate related to mean arterial pressure in a hemorrhage model; the highest survival rate was found in the group with the lowest blood pressure, and mortality

was greatest and mean survival time shortest in the group with the highest blood pressure [13].

Applied to extrication of the patient, we must realize that stabilization of hemodynamic parameters often cannot be achieved at the accident scene, and that normalization of blood pressure is not a worthwhile goal and may even be dangerous in the absence of hemorrhage control. However, replacement of blood loss is only one aspect of prehospital care. "Scoop and run" also implies unplanned and unsafe disentanglement with the potential danger of further damage and impairment of coexisting injuries, neglects the risks of respiratory distress, and seems to deny the great advantage of relief by merciful analgesia and anesthesia before extrication and transport. Our goal is not to try to stabilize the patient inside the wreckage, but rather we aim for a condition that is safe with a minimal amount of stress. This means that analgesia is provided for patients with minor injuries, and if airway control can be achieved, we try to perform general anesthesia for multiple trauma patients while they are still inside the car, and before the technical part of the rescue is performed.

For analgesia, opioids such as morphine, fentanyl, or ketamine in an analgesic dosage (0.25–0.5 mg/kg) are used, and for induction of general anesthesia we prefer ketamine (1–2 mg/kg) in the absence of central nervous damage, together with succinylcholine to facilitate endotracheal intubation. In the unconscious patient with potential severe brain damage, fentanyl and etomidate are substituted for ketamine. Controlled ventilation with a minimum FiO_2 of 0.5 is performed and monitoring, which includes pulse oximetry, noninvasive blood pressure measurement, ECG, and capnography, is established.

Consulting with the Technical Team on the Best Way To Extricate the Victim

Parallel to the medical supply, the paramedic and the technical leader should maintain close contact with the EMP and utilize their "time off" to develop a plan for extrication. Rescue personnel are well trained for standard situations, but emergency medicine gains its most fascinating impulses from the art of improvisation and adaptation to the individual circumstances, and there is always at least one good solution for each scenario. This optimal solution should be strived for and discussed briefly between the two partners of the rescue team at this stage of action [14].

Disentangling the Patient

The basic principle of disentanglement is to remove the vehicle from the patient, not the patient from the vehicle [5]. All parts of the wreckage that keep the victims entrapped are removed carefully, and special skills and constant training are necessary to find the best approach for the different components of the cars that are involved. While the EMP and paramedic continue medical treatment

(monitoring, infusion, ventilation, if necessary), one fireman should be responsible for protecting the patient and medical team against the effects of the technical rescue.

A blanket should be used to prevent dispersion of broken glass, and special care is necessary if open-ended metal parts are cut by hydraulic means. The latter can develop high velocity when torn off and may represent a serious danger for everyone nearby. Even when one tries to pull all the broken glass out of the window frame, there is usually enough left in it to cut hands and arms unless the frame is covered with a blanket or wide tape.

Safe disentanglement is time consuming, but for the well-being of the team and the prevention of further harm to the patient it makes little sense to work too quickly, even in view of the importance of the preclinical time factor.

Immobilization of the Patient

As soon as jamming vehicle parts are removed and free access to the entire patient is gained, you should try to accomplish medical supply to the parts of the body not previously accessible – primarily, hemorrhage control to the lower extremities.

In all cases of high-velocity injuries, and especially in unconscious patients, one must presume possible spinal injuries and attempt to immobilize the patient as soon as possible. Cervical spine control should have already been performed at an earlier stage of extrication while managing the airway (see "Providing Medical Care to the Patient"). Various effective cervical spine immobilization collars are available, and it is necessary to have different sizes in supply to guarantee an individual's safe fixation. The appropriate collar size can quickly be selected by measuring the distance between the chin and shoulder of the patient with one's hand and transferring this distance to the widths of the available collars.

Two persons are necessary to fix the collar: one immobilizes the head in a neutral position while the other adapts and affixes the different parts of the collar. If the trachea of the patient must be intubated for airway control, it must be appreciated that laryngoscopy is difficult with a collar in place. It is safer to perform endotracheal intubation with a second person stabilizing the cervical spine by means of manual on-line immobilization and to refix the collar when the procedure is finished.

Safe removal of a motorcycle helmet requires trained staff, and four hands. One person moves the helmet upwards with a slight axial traction while the assistant stabilizes the chin and neck of the patient with two hands and forearm, thus fixing the head and neck relative to the shoulder, and continues on-line immobilization until the first person replaces the helmet with a cervical collar [15].

For immobilization of the patient inside the wreckage we try to use the scoop stretcher (Fig. 3), which enables complete stabilization of the whole body and prevents axial or rotational deviation at this sensitive stage of rescue. The patient is manually fixed in position by the medical team while the back of the seat is removed by the technical partners. Subsequently, the two parts of the scoop

stretcher are installed, and the victim can then be carefully placed in the horizontal position.

For difficult and cramped situations where scoop stretcher or spine board cannot be used, short immobilization systems may be an advantage. The Kendrick Extrication Device (Mediked, El Cajon, CA, USA) is a flexible immobilization set for the upper part of the body with a stiff plastic portion for the back, which can be attached even to a patient in an exposed position [16].

Packaging the Patient and Removal from Entrapment

With the victim immobilized on a scoop stretcher, at least four persons are needed to lift the patient out of the wreckage (Fig. 3), an action that should be controlled by the EMP. It is the task of the EMP to make sure that the patient's clothes are free from entanglement, and that intravenous lines, infusions, monitoring devices, and tubes are not squeezed or displaced during this maneuver. It is a common experience that the line or tube inserted with the highest output of endogenous catecholamines (e.g., chest tube or cricoid puncture) is the first one lost when removing the patient. Therefore, an important aim is the best possible fixation of the lines.

Unfortunately, in severe trauma victims with cold, sweaty skin, who are covered with blood and tempered glass and are completely wet from pouring rain, no ideal fixation method exists. Under these conditions the best approach is to dry the part of body that the tape will be fixed to, apply the best sticking tape, and secure the fixation by means of a circular bandage. In the case of controlled ventilation it may be advantageous to disconnect the endotracheal tube from the bag or transport ventilator while the patient is being removed, provided that good oxygenation permits a short disruption of the oxygen supply. The exit route of the victim is usually in the direction toward the back of the car and then up and out of the cut off roof of the vehicle.

As standard equipment, our fire brigade carries a tent (Fig. 8) that can be quickly assembled, with or without its floor, for the main purpose of maintaining victims when transport facilities are insufficient. In the event of inclement weather conditions such as cold and pouring rain (unfortunately, more often the rule than the exception), it may be worthwhile to consider setting up such a tent, without its floor, over the wreckage to provide better working conditions for the rescue team and to protect the patient from additional hypothermia.

Once freed from the wreckage, the patient is placed on a vacuum mattress and prepared for transport to the hospital. Before covering the victim with warm blankets it is necessary to confirm that all the lines and monitoring devices are working and, most importantly, to ensure that the endotracheal tube is still correctly positioned.

At this stage of the rescue the medical team is again in a familiar treatment setting and can proceed with standard clinical procedures. A safe extrication has been accomplished, but the time factor must still be kept in mind, and there is little reason now not to "scoop and run."

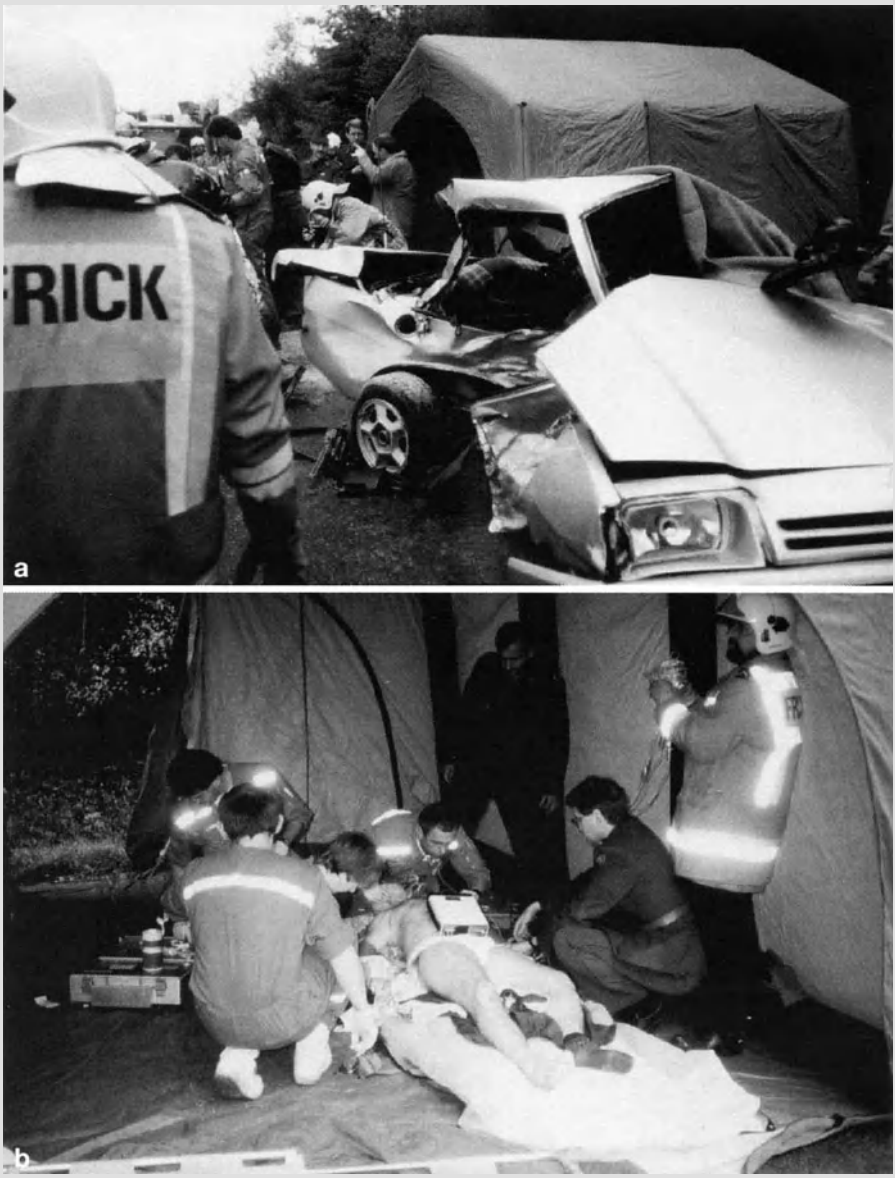

Fig. 8a, b. Inflatable tent

Conclusion

Two different teams with different skills, education, purposes, and resources meet one another in a stressful and dangerous situation. There is little time for "rescue team" formation, but the patient's outcome is highly dependent on the "new"

partnership's ability to communicate effectively, understand priorities, accept mutual leadership, utilize given resources, and perform a safe and standardized rescue procedure following an agreed upon schedule. Thus, the optimization of these factors helps the patient to receive the most appropriate treatment without being lost between the rescuers from the medical and fire brigade teams.

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Early Intubation in Trauma Patients

D. Nast-Kolb, A. Trupka, and C. Waydhas

Introduction

The ABCs of both initial assessment and resuscitation of a trauma victim start with "A" for "airway." Thus, control of the airway has the highest priority during the first minutes of trauma care. The management of "B," representing "breathing and respiratory function," is closely related to the initial steps of airway management. Reestablishing and maintaining adequate ventilation, oxygenation, and tissue perfusion therefore represent the main objectives in resuscitation of patients with multiple injuries. Achieving these goals in life-threatening situations often requires emergency interventions at the scene to reduce the incidence of acute posttraumatic complications. These early interventions are also crucial in preventing late complications such as (multiple) organ failure, the most frequent cause of late death in trauma patients. This is achieved by: (a) obtaining an adequate airway and by respiratory management and (b) immediate control of hemorrhage along with circulatory support by aggressive volume resuscitation.

Assessment of the Airway and Respiratory Function: Pathophysiological Principles

Assessment of the Airway

Due to the urgency of the situation initial assessment of the airway is entirely clinical. The absence of air movement through nose and mouth, chest wall excursions, and breath sounds should entail a high suspicion of apnea. Increased respiratory rate, inspiratory stridor, anxiety, the use of accessory respiratory muscles, sternal and supraclavicular retractions, and, as a very late sign of preterminal hypoxia, cyanosis may indicate a compromised airway. Note that "noisy beathing is obstructed breathing." Upper airway obstruction occurs most commonly in unconscious patients lying in the supine position. In this position the relaxed tongue occludes the posterior oropharynx. Other causes of upper airway obstruction include aspiration of foreign bodies (gastric contents, blood, dentures, tissue fragments in maxillofacial injuries), blunt or penetrating laryngeal or tracheal injury (with submucosal hemorrhage, edema, aspirated blood,

or tracheal collapse), expanding hematomas of the neck with laryngeal or tracheal compression, and the extremely rare "Andy Gump" mandible (bilateral fracture of the mandible with pharyngeal obstruction by the posteriorly collapsed tongue).

Assessment of Respiratory Function

Breathing and respiratory function are assessed by determining the respiratory rate and tidal volume, by examining the symmetry of chest wall excursions (to identify a flail chest), by auscultation and percussion of the lungs, and by pulse oximetry. Respiratory function in multiple trauma patients may be compromised by two major pathophysiological mechanisms: chest trauma and shock-related alterations.

Chest Trauma. Direct impact of mechanical forces on the chest induces chest wall injury, parenchymal lung lesions, intrapulmonary hemorrhage, and alveolar collapse, with a 50%–70% risk of very early respiratory failure [1–4]. Lacerations of the parietal pleura, costal or sternal periosteum, and intercostal nerves may cause severe pain with impairment of respiratory movements, tachypnea, and regional or general hypoventilation. Dead space increases, while alveolar ventilation is reduced.

Hemothorax and pneumothorax also contribute to reduced lung volumes and increased shunt perfusion in collapsed or compressed lung areas, with a further compromise of respiratory function. A life-threatening tension pneumothorax must be diagnosed clinically rather than radiologically, as immediate chest tube decompression is mandatory (see "Endotracheal Intubation: Technique and Pitfalls").

Serial rib fractures and flail chest may lead to respiratory distress by multiple factors. Inefficient ventilation and respiratory failure result from pain with limited chest wall movements, inability to cough effectively with accumulation of secretions, atelectasis leading to pneumonia, paradoxical movements of a flail segment in an unstable chest, and the nearly always associated lung contusion. The pathophysiology is therefore related not only to the chest wall instability but more importantly to the underlying pulmonary contusion and cardiovascular alterations that may develop in some patients [5].

The basic defect in patients with lung contusion is loss of microvascular integrity. Mild injuries result in focal edema, congestion of pulmonary capillaries, and limited interstitial and alveolar hemorrhage. In more severe cases increasing amounts of interstitial and intra-alveolar blood are seen, up to a large intrapulmonal hematoma dissecting along the blood vessels. The pulmonary contusion leads to interstitial edema in the surrounding lung tissue. Diffuse interstitial edema may also be observed progressively in the opposite lung. These changes are thought to result from mediator-related increases in capillary permeability and alterations in alveolar surfactant. This mediator release and activation is probably initiated in areas of injured pulmonary capillary endotheli-

um and is similar to the systemic inflammatory response to injury that leads to (multiple) organ failure [5, 6].

Additionally, debris, blood, edema fluid and secretions result in occluded bronchioles and unventilated alveoli and in atelectasis. These alterations induced by pulmonary contusion finally lead to ventilation-perfusion mismatches and increased shunting, with hypoxemia.

Shock-Related Alterations. The second pathophysiological mechanism of respiratory failure in trauma patients is shock-related alterations of the microcirculation and capillary homeostasis, coupled with an inflammatory mediator response. This causes very early morphological changes (e.g., leukostasis, endothelial swelling, interstitial edema) in lung parenchyma, as well as in all other shock organs, often without evident effects on blood gases or X-rays at this time [7, 8]. If adequate treatment is provided, these early pathomorphological changes are reversible without deterioration in organ functions [9, 10].

The risk of posttraumatic organ failure increases with higher Injury Severity Score (ISS) and in patients with multiple major fractures or extensive soft tissue injury due to overwhelming mediator release and cellular activation [6]. With these data in mind, early prophylactical intubation of severly injured patients seems reasonable, as early pulmonary dysregulation is not always clinically detectable. These microcirculatory lesions may be underestimated early in the course after trauma and with inappropriate treatment may lead to respiratory or other organ failure as they aggravate tissue hypoxia in hypovolemic-traumatic shock.

Indications for Endotracheal Intubation in Trauma Patients

Upper Airway Obstruction

Upper airway obstruction represents an emergency indication for endotracheal intubation (see above), especially in patients with obtunded consciousness and oropharyngeal obstruction by a posteriorly displaced tongue. Other indications for emergency intubation are injuries to the larynx and trachea with airway obstruction, expanding neck hematomas with tracheal compression, and maxillofacial injuries with enoral bleeding, loose tissue fragments, and a very high risk of aspiration. Severely injured patients, who already have aspirated gastric contents or blood, should be intubated to avoid further aspiration, to facilitate pulmonary toilet (bronchoscopy, lavage) and to start artificial ventilation if respiratory function is deteriorating [11].

Profound Circulatory Shock

Intubation is an accepted standard procedure in severely injured patients during cardiopulmonary resuscitation (CPR) or in profound circulatory shock [12, 13].

All of our patients intubated during CPR had sustained blunt injuries and succumbed during the first 24 h after injury. This reflects the extremely poor prognosis of patients with blunt injuries requiring CPR, in contrast to patients with penetrating injuries, who have a clearly better prognosis [12].

Chest Trauma

Multiple trauma patients suffering from major chest trauma seem to have an improved outcome following early intubation (EI) and ventilation, regardless of whether there is an initial respiratory distress or not. Only with intubation and artificial ventilation can adequate lung inflation and oxygenation be provided in noncompliant and partially atelectatic injured lungs. In multiply injured patients with chest trauma, late intubation – as compared to EI within 2 h after trauma – resulted in a higher incidence of respiratory failure (55% versus 48%), a similar rate of organ failure (55% versus 58%), and a higher death rate (22% versus 16%), despite a highly significant lower ISS (26 versus 36; $p \leq 0.001$) [14]. These findings stress the need for EI in multiply injured patients with severe thoracic trauma [1, 2, 14, 15]. A more conservative approach may be tried in patients with isolated severe thoracic injury, adequate ventilation, a patent airway, and no signs of apparent respiratory failure. These patients should be admitted to an intensive care unit (ICU), with close observation and monitoring of their hemodynamic status and respiratory function (frequent physical examination, ECG, oximetry, arterial blood gases, chest X-rays, serial spirometric testing).

Baseline therapeutic principles consist of supplemental oxygen, adequate analgesia (patient-controlled analgesia, epidural catheter analgesia, intercostal analgesia), chest tube drainage of hemo-pneumothorax, fluid restriction, and aggressive pulmonary physiotherapy (suctioning, incentive spirometry, early mobilization, and humidification of air) to clear bronchial secretions and avoid atelectasis and pneumonia. Intermittent positive pressure breathing, nebulized bronchodilator administration, postural drainage, cupping or clapping, and therapeutic fiberoptic bronchoscopy to suction secretions and reexpand atelectasis are often necessary. Intubation and mechanical ventilation are indicated if there are signs of respiratory decompensation and/or patient exhaustion. The indications for endotracheal intubation and mechanical ventilation in severly injured patients (modified from [16] are the following:

- Clinical signs of fatigue or exhaustion
- Respiratory rate < 10 or > 29/min
- PaO_2/FiO_2 ratio < 200
- $PaCO_2 > 55$ mmHg at $FiO_2 > 0.5$
- $PaO_2 < 60$ mmHg at $FiO_2 > 0.5$
- Arterial oxygen saturation $< 90\%$ ($FiO_2 = 21\%$)
- Preexisting chronic pulmonary disease

Respiratory Failure

Severely injured patients with evidence of respiratory distress require intubation, regardless of the presence of thoracic injuries, to avoid hypoxemia and tissue hypoxia (see the indications listed in the previous section).

Head Injury

A number of airway and respiratory disturbances may be observed following severe head injury. Hypoxia, hypercarbia, and aspiration may result from upper airway obstruction (relaxed tongue), depressed or absent protective airway reflexes, abnormal breathing patterns due to irritation of vegetative centers in the brain stem, and associated injuries (chest trauma, hypovolemic shock). There are thus three indications to intubate patients with severe head injury. The first indication is the need to secure the commonly obstructed airway in unconscious patients and to prevent aspiration. The second is to prevent hypoxia and hypercarbia, with their detrimental effects on cerebral oxygenation and intracranial pressure (ICP). Early hypoxia, hypercarbia, and hypotension have been demonstrated in up to 40% of patients with severe head injury, especially when multiply injured, and represent important "secondary insults" to the injured brain and are correlated with significantly poorer outcome from head injury (see Stocker et al., this volume).

Also, recent studies have demonstrated a significantly improved outcome in patients with severe head injury who were intubated and ventilated already in the prehospital phase [17–21]. The third indication is the possibility to avoid hypercarbia, which is associated with cerebral vasodilatation and increasing ICP, and to establish therapeutic hyperventilation to reduce ICP in cases of diffuse hyperemic cerebral swelling. Recent studies suggest that prophylactic hyperventilation in the prehospital phase may be harmful, leading to uncontrolled cerebral vasoconstriction, with the risk of cerebral ischemia. Controlled hyperventilation may be effective only in the presence of diffuse hyperemic cerebral swelling, as diagnosed by transcranial Doppler monitoring, jugular venous oxygen saturation measurement, and continuous ICP monitoring, [22]. According to these indications, every head-injured patient with a Glasgow Coma Score (GCS) of 8 or below requires immediately intubation at the scene [21]. Also intubation is necessary in patients with a GCS of 9 or more in the case of agitation or inadequate ventilation.

Prophylactic Early Intubation Due to the Severity of Injury

Multiple organ dysfunction syndrome (MODS), with or without infection, is the leading cause of late death in severely injured patients [23–25]. Soft tissue injury with extensive mediator release, circulatory shock, hypoxia, and bacterial toxins (translocated from the gut or from septic foci) are able following an injury to induce a generalized humoral and cell-mediated inflammatory response, with

endothelial and cellular damage in all organ systems, which may ultimately culminate in MODS [6, 10, 26–28]. Despite the extensive experimental and clinical work on this topic over the past two decades the pathophysiological alterations following traumatic shock are not fully understood. Also, our ability to salvage patients with MODS has not appreciably improved over the past 20 years [25–28].

Among these posttraumatic organ failures, respiratory failure and liver failure have the highest incidence (30%–50%). Respiratory failure is present in nearly all cases of MODS, occurs very early, and often leads to intractable progressive adult respiratory distress syndrome as the major problem in lethal MODS. Posttraumatic respiratory failure has two major causes: direct parenchymal lung damage in chest trauma and indirect lung damage mediated by a systemic inflammatory process, initiated by traumatic-hypovolemic shock [4, 8,24]. Experimental studies and human lung biopsies have shown early morphological changes in the lung within 1 h after multiple trauma, prior to any deterioration of arterial blood gases. If the underlying shock state is adequately treated, these early changes are fully reversible [7, 9,10].

The prevention of early respiratory and other organ failures plays a predominant prognostic role, as the final outcome is closely related to the number of failing organ systems [3, 23, 27]. Because of varying definitions in the literature the reported mortality rates of respiratory failure, adult respiratory distress syndrome, and MODS range from 20% to 75% [2, 24, 27, 28]. No causal treatment is available at present, and one must therefore consider all possible symptomatic and prophylactic therapeutic interventions for counteracting the uncontrolled activation and release of inflammatory mediators.

With this in mind, we carried out a prospective study to evaluate the prognostic value of EI in multiply injured patients (Table 1). In addition to the classical indications, prophylactic EI was administered to all severely injured patients with at least two injured body regions with Abbreviated Injury Scale (AIS) score greater than 3 or three major fractures with a known or suspected ISS above 24 [6, 29–31]. Since ethical considerations preclude conducting such a study in a randomized way, we were able to compare patients only in terms of intubation during the first 2 h after trauma versus later intubation. The cutoff point of 2 h was chosen as this seemed to be a reasonable time for transport to the emergency room (85% of patients directly admitted to our hospital arrival within 75 min after trauma) and for performing the diagnostic procedures necessary to decide about EI. Due to our trauma management strategy EI was performed in 81% of the patients ($n = 106$), and in 15% intubation was performed later in the course (delayed intubation, DI); six patients (4%) were not intubated.

The main indications for EI were: (a) unconsciousness following severe head injury in 45 patients (45 EI, 0 DI), (b) major chest trauma in 40 cases (31 EI, 9 DI), and (c) injury severity in the absence of the two above indications in 40 patients (30 EI, 10 DI). The difference in mean ISS between EI patients (ISS 39) and DI patients (ISS 29; $p < 0.005$) raises some difficulty in directly comparing organ failure and survival rates between groups. However, it is wellknown that increasing ISS values are correlated with higher organ failure and death rates [30, 31]. Thus a lower incidence of organ failure, MODS, and death should be

Table 1. Posttraumatic organ failure and mortality in patients with multiple injuries and early or delayed intubation ($n = 125$)

	Main indication		
	Chest trauma (AIS ≥ 3)	Severity of injury	All patients
Early intubation (< 2 h)			
n	31	30	106[a]
ISS	36*	33	39**
Organ failure	58%	60%	67%
Respiratory failure	48%	47%	45%
Deaths	16%	14%	15%
Delayed intubation (> 2 h)			
n	9	10	19
ISS	26	32	29
Organ failure	55%	80%	63%
Respiratory failure	55%	40%	42%
Deaths	22%	30%	26%

* $p < 0.01$; ** $p < 0.001$.
[a] All patients with severe head injury ($n = 45$) were intubated early.

expected among the less severely injured DI patients. However, in the DI group we observed similar incidences of respiratory failure (42% versus 45%) and organ failure (63% versus 67%) and even higher MODS (37% versus 28%) and death rates (26% versus 15%). This underscores the value of EI for multiple trauma patients, leading to less posttraumatic organ failure and improved outcome. This conclusion is reinforced by the fact that the hemodynamic condition of the EI patients (higher infusion and transfusion requirements throughout the resuscitation period) were liable to increase the risk of posttraumatic organ failure in the EI group. Despite a significantly worse hemodynamic condition on admission and a significantly higher ISS, no differences in respiratory parameters (PaO_2/FiO_2 ratio, arterial pCO_2) were observed between groups. This indicates that at this early point (within 2 h after trauma) – excluding evident respiratory failure – respiratory parameters may not be helpful in deciding whether to intubate or not. We conclude that EI of severely injured patients may be indicated even in the absence of early apparent respiratory distress (Table 1).

Injury severity was similar in the small but interesting subgroup of patients who were intubated as indicated by the severity of their injuries and in the absence of head or chest trauma (EI, ISS = 33; DI, ISS = 32). The small size of these subgroups may be responsible for the absence of a statistically significant differences in the rate of organ failure (DI 80% versus EI 60%) and death (DI 30% versus EI 14%) while, again, smaller rates of organ failure and death were seen in the EI group. As the risk of developing posttraumatic organ failure seems to increase sharply in severely injured patients with an ISS higher than 24, we recommend EI of these patients [14].

Transport of Severely Injured Patients

The decision to intubate should be made more liberally in patients being prepared for land or air transport. If there is any doubt about the condition of the patient's airway or any sign of progressive deterioration of the respiratory or circulatory functions, the patient should be intubated regardless of the indications discussed above.

Endotracheal Intubation: Technique and Pitfalls

In general, every trauma patient should initially have supplemental oxygen at a high flow rate (8–10 l/min) via nasal prongs or face mask. If simple maneuvers of airway management (chin lift, jaw thrust, removal of foreign material from the oropharynx, suction, oropharyngeal/nasopharyngeal airway) are not sufficient to secure a patent airway, or if any of the outlined indications above is present, intubation should be performed. However every airway maneuver, especially endotracheal intubation, may be problematic in severely injured patients. Every trauma patient with a reduced level of consciousness, alcohol or drug ingestion, and inappropriate or impossible clinical assessment of the cervical spine and peripheral neurological status must be assumed to have significant cervical spine injury with the risk of neurological deterioration during neck manipulation. There may be an occult dislocation or unstable fracture of the cervical spine or an incomplete spinal cord lesion that can be aggravated by uncontrolled manipulation of the neck [32]. Therefore it is standard practice to control the airway of a every trauma patient with a possible cervical spine injury without neck manipulation (in-line manual cervical immobilization), followed by securing the cervical spine with rigid cervical collars (e.g., Stiffneck) until injury has been excluded by an appropriate series of X-rays, including all seven cervical vertebrae. Soft and semirigid collars (e.g., Philadelphia collar) have no place in prehospital trauma care as they do not stabilize the spine effectively.

Prior to intubation great attention must be paid to pain relief and sedation, even in comatose patients. Patients with brain injury may show an increase in ICP during intubation due to inadequate sedation and analgesia. Additionally, it should be assumed that every trauma patient has a full stomach, which requires special precautions. Although the use of neuromuscular blocking agents has been shown to be safe and effective in the hands of experienced physicians, their use cannot be widely recommended because failure to adequately ventilate a paralyzed patient results in anoxic brain injury or death. The initial attempt at intubation should be performed without the use of muscle relaxants. If at all, only skilled personnel familiar with neuromuscular blocking agents should use these drugs, ascertaining that the airway can be secured with bag-mask ventilation, endotracheal intubation or by surgical means (translaryngeal jet ventilation, cricothyroidotomy). Either succinylcholine or vecuronium bromide are recommended for paralysis with respect to the contraindications and side effects [33, 34].

We suggest the following protocol for drug-assisted intubation. For different indications, side effects and (dis)advantages of each drug the reader is referred to Kerz and Dick (this volume). Latex gloves should be worn, and eye protection is recommended. The points in the protocol are:

1. Preoxygenation and ventilation with 100% oxygen by face mask and assisted ventilation
2. Local (e.g., lidocaine) or systemic anesthetics to blunt cardiovascular reflexes, especially in nasotracheal intubation
3. Adequate sedation and analgesia (recommended drugs: midazolam, fentanyl, ketamine, etomidate, thiopental)
4. Cricoid pressure (Sellick's maneuver) during bag-mask ventilation, and intubation to reduce the risk of regurgitation and aspiration
5. Endotracheal intubation

Several techniques of endotracheal intubation are available. We recommend laryngoscopic orotracheal intubation with in-line manual cervical spine immobilization as the standard procedure. Nasotracheal (blind or light guided) or fiberoptic intubation is usually considered safer in patients with a known or highly suspected cervical spine fracture as it requires fewer neck movements. Nasotracheal intubation is contraindicated in patients with facial or basilar skull fractures due to the risk of perforation of the nasopharynx, intracranial tube placement, and infection. Alternative intubation procedures using light-guided intubation or digital techniques are presumed to lessen the risk of neck movements, but no studies have been performed to confirm this.

After intubation and before mechanical ventilation is started, proper placement of the tube must be confirmed to rule out esophageal or right main stem bronchus intubation. The only indication for creating a surgical airway is inability to intubate the trachea (edema of the larynx, laryngeal or tracheal trauma with hemorrhage or anatomical alterations). This may be achieved by translaryngeal jet ventilation or cricothyroidotomy [11].

Mechanical ventilation is the most common cause of tension pneumothorax, with an increased risk in patients with chest trauma when positive end-expiratory pressure ventilation is used. Other causes are open chest wounds, central venous catheterization and spontaneous pneumothorax (ruptured bullae). Tension pneumothorax develops when a "one-way valve" air leak occurs, either from the lung or through the chest wall. Clinical signs are a rapidly deteriorating hemodynamic condition (hypotension, tachycardia) along with respiratory distress in a spontaneously breathing patient, or increasing inspiratory pressure in ventilated patients, along with decreasing arterial oxygen saturation. Cyanosis is a late manifestation. Unilateral absence of breath sounds, distended neck veins, hypertympanitic percussion over the affected side are additional signs, but these signs are sometimes difficult to identify at the scene with a patient in the supine position and a very noisy environment. Thus a high index of clinical suspicion is necessary to diagnose and treat tension pneumothorax promptly. In our own trauma service a tension pneumothorax has been observed three times more frequently in patients with thoracic injury arriving intubated and ventilated in

Table 2. Incidence of pneumothorax and tension pneumothorax in patients with chest trauma upon hospital arrival ($n = 51$)

	Pneumothorax		Tension pneumothorax	
	n	%	*n*	%
Intubated and ventilated patients ($n = 29$)	14	48	4	14
Nonintubated patients ($n = 22$)	7	32	1	5

Differences nonsignificant

hospital than in spontaneously breathing patients (Table 2). We therefore recommend prophylactic chest tube drainage on the affected side in every trauma patient with significant chest injury (subcutaneous emphysema, serial rib fractures, lung contusion, flail chest, open chest wounds) who has been intubated for any reason.

Conclusion

The potential presence of a concomitant cervical spine injury must be a major concern in every trauma patient, requiring any airway procedure, especially endotracheal intubation. In-line manual cervical immobilization during airway maneuvers followed by the use of rigid cervical collars (e.g., Stiffneck) reduces the risk of spinal cord damage. Indications for emergency prehospital endotracheal intubation in trauma patients are:

- Upper airway obstruction that cannot be cleared with simple maneuvers
- CPR or profound circulatory shock states
- Acute head injury with GCS < 8
- Respiratory failure

Additionally, there is strong evidence that prophylactic EI in polytraumatized patients without signs of circulatory or respiratory dysfunction may reduce the incidence of posttraumatic (multiple) organ failure and hereby improve outcome in:

- Multiply injured patients with major chest trauma (AIS > 3)
- High injury severity with ISS > 24
- A combination of two or more major fractures with extensive soft tissue injury (crush injury) and a high risk of posttraumatic organ failure, due to overwhelming mediator release and cellular activation (e.g., bilateral femoral fracture or pelvic plus femoral fracture)

In patients being prepared for air or ground transport, intubation should be performed even more liberally, especially if there is any possibility that respiratory or cardiocirculatory functions may deteriorate.

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Early Ventilation in Trauma Patients

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Introduction

Acute respiratory failure is frequently encountered in multiple trauma patients. Maintenance of airway patency and adequacy of ventilation are the top priorities of the resuscitative protocol. Severe clinical situations (cardiorespiratory arrest, airway obstruction, deep coma) represent a clear indication for emergency tracheal intubation and mechanical ventilation. However, more subtle decision making is necessary in a conscious or obtunded, spontaneously breathing patient with moderate shock due to multiple traumatic lesions. In such a situation early respiratory support should be based on an understanding of the mechanism of trauma. While unnecessary intubation should be avoided in patients who most likely will remain stable, early mechanical ventilation may significantly contribute to the decrease in morbidity of those who subsequently deteriorate. Adequate and timely respiratory support may diminish the incidence of aspiration, atelectasis, acute respiratory distress syndrome (ARDS), pulmonary infection, and intracranial hypertension.

Choice of the early mode of ventilation should be adapted to the particular features of respiratory pathology in different categories of trauma. Causes of postraumatic respiratory failure are listed below (the management of upper airway patency – problems of full stomach and difficult airway, techniques of tracheal intubation – are presented elsewhere in this volume):

- *Upper airway injury or dysfunction*
  - Foreign body obstruction
  - Soft tissue obstruction (tongue)
  - Oropharyngeal edema/hematoma
  - Maxillofacial injury
  - Laryngeal and tracheal laceration, edema, hematoma
  - Compressive cervical hematoma
- *Thoracic trauma*
  - Thoracic wall injury, flail chest
  - Pneumothorax, hemothorax
  - Pulmonary injury (contusion, laceration, hematoma, pneumatocele)
  - Tracheobronchial lacerations
  - Diaphragmatic hernia

- *Other pulmonary lesions*
  - Aspiration
  - Smoke inhalation
  - Near drowning
  - ARDS (postraumatic, neurogenic, toxic)
  - Fat embolism
  - Pulmonary edema of cardiac origin
- *Central nervous system disorders*
  - Severe head injury
- *Others*
  - Drug overdose, alcohol, poisoning, hypoxic encephalopathy
  - Cervical and upper dorsal spine injuries
  - Multiple severe traumatic injuries
  - Shock
  - Electrocution

Indications for Early Respiratory Support

Multiple Trauma Patients

There are three essential indications for early respiratory support in multiple trauma patients: impairment of pulmonary gas exchange and/or respiratory mechanics, presence of hypovolemic shock, and presence of central nervous system disorders associated with severely altered airway reflexes. Impaired pulmonary gas exchange and circulatory shock result in decreased oxygen delivery to injured organs, aggravate the hypoxic tissular damage, induce the release of cytokines with a subsequent activation of macrophages and neutrophils, precipitate pulmonary and systemic microvascular alterations and lead to the development of multiple organ dysfunction syndrome (MODS). ARDS is frequently the precursor of MODS, suggesting that altered pulmonary function has a key role in subsequent organ failure [1, 2]. Traumatic alteration of airway reflexes may lead to aspiration, atelectasis, and early pneumonia, and these complications may result in sepsis associated with ARDS or MODS [3].

The incidence of posttraumatic pulmonary complications varies in different categories of trauma patients. It is therefore important to define the group at risk in whom the early and adequate respiratory support should be particularly beneficial. A recent study analyzed the incidence of pulmonary complications and the risk factors present at the scene of the accident or at admission [4]. Pulmonary complications occurred in 11.2% of the patients and represented one-third of all disease complications. Severe head and chest trauma [Abbreviated Injury Scale (AIS) > 3) were the most common traumatic lesions and accounted for 75% of pulmonary complications. The contribution of severe trauma to the extremities, pelvis, and abdomen to the development of pulmonary complications was much less important (16%). The common risk factors for atelectasis, ARDS and

pneumonia were: (a) blunt mechanism of injury, (b) Injury Severity Score (ISS) higher than 16, (c) trauma score less than 13 at admission, (d) coma and severe head injury (AIS > 3; Glasgow Coma Scale < 8), and (e) surgery to the head. The risk factors for the association of pneumonia, ARDS, and respiratory failure (defined as the need for prolonged ventilation and as difficulties in weaning) were: (a) age > 55 years, (b) ISS > 16, and (c) chest injury (AIS > 3). The authors concluded that patients at high risk of pulmonary complications are those with blunt mechanism of injury and severe trauma who are admitted in shock and coma, have significant injuries to the head and chest, and undergo surgery. Prospective research on the pathogenesis of these complications and on the development of more efficient therapeutic modalities should be focused on this category of trauma patients [4].

Patients with Central Nervous System Injury

Patients with mental status altered by head injury, hypoxia, alcohol or drugs, in combination or alone, form a population with a particular need for early airway protection and respiratory support. Those who are severely obtunded or comatose present with diminished or absent airway reflexes and a distended stomach. The incidence of preadmission respiratory failure caused by regurgitation, vomiting, and aspiration of orotracheal and gastric contents or blood is very high in these patients, who should be considered as having an acute respiratory emergency [3, 5]. In some patients the acute alteration of mental status manifests itself by disorientation, agitation, and combativeness, alternating with occasional spells of somnolence. These symptoms may be due to the effects of drugs or alcohol but may also result from an associated cerebral injury. These patients must be sedated to make possible the recuscitative maneuvers, transport, and diagnostic procedures. However, even a gradual and incremental administration of benzodiazepines and/or narcotics may potentiate the effect of drugs and alcohol and dangerously increase the risk of aspiration and respiratory depression in unintubated individuals. A safer alternative is a management protocol which includes carefully planned intubation with the use of local anesthetics, intravenous medication, and if necessary curarization, followed by respiratory support, facilitated by a continuous sedation.

Severe head injury is the most frequently encountered form of CNS disorders in trauma patients and is also the leading cause of respiratory failure. The respiratory alterations following craniocerebral trauma are due not only to the inhibition of airway reflexes but also to important disturbances of the regulatory mechanisms of pulmonary circulation and ventilation. Respiratory arrest of a variable duration may immediately follow the traumatic impact. Massive release of catecholamines and inflammatory mediators of cerebral origin into the systemic and pulmonary circulation is the main cause of pulmonary dysfunction [6].

Neurogenic pulmonary edema is a fulminant, albeit rare, form of respiratory failure that results from a sudden increase in hydrostatic systemic and pulmonary

pressures causing a permeability defect in pulmonary capillaries [7]. Other, less dramatic, forms of pulmonary dysfunction are characterized by ventilation/perfusion disturbances due to the alteration in hypoxic vasoconstriction, increase in extravascular lung water due to altered pulmonary capillary permeability, and elevated airway resistance due to neurogenic bronchoconstriction [8]. Respiratory function may also be impaired by the presence of abnormal respiratory patterns (Cheyne-Stokes respiration, central neurogenic hyperventilation) and by associated extracranial disorders: thoracic injury, spinal injury, multiple traumatic lesions, fat embolism, and shock. Hypoxia, which is observed at the early stage in most severe head trauma patients, alters cerebral hemodynamics and increases intracranial pressure (ICP). Equally deleterious is hypercapnia, due to airway obstruction, which frequently occurs at the site of the accident [9]. Both hypoxia and hypercapnia, especially if associated with systemic hypotension, may unfavorably influence the neurological prognosis [10]. Therefore the institution of early adequate respiratory support in head-injured patients is important.

The immediate on-site maneuvers should assure airway patency and administration of oxygen via a face mask if spontaneous respiration is sufficient, or by manual bag and mask ventilation with cricoid pressure in patients with severe respiratory deterioration. At the same time a rapid neurological examination (state of consciousness, pupillary response, presence or absence of spontaneous movements, and signs of peripheral neurological deficit) should be performed. The initial assessment should also include the observation of respiratory patterns (diaphragmatic respiration may indicate spinal injury) and a rapid evaluation of other traumatic lesions. In particular, a close search for the presence of maxillofacial injuries and signs of cervical lesions (hematoma, displacement of trachea, subcutaneous emphysema, status of carotid pulses) is of utmost importance.

The feasibility of emergency tracheal intubation must be rapidly decided upon. If the situation permits, the intubation should be carefully planned and performed only after neurological examination. The oral route of intubation is preferred because of frequent lesions of the cribriform plate.

The usual precautions concerning the full stomach and cervical spine protection are mandatory. Sedation and/or curarization is recommended in order to shorten the duration of intubation and avoid increases in ICP during airway manipulation. The choice of the pharmacologic agent should be based on the hemodynamic status of the patient, severity of coma, time allowed for intubation, and experience of the care provider.

Midazolam, which is frequently used because it assures hemodynamic stability, may not provide for sufficient sedation in agitated and combative patients. Propofol and thiopental may cause hypotension in hypovolemic patients, and etomidate carries the risk of inhibition of adrenal steroidogenesis [11]. Muscle relaxants can be used to facilitate intubation in patients in whom no particular technical difficulties are foreseen. Succinylcholine, a depolarizing muscle relaxant, has a rapid onset and short duration, but presents several undesirable effects (increase in intragastric and intraoccular pressures). Vecuronium, a long-lasting nondepolarizing agent, assures hemodynamic stability and

does not promote histamine release. A recent study reported that high-dose vecuronium may offer intubating conditions that are as good as those of succinylcholine in head injured patients [12]. Newer nondepolarizing agents, with a shorter time of onset, are being evaluated. If tracheal intubation is not feasible, and an emergency airway is necessary, cricothyroidotomy is the procedure of choice. A recent study reported 97% of successful intubations in a large group of head trauma patients [12]. All unsuccessful intubations (3%) occurred in patients with complex maxillofacial fractures and had to be followed by cricothyroidotomy [12]. However, if endotracheal intubation seems technically difficult, but the patient's respiratory status is stable, and the time of transportation will not be too long, transfer to a hospital facility for fiberoptic intubation or tracheostomy under controlled conditions may be preferable.

The aim of early respiratory support in severe head injury is to prevent cerebral hypoxic damage and to contribute to the control of intracranial pressure. Because of the frequent impairment of gas exchange, controlled mechanical ventilation (CMV) with positive end expiratory pressure (PEEP) must frequently be employed to achieve an acceptable level of arterial oxygenation [13]. However the safety of PEEP in patients with head injury was largely debated in the last years [13, 14]. Theoretically, elevated intrathoracic pressure, which is essentially transmitted to the intracranial contents via the venous system may cause a decrease in cerebral venous outflow with a subsequent increase in ICP. At the same time, PEEP-related changes in the systemic circulation (decreased venous return, decreased systolic blood pressure) may further deteriorate the Cerebral Perfusion Pressure (CPP). However, a compensatory waterfall mechanism in cerebral venous circulation prevents the transmission of intrathoracic pressure changes to the cortical veins. This mechanism is particularly effective when intracranial hypertension exists, and the pressure in cortical veins is elevated [14].

The overall effects of elevated intrathoracic pressures on cerebral hemodynamics depend on the interactions of the thoracic wall, pulmonary, and cerebral compliance (the "stiffer" the lung, the less pressure in transmitted to the brain), the status of cerebral vascular autoregulation, and the systemic circulation [15]. In patients with impaired autoregulation and reduced CPP the lowest possible end-inspiratory pressure should be employed. High-frequency ventilation, which permits a decrease in the peak airway pressure during a respiratory cycle, was proposed for brain-injured patients with elevated ICP. However, its superiority over CMV has not been demonstrated, and its use for early ventilatory support is technically difficult [16]. A careful control of CO_2 levels and avoidance of hypercarbia are very important. Moderate hyperventilation (pCO_2 28–30 mmHg) is advocated during the early respiratory support to lower ICP levels in patients at risk for intracranial hypertension. However, as soon as ICP monitoring is available, normocapnic ventilation should be reinstalled in patients with normal ICP. Indiscriminate prophylactic hyperventilation, which may aggravate cerebral ischemia, has been reported to be deleterious [17]. Early and long-lasting use of neuromuscular blocking agents in patients with head injury is an essential part of the treatment of intracranial hypertension in many institutions. The aim of this

"early paralysis," adjunctive to sedative therapy, is to prevent increased ICP during posturing, tracheal aspiration, and patient manipulation. A recent multicenter study reported that the routine use of neuromuscular blocking agents contributes to morbity by prolonging hospital stay and increasing the frequency of septic complication, in particular pneumonia [18]. The authors concluded that "pharmacological paralysis" should be reserved for initial intubation, hospital transport, and diagnostic procedures. The patient should be weaned from neuromuscular blocking agents as soon as possible unless continuation of these agents is specifically indicated for ICP control [18].

Patients with Chest Trauma

Patients with major chest injuries due to blunt or penetrating trauma frequently require early respiratory support. Blunt thoracic injuries are more common and more frequently associated with injury to other systems. The morbidity rates related to pulmonary traumatic lesions are high, but the early mortality is usually due to nonpulmonary injuries such as associated cerebral trauma or uncontrollable hemorrhage from great vessel laceration [19]. However, some early manifestations of trauma to the chest (open or tension pneumothorax, rapidly increasing hemothorax, airway obstruction, cardiac tamponnade, air embolism) are life threatening and require rapid therapeutic intervention.

The main causes of respiratory failure in major chest trauma are:
- Impairment of respiratory mechanics due to flail chest
- Hypoventilation resulting from rapid and shallow breathing due to acute pain from broken ribs
- Obstruction of airways by blood, edema, and secretions
- Lung compression or collapse due to tension pneumothorax, massive hemothorax, or diaphragmatic hernia
- Presence of pulmonary contusion resulting from direct transmission of kinetic energy to pulmonary parenchyma
- Pulmonary edema due to cardiac failure from traumatic myocardiac injury or preexisting disease
- Indirect pulmonary damage associated with shock and release of acute phase mediators into the pulmonary circulation

Increased work of breathing, decreased alveolar ventilation, and decreased arterial oxygenation are the common features of these different forms of pulmonary disability [20]. The decision to intubate and ventilate in blunt thoracic trauma should be based on clinical examination, continuous observation, and sequential evaluation of gas exchange. Imaging techniques, in particular thoracic computed tomography (CT), reveals the extent of thoracic wall, pleural, mediastinal, and parenchymal lesions [21]. Thoracic CT is particularly useful in visualizing minimal extra-alveolar air collections, which may lead to a tension pneumothorax during positive pressure ventilation. Such collections must be drained in advance [22].

The presence of tachypnea, tachycardia, low systemic blood pressure, and poor arterial blood gasses, together with the presence of other injuries have been indentified as risk factors for early respiratory insufficiency [23]. On-site emergency intubation should be reserved for patients with overt severe respiratory distress or hypoxia, for those with less severe thoracic injuries combined with hemodynamic or cerebral failure, and for patients transported by air. Here again, oral intubation facilitated by sedation and local anesthesia is the method of choice.

Clinical signs of pneumothorax require the placement of chest drains before the initiation of positive pressure ventilation. A polyethylene 16-F catheter connected to a one-way Heimlich valve may be inserted on the site of the accident as a temporary measure for treatment of pneumothorax.

The presence of a flail chest and pulmonary contusion are the main indications for the early respiratory support in chest trauma patients [24]. In the past few years the importance of pulmonary contusion in determining the need for ventilator assistance has been emphasized [25]. The frequent combination of flail chest and pulmonary contusion implies that the patient has sustained a high-energy injury to the intrathoracic organs. However, the extent of the flail injury depends on thoracic compliance. In young patients with very compliant chests severe bilateral pulmonary contusion with extensive intraalveolar hemorrage, pulmonary hematomas, and pneumatoceles may occur with very little injury to the chest wall itself. In contrast, elderly patients may develop a serious flail chest injury after a minor trauma with very little underlying parenchymal damage. Full respiratory support for flail chest to assure "internal stabilization of chest wall" is no longer a general rule [26]. A more selective management protocol combining epidural analgesia with partial respiratory support via an endotracheal tube or face or nose mask (CPAP, pressure support, BiPAP, ARPV) and intensive chest therapy is frequently employed today in patients with unstable chest wall fractures associated with minor to moderate parenchymal lesions. However, if it appears that full ventilatory assistance for flail chest is required because of the importance of respiratory or hemodynamic failure, early implementation of mechanical ventilation is very important. Delayed ventilatory assistance, preceded by clinical evidence of hypoxia and inadequate ventilation lasting for several hours, increases the mortality rate significantly [25, 26].

The objective of respiratory support in chest trauma patients is to reduce the work of breathing, prevent or reverse atelectasis, recruit collapsed alveoli, and equalize ventilation and perfusion in the lung units. This objective may be achieved by adaptation of the ventilatory strategy to the patient's respiratory requirements. Hypoxic patients with extensive pulmonary contusion must be deeply sedated in the early postraumatic period.

Controlled ventilation with PEEP or inversed ratio ventilation with pressure control frequently represent the initial mode of ventilation. In patients with an unilateral lesion the effect of positioning with the least involved lung in the dependent position should be tried [27]. In the rare case of a massive air leak from a bronchopleural fistula, no single ventilatory mode has been shown to be more effective than the other, although in some cases a succesful use of independent

lung ventilation with high frequency ventilation has been reported [28]. However, the majority of patients with thoracic trauma can be managed with various types of partial respiratory support with or without intubation, using systemic and regional anesthesia in the place of sedation. Early use of fiberoptic bronchial toilet and mucolytics are a routine part of this management protocol.

Other Indications

Other indications for early full or partial respiratory support include patients with cervical spinal cord injury and tetraplegia, those with upper dorsal spinal cord injury and respiratory insufficiency, those involved in an explosion or fire in a closed space with the risk of inhalation injury, and those who are the victims of near drowning. In the past few years small and compact portable respirators have been developed that are able to provide sophisticated modes of respiratory support and are equipped with a well-functioning alarm system [29].

The noninvasive techniques for evaluating oxygenation and alveolar ventilation – pulse oxymetry and capnography – are incorporated in miniaturized and portable monitors. Because of these technological advances the same well-adapted respiratory strategies may be employed on the scene, during transportation, during diagnostic procedures, and in the ICU.

Pitfalls and Complications of Early Respiratory Support

Complications related to early respiratory support in trauma patients include provider-related iatrogenic injuries during airway instrumentation, adverse systemic and cerebral hemodynamic effects of positive pressure ventilation with PEEP, and development of new barotrauma in patients with thoracic injury [13, 22, 30]. Pulmonary injury related to ventilation with large lung volumes and high peak airway pressures and the development of nosocomial pneumonia are related to the prolonged use of ventilatory support [3, 30, 32]. Early pulmonary infections, frequently observed in multiple trauma patients, are probably related to preadmission aspiration [3]. Sedation and paralysis, which are frequently used to assure the patient's comfort and eliminate patient-ventilator asynchrony, have several deleterious effects. Sedation produces vasodilation and hypotension in hypovolemic patients and reduces the possibility of neurological surveillance. Neuromuscular paralysis immobilizes the patient, favors retention of secretions, atelectasis, and septic complications, and may lead to long-lasting neuromuscular disorders [32].

For these reasons a patient-controlled analgesia regimen by intravenous or epidural route together with a partial respiratory support with or without intubation should be used whenever possible.

Conclusion

Early emergency ventilation must be employed in several categories of trauma patients, in particular in the presence of major cerebral or thoracic injuries and in multiple injuries. Ventilatory strategies should be adapted to the type of injury. Recent technical advances allow for using sophisticated modes of ventilation early, and for evaluating the impairment in pulmonary gas exchange and the effects of therapy. Excessive sedation and longlasting neuromucular blockade associated with full respiratory support may be deleterious.

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Analgesia and Sedatives in Emergencies

T. Kerz and W. F. Dick

Introduction

In addition to securing vital functions according to the ABCs, analgesia and sedation in trauma patients are of crucial importance. Acute pain is considered transient in nature. However, as trauma harms the patient not only by direct effects on body tissues but also psychologically and by centrally mediated effects, there is always a chance of conversion into chronic pain. The experience of pain is modulated by fear, anxiety, and previous experience of pain. Pain and anxiety contribute to morbidity and mortality. Thus, analgesia and sedation should help to attenuate the patient's reaction to trauma as far as it is influenced by pain and anxiety, to support emergency care procedures, and hopefully to prevent acute pain from becoming chronic.

This chapter addresses the body's reaction to pain and trauma in general, and reviews different methods and a variety of drugs used for analgesia and sedation in acute trauma patients.

The Trauma/Stress Response

Pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" [1]. Others define pain as "whatever the patient says it is" [2], to underline individually differing grades of perceptions. From a pathophysiological viewpoint, acute pain originates from activation of different nociceptors whereas the sensation of pain is produced in the cortex.

Nociceptors are of two major types: high-threshold mechanoreceptors (Aδ fibers) and polymodal nociceptors (C-fibers). Aδ fibers are myelinated and conduct at 5–25 m/s. They respond to strong pressure and severe thermal injury. Activation of these fibers results in sharp and localized pain ("first" pain), rapid in onset. C-fibers are unmyelinated and have a conducting speed of 1–2 m/s. They respond not only to pressure but also to heat and algesic substances and are therefore referred to as polymodal. C-fiber activation leads to dull, poorly localized, and prolonged pain. All types of nociceptors are subject to plasticity when activated; that is, their sensitivity increases after injury.

From peripheral nociceptors, somatic afferent inputs enter into their cell bodies in the dorsal horn ganglia in the spinal cord, whereas visceral afferent inputs are mediated by the same type of fibers but enter the spinal cord by sympathetic, parasympathetic, and splanchnic nerves or via ventral roots. The dorsal root is also the origination of ascending pain pathways such as the spinothalamic or spinoreticular tract after segmental crossing of dorsal horn cell axons has occurred. Impulses travel through the spinothalamic tract to the ventroposterior and medial thalamic nuclei and are then projected to associative and somatosensory areas of the cerebral cortex. Spinoreticular neurons are held responsible for autonomic responses and for arousal and affective sensations.

In response to stress ACTH-releasing hormone is secreted mainly from the paraventricular thalamic nucleus which activates the hypothalamo-pituitary-adrenal axis (HPA) and the sympathetic system. Central administration of ACTH-releasing hormone in large doses produces anxiety [3]. Several cytokines can also activate the HPA [4] and stimulate ACTH and cortisol secretion. Arousal, anxiety, and vigilance are stimulated by norepinephrine release throughout the brain, originating from the locus ceruleus [3].

Many substances which are liberated by trauma can change the threshold and activity of peripheral nociceptors. Pain receptors may be activated by cyclooxygenase metabolites (the "prostanoids") and sympathomimetic amines. Histamine, serotonin, bradykinin, prostaglandins, leukotrienes, cytokines, dopamine, and noradrenaline contribute to nociceptor upregulation [5]. Prostaglandin-mediated activation of adrenergic α-receptors has been held responsible for sympathetic hyperalgesia [6].

Furthermore, not only peripheral but also central sensitization of dorsal horn neurones (or "neuroplasticity") occurs, due to the nociceptive input to the spinal cord. When these alterations last, pathological pain states might result, with pain occurring even in the absence of a distinct stimulus and with exaggerated responses to inocuous stimuli. Recently these findings have led to the "preemptive-analgesia" approach, preventing sensitization by blocking afferent pathways before setting the stimulus [7]. For example, postoperative pain is reduced when opiates are given before the start of surgery.

Pain increases the blood levels of catecholamines, cortisol, renin, vasopressin, glucagon, free fatty acids, and lactate [8]. Increased levels of stress hormones lead to hyperglycemia, insulin resistance, protein catabolism, and water and sodium retention. In head trauma patients catecholamine levels are correlated directly to the severity of injury measured with the Glasgow Coma Scale [9].

The neural input from afferent nerve fibers in the surgical wound is important in the response of the HPA. This has been certified many times by supplementing general anesthesia with epidural or spinal anesthesia, which prevents increases in ACTH and cortisol blood levels whereas general anesthesia alone results in increases in stress hormones. For example, after hip surgery ACTH and cortisol levels remain almost stable when general and epidural anesthesia are combined [10].

However, factors other than direct neural input most probably contribute to activation of the stress response. When upper abdominal surgery (pancreato-

duodenectomy) was performed under combined general and epidural anesthesia, an increased level of endotoxin was observed which was followed by elevation of tumor necrosis factor-α and interleukin-6 levels [10]. These two cytokines in turn stimulate ACTH and cortisol secretion and stimulate the adrenal response to ACTH [11]. Recently a molecular response to surgical stress was shown in rats when heat shock protein gene expression was induced simultaneously with activation of the HPA axis [12].

The degree of hormonal responses is positively correlated with the severity of surgical stress and the injury severity score. Yet, there is marked variability in sympathetic responsivity to surgical trauma.

Effects of Pain on Pulmonary Function

Trauma, especially blunt chest trauma or upper abdominal surgery, leads to substantial, restrictive changes in ventilatory patterns as vital capacity is reduced to 40%–60% of normal values [13]. Functional residual capacity and tidal volume-decrease while the respiratory rate increases. A fall in the functional residual capacity below the closing volume results in ventilation/perfusion mismatch and atelectasis, thereby finally generating hypoxemia and promoting pneumonia. These changes return to normal after 7–14 days. In the meantime these patients are not able to cough effectively and to clear secretions.

Cardiovascular Effects

Hypertension, tachycardia, and increased myocardial contractility all emanate from pain and activation of the sympathetic nervous system. As a result myocardial oxygen consumption is increased. At the same time volume depletion with the resulting hypotension, anemia, and hypoxia may contribute to decreased myocardial oxygen supply. The net result is myocardial ischemia and infarction. High levels of endogenous catecholamines may cause myocardial damage, and are associated with higher mortality rates [14]. Hackenberry considered elevated catecholamine levels to be responsible for myocardial injury in patients with severe head trauma [15]. Coronary spasms can be triggered by high levels of sympathometics. Erythrocyte flexibility is decreased by high levels of catecholamines, hampering the microcirculation and contributing to organ hypoxemia and dysfunction. Also, coagulation disorders are promoted by catecholamines [16].

Psychological Effects

Pain causes distress and suffering. Patients may feel afraid or even panic. Depression, delirium, and psychotic reactions have been described. However, the perception of pain is subject to wide interindividual variability. Mood, person-

ality, attention, expectations, and social and ethnic factors influence the degree of pain perception. Intensive care procedures, such as ventilatory support, have been described as unpleasant and stressful by around 50% of patients, thus pointing to the need for adequate stress reduction [17]. Helplessness and panic have also been recorded.

Other Effects

Pain may contribute to thromboembolic complications, as immobilization reduces pain but increases venous stasis. Pressure sores are more likely to develop in immobile patients, who do not move for fear of pain. Several studies have shown that trauma/surgery impairs the body's immune response. Pain also reduces gastrointestinal motility.

Preclinical Management of Analgesia/Sedation

The issue of sedation and analgesia is not only a question of reducing human suffering. Because pain and anxiety may aggravate the trauma response, analgesia and sedation contribute to sucessful management of various disorders and to prevention of life-threatening complications. High doses of systemic opioids at least partially blunt the hormonal response to surgery [18].

