N.D. Reis E. Dolev (Eds.)

Manual of Disaster Medicine

Civilian and Military

With 82 Figures
and 25 Tables

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Preface

This book is not a learned treatise. Its purpose is to render practical instruction to all those physicians, surgeons, administrators, and paramedical personnel who have to act in war or disaster situations. As a manual it is in no way a substitute for existing detailed and specialized texts on the various aspects of trauma surgery and management: rather have we attempted to collate the most essential knowledge required to organize and afford medical aid whenever civilian or military disaster strikes. The man on the spot is hardly ever a specialist, hence the need for a simple general manual of instruction.

The organizational aspects of medical services in war are very similar to those required for coping with a disaster: indeed the military are often called to cope with civilian disasters. Our duty is to be prepared, so as not to lose life and limb for lack of foresight.

There are repetitions in the book for which no apologies are made, since they always concern invaluable knowledge.

The chapters are organized to afford an understanding of the organization of medical services, the cause of wounds and traumatic disease, and the pathophysiological processes resulting from the different kinds of trauma. The management of treating casualties is divided in each section into the three basic echelons: on site (at the actual place of wounding), at the medical (battalion) aid station and in the field or base hospital.

Much of the treatment assigned to the hospital is in the realm of surgical specialities: nevertheless, an understanding is important for general physicians, who often must cope without the aid of specialists in a disaster situation.

Readers' criticisms and suggestions are most welcome. Finally we wish to express our sincere thanks to Mrs. Orah Naor, our editorial assistant, whose patience and industry were essential to the completion of this book.

Haifa, Spring 1989

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Part 1

Organization of Medical Services

1.1 Organization of Military Health Support

E. Dolev

Introduction

Military medicine includes medical organization, prevention, and treatment. As a component of the military organization, the military health services maintain a high level of readiness, preparedness, and flexibility. Medicine in the field aims to care for battle casualties end civilian disasters by treatment and evacuation. Through adequate medical organization, surgical and medical treatment are given to casualties in the most efficient and professional way. Military medicine on the battlefield has a threefold mission: to save life, to alleviate suffering, and to return soldiers to duty.

Modern battlefields present new problems for military medicine. The most challenging and demanding is the situation of mass casualties, with many of the victims seriously wounded. Such a situation dictates the application of basic principles, namely, echeloning and medical evacuation. In the military scenario the patient is treated through an evacuation chain which means that he is being cared for by more than one surgeon, or by more than one surgical team: this system is equally applicable to civilian disaster situations.

The cardinal causative agents in military injury are fragments: high-velocity missiles and explosives. These agents cause a variety of injuries. In open areas most of the injuries are in the limbs, and about 10% are burns. In closed areas, mainly in urbanized terrain, the typical injuries are blast, crush, and multitrauma. These same injuries are characteristic of terrorist activities.

Comparison of data from various military conflicts is based on three basic definitions:

Killed in action (KIA):	The number of soldiers who died before being seen by a medical officer / The total number of live casualties.
Died of wounds (DOW):	The number of soldiers who died after being attended by a medical officer / The total number of live casualties.
The mortality rate:	The total number of soldiers who died (DOW + KIA) / The total number of casualties.

KIA in modern conflicts has been about 20%–25%. DOW mirrors the ability of military medicine to cope with war injuries: In recent military conflicts it has been about 2%.

Factors to Consider

Planning health support for units on the battlefield is based on the factors described in the following section.

Knowledge of the mission

The important question is: What is the plan for a certain operation?

The situation

The enemy

1. Strength and disposition, including morale, willingness to fight, endurance, and motivation.
2. Types of weapons — special medical problems resulting from various types of weapons, e.g., high velocity missiles, fragments, explosives, automatic weapons, chemical, biological, and nuclear weapons, "unconventional weapons" being a special problem.

Friendly force

1. Strength and disposition — types of units, strength, training, ability to fight by itself and in comparison with the enemy.
2. Logistic situation includes the ability to support the units and the level of various materials in the depots.
3. Weaponry — the hazards of friendly weapons.

Area of operations

1. Terrain — different types of terrain influence differently the tactical maneuvers, the ability to concentrate units, including medical units, or to locate casualties. There are characteristic injuries in different terrains.
2. Weather influences mainly the possibilities to evacuate casualties by helicopters and airplanes. It also affects the ability to reinforce and resupply combat units.
3. Civilian population might be affected by hostilities. On the other hand, it might be a source of disease hazards for the units involved.

Preventive medicine and epidemiology

1. Presence of diseases.
2. Status of immunization.

3. Clothing and equipment, including protective garments, fireproof suits, antiballistic flaks, etc.

Health services support analysis

Casualty estimates

1. Numbers — according to the enemy, weaponry involved, terrain, weather, and type of operation. Planning health services support based on an estimation of 10% casualties of any force per day covers practically all forms of operations, excluding unconventional warfare.
2. Types of casualties — depending on the type of operation, terrain, weaponry. While in armored battles the typical injuries are burns and soft tissue wounds; in fighting in closed areas the typical injuries are in the head, neck, and chest. Traumatic rhabdomyolysis (the crush syndrome), blast, and multiple injuries are also common in this type of warfare.
3. The possibility of mass casualty situations. This extremely demanding situation arises mainly in built-up areas when many people are injured simultaneously, due to explosion. Here, the medical organization and proficiency are crucial in order to initiate the process of triage which is the only way to handle the situation.

Requirements

1. At the front line — numbers and qualifications of personnel and medical units needed for certain types of operations according to the above specifications.
2. Hospitalization — numbers of surgical teams, their ability to perform, pre- and postoperative intensive care units attached to the surgical teams; the ability to hospitalize patients in various echelons at different locations, and how to locate these units according to the military scenario and the estimation of casualties.
3. Supplies — the limiting factor for adequate medical treatment may be blood supplies. Generally, the quantity of blood needed corresponds to half the number of estimated casualties.
4. Special attention is required for the establishment of alternative systems of communication. Without communications, control and regulation of the various groups of patients cannot be secured and guaranteed.

Medical units

1. Organic units are a part of the force itself.
2. Attached units — there are several kinds of medical units which are attached to the force by necessity (estimated casualties and types of injuries). Attached units may include surgical teams, resuscitation teams, etc.

3. Supporting units are medical units which are located in the area of operations or nearby, to which casualties may be evacuated according to plans. However, these units are not organic or attached; they receive their orders from a superior headquarters.
4. Required units are those which are needed according to the medical plan, based on the military scenario and its consequent estimates. They should be sent to the arena by superior headquarters upon request.

Preventive medicine

1. Immunization.
2. Prevention of infection, cold injuries (adequate clothing, etc.), and dehydration.

Evacuation

Battle casualties are evacuated from the front line for two reasons: (a) to give the injured soldier proper medical and surgical treatment, and (b) to allow the tactical, combat unit to continue to perform its mission on the battlefield. Caring for the wounded is one of the commander's responsibilities and interferes with the ability to continue the mission.

Evacuation is an essential component of military medicine and for a long time has been one of the main differences between military and civilian medicine. In civilian practice, medical evacuation is usually not organized as a process, and in many instances medical facilities are located close to the site of the event. Thus, the problem of evacuation and its regulation is usually not considered an important issue.

Evacuation is not just the logistic transportation of battle casualties from the front line to a medical facility or from a forward facility to a rear hospital: it is a medical process.

Medical evacuation begins with the selection of battle casualties for transportation. This selection takes into account: the priorities for evacuation which are based on the patient's medical condition and the availability of the means of evacuation/transportation; the tactical situation; and the conditions at the various medical and surgical facilities at the subsequent echelons. For example, casualties who are hemodynamically unstable should not be evacuated before being resuscitated and stabilized.

During the process of evacuation the patients are cared for by medical teams who monitor their hemodynamic and respiratory condition, anticipating complications and coping with them as they arise.

Medical planning should consider tactical situations which may preclude or slow down evacuation from a particular location at a particular time, due to combat circumstances.

Due to the experience of recent military conflicts, medical evacuation has become identified with aeromedical evacuation; yet due to tactical situations, weather conditions, and many other variables, this type of evacuation

cannot be guaranteed. Thus, any plan for medical evacuation from the battlefield must consider ground-based alternatives.
A very efficient evacuation system has its own limitations: though it contributes to the salvage of many lives, it also brings about the evacuation of hopeless cases to medical facilities, thus, paradoxically, increasing the number of those who eventually die of their wounds (DOW) at the hospital level.
It should be remembered that the most effective treatment for "battle fatigue" and other mental problems caused by performance under fire is given as soon and as close to the event and its site: Hence the hasty evacuation of psychiatric cases is contraindicated. However, the general policy is to rid the front line of casualties, usually bringing about their transfer to the rear. Thus, too efficient an evacuation system which rapidly evacuates psychiatric cases jeopardizes their appropriate treatment.

Return to duty (RTD)

This is one of the main differences between military medicine and civilian medicine. In civilian practice the mission of medicine is simply to care for casualties, while in the military framework medicine is committed also to the mission: it supports the effort to win the battle. Thus, one of the tasks of military medicine is to direct lightly wounded soldiers and those who have completed their convalescence period back to their units, since they are a major source of reinforcement of trained and experienced soldiers.

Mode of operation

The most efficient way to reduce morbidity and mortality on the battlefield is the prevention of injuries. When waging war, the use of appropriate antiballistic and fireproof garments, eye goggles, and other protective measures are to be enforced.
Immediate first aid and rapid resuscitation have been demonstrated to contribute to higher survival rates among battle casualties. In order to reach a high level of preparedness among the soldiers, first aid should be taught in every military course. Thus all soldiers will be able to dress themselves or their comrades properly and immediately upon wounding.
Resuscitation and stabilization achieved by trained medical orderlies and medical officers (physicians) is the next step in caring for battle casualties, prior to evacuation to a field/evacuation hospital: Here, only life-saving surgical procedures are performed. For the rest of the patients, professional medical treatment is given either to ensure further, safe evacuation, or to return them to duty.
Based on individual skills, organization and treatment are intertwined and coordinated, resulting in a system planned carefully during peacetime which is most efficient during wartime. This military system can then be equally applied to any civilian disaster situation which may arise.

1.2 Organization of Medical Services in the Field

Y. Besser and B. Lev

The goals of the medical service are to save lives and limbs, to alleviate suffering, and to return soldiers to active duty after the shortest period of incapacitation.
In planning the following factors are to be considered:

1. The number of casualties
2. Types of casualties
3. The medical facilities available
4. Geographical factors
5. Tactical situation

First aid, evacuation, hospitalization, supply, laboratory, blood bank, dentistry, and preventive medicine are the services required. These services should act rather than react to changing situations (the ability to shift medical resources as the tactical situation changes). The principles for the development of medical units in the field are:

1. Proximity. Medical units are deployed as close to the area of combat or disaster as possible; i.e., in the combat zone, in order to afford rapid medical care to any casualty shortly after being wounded.
2. Flexibility. In order to shorten the possible lag period between wounding and medical treatment, medical support units are shifted to meet changing requirements.
3. Mobility. The capacity for rapid movement is needed in order to afford support in real time and to perform efficiently within the combat zone. All medical units should be able to serve as ground, airborne, or seaborne units.
4. Continuity. Casualties should receive optimal treatment from the moment of injury throughout the chain of treatment until discharge from the medical system. This principle, also known as "echeloning", differs in military and civilian medical services. The field medical service is characterized by a multistage system involving several teams that evaluate and treat sequentially the same patients. The distance between echelons is dictated by the terrain and the battlefield condition. Transportation time-span between the echelons should be as short as possible.

5. Control. A senior medical officer is responsible for planning and coordinating the medical services.
6. Communications. A communication system is vital: Ideally a radiotelephone system should be the means of communication but at times runners and messengers may have to do the job.
7. Area support. Medical services are not bound to the organic combat forces but are deployed according to the principle of regional area support. Medical units should be located along the routes of movement and within the compartment of the terrain. The echelons are the most effective and efficient means of providing medical care in the field, allowing rapid evacuation of those requiring definitive treatment and early return to duty of minor casualties.

The treatment in the echelons should not be a matter of rigid prescription: Functions may be expanded or contracted at each level as needs dictate. Echelons may be bypassed for increased efficiency; on the other hand, echelons can be combined in order to address a specific problem.

The Echelons

Care at each echelon is determined by the following interacting factors:

1. Urgency of the casualty/patient's needs
2. Mobility of medical personnel or the facility
3. Capabilities: medical personnel, facilities, equipment, and supplies
4. Workload: arrival time at the echelon relative to its treatment capacity
5. The ability of an echelon to perform in relation to an expected workload

Transportation links are considered in determining the placement of facilities, which once arranged will determine routes of evacuation and resupply.

First Echelon — On Site Self-Aid — First Aid

First aid is provided by injured soldiers themselves (using personal battle dressing) or by a comrade who has been trained in first aid (buddy aid). This includes control of hemorrhage, improving ventilation, immobilization of fractures, and protection of wounds.
In this echelon, first aid is also provided by a medically trained aidman. Aidman care includes: CPR procedures (cardiopulmonary resuscitation), prevention and treatment of hypovolemic shock, fracture splinting and wound dressing, pain relief, and evacuation.
The **battalion aid station** (BAS) or its equivalent, the **medical aid station** (MAS), is the first medical facility staffed with at least one physician and a

team of trained medics headed by a senior medical aidman and equipped to provide medical care within the first echelon. The aid station is established as forward in the battalion area as the tactical situation permits. Casualties may arrive at the station immediately after being injured or several hours later, depending on the battlefield situation. As the battalion moves the BAS may be required to move swiftly. Its proximity to the combat zone dictates its mobility requirements: Therefore it will be mobilized on a half-track armoured personnel carrier (APC) or alternatively on four-wheel-drive vehicles.

Medical treatment includes physical examination and resuscitative procedures as indicated.

At the aid station the following procedures may be performed:

1. Triage. Patients/casualties with minor wounds or illness are treated and are sent back to duty as soon as possible.
2. Insertion of intravenous catheters, performance of venous cutdowns, tracheotomy, central venous pressure measurement, and intercostal drainage.
3. Administration of crystalloid solutions and plasma volume expanders for treatment of hypovolemia.
4. Splinting and bandaging.
5. Administration of antibiotics.
6. Recording — brief but complete and accurate, including:
   - Personal ID
   - Description of the injury: location of external wounds, burns, fractures, and bleeding points.
   - Treatment: fluid administration, drugs, blood, and narcotics.
   - Evacuation priority.
   - Requesting, monitoring, and establishing priorities for aeromedical and ground evacuation.

The BAS is also responsible for resupplying aidmen with medical equipment.

Second Echelon — The Medical Company

The tasks of the medical are to provide medical support to those units which have no organic medical element or to reinforce the BAS as required. The company has a patient-holding capacity.

The front-line medical company consists of four identical medical units, two ambulance platoons, a logistic and a decontamination team. The main tasks of such a company include:

1. Receiving casualties from the BAS as well as from other medical teams within the support area.
2. Triage: setting up priorities for medical treatment and further evacuation.

3. First psychiatric aid for combat fatigue casualties.
4. Resuscitative treatment for all categories of casualties requiring evacuation.
5. Definitive treatment for patients who can be returned to duty within a short time.
6. Emergency dental treatment.
7. Evacuation of casualties to rear medical facilities.
8. Supply medical equipment to the company platoons and battalion aid stations within the support area.

The medical company is staffed by 4 physicians, 1 dental officer, 2 social workers, 28 medical aidmen, and 20 stretcher bearers and drivers.

Third Echelon — The Surgical Unit or Field Hospital

The surgical unit is located behind the second echelon within the division support area. Its main task is to provide resuscitative surgery and medical treatment necessary to prepare the critically injured for further evacuation. The unit is equipped with basic laboratory, pharmacy, and X-ray facilities as well as an operating theater and intensive care beds. In addition it provides dental, preventive medicine, and medical supply services.
The unit provides the following services:

1. Resuscitative surgery and medical treatment necessary to prepare critically injured patients for further evacuation to definitive treatment facilities.
2. Surgery is performed only to save life and limbs and to stabilize the patient for further evacuation.

The unit is usually located 30–40 km from the front line and is usually the last field facility before evacuating casualties to definitive treatment facility (such as a rear hospital): sometimes the distance is too great, and it is then necessary to establish another field medical facility (such as an evacuation hospital) along the route of evacuation.
Medical services in the field include the following additional components: dental services, health service logistics, and preventive medicine.

Dental Services. In the battlefield area dental services should provide treatment for urgent, incapacitating, dental problems only. The dental services are allocated to existing medical facilities. The service should be mobile; thus it requires its own means of transportation.

Preventive Medicine Services. Morbidity and mortality risks in the field are:
- Heat injuries
- Cold injuries
- Diarrheal diseases

- Diseases transmitted by insects
- Diseases caused by existing physical or mental disabilities
- Environmental and occupational hazards

Individual soldiers and commanders are responsible for taking preventive measures against environmental threats. Adherence to preventive routines, motivating subordinates, and enforcing these measures as well as ensuring the supply of adequate quantities of water and food will reduce the risk of illness from these causes. Estimation of the environmental risk factors will help in planning an effective health services support to the field forces.
The planning process includes estimation of the needs for preventive medicine supplies and equipment and plans for the use of special preventive medicine teams, field sanitation teams, and local public health personnel to support the local population.

Health Services Logistics. Medical logistic planning covers to following:

- Medical supplies (pharmaceuticals, fluids, blood, splints, and bandages)
- Equipment
- Laboratory reagents
- Biomedical equipment maintenance
- Routes and means of transportation for medical supplies
- Resupply (following a plan that estimates the need for supplies for a given number and distribution of injuries)

Health services logistic plans include in-theater medical maintenance and medical supply. Planning for supplies and maintenance should be a continuous process according to changing circumstances and needs.
The maintenance program includes:

- Prevention of equipment failure.
- Early detection and correction of system failure.
- Minimizing requirement for new equipment.

Biomedical equipment maintenance sections should be established in the field and categories of maintenance at each echelon/level should be stated and authorized.

Evacuation

Evacuation of battle casualties is a medical process and not mere transportation from one echelon to another. The goals of medical evacuation are to minimize morbidity and incapacitation, and to improve the quality of care by organization and regulation.
Evacuation policy should determine the maximal period of time that a casualty may be held within the theater for treatment. The policy enables the planner to compute the needed number to beds, medical units, and distribution and location of medical facilities.

The factors that determine the evacuation policy are:

1. Nature of the tactical operation: duration, magnitude, type of disasters or combat, weapons that will be employed (conventional, chemical, nuclear).
2. Number/type of casualties/patients.
3. Means of evacuation — ground or airborne.
4. Availability of reinforcements.
5. Duration of evacuation to a definitive medical treatment facility.

Medical escort is essential in any evacuation (ground or air) in order to maintain and continue the treatment of the casulty. Evacuation should follow the stabilization of the casualty: evacuation before the patient has been stabilized will be considered only when the chance of stabilization in the field is smaller than in the rear hospital (the evacuation time is taken into consideration). Evacuation priorities should be set and used as guidelines.

Evacuation is performed by ground vehicles or aircraft. Ground evacuation ambulances are designed and equipped to provided transportation and emergency medical treatment to patients enroute to a medical facility. Field ambulances are truck ambulances or APC. Each ambulance can accommodate four litters or six ambulatory patients.

Evacuation by air is preferred under most circumstances and is performed by helicopters or fixed-wing transportation aircraft.

1.3 Organization of Medical Services in Disaster Areas

Y. Adler

Disasters may be defined as a disruption of the human ecology, which the affected community cannot overcome with its own resources. Medically, it creates a temporary situation in which the demands for health services exceed the available supply of manpower, knowledge and resources. Some disasters cause severe damage to the infrastructure in the affected area, thus disrupting transportation, communication, electricity, water supply and health services. Such occurs in wars, earthquakes, avalanches, floodings and severe storms. Other disasters may cause increased morbidity and mortality in the affected population, without damage to the infrastructure, e.g. industrial and nuclear disasters, famine and communicable diseases.

Disasters are often classified into two main aetiological groups: natural, e.g. drought, earthquakes, floodings, tidal waves and high winds, and man-made, e.g. wars and terrorist activities, chemical and nuclear accidents, fires, major transportation accidents, famine and epidemics. Table 1 summarises the effects of different types of disasters.

According to recent data tropical cyclones, floods and earthquakes have affected some 194 million people during the decade 1970–1979. The average number of recorded disasters seems to have increased by 50% in the last decade (Table 2). Six times as many people died from disasters in the 1970s than in the 1960s (Table 3).This increase in the number of disaster victims may be explained by the fact that people continue to change their environment, thus making themselves more vulnerable to the effects of disasters. Urbanisation has increased immensely in third world countries — causing more people to live in disaster-prone areas. Thus the leak of 40 tons of isocyanate from a chemical plant in Bhopal, India, in 1984 caused more than 2000 deaths, mainly among the large population living in primitive dwellings adjacent to the plant.

Extensive damage is usually caused by earthquakes to villages and poorer living areas in the suburbs of large towns, where houses are built with cheap building material and without observing anti-seismic building standards.

The severe famine and the spread of infectious diseases causing hundreds of thousands of casulties in the last decade in the Sahel belt in Africa was mainly caused by political and agricultural mismanagement — more than

Table 1. Effects of major disasters (from PAHO 1981 Emergency health management after natural disaster)

Effect	Earthquakes	High winds (without floodings)	Tidal waves/ flash floods	Drought	Industrial nuclear accidents
Deaths	Many	Few	Many	Many	Moderate
Severe injuries requiring extensive care	Overwhelming	Moderate	Few	None	Many
Increased risk of communicable diseases	Potential risk following all major disasters (probability rising with overcrowding and deterioration of sanitation)				
Food scarcity	Rare	Rare (may occur due to factors other than food shortage)	Common	Common	None
Major population movements	Rare	Rare (may occur in heavily damaged urban areas)	Common	Common	Common

Table 2. Average recorded disasters per year

Type of event	1960s	1970s
Drought	5.2	9.7
Flood	15.1	22.2
Civil strife/conflict	4.1	6.8
Tropical cyclone	12.1	14.5
Earthquake	6.9	8.3
Other disasters	10.8	19.5
Total	54.2	81.0

by recurrent years of drought. Some of these countries have been engaged for years in civil wars, spending huge sums of money on armaments instead of introducing modern agricultural development programmes, sorely needed for the rapidly expanding populations.

Table 3. Number of people killed in disasters per year

Type of event	1960s	1970s
Drought	1101	23110
Flood	2370	4680
Civil strife/conflict	300	28 840
Tropical cyclone	10750	34360
Earthquake	5250	38970
Other disasters	2890	12960
Total	22570	142920

Stages of Disasters and the Requirement of Health Services

Natural and many "man-made" disasters occur without any advance warning. The usual picture immediately after the impact is one of utter confusion and disruption of the local government and leadership. The phases of the disaster and the necessary measures to be taken are outlined in Table 4.

Table 4. Phases of a disaster (from PAHO 1982 Environmental health management after natural disaster)

	Predisaster	Postdisaster											
		Days											
Phase measure	0	2	4	6	8	10	12	14	16	18	20	22	24	26
1 Predisaster	←———													
2 Emergency														
Immediate		⊢—⊣												
Consolidation			⊢———⊣											
3 Rehabilitation														
Short-term			⊢————				———⊣							
Long-term							⊢————							→

——— Anticipated duration of measure

The Predisaster Phase

Preventive measures to mitigate the damages of any possible disaster must be taken. A national disaster committee should be established by the government, which will propose a national plan for disaster prevention and management and oversee its implementation (Fig. 1). Regional and local committees should also be nominated, receiving their directions from the national committee. The committees ideally include representatives of government, health services (primary, hospital and public) and other rescue organisations (police, fire protection, civil defence).
The national plan must incorporate the following objectives:

1. Prevention of natural and man-made disasters by hazard mapping of potential disaster areas, e.g. seismic active areas, areas prone to flooding or to tidal waves, hazardous industrial locations, etc.
2. Introduction of laws regulating anti-seismic building codes, the storage, transportation and disposal of hazardous material, protection against industrial spillage or nuclear accidents, etc.
3. Preparing an inventory of all relevant resources in manpower, material and equipment necessary for intervention in the disaster area. A survey of public and private buildings which could be used as temporary shelters for evacuees should be made and necessary equipment (mattresses, blankets, cooking utensils and sanitary installations) stockpiled.

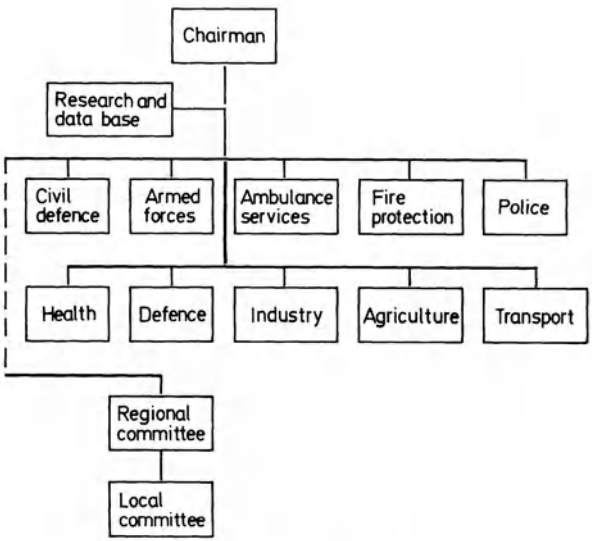

Fig. 1. Organisation of a disaster committee

4. Mobile, self-contained units for disaster intervention should be established, equipped and trained, including the following elements:
Rescue teams led by building engineers equipped with earth-moving, lifting and cutting devices necessary to extricate victims from collapsed buildings.
Mobile medical teams including physicians trained in emergency medicine, nurses, paramedics or medical orderlies, litter bearers and ambulance drivers. These units will be deployed at the disaster site, preferably in a large open space or room easily accessible to the litter bearers and close to a road to be used for evacuation. Ambulances or other vehicles fitted for the transport of casualties should be available to each team.
The casualty collecting station should be marked in daytime with an easily identifiable sign and at night by a coloured light. The units should be equipped with intravenous fluids in adequate amounts, bandaging and splinting material, endotracheal tubes and bags for assisted ventilation, suction pumps and antibiotics for i.v. or i.m. administration. Surgical instruments may be limited to trachiotomy, intracostal drainage and venous cutdown sets.
The treatment provided by these "forward" medical teams should include only the necessary steps to stabilise casualties for further transportation and final surgical treatment in a hospital. Triage at the arrival of casualties should always be performed by the senior medical officer who will establish priorities of treatment and evacuation to the proper medical facility. Victims dead on arrival should be removed to a specially designated collecting area — after an identification has been done on site.
Fire protection services will receive priority at the disaster site, extinguishing fires, pumping air to trapped victims and evacuating smoke and water from flooded areas. The fire chief may be the best qualified officer to command the rescue teams on the scene.
Police forces will be responsible to maintain law and order, preventing looting, directing traffic and the evacuation of homeless people. Roads will have to be blocked to prevent unauthorised people entering the site. They will also be responsible for the identification of the dead and dissemination of information to concerned relatives and the public in general. In some countries the senior police officer may be the coordinator of all emergency services in the disaster area.
Civil Defence units may be mobilised to reinforce rescue teams and medical teams at the site. In some countries the Civil Defence will assume responsibility and command of the disaster area. If this is the case, all rescue services including the medical units will be placed under the command of the senior Civil Defence officer on the site.
Armed Forces units may be recruited to assist the civilian rescue teams. The armed forces may be in command of resources of great importance

to the rescue operation, such as radio communication systems, transportation, including helicopters, engineers and mechanics with heavy equipment, medical field units including field hospitals, etc. In major disaster situations a state of national emergency may be declared with transfer of all powers in the disaster area to the commanding officer of the Armed Forces.