The first step to diminish pain and anxiety levels is that of a concentrated, skillful, and calm first-care provider. Comfort, psychological assistance, and verbal reassurance all are necessary measures and will invariably create confidence in the patient. Early immobilization of extremity fractures reduces pain. Many patients, however, need an analgesic or sedative for definite pain relief and anxiolysis.

Analgesia

In trauma patients the ideal analgesic drug should be easy to administer and provide analgesia effectively and quickly. Sedation and anxiolysis can to a certain extent be regarded as desirable side effects. Analgesics should have a rapid onset and a short to moderate duration of action. They should not depress respiratory or hemodynamic patterns. Nausea and vomiting or urinary retention are also unwelcomed side effects. Analgesia in trauma patients must consider the severity and site of injury. For example, in spontaneously breathing patients with head trauma, opioids should be administered cautiously, as hypoventilation results in a rise of pCO_2 and a subsequent rise in intracranial pressure (ICP). According to the severity of trauma, effective prehospital analgesia and reduction of stress response sometimes requires the induction of anesthesia. In contrast, a patient with a single limb fracture may be satisfied by a small amount of any given opioid or a peripheral analgesic such as acetaminophen.

Analgesics can be classified into four groups: (a) inhalation analgesics, (b) nonsteroidal anti-inflammatory drugs, (c) hypnoanalgesics (narcotics), and (d) ketamine

Inhalation Analgesia. Nitrous oxide/oxygen mixtures (Entonox) have been used for the prehospital phase since 1969 [19], and results show marked to complete relief of pain in 65% of patients. In a recent report all 63 ambulance services surveyed in the United Kingdom preferred Entonox, and only 5 of them administer other analgesics [20]. Entonox has sedative, amnestic, analgesic, and anxiolytic features and is effective and safe in both adults and children. Acute tolerance to its analgesic effects has been described [21].

Entonox can be administered easily by way of a demand inhalational unit, which is controlled by the conscious patient himself. Safety is further increased by the short half-life of the agent. Analgesia begins 20 s after the onset of inhalation and levels off 3–5 min after withdrawal. Most conveniently, the mixture is marketed in a single cylinder containing 50% of each gas. At temperatures below −6°C the nitrous fraction separates out and falls to the bottom of the cylinder. A patient inhaling from such a cylinder receives almost pure oxygen first, but as the cylinder empties, the concentration of nitrous oxide increases to almost 100%. Nevertheless, such conditions are uncommon in everyday practice. Insufficient analgesia has been reported in about 6% of patients.

Diffusion hypoxia is not a risk in healthy subjects, when Entonox in a 50% ratio is replaced by room air [22]. Entonox analgesia is contraindicated in patients who cannot self-administer the gas, and those who suffer from head trauma, pneumothorax, pulmonary edema, acute abdomen, middle ear effusions, or severe cardiovascular depression. Prolonged administration periods of more than 2 h result in bone marrow depression and leukopenia. Nausea may occur in some patients. N_2O increases the awareness of the surroundings, noises being unpleasant for the patient.

Although Entonox is safe and effective and has remarkably few side effects, the list of contraindications and the need for an additional apparatus has prevented the method from gaining broad acceptance outside the United Kingdom.

Nonsteroidal Anti-inflammatory Drugs. Nonsteroidal anti-inflammatory drugs (NSAIDs; "nonnarcotic" analgesics) act by blocking the action of cyclooxygenase on arachidonic acid. Thus, the production of prostanoids is inhibited and peripheral sensitization of pain receptors is diminished, as histamine or bradykinin action depends on prostaglandins. Ketorolac in addition blocks the lipoxygenase pathway, thereby preventing the formation of leukotrienes. NSAIDs have a mild to moderate analgesic activity and exhibit anti-inflammatory and antipyretic effects. For short-term use they may be as effective as morphine. NSAIDs have an opiate-sparing but no sedating effect [23]. Combination with a sedative or hypnoanalgesic agent is advisable to calm the patient, relieve anxiety, and relieve more severe pain.

Combination of either a NSAID or an opioid with an anticholinergic such as scopolamin, can be useful for relaxation of the smooth musculature. Side effects

Table 1. Nonsteroidal anti-inflammatory drugs

Drug	Route of administration	Dose (mg)	Dose interval (in hours)	Comments
Acetaminophen	po	500–1000	6–8	For children older than
	pr	500	6–8	3 months
Aspirin	po	300–1000	4	Not for children
	iv	500–1000	4–6	
Diclofenac[a]	po	50	6–8	Maximum 150 mg/day
	pr	50–100	6–8	
	iv	50–100	6–8	
Ibuprofen[a]	po	200–400	6–8	Maximum 1200 mg/day
Indomethacin[a]	po	25–50	6–12	
Ketorolac	po	10	6	
	iv	10–30	4–6	
Novaminsulfon	po	500–1000	6–12	Must be injected slowly
	iv	1000–2500	8–12	
Piroxicam[a]	po/pr	10–20	12–24	Maximum 40 mg/day

po; Oral; pr, rectal.
[a] Indicated only with pain of inflammatory origin.

are allergy and severe bronchoconstriction induced by enhanced leukotriene production. Prolonged NSAID therapy can result in renal failure when renal blood flow depends on vasodilating prostaglandins, as in hypovolemic patients or patients with chronic renal failure. For these reasons ketorolac was recently withdrawn from the German market. At least theoretically, NSAIDs given to trauma patients could enhance bleeding because of inhibition of thrombocyte cyclo-oxygenase, but this problem has not been studied yet. Table 1 presents a dosing regimen and pharmacokinetic data for some NSAIDs. Unfortunately, the only preparations available for intravenous administration are aspirin and novaminsulfone.

Hypnoanalgesics (Narcotics). Opiates (naturally occurring, e.g., morphine) and opioids (synthetical, e.g., fentanyl) are the first-line drugs in the treatment of severe pain. Until recently opioids were thought to induce physical and psychological dependency, but this has not been validated when opioids were used for acute pain relief. Morphine is the standard drug against which all others are measured. Opioids lower the sensitivity of the respiratory center to CO_2, which results in hypercapnia and a decreased respiratory rate. Apnea may occur, but patients will take a breath when requested to. When given for pain relief, the respiratory-depressant effect can be titrated against the degree of pain. Opioids can produce muscle rigidity to an extent that renders bag-mask ventilation

impossible. In this case small amounts of nondepolarizing muscle relaxants, benzodiazepines (BZDs), or barbiturates are helpful.

In hypovolemic patients, opioids should be used cautiously, as hypotension can be aggravated. All opioids stimulate central vagal nuclei and can provoke bradycardia. Nausea and vomiting are further side effects (especially with tramadol) but can be prevented by concomitant administration of neuroleptics such as triflupromazin or haloperidol. Slow injection also prevents nausea. In patients with renal failure a prolonged action of opiods has been described [24].

As all narcotics exhibit analgetic effects, the decision to choose one narcotic over another should be governed by their respective desired or undesired side effects. Morphine and meperidine liberate excessive amounts of histamine and may provoke severe bronchoconstriction, an effect not seen with fentanyl-like narcotics. These drugs should therefore be used cautiously in asthmatics. Also the sedative effects of opioids are relative to the drug used. Morphine, piritramid, and nalbuphine exhibit considerable sedating action, whereas meperidine, tramadol, and pentazocine cause almost no narcotic effects. Buprenorphine has a moderate effect in this regard.

Opioids can exhibit detrimental effects on the cardiovascular system. Meperidine increases heart rate and lowers arterial blood pressure. Pentazocine does not lower blood pressure but increases heart rate and rises pulmonary artery pressure. Neither of these drugs should be used in cardiac patients.

In acute trauma the speed of onset of pain relief is important. Intravenous administration is therefore the most suitable route for administration. The time-to-effect is shortest with this route, distribution is highly predictable, and drug effects can easily be monitored and titrated up to the desired level. Other routes of application include the transdermal administration of opioids (e.g., fentanyl) with relative ease of administration but with slow and unpredictable onset. In children rectal administration of drugs is common practice and has been shown to be effective for opioids, benzodiazepines, and barbiturates. Sufentanil, fentanyl, ketamine, and midazolam have been found to be reliable when administered intranasally. Buprenorphine has a satisfactory range of bioavailability after sublingual delivery and is therefore often administered by this route [25].

The amount of drug given must be titrated against the patient's reaction. Pharmacokinetic data must be kept in mind, such as the fact that the effect of alfentanil begins after some 2 min while morphine takes 5 min for time-to-onset and around 15–30 min for time-to-maximum effect. If repeated too early, overdosing will result. Attention should be paid not to mix agonist (e.g., fentanyl, morphine) and mixed agonist-antagonist drugs (e.g., pentazocine, nalbuphine, buprenorphine), as use of the latter can result in acute withdrawal syndromes when pretreatment consists of pure agonists. This problem is even more pronounced in patients who take opioids on a regular basis. However, some authors have recommended antagonization of fentanyl overdosing with nalbuphine, as this drug does not relieve analgesia [26]. The action of pure agonists can be reversed by naloxone, but antagonization of buprenorphine is impossible. Furthermore, the dose-response curve in pure agonists is linear, whereas

Table 2. Opioids for acute pain relief

Drug	Dose (mg)	Comparative potency	Time to onset/time to peak (min)	Comments
Morphine	2–5–10	1	5–15/15–30	Sedation, hypotension
Alfentanil	0.5–1	73	0.5–1/1–2	Extremely short distribution half-life
Buprenorphine[a]	0.1–0.3	33	10–15/20–30	No reversal with naloxone, sublingual administration possible
Fentanyl[b]	0.05–0.1	292	2–3/5–7	Short distribution half-life
Hydromorphone	0.5–1	5	5–10/30–45	–
Meperidine	25–50	0.53	2–5/15–45	Tachycardia, metabolite has convulsant properties
Methadone	2–5	4	5–15/30	Long elimination half-life
Nalbuphine[a]	10–20	0.7	2–3/10–20	Sedation
Pentazocine[a]	10–30	0.3	5–10/10–20	Tachycardia, high systemic and pulmonary artery pressure, no sedation, dysphoria
Piritramid	3.75–7.5	0.66	3–5/15–20	Sedation
Sufentanil	0.01–0.05	4520	0.5–1/3–4	Sedation, short half-life
Tramadol[b]	50–100	0.1	5–10/15–45	High incidence of nausea, almost no sedation

[a] Mixed agonist-antagonist.
[b] Narcotics used at our institution.

increasing the dose of mixed agonists-antagonists does not increase analgesia but increases side effects or even diminishes analgesia [27].

Table 2 presents comparative features of opioids as well as pharmacokinetic data. Only the intravenous route is considered, and no dosing intervals are given as the effect should be titrated. Sufentanil has been included although it has pronounced hypnotic virtues and a very short half-life. There have been conflicting results concerning the effect of sufentanil on ICP in patients with severe head trauma. Even the most potent opioid is not a total anesthetic and must be combined with a drug acting as a hypnotic [28].

Instead of using miscellaneous substances, our institution follows the policy of restricting the number of narcotics to two (indicated in Table 2). Also, only experienced emergency physicians are allowed to inject analgesic medication.

Ketamine. Ketamine, a phencyclidine derivative, is a drug that produces either analgesia or anesthesia depending on the dose given. An intravenous dose of 0.25–0.5 mg/kg or an intramuscular dose of 0.5–1 mg/kg produces analgesia with moderate narcotic effects, whereas an intravenous dose of 2 mg/kg results in a trancelike patient after 2–3 min, lasting for 10–15 min. When given intramuscularly at a dose of 5–10 mg/kg, onset of effect occurs after about 5–8 min, but recovery time is prolonged (30–120 min). Ketamin 0.5 mg/kg intramuscularly results in effective analgesia for about 10 min, without mental obtundation [29]; increasing this dose to 1 mg/kg results in an almost pain-free period of about 30 min [29].

Ketamine administration uncouples the cortical and limbic systems, and its action has been described as "dissociative." The analgetic action of ketamine does not seem to be mediated via opioid receptors as it is also effective in the presence of naloxone [30], but the opposite has also been reported. Ketamine may induce spontaneous movements, not related to pain sensations, and increase muscle tone. Airway reflexes are maintained, but respiratory depression occurs when high doses are given rapidly. Salivation is a main adverse reaction that can be abolished by an anticholinergic agent such as atropine or glycopyrrolate. Ketamine induces hallucinations, unpleasant dreams, and misperception of auditory and visual stimuli in 50% of patients. Addition of a benzodiazepine can alleviate these side effects. Benzodiazepines or neuroleptics reduce the nausea and vomiting, associated with about 50% of cases, when ketamine is used as a sole substance.Ketamine has a centrally acting sympathometic effect which results in increases in blood pressure and heart rate. For this reason ketamine has been recommended for hypovolemic trauma patients. For example, analgesia for extrication of patients trapped in car wrecks can be achieved without hemodynamic compromise. Exogenous catecholamine requirements are diminished when ketamine/midazolam is compared to fentanyl/midazolam [31]. However, in patients having exhausted all sympathetic reserves, its direct myocardial depressing activity can lead to severe hypotension. As myocardial oxygen consumption increases with sympathic stimulation, ketamine should be used cautiously in patients with coronary artery disease. This sympathetic effect is beneficial in asthmatic patients, where bronchodilation is needed. In patients with head

trauma, ketamine administration results in elevations of ICP because of CO_2 retention; this effect can be counteracted by artificial respiration therapy.

In our institution ketamine is the drug of choice in hemodynamically compromised patients with or without head injury. Small amounts of a benzodiazepine are added immediately before ketamine injection.

Sedation

Mild (conscious) sedation in a fearful, distressed patient reduces the level of anxiety and facilitates treatment. More profound sedation (or, better, anesthesia) is required in a patient who needs mechanical ventilation. Theoretically, this can be achieved with any drug presented in this chapter, but pharmacodynamic and -kinetic features vary and must be considered. The low distribution volume in hypovolemic patients should be kept in mind, and the dose of the sedative drug should always be titrated in order to prevent unintentional apnoea. Concomitant use of an analgesic drug or alcohol increases both potency and side effects of the sedatives.

Five different groups are in use currently: benzodiazepines, barbiturates, major tranquilizers, etomidate, and propofol.

Benzodiazepines. Diazepam, midazolam, and flunitrazepam are the three most commonly used BZD (Table 3). BZDs bind specifically to receptors in the frontal and occipital cortex and in the limbic system. Their effect is sedative, anxiolytic, anticonvulsive, and hypnotic (according to the dose given). After BZD administration, stress-induced increases in plasma-norepinephrine levels and blood pressure are reduced [32]. Anterograde amnesia is a common feature, although most pronounced with midazolam. Sedative or hypnotic effects are greater with flunitrazepam or midazolam.

BZDs exhibit a "ceiling-effect," that is, there is no linear relationship between the degree of sedation and the plasma-concentration. Once all the receptors are saturated with BZDs, only the side effects increase. However, when used as a sole substance, there is almost no effect on the cardiovascular system. When combined with analgesic drugs, BZDs sometimes do exhibit hypotensive effects.

Table 3. Benzodiazepines

Drug	Dose (mg) iv	Time-to-onset (min)/ duration (h)	Comments
Diazepam	0.1–0.4	1–3/1–3	Painful injection
Flunitrazepam	0.015–0.03	2–4/6–8	Painful injection, hypotension
Midazolam	0.05–0.3	1–3/0.5–1.5	Apnea with high doses
Flumazenil	0.3–2	1–3/0.3	Short half-life

Time-to-peak effect is 2–5 min with diazepam and flunitrazepam and 2–3 min with midazolam. In terms of their elimination half-lives BZDs can be divided into long-acting (diazepam), medium-acting (flunitrazepam), and short-acting (midazolam) substances. For this reason midazolam has replaced diazepam as the drug of choice in critical care medicine. Its elimination half-life is only 1–4 h, compared with 24–50 h for diazepam. Accordingly, return to baseline function is earlier when midazolam is used for conscious sedation than when diazepam is used. However, midazolam kinetics in severely ill patients can change, and absence of metabolism has been described.

Midazolam titration should be started with an initial dose of 1–2 mg, and the patient's reaction be observed for 2–3 min. In many cases conscious sedation is sufficient with such a dose whereas a young, healthy patient may need 5–7.5 mg. Because midazolam is water soluble, no solvents are needed, and pain with injection is less common than with other BZDs. Paradoxical reactions are possible in about 10% of patients receving BZDs. The incidence of apnea is similar whether midazolam or thiopental are given for induction of anesthesia. Both diazepam and midazolam decrease upper airway reflex sensitivity [33]. However, BZDs have remarkably low toxicity and few side effects.

Flumazenil, a benzodiazepine antagonist, reverses all BZD-induced effects at a dose of 0.3–2 mg. Flumazenil is virtually devoid of any intrinsic action. Should BZD-overdosing occur, reversal is easy when flumazenil is titrated to the desired effect. Elimination half-life of flumazenil is remarkably shorter than that of midazolam (1 h vs 1.5–3 h), and re-sedation must therefore be monitored closely, or a continuous flumazenil infusion should be started.

In our EMS system we use diazepam for conscious sedation when a long-lasting effect is desired (e.g., in psychiatric or hyperventilating patients). In all other cases (e.g., sedation for artifical ventilation) we rely on (titrated) midazolam. Flumazenil is used as a diagnostic tool for suspected BZD overdose.

Barbiturates. Barbiturates are derivatives of barbituric acid, which itself possesses no hypnotic activity. γ-Aminobutyric acid (GABA) activity in the central nervous system is increased by barbiturates. In extremis, cerebral function can be depressed to an isoelectric EEG. Barbiturates dose-dependently induce sedation and hypnosis and are suitable for induction of anesthesia (Table 4). Thiopental and methohexital are ultrashort-acting substances, with hypnosis lasting 5–10

Table 4. Barbiturates

Drug	Dose (mg/kg) iv	Time-to-onset (min)/ duration (min)	Elimination half-life (h)	Comments
Methohexital	1–2	0.5–1/5–10	1–2	Pain on injection
Pentobarbital	2–5	1/15	20–50	–
Thiopental	2–6	0.5–1/5–15	5–12	–

min after the usual induction dose. Pentobarbital is short-acting, with hypnosis lasting for about 15–20 min. When repeated doses are given, there is considerable accumulation, with prolonged awakening times. Respiratory depression must be carefully monitored. Hiccups and coughing are seen with methohexital in about 30% of patients, but this rate is lower with the other drugs.

Barbiturates reliably decrease high ICP by diminishing cerebral oxygen demand. Even in normal subjects barbiturates lower blood pressure and cardiac output, with a compensatory increase in heart rate. This effect is dose related and depends on the speed of injection. In hypovolemic patients heart rate and systemic vascular resistance are already maximally elevated, and compensatory mechanisms are exhausted. Barbiturates may thus induce severe hypotension. Barbiturates are further contraindicated in patients with status asthmaticus or a history of acute intermittent porphyria.

Subanesthetic doses heighten airway reflexes, and laryngospasm can result. Furthermore, barbiturates are hyperalgesic in low doses. Because of these features, the sometimes marked cardiovascular depression and the short action, we do not use them for either short- or long-term sedation. However, when the patient suffers from severe head trauma, and there is no cardiovascular depression, we use thiopental for induction of anesthesia because of the beneficial influence on ICP. Once intubated, anesthesia is maintained by repeated injections of fentanyl/midazolam.

Major Tranquilizers. Sedation is also a common feature with neuroleptics such as the butyrophenones (haloperidol, droperidol) and the phenothiazines (chlorpromazine, thioridazine, trifluprom azine; Table 5). With haloperidol, sedation is only moderate, whereas droperidol was used as the hypnotic agent in neuroleptanesthesia. Chlorpromazine and trifluprom azine have potent sedative and fewer antipsychotic features. The antipsychotic effect of thioridazine is more pronounced. Phenothiazines act as antihistamines and lower the epileptic threshold.

This group of drugs was developed for use in psychiatric patients, and their sedative action is only a side effect. Furthermore, patients appear to be calm, but

Table 5. Major tranquilizers

Drug	Dose (mg)iv	Time-to-onset (min)/ duration (h)	Elimination half-life (h)	Comments
Haloperidol	2.5–5 every 30 min	10/12–24	10–36	Antipsychotic, up to 300 mg/day reported
Droperidol	0.625–1.25, 2.5–5	3–10/2–4	6–12	Antiemetic dose antipsychotic dose
Chlorpromazine	30–300	1–3/2–4	–	–
Trifluprom azine	2.5–5–10	1–3/4–6	–	–

later recall inner agitation and apprehensiveness. Major tranquilizers impede thermoregulation, can depress the bone marrow, and lower blood pressure because of their sympatholytic virtues. Hypotension is even more pronounced when opioids are given jointly. Administration of phenothiazines may result in ventricular fibrillation, and the use of haloperidol in torsade de pointes [34]. Extrapyramidal side effects such as akathisia, dystonia, or dyskinesia have a low occurrence rate with short-term use but develop in about 5%–36% of patients when used for prolonged periods [35].

Major tranquilizers are useful in the emergency department for treatment of delirium of unknown origin. A period of 30 min should be allowed to pass before injection is repeated. Riker et al. reported on successful continuous haloperidol infusion for control of agitation in critically ill patients, at an average daily dose of 269 mg [35]. With haloperidol, sedation is the first action, with antipsychotic effects emerging only after several days. Therefore its use should not be discontinued too early because of presumed ineffectiveness. Anxiety is not relieved, and benzodiazepines are superior for this indication. Droperidol or triflupromazine are useful in low doses when a patient presents with (opioid-related) nausea or vomiting.

In our EMS system there is no well-defined policy regarding the use of these drugs, and administration is left to the discretion of the emergency physician. In delirant patients the titration of haloperidol, given concomitantly with small increments of midazolam, has been sucessful. Antiemetic therapy with major tranquilizers is performed only when opioids cause nausea.

Etomidate. Etomidate is an ultrashort-acting, barbiturate-free hypnotic which acts GABA-like. Etomidate is almost the ideal hypnotic as it causes only minimal hemodynamic depression, hypnosis in one arm-brain circulation time, reduction in ICP, and rapid recovery. At a dose of 0.2–0.6 mg/kg (mean 0.3 mg/kg) it is an ideal induction agent when preservation of cardiovascular stability is mandatory. Time-to-onset of effect is around 30 s, distribution half-life is around 3 min, and elimination half-life around 4 h.

Etomidate may induce apnea, especially in elderly patients, and the drug has no analgesic activity. Another reason for combination with an opioid or benzodiazepine is to reduce the incidence of myoclonic activity, otherwise occurring in up to 70% of patients. An additional advantage of adding a BZD/opioid is a more profound anesthesia than with etomidate alone. Pain can occur on injection, but recently a lipid-containing formula was released which has reduced the incidence of pain.

The main adverse effect of etomidate is suppression of adrenal steroidogenesis, even after a single injection. Although this may have no effect on the clinical course of patients, Ledingham et al. reported higher mortality in trauma patients sedated with etomidate than in those sedated with opioides and BZDs [36]. Therefore, long-term etomidate sedation is not recommended.

We use etomidate for induction in the preclinical setting if the patient is hemodynamically unstable (unless ketamine is used). It is always combined with an opioid or a BZD. Further anesthesia is maintained with fentanyl/midazolam.

Propofol. Propofol comes in a lipid-emulsion as it is insoluble in water. Propofol reduces pre- and afterload and depresses cardiac function more than thiopental, especially when given as a single bolus. Slow injection can reduce the degree of cardiovascular depression. In Pinaud's study in head trauma patients, propofol reduced cerebral perfusion pressure as a result of lowering mean arterial pressure [37]. The author did not recommend its use under emergency conditions.

Propofol has no analgesic action. When combined with opioids, bradycardia is a relatively common feature, which necessitates parasympatholytics such as atropine. Unlike etomidate, it does not suppress adrenal steroidogenesis.

After an induction dose of 1.5–2.5 mg/kg, loss of consciousness occurs after approximately 30 s, and recovery is rapid (approximately 6 min after the initial dose). Elimination half-life is 3–6 h. Cognitive functions recover remarkably quickly after propofol anesthesia.

Propofol 1–3 mg kg^{-1} h^{-1} effectively sedates patients requiring mechanical ventilation, and no cumulative effects have been seen in this setting. Because of this lack of cumulation propofol can be used to wean patients from long-term BZD use. BZD serum levels fall below the hypnotic threshold, and termination of propofol-infusion awakes the patient after 20–30 min. When given in small veins, pain on injection occurs in up to 40% of patients. Propofol can be used safely in patients with liver or renal disease.

Propofol is especially valuable when prompt recovery is important (e.g., repeated neurologic examination), or when the patient is scheduled for only short-term mechanical ventilation. In the preclinical setting we do not use it for fear of adverse hemodynamic effects in hypovolemic patients. After all, continuous infusion of propofol for sedation after induction with another, more favorable, agent is a treatment modality that we regularly use in our ICU.

Conclusion

No ideal agent exists for analgesia/sedation in emergency situations. Low to moderate pain states can be treated with NSAIDs, but severe pain requires effect-titrated intravenous opioid administration. Adequate sedation can be achieved as a side effect of opioid administration. For anxiolysis and stress reduction benzodiazepines (especially midazolam) can be titrated or coadministered to the desired effect. In both cases provisions must be made to treat major side effects such as respiratory depression or hypotension.

In hemodynamically unstable patients, ketamine administered intravenously or intramuscularly provides reliable analgesia without cardiovascular depression. Benzodiazepines and/or atropine should be given concomitantly. Etomidate is an alternative in this group of patients as it has no pronounced effect on cardiovascular parameters. BZD or opioid administration prior to etomidate injection is strongly advised. Major tranquilizers are indicated in delirium of unknown origin and should always be combined with BZDs. The place of propofol in prehospital care remains to be determined, but it is invaluable in short-term sedation, and when repeated neurologic assessments are indicated.

Most importantly, drug therapy in emergencies should be restricted to the use of substances whose effects are well-known by the emergency care provider, and whose adverse effects can be managed safely.

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Causes of Shock in the Severely Traumatized Patient: Emergency Treatment

W. Ertel and O. Trentz

Introduction

Shock-like states are typical systemic reactions of severe trauma. Although in most severely injured patients, hemorrhagic shock is responsible for circulatory instability, continuous posttraumatic shock may be augmented by massive tissue destruction, spine and brain injury, or penetration of microorganisms through destroyed mucosal or skin barriers with consecutive endotoxemia. Tremendous progress in preclinical treatment of traumatized patients has been achieved with regard to fluid resuscitation and ventilatory support. However, a high number of patients who survive the early critical period after injury die in the late posttraumatic period because of an increased susceptibility to infections.

Although the relationship between severe shock and late infectious complications is not obvious, recent studies clearly demonstrate a close relationship between traumatic shock, systemic inflammatory response syndrome (SIRS), infectious complications, and multiple organ dysfunction syndrome (MODS) [1, 2]. It has become apparent over the past 5 years that serious injury with severe shock results in significant depression of essential immune functions, which causes a high incidence of posttraumatic infections with high morbidity and mortality [1, 2]. Therefore rapid recognition and successful treatment of traumatic shock not only improve the survival in the early period after severe injury but also dramatically affect the incidence of complications in the late posttraumatic course.

Classification of Shock

Shock has been recognized as a condition for over 100 years, but a clear definition of the complex pathophysiological alterations has only emerged slowly. Blalock [3] described shock in 1940 as peripheral circulatory failure due to a discrepancy between the size of the vascular bed and the volume of intravascular fluid. Today, shock is defined as a state of reduced tissue perfusion leading to generalized cellular hypoxia with a continuous discrepancy between delivery and consumption as well as utilization of oxygen [4]. The resulting deficit of energy as a consequence of decreased intracellular ATP levels leads to cell dysfunction and finally cell death with consequent vital organ damage and MODS [5].

The three essential and most common forms of shock [6] that occur in the early period after trauma are: hypovolemic (hemorrhagic) shock, cardiogenic shock, and spinal shock. In addition, in severely injured patients one often observes traumatic-hemorrhagic shock, which reflects severe hemorrhage combined with extensive tissue destruction and systemic trauma reactions including neuroendocrine response, symphaticoadrenal activation, release of proinflammatory mediators and oxygen free radicals, and a metabolic response. Because of the pathophysiological alterations caused by tissue destruction and the specific trauma reaction the hemodynamic and pulmonary alterations after traumatic-hemorrhagic shock are much more pronounced than in pure hemorrhagic shock (e.g., GI bleeding).

All four forms of shock invariably show one or more of the following separate, but interrelated pathophysiological alterations: loss of intra- and/or extracellular fluid, pump failure, and decreased vascular resistance. Hypovolemic shock in traumatized patients includes a significant loss of fluid in the form of whole blood, plasma, or extracellular fluid through hemorrhage in and around areas of injury. The following injuries, isolated or in combination, cause severe hemorrhagic shock with manifest symptoms: trauma with disruption of the spleen and/or liver, fractures of the pelvis or long bones, injuries of major vessels, and extended soft tissue trauma including subtotal or total amputation. Cardiogenic shock implies a failure of either left ventricular ejection caused by extended myocardial contusion, or a failure of left ventricular filling (obstructive shock) which occurs through cardiac tamponade, tension pneumothorax, or vena caval obstruction. Spinal shock is characterized by a generalized interruption of vasomotor control, as occurs after trauma to the spinal cord or after severe brain injury.

Physiological Changes

After loss of intravascular volume, arterial blood pressure remains normal as long as the total peripheral vascular resistance can be increased to compensate the reduction in cardiac output. Through this autoregulation blood losses of less than 10% of the total intravascular volume can easily be compensated without a decrease in cardiac output or mean arterial pressure. Blood losses greater than 10% result primarily in a reduction of cardiac output. If the blood loss exceeds 20%–30% of total blood volume, a decrease of cardiac output below 40% results in a significant decrease of the mean arterial pressure.

Total blood flow to the heart and the brain is maintained while vasoconstriction occurs in all other organs which are not essential for immediate survival. Thus, the great increase in peripheral resistance in these organs (skin, kidney, liver, spleen, gut) leads to significant reduction in blood flow to these organs while providing a life-saving diversion of cardiac output to the brain and myocardium. Hypoxia in the liver, spleen, and kidney causes activation of tissue macrophages with increased release of proinflammatory and immunosuppressive mediators [7], as well as hypoxic destruction of parenchymal cells resulting in organ dysfunction

and organ failure. Moreover, a long-lasting shock-like state with decreased blood flow in the gut enhances the translocation of microorganisms into the lymph nodes and portal vein [8].

If the adaptive homeostatic mechanisms can no longer compensate for the continuous reduction of intravascular volume, blood pressure rapidly decreases, resulting in low flow conditions and whole body ischemia. The reduction in intravascular volume is generally associated with tachycardia via an excitation of the sympathicoadrenal nervous system with simultaneous inhibition of the vagal-medullary center.

The decrease in cardiac output, which is caused by the loss of intravascular volume and a consequent decrease in the venous return, results in a very rapid increase in peripheral resistance and generalized venous constriction. Moreover, extravascular, extracellular fluid is moved into the circulation, leading to hemodilution. Since this process does not occur as rapidly as the above mentioned compensatory mechanims, the hematocrit remains stable in the very early period after trauma, despite massive blood loss.

The biochemically measurable changes during shock are: (a) changes invoked by the pituitary-adrenal response to stress, (b) alterations as a result of reduced organ perfusion, and (c) changes brought about by failing functions within specific organs. The immediate effects, seen from the symphaticoadrenal activity, are associated with high circulating epinephrine levels. Further alterations induced by the symphaticoadrenal response include a negative nitrogen balance, retention of sodium and water, and a notable increase in the excretion of potassium.

These pathophysiological changes lead to a decreased blood flow ("low-flow state") in vital organs with a reduced delivery of oxygen, thus resulting in a mandatory change in metabolism from aerobic to anaerobic. The net result of this low-flow state represents a significant reduction of intracellular ATP levels and an enhanced production of lactic acid ("hidden acidosis") [9]. In addition, persisting hypoxemia activates macrophages to produce large amounts of so called proinflammatory mediators (tumor necrosis factor-α and interleukins 1β, 6, and 8). These mediators, in combination with oxygen free radicals and proteases, seem to be responsible for shock-induced MODS [10, 11].

Diagnosis

The symptoms of hypovolemic shock are classic and usually easy to recognize. These signs are generally characteristic of decreased peripheral blood flow with significant hypotension and are contributed to by the effects of excessive adrenal-symphathetic activity with tachycardia. In young patients signs of shock may be hidden for a long time, which leads to a sudden circulatory collapse with a life-threatening shock-like state. Young traumatized patients who sustain hemorhagic shock appear restless and anxious, followed by great apathy.

Shock in the aged patient may result in early impairment of cardiac and pulmonary functions, which rapidly complicates the situation. For these reasons

Table 1. Grading of shock (from [12])

Degree of shock	Blood pressure	Pulse quality	Skin temperature	Skin color	Circulation in skin	Thirst	Mental state
Slight (blood loss 10%–20%)	Increased	Normal	Cool	Pale	Definite slowing	Normal	Clear and distressed
Moderate (blood loss 20%–40%)	Decreased	Decrease in volume	Cool	Pale	Definite slowing	Definite	Clear and some apathy unless stimulated
Severe (blood loss > 40%)	Decreased to nonrecordable	Weak to imperceptible	Cool	Ashen to cyanotic	Very sluggish	Severe	Apathy to coma

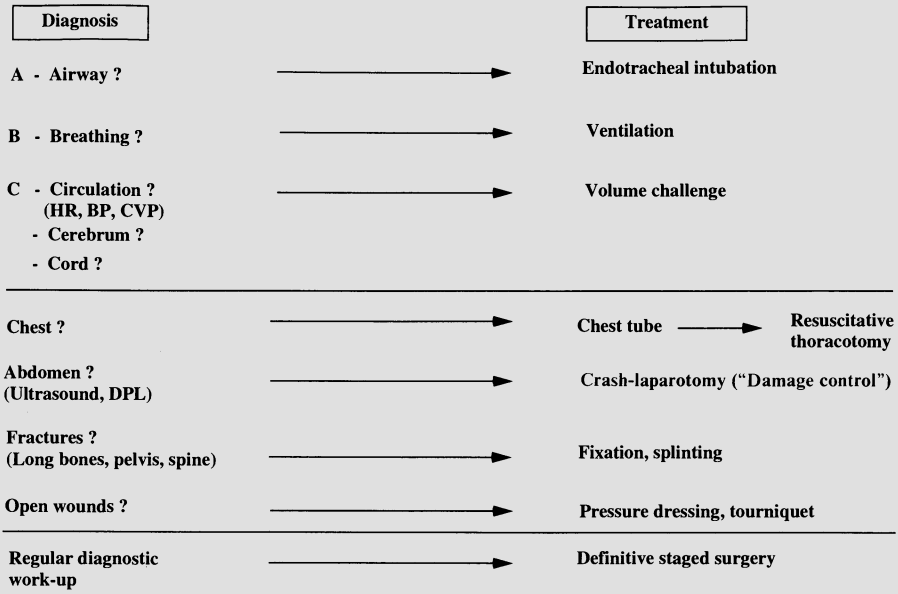

Fig. 1. Management of the severely injured patient in shock

injured patients with shock require individual consideration with regard to severity of injury, age of the patient, and duration of the shock episode. Moreover, the importance of defining criteria for the recognition of shock lies in the early impression of impending disaster which may be apparent for some time before blood pressure and metabolic changes become evident. Table 1 summarizes the early signs and symptoms, as they occur during hemorrhagic shock and with regard to the severity of illness [12].

In cardiogenic shock patients demonstrate tachycardia, arrhythmias, increased central venous pressure, and in the case of cardiac tamponade a paradoxic pulse. However, there are some differences in the classic picture of hypovolemic shock and spinal shock, where the pulse rate is normal or often decreased. In addition, the pulse pressure is wide, the pulse feels strong rather than weak, and the skin is dry, warm, and even flushed. The reduction in cardiac output is accompanied by a decrease in resistance of arteriolar vessels.

Physical examination of the traumatized patient must be carried out immediately after the admission of the patient in the emergency room, with special attention to ventilation, circulation, and external or internal sources of serious bleedings (Fig. 1). Evaluating the effectiveness of ventilation by auscultation of the chest permits the diagnosis of a (tension) pneumothorax. In addition, registration of blood pressure, pulse rate, and central venous pressure is essential for indicating hypovolemic or cardiogenic shock. Examination of the abomen and thorax, including ultrasound, provides evidence of acute intraabdominal bleeding, hemothorax, or cardiac tamponade. Inspection of the pelvis and of the extremities further allows diagnosis of long bone fractures and severe tissue

injury (Fig. 1). This information is helpful in identifying shock-like states in most patients and leads to a prompt treatment.

Emergency Treatment

The therapy of shock must be started promptly and focus on four pathophysiological alterations: (a) oxygen transport capacity, (b) cardiac output, (c) hemoglobin-oxygen saturation, and (d) hemoglobin-oxygen affinity. These parameters are closely related to each other; the available oxygen transport capacity has been quantitated by Nunn and Freeman [13] as: available oxygen = cardiac output × arterial O_2 saturation × hemoglobin concentration × 1.34. In a resting human the available oxygen is 250 ml/min, which can be increased to 1000 ml/min under stress conditions. However, the severely injured patient requires an increased delivery of oxygen to the tissue, which depends on the available oxygen transport capacity. If two of the variables decrease at the same time, the effect on available oxygen equals the product of the individual changes. Thus, if cardiac output and hemoglobulin are both half of normal, the available oxygen is reduced to one-quarter, or 250 ml/min. This value represents the most critical level in injured patients and is compatible with life for only a short period of time. To correct the reduced availability of oxygen in the tissue effective treatment of shock must consider three specific modalities: fluid replacement, ventilation therapy, and drug therapy.

Hypovolemic Shock

Hypovolemia is the most common cause of shock in traumatized patients but is most readily responsive to rapid and correct therapy. After obvious extensive injury, aggressive fluid replacement is the first choice, provided the atrial filling pressure is adequate to produce an effective cardiac output (exclusion of cardiogenic shock). This essential information is gained by clinical observation of the pressure in the neck veins or, better, by measurement of central venous pressure. Bickell et al. [14] suggested treating hypotensive patients with penetrating torso injury with delayed fluid resuscitation until surgical control of hemorrhage.

At least two large-gauge cannulas are inserted immediately into appropriate veins. Preferably the arms are used for percutaneous puncture, while a saphenous venous cutdown at both ankles can provide rapid access. The type of fluid that should be used for the primary treatment of hypovolemic shock is still under discussion, but many authors now recommend a combination of crystalloid and colloid solutions with a ratio of 3:1 in favor of crystalloids [4]. Volume replacement with albumin or fresh frozen plasma does not have a positive effect. If blood loss has been severe (> 40% of total blood volume) or hemorrhage continues, accurately typed and cross-matched packed red cells should be given. However, it must be emphasized that the infusion of packed red cells may increase

the disturbances in microcirculation through an elevation of viscosity. Recently, the so-called "small-volume resuscitation" with hypertonic-hyperoncotic solutions has been clinically tested [15].

One should avoid positioning the severely injured patient with brain or chest injury in a head-down (Trendelenburg) position as an adjunct in the treatment of hypovolemic shock. The Trendelenburg position increases intracranial pressure and simultaneously decreases lung volume because of displacement of the diaphragm and intra-abdominal contents. The G-suit and the military anti-shock trousers which have been used predominantly in the United States to control exsanguinating hemorrhage after severe pelvic injury or abdominal trauma, have failed to achieve hemodynamic stability, but cause serious ischemic damage in the lower extremities.

Ventilation therapy should be used aggressively in patients with shock after severe trauma or with brain injury [16]. Airway control allows for endotracheal suction with removal of secretion or aspirated blood, and optimizes oxygen delivery.

Vasopressors can be used in cardiogenic and spinal shock. Although vasopressors have been popular in the therapy of hypovolemic shock in recent years, they decrease tissue perfusion by raising peripheral vascular resistance. Therefore, catecholamins and other vasopressors should be used only in patients with cardiac arrest or spinal shock, where peripheral vascular resistance is significantly decreased. Additionally, the prophylactic use of bicarbonate solution is not recommended because of its paradoxic effects on tissue pH. The basis of treatment is to correct the anaerobic metabolism by restoring tissue perfusion, instead of loading the tissues with buffered solutions.

Cardiogenic Shock

Cardiogenic shock in traumatized patients is due either to cardiac tamponade or compression of the pericardium from tension pneumothorax, or to cardiac contusion with extended myocardial infarction. If the origin of pump failure is a tension pneumothorax or cardiac tamponade, the insertion of a chest tube or a pericardiocentesis should be carried out immediately. In the case of myocardial contusion with significant reduction in blood pressure, drugs with positive inotropic action may be effective.

Spinal Shock

Spinal shock occurs generally in injured patients with a fracture of the spine and compression or dissection of the spinal cord. In the management of injured patients with spinal shock, where significant blood loss into the areas of injury surrounding the cord is often found, a balanced therapy of fluid administration and vasopressors such as phenylephrine is recommended.

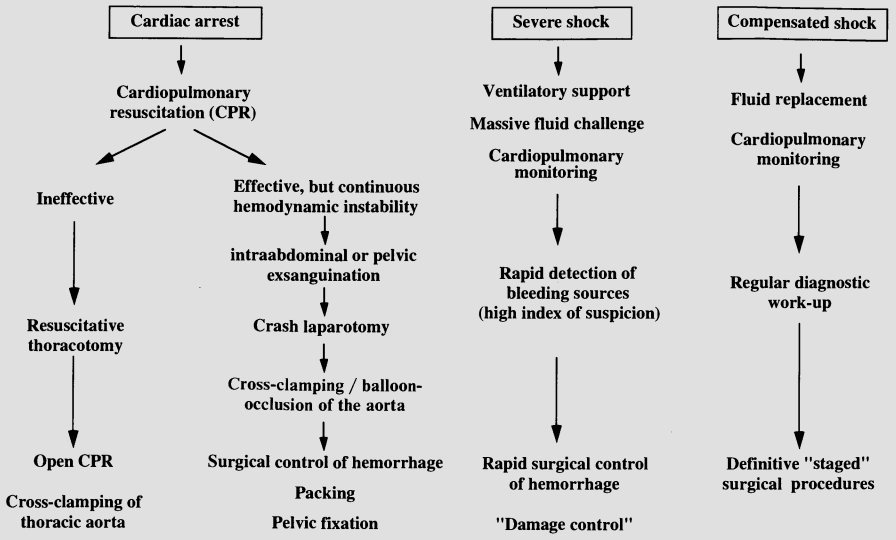

Fig. 2. Shock: algorithm of acute treatment

Traumatic-Hemorrhagic Shock

The basic tenets in treating patients with multiple injuries and a shock-like state are (a) that life-threatening injuries take priority, and (b) that overall responsibility for care should rest with the trauma team leader. Patients with severe injuries are divided into three categories with regard to the diagnostic and operative management (Fig. 2): those with cardiac arrest, those with unstable vital functions, and those with stable vital signs. In severely injured patients with cardiac arrest, basic and advanced cardiac life support must be carried out at the scene. If cardiac arrest persists after arrival of the patient in the emergency room, emergency thoracotomy [17] allows for open resuscitation with optimization of cardiac output, release of cardiac tamponade, control of great vessel and cardiac bleeding (penetrating injuries), and redistribution of the available blood to vital organs (brain, heart) by cross-clamping the descending aorta (Fig. 2).

In exsanguinating injuries of the abdomen or crush trauma of the pelvis, laparotomy in the emergency room is indicated to clamp the abdominal aorta under the diaphragm or to insert an aortic balloon occluder [18]. This is followed by rapid operative control of extensive bleeding. The operative techniques depend on the location of injury, using partial (liver) or total organ resection (splenectomy), Pringle's maneuver, or packing (liver, pelvis) (see Battistella and Blaisdell, this volume). It should be emphasized that trauma victims who have cardiac arrest before they arrive at the hospital only have a 3%–8% likelihood of survival [19].

Primary treatment of patients with severe shock and unstable hemodynamic conditions includes rapid establishment of an airway and circulatory assistance. Most of these patients need emergency operations to control hemorrhage. In

abdominal trauma combined with vascular and visceral injury, additional physiological derangements such as coagulopathy, hypothermia, acidosis, and contamination occur. In these patients one uses "damage control" with initial control of hemorrhage and contamination followed by intraperitoneal packing and rapid closure. This procedure allows for rapid resuscitation in the ICU to achieve stable hemodynamic conditions and subsequent definitive reexploration [20].

No time should be wasted in getting the patient into "operative condition" or attempting to establish a definitive diagnosis by laboratory or X-ray means. It should be emphasized that the mean arterial pressure in patients with severe shock is highly susceptible to anesthesia with inhalation narcotics, which decrease vasotonus and thus augment the shock-like state. Moreover, emergency laparotomy can impair the existing shock-like state by sudden decompression of the large intraabdominal veins, which can result in massive venous pooling.

The third group of patients show signs of shock because of injuries which obviously require surgical intervention. Because of compensated shock with stable vital signs, X-ray or computed tomography is able to precisely define potentially associated injuries. These patients undergo surgery within 1–2 hours. Operative management includes repair of liver and spleen injuries, primary stabilization of major fractures including shaft fractures of long bones, unstable injuries of the spine, the pelvis, and of large joints ("day one surgery"; see Biert and Goris, this volume). Primary fracture fixation not only decreases blood loss but also reduces stress, pain, and tissue destruction and thus results in a decreased incidence of ARDS and MODS [21, 22].

Conclusion

The adequate and successful therapy of shock-like states in severely injured patients still remains a challenge for the trauma team. Although great progress has been achieved in the prehospital treatment of traumatic shock, with a significant reduction of mortality at scene or during transport to the emergency room, the long-term effects of insufficient or delayed shock treatment, such as immunosuppression with increased susceptibility to infection or continuous systemic inflammation with consequent MODS, are deleterious for the host. To avoid or minimize the incidence of these posttraumatic complications aggressive treatment of severely injured patients with airway control, ventilation, and sufficient fluid replacement must be carried out. Diagnostic procedures depend on the stability of vital functions. A rapid and, if possible, definitive treatment of fractures of the pelvis, spine, and long bones decreases the risk of posttraumatic ARDS or MODS, thus leading to reduced morbidity and mortality of severely injured patients.

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Volume Infusion in Traumatic Shock

J. S. Mondy III and F. W. Blaisdell

Introduction

The term shock is used to describe a clinical syndrome caused by inadequate delivery of nutrients, including oxygen, to the tissues. Even though shock may be manifested by generalized physiologic derangements, the pathophysiology takes place at the cellular level. In victims of trauma inadequate cellular perfusion can be attributed to hypovolemia from blood loss (hemorrhage), increased microvascular permeability, or redistribution of intravascular volume in the capacitance vessels [1]. The end result is a vicious cycle of oxygen debt leading to metabolic acidosis, cellular dysfunction, and ultimately cellular death [2].

The goals of resuscitation in traumatic shock are to correct this deficit in plasma volume and restore oxygen delivery to the tissues. Our understanding of the pathophysiology of shock resuscitation is based in large part on the research of Shires et al. who found that in a model of hemorrhagic shock there is a significant reduction in skeletal muscle cell membrane potential which is associated with a shift in water, sodium, and chloride from the extracellular to the intracellular space, resulting in cellular edema and a decrease in the interstitial fluid volume [3]. The goal of volume infusion therefore is restoration of the plasma and interstitial fluid volumes and correction of the electrolyte deficit. Although shock and resuscitation both occur as a continuum, for practical purposes, the discussion of volume infusion in traumatic shock can be divided into three phases: prehospital resuscitation, hospital resuscitation (both in the emergency and operating rooms), and postoperative resuscitation.

Prehospital Resuscitation

The issue of prehospital institution of fluid resuscitation in trauma has stirred much controversy in the literature. Whereas in the setting of cardiac arrest prehospital care is definitive, in the trauma patient prehospital care is resuscitative only, and definitive care must be given in a hospital setting. In metropolitan centers it has been shown that transport time to the trauma center where definitive surgical intervention is available is less than the time needed to establish intravenous access [4]. Similarly, the small amounts of isotonic fluids given during transport were found to have no influence on survival [4, 5]. In patients with only

a mild degree of shock, vascular access is possible, but the benefit of fluid administered is negligible.

In patients with severe levels of shock, however, the venous system is in spasm and peripheral IV access is difficult to establish. In these critical patients the rate of fluid administration must approach that of the bleeding to be of any benefit, and even when bleeding rates are very high (on the order of 75–100 ml/min), the benefit of IV therapy has been demonstrated only for prehospital times exceeding 30 min [6]. These findings have brought about the institution of a "scoop and run" or "load and go" policy for many trauma systems to minimize the time to definitive intervention.

The advent of hypertonic fluid resuscitation has resulted in a reevaluation of the potential benefit of prehospital fluid administration. The concept of hypertonic fluid resuscitation came from the demonstration that 200–300 ml 7.5% sodium chloride solution was as effective as 2 l isotonic fluid administered during the same time period [7]. Animal model studies of fixed volume hemorrhage have shown that significant attenuation of the endocrine response to hemorrhage can be achieved with a fluid resuscitation that restores blood volume but does not restore mean arterial pressure [8]. This may be an especially useful concept if transport times are long. Hypertonic saline resuscitation is based on the redistribution of fluid and electrolytes from the expanded intracellular space to the extracellular space. Nakayama and coworkers demonstrated that resting membrane potential, intracellular water, and intracellular sodium and chloride all return to normal after hypertonic, but not an equivalent isotonic, resuscitation [9]. These transient effects on the cardiac output and mean arterial pressure can be augmented and sustained for 3 h by the addition of colloid in the form of dextran which selectively partitions the "borrowed" intracellular water in the intravascular space. The administration of 4 ml/kg 7.5% NaCl/6% dextran 70 brought about the plasma volume expansion of approximately 10–12 ml/kg [10]. Other effects of hypertonic saline may include the direct improvement of cardiac performance with increased contractility and precapillary dilatation resulting in decreased aortic input impedance [11, 12]. In simulated field resuscitation and transport situations, a bolus of hypertonic saline and dextran restored cardiac output and blood pressure for 30 min and was found to decrease by a factor of 6 the volume of subsequent isotonic lactated Ringer's needed to maintain cardiac output [13]. No benefit is conferred by increasing the hypertonic sodium chloride content above 7.5%; however, increasing the concentration of dextran does improve the hemodynamic responses during resuscitation [14]. The ability to achieve a greater initial increase in cardiac output and more sustained increase in plasma volume with smaller doses of hypertonic saline with increased concentrations of dextran 70 [15] may have even wider applications in military trauma where there is concern about the administration of high salt loads in the setting of preexisting dehydration [16].

The safety of the peripheral administration of these hypertonic and hyperoncotic fluids in doses approximating 4 ml/kg (250 cc) has been studied extensively in animal models and clinical trials in a civilian urban setting [17–19] and in patients undergoing helicopter transport [20, 21]. These trials have shown an

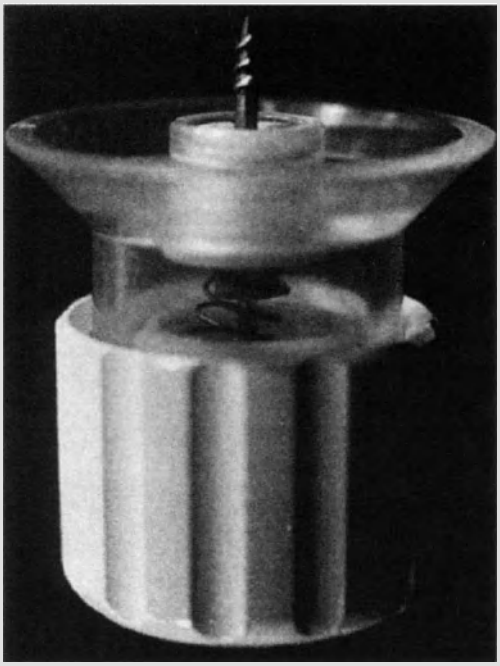

Fig. 1. Sternal needle for hypertonic saline infusion. This needle has a screw tip so that the needle spins freely when the marrow cavity is entered

improved survival rate of patients given hypertonic saline, especially those with severe head injuries [20]. More recent data have brought into question the usefulness of added dextran with its attendant increased expense. Two clinical studies of trauma patients, in both urban and helicopter transport settings, with rapid transport times and aggressive supplemental fluid resuscitation have found no added benefit of the dextran component [22, 23]. The colloid, however, may still provide added benefit in the setting of military or mass casualties in which the prolonged cardiac effects and relatively small volumes become important from a logistic standpoint.

An added attraction of small-volume hypertonic resuscitation is that these fluids may be administered by intraosseous infusion into the marrow cavity, therefore avoiding the difficulty of establishing peripheral intravascular access. The use of a recently developed sternal infusion needle (Fig. 1) provides safe and quick access to the sternal marrow cavity for hypertonic resuscitation even during rough transport, and it provides rapid normalization of blood pressure and cardiac output [24]. The use of hypertonic saline via sternal marrow infusion provides for improved hemodynamic resuscitation of trauma patients while minimizing delays in transportation to definitive care.

Emergency Room Resuscitation

Once the patient arrives in the emergency resuscitation suite, initial assessment follows the guidelines set forth in the Advanced Trauma Life Support Course of

Table 1. Clinical classification of hemorrhagic shock

Mild shock (up to 20% blood volume loss)	
Pathophysiology	Decreased perfusion of nonvital organs and tissues (skin, fat, skeletal muscle, and bone)
Manifestations	Pale, cool skin; patient complains of feeling cold; hypotension if intoxicated with alcohol
Moderate shock (20%–40% blood volume loss)	
Pathophysiology	Decreased perfusion of splanchnic organs
Manifestations	Oliguria, postural hypotension, mild agitation; pregnant patient shows signs of mild shock and fetal distress
Severe shock (40% blood volume loss or greater)	
Pathophysiology	Decreased perfusion of the heart and brain
Manifestations	Restlessness, agitation, coma, cardiac ischemic changes, cardiac arrest

the American College of Surgeons [25]. After establishment of an adequate airway and breathing, the next step is to assess the adequacy of circulation. Shock can be thought of in three general levels: mild, moderate, and severe (Table 1).

In *mild shock* perfusion of vital organs is preserved despite peripheral vasoconstriction. This is manifested by cool extremities, poor capillary refill, and a subjective complaint of feeling cold. Urine output is maintained. Tachycardia may be present; however, the absence of tachycardia should not be taken as reassuring because significant levels of shock may exist with normal heart rates [26].

In *moderate shock* there is impaired perfusion of vital organs except for the heart and brain. Urine output can be a convenient guide in monitoring this level of shock and should be maintained at 0.5 cm kg^{-1} h^{-1} in the adult to assure adequate renal perfusion. However, urine output must be interpreted with caution because it may be misleadingly elevated in patients who are intoxicated, those who have glucosuria, and in those who have previously received hypertonic saline.

Severe shock is manifested by signs of impaired perfusion of the heart and brain, including agitation, restlessness, coma, cardiac dysrhythmias, ischemic changes on electrocardiogram, and ultimately cardiac arrest.

Assessment and treatment of shock proceed simultaneously. Adult patients who manifest shock by any of the above definitions should be given a rapid intravenous infusion of 2–3 l (30 ml/kg) isotonic crystalloid. Venous access can be obtained by a large-bore percutaneous catheter, venous cutdown, or central lines. In the patient suffering from severe shock, saphenous venous cutdown at both ankles provides rapid and reliable access. In an adult the saphenous vein can accept the entire cross-section of intravenous tubing; in a child a large-bore

(12–16 gauge) venous catheter may be used. After the initial bolus infusion, the level of shock should improve, thus allowing placement of percutaneous central lines if deemed necessary.

Fluids should be administered rapidly and should be warmed to 37°C if possible. Commercially available compressor-driven rapid infusors have the capability to provide up to 1 l/min at 37°C. This may be especially important in those patients who have had short prehospital times yet still arrive in deep shock. Rapid reversal of hypovolemia, as well as prevention and perhaps reversal of hypothermia with its attendant development of coagulopathy, expedites definitive surgical intervention [27]. Hypothermia in trauma victims is directly related to the severity of injury and is associated with increased mortality [28].

Isotonic crystalloid solutions are the fluids of choice during initial hospital resuscitation as they achieve restitution of both plasma volume and interstitial volume. Colloid solutions are readily available and are effective volume expanders. However, they are expensive and in the setting of the diffuse microvascular permeability of shock have been shown to be no more effective than isotonic crystalloid and to increase the incidence of pulmonary edema [29]. Colloids are therefore reserved for replacement of abnormally low serum proteins and coagulation factors late in the postinjury course once vascular permeability has been corrected.

Crystalloids with a mild buffering capacity are recommended because of the variable degrees of acidosis accompanying shock. Lactated Ringer's solution or normal saline may be used. Sodium bicarbonate may be added to the saline to compensate for the acid chloride load. Normal saline is preferred if blood is to be administered because the calcium contained in lactated Ringer's may neutralize the citrate in banked blood and allow clotting of the blood in the tubing. Dextrose-containing fluids are to be avoided during resuscitation because of incompatability with transfusion should the same line be used for blood, the worsening of shock-associated hyperglycemia, and the increased risk of inappropriate osmotic diuresis.

In general, the use of blood transfusions is limited to the operating room. Patients who continue to demonstrate hemodynamic instability after initial fluid resuscitation with 2–3 l crystalloid usually have major injuries requiring prompt, operative intervention. Once hemostasis is achieved, blood products may be administered, with preference given to return of shed blood via the use of a reclamation and auto transfusing system. However, the use of blood products is not without risk and may in some cases be harmful. Transfusion of blood products may cause bleeding diatheses, hyperkalemia, acidemia, and pulmonary insufficiency [30]. The risk of viral transmission must also be considered. All patients in shock have increased blood viscosity and therefore may actually benefit from lower hematocrits. Trauma patients, who tend to be otherwise young and healthy, tolerate hemodilution to a hematocrit of 20% quite well as long as vascular volume is maintained [31]. Older patients with limited cardiac reserve tolerate hematocrit values of 30%. Because 50% acute blood volume loss is the point of mortality, all patients who arrive in the emergency room alive have

sufficient red cell mass remaining, so that restoration of blood volume with crystalloid should not result in an hematocrit of less than 20%.

During initial resuscitation the patient should be surveyed to verify the type of shock, which may be thought of as hypovolemic, relatively hypovolemic, or cardiac. Differentiation among these three may be aided by assessment of the neck veins. *Hypovolemic or hemorrhagic shock* is the type most frequently encountered in the trauma patient, and it results from internal or external blood loss. The neck veins are initially flat. Once the source of hemorrhage is controlled, fluid resuscitation is curative. If the neck veins are distended after vigorous initial fluid resuscitation, *cardiac forms of shock* should be considered. These may take the form of cardiogenic shock from coronary artery disease or myocardial contusion, or cardiac compressive shock from tension pneumothorax or pericardial tamponade. Treatment involves either pneumothorax decompression by a tube thoracostomy, pericardiocentesis, or careful cardiac monitoring and pharmacological support to prevent further myocardial damage. Relative hypovolemia or *neurogenic shock* occurs in spinal shock in which the vascular space is expanded relative to the plasma volume. Spinal shock results in "warm" shock, with cutaneous perfusion preserved at the expense of central perfusion. The early stages of spinal shock show none of the manifestations of mild shock except for hypotension. However, a compromise of vital organ perfusion is indicated by minimal urine output. In the latter stages the patient may become hypotensive and manifest the symptoms of hypovolemic shock. In spinal shock pressor drugs may be of value in the initial therapy, but the basis of treatment remains fluid resuscitation with isotonic crystalloid to fill up the expanded vascular volume.

Full resuscitation in the trauma patient with both hypovolemic shock and closed head injury presents a challenge [32]. The goal of resuscitation is still restoration of tissue perfusion; however, aggressive administration of fluid may worsen cerebral perfusion via increased intracranial pressure from cerebral edema. The dilemma in fluid management of these patients is achieving vascular expansion without excessive increases in total body water [33]. In animal models of hemorrhage, both with and without head injury, resuscitation with isotonic fluids causes increases in intracranial pressure (ICP) above baseline [34–36]. Efforts to maintain reduced ICP are warranted because increased ICP following head injury worsens neurological outcome [37]. Osmotic agents such as mannitol have traditionally been used to lower ICP; however, the obligate volume loss from osmotic diuresis may actually lower cerebral perfusion pressure by causing arterial hypotension. Because of its osmotic effects and its ability to restore hemodynamic parameters with very low volumes, hypertonic saline is an attractive alternative in these patients. Using a large animal model of hemorrhagic shock in head injury, Battistella and colleagues [38] demonstrated that hypertonic saline resuscitation abolishes the increases in ICP seen with conventional resuscitation and results in a decrease in ICP compared to baseline. The postulated mechanism of action is dehydration of areas where the blood-brain barrier is intact. Edema in the area of injury is not affected by the type of resuscitation fluid. The same group have also found that cerebral metabolism is

not adversely affected by cellular dehydration caused by hypertonic saline, and further that brain function as measured by the flash evoked potential is preserved until very high levels of serum sodium and osmolarity are reached [39, 40]. These extreme values have not been seen using the hypertonic regimens studied clinically. Freshman and coworkers have demonstrated that 7.5% NaCl is equally effective as 20% mannitol in treating the increased intracranial pressure of a space-occupying lesion while conferring the advantage of rapid cardiovascular resuscitation [41]. As stated earlier in the section on hypertonic prehospital resuscitation, improved survival in patients with head injuries using these regimens has been demonstrated clinically [20]. More clinical studies are now needed to document this improvement in ICP because maximal brain swelling often does not occur until 12–18 h after injury [42].