The Immediate Phase (Impact Phase)

This is usually brief and may last seconds (as in earthquakes), hours or days as in floodings, industrial or nuclear accidents, or weeks and months as in famine or war. During this critical period, the affected population may usually have to cope with its own resources before outside help can arrive. It is essential that the affected community be familiar with its disaster plan and able to activate it immediately. Local authorities will have to take command and activate the local emergency units and system. Regional and national authorities will have to be notified without delay about the extent and location of the disaster. An immediate assessment of damages and injuries to the population in the affected area must be made by local authorities and special "assessment teams". All components of the emergency and disaster system prepared and ready according to the directives of the disaster plan will have to be alerted immediately and all personnel called to their respective posts.

Hospitalisation

Casualties are to be evacuated from the disaster area to the nearest hospitals by ambulances and all available means of transportation. At the entrance to each hospital the triage officer — preferably a senior surgeon — will decide on priorities for medical treatment and direct the flow of casualties to the proper treatment areas. Each hospital will deploy its manpower and facilities according to the disaster plan. The plan will include the following:

1. The objectives of the plan and the responsibilities of the disaster coordinator and members of the hospital administration.
2. The alert, namely who is authorised to initiate the internal and external call-up. The call-up may be by an internal loudspeaker system in the hospital or by pagers and by telephone calls for calls outside the hospital.
3. Definition of the following functionaries: the hospital administration, the chief surgeon of the Emergency Department (ED), the triage officer at the entrance to the hospital, advisers to the ED from various speciali-

ties, the chief nurse in the ED, registration and recording personnel, medical treatment teams in the ED, operation theatre personnel, X-ray department personnel, laboratory personnel, nursing teams of pre- and postoperative care units, security officers and guards at the entrance to the hospital, social workers and psychological/psychiatric support teams, public relations personnel to receive and brief members of the press and TV crews and run an information service.

4. The plan should include a flow chart specifying the flow of patients inside the hospital (Fig. 2).
5. The chief surgeon, responsible for the reception and treatment of all patients in the ED, should receive a list of the patients admitted to the ED. This list is to be prepared by the office of registration and should include each patient's name, sex and age. The surgeon will state the diagnosis, severity of injuries and plan of treatment for each patient. This list is of great importance for the control of patient flow in the hospital. A copy of this list should be disseminated to the chief administrator and public relations office.
6. Debriefing sessions should be done regularly after each disaster drill or exposure to real disaster. Lessons have to be learned and mistakes corrected.
7. Medical equipment should be stockpiled in all relevant units in the hospital. Treatment and resuscitation carts in sufficient numbers should be kept in proximity to the ED. Additional emergency trolleys or army-type litters should also be readily available. Several hospitals have developed special aprons for ED nurses with several pockets holding the most necessary items for initiating i.v. lines, bandaging wounds and drawing samples for routine tests and crossmatching of blood.

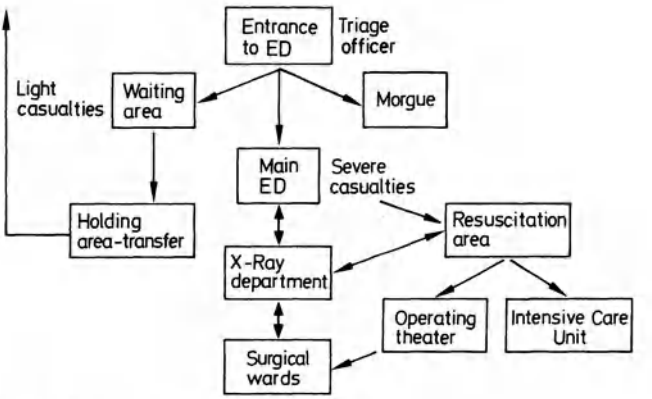

Fig. 2. Flow chart of patients in hospital

8. Registration forms for casualties should be prepared in plastic bags and include the following items (all items to be marked with the same number for easy identification): a plastic identification bracelet to be attached to the patient's wrist or ankle, an ED chart, all necessary forms for ordering X-rays, routine blood examinations, etc., a large plastic bag for the storage of personal belongings.

Evacuation of Casualties

At the site of a disaster severe casualties are medically stabilised and transferred to the nearest hospitals by ground transport. In some instances, in inaccessible areas, air evacuation may be necessary. Casualties must be sorted or "triaged" by the most experienced surgeon present who will decide on priorities for treatment and evacuation.
Many of the light casualties will seek medical assistance on their own in the nearest local hospital. This hospital may soon be overfilled and cease to function. In order to "protect" this hospital, medical triage will have to be performed at the entrance to the ED and light casualties may have to be directed to an ambulatory treatment facility, e.g. the outpatient clinic or a Red Cross station. This hospital will become a "sorting" hospital, stabilising and resuscitating all medium and severe cases and operating only on the most severe cases who would not survive further transportation. All other casualties will have to be distributed to other hospitals, located further from the disaster area, which thus become "receiving" hospitals. It is not recommended that medical personnel be removed from hospitals in the disaster area to reinforce teams at the disaster site. In mass casualty situations, when surgical teams are overburdened, the most afflicted casualties, suffering from multiple thoraco-abdominal or severe head trauma (having the worst prognosis), may have to be treated as an "expectant" group. These casualties will be operated on only after the "urgent" group of patients (needing less extensive surgery) have received definitive treatment. Most of the oper-

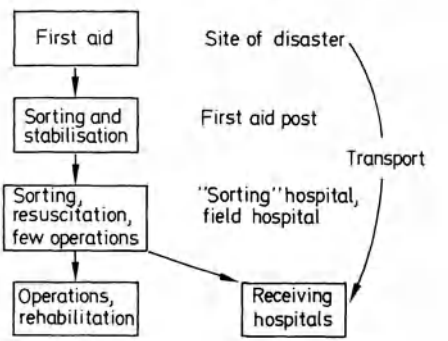

Fig. 3. Chain of evacuation

ations will be performed in fixed hospitals, but in some rural disaster areas located far away from hospitals, mobile field hospitals may be deployed in order to shorten the chain of evacuation (Fig. 3).
A field hospital should be self-contained in order not to put an additional burden on the already overtaxed supply system. Only severe, urgent cases should be operated on in these facilities in order to maximally utilise their potential to stabilise and prepare a large number of patients for further evacuation.

Injury Patterns

Types of injuries and their locations will vary according to the different causes of disasters. In earthquakes and landslides death may be instantaneous as a result of the sudden crush of head, chest or abdomen or the suffocation caused by an inadequate supply of air. In floodings, death may be caused by drowning or hypothermia. The ratio of dead to injured is determined by the effectiveness and speed of rescue operations (Table 5).
A field hospital deployed on the 3rd day after the earthquake in Guatemala in 1976 received most of its casualties during the following 4–5 days, the number of survivors decreasing after that time (Fig. 4).
Most of the patients admitted to hospitals after earthquakes suffer from injuries to their extremities (Table 6).
Many survivors trapped in collapsed buildings for more than 6 h suffer from extensive crush injuries. The massive rhabdomyolysis caused by prolonged ischaemia to their limbs will cause myoglobinaemia after their extrication from the rubble, with ensuing metabolic acidosis, hyperphosphataemia, hyperkalaemia, hyperuricaemia, coagulation defects and volume depletion. Immediate treatment with massive infusions of electrolyte solutions and sodium bicarbonate may prevent the fatal outcome of renal failure, as has

Table 5. Ratio of killed to injured (from Beinin 1985)

Date and place	Number killed	Ratio of killed to injured
1929, North Iran	3253	1:0.34
1930, Tadzhikistan	151	1:1.4
1931, Armenia	231	1:3.8
1934, Armenia	456	1:2.6
1943, Java	213	1:15.3
1960, Marocco	12000	1:1
1963, Yugoslavia	2000	1:1.7
1976, Guatemala	22000	1:3.4

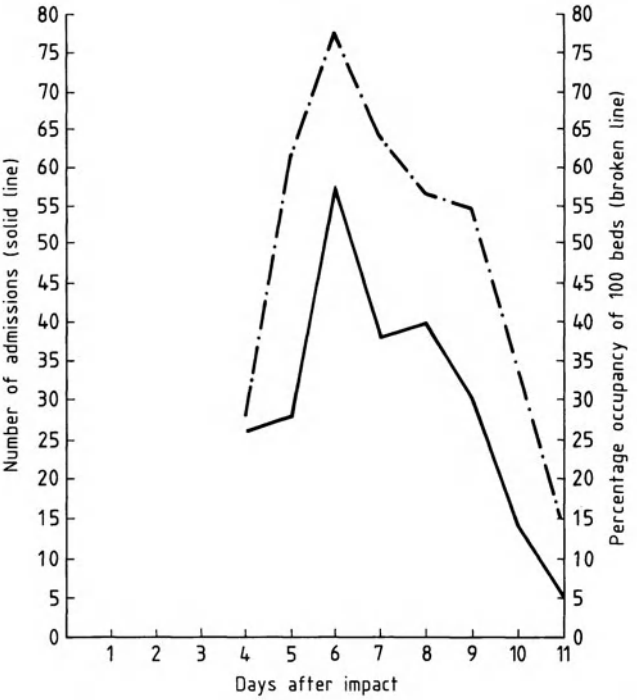

Fig. 4. Admissions and occupancy rates at the field hospital in Chimaltenango, Guatemala, 1976 (from PAHO 1981 Emergency health management after natural disasters)

Table 6. Topographical lesions in 550 hospitalised cases (from Villazon-Sahagun)

	Number	% Total	% Patients
Head and neck	178	20.7	32.3
Spinal cord	36	4.2	6.5
Upper extremities	224	26.1	40.7
Lower extremities	284	33.1	51.6
Thoraco-abdominal	136	15.8	24.7
	858		

been learned from recent experience with in victims trapped in collapsed buildings in southern Lebanon.

A large number of victims suffer from multiple injuries as witnessed in the earthquake of El-Asnam, Algiers, in October 1980 (Table 7).

Table 7. Patients diagnosed 12–16 October 1980 in emergency hospital Khemismiliana after earthquake of El Asnam, Algiers, on 10 October 1980 (from Journal of WAEDM 1 [suppl I] 1985)

Injuries	Occurrence
Contusions and bruises	91 (74%)
Infected injuries	83 (67%)
Closed fractures	80 (65%)
Open fractures	7 (7.5%)
Limb fractures	58 (47%)
Head fractures	12 (9.8%)
Pelvic fractures	13 (10.5%)
Thorax fractures	4 (3.3%)
Spine fractures	1 (0.8%)
Abdominal injuries	4 (3.3%)
Osteomyelitis	2 (1.6%)
Pregnancies (1 caesarian)	3 (2.4%)
Injuries not attributable to the earthquake	4 (3.3%)
Infectious diseases	3 (2.4%)
Total injuries diagnosed	365
Total number of patients	123
Average number of injuries per patient	3

Rehabilitation and Reconstruction

This phase of the disaster management will begin almost immediately after the acute (impact) phase of the disaster. It may continue for weeks, months or years. The main objectives of the health services will be to minimise additional exposure and risks to the affected population and to re-establish pre-disaster services. The health risks to the population are as follows:

1. Interruption of regular services for specific medical conditions, e.g. pregnancy, diabetes mellitus, renal dialysis.
2. Adverse environmental conditions, e.g. lack of shelters from exposure to low or high temperatures, lack of proper food and safe drinking water.
3. The large number of people crowded in refugee camps without proper sanitary conditions (water, soap and toilets) and communal feeding will cause an increased incidence of communicable diseases. This in turn will cause an increased morbidity and mortality in small children and aged people, whose immunity and resistance are lower.
4. The inadequate control of disease vectors and pests will contribute to the incidence of diseases.

5. Psychological stress reactions will appear, usually at a later stage, in many of the survivors who have witnessed personal tragedies. Panic is not usually encountered, even in sudden disasters, and the majority of the affected population react quite rationally.

Preparations for this stage of disaster management should be planned by the health services well in advance. Physicians and nurses should receive education and training in providing preventive and curative services to a population affected and displaced by a disaster. The necessary equipment should be stockpiled in regional depots in order to prevent their destruction and make them readily available in any location. The equipment should include tents, field beds and mattresses, blankets, diapers (disposable), clothing and footware, water containers, tablets for purifying water, eating and cooking utensils, soap, toilet paper and tools to dig field latrines, disinfectants and pesticides. Medical equipment should include basic instruments necessary to run a dispensary containing essential drugs such as antibiotics, analgesics, sedatives, oral rehydration powder to prepare a drinking solution for dehydrated patients, etc. Immunisation will not usually receive high priority except for anti-tetanus booster doses for injuries and vaccination against measles in pre-school children.
The identification of the dead and their burial and the removal of animal carcasses should receive high priority for moral and medical reasons. Removal of debris and collapsed buildings and the re-establishment of roads, water and power supply and communication will be the task of non-medical services and should be included in the overall plan. Past experience has shown that the vast majority of evacuated disaster victims prefer to return to the location of their previous homes. The government must decide on the resettlement policy. Reconstruction of private houses and public buildings should commence as early as possible to prevent additional exposure and damage to the affected population.

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1.4 Organization of Civil Hospitals During Sudden War/Mass Casualties

J. M. Brandes

In countries in which hospitals are civilian and evenly spread, evacuation and transportation from a disaster area is relatively simple, and those hospitals should be utilized in a state of emergency. However, the preparation and organization for such an emergency is complicated. This chapter describes what has to be done by the hospital management to solve the problems of mass casualties in a disaster or at the outbreak of hostilities. The similarities and differences of those two occurrences will be discussed.

Emergency and disaster situations with mass casualties can occur anywhere in the world. The situation of mass casualties is defined for any hospital as an occurrence in which the number of casualties arriving exceeds the normal admission capacity during peacetime. Such casualties may be from road accidents (train collision with bus), subversive actions (car bombs, explosions), wounded soldiers or civilians airborne to the hospital, massive ammonia (or other gases) poisoning from a nearby chemical plant, earthquake, ships sinking, air plane crash, etc. These happen without warning, with casualties flooding the emergency ward with at best very short notice. Usually there will be some time for preparation after the first alarm, but there is never enough time for improvization! Therefore, it is vital to prepare contingency plans for all the possible scenarios.

Table 1 compares the similarities and differences in war and peacetime which have to be considered by management while preparing contingency

Table 1. Comparison between some conditions of peace and war

	Peacetime	War
Preparatory alarm	none	possible
Time for improvization	none	available
Casualties	mostly local	national
Organization, manpower, etc.	local	Ministry of Health, Defence and Labor
Time for advanced evacuation	none	Available on national and frontier basis

plans. Any disaster situation must be evaluated as soon as possible by the highest ranking personnel of the hospital, preferably the director. The state of emergency can be of short duration (hours) or long (days, weeks). Organization of the hospital during wartime is different:

1. Evacuation of patients from hospital to homes or other institutions
2. Reassignment of most of the medical departments into surgical or other departments (e.g., enlarge burn unit and ICU, change dialysis area to postoperative area because it is near the operating theater or for some other local reason, enlarge the orthopedic department)
3. Apply prepared contingency plans of hospital enlargement to hotels, schools, and various other places. From 800 to 1200 beds on campus can be created and from up to 1800 beds in the annexes outside campus.
4. Enlarge manpower by drafting civilians (Ministry of Labor), high school pupils (pretrained), military and paramilitary organizations, and through volunteers.

The following are the basic principles on which the management of the hospital is based during a mass casualties emergency (many tasks are performed simultaneously, so this list does not determine priorities).

Disaster Contingency Plans. For the various possibilities according to your geographical locality (e.g., proximity of refineries, chemical industries, harbor, river, earthquake, volcano, airport, etc.) contingency plans should be drawn up. There should be an information file available on each specific contingency and its specific problem (e.g., noxious agents and their antidote). The data regarding an occurrence should be analyzed and solutions found, and if changes are needed, implement them at once! Never postpone for "better times" – they never will come. Then simulate the situation: train and drill the staff and the hospital. In training never spare the management or the personnel.

Decide on the Evacuation Plan. Evacuate as many of the incumbent patients as you can according to a preplanned schedule, whether it is to their homes, the community, or to other institutions. Provide them with a disease summary, instructions, and, if possible, medication for at least 24 h. Prepare means of transportation and contact families, etc. Arrange an evacuation center (EC) on campus at an appropriate place. Have a senior physician specializing in internal medicine head the EC with the help of a senior nurse administrator, clerks, transportation, nurses, social workers, and stretcher carriers (Fig. 1) Use the information available to decide upon the dimensions of the evacuation. Calculate, according to the size of the hospital and the event, the modular schedule for the evacuation: e.g., in an 850–bed hospital, decide to have 20% increments of levels of evacuation, up to total evacuation. It possible, keep neighboring hospitals informed of the changing evacuation plans.

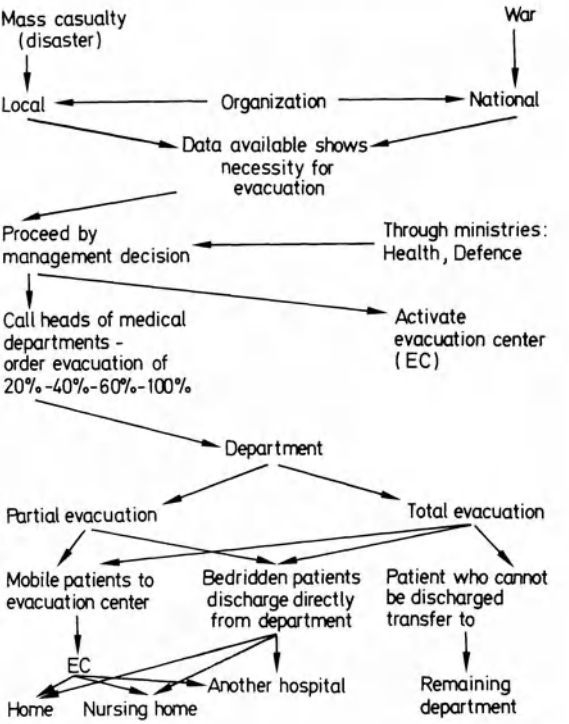

Fig. 1. Hospital evacuation planning

Core Assignment and Reassignment of Beds. Beds are allotted according to a tentative core assignment based upon estimates from previous disasters. The allocation should be very flexible whilst making allowance for large numbers of versatile multipurpose beds which can be used according to circumstances. The reassignment should be based mainly on the shift from internal medicine and pediatrics toward surgical and orthopedic beds. Always prepare as many intensive care unit (ICU) beds as possible, and monitors and respirators (of which one can never have enough!). Do all that according to local variables. Try and arrange for blocks of beds with a common purpose; arrange them according to proximity to other facilities (e.g., operating room); remember that transportation within the campus is going to be a problem.

Expansion of the Hospital. Areas can be prepared to serve various purposes. The amount of additional beds depends on the physical location of the hospital in relation to potential expansion areas, e.g., hotels, motels and schools, and on the trained personnel available to take over the com-

mandeered sites outside campus. When authorizing the use of a hotel or a childrens' hospital in the area, be sure that its elevators can take an adult stretcher (many such practicalities must be taken into account: the snags are identified during the simulated exercises of the contingency plan). While the evacuation from the hospital to other places goes on, the emergency area has to be enlarged considerably, and large areas are allocated for mild cases, which can be conveniently located in outpatient clinics, large waiting rooms, restaurants, etc., all of which should be at ground level. This reserves the limited space with the facilities of an emergency room for the treatment and triage of the medium and severe cases.

Manpower Availability. During wartime many young doctors and nurses are drafted, and their vacancies should be filled according to contingency plans. The manpower available in the area, such as high school pupils (14–18 years old), should be trained for various jobs, e.g., stretcher carriers and messengers for the blood bank and radiology. On a yearly training basis they are taught their jobs in the hospital during disaster. Their compensation is twofold: good services to the community (recognized as credit in the school) and training in cardiopulmonary resuscitation and first aid for their own benefit.

Institute a long-term program for reassignment and training of personnel for versatile roles, e.g., train the pediatrician to be the expert on fluid and electrolyte belance in the burns unit. Train the midwives to be operating theater nurses. Hold courses all year round on the use of mechanical respirators, etc.

Other manpower resources are the volunteer organizations associated with the hospital such as women auxiliaries, retired people who are trained in technical skills (to deal with the generators, elevator maintenance, etc.). Bring them in. In wartime, contingency plans for manpower are furnished by the Ministry of Labor. Change to 12-h shifts. Prepare kindergartens for the working mothers, arrange sleeping quarters for volunteers and families.

Law Enforcement. On campus, security guards, police and army should be available to control panic or riot and visitors. All approach roads to the hospital, especially from the helipad, must be cleared and kept clear.

Traffic and Transportation on Campus. To ensure the flow of traffic within the campus, two major issues should be addressed. All stretchers (for disaster) should be built on wheels with a folding option (for storage purposes). Since there will be a shortage of stretcher bearers, this system allows one man to push one stretcher carrying at least one casualty. Secondly, put up additional "traffic signs" naming the "new" departments. If possible, have color code strips for the mainstream traffic: to ICU, operating room, evacuation, helipad, etc.

Information and Communication. Control and decision making depend on information and feedback. Establish various means of gathering and giving information. Different types of communication system include:

1. Messengers who know their way around the campus
2. Telephones
3. General alarm systems that convey identical orders to all the departments
4. Information messages sent from a central computer station to peripheral stations through computers (on-line information), and if computer tabulation is disrupted have prepared statistical staff tables that are filled in manually with the information
5. Telecom, both inside and outside. Walkie talkies and Motorolla should be issued to key personnel and stations. Use a different wavelength than usual, otherwise the transmission may be jammed by a regular communication
6. Institute an emergency recruitment call system: those on shift each call four people who in turn each call another four according to profession and locality of their homes, so that grouped transportation can be arranged, and rapid dissemination of the emergency call achieved

Support Groups. Arrange for social workers, psychologists, and psychiatrists as support groups to the wounded and their families. If the occurrence is extraordinary, like many wounded children or many cases of burns or extended time of the state of emergency, then psychological support for the personnel also needs to be considered.

Information Center. Arrange for a center in the outskirts of the campus where up-to-date information on the state of the wounded is available for the families. During disaster expect many people to come to look for their relatives; a peripheral information center can supply some of the need for information. Clerks and social workers should man this center.

Triage and Resuscitation. Triage should take place outside the campus on the parking lot or at some similar site. This first quick triage is performed to separate out medium and severe injuries (which are transferred on stretchers to an enlarged emergency room of 25–100 beds) from the lightly injured who can walk or are wheeled to a concentration area preassigned for that purpose. Normally, patients can be moved back and forth to the emergency room after having been examined in the radiology department or elsewhere. However, during a mass casualty occurrence, the patients move in only one direction out of the emergency room (with no return option) towards radiology, operating room, or definite departments. This one-way traffic forces the triage officer in the emergency room to make definite decisions as to the destination of injured, for the sake of keeping the emergency room as empty as possible to receive the next wave of casualties.

No matter how meticulous the triage has been, it is always necessary to reevaluate. This is done at each of the destinations mentioned by a surgical officer delegated to this specific task. The two classical bottlenecks are within the waiting areas of the radiology department and the reception area of the operating room. Organize for resuscitation team and triage officers in both places. Here priorities for operation can be changed according to the condition of the injured and the facilities available. In addition, a very important group of 3–4 specialists circulates in the various departments and reevaluates diagnosis and treatment. Errors and omissions of diagnosis in various mass casualty occurrences run between 25%–40%.

Equipment. Arrange a special warehouse to store only equipment for states of disaster or emergency. Twice yearly, check all the equipment for availability and serviceability. Use solutions, antibiotics, drugs, etc. before the expiry date, and restock at once with new equipment and supplies so as never to fall below par.

Blood Banks. The storage of blood is normally centralized and works on statistical demands and a margin of safety. However, during wartime or in disasters when transportation becomes impossible or unreliable, facilities and trained personnel for blood donation are mandatory. Have a long list available of potential walk-in donors, e.g. medical students, nurses, high school students, and others in the neighborhood, All this has to be prearranged together with facilities for calling the donors by telephone, radio, TV, or cars with loudspeaker.

Skeleton Scenario of Mass Casualties Occurrence

The development of events in a hospital during a mass casualties occurrence can be described in three phases.

Phase I. Opening Data

Casualties arrive either at short notice or without any previous alarm, and information is received that there are more casualties to be expected. The optimal personnel (those best qualified to deal with the injuries) available at the hospital at that time are called through an alarm system to the emergency room or other appointed areas, to triage and provide treatment. The problems to be solved include:

- Deficiency in manpower such as doctors, nurses, technicians, secretaries, etc., both qualitative and quantitative
- Reorganization of manpower and equipment while treating the casualties that pour in
- Not enough senior people for appropriate decision making

Different responsibilities from those in peacetime in medical and administrative decisionmaking

Solutions

- Arrange for an alarm code system whereby all available personnel are called to the hospital while the "in-house" personnel reorganize and start treatment
- The senior surgeon present in the hospital automatically becomes the acting chief triage officer.
- The senior nurse present is responsible for the activation of the hospital emergency contingency plan and the preparation for phase II
- Training and periodic drilling of all hospital employees in their tasks when dealing with mass casualties.

Phase II

About an hour later many of the personnel will have arrived at the hospital. This coincides approximately with the time when the most extensive use of the medical facilities is needed: radiology, operating rooms, blood bank, laboratories, etc. The problems to be solved include:

- Not all the key workers are in the hospital and temporary substitutes take their place
- The distribution of the various functionaries is not necessarily the one that is optimal for the given stage
- The demand for services is usually beyond their capacity to supply a specific service, e.g. long queue in the radiology department, deficiency in the blood supply, etc.
- Communications tend to be jammed

Solutions

- Organize emergency headquarters that will manage the hospital and distribute the personnel arriving at the hospital
- Organize the streamlining and transfer of casualties between the various central points in and out of the hospital
- Prearrange storage of equipment and supplies in nearby special warehouses in sufficient quantity to last until phase III: resuscitation charts, stocks of blood, plasma, IV fluids, splints, bandages and surgical dressings, treatment and transfusion cards, etc.
- Prepare alternative communications systems e.g., messengers, walkie talkies, etc.

Phase III

After about 5 h most of the key people will be in the hospital, the casualities triaged, and primary treatment and resuscitation given. At this stage the

hospital may be stretched to its ultimate potential capacity and cannot admit more casualties. Arrangement for transfer to other hospitals by various means of transportations (buses, trains, helicopters, ambulances) should be considered and dealt with.

If more casualties are due and the hospital has the capacity and the means to treat them, then continued evacuation of previous inpatients and preparations for the new wave should be in progress. The pressure at the bottlenecks of action (emergency room, radiology, etc.) usually subsides now, and one should return if possible to a normal working rhythm whilst reinforcing those departments that took the main thrust and burden.

This chapter does not deal with the options of a tertiary care facility: however, they are worth mentioning: CAT, MRI, and US imaging from the diagnostic point of view and, in addition to the regular surgery and orthopedics groups, cardiovascular, neurological, plastic, maxillofacial surgeons, ICU, ICCU specialists and equipment. At hospitals with a helipad a multidisciplinary flying squad could be prepared that can be sent by helicopter to help and organize the first aid at the disaster site.

Summary

To work out a specific disaster contingency plan a lot of work must be done. One successful formula can be based on five elements in a loop: (1) Analysis of the problem, (2) formulation of a solution leading to conceptual (3) changes which have been (4) simulated by computer, and then (5) drilled to establish the present disaster contingency plan.

1.5.1 Organization of Civil Hospitals: Department of Radiology

A. Rosenberger

The radiology department is a vital link in the proper care of the injured patient. Diagnoses and priorities are established, confirmed or changed, and care is planned accordingly. In order to cope with a large number of casualties, changes are made in the department and in the work routine. The change encompasses administration, patient flow, continuing, treatment of patients within the confines of the department staffing and radiological staffing.

Layout. Ideally, the department should be situated as near as possible to the triage area, thus facilitating transportation and communication. The examination rooms and waiting areas in the department itself should be spacious enough to accommodate stretchers and the resuscitation equipment accompanying the trauma patient. Oxygen outlets and suction must be available. During the waiting period before and after the radiological work-up, vital signs must be monitored and supportive treatment has to continue.

Administration. The clerks in charge of identification of the wounded, registration and administrative work should be placed at the entrance of the admission area to the department of radiology. Anonymous patients lacking personal data have already received some identification mark or number in the triage area, and these marks should be retained without any changes on the files, envelopes and folders in the radiology department until definite identification becomes available.
The written report of the radiologist together with the radiographs should accompany the patient when leaving the radiological department on completion of the examination. A second envelope containing copies of the reports is retained in the department. During hospitalisation, the envelope accompanying the patient should remain in the ward to permit ready consultation and follow-up on the floor.

Patient Flow. Patient flow should be unidirectional, i.e. from the triage area to the radiology department and from there either to the operating theatre or to the ward. The wounded should never be returned to the triage area. (Diagnosis is completed in the radiology department, allowing for a decision on the patient's treatment and next destination.)

Treatment Staffing. Injured patients require intensive, continuous supervision. Therefore, trained nursing staff in adequate numbers must be present in the radiology department. The presence of at least one physician is necessary (preferably a surgeon experienced in traumatology). Any sudden or unexpected change in the patient's condition requires on the spot decisions on alterations in priorities of examinations, treatments and destinations.

Radiology Staffing. A senior radiographer should be present at all times in the waiting area adjacent to the registration clerk. His duty is to direct the patients into the appropriate examination rooms according to the type of examination requested and the status of the patient. He directs the flow of the patients into and out of the examination rooms. He is in constant contact with the reporting area to ascertain whether the radiological work-up has been concluded and orders the transfer to the appropriate destination. Each room should be worked by two radiographers to speed up the pace of work.

Reporting is best done by a team of two radiologists who maintain consultations with the surgeons in charge of the wounded. Quality work is done in the radiology department only. The use of mobile units in the triage area should be reserved for exceptional emergency cases only. The number of such cases is small, and therefore financial investment and the dispersal of radiologists and radiographers is not justified on a large scale for this purpose.

1.5.2 Essential Clinical Laboratory Services

O. Zinder

Introduction

In times of disaster or war the routine functioning of clinical labs must give way to an emergency service geared to the immediate assessment of mass casualties and to follow their progress in the emergency room, the operating theatre or the intensive care ward. Some of the tests must be available quickly in the triage and emergency room area, yet can only be performed in a properly equipped central laboratory, remote from the emergency treatment area.
At the same time the laboratory service serving severely ill, hospitalised patients as well as the community must be maintained.
The establishment of an active emergency lab, easily convertible into a major service status during war or emergency, is a cardinal requirement of preparedness in all hospitals, large or small. The instruments and reagents necessary for the required laboratory tests should be in daily use to ensure their potency and accuracy, and the laboratory personnel should be prepared to transfer quickly and smoothly their activities to the emergency lab. Only thus can the laboratory provide the medical staff with the tests and information so vital to the efficient treatment of disaster victims.

Preparedness and Training

The proper provision of rapid, reliable and effective laboratory tests is essential when disaster strikes and may save many lives. If, however, the instrumentation and personnel are not ready, and the proper preparations have not been made and practised well in advance, the service is liable to break down. A disastrous bottleneck may then form, threatening the very existence of the clinical emergency effort. The tasks to be carried out in disaster or war must be written out clearly and disseminated to all involved personnel. These instructions must be learned by rote, and periodically rehearsed. Logs must be kept on the maintenance and quality control of all instruments and procedures to be used in emergencies to make sure they

will work efficiently when needed. Each worker must know his responsibilities and the location of his activities, and substitutes for each function should be trained to ensure the smooth transition from routine work into disaster-level activity. Senior staff members should be made personally responsible for the implementation of these advance preparations and for overseeing the proper application of the emergency plans when a disaster occurs.

By constantly practising these precautions and preparations, the laboratory services will be ready to face the challenge and be an invaluable asset to the medical effort during war or disaster.

Essential Laboratory Tests in a Disaster Situation

The design and operation of laboratory services in war and disaster depend on the tests which must be provided and the instrumentation necessary to carry them out (Table 1). Additional tests are also listed in Table 1 which help to cover a variety of disaster medicine contingencies.

Table 1. Essential laboratory tests and their appropriate instruments during war and disaster

Procedure	Instrument
Standard emergency tests	
Biochemistry	
Blood gases	Blood gas analyser
Electrolytes	Whole blood electrolyte instrument
Blood bank (typing and cross-matching)	Manual test
Haematology	
Blood cell count	5-parameter cell counter
Clotting factors	Semi-automated or manual
Microbiology (culture and sensitivity)	Rapid culture instruments (based on CO_2 production, turbidity or luminescence)
Additional laboratory tests	
Kidney function, proteins, enzymes (crush injuries)	Automated spectrophotometer
Proteins (burn injuries)	Automated spectrophotometer
Chromatography	Semi-automated or manual rapid toxicological screens (toxic and chemical injuries)

The Base Hospital

The Emergency Laboratory

The capability must exist to perform the essential laboratory tests simultaneously with the delivery of casualties to the hospital, as often there is no prior warning. This requires the establishment of a round-the-clock emergency laboratory service within the framework of the existing laboratory services of the hospital. Table 2 lists the instruments with which this lab should be equipped to be able to provide both the biochemical and haematological tests necessary, as shown in Table 1.
The instruments must be in constant use and properly calibrated and maintained in order for them to be useful when disaster strikes. There is no point

Table 2. Recommended emergency laboratory instrumentation and tests

Instrument	Type	Tests
Blood gas analyser	Capillary sample Rapid turnaround Automatic calibration	pO_2, pCO_2, pH
Electrolyte analyser	Whole blood sample Semi-automatic or automatic sampling Multiple analyte determination	Na^+, K^+ Cl^- HCO_3^- Ca^2
Spectrophotometer	Multiple analyte determination (easy shift from one to another) Semi-automatic or automatic sampling	Bilirubin BUN Calcium Creatinine Enzymes (CPK, SGOT, amylase cholinesterase) Glucose
Blood cell count	Multiple results Rapid turnaround	Haematocrit Haemoglobin RBC WBC
Clotting	Semi-automated	Fibrinogen Clotting factors
Toxicology	Manual multi-sample thin-layer chromatography (recommended) or: Dedicated drug monitoring instrument	Levels of poisonous substances (macro scale, intended to differentiate between lethal, toxic and non-toxic levels)

in keeping inventories of materials and instruments on a shelf marked "Disaster" which are not in routine use, as they will be found to be out of order or incorrect precisely when they are needed. Back-up instruments must also be periodically used, maintained and checked for the precision of their results. An inventory of a 2-weeks' supply of reagents and materials must be kept and rotated into use on a regular basis.
By sticking meticulously to these rules all that needs to be done upon receiving notification of the onset of a disaster is to step up the operation of the laboratory and to add staff.

Location

The emergency laboratory is often situated in the vicinity of the emergency room and/or the triage area. It should not have to be moved from its accustomed location during war or disaster. This laboratory should be connected to emergency power, water supplies and waste disposal.
Nevertheless, the location of the emergency lab should ideally be within the area of the hospital's laboratory complex and be connected up to the emergency room area by tube (pneumatic or carriage) system, for delivery of specimens, and by a communications network (computer-telephone lines and intercom) to report results. The advantage of this is threefold:

1. The emergency area is not cluttered up with personnel not directly attending the injured
2. The emergency laboratory service can be carried out more efficiently and quickly when not in the vicinity of the hectic activities surrounding the injured
3. The emergency lab can draw upon staff, reagents, instruments, etc. to be found in the other, more extensively equipped and staffed laboratories

An additional possible location for emergency laboratory testing is the operating theatre. This, however, should be kept to the basic minimum of the really extremely urgent tests: blood gases, electrolytes and haemoglobin. In order for these tests to be available, dependable and valid during a disaster, they must be carried out by qualified laboratory personnel, trained specifically for this purpose, who will report to the operating theatre area together with the portable instruments necessary immediately upon notification of the disaster and the impending flow of injured to the hospital. They will set up their instruments in an area central to the operating theatre which has been preordained for their activities, calibrate them and be prepared for collection of samples (by hand) and transmission of results (intercom). They will also be responsible for delivery of specimens to the central lab area (by tube) and the collation of these results.
This work cannot, and should not, be carried out by operating theatre personnel since they are not professionally versed in the proper operation of

the instruments, are not experienced in overcoming faults during operation and certainly cannot work at the speed demanded by a disaster situation, as professional laboratory workers can.

Personnel

A disaster situation supersedes all other hospital activities, including routine laboratory testing. This relieves many laboratory workers from their normal, daily activities and allows them to reinforce the emergency laboratory services (adding staff to the emergency lab, and activating the operating theatre laboratory services).

Community Laboratory Services

War or disaster result in the clearing of general hospitals to accommodate the injured. Many non-critically sick patients will be sent to their homes to be cared for by community medical services and outpatient clinics. This will result in an increased routine laboratory workload in the community medical services. All available personnel should be put in a full-shift and/or overtime post in order to exploit fully the existing facilities.

1.6 Equipment for Forward Casualty Care: The Battalion Medical Officer Kit

R. R. Rozin and J. M. Klausner

Today's battlefield is inundated by hand-held, guided, ground-to-air missiles which, together with intense tank battles, preclude immediate helicopter evacuation of casualties. The battalion medical officer must be prepared for care and support of the casualty in the field for long periods of time until evacuation to the rear becomes feasible.
It is therefore important to devote the utmost attention to the equipment and medications to be allocated to the medical officer for the treatment of combat wounded in the forward clearing stations.

Principles for the Choice of Equipment

The equipment and medication put at the disposal of the battalion medical officers should enable them to deliver in the field treatment similar to that given to a casualty in a well-equipped civilian emergency room. Since the officers cannot carry with them to the field all the equipment found in an emergency room, the choice must be very exacting.
The equipment must be light, compact, easy to use, and multifunctional and have the ability to withstand adverse conditions: The advent of plastic materials and disposable equipment have made resuscitation tools light and portable.
The contents of the military officer's bag should reflect the aim to counteract the systemic effects of the injury, and not only treat the local wounds (see Table 1).

Resuscitation and support during evacuation require:

- Respiration and airway control
- Blood volume control
- Hemostasis and wound control
- Prophylactic treatment
- Monitoring

Table 1. Functional classification of the equipment in the medical officer's kit

Respiration and airway control	Blood volume control	Wound control	Prophylactic treatment	Monitoring
Laryngoscope	Venesection sets	Tourniquets	IV fluids	Stethoscope
Oral airways	Central vein sets	Personnel pressure dressings	Nasogastric tubes	Sphygmomanometer
Endotracheal tubes	IV sets	Large abdominal dressings	Urinary catheters	Central vein sets
Face mask	Intracath	Burn dressings	Systemic antibiotics	Urinary catheters
AMBU bag	Pressure administration sets	Bags of electrolyte solution	Steroids	Nasogastric tubes
Suction pump	Blood substitutes	Local antibiotics	Sodium bicarbonate	Medical record charts
Trocar intercostal tubes,	Steroids	Splints	Diuretics	
Heimlich valves, tracheostomy sets	Electrolyte solutions		Analgesics	Thermometer

Equipment for Respiration and Airway Control

The primary and most urgent actions taken by the MO are the establishment of a free airway and ascertaining that respiration is resumed. The face mask and oral airway will suffice in most cases of airway obstruction. A laryngoscope and endotracheal tubes are used for performing intubation in most cases of respiratory arrest or head and neck trauma. When intubation cannot be performed, a tracheostomy set is available: The set must be self-contained and hence include all the instruments and materials needed for the performance of this procedure.

Respiratory difficulties may also arise from mechanical derangements of the chest wall and the pleural cavity. Pneumo- and hemothorax present immediate life-threatening situations. In order to drain the pleural cavity rapidly, a trocar intercostal tube is provided. The introductory set includes the trocar, with its tube, a one-way Heimlich valve, and connecting tubes to a plastic drainage bag (in addition to the other instruments and materials needed for the procedure). With this equipment, intercostal drainage can be

achieved while both the casualty and the medical officer are in the lying position, as may occur under fire.
The AMBU bag with a non-rebreathing valve provides an excellent solution for assisting respiration. The casualty can be respirated with the aid of the AMBU even by a minimally trained medic. The bag itself can be connected to a face mask, to an endotracheal tube, or to a tracheostomy tube.
A leg-operated vacuum pump is provided to suck out secretions from the mouth, the endotracheal tube, or tracheostomy. It can be powered by foot pressure or by the flow of gas from an oxygen tank when this is available.
This set of equipment provides the medical officer with the capability to perform the full gamut of respiratory resuscitation procedures. After intubation or chest drainage are established, the casualty can be prepared for safe evacuation over land or by helicopter.

Equipment for Blood Volume Control

Equipment must be available to provide the means to restore blood volume via various venous routes and at different rates. Peripheral vessels may be accessed with the aid of regular IV sets with large bore needles. The Intracath permits the utilization of central veins for restoring blood volume rapidly. Medical officers must be versed in introducing these catheters into the subclavian or jugular veins. Only rarely should there be a need to perform venous cutdown for vein access. However, an instrument set for venous cutdown is provided for this eventuality.
Two problems exist with rapid fluid administration in the field. The first is the need to create hydrostatic pressure for the IV administration. The infusion bottles must be hung high to achieve rapid flow of the solution, and this of course can present an easy target in a battlefield or be an obstacle during transport. To offset this problem, a pressure adminitration set creates steady pressure on the IV bag. This allows a rapid and even rate of infusion while the bag is laid beside the casualty.
The second problem is that of air accidentally entering into the infusion line. To remedy this, the drop chamber is especially designed so that air cannot possibly enter even when the IV bag is laid on its side or fluids are administered under pressure.
Two other important modifications are incorporated into the IV set. One is the placing of a Luer lock where the line connects with the needle so that it cannot be accidentally disengaged. The second is an accurate and sturdy drop mechanism to control the rate of infusion.
Volume replacement in the field is best accomplished with electrolyte solutions. Ringer's lactate has proved itself in all the recent wars, and it remains the mainstay of blood loss replacement in the field. Other blood substitutes are available. Currently the medical officer is provided with Haemaccel.

All the IV sets have filter chambers, and they can be used for blood administration when blood is available at the regimental aid station or during helicopter evacuation; the sets need not be changed.
Steroids are provided for IV administration.

Equipment for Wound Control

It is imperative to stop bleeding and occlude open wounds to prevent further contamination. The standard military personnel sterile bandages are ideal for this purpose. Large occlusive bandages are also supplied for dressing extensive abdominal or chest wall wounds.
Debridement and cleansing of contaminated open wounds is achieved by forcefully flushing the wound with the sterile Hartmann solution. The IV bag is compressed with both hands, and its spout is used to direct the stream of fluid into the wound. Local antibiotic ointments are also provided for use on mucus membranes and in the eye.
Slings are used to immobilize and splint wounded or fractured limbs. Tourniquets are provided to arrest bleeding but are used only when pressure dressings are not sufficient to do so.
Specialized aluminated dressings are provided to cover burns. These dressings can be shaped according to the size and location of the burned areas to be covered, and they are both non-adhering and prevent evaporation. Metal mesh splints, which are rolled when packed, are used for fracture immobilization.

Equipment for Prophylactic Treatment

The medical officer's kit must provide for problems which either occur inevitably or are highly probable as a consequence of the initial wounding. Early treatment can prevent some of these consequences.
Gastric dilatation occurs frequently in response to head injury, abdominal injury, spinal injury, burns, or severe soft tissue injuries, causing vomiting, abdominal distension, bleeding from the stomach lining, and respiratory distress due to pressure on the diaphragm. This dilatation can be prevented or alleviated by emptying the stomach and suction. A nasogastric tube is provided to be introduced even in the field.
Large quantities of electrolyte solutions must be used for blood volume replacement in order to maintain normal blood pressure. Inevitably diuresis ensues, and the casualty must evacuate large quantities of urine. Urination, though, is difficult because of pain, recumbence and the use of narcotics. The urinary bladder becomes distended, causing discomfort and pain. Catheterization of the urinary bladder is therefore performed in the field prior

to evacuation in anticipation of this problem. (Restlessness in patients with head injuries is frequently due to distended bladder!) Sepsis is a much dreaded complication of the war wound. To be effective, prophylactic antibiotic treatment should be started as soon as possible. Crystalline penicillin is provided as the mainstay antibiotic agent. It is given in doses of 5 million units starting with the first IV bag. Additional antibiotics are provided to be used for severe contaminated wounds such as penetrating abdominal injuries or very extensive soft tissue damage: chloramphenicol is provided for IV administration.

IV sodium bicarbonate solution is used whenever metabolic acidosis is anticipated. Prolonged hypotension or severe wounding followed by intensive resuscitation require the rapid correction of acidosis with sodium bicarbonate.

Diuretics are provided for establishing urine flow in kidneys endangered by severe hypovolemia. Furosemide is supplied for IV adminstration.

The medical officer often sees the casualty soon after wounding and before pain becomes a problem. Whenever there is no contraindication to analgesics, they are given in an adequate dose. Morphine is provided for IM and IV administration.

Equipment for Monitoring

Bulky, heavy monitoring machinery cannot be carried in this echelon. Hence maximal use must be made of diagnostic equipment for the purpose of monitoring the casualty's vital signs: Stethoscope and sphygmomanometer are therefore the only designated monitoring tools in the kit. (The din of the battlefield or disaster area may preclude their use.)

Central venous pressure can be measured during IV fluid administration by using the plastic tubing of the IV set. This is done by momentarily disconnecting the tubing from the solution bag and measuring the height of the column of water in the tubing.

A measure of vital organ blood perfusion can be obtained by observing the urine flow from the catheter into the plastic collection bag.

The volume of blood loss from chest wounds can be estimated by measuring the contents of the bag connected to the intercostal chest drain. Measuring the fluid aspirated from the nasogastric tube gives a rough estimation of intraluminal intestinal fluid loss. Hypothermia is diagnosed and its treatment is monitored by repeated measurement of the rectal temperature.

The importance of keeping records cannot be overemphasized: under battle conditions this may be very difficult and at times recording is reduced to scribbling on the casualty's forehead or chest. Stiff and easily attachable record cards are provided in the kit so that the medical officer can record his observations and treatments for use by the next medical echelon.

The Kit and Its Contents

The equipment itemized in Table 2 can be fitted into one backpack. It can be carried in the field without too much effort. The backpack is made of a water-resistant material and is systematically compartmentalized. The immediate essential respiratory equipment is concentrated in the middle compartment and is accessed easily. Dressings and splints are placed in the side

Table 2. Itemised list of the contents of the battalion officer's kit

Instruments	*Medications*
Set for performing - Venesection	Injection - Adrenalin
- Tracheostomy	- Sodium bicarbonate
- Intercostal drainage	- Sodium chloride
- Nasogastric intubation	- Atropine sulfate
- Central vein catheterization	- Pramine
	- Furosemide
	- Morphine
	- Crystalline penicillin
General suture set	- Chloramphenicol
Sphygmomanometer	- Prednisolone pivalate
McGuile forceps	- Diazepam
Laryngoscope	Ointments - Chloramphenicol
Suction pump	- Sulfacid
Face mask	- Lidocaine
AMBU bag with non-rebreathing valve	IV fluids - Ringer's lactate
Stethoscope	- Haemaccel
Oral airways	
Primary IV sets	
Dressings	*Miscellaneous*
Standard cotton bandages	Pressure administration set
Ace bandages	Head lamp and batteries
Roller gauze	Extension tube for AMBU
Personnel dressings	Disposable syringes 2 cc and 10 cc
Abdominal dressings	Hypodermic needles
Burn dressings	Medical report forms
Arm slings	Rubber tourniquets
Gauze pads	Prepacked suture material
Adhesive tape	Thermometer
Alcohol swabs	Intracath IV catheters
Splints—metal mesh	Intercostal trocar set
	Heimlich valve
	Endotracheal tubes
	Scissors
	Safety pins

compartments. The remaining equipment is placed in a logical manner in well-marked, color-coded compartments. The kit can be completely opened and spread out so that it serves as a mobile first-aid station. The covering flap is used for laying out the equipment being used.
The kit contains sufficient supplies to treat three casualties requiring respiratory control and several others requiring other treatment. Additional supplies are provided in another bag which is carried by the accompanying aidman.

The Medical Officer

This kit provides battalion medical officers with as sophisticated resuscitation equipment as can be carried. To utilize it well, they must be trained in basic life support techniques so that they can perform definitive resuscitation procedures. Medical officers of today must deal not only with the immediate threats of the wound, but also anticipate future complications. They have to deal not only with the wound itself but with its systemic effects. For this purpose, they have to acquire essential skills:

Endotracheal intubation
Tracheostomy
Intercostal drainage
Central vein catheterization
Venesection
Nasogastric intubation
Limb splinting
Wound dressing
Airway suction
Suturing
Urinary catheterization
Central vein pressure reading
Use of AMBU bag

The Casualty

The availability in the field of sophisticated resuscitation equipment increases options for triage. Casualties in the field have a greater chance of surviving because they can be intubated and their respiration assisted for long periods of time. Their hemodynamic state can be rapidly improved by the infusion of large volumes of fluids. This stable state can be maintained and monitored repeatedly for long periods with the means at the disposal of the battalion medical officer. This can be demonstrated much better when specific injuries are discussed. For example, the following treatment

can be given in the field to the casualties with an abdominal injury soon after injury. They are intubated for better oxygenation. Nasogastric and Foley catheters are introduced. One or two central veins are accessed with Intracath, and large volumes of fluids are infused rapidly. One or more antibiotic agents are started by IV administration. Analgesics are administered, and the casualties supported and monitored for long periods until definitive treatment can be carried out.

Casualties with brain injuries can be intubated and hyperventilated (to prevent or reduce cerebral edema). The stomach is emptied to prevent aspiration, and their hemodynamic state is supported and monitored via a central vein. The immediate dangers of respiratory embarassment and aspiration are therefore eliminated, and they can be maintained for long periods of time. At the same time, future complications and consequences are addressed.

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1.7 The Evacuation Hospital: Concept, Modes of Operation, and Organization

R. R. Rozin and E. Dolev

Introduction

A military field hospital is usually located not far from the combat zone, so that battle casualties may be treated as soon as possible. When the number of wounded is moderate, the hospital can give adequate definitive treatment to them all.
However, the scenario is usually different: large numbers of battle casualties arrive at all times, often without warning, overwhelming the hospital facilities, and it becomes no longer feasible to give the full treatment required to every casualty. To cope with this situation, the field hospital must convert itself to an evacuation hospital.

Functions

The functions of an evacuation hospital are to sort out all the wounded brought to it from the front line (triage), resuscitate them, and render all supportive treatment nececessy to prepare them for transport by air to another hospital in the rear. Only two groups of wounded remain in the hospital: the very severely wounded who cannot be rendered transportable without surgery, and the very lightly wounded who can be returned to their units after a minium period of hospitalization. The concept of the hospital acting as a triage center or an evacuation hospital can also be applied in a civilian setting to disasters or mass casualty situations.
These functions are fulfilled by:

1. Assessment and sorting of the wounded.
2. Immediate treatment of life-threatening respiratory failure and blood volume loss.
3. Surgical intervention for those casualties considered otherwise unlikely to survive transfer to base hospitals.
4. Surgery for the salvage of limbs.
5. Provision of hospital facilities for the very lightly wounded casualties who can be returned to the front or sent home within 48 h.

6. Provision of hospital facilities for shell-shocked casualties for longer periods of time.
7. Holding facilities for casualties until conditions are favorable for their evacuation.
8. Selection of appropriate rear facilities to which the evacuees will be sent.