Operative and Postoperative Shock Resuscitation

Despite emergency room resuscitation and operative intervention, shock may still plague the trauma patient. In these forms of shock increased microvascular permeability results in fluid losses from the intravascular into the "third" space and persistent hypovolemia. Increased permeability may accompany severe trauma or immediately follow resuscitation. This is referred to as "traumatic shock." Experimental evidence from studies of extremity ischemia and crush injuries indicate that diffuse intravascular activation of inflammatory and coagulation cascades takes place with reperfusion [43], and that the target of this intense inflammatory response is the microvascular endothelium which then loses integrity [44]. This state is usually associated with peripheral vasoconstriction and decreased cardiac output. It develops gradually in the first 24 h following injury and is manifest most dramatically in the lung where it is referred to as the adult respiratory distress syndrome (Fig. 2).

When this increased permeability type of shock follows injury by several days, it is usually due to products of bacterial action and is termed septic shock. Characteristically the cardiovascular changes are hyperdynamic, consisting of high cardiac output and low vascular resistance. A source of infection may or may not be identified. Any source of inflammation may create this systemic inflammatory response, including large wounds, toxins or bacteria released from the gut, and secondary infection in the peritoneal cavity or lungs. Crystalloid infusion remains the treatment of choice, as Holcroft and Trunkey have shown that colloids extravasate into the interstitial space and may actually aggravate tissue edema [45]. Even though crystalloid infusion may aggravate peripheral edema, mobilization of this third space fluid can be augmented by the use of diuretics after the vascular system has repaired its integrity (generally indicated by a spontaneous increase in filling pressures, increased urinary output, and negative fluid balance). Therapy for this systemic inflammatory response syndrome is currently supportive and consists of restoration of oxygen delivery and elimination of the source of ongoing inflammation by treatment of infections, débridement of wounds, and stabilization of fractures.

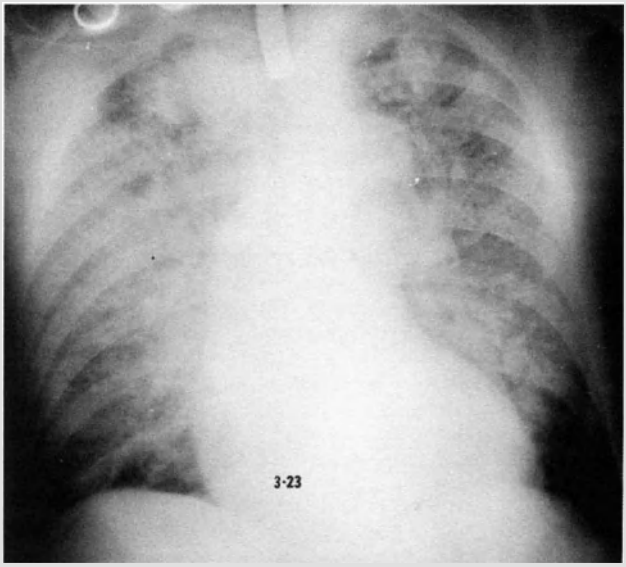

Fig. 2. Chest X-ray in a patient with the respiratory distress syndrome. Diffuse reticular infiltrates are characteristic of the initial parenchymal changes

The prognosis for morbidity and survival following trauma relates to the amount of third space fluid losses that take place during the first 24 h after injury. In a study by Vassar et al. [46] those trauma patients who had fluid retention of 3 l or more had a higher risk of pulmonary failure and subsequent death. It is felt that fluid requirements reflect the severity of the initial shock, and that this determines subsequent morbidity and mortality. This concept has been used as the basis for an ICU point system for prediction of outcome in trauma patients [47].

Conclusion

In conclusion, low volume hypertonic saline shows promise for resuscitation of trauma patients in the field and can be given by sternal marrow infusion even under the most adverse conditions of transport. Hypertonic solutions also are beneficial in the initial resuscitation of patients with concomitant head injuries. Warmed, isotonic crystalloid then plays the primary role in hospital management of shock resuscitation because of ready availability, safety, and proven capability of vascular and interstitial expansion. Transfusion of blood for trauma is individualized for each patient but in general is used when hematocrit values drop below 20%–30%, depending upon the underlying health and reserve of the patient. Colloids are used for specific indications of replenishment of coagulation factors or serum proteins and are not used primarily for resuscitation. The fluid requirements of trauma patients reflect the severity of the initial insult and can be used to predict survival.

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End-Points of Resuscitation

J.-L. Vincent and P. Manikis

Introduction

Polytrauma is the leading cause of death during the first four decades of life. It is also a major cause of morbidity, requiring prolonged hospital care and entailing elevated treatment costs. Improvements in emergency transportation have increased the number of survivors on arrival at hospital, while emergency resuscitation and advanced surgical techniques, aided and directed by modern imaging processes, have reduced early mortality. There remains, however, a significant proportion of patients who suffer multiple organ dysfunction syndrome (MODS) at a later stage, and unfortunately many of these patients die. Improved early resuscitation techniques, including more aggressive practices, could conceivably help to prevent morbidity and mortality in such circumstances.

Traumatic shock is characterized by a reduction in blood volume, resulting initially from a decrease in cardiac output followed promptly by a diminution in arterial blood pressure (ABP). These alterations provoke a sympathetic response, leading to peripheral vasoconstriction, increased heart rate, and decreased myocardial contractility. The subsequent redistribution of blood flow to vital organs results in reduced skin perfusion and decreased urinary output. Hence, assessment of adequate resuscitation is often based upon these parameters.

There are, however, three important limitations to this practice. First, each of these parameters lacks both in sensitivity and specificity. Second, even when global parameters have been restored to within acceptable limits, the possibility exists that resuscitation is incomplete at the microvascular level [1]. Indeed, resuscitation may restore, but does not necessarily maintain, hepatic function [2]. More recently, Wang and coworkers described a murine model of traumatic hemorrhage, in which resuscitation did not restore cardiac output or blood flow to the brain and skeletal muscle, and thus was associated with late mortality [3]. In addition, altered tissue perfusion may persist despite normalization of traditional parameters. Third, trauma is associated not only with hypovolemia but also with the release of a number of mediators, such as tumor necrosis factor-α and oxygen free radicals. Some of these mediators are also associated with sepsis, so that a "sepsislike" response may be observed even in the absence of infection. This inflammatory response may include moderate hypotension and vasodilation and also some maldistribution of blood flow, leading to secondary hypovolemia with reduced venous return. Meanwhile, these media-

tors further impair myocardial function and therefore complicate the resuscitation process.

Fluid administration is sometimes limited by fear of inducing pulmonary edema. Fluid overload should obviously be avoided; and cardiovascular monitoring is therefore advisable in complicated cases. Nevertheless, posttraumatic pulmonary edema is related more to sepsis than to fluid overload [4], and, importantly, elevated blood flow may help to reduce the septic response [5]. Moreover, patients do not usually die from respiratory failure but rather from MODS. The primary mechanism in organ damage is severe reduction in oxygen delivery, the effects being most detrimental when this reduction is prolonged. For these reasons maintenance of oxygen delivery should receive the highest of priorities.

Clearly, traditional assessment of posttraumatic resuscitation is often based on parameters which may be misleading since they can be influenced by more complicated factors. Informed reinterpretation of these parameters is necessary, taking into account their complex interactions, if early resuscitation is to attain satisfactory endpoints.

The following list summarizes the traditional parameters used in resuscitation, together with more advanced parameters and indices of oxygen extraction which may help to provide more information about the level of resuscitation:

- *Traditional parameters*
  - Arterial blood pressure
  - Heart rate
  - Central venous pressure
  - Urinary output
  - Skin perfusion
  - Hematocrit
- *More advanced hemodynamic parameters*
  - Cardiac output, DO_2, VO_2
  - SvO_2 and oxygen extraction
- *Indices of tissue oxygen availability*
  - Base deficit
  - Blood lactate
  - Gastric intramucosal pH (in conjunction with the above)

Traditional Parameters

Traditional methods of resuscitation usually rely on monitoring the following parameters: ABP, heart rate, central venous pressure, urinary output, and various other clinical indicators, including skin perfusion. Hematocrit levels are also surveyed to assess the degree of blood loss [6, 7]. Such an evaluation is unsatisfactory because none of these factors is sufficiently sensitive or specific. In particular, each may each be influenced by other factors.

Arterial Blood Pressure. ABP remains normal until bleeding is severe, usually exceeding 25 ml/kg [8]. Although persistent hypotension is a strong element in incomplete resuscitation, a precise level is difficult to define for all patients since some may have relatively low ABP under normal conditions. More importantly, the maintenance of ABP within acceptable limits does not rule out incomplete resuscitation, for regional underperfusion may remain despite normal ABP. Orthostatic hypotension may provide a more subtle indication of hypovolemia since it can be detected by monitoring ABP in both the supine and sitting positions. This is not common practice, however, as it is difficult to perform in polytraumatized patients. Hence, resuscitation cannot be monitored reliably by ABP alone.

Heart Rate. Tachycardia may indicate hypovolemia but can also result from low hemoglobin levels, the stress response (through the influence of catecholamines), fever, etc. Furthermore, some patients – particularly the elderly – may not develop tachycardia despite hypovolemia. Wo et al. [9] have demonstrated the lack of correlation between measurements of ABP, heart rate, and cardiac output during emergency resuscitation.

Central Venous Pressure. Cardiac filling pressures are not reliable indicators of volume status as they are affected not only by blood volume but also by cardiac performance, vascular compliance, and the ventricular pressure/volume relationship. In addition, a low cardiac filling pressure can be compatible with either hypovolemia or normovolemia. Central venous pressure (CVP) is particularly unreliable since a low value may be associated with a high left filling pressure in patients with left ventricular dysfunction. Alternatively, high CVP may indicate pulmonary hypertension rather than hypervolemia. Obviously, the CVP may also rise sharply in the presence of acute conditions such as tension pneumothorax, cardiac tamponade, or myocardial contusion.

Urinary Output. Renal failure can reduce urinary output, even when the volume status is acceptable. Urinary output is therefore an unreliable parameter, particularly when the risk of organ failure is high.

Other Clinical Signs. Other clinical signs, such as mental status, are unreliable. For example, mental status may be altered by head injury or administration of sedative or analgesic drugs. Also, skin perfusion is difficult to evaluate satisfactorily.

Hematocrit. As a measure of the relationship between red blood cells and plasma, the hematocrit is an important parameter in assessing transfusion requirements but is a poor predictor of total blood volume. Therefore the hematocrit is unsatisfactory in the determination of fluid needs. Evidently, effective direct assessment of tissue oxygen availability calls for the identification of more specific and reliable parameters than those traditionally used.

Oxygen Delivery

Oxygen delivery (DO_2) must be sufficient to meet oxygen requirements (VO_2). Importantly, VO_2 increases in trauma due to the stress and inflammatory response. Several studies have shown that survivors from trauma have superior DO_2 than nonsurvivors [10–12]. Moore et al. studied 39 polytrauma patients according to a protocol designed to increase DO_2 to greater than 600 ml min^{-1} m^{-2} with the aim of achieving a VO_2 of greater than 150 ml min^{-1} m^{-2}. Fifteen (38%) of these patients did not attain the prescribed $\dot{V}O_2$ by 12 h. Interestingly, these so-called nonresponders had equivalent or better trauma scores (e.g., Revised Trauma Score; Injury Severity Score, ISS) than the responders, but their low $\dot{V}O_2$ and higher mean lactate levels (3.4 ± 0.5 mmol/l vs. 2.4 ± 0.2 mmol/l) proved significant independent predictors of MODS [12].

Bishop et al. investigated 90 polytrauma patients and found that survivors had higher values of cardiac index, DO_2, and $\dot{V}O_2$ than nonsurvivors [13]. Notably, their findings indicate that a time factor needs to be taken into account since patients who attained supranormal levels within 24 h of hospitalization were more likely to survive than those who did not. There were no significant differences in trauma scores that could otherwise account for this observation.

Invasive hemodynamic monitoring is not needed in all patients, but it may be helpful in complex cases, especially in elderly patients and patients with compromised hemodynamic status. In a study of 60 multiply injured patients aged over 65 years, Scalea et al. found that initial blood pressure of less than 150 mmHg, multiple fractures, acidosis, and head injuries were reliable predictors of mortality [14]. In a subsequent study the same team used invasive monitoring in 15 patients suffering from one or more of these factors. At this stage the mean time from emergency department admission to intensive care monitoring was 5.5 h and overall mortality was 7%, with a high incidence of MODS. The researchers suspected that organ failure could be attributed to a prolonged low flow state and consequently reduced the mean monitoring time to 2.2 h. Overall survival was thus increased to 53%. These findings support the theory that an extended period of reduced oxygen consumption results in an oxygen deficit which patients with compromised cardiac performance find difficult to restore subsequently.

Scalea et al. noted that elderly patients are at particular risk in these circumstances, and that aggressive and early intervention can significantly increase their chances of survival [14]. Although the elderly are often associated with poor cardiovascular reserve, young patients can be at risk as well. Abhou-Khahil et al. studied 39 patients aged under 40 years who had penetrating trauma and received more than 6 U blood intraoperatively [15]. They showed that survival was more likely when hemodynamic status was stabilized within the first 24 h of hospitalization. Stabilization was defined as $\dot{V}O_2/DO_2$ independence with normal lactate levels. These authors also advocated early invasive monitoring to establish the individual's requirements.

Optimally, one would increase DO_2 until $\dot{V}O_2$ were stabilized [15], but this frequently is an unrealistic goal, because of difficulties in regulating DO_2 over a wide range. Moreover, the nature of many interventions is such that $\dot{V}O_2$

increases correspondingly, so that no plateau in the $\dot{V}O_2/DO_2$ relation is ever reached.

Oxygen extraction may be a particularly interesting parameter to monitor since this reflects the balance between $\dot{V}O_2$ and DO_2. In the study by Abou-Khalil et al. survivors initially had higher mixed venous oxygen saturation (SvO_2) than nonsurvivors ($73\% \pm 3\%$ vs. $63\% \pm 4\%$, $p < 0.03$) [15]. Further studies are needed to define the O_2 extraction ratio, commensurate with survival in polytrauma.

Lactate Levels and Base Excess/Deficit

As early as 1959 Crowell et al. incriminated oxygen debt as the common determinant of irreversible hemorrhagic shock [16]. Lactate levels and base excess/deficit have been shown to correlate with the degree of oxygen debt and can therefore be used to monitor the degree of tissue hypoxia. During hemorrhagic shock in dogs lactate and base deficit levels offered the best prediction and most accurate estimation of oxygen debt [17]. Also in acute hemorrhage in dogs, the severity of hemorrhage is best reflected by base deficit levels [8].

The importance of hyperlactatemia as a predictor of mortality following shock due to various causes has been well established [18–20]. Serial measurements of lactate levels are correlated with the severity of organ failure following sepsis [21]. Patients' admission levels of base deficit and blood lactate are highly significant predictors of death following trauma [20]. Although base deficit has proven the most reliable of the two parameters, its interpretation may be more difficult in the presence of renal failure [20]. Rutherford et al. also stressed that base deficit can represent a sensitive measure of the degree and the duration of inadequate perfusion [23]. Interestingly, base excess and lactate levels do not change rapidly, so that the time scale of their measurement has some clinical value. Lactate levels,

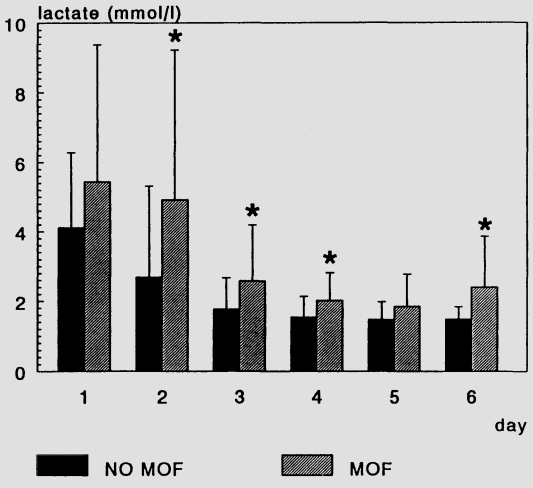

Fig. 1. Mean lactate levels (± SD) of 51 patients with multiple trauma divided into those with ($n = 18$) and without ($n = 33$) MOF (* $p < 0.05$). (From [25] with permission)

in particular, take time to return to normal and this normalization period provides important additional information about the response to fluid resuscitation [21, 24, 25].

Roumen et al. studied the time course of blood lactate in 56 multiple trauma patients and evaluated several scoring systems [25]. Interestingly, the APACHE II score was not correlated with subsequent acute respiratory distress syndrome (ARDS) and MODS nor with lactate levels. Indeed, Polytrauma Score (PTS), Sepsis Severity Score (SSS), and ISS were the only scoring systems predictive of ARDS, while PTS and ISS were the only systems predictive of MODS. It was discovered that lactate levels were higher and remained higher in patients who developed ARDS and organ failure (Fig. 1). Therefore these researchers concluded that a rapid and aggressive early resuscitation should be employed to reduce hypoxic tissue injury. These results agree with those of Moore et al. who found that patients with MODS had higher lactate levels [12]. Recently, Manikis et al. confirmed and extended these findings by showing that lactate levels correlated with the development of organ failure and also with survival [26]. Moreover, they found that in addition to the initial and the highest lactate levels, the duration of hyperlactatemia was also correlated with outcome.

Other Biological Indicators?

The anatomic structure of gastric mucosa is at particular risk of ischemia since the splanchnic blood flow is sacrificed earlier than other regions when cardiac output is diminished. Therefore monitoring of gastric intramucosal pH (pH_i) may provide a good indication of the presence of splanchnic ischemia. It may be particularly attractive to combine pH_i and lactate measurements [27]. Measurements of bowel tissue oxygen tension may be extremely valuable, but they are clinically not available [28].

Another suggested biological indicator is the acetoacetate to hydroxybutyrate ratio, which may be correlated with the mitochondrial redox state. These measurements are more complicated than those discussed previously and are not necessarily more reliable [17].

The Problem of Uncontrolled Hemorrhage

Importantly, most of the previously discussed parameters have been investigated during controlled hemorrhage. Several studies have stressed the potential harm of aggressive fluid resuscitation before the source of hemorrhage is controlled [29–31]. In particular, massive blood loading may lead to dilution of coagulant factors and hypothermia and may thus inhibit clot formation [30]. Therefore, despite the many sound arguments in favor of early and aggressive resuscitation, it is probably unwise to try to obtain supranormal levels of DO_2 rapidly before hemorrhage has been controlled. To do so may result in harmful consequences by actually increasing the bleeding.

Conclusion

Many experimental and clinical studies strongly suggest that an aggressive and more complete resuscitation may reduce morbidity and mortality in polytrauma. In particular, higher DO_2 and $\dot{V}O_2$ values have been associated with lower incidences of MODS, ARDS, and mortality. Some investigators have recommended the maintenance of supranormal cardiac indices and DO_2 values. The limitation of this approach is that O_2 needs cannot be defined easily, so that this may lead to excessive administration of fluids and vasoactive agents. It is probably better to use an index of oxygen metabolism such as blood lactate levels and base deficit as a guide. Base deficit is currently the best indicator of resuscitation in the absence of intricate abnormalities such as renal failure. Indeed, analysis of base deficit may even surpass lactate measurements in its predictive capabilities, but it should be remembered that lactate is the more reliable one in more complex scenarios.

The total duration of the hypoxic period is a crucial determinant of organ failure. Many studies have indicated that the longer the resuscitation time, the higher is the likelihood of development of organ failure. Hence, resuscitation should be aggressive and prompt, a corollary being that hemorrhage must be controlled first. Finally, repeated evaluations are recommended. In particular, the time course of lactate levels has been shown to be very useful.

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Standard Diagnostic Workup of the Severely Traumatized Patient

L. P. H. Leenen and R. J. A. Goris

Introduction

The diagnostic workup of severely traumatized patients has evolved rapidly in recent years, with progressive integration of diagnostic and therapeutic procedures. This chapter addresses the clinical examination, additional diagnostic laboratory investigations, and imaging techniques used in evaluating severely traumatized patients.

Clinical Examination

Despite the rapid development of new diagnostic techniques and procedures in medicine clinical examination is still of primary importance during the first hour after injury. The Committee on Trauma of the American College of Surgeons has developed a protocol in which the same sequence of examinations is to take place in every trauma patient, whether stable or unstable. This protocol has evolved into the Advanced Trauma and Life Support (ATLS) course, which addresses the workup of the trauma patient in the first hour [1]. Primarily ATLS addresses vital functions to provide adequate oxygen delivery to vital organs [1]. The factors of airway, breathing, and circulation (ABC) are assessed systematically, and diagnostic procedures and resuscitation are integrated. Towards the end of the survey further action is prescribed by the nature of the injuries. Various tasks are performed simultaneously by the trauma team. The tasks and responsibilities of the various team members should be outlined before the patient arrives [2] (Table 1).

Primary Survey

The primary survey addresses vital functions and includes a quick examination of the patient. Any problem encountered during the primary survey should be solved immediately rather than proceeding to the next step of the survey (e.g., the airway should be patent before measuring blood pressure). The sequence of care given the injured person during the first hour is:

Table 1. Example of tasks in an eight-member trauma team

	Main tasks	First 10 min	After first 10 min
Trauma surgeon (T1)	General management Coordination Trauma scores	Primary survey Secondary survey Estimate blood loss Order blood Indicate imaging sequence	Secondary survey Indicate consultants Assess X-rays with R Guide diagnostics Guide treatment
Senior resident (T2)	Surgical procedures	Perform cricothyroidotomy Insert thoracic drain(s) Insert two IVs, venous cutdown	Peritoneal lavage Stays with patient
Junior resident (T3)	Documentation Immobilization	Immobilize C-spine 35 ml art. blood sample Blood gas Serum for cross-matching	Emergency dressings Splinting fractures Documentation Phone consultants, OR, ICU
Trauma nurse 1 (N1)	Assist T2	Obtain blood pressure Assist T2	Assist T2 Prevent hypothermia Stays with patient
Trauma nurse 2 (N2)	"Shuttle"	Handle, dispatch blood samples Get O neg blood Remove clothing	Obtain identification Gather/secure rings, prostesis Get cross-matched blood Dispatch further blood samples
Anesthesiologist (A1)	ABC	Free airway, intubation Bronchial suction Ventilation	Gastric tube Arterial line CVP line Sedation and analgesia Monitor fluid infusion
Anesthesia aid (A2)	Assist A1	Assist intubation Prepare medication Hook-up monitoring	Assist A1 Obtain further blood samples
Radiologist (R)	Imaging	Lateral C-spine X-thorax X-pelvis	Obtain, analyze further imaging

- *Primary care*
  - *A*irway maintenance and cervical spine control
  - *B*reathing and ventilation
  - *C*irculation with hemorrhage control
  - *D*ysfunction of the central nervous system
  - *E*xposure of the patient

- *Resuscitation phase*
- *Secondary survey*
- *Definitive management*

Airway and Cervical Spine Control. In every trauma patient, especially after blunt injury, the cervical spine is assumed to be damaged unless proven otherwise. The cervical spine is stabilized manually or with a stiff cervical collar. The collar should match the patient's neck and must allow visualization of the trachea and cervical veins. Hyperextension of the neck must be avoided.

The patient is talked to. When the patient replies coherently and is talking with a normal voice, a patent airway and good cerebral perfusion can be assumed. When not, the airway should be examined. The mouth is opened and cleared of debris and/or vomit, preferably with a rigid aspiration device and a Magill forceps. A chin lift maneuver is performed to clear the tongue from the airway, and this position is maintained with a nasopharyngeal airway. Maxillofacial injuries, obstructing the airway, should be dealt with by jaw thrust, pulling away the fractured parts, and by early intubation or a cricothyroidotomy.

When there is no gag reflex, the airway should be protected with a cuffed orotracheal tube. During intubation the cervical spine should be kept in line manually by an assistant. The method of airway management depends on the clinical situation; in most cases orotracheal intubation suffices. In agitated patients succinylcholine can be used for paralysis [3]. The position of the tube is evaluated with a chest X-ray. Multiply injured patients should recieve 100% oxygen, 6 l/min with a nasal canula or 12 l/min with an oxygen mask [4].

Evaluation of the cervical spine is essential during the early assessment. In the unstable patient a cross-table lateral cervical spine film and clinical judgment guide the initial airway management [5]. However, in the stable patient extensive evaluation of the spine should be performed (see below).

Breathing. Free the thorax of clothing, and inspect both sides. Evaluate the movements of the thorax and be sure that both sides ventilate adequately. If one side is fixed in maximal inspiration, a tension pneumothorax or massive hemothorax is present. Notice paradoxical respiratory movements of the chest wall and of the sternal area, indicating flail chest. Dissociation between thoracic and abdominal breathing is an important sign, indicating the presence of a high cervical spinal cord injury. Active expiration using the abdominal muscles (auto-PEEP) may indicate excessive intrathoracic pressure, from pneumothorax or from elevation of the diaphragm by increased intra-abdominal volume, for example, by blood. Auscultate both sides for breath sounds and count the respiratory rate. Clues to hypoventilation are diminished breath sounds and decreased or asymmetric chest wall excursions. Causes of hypoventilation are upper airway obstruction, leaking face mask, head injury, spinal cord transsection, flail chest, open thoracic wounds, (tension) pneumothorax, hemothorax, intubation into the main brochus, and lung contusion.

Table 2. Rough estimate of expected blood loss due to fractures

Area	% blood volume	Liters in adults
Pelvis	20–100	1–5
Femur	20–50	1–2.5
Spine	10–30	0.5–1.5
Tibia, humerus	10–30	0.5–1.5
Midfoot, ankel	5–10	0.2–0.5
Radius, ulna	5–10	0.2–0.5
Rib	2–4	0.1–0.2

Compound fracture: add 50% of expected blood loss for each.

Circulation. After establishing airway patency and ventilation, optimalization of the circulation should be addressed. As hypovolemia is the most likely reason for cardiovascular collapse in the trauma patient, institution of volume infusion is the next step. Two large-bore (14-gauge) cathethers are placed in the antecubital veins. In cases of evident hypovolemia 1 l crystalloids is infused rapidly.

When peripheral veins are collapsed, a venous cutdown is performed, preferably in the saphenous vein at the ankle, and a large bore catheter introduced. Central venous catheters are initially avoided because of the lack of sterile conditions in the shock room and because of possible complications induced by their insertion. Also, it should be realized that the volume infused per unit of time depends on the diameter and length of the cathether, not on the diameter of the vein [1].

Blood pressure can be rapidly assessed by clinical parameters as skin color, carotid pulse (systolic pressure > 60 mmHg), femoral pulse (systolic pressure > 70 mmHg), and radial pulse (systolic pressure > 80 mmHg) [3].

The expected amount of blood loss should be estimated roughly, depending on the fractures present (Table 2) and cross-matched blood ordered accordingly.

Disability. Central nervous system functions are assessed by examination of the pupils and the response to vocal and painful stimuli. The Glasgow Coma Scale is applied and the score recorded in the secondary survey. Changes in the patient's responsiveness require a thorough reevaluation of oxygenation, ventilation, and circulation.

Exposure. The primary survey ends with obtaining full exposure of the patient by removing all clothes. Thereby the patient should be moved as little as possible, which is best done by cutting off the clothes with a pair of large scissors. Take notice of any bruises, ecchymoses, and other skin marks (e.g., from seat belts), indicating internal injury. Hypothermia is avoided by covering the patient with blankets and/or a warming mattress.

Secondary Survey

After completion of the primary survey, which assumes the patient is stabilized, a secondary survey is performed. The secondary survey consists of a detailed physical examination, according to a standardized protocol, from scalp to toe, performed by one of the members of the team. During this examination the ABC and neurological status should be reevaluated at regular intervals and the results recorded. Deterioration of the neurological status is not necessarily due to brain damage but can also be caused by hypoxemia and/or hypovolemia.

Head, Neck, and Upper Extremity. The head is examined first. The eyes are evaluated for penetrating or blunt trauma. Pupilary function and corneal reflexes give important clues in the unconscious patient. Bilateral myosis indicates drug intoxication. Unilateral mydriasis indicates an intracranial mass lesion. Bilateral mydriasis in a normoxic patient indicates brainstem contusion or decerebration. Vision is evaluated by investigating eye movement by the confrontation method. The fundi should be examined for bleeding or edema. The eye should be evaluated early, as edema frequently inhibits a proper survey in a later stage.

The responsive patient is asked to close the eyes firmly, and subsequently to show the teeth (facial nerve). The scalp is palpated to search for fractures and major skin lacerations. The face is palpated symmetrically for deformities. Especially, the orbital rims and the jugular bones are palpated.

The responsive patient is asked to clench the teeth and to feel whether teeth occlusion is normal. If this is not the case, a fracture of the maxilla and/or mandibula is present. The upper incisors are grasped between the tumb and the index and middle finger and moved back and forth to detect midface injuries, precluding nasogastric intubation.

The cervical spine should be assumed fractured and be protected until adequate X-rays have shown otherwise. No neck pain and the absence of paresthesia in the upper extremities and of neurological deficit rules out cervical problems; however, other injuries may mask a cervical spine problem. Look for bruises in the cervical area, for deformity, tracheal displacement, and distended veins, clues for cervical and thoracic injury. Penetrating wounds should not be probed but thoroughly evaluated.

Ask the responsive patient to raise the right or left arm. If this is possible, ask him to squeeze your hands firmly. Ability to execute these commands properly without pain demonstrates the anatomical and functional integrity of the major structures of the upper extremities. Paresis/paralysis of the elbow flexors indicates spine injury at the level of C5, of wrist flexors at C6, of elbow extensors at C7, of finger flexors at C8, of finger spreading at T1. Assess the radial pulses on both sides.

Thorax. The chest is inspected visually; hemorrhage and bruises give clues to hidden pathology. Rotate the patient gently to inspect the posterior aspect of the thorax and back, as, for example, a stab-wound may otherwise go undetected. The clavicles, ribs, and sternum are palpated. Subcutaneous emphysema is noted. Ask

the responsive patient to take a deep breath. Look (again) for symmetry of thoracic wall movements and abnormal motion. Pain indicates the presence of rib or sternal fractures. Gently compress the thorax anteroposteriorly and laterally. This is painful in the presence of fractured ribs and/or sternum. Upon auscultation, a pneumo- or hemothorax is indicated by the presence of diminished breath sounds, anteriorly or posteriorly, respectively. Crepitation may indicate a lung contusion. Cardiac tamponade can be assumed when finding distant heart sounds, distended cervical veins, and a narrow pulse pressure, although not all criteria may be present.

Abdomen. The abdomen is truly the "black box" of the trauma patient. The patient may be irresponsive, while in the responsive patient severe pain from other injuries may mask the pain due to intra-abdominal injury. Intra-abdominal blood or leaking bowel contents are isotonic and produce few abdominal signs and symptoms in the first hours after injury. A "hollow viscus" perforation thus becomes apparent only after the onset of peritonitis after several hours and hollow viscus ischemia only after 1–2 days. Clearing the abdomen is an absolute must in the trauma patient and should include abdominal lavage, echography or computed tomography upon the slightest suspicion.

The abdomen is visually inspected for ecchymoses and bruises. In car occupants bruises due to seat-belt injury should be especially looked for, as they indicate severe compression of the abdominal contents, with possible laceration of the liver, mesentery or bowel, or even a tear of the intima of the abdominal aorta resulting in occlusion or dissection. Contrary to ancillary practice, an increase in abdominal girth is a poor indicator of intra-abdominal bleeding. Indeed, half of the circulating blood volume is required to increase abdominal girth by 2 cm.

Gently palpate the abdomen in search of tenderness, muscular guarding, or abnormal swelling. Palpate both kidneys in search of a perirenal hematoma. If upon percussion the Traube space is obtunded, a splenic laceration is likely to be present. Gently compress the pelvis anterioposteriorly and laterally. If pain is induced and/or abnormal motion present, a pelvic ring fracture is probable. In this case, ordering 10 U more of cross-matched blood is appropriate.

Inspect the urethral orifice. If blood is noticed, urethral catheterization is contraindicated as an urethral rupture is present, and an urethrography is ordered. The perineum is inspected for hematoma and lacerations, and sensitivity of the perineal area assessed. A (vaginal and) rectal examination is performed, assessing the tonus of the anal sfincter and the presence of vaginal and/or rectal lacerations. In male patients a high-riding prostate indicates the presence of an urethral rupture.

The Lower Extremities. Inspect the lower extremities for abnormal position or movements, lacerations, and soft tissue injuries. Ask the reponsive patient whether he can move, and then to lift the right respectively left leg. If this can be performed, the major functions and structures of the lower extremities are intact. In this case gently lift each leg from the examination table and tap gently on the

heel. More subtle fractures can thus be located by the pain induced by axial compression. Paresis/paralysis of the hip flexors indicates a spine-injury at the level of L2, of knee extensors at L3, of foot dorsiflexion at L4, of great toe extension at L5, of foot plantar flexion at S1. Assess the sensitivity of both lower extremities, especially in the area of the peroneal nerves. Palpate the arterial pulses in the groins, of the tibial and posterior tibial arteries, and the arteria dorsalis pedis.

Medical History

A simple list of relevant items should be obtained from the patient, his relatives, or ambulance crew. The presence of allergies (A), medication (M), a past medical history (P), last meal (L), and events leading to the injury (E) should be asked for (AMPLE) [1]. After completion of this secondary survey, which should take only a few minutes, further radiological studies relevant to the secondary survey can be obtained.

Standard Laboratory Investigations

Upon admission blood is obtained for arterial blood gas analysis, blood group, and rhesus determination, for cross-matching, hemoglobin, platelets, blood coagulation testing. In elderly patients tests for blood glucose, blood urea nitrogen, and electrolytes are also ordered.

Standard Roentgenological and Ultrasound Workup

Radiological and echographic evaluations of the trauma patient quickly give important clues on the different injuries and the management of the patient. However, obtaining these images should not interfere with resuscitation and appropriate surgery.

In the unstable patient the following X-ray images are obtained:

(a) cross-table cervical spine – 98% of all neck injuries are diagnosed by cross-table cervical spine X-ray examination;
(b) AP thorax, as soon as possible, an AP X-ray of the thorax is obtained – this X-ray serves as a baseline for comparison with later thoracic X-rays;
(c) AP pelvis.

In stable patients, in addition to the above X-rays, more extensive imaging with X-ray, computed tomography, echography, or magnetic resonance imaging is warranted, depending on the findings of the secundary survey. These further diagnostic procedures are outlined by Heijstraten, Glaser et al., Childs and Blaisdell, and Rommens "Diagnostic Procedures in Spine, Pelvic,and Extremity Injuries" (this volume).

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Radiology in Chest Trauma Patients

F. M. J. Heijstraten

Introduction

Trauma caused by motor vehicle accidents is the leading cause of death among persons under the age of 40 years [1]. Thoracic injuries account either directly or indirectly for approximately 50% of these deaths. On the other hand, serious injury to the thoracic contents can exist while the clinical symptoms are mild or even absent. For these reasons a careful radiological investigation of the chest is important in all patients with known chest trauma and in those involved in serious accidents, as most of the significant injuries are fortunately demonstrated during the radiological investigation.

The conventional chest film remains the most important imaging technique for the initial evaluation and follow-up of patients with trauma to the chest, while computed tomography (CT), sonography, and angiography can add important information in some patients.

Radiological Equipment

Modern emergency X-ray examination rooms should be equipped with C or U arm equipment with a large radius and a floating or multidirectionally movable table top, allowing for simultaneous radiological procedures and resuscitation maneuvers. Plain radiographs should be made with grids to optimize quality. A large field image intensifier with a spotfilm and/or digital subtraction facility is required for adequate radiological assessment of the trauma patient [2]. Angiography of the thoracic vessels can be performed without moving the patient to another room. Ultrasound (US) equipment is now considered essential in the radiological emergency room. In chest examination US is used primarily to look for the presence of pleural or pericardial effusions and dissection of the descending aorta. A CT scanner should optimally be located adjacent to the trauma room. CT should be performed whenever a mediastinal lesion or hemorrhage is suspected [3].

It is essential that a radiologist be part of the trauma team and "on the spot" to evaluate the radiological findings in chest trauma patients. The sequence of radiological examinations is determined in close cooperation with the other members of the trauma team. The radiologist follows the imaging, evaluating films, guiding supplementary views, and interpreting new findings. The first and

often only examination of the chest trauma patient is a plain chest X-ray. More sophisticated X-ray examinations are performed as a consequence of findings on the chest X-ray and/or the mechanism of the injury.

Trauma to the Thoracic Spine

Spinal injury is frequently seen in high velocity trauma. Unstable fractures are occasionally present without causing neurological damage at the onset [4]. It is imperative that such fractures be detected before neurological damage ensues. It is recommended that lateral and frontal films of sufficient penetration be obtained in all severely injured patients to visualize the vertebral bodies and the neural arch. Careful attention should be paid to the vertical axial alignments, interpedicular distances and paravertebral soft tissues. Frontal views and CT scans can be obtained as required. Thoracic spinal injury may be associated with mediastinal hemorrhage, particularly in the superior mediastinum. Indeed, mediastinal hemorrhage from small vessels may be seen as often with spinal injury as with damage to the aorta or great vessels [5].

Trauma to the Chest Wall

Trauma to the chest wall occurs in 40% of all patients with blunt chest trauma. Fractured ribs, claviculae, and scapulae are the most common causes of pleural and lung damage. Fractured first and second ribs are not often combined with other rib fractures; they are often associated with a laceration of the airway or vascular injury [6]. Fractures of the lower ribs should increase suspicion of splenic, hepatic or renal injury, confirmation of which should be sought by appropriate investigations such as sonography or CT. Flail chest is the most important chest wall injury caused by blunt injury. It occurs when there are three or more double rib fractures that cause a segment of the chest wall to become abnormally mobile and allow paradoxical movement during inspiration.

Combined nerve and vascular injury often occurs in the case of scapulothoracic dissociation. In such cases emergency angiography is required before surgery to determine the location of the arterial injury (Fig. 1) [7]. Sterno-clavicular dislocation may be of great significance if the medial end of the clavicle is displaced posteriorly and impinges on the trachea and the vessels in the root of the neck. This is best seen with CT [8].

The presence of air in the soft tissues may accompany laceration of the parietal pleura and is often associated with a pneumothorax. Rib fractures (3–9) can be identified with conventional chest X-rays; lower rib fractures (10–12) can often be diagnosed better on an abdominal scout film. The possibility of laceration of the underlying lung or liver and spleen by a rib fragment must always be kept in mind.

Sternal fractures (often best seen on CT) can create large anterior mediastinal hematoma's by laceration of internal mammary vessels (Fig. 2). The possibility of an underlying myocardial or pericardial injury is high.

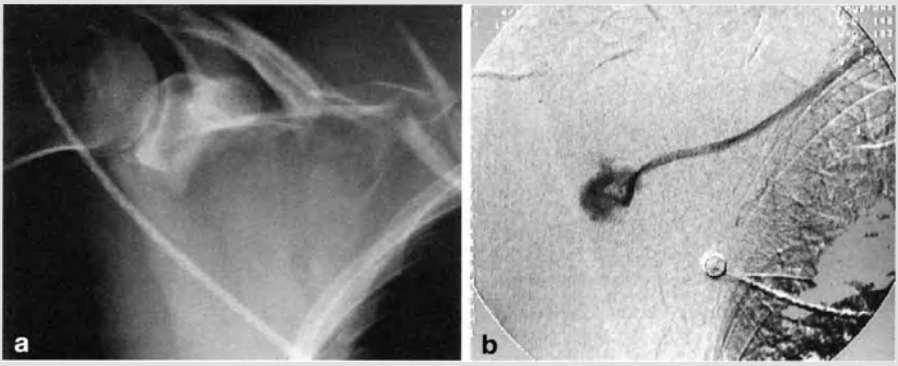

Fig. 1a,b. Scapulothoracic dislocation. At angiography dissection of the subclavian artery was found

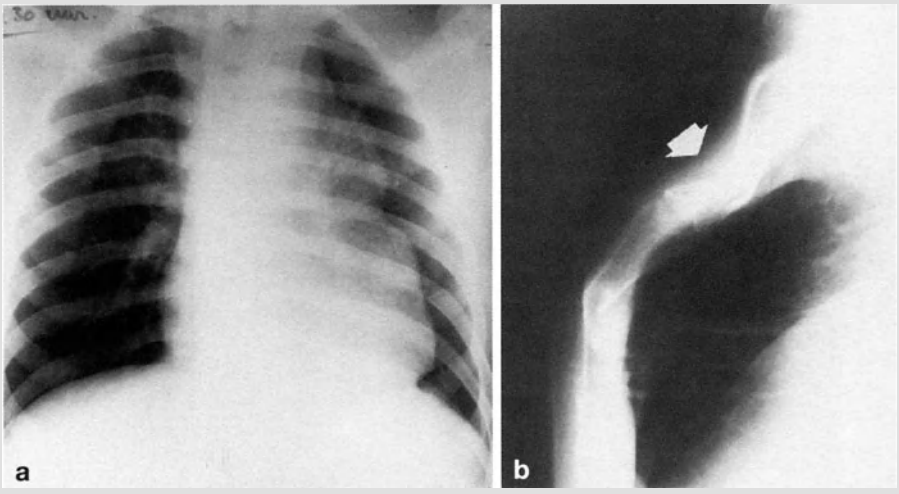

Fig. 2a,b. Anterior mediastinal hematoma. **a** Large anterior mediastinal hematoma, with typical undulating contour. The aortic arch was normal at angiography. **b** Lateral chest X-ray shows sternal fracture (*arrow*)

The Pleural Cavity

Pneumothorax is the second most common injury following chest trauma, occurring in up to 40% of patients. A tear of the visceral pleura, combined with a rib fracture, is the most frequent underlying mechanism. The diagnosis rarely poses a problem, but sometimes a pneumothorax can be small or loculated (Fig. 3). In the supine position a pneumothorax tends to collect in the anterior-inferior costodiaphragmatic angle, producing a "deep" costophrenic sulcus (deep sulcus sign), a "double-diaphragm" contour, hyperlucency in the lower thorax

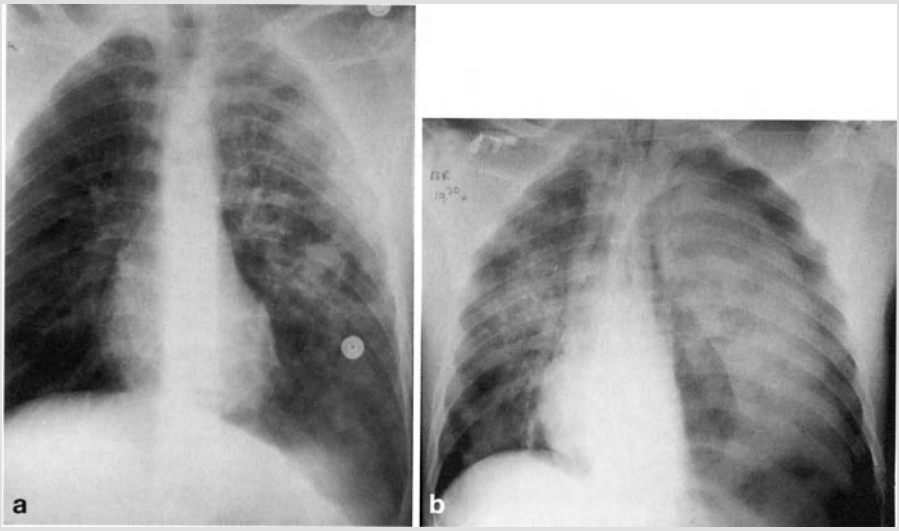

Fig. 3a,b. Basal pneumothorax. **a** Contusion of the left lung with two areas suspect for basal pneumothorax; this was thought to represent a ruptured diaphragm. **b** After a negative laparotomy a tension pneumothorax developed

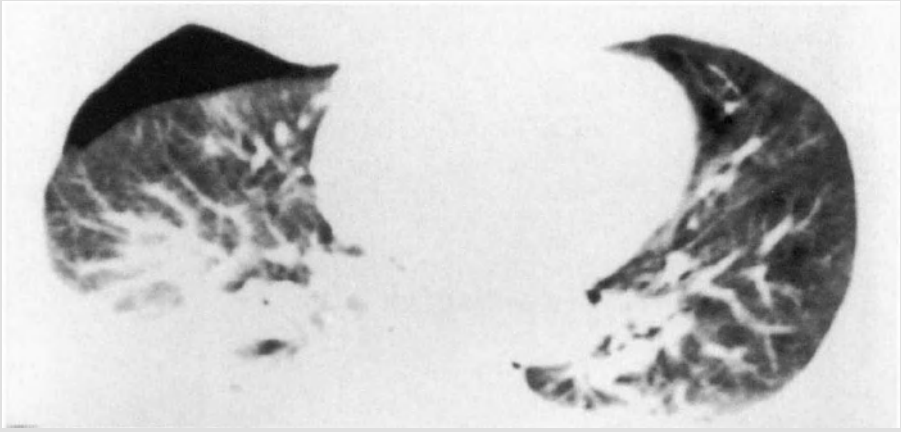

Fig. 4. Pneumothorax. In the supine position a small pneumothorax is best seen with CT

and upper abdomen, and a sharp demarcation of the cardiac apex [9, 10]. CT is the most sensitive technique to detect a small pneumothorax that lies anteriorly in the supine patient (Fig. 4). All abdominal CT scans should be initiated at the lung bases and viewed with lung window settings to detect a subtle pneumothorax. All pneumothoraces have the potential to become larger and develop tension components. It is important to detect pneumothorax before surgery to provide

prophylactic thoracostomy tube placement. Tension pneumothorax is recognized by displacement of the ipsilateral diaphragm inferiorly, shift of the mediastinum to the contralateral side and spreading of the ipsilateral ribs.

Traumatic pleural effusions can consist of blood, transudate, or chyle. Venous bleeding results from laceration to the lung or pleura and is likely to be self-limiting. A rapidly expanding pleural effusion in the setting of acute trauma is more likely of arterial origin such as an intercostal, internal mammary or diaphragmatic vessel. Arterial bleeding may displace the mediastinum contralaterally. If the bleeding is extrapleural in origin, i.e., the subclavian artery, the blood appears loculated and forms a bulge convex toward the lung. Because pleural fluid is outside the lung, bronchovascular markings are preserved. Small pleural effusions are more easily detected with CT or US. Drainage of a pleural effusion is mandatory to evaluate the mediastinal and diaphragmatic silhouette.

Pneumomediastinum

Pneumomediastinum is most often caused by air released from ruptured alveoli. A sudden increase in intra-alveolar pressure may result in alveolar rupture, with the air subsequently traveling along the peribronchial connective tissue into the mediastinum. Iatrogenic causes of pneumomediastinum include barotrauma secondary to overpressure, ventilation via improperly placed endotracheal and nasogastic tubes, and as a complication of tracheostomy. Visualization of a pneumomediastinum is usually accomplished on a chest X-ray. Radiographically pneumomediastinum is best detected by visualization of the parietal pleura along the left mediastinal border. Linear streaks of gas frequently extend into the cervical soft tissues. Air in the mediastinum creates a sharply defined aortopulmonary space, a sharp contour of the descending aorta, that often can be followed into the upper abdomen. Sometimes a "continuous diaphragm sign" under the

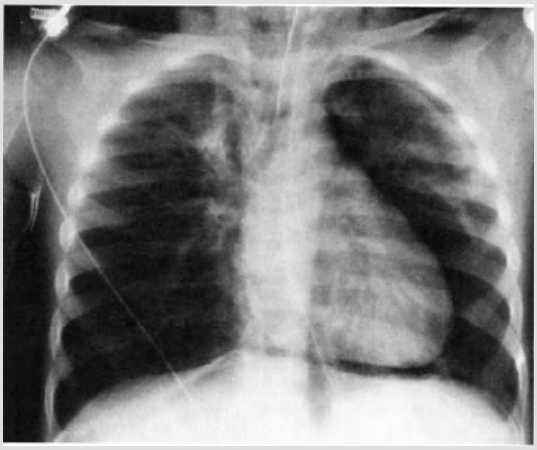

Fig. 5. Pneumomediastiunum. Demonstration of a "continuous diaphragm sign" caused by mediastinal air collection. Note also air collection along the left heart border and cervical subcutaneous emphysema

cardiac shadow can be seen (Fig. 5). CT is unnecessary for confirmation in most instances.

Other mechanisms of mediastinal emphysema include: (a) from a pneumothorax via a tear in the visceral pleura, usually at the hilus of the lung, and (b) from a bronchial rupture, with or without a pleural lesion.

Traumatized Lung Parenchyma

Pulmonary contusion occurs in 30%–70% of blunt chest trauma victims and results form direct transmission of energy through the chest wall [11]. Pulmonary contusion is characterized by lesions of the capillaries in the alveolar walls and septa, and leakage of blood into the alveolar lumen and the interstitium; the structural integrity of the lung, however, is maintained. The radiographic appearance is variable and ranges from poorly defined infiltrates without segmental or subsegmental distribution to large consolidations (Fig. 6a) [12]. Radiographic changes appear and evolve within 4–6 h in most patients; however, in most instances they are visible even on the admission chest X-ray. It is important to make follow-up chest X-rays regardless of whether the initial chest X-ray is abnormal. A contusion resolves within 7–10 days while blood in the alveolar bed resolves within 24–48 h.

Pulmonary lacerations can be caused by blunt or penetrating injuries. In nonpenetrating trauma, the laceration may first be masked by diffuse contusion and is recognized only after resolution of the contusion (Fig. 6b,c). The laceration implies disruption of the lung architecture with the formation of spaces containing air (traumatic pneumatocele) or blood (traumatic pulmonary hematoma) as a result of involvement of the bronchial tree or pulmonary vasculature, respectively (Fig. 6). CT identifies pulmonary laceration more accurately than do plain films. Lacerations resolve slowly over 3–5 weeks and may leave a residual coin lesion for several months. Lacerations of the lungs are generally benign but can lead to complications such as bronchopleural fistula and/or infection. Surgical resection is sometimes required.

Atelectasis is a frequent complication of blunt trauma to the chest and can have the following pathogenesis: (a) due to diminished ventilation caused by pain from rib fractures; (b) bronchial rupture or blood, filling in the bronchial lumen; (c) endobronchial plugs (mucus); and (d) selective bronchial position of the endotracheal tube.

Radiographic features are variable, from platelike atelectasis to large segmental, lobar, or total lung consolidations. The larger the atelectasis the more volume changes are apparent.

Aspiration

Due to loss of consciousness, emergency intubation, and vigorous resuscitation, blunt thoracic trauma can be complicated by aspiration. On the chest X-ray areas

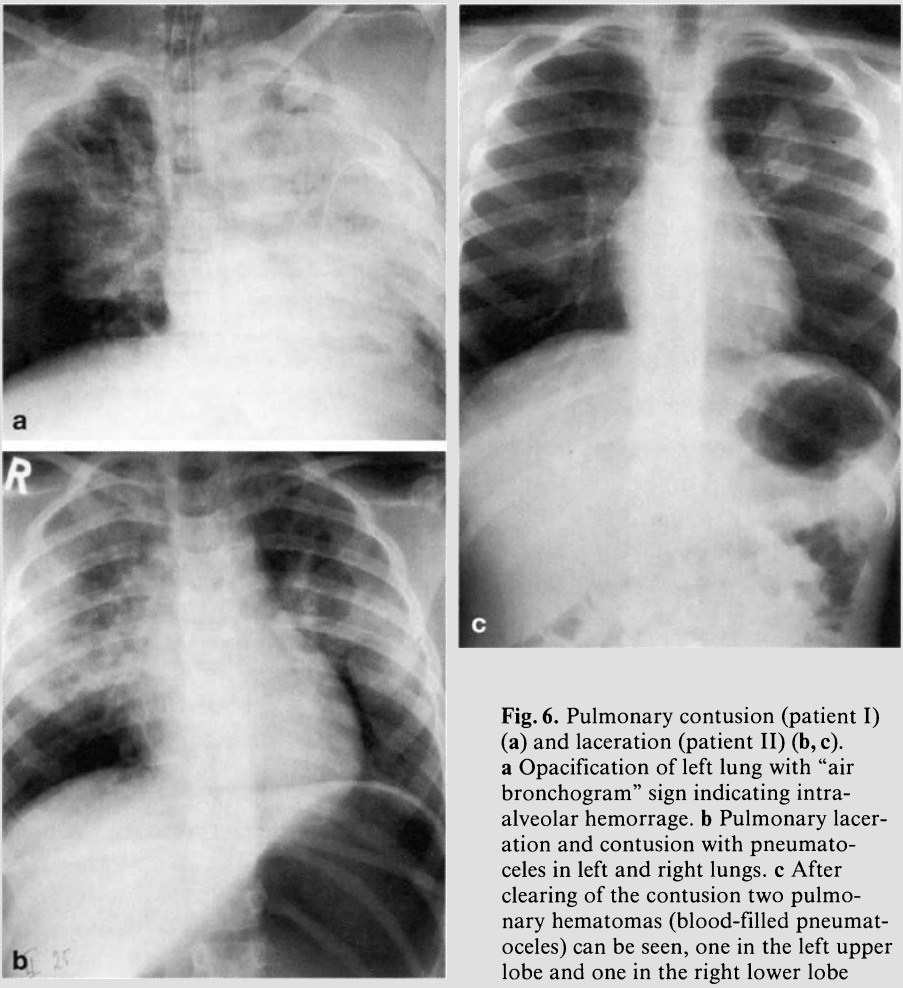

Fig. 6. Pulmonary contusion (patient I) (**a**) and laceration (patient II) (**b, c**). **a** Opacification of left lung with "air bronchogram" sign indicating intra-alveolar hemorrage. **b** Pulmonary laceration and contusion with pneumatoceles in left and right lungs. **c** After clearing of the contusion two pulmonary hematomas (blood-filled pneumatoceles) can be seen, one in the left upper lobe and one in the right lower lobe

of consolidation can be seen (most often lower lobes) that are often indistinguishable from lung contusion. Clearance depends on the pH of the gastric contents; if the pH of the aspirate is low, greater lung damage occurs and healing is delayed.

Traumatized Diaphragm

The phrenic nerve is rarely damaged by nonsurgical trauma. Thus, unrelated elevation of one diaphragm is more likely to have another cause, such as preexisting diaphragmatic eventration, gaseous distension of bowel, subpulmonic effusion, or partial lung collapse. Diaphragmatic injuries are more often related to abdominal trauma than to one of the chest. Associated injuries of liver, spleen, and bowel are frequent. The posterior and posterolateral segments are most

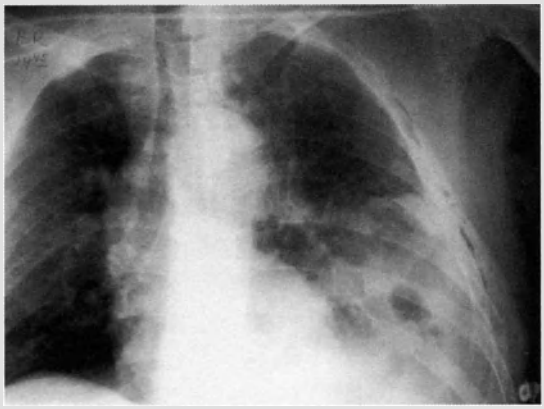

Fig. 7. Diaphragmatic rupture. The left hemidiaphragm cannot be delineated. Opacification of the left lower hemithorax with gas filled bowel loop. At operation a complete tear of the left hemidiaphragm was found

commonly involved. The left diaphragm is more commonly involved than the right, with a ratio of 2:1 [13].

In the case of a small laceration of the left hemidiaphragm, a bowel loop, the stomach, or the spleen may be found in the chest, whereas in the case of a large tear in the right diaphragm the liver may be herniated into the chest.

Conventional plain films can disclose an apparently elevated, unsharp diaphragm or a mass or pseudoinfiltrate at the base of the lung. Sometimes gas-filled bowel loops in the chest or an abnormal position of the nasogastric tube can be seen (Fig. 7). Barium studies can be very helpful in making the correct diagnosis when an extrinsic narrowing occurs in the contour of the stomach or bowel at the point where they pass the diaphragmatic tear. This makes exact localization of the diaphragm possible [13]. A large hemothorax can conceal the diaphragmatic rupture. CT, magnetic resonance imaging, and US have been recognized as important additional techniques for imaging diaphragmatic injuries [14].

Traumatized Heart and Pericardium

Trauma to the heart may represent a surgical emergency, and no time should be wasted with conventional X-ray or CT. An echocardiogram should be performed as quickly as possible, and if pericardial fluid is present, emergency drainage should be performed. In addition to myocardial contusion and rupture, cardiac injuries include damage to the pulmonary arteries, valvular incompetence, traumatic interventricular septal defects, and pericardial bleeding.

Cardiac tamponade is rather unusual in patients with blunt chest trauma, unless it is associated with aortic rupture, coronary laceration, or mediastinal hemorrhage [15]. A rapid accumulation of blood in the pericardial space often causes tamponade and severe hemodynamic problems without altering the radiological appearance of the cardiac silhouette. Sometimes a nonspecific enlargement of the cardiopericardial contour can be seen (Fig. 8). Cardiac

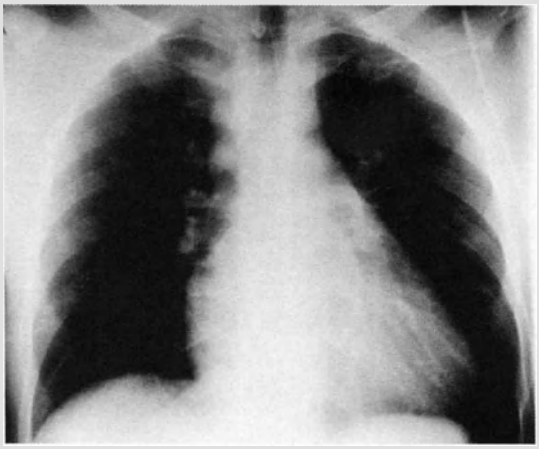

Fig. 8. Cardiac tamponade. A nonspecific enlargement of the cardiac silhouette is visible. Note the distension of caval and azygos silhouette

tamponade is reflected on CT by distension of the inferior vena cava, hepatic and renal veins.

Pericardial effusions in supine patients, surprisingly, are more frequently present in the anterior portion of the pericardial sac than in the posterior one. This allows CT or US to be used safely for percutaneous drainage of pericardial effusions.

Pericardial rupture represents a rare consequence of serious thoracic trauma and was diagnosed in only 0.11% of 20000 admissions in a large trauma center over a 10-year period [16]. Typically the diagnosis is established intraoperatively during resuscitation or surgery for associated injuries or at autopsy. Pneumopericardium may appear in the presence of pneumothorax as air enters through the pericardial disruption.

Traumatized Airways

Trauma of the tracheobronchial tree occurs most commonly at the distal end of the trachea or main bronchi. Subcutaneous emphysema, tension pneumothorax, hemoptysis, and airway obstruction account for the most common clinical signs observed at admission. The chest X-ray shows pneumothorax, pneumomediastinum, and subcutaneous emphysema. In some instances the ipsilateral lung fails to collapse in the presence of a large pneumothorax. If transection of the main bronchus is complete, and in the presence of a large pneumothorax, the lung falls to a dependent position in the hemothorax, producing the "falling lung sign." Another sign indicative of bronchial rupture is persistent pneumothorax in spite of adequate drainage [17]. Urgent bronchoscopy is mandatory for the definitive diagnosis of tracheal or bronchial rupture.

Mediastinal Hematoma

Blunt trauma to the chest is commonly associated with local or diffuse bleeding into the mediastinum. Bleeding into the mediastinum may arise from rupture of mediastinal veins or arteries. Clinical evidence of continued bleeding should lead to angiographic evaluation of internal mammary and intercostal arteries.

Thoracic Aorta Injury

In the United States 25% of the more than 150000 deaths occuring each year from trauma result from thoracic injury [18]. Several investigators have demonstrated that rupture of the thoracic aorta occurs in 10%–30% of patients sustaining fatal blunt trauma [19]. In an autopsy study Parmely et al. found that 85% of patients with aortic rupture die shortly after trauma, before reaching the hospital [20]. For patients who do reach the hospital with aortic damage it is essential that early diagnosis be made prior to prompt surgical intervention.

The currently accepted mechanism to explain traumatic aortic rupture from rapid deceleration involves a combination of traction, torsion, hydrostatic, and shearing forces [21]. The most common location for aortic rupture is at the site of the ligamentum arteriosum. The transverse or longitudinal intimal and/or medial tear is contained either by the adventitial wall or the mediastinal pleural wall.

The clinical diagnosis of aortic rupture is very difficult. A difference in blood pressure between the upper and lower extremities (caused by an intimal flap at the site of the rupture) can be a helpful sign. The most important clues for the diagnosis of a traumatic aortic rupture are the pathogenesis of the trauma and the findings on the plain chest X-ray. The conventional supine chest film is in most cases the first and often only indicator of major injury to the thoracic aorta. Ideally, an upright chest radiograph should be obtained, but this is often impractical because of pelvic, spinal, and head injuries or shock. There are many indirect signs of aortic tear, but they are never absolute and may be either false positive or false negative.

On the plain chest X-ray, the following signs may indicate the presence of a thoracic aortic rupture.

The Widened Mediastinum. Many signs of mediastinal hematoma have been reported to be associated with aortic rupture [22]. Widening of the superior mediastinum as a result of mediastinal hemorrhage is visible in the majority of patients with aortic tears. However, this widening can be very mild or even absent. Moreover, mediastinal hematoma can also be caused by rupture of small intrathoracic vessels or by a fracture of the thoracic spine [5]. The mediastinum is difficult to evaluate on a supine chest X-ray because of the short focus-film distance, rotation of the patient, and mediastinal widening in large and obese patients. Woodring et al. [22] concluded that a diagnosis of mediastinal hematoma may be made if the aortic contour is unsharp, or if one of the following signs is positive: mediastinal width greater than 8 cm at the level of the aortic

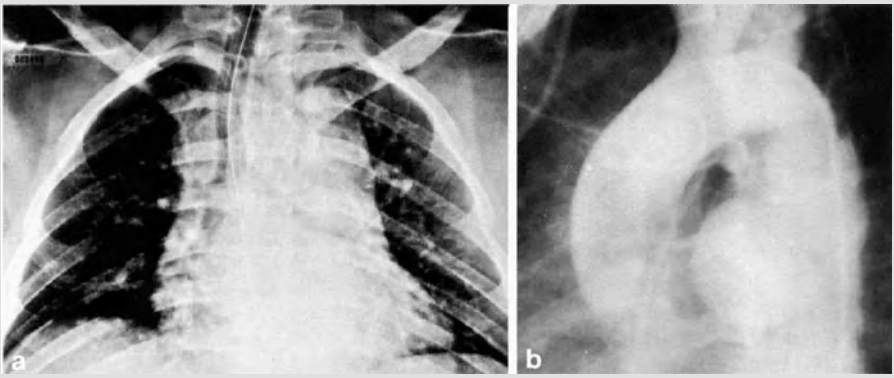

Fig. 9a,b. Aortic rupture. **a** Blunt chest trauma: widened mediastinal shadow; tracheal displacement to the right; deviation of nasogastric tube to the right; abnormal aortic contour. **b** Angiography: aortic rupture, with large false aneurysm below the isthmus

knob, apical cap, or widening of the right paratracheal stripe more than 5 mm deviation of the nasogastric tube (Fig. 9a,b). If both of the last two signs are negative, there is a 98% probability that an aortic rupture is absent.

Deviation of the Nasogastic Tube to the Right. The sensitivity of rightward nasogastric tube displacement is only 60%–70% [23]. Thus absence of this finding does not exclude the diagnosis, and the decision regarding aortography must be based on other radiographic criteria.

Right Paratracheal Stripe Widening. Normally the paratracheal stripe in supine patients is less than 5 mm. Conversely, aortic injury was present in 23% of patients with a paratracheal stripe wider than 5 mm [22].

Widening of the Right and Left Paraspinal Lines. This is a rather specific and sensitive sign for mediastinal hematoma, especially on the right side and especially in combination with a broadened paratracheal stripe, a nasogastric tube displaced toward the right, and an opacified pulmonary window [24]. Widening of the paraspinal lines also occurs in cases of spinal fracture.

Left Apical Cap. The sensitivity of this sign is rather low. Simone et al. concluded this on the basis of findings in 45 patients suspected for aortic rupture [25]. An apical cap is often present in left hemothorax and upper rib fractures.

Obscuration of the Aortic Knob. The sensitivity of this sign was 100% in the series of Livoni and Barcia [26]. Specificity, however, is low. In the series reported by Marnocha and Maglinte no case was found with aortic rupture if the aortic knob and contour appeared normal, and the trachea and nasogastric tube were not deviated [27].

Fracture of the First and Second Rib. There is no relationship between first and second rib fractures and vascular mediastinal injuries [28].

Rightward Deviation of the Trachea, Downward Displacement of the Left Main Stem Bronchus, Opacification of the Aortopulmonary Window and Hematothorax. These are less sensitive and specific signs.

In summary, the chest roentgenographic findings of aortic rupture are frequently nonspecific. Any or all of these suggestive signs may be present in the absence of significant aortic injury. Conversely, in good clinical practice a rather large percentage (30%–70%) of negative aortographies must be accepted in search for aortic rupture [29].

Contrast enhanced CT (in stable patients) can be helpful to select patients for aortography. If no signs of mediastinal hematoma are found on CT, the probability of aortic laceration is extremely low. CT can also help to reduce the number of negative angiographic findings. If signs of mediastinal hematoma are found on CT, angiography is mandatory [3, 30, 31]. In high-risk patients, as defined by the type of trauma and the chest X-ray findings, aortography must be performed following the X-ray study.

New developments in diagnosing rupture of the thoracic aorta include transesophageal echography for ruptures located in the descending aorta.

In 5% of patients with aortic rupture the lesion is not detected at admission, and a false aneurysm is detected later by chance or because of symptoms (dysphagia, dyspnea, chest pain). These patients require surgery because of the increasing chance of rupture with time [24].

Fat Embolism Syndrome

Fat embolism syndrome should be considered in patients with radiographically clear lungs who develop sudden shortness of breath, and who have long bone fractures (see Biert and Goris, this volume). The lung lesions consist of interstitial and intra-alveolar hemorrhagic edema. Initially the chest radiograph is normal. After 12–72 h bilateral bronchovascular haziness occurs, changing into ground-glass opacities and lung consolidation with air bronchograms. In nonfatal cases clearing usually occurs within 1 week. In severe cases no resolution occurs and adult respiratory distress syndrome (ARDS) can develop.

Adult Respiratory Distress Syndrome

ARDS is a condition of unknown cause in which the pulmonary capillary permeability to fluid and protein is altered, thereby allowing proteinaceous fluid to exude into the interstitial tissues of the lung and eventually into the alveoli.

The clinical signs of tachypnea and hypoxia precede by 12–24 h the changes which become visible on the chest X-ray. The chest X-ray shows bilateral uniform

lung consolidations with air bronchograms. The heart is usually normal in size, and pleural fluid is absent. The lung consolidation can remain unchanged for several weeks; barotrauma (pneumothorax, mediastinal emphysema, and interstitial emphysema) is, however, frequent in these patients, reflecting the increased airway pressures required to maintain ventilation. If the patient survives, clearance of pulmonary abnormalities can take several months.

The radiological presentation is strongly determined by the underlying lung pathology and by the possible complications of the therapy. On the basis of the radiological findings alone it is difficult to differentiate ARDS from other causes of diffuse lung consolidation such as massive contusion, pulmonary edema, and diffuse infection. Posttraumatic ARDS should be suspected in patients with severe respiratory distress and diffuse lung consolidation that shows no tendency to resolve after the first week.

Conclusion

The plain chest X-ray is the most useful tool in the evaluation of thoracic trauma victims. Careful evaluation of the spine, chest wall, pleura, lung parenchyma, diaphragm, and mediastinum is mandatory. Repeat chest X-rays are useful in follow-up and after the placement of IV lines, intubation, and resucitation. The plain chest X-ray gives a number of clues to the diagnosis of aortic arch rupture. CT, sonography, and sometimes angiography can give important additional information in some patients.

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The Role of Ultrasound in the Management of Blunt Abdominal Trauma

K. Glaser, J. Tschmelitsch, A. Klingler, and G. Wetscher

Introduction

Patients with blunt abdominal trauma are often hemodynamically unstable and frequently unconscious. A decision for laparotomy must to be established rapidly in the case of severe intra-abdominal injuries in order to keep mortality as low as possible; however, only 30% of our patients with blunt abdominal trauma had such lesions necessitating surgery. Diagnostic investigations immediately after admission therefore aim at providing reliable criteria to identify the patients who require emergency surgery. The amount of free fluid in the abdomen is more important in this than the exact location of the injury. Peritoneal lavage, computed tomography, ultrasound (US), and recently laparoscopy have been reported to provide good results in the diagnostic management of patients with blunt abdominal trauma. In our hands, US has proven both reliable and accurate in identifying blunt abdominal trauma.

US Examination

We retrospectively studied 1151 patients with blunt abdominal or thoracic trauma treated in our Department between January 1980 and January 1990; there were 670 men and 481 women, with a mean age of 52 years. The US investigation (Combison 100, Kretz-Technik, Zipf, Austria; Sonoline SL-2, Siemens, Germany) was performed immediately in the emergency room. US began in the right upper abdomen and right flank, with the patient lying on his back. In addition to inspection of the liver, the right kidney and right retroperitoneal space, special attention was paid to Morrison's pouch, an area where intra-abdominal fluid first shows in the event of liver rupture or lesions of upper abdominal organs. The left upper quadrant and left flank were then examined for evaluation of the spleen, left kidney, pancreatic tail, and left retroperitoneal area. At the same time the left and right thoracic cavity were investigated for free fluid. The pancreatic head and body were identified in the middle upper abdomen. Peripancreatic fluid combined with retrocolonic fluid along the ascending and descending colon was of interest with respect to the diagnosis of pancreatic rupture. The middle lower abdomen was evaluated for free fluid in the pouch of Douglas.

Patients who did not seem to require surgery underwent careful clinical, hemodynamic, and US observation. If they became secondarily unstable during observation in spite of adequate blood transfusion, and if increasing amounts of intra-abdominal fluid were found in US, operative exploration was performed immediately.

After the abdomen had been investigated ultrasonographically, we routinely searched for intrathoracic fluid collections.

Diagnostic Accuracy of Ultrasound

The diagnostic accuracy of US was assessed comparing the amount of intra-abdominal free fluid found as indication for operation to surgical results and to the clinical course in conservatively treated patients followed up over 4 weeks. Of the 1151 patients the initial US examination revealed no pathological findings in 506 (44%). In three of these cases (0.6%) clinical deterioration and free intra-abdominal air required surgery, which revealed bowel perforation not diagnosed by ultrasound (37% of the total eight bowel perforations). The patient's condition remained stable in 461 (91%), and 42 developed minor amounts of free fluid (no operation performed).

Intra-abdominal and/or intrathoracic lesions were found in 645/1151 patients (56%). Of these, 238 (37%) were in a stable condition and were treated conservatively (Table 1); organ lesions, such as contusions, subcapsular or intraorgan hematomas, were observed in 115, and tears, while in 123 only free fluid was detected without a visible organ lesion. All of these patients continuously showed stable vital functions, as assessed by clinical observation and repeated US examination, and surgery was not required. A true-negative diagnosis with respect to the indication for laparotomy was established by US in 741 (64.4%) of 1151 patients. In three of eight cases with bowel perforation US results thus proved falsely negative (0.3% of all patients).

Table 1. Patients with pathological findings as established by initial ultrasound ($n = 645$)

Pathological findings	*n*	%	Operation (*n*)	Conservative therapy (*n*)
Liver lesions	90	14.0	27	63
Splenic lesions	83	12.9	34	49
Kidney lesions	59	9.1	11	48
Pancreatic lesions	9	1.3	6	3
Bowel injuries	5	0.8	5	0
Mesenterial tears	3	0.5	3	0
Hemo/pneumothorax	260	40.3	260	0
Increasing amount of free fluid	22	3.4	18	4
Others (diaphragm, hematomas, etc.)	114	17.8	43	71

US examination indicated operative treatment in 407/1151 patients (35.3%), and in 395 cases (97%) the operation was considered necessary. The preoperative US diagnosis was therefore true positive in 34.1% of all patients. In 12 (3%) laparotomy was not necessary a posteriori. In 7 of these the false-positive US finding was due to diapedesis of blood from a retroperitoneal hematoma or from vertebral fractures into the peritoneal cavity, simulating intra-abdominal bleeding. Five patients had only minor intra-abdominal injuries, and bleeding had already subsided spontaneously. In these 12 patients the US diagnosis was considered false positive. Thus US had an overall sensivity of 99%, specificity of 98%, positive predictive value of 97%, and negative predictive value of 99%.

Peritoneal Lavage, Computed Tomography, or US?

Peritoneal lavage and computed tomography are frequently used as diagnostic procedures in blunt abdominal trauma. Peritoneal lavage may cause complications [1–8] and does not reliably estimate the amount of intra-abdominal blood [6, 9–11]. Computed tomography is time consuming and cannot be performed at the bedside [3, 12]. Furthermore, computed tomography is expensive, and repeated examinations are not practicable.

In our Department 45000 US investigations have been performed by surgeons since 1979. There was a steep learning curve, since surgeons receive immediate feedback. Since 1982 US examinations have also been carried out by our residents. According to the guidelines of the Austrian Society of Ultrasound in Medicine, 500 investigations under supervision of a US tutor are necessary to be able to interpret intra-abdominal US findings. Therefore, provided the required experience has been obtained, US is our first choice in evaluating blunt intra-abdominal injury.

US-Based Indications for Laparotomy

Even if intra-abdominal blood is found by US, the surgeon has to decide whether laparotomy is indicated, since minor bleeding may subside spontaneously.

Therefore the ultrasonographer must answer the following questions:

(a) Is intra-abdominal or intrathoracic free fluid present, and what is the nature of this fluid?
(b) Is the amount of blood clinically relevant?

If less than 500 ml blood is found more than 3 h after injury, there is no indication for laparotomy in patients with stable vital functions or requiring no more than 4 U blood for stabilization. On the other hand, 500 ml blood detected no more than 30 min after trauma is a strong indicator for surgery.

During the first work-up of the injured patient it is often impossible to directly detect by US which organ is injured. Also, fresh blood clots in ruptured organs, such as liver or spleen, can barely be identified from the normal aspect of that

organ's parenchyma, as blood clots initially show a similar US pattern [13]. Only after 2–4 h the visibility of blood clots by US is enhanced by alterations of the hemoglobin-structure within the clot. However, it is clear that the main importance for US during the acute phase lies in the detection and quantification of free fluid.

Large retroperitoneal hematomas constitute a difficult diagnostic problem for US. First of all, the amount of blood loss into the retroperitoneal structures is often important. Patients are therefore in an unstable condition or need a large amount of blood for stabilization. Secondly, there is generally a noticeable amount of free intra-abdominal fluid since blood or plasma is passively pressed through the serosal monolayer of the retroperitoneum. Finally, large retroperitoneal hematomas induce a paralytic ileus. On the whole, these patients show a variety of findings, which renders further conservative management impossible and necessitates exploratory laparotomy. These patients may be candidates for laparoscopic evaluation.

Bowel perforations are generally difficult to diagnose by US. Large ruptures may present with a large amount of free intestinal fluid which shows echogenic spots similar to falling snow. In doubt, US-guided fine needle aspiration reveals the nature of the fluid. Small perforations, or secondary perforations due to a ischemia, can be detected only during the follow-up period. This delays surgery and increases mortality.

The three false-negative US findings in our series were due to bowel perforation. The sensitivity of US with respect to bowel perforation was only 63%. Therefore in case of severe blunt abdominal trauma with large contusion marks on the abdominal wall, exploratory laparotomy should be performed even if the US findings are negative.

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Diagnostic Procedures in Abdominal and Retroperitoneal Injury

E. W. Childs and F. W. Blaisdell

Introduction

In the United States trauma is the fourth leading cause of death and the most frequent cause of mortality in persons under the age of 45 years [1]. The distribution of abdominal trauma is almost evenly divided between penetrating and blunt. With penetrating trauma the ability to assess the injury is relatively easy, whereas in blunt trauma the injury is often more subtle, particularly when there are major associated injuries such as rib fractures, extremity fractures, or an altered sensorium due to intoxicants or head injury. In all instances a high index of suspicion must be present on the part of the clinician, because the rule is "blood somewhere, blood nowhere, blood must be in the abdomen." The extremities, neck, and chest can be readily examined, but the abdomen represents the "black box" where either retroperitoneal injury, intraperitoneal injury, or both can exist without obvious external manifestations. Prior to the advent of current diagnostic modalities up to 17% of patients with abdominal trauma died secondary to unrecognized intra-abdominal bleeding [2, 3]. The purpose of this chapter is to discuss the diagnostic procedures available for the evaluation of intraperitoneal and retroperitoneal injury.

Diagnostic Adjuncts

Peritoneal Lavage

Paracentesis was first used for the evaluation of trauma, pancreatitis, and peritonitis by Neuhof and Cohen in 1926 [4]. In 1965 Root et al. expanded this idea with the addition of lavage [5]. This ingenious addition increased the sensitivity of the procedure in trauma patients and led to its increased use. The accuracy of the initial physical exam in detecting intra-abdominal injury has been reported to be only 16% and 45% [6–8]. A delay in diagnosis is associated with an increase in morbidity; therefore, peritoneal lavage has been applied as a more objective means of making the diagnosis of intra-abdominal injury. When the patient in the emergency room is unstable and requires large volumes of fluid to maintain blood pressure, a rapid trip to the operating room is appropriate. When the instability appears to be caused by bleeding associated with scalp lacerations,

fractures (such as those of the pelvis), or when there is an associated urgent problem, especially head injury, peritoneal lavage is performed immediately to determine whether the first therapeutic maneuver should be laparotomy for abdominal hemostasis. In the patient with a possible major thoracic injury such as a ruptured thoracic aorta, peritoneal lavage is carried out prior to obtaining an angiogram. The major advantages of peritoneal lavage are: it is rapid, it can be performed in the emergency department, it lends itself to early diagnosis and triage, and the patient can remain under continuous observation by the physician.

There are two basic techniques for introducting the catheter into the peritoneal cavity, open and closed. The two techniques are similar in regards to accuracy and safety [9]. The open technique is espoused by the American College of Surgeons and taught through the Advanced Trauma Life Support program [10]. We prefer the open technique because it is safe, easy, and reliable. The infraumbilical region is prepared and draped in a normal sterile fashion. The area is infiltrated with a local anesthetic (1% lidocaine). A nasogastric stomach tube and a bladder catheter are placed prior to incision. A linear incision is made in the midline approximately 3.5 cm in length, depending on the thickness of the subcutaneous tissue, and carried down to the linear alba. A 5-mm incision is made in the fascia exposing the peritoneum. The peritoneum is grasped with hemostats or forceps, incised, and the abdominal cavity entered. The lavage catheter is carefully passed along the parietal peritoneum and then advanced downward into the pelvis. The fascial defect is closed in a running fashion using no. 1 polyglycolic acid suture. A syringe is attached to the end of the catheter for aspiration, 10 ml gross blood is considered a positive tap. If 10 ml gross blood is not returned, one liter of normal saline is infused into the peritoneal cavity. The bag is then placed to dependent drainage for removing of at least 750 ml. Laboratory analysis for RBC, WBC, bile, amylase, and vegetable matter is performed. A positive result includes 100000 RBC/ml, 500 WBC/ml, 175 U/100 ml amylase, the presence of bilirubin, and detection of vegetable matter.

The WBC count in lavage fluid is being questioned as to its clinical levels of significance [11]. Root has recently supported the use of WBC count and related its application to the timing of laboratory collections. In his 1967 experimental work he noted marked polymorphonuclear neutrophil leukocyte release to chemical or bacterial exposure beginning within 2 h of exposure. Pancreatic secretions in the peritoneal cavity provided the earliest response of 660 WBC/ml in 1 h and gastric juice produced 200 WBC/ml. Over 5.5 h bile and fecal suspension produced WBC counts in the 10000–12000/ml range. He concluded that when the time from injury exceeds 180 min minimum, the WBC count closely correlates with the presence of intra-abdominal injury [12].

The overall sensitivity of peritoneal lavage ranges between 95% and 98% [13]. The morbidity of peritoneal lavage is less than 1% and reflects operative experience. Those include perforation of bowel and/or mesentery, bladder, and vascular structures. Relative contraindications are previous abdominal surgery, a gravid uterus, and massive obesity.

False-negative studies can result when there are unsuspected intra-abdominal adhesions that compartmentalize the abdominal cavity; when there are retroperi-

toneal injuries such as duodenum, pancreas, rectum or kidney; when there is an associated diaphragm laceration; or when there is bowel injury with minimal initial contamination.

Computed Tomography

The use of computed tomography (CT) for evaluation of blunt abdominal trauma received considerable attention in the early 1980s [14]. The procedure is noninvasive and offers evaluation of both the intraperitoneal and retroperitoneal structures. In contrast to diagnostic peritoneal lavage (DPL), CT is organ specific. CT is best suited for hemodynamically stable, blunt trauma patients presenting to the emergency department with equivocal abdominal examinations or for patients whose serial hematocrit values show an unexpected drop and the source is not readily apparent [16]. It is also valuable for patients with a relative contraindication for DPL, for example, multiple previous abdominal operations and obesity.

The procedure optimally involves administering 200–400 ml 1% diatrizoate by mouth or by nasogastric tube at least 30 min prior to the scan and 100 ml IV iodinated solution [15]. The scan should extend from the diaphragm through the pubis, with 1-cm cuts to the level of the lower poles of the kidney and 2-cm cuts thereafter.

The limitations of the procedure include availability of experienced radiologists, distance from the emergency department, variability of scanners, patient cooperation, and incompatibility with oral and intravenous contrast. Certain injuries such as ruptured bowel and isolated pancreatic injuries can be difficult to diagnose, particularly when the scan is performed shortly after the time of injury [13]. In the evaluation of acute abdominal injuries, the sensitivity for demonstrating solid viscus injury or hemoperitoneum ranges between 92% and 96.5%, with a specificity from 90% to 99% [17].

Ultrasound

The use of ultrasonography in the evaluation of blunt abdominal trauma has gained wide acceptance in Europe; however, its use in the United States has been limited [22, 29]. Gruessner reported on 71 patients in Germany with blunt abdominal trauma and obtained a sensitivity of 84%, specificity of 88%, and accuracy of 86% using sonography [18]. Dietrich et al. performed an ultrasound study in less than 1 min at bedside [19]. Tso et al. trained trauma fellows with at least 1 h of theoretical training and 1 h of practical training, and their results were similar to the above in regards to sensitivity, specificity, and accuracy [20]. Rozycki reported a 10% difference in the interpretation of the surgeon and radiologist [21]; the radiologist reviewed the studies from a static view usually within 24 h.

The potential benefit of ultrasound is its lack of invasiveness, proximity to patients in monitored areas, and its ability to be performed by in-house trauma

personnel. The disadvantage of a sonogram is that it is not organ specific and provides limited information on obese patients or patients with subcutaneous air. Approximately 200 cm^3 free peritoneal fluid is needed to be detected via ultrasound. This could present a problem in patients with clotted blood or loculated fluid secondary to intra-abdominal adhesions.

Ultrasound may be useful to surgeons in assessing patients in an acute setting. The learning curve of increased accuracy is approximately 200 examinations [21]. For the surgeon to become proficient with this procedure it must be incorporated into general surgery training programs.

Laparoscopy

Laparoscopy was first performed nearly 90 years ago [23, 24]. Gazzaniga and associates proposed the use of laparoscopy in the evaluation of trauma patients in 1975 [25]. With the advent of laparoscopic cholecystectomy there has been a resurgence of interest in peritoneoscopy or celioscopy in the evaluation of abdominal disease and injury [26]. Several authors have advocated the use of laparoscopy in the emergency setting for the diagnosis of intra-abdominal injury [23, 24, 27, 28]. There is potential for both diagnosis and therapeutic intervention using a laparoscope in trauma patients. There are several limiting factors in its use on trauma patients that revolve around cost, the operating versus emergency room environment, anesthesia, and the complications of pneumothorax and gas emboli.

Salvino et al. evaluated 75 patients in a prospective fashion with both DPL and laparoscopy; 93% of the procedures were performed in the emergency department. Fifty-nine percent of their patients were injected with a local anesthetic, and no patients required intubation secondary to the procedure only. Their results were that three patients with negative DPL had injuries that were found laparoscopically and three with positive DPL had only minor injuries. These six patients in whom the injuries were identified were treated by observation alone. They concluded that management based on laparoscopy in addition to peritoneal lavage could potentially improve the care of 8% of their cases [24].

Laparoscopy may be performed under local and/or general anesthesia, A small incision is made in the infraumbilical region. The Veress needle is used to infuse the abdominal cavity with 12–15 cmH_2O pressure of carbon dioxide or nitrous oxide. Once the abdominal cavity is insufflated, a 10/12-mm trochar is inserted at the infraumbilical region to gain access to the peritoneal cavity. Subsequent trochars are placed as necessary.

The advantages of laparoscopy are that it is organ specific and may be used for diagnosis as well as therapeutic interventions. Laparoscopy can be performed in the emergency department or operating room under local anesthetic without marked discomfort. The disadvantages include the cost and lack of trained personnel to both operate and assist in set-up of the procedure. The risk, particularly related to the trauma patient, includes tension pneumothorax, air embolism, and cardiovascular compromise.

Minilaparotomy

In many instances a minilaparotomy may be more advantageous than peritoneal lavage in a patient who is going to require prolonged anesthesia for treatment of other injuries. This is particularly true when prolonged orthopedic procedures are to be carried out. Our policy is to fix all major fractures within the first 24 h of injury, but the surgery often involves many hours under anesthesia. Under these circumstances, anesthesiologists and the general surgeons feel far more comfortable knowing from direct inspection that there is no major abdominal injury.

A minilaparotomy is usually carried out using an epigastric midline incision. An incision of approximately three to four fingerbreadths is made through skin, fascia, and peritoneum. A standard suction device is then inserted down into the pelvis into the right and left lower quadrants and up under the diaphragm. The absence of blood or intestinal contents in any of these regions rules out quite effectively any serious peritoneal process. To complete the procedure sponge sticks are used to push the stomach down and assess the gastrohepatic area for evidence of a central retroperitoneal hematoma that may be associated with major vascular or pancreatic injury. If there is no evidence of hematoma, critical retroperitoneal bleeding is fairly effectively ruled out, and a major retroperitoneal injury requiring surgical intervention is unlikely. Pelvic or lateral retroperitoneal hematomas that have not manifested themselves clinically are usually of no consequence and are managed nonoperatively in most instances. The risk of minilaparotomy is minimal and far outweighs a missed intraperitoneal injury.

Management: Penetrating Versus Blunt Trauma

Penetrating Injuries

Evaluation of gunshot wounds is relatively straightforward. The path of the missile can usually be traced by noting entrance or exit wounds or, in the case of an entrance wound only, by the use of X-rays to locate the wounding missile. All penetrating injuries occurring between the 6th intercostal space and the inguinal ligament must be assumed to be intraperitoneal. The diaphragm can rise as high as the 4th or 5th interspace; therefore, injuries in this region also carry a possible risk of peritoneal penetration.

Physical examination that demonstrates abdominal tenderness is diagnostic of injury and requires laparotomy, despite the lack of evidence of injury by parallel diagnostic studies. The chest should first be ruled out as a source of instability by a chest X-ray. Missile fragments below the diaphragm require operation and no further diagnostic procedures. Penetrating knife and gunshot wounds require immediate exploration in an unstable patient. Gunshot wounds of the abdomen benefit from operation, as 90% of these types of wounds are associated with intra-abdominal injury, and selective management is rarely indicated unless the wound is clearly superficial or subcutaneous. Knife wounds are more subtle and more difficult to evaluate. Knife wounds of the chest, 6th interspace or below, may

dictate laparotomy if there is any evidence of abdominal tenderness or any possibility of diaphragm penetration. Even when there is evidence of thoracic bleeding, there is always the possibility that the source of the bleeding is intra-abdominal as a result of diaphragm penetration. Because of the larger mass of tissue that must be penetrated before a hollow viscus is reached, stab wounds posterior to the posterior axillary line may be managed selectively.

Peritoneal lavage and CT of the abdomen are not particularly reliable in ruling out abdominal injury following penetrating injuries of the chest because the lavage fluid that bathes a site of abdominal viscus injury may be sucked up into the chest. On occasion, if a chest tube has been placed, diaphragmatic injury may be identifiable by the appearance of a gush of lavage fluid from the chest tube as lavage is initiated. CT is not reliable because knife wounds, even when they penetrate the bowel or some other critical structure, may not be apparent on the X-ray examination until major bleeding or advanced inflammation from the leak of bowel contents has developed.

If the size of the wound permits, we explore stab wounds with a gloved finger. If there is overt peritoneal penetration, operation is indicated. If peritoneal violation is not overt, particularly in the obese patient, we extend the subcutaneous portion of the laceration in the emergency room to identify fascial penetration. This procedure is carried out using local anesthesia. If there is evidence of fascial penetration, we advise laparotomy, although we recognize that this is controversial and many centers use close observation so as to avoid a 20%–30% incidence of negative laparotomy. One alternative is to use peritoneal lavage to ascertain the presence of bleeding or bowel contamination. Laparoscopy can also be used in this situation to prevent an unnecessary celiotomy [30].

Blunt Injuries

Blunt trauma that is confined primarily to the abdomen or lower half of the chest is best evaluated by clinical assessment and by serial hematocrit values and WBC counts. The presence of abdominal tenderness, particularly when it increases during observation, is an indication for laparotomy despite apparent stability. A falling hematocrit with no other explanation than the abdominal injury is an indication for operation. Generally we consider a fall in hematocrit of more than 6 points with no other source of the blood loss an indication for some type of definitive evaluation.

In patients in whom the ability to monitor abdominal bleeding has been lost because of the associated injuries, ancillary diagnostic procedures are of value. These include patients with severe head injuries, patients with blood loss from other sites, and those in whom the presence of rib fractures or pelvic fracture may make abdominal assessment difficult.

If the patient has had major blood loss and requires anesthesia to treat specific injuries such as major scalp lacerations or fractures, we carry out peritoneal lavage in the operating room after induction of anesthesia. If the patient is unconscious with evidence of major head injury, before taking the patient to

X-ray for head CT, we initiate peritoneal lavage. The problem with immediate emergency CT is that the patient is lost from physician contact. If urgent X-ray, CT, or magnetic resonance imaging is necessary for central nervous system injury, abdominal injury should be ruled out by a prompt peritoneal lavage, with the fluid drainage continued as the patient is transported to the X-ray department.

Other patients who are conscious and alert, and who have no manifestations of bleeding are followed closely clinically. Hematocrit values are monitored every 4 h for the first 24 h. If the hematocrit drops 6 points with no obvious explanation, and the abdomen is benign, we perform CT to establish the presence or absence of abdominal fluid, major organ injury, or retroperitoneal injury. A negative CT result provides evidence that the source of the hematocrit drop is not in the abdomen or retroperitoneum. If the patient develops abdominal findings associated with a drop in the hematocrit or a rise in WBC count, an exploratory laparotomy rather than a diagnostic procedure is indicated.

Conclusions

Use of the various diagnostic modalities described will vary depending upon the experience and expertise available in each individual environment (Table 1). We use open peritoneal lavage as our primary initial diagnostic modality. This has the advantage that it can be carried out in the emergency department where the patient can be kept under observation. It is used in patients who present in shock but have other sources of blood loss, in patients in whom a reliable abdominal examination cannot be carried out because of head injury or an altered consciousness from other causes, and in patients who are in need of urgent operation to treat other injuries such as those to the chest.

CT evaluation is reserved for cooperative and stable patients whose hematocrit values or WBC counts suggest pathology, but whose examination is relatively benign. Most often CT is used for follow-up several hours after admission or to

Table 1. Diagnostic procedures: relative advantages and disadvantages

	Advantages	Disadvantages
Peritoneal lavage	Patient under observation; quick	Invasive procedure; painful for the awake patient
CT	Noninvasive; organ specific	Requires patient cooperation or heavy sedation
Ultrasound	Noninvasive; fluid recognized	Operator experience required; bowel and organ injuries missed
Laparoscopy	Less invasive than laparotomy; accurate	Invasive, requires anesthesia; air embolism, pneumothorax
Minilaparotomy	Less morbid than laparotomy; accurate	Invasive, requires anesthesia; less accurate than laparotomy

facilitate early discharge of patients who have equivocal findings. Minilaparotomy or possibly laparoscopy is of value when the patient has multiple associated injuries that require prolonged anesthesia to treat such as major orthopedic injuries or severe chest injuries. While on the operating table the patient is lost to clinical evaluation and may manifest operative instability. Under these circumstances, knowing that the abdomen is benign provides great reassurance to all involved in the case.

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Diagnostic Procedures in Spine, Pelvic, and Extremity Injuries

P. M. Rommens

Introduction

Although spine and extremity injuries seldom are life threatening, they account for the greater part of late morbidity and disability in surviving polytraumatized patients. The approach taken during the first hours can make the difference between complete recovery and a life of disability. In the acute phases of reanimation, life-saving operations, and stabilization there is a risk of overlooking or delaying the diagnosis of "less important" lesions. A quick, accurate, and complete clinical examination of all body regions and an appropriate diagnostic check-up is therefore absolutely necessary. To save time and enhance efficiency in the initial management, the exact sequence of all diagnostic and therapeutic investigations must be fixed in a "polytrauma protocol" for diagnostic procedures involving the spine, pelvic ring, and extremities.

The Prehospital Phase

An initial clinical examination is made by the emergency physician and ambulance crew at the accident site. Information about painful body areas, such as the thoracal or lumbar spine, wrist joint, and foot, can be given by a responsive patient before intubation and ventilation. Gross abnormalities of the extremities, suggesting fractures, dislocations, or soft tissue trauma, should be noted before the extremities are splinted. A quick examination of the vascular and neurological status of the extremities can provide information that is extremely useful for subsequent management.

Consecutive vascular and neurological check-ups may reveal a deterioration in one or more injuries. All information on existing or suspected injuries should be collected in a written note attached to the medical chart of the patient in the admitting hospital. These data become the basis for the first diagnostic procedures on the extremities performed in the emergency department.

The In-Hospital Phase

Immediately after admission to the emergency department the diagnostic procedures in patients with unstable vital functions focus on the search for life-

threatening injuries. Therefore a lateral or cross-table roentgenogram of the cervical spine, AP view of the chest, and AP view of the pelvis are taken in this sequence in all severely injured patients. Diagnostic procedures for the C-spine and pelvic ring are analyzed in this chapter.

The Pelvic Ring

The AP X-ray of the pelvic ring is an essential part of the primary investigation of the polytraumatized patient. However, pelvic views, obtained in the shock room are not made under optimal conditions; the patient is only partially undressed or malpositioned on the table, and a table with grid or filter is seldom available. Only fractures or lesions of the ventral pelvic ring and important dislocations in the dorsal pelvic ring can be recognized on these views with any certitude. If any bony lesion of the ventral pelvis is seen, another lesion of the dorsal pelvis must be suspected and searched for.

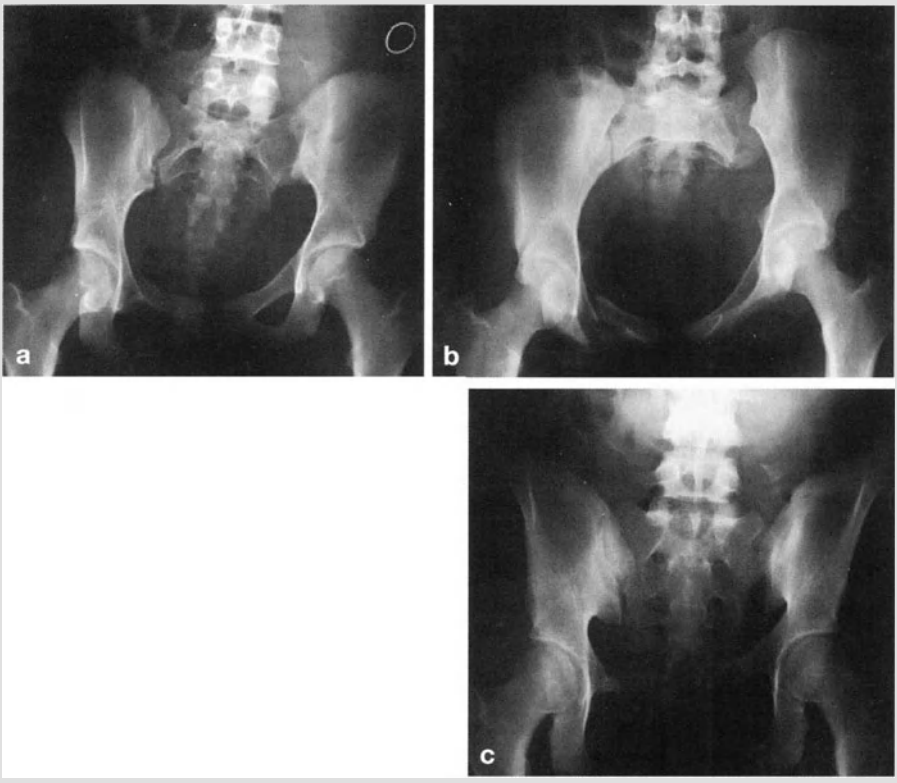

Fig. 1. a AP pelvic view of a 15-year-old girl who had been run over by a truck. **b** Pelvic inlet view. Dislocation of the left sacroiliac joint and dorsal displacement of the left iliac wing. **c** Pelvic outlet view. Diastasis of the left sacroiliac joint and fracture of the transverse process of the fifth lumbar vertebra

For a more precise diagnosis the AP view of the pelvis must be repeated under better circumstances and completed with pelvic inlet and outlet views [1, 2]. The patient is undressed and lies in a correct supine position on the table. The center of the X-ray beam is directed perpendicular to the pubic symphysis. For pelvic inlet and outlet views the patient remains in a supine position, but the X-ray beam is directed 40° cephalad, or caudad. The pelvic inlet view best demonstrates fractures or crush lesions through the anterior cortex of the lateral mass of the sacrum and through the sacral foramina. A rotational and/or ventrodorsal displacement of one hemipelvis can also be seen very well. The pelvic outlet view offers an AP view of the sacrum. Fractures through the sacral foramina or craniocaudal displacements of one hemipelvis can be discovered on this radiograph (Fig. 1).

However, even pelvic overviews of optimal quality do not always allow a proper assessment of the dorsal pelvic ring because of the superposition of multiple bones and soft tissues. Exact assessment of the sacrum and sacroiliac joints is particularly difficult. Several authors report a ratio of 30%–60% false-negative or false-positive findings in this area [3–5]. Nevertheless, a complete assessment of the dorsal pelvic ring is of the utmost importance for planning treatment. Dorsal pelvic ring injuries interfere with the transfer of body weight from the vertebral column to the lower extremities. The distinction between vertically stable and unstable pelvic ring structures, which is of great prognostic value, depends exclusively on the structural integrity of the dorsal pelvic ring.

A complete inventory of ligamentous and bony pelvic lesions can be made only with the help of computed tomography (CT), assessing the dislocation of the various fracture fragments, their relationship to the spinal canal, the degree of instability of the pelvic ring, and the severity of sacroiliac lesions [4, 6]. An additional advantage of CT is the ability directly to visualize associated intrapelvic and retroperitoneal injuries. (Fig. 2).

This information allows a correct decision as to the indication for and timing of surgery, the most appropriate operative technique, and operative approach. Disadvantages of pelvic CT are that the patient must be transported to the CT room, and that caring for vital functions is difficult when the patient is inserted into the CT machine. Whether the pelvic CT is performed immediately or after the first operative phase depends on the hemodynamic condition of the patient and the need of other urgent CT examinations (e.g., skull, thorax, abdomen; Fig. 2).

The Cervical Spine

Injuries of the cervical spine occur in about 3% of all trauma victims and account for 10%–15% of all spine injuries. In 3%–25% of patients with a cervical spine lesion delayed diagnosis, misinterpretation of radiographs or impermissible manipulation of the cervival spine leads to a deterioration in the neurological status [7–10]. During resuscitation and reanimation manipulations must be carried out before the cervical column is cleared. To prevent secondary and iatrogenic spine injuries, the cervical spine must be immobilized throughout

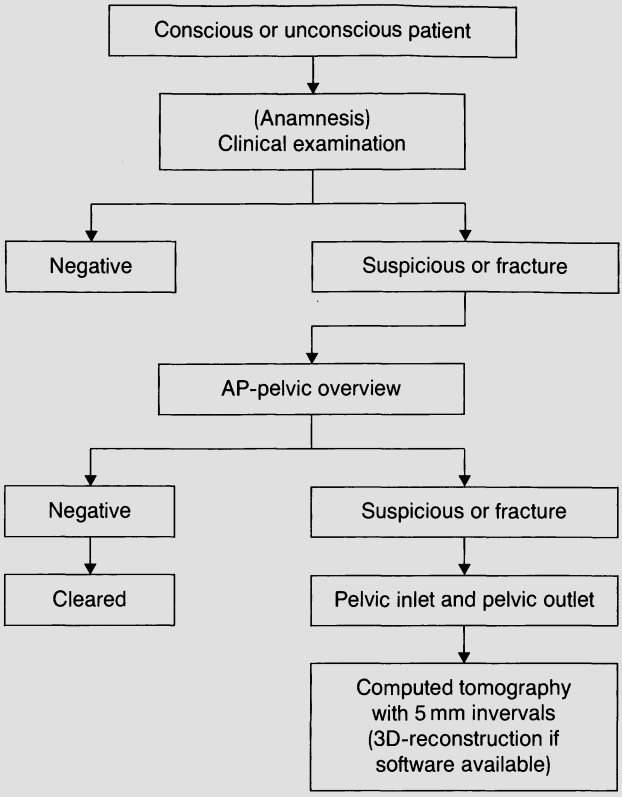

Fig. 2. Pelvic ring evaluation protocol for acutely injured patients

reanimation, until evidence or exclusion of C-spine injury has been obtained. Every trauma protocol should therefore include early X-ray examination of the C-spine, to assure that no lesion remains unrecognized (Fig. 3).

The first radiograph to be obtained is the cross-table lateral view of the C-spine. In emergency situations this is the only radiograph of the spine that is really needed. For a radiograph of good quality the C-spine must be sufficiently immobilized. In uncooperative or restless patients and in traumatized children this may require intubation and relaxation. As many lesions are localized at the craniocervical or cervicothoracic junction, it is extremely important to visualize the whole cervical spine. The cervicothoracic transition is often masked by bony or soft tissue structures of the shoulder girdle. Both shoulders can be pulled down by axial traction on the upper extremities (Fig. 4). An alternative is the "swimmer's view," in which one arm is positioned upright along the head of the patient. As it is impossible to exclude a C-spine injury with only a lateral X-ray, the C-spine remains suspected until a standard series shows otherwise. Other emergency investigations should not be delayed because of an inappropriate lateral C-spine radiograph.

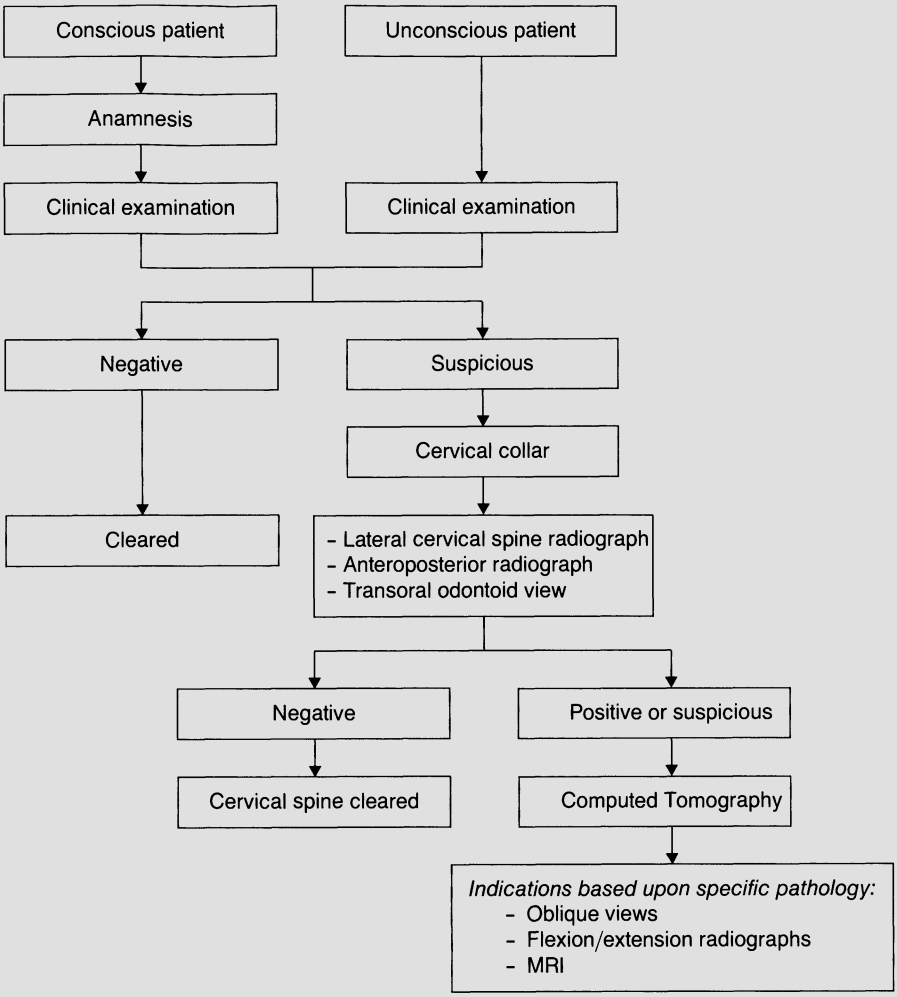

Fig. 3. Cervical spine evaluation protocol for acutely injured patients

In the "standard" C-spine series the lateral view is completed with AP and open-mouth odontoid views. To minimize misdiagnosis these radiographs are analyzed systematically. First, it is ascertained that seven vertebrae including the cervicothoracic junction are visible. Second, radiological signs of unstable spinal injuries are sought, such as displacement of vertebrae, widened interspinous spaces, widened apophyseal joints, and a widened vertebral canal [9]. A discrepancy between the anterior and posterior bone height of a vertebral body, and widening of the prevertebral soft tissue space may also indicate the presence of a C-spine injury [5].

If no lesion is found on the standard C-spine series, and the patient is conscious, and has no neck pain and no neurological deficit, further investigation

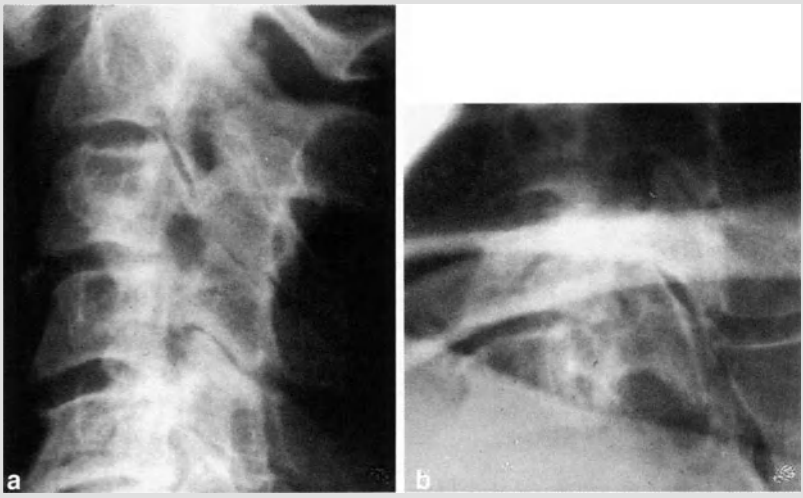

Fig. 4. a Lateral C-spine in a 65-year-old man after a traffic accident. Only the vertebrae C2 to the upper half of C6 are visible. **b** Swimmer's view shows dislocation between C6 and C7

is not necessary. In all other cases further imaging of the C-spine is required. Here CT offers the highest resolution diagnosis, with 98% sensitivity, and 100% sensitivity when combined with a three-view X-ray series of the C-spine [11]. CT is superior to any other method for evaluating the integrity of the spinal canal and detecting bony fragments in it. The quality of the images is not related to the position of the patient, and the examination can easily be combined with CT of the skull, thorax, abdomen, or pelvis.

Oblique views and/or flexion/extension views of the C-spine can also be beneficial. Oblique views enable a better interpretation of pedicles, intervertebral foramina, facet joints, and lamina. Flexion-extension radiographs are indicated when no bony lesion is discovered, but a segmental instability suspected. They must be obtained under image-intensifier control and in the presence of a physician [12].

Magnetic resonance imaging (MRI) enables the detection of abnormalities in both bony and soft tissue structures. MRI is superior to CT for detecting injuries to the spinal cord, such as intraspinal hemorrhage, cord edema or contusion, and lesions of nerve roots. However, its use in the primary diagnostic work-up of the severely injured is limited, because of the time needed for examination and because the anesthetic equipment contains iron, which cannot be tolerated in the neighborhood of the magnet. Also, adequate diagnosis of discoligamentary injuries by MRI is possible only some 2 days after injury, as the accompanying hematoma can be distinguished from fat tissue only after hemoglobin is partly degraded.

The Thoracolumbar Vertebral Column

The great majority of lesions of the vertebral column are thoracolumbar fractures. Dislocations and fracture dislocations are more frequently localized in the

cervical region, while fractures are seen more often in the thoracic and lumbar region. Lumbar fractures are twice as frequent as thoracic fractures, but most of them occur at the thoracolumbar junction. These lesions originate from high-energy accidents, such as falls from a great height, traffic accidents, and deceleration injuries in sports.

Anamnesis reveals the direction of impact on the vertebral column: vertically acting forces (fall), forced flexion-extension (frontal crash), laterally acting forces (lateral crash) or a rotational forces (swing movement). Clinical and neurological examination are especially important for localizing the injury. The sensibility and muscle force of the trunk and lower extremities are assessed, as well as the sensibility of the perineum and the function of the external anal sphincter. The latter is important to make the distinction between total paraplegia and paraplegia with sacral sparing, or to confirm the diagnosis of a cauda equina syndrome. Neurological examination enables patients to be categorized in terms of the Frankel scoring system: A, no motor or sensory function; B, preserved sensation only; C, preserved motor function (nonfunctional); D, preserved motor function (functional); and E, completely normal.

Conventional radiographs are indispensable for confirming the diagnosis. AP views of both the thoracic and lumbar vertebral column are easy to obtain in polytraumatized patients lying in the supine position. This is not the case for the lateral views, as many patients cannot be turned because of concomitant lesions, and because this manipulation is not without danger in the presence of an unstable vertebral fracture. Nevertheless, lateral views of the thoracolumbar column should be obtained, on which the alignment and form of the vertebral bodies can be evaluated.

On the AP X-rays, the following signs should be sought: diminished vertebral height, widening of a vertebral body with spreading of the pedicles, scoliotic deformity to the right or left with the tip of the angle situated on one vertebra, fracture of transverse pedicle(s), and translation of one vertebral body to the other in a horizontal plane. On the lateral X-rays the following signs can be recognized: wedge-shaped flattening or diminished height of the vertebral body, kyphotic deformity of the vertebral column with the tip of the angle situated on one vertebra, and displacement of the posterior wall of the vertebral body, and translation.

The main disadvantage of conventional X-rays is that the vertebral canal is not visualized well. CT images with sagittal or frontal reconstructions, are therefore indispensable for decision making in vertebral fractures, as they identify fractures of the vertebral arch, dislocations or fracture dislocations of the small articulations, dislocation of each fracture fragment, the presence of fracture fragments in the spinal canal, and narrowing of the spinal canal [13] (Fig. 5). Although CT scans and reconstructions of vertebral fractures are not the most urgently needed images, they can easily be combined with CT examination of the skull, abdomen, or thorax. Definitive treatment of these injuries should be based upon exact and complete radiological information. MRI has the same advantages and disadvantages as in lesions of the cervical spine. Its use in the primary evaluation of the thoracolumbar lesions is presently not widespread (Fig. 6).

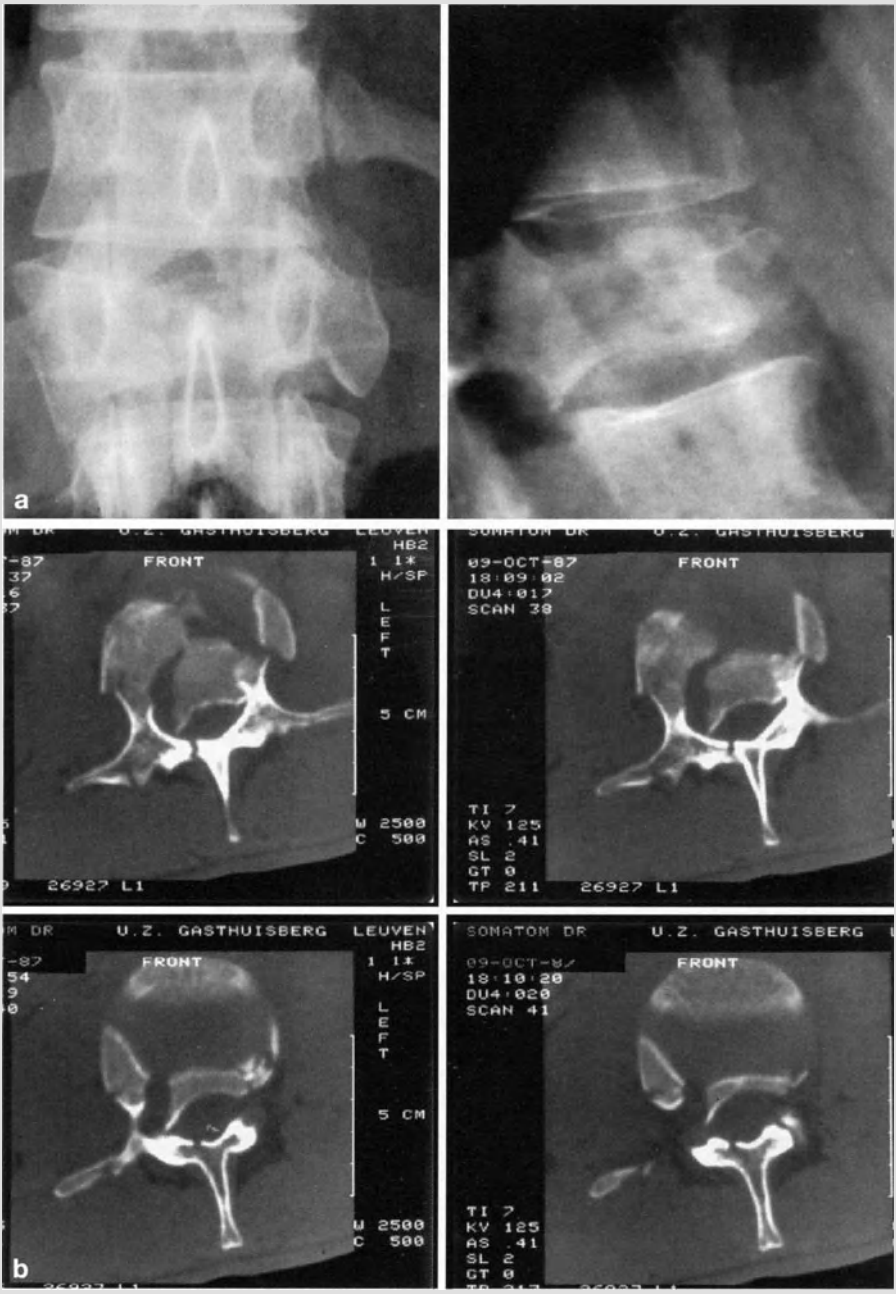

Fig. 5. a Burst fracture of L1 in a 27-year-old man after a motor vehicle accident. Note the decreased height and the broadening of the vertebral body on AP and lateral views, the vertical gap in the vertebral body on the AP view, and the protrusion in the spinal canal of the fractured posterior aspect of the vertebral body in the lateral view. **b** CT of the same level show a nearly complete obstruction of the spinal canal

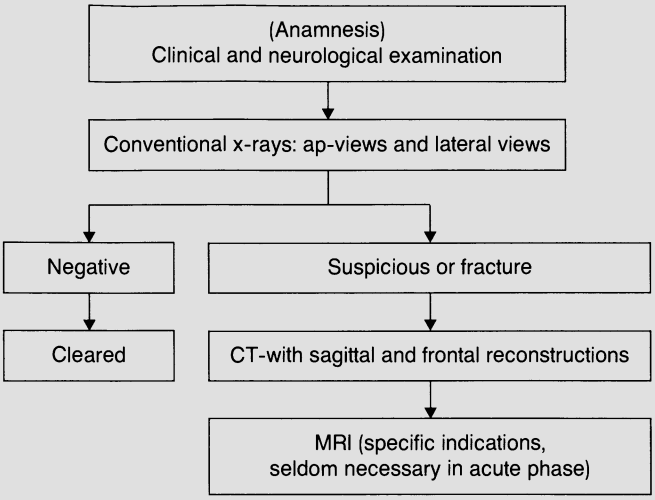

Fig. 6. Evaluation of the thoracolumbar spine

Extremities

More than 75% of patients with severe blunt injury have one or more extremity lesions. The lower extremities are more often injured than the upper ones. Severe soft tissue damage is more frequently seen in polytraumatized patients than in those with solitary lesions. Diagnosis of extremity lesions is based almost exclusively upon anamnesis, clinical examination, and conventional radiography. Responsive patients can provide important information as to sites of tenderness, pain, or altered sensibility. Clinical examination yields information on the presence of deformities, abnormally fixed position of an extremity, and soft tissue injuries. Attention is paid in each extremity to areas of deficient sensibility and altered skin temperature or color. All findings must be noted on the medical chart, indicating the need for further radiological investigations, to be performed at a suitable time, depending on the presence of other more urgent injuries.

If a neurological deficit is noted, somatosensory evoked potentials provides exclusive information immediately after injury for the correct diagnosis of neurological damage. The EMG shows alterations only some 3 weeks after trauma, and must therefore be delayed.

Evaluation of Soft Tissue Damage. Correct assessment of soft tissue damage is very important for deciding on the appropriate surgical therapy. Soft tissue damage in closed fractures is classified according to Tscherne [14], and open fractures according to Gustilo [15] (Table 1). The Gustilo classification grades open fractures in terms of the mechanism of injury, degree of soft tissue damage, fracture configuration, and level of contamination. In type I open fractures, the wound is less than 1 cm long, usually originating with a piece of bone piercing the

Table 1. Classification of soft tissue injuries in closed and open fractures

	Soft tissue damage	Fracture severity	Contamination
Closed Injuries			
(Tscherne classification)			
Grade 0	–	+	–
Grade I	+	+ to ++	–
Grade II	++	+ to +++	–
Grade III	+++	+ to +++	–
Open injuries			
(Gustilo classification)			
Grade I	+	+ to ++	+
Grade II	++	+ to +++	++
Grade IIIa	+++[a]	+ to +++	+++
Grade IIIb	+++[b]	+ to +++	+++
Grade IIIc	+++[c]	+ to +++	+++

[a] Sufficient soft tissue coverage.
[b] Insufficient soft tissue coverage.
[c] Arterial injury.

skin. There is little soft tissue damage and no signs of crush. The fracture type is generally simple. In type II open fractures, the wound is larger than 1 cm, but there is no extensive soft tissue damage. The fracture type, crush lesions, and wound contamination are moderate. In type III open fractures, there is extensive soft tissue damage to the muscles, skin, and/or neurovascular structures. The wound is severely contaminated. Type IIIA open fractures have sufficient coverage of the fracture site with vital soft tissues. Type IIIB fractures are characterized by periosteal stripping and insufficient soft tissue coverage after débridement. Type IIIC fractures are associated with arterial injury, which requires operative repair.

Closed soft tissue injuries are divided into four levels of severity [14]. In Grade 0 lesions soft tissue damage is absent or negligible. The fracture has a simple configuration. In Grade I injuries there is superficial damage caused by pressure of a fracture fragment from within. The fracture has a moderately severe configuration. In Grade II soft tissue damage and direct trauma has caused a deeper contusion, combined with skin and muscle damage. The fracture configuration is moderate to severe. Impending compartment syndromes are included in this category (see Rommens "Prevention of Local Complications," this volume). In grade III closed soft tissue damage the skin is extensively contused, and muscle damage may also be severe. To this category belong fractures with major vessel injury, decompensated compartment syndromes, and extensive subcutaneous avulsions. Although a precise clinical examination of the extremities may reveal most of the soft tissue damage, the ultimate categorization may not be possible until operative exploration and débridement have been performed.