Triage

The wounded are sorted into the following categories:

1. Unfit for evacuation in spite of adequate resuscitation: surgical treatment required in the evacuation hospital.
2. Fit for evacuation, or can be made fit after additional resuscitation.
3. Lightly wounded or shell-shocked.
4. Dying.

Treatment Priorities

Priorities of treatment are:

1. Further resuscitation and urgent surgical treatment at the evacuation hospital.
2. Further resuscitation and treatment before evacuation in preparation for sugery in a rear hospital.
3. Minor surgery and psychotherapy.

The main indications for operation in an evacuation hospital are:

- Massive hemorrhage in the abdomen, chest or cranium.
- Progressive peritoneal irritation with deterioration of vital signs.
- Extensive damage to a limb.
- Arterial damage endangering a limb.
- Injuries endangering the airway in the upper respiratory tract.

Location

The evacuation hospital should be located out of artillery range in a well-protected area. It should be located on a road intersection accessible to all the front units it serves. It should have a helicopter landing pad and preferably a runway nearby for fixed-wing aircraft used in the evacuation of wounded to the rear.

Construction

The hospital should be mobile so that it can be constructed even in remote areas. It should be constructed in such a way that its largest parts can be moved on a single truck or a large helicopter. According to the geographical region where it has to function, it should be fitted either with heating facilities or with airconditioning. The operating and intensive care facilities should be in a hard-shell structure and be well ventilated.

Layout

The hospital should be laid out in such a way that distances between the various facilities of the hospital are minimal. Since entry into the hospital is through the triage area, the latter should be located centrally, with all other facilities fanning out from it. On leaving the triage area, the casualty is transferred to either intensive care, hospitalization ward, operating room, psychiatric ward, or delay area. Most casualties will eventually end up in the delay area prior to evacuation to rear medical facilities. A suggested layout is presented in Fig. 1, which shows the possible movement between the various facilities.

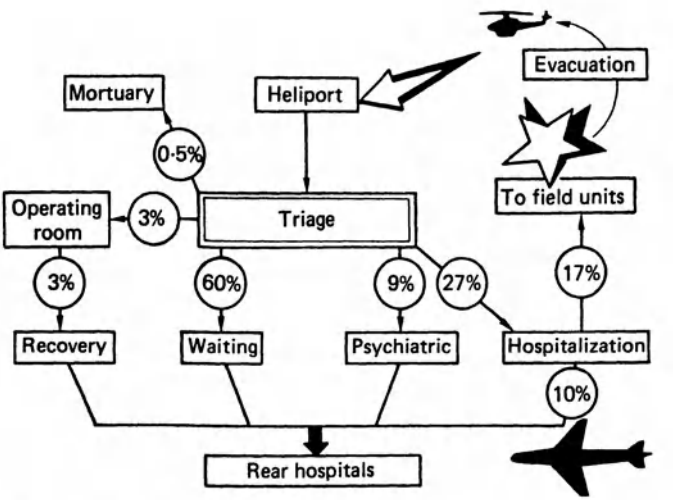

Fig. 1. Organization of an evacuation hospital

Triage Area

The area assigned for triage is proportional to the capacity of the hospital itself. No more than 36 casualties should be sorted at the same time in one area. If the triage capacity is bigger than this, it should be split up into smaller units, in order to avoid the commotion, noise , and confusion inherent in triage and intensive resuscitation. The triage area (Fig. 2) should be large and spacious with no hidden corners. Having one entrance and one exit simplifies the flow of casualties and personnel. The area nearest to the entrance is reserved for the most severely wounded, and empty stretcher racks without stretchers are spaced so as to facilitate ease of access and working room for the medical staff. The central area is assigned to the moderately injured and less equipment is stored in this area. The farthest part of the room is assigned to lightly and walking wounded. Only bandages and dressings are kept there. A dispensing station is set up in the center of each triage area for rapid and supervised dispensing of drugs and other medications. Mobile X-ray units are set up in each triage area to avoid moving patients unnecessarily; removal of a patient in order to X-ray him in another area carries the risk of his deteriorating unnoticed. X-Rays are considered sufficiently important to warrant the authorization of the chief triage surgeon. Mobile blood banks are set up in each triage area.

Operating Rooms

The operating rooms are situated immediately next to the triage area, since surgery is often a vital part of the resuscitation of more critically wounded casualties. They should be equipped with the best operating tables, good

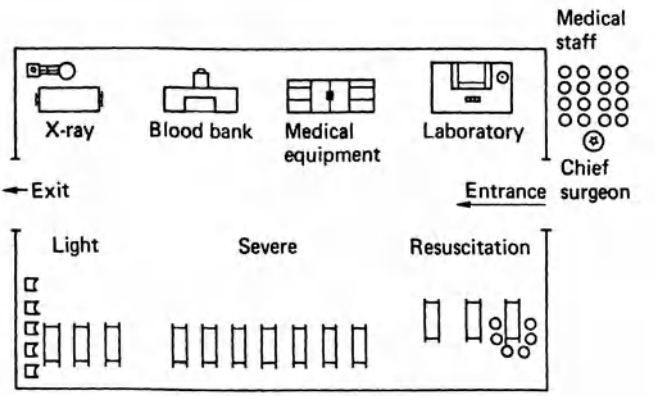

Fig. 2. Triage area

lighting, adequate supplies, quality surgical instruments, suction pumps, drugs, etc. All sterile equipment should be prepacked although there should be facilities for immediate resterilization. Much of the equipment — drapes, etc. — can be obtained in disposable form. Diathermy equipment should be available. Anesthesia equipment should also be of the best quality with a circle absorber and a ventilator.

Intensive Care

An intensive care facility is provided for the most severely wounded casualties who require respiratory assistance or intensive fluid replacement therapy. Casualties awaiting surgery and those after surgery should be concentrated in this area. It is inefficient to disperse the various groups of casualties requiring intensive care; by concentrating them in one area they can be managed best and all the specialized equipment can be concentrated in that one area. The intensive care facilities include monitors, a defibrillator, automatic respirators, and suction equipment. Large quantities of disposable equipment similar to that used in rear hospitals should be available.

Delay Area

This special area is for maintaining casualties until transport is available for their further evacuation. It is important to keep the triage area as empty as possible for the incoming wounded, so as soon as a casualty is stabilized, he should be removed to the delay area. This must be equipped for treating severely wounded casualties for periods of many hours, as evacuation may be postponed because of weather or enemy activity: monitoring equipment and a dispensing unit for drugs and intravenous fluids will be needed.

Radiography

The hospital must have X-Ray equipment — at least one unit, and more in bigger hospitals. At least one unit must be mobile so that it can be moved into the triage area and function during the receiving and sorting of casualties.

Blood Bank

Each evacuation hospital must have its own blood bank. When the hospital supports a major front or disaster it stores enough units of blood to supply the other medical units operating within its vicinity. The demands for blood are predictable; the average casualty will require 0.6 units of blood during

his stay in the hospital. Therefore, a minimum of 60 units are needed per 100 triaged casualties, and an additional 10 units to allow for cross-matching. Whenever possible, the blood bank should be supplied with a freezer to store fresh frozen plasma and should have facilities to store blood components. The blood bank is situated centrally since blood will be required in the triage area, in the operating theaters, and in the intensive care area. It has equipment for collecting blood so that blood can be donated by troops in the vicinity of the hospital. The blood bank should be modular, so that a section of it can be moved into the triage area and function there independently.

Psychiatric Facilities

Shell-shocked casualties constitute about 10% of all casualties. Modern psychiatric practice requires that these casualties be treated together with other wounded and are in no way to be segregated. The facilities for treating them should therefore be set within the hospital and provision must be made for hospitalizing these casualties for a few days with the aim of returning them to their units.

Hospitalization

Hospitalization facilities are set up according to the aims of the hospital in question. If it is expected that casualties will remain in the hospital postoperatively and until they are fit to travel, the hospitalization facilities are extensive and sophisticated. In a hospital functioning as an evacuation hospital and retains only casualties with light wounds and for a short period of time, there is no need to elaborately equip such a facility; only the bare necessities are installed.

Pharmacy

The stocks of drugs, intravenous fluids and disposable medical supplies are stored and dispensed by the pharmacy, which has a large storage space, refrigeration facilities, loading and unloading ramps, and equipment for transporting supplies within the hospital itself. Daily rations are drawn by the various hospital departments and the pharmacy is stocked to work under seige or adverse conditions without restocking for a predetermined period of time. A dispensing station should be set up in the triage area to function during the reception of casualties. This station also dispenses equipment and drugs to the corpsmen for the treatment of casualties.

Administration and Nonmedical Personnel

The administrative component of an evacuation hospital provides all the logistic support for the efficient running of the hospital. In addition to cooks, technicians, guards, drivers, mechanics, etc., the hospital needs some specialized personnel. It requires electricians, generator operators, airconditioning experts, oxygen handlers, electronics experts, and technicians versed in the care of respiratory, X-ray, and anesthesia machines. Since communications with other forward and rear medical facilties and with headquarters are extremely important, there should be an adequate number of communications experts to man the wireless and telephone facilities. The adminstrative staff also double as stretcher bearers and traffic controllers during periods of intense medical activity. They take charge of the helipad and transport within the hospital and to and from it. The administrative facilities and stores should be set alongside the hospital but not too close so as to separate the traffic and ease congestion.

Medical and Paramedical Personnel

The size of the medical staff is of course related to the size of the hospital and its intended activity. Field hospitals require very specialized medical personnel. There should be well-trained operating room nurses, intensive care nurses, emergency room nurses, and a large number of corpsmen trained for service in a field hospital. When the hospital is bigger there is a need for physiotherapists, psychologists, medical recorders, etc. The organization of personnel is of prime importance because the hospital is thrown into periods of peak activity when casualties are brought. in. The modern battlefield or disaster area may preclude the orderly evacuation of casualties from the field; they are usually evacuated during fulls in intense activity and therefore tend to arrive at the hospital in waves. The arrival of large numbers of casualties at once requires the full complement of personnel to handle the impact of this large influx. Therefore, all personnel should be assigned to the triage areas except for a small number who maintain and run the hospital's other facilities.

The medical personnel are divided into three identical teams which rotate in 8-h shifts except for those occasions when the casualty influx is so high that the entire resources must be committed simultaneously. Each team is headed by a senior surgeon who also acts as the chief sorting officer. Each team comprises three anesthetists, a chest or vascular surgeon, a urologist, an orthopedic surgeon, two senior surgons, and an additional specialist acting as junior surgeon for the group. A neurosurgeon, an ear nose and throat

surgeon, and an ophthalmologist are on call for consultation when needed. The chief surgeon, in his role as chief sorting officer, is in sole charge of the initial triage of casualties. He plans the division of work and methods, and makes final decisions regarding priorities of surgery and evacuation.

Principles of Operation

Evacuation is never as urgent as resuscitation: evacuation takes place after hemodynamic stabilization, and only after the initiation of all required resuscitative measures. The stabilized wounded tolerate transportation over long distance, thus eliminating the need to set surgical hospitals close to the front.

Under the extreme conditions of war or disasters, appropriate resuscitation reduces the urgency of evacuation. In order to obtain the optimal results evacuation should be commanded, controlled, and coordinated by medical headquarters, which should preferably be situated at the evacuation hospital, so that the data concerning battle casualties and their treatment are available in real time.

In a disaster, the nearest hospital becomes the triage center or evacuation hospital according to the same principles which apply in the military context. In a civilian hospital operating as an evacuation hospital, only a small percentage of the casualties/victims should be operated upon; any operation that can be avoided should be avoided, and every operation that can be delayed should be delayed.

In special cases, a mobile surgical unit — military or civilian — can reinforce a regional hospital and transform it into a triage center or evacuation hospital, making possible the sorting, resuscitation, and supportive treatment necessary to prepare casualties for transportation.

1.8 Aeromedical Services

U. Y. Dreyfuss

Introduction

The purposes of the aeromedical service (AMS) are: The rapid despatch of emergency medical services to the scene of injury, thereby shortening the therapy-free interval to a minimum, and aerial transportation of the injured under close medical supervision to a suitable medical facility. There are two types of (AMS) operations: stand-by operations (launched immediately following an accident and flown from the scene of injury to a hospital) and pre-scheduled operations (where patients are flown between hospitals). Stand by AMS operations include scene missions (SM) and air rescue (AR). Pre-scheduled AMD operations include interhospital transfers (HT) and repatriation flights (RF).
The advantages of aeromedical services are those of flight as compared with ground transportation: casualties are under medical supervision, expeditiously overflying natural obstacles like forests, lakes, mountains, hostile terrain, and urban obstacles such as narrow streets and traffic jams. The limitations are: bad weather, high cost, and the difficulty of remaining operational at night (unless aircrafts and aircrews capable of operating at night are available.).

Scene Missions. An AMS team and its equipment is flown to the scene of injury. There casualties are evaluated and receive primary treatment, including resuscitation and advanced life support services. Thereafter, the casualties are transported by air to a medical facility, under the supervision of the AMS team. SM are flown by helicopter.

Air Rescue. For persons injured in remote or hostile areas which cannot be reached by conventional first-aid teams, AR is required. The need to receive primary treatment at the scene of injury is compounded by the need to be extracted from those areas. AR is performed by specialists, who, in addition to expertise in on-site primary medical care, have mastered AR techniques. Most AR missions are flown by helicopters.

Interhospital Transfer. Hospitalized patients may sometimes need to be further transferred; from the scene of injury, a patient might be transported first to a nearby hospital. This hospital may not be able to cope with some of the patient's specific problems (neurosurgical, vascular, burns, unavailability of intensive care, etc.). Transfer to another hospital where such services are available is then indicated. Most HT are flown by helicopters, although if the flight involves long distances, fixed-wing aircraft may be preferred.

Repatriation Flights. Injury or disease may strike far from home. In a foreign country patients may find themselves in a hospital where medical standards differ from those of their home country, where an unfamiliar language is spoken, or they may lack financial cover for the long-term treatment required. Patients may therefore wish to be transferred to a medical facility in their home country.

A hospital near the scene of war or disaster situations may quickly become overloaded. Many patients will soon be fit for transportation to other medical facilities, further away from the scene of events. Aircraft flying personnel and supplies into the area can be used for outgoing aeromedical transportation. Usually, RF are flown by fixed-wing aircraft, although large helicopters might serve the same purpose if the transportation distance is relatively short (up to 100 km).

Organization and Operation

Personnel

AMS personnel are trained to provide primary medical care under field conditions and during flight. The team must be familiar with the primary diagnosis and treatment of all kinds of injuries and acute diseases, and master many medical, technical, and operational skills:

1. Endotracheal intubation (oral and nasal)
2. Intercostal drainage
3. Peripheral vein cannulation
4. Central vein cannulation (subclavian, internal jugular, femoral)
5. Catheterization of the bladder
6. Cricothyriotomy
7. Cardiopulmonary resuscitation and advanced life support
8. Dressing of all types of wounds
9. Splinting of all types of fractues and dislocations (with special emphasis on spinal fractures)
10. Aquaintance with the type and action of all drugs included in the AMS kit
11. Technical skills: (a) placing of patients on stretchers, (b) loading and unloading of stretchers on and off the evacuation aircraft.

12. Operational skills: (a) use of the aircraft communication system, (b) acquaintance with all safety and emergency procedures of the aircraft, (c) AMS teams operating with helicopters equipped with a winch must be familiar with down- and up-winching techniques.

Two members are the minimum requirement for an aeromedical service team. AMS missions are usually initiated for the severely injured and diseased; on-scene work must be efficient and quick. Therefore, team work is of the utmost importance. Single-handed performance of emergency procedures is very difficult indeed. Physicians, nurses and paramedical technicians can comprise AMS teams. A combination of any two of these disciplines is acceptable. Including a physician is of advantage since he is more capable of making decisions on the spot and also of executing them. Nurses with appropriate training and experience in emergency medicine also function well as the leading figure of the team in decision-making and carrying out emergency procedures.
Treating patients at the scene of injury might be difficult. The terrain could be rough. Crowds will gather and often disturb the AMS team at its work. Many AMS missions are done at night when poor illumination might cause problems. Bad weather can adversely affect the work at the scene of injury.

Equipment

Equipment for AMS should be portable. Equipment and drugs must be in sizes and dosages suitable for the treatment of children as well as adults. Equipment should be reevaluated once a year, and nonessential items discarded. If the AMS team operates with medically designated aircraft ("Medicopters"), some of the equipment can be housed permanently on the aircraft, and electrical appliances can be connected to its electrical system. With such a mode of operation, a medical bag and a hand-operated suction device are all that are needed outside the aircraft. If the aircraft is of the general purpose type, the equipment must be limited by size and weight, so as to be portable by the team of two.
A further consideration is the radius of operation. For missions of the "scoop and go" type, with flying time to the medical facility of less than 15 min, only limited equipment is necessary. If the service is designated for long-range aeromedical transportation, more items are necessary, since more comprehensive treatment is indicated at the scene of injury and during flight.
The capability of the medical team as well as the carrying capacity of the aircraft are further factors to be considered: a medical team of two can provide primary medical treatment simultaneously to no more than two severely injured individuals or to three moderately severe casualties. Small helicopters (Bo-105, Bo-117, Bell 206) can carry only 2 litter patients. Me-

dium-sized helicopters (Bell 205, Bell 212) will carry up to 6 patients on litters. Large helicopters and cargo aircraft have a carrying capacity exceeding 15 patients on litters but will usually be operated by more than one medical team. Therefore, an appropriate medical kit should exceed neither the working capacity of the medical team nor the carrying capacity of the aircraft.
The equipment for a two-man AMS kit should contain the items detailed:

1. Set for endotracheal intubation (laryngoscope, tubes of various sizes, including sizes for children)
2. Set for intercostal drainage
3. Set for the cannulation of peripheral and central veins
4. Surgical set for minor operations
5. Set for infusion of IV fluids
6. Dressing material in various sizes (including dressing material for burns)
7. Splints for immobilization of the upper and lower extremities (for AMS missions flown in helicopter and pressurized fixed-wing aircrafts, inflatable soft splints are more suitable than hard ones, since they occupy less space when not in use)
8. Ventilator (Ambo bag and mask or other portable mechanical ventilator) and oxygen cylinders
9. Cardiac monitor-defibrillator
10. Suction machine (operated by battery as well as by aircraft current)
11. Drugs

The equipment should be kept in water-repellent bags. They should be easy to carry and the weight of each bag should be limited to what one person can carry comfortably.
Personal and safety equipment should include: overall, boots, flying jacket, safety helmet with built-in earphones and life-vest (if operating over sea).

Operational Considerations

In order to operate an efficient AMS, many functions must be coordinated. This will be done through a dispatch center (DC) which is usually located at or near the AMS base. The location of the base should be either in a major hospital (if the service is designated to serve primarily this hospital) or in the geographical center of the area to be served. (An airport where technical maintenance services can easily be provided is a good location.)
A DC is responsible for coordinating the following functions:

1. Emergency services in the area (police, amublance service, fire brigade, etc.); often they are the first responders at the scene of injury and alert the aeromedical service

2. Main functions in the hospitals (emergency room, operation room, etc.)
3. Assure at all times the availability of the medical crew, aircrew and maintenance service for the immediate execution of missions
4. Keep a service logbook on all incoming calls and executed missions
5. Receive mission reports and maintain a follow-up service on the patients treated and flown by the service

The availability of the medical, air, and maintenance crews on duty must be such as to allow for take-off within 5–7 min from the moment an emergency call is received at the DC. This is best assured by providing each member with a paging device. The aeromedical team uses a standard report form to describe flying to the scene and back, pick-up location, and name of hospital of delivery.

Interhospital transfers and repatriation flights usually involve longer flights. The advantage of such missions is that they can be planned in advance, and all the necessary precautions can be taken. It is of utmost importance to get all the necessary medical information about the patients who are going to be transferred. Pick-up and delivery times should be fixed in advance, so that both the sending and receiving medical facilities can get ready. At the receiving facility the patient should be handed over to a medically competent person (usually a physician) who will also receive all accompanying documents. The name and function of this person should be registered on the AMS report form.

The medical requirements of air rescue and scene missions are equal; however, in a rescue mission the medical team or rescue specialist will first have to reach the patients, evaluate them, and, if necessary, perform life-saving procedures prior to extraction. Following extraction, further scene first aid will be given.

Usually, it is very difficult to administer treatment during flight. This means that the entire treatment necessary for the safe transportation of the patients to a medical facility has to be completed beforehand. Life-saving procedures, like keeping a free airway, assuring efficient ventilation and circulation must always precede transportation. Wound dressing and splinting of fractures should also be completed prior to take off. The decision whether to perform other procedures at the scene of injury depends on how long these procedures take as compared with the time necessary to deliver the patient to a hospital. If the flight time is shorter than the estimated time necessary to complete the treatment, the patient should be flown to the hospital where it can be done under more favorable conditions.

Following the AMS mission, a detailed report should be filed on the person transported:

1. Name, age, sex, ID number, address, phone number
2. Circumstances of injury/disease
3. Type of injury/disease

4. Detailed information on the treatment at the scene of injury and during the flight
5. Delivered at what hospital, and to whom?
6. Type of aircraft
7. Names of AMS team (physician, nurse, PMT, pilots)
8. Data on flying time (give details date and time) for base to pick-up zone, time spent at pick-up zone, pick-up zone to hospital, time spent at hospital, and hospital to base.

Aeromedical Services in Disaster Situations

The main difference in disaster AMS as compared with routine AMS is in the number of casualties that will potentially need AMS. These numbers may exceed by far the available AMS resources. Many of the casualties who normally would qualify for aeromedical evacuation, will not be able to receive this service because of its limited availability. This situation will necessitate a very thorough triage system at the scene of events. A senior medical person, preferably with extensive experience in trauma, must be designated for the position of a "scene medical coordinator". His responsibility will be to evaluate all casualties and decide upon priorities in evacuation:

1. Top priority for evacuation by air will be reserved to severely injured persons who, if brought expeditiously to a hospital capable of treating their injuries, have a good chance of survival. Saving a limb or sight are also an indication for first-priority evacuation.
2. Severely injured persons with a poor chance of survival will be kept in the area until all the casualites mentioned in 1 have been evacuated, or until further means of transportation have been made available.
3. Casualties with mild or moderate severe injuries will remain in the area until all the more severely affected ones have been evacuated. Later they will be transported, usually by ground transportation, to nearby medical facilities.
4. The medical coordinator must be familiar with all the medical facilities inside and outside the area of disaster (size and professional capabilities). This will enable him to distribute casualties in an optimal way, so that nearby hospitals will not become overloaded, while the farther ones remain relatively idle. The medical coordinator will order the evacuation of casualties with special problems (craniocerebral injuries, major vascular injuries, burns, etc.) to a hospital capable of dealing with such problems.
5. Local medical and general supply resources may be used up quickly and limit the efficiency of the AMS, or even cause it to cease functioning. All will then depend on early relief from outside the area of disaster.

War Aeromedical Services

Wars cause both military and civilian casualties. Modern armies operate AMS for its injured soldiers. Unfortunately, the number of casualties amongst civilians may exceed manyfold those of the military personnel. Whether military resources will be available for civil casualties is doubtful. It is further unknown whether civil AMS will be able to continue to serve the civil sector during war, since all helicopters and transport planes are highly vulnerable in war situations. Air superiority is an absolute prerequisite for the safe operation of any aerial evacuation service. From a purely medical point of view and presuming that there is no shortage in medical supplies, AMS can continue to operate during a war, in a manner similar to that of peacetime.

Clinical Considerations

The treatment given by an AMS is influenced by the type of aircraft (helicopter or fixed-wing airplane), flight altitude, and duration of the flight. Helicopters usually fly at low altitude (300–600 m). At such altitudes pathophysiology due to the physical behavior of gases under reduced atmospheric pressure can be disregarded. Difficulties and problems in treating patients in helicopters result from the noise, vibration, dust, and space restriction.

Noise. Inside a helicopter the noise may reach 100 dB. At such a level it becomes difficult for the team to communicate with the patient or among themselves. Auscultation or blood-pressure measurements are virtually impossible. The noise is unpleasant for the patient and may increase apprehension and anxiety, especially in those who have never flown. Earphones or earplugs are mandatory.

Vibration. A helicopter engine generates vibrations of low frequency (20–40 Hz). This sense of vibration can be disturbing for the patient and has an accumulating effect of fatigue during long flights.

Dust. Rotor wash raises dust around the aircraft during landing and take off. To avoid penetration of the dust into airways (especially when the casualty is intubated or has a tracheostomy) and wounds, the patients should be kept well away from the landing site of the helicopter until the rotor has come to a stop. All wounds should be dressed and the patient covered with a blanket prior to boarding. The blanket must be attached well to the litter, to avoid it being blown away by the rotor wash.

Restricted Space. In most helicopters used for the AMS, the cabin space is very limited. The performance of therapeutic procedures in-flight is therefore awkward. If at all possible, all primary care procedures should be completed prior to aerial evacuation.

The clinical aspects of evacuating patients in nonpressurized fixed-wing aircrafts are influenced by two physical principles, Boyle's law and Dalton's law.

Boyle's law states that the volume of gas is inversely proportional to its pressure (temperature remaining constant). This means that as altitude increases, gas expands. The clinical consequence of this law is that any gas enclosed in a body cavity will expand with altitude, which has several pertinent clinical consequences. As a rule, patients who, during surgery, have had air introduced into enclosed spaces should not be evacuated by air during the first postoperative week.

If aerial evacuation is absolutely essential, the following patients should be flown at the lowest safe flying altitude: (a) patients after intestinal surgery, where entrapped air in the gut might rupture an anastomosis; (b) patients after intracranial surgery, or with a fractured base of the skull, where air might have entered into the cranium; and (c) patients after intraocular surgery.

Patients after intrathoracic surgery or suffering from a pneumothorax can be transported safely by air, provided that a chest drain has been inserted and connected to a Heimlich valve (or a simple underwater trap bottle if the special valves are not available). Prior to the flight, it is vital to ascertain that the chest drain is functioning properly.

Prior to aerial evacuation, retention balloons of endotracheal tubes and Foley catheters should be filled with saline solution instead of air, to avoid uncontrolled expansion or explosion of the balloon.

For the administration of infusions only plastic bags should be used (using glass bottles, the expanding air will increase the pressure in the bottle and by so doing will accelerate the rate of infusion).