Conventional X-Rays. X-rays must be obtained for all extremity lesions in two directions: AP and lateral. If this is not possible, two X-rays perpendicular to each other must be taken. In long bone fractures the proximal and distal articulation must also be visualized, as fractures of the diaphysis may run as a fissure into the proximal or distal joint. Also, some fractures are associated with dislocations of the proximal or distal joint (e.g., Monteggia, Galeazzi, and Maisonneuve fracture dislocations). This information may totally change the treatment of the fracture. If intra-articular fractures are present, additional oblique views may be desirable. For optimal X-ray assessment, pneumatic or vacuum splints are much more suitable as temporary immobilizers than metal or thermoplastic splints.

In the stepwise search for injuries in the polytraumatized patient, less important lesions may initially be missed, such as fractures or dislocations of small articulations of fingers or toes [8]. Even dislocations of the femoral head or shoulder joint have been missed. Ligamentous lesions are not visualized on X-rays, even when severe, and should be systematically looked for clinically, especially in the knee joint and the Lisfranc articulation. Delayed or missed diagnosis may lead to life- or limb-threatening complications and to permanent functional impairment. This is best prevented by repeated, thorough clinical and radiological examination of the patient, after reanimation and life-saving surgery, and after the primary operative phase.

Angiography. Vascular lesions, although rare in extremity injuries, dramatically aggravate the prognosis of an extremity injury and must be recognized and treated early to prevent severe functional sequelae. The first clinical examination of the patient must include evaluation of the vascular status of the extremities. Skin color, temperature, capillary refill, and peripheral arterial pulses must be controlled on both sides and compared with each other. Clinical symptoms may be misleading when the patient is hypothermic – when hands and feet feel cold, look pale, and are swollen by soft tissue damage. When there is doubt, Doppler sonography is necessary to evaluate continuity of the arteria brachialis, radialis, ulnaris, and arteria dorsalis pedis and tibialis posterior in the lower extremities. If available, color duplex sonography is preferred. If doubt still persists, angiography must be performed as soon as possible. Arterial digital substraction angiography can be carried out more quickly than selective arteriography, and with less contrast solution [16]. If high-contrast resolution is necessary in a specific area of the extremity, selective arteriography is the better solution (Fig. 7). Single-shot angiography may be helpful in emergency procedures for perforating injuries.

There is a greater risk of vascular lesions in mangled extremities, extremities with serial fractures, supracondylar fractures of the humerus, elbow dislocations, severely displaced distal femoral fractures, knee dislocations, and proximal tibial fractures. Also, after closed reduction and operative fracture treatment, the vascular status of the traumatized extremity must be controlled a number of times to be able to recognize impending or total ischemia immediately.

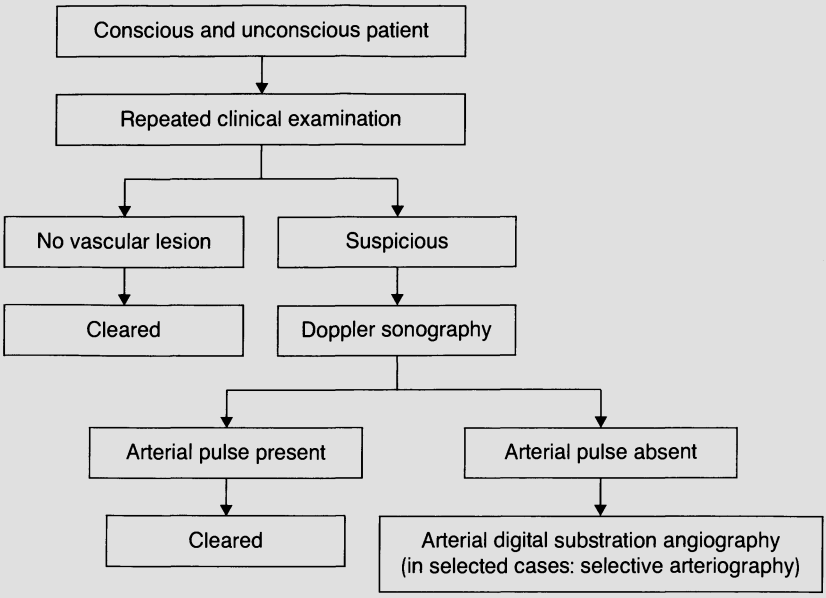

Fig. 7. Evaluation of vascular lesions

Ultrasound Examination. Ultrasound examination is recommended for the detection of tendon lesions (e.g., patella tendon, achilles tendon), muscle ruptures, fluid collections in the soft tissues and intra-articular lesions (e.g., acromioclavicular joint, rotator cuff), and non-radio-opaque foreign bodies (e.g., wood, plastics). The diagnostic procedures for most of these injuries do not belong to the acute posttraumatic phase.

The Acetabulum. Fractures of the acetabulum are more frequent in polytraumatized patients than as a solitary lesion. The diagnostic procedures needed to assess an acetabular fracture properly have no priority in polytraumatized patients, but must be performed with great care. The acetabulum is a three-dimensional structure which originates from the fusion of three bones: os ilium, os ischii, and os pubis. These three bones are oriented in different planes, which do not coincide with the frontal, sagittal, or coronal plane. Classical X-rays project the three-dimensional pelvis in one plane, with superimposition of soft tissues, bowel contents, and bony structures.

An AP view of the pelvic ring will already have been obtained in the emergency room. If a fracture line is detected in the neighborhood of the acetabulum, ala and obturator views are obtained. For the ala view the patient is rotated 45° to the traumatized side, and for the obturator view, 45° to the nontraumatized side, while the X-ray beam remains perpendicular to the X-ray table (Fig. 8). A thorough study of these three overviews should make it possible to recognize most of the fracture patterns.

CT provides more detailed information about the bony structures of the acetabulum and the surrounding soft tissues. CT images visualize transsections of

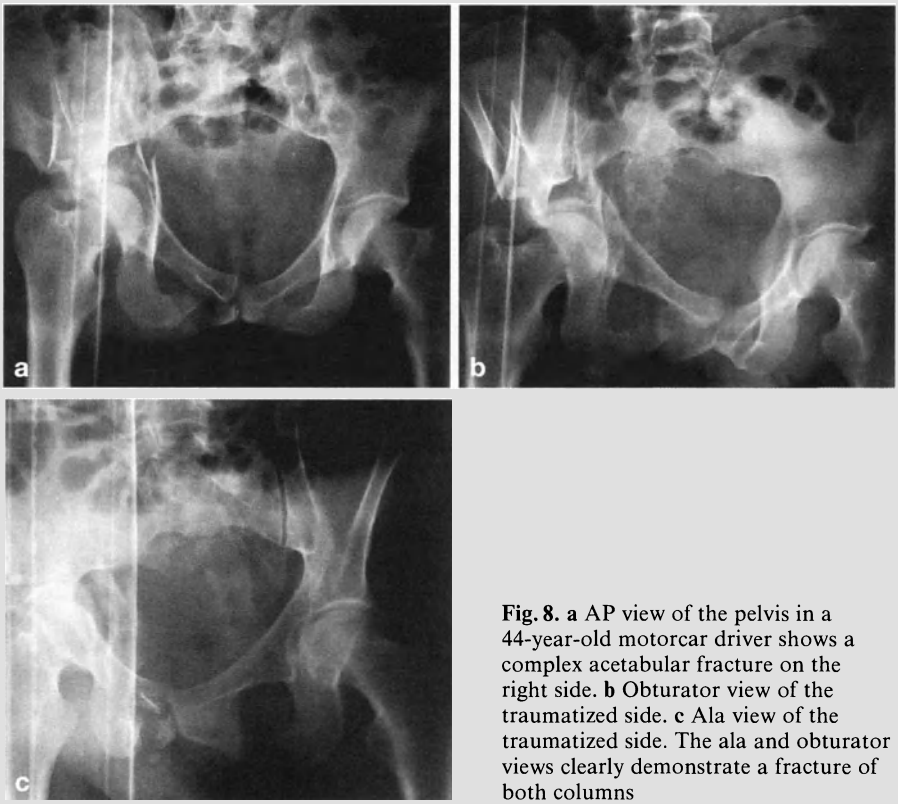

Fig. 8. a AP view of the pelvis in a 44-year-old motorcar driver shows a complex acetabular fracture on the right side. **b** Obturator view of the traumatized side. **c** Ala view of the traumatized side. The ala and obturator views clearly demonstrate a fracture of both columns

the acetabulum at a specific level. The fracture must then be reconstructed, following the fracture lines on the consecutive images. This requires three-dimensional conceptualization, and experience (Fig. 9). Three-dimensional images give the same information of the acetabular fracture as do CT images, but the acetabulum is exposed as a whole, which facilitates interpretation of the lesion. The articulation can be analyzed from each direction. Even direct visualization of the lunate cartilage is possible once the femoral head is substracted from the images.

Conclusion

Optimal management of polytraumatized patients is based upon proper understanding of the urgency and severity of all injuries. It is the task of the trauma surgeon to decide upon the sequence of diagnostic and therapeutic procedures, knowing the possibilities and limitations of each diagnostic procedure, in order to avoid delay and the missing of single lesions.

Repeated clinical examination of the patient remains the basis of the diagnostic work-up. A high-quality lateral X-ray of the cervical spine followed by an AP view

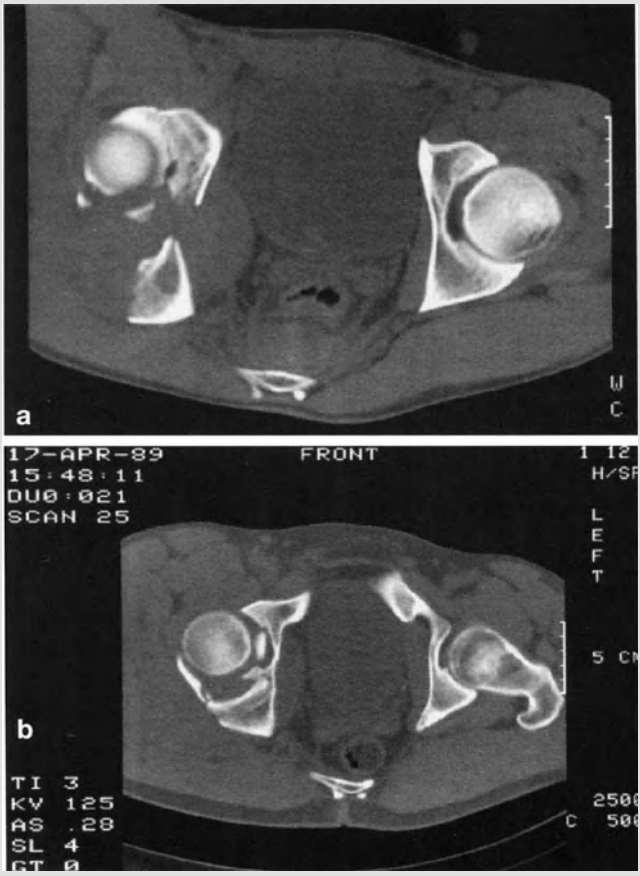

Fig. 9. a CT of the acetabulum showing significant dorsal displacement of the dorsal column. **b** CT of the acetabulum showing dorsal impaction fracture with an intra-articular fracture fragment

of the thorax and the pelvis, must be obtained before any other investigation. Evaluation of extremity injuries includes correct examination of soft tissue damage and conventional X-rays in two directions. Specific injuries may require angiography, tomography, CT, or ultrasound examination. If necessary, examinations must be repeated until all injuries are identified or excluded [17].

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Anesthesiological Management of the Severely Traumatized Patient in the Operating Theatre

L. H. D. J. Booij

Introduction

Severely injured patients often require anesthesia for diagnostic procedures, emergency surgery, and delayed surgery. The objective of trauma anesthesia is:
(a) to stabilize and support the vital organ functions,
(b) to suppress and/or prevent the stress resulting from diagnostic and surgical procedures,
(c) to start or continue correction of the physicochemical imbalances resulting from trauma and surgery, and
(d) to initiate measures that guarantee pain treatment and stress prevention in the postoperative period.

The Emergency Room

Anesthetic care for the trauma patient in the hospital should start in the emergency room and be continued in the operation theater up to the ICU, preferably by the same team. Meticulous monitoring of the patient, looking for changes in signs, can guide supportive treatment and prevent unnecessary morbidity and mortality. Such monitoring should include:
(a) mental status (level of responsiveness; Glasgow Coma Scale, GCS),
(b) respiration (rate, volume, symmetry, quality, end-tidal CO_2, pulse oximetry, chest X-rays),
(c) circulation (ECG, blood pressure, heart rate, urine output, blood loss, fluid administration),
(d) metabolic status (blood gases, electrolytes, blood urea nitrogen, blood sugar, temperature), and
(e) neurolgical status (mental status, motor function, muscle tone, cervical X-rays).

Pitfall 1: The Inexperienced Anesthesiologist

Trauma anesthesia is technically difficult (intubation problems, difficult vascular access, medical problems, etc). Trauma patients belong to all age groups, may

have various concurrent diseases, and use various drugs. Frequently information about these matters is not available. Trauma patients can be pregnant, intoxicated with alcohol or drugs, and have a full stomach. Furthermore, surgery may involve all areas of the human body. Any of these factors may entail specific problems and complications. Therefore trauma anesthesiologists should have a substantial experience in the whole field of anesthesia. Trauma care is not a field for inexperienced staff and newcomers.

Pitfall 2: Lack of Information on the Mechanism of Trauma

Many of the problems arising during anesthesia are the result of the mechanism of injury. Therefore the anesthesiologist should know whether the trauma was blunt, penetrating, thermal, or mixed. Was deceleration (traffic, fall), fire (burn, smoke, soot), electricity, chemical substances (inhaled, ingested, skin contact), biological material, a penetrating projectile (gunshot), or nuclear radiation involved? Knowledge of the mechanism of injury may help in identifying occult problems and avoiding iatrogenic complications of the existing condition. Examples include the presence of a fractured cervical vertebra in relation to endotracheal intubation, and the presence of an undiagnosed pneumothorax in relation to the development of a tension pneumothorax, due to positive pressure ventilation or the use of nitrous oxide. Information is also necessary – but frequently not available – on other topics, such as the existence of allergies, concurrent diseases, co-medication, and last ingestion of food. Furthermore, supporting data for anesthetic care such as laboratory testing, pulmonary function tests, and ECG often are not available. Important information, however, can be obtained from scoring systems such as the GCS, the Injury Severity Score, or the Revised Trauma Score. The ASA physical status classification system has also proven to be of great value in trauma anesthesia. Lack of information on these factors may increase the risk of anesthesia because potential problems cannot be anticipated.

Pitfall 3: Sedation Is Unsafe

Patients in the emergency room often are anxious, agitated, fearful, and uncooperative with diagnostic and treatment procedures. This is especially a problem in intoxicated patients and in those with head trauma. Pharmacolgical sedation of these patients is dangerous because they may lose control of their airways, vomit, and aspirate, and may become apneic. With general anesthesia and endotracheal intubation, airway control is guaranteed. Many of these patients need further anesthesia for surgical treatment in any case, while others need intubation for artificial ventilation in the ICU. Sedative drugs may also produce hypotension. Thus, anesthetizing these patients in the emergency room is the safest method. In the emergency room a number of vital parameters should be monitored; these are discussed below. In the ICU sedation is frequently indicated. Patients needing sedation are generally intubated and artificially ventilated; thus

respiratory side effects are not as dangerous as in the prehospital period or in the emergency room. It must be remembered that muscle relaxants do not sedate the patients but only paralyze, causing severe stress in the awake patient. A problem especially in the ICU is the accumulation of drugs. This leads to prolonged effects and difficulty in weaning the patient from artificial ventilation.

Pitfall 4: The Unwanted Side Effects of Anesthetics and Muscle Relaxants Used for Intubation

Airway management is an important task in the emergency room. Some patients (the deeply comatose) can be intubated without administration of anesthetics or muscle relaxants; however, the majority must be anesthetized and paralyzed first. Administration of such drugs may lead to problems. Anesthetic agents may cause vasodilatation, resulting in hypotension and aggravation of shock. Muscle relaxants may cause problems depending on the type and the pharmacological properties of the individual drug. The most frequently used relaxant for intubation in emergency care still is suxamethonium, a depolarizing relaxant with rapid onset and short duration of action. It is associated with many side effects, including potassium release, fasciculations, and muscarinic effects. Hyperkalemia after suxamethonium administration is seen especially in patients with neuromuscular diseases and after burn trauma. In children suxamethonium frequently leads to initial rigidity of the masseter muscles, making endotracheal intubation difficult or even impossible. Other side effects include increased intragastric and intra-abdominal pressure, with increased risk of regurgitation and aspiration, and increased intracranial and intraocular pressures, which is undesirable in patients with head injury or ocular injury. Most nondepolarizing muscle relaxants have a slow onset of action, resulting in a delay of 4–6 min between their administration and the possibility to intubate the trachea. Rocuronium bromide, a new steroidal nondepolarizing muscle relaxant, has a rapid onset of action, making endotracheal intubation possible within 1 min, and does not increase intracranial or intraocular pressure [1]. The intubation conditions are reported to be similar to those after suxamethonium. Its duration of action is comparable to that of vecuronium bromide, thus markedly longer than that of suxamethonium. It can, however, be easily reversed, even at deeper neuromuscular blockade. Rocuronium bromide thus seems to be the relaxant of first choice to facilitate endotracheal intubation in trauma patients.

Pitfall 5: Problems with Endotracheal Intubation

Endotracheal intubation is indicated in patients with a GCS score of less than 9, class III or IV shock, persistent hypoxemia, airway obstruction, and in the uncooperative patient requiring evaluation. Intubation may also be needed in anticipation of future problems, i.e., in patients with inhalation injury, when edema of the pharynx and glottis may occur, when intubation in a later stage is

extremely difficult or even impossible. Also prophylaxis of respiratory failure requires artificial ventilation in a number of situations. The technical difficulties associated with endotracheal intubation of a severely traumatized patient, may aggravate the risk of cervical spinal injury, regurgitation and aspiration, and other intubation complications [2].

Use of fiberoptic laryngoscopy and blind nasal intubation have been advocated. Blind nasal intubation, however, requires more manipulation of the neck and has a higher failure rate than oral intubation. Furthermore, nasal intubation increases the risk of infection and bleeding in the presence of nasal trauma or maxillofacial fractures, and sinusitis may develop. In the presence of fractures of the cribriform plate, fault route intubation into the cerebral substance has been described, as with the introduction of nasogastric tubes. Nasal intubation is slower in execution than oral intubation [3]. For all these reasons nasal intubation should be abandoned as the initial route of intubation in trauma patients, and oral intubation preferred.

With oral intubation, direct visualization allows inspection of the upper airway, glottis, and first part of the trachea and cleaning the airway of foreign debris. Direct laryngoscopy can be used in patients with cervical injury when intubation is performed by an experienced anesthesiologist, and if the head is fixated, with axial traction applied by a third person [4].

Intubation may be technically impossibile because of upper airway damage, in maxillofacial trauma, edema formation, inability to open the mouth. Or the presence of an orofacial wound itself may interfere with endotracheal intubation. In such situations ventilation with a mask and bag may also be impossible, and immediate tracheostomy or cricothyroidotomy is indicated. Cricothyroidotomy should not be performed in those under 6 years of age since at this age the cricoid ring is the narrowest portion of the airway, and scar tissue formation may result in progressive airway obstruction.

When damage to the lower airway is suspected, bronchoscopy is indicated prior to intubation. If a tracheal injury is found, the endotracheal tube is passed over the fiberoptic bronchoscope and distally from the injury. In the presence of distal tracheal lacerations the use of extracorporeal oxygenation must be considered. Unless the exact location of the tracheal injury is known, cricothyroidotomy or transtracheal ventilation is contraindicated.

Many recommend intubating severely injured patients in deep shock without the administration of any drugs, or after administration of muscle relaxants only. However, laryngoscopy and intubation result in an important transient increase in intracranial pressure and in severe cardiovascular responses, which can be prevented only by adequate anesthesia [5]. In my opinion, anesthetic agents thus should always be administered when intubating patients with head injury.

Pitfall 6: The Risk of Aspiration

After trauma, gastric emptying may be delayed for 24–48 h, and frequently the patient ingested food or alcohol shortly before injury. Aspiration may occur

because the protective pharyngeal reflexes are abolished by induction of anesthesia and administration of muscle relaxants, or in comatose patients. Furthermore, the presence of a full stomach, anxiety, and pain from trauma, the consumption of alcohol and/or drugs, and elevated intracranial pressure favor vomiting, regurgitation, and aspiration [6]. To prevent aspiration (and gastric insufflation) cricoid pressure should be applied and maintained during mask ventilation and after administration of muscle relaxants, until endotracheal intubation is completed [7]. Insertion of a gastric tube to evacuate gastric contents is controversial, as it may induce vomiting and may interfere with the lower esophageal spincter mechanism. When vomiting occurs, solid particles may cause bronchial obstruction, which should be treated by cleaning of airways and bronchi. The development of aspiration pneumonitis depends on the amount and acidity of the aspirated material [8]. It is not absolutely prevented by antacids. Thus, rapid sequence induction under application of cricoid pressure (Sellick's maneuver) remains the most valuable prevention of aspiration. If aspiration occurs, application of PEEP ventilation, increase in inspiratory oxygen concentration, administration of broad spectrum antibiotics, and correction of acid-base balance disturbances are important. Administration of steroids is probably of no value.

Pitfall 7: Treatment of Pain in the Emergency Room

Pain induces deleterious effects on many physiological functions (see Kerz and Dick, this volume). However, pain treatment may mask some of the symptoms of the injury and induce respiratory depression when narcotic analgesics are administered. With proper titration of analgesics the fear for respiratory depression and decrease in consciousness is unrealistic. In a number of cases peripheral nerve blocks (including intercostal blocks in chest trauma), or regional anesthetic techniques may be applied. In the severely injured patient, systemic administration of narcotic analgesics is, in my opinion, indicated to prevent pathophysiological changes. In some cases the administration of nitrous oxide may be considered, keeping in mind its deleterious effects on pneumothorax, intracranial pressure, and distension of lacerated bowels. Pain treatment is certainly indicated during surgical procedures and in the ICU. All available techniques (intravenous, regional, etc.) are valuable there. For narcotic analgesics it is recommended to use intermittent dose techniques in the emergency room. In the operating room continuous infusion may be considered. In both situations preferably short-acting drugs are used.

Pitfall 8: The Intoxicated Patient

The association of alcohol and drugs frequently makes the patient uncooperative [9]. Mortality in intoxicated trauma patients is four times higher than that in nonintoxicated patients [10]. Alcohol can also cause cardiac dysrhythmias, and

vasodilation with hypothermia. The effect of many anesthetics is potentiated and prolonged by alcohol intake [11]. Narcotic abuse may result in central nervous system and respiratory depression. Myosis is a common discriminatory sign in such patients. Cocaine can cause severe effects on the cardiovascular system, with increased coronary artery resistance [12]. This can lead to acute myocardial infarction if oxygen consumption is increased or supply decreased. Cocaine can also induce ventricular fibrillation, asystole, and epileptic seizures. Cocaine-addicted patients in shock can have a relative bradycardia [13]. Cocaine increases the minimal alveoloar concentration (MAC) for halothane. Withdrawal effects may develop, depending on the intoxicating substance. These effects must be treated either symptomatically or by substitution of the substance on which the patient is dependent. Important opioid withdrawal symptoms with implications for anesthesia are: nausea and vomiting, lacrimation or rhinorrhea, pupillary dilatation, sweating, diarrhea, fever, abdominal pain, anxiety, and insomnia.

The Operating Room

Monitoring of vital signs and treatment of respiratory and cardiovascular problems which began in the emergency room are continued in the operating room. Other monitoring parameters are added, depending on the type and extent of the surgical procedures to be performed. Monitoring should include devices for the following:

- Ensuring adequacy of ventilation and oxygenation: pulse oximetry, oxygen concentration, capnography, anesthetic gases, arterial blood gas analysis, airway pressures, ventilatory rate and volume, mixed venous oxygen saturation
- Ensuring hemodynamics and tissue perfusion: ECG, ST segment analysis, nonivasive blood pressure, intra-arterial pressure, central venous pressure, cardiac output, pulmonary arterial pressure, pulmonary capillary wedge pressure, urinary output, blood loss, hemoglobin concentration, hematocrit
- Regulating the temperature: central and peripheral temperature probes, heating blanket/matress, fluid warming systems, humidification of anesthetic gases
- Monitoring metabolic and clotting status: electrolytes, blood urea nitrogen, glucose level, blood gas analysis, determination of coagulation factors, bleeding time
- Evaluating neurological status: intracranial pressure, electroencephalogram, somatosensory evoked potentials, pupil diameter
- Evaluating neuromuscular transmission

Pitfall 9: Increased Risk in Acute and Trauma Anesthesia

Age, emergency surgery, and poor physical status are clearly factors that increase anesthetic morbidity and mortality. The overall risk of anesthesia-related mortality is estimated to be approximately 1–2 in 10000 patients. These morbidity

and mortality rates double when the same operation is executed as an acute case. Also in elderly, the risk is increased. Some 75% of anesthesia-related morbidity and mortality is the result of human failure. In the haste involved in emergency resuscitation, patient transfer, and trauma surgery human errors are likely to occur more frequently. Also, the higher the ASA classification, the more likely it is that a complication will lead to fatal outcome [14].

Pitfall 10: Insufficient Monitoring and Vascular Entrances

In trauma patients monitoring of vital functions is necessary for anesthesia safety and serves a therapeutic guidance role. With monitoring it is easier to evaluate the results of the cardiovascular and respiratory support provided, and to evaluate other physiolgical functions such as urinary output, metabolism, and neuroendocrine functions. All monitoring needed during the long surgical procedures should be started before surgery because, after surgery has started it is often impossible to reach the patient for further application of devices. This is also the case for inserting an urinary catheter and for acquiring vascular access. At least two large-bore intravenous catheters, and regularly also an intra-arterial line, are thus demanded from start. In some cases a multilumen catheter for cardiac output measurement must be introduced for the same reason. If abdominal or thoracic vessel cross-clamping is anticipated, such intravascular catheters must be placed in a position that the circulation will be reached under all circumstances, i.e., in the upper part of the body. The parameters to be monitored are listed above.

Pitfall 11: Anesthesia in the Patient in Shock

Acute surgery is frequently required for uncontrollable shock. While shock is an important cause of early mortality, slow and inadequate correction of shock contributes to the development of sepsis, multiorgan failure, and late mortality. Shock and its treatment is extensively discussed in other chapters of this volume, but because of its impact on anesthesia some aspects are repeated here. Hemorrhagic shock can be divided in four classes in terms of degree and signs (Table 1). It must be realized that, as mentioned above, cocaine addicts in shock can have relative bradycardia. Also, the use of beta-blockers may influence the heart rate response. Class III and IV shock are absolute indications for endotracheal intubation and artificial respiration because ventilatory rates of 35 and more demands an oxygen consumption that exceeds oxygen supply. Thus, in these conditions the amount of oxygen available for the other tissues decreases. Shock does interfere with the induction of anesthesia. Most anesthetic agents have sympatholytic effects, and thus result in vasodilatation. Many also have negative inotropic and chronotropic effects on the myocardium. Thus decreases in heart rate, blood presure, and cardiac output occur during induction of anesthesia. Because of hypovolemia the initial volume distribution of anesthetic drugs is

Table 1. Classes of shock (signs are for adult patients)

Signs	Shock classes			
	I	II	III	IV
Blood loss (ml)	< 750	750–1500	1500–2000	> 2000
Loss% total (%)	< 15	15–30	30–40	> 40
Heart rate (1/min)	> 100	> 100	> 120	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse width	Normal	Decreased	Decreased	Decreased
Respiration rate(1/min)	14–20	20- 30	> 35	> 35
Urinary output (ml/h)	> 30	20–30	5–15	Negligible

decreased, leading to increased peak plasma concentrations and to more marked pharmacological drug effects. Therefore careful induction with reduced dosages of the drugs is absolutely required.

Pitfall 12: Crystalloids or Colloids

The aim of fluid resuscitation is to restore circulating blood volume and extracellular extravascular volume, thereby maintaining adequate oxygen transport, and to correct coagulopathies. Fluid resuscitation thus concerns administration of fluids, blood, and blood components (Mondy and Blaisdell, this volume). A number of points must be mentioned in the context of the present chapter. Crystalloids replace whole body water loss very effectively, whereas colloids restore circulating blood volume and oxygen consumption more efficiently. When more than 30% of the circulating blood volume is lost, blood or blood components must be administered. When less than 30% is lost, only administration of crystalloids is sufficient in an amount of three to four times the volume lost. A practical approach is to start in both cases with 1–2 l crystalloids, followed in the latter case by 1–2 U colloids or packed red cells.

Important in shock patients is the restoration of the oxygen-carrying capacity, i.e., hemoglobin concentration (transport capacity), cardiac output (volume), and partial oxygen pressure (saturation). A hematocrit of 0.28–0.30 seems optimal. The pathophysiolgical rationale for blood transfusion is thus correction of a lowered oxygen transport capacity that accompanies a lowered hematocrit value. In the early phase of treatment completely cross-matched blood or blood components are frequently not available, and type 0 rhesus negative blood is administered. Autotransfusion of shed blood is a valuable method to limit the use of homologous blood for replacement. Large quantities can be collected from the four body compartments, which can contain significant amounts of blood, i.e., the abdomen, thorax, major extremity injuries (femur, pelvis), and retroperito-

neum. The shed blood is washed to remove bacteria, tissue debris, and other contaminants. The erythrocytes are retransfused to the patient after centrifugation.

Pitfall 13: Massive Blood Transfusion

Massive transfusions, exceeding the patient's own blood volume (10 U or more) are a potential source of many problems. Coagulation disorders may occur from insufficient amounts of coagulation factors available in the transfused blood (platelets and factor VIII). Furthermore, disseminated intravascular coagulation may be triggered by thromboplastins released from injured tissue and hematomas. Administration of platelets and/or fresh plasma or cryoprecipitate may become necessary. Platelet concentrates contain significant levels of coagulation factors (II, VII, IX, XI, XII in normal and V, VIII in lower amounts) [15]. Massive transfusion may result in hypothermia, which may induce coagulation disorders and shivering, with increased oxygen consumption. If citric acid is used as anticoagulant, citric acid intoxication may occur in the form of hypocalcemia and acidosis. Because administration of calcium is accompanied by adverse effects more frequently than citric intoxication is seen, calcium administration can no longer be advocated. Acidosis as a result of infusion of old blood with a low pH should be corrected only on the basis of blood gas analysis because the degree of acidosis is variable, while overcorrection may lead to alkalosis with cardiac arrhythmias and hypernatremia [16]. Although large amounts of potassium are infused with massive transfusion, hyperkalemia does not constitute a real problem. Not only do most trauma patients have a large intracellular shift of potassium, but, potassium is also rapidly excreted via the kidneys. Old blood has a decreased 2, 3-diphosphoglycerate level, which results in an increased hemoglobin affinity for oxygen and a decreased dissociation of oxygen from hemoglobine in the tissues.

Pitfall 14: Choice of the Anesthetic Technique

In severely injured patients general anesthesia is the main choice. Because most anesthetics decrease myocardial contractility and cause vasodilatation, the drugs and their dosage must be meticulously chosen [17]. There is no ideal anesthetic drug in trauma patients, and it is likely that the skills and knowledge of the anesthesiologist is of more importance than the choice of drug. Thiopentone must be administered with care because it is a strongly vasodilating drug, causing myocardial depression. Ketamine is an anesthetic with sympathomimetic effects. It increases heart rate and blood pressure, which may be desirable in shock patients; however, it also increases intracranial pressure. Etomidate decreases corticosteroid synthesis and release, and in shock patients leads to further decrease in blood pressure. Midazolam has the benefit of a strong amnesic effect but has a slow onset and also is a cardiovascular depressant. The

vasodilating effect caused by propofol is more pronounced in hypovolemic patients. Inhalational anesthetics all have strong cardiodepressant and vasodilatory effects. Nitrous oxide furthermore reduces the inspired O_2 concentration, and shifts the oxygen dissociation curve to the right. Because of its rapid diffusion and high blood/gas partition coefficient, it also increases the volume of sequestered gas (pneumothorax, bowel distension, pneumoencephaly). It therefore should not be used if the circulating volume has not yet been restored, and if expansion of closed gas spaces has not been controlled. Halothane and enflurane depress the baroreceptor responses, impairing reflex tachycardia. All inhalational anesthetics increase cerebral blood flow and intracranial pressure, especially in spontaneously breathing patients (CO_2-mediated effect). Nitrous oxide also has this effect [18]. Both intravenous and inhalational anesthetics decrease cerebral oxygen consumption, and thus have potentially protective effects on the injured brain.

Muscle relaxation is generally required in trauma patients. Suxamethonium has many disadvantages (see above). It nevertheless is used for rapid intubation of trauma patients. The steroidal muscle relaxants (pancuronium, vecuronium, pipecuronium, rocuronium) can be used safely; however, histamine release and subsequent bronchospasm, hypotension, and anaphylactoid reactions may occur with the dibenzylisoquinolines (tubocurarine, atracurium, mivacurium, doxacurium). Morphine and meperidine may also release histamine. Fentanyl and its analogues are safer in this regard, but may induce more pronounced muscle rigidity and bradycardia. All opioids should be titrated according to the need of the individual patient. Narcotic analgesics should not be administered to spontaneously breathing head injured patients because of the risk of respiratory depression and subsequent increase in intracranial pressure. The pharmacokinetic profiles of all drugs are altered in trauma patients. These result in different pharmacodynamic characteristics, which must be anticipated. In general, decreased doses of anesthetic drugs are needed. Also the duration of action of drugs is prolonged, especially when the drug is extracted in the liver [19]. Their elimination depends on liver blood flow, which is decreased in shock. In choosing the proper drug for anesthesia, the transfer to, and later treatment in ICU, should also be taken into consideration.

In some situations locoregional is preferred, usually in combination with general anesthesia, i.e., for reimplantation of (parts of) extremities. Locoregional techniques promote tissue perfusion and graft survival. It must be recalled that regional anesthesia is contraindicated in patients with coagulation disorders because of the risk of bleeding at the puncture site. Spinal anesthesia and probably epidural anesthesia are contraindicated in patients with increased intracranial pressure because of the risk of accidental dural puncture. When liquor is lost, acute herniation of the cerebellum (foramen magnum) or cerebrum (tentorium) may occur. This can be lethal or result in increased brain damage.

Pitfall 15: Long Duration of Operations

It has been demonstrated that a favorable outcome is more likely in multiple trauma patients when all injuries are corrected at initial surgery [20, 21]. Emergency surgery in these patients can therefore take a long time, which brings special problems. After induction of anesthesia neurological examination is almost impossible because the reflexes are depressed. Meticulous attention to minor clinical signs of neurolgical detoriation is thus important. Unexplainable cardiac arhythmia, unexplainable changes in hemodynamics or respiration, in pupil size (dilatation) and position, and in body temperature must alert one to a possible deterioration of the central nervous system. If possible, and depending on the existing trauma, neurophysiolgical monitoring must be used, using echo-Doppler or EEG. Other problems of prolonged anesthesia are accumulation of anesthetic drugs, hypothermia, massive blood loss, and loss of vigilance of the team.

Pitfall 16: Hypothermia

Severe trauma patients frequently arrive in the hospital with hypothermia [22]. Exposure of large parts of the body, open chest and abdomen during prolonged surgery, and massive blood or fluid infusion contribute to the development of hypothermia during treatment. Patients with drug or alcohol intoxication often have extreme vasodilatation, making them more prone to heat loss. Hypothermia affects all organ systems, alters blood coagulation, increases blood viscosity, induces peripheral vasoconstriction, causing increase in cardiac workload and dysrhythmias, and decreases heart rate, cardiac output, and blood pressure [23]. Hypothermia also shifts the oxygen dissociation curve to the left, while the metabolism and excretion of drugs is decreased. In the early stages of hypothermia ventilation is centrally stimulated, but ventilation then decreases. When hypothermic patients awake from anesthesia, shivering may occur, leading to increased oxygen consumption and myocardial ischemia. Hypothermia furthermore decreases cerebral perfusion. Trauma patients with hypothermia below 32° C have a mortality of 100%, regardless of the severity of injury, degree of hypotension, or amount of fluid replacement [24]. Use of blood/fluid warming devices, heating matresses and heating blankets, heating and humidification of fresh respiratory gases, and meticulous covering of the patients are necessary preventive measures.

Pitfall 17: Awareness

In trauma patients special attention must be paid to the problem of awareness. Awareness is the result of underdosing anesthetics [25]. In trauma patients, lower doses of anesthetics are often administered because of the fear for hypotension. Patients normally move when anesthesia is too light, but this is impossible if they

have received a muscle relaxant. Reliance on an opioid-based technique may increase the risk of awareness [26]. Awareness can be prevented by administration of benzodiazepines or continuous inhalation of anesthetics.

Pitfall 18: Awakening of Patients After Trauma Surgery

In severely traumatized patients optimal monitoring and support of respiration and circulation should be continued after emergency surgery. Indeed, prolonged postoperative ventilation and critical care treatment have been shown to prevent or treat multiple organ failure [27]. Indications for admission to the ICU and for continuing artificial ventilation are presented by Van Dalen (this volume). Extubation of the trachea therefore should not be performed until all potential disturbances in gas exchange are ruled out. Extubation also is contraindicated in the presence of lung and upper airway injury, unless edema formation and other respiratory reactions are unlikely to occur, in patients with shock, fat embolism, or serious head injury.

Pitfall 19: Existing or Developing Respiratory Failure

The symptoms and problems of respiratory failure of whatever origin are not always present at admission, i.e., in patients with pulmonary contusion, inhalation injury, maxillofacial injury, burns, and aspiration. These symptoms frequently appear during emergency surgery or in the ICU and require immediate treatment. The result of respiratory failure is hypoxia, hypercapnia, and increased repiratory workload. The cause of respiratory failure must be treated if possible. In most of these patients endotracheal intubation and artificial ventilation (PEEP) are indicated. Adequate and early treatment of shock is one of the major parts in the prophylaxis of respiratory failure. Repeated blood gas analysis should be performed as soon as possible, and monitoring with pulse oximetry is recommended throughout the emergency room and operating room period.

Pitfall 20: The Patient with Head or Spinal Cord Injury

Patients with a GCS score of less that 9 have severe brain injury. Secondary brain injury contributes largely to morbidity and mortality, while it can frequently be prevented. Factors contributing to secondary injury are hypoxia, hypercapnia, excessive hyperventilation, systemic hypotension, increased intracranial pressure (ICP), posttraumatic cerebral arterial spasm, and transtentorial (uncal) and cerebellar (tonsillar) herniation. Prompt resuscitation of the ventilation and circulation prevents secondary hypoxic brain damage in many cases (see Stocker et al., this volume). Only about 20% of the patients with head injury undergo operation immediately. During surgery an impeccable airway should be established and maintained, as well as cardiovascular stability and optimal intracranial

circulation. Cerebral blood flow is constant (autoregulation) at mean arterial pressures between 60–160 mmHg (50–150 mmHg cerebral perfusion pressure), decreases linearly with $PaCO_2$ of 25–55 mmHg (3% per mmHg), and increases with hypoxic PaO_2 below 50 mmHg. When ICP increases, cerebral blood flow decreases.

Hypotension in head injured patients usually indicates the presence of sites of hemorrhage or high spinal cord injury. The common response to head trauma is hypertension and tachycardia. Release of high ICP by craniotomy may induce sudden hypotension. PEEP ventilation may hinder venous outflow, and thus also decrease cerebral perfusion pressure. Most IV anesthetic agents decrease cerebral metabolic rate (normal oxygen consumption 3–3.5 ml $100\,g^{-1}\,min^{-1}$) and cerebral blood flow, as long as CO_2 retention is prevented. Barbiturates, etomidate, and propofol decrease ICP, but favor hypovolemic hypotension. Ketamine, however, increases ICP. Synthetic narcotic analgesics (fentanyl and analogues) increase ICP [28, 29], except when blood pressure is supported [30]. Halothane increases cerebral blood flow and should probably not be used in head-injured patients. Isoflurane, desflurane, and sevoflurane do not influence ICP when administered in doses below 1–1.5 MAC. Nondepolarizing relaxants that cause histamine release (atracurium, tubocurarine etc.) occasionally increase ICP. Clean steroidal relaxants, such as vecuronium and rocuronium, are therefore to be recommended. Increased central venous pressure causes increased ICP because it increases cerebral venous outflow resistance. Therefore resuscitation should take place under strict monitoring of the central venous pressure to avoid hypervolemia.

Brain protection is the ultimate goal of care for head-injured patients. This reduces ICP, maintains cerebral blood flow, decreases cerebral metabolic rate, and scavenging free radicals prevents seizure activity, decreases sympathetic action, decreases intravascular sludging (hemodilution), and prevents calcium shifts (nimodipine, nifedipine, flunarizine). Many principles in the care of head injury also apply for spinal cord injury. The level of injury determines the symptoms and complications. Endotracheal intubation is one of the main reasons for secondary trauma [31]. Injury at the fifth cervical vertebra wipes out intercostal muscle activity, making respiration completely dependent on the diaphragm. Injury at higher levels causes complete repiratory arrest. However, edema of the cord is frequently the cause of transient respiratory depression or arrest in patients with damage at lower levels. It thus is difficult to determine whether the patient will need life-long artificial ventilation. Another complication is a limited response to volume stress with the development of pulmonary edema and spinal shock.

Conclusion

Trauma anesthesia is a field for experienced anesthesiologists because of the many pitfalls involved. No ideal anesthetic agent or technique for the severely injured patient exists. A choice should be made on the basis of the physical status of the

patient, the time course of the trauma disease, and the pharmacolgical characteristics of the individual anesthetic drugs and techniques. Knowledge of, and proper preventive action to the individual pitfalls will minimize the complication rate.

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Therapeutic Sequences in the Acute Period in Unstable Patients

O. Trentz and H. P. Friedl

Introduction

This chapter deals with emergency treatment of severely or multiply injured victims in the resuscitation room who upon admission obviously are dying or unstable or have decompensated vital functions. Most commonly these critical conditions are exsanguinating hemorrhage, acute pump failure, obstruction of airways, mechanical failure of ventilation, or severe brain damage with tentorial herniation. A rapid survey suggests whether the trauma victim is unstable or dying, which requires urgent clearing and securing of airways, ventilatory support, massive volume infusion, and control of rapid external bleeding. Here, treatment precedes diagnosis! The first priorities of the advanced trauma life support (ATLS) course address the traditional ABCs of resuscitation: A = airway and cervical spine control, B = breathing, and C = circulation, cerebrum, cord. If effective, resuscitation cannot be achieved by conventional measures such as intubation, ventilation, and volume replacement, immediate life-saving surgical procedures are required.

Prerequisites

An uncoordinated approach to trauma management in an empirical trial-and-error manner results in a high rate of preventable deaths. Optimal results can be expected only by a well-organized trauma care system, backed up by designated trauma centers. Hospitals that provide trauma care should maintain:

- A defined trauma team
- A central, easily accessible shock-trauma resuscitation suite, equipped with all necessary diagnostic (e.g., X-ray, echography) and therapeutic facilities
- Consensus over responsibilities and leadership ("trauma captain")
- Prepared equipment, sets and trays for urgent procedures, and resuscitation trolleys
- Defined and trained algorithms and protocols to enforce strictly trauma "policy and procedures"

Surgical Access to Life Support Systems

If the ABCs of resuscitation cannot be achieved by conventional measures, immediate surgical access to life support systems must be established:

(a) the inability to intubate the trachea (including with fiberoptic assistance), i.e., in severe maxillofacial or neck injuries, is (the only) emergency indication for creating a surgical airway by cricothyroidotomy;
(b) whenever sufficient and secure IV lines cannot be established by percutaneous techniques, surgical cut-downs are mandatory.

Sufficient means at least two large-bore lines, one above and one below the diaphragm. Large-bore trauma tubings (exchange kits/introducer catheters, at least 7 F) are placed in central veins and secured by sutures. Cut-downs (below the diaphragm) should start synchronously with percutaneous insertions (above the diaphragm). Cut-downs are performed bilaterally in the saphenous veins at the ankle or in the saphenous or femoral veins at the groin. After initiating IV lines, a massive initial fluid challenge is given with a 2-l bolus of warmed Ringer's lactate using pressure bags. Depending on the hemodynamic response, the fluid challenge is continued and warmed blood (pressure bag) transfused as soon as possible.

Life-Saving Decompression of Body Cavities

Tension Pneumothorax. Increasing respiratory distress, despite or soon after intubation and ventilation, with rising airway pressure and distended neck veins indicates a tension pneumothorax requiring decompression by needle thoracostomy followed by tube thoracostomy. The chest tube is inserted in the fifth intercostal space along the midaxillary line or above the level of the nipple through the anterior chest wall after exploring the pleural cavity with the index finger. The tube is guided anteriorly and superiorly. For massive hemothorax another tube should be directed under digital control posteriorly and inferiorly. In the acute trauma setting, only large-bore chest tubes (30–40 F) should be used. The tube should be connected as soon as possible to a closed water-seal drainage system (commercial tube drainage system) with 20–30 cm water suction.

Cardiac Tamponade. When pericardial tamponade is suspected, pericardiocentesis via the paraxiphoid route may confirm diagnosis and release tamponade. Under severe trauma conditions this decompression buys time for performing emergency thoracotomy or median sternotomy. During crash laparotomy transabdominal pericardiotomy through the subxiphoid membranous diaphragm can rule out cardiac tamponade.

Intracranial Hypertension with Impending Tentorial Herniation. The most important measures to protect the injured brain from secondary brain damage

and increasing intracranial pressure are restoration and maintenance of adequate cerebral perfusion and oxygenation. Focal brain injuries with acute subdural or epidural hematomas (especially after falls or assaults) can take a precipitous downhill course, with increasing mydriasis and contralateral motor deficit. Under these specific circumstances emergency surgery without confirmation by computed tomography can be life saving. Exploratory burr holes on the side of the enlarged pupil and fracture can find and release an extracerebral mass. Once the burr hole has removed the compressing clot, a definitive craniotomy is possible.

Compression of the Cervical Cord. Injuries with a high index of suspicion for cervical spine lesions require protection of the C-spine during extrication and emergency treatment with a semirigid collar or a spine board. A cross-table lateral C-spine X-ray is mandatory as soon as possible. Once a fracture-dislocation or jumped/locked facets of the cervical spine are identified, immediate closed reduction (Gardner-Wells tongs) and sufficient splinting (collar and traction) are necessary.

Resuscitative Thoracotomy

Resuscitative thoracotomy in the emergency room should be performed only in trauma victims with signs of life, either at the very moment of admission or when in closest proximity to the hospital under rapid transportation [1]. Chances are better in penetrating than in blunt trauma and in patients who are *in extremis* or rapidly deteriorating, despite adequate resuscitation. Under these circumstances indications for resuscitative thoracotomy are given in penetrating chest trauma with acute deterioration, exsanguinating hemorrhage, pericardial tamponade, and cardiac arrest. In blunt trauma emergency thoracotomy is indicated only with on-the-spot deterioration or observed cardiac arrest.

Through a left anterolateral thoracotomy (fourth or fifth intercostal space, sternum to midaxillary line), a rapid and wide entry into the chest is performed. Depending on the findings, the first measures are cross-clamping of the descending thoracic aorta or the pulmonary hilum or opening of the pericardial sac longitudinally anterior to the phrenic nerve and starting cardiac massage (and internal defibrillation).

Penetrating wounds to the heart should be occluded with finger tamponade or an inflated balloon-catheter and then closed with sutures (4 mm bites of 2–0 Prolene) pledgetted with Teflon felt. Pericardium bolsters may be required, at least at the thinner and friable muscle of the right ventricle. Atrial injuries can be closed by vascular clamping, which facilitates repair with a running suture. A brief (2–3 min) inflow occlusion of the superior and inferior vena cava using clamps or tourniquets can be helpful for the repair of a myocardial wound. As soon as there is a positive response with improvement of vital signs, the patient is transported to the operating theatre for definitive surgery.

Control of Exsanguinating External Hemorrhage

Dural Sinus Bleeding. Open and penetrating head injuries can be accompanied by excessive hemorrhage from transsected dural sinuses. Control of hemorrhage requires a trephine opening near the bone wound and an enlarging circumferential craniotomy using a rongeur. Dural sinus bleeding can be stopped by lateral suture repair enforced by a patch of pericranium or fascia. Only the anterior sagittal sinus may be controlled by suture ligation.

Penetrating Wounds of Central Large Arteries. Due to the accompanying life-threatening bleeding these wounds demand rapid central vascular control by cross-clamping or balloon occlusion followed by lateral repair or application of an intravascular shunt until definitive reconstruction is possible or a bypass installed. To gain vascular control of the thoracic outlet an extended emergency exposure by median sternotomy, "book" or "trapdoor" thoracotomy (supraclavicular incision, partial sternotomy, and anterolateral thoracotomy) may be necessary.

Traumatic Amputation and "Mangled Extremity." Hemorrhage control in complete traumatic amputation is easily obtained by pressure dressings, tourniquet, or vascular clamping. Near-amputations in severely or multiply injured patients are completed with preliminary bleeding control. Mangled or crushed extremities in patients with severe polytrauma require primary amputation as a life-saving measure. For temporary bleeding control pressure dressings with pneumatic splinting or tourniquet can be applied.

Open Pelvic Fractures. Exsanguinating hemorrhage from open pelvic fractures cannot be controlled by external compression such as pneumatic splinting (MAST). These patients can be saved only by aggressive surgical bleeding control, including stabilization of the pelvic ring by external or internal fixation and pelvic packing.

Control of Exsanguinating Hemorrhage into the Body Cavities

Massive Hemothorax

Apart from resuscitative (emergency room) thoracotomy, urgent (operating room) thoracotomy is indicated with severe thoracic hemorrhage, i.e., blood loss via the chest tube exceeding 1500 ml at the time of thoracostomy or 500 ml for 1 h or 200 ml/h for more than 4 h. Severe pulmonary lacerations and greater vessel injuries are controlled by cross-clamping and suture repair or by definitive surgery. Rapid recognition of blunt traumatic aortic rupture requires a high index of suspicion after major deceleration trauma. Transesophageal sonography in the emergency room is the method of choice to diagnose aortic rupture in the unstable patient. An open, sucking chest wound after penetrating trauma requires a large-

bore chest tube and sterile air-tight occlusive dressing, which buys time for definitive surgery. Ongoing severe hemorrhage, with chest tube output greater than 500 ml within the first hour, requires urgent thoracotomy. Generally speaking, massive hemothorax allows for autotransfusion, which provides the patient immediately with type-specific cross-matched warmed blood.

Massive Hemoperitoneum

A patient who is dying or is highly unstable *in extremis* due to exsanguinating hemorrhage into the abdominal cavity requires resuscitative crash laparotomy (Fig. 1) in the emergency room. If a resuscitative thoracotomy is necessary, preliminary cross-clamping of the descending thoracic aorta should be performed before crash-laparotomy to avoid cardiac arrest due to sudden decompression of the tamponaded abdominal cavity. As an alternative the supradiaphragmatic aorta can be occluded by a balloon catheter (occluder 22-F) from the groin via the external iliac artery [2]. Crash laparotomy is performed by a midline abdominal incision, which gives rapid access and wide exposure and allows for extension into the chest. If possible, intra-abdominal blood should be collected for autotransfusion by a suction tip passed through a preliminary mini-laparotomy before full-sized midline exposure. If supradiaphramatic control of the aorta cannot be performed, manual compression of the subdiaphragmatic aorta is carried out first. This manual aortic compression is transferred to an assistant. Next, a rapid check of all four quadrants (upper left quadrant first and then counterclockwise) with rapid packing to control venous bleeding. After tight packing the manual aortic compression (or aortic occlusion) can be released to check bleeding control.

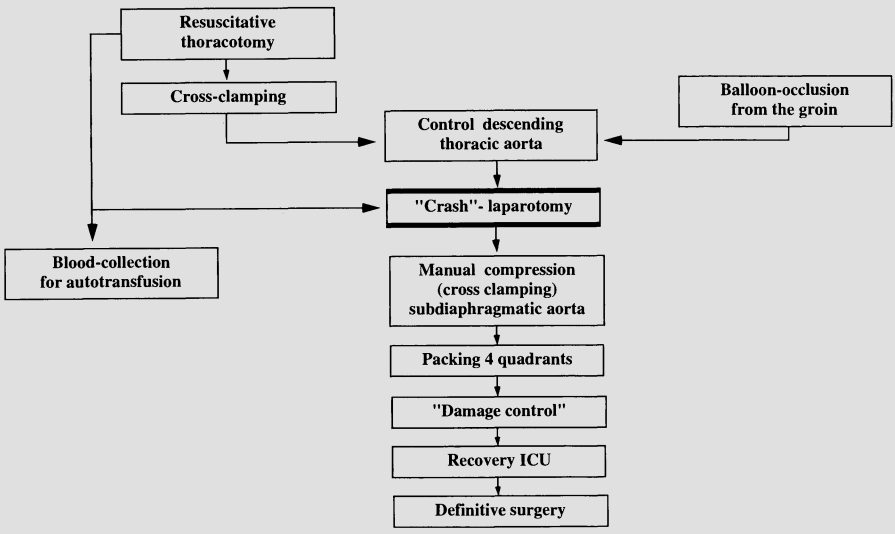

Fig. 1. Crash laparotomy in exsanguinating hemorrhage

In the presence of an obviously ongoing arterial hemorrhage, cross-clamping of the subdiaphragmatic aorta or inflow control of an identified injured organ, are performed. As soon as a positive hemodynamic response is obtained, the patient should be transported to the operating room for definitive surgery. In penetrating torso trauma with severe hypotension some authors recommend surgical control of hemorrhage prior to massive volume challenge [3]. Patients *in extremis* with severe polytrauma or extensive penetrating abdominal injuries have a better chance to survive when definitive repair of intra-abdominal injuries is delayed after effective resuscitation and stabilization in the ICU. In such situations only damage control is performed after crash laparotomy [4]. This includes:

(a) control of hemorrhage,
(b) control of contamination,
(c) intraperitoneal packing, and
(d) rapid provisional closure.

Hollow viscus injuries are closed by running sutures or with a linear stapler. Definitive repair, reconstruction of bowel continuity, and colostomies are performed after 8–16 h of recovery under much better conditions [4, 5].

Massive Hemorrhage Due to Crushed Pelvis

Pelvic crush and disruption of the pelvic ring (open book, vertical shear injuries) can produce exsanguinating hemorrhage into the retroperitoneum and the peritoneal cavity (Fig. 2). Apart from aggressive volume challenge these patients require immediate stabilization of the pelvic ring by external fixation or a pelvic compression clamp. With good response, diagnostic work-up can be completed and pelvic reconstruction done as staged surgery. If the patient remains unstable, laparotomy is mandatory for active bleeding control. In these circumstances

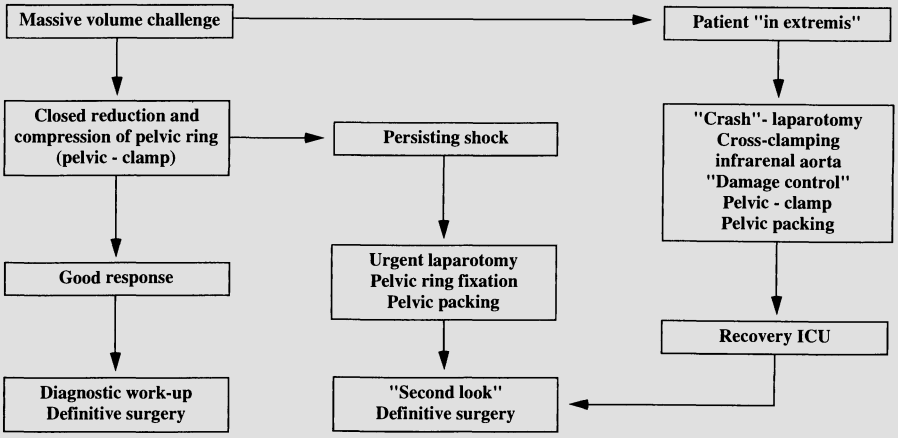

Fig. 2. Severe hemorrhage from pelvic crush trauma

internal fixation of the pelvic ring may be advisable, followed by tight pelvic packing. Exsanguinating hemorrhage in open pelvic injuries may require crash laparotomy with cross-clamping of the infrarenal aorta. After a positive hemodynamic response, pelvic stabilization and packing are mandatory [6].

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Surgical Procedures in the Stabilized Patient

H. Tscherne and G. Regel

Introduction

It is has been over 50 years since Cuthbertson described the metabolic response of patients suffering bone and nonbone injury. In his clinical study he characterized all metabolic changes as resulting from "tissue injury" itself. We realize today the fundamental nature of this insight, and that therapeutic progress requires better comprehension of the physiological and biochemical events. According to this principle, both diagnostic and therapeutic procedures are oriented to the pathophysiological mechanisms following trauma at different phases in the posttraumatic course. In this course we must distinguish four different periods:
(a) acute or reanimation period (1–3 h),
(b) primary or stabilization period (1–72 h),
(c) secondary or regeneration period (3–8 days), and
(d) tertiary or rehabilitation period (after the 8th day).

Definition of the Primary Period

The primary period starts at the time that vital functions stabilize. This means:
(a) after treatment of mass bleeding from blunt thoracic, abdominal, or pelvic injuries;
(b) after providing adequate ventilation (e.g., prevention of pneumothorax and clearing of the major airways); and
(c) after treatment of intracranial mass bleeding (epi- and subdural hematoma).

These three steps in acute treatment should guarantee stabilization of the patient in the first hours after trauma. Only in rare cases is this period prolonged up to 72 h [1, 2]. The primary period thus begins after treatment of life-threatening injuries. It consists of further diagnostic procedures and treatment of injuries that are not directly life threatening but may become life endangering or severely disabling if not treated promptly (Fig. 1).

Further diagnostic procedures include:
(a) assessment of laboratory parameters;
(b) advanced cardiovascular measurements, such as pulmonary hemodynamics (pulmonary artery pressure and cardiac index; this is especially import-

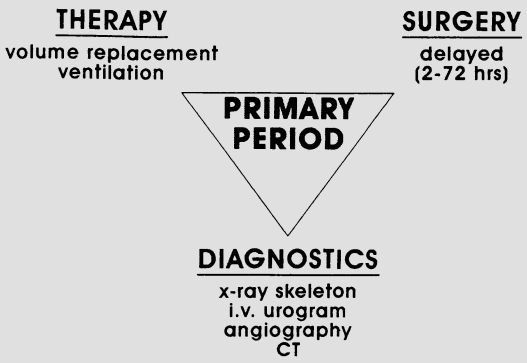

Fig. 1. Therapeutic concept in the primary period, consisting of continuous resuscitation and secondary priority operative regimens after finishing diagnostic procedures

ant in patients with a prolonged shock phase and those with thoracic trauma);

(c) diagnostic exclusion of vascular injuries (Doppler sonography, angiography, digital subtraction angiography);

(d) diagnostic exclusion of injuries of the urogenenital system (e.g., retrograde cystography); and

(e) X-ray examination of the skeletal system (fractures and articular injuries).

After further assessment and with continuing hemodynamic stability, injuries of secondary priority can be treated surgically. These surgical procedures are also called "delayed primary" or "day 1" surgery. Some of these injuries can be dealt with simultaneously, such as maxillofacial injuries and extremity fractures. The priorities in the surgical treatment in the primary period are as follows:

1. Brain injuries
2. Eye and facial injuries
3. Progressive compression of the spinal cord
4. Visceral injuries
5. Musculoskeletal injuries
   - Fractures with concomitant major vessel injury (with severe compartment syndrome, with open soft tissue injuries, with open joint)
   - Closed shaft fractures
   - Pelvic ring injuries
   - Unstable spine fractures

Brain Injuries

Evacuation of intracranial bleeding (especially epidural and subdural hematoma) should be performed in the acute period. All other injuries of the cranium are treated in a delayed primary approach. Further assessment and analysis of brain injury should be performed at the end of the acute period. Localization, extent, and severity of the brain injury are best evaluated by computed tomography (CT) at this stage. CT is indicated in patients with:

(a) primary unconsciousness (Glasgow Coma Scale < 8),
(b) focal neurological signs,
(c) open brain injuries,
(d) worsening of clinical and neurological status, or
(e) skull fractures.

The cause of unconsciousness must be analyzed and a relationhip to other life-threatening injuries ruled out. Focal neurology in conscious patients can be related to a former injury; in all other cases a lesion of the carotic artery must be excluded. An increase in a neurological deficit is always a sign of intracranial bleeding or edema formation and an absolute indication for CT.

The risk of cerebrospinal fluid leakage and consequent intracranial infection makes open head injuries and skull base fractures serious injuries that must be operated on in the primary period. An operation should be considered in cases of open, depressed skull fractures with or without space-occupying hematoma, compound fractures of the rhinobasis and orbita, dislocated fractures of the dorsal wall of the frontal sinus, and continuous leakage of cerebrospinal fluid, especially of the frontobasis. The operative approach depends on the type and localization of the fracture. The transsphenoidal and fronto-orbital approaches are less traumatic but offer only limited visibility. The transfrontal intradural approach offers by far the best view of the frontobasis and is therefore indicated in most cases.

Continuous measurement of intracranial pressure is required after primary treatment of the intracranial lesions. An indication for intracranial pressure measurement is present in all cases of intracranial mass lesions, midline shift, elapsed basal cisterns, and in those with deterioration of the neurological status during the posttraumatic course.

Eye and Maxillofacial Injuries

Perforating injuries of the eyes and extensive lesions of the facial soft tissues require immediate operative treatment at the beginning of the primary period. In most cases a simultaneous approach to these injuries and to extremity fractures is possible and recommended to shorten this operative phase. Maxillofacial fractures are normally treated in a two-step approach. Initially the unstable fractures are only splinted and wired, with final reconstruction and stabilization being performed only after facial swelling has resolved to prevent infection and prolonged wound healing.

Compression of the Spinal Cord

Progressive compression of the spinal cord is an absolute indication for operation in the primary phase. In any case where the neurological status cannot be verified (e.g., primary unconsciousness), a complete assessment must be performed (X-ray

of thoracic and lumbar spine, AP and lateral views, if indicated CT scan). Recovery from spinal cord injury depends on the amount of initial damage or contusion of the cord, as well as on mechanical factors such as compression of the neurological structures by fragments of bone and intervertebral disk. Although the surgeon cannot directly alter tissue disruption, movement of the injured spinal cord or persistent compression of the neural structures by fragments of bone or disc can be prohibited in an attempt to preserve the remaining neural integrity. Immediate stabilization of these fractures protects the spinal cord and allows early mobilization of the patient.

Visceral Injuries

Visceral injuries are not always associated with an intra-abdominal mass bleeding, but may still lead to a life-threatening situation if not diagnosed early in the primary period. Patient history is most useful in this respect since the accident mechanism often provides important information on specific injuries. In particular, thoracic and vertebral injuries are often combined with abdominal trauma. Routine diagnostic criteria are considered unspecific (laboratory parameters, ultrasound, etc.) while the diagnostic value of computer tomography is still under discussion.

Ruptures of the diaphragm, for instance, are rare (1%–7% in multiple trauma patients) and are often overlooked or are concealed by the presence of an ipsilateral thoracic injury (lung contusion, hemothorax) during the primary diagnostic procedures. Clinical evaluation leads to definitive diagnosis (chest X-ray, and in case of doubt imaging of the – usually intrathoracally displaced – stomach via the nasogastric tube). In 80% of cases laparotomy is indicated.

Injuries of the small intestine and mesenterium are the most frequent bowel lesions in blunt abdominal trauma (3%–18%). A typical injury mechanism is the so-called "seat-belt syndrome," where a submarining effect leads to bursting of the intestine. A secondary perforation may occur after a delay of several days. The most reliable diagnostic procedure is peritoneal lavage. After diagnosis, laparotomy is indicated immediately. Colon injuries are rarely seen after blunt trauma. Colon lesions are more often diagnosed in association with pelvic injuries. A laparotomy is obligatory, and, depending on the type of lesion, a primary suture or resection with or without temporary colostomy is indicated. Injuries of the pancreas and duodenum are also often concealed after blunt abdominal trauma. Diagnosis is very difficult in these cases, and clinical symptoms are often unspecific. CT is recommended, especially in pancreatic trauma. Laboratory parameters are not correlated with the severity of the injury and may reach abnormal levels only after some delay.

Injuries of the urinary tract are often diagnosed during the initial phase, when hematuria occurs. The first step in the diagnostic procedures is to exclude a lesion of the urethra or bladder with the help of retrograde urethro-cystography (84% sensitivity). The most frequent injury of the urethra is the indirect, intrapelvic type. Bladder ruptures are generally located in the extraperitoneal portion. Both

lesions are often associated with pelvic fractures (transpubic instability, rupture of the symphysis) in polytraumatized patients. They can lead to a life-threatening situation if diagnosed or treated with delay. These injuries require surgery in the primary period. Treatment of the bladder injury depends on the (extra- or intraperitoneal) localization of the lesion. In extraperitoneal injuries the pelvic fracture is stabilized first; the retroperitoneum is then explored, the hematoma evacuated, and finally the bladder sutured. In intraperitoneal lesions laparotomy is necessary. In both cases a transurethral catheter is installed and antibiotics given. Lesions of the urethra are also treated in this phase, with immediate suturing and a transurethral catheter.

Blunt trauma leading to injury of the kidneys can be best diagnosed by intravenous pyelography, which shows the functioning and contour of both kidneys. Parenchymal lesions are best visualized by CT.

Contusions and smaller subcapsular hematomas are treated conservatively. Extensive parenchymal lesions require surgery. A transperitoneal approach is recommended since these lesions are often associated with other intra-abdominal injuries. The indication for operation is given not only by the extent of bleeding but also by the loss of kidney function and by the potentially associated lesion of the ureter.

Musculoskeletal Trauma

The general principle regulating fracture treatment is stable osteosynthesis. Among the various musculoskeletal injuries, first priority goes to open joint and shaft fractures, to fractures with a vascular injury or compartment syndrome (see "Definition of the Primary Period").

Fractures with Concomitant Vascular Injury

Ischemia may occur systemically in cases of severe hemorrhage or locally in case of severe limb fractures with vessel injury (Fig. 2). In either situation reperfusion starts at the time of resuscitation and initiates a cascade of mediators which lead to

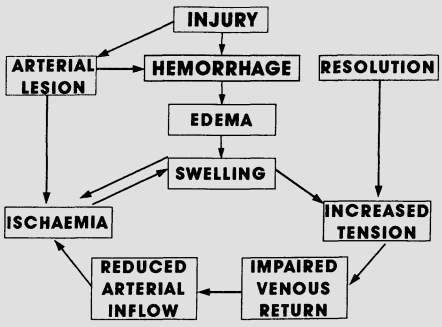

Fig. 2. Pathophysiology and relationship of vascular injury and ischemia

a typical reperfusion injury. This is characterized by morphological evidence of neutrophil sequestration, increased microvascular permeability, and protein leakage. An important step in this series of events is neutrophil adherence to endothelial cells. Only adherent neutrophils are able to damage the endothelium by releasing proteases. Ischemia alone does not end in increased vascular permeabilty or neutrophil sequestration but to loss of organ function and cell death depending on the duration of ischemia.

The prognosis of a fracture with concomitant vascular lesion depends on the duration of ischemia and degree of reperfusion disturbances. Loss of muscle function is seen as early as 2–4 h after ischemia, and irreversible destruction develops after 4–6 h. Nerve tissue may show functional impairment after even 30 min, and irreversible damage after 12–14 h of complete ischemia. Several changes of the capillary endothelium are seen after only 3 h of ischemia. The result is increased permeability, with postischemic swelling of the soft tissue. Potential causes of muscle swelling after injury are numerous, including direct injury, interstitial swelling from impaired venous return, ischemia, and reperfusion. In multiple trauma patients this destruction may be aggravated by the presence of hypoxemia [4, 5].

Because of their fixation to the bone the following arteries are susceptible to vascular injuries : the subclavian artery near the clavicula, the brachial artery at the shaft of the humerus, the femoral artery in the region of the femoral shaft, and the popliteal artery at the knee joint. A dislocation of the knee joint is associated with a rupture of the popliteal artery in 50% of cases [6]. In relation to this the development of a compartment syndrome is important (Fig. 3). Compartment syndromes lead to an increase of intrafascial pressure with irreversible muscle, nerve, and vascular damage. In isolated injuries the compartment pressure is pathological between 30 and 40 mmHg. In this case an immediate fasciotomy is necessary. In multiple trauma patients this is not always recommended, since hypoxemia can lead to irreversible changes even at far lower pressures. At high risk are patients with comminuted closed fractures of the proximal and distal tibial or complex foot injuries [6].

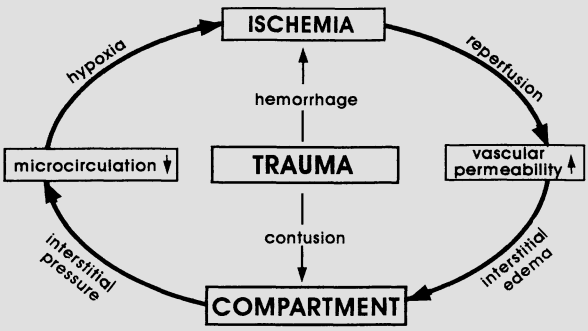

Fig. 3. Relationship between ischemia and compartment syndrome and their consequences for the microcirculation and vascular permeability

Open Fractures

All open fractures are treated during the primary period [6–8]. This includes extensive débridement, possibly exploration of the vasculature, and stable fixation of the fracture (Fig. 4). The type of osteosynthesis is chosen, depending on the degree of soft tissue injury. Formerly, fractures with associated severe soft tissue injury were generally treated with external fixators, but today unreamed intramedullary nailing systems can be used with low risk, even in shaft fractures with IIIb and IIIc open injuries. Most important here is the treatment of the soft tissue injury. Especially in multiple trauma patients, primary closure of the soft tissues is not recommended. Relative hypoxia potentially leads to delayed soft tissue healing and to increased susceptibility to infection. Therefore in multiple trauma patients secondary closure of soft tissues is recommended [6]. However, adequate soft tissue coverage of the "hardware" must be obtained. After this, artificial skin (i.e., Epigard) is used in the primary period, and secondary closure is performed after 5–10 days, possibly following sequential partial wound closure (Fig. 4).

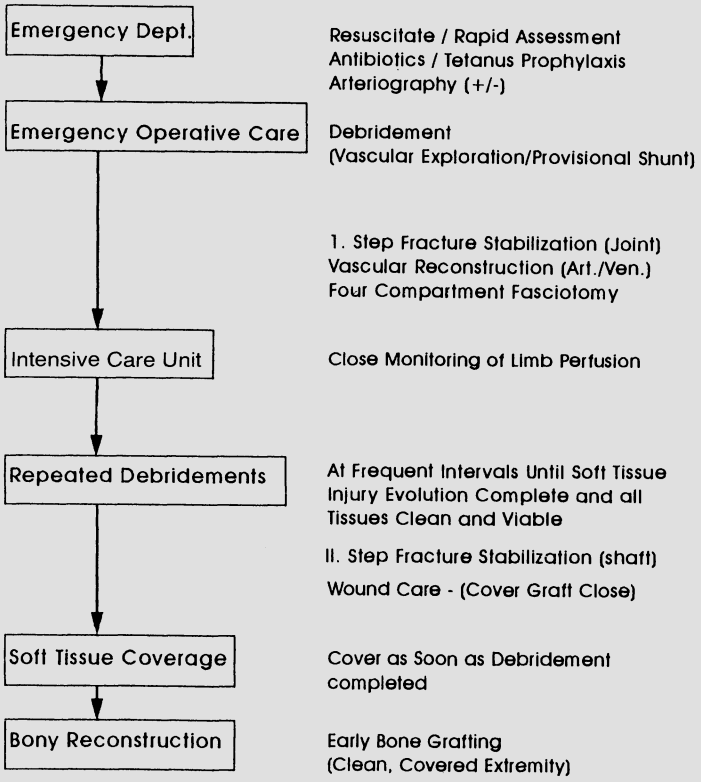

Fig. 4. Standardized regimens for treating open fractures. Soft tissue reconstruction (i.e., local or free vasularized flap) is obligatory at the beginning of the secondary period

Soft tissue defects with denuded bone need a special reconstructive approach. In such cases, local flaps or – with extensive defects – free revascularized soft tissue transfer (preferably a latissimus dorsi flap) are recommended. To prevent late complicatons (osteitis), this reconstruction has to be performed not later than 2–5 days after injury. Open intra-articular fractures are also treated initially with débridement, reconstruction of the articular surface with a minimum of implants, and external fixation bridging the joint. Definitive internal fixation of the joint component to the shaft is delayed until the rehabilitation period.

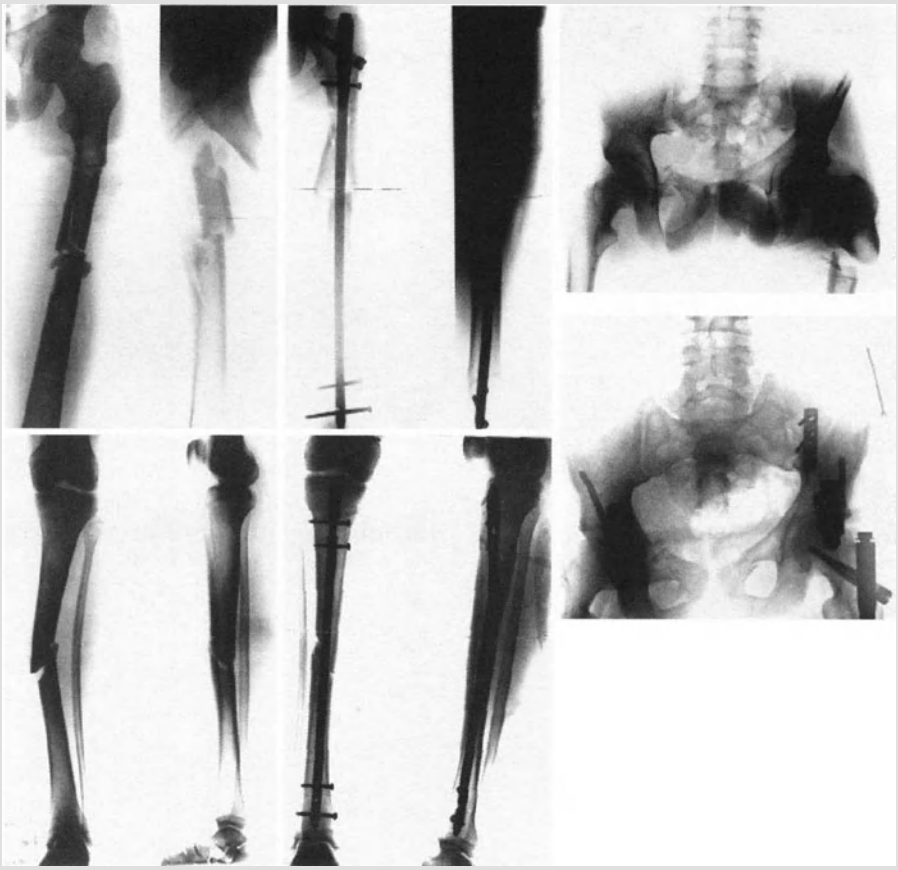

Fig. 5. A 21-year-old multiple trauma patient. Monteggia injury (*left*) (*not shown*), type C pelvic injury with transiliac fracture dislocation (*left*), sacroiliac disruption (*right*), transpubic instability (*right and left*), grade I open proximal femur fracture (*left*), and grade II open tibial shaft fracture (*left*). Operative treatment according to the priority of fracture treatment: (a) temporary fixation of femur with a distractor, (b) stabilization of the tibia with an unreamed nail, (c) with stable vital functions, fixation of femur with unreamed nail after removal of distractor (simultaneously, stabilization of Monteggia injury by a second team), (d) with continuously stable vital functions, stabilization of the pelvis with screws and plates, external fixation of the "open book injury"

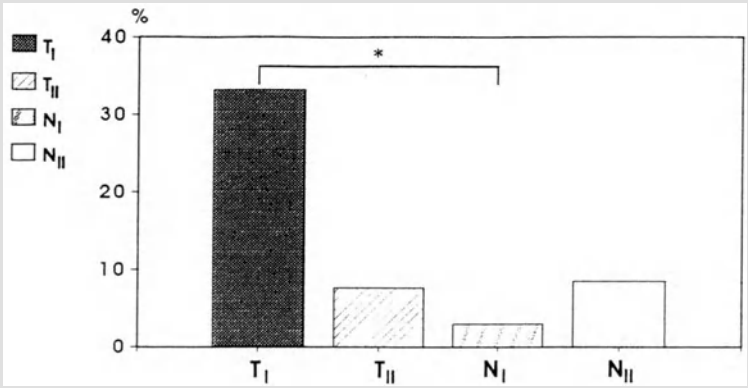

Fig. 6. Incidence of adult respiratory distress syndrome in patients operated primarily (*I*) and secondary (*II*), with intramedullary stabilization of the femur (reamed technique), distinguishing patients with (*T*) or without (*N*) associated thoracic trauma

Closed Fractures

Stable fixation of fractures minimizes pain, secondary soft tissue injury and the danger of fat embolization [8, 10–13]. Stabilization of extremity fractures therefore reduces stress, traumatic shock, and posttraumatic complications, leading to lower morbidity and mortality [12, 13] (see Biert and Goris, this volume). Stabilization of fractures of the lower extremity (femur and tibial shaft) are especially important in this respect. The priority of fracture treatment is: tibia, femur, pelvis, spine, upper extremity. Alternative fixation methods must be used to respect this sequence. In dealing with ipsilateral fractures of the lower extremity, we for example use temporary fixation of the femur with the large AO distractor, stabilize the tibial fracture with an unreamed nail, and finally perform fixation of the femur (Fig. 5). In all cases primary stabilization of the femur remains the major goal in the multiple trauma patient. If severe trauma with additional pulmonary injury is present, reaming of the medullary canal should be avoided (Fig. 6) [5, 18–21]. Unreamed nailing procedures are presently favored and seem to be an alternative in this special situation [19, 20]. For temporary stabilization in a critically injured patient an external fixation device can be applied temporarily. Secondary intramedullary nailing is then performed safely after an interval of at least 2–3 days. The requirements to perform these prolonged operations are described by Biert and Goris (this volume).

Limb Salvage Versus Amputation

With the development of modern microsurgical procedures surgeons are often able to "salvage" revascularized limbs that would formerly have been bound for certain amputation. The outcome of limb salvage in such injuries, however, may be less desirable for the patient than that which might be expected from immediate

Table 1. Mangled Extremity Severity Score

Characteristics	Injuries	Points
Skeletal/soft tissue		
Low energy	Stab wounds, simple closed fractures, small-caliber gunshot wounds	1
Medium energy	Open or multiple-level fractures, dislocation, moderate crush injuries	2
High energy	Shotgun blast (close range), high-velocity gunshot wounds	3
Massive crush	Logging, railroad, oil rig accidents	4
Shock		
Normothensive	Blood pressure stable in field and in operating room	1
Transiently hypotensive	Blood pressure unstable in field but responsive to intravenous fluids	1
Prolonged hypotension	Systolic blood pressure less than 90 mmHg in field but responsive to intravenous fluid only in operating room	2
Ischemia		
None	A pulsatile limb without signs of ischemia	0
Mild	Diminished pulses without signs of ischemia	1
Moderate	No pulse by Doppler, sluggish capillary refill paresthesia, diminished motor activity	2
Advanced	Pulseless, cool, paralyzed and numb without capillary refill	3
Age		
< 30 years		0
30–50 years		1
> 50 years		2

amputation [23–25]. Also, increased patient morbidity and even mortality are associated with failed attempts at limb salvage. Decision making protocols have looked at absolute and relative indications for limb salvage, also considering concomitant injuries or associated polytrauma [23, 24]. The Mangled Extremity Severity Score classifies these injuries and thus attempts to assist in decision making [23] (Table 1). A score of 7 points or more is highly correlated with the decision to amputate. Treatment of the severely injured limb is standardized once a limb salvage attempt has been chosen (Fig. 4).

Unstable Pelvic Ring Fractures

Pelvic instability, especially in multiple traumatized patients, requires an aggressive and well-planned therapeutic regimen [28–30]. Early physical and radiological assessment is necessary to evaluate concomitant injuries and to plan

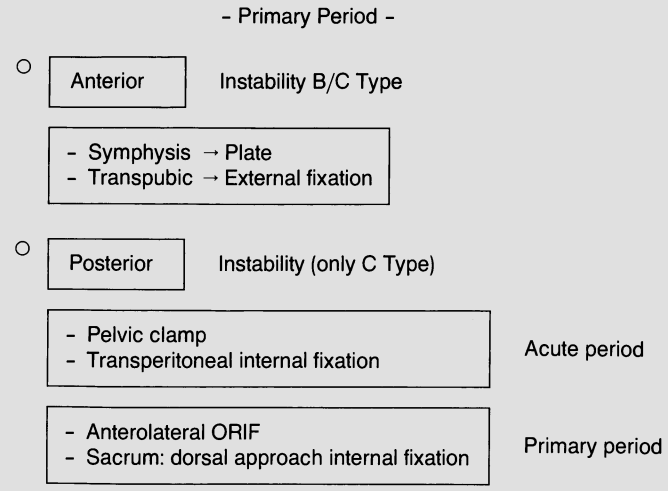

Fig. 7. Algorithm for treating unstable pelvic injuries in the primary period, considering the need for laparotomy and the different stabilization methods for the anterior and posterior pelvic ring

treatment. In the acute phase the main goal is to prevent massive bleeding. A simple three-step emergency algorithm has been developed to faciliate primary hospital resuscitation in unstable pelvic fractures [29, 30]. Before approaching a massive external or internal hemorrhage in the pelvic region an extrapelvic source must be excluded by routine chest X-ray and ultrasonography of the abdomen. If the hemorrhage is definitely caused by an intrapelvic injury, it must be determined whether it is possible to stabilize the patient hemodynamically. When hemodynamic stabilization is achieved, further examination of urological and neurological systems and definitive radiographic examination should follow. If, however, the patient remains hemodynamically unstable following initial resuscitative measures, massive blood transfusions should be instituted immediately. Additionally, in the presence of a significant posterior pelvic instability acute reduction and compression of the posterior pelvic ring, using a so-called "pelvic clamp," may be life saving in terms of hemorrhage control [29, 30]. In all other cases immediate surgical intervention is needed (Fig. 7). In all patients who then become hemodynamically stable further assessment can proceed.

When the primary evaluation indicates pelvic ring instability or fracture, oblique pelvic radiograph views (inlet/outlet) are completed as early as possible and the pelvic injury is classified (see Rommens "Diagnostic Procedures in Spine, Pelvic, and Extremity Injuries," this volume). After classification unstable fractures should be treated in the primary operative period. This is absolutely indicated, as only stable fixation of the fracture can prevent further hemorrhage, even in a stable hemodynamic situation, especially from the presacral and paravesical venous plexus. For stabilization of the pelvis both internal and external fixation methods are suitable. Symphyseal ruptures are treated with plate

osteosynthesis. External fixation is an adequate method of stabilization for transpubic instabilities in type B injuries and after posterior internal fixation of type C injuries. For stabilization of the posterior pelvic ring the supine position is preferred whenever possible. The anterolateral approach provides good visualization of the ipsilateral iliac wing and sacroiliac joint. Especially in polytrauma patients the supine position allows access to the patient for simultaneous procedures. Displaced sacral factures must be considered part of an unstable pelvic ring fracture (type C), and internal stabilization is therefore required. Complex pelvic fractures are combined with additional severe intra- or extrapelvic soft tissue trauma [30]. These concomitant injuries must be dealt with following control of the massive bleeding and internal stabilization of the pelvis,.

Unstable Spine Injuries

Acute spinal injuries with neurological involvement and/or relevant bony deformity, resulting in instability of the segment, are generally treated operatively, as nonoperative management does not always result in a stable situation. Also, increasing bony deformity, as a result of instability, can often lead to persistent pain or later neurological pathology [32–34].Furthermore, early operative treatment facilitates patient care and significantly shortens the length of hospital stay and immobilization, especially in multiple trauma patients [6, 22, 32, 34]. Most fractures and dislocations are reduced in a closed manner as soon as possible. CT subsequently helps in determining the exact location and type of the spinal injury.

Since immobilization of a cervical spine fracture is possible only with a halo fixator or continuous traction, an early operative approach is recommended. In the upper cervical spine (C0-C2) we prefer a dorsal approach for the C0-C1 junction, a ventral approach with screw fixation for dens fractures, and a ventral fusion of C2-C3 for Hangman's fractures. Unstable fractures of the lower cervical spine are generally treated via the anterior approach. This allows decompression of the spinal cord when a neurological deficit has been confirmed. Stable fixation is accomplished after decompression and removal of the disk with intercorporal spondylodesis. Stabilization is completed with an anterior plate fixation (H plate).

The treatment of thoracolumbar spine injuries is accomplished nowadays with closed reduction and correction of alignement. Early posterolateral decompression with consecutive transpedicular stabilization is then recommended. With burst fractures an additional ventral spondylodesis is required. In multiple trauma patients a two-step approach is then advisable. Unstable fractures of the thoracic spine seldom require treatment in the primary period. These are always associated with severe thoracic trauma and may extremely destabilize the thorax. ICU treatment is nearly impossible if the unstable spine is not fixed. Fracture dislocations in this region usually lead to a hemothorax after rupture of the pleura and bleeding from a segmental artery. The main goal of treatment is decompression of the spinal cord, followed by adequate stabilization. Operative technique and fixation device must guarantee a short operation period, a simple use of the device and preferably a short fusion of the spine.

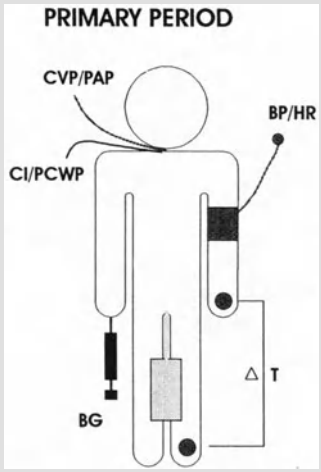

Fig. 8. Standardized monitoring of multiple trauma patients during the primary period. Monitoring includes pulmonary artery catheter (*CVP, PAP*) continuous measurement of arterial blood pressure (*BP*), and arterial blood gases (*BG*), body temperature difference (core to peripheral; *T*) and urinary catheter

Intensive Care Management During the Primary Period

Monitoring

After the primary operative period multiple traumatized patients require sensitive monitoring to permit immediate detection of signs of prolonged shock. Helpful parameters include: cardiac index, difference between extremity and core temperature, urinary output, and blood gas analysis (especially base excess). A pulmonary artery catheter, arterial line, and continuous capillary oxygen tension measurements are routine monitoring instruments in this issue (Fig. 8).

Treatment of Prolonged Hemodynamic Instability and Hypothermia

Hemodynamic instability in this period should be met with massive volume replacement, preferably with blood and blood plasma (i.e., fresh-frozen plasma), which provide the best volume effect and substitute for consumed clotting agents. Another target in ICU is the prevention of prolonged hypothermia [35, 36]. Warming of the gastric fluid, peritoneal lavage, and bladder lavage are recommended to prevent a decrease in core temperature below 32°C, the pathological cutoff point. Warming also has a positive effect on peripheral vasoconstriction [36].

Improvement in Pulmonary Function

The lung is known to be an important target organ in patients with multiple trauma; it is the first organ to deteriorate and is a major trigger factor for further organ damage [37]. Mortality is high in multiple injured patients with associated

blunt chest trauma, ranging from 15% to 50%. For this reason early diagnosis and treatment of the chest injury are essential for improving prognosis. Chest X-rays are unreliable in the primary period, and their findings are not correlated with clinical outcome or respiratory status. The diagnosis of "lung contusion" based on chest X-ray findings is often delayed, and respiratory insufficiency may develop before a diagnosis is made. Bronchoscopy therefore appears to be a more sensitive method of diagnosis, visualizing parenchymal and bronchial lesions [38]. The bronchoscopic removal of blood and aspirated fluid also helps to reduce postoperative complications, such as pneumonia. Bronchoscopically obtained bacteriological specimens are more representative than those taken from the tracheal secretion. A further advantage of bronchoscopy is the early detection and localization of bronchial injuries, leading to a bronchopleural fistula [39]. The indication for bronchoscopic occlusion technique of third and fourth order bronchi is:

(a) when the loss of mean minute volume is more than 40%, and changes of the ventilation mode shows no success, and
(b) when the patient is in a poor respiratory condition which prohibits extended operative intervention with resection of lung parts [39].

An operative approach is indicated with:

(a) rupture of a main bronchus or trachea, or
(b) extensive parenchymal defect at the pleural surface of the lung.

Another target in the treatment of respiratory insufficiency is to prevent or abolish developing atelectasis. Atelectasis can occur and impair pulmonary gas exchange especially with thoracic trauma but also with increasing pulmonary interstitial edema [40]. This leads to an increase of shunt fraction and hypoxemia, mechanisms contributing to the aggravation of traumatic hemorraghic shock. Therefore mechanical ventilation is adapted in a manner to prevent atelectasis, which is best accomplished by means of combining a limited maximal inspiratory pressure and establishing "intrinsic PEEP" [40]. Both help to minimize the injury caused by mechanical ventilation and provide an immediate "opening" of atelectatic areas [40]. The optimal flow, tidal volume, and pressure regimens are assessed under continuous arterial oxygen monitoring immediately after admission of the patient to ICU. These regimens must be controlled carefully in the first hours after admission and often need modification in this period preceding the establishment of a stable respiratory situation.A variety of studies, have clearly shown that patient positioning affects pulmonary oxygenation capacity. According to the gravity areas described by West et al. [41], fluid accumulates in the dorsal dependent areas of the supine patient. Significant atelectasis may develop in this region. In a patient prone to a variety of pulmonary complications especially due to interstitial posttraumatic edema, a change in body position is therefore a causal treatment with respect to the redistribution of pulmonary fluids. Many centers use intermittent prone and supine positioning in patients with pulmonary failure [41]. This method, however, is difficult to use in patients with abdominal trauma who are treated by the open abdomen technique.In addition, there is a high risk of dislocation of chest tubes and other essential lines.

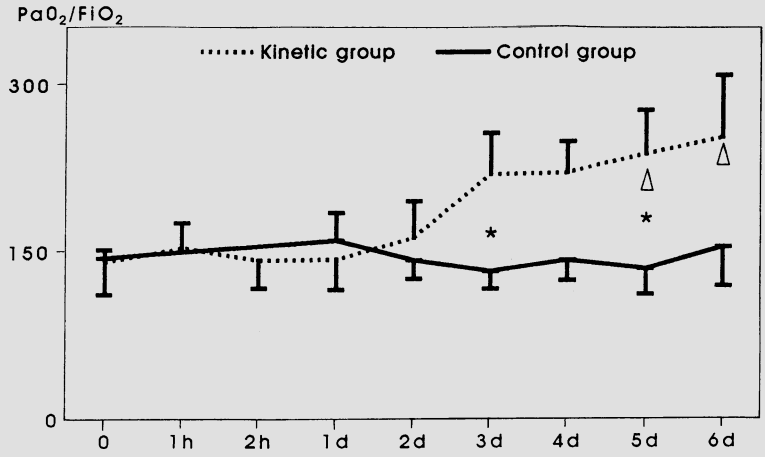

Fig. 9. Comparison of oxygenation ratio (PaO_2/FiO_2) in patients treated with standard and continuous kinetic bed positioning

Alternatively, a specialized bed allows continuous rotation along the body axis [42]. This device significantly improves the ability to perform nursing maneuvers, even in patients with an unstable thorax or with an open abdomen. Compared with patients submitted to conventional supine positioning, a significant improvement in oxygenation is measured within 3 days after positioning (Fig. 9). Reduction in interstitial fluid accumulation then follows, with a delay of hours to days. Changing body position therefore appears useful in patients with posttraumatic pulmonary failure. It probably does not replace extracorporal oxygenation in patients with endstage lung failure.

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Monitoring and Treatment of Acute Head Injury

R. Stocker, R. Bernays, T. Kossmann, and H. G. Imhof

Introduction

Head injury is still one of the leading causes of mortality and disability in young persons. After a period of dissatisfaction and frustration in treating these patients, the past few years have brought new insights in the pathophysiology of head trauma. These developments have provided new impulses for monitoring and treatment modalities.

The outcome of head injury is influenced markedly by secondary brain injury caused by biochemical and pathophysiological changes. Depending on the type and extent of the primary damage and on the severity of accompanying injuries the sequelae of head trauma are substantially affected by the promptness and quality of initial resuscitation, diagnostic procedures, surgical interventions, and posttraumatic intensive care.

Since the central nervous system (CNS) contains the sites of regulation for many organ systems (e.g., the circulatory, respiratory, endocrine, and immune systems), head injury not only affects the brain itself but disorganizes the whole organism. This has consequences for the treatment of the brain-injured patient. A comprehensive approach is required to create optimum conditions for the prevention of secondary brain injury and extracerebral damage and for the recovery of not definitively destroyed brain areas.

Basic Pathophysiological Principles

Primary Injury

The primary injury occurs within milliseconds to minutes and results from the mechanical effect of forces applied to the scull and brain at the time of impact (e.g., contusions to the surface of the brain, diffuse axonal injury and white matter damage, diffuse vascular injury). This results in severe focal or generalized neurological disturbances and in neuropathological changes which can lead to delayed deafferentiation and/or to secondary events [1].

There are two main categories of primary brain injury [2]. The first is diffuse injury, caused by a sudden acceleration or deceleration of the head, which leads to a brief cerebral concussion and to prolonged posttraumatic coma due to diffuse

axonal injury. This type of injury may sometimes be accompanied by subdural hematomas resulting from ruptured bridging veins and the contre coup contusion. The second type of primary brain injury is the focal injury, caused by direct blows to the skull leading to contusions, brain lacerations and (extradural, subdural, intracerebral, subarachnoidal) hemorrhage. Diffuse and focal brain injury have different mortality rates (24% versus 41%, according to the Traumatic Coma Data Bank in the United States), suggesting relevant differences in their pathophysiology. Even with the same injury type the mechanism that produces the damage significantly influences outcome [3].

Pathophysiological Cascade in Diffuse Brain Injury. The initial impact leads to a primary defect in the axonal membrane, resulting in ionic shifts (particularly of calcium) within the axon and in axonal depolarization. This results in impaired transmission within the neuronal network, which leads to widespread neurological dysfunction and primary coma not caused by compression. The primary forces cause axonal strain, which tends to occur at the nodes of Ranvier. This can lead to disruptive damage (immediate axonotmesis), followed by either degeneration or regeneration. More commonly, nodal strain leads to internal axonal damage and results in either secondary axonal degeneration, caused by excessive accumulation of calcium ions within the cells or in self-repair with restoration of presumably normal axonal structure and function [4].

Pathophysiological Cascade in Focal Injury. A focal blow to the skull causes local mass effects from contusion or hematoma. This can secondarily cause brain shift, herniation, and brain stem compression – events preceding the loss of consciousness. Prolonged compression results in brain stem hemorrhage. On the cellular level focal injury is believed to cause concentric areas of progressive damage severity (penumbra zone) around the point of direct impact. Localized structural disruption may occur in the area closest to the primary insult. The surrounding area includes primary traumatic damage without destruction of the tissue itself. The next surrounding area potentially subjected to delayed insult includes a zone of ischemia and edema, influenced by inflammatory and cytotoxic mechanisms [4]. Not infrequently diffuse and focal damages are combined, featuring elements of both types of injury.

Secondary Brain Damage

Secondary brain injury occurs minutes, hours, or days after the initial impact. Secondary brain injury represents complicating processes initiated by the primary injury, such as ischemia/reperfusion, brain swelling and edema, intracranial hemorrhage, and intracranial hypertension. These may substantially aggravate the primary damage and/or prevent recovery of not definitively destroyed brain areas. Factors that aggravate the initial injury include hypoxia, hypotension, anemia, hypovolemia, hyper- and hypocarbia, and hyperglycemia. Prevention or prompt treatment of these secondary factors improves outcome after head injury.

Seizures, infection, and sepsis may add to the effects of secondary brain injury and must also be prevented or treated promptly.

Ischemia is the leading cause of secondary brain damage [4]. This is due either to inadequate gas exchange (e.g., hypoxemia, severe hypocapnia), to impaired systemic and/or cerebral circulation [e.g., hypotension, increased intracranial pressure (ICP) with reduction in cerebral perfusion pressure (CPP)], or to increased oxygen demand without adequate increase of oxygen supply (e.g., high fever, seizures). Postmortem examination reveals ischemic cell destruction in more than 85% of patients who die from acute head injury [5, 6]. Areas prone to ischemic damage are located predominantly within the arterial boundary zones of the cortex. Many of these ischemic episodes occur at the scene of the accident, during transfer to or within the hospital, especially if initial resuscitation and surgical intervention is delayed, or monitoring is insufficient. Considerable risks leading to ischemia are also present in the posttraumatic course (e.g., ICP increases, cardiovascular instability, uncontrolled hyperventilation, epileptic seizures), while these often can be prevented by well-trained personnel, adequate monitoring, and immediate treatment.

Increases in ICP with potential decrease in CPP (CPP = MAP-ICP-$P_{jugular\text{-}venous}$) after severe head injury is a common feature, even in unconscious patients with minor or no pathological findings on computed tomography (CT) [7] and contribute considerably to secondary damage, morbidity, and mortality. Because the brain is enclosed in a nondistensible compartment, intracranial volume is constant. Volume relationships are described by the Monro-Kelly relationship: $V_{intracranial} = V_{brain} + V_{CSF} + V_{blood} + V_{mass\ lesion} = \text{constant}$.

Intrinsic compensatory mechanisms can counterbalance increases in volume due to mass lesions, edema, or augmented cerebral blood volume, for example, by depleting CSF or by brain compression, provided sufficient time exists for these compensatory mechanisms to become effective. Rapid alterations in intracranial compliance following head injury can result in important adverse changes in ICP, with relatively small changes in the volume of any of the intracranial volume components. The pressure-volume index (PVI) is a measure of the change in ICP secondary to changes in intracranial volume. ICP in normal individuals is less than 10 mmHg. Pathological increases in ICP or decreases in CPP after head injury are correlated quantitatively and qualitatively with outcome [8]. Early ICP elevations are due to expanding lesions within the skull caused by interstitial vasogenic edema (microvascular damage, breakdown of the blood brain barrier) and/or vascular engorgement with an increase of cerebral blood volume (CBV). Later, vasogenic brain edema is often combined with cytotoxic intracellular edema. This occurs as a consequence of ion pump failure in the cell membrane as a consequence of intracellular calcium overload or ischemia [9].

The end-points of an uncontrollable ICP increase include ischemia and infarction, pressure necrosis in parahypocampal gyri, tentorial and transforaminal herniation, brain stem infarction, and interruption of cerebral circulation. Several mediators (i.e., bradykinin, arachidonic acid, histamine) can induce the breakdown of the blood-brain barrier and induce vasogenic and/or cytotoxic brain edema [10]. Free radicals are released under several pathological conditions

(e.g., reperfusion after ischemia) but induce only irregular tracer leakage. They also have been found to induce cytotoxic edema. The role of cytokines (e.g., interleukins, ILs) in the development of brain edema has not yet been elucidated.

The excitatory amino acids glutamate and aspartate appear to play a major role in the events leading to cytotoxicity after head trauma [11]. They induce an overactivity of the *N*-methyl-D-aspartate-type glutamate receptor (NMDA receptor), which leads to a marked augmentation in calcium channel permeability. The increase in intracellular calcium concentration stimulates protease activity and activates lipases. It further deregulates control systems in damaged brain areas which normally protect the cell against cytotoxic effects from excitatory amino acids and induce irreversible destruction of the mitochondria. The consequence is an increase in extracellular potassium and the breakdown of ATP-dependent ion pumps, followed by cell death and release of water, calcium, and excitatory amino-acids, which in turn leads to astrocyte swelling and again activation of the NMDA receptor of neighboring cells.

Systemic Sequelae of Head Injury

Acute head injury causes profound alterations in the organ systems, teleologically directed toward preservation of vital organ functions, and to adequate oxygen and substrate delivery to the brain. Examples include the cardiovascular response and autoregulation of the cerebral vascular bed in order to maintain cerebral perfusion and blood flow. Neurohumoral responses mediated by the brain injury itself or by pain, stress, hemorrhage, or humoral mediators may result in disturbances of the neurohumoral axis. For example, hypersecretion of ADH (syndrome of inappropriate ADH secretion) may predispose to water intoxication; conversely, injury to the pituitary gland or hypothalamus may impair production or release of ADH [12]. Additionally, the pituitary response to head injury includes the release of growth hormone and ACTH, which induce the release of steroids.

Hypothalamic activity regularly leads to profound sympathetic stimulation, with release of norepinephrine, epinephrine, and dopamine. Both humoral and sympathetic stimulation predispose to fluid and electrolyte disorders, hyperglycemia, protein catabolism, and an increase in aldosterone secretion.

Catecholamine release is part of a generic central nervous response to injury and stress [13]. Within seconds after trauma the increase in norepinephrine and epinephrine release may be 100-to 500-fold and induce acute changes in serum electrolytes (e.g., hypokalemia), myocardial ischemia, subendocardial necrosis [14], repolarization abnormalities, arrythmias, hypertension, disturbances of vasoregulation, and neurogenic pulmonary edema due to endothelial damage.

Release of nonspecific substances from the brain can induce multisystem dysfunction by initiating unspecified biochemical cascades; conversely, these cascades may result from systemic trauma and affect cerebral function. For example, the highest concentration of tissue thromboplastin is located in brain

tissue, whereas tissue plasminogen activator is found in the choroid plexus and meninges. Injury to the cerebral parenchyma therefore may result in disseminated intravascular coagulation or in a fibrinolytic consumptive coagulopathy [15].

As mentioned above, activation of cytokine cascades (i.e., IL-1, IL-2) occurs, but their role in mediating systemic sequelae is not yet fully understood [16].

Further systemic risks of brain injury include impairment of cardiovascular and respiratory functions, loss of voluntary and reflex control, resulting in oropharyngeal airway obstruction, loss of the coughing reflex, immobilization with consecutive disorders in calcium metabolism, muscle atrophy and potential flebothrombosis, and gastrointestinal dysfunction potentially leading to ileus and stress ulcer bleeding. Additionally, many pathological events arise from artificial ventilation, infectious complications, or concomitant injuries.

Monitoring in Acute Head Injury

Basic Monitoring

Basic monitoring after severe head injury includes common cardiovascular and respiratory monitoring devices (e.g., ECG, arterial line, central venous line, urinary catheter, arterial blood gas analysis, capnography) as well as special monitoring techniques all of which are discussed in detail below.

Monitoring of Intracranial Pressure

At our institution continuous ICP monitoring is performed regularly in every patient with: (a) a Glascow Coma Score (GCS) below 8 after initial resuscitation, (b) a GCS of 8 or higher with positive CT findings if the patient is intubated or neurological assessment is impossible, or (c) a neurological deficit in patients in which neurological reevaluation is impossible due to long-term sedation (major surgery, need for controlled ventilation, sedation for other reasons). An intraventricular catheter is used as the primary monitoring device. This allows continuous registration of ICP and treatment of ICP increases by means of cerebrospinal fluid (CSF) drainage. In patients with minor head trauma requiring ICP monitoring and in patients in whom the use of an intraventricular device is not feasible (e.g., with major brain shift), a subdural epiarachnoidal catheter (Wilkinson cup) is used. Epidural devices are not used because of their lower reliability. In some special cases we also measure ICP by means of camino catheters (Camino, San Diego, CA). ICP is registered continuously and treated according to protocol.

Bedside Jugular Bulb Oxymetry and Sequential Measurement of Arterial to Jugular Differences in Lactate Concentration

The insertion of a fiberoptic catheter (Oximetrix 5.5-F PA catheter, Abbott, North Chicago, IL) in the jugular bulb allows continuous registration of the global relationship between oxygen delivery and oxygen consumption of the brain, assuming that the hemoglobin concentration is kept constant [17]. However, this method cannot identify the simultaneous occurrence of luxury perfusion and ischemia. Therefore we also sequentially measure arterial to jugular differences in lactate concentration [18]. The combination of these two techniques allows the monitoring of adverse effects of hyperventilation and the avoidance of ischemia induced by hypocapnia. This monitoring is used in all normoventilated patients in whom ICP increases above 15 mmHg in spite of mild osmotherapy. Since January 1992 we have used 210 jugular bulb catheters in 180 patients. In our experience, desaturations and/or widening of arterial to jugular differences in lactate occurs very frequently (in more than 70% of patients), even during moderate hyperventilation and during the whole course of treatment. Hence, we believe that blind hyperventilation after head injury should be avoided [19].

Registration of EEG and Somatosensory Evoked Potentials

We perform sequential registration of EEG and somatosensory evoked potentials (SEP) in all patients in whom clinical assessment of neurological function is not possible. The first investigation takes place within the first 24 h. It includes an assessment of the EEG [wave form, modulation of frequency spectra (vigilance), reactivity induced by external stimulation] and evaluation of SEP components. By stimulation of the median nerve at the wrist SEP are recorded at several points between the midpoint of the clavicle and the scalp, overlying the area of the sensomotor and frontal cortex [potentials of the posterior nuclei C2 (N14), thalamus (N15), somatosensory (N20) and frontal cortex, central conduction time (CCT)]. The SEP of each side is graded in terms of alterations in potential and CCT (normal SEP, grade I; pathological SEP, grades II-IV). Retrospective analysis in 108 patients admitted to our ICU showed a significant correlation between SEP grade and GCS ($p < 0.005$), pupillary function ($p < 0.0005$), and outcome (especially if the N20 component was missing bilaterally; $p < 0.005$) [20]. The combination of the two investigations allows the estimation of outcome [20]; the detection of epileptic activity and therapy control requires repeated EEG registrations. During barbiturate coma, continuous EEG registration is mandatory to maintain a safe relationship between benefit and adverse effects. Our goal is to achieve a burst suppression pattern with 6 bursts/min and a burst suppression relationship of 1:1.

Transcranial Doppler Investigations

Especially patients with blood in the subarachnoidal space are at risk of developing vasospasm and ischemia. Determination of blood flow velocity (V) in the middle cerebral artery (MCA) and in the intracranial portion of the internal carotid artery (ICA) and computation of the VMCA/VICA ratio and pulsatility index permits the detection of vasospasm and for surveillance of treatment. Vasomotor CO_2 reactivity and cerebral autoregulation can be tested by monitoring flow velocities in either the middle cerebral arteries as a function of changes in arterial CO_2 or arterial blood pressure. Maintained CO_2 reactivity and autoregulation seem to be of positive prognostic value.

Computed Tomography Follow-Up

CT often does not demonstrate all pathological consequences of brain injury within the first hours or days after injury. Furthermore, secondary injuries are not infrequent and are not detected by early CT. In 45% of 129 patients CT performed in the later posttraumatic course showed a deterioration, a finding independent of the initial GCS and the initial diagnosis of circumscribed or diffuse injury. The deterioration most often observed was bleeding into a preexisting contusion or even into a region that looked normal on the first investigation. In the vast majority of cases this occurred within the first 2 weeks after trauma. Therefore we repeat the CT early in the posttraumatic period, even in patients with mild to moderate head injury. For the same reason we believe that patients with diffuse brain injury should be transferred to a neurotraumatological unit for specialized management, independent of the need of immediate surgical intervention [21].

Neuroimmunological Monitoring

The role of cytokines in the pathophysiology of acute brain injury is still unknown. We performed an enzyme-linked immunosorbent assay analysis of CSF samples obtained from CSF drainage in nine patients whose ICP exceeded 15 mmHg within 3–14 days after injury. Peripheral blood samples were assayed concomitantly. Preliminary results showed the presence of IL-6 in all samples. IL-6 was released abundantly during the first days (days 1–8) [22]. IL-6 levels in CSF were highest in patients with the most severe brain injury, while only small amounts were found in serum [23]. When tested in vitro, IL-6 stimulated the migration activity of cultured astrocytes in a dose-dependent manner, and promoted the synthesis of transforming growth factor-β [24]. IL-2 receptor (IL-2r) was measured only in serum and peaked in the middle or towards the end of the study period. These results indicate that major differences in terms of lymphocyte activation and function occur between the CNS compartment and the systemic circulation. IL-8 was found in the CSF of all patients and peaked in the second week after injury. IL-8 was also observed in the serum but never in the same

amount as measured in the CSF. Our results clearly show that the cellular immune system of head-injured patients is activated in the central nervous compartment and in the systemic circulation. The role of cytokines in the inflammatory events and immunomodulation after head injury, their value as indicators for the severity of brain damage, and for the efficiency of treatment need further investigation.

Pressure Volume Index Timed Wake-Up in Head-Injured Patients

The necessity of sedation after head injury is clearly established, but the timing of wake-up is unclear and critical, because stopping sedation and weaning from mechanical ventilation is often associated with elevated ICP and deterioration in CPP [25, 26]. To coordinate respiratory care with protection of the brain against secondary cerebral injuries we use the PVI, as defined by Marmarou [27, 28], as a predictor of outcome upon waking-up. Outcome is defined as an ICP remaining below 15 mmHg or rising above 15 mmHg, which requires resedation and continued mechanical ventilation. PVI is a valuable parameter for defining brain tightness or cerebral compliance [29], which represents the compressibility of the brain's vascular system [30]. In clinical practice PVI is a valuable decision-making tool for further diagnosis and therapy [31, 32] and is an indicator for an impending rise in ICP [33, 34] and for prognosis of neurological outcome. Some authors have criticized PVI calculations [35], stating that they are difficult to perform, time-consuming, susceptible to artifacts, and without correlation to pulsatile ICP changes.

PVI Measurement Protocol. We improved PVI calculations by introducing a new, easy-to-use, computer-based protocol which produces reliable and stable PVI values during 30 min of bedside testing. There may be considerable variability in sequential PVI values, due partially to baseline floating (i.e., B-waves) [36, 37]. In a majority of recorded pressure curves under ICU conditions it is insufficient to consider only cardiac and respiratory cycles to choose P_o and P_p (P_o = median preinjection/withdrawal ICP; P_p = median postinjection/withdrawal ICP value) (Fig. 1). Using Marmarou's standard equation, we developed a computer-based protocol for sampling PVI values, which significantly improves the technique for determining PVI (Fig. 1). PVI calculations are started within the first 24 h after insertion of a ventricular catheter and are repeated at 24-h intervals. Preceding a PVI study ICP is recorded at slow speeds over 5 min for assessing the recording quality and possible baseline floating.

In patients with an ICP below 15 mmHg 0.5–3 ml Ringer's solution at room temperature is injected at 1 ml/s into the lateral cerebral ventricle; in those with an ICP above 15 mmHg 0.5–3 ml CSF is drained at 0.5 ml/s. Usually 5 (no baseline floating) to 15 (considerable baseline floating) PVI calculations, bolus injections, and CSF drainages of defined volumes alternately are performed per study. One study is conducted per day. Using a mouse driven computer cursor, two-time

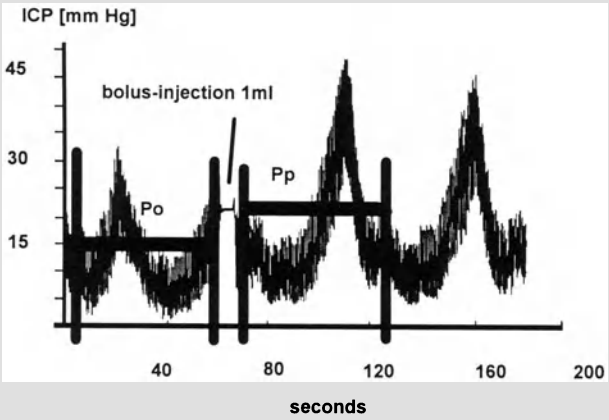

Fig. 1. ICP alterations induced by small intraventricular bolus injection. Time windows of P_o and P_p determined by the slowest underlying wave period. P_o, median preinjection ICP value; P_p, median postinjection ICP value

windows are selected before and after injection/withdrawal (Fig. 1). From these, P_o and P_p are determined and PVI is calculated. Depending on the amount of baseline floating, this PVI calculation is repeated 5–15 times. A median PVI is then calculated and recorded as the PVI for that day. A PVI study takes approximately 30 min per patient.

Hardware and Software Used for Recordings. A commercially available analogue/digital data acquisition board (8-channel, 12-bit resolution, 100 kilosample/s, Keithley MetraByte) with data acquisition and analysis software (Easyest LX, Keithley Asyst) has been installed in a 486-PC. This equipment enables us to program automated recording protocols and bedside PVI calculations of the recorded data. All recordings and calculations can be visualized on the PC screen and printed out. PVI calculations are able to detect the patients likely to develop elevated ICP during the weaning phase. Indeed, several patients with a PVI below 18 ml underwent unsuccessful wake-up attempts due to elevated ICP. PVI calculations also detect patients who would tolerate wake-up. Further experience and a higher number of investigated patients are necessary to confirm the reliability and show the range of confidence of this computer-based PVI assessment system for timing of wake-up of head-injured patients after sedation or barbiturate induced coma to prevent secondary cerebral insults during this critical phase [38].

Treatment Protocol of Acute Head Injury

Goals of Treatment

The main goals of treatment include:

1. Immediate life support, especially achievement and maintenance of adequate gas exchange and circulatory stability by means of intubation, artificial ventilation, adequate volume replacement and, if necessary, vasoactive drugs
2. Early diagnostic procedures and, if needed, surgical treatment (evacuation of mass lesions); repeated CT for detection of surgically treatable pathological findings
3. Profound sedation and analgesia to avoid stress, fighting against the ventilator, and cardiorespiratory instability
4. Achievement and maintenance of optimal conditions (cerebral perfusion, cerebral oxygen balance), allowing recovery of damaged brain areas and prevention of secondary brain injury, monitored by continuous ICP registration, jugular bulb oximetry, assessment of the arterial to jugular difference of lactate, transcranial Doppler investigations, and repeated or continuous EEG registration
5. Prevention of stress ulcers, maintenance of gut integrity, pneumonia prophylaxis, employing early enteral feeding; prevention of hyperglycemia (risk of CNS acidosis)
6. Repeated bacteriological sampling and anti-infectious treatment if necessary

Therapeutic Tools

In addition to basic intensive care, the following therapeutic tools serve to improve conditions which allow prevention of secondary damage.

Monitoring. To optimize the circulation we use pulmonary artery catheters liberally. This tool may help to optimize fluid/volume replacement and guide catecholamine support. In our unit the use of a pulmonary artery catheter is mandatory in patients in whom barbiturates are used, because of their circulatory depressant properties.

Cerebrospinal Fluid Drainage. CSF drainage reduces the volume normally present in and taken up from the CSF compartment. The daily amount of drained CSF can exceed daily production, demonstrating a net drainage from the interstitial space. In moderate brain edema CSF drainage is often sufficient to control ICP.

Hyperventilation. Where cerebral autoregulation and CO_2 responsiveness are preserved, a decrease in pCO_2 by 2–4 mmHg can result in a 1 mmHg decrease in ICP due to cerebral vasoconstriction and a concomitant decrease in cerebral blood volume. In our experience (see above) and according to several recently published papers, uncontrolled and long-term hyperventilation may be danger-

ous because hypocapnia induces ischemia and loss of bicarbonate buffer, leading to CSF acidosis [39]. Therefore the effects of hyperventilation should be monitored at least by means of jugular bulb oxygen saturation and arterial to jugular lactate differences. Regretfully, there exists as yet no other bedside monitoring that is more feasible and reliable for assessing cerebral blood flow.

Osmotherapy. The use of mannitol in controlling intracranial hypertension is well established. In addition to its properties for decreasing brain water content, there is some evidence that mannitol reduces cerebral blood volume through vasoconstriction [40]. Additionally, mannitol may serve as an oxygen radical scavenger. Potential disadvantages include induction of osmotic diuresis with concomitant hypovolemia, electrolyte disorders, and adverse effects on blood viscosity. Fast infusion and/or administration of large amounts may lead to a rebound increase of ICP. We therefore restrict the administration of mannitol if serum osmolarity exceeds 315 mOsm/l. In all cases the infusion rate is low and the bolus given is small (for example, 25 ml within 30 min).

Mild Therapeutic Hypothermia. Recent experimental studies and patient data have demonstrated that mild hypothermia (33°–34°C) may improve outcome after severe head injury [41, 42]. It has been demonstrated that hypothermia significantly reduces ICP, CBF, and cerebral metabolic rate of oxygen ($CMRO_2$) and increases CPP. Additionally, there is some evidence that hypothermia reduces the excretion of excitatory amino acids and stabilizes cell membranes. Available data show no adverse effects. However, it is not clear whether the attenuation of the immune response to infections induced by hypothermia poses a higher risk of infection. Hence we keep our stable patients within a normal temperature range (36°–37°C) and combine mild hypothermia with the use of barbiturates in the others.

Barbiturate Coma. Although the lowering effect of barbiturates on ICP is well accepted, controversy still exists about the quality of outcome if barbiturates are administered. Barbiturates decrease $CMRO_2$ and CBF in a dose-dependent manner by decreasing synaptic transmission, presumably by affecting GABA transmission. Barbiturate-induced isoelectric EEG results in a reduction of $CMRO_2$ and CBF by approximately 50% and in an increase in intracellular energy stores. Barbiturates increase cerebrovascular resistance and decrease CBV and ICP [43]. Other postulated or verified actions include the protection of compromised penumbra zones of ischemia, shunting of blood to regions of reduced perfusion, anticonvulsant effects, stabilization of lysosomal membranes, decreasing excitatory neurotransmitters and intracellular calcium, and free radical scavenging.

A number of risks accompany the high-dose administration of barbiturates. Barbiturates are cardiovascular depressants, impair gastrointestinal and immune function, and are potent liver enzyme inductors. Because of their negative effects on cardiovascular function we start barbiturate coma only in patients with stable cardiovascular function, who are monitored by means of a pulmonary artery

catheter and are not responsive to conventional therapy of increased ICP (CSF drainage, jugular bulb guided hyperventilation, osmotherapy). To obtain an optimal relationship between benefit and adverse effects therapy is guided by continuous EEG registration. Achievement of a burst suppression pattern is the main goal. The therapeutic end-points include: (a) success (ICP within normal range for 24 h, ICP control with standard therapy is possible after withdrawal of barbiturates), (b) failure (diagnosed brain death, uncontrollable ICP). (c) development of intolerable side effects requiring discontinuation of barbiturates (hypotension or low output not responsive to volume loading, inotropes or vasoactive drugs).

Step-by-Step Treatment After Severe Head Injury

Basic Treatment. The points of basic treatment are:
- Early intubation (if possible at the site of the accident) and artificial ventilation; goals:
  (a) $pO_2 \geq 13$ kPa,
  (b) normocapnia,
  (c) no prophylactic hyperventilation (risk of ischemia)
- Sufficient analgesia, sedation and relaxation
- Maintenance of normal body temperature, prevention of hyperthermia and hyperglycemia
- Aggressive circulatory stabilization by means of volume/fluid replacement and catecholamines if necessary; goals:
  (a) mean arterial pressure > 80 mmHg,
  (b) normovolemia,
  (c) hematocrit > 30,
  (d) no antihypertensive drugs up to MAP = 130 mmHg (range of autoregulation) if patient is adequately sedated (demand hypertension),
  (e) maintenance of a CPP $\geq$ 70 mmHg
- No routinely performed head elevation (lowers CPP, increases volume sequestration)
- Surgical intervention if necessary (CT)
- Continuous ICP monitoring (ventricular drainage catheter if possible, subdural Wilkinson cup catheter, camino catheter)
- Administration of nimodipin in cases with Doppler sonographic signs of vasospasm

ICP Increases. In the case of increases in ICP (threshold ICP > 15 mmHg > 5 min) the following are recommended:
- Deepening of sedation, analgesia, muscle relaxation
- CSF drainage if possible
- Insertion of a fiberoptic jugular bulb catheter
- Hyperventilation as long as
  (a) $Sv_jO_2 > 60\%$,

(b) a-jDL < 0.2 mmol/l,
(c) ICP lowering by means of decrease in $PaCO_2$ is possible
- Osmotherapy: mannitol IV 25–50–100 ml (slowly; risk of rebound) as long as serum osmolarity is < 315 mOsm/l
- Moderate hypothermia (± 34 °C)
- Barbiturate coma under continuous EEG registration; goal:
  (a) burst suppression pattern, < 6 burst/min,
  (b) burst suppression relation 1:1
  (Start with thiopental 10 mg kg^{-1} min^{-1}; this therapy may lead to hemodynamic deterioration and requires close monitoring and cardiocirculatory support, guided by the use of a pulmonary artery catheter)
- Increase of MAP to meet a CPP > 70 mmHg
  (If cause of ICP increase is unknown: CT)

Wake-Up. The wake-up procedure includes stopping sedation and muscle relaxation. The ventilator settings are changed as soon as possible to an assisted ventilation mode.