Dalton's law states that each gas in a mixture of gases behaves as if it alone occupied the total volume and exerts a pressure (the partial pressure) independent of the other gases present. The sum of the partial pressures of individual gases is equal to the total pressure. Thus, patients with severe anemia (Hb $<$ 7 mg/100 ml), who might be well compensated at sea level, become hypoxic at a flying altitude which would normally be well tolerated (up to 3000 m). Anemic patients should therefore have oxygen supplemented during flight. Patients suffering from pulmonary or cardiac insufficiency should also have oxygen supplemented during flight. Any patients with a head injury will tend to develop cerebral edema, causing compression of intracerebral vessels which leads to cerebral hypoxia. Consequently, oxygen should be supplemented during flight. Hyperventilation is also indicated since, by causing hypocapnia, cerebral edema is reduced.

Most fixed-wing aircraft used nowadays for AMS are pressurized to a flight

altitude of 1500–2700 m. Under such conditions the clinical problems mentioned above rarely manifest themselves. However, the transport of patients in such aircraft raises other problems.

Dry Air. The humidity of the air in a pressurized aircraft is very low (< 1 U%). Intubated or tracheostomized patients must then be provided with a means to humidify the inspired air. The danger of corneal damage in comatose patients is increased. Some eye ointment should therefore be introduced prior to the evacuation of comatose patients in a pressurized aircraft.

Duration of Flight. Many repatriation flights may be of long duration. Thus, patients may have additional needs such as routine medication (insulin, digoxin, etc.). Dressings and bedding may have to be changed en route, and the appropriate material must be available.

Motion Sickness. Anybody in flight may develop motion sickness due to bad weather conditions or to personal susceptibility. Aspiration of vomitus, especially in semiconscious patients, is the main danger. These patients must be watched carefully, and an efficient suction device must be available at all times. It is further recommended that such patients be intubated prior to evacuation.
Similarly, a patient whose jaws have been wired for a fracture of the mandible cannot expel vomitus unless the intermaxillary wires are released. Such a patient should never be transported without first making sure that a cutter for the quick separation of the wires is available.

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Part 2

Mechanisms/Causes of Injury: Pathophysiology and Management

2.1 Missile Wounds

N. D. Reis

Introduction

Knowledge of the physical events caused by the penetration of a missile into the body leads to an understanding of the appropriate treatment. The damage caused by a bullet or shrapnel striking the body depends on: (a) velocity; (b) the physical properties of the tissues; (c) size, weight and shape; and (d) stability.

Velocity. High-velocity bullets are fired from modern military rifles; they travel at 800–1000 m/s. Hand guns fire low-velocity bullets (around 200 m/s). Bullets may have various motions during flight depending on their velocity, size, shape, or stability, and the properties of the gun barrel.
The kinetic energy (K) of a missile in flight is half the mass (m) multiplied by the square of the velocity (V):

$$K = \frac{mV^2}{2}$$

It is the transfer of this kinetic energy to the tissues of the body which causes the damage, velocity being a paramount factor. The amount of this destructive energy expended when a missile strikes the body is expressed in the formula:

$$K = \frac{m(V_1^2 - V_2^2)}{2g}$$

where V_1 is the initial velocity, V_2 the final velocity, and g the acceleration due to gravity.
The Russian AK-47 (Kalachnikov) rifle fires a 7.62-mm bullet which has more than twice the weight of the American M16 5.56-mm (3.5g) bullet, but their destructive effects on tissue are similar owing to the latter's greater velocity (1000 m/s).

Physical Properties of the Tissues. The extent of damage done in tissues is related to the rate and amount of energy given up by the missile; thus the passage through soft tissues (which behave like a fluid) deprives the missile of less energy at a lesser rate than when a bone is struck. Tissue damage is also increased by yaw and tumble, these being promoted when a bullet meets with the resistance of the tissues: the longer the missile tract and the greater the density of the tissue, the greater the tendency to increased yaw and its conversion to tumble.

Size, Weight, and Shape. Shrapnel from the explosion of shells, mines, rockets, etc. varies in size, shape, mass, and velocity and may therefore behave as a high- or low-velocity missile. Secondary missiles (objects which have become dislodged and themselves turned into missiles by the primary missile or explosion, e.g., rocks, building materials, metallic fragments inside armoured vehicles, clothing) have a low velocity but may inflict grievous wounds owing to their size, mass, and ragged shape, tearing and crushing the tissues.

Missile Stability. A bullet in flight is spin-stabilized. Forces acting upon it change the profile of the impact surface and the type of movement of the bullet along its trajectory. Yaw is a deviation of the long axis of the bullet from its line of flight. Tumbling is a rotation around a transverse axis so that the front and back of the bullet face the flight path alternately.
Spin can give rise to two deviants: precession (a circular yawing shaped like a spiral) and nutation (a movement in circles forming a rosette). A stable bullet may well cause a through and through wound giving up less than 20% of its energy (causing relatively limited destruction); however, the same bullet when unstable may dissipate 70% of its energy, causing a severe wound.

Mechanism of Wounding in the Tissues

Cleaving, Tearing and Contusion. Low-velocity missiles (hand-gun bullets, knives, bayonets) forcibly penetrate and separate the tissues, contusing, crushing, and tearing only those tissues which form the intimate sleeve of the missile path. Energy is not dissipated around the wound tract; consequently, near-by structures are not damaged. There is no additional "occult" tissue damage.

Explosion. A high energy missile striking the body creates two additional wounding mechanisms which are in fact an explosion within the body.
(a) Shock wave: the entering missile drives a circular shock wave before it for about one-millionth of a second. For that fraction of a second tissue pressures may rise to 100 atm. The shock wave travels at around 1500 m/s

and is conducted along bones and blood vessels, damaging tissues at a distance.
(b) Cavitation: As the tissues are propelled violently forwards and sideways, a cavity 40 times the size of the missile's diameter is blown up, reaching its ultimate size after the passage of the bullet. Now, having developed a negative pressure, the cavity collapses, sucking in dirt, foreign bodies, and bacteria from both the entry and exit wounds. These contaminants are retained within the permanent cavity observed at surgical exploration.
"Explosive" cavitation is compounded by shattered bone which behaves like a secondary missile. Solid, homogeneous, dense, friable tissue such as liver, spleen, brain, and muscle contain few or no elastic fibers and are extremely susceptible to damage by cavitation, whilst elastic, tougher tissues such as lung and skin are more resistant.
Wounds in which the shock wave and cavitation phenomena have taken place are those in which "unseen, hidden" trauma is present, i.e., there is a more massive and extensive tissue death around the missile tract than is immediately apparent. Remote damage to the intima of a major blood vessel, to a nerve, or to the spinal cord is made possible by these mechanisms.

Distribution of Missile Wounds in War

Recent conflicts have shown that over 60% of missile wounds occur in the extremities. About 15% are in the thorax and abdomen and 15% in the head and neck (including the eyes). Some 30% are multiple missile wounds affecting various parts of the body.

Prevention

Steel helmets, armored vests, and protective glasses are effective in reducing the number of injuries and often convert a potential fatality into a treatable casualty.

Clinical Treatment

Experiments have shown that a high energy missile wound contains a volume of dead tissue of around 500 cc. A constant awareness of the widespread destruction which may well underlie a relatively innocent-looking

external wound together with the knowledge that remote injuries may present themselves is the basis of the treatment protocol: wide, and repeated excision of wounds to achieve as complete a removal as possible of devitalized tissues and foreign bodies. Assume all wounds are contaminated by bacteria. Assure free drainage, and then close the wound only after it is certain that no dead tissues remain and never sooner than the 5th day.

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2.2 Blast Injuries

M. Michaelson and N. D. Reis

Basic Physics

The detonation of explosives creates an expanding volume of heated gas which gives rise to a supersonic wave of pressure which causes a shock wave. This shock wave has three phases:

1. The Positive Phase. At detonation an instantaneous rise to very high pressures occurs (1000 atm) for a very short period (5–10 ms). This creates a shock wave. Damage is proportional to the peak pressure and its duration. The blast moves away from the center of the explosion as an expanding ball of hot gas which may reach a velocity of 3000 ms but quickly falls to the speed of sound at a variable distance.
Pressures of thousands of psi may be recorded at the center, falling off with distance (by the cube of the distance from the center).
The shock wave behaves like sound, so it can pass through an obstructive barrier, reflect from it, and/or flow all around it, injuring persons sheltering behind it. A reflected wave may intensify its damage effects. The positive phase is responsible for the primary blast injury.

2. The Negative Phase. This phase is 10 times longer but is of a much lesser magnitude than the positive phase. It is caused by the suction force which follows immediately upon the blast wave. It is not associated with primary injury to the body.

3. Movement of Air. Expanding air follows the shock pressure wave. This is called "the blast wind" and can reach a velocity of 742 kmph (460 mph). This phase, together with the missiles, is responsible for the secondary and tertiary injuries.
Injuries from a blast are divided into three categories:

1. *Primary* blast injury which is caused directly by the impaction of the positive phase pressure wave on the body.
2. *Secondary* blast injury which is caused by flying objects (missiles) such as glass, wood, and parts of the shell or bomb.
3. *Tertiary* blast injury is sustained when the victim is thrown and injured by collision with stationary objects.

(The two last categories are dealt with elsewhere since they are not unique to blast injury.)

Density of the *environment* in which the explosion takes place determines which category of blast injury predominates. In *underwater blast,* primary blast injury tends to be greater: the shock wave travels much more quickly in water, and thus the lethal radius of blast in water is about 10 times that in air. The high density of water, which is similar to body density, practically cancels out the secondary and tertiary effects of blast but increases the severity of the primary blast injury. Because of this similar density, the body absorbs the shock wave in water, while in air, part of the energy is reflected.

The underwater phenomenon similar to the blast "hurricane" in air is the water ram. Unlike explosions in air, the forcible displacement of water powerful enough to cause injury occurs only in close proximity to the explosion.

In air, a blast shock wave having a short wavelength (high-pitched bang) is potentially more injurious than one of long wavelength (low-pitched, booming bang).

The eardrum ruptures at about 7 psi above atmospheric pressure whilst lung and abdominal injuries are caused at over 30 psi above atmospheric pressure.

Even though a victim may escape major injuries from the blast shock wave, the blast "hurricane" may fling him through the air for a considerable distance onto the ground or against any other obstructing object. In air therefore, there is a greater danger of injury by secondary and tertiary effect, i.e., flying objects and danger of sustaining a severe blunt injury.

In solid blast, the blast pressure wave is transmitted directly from a solid object in contact with some part of the body (the wall of the tank, the deck of the ship). Such pressure waves passing through the body disrupt blood vessels, lacerate internal organs, or cause fractures away from the source of contact.

Pathophysiology

Three physical phenomena related to the blast wave may cause damage to the living body:

1. *Spallation* is the tendency of the surface between two media of different density to disrupt when a shock wave is transferred from the denser medium to the medium which is more compressible. An example is the water spout that is created by an underwater explosion.
2. *Implosion* is the compression of gas bubbles in liquid media. These bubbles absorb energy and, after the shock wave passes, re-expand to a

greater diameter and act as multicenter explosions. The same is true for any gas-containing organ.

3. *Inertial effects.* Due to the differences in acceleration caused by the shock wave between two different organs of different density, tears of the adjoining pedicles may occur. Consequently, the body sites most likely to be injured by blast are the gas-tissue interfaces. The organs which are most susceptible to blast injury are: auditory system, lung, and intra-abdominal viscera.

Localization of Blast Injury

1. Auditory system. The eardrum is the most susceptible organ to injury by blast in air. The eardrum ruptures at a much lower pressure (5–10 psi) than other organs. Therefore the absence of an injury to an unprotected ear in blast trauma in air practically rules out primary blast injury (this is not true for underwater blast!).
In the ear the blast wave may cause:

- rupture of the tympanic membrane
- dislocation of the ossicles
- damage to the inner ear

2. Lungs. Blasts of above 30 psi may cause tearing of the alveolar parenchyma, producing hemorrhage and edema into the interstitial and intra-alveolar space. Such lung lacerations can cause hemothorax, pneumothorax, pneumomediastinum, and air embolism.

3. Intra-abdominal Viscera. The gas-containing organs (especially the large bowel) may be damaged by the blast wave: the damage ranges from serosal hemorrhages to multiple perforations of hollow organs and lacerations of solid organs. Late perforation and ruptures may occur days after injury. Abdominal and lung injuries are more frequent in underwater blast than in air blast.

4. Central Nervous System. Brain injury is caused by air embolism or is secondary to a gross elevation of the venous pressure as a result of blast to the chest and abdomen. There is no direct damage to the brain from the blast (see Air Embolism).

Diagnosis (Fig. 1)

Pitfalls

1. Primary blast injuries are difficult to diagnose because they may be accompanied by severe secondary and tertiary injuries which are in them-

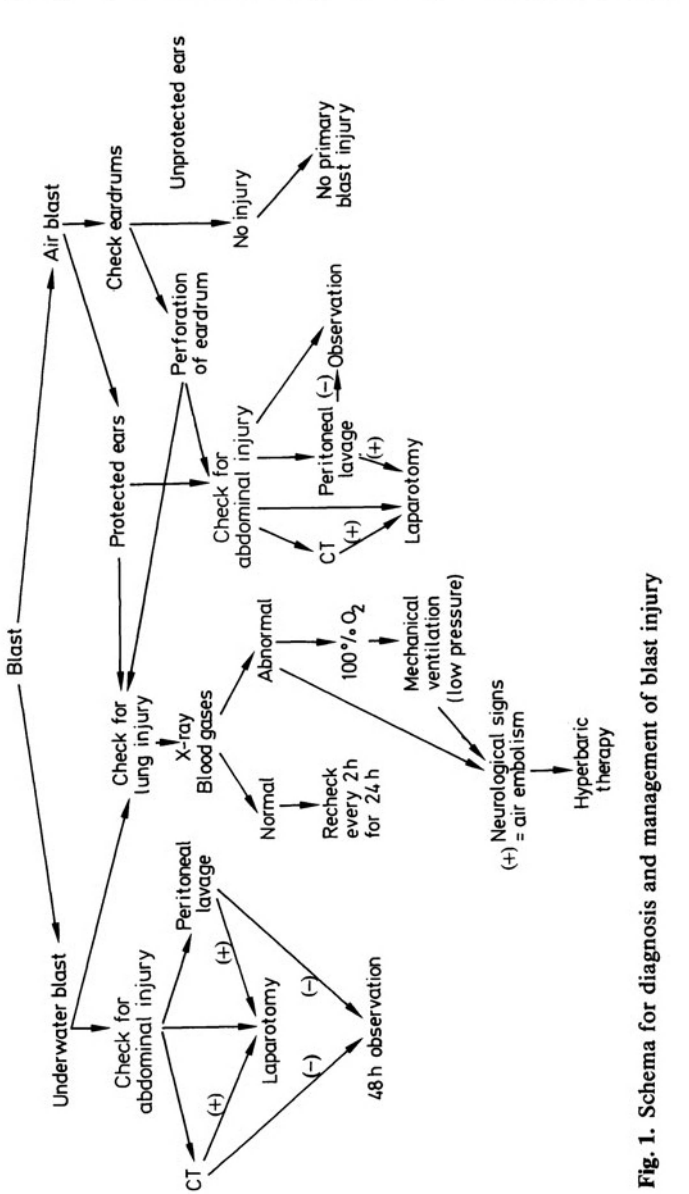

Fig. 1. Schema for diagnosis and management of blast injury

selves life threatening: the signs of primary blast injuries tend to be overlooked.
2. External signs of injury may be absent.
3. The signs and symptoms may present late.
4. The casualty may be restless, fearful, and present with a tremor: exclude blast injury before labelling him as "battle fatigue."

Signs and Symptoms

1. Auditory system. Sudden, transient, painless deafness lasting some minutes is the usual symptom. This is often followed by pain, dizziness, tinnitus, and minor bleeding from the external auditory canal. Diagnosis is confirmed by otoscopy, which reveals a tear of the tympanic membrane.
2. Lungs. Chest pain, epigastric pain, restlessness, tachycardia, cough, blood-stained sputum, shortness of breath, and shock occur in a variety of combinations.

Local signs:

- Physical signs of pneumomothorax or hemothorax
- Fine crepitations

Laboratory findings:

- Blood gases: hypoxemia
- X-ray: Pneumothorax, hemothorax, mottled lung field as in adult respiratory distress syndrome
- Pneumomediastinum
- Interstitial emphysema

Note: The signs and symptoms of blast injury to the lung may develop hours (or days) after the injury!

3. Abdomen. The surviving victim complains of having been "kicked in his belly" — the pain is acute, and he feels "winded." Early signs: abdominal pain, tenderness, rigidity and rebound, urge to defecate, acute abdomen, nausea, vomiting, rectal bleeding. Late signs: onset of peritonitis indicates late perforation. X-ray of abdomen reveals free air in the peritoneal cavity. Peritoneal lavage — bloody tap.
(CT of abdomen if available).
4. Central nervous system.
Signs range from minor mental changes to deep coma: focal neurological findings. Diagnosis requires brain CT scan for hemorrhage.

Treatment (see Fig. 1)

On Site and Medical Aid Station

Every victim who was in the water during an underwater blast is treated as suffering from blast injury. Every casualty wounded by shells, mines, or hand grenades should be checked for blast injuries. First treat life-threatening, secondary and tertiary injuries.
Check the eardrums: Intact eardrums, in unprotected ears, in air rules out primary blast injury. If the eardrums are torn, no treatment should be given. Avoid all local applications.
With lungs, treat pneumo- and hemothorax. A chest tube should be inserted the moment diagnosis is made (see Lung Injury), and 100% oxygen should be administered by mask on suspicion of lung involvement. Intubation and ventilation with positive pressure should be avoided for as long as possible to prevent air embolism. The victim should be recumbent on a stretcher. A nasogastric tube should be inserted and a large IV line started on suspicion of abdominal involvement. Shock should be treated along the lines described elsewhere (see Shock).

Hospital

Ear injuries are treated conservatively and usually heal by themselves.
Upon arrival at the hospital the abdomen should be investigated. X-rays may show free air in the peritoneal cavity. If the patient is hemodynamically stable a CT of the abdomen or a peritoneal lavage should be performed. This should be followed by laparotomy. Patients with suspected abdominal blast trauma should be admitted and followed up for at least 48 h.
The most difficult injury to treat is a lung injury, excluding pneumothorax and hemothorax. An X-ray of the chest and analysis of blood gases should be done immediately upon arrival. These are the two tools for diagnosis and follow-up of the chest injury. Hypoxia should be treated with 100% oxygen. If this is not successful, intubation and mechanical ventilation are begun at the lowest possible pressure to avoid massive air emboli. Otherwise this lung injury is treated as an acute respiratory distress syndrome (see Lung Injury).
On the suspicion of air embolism the patient should receive 100% O_2 and be treated in a hyperbaric oxygen chamber. This is an emergency, and no time should be wasted.

Prevention

- Use earplugs or earpads
- Take cover whenever possible behind solid object or, better, get underground

- In water, float on your back
- Wear padded, protective clothing

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2.3 Pathophysiology of Burn Injuries

R. Shafir and J. Weiss

Introduction

A burn is the disruption of the integrity of the skin covering the body. This can be caused by exposure to thermal changes, such as heat or cold, chemicals (acids, alkalines, phosphorus, etc.), and electric current. The degree of the disruption is determined mainly by the amplitude of the impact, the length of its action, the thickness of the skin, and the percentage of affected body surface area. This chapter is devoted to the most frequently encountered burn, the heat burn.

When affecting only 10%–20% of the body surface area (BSA) it will cause no problem beyond the localized skin lesion. However, when large surface areas are burned a severe disease — the burn syndrome — develops, initiating a process of disruption of cellular, metabolic, thermoregulatory, hemodynamic, and immunologic activities.

The immediate pathologic processes caused by to the damage to the skin, its cells, and microvasculature are hyperpermeability, edema, and shock. Later pathologic processes are initiated by the exposure of deep tissues to the environment. The skin is a barrier protecting deeper tissues from infection, dehydration, and heat-energy loss. When large areas of it are damaged, a two-way passage is opened which leads to total body and specific organ failure.

Morphologic Changes in the Skin

In skin damaged by heat, three zones can be recognized. The most superficial (nearest to the impact source) is the zone of coagulation, in which capillary flow immediately ceases and the cells are completely destroyed. No recovery is possible. Deeper and more peripheral, is the zone of early stasis, in which the microvasculature dilates and leakage causes edema formation. Within minutes and up to 3–4 h the vessels become clogged by microthrombi and red cell aggregates. No epidermal cells survive in this area, but the deeply seated adnexal epithelium which serves as a source for regeneration of the epidermis remains alive.

The least severe damage occurs in the zone of delayed stasis. Most cells in this zone survive the direct injury. Here vessels leak for 4–16 h and become blocked at a later stage. The damage in this zone can be reversed by early cooling; this should be initiated instantaneously and remains partially effective even if started within the first postburn hour. It is essential to prevent desiccation of this partially damaged zone by the use of temporary biologic or artificial coverage, thus preventing the death of the zone of delayed stasis and avoiding the development of a full thickness burn.

Capillary Permeability and Edema

The major problem which the burn patient faces in the early postburn phase is the development of edema and shock. These are caused by a chain of events started by the injury to the microvasculature and the stimulation of the humoral mediators of inflammation. The first mediators to appear in the damaged tissue are histamines, bradykinins, and serotonin; these are activated by abnormal proteins in injured tissues. These mediators cause the early phase of hyperpermeability of the microvasculature with immediate leakage of fluids. They also activate the phospholipase A2 which releases arachnoidic acid from the membranes. This acid is converted to biologically active products such as thromboxane in platelets, prostacyclins in endothelial cells and leukotrienes in other cells; vasoconstriction and platelet aggregation (thromboxane), vasodilation (prostacyclin) and increase in permeability (leukotriene). This is the late phase of hyperpermeability, leakage, and edema formation, which continues for about 24 h or longer.

The extensive extracellular edema in the burn patient is the result of four interacting forces: (a) increased capillary hydrostatic pressure due to vasoconstriction of venules and clogging with clumps of platelets, (b) reduction in the plasma protein concentration due to leakage, (c) blockage of lymphatics by cells and debris thereby reducing lymphatic flow and reabsorption of extravascular fluids, and (d) the increased porosity of the capillary endothelium due to changes in cell configuration.

These pathological processes start at the site of injury, but the activated products also affect the vasculature of tissues and organs remote from the burned surface. These include the viscera, in which the clinical expression of edema is ileus, gastric dilatation, and sometimes laryngeal edema. The leaking fluid contains water, electrolytes, and proteins, mainly of low molecular weight (albumin). Sodium loss is relatively greater than water loss possibly due to failure of the "sodium pump" and intracellular sodium retention. No drug is known to be effective in preventing or reversing the pathogenesis described.

The natural history of the severely burned goes on to the development of oligemia and dehydration due to fluid leakage: the cardiac output de-

creases, peripheral resistance increases, and the perfusion of vital organs is diminished. Failure of these organs progresses into shock and renal shutdown. Adequate fluid replacement prevents this deleterious process at this stage.

Hypermetabolism

The dominant pathophysiologic process of the next few days (which also continues until complete healing of the burn wound) is the hypermetabolic state of the patient. Fluid and heat are lost through the uncovered tissues. The loss of heat requires appropriate heat production by "burning" of body mass; this in turn leads to a catabolic state with a negative nitrogen balance. The metabolic rate is very high, not only due to heat loss but also by an endogenous reset of metabolic activity through an elevation of the set point of the "thermostat" in the hypothalamus.
Reduction of energy expenditure can be avoided by raising environmental temperature. Preventing the conversion of body mass into energy is achieved by hyperalimentation. Various hormone-secreting organs (pituitary, thyroid, adrenals) influence and react to these metabolic processes.

Immune Mechanism and Infection

Sepsis is the main cause of death in the burn patient. The infection is inflicted by the disruption of the integument, allowing invasion of microorganisms into the tissues: the burned skin, the respiratory mucosal lining destroyed by an inhalation injury, and the mucosal lining of intestine damaged by hypoperfusion due to dehydration and shock. The way is open for environmental and endogenous organisms to colonize the burn. The pathogenic endogenous organisms (which are harmless in the healthy human) become virulent due to the depletion of the cellular and humoral elements of immunity.
The normal inflammatory mechanisms are disrupted by the failure of the microcirculation (stasis and clogging prevent the delivery of the cellular components to the site of injury), phagocytosis, and local cellular immune function, and the activity of the opsonic and complement system are impaired by the hypoproteinemia. The specific immune system which usually responds to particular antigens is also severely impaired. The B- and T-lymphocytes fail to recognize the bacterial antigens, thereby delaying the phagocytic activity of the macrophages. The system is also depleted of its immunoglobulins which take part in the antibody response to infection. This again is the result of protein deficiency, hypercatabolism, and malnutrition.

Inhalation Injury

Inhalation injury ranks next to infection as the cause of death in the burn patient. Injury to the pulmonary system due to the direct heat impact is exceptional and occurs only when concentrated high temperature steam is inhaled. Dry inhaled material cools off prior to its entering the bronchial tree. Carbon monoxide inhalation does not cause pulmonary injury but generalized poisoning. The damage is due to tissue hypoxia due to CO combining with hemoglobin, thereby reducing the oxygen transport capacity of the blood. True inhalation injury is the consequence of inhaling smoke from burned organic or man-made materials. The inhaled toxic gas irritates the respiratory mucosal tract and causes pulmonary edema, mucous secretion, clogging of the bronchial tree, and atelectasis formation. In more severe injuries the mucosa sloughs off and a mucopurulent membrane develops. Inhalation injury has a more deleterious effect if occuring concomitantly with a skin burn. Edema and microthrombi formation in the respiratory tree and the direct injury to the bronchi and alveoli cause severe respiratory failure and death.

Summary

The interacting pathological processes composing the burn syndrome have been described. The role of the endocrine system, the coagulation-fibrinolysis system, and the blood cellular elements have not been described in detail for the sake of simplicity and clarity. Each is complex, and together they make burns a most complicated disease, involving the whole organism. It is therefore difficult to find specifically targeted modes of treatment that would prevent the development of insufficiencies of the different organs and systems. An understanding of burn pathophysiology to date leads to the following major aims in treatment: prevention of microcirculatory derangement and edema, preventing infection, and reversing the catabolic to an anabolic state.

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2.4 Cold Injuries

S. Brill

Introduction

Excessive body heat loss, from exposure to a cold climate without proper insulation, causes: superficial cold injuries (trench foot and frost bite) or accidental hypothermia in the severe and rarer cases.

Superficial Cold Injuries

Trench Foot

Pathophysiology

The physiological reflex of peripheral vasoconstriction evoked by exposure to ambient temperature in the range of 0°–10°C helps to maintain body core temperature. However, prolonged vasoconstriction (above 12 h) combined with increased conductive heat loss caused by wet garments results in damage to skin, blood vessels, nerves, and muscles. Aggravating factors include malnutrition and physical exhaustion.

Signs in the limb are:
- Cold and pale skin
- Feeble or absent peripheral pulses
- Reduced sensation (touch, pain, temperature, vibration)

Signs following rewarming of the limb:
- Hot, dry, reddish skin
- Formation of blisters and edema
- Severe local pain
- Focal skin necrosis

Late complications:
- Constant pain
- Pain upon exposure to cold
- Raynaud's phenomenon
- Local hyperhydrosis
- Local fibrosis limiting range of joint movement

Treatment

1. On site: First aid consists of minimizing further exposure to cold, e.g. change to dry clothing in a warm shelter when possible.
2. Medical aid station or primary medical facility: Rewarming should be started only if a second prolonged exposure to cold during evacuation to a field or base hospital can be avoided.
3. Field or base hospital: The affected limb is rewarmed by immersing it in warm water (37°–42°C) followed by oral analgesics, bed rest with the limb elevated (see also under "Frost Bite").

Frost Bite

Pathophysiology

Exposure to temperatures below freezing point leads to the formation of extra- and intracellular ice crystals and obstruction of the peripheral blood vessels, causing hypoxia. The severity and extent of the damage are directly related to the temperature and period of exposure. Because of the physiological reflex of peripheral vasoconstriction, the distal parts of the body (tip of nose, finger- and toetips, ears) are mainly affected.

Signs in affected part (before rewarming):
- Cold and pale skin
- Feeble or absent peripheral pulses
- Reduced sensation (touch, pain, temperature, vibration)

Signs following rewarming of affected limb:
Grade I — Immediately after rewarming the skin is red, hot, and dry, followed a few hours later by severe pain and edema. The edema disappears in 5–10 days, after which the skin starts to peel off.
Grade II — In addition to the damage in grade I there is a decrease in the sensation of touch, the appearance of blisters or blebs within 6–12 h followed by crust formation 10–24 days later.
Grade III — Skin necrosis and ulcerations. The healing process is longer (2 months on average) and may be complicated by infection.
Grade IV – This is the severest form, the damage extending from skin to bone. Following rewarming the skin has a purple-blue color, no sensation, and usually no edema or blisters. In about 1 month, a line of demarcation is noted on the skin, extending a few weeks later to the bone. Fingertips are most commonly affected.

Treatment

1. On site and first aid station:
   Minimize further exposure to cold:
   - Dry clothing and blankets

- Warm shelter
- Hot beverage
- Evacuation in a sheltered vehicle

Do not:

- Massage frozen limb
- Expose to open fire or hot water >43°C

2. Field hospital:
   - Attempt rewarming only if conditions ensure warm surroundings after the procedure and during evacuation
   - Rewarm affected limb by immersing it in warm water (37°–42°C) until there is a clear change in skin color
   - Separate affected fingers by sterile gauze
   - Avoid opening blisters
   - If conditions permit (clean rooms) avoid dressings and leave open
3. Base hospital:
   The definitive treatment should include:
   - Daily debridement of necrotic skin
   - Skin grafting
   - Amputation should be postponed as long as possible, until a clear demarcation line appears

Accident Hypothermia

Definition. This is a clinical state in which rectal temperature drops below 34°C following uncontrolled exposure to cold.

Diagnosis. If the patient is cold and has any of the following signs or symptoms, he is considered to have severe hypothermia:

- Depressed vital signs
- Altered level of consiciousness including slurred speech, staggering gait, decreased mental skills
- No shivering in spite of being very cold
- Rectal temperature below 32°C (90°F)

An associated significant illness or injury may be present and may have caused the development of the hypothermia.
If the patient is cold but does not have any of these signs or symptoms then he has a mild or moderate hypothermia.

Treatment

1. On site and first aid station:
   - Move the patient very gently to the nearest shelter (vehicle, building, etc.)

- Replace wet clothing with dry clothing or coverings, searching meanwhile for associated injuries
- Carefully assess the presence or absence of pulse or respirations for 1–2 min. If no pulse or respirations are found, start cardiopulmonary resuscitation (CPR) using mouth-to-mouth ventilation.There is some controversy regarding performing CPR on the very cold heart. Until information becomes available to the contrary, start CPR on all pulseless apneic victims
- In order to prevent a further decrease in core temperature, apply external warm objects to head, neck, axilla, and groin, e.g., hot water bottles or chemical heat packs (wrapped in a towel and watched carefully to avoid skin burns)
- Until examined by a physician, even if the patient is conscious, *avoid:*
  Rubbing the patient's extremities
  Giving warm beverages, alcohol, or cigarettes
  Putting the patient in a shower or bath
- Transfer to a medical facility in a heated vehicle or aircraft (18°–22°C)

2. Medical aid station and field hospital:
   - Basic treatment as indicated above
   - Treat further to the level of your ability and as your equipment, staff, and skill dictate
   - Initial attention is to ABC of CPR as needed
   - If the patient is unconscious, add:
     An endotracheal tube if warmed air or oxygen are available and after a careful neck evaluation
     Urinary bladder catheter
     Nasogastric tube if the aiway is protected
   - IV Therapy:
     a) Should be started only if warm solutions are available
     b) IVs are difficult to start in cold patients (do not delay transport and evacuation by taking a long time to start one)
     c) A central venous pressure line may be started but great care must be taken to place it in the superior vena cava and not in the right heart — due to the increased irritability of the cold heart, this could trigger off dangerous arrhythmias
     d) Cardiac monitoring - if possible
   - Active rewarming should not be started at this level

3. Base hospital:
   Evaluation:
   - Initial attention to ABC of CPR as required
   - Vital signs including rectal temperature using a special thermometer (range 0°–50°C)

- Brief physical examination: check skin temperature, level of consciousness, shivering, cardiopulmonary state, and seek associated trauma
- 12-lead electrocardiogram
- Laboratory:
  Blood (complete blood count, BUN, creatinine, electrolytes, glucose, platelets, partial thrombin time, prothrombin time, liver function tests, amylase)
  Arterial blood gases
  Urine (urinalysis, sodium, and osmolality)

Monitoring and treatment:

- Cardiopulmonary monitoring
- An IV and/or central venous pressure line (see above). Rate of infusion should be determined by patient's level of hydration and laboratory data.
- In the unconscious patient, add:
  Endotracheal/nasotracheal tube after careful neck examination
  Always ventilate with warm moist air at lowest oxygen percentage needed
  Nasogastric tube, if airway is protected
  Urinary bladder catheter

Rewarming:

- Recommended possibilities include:
  External methods (gradual spontaneous rewarming, warming blankets, mattresses, etc., tub bath)
  Internal methods (warm air inhalation/ventilation, peritoneal lavage, warm IV fluids, extracorporeal circulation by A-V shunt)
- The recommended temperature is 40°–42°C (105°–110°F) for all methods
- Regardless of the methods chosen for adding heat, the patient must be under total physiologic control in order to deal with his metabolic needs
- Tub bath is the most rapid method and requires immediate laboratory results and extremely close physiologic monitoring of blood pressure, heart rate, electrolyte and acid/base status to maintain control of the situation
- For severe hypothermia without signs of life (requiring CPR):
  Warm the core as rapidly as you can, using one or more of the methods (for example, warming mattress, warm air inhalation, and peritoneal lavage) trying to raise core temperature above 30°C (85°F)
  For severe hypothermia with life signs use your judgement utilizing one or more of the above methods

Common complications:

- Rewarming shock – rapid rewarming (tub bath) may lead to a marked drop in blood pressure due to the inability of the constricted blood

volume to fill the newly dilated blood vessels. When this occurs, rewarming should be stopped and infusion rate of liquids increased
- Severe acidosis, to pH 7 due to liver and kidney damage: administer sodium bicarbonate
- Hypoglycemia — due to increase in metabolic demands in a patient with depleted glycogen stores: administer IV glucose solution
- The "after do" phenomenon — this refers to a drop in core temperature induced by cold blood returning from the periphery due to rewarming-induced peripheral vasodilation. This can be avoided by using internal as well as external rewarming methods
- Cardiac arrhythmias:

  Usually atrial arrhythmias that convert spontaneously with rewarming

  Ventricular fibrillation in the very cold patient is treated with CPR, adding heat and after rectal temperature reaches approximately 30°C (85°F) by defibrillation

Prevention of Cold Injuries

1. Minimize heat loss by using:
   - Several layers of dry clothing
   - Water-resistant and wind-protected outer garment
   - Leather gloves with an inner wool layer
   - Water-resistant leather shoes
   - Proper head and ear coverings
2. Increase heat productions by vigorous physical activity. When sweating starts, stop activity or enable sweat evaporation by controlled garment ventilation
3. Keep ingesting hot beverages and high caloric food
4. Following prolonged exposure to cold, resting in a warm shelter (18°–22°C) is highly recommended
5. Avoid alcohol and smoking

2.5 Heatstroke and Heat Exhaustion

Y. Epstein, Y. L. Danon, and Y. Shapiro

Introduction

Human core temperature is closely regulated and kept within a narrow range: 36.5°–37.5°C at rest and up to 38.5°C during physical effort in a hot environment. In order to maintain a constant body temperature, the heat generated in the body (metabolic heat which depends on muscular work) and the heat absorbed from the surroundings (external heat load) must be dissipated. In warm environments two mechanisms participate in heat dissipation: the cardiovascular system conducts heat to the surface of the body (vasodilation) from which it is then lost to the environment by conduction and convection and the evaporation of sweat. Sweating is the main mechanism by which heat is dissipated from the skin to the environment. When heat accumulation exceeds heat dissipation, body temperature rises.
The loss of body fluids due to sweating together with a rise in body temperature may result in organ system damage unless prevented or effectively treated.

Heat-Related Injury

Dehydration

Pathophysiology. The relative contribution to heat loss of sweat evaporation and heat exchange by radiation and convection depends on environmental conditions. When the ambient temperature approaches skin temperature, sweat evaporation only dissipates heat. The rate of sweating depends on environmental conditions, clothing, and the level of physical activity. Body fluid loss by sweating can therefore vary greatly, and sweating rates as high as 1000 g/m^2 per h are common.
During physical exercise in the heat, sweat output exceeds water intake, resulting in a reduction of total body water (dehydration). Thirst is not a good index of body water requirements; therefore, ad libitum drinking often results in imcomplete fluid replacement or "voluntary dehydration."

The symptoms of dehydration are listed in Table 1. Thirst is present after incurring a water deficit of 2% of body weight but does not increase in intensity as dehydration progresses. A 4%–6% body weight water deficit is associated with anorexia, impatience, and headache, whereas 6%–10% is associated with vertigo, dyspnea, cyanosis, and spasticity. Dehydration levels of more than 10% are life-threatening. Hyperosmolarity and decreased plasma volume lessen the thermoregulatory response, resulting in an elevated core temperature.

Principles of Prevention and Treatment. Since thirst is not a reliable index of water loss, a potential victim should drink often, even though he feels no thirst. At least 300 ml should be imbibed every 30 min during physical activity in the heat. The amount of fluids to be drunk are best monitored by urine color: the darker the color, the more concentrated is the urine, indicating that more fluids should be consumed. However, hyperhydration gives no advantage during exercise in the heat except for the delay in the development of hypohydration. If mild to moderate dehydration occurs (< 5% body weight), fluids given by mouth is the treatment of choice. For higher levels of dehydration (> 6% body weight), intravenous normal saline solution is infused at a rate of 1.5–2 liters within the 1st hour; there after, infusion will be regulated according to urine output. If a subject collapses during exercise he must be treated as a heatstroke patient.

Salt Replacement. Sweat is ordinarily hypotonic relative to plasma (sodium concentration of 20–30 mEq/liter). Therefore, in dehydration the plasma will tend to become hyperosmotic. It is well-established that salt loss by sweating is relatively small and that there is *no need* for additional salt intake to replace "what was lost in sweat".

Table 1. Signs and symptoms characteristic of dehydration

Water deficit (percentage of body weight)		
2%–5%	6%–10%	> 10%
Thirst	"Cotton mouth"	Delirium
Vague discomfort	Dyspnea	Inability to swallow
Anorexia	Cyanosis	Dim vision
Impatience	Dizziness	Oliguria/anuria
Headache	Stumbling	
Apathy (or clamorous)	Rombergism	
Tingling		

Heat Illness

Heat illness ranges from minor aberrations in physiology attended by minimal clinical symptoms, to full-blown heatstroke which is accompanied by major clinical manifestations and may be fatal.

Heat Edema

Swelling of the feet and ankles are often reported by nonacclimatized individuals. Usually no underlying cardiac, renal, hepatic, venous, lymphatic, or hypoproteinemic disease is found, and the edema tends to resolve after several days of acclimatization; if the swelling persists, simple elevation of the legs or a support hose is employed.

Heat Syncopy

Exposure to heat results in a redistribution of blood, more being located at the body surface and periphery (peripheral vasodilation); in combination with intravascular hypovolemia this may result in an inadequate central venous return. The concomitant drop in cardiac output may be insufficient to maintain cerebral perfusion and consciousness. Protracted standing in hot environments should be avoided. Lying down is the cure.

Heat Exhaustion

Heat exhaustion (Fig. 1) is the result of a cardio-circulatory failure caused by extreme peripheral vasodilation. The syndrome is characterized by a mild elevation in body temperature, tachycardia (> 170 beats/min), minor aberrations in mental function, dizziness, nausea, and headache. Hypohydration contributes but is not an essential factor for the development of the syndrome. Heat exhaustion characteristically occurs in hot-wet climates or other circumstances in which evaporation of sweat is limited (protective garment).

Treatment. Remove the patient to a shaded area, and cool him by any means available. After a few hours rest the patient can usually be discharged. Under field conditions heat exhaustion should be regarded as heatstroke and treated as such, until proven otherwise.

Heatstroke

Heatstroke (Fig. 1) occurs when a high body temperature (usually above 41 °C) results in cell, tissue, and organ damage. Contrary to previous beliefs, in most cases the sweat glands are still very active at the stage of heatstroke collapse. Dehydration often accompanies heatstroke but is not an essential factor in the development of the syndrome.

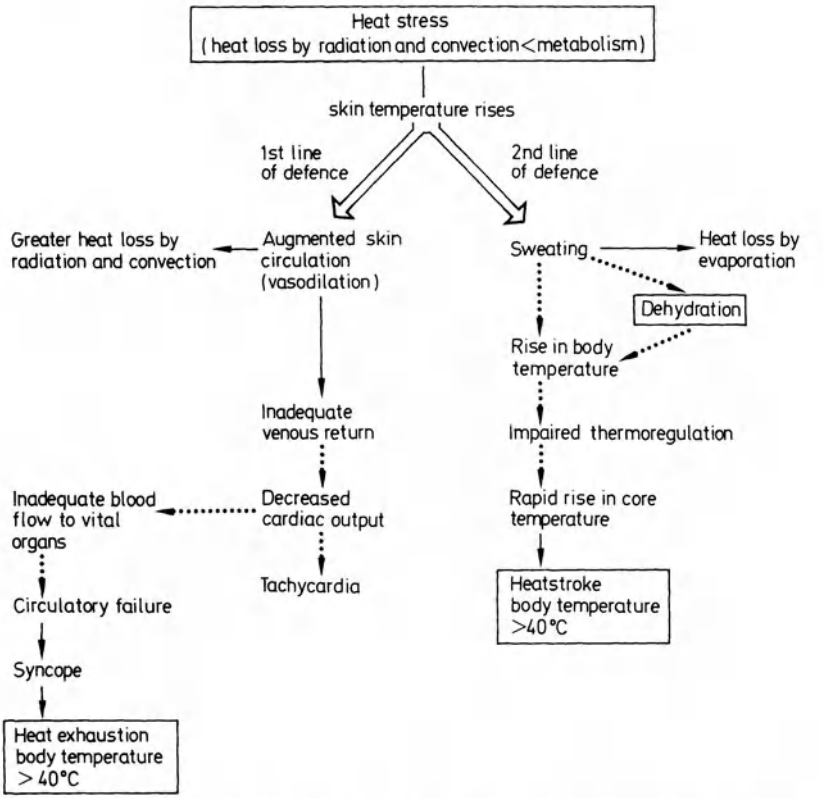

Fig. 1. The various forms of heat illness arising from failure of responses to heat stress (dotted lines). Modified from Belding and Hatch 1955

Recognition and Differential Diagnosis. Heatstroke is usually easy to diagnose (Fig. 2). In any collapse occurring during physical exercise, even under relatively mild climatic conditions, heatstroke (or heat exhaustion) must be suspected. The on-site diagnosis of heatstroke is based on the rectal temperature measured immediately upon collapse. Rectal temperature above 40.0 °C is usually regarded as a positive diagnosis of heatstroke. However, a lower temperature does not exclude the diagnosis. A delay in temperature measurement may be misleading, since the temperature may have fallen with the cessation of exercise. In hospital, measurements of SGOT, SGPT, and LDH may be useful in defining heatstroke. In most febrile states and in heat exhaustion these enzymes will be normal or only minimally elevated, while they are significantly elevated in heatstroke.

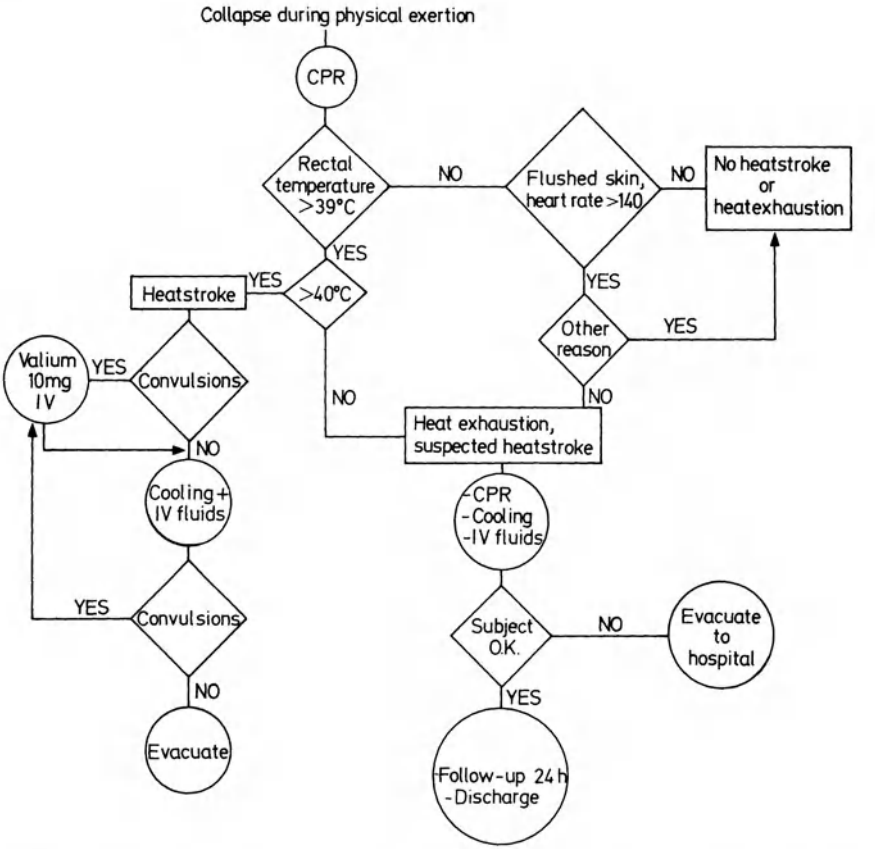

Fig. 2. Differential diagnosis and treatment on site of heatstroke and heat exhaustion

Central Nervous System. Disturbances of the CNS often dominate the early course of heatstroke. Confusion, irrational behavior, and a sudden change in or loss of consciousness are characteristic. Delirium, stupor, and coma may be accompanied by a Babinski reflex and convulsions. Other findings are: decorticated-like position, loss of sphincter control, flaccid palsy, or hemiplegia. In most cases the cerebrospinal fluid is normal. CNS symptoms are usually directly related to the duration of the hyperthermic phase and to circulatory failure. At autopsy edema, petechial hemorrhages, degenerated Purkinje cells, and edematous pycnotic cells are found in the brain. Rarely hemiplegia, ataxia, mental disturbances, and changes in personality remain as permanent sequelae.

Cardiovascular System. Tachycardia is commonly present. The ECG may show conduction disturbances and/or nonspecific ST-T wave changes. Circulatory failure is usually found in heatstroke. Hypovolemic shock occurs in about 20%. The skin is pale; cardiac output is low, diastolic blood pressure is low, and consequently the pulse pressure is high.

Clotting Dusfunction. Disseminated intravascular coagulopathy (DIC) is common. Clotting disturbances are mediated by a drop in the platelet count, hypoprothrombinemia, hypofibrinogenemia, elevated fibrinogen split products and increased capillary fragility. Clotting dysfunction peaks at 18–36 h after the acute phase of heatstroke (which explains the typical findings at autopsy of disseminated thrombosis in various organs and especially in the brain, kidney, liver, and gastrointestinal tract).

Acid-Base Balance. Hyperventilation and elevation in body temperature are associated with primary respiratory alkalosis, which is replaced later on by metabolic acidosis as a result of increased glycolysis and the development of hyperlactemia (Fig. 3).

Renal Dysfunction, Fluid and Electrolyte Balance. Hypohydration is not an essential factor in the development of heatstroke. However, many heatstroke cases are subjects who indulge in intense physical activity in a hot climate and who sweat excessively without adequately replenishing water loss. Hypohydration of more than 10% combined with renal arteriolar intravascular clotting due to DIC leads to acute renal failure. Plasma sodium levels are usually normal or elevated. Temporary hypokalemia, due to respiratory alkalosis, could turn into hyperkalemia as a result of metabolic acidosis. Usually hypocalcemia and hypophosphatemia are present. Oliguria and anuria are characteristic fearures, the urine being described as "machine oil" with low specific gravity. Red and white cells, hyaline and gran-

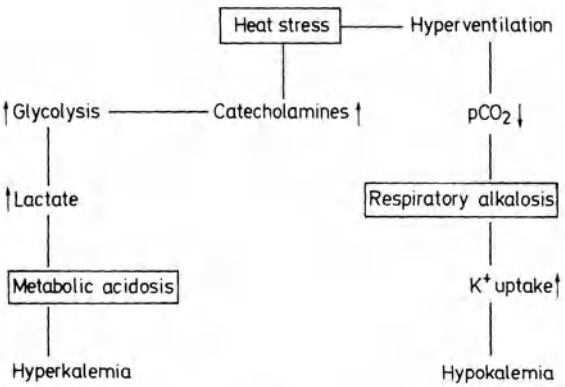

Fig. 3. Acid-base balance in heatstroke

ular casts, and mild to moderate proteinuria are commonly seen. The urine usually contains ketones. The incidence of renal failure peaks 24 h post-collapse and may last for several days.

Hepatic Dysfunction. Some 12–24 h after heatstroke bilirubin and transaminase levels are found to be elevated, and they peak within 72–96 h. Elevated hepatocellular enzymes reflect hepatocellular damage. Centrilobular necrosis, dropping out of hepatocytes, diffuse monocellular infiltrates, and widespread membrane damage are found at autopsy.

Gastrointestinal Tract. Gastrointestinal dysfunction is often present and reflects poor perfusion and DIC leading to gastroparesis, ileus, gastrointestinal ulceration, and massive bleeding. Diarrhea and vomiting are seen.

Laboratory Findings. (a) Clotting dysfunction is represented by a drop in platelet count and in clotting factors V and VIII, hypoprothrombinemia, increased fibrin products, and hypofibrinogenemia. (b) White blood cell count is elevated and may be in the range of $20–30 \times 10^3$ per mm^3 or even higher. (c) Electrolyte analysis reveals hypokalemia in the early stages of heatstroke, which in some cases turns into hyperkalemia. Sodium levels are usually within the normal range or slightly elevated, reflecting a state of dehydration. Hypophosphatemia is frequently described as a result of the respiratory alkalosis which accompanies the early stages of heatstroke.

Treatment. The prognosis of heatstroke depends on the body temperature, the duration of the hyperthermia, and the development of complications. *It is thus crucial to reduce the body temperature as quickly as possible and evacuate the heatstroke victim to hospital for the treatment of complications.*
On site and at a medical aid station, cooling is of primary importance and is initiated as soon as possible, certainly prior to any time-consuming investigation. The patient is removed to a shaded area, his clothing is removed, and any available cooling measure applied. The quickest and most effective way of cooling is immersion in tap water or pouring large quantities of water over the patient. (Icy water causes vasoconstriction and a drop in cooling efficiency!). When the body temperature (rectal measurement) drops to 38.5°C, cooling is stopped to avoid a hypothermic overshoot.
Convulsions are treated, prior to cooling, by a slow IV administration of diazepam 10 mg, repeated until the convulsios are controlled.
Fluid replacement is also urgent: 1.5–2.5 liters of an electrolyte solution are infused within the first hour and another liter during the next hour. An urinary catheter is inserted, and thereafter fluids are given in accordance with the urine output. Hemodynamic parameters and the acid-base balance are monitored.
In hospital the measures outlined in Fig. 4 are carried out. Renal failure occurs in most cases of heatstroke with high values of BUN and creatinine. Anuric patients should be given 0.25 mg/kg mannitol injected IV or 80–100 mg of furosemide. A lack of diuretic response indicates that dialysis treatment

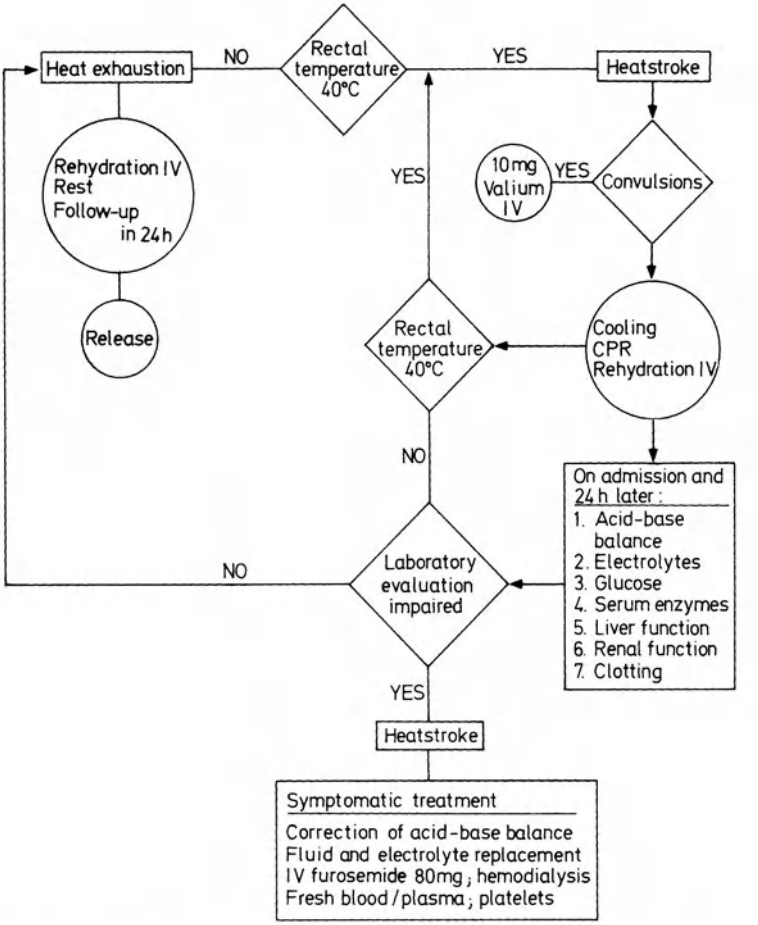

Fig. 4. Differential diagnosis and treatment in hospital of heat exhaustion and heatstroke

will be required. Heparin and other anticoagulants are ineffective against DIC. In severe cases give fresh plasma or fresh blood.