The first wake-up is performed only if the following conditions are met:
- No ICP increase above 15 mmHg within 24 h under normothermia and normoventilation without treatment
- PVI > 18 ml
- Amount of CSF drainage below 50 ml/24 h
- No signs of intracranial hypertension on CT
- Sv_jO_2 and a-jDL within normal range

The wake-up is stopped and treatment resumed if ICP increases above the threshold for more than 5 min.

Discontinuation of ICP Monitoring. ICP monitoring is discontinued as soon as a neurological assessment is possible, and no therapeutic interventions have been necessary over the past 24 h.

Epidemiology and Our Own Results

Since we established multimodality monitoring and treatment at the beginning of 1992, 316 patients with isolated or combined acute head injury have been treated. Of these, 71% were men and 29% women; 54% were younger than 40 years and 18% older than 60 years. In 41% the initial GCS was lower than 8, in 23% between 8 and 12, and in 36% higher than 12. Overall mortality was 17%; 27% died during the first day after injury and 48% within the first 48 h after admission. Causes of early death were uncontrollable intracranial pressure or admission to confirm brain death.

Glascow Outcome Score (GOS) was assessed within 4–6 months after injury. GOS was 2 in 2%, 3 in 13% (more than half of these patients were older than 55 years), 4 in 20%, and 5 in 65%. There were no survivors who persisted in vegetative state (GOS 1).

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Treatment of Abdominal Injuries

F. D. Battistella and F. W. Blaisdell

General Principles

Exploration of the abdomen for trauma is best carried out utilizing an upper midline incision from the xiphoid to the umbilicus. Once pathology is encountered in the abdomen, the incision can be extended around the umbilicus and down to the pubis if necessary to permit adequate exposure of the injuries and an inspection of the entire abdominal cavity. The incision can also be carried upward as a midline sternotomy if necessary to treat a thoracoabdominal injury.

Upon entering the abdomen, initial assessment should be made for bleeding. If blood is encountered, all four quadrants of the abdomen should be rapidly inspected, and when bleeding is detected that quadrant should be temporarily packed while assessment of the remaining quadrants is completed. This prevents one from concentrating on the wrong lesion, such as proceeding with splenectomy in the face of a massive injury to the right lobe of the liver. After a quick inspection of the four quadrants and a brief look at the central portion of the abdominal cavity, the area perceived as the source of the major hemorrhage should be uncovered and the pathology assessed; the injury with the most active and vigorous bleeding should be given initial treatment priority.

Following blunt trauma the lesions most often responsible for bleeding are those of the spleen, liver, and mesentery (in that order), with retroperitoneal bleeding from a kidney rupture a distant fourth. In penetrating trauma the probability of bleeding corresponds to the mass of the organ; therefore, the liver is most commonly responsible for hemorrhage, followed by major blood vessels in the mesentery or in the retroperitoneum and, lastly, the spleen.

Treatment of Specific Abdominal Injuries

Splenic injury is the most common problem encountered overall and is most often responsible for major ongoing abdominal hemorrhage. The decision of whether or not splenectomy is required relates to the extent of the injury and the amount of hemorrhage encountered at laparotomy. If blood loss is more than 1 l, and the spleen is the primary source, a splenectomy is usually indicated. If blood loss is less than 500 cm^3 at the time of laparotomy, there is a high probability that the spleen can be saved; most injuries other than those to the hilum lend themselves to

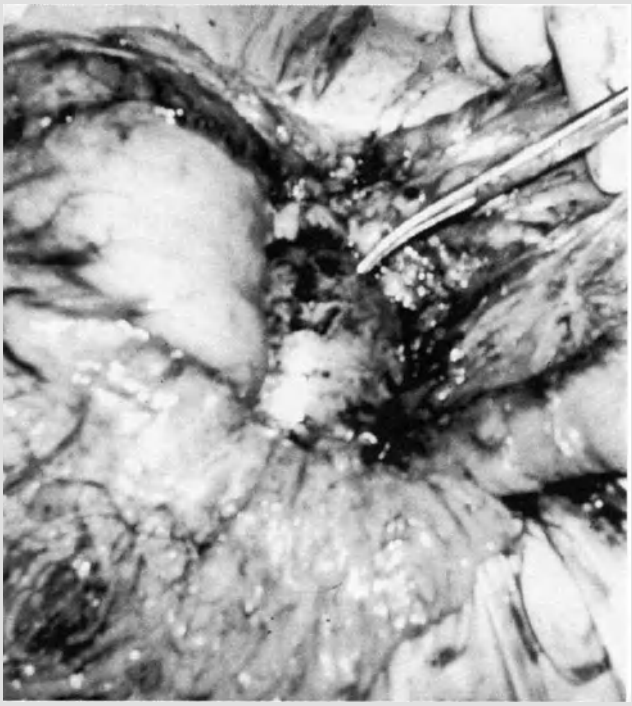

Fig. 1. Repair of a deep splenic capsular laceration can be performed using strips of Teflon as bolsters to prevent mattress sutures from cutting through the spleen

splenic salvage. When the blood loss is between 500 and 1000 cm^3, splenic salvage may or may not be indicated. When there are other associated major abdominal or major orthopedic injuries, splenectomy is usually the optimal treatment. The exception is with children, in whom greater efforts should be made towards splenic salvage. If this is not possible, replantation of splenic tissue slices in the omentum may be of some value [2]. If blood loss has been modest, blood and clot around the spleen should be removed. If the spleen is oozing, the area should be packed and the remainder of the exploration and treatment carried out. If the initial blood loss is minimal, no treatment is necessary while other injuries are addressed. If splenic oozing persists, application of a hemostatic sponge or microcrystalline collagen is indicated. If this does not control the bleeding, and the spleen appears salvageable, it should be mobilized medially, the short gastric communications divided, and a vascular clamp placed across the hilum of the spleen. In a dry field the injury can be fully assessed, and specific treatment such as repair of a capsular injury or hemisplenectomy can be carried out (Fig. 1).

Most *liver injuries* are associated with spontaneous hemostasis. If inspection of a liver injury following evacuation of blood and clot reveals minimal bleeding, the liver should be left alone and reinspected prior to abdominal closure. Similar treatment criteria as for splenic injuries can be applied to liver injuries. If the blood loss has been less than 500 ml, most liver lesions can be treated with evacuation of the hemoperitoneum and benign neglect. If the blood loss is more than 500 ml, drainage is required; however, unless bleeding is active no treatment

other than the application of hemostatic sponges or microcrystalline collagen should be required. Occasionally a biologic pack of omentum placed over the injured area offers a better alternative. In our experience, 90% of liver lesions can be managed by one of these methods. In the few cases in which active bleeding persists the liver should be mobilized to allow inspection of the entire depth of the laceration. Utilizing an extended midline incision, the lateral attachments of the liver can be divided, and the liver is then brought up into the field for inspection. If hemostasis is obtained by a vascular clamp placed across the portal triad, the depth of the laceration can be inspected for the source of the hemorrhage and these blood vessels oversewn.

If mobilization of the liver and application of a portal triad clamp results in persistent or aggravation of the bleeding, the problem is most likely hepatic vein injury. These injuries are best treated by packing the liver to control the hemorrhage. This should be successful in two-thirds to three-quarters of these instances. The patient's abdominal incision is then closed,, and reoperation is carried out 24–36 h later for removal of the packs. At this time hemostasis is usually complete and drainage of the perihepatic area is all that is necessary. If devitalized liver tissue is present, it should be débrided or resected.

In 3%–5% of liver injuries, particularly those with extensive posterior injury, there is uncontrollable hemorrhage due to major hepatic vein tears at the level of the vena cava. In such cases a caval shunt or bypass is indicated [3]. This is best accomplished by extending the abdominal incision to a midline sternotomy and placing a transatrial catheter so as to shunt blood from the infrarenal vena cava to

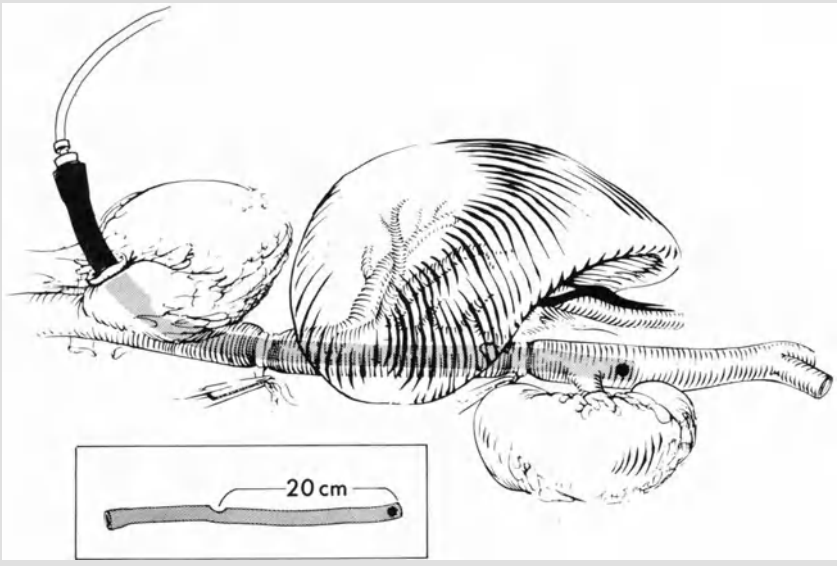

Fig. 2. Use of a transatrial vena cava shunt. When the portal triad is clamped and tapes pulled up around the cava above and below the liver, the liver and retrohepatic cava can be exposed in a dry field

the atrium (Fig. 2). An alternative is to control the bleeding with packs while the femoral vein and the jugular vein or right atrium are cannulated for caval bypass. Following decompression of the inferior vena cava the application of a hilar clamp will provide complete hemostasis. The presence of a dry field permits repair of the venous injury and/or resection of the damaged liver.

Mesenteric lacerations are generally peripheral where hemostasis can be easily achieved. Occasionally, particularly with penetrating trauma, a central vascular injury is present. If the bleeding cannot be easily controlled in the small bowel mesentery, a vascular clamp placed across the base of the mesentery just inferior to the pancreas may control the bleeding so that vascular repair can be performed. If this is not successful in controlling the bleeding, the origin of the mesenteric artery can be reached by an extensive Kocher maneuver combined with mobilization of the right colon and the mesentery (Fig. 3). An alternate approach is the opposite maneuver in which spleen, stomach, pancreas, and left colon are mobilized medially and ventrally. This exposes the aorta from the diaphragm to its bifurcation and permits control of the mesenteric vessels and also the celiac axis. With the origin of the mesenteric artery cross-clamped, distal dissection is carried out for exposure and repair of any mesenteric artery or vein injury.

Kidney bleeding is manifested by a hematoma in the lateral retroperitoneum. If the hematoma is not expanding at the time of operation, it should not be entered.

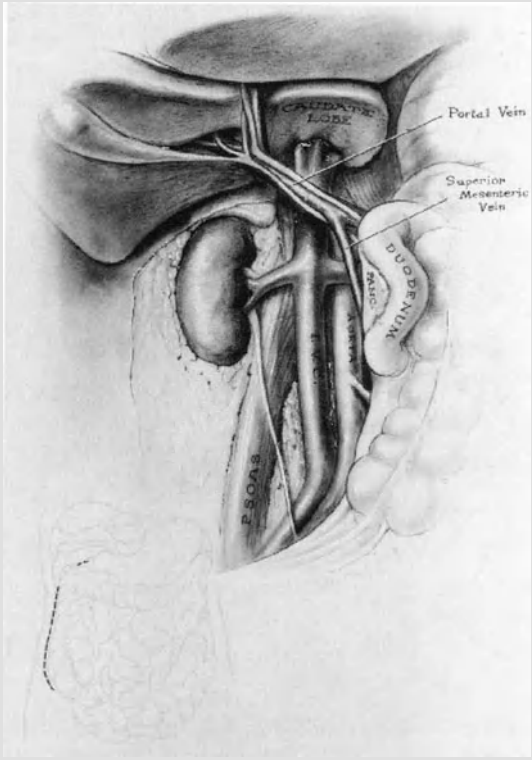

Fig. 3. Exposure of the superior mesenteric artery, aorta, and extrahepatic vena cava. This is accomplished by mobilizing the right colon, duodenum, and head of the pancreas upward to the left

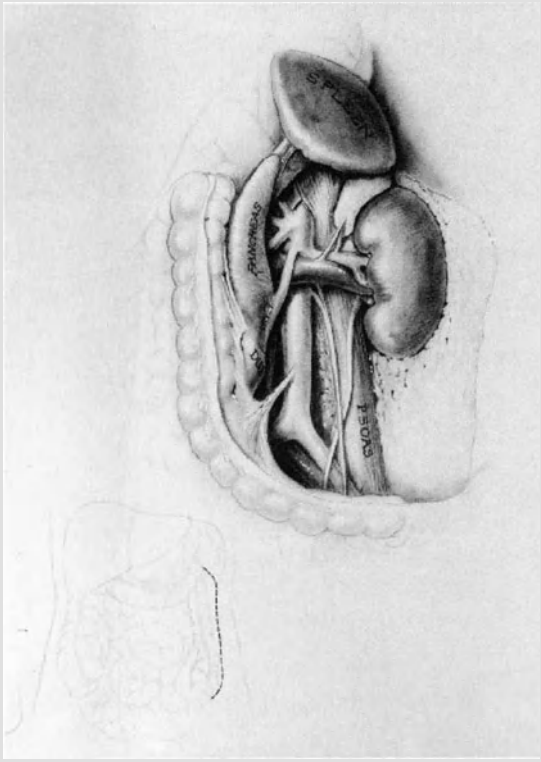

Fig. 4. Exposure of the entire aorta and its branches from the diaphragmatic hiatus to the aortic bifurcation is accomplished by mobilizing the abdominal viscera upward and to the right

If the hematoma is obviously arterial, the paraduodenal aorta should be exposed and dissection carried out to identify the origin of the appropriate renal artery. This is then cross-clamped to control the bleeding while the kidney is mobilized. If bleeding is massive, the best approach is simply to retract critical overlying viscera medially away from the retroperitoneum, rapidly mobilize the kidney, apply a hilar vascular clamp, and follow with an immediate nephrectomy.

Aorta and iliac artery injuries can be a major challenge, and the mortality rate for suprarenal aortic injuries is extremely high [4]. When the possibility of a suprarenal aortic injury is identified, the midline incision should be carried across the costal margin to the left and the chest entered to control the descending aorta. Following this the spleen, stomach, and viscera can be mobilized upward from left to right exposing the retroperitoneal aorta from diaphragm to aortic bifurcation (Fig. 4). If the injury appears to lie at or below the renal arteries, an aortic occluder can be used for proximal control while the aorta is exposed by mobilizing the viscera upward to the left. After proximal and distal control have been obtained, the occluder can be released and the aortic injury identified and repaired. Injuries in the region of the iliac arteries should initially have aortic control, followed by progressive distal dissection until the specific iliac artery can be isolated and controlled.

Venous injuries constitute a far greater problem than arterial injuries. Venous flow is equivalent to that of an artery, but the abundant collaterals and friability of the vessel wall make these injuries more difficult to manage than those to corresponding arteries. The sequence in controlling hemorrhage is as follows:

1. Proximal control of arterial injuries usually stops hemorrhage because with shock distal collateral flow is relatively modest, and back-bleeding is not a major problem. Venous injuries, conversely, require both proximal and distal control because distal pressure is raised, causing veins to bleed vigorously in both directions.
2. Although cross-clamping of the artery during shock results in little change in collateral flow, cross-clamping of veins results in a marked increase in pressure, and collateral bleeding is augmented dramatically in all branches entering the injured segment. Proximal and distal control therefore does not necessarily result in control of hemorrhage.
3. Arteries have integrity and hold sutures well. Veins often have the consistency of wet tissue paper and tear with the application of clamps or when sutured under tension.
4. Suture lines in large arteries rarely produce thrombotic problems, whereas suture lines in veins that expose raw surface produce a high risk of local thrombosis and embolism. Moreover, suture lines in veins tend to contract and obstruct flow with the passage of time, whereas this is unusual in arteries.
5. Prosthetic substitutes work relatively well in the arterial system and poorly or not at all in the venous system.

Management of suprarenal vena cava injuries is similar to that recommended for hepatic venous injury above. For injuries below the liver a Kocher maneuver and mobilization of the right colon permits exposure of the vena cava and the right iliac veins (Fig. 3). Venous injuries can usually be controlled by direct pressure while dissection is carried above and below the area of injury. Even so, the infrarenal vena cava has abundant lumbar collaterals, and those entering the injured segment require isolation and ligation or lateral control before adequate hemostasis can be obtained to permit repair. Iliac venous injuries are controlled by direct pressure while dissection is carried out proximally and distally. Internal iliac lesions are often extremely difficult to control, and packing the pelvis may be the best means of managing these difficult injuries. Packs can then be removed 24–48 h later.

In *pancreatic injuries* the most difficult aspect of management is determining the degree of injury. Subcapsular edema and hematoma may restore the profile of a badly damaged organ. Simple injuries can be ignored or treated with simple drainage, but major disruptions of gland integrity can result in devastating problems relative to pancreatic fistulae and infection. The most common site for blunt trauma injury to the pancreas is at its neck, the level where the mesentery vessels pass under the pancreas. Penetrating injuries at the neck or body of the pancreas or blunt injuries associated with loss of pancreatic integrity are best treated by distal resection. If the injury is an isolated problem, careful dissection may permit separation of the splenic artery and vein from the pancreas and

preservation of the spleen [5]. However, when there are associated injuries or where there has been major blood loss or major damage of the pancreas, the resection is expedited by sacrificing the spleen. Rapid mobilization of the spleen can be carried out in conjunction with the tail of the pancreas so that a vascular clamp can be applied across the pancreas immediately proximal to the area of injury. In a bloodless field distal pancreatic resection can then be carried out, the pancreatic stump oversewn, and the splenic artery and vein ligated.

The most difficult pancreatic injuries to manage are those to the head or those combined with duodenal lesions. Unless there is a massive, devascularizing injury, a Whipple procedure which carries a high mortality is rarely indicated. Crush injuries of the head of the pancreas combined with duodenal blowout are best treated with "duodenal diverticulization." The greater curvature of the stomach is opened in the distal third, the pylorus oversewn from within the stomach, and a gastroenterostomy is created. Following this, any injuries which are reparable in the duodenum are sutured, and liberal drainage of the head of the pancreas is established with dorsal and ventral drains. Simple pancreatic and duodenum fistulas usually heal in the absence of distal obstruction, and (provided they are adequately drained) serious problems with infection are not a problem. Simple penetrating lesions of the head of the pancreas are best treated by drainage.

When *small intestinal injuries* involve anything more than simple lacerations, they are best treated by resection and primary anastomosis of the injured area. Simple perforations can be repaired with a single layer of suture. If there is any question about the bowel's viability, particularly if there has been an associated mesenteric injury, a second-look procedure should be carried out 24 h later.

Colon injuries which are isolated and not associated with other major injuries can be treated with primary repair if patients are operated on within 3–4 h of injury, the bowel is not devitalized, and contamination is not extensive. If there is any doubt about the extent of the injury or the degree of contamination, the involved portion of colon should be resected and a proximal colostomy and Hartmann's pouch created. Penetrating injuries of the rectum or blunt injuries associated with open pelvic fractures and perineal lacerations are best treated with proximal diverting colostomy and mucous fistula. Following this, the anal sphincter should be dilated, followed by irrigation of the distal segment until clear.

Postoperative Management

When injuries are confined to the abdomen, most patients do well, and serious postoperative complications are relatively rare. If such a patient does not do well in the first few days following surgery, reoperation is indicated. Whenever there is question of viability of specific organs or hemostasis has not been complete, a planned reintervention 24 h later is optimal, even if the patient is doing well. The decision for reoperation should be made at the time of initial operation. Therefore in these cases the surgeon is well advised to leave intra-abdominal packs if needed to facilitate hemostasis or to perform a temporary abdominal wall closure so as to

require reoperation. This prevents the temptation not to reoperate if the patient is doing reasonably well in the first 24 h. If reoperation is delayed until complications are obvious, morbidity will be extremely high and mortality will be increased. Anticipation of problems is the best means of prevention.

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Treatment of Extremity Injuries in Polytraumatized Patients: Timing of Osteosynthesis and Other Important Factors

J. Biert and R. J. A. Goris

Introduction

The in-hospital management of polytrauma patients can be divided into four periods [1]. In the *acute period* problems of the ABC (airway, breathing, circulation) are addressed. When vital functions have been stabilized, the *primary period* starts, with time for further diagnostic procedures and for a second series of operations, addressing injuries that may become life-threatening in the near future, or which may lead to severe disability if not treated early. The *secondary period* begins after 48–72 h, when the patient has been optimally stabilized. During the secondary period, operative reconstruction may be performed of major closed joint injuries (e.g., acetabular fractures) and closed fractures of the forearm. The *tertiary period* follows after 6–7 days and is determined by two events, septic complications and rehabilitation of the patient. Optimal rehabilitation can start after only operative stabilization of all major fractures, or consolidation of such fractures if treated conservatively.

The timing of operative stabilization of extremity fractures in polytrauma patients has been a matter of extensive controversy. Classically, these operations were performed during the tertiary period. Nowadays there is evidence that in these patients major long bone and pelvic fractures should be fixated operatively in the primary period whenever possible. Another controversy concerns the most appropriate type of osteosynthesis for operative fixation of long bone and pelvic ring fractures in polytrauma patients. While in patients with isolated fractures intramedullary nailing has largely replaced the use of external fixators and shaft plating, there is concern that in polytrauma patients nailing may increase the risk of respiratory insufficiency.

Concerning the treatment of unstable pelvic ring fractures, adequate fixation is an important factor in stopping hemorrhage from these fractures. In this respect, the use of a pelvic clamp, arterial embolization, and external or internal fixation have recently been advocated. Also, attempts to salvage a severily injured limb in a polytraumatized patient requires a different approach than in patients in whom this is the only injury. Polytraumatized patients are especially at risk for developing a compartment syndrome, and in cases of severe extremity injury with extensive swelling preventive fasciotomy may be the safest way to avoid further harm.

Rehabilitation programs in patients with multiple injuries are usually initiated in a late phase. The catabolic metabolism, typical for the severe trauma patient,

easily leads to muscle waisting, while lack of exercise produces joint stiffness in all extremities, injured or not. Treatment of these patients should therefore include early exercise.

These topics are the subject of the present chapter.

Timing of Osteosynthesis

On theoretical grounds, performing operative stabilization of major fractures (femur, pelvis) on the day of injury is attractive:

- Surgical procedures are much easier to perform.
- The generally young and active trauma patient is still in a good nutritional and immunological condition. This condition deteriorates during the following days or weeks, which may increase the risks of surgery.
- The presence of one or more "floating" major bone fractures makes nursing extremely difficult and uncomfortable to both patient and nurse. Stabilizing these fractures allows for optimal nursing, as the patient can be moved and turned without undue problems. This prevents decubitus wounds.
- The position of the "upright chest" can be attained at an early stage, which improves respiration and expectoration.
- Since fewer analgesics are required, the patient is more alert. Thus active mobilization can start at an earlier stage, a prerequisite for the prevention of thromboembolic complications and for attaining optimal functional outcome of the fractured extremities.
- Early osteosynthesis helps in preventing fat embolisms, acute respiratory distress syndrom (ARDS), and sepsis and decreases mortality (see following two sections).

Classically, the arguments in favor of late osteosynthesis have been that these operations can be planned more carefully, and that more experienced surgeons perform the operations. However, in a modern trauma center optimal care should be possible around the clock. The major problem with delayed osteosynthesis is that these operations often have to be further delayed because of the poor condition of the patient, focal infection, or sepsis. The arguments in favor of early osteosynthesis are:

- Prevention of ARDS, sepsis
- Patient in optimal condition
- Optimal nursing possible
- Early mobilization
- Upright chest, expectoration
- Prophylaxis of thromboembolism
- Fewer analgesics required
- No skin colonization

Those against early osteosynthesis include:

- Not fully stabilized patient (?)
- Planning not possible (?)
- No experienced manpower present 24 h per day (?)

Early Osteosynthesis and Prevention of Fat Embolism

Fat embolism may be viewed as one of the mechanisms leading to ARDS, with specific clinical and morphological characterics, such as the appearance of petechiae, high fever, a sudden unexplained drop in hemoglobin concentration, and intravascular fat particles in the lung, brain, and other tissues. Microscopically, pulmonary fat emboli are present in all trauma victims, dying between 12 and 24 h after injury, a finding unrelated to the cause of death [2]. The incidence of morphological fat embolism in patients dying after 24 h declines progressively with time. Massive pulmonary fat embolization is present in patients with multiple fractures, but very few show any clinical symptoms of fat embolism [2]. From these data it is clear that a distinction should be made between the morphological findings and the clinical fat embolism syndrome.

The value of early osteosynthesis of major fractures in the prevention of fat embolism has been well documented [3]. However, recently it has been shown that intramedullary reaming may enhance pulmonary fat embolization. This is discussed under "Early Osteosynthesis in Patients with Severe Thoracic Injury").

Early Osteosynthesis and Prevention of ARDS and Sepsis

In severely traumatized patients 90% of late deaths are caused by ARDS and multiple organ dysfunction syndrome (MODS) [4, 5]. An important factor correlating with death is the presence of one or more major fractures not stabilized by external or internal fixation [4, 5]. Possibly because of this factor ARDS is the cause of death with the lowest average Injury Severity Score (ISS) [4, 5]. It has also been shown that high plasma levels of the granulocyte enzyme elastase are correlated directly with an increasing Hospital Trauma Index (HTI) for limb injury and with an increasing incidence of ARDS and MODS [6]. On the other hand, secondary surgery leads to a release of inflammatory mediators comparable to severe accidental trauma, possibly increasing the risk of pulmonary failure [7].

The risk of developing organ failure after secondary surgery for trauma can presently be estimated using respiratory and inflammatory parameters [7]. Predictive for organ failure are: (a) for operations carried out on postinjury days 2 and 3: a PaO_2/FO_2 ratio below 280; (b) for operations performed after day 3: a platelet count above 180000×10^6/ml, C-reactive protein above 11 mg/dl, and plasma elastase above 250 ng/ml.

It has been shown experimentally in dogs that a femur fracture treated with operative stabilization has no effect on arterial oxygen tension while without stabilization arterial oxygen tension falls by 10% [8]. Ventilated pigs that received a combination of a femur fracture and missile trauma developed no respiratory or circulatory failure provided the femur fracture was operatively stabilized at an early stage [9].

Numerous studies have been published concerning the prophylaxis of ARDS and MODS in polytrauma patients with major fractures [1, 3–5, 10–20]. All studies demonstrate that early osteosynthesis reduces morbidity and mortality,

due to a lower incidence of ARDS and sepsis, as well as ventilator and ICU days. Only one study has been performed in a prospective, randomized way [13]; it reported the same overall results as the retrospective cohort studies as to time on the ventilator, ICU days, and decreased late septic mortality rate. In addition, this study demonstrated that a delay in fracture stabilization of 48 h led to an average of six additional days in the ICU, with twice the hospital cost.

Early osteosynthesis of major fractures in polytrauma patients has thus been shown consistently to decrease the incidence of fat embolism, ARDS, and sepsis and to increase the chances of survival.

Early Osteosynthesis in Patients with Severe Brain Injury

Concerns have been expressed as to early osteosynthesis in the coma patient with multiple fractures, as monitoring brain function is impaired by prolonged anesthesia. On the other hand, in most neurosurgical units patients with an initial Glasgow Coma Scale (GCS) below 9 are routinely ventilated in the ICU and may receive high doses of barbiturates, creating a situation similar to general anesthesia. Also, rapid restoration and maintenance of adequate oxygen transport are the single most important factors in preventing secondary brain damage [21]. This can be achieved in either the operating room or the ICU. Prior to operative fracture stabilization computed tomography (CT) of the brain should be carried out in these patients, and intracranial pressure monitoring should be started at an early stage. Operative fracture stabilization further allows for placing the brain-injured patient in a semirecumbent position at an early stage, which contributes to decreasing intracranial pressure.

A positive effect of early fracture stabilization on preventing secondary brain damage, mortality, and ease of nursing has been shown in several clinical studies [10, 11, 16–20]. Outcome of brain injury, as measured by the Glasgow Outcome Scale, was also better in patients with early fracture fixation than in a group receiving conservative treatment, although this difference did not reach the level of statistical significance [10, 16]. In children with severe brain injury and a fractured femur, early osteosynthesis, as compared to conservative treatment, decreased average ICU stay from 34 to 14 days and hospital admission from 120 to 63 days [19].

In the brain-injured patient the presence of painful stimuli generated from insufficiently immobilized fractures contributes to restlessness and to an increased requirement of sedatives and analgesia. It has also been shown experimentally that stimulation of the C-fibers of the sciatic nerve reduces cerebral blood flow in animals with restricted cardiovascular activity [22].

It is concluded that there is no reason for concern about a negative effect on mortality of early osteosynthesis of major fractures in patients with severe brain injury. Actually this approach facilitates the care for these patients and may improve outcome of brain injury. Furthermore, if these patients survive, they should be offered the best possible chances of functional recovery of the extremity injuries, especially if a permanent neurological deficit results.

Early Osteosynthesis in Patients with Severe Thoracic Injury

Patients with long bone fractures, concurrent blunt chest trauma, and pulmonary contusion have a higher incidence of pulmonary morbidity and death than patients with the same ISS but without long bone fractures [23]. Concerns have been expressed as to the potential detrimental effects on pulmonary function of early osteosynthesis in these patients, especially with intramedullary reaming and nailing [12, 23–25]. Increased trauma to the patient at an inappropriate time, when inflammatory mediators are maximally activated, may indeed result in increased pulmonary inflammation. Also, experimental intramedullary nailing of the unbroken femur in sheep induces additional pulmonary damage, especially in the presence of pulmonary contusion [25]. In another similar study, no increased pulmonary capillary permeability was found with reaming only, except in the presence of hypovolemic shock [26]. These findings, however, may not be relevant to the clinical problem, as the real question is whether nailing a broken femur induces more pulmonary damage than leaving a broken femur without proper stabilization.

Few clinical studies have addressed this problem, and in the available studies the severity of pulmonary damage has been poorly defined, patients were not stratified according to the severity of pulmonary contusion, and osteosynthesis often was not performed within 24 h of injury. In one study it was concluded that early fixation did not protect against pulmonary dysfunction or death, while the study actually showed that early osteosynthesis was safe as it did not increase morbidity or mortality [23].

We performed a retrospective study of patients admitted with severe pulmonary contusion, stratified according to the HTI of thoracic injury and to the severity of pulmonary contusion [28]. Group A consisted of patients without long bone fractures or without early osteosynthesis of such fractures, and group B of patients with one or more major bone fractures treated by early osteosynthesis. The course of the PaO_2/FO_2 ratio showed no significant differences between the groups despite a significantly higher ISS in group B. There was also no difference in the incidence of ARDS, duration of artificial ventilation, or length of ICU stay [28].

Recently it has been shown that a temporary deterioration of lung function and a rise in pulmonary artery pressure, as seen in reaming and nailing of femur fractures, can be largely prevented by the use of unreamed femoral nails [24, 25]. It is nevertheless doubtful whether the worries about secondary pulmonary damage can be circumvented by using a two-stage procedure, with the primary application of an external fixator followed later by internal fixation.

It is concluded that early operative fixation of major fractures is a safe procedure in patients with pulmonary injury. However, there are still questions to be answered concerning the choice of osteosynthesis.

Early Osteosynthesis, Fracture Healing, and Risk of Osteitis

Early osteosynthesis does not prolong the period of fracture healing or increase the incidence of osteitis in the multiply traumatized patient [4, 13]. In any case, osteitis can presently be treated well if diagnosed early.

Prerequisites for Early Osteosynthesis

Performing early osteosynthesis in polytraumatized patients requires a number of prerequisites:

1. The patient should have stable vital functions, as shown by a PaO_2/FO_2 above 280, and a stable arterial blood pressure within normal limits.
2. All important traumatic lesions should have been identified and, where appropriate, treated. This includes i.e., clearance of the abdomen by diagnostic peritoneal lavage, ultrasound or CT, and prophylactic pleural drainage when rib fractures are present.
3. The patient should not be hypothermic. Core temperature should be above 35°C.
4. Hypothermia should be prevented by all possible means, including an elevated temperature in the operating room, the use of warming devices for the patient, and warming devices for infusion fluids.
5. Blood coagulation should be within normal limits. Platelet counts should be above 100000×10^6/ml.
6. Monitoring of vital functions should be performed as optimally as in the ICU.
7. If significant blood loss is expected, enough units of type-specific, cross-matched blood should be present, and a cell saver should be used.
8. Patients with a GCS below 9 should receive brain CT and have an intracranial pressure measurement device implanted before starting the operation.
9. Osteosynthesis is performed by a team, experienced in all forms of osteosynthesis techniques.
10. Intramedullary reaming should be avoided if possible.

What Fracture Should Be Operatively Fixated Early?

Every polytraumatized patient presents with a specific set of problems. There are general rules to be adapted to this individual situation. This is especially true for the sequence of osteosynthesis in the polytraumatized patient. General rules include:

1. Dislocated joints should be repositioned at the earliest convenience.
2. Fractures or fracture/dislocations of the spine with progressive neurological deterioration and depressed skull fractures are treated first.

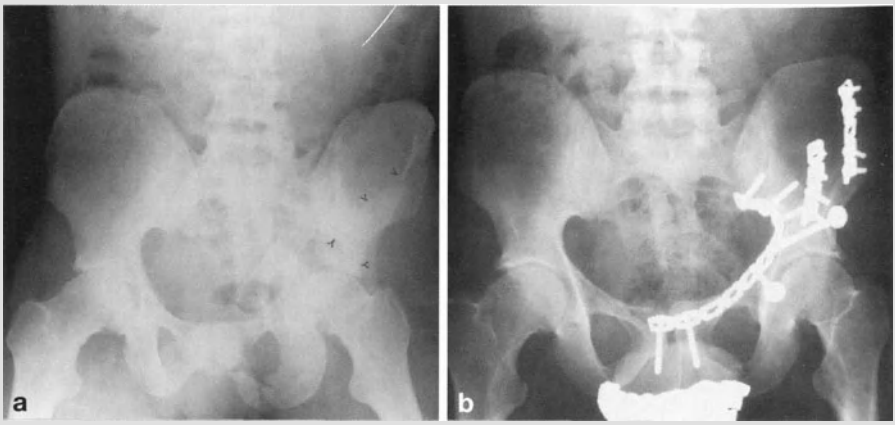

Fig. 1. a AP pelvis in a 42-year-old man after lateral impact car injury. Central dislocation of the femoral head, comminuted acetabular fracture with central dislocation and tilting of the acetabular roof (*arrows*) and fractures of both rami pubis. The patient had no other injuries, was hemodynamically stable, and further examination revealed no bowel or urinary tract injury. A transcondylar traction was temporarily applied to the femur. **b** Internal fixation was planned on the basis of pelvic CT and performed 48 h later

3. Fractures and joint injuries associated with vascular injuries should be treated within 6 h after injury, as dictated by the vascular injury.
4. Open fractures and joint injuries should be treated within 6 h after injury due to the increasing risk of infection after this period.
5. Next in the line of priorities are fractures of: femur and pelvis; tibia and humerus; fracture-dislocations of the spine without neurological deterioration; maxillofacial fractures.

Major joint reconstructions, i.e., of acetabular fractures and closed fractures of the forearm, do not require operative stabilization in the primary period and can safely be delayed to a more appropriate time (Fig. 1). This also is the case for bone grafting and closing large skin defects with skin grafts. If possible, simultaneous reconstruction should be performed by two teams to decrease operation time.

The Unstable Pelvic Ring Fracture in the Polytraumatized Patient

Enormous energy is required to induce a fracture of the pelvic ring. At the time of injury, major deformation of the involved tissues results, with extensive crushing and tearing of soft tissues and possible injury to all intra- and juxtapelvic structures. From the outside, internal injuries escape detection unless carefully sought.

The management and examination of a patient with an unstable pelvic ring injury should include:

1. Starting two large bore IV catheters and appropriate IV infusion.
2. Ordering at least 10 U cross-matched type-specific blood.
3. Pelvic inlet, outlet, ala and obturator X-rays, a pelvic CT scan (see Rommens, this volume).
4. Examination of the urethral orifice for blood; urethrography to identify a rupture of the urethra; urethral catheterization if the urethra is intact; installation of a suprapubic bladder catheter if the urethra is damaged; cystography to identify a bladder rupture.
5. Neurological examination of the perineal area, especially the anal region.
6. Rectal examination for possible tears and/or the presence of rectal blood; if in doubt, diatrizoate (Gastrografin) rectography.
7. Vaginal examination for possible vaginal tears.
8. Examination of arterial pulsations in the common femoral arteries.
9. Neurological examination of both legs to identify injury to the sciatic and femoral nerves.
10. Checking for diaphragmatic tears.
11. Searching for tearing or degloving injuries to the skin and subcutis.

A possible approach to the unstable ring fracture with persisting hemorrhage is [29, 30]:

1. Midline laparotomy, packing the pelvis, direct hemostasis as far as possible, with suture repair of tears in major vessels.
2. Extensive débridement and irrigation if soiling has occurred.
3. Evaluation and repair of injuries to the intra-pelvic organs.
4. Internal and/or external fixation of the pelvic ring fracture.
5. Diverting colostomy in cases with rectal or anal lacerations, and/or with extensive perineal wounds.
6. If oozing persists at the end of the operation, packing the pelvis before closing the fascia.
7. Performing a colonic washout through the colostomy.
8. Extensive excision and débridement of all external lacerations.
9. Leaving all skin wounds open.
10. Scheduled surgical revision after 24–48 h.

An alternative approach is to limit operative exploration to lesions of the bladder and rectum when indicated, to install an external fixator, and to obtain further hemostasis by selective arterial embolization. It should be kept in mind that – in the absence of disturbances in blood coagulation – instability of the pelvic ring fracture is the main cause of persistent bleeding. Stabilizing the pelvic ring fracture has significantly improved survival in these patients [14, 17, 29, 30].

Limb Salvage

The development of microsurgical techniques has increased the chances of salvaging amputated or semiamputated limbs. In the polytraumatized patient,

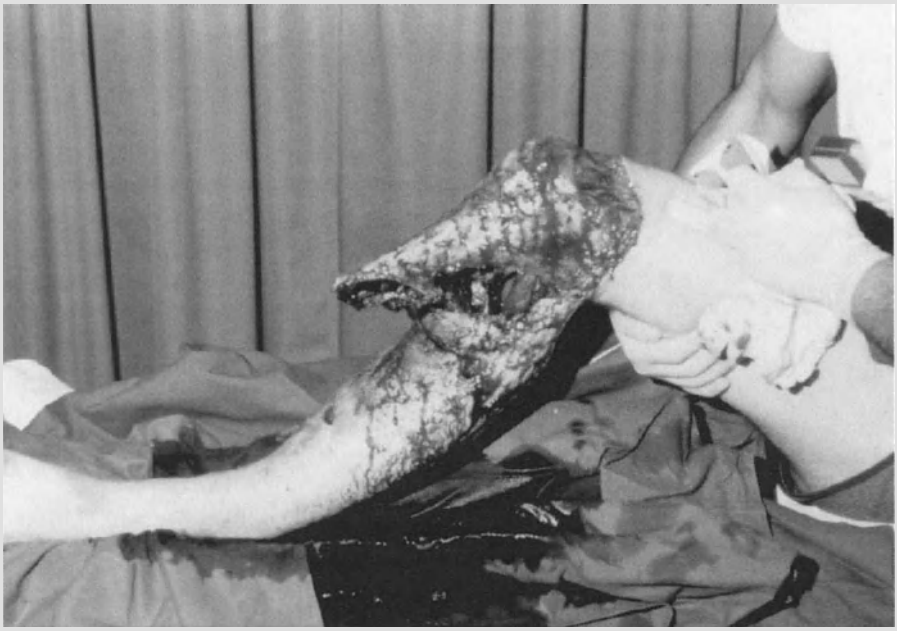

Fig. 2. A 20-year-old diabetic man who, sitting on a motor-bike, was caught with one foot behind the bumper of a car heading the other direction. The skin in front of the knee joint is avulsed from the thigh and pulled over and behind the fractured tibia. All tissues, including the popliteal artery and vein, were lacerated except the skin on the back-side of the knee and the tibial nerve, as seen macroscopically in the wound. No sensibility or motor activity of the foot and lower leg. As the above injury required a temporary lenghtening of the leg by approximately 20 cm, it is clear that the integrity of the sciatic nerve or the roots of the lumbar plexus should have been dysrupted. Therefore a primary amputation was performed

however, salvaging such extremities with severe soft-tissue injury likely increases the systemic inflammatory response, increases the risk of local septic complications, and requires a prolonged operation in the primary period. Therefore salvage or replantation is not recommended in patients with severe head injury or other injuries requiring intensive treatment (Figs. 2, 3).

A two-stage replantation procedure with initial osteosynthesis and revascularization, followed 3 days later by final débridement, nerve, and tendon suturing and soft-tissue coverage, may help in reducing the length of the primary operation [31].

Successful replantation of the lower extremity has been reported in children. In adults, however, replantation of a lower extremity is controversial, even if it is the only injury [32]. Indeed, injury to the posterior tibial nerve most likely results in the permanent absence of protective sensibility of the plantar side of the foot, which is the minimal requirement for obtaining a useful extremity. In mangled extremities it has been shown that sciatic or tibial nerve damage and failure of arterial repair are the only significant factors for the prediction of future amputation [33].

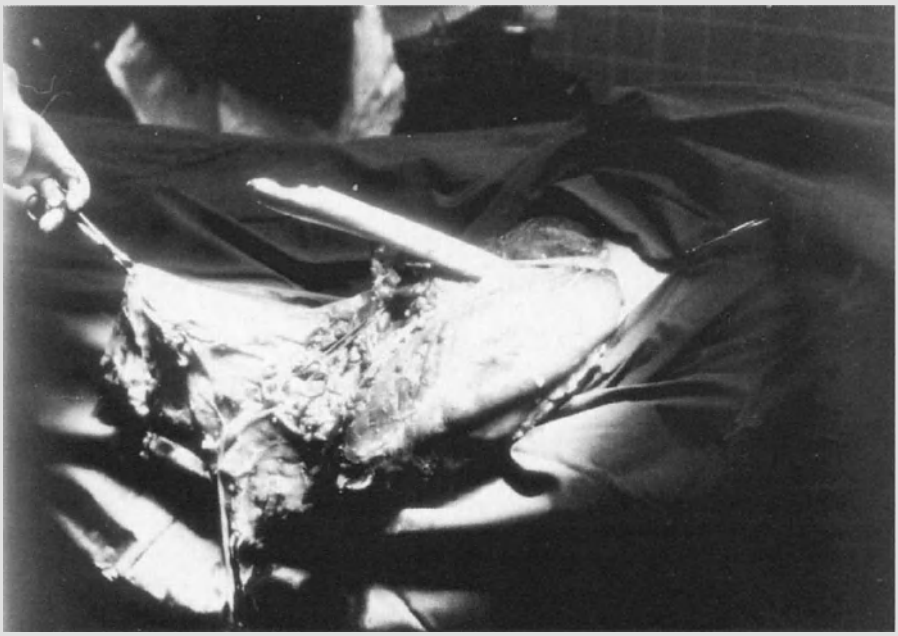

Fig. 3. An 18-year-old man who was run over by a fully loaded truck. Crush injury with extensive skin loss and degloving of the right thigh, from the groin up to the knee joint. Surprisingly, motor and sensory functions of the foot were normal, and the superficial femoral artery and vein were intact. There was also a subtrochanteric fracture of the left femur. An attempt at salvage was performed, with extensive débridement, fixation of the femur fractures, and secondary skin grafting. Secondary amputation was necessary 8 weeks later because of severe sepsis

In the polytraumatized patient therefore the balance between replantation and primary amputation of a lower extremity should favor the latter. An amputated lower extremity admittedly leads to significant disability [34], while good results of replantation have been reported in selected patients with an isolated injury [32]. However, a good amputation stump may actually provide for a better functional outcome, especially in the young and fit patient, than the remnants of a mangled extremity with an unhealed fracture and persisting local infection. When performing a primary amputation, parts of the amputated leg can be used for reconstruction of other defects, i.e., for bone grafting or partial thickness skin grafting.

In the upper extremity amputation leads to significant disability, while the prospects of a successful replantation are much better than for the lower extremity. Accordingly, in the polytraumatized patient replantation of the upper extremity should be considered more positively.

Preventive Decompression of Muscle Compartments

After severe soft-tissue injury excessive swelling may be expected in the affected muscle compartments both from local hemorrhage and from increased capillary permeability. As the fascial structures surrounding each compartment are rigid, an increase in compartmental volume inexorably leads to increased compartmental pressure, eventually resulting in a shut-off of the capillary perfusion and subsequent necrosis. In the polytraumatized patient this development is enhanced when arterial pressure is decreased or (local) venous pressure increased. Elevation of the affected extremity also decreases the "critical closing pressure," while traction on the affected extremity increases compartmental pressure, thereby increasing the chances of a compartment syndrome.

On the other hand, the clinical signs and symptoms of a developing compartment syndrome remain largely unnoticed in the polytraumatized patient, as the patient may be unable to communicate. Also, compartmental pressures is difficult to measure accurately and in all compartments at risk. The deep posterior compartment of the lower leg is difficult to reach with a probe, and this procedure is not without risk of vascular injury. Furthermore, it has been shown that the intracompartmental pressure has large variations from the knee to the ankle depending on the distance from the fracture [35]. This implies that one is never sure of having measured the highest pressure.

Especially in the polytraumatized patient we therefore advocate preventive dermatofasciotomy of all compartments that sustained a severe soft-tissue injury, i.e., after high-speed traffic accidents, high-velocity gunshot wounds, or other high energy transfers, and after prolonged ischemia of an extremity, i.e., after repair of a vascular injury.

Operative decompression should be performed within the first hours after injury, thus in the primary period. In the lower leg a four-compartment dermatofasciotomy is best performed through a parafibular incision, with special attention to decompression of the important deep posterior compartment. Failure of adequate decompression results in necrosis of most structures within the affected compartment, with severe late disability, and may result in generalized effects such as myoglobulinemia with renal failure.

In patients with severe soft-tissue injury it is advisable to bring the distal part of the extremity into a functional position by external splinting or external fixation. To prevent pes equinus in lower leg injuries an additional small external fixator is applied from the tibia to the first metatarsal bone, with the ankle joint at 90°. The application of a plaster of Paris splint is not recommended in this respect as it generally does not prevent pes equinus and impairs the care of possible extensive wounds. Furthermore, the external fixator facilitates nursing and allows for suspending the injured leg.

Special Considerations in Polytrauma Patients

The first objective in treating the polytraumatized patient with extremity injuries is the survival of the patient. As the extremity injuries are thus not the primary objective, these injuries do not always receive optimal treatment. To shorten the time spent in the operating room external fixation without anatomical reposition may be chosen, preferably to anatomical reposition and internal osteosyntheses. A definitive operation is performed at a later stage to achieve the best result.

In polytrauma patients the possibility of occult injury should be considered. The factors contributing to missing these injuries are: significant polytrauma with an obvious injury of the ipsilateral limb, unstable patient, altered sensorium, splints, inadequate radiographs, radio-opaque objects distracting attention, and minor signs [36]. Therefore, a high index of suspicion is required, with a new complete survey of the patient performed at the end of the operative procedures of the primary period. Intensive cooperation with the radiologist also improves the number of correct diagnoses of extremity injuries.

Aftercare

The patient with an isolated extremity injury benefits from active exercise, muscle training, and early weight bearing to gain functional recovery of the injured limb at an early stage. This prevents complications such as muscle wasting, joint stiffness, and tromboembolic disease.

Early and intensive treatment by a physiotherapist is needed in polytraumatized patients, including early active and/or passive movements of the injured and uninjured limbs. Continuous passive movement devices are useful, especially for joint injuries. Passive exercises, together with the application of removable spints or external fixators, should prevent joint contractures (i.e., pes equinus).

The patient should receive an intensive rehabilitation program, if necessary in a rehabilitation center. Early consultation of a rehabilitation specialist improves late functional results; this is only one of the many factors independent of the extent of physical impairment that affect the outcome. Early identification of and attention to a possible postinjury depression might improve the final outcome in polytraumatized patients [37].

Conclusion

Adequate stabilization of major fractures has become an integral part of caring for the multiple traumatized patient. Early osteosynthesis contibutes to a lower rate of pulmonary insufficiency, to a lower mortality, and to earlier mobilization of the patient. In severe pelvic fractures, operative stabilization also contributes to hemostasis. Other essential steps for an optimal functional outcome include adequate fasciotomies, spinting joints to prevent contractures, and early active

and/or passive exercises. Salvage or replantation of a severely injured extremity should be considered with great care, as this may substantially lengthen time in the operation theater and may contribute to later sepsis.

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Prevention of General Complications: Hypothermia, Coagulation Disorders, Infection and Acute Respiratory Distress Syndrome

P. M. Suter

Introduction

The most important measures in preventing posttraumatic complications are part of the appropriate initial resuscitation and of the correct priorities of surgical and medical management. Fast restoration of vital organ perfusion, oxygenation, and early surgical intervention is life saving in the early hours, decreases complications for the first weeks, and preserves vital organ function for the rest of life. The most important causes of morbidity and mortality between the second week and the first few months after trauma are (a) vital organ failures such as those of the respiratory system and the kidney and (b) secondary infections, pneumonia and blood stream infection. Frequently there is an interrelation between these two elements. Two other factors are recognized risk factors for both vital organ dysfunction and posttraumatic infection, namely hypothermia and coagulation disorders.

The present chapter reviews briefly the pathophysiology of these complications and the methods that have been proposed to prevent them, or at least to decrease their incidence after trauma. In addition, some therapeutic aspects are discussed.

Hypothermia

Hypothermia is not uncommon in the trauma patient under the following conditions: long time interval between the accident and arrival in a protected environment, contact with cold, well-conducting surfaces (water, ice, metal), cold environment, substantial blood loss, large volume of replacement administered, recent alcohol intake or other drug intoxication, and wind. Hypothermia markedly impairs survival in the trauma patient [1] due to the following pathophysiological effects on organ and cellular function:

- *Central nervous system*
  - Somnolence, coma
- *Heart and circulation*
  - J-junction elevation on the ECG
  - Arrhythmias

  - Ventricular fibrillation < 28 °C
  - Asystoly < 22 °C
  - Vasoconstriction (venous and arterial)
- *Respiration and acid-base regulation*
  - Decreased minute ventilation
  - Altered airway reflexes
  - Disturbed pH regulation
- *Kidney*
  - Decreased renal blood flow
  - Increased diuresis
- *Gastrointestinal tract*
  - Ileus
  - Pancreatitis
- *Metabolism*
  - Increased metabolic rate by shivering
  - Fall in oxygen consumption and CO_2 production at lower temperature
  - Hyperglycemia
- *Hematology, coagulation*
  - Granulocytopenia
  - Platelet dysfunction
  - Disseminated intravascular coagulation (DIC)

Pathophysiological Effects of Hypothermia on Vital Organ Systems

The brain tolerates hypothermia very well. Clinical signs include an impaired intellectual function below 34 °C followed by progressive somnolence, and unconsciousness below 28 °C. On rewarming these changes are rapidly and completely reversible.

The cardiovascular function in hypothermia is characterized by progressive sinus bradycardia and decreased cardiac output and coronary blood flow [2], in part as a consequence of a lower metabolic rate. A lower than normal heart rate, cardiac output and systemic arterial pressure are well tolerated in this situation. Typical electrocardiographic abnormalities include a J-junction elevation observed below 33 °C. Atrial fibrillation and AV blocks can also be seen. Ventricular fibrillation is common below 28 °C and asystoly occurs usually between 20° and 22 °C. A marked arterial and venous vasoconstriction causes centralization of the blood pool. The respiratory system adapts to the metabolic rate, first to the increased demands during shivering and then to the decreased needs for oxygen uptake and CO_2 elimination at lower temperatures. A markedly lowered minute ventilation and respiratory frequency are the rule. Apnea is usually seen below 24 °C. Aspiration of gastric content is facilitated by the blunted airway reflexes.

In humans, the regulation of pH probably occurs according to the alpha-stat control, i.e., pH changes with temperature, with an alkaline shift during hypothermia (0.015 pH units per degree Celsius) by relative hyperventilation. This regulation better preserves some enzyme and protein functions, myocardial performance and normal cerebral blood flow autoregulation [3]. During mechanical ventilation this physiological adaptation can be maintained by not correcting pH and $PaCO_2$ for the current body temperature, taking 7.40 and 40 mmHg, as measured by the blood gas analyzer at 37°C, as the desired end-point.

Kidney function is altered by the decreased renal blood flow and glomerular filtration rate. Despite this, diuresis may increase, partly as a result of central hypervolemia. Oliguria or anuria is more common in the trauma patient with massive hemorrhage, however.

Complications of hypothermia in the digestive tract are essentially ileus and pancreatitis.

The metabolic rate is markedly increased during shivering. With progressive hypothermia this reaction disappears, and oxygen consumption as well as CO_2 production are markedly decreased. As in other situations characterized by a marked stress, hyperglycemia is common.

Hematological abnormalities in hypothermia include granulocytopenia, platelet dysfunction, and quite frequently DIC. The latter two factors may be responsible for excessive bleeding in the trauma patient. Oxygen release from hemoglobin is impaired, and this can decrease tissue and cellular oxygenation.

Monitoring in Hypothermia

Electronic thermometers must be used when hypothermia is suspected. This measurement must be repeated in multiple sites, for example, sublingual, esophageal, rectal. With special probes, temperature can also be obtained from the pulmonary artery (by a Swan-Ganz catheter equipped with a thermistor), tympanic membrane, and bladder [4]. The best site is the tympanic membrane, which closely reflects the cerebral temperature. Continuous surveillance of temperature throughout rewarming is mandatory for a good clinical management, and should be done at two sites simultaneously.

An arterial line is mandatory in the hypothermic patient for both correct arterial pressure assessment and frequent blood gas analysis. Pulse oximetry on the ear lobes or digits is frequently ineffective due to severe vasoconstriction. The bridge of the nose is a more appropriate site and probes for this location exist.

Endotracheal intubation may be indicated for airway protection or ventilatory support. A naso- or orogastric tube is also frequently needed, as are continuous ECG monitoring and a Foley catheter.

Prevention of Hypothermia

Heat loss occurs through the mechanisms of radiation, evaporation, conduction, and convection. The trauma patient must be brought as rapidly as possible into an

environment protecting against heat loss, and the surrounding area (ambulance, emergency room, operating theater) should have an appropriate ambient temperature. Wet clothes must be removed, and the patient should be covered by dry blankets. The scalp has a high blood flow allowing important heat loss; it must be covered completely in bald patients and small children. When large amounts of intravenous fluids are administered, these should be warmed to about 40°C. This is of particular importance when the body temperature is already lower than normal [5]. Various types of fluid warming systems exist. The use of a heated humidifier for inspired gases is recommended, with inspired temperatures between 36 and 40°C. These preventive measures are usually sufficient to avoid a marked drop in temperature and also to correct mild hypothermia, i.e., 32°–34°C. Shivering can be decreased by radiant skin warming or warm blankets and in intubated and ventilated patients by sedation and, if necessary, neuromuscular blockade.

Rewarming of the Severely Hypothermic Patient

Simultaneously with other measures and therapies for resuscitation and surgical management after trauma, body temperature should be brought close to the normal range. Surface rewarming is adequate in mild hypothermia, for instance, by using warm air circulation systems such as the BAIR Hugger. Warming of respiratory gases is efficient for the prevention of heat loss but not for rewarming. Warm gastric or bladder irrigation is readily available but insufficient in severe hypothermia. Isotonic solutions with a temperature between 36° and 40°C should be used.

The most efficient methods for rewarming are pleural and/or peritoneal lavage with warm fluids (36°–43°C) and extracorporeal circulation. These techniques have the advantage of warming the "core" faster than the "shell" of the organism. This is of importance because cardiac contractility is impaired in hypothermia and care should be taken that oxygen consumption does not increase faster than cardiac performance. Indications for extracorporeal circulation include body temperatures below 25°C or cardiac arrest with core temperature below 30°C. Peripheral vasodilation occurs during rewarming, which can cause a marked drop in arterial blood pressure. Rapid volume expansion (with warm fluids) may be necessary. Blood gases and pH must be monitored closely by the alpha-stat method (see above), i.e., not correcting for actual body temperature, for pH and $PaCO_2$, whereas correction of PaO_2 for temperature is advisable. Inotropic and vasoactive drugs such as dobutamine and dopamine are mostly ineffective and arrhythmogenic during hypothermia. Severe hyperkalemia is frequently a marker of death before hypothermia, or of crush injury, hemolysis, or chronic renal failure [6, 7]. The absence of signs of life despite rewarming above 30°C can be considered to indicate death in most situations.

Coagulation Disorders

A normal coagulation system is crucial for a physiological response to injury and adequate conditions for surgery. A number of pathological elements can disturb the clotting factors and the cascade required for a good hemostatic function after trauma.

Pathophysiology of Coagulation Abnormalities in the Trauma Patient

A number of mechanisms lead to impaired blood coagulation after trauma. The most frequent of these include: significant loss of essential coagulation factors by bleeding, intrinsic or extrinsic activation of the clotting cascade initiated by the release of tissue factor, contact activation after vascular damage. In later phases sepsis can contribute to coagulation abnormalities in these patients through an accelerated procoagulant turnover induced by activated neutrophils and their mediator products such as elastase, or platelet activating factor and factor XII. The loss of coagulation factors and platelets during or after injury, bleeding, and surgery can be significant. Simultaneously, a marked dilution of these elements takes place during massive volume replacement therapy, administered to maintain volemia, with crystalloids and artificial colloids. With the continuing activation of the coagulation cascade the changes in procoagulant and inhibitor turnover become more relevant for the importance and persistence of coagulation abnormalities.

Activation of clotting becomes independent of physiological need and is self-stimulating. This is due to an inability of the available inhibitor potential to restrict fibrin formation. As a consequence low-grade intravascular coagulation ensues and an increasing consumption of clotting factors, inhibitors, and platelets occurs. DIC, fibrinogen depletion, thrombocytopenia, and fibrinolysis develop, which aggravate posttraumatic and surgical bleeding [8]. DIC can also be induced or facilitated by hypothermia or prolonged circulatory shock. Which of the possible causative factors is actually responsible for insufficient coagulation may not be important for the therapeutic approach, but the essential preventive measures can be derived from the possible pathophysiological pathways:

- Loss of coagulation factors and platelets by bleeding
- Dilution of clotting factors by massive blood volume expansion with crystalloids or artificial colloid solutions
- Release of tissue factor and contact activation
- Intravascular coagulation due to inefficient inhibitors
- Hypothermia or prolonged shock causing DIC
- Superinfection and sepsis: neutrophils activated by endotoxin release, elastase, platelet activating factor, and factor XII

A rare cause of secondary clotting abnormalities is a transfusion reaction. Administration of noncompatible blood products is usually followed by hemoly-

sis and consumption coagulopathy. The diagnosis is generally clinical, but appropriate laboratory examinations such as the Coombs test must be carried out to confirm the diagnosis.

Prevention and Treatment of Coagulation Abnormalities

The normal regulation of the coagulation system is the result of a fine balance between procoagulant, i.e., clotting factors, and inhibitory, i.e., antithrombin III, protein C and S activity. The balance is also influenced by vascular endothelial components and platelet function [9]. All factors facilitating or causing coagulation disorders listed above require rapid and adequate treatment. Of utmost importance is rapid stabilization of cardiovascular function and respiration, correction of hypothermia and acidosis.

In most instances of severe blood loss the administration of erythrocytes and plasma must be considered. On the other hand, maneuvers limiting loss of blood and coagulation factors may be worthwhile. For instance, Bickell et al. [10] have recently shown that delayed administration of intravenous volume replacement in penetrating torso injuries can decrease coagulation problems and mortality. In this investigation the infusion of crystalloid and colloid fluids was delayed until arrival in the operating room. This resulted in less intraoperative bleeding, fewer postoperative complications, and a better survival rate. This study illustrates that in the context of marked bleeding, the danger of low intravascular volume and borderline arterial pressure for vital organ perfusion must be weighed against restoration of circulating blood volume, leading to dilution of clotting factors and increased intravascular pressure, both causing increased bleeding.

A battery of coagulation tests should be obtained in the trauma patient with clinically manifest coagulation disorders. Global tests such as prothrombin time, activated partial thromboplastin time, and thrombin time are useful for obtaining a general idea of the clotting status or to confirm the clinical impression. In many situations, however, a more precise analysis and a dynamic assessment at several time points are indicated.