Cooling and hydration may usually be sufficient to correct the acid-base imbalance and electrolyte changes. A closer observation of the fluctuation pattern of the changes and the development of renal and/or hepatic disorder may dictate more aggressive intervention.

Prognosis. Rapid reduction of body temperature, arrest of convulsions, proper management of rehydration, with a quick evacuation to a medical

center will result in >90% survivability. A delay in these urgent procedures may result in more than 80% fatalities. Recovery from heatstroke is in most cases ad integrum (the vast majority of survivors from heatstroke recover without any permanent damage).

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2.6 Immersion Hypothermia

P. Halpern

Pathophysiology

The maintenance of a constant body temperature in water, as in air, is based on a fine balance between heat energy produced in and absorbed by the body and heat lost from the body. The maintenance of this balance is much more difficult in water for several reasons. The thermal conductivity of water is 25 times that of air, and the specific heat is about 1000 times greater. Heat loss by conduction and convection is therefore more rapid in water than in air, and these are the main routes of heat loss during immersion. Most forms of passive external insulation adequate for air exposure lose their efficacy in water. Specialized clothing is required, which is likely to be available only in diving situations or carefully prepared exposures. Accidental immersion therefore usually leaves the victim without effective insulation.

It is most difficult to assume an optimal heat-conserving posture in water. Physical effort must be expended to maintain flotation, resulting in a higher energy production on the one hand but also in a higher energy loss due to peripheral vasodilation and to increased water convection around the body. The scalp is a major avenue of heat loss because of the inability of its vessels to constrict in response to cold. During immersion, the heat lost through the scalp may account for up to 55% of total body losses. In diving with compressed gas, respiratory heat loss may become an important factor, as the diver breathes very cold and dry gas. In deep diving operations where helium is the main gas breathed, respiratory heat loss may become the major factor.

Other factors affecting heat balance include thickness of the subcutaneous fat layer (fat people have a significantly higher capacity of withstanding cold water exposure), drugs (e.g., alcohol causes vasodilation, resulting in higher heat loss, while marihuana and other psychotropics impair judgement and may inhibit necessary defensive activity) and general physical condition (fit people may survive longer than unfit or chronically ill subjects). Adaptation to cold occurs over a period of days. Previously cold-acclimatized people may better withstand cold exposure. When the victim becomes physically and/or mentally incapacitated by hypothermia, the sec-

ondary hazard of drowning becomes a significant to life before hypothermia per se is life-threatening.
Consequently, the thermoneutral temperature of water is 33 °C as opposed to 25 °C in air. Since water at this temperature is rarely encountered outside a bathtub, almost all exposures to water have the potential of causing hypothermia.
The pathophysiology of immersion hypothermia is similar to that occurring in air. It is always a rapid onset-type hypothermia. Dehydration may be more pronounced as the cold-induced diuresis is augmented by immersion. Local effects of cold, such as chilblains and frost bite, are not usually a problem except in freezing water.
A specific case is the so-called immersion syndrome appearing when a victim is suddenly exposed to very cold (less than 10 °C) water without protection. It is *not* related to hypothermia and occurs within seconds to minutes of exposure. The initial response is a very severe hyperventilation which is impossible to control. If the victim is unable to raise his mouth above the surface within seconds, drowning will be immediate. If drowning does not occur, the continued hyperventilation may cause confusion, muscle rigidity, and even dysrhythmias, all of which predispose to drowning. Some persons, especially those with previous coronary artery disease, may develop fatal dysrhythmias due to the massive sympathetic stimulation of cold water on the skin, or, if water enters the larynx, a strong vagal reflex. A sudden increase in myocardial workload and perhaps coronary vasospasm may produce myocardial ischemia.

Diagnosis and Treatment

All victims of accidental immersion must be suspected of being hypothermic, and the rectal temperature should be measured with a hypothermic thermometer. Temperatures between 33° and 36 °C denote mild hypothermia. Patients with rectal temperatures below 33 °C are severely hypothermic.
The mildly hypothermic patient may be treated on site or at the nearest sheltered facility. He is conscious, if often slow of thought and of movement. He is usually shivering. Only passive heating is required. He should be dried and covered with many layers of blankets, or at least put in a warm environment. Hot drinks are helpful, but not too hot, as he will not appreciate a scalded tongue once he is again in full possession of his senses. While hospitalization is not required if other conditions (near drowning, dehydration, injuries) do not warrant it, the patient should be observed until fully warmed up, as evidenced by the appearance of sweat.
Severely hypothermic patients should be treated as emergency cases. All such patients should be transferred to a major medical facility following

initial resuscitation and stabilization. An important dictum to remember is that no hypothermia victim should be pronounced dead until warm and dead. Indeed, severely hypothermic persons may look dead to a superficial observer: the skin is bluish and cold, respirations and pulse may be nearly undetectable, the pupils mydriatic and unresponsive to light, muscles stiff, and reflexes unobtainable. The initial examination of the patient is therefore important. The pulse should be sought after only in the carotid arteries, and one must remember it may be as slow as 30 b.p.m. Respirations may be extremely shallow and slow. Do *not* rush to start cardiopulmonary resuscitation before a careful assessment of cardiovascular status. The hypothermic heart is exquisitely sensitive to stimulation. Unnecessary cardiac compression or rough handling during too hasty transport may precipitate fatal dysrhythmias. Only if cardiac standstill or ventricular fibrillation are diagnosed should external cardiac compressions be started and intubation performed. During resuscitation chest compressions may be done at a rate of 30–40 per min and hyperventilation avoided. Anti-arrhythmic medications are very often ineffective until the heart is warmed above 31°–32°C. Therefore, vigorous heating should be commenced quickly and overtreatment with drugs avoided. A fuller discussion of rewarming methods is found in the chapter on cold injury.

A difficult but often crucial decision is whether to begin the rewarming process at field level or to transfer the patient first to a fully equipped medical facility. Resuscitation from cardiac arrest or ventricular fibrillation requires on-the-spot rewarming to 31°–32°C. However, if swift transportation under medical care is available, then a stable patient should be rewarmed only at a well-equipped station, or, better, in a hospital equipped for sophisticated care.

The complications which may arise during rewarming are: "afterdrop", a further decrease in temperature at the start of rewarming due to return of very cold peripheral blood to the core; hypotension due to vasodilation in the hypovolemic and cardiodepressed patient; acidosis; hypoglycemia; dysrhythmias.

Prevention

Divers are usually well protected by diving suits and are knowledgeable about the dangers of cold exposure. It is the accidental immersion victim who is in great danger. Prophylactic measures if any warning of impending immersion is given include:

1. Warm but preferably unrestricting clothing; even when wet, some insulating value still remains
2. Head protection is especially important; wool is best
3. Warming up prior to immersion so as to start with a "reserve" of heat.

4. Drinking (preferably warm) copious fluids
5. Avoiding alcohol and hypnotic drugs
6. Donning flotation gear, making sure that all other survival equipment is handy but secure
7. Trying to enter as part of a group, and once in water staying close to the group

Once in the water:

1. Avoid unnecessary exercise. If swimming should prove necessary it should be controlled, not strenuous, and only in the direction of a specific target.
2. If swimming is not required, the "HELP" (Heat Exchange Lessening Posture) is assumed: knees to chest, encircle and hold with both arms. In a group 2–3 people should huddle together and try not to move.

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2.7 Near Drowning

P. Halpern

Pathophysiology

1. *Pulmonary.* Aspiration of liquid and of particulate foreign material: Pulmonary edema of the high capillary permeability type, atelectasis; high shunt fractions; bronchospasm; secondary bacterial infection fairly common (Fig. 1)
2. *Central nervous system:* (a) Immediate: Anoxic-ischemic global brain damage ranging from mild and reversible to brain death. (b) Delayed: Deterioration due to brain edema; epilepsy, cerebrovascular accident (may be the cause for drowning)
3. *Acid-base.* Severe metabolic acidosis
4. *Electrolytes:* Hypernatremia, hyperchloremia in seawater. Hyponatremia and hemolysis-induced hyperkalemia in fresh water (abnormalities usually minor)
5. *Cardiovascular.* Anoxic-ischemic myocardial damage. Hypervolemia in fresh water and hypovolemia in seawater. Shock may be hypovolemic or cardiogenic. Acute MI or dysrhythmia may have caused drowning!
6. *Renal.* Acute renal failure due to anoxia, shock, and hemolysis
7. *Hematologic.* Disseminated intravascular coagulation may ensue especially in freshwater drowning

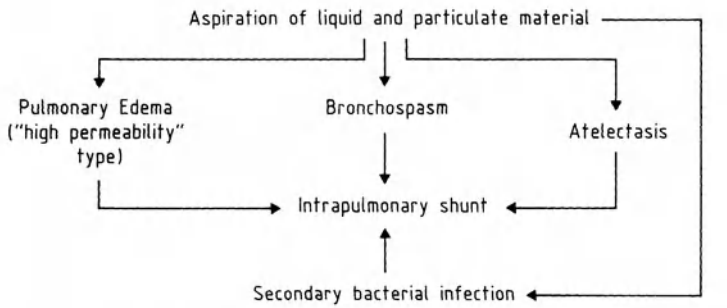

Fig. 1. Pathophysiology-pulmonary effects of near drowning

8. *Hypo-hyperthermia*. Victims are variably hypothermic depending on the water temperature and the length of exposure. Hypothermia complicates the hemodynamic and metabolic picture. Hyperpyrexia may ensue after a few hours. Severely hypothermic patients may look dead and beyond salvage whilst actually being resuscitatable.
9. *Associated conditions*. Myocardial ischemia, fractures of the limbs, fractures of vertebral column, internal injuries of the abdomen and chest, hyper/hypoglycemia, head injury, marine animal bites, and epilepsy may be etiologic factors in the drowning episode as well as posing significant therapeutic problems afterwards

Therapy

The rapidity of administration of initial resuscitation is the main factor in prognosis.

On Site and First Aid Station

Immediate

1. Assess respiration and pulses and start ABC of cardiopulmonary resuscitation immediately. Mouth to mouth ventilation may be started whilst the victim is still in water.
2. Clear the airway of foreign material, but do not attempt to evacuate the aspirated water. If patient vomits repeatedly (stomach is usually full of water) and external cardiac compression is *not* required, place patient in lateral decubitus position. Artificial ventilation may be continued in this position.
3. Assess carefully for associated injuries whilst continuing cardiopulmonary resuscitation.
4. Supplemental oxygen should be administered as soon as available.
5. Tracheal intubation is indicated: as part of cardiopulmonary resuscitation; in extreme hypoxia; in the unconscious patient; for deep shock. Maintain cricoid pressure and/or head-down position during intubation to minimize aspiration of gastric contents.

Emergency room (Hospital)

1. Continue CPR (intubation, medication, etc.) as required
2. Administer supplemental oxygen; monitor ECG
3. Initial laboratory work-up: arterial blood gases; chest X-ray; 12-lead ECG; electrolytes, hematocrit, BUN, glucose; rectal temperature (hypothermic thermometer)

4. Start IV with D5W for sea water and normal saline for freshwater near drowning and adjust fluid type and rate of administration according to hemodynamics and lab results
5. Insert nasogastric tube. If patient is unconscious, do tracheal intubation first
6. Introduce Foley catheter to monitor hourly urine output
7. If patient is in shock, insert central venous line
8. Correct acid-base balance: give bicarbonate IV if metabolic acidosis is very severe (pH < 7.10), and artificial ventilation for hypercarbia
9. Prophylactic antibiotics: only to be used in high-risk situations, such as near drowning in polluted water, or with underlying pulmonary or systemic disease

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2.8 Crush Injuries and Crush Syndrome

M. Michaelson and N. D. Reis

Crush Injury

The local result of prolonged continuous pressure is crush injury. In survivors of prolonged crushing we are mainly concerned with crushed limbs.

Diagnosis

Diagnosis is by the history of the crushing event. On site there is an area of blanched or contused skin and variable motor and sensory loss. The major limb artery is almost always patent. Upon release, at first there is little pain. Within hours the crushed area swells to a tense brawny consistency, and the pain becomes acute, especially on applying local pressure or with movement of the adjacent joints.
Most crush injuries are blunt closed injuries, but when the crushing object is jagged or sharp edged, a laceration of varying extent is observed. Crushing by a rolling heavy object may cause closed or open separation of the skin from the deeper tissues (degloving): this is not caused by prolonged application of pressure and is therefore not included in the crush injury definition. A fracture may be present in the crushed area or remote from it.

Treatment

Figure 1 outlines the treatment schedule for crush injuries. *On site and medical aid station:* cover an open wound with a sterile dressing. Provide a stretcher for lower limb crush. Gently splint the limb when a fracture is present. Evacuate (see crush syndrome) the patient as soon as possible. In the *field and base hospitals* the important principle is to not convert a closed crush to an open one. Mortality in crush injury is associated with sepsis, and sepsis is associated with an open wound containing dead tissue. The treatment for a closed crush injury involves observation, applying anal-

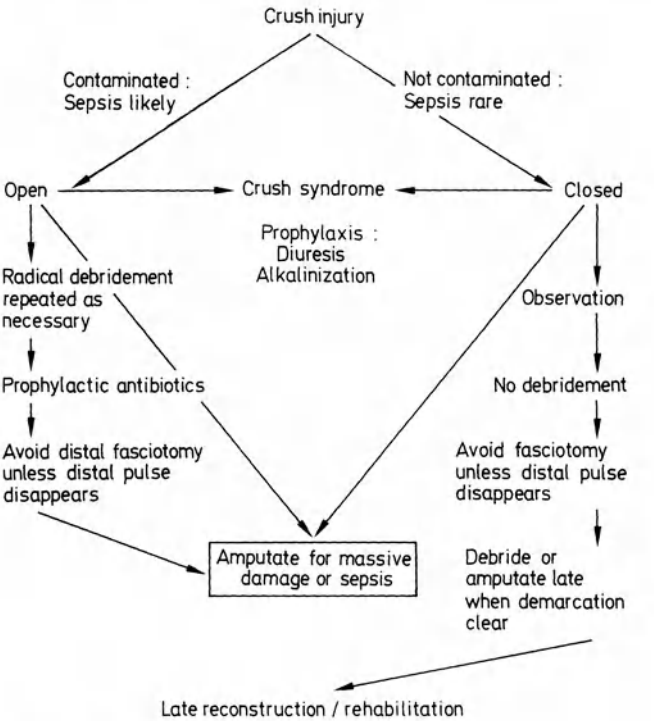

Fig. 1. Principles of treatment of a crush injury

gesia, keeping the patient recumbent, and splinting any fractures. There are several proscriptions:

1. Never excise crushed skin, and never debride unless clear demarcation of the gangrenous area is observed after a week or later. Even then, as long as the necrotic areas are not infected and there is no sepsis, perform amputation or radical local debridement only when the dead tissues are clearly demarcated from the living.
2. Never perform prophylactic local or more distal fasciotomy unless the foot or hand are endangered by the occlusion of the distal arterial blood supply. In that case, fasciotomy must be combined with radical local debridement of the crushed tissues.
3. Never operate on a fracture in a closed crushed area: treat conservatively. Avoid skeletal traction pins in or near the crushed area.
4. Amputate through healthy tissue for impending or established sepsis (sepsis in a closed crush will usually have been introduced by previous ill-judged surgery). Only rarely does one amputate primarily through in-

tact tissue for massive limb destruction when there is no chance for achieving a functional result better than a prosthesis.
Late drainage of "liquified muscle" sterile abscesses should be undertaken. The long-term management is dictated by the permanent damage and is according to established principles of reconstructive surgery and rehabilitation.

When an open crush is present, the treatment program changes:

1. Primary radical debridement or primary amputation is performed if damage is severe (as above). Once splinted, the patient should be encouraged to rest and given analgesia.
2. Repeat excision under general anesthesia every 48 h as indicated by the local appearance of the wound.
3. Prophylactic antibiotics must be administered (see chapter on treatment of wounds).
4. Distal fasciotomy is called for only if the distal pulse is obliterated (never prophylactically).
5. Surgery is followed by delayed primary or secondary wound closure by skin suture or graft.

Crush Syndrome

Crush syndrome is post crush injury, myoglobin-induced, acute renal failure. This syndrome develops in victims of catastrophic events, such as earthquakes, mine and train accidents, and war bombardment injuries (it is also described as rhabdomyolysis in nontraumatic conditions such as alcoholism and drug abuse). In all of these conditions the patients are pinned down for a prolonged period before being rescued. On release, there are no immediate signs of the severity of their condition, and yet they may rapidly develop hypovolemic shock, oliguria, and acute renal failure. The crush syndrome carries high mortality and morbidity rates.

Pathophysiology

Crush syndrome develops in patients who have suffered a crushing injury in which a large bulk of muscle is damaged. Crush injury to the torso is fatal because the rescue team cannot reach the patient in time to save him from acute cardiorespiratory failure. Crush syndrome is a reperfusion syndrome.
As a result of the reestablishment of circulation in the affected tissues after crush injury, there is leakage of fluids from the intravascular space into the

damaged muscle tissue with the creation of voluminous edema and consequent hypovolemic shock. The breakdown products of injured muscle pour into the bloodstream. The resultant severe hyperkalemia may endanger the patient's life. The muscle pigment, myoglobin, and the acid radicals from disintegrated tissue, having entered the blood circulation, in combination with hypovolemia promote the onset of acute renal failure. The histological changes include necrosis, desquamation, regeneration, and fibrosis of the whole nephron, usually with the presence of pigmented casts, the most severe damage being typically in the ascending loop of Henle and the distal convoluted tubule.

In summary, renal damage is caused by a combination of:

1. Hypovolemic shock, which by itself, if prolonged and severe enough, may cause acute renal failure
2. Deposition and blockage by myoglobin in the ascending loop of Henle and the distal convoluted tubule (especially when the pH of the environment is less than 6)
3. Toxic effect of myoglobin on the tubule cells when the pH is below 6

Injured muscle cells cannot retain their high concentration of intracellular potassium, much of which enters the circulation, and the injured kidney cannot excrete the surplus potassium, so that a severe hyperkalemia sets in. This may lead to slowing of conduction in the heart muscle, complete heart block, ventricular fibrillation, and, finally, cardiac arrest.

Diagnosis

When patients are dug out from under debris or released from crushing by other causes, external bleeding is not observed. The blood pressure is normal, although the pulse rate may be high. They are conscious and without any signs of external injury, except for local impressions made in the skin by the pressure, and they are often very alert and talkative. There are usually no physical complaints beyond mild pain in the crushed area.
As time goes by the condition deteriorates: the blood pressure drops, the pulse quickens, edema of the injured limb(s) develops, and hypovolemic shock ensues. The central venous pressure is low. The urine is smoky brown or reddish in color and contains myoglobin. Laboratory investigations reveal raised hematocrit, high potassium, high phosphorus, high creatinine phosphokinase, high BUN and creatinine, thrombocytopenia, and myoglobinemia.

Treatment

The patients are in danger of developing oliguria and acute renal failure unless early treatment is instituted (the local treatment of the crush injury has no influence on the development of acute renal failure). Once a crush injury has occurred, its local effects cannot be avoided, but its consequences, namely crush syndrome, can be prevented. The aim of treatment is to prevent hypovolemic shock, acute renal failure, and hyperkalemia. This is accomplished by replacing fluids to achieve a diuresis of 300 ml/h and maintaining a pH above 6.5 in the urine. As a result, hyperkalemia is eliminated (the massive load of K^+ will be washed out by the diuresis), and shock and myoglobin tamponade of the tubules are prevented. To achieve these goals, treatment must be started as soon as possible; therefore it should be instigated at the injury site. On-site management involves diagnosis, immediate treatment, and evacuation.

Diagnosis. The further removed from the injury site in time and space, the harder is becomes to dianose crush injury and syndrome. The patient arriving at the emergency room amongst mass casualties is easily misdiagnosed for the lack of impressive signs and symptoms. The place to diagnose potential crush syndrome is at the catastrophe site. The clues are:

a) the situation of the casualty: every casualty who was pinned down or trapped for hours or whose rescue was delayed should be considered as a potential crush syndrome victim until proven otherwise.
b) Signs of crush injury, i.e. patchy hypoesthesia and motor deficiencies (there is no edema of the limbs at the time of rescue).
c) A dark smoky red or brown colored urine.

If one or all of the above are present, a presumptive diagnosis of crush syndrome should be made and inscribed in the casualty's records or directly on the skin.

On-Site Treatment. Even before the victim is fully rescued treatment should be started. An intravenous line must be installed as soon as the rescue team are able to enter a vein, and a large amount of K^+-free crystaloids infused in order to induce diuresis. (It should be taken into consideration that the victim entrapped for a long period of time is dehydrated, even without being injured). Blood transfusion is contraindicated. There is no blood loss, and the patients are already in hemoconcentration. A thorough physical examination follows, and the findings should be recorded in the patient's records. The first urine sample must be observed by the physician. If the casualty is unable to pass urine, sterile, on-site catheterization of the bladder is mandatory to monitor urinary output. On-site treatment should be as fast as possible; delay in the transfer of the patient to an intensive care unit is to be avoided if such a facility is available.

Evacuation. The moment the patients are fully released, they should be checked for other life-threatening injuries that may take precedence. First aid is administered. They should then be transferred in the quickest possible way (preferably by helicopter) to a hospital able to give definitive intensive care.

The Medical Aid Station and Field Hospital

Patients suspected of crush syndrome should be sent directly to a base hospital. However, should transfer to the medical aid station or field hospital be dictated by prevailing conditions, he should be treated along the lines to be described for the base hospital.

The Base Hospital

Even though the patient may show no signs of developing a critical condition, he should be cared for in an intensive care department whenever possible. Other injuries are treated simultaneously but on no account may the treatment for crush syndrome be delayed. The continuing aim is to prevent acute renal failure. This is done by maintaining a diuresis of above 300 ml/h and an urine pH above 6.5. To achieve this the following protocol is recommended (Fig. 2).

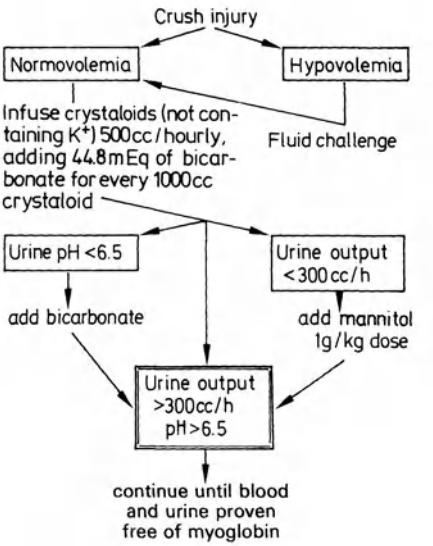

Fig. 2. Prevention of acute renal failure in crush syndrome

On arrival a central venous line and a urinary catheter are installed.
If the patient is in shock and anuric, a fluid challenge is given (see chapter on shock). On achieving a normal blood pressure, 500 ml of K^+-free crystalloid fluids are infused hourly. An ampoule (44.5 m Eq) of sodium bicarbonate is added to every second infusion bag. If this fails to produce 300 ml of urine an hour, then 1 g per kg mannitol is infused. If urinary pH is less than 6.5, more bicarbonate is added, but if the pH of arterial blood is alkalemic, then acetazolamide (Diamox) 250 mg is given i.v. Blood pressure, pulse rate, central venous pressure, urinary output, and pH are monitored hourly. Every 6 h electrolytes and osmolarity in blood and urine are measured. Blood urea nitrogen, creatinine, creatinine phosphokinase, complete blood count, and myoglobin in blood and urine are monitored every 24 h. The patient is fed a high caloric intake of 30–35 Kcal/kg per day.
This protocol is stopped only when the blood and urine are free of myoglobin. If treatment along these lines is started early, acute renal failure and hyperkalemia (which are the causes of the high mortality and morbidity) are prevented. If acute renal failure does occur or is already established, then it should be treated as any acute renal failure by dialysis (peritoneal or hemodialysis). Acute renal failure is a self-limiting disease: the patient should be dialysed for 2–4 weeks as required.

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2.9 Chemical Warfare and Disasters: Medical Organization and Treatment

I. Shalit

Lack of experience and ignorance contribute to the anxiety existing among soldiers and medical echelons with regard to chemical warfare.
Chemical warfare will result in mass casualties. This fact has several vital implications: a disaster plan for such an event is imperative; the self-treatment of persons involved is of paramount importance; specific modes of evaluation and treatment of mass casualties must be available and must have been well exercised and drilled by all concerned.
Chemical casualties are dispersed within a contaminated area. This creates a difficult problem of decontamination. It has to be decided beforehand whether casualties will be treated inside a contaminated region, thereby creating the problem of protecting the medical echelons involved, or outside that area — after decontamination of the casualties. The latter possibility protects the medical echelon but may cause a significant delay in specific medical treatment.
The best protection against chemical agents is the use of masks and protective garments. No medical treatment is comparable to prevention. However, long periods of use of special garments and masks are inconvenient and bring about some physiological hazards (dehydration, for example). Therefore, one cannot use the protective measures continuously, i.e. only for short periods of time. Hence good intelligence and a high level of readiness are imperative in order to optimize the use of protective methods. Guidelines for defense of civilians are given in Table 1.
The two main groups of chemicals in military use are vesicants and nerve agents.