Replacement of coagulation factors should be as specific as possible. In the trauma patient with massive blood loss the early hours of surgical and medical management usually require the administration of fresh-frozen plasma. Prothrombin time should be kept between 30% and 50%. During and after massive transfusion, plasma calcium should be monitored and substituted if necessary. A platelet count below $20000/mm^3$ in the presence of bleeding is considered an indication for substitution. Specific coagulation factor preparations, for instance, prothrombin complex concentrate, are available today. Their administration provides rapid procoagulant substitution and they decrease the danger of transmission of certain viral diseases, but they are expensive.

Inhibitors of the coagulation cascade may be indicated when the inhibition potential is low and intravascular coagulation occurs. The main physiological inhibitor is antithrombin III. Its administration decreases thrombin generation in

sepsis and shock [11, 12]. After restoration of adequate antithrombin III levels, heparin should be added at low doses to maintain an appropriate procoagulant-inhibitor balance.

Infection and Antibiotic Policy

The trauma patient is at high risk for secondary and hospital-acquired infection. Several factors are responsible for an increased risk of contamination and a high susceptibility to develop serious infections.

Pathogenesis and Diagnosis of Infection

The great majority of patients with severe trauma develop clinical signs and symptoms of acute inflammation, i.e., fever, leukocytosis, tachycardia, tachypnea, and hyperglycemia. This reaction to aggression and stress is physiological and results from the activation of a number of elements of the immune system, including leukocytes, cytokines, coagulation, and complement systems. All too frequently this systemic inflammatory reaction syndrome [13] is ascribed to severe infection, leading to administration of antibiotics in the absence of any verified microbial involvement. This frequently inadequate, too early, and wide-spectrum antibiotic therapy exposes the patient to the toxic effects of these drugs, can make a correct microbial diagnosis impossible, and leads to the emergence of resistant bacteria [14]. Therefore a precise differential diagnosis, frequent cultures of blood and other body fluids, and restrictive antibiotic policy are essential parts of the management of the trauma patient with suspected infection.

Standard hospital hygiene has decreased cross-contamination and nosocomial infection enormously over the past 10 or 20 years. It is uncommon to see an important incidence of exogenous or cross-infection in a modern surgical ICU. Clearly, the large majority of ICU-acquired infections are due to endogenous infections, i.e., bacteria translocated from naturally colonized areas such as the nasopharynx or the gastrointestinal tract to normally sterile regions such as the lower respiratory tract or the peritoneum. This does not mean that hand-washing and other clearly efficient hygienic measures must not be controlled and reinforced regularly.

Important promoting factors in the pathogenesis of infection after trauma include:

- Disturbance of normal barriers between host and microbial environment [36]
- Altered immune defences: leukocyte activity, liver function
- Colonization and adherence of bacteria to epithelial cells and plastic surfaces in normally sterile localizations
- Invasive procedures (including surgery, endovascular, tracheal, bladder catheters, etc.)
- Cross-contamination from infected sites or other patients

Patients who sustain multiple injuries are susceptible to endogenous and exogenous infection because a number of defence mechanisms are severely altered. This concerns certain physiological barriers, general alterations of the host defence mechanisms, and the frequent need for invasive procedures for diagnostic, and therapeutic purposes. Thus the normal symbiosis between man and his microbial flora (in the gastrointestinal tract, the upper airway including nose, sinuses, and oropharynx) is disturbed by changes in defence mechanisms and foreign bodies breaking down these barriers.

Prevention of Secondary Infections

Awareness of the specific mechanisms of infection in the trauma patient forms the basis of efficient prevention and therapy. Similar to other serious complications in this type of patient, appropriate early management is crucial to decrease the importance of this problem.

Preventive measures to decrease secondary infection after trauma include:

- Early and complete surgical therapy of traumatic lesions (débridement, coverage, fixation of fractures)
- Mobilization
  - Never leave a patient in supine position for more than a few hours (except specific indication)
  - Semirecumbant position decreases gastropharyngeal-tracheal reflux
  - Early ambulation
- Appropriate hospital hygiene – thorough hand-washing
- Extubation as early as possible
- Removal of all nonvital invasive catheters and tubes
- Early enteral nutrition
- Selective gut decontamination remains controversial
- Frequent microbiological analyses in patients at high risk

Breakdown of physiological barrier function normally cannot be avoided, but good surveillance of the entry sites of all catheters and tubes, strictly sterile procedures for their introduction, and daily care of these devices decrease superinfection markedly. It cannot be emphasized enough that early removal of all nonvital foreign bodies and devices, including the endotracheal tube, decreases the incidence of infection.

Appropriate positioning and position changes have been advocated for many years to decrease pulmonary superinfection and skin problems. No patient should remain in the supine position and flat on his back for more than a few hours per day (except in specific indications) because a significantly greater reflux from the gastrointestinal tract into the trachea occurs in this position [15]. The risk of nosocomial pneumonia due to enterogenic organisms increases dramatically in the presence of an oro- or nasogastric tube, an endotracheal catheter, the use of histamine-2 blocking agents, and a high gastric pH.

Administration of an antibiotic solution in the oropharynx and stomach, introduced by Stoutenbeek more than 10 years ago [16], can decrease colonization and infection of the lower respiratory tract and lung. Although this selective digestive decontamination technique remains a subject of intense debate, it can clearly decrease infectious complications and mortality in certain patient categories [17].

Early enteral nutrition is another efficient measure to decrease the microbial load of the upper digestive tract and improve its barrier dysfunction after trauma. However, it is not entirely clear whether translocation of endotoxin and bacteria from the intestinal lumen to the blood stream is a frequent mechanism of sepsis in man.

The administration of ulcer prophylaxis does not seem justified today in all trauma or ICU patients [18]. However, this prophylaxis is still indicated in those requiring mechanical ventilation, or if coagulation disorders are present. Preference is usually given to sucralfate as the agent of choice because it is associated with a higher intragastric pH, less important gastric overgrowth of bacteria, and decreased incidence of nosomial pneumonia in this situation [19, 20].

Antibiotic Prophylaxis and Therapy

Prophylactic administration of antibiotics is indicated only in the following situations: (a) when potential contamination occurred as a consequence of trauma and surgery, (b) if foreign material was implanted, and its superinfection could constitute a major threat to survival or vital functions of the patient, or (c) if the patient is in a particularly vulnerable situation due to severe underlying diseases, and thus impaired normal host defenses.

Antibiotic prophylaxis must always be of short duration (24–48 h) and the doses chosen to achieve appropriate tissue levels, thereby limiting side effects.

Specific antibiotic therapy is indicated when a diagnosis or a serious suspicion exists for microbial involvement in the inflammatory or infectious process. Although it is clear that early adequate antibiotic treatment of a real infection improves outcome, it must be kept in mind that inappropriate antibiotic therapy bears the dangers of impairing a correct microbial diagnosis, increasing the emergence of resistant germs and the incidence of fungal infection, and contributing to the high costs of therapy.

Acute Respiratory Distress Syndrome

Some 20%–30% of patients who have sustained multiple trauma develop a severe pulmonary parenchymal failure associated with typical clinical, morphological, radiological, and lung function changes. This disease was termed adult respiratory distress syndrome (ARDS) by Ashbaugh and Petty in 1967 [21], but it has

recently been renamed acute respiratory distress syndrome because it also occurs in children [22].

Pathophysiology

The hallmarks of ARDS include an acute, overshooting inflammatory reaction, interstitial and alveolar edema, fibrin deposition in the airspaces, and subsequent fibrotic transformation or remodelling of the lung. As described elsewhere in this volume, ARDS is frequently but not always a part of the multiple organ dysfunction syndrome (MODS). Similar cellular interactions and mediators are involved in both syndromes [23–26]. It is not very clear today why this inflammatory reaction is limited to the lung parenchyma in some patients while similar changes are seen in a number of different organs and tissues in others. Playing a role are the local recruitment and activation of macrophages and leukocytes, mediator release, cell-to-cell signaling and the susceptibility of vascular endothelial cells to these signals, and permeability in different organs [27].

The most frequently observed predisposing conditions associated with ARDS in the trauma patient are: prolonged hypovolemic shock, nonstabilized fractures of long bones and fat embolism syndrome, severe soft tissue injury, inhalation of gastric content, and sepsis.

ARDS still has a high mortality despite extensive research in new areas of therapy, including anti-inflammatory agents, anti-cytokine antibodies, sophisticated methods of mechanical ventilatory support, and extrapulmonary gas exchange techniques. Most deaths, however, are not due to hypoxemia but to pulmonary superinfection, septicemia, or MODS [28]. The damaged lung parenchyma in ARDS may be more susceptible to superinfection, and the generalized inflammatory response could constitute an inappropriate defence state in the presence of an important microbial load.

Secondary damage to the lung parenchyma by large tidal volume ventilation and (possibly) by high inflation pressures is more and more recognized as an important additional pathophysiological mechanism of barotrauma and volotrauma [29] in ARDS. Positive pressure ventilation can also cause an increase in capillary and alveolar permeability [30, 31]. These side effects of mechanical ventilation are possibly involved in the nonrecovery of pulmonary function and the high mortality associated with prolonged forms of ARDS.

Prevention of ARDS

Rapid and correct treatment of the underlying conditions known to be associated with a higher incidence of ARDS is of utmost importance. However, massive transfusion and volume loading may worsen pulmonary function in the presence of an altered capillary permeability. Therefore the precise titration of intravascular volume therapy to adequate cardiac filling pressures, associated with low-dose vasopressor administration to maintain an acceptable systemic arterial pressure,

is frequently the target of this treatment. A negative or equilibrated fluid balance after initial resuscitation is advisable and decreases the severity and mortality of established ARDS [32].

Appropriate early management of the respiratory function after trauma includes oxygen therapy, adequate alveolar ventilation, CO_2 elimination in respect to the needs of the organism, and securing the airway if necessary. The use of prophylactic mechanical ventilation or prophylactic application of positive end-expiratory pressure has not been shown to decrease the incidence of ARDS after trauma nor to improve outcome [33]. The early diagnosis and appropriate therapy of infection and sepsis is an important and efficient measure for decreasing the incidence of ARDS and MODS in the trauma patient. Prophylactic antibiotic therapy should be used only in clear indications and for short time periods. The diagnosis of pulmonary superinfection is difficult to make in the injured patient in general and in ARDS in particular. An antibiotic window (e.g., no antibiotic treatment) for 24–48 hours, followed by a bronchoalveolar lavage procedure gives the best chances to exclude or to confirm the diagnosis of lower respiratory tract infection, and pneumonia. This approach allows in most situations an adequate, specific antibiotic therapy.

Efficient drug therapies to prevent or treat ARDS are not yet available. As mentioned above, all well-conducted major trials for prevention or early treatment of ARDS by anti-inflammatory agents, including corticosteroids, have shown no benefit and some have shown even harmful effects [34]. However, some evidence is emerging that in a much later stage, i.e., the fibroproliferative phase, for example, after 10–15 days evolution, corticosteroids could decrease the fibrotic transformation and be potentially useful [35].

Overall quality of care determines outcome in the trauma patient developing serious complications such as sepsis or ARDS. Meticulous attention to hospital hygiene, infection prevention and recognition, and general therapeutic principles including mobilization, anticoagulation, and nutrition, are as important as efficient initial resuscitation and surgical management.

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Prevention of Local Complications

P. M. Rommens

Introduction

Suboptimal care, especially in the severely injured patient, inevitably leads to acute or late complications, which significantly influence early morbidity, hospital stay, mortality, and expense and compromise the functional end result of the surviving patient. The most feared complications in trauma patients are compartment syndromes, thromboembolism, infection, and decubitus. This chapter demonstrates the importance of aggressive shock treatment and optimal soft tissue handling in the prevention of these complications.

Compartment Syndromes

Although much attention has been paid to compartment syndromes in the last decade, the pathophysiology and early symptoms of this horrible complication after trauma or surgery of the extremities is still insufficiently known. Once established, compartment syndromes nearly always lead to irreversible damage of the soft tissues with disabling sequelae (Fig. 1). Awareness of the possibility that a compartment syndrome may develop, recognition of the early symptoms and signs, and immediate accurate treatment can prevent many problems for patient and surgeon.

A compartment syndrome is a condition in which increased pressure in a muscle compartment reduces capillary blood flow below a critical level necessary for tissue viability [1]. The "critical closing pressure" of the capillaries is also affected by a decreased systemic (shock) and/or local arterial blood pressure (vascular injury, elevation of the extremity). Severe trauma patients are therefore especially at risk of developping compartment syndromes. Most frequently the four compartments of the lower leg are involved, but compartment syndromes also exist in the forearm, foot, hand, thigh, and buttocks [2, 3]. Many conditions may provoke the syndrome:

(a) factors decreasing local arterial blood pressure such as shock, vascular injury, or elevation of an extremity,
(b) factors decreasing compartment size such as constrictive dressings, casts and skeletal or especially skin traction, and

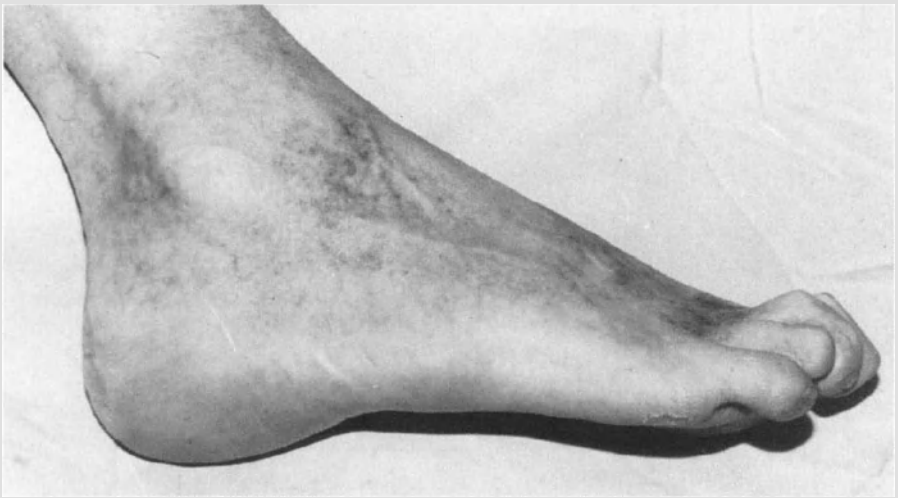

Fig. 1. Typical sequelae of a compartment syndrome of the lower leg: rigid pes equinus and claw toes

(c) factors leading to an increase in compartment contents such as accumulation of blood, edema, or a combination of the two. Accumulation of blood occurs after laceration of an intracompartimental blood vessel or after injury in patients receiving anticoagulant therapy. Edema is seen in postischemic swelling (reperfusion syndrome), after prolonged immobilization with limb compression, and after thermal injuries.

The pathophysiological cascade begins with an increased permeability of the capillary wall. This may be caused by direct soft tissue trauma or by the release of serotonin, histamine, free radicals, and other mediators due to ischemia. Posttraumatic or hypovolemic shock intensifies these disturbances by systemic mediator release. Therefore, adequate treatment of hypovolemia contributes to the prevention of compartment syndromes. Due to the increased capillary permeability osmotic pressure changes on both sides of the capillary wall, with a decrease in the arteriovenous osmotic pressure difference. As tissue pressure increases, the net arteriovenous pressure difference decreases further, together with tissue perfusion. This again leads to the release of vasoactive mediators, closing a vicious circle. Below a critical level of partial oxygen pressure, muscle necrosis and damage of the nerves occurs [2].

The first and leading symptom of an impending compartment syndrome is pain. This pain is not related to the primary injury but is localized in the whole compartment affected. Pain is intense and unrelenting [1]. Stretching of the muscles of the compartment enhances the pain, while no or only little pain relief is provided by analgesics. Children unable to express their pain are restless. The only patients with impending compartment syndrome who do not complain of pain have an associated or combined central (e.g., coma) or peripheral neurological

deficit (e.g., neuropraxis). The clinical manifestation of the increased intracompartimental pressure is a swollen, tense compartment. However, pain and palpation of the tension of a compartment are subjective findings and may be misleading in patients with severe trauma.

Paresthesia of the skin area corresponding to the nerve(s) passing through the attached compartment may be an early finding. Hypesthesia and later anesthesia occur. Paresis is a common finding but is also seen in severe muscle trauma, in ischemia, or after nerve involvement. The main differences between a limb with a compartment syndrome and ischemia are skin color and the presence of peripheral pulses in the former. Skin color remains pink in the patient with an impending compartment syndrome while the ischemic limb is pale. Finding peripheral pulses may be difficult because of swelling. In case of doubt Doppler sonography must be performed.

Almost all compartment syndromes can be diagnosed clinically. Only in patients with central or peripheral neurological disturbances may clinical examination be misleading. Compartment pressure can be objectivated with a slit catheter connected to a pressure transducer. The difficulty is not in measuring the intracompartmental pressure but in defining the pressure threshold for fasciotomy. Some authors recommend fasciotomy in patients with an intracompartmental pressure 20–45 mmHg less than the diastolic pressure. Others recommend fasciotomy if intracomparimental pressure exceeds 30 mmHg and is combined with other positive findings. Nevertheless, each indication for fasciotomy should be individualized as the pressure threshold is only a relative indication. Blood pressure, peripheral perfusion, evolution of complaints, patient reliability, and cooperation must also be taken into account [1]. We advocate early and extensive fasciotomy without pressure measurement in every patient in whom a compartment syndrome may be expected (Fig. 2). Hesitation with repeated pressure measurements and endless discussions delay therapy. Fasciotomy delayed is fasciotomy too late [4].

In the forearm a single skin incision beginning proximally to the antecubital fossa and reaching distally to the carpal tunnel is recommended. Proximally the lacertus fibrosus and distally the carpal tunnel are opened. The skin incision on the dorsal side is much shorter than that on the volar side. Skin incisions are never closed immediately after fasciotomy. The correct terminology for this procedure should therefore be "dermatofasciotomy."

In the lower leg we advise two skin incisions to decompress the four compartments. An anterolateral skin incision halfway between the tibial crest and the fibular shaft releases the anterolateral and peroneal muscle compartments. The superficial peroneal nerve, which lies in the lateral compartment just near the intermuscular septum, must be identified and preserved. The second skin incision is situated on the posterior medial side of the lower leg and decompresses the superficial and deep flexor compartments. Here the saphenous nerve and vein must be identified and preserved, and the skin incision left open. The alternative is a single long incision along the fibula, allowing for opening up all four compartments.

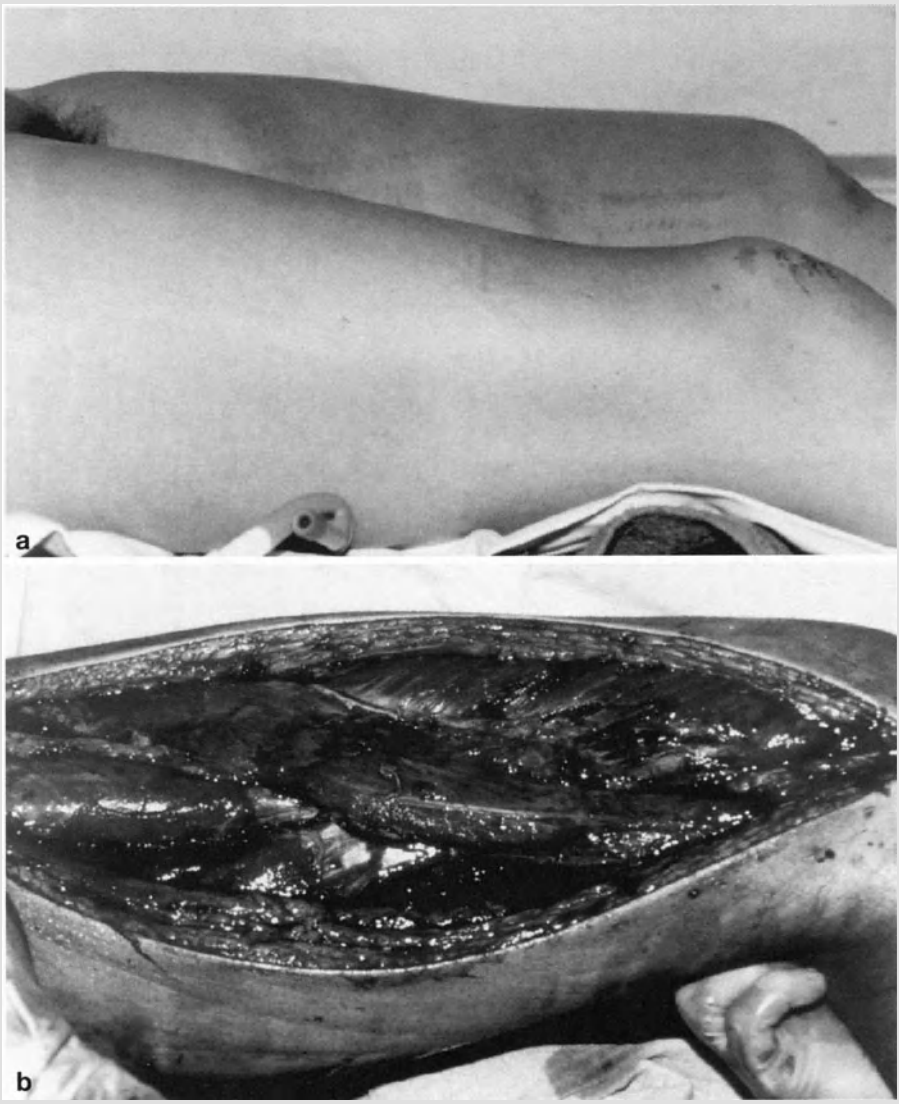

Fig. 2. a Extreme swelling of the left thigh in a 23-year-old girl with cerebral contusion and left femoral fracture. **b** Medial decompressive fasciotomy after intramedullary nailing of the femur

Skin incisions in the thigh, upper arm, and buttocks can be single. They must be large enough to enable safe decompression of all muscle compartments.It should be realized that after decompressing the compartiment additional swelling may occur due to reperfusion injury. Radical scavengers, such as mannitol, may contribute to decreasing this reperfusion injury. To preserve the elasticity of the skin elastic vessel loops can be attached to the skin margins. After daily wound

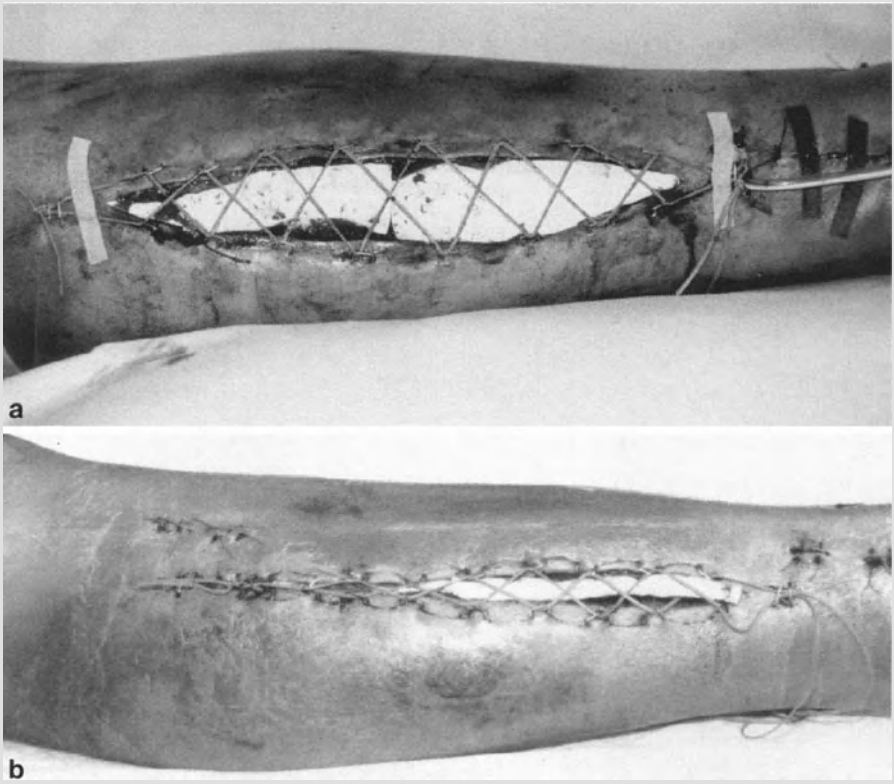

Fig. 3. a Vessel loops are attached to the skin margins immediately after decompressive dermato-fasciotomy of the lower leg. The skin defect is covered with a synthetic skin substitute. **b** After 5 days the area of the skin defect is gradually decreased due to the elastic action of the vessel loops. Wound closure without skin grafting can be performed

care these vessel loops are tightened, thus gradually decreasing the area of skin defect (Fig. 3).

Abdominal Compartment Syndrome

A special form of compartment syndrome may affect the abdominal cavity. Due to a retroperitoneal hematoma and to bowel paralysis, intra-abdominal volume may increase significantly, leading to increased intra-abdominal pressure. This results in elevation of the diaphragm with impaired respiration, impaired venous return to the heart, and decreased renal perfusion by obstruction of the inferior caval vein. In extreme cases an inferior caval vein syndrome may result, with hypotension, extensive edema of the lower extremities, and decreased urinary output, even renal failure. If this complication is anticipated after laparotomy, the

abdominal wall should be closed without tension, using prosthetic material. Subsequently the abdominal wall defect can progressively be reapproximated untill tension-free closure is possible.

Thromboembolism

Although the epidemiology of thromboembolism has been well studied in orthopedic patients and risk factors identified [5, 6], little is known about this problem in (poly)traumatized patients. Various studies estimate the risk of deep venous thrombosis (DVT) after trauma between 6% and 60%. Where real risks are not precisely known, no safe policy of prevention can be worked out. It follows that until now no method of prophylaxis of DVT applicable and effective for all trauma patients has been elaborated. In a recent randomized prospective study, Knudson et al. [5] identified the following risk factors for DVT after trauma:

- Age over 30 years
- Pelvic fracture
- Spine fracture with paralysis
- Paraplegia
- Coma (Glasgow Coma Scale < 8)
- Immobilization longer than 3 days
- Lower extremity fracture
- Direct venous injury
- Pregnancy
- Presence of femoral vein catheter
- Injury Severity Score > 16

Polytraumatized patients with an ISS greater than or equal to 16 can be regarded as high-risk patients for DVT because they generally have a combination of two or more risk factors. Risk factors are related to damage of the venous endothelium (e.g., direct venous trauma, fracture), to venous stasis (e.g., paralysis, prolonged immobilization) and to hypercoagulability (tissue injury) [7]. Especially patients with paraplegia are at high risk.

Also methods of surveillance and detection of a DVT pose problems. Many trauma patients who develop DVT have no clinical signs. Clinical examination is therefore not reliable [8]. Methods for detection of DVT include: light reflection rheography, duplex sonography, ^{125}I-labeled fibrinogen scans, and venography. Duplex venous sonographic imaging seems to be the most suitable detection method if performed by a qualified physician with a high-quality device [9]. It can be performed repeatedly at the bedside. A normal light reflection rheography examination is very accurate as a negative predictor, but abnormal rheographical results require further diagnostic studies since the specificity is not so high [10]. ^{125}I-labeled fibrinogen scanning is a very sensitive examination for DVT. However, it cannot be used routinely or as a preventive screening method since scanning is necessary 2 h and 1 day after administration, and since it may be impossible to perform in severely traumatized patients.

Venography is still considered the gold standard for diagnosing DVT. Because of the technical difficulties, invasiveness of the examination, necessity of contrast solution, and the need of transport to a radiology department it is useless as a preventive screening test.

The next question is which measure is most appropriate to prevent DVT. Compressive stockings, sequential pneumatic compression, and the daily administration of low-dose heparin are alternatives. Compressive or graduate elastic stockings are the first preventive measure and must be used in all trauma patients [11, 12]. If two or more risk factors are present, stockings should be combined with the use of sequential compression, which significantly increases blood flow velocity in the femoral vein and prevents venous stasis. In high-risk patients and patients with direct venous injury low-dose heparin or its fractionated derivates (5000–7500 IU once daily) should be additionally administered subcutaneously [13]. As the risk of DVT extends several weeks beyond the in-hospital period, and as many patients are less mobile at home than in the hospital, continuation of prophylaxis after hospitalization is recommended [14]. Contraindications for heparin and its derivates are a history of cerebrovascular accident, recent neurosurgery, uncontrolled hypertension, active hemorrhagic lesions of the gastrointestinal tract, and bleeding disorders due to deficiency of clotting factors. Some authors recommend the prophylactic insertion of vena caval filter in patients with the highest risk of DVT or in patients for whom other preventive measures are not available.

Infection

Infectious complications are seen exceptionally in the acute management of (poly)traumatized patients, but their later occurrence can be influenced or prevented in the first hours of treatment. The most important preventive factors are efficient shock treatment, early and aggressive débridement, meticulous soft tissue handling, leaving traumatic wounds open, mandatory second look, and delayed primary suture or grafting. The administration of antibiotics is not important in this respect.

Accurate soft tissue handling begins at the scene of the accident. Before transport, wounds are rinsed and covered with sterile dressings. Wound cleansing is performed with any watery solution available. Antiseptic solutions are not necessary in this phase of treatment. The main effect comes from mechanical rinsing, with removal of small contaminated particles from the wound. After abundant rinsing the wound is covered with sterile dressings that prevent secondary contamination. Thereafter the traumatized extremity is immobilized after continuous and careful longitudinal traction. Restoration of the anatomical axis decompresses the soft tissues around the fracture fragments and significantly diminishes pain. Immobilization with a pneumatic splint including the proximal and distal joint prevents redislocation of fracture fragments and secondary injury to the soft tissues. Early reduction and immobilization must also be carried out in open fractures. The risk of deep wound contamination in reducing open fractures

is smaller than the risk of secondary soft tissue damage in unreduced open fractures. Secondary soft tissue damage superimposed on the primary damage is often responsible for wound healing disturbances with hospital acquired infections as a consequence. The micro-organisms of these acquired infections are much more difficult to treat than those of the primary wound contamination [16].

It is clear that adequate shock treatment is also effective in the prevention of subsequent infectious complications. Aggressive volume substitution and a normal cardiac output with good systolic blood pressure contribute to optimal tissue oxygenation while prolonged shock or hypoxia may enhance necrosis of already traumatized soft tissues [17].

In hospital the dressings and splints are left in place until definitive care can take place in the operating room. The only effect of removing adequately applied dressings in the emergency room is an increased risk of infection.

Surgical débridement is the basis for preventing infection. Wound contamination is a surgical emergency; thus, débridement should be performed as soon as possible [3, 18, 19]. This consists of wound cleansing, disinfection of the surrounding area with antiseptic solutions, and removal of all foreign bodies and devitalized tissues. Débridement must be performed under sterile conditions.

Prevention of infection in trauma patients requires:
- Aggressive shock treatment
- Care at the scene of accident
  - Wound rinsing and wound coverage
  - Reduction of (open) fractures
  - Immobilization with splints
- Early wound débridement
  - Wound rinsing
  - Removal of foreign bodies and devitalized tissues
  - Disinfection
- Meticulous soft tissue handling
- Open wound treatment
- Preventive administration of antibiotics
- Booster immunization against tetanus

The surrounding skin is cleansed mechanically with soap or a detergent, shaved, and disinfected with a powerful desinfectant such as iodine 2% in alcohol 70% or betadine iodine. Care must be taken that the disinfectant does not enter the wound itself. The wound and neighboring skin are then draped with sterile dressings. Thereafter, hemostasis is obtained, not by blind clamping of bleeding areas, as this aggravates tissue necrosis and may damage important structures, but by isolated clamping or coagulation of every bleeding vessel.

Débridement follows with careful wound inspection. Therefore it may be necessary to enlarge the skin wound. The integrity of the bony, ligamentary, and soft tissue structures must be evaluated separately. All comtaminated and contused structures are removed with scalpel or scissors so that only vital and clearcut wound margins remain. Specialized structures such as nerves, vessels, and tendons are not removed but cleansed with saline. The viability of degloved

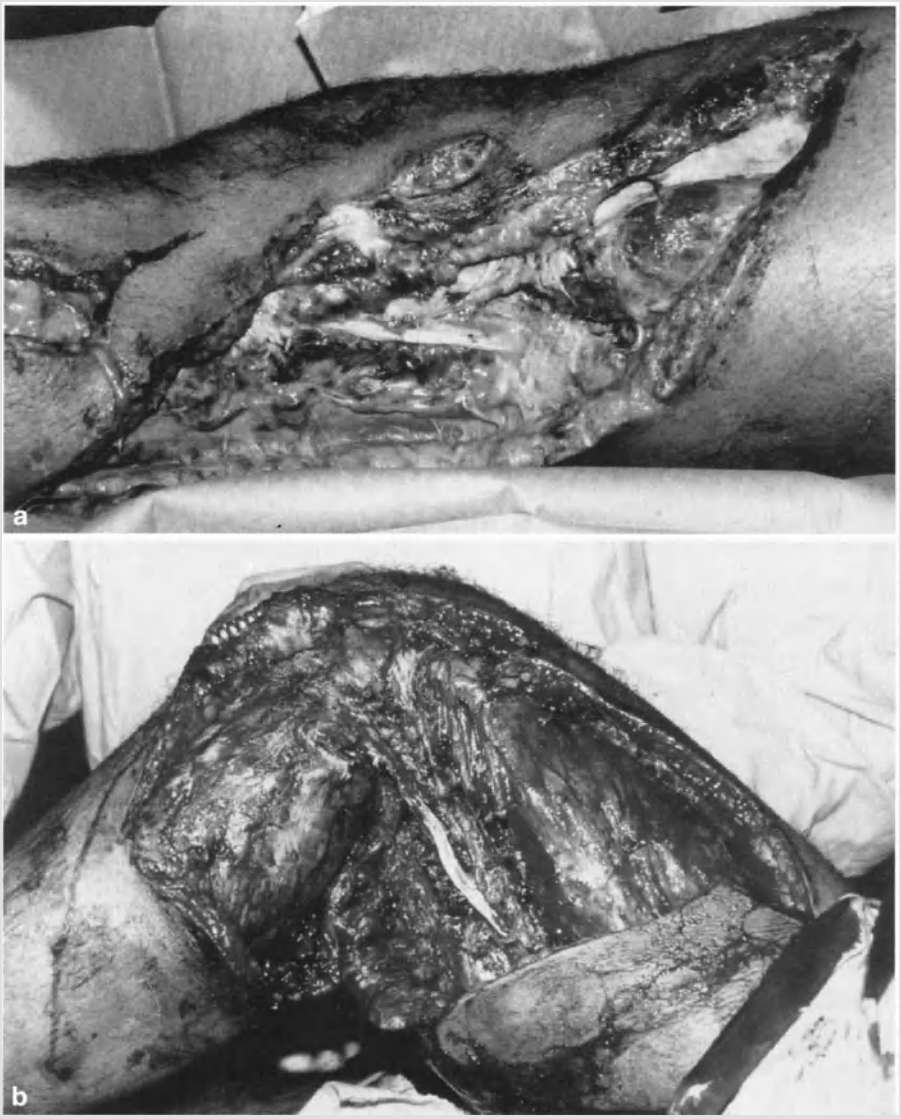

Fig. 4a, b

skin must be examined carefully. If the skin perfusion is critical or interrupted, avulsed skin should be removed and used as split-thickness skin grafts [3]. After wound débridement the wound is left open unless it is not older than 6–8 h, no important wound contamination is present, skin margins are well perfused and can be closed without any tension, and the wound is not situated in the neighborhood of a contaminated or infected area. Stab or bite wounds are never

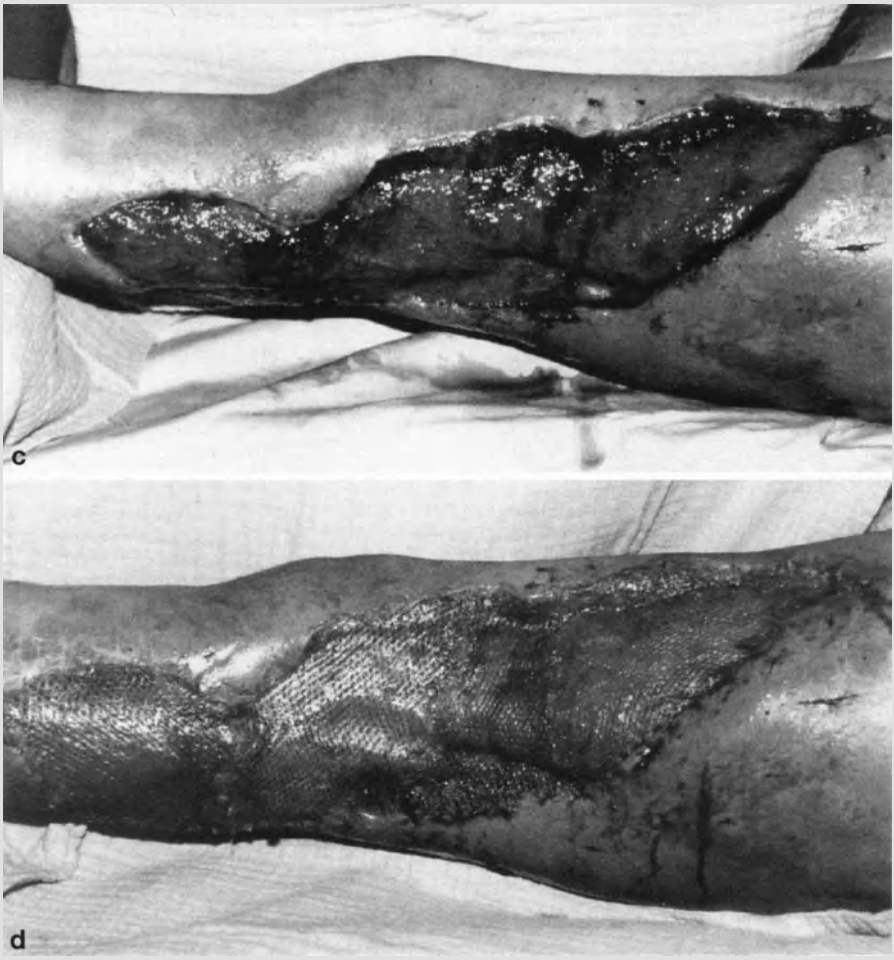

Fig. 4. **a** Severely contaminated wound of the knee region in a 30-year-old man 5 days after a motorcar accident in central Africa. Situation at admission in our hospital. **b** After débridement only vital soft tissue structures and clearcut wound margins remain. The wound is left open. **c** One week after débridement the wound is covered by fresh granulation tissue. No signs of infection. **d** The wound is coverd with split-thickness skin grafts 10 days after admission

closed. Primary wound closure has the advantages of quicker wound healing, less scar tissue formation, and better functional results. If there is any doubt of the viability of remaining soft tissues, the wound is also left open and wound revision planned 24–48 h after the first débridement. Secondary wound closure is carried out only with a clean wound base and well vascularized wound margins. The use of split-thickness skin grafts is often necessary (Fig. 4).

The benefit of prophylactic administration of antibiotics is well documented in trauma surgery. The purpose of this administration is to reduce the amount of

potentially pathogenic micro-organisms to beneath a critical level for the induction of infection. Administration must be continued as long as the risk period exists. In polytraumatized patients fracture treatment, stabilization of open fractures, management of severe soft tissue damage, acute implantation of a prosthesis, or metal implants must be regarded as contaminated operations.

The choice of the antibiotic agent is influenced by the micro-organisms, most often found in muskuloskeletal surgery: *Staphylococcus aureus*, *S. epidermidis*, and less frequently Enterobacteriaceae. The antibiotic agent which is chosen for prevention cannot be the same as the agents which are used for the treatment of postoperative infections with resistant micro-organisms.

In patients who only need surgical interventions on the musculoskeletal system we advise a first- or second-generation cephalosporin (cefazoline or cefamandol) at the induction of anesthesia, repeated twice within the first 24 h after surgery [20]. In patients also requiring exploratory laparotomy or "clean" interventions in the abdomen (splenectomy, splenoraphy, cholecystectomy) the same shedule can be used. When patients need colorectal surgical interventions, a combination of cephalosporin, aminoglycosides, and metronidazol should be administered on the day of surgery (three administrations of cephalosporin, two administrations of aminoglycosides and metronidazole). On admission the status of the patient as regards immunization against tetanus must be determined. If the patient or the patient's relatives cannot answer this question, a booster vaccination must be given.

Decubitus

Decubitus is defined as any degenerative change of the skin and subcutaneous tissue which is caused by pressure or sliding forces on these tissues. Skin defects, wound margins, and wound basis of decubitus lesions are generally contaminated by a mixture of hospital-acquired micro-organisms. These skin defects are situated in body areas which are difficult to cover with simple surgical measures, making decubitus wounds serious, sometimes life-threatening complications in trauma patients. The best way to deal with this problem, is prevention.

At risk are patients requiring prolonged immobilization: the polytraumatized, unconscious, those needing sedation or muscle relaxation and intubation with mechanical ventilation, and those with conservatively treated injuries (skeletal traction, plaster casts). Other risk factors are preexisting diseases of the arteries and veins (atherosclerosis), diminished peripheral circulation (hypovolemia, hypotension, dehydratation), and a poor nutritional status. Also, patients with hypoesthetic or anesthetic body regions (e.g., paraplegia, plexus lesion, paresis of one peripheral nerve) do not feel pain in areas with impending decubitus. Predilection areas for decubitus are regions with prominent subcutaneous skeletal structures subjected to high pressure or a continuous low pressure, such as the occipital region, greater trochanters, olecranon, calcaneus, head of the fibula, and tip of the scapula. The first signs of decubitus are local pain and redness of a skin area. In a second stage, epidermolysis is seen. The

third and fourth degree are superficial, respectively deep, skin defects with or without deep necrosis.

Primary preventive measures can be general or local. An aggressive treatment of hypovolemia and shock and early operative stabilization of extremity lesions contribute to the prevention of decubitus as they favor adequate tissue oxygenation and early mobilization respectively. The treatment of concomitant diseases such as diabetes mellitus, hypoalbuminemia, heart decompensation, or infections of the respiratory or urinary tract also optimalizes the general condition of the patient and helps to prevent the onset of decubitus [21]. Adequate early, preferentially oral feeding also contributes in preventing decubitus. Local measures are the task of the nursing staff but must regularly be controlled by the physicians. The patient's position must be changed at regular intervals. In high-risk patients, such as intubated and ventilated persons or paraplegics, a schedule for changing body position is best worked out. The position of the patient must be chosen in a way that predeliction areas do not receive high pressures. Air suspension beds may be of benefit for high-risk patiens [22]. Body regions at risk must be controlled and taken care for. Weakening of the skin by the uncontrolled loss of body fluids must be prevented by regularly changing moist dressings and bandages. Conscious patients should be stimulated for mobilization by regular social contacts and exercises with the physiotherapist. In patients with already existing decubitus measures are taken to prevent aggravation of these lesions or permanent disability as a consequence of decubitus. Also in secondary and tertiary prevention, general measures as mentioned above, are as important as local measures. Surgical débridement is always necessary and plastic surgery with muscle rotation flaps or free flaps is sometimes necessary [23, 24].

Secondary Nerve Damage

During their period of incapacitation severe trauma patients are at high risk of developing secondary nerve damage due to prolonged or undue local pressure on areas at risk such as the elbow (ulnar nerve) and knee (peroneal nerve). At all times during their extrication, transport, diagnostic procedures, operations and in ICU attention should be given to positioning the patient and protecting these areas in a safe way.

Conclusion

Each physician dealing with (poly)traumatized patients must be aware of possible complications, have the knowledge to make an early diagnosis, and possess the technical skills to treat them adequately. Optimal care prevents long-tern morbidity and late sequelae by preventing complications. Prolonged shock and suboptimal peripheral tissue oxygenation play a decisive role in the onset of compartment syndromes, infection and decubitus. Careful handling of soft tissues, beginning at the scene of accident, adequate débridement, and

open wound treatment can prevent compartment syndromes and infection. Recognition of the risk factors of thromboembolism and decubitus help us to take optimal preventive measures in each individual trauma patient. Teaching of (poly)trauma management must include teaching of the pathophysiology, symptomatology, diagnosis, prevention and therapy of possible complications.

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The Severe Trauma Patient in the ICU

R. van Dalen

Introduction

Before admission of a trauma patient to the intensive care unit (ICU) hemodynamic and respiratory parameters should have been stabilized, diagnostic procedures carried out, and emergency surgery performed as required [1, 2]. During ICU stay three main points should be addressed: (a) close monitoring of vital organ functions, (b) prevention of late complications, and (c) treatment of disturbances in vital organ functions. This chapter discusses these points briefly.

Close Monitoring of Vital Organ Functions

At the end of the primary period the trauma patient is again extensively examined for missed injuries and developing complications such as compartment syndromes [3, 4]. A full physical examination must be performed again, looking carefully for symptoms of severe trauma, i.e., leakage of cerebrospinal fluid from the nose or from the ears (fracture of the base of the skull), unilateral or bilateral paresis or paralysis of extremities (central nervous system trauma), or subcutaneous air in the neck or at the chest as can be seen in tracheobronchial disruptions and lung injury, especially in the presence of rib fractures.

Attention must be paid to the radiographic signs of aortic disruption on recurrent chest X-rays, as such signs may develop progressively during the following hours or days. If these signs are present or suspected, a contrast-enhanced computed tomography or arteriography must be carried out [4, 5]. Repeated chest X-rays are also useful to detect a ruptured diaphragm, as signs may develop only after some delay. ECG monitoring is required to detect supraventricular tachycardias, atrial fibrillation, ventricular extrasystoles, and repolarization disturbances, as may be seen with myocardial contusion [6]. Of course, monitoring of circulation and ventilation is of utmost importance [3].

Postoperative hypotension may be the result of undiagnosed or recurrent hemorrhage or, frequently, of hypovolemia during the phase when the patient is being rewarmed after a period of hypothermia and vasoconstriction. In addition, third space fluid loss is present in the first few days. Therefore, hemoglobin concentration and fluid balance must be measured frequently during the first 12–24 h and urinary output must be at least 1 ml $kg^{-1} h^{-1}$.

Adequate ventilation and circulation are needed to restore and maintain tissue oxygenation. The risk of complications in the early postoperative period is one of the reasons to leave the patient on the ventilator until he has been proven to be stabilized (see "Prevention of Late Complications"). Most of the patients can be ventilated with a tidal volume of 7–10 ml/kg body weight and a respiratory rate of 14–16 per minute. Medium-sized tidal volumes are preferred to prevent barotrauma by avoiding high inspiratory peak pressures. Depending on the degree of parenchymal lung disease, for example, pulmonary contusion, an FiO_2 is chosen between 0.40 and 0.60. Usually, 4–6 cmH_2O positive end-expiratory pressure (PEEP) is sufficient to improve functional residual capacity. This PEEP level does not significantly affect the circulatory parameters.

Coagulation disorders are frequently present in the polytrauma patient after massive hemorrhage. In most patients thrombocytopenia and consumptive coagulopathies can be observed [7]. Causal therapy is repair of inadequate surgical hemostasis, if present. Platelet transfusions should be administered while bleeding is still going on, and the platelet count is less than 50×10^9/l. The use of fresh frozen plasma (FFP) is indicated only in patients with massive hemorrhage [8]. Because of the potential risks of blood components, neither platelets nor FFP should be given for prophylactic reasons. Prophylactic platelet transfusions are indicated for the prevention of intracerebral hemorrhage only if the platelet count is less than 10×10^9/l.

Prevention of Late Complications

During the first day in the ICU adequate actions must be taken to prevent late complications. Risk factors for deep venous thrombosis include bed rest, immobilization, and trauma to the extremities. Therefore prevention should be started in the early postoperative period, unless bleeding is still substantial. Administration of low molecular heparin in a daily dose of 7500–10000 U subcutaneously is effective in preventing deep venous thrombosis and pulmonary embolism. The risk of bleeding is only slightly increased.

Prevention of acute respiratory distress syndrome (ARDS) by optimal respiratory support is an important task of the ICU in treating polytrauma patients. In this respect prophylactic mechanical ventilation is most helpful. Initially, the discussion on this matter was confused by the fact that only the additional effect of PEEP was studied instead of mechanical ventilation by itself. The addition of PEEP at a level of 8 cmH_2O to 44 mechanically ventilated patients at risk of ARDS, including 31 trauma patients, resulted in no significant difference with controls [9]. However, nonventilated polytraumatized patients with femoral fractures initially treated with traction all subsequently needed therapeutic mechanical ventilation [1]. In addition, the mean number of ventilator days was 21, compared to 8.7 in the patient group ventilated prophylactically [1]. Also, the number of positive blood cultures rose from 0.5 to 3.7 per patient in the primarily nonventilated group compared to the patients who were ventilated prophylactically [1].

Table 1. ARDS prevention scale

	Points
Flail chest	10
Intestinal perforation	6
$PaO_2 < 8.0$ kPa ($FiO_2 = 0.21$)	5
Fractured femur or pelvis, each	5
Cerebral contusion	4
Initial blood pressure $\leqslant$ 80 mmHg	4
Liver rupture	4
Splenic rupture	3
Blood transfusions $\geqslant$ 4 U	3
Fracture of humerus, tibia, vertebra or Le Fort III, each	3
Fracture of forearm, Le Fort II, each	2
Fracture of ankle, wrist, rib, mandibula or foot, each	1

Note: Patients with GCS < 8 are always intubated and ventilated.

In recently published combined clinical and laboratory studies different cytokine patterns were observed early after injury in patients developing ARDS and multiple organ dysfunction syndrome (MODS), compared to patients without ARDS/MODS. On the first and second day of admission serum concentrations of tumor necrosis factor-α (TNF-α), interleukin (IL) 1 and IL-6 in patients developing ARDS or MODS were higher than in the non-ARDS group [10].

In an experimental study in rabbits it was shown that even mild hypoxia dramatically potentiates the inflammatory response induced by complement activation, resulting within 4 h in an ARDS-like syndrome [11]. As complement activation has been well documented in severe trauma patients [12], it is reasonable to assume that prophylactic mechanical ventilation contributes to the prevention of ARDS by preventing hypoxia.

The question remains as to which trauma patients should be ventilated prophylactically and for how many hours or days. In our ICU an ARDS prevention scale was designed empirically in 1976 (J.S.F. Gimbrere). The scale is still in use in a slightly modified form (Table 1). If a trauma patient scores 10 points or more, mechanical ventilation is provided for 24 h and continued until the patient can sustain production of adequate blood gas levels on spontaneous respiration. In 1983 only one out of 314 patients scoring 9 points or less developed ARDS, compared to 8 patients out of 27 with 10 or more points who were ventilated prophylactically. This indicates the predictive value of the scale, but its value with respect to prevention of ARDS has not been prospectively studied.

In trauma patients ARDS is not an independent disease entity, but often the first organ failure in a series of biological events that can result in MODS [13]. The mortality rate of MODS is high and depends on the number of failing organs. As there is no specific treatment for MODS, attention should focus on possible methods of prevention.

For a better understanding of preventive measures, the current concepts in the pathophysiology of MODS are discussed briefly. Adequate oxygenation and perfusion of tissues play a key role in maintaining homeostatic systems. Hypoxia is the most potent stimulus for macrophage activation, and activates the arachidonic cascade as well. Also, during shock or hypoperfusion from whatever cause intestinal permeability may be increased as a result of impaired oxygen delivery to the gut mucosa [13]. The increased permeability may result in bacterial translocation, including the passage of endotoxin from the intestinal lumen into the portal circulation. This again leads to activation of neutrophils, macrophages and plasma protein cascades, including the complement and coagulation systems.

Prolonged activation of macrophages results in excessive production of cytokines. Elevated serum levels of TNF-α, IL-1, and IL-6 have been observed in septic patients [13–15] but also in non-infected burn patients [13] and trauma victims without sepsis [10]. Furthermore, higher concentrations of these cytokines are associated with an increased risk for subsequent ARDS and MODS [10]. The cytokine cascade can further increase intestinal permeability, thereby potentiating the translocation of bacteria and endotoxins.

Tissue injury, expressed as increased capillary permeability and interstitial edema, may result from interactions between the endothelial cells and activated leukocytes [13]. Autopsy data in trauma victims and in those who died within 24 h after trauma revealed evidence of intracellular and/or interstitial permeability edema and infiltration. These changes were found in organs primarily injured by trauma but also in organs not primarily injured [16].

In summary, in the polytrauma patient hypotension may result in hypoxia-mediated activation of macrophages and neutrophils in different organs. This can be amplified by any subsequent event, i.e., recurrent hypotensive episodes or infection [13].

From this concept it can be concluded that the key points in the prevention of MODS are restoring and maintaining adequate tissue perfusion and oxygenation as soon as possible after injury, and preventing all causes of (ongoing) complement activation. In trauma patients the best way to limit the inflammatory response is immediate repair of all injuries, including long bone fractures [1, 2, 17–19]. The policy of early stabilization of fractures results in a lower number of ICU and ventilator days, a reduced incidence of ARDS and MOF, and even a lower mortality rate [20].

Successful prevention of infection may also reduce the incidence of MODS and sepsis. In the eighties, it was suggested that bacterial infections in trauma patients could be prevented during their ICU stay by selective decontamination (SD) of the oropharyngeal region and the gut, combined with systemic antibiotic therapy in the first few days after admission [21, 22]. In many subsequent studies heterogeneous groups of ICU patients were included, trauma patients being only part of them. Recently, a double-blind randomized trial on the use of SD in 72 trauma patients during their ICU stay, found no significant benefit [23]. However, it must be kept in mind that in this study cefotaxime was administered to both the study and the placebo group [23].

Recently published meta-analyses and reviews of SD studies have not revealed any beneficial effect on mortality rate [24–26]. Some studies have described the emergence of resistant micro-organisms [26, 27]. Furthermore, the reduced respiratory tract infection rate, as observed in many SD studies, can be criticized because the diagnostic criteria used in most have been rather inaccurate [27]. For these reasons, SD cannot be advised for successful prevention of infection in trauma patients.

Effective preventive methods for infection include radical débridement of soft-tissue injuries, and replacement within 24 h of all intravascular lines that were inserted in the acute period, under suboptimal conditions.

Successful antibiotic prophylaxis has been described in patients with open fractures and in patients after penetrating abdominal trauma. Antibiotics should be used early in both groups. In the former group a first-generation cephalosporin is preferred, whereas in the latter the antibiotics chosen must be active against both aerobic and anaerobic micro-organisms. In both patient groups the antibiotics must be administered as early as possible, and for only a short period of time, i.e., 24 h or less (for review see [30, 31]).

As mentioned above, increased intestinal permeability may play a role in the pathogenesis of MODS. Therefore it is not surprising that efforts have been made to preserve the integrity of the intestinal mucosa and to maintain its barrier function. Not only adequate tissue perfusion and oxygenation are important but also nutrition. It has been suggested experimentally that the enteral route of alimentation is superior to the intravenous route [28]. This was confirmed clinically by a lower incidence of infections in trauma patients with enteral nutrition than in those with parenteral nutrition [29]. Furthermore, it is essential to start enteral feeding early in order to maintain structure and function of the intestinal mucosa. Oral nutrition can be administered through a nasogastric or nasojejunal tube or via a surgical jejunostomy. The risk of acalculous cholecystitis is also lower by enteral feeding than by intravenous nutrition. Early nutritional support (within 12–72 h after trauma) may also attenuate the posttraumatic hypermetabolic response [29].

Treatment of Disturbances of Vital Organ Functions

It was pointed out above that prevention is an important aspect of the first 24 h in the ICU. If necessary, however, therapeutic aspects must also be considered. First of all, hemodynamic and respiratory stability are crucial. If the patient is still bleeding, one must be aware of preexistent use of anticoagulant therapy. If so, the anticoagulant must be antagonized by the intravenous administration of 10 mg vitamin K and by a concentrate of the coagulation factors II, VII, IX, and X. FFP can be used in patients with massive hemorrhage. The initial dosage is 2–4 U after the patient's total blood volume has been replaced by erythrocytes and infusion fluids. Platelet transfusions are needed when the platelet count is less than $50 \times 10^9/l$ and bleeding is still present. Further administration of coagulation factors should be guided by the results of laboratory tests [7, 8]. Of course,

surgical reintervention has the highest priority if surgical hemostasis is inadequate.

As mentioned above, the risk of complications in the early postoperative period is one of the reasons to leave the patient on the ventilator initially. Varying degrees of pulmonary dysfunction are observed after major trauma [32]. This may result from the injury itself (lung contusion) or from aspiration, ARDS complicating shock, multiple transfusions, or atelectasis. Several modes of mechanical ventilation are available nowadays, not only intermittent positive pressure ventilation and synchronized intermittent mandatory ventilation but also pressure regulated volume controlled ventilation, pressure support, and in patients with low pulmonary compliance pressure-controlled ventilation. Unfortunately, it is not possible to prefer one mode or another because of the absence of comparative studies.

In general, one must be aware of the risks of barotrauma by preventing inspiratory peak pressures above 35–40 cm H_2O. Furthermore, it is advocated to prevent muscle atrophy by choosing a supportive ventilatory mode that allows the patient to use his own respiratory musculature. PEEP is needed to maintain PaO_2 at a level of at least 8.0 kPa with an FiO_2 0.60 or less. If a hemo- and/or pneumothorax are still present at the time of admission to the ICU, these must be treated by insertion of chest tubes. In some cases more than one tube is needed to prevent the occurrence of tension pneumothorax.

In 47% of trauma patients admitted to our tertiary-care ICU cerebral contusion was the main problem. Of the total number of ICU days for trauma victims 59% were spent with the neurotrauma group. The mean number of ICU days was 11.7 for the neurotrauma patients versus 7.2 days for the nonneurotrauma group. Hence, the presence of an adequate resuscitation protocol for neurotrauma patients is necessary.

Prevention of secondary insults to the already injured brain is of utmost importance [33]. Secondary insults often result from hypoxemia, hypotension, hypercapnia or severe hypocapnia, and anemia. During the first ICU day (and also thereafter) strict attention must be focused on the prevention of these insults. Blood pressure must be monitored continuously by means of an intra-arterial catheter and arterial oxygen saturation measured using pulse oximetry. The cerebral perfusion pressure (CPP) can be calculated from the difference between mean arterial and mean intracranial pressure (ICP).

Therapeutic measures are aimed at maintaining the cerebral perfusion pressure above 70 mmHg and the intracranial pressure below 20–25 mmHg [33, 34]. At a perfusion pressure above 70 mmHg, no further increase in jugular venous oxygen saturation has been observed. This suggests that 70 mmHg is optimal for patients with head injury [33]. The oxygen saturation level in the jugular bulb should be between 55% and 85%. Values below 55% are indicative of a reduction in cerebral blood flow, whereas values above 85% indicate cerebral hyperemia [33].

Intracranial hypertension can be caused by water overload, resulting in dilutional hyponatremia. Therefore the infusion of hypotonic fluids must be avoided. Fluid intake should be normal as there is no indication for dehydration.

The combination of hypovolemia and sedation can be dangerous because of an increased risk of hypotension [33, 34]. If raised ICP is not associated with rotation of the neck, insufficient sedation, or hypercapnia, it can be treated by mild hyperventilation.

The $PaCO_2$ should not be decreased below 3.5–4.0 kPa in order to prevent ischemia due to severe cerebral vasoconstriction [35]. If mild hyperventilation is not sufficient to achieve normalization of ICP or CPP, mannitol can be administered by bolus infusion of at least 0.5 g/kg. Subsequent doses must be adjusted (upwards or downwards) to the response of ICP and CPP [33, 34]. The frequency of mannitol administration is six to eight times per day, but it must be stopped when serum osmolality rises to 320 mOsmol/l, or signs of renal failure develop.

If these actions are not effective in normalizing ICP and CPP, the last step in routine daily practice may be the administration of barbiturates, such as pentobarbital. Barbiturates reduce the cerebral metabolic needs, thereby reducing the discrepancy between oxygen consumption and oxygen supply, but they have a strong hypotensive effect. A significant improvement in outcome has not been shown using barbiturates [36]. At the moment no other drugs are useful. Cortisteroids [37, 38] and the calcium-entry blocker nimodipine [39] have not proven effective.

Conclusion

During the first day after ICU admission trauma patients must be monitored carefully. A physical examination must be carried out again, looking for missed injuries, compartment syndromes, subcutaneous emphysema, and neurological lesions. Recurrent chest X-rays must be made in search of injury to the great vessels and of a ruptured diaphragm.

Close attention must be paid to restoring and maintaining adequate tissue perfusion, oxygenation, and nutritional status because these factors are important in preventing ARDS and MODS. Prophylactic mechanical ventilation is advised in more severe cases. Adequate fluid resuscitation must be performed to prevent hypotensive episodes. Nutrition must be started early, i.e., within 12–72 h, preferably via the enteral route. Infection prevention deserves attention, but SD of the gut is not effective in reducing mortality. In head-trauma patients actions must be taken to prevent secondary brain damage and to normalize ICP and CCP.

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