Vesicants

The most important group among these agents is the mustard group. They cause chemical burns of the skin and mucosae and at a later stage result in systemic reactions. There are two different mustards: sulfate mustard (also known as yperite or mustard gas) and nitrogen mustard (which besides being a dangerous vesicant is also in use in cancer chemotherapy).

Table 1. Practical guidelines for civilian defense against chemical warfare agents

Shelter	Stay indoors. Shut all windows and doors. Move towards inner spaces, closets, etc. Seal openings with adhesive tapes. If possible, prepare ahead of time food and drinking water supply in sealed plastic containers. Cover the containers with plastic bags and seal tightly.
Protective equipment	Avoid contact with the chemical agent. Roll down sleeves. Use impermeable material such as plastic overgarments, gowns, blankets, etc., to cover exposed skin areas. Protect hands with gloves or plastic bags. If a protective chemical warfare mask is not available, use regular towels soaked with sodium bicarbonate (baking soda) solution (25 g for each 1000 ml water). Breathe through the towel, shifting it from time to time to breathe through wet areas.
Decontamination	Remove all droplets of chemical agent from the skin using clean gauze or cotton wool. Do not rub the skin. For effective skin decontamination use commercially available tubes containing Fuller's earth powder. Disperse powder over exposed skin areas. Leave powder for 1 min and remove gently with a clean gauze or cotton. Do not rub powder into the skin. If a powder is not available, use water and regular soap to remove the chemical agent from the skin. Baking soda solution can also be used for decontamination of skin including the facial area (avoid eye penetration).
After the attack	When the area is declared clean, remove all protective equipment cautiously. Use rubber gloves to protect your hands while removing contaminated material or use tweezers or similar devices. Put all contaminated material in plastic containers. Seal the containers and label them appropriately. When leaving the house or shelter (after the area is declared clean), move opposite to wind direction.

The mustards are oily liquids which evaporate very slowly at room temperature. They are persistent agents capable of contaminating a specific area for long periods of time. They cause damage either by penetration through the skin or the conjunctiva or through the lungs on inhalation.

Mechanism of Action

Mustards are alkylating agents which form strong covalent "bridges" between complementary DNA strands. Thus they impair the ability of the cells to replicate genetic substance; consequently, the most affected tissues are those in which there is continuous cell division, e.g., skin, mucous mem-

branes, and bone marrow. There is a latent period of 1–24 h between exposure and the appearance of clinical signs and symptoms. This latent period depends on the concentration of the toxic substance, time of exposure, and the tissue systems or organs involved.

Clinical Manifestations and Diagnosis

In both types of mustard poisonings are similar. Mustard gas affects mainly the skin and eyes, whilst nitrogen mustard causes mainly systemic effects.

Eye Involvement. Symptoms commence 4–12 h after exposure to a low concentration of sulfate mustard, or 1–3 h after exposure to a high concentration. Symptoms appear more rapidly when nitrogen mustard is involved. Eye symptoms are divided into four categories:

1. Mild conjunctivitis including irritation, lacrimation, and photophobia. The signs are injection and congestion of the conjunctiva. Healing takes from 1–2 weeks.
2. Conjunctivitis with mild keratitis leads to severe pain and blepharospasm. The conjunctiva is edematous and congested. Healing takes 2–6 weeks.
3. Moderate keratitis is characterized by corneal erosions and iritis in addition to all the symptoms and signs mentioned above. Healing is prolonged (2–3 months), and the patient must be treated in a hospital.
4. Severe keratitis is a rare form occurring as a result of direct exposure to mustard droplets. There are opacities and ulcers in the cornea, and the cornea may be perforated. The eye is permanently damaged.

Skin Involvement. Liquid mustard penetrates the skin rapidly and reaches the blood vessels. Penetration is increased in hot and moist skin areas (arm pit, inguinal region, etc.). After a latent period of 6–12 h, lesions which resemble 1st degree burns appear over the skin and develop later into vesicles comparable to 2nd degree burns.
As a result of long or massive exposure, necrotic ulcers appear at the affected sites which heal very slowly, leaving a residual scar and hyperpigmentation. The mustard absorbed through the skin causes systemic manifestations such as fever, nausea, and weakness that appear several hours after exposure.

Respiratory System Involvement. Coughing, sore throat, and mucoid secretions from the upper respiratory tract appear 10–12 h after exposure to mustard vapors. In severe exposure, mucosal pseudomembrane similar to diphtheria is observed and is followed several days later by fever and the signs of chemical pulmonary edema. There is a tendency to a secondary infection in the damaged lungs. Those who survive the pulmonary intoxica-

tion go through a very long convalescence and healing is never complete: Residual damage to the respiratory system is to be expected in the form of chronic bronchitis, bronchiectasis, and/or interstitial fibrosis. There is an increased frequency of lung tumors among survivors.

Gastrointestinal System Involvement. Swallowing mustard drops is often lethal. Some 20–60 min after the event, the victim may complain of abdominal pains, colics, nausea, and vomiting. Usually the body temperature rises. Several hours later, the victim suffers from diarrhea (often bloody). At this stage there are widespread ulcerations along the gastrointestinal tract. Dehydration and severe electrolyte disturbances follow.

Bone Marrow Involvement. Damage is observed about 12 h after exposure to nitrogen mustard. Within a few days the bone marrow becomes aplastic.

Other Systemic Manifestations. General weakness and systemic fever appear in most people affected. After massive exposure, neuropsychiatric manifestations such as depression, sleep disturbances, and rarely convulsions may be anticipated.

Treatment

There is no specific antidote. Treatment is symptomatic and supportive. It includes analgesics, spasmolytics, and antitussive agents. Bacterial pneumonia should be anticipated and antibiotic treatment instituted.
If the eyes are affected, they should be washed out with water or saline immediately. Local anesthetic eye drops are recommended to reduce blepharospasm. Pupil dilators should be administered to avoid synechiae. Secondary eye infection is prevented by the use of sulfacetamide or antibiotic eye drops.
Mustard burns should be treated just like any chemical burn. The treatment includes analgesics, local application of ointment (in certain areas), monitoring of water and electrolyte balance, and supplying adequate amounts of proteins and calories.

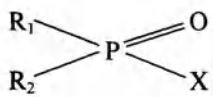
Military/Medical Aspects

Mustard agents have the capability to penetrate the skin, clothes, and even through protective garments. Thus the goal should be the early decontamination of any victim. Any delay may bring about more absorption of the material. This means that every soldier should have a personal decontamination kit and know how to use it.
During the First World War nitrogen mustard gas was considered a lethal agent due to the many deaths attributed to secondary bacterial pneumonias.

Today it should be considered only as a "neutralizing" chemical due to the advent of protective garments, masks, and better medical treatment. The main problem in the treatment of affected combatants is the eye involvement.
Medical services on the battlefield should be ready for nonconventional attacks. Readiness and preparedness mean the ability to cope with a mass casualty situation in which the causative agent may be a mustard agent and the ability to operate in a contaminated area. Hospitals should be trained to cope with such problems in order not to be overwhelmed by the many severely burned casualties.

Nerve Agents

The most common nerve agents in use are the organophosphates:



where X is a halogen or another negative radical, and R_1 and R_2 are carbohydrates. The organophosphates are systemic poisons which act on the cholinergic synapses in the central and the peripheral nervous systems.

Mechanism of Action

The neurotransmitter acetylcholine (ACh) is secreted at the proximal nerve site of cholinergic synapses. The ACh is bound to receptors at the postsynaptic membrane and evokes an action potential there. This synaptic transmission is interrupted by the enzyme acetylcholinesterase which metabolizes ACh and hence its action on the membrane stops. At the motor endplate this esterification stops muscle contraction or stops the secretion of a cholinergic gland, etc.
The organophosphates act at the cholinergic synapse through binding acetylcholinesterase. This binding abolishes the action of the enzyme, resulting in a continuous reactivation of the cholinergic synapse without the physiological pauses in their action.
This continuous reactivation causes permanent contraction of smooth muscles (e.g., intestinal contractions or bronchospasm) and, later, tetanic paralysis of voluntary muscles and continuous activity of secretory organs.
The chemical bond between the organophosphate and the enzyme acetylcholinesterase is very stable (i.e., it does not undergo spontaneous hydrolysis). It can be hydrolyzed by the oxime group of substances. Oximes have a

high affinity for organophosphates resulting in the disconnection of acetylcholinesterase from the organophosphate, while creating a stable compound of oxime-organophosphate. The released enzyme resumes its normal activity in the synapse and reverses the signs of intoxication. Some of the organophosphates, after binding to actylcholinesterase, undergo a process of further bond stabilization called "aging". This stable bond cannot be hydrolyzed by oximes. The only way to overcome the "aging" situation is by de novo synthesis of acetylcholinesterase in the synapse. This may be a long process, and clinically such organophosphates cause a severe and sustained intoxication.

Nerve agents used as chemical warfare agents, in contrast to organophosphates used as insecticides, are characterized by a more rapid "aging" process of their bond with acetylcholinesterase, and by their significantly lower toxic doses.

The battlefield nerve agents fall into two main groups.

G substances are evaporating chemicals which penetrate the body mainly through the respiratory system. These substances may contaminate an area for up to several hours. While the time of "aging" for some of these chemicals is several hours, other, more dangerous ones undergo "aging" within minutes.

V substances are persistent (nonevaporating) chemicals. They affect the body by penetrating through the skin and mucosae, though people may be affected through the respiratory system by aerosols. These substances may contaminate an area for weeks. Their toxicity is 10 times higher than the G substances; however, their "aging" time is very long (>48 h).

Clinical Manifestations and Diagnosis

The clinical picture in nerve agent casualties is best described according to the three types of cholinergic synapses affected:

1. Muscarinic: sweating, lacrimation, contracted pupils, disturbances, bronchospasm, watery diarrheas, polyuria.
2. Nicotinic: fasciculations, muscle twitching and contractions, muscle weakness, flaccid paralysis.
3. Central nervous system: anxiety, nausea and vomiting, stupor, coma, epileptic fits, paralysis of vital centers (including the respiratory center).

Generally, victims will develop a combination of the above symptoms. Exposure to low concentrations of nerve agents will result in a gradual appearance of muscarinic symptoms along with local fasciculation. Anxiety, restlessness, and confusion appear in mild intoxications as well. Without treatment, general fasciculations followed by flaccid paralysis will ensue.

In people exposed to high concentrations of nerve agents, the full spectrum of signs and symptoms listed above will appear within a short time. Owing to the combined effects of bronchospasm, bronchial secretions, respiratory muscle paralysis, and respiratory center depression, a severe impairment of the entire respiratory system occurs, leading to early respiratory arrest and death.

Late effects. Aside from the acute clinical picture, there are late and delayed effects of organophosphate poisoning. Patients who are apparently at a convalescent stage may develop late effects unexpectedly. These can be divided into three groups:

1. Continuing manifestations of acute poisoning. The central nervous system is mainly involved. The effect of the organophosphates may last for weeks or months. Restlessness, insomnia, depression, and lack of concentration are common. Myosis may persist for days or weeks.
2. Delayed neurotoxicity may appear 1–2 weeks after exposure and manifest as peripheral polyneuritis. Later the pyramidal system may become involved, leading to an upper motor neurone disease. These phenomena have been described only in pesticide poisoning.
3. Late cardiac manifestations. Ventricular tachycardia has appeared suddenly in a small number of cases that were recovering clinically from an acute poisoning. Rarely a prolonged QT segment in electrocardiograms was observed prior to the appearance of ventricular tachycardia. The cause of this phenomenon is not known.

These late clinical manifestations bear important military implications. The mental, neurological, and ocular symptoms bring about the long-term neutralization of the victims. Affected soldiers may not be fit to return to combat areas for several weeks or longer. To date there is no way to predict, prevent, or treat these late manifestations.

Treatment

Organophosphate poisoning is a medical emergency requiring adequate oxygenation, decontamination, specific antidotal treatment, and supportive treatment.

Oxygenation. Ensuring patent airways, suction from the upper respiratory tract, ventilation, and oxygen enrichment is imperative.

Decontamination. To stop further exposure to the noxious agent decontamination is necessary. Washing with water and detergent is usually sufficient. In the battlefield soldiers should be equipped with a personal decontamination kit comprising either liquid detergent or dry absorbent powder. In secondary and tertiary echelons of medical treatment, a decontamination area

should be organized. A schematic description of such a decontamination area is illustrated in Fig. 1.

Antidotal Treatment. To counter the organophosphate action and restore the acetylcholinesterase activity to normal, the main antidotes are atropine and oximes.

Atropine is a cholinolytic substance which competes with ACh on the muscarinic receptor, blocks the effect of the excess of neurotransmitter, and causes the disappearance of the muscarinic signs and symptoms. Atropine crosses the blood-brain barrier very slowly and does not reach the nicotinic synapses. Thus atropine is only a peripheral muscarinic blocker. It should be administered as early as possible and in adequate doses. The total dose required for each individual is difficult to predict, therefore it should be administered by repeated small doses, while continuously monitoring the clinical condition of the patient.

An initial dose of 1-2 mg atropine should be injected IM in mildly affected cases and IV in severe cases. Onset of action is 20 min after IM injection and 3 min after IV injection. This repeated recommended dose should be administered every 15-20 min in mild cases and every 3-5 min in severe cases. Repeated administrations should be continued until mild signs of atropinization are noted (dry and red skin and dry mucosal membranes). At this

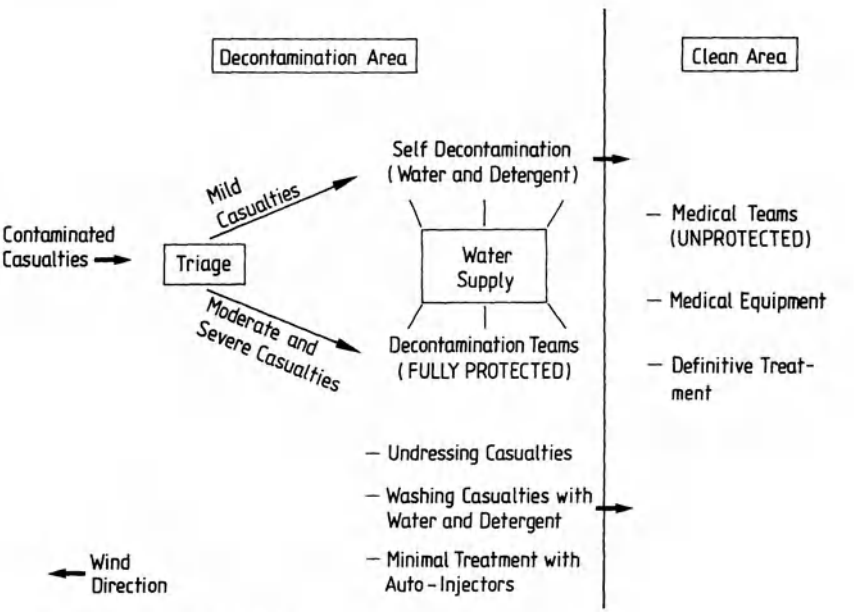


Fig. 1. Arrangement of a typical decontamination area

stage, the muscarinic signs and symptoms subside, and the respiratory state improves. Usually the atropine dose needed by mildly affected patients during the first 24 h is 4–8 mg. Severely affected cases may need as much as 100-400 mg during the first 24 h. Most of this quantity is given during the first 6 h after the poisoning. As the patient's condition improves, there is a gradual decrease in the amount of atropine needed in order to achieve atropinization. There is no hard and fast rule for how long to continue to treat. Based on a literature survey it is recommended to sustain mild atropinization for 24 h in mildly affected cases and 48–72 h in severe cases.
The widely used *oxime* is pralidoxime (2-PAM). The newer obidoxime chloride (Toxogonin) has two active sites on one molecule and is considered to be a better reactivator of acetylcholinesterase than 2-PAM. Trimedoxime (TMB-4) is used mainly in eastern Europe, and pralidoxime mesilate (P_2S) is the only oxime active by the oral route.
The oximes are active mainly at the nicotinic synapse and are not efficient for central or muscarinic effect. They improve the activity of the affected respiratory muscles and abolish fasciculations. Thus, the oximes together with atropine make up a complementary therapeutic regime for organophosphate poisoning, combining activity on muscarinic and nicotinic synapses.
In contrast to atropine which is given in small repeated doses, oximes are given in a fixed dose. The adult dose of 2-PAM is 1 g every 2 h. The dose of Toxogonin is 0.25 g every 2 h. The maximal dose is 0.75 g in mild cases and should not exceed 2 g even in severe cases, due to hepatotoxicity.
Oximes should be injected IV very slowly in order to prevent adverse effects, such as nausea, dizziness, and hypotension.

Military/Medical Aspects

Several unique problems arise when nerve agents are used as chemical warfare agents in the battlefield. The organization of medical services in the battlefield is complicated by the problem of decontamination whilst treating and evacuating patients in a contaminated area. Triage of mass casualties under such conditions has to be aimed at rescuing those with reasonable chances for survival and not necessarily towards those who are more severely affected. The use of respirators as an essential part of resuscitation of poisoned patients creates a major logistic problem in the mass casualty situation in the battlefield, and even secondary echelons in noncontaminated areas are unlikely to be equipped with the number of respirators required.
Realizing the above problems, it is apparent that military/medical authorities have to create a specific doctrine for medical treatment and organization under chemical attack. Preparedness, readiness, and appropriate equipment are necessary to achieve these goals.

Self-Treatment

The main problem with military nerve agent poisoning is the rapid development of the clinical picture. Treatment is efficient only when applied within minutes of poisoning. The only way to ensure early antidotal therapy is to equip all soldiers with antidotes for self-administration and teach them how to use them. One such method is by providing automatic syringes containing atropine and oxime. The mixture offers better treatment than either compound alone, and in addition the early treatment with oxime reactivates the acetylcholinesterase before the rapid "aging" process takes place.
An example of such a mixture is the TAB automatic syringe which includes: the oxime TMB_4 (trimedoxime, T), atropine (A), and benactyzine (a central cholinolyte, B).
A self-injection of this mixture, and later 1–2 additional injections of atropine offers a significantly better treatment than atropine only.
Equipping every soldier with an automatic syringe may result in the injection of the mixture into a healthy individual. This may cause several adverse effects such as blurred vision, tachycardia, dry mouth, dry and red skin, and drowsiness. It is essential therefore to assure the appropriate use of automatic syringes in the battlefield. Hence, military/medical authorities have to decide how many syringes each soldier will have, and in addition, design a training program that will diminish the chance of mistaken injections.

Pretreatment

Since postexposure treatment for nerve agent poisoning has several problems (e.g., the need for rapid treatment and the "aging" process), there is a constant search for drugs that can be taken before exposure to the nerve agents and offer protection either alone or by facilitating the postexposure antidotal therapy. The oxime P_2S has been proposed as pretreatment: In order to create an effective level in the tissues P_2S tablets must be ingested every few hours. In experimental animals pretreatments with P_2S improves the resistance to nerve agents by facilitating the efficacy of antidotal treatment administered after exposure.
An additional group of drugs, the carbamates, has been advocated as efficient pretreatment agents. These agents are weak, reversible inhibitors of acetylcholinesterase (the exact mechanism of action is as yet unknown). Several of these agents are already in clinical use (e.g., pyridostigmine in myasthenia gravis). In experimental animals pretreatment with pyridostigmine, followed by postexposure atropine administration, was significantly better than postexposure treatment alone. Pyridostigmine has several advantages over P_2S. It offers better protection against the most rapidly "ag-

ing" nerve agent, the daily amount of tablets required for adequate protection is smaller, and the frequency of associated side effects is much lower.

In conclusion, equipping every soldier with an oral pretreatment drug in addition to automatic syringes containing antidotal mixture is likely to improve the outcome of nerve agent casualties in the chemical battlefield.

2.10 Medical Aspects of Thermonuclear Disaster

C. Llewellyn

Mass casualties, whether from conventional, biological, chemical, or nuclear warfare, can overwhelm any medical system. This chapter focuses on the immediate (first 2 days) management of casualties caused by tactical and strategic nuclear weapons but is equally applicable to the handling of casualties from radiation accidents.

Nuclear weapons produce injuries by blast, thermal radiation, and ionizing radiation injury. As shown in Hiroshima and Nagasaki (Table 1), large *strategic nuclear weapons* (20 kilotons to megatons) produced few surviving casualties with *only* radiation injury. Blast and thermal effects killed most of those with high doses of radiation. The radiation illness predominantly observed was the sublethal hematopoietic syndrome.

Low yield *tactical nuclear weapons* (0.1–20 kilotons) produce significant radiation exposure extending well beyond lethal burn and blast distances from ground zero. Thus, surviving casualties may exhibit the full spectrum of the acute radiation syndrome; central nervous system, microvascular, gastrointestinal, and hematopoietic syndromes may be seen, either alone or in combination with blast and thermal injuries.

For the casualty with a potentially survivable radiation exposure there is no immediate life-threatening hazard. Initial treatment efforts should focus on

Table 1. Types of injuries observed in Hiroshima and Nagasaki by strategic (high yield) nuclear weapons

Injury	Hiroshima (12.5 KT)	Nagasaki (22 KT)
Deaths	64500	34200
Burns and other injuries	67%	63%
Blast trauma and other injuries	53%	42%
Radiation and other injuries	29%	24%
Burns alone	23%	15%
Blast trauma alone	30%	30%
Radiation alone	8%	13%

KT, kilotons

resuscitation and stabilization of other injuries, deferring assessment of possible radiation injury. Triage and treatment should be conducted without concern for the probability of radiation injury. Only for patients in whom radiation sickness is the only medical problem should a preliminary diagnosis of radiation injury be made, based on prodromal signs and symptoms of radiation injury.

The Contaminated Patient and Decontamination

The irradiated patient is not a health hazard to medical personnel unless externally contaminated by radioactive material. If radiation monitoring equipment is unavailable, all patients should be decontaminated prior to entering a medical facility. Resuscitation and stabilization for life-threatening injuries must be accomplished prior to decontamination.

Short-term goals of decontamination are: minimizing internal translocation of contamination, reducing the radiation dose to the body, and preventing the spread of contamination to the environment and others in it. If monitoring equipment is available, only patients with detectable levels of contamination need be decontaminated.

In Appendix 1 a protocol is provided for handling a contaminated patient in an emergency department. Primary decontamination — removing the patient's clothing and shoes — will reduce contamination by 70%–90%. Gentle washing all exposed body surfaces with a mild soap or detergent will remove most of the remainder. Producing trauma to the skin must be avoided.

Wounds must be presumed contaminated, and they must be decontaminated first before proceeding to unbroken skin. Copious irrigation with saline or 3% hydrogen peroxide will remove most contamination.

Internal deposition and incorporation of radioactive material inside cells by inhalation or ingestion is a potential problem; treatment goals are reducing absorption and accelerating elimination or excretion. Information on decorporation agents and management of internal contamination can be found in the Further Reading.

Medical treatment personnel can be adequately protected from contamination by the use of the usual surgical attire: surgical gloves, scrub suits, gowns, hoods and masks, and waterproof shoecovers. Decontamination procedures, including the removal of contaminated clothing and disposal of potentially contaminated waste and fluids, must be practiced frequently by all medical units following well-defined, standard operating procedures.

It must be reemphasized that the irradiated patient, if not contaminated or after complete decontamination, is not radioactive and therefore not a hazard to other personnel. Just as a thermal burn casualty does not radiate heat, the irradiated casualty does not give off radiation. Potentially surviva-

ble radiation injuries, if uncomplicated by other burn, blast, or traumatic injuries, are not acutely life threatening, and therefore not true emergencies.

The Acute Radiation Syndrome

Three major organ systems with different levels of radiation sensitivity respond to penetrating radiation and thereby contribute to the syndrome (Tables 2 and 3).

1. *Cardiovascular/CNS syndrome* (over 3000 rads).
   Supralethal dose, always fatal. Immediate nausea, vomiting, bloody diarrhea, irreversible hypotension, apathy, ataxia, convulsions, and coma. No prodrome or latent phase. Progression to overt clinical illness in 3–6 h. Death within 48 h. Lesion involves endothelial radionecrosis and vascular collapse.
2. *Gastrointestinal syndrome* (1000–3000 rads).
   Prompt onset (3–12 h) of profuse diarrhea, nausea, and vomiting. Patient becomes asymptomatic in 24–48 h: lymphocyte depression. Latent period of 1 week or less followed by denuding of GI tract leading to profuse diarrhea, fulminant fever, infection, and hemorrhage, either leading to death or the hematopoietic syndrome.
3. *Hematopoietic syndrome* (200–1000 rads).
   Prodromal phase — Nausea, vomiting, and anorexia. Onset in 2–6 h with higher dose; 6–12 h with lower doses. Patient is asymptomatic within 24–48 h: lymphocyte depression.
   Latent phase — From a few days to 2 or 3 weeks.
   Sequence of changes in blood elements resulting in pancytopenia.
   Overt clinical illness phase — Begins 30–40 days after exposure. Illness due to pancytopenia, with infection and hemorrhage requiring sophisticated treatment.

Table 2. Categories of acute radiation effects

Form	Dose (rads)
Subclinical	0–75
Hematopoietic	50–1000
Gastrointestinal (GI)	800–2500
Central nervous and cardiovascular systems (CNS-CV)	Above 2500

Table 3. The acute radiation syndrome

Dose (rems)	1 h	2–6 h	6–8 h	24–48 h		1 2 3 4 Presenting type of illness	Treatment required from	Outcome	
50–150		?	Symptoms reach maximum	Prodromal symptoms subside	LATENT PERIOD	Little clinical upset Perhaps only laboratory evidence of blood upset	?	Recovery likely	50–150
200–400		□ Nausea, vomiting				← Latent period 2–3 weeks → Clinical and laboratory evidence of blood upset	3rd–4th week	50% or more recover	200–400
* 400–600		□ Nausea, vomiting		Symptoms may continue for several days ...		← L.P. → 1–2 weeks Severe clinical evidence of blood upset. Gastrointestinal upset at higher doses.	2nd week	50% or more die	400–600
600–1400		□ Nausea, vomiting, diarrhea		... and may merge into manifest illness →		← L.P. → 0–7 days Severe gastrointestinal upset. At lower doses patient may survive long enough to show severe blood upset later.	1st week	Death likely	600–1400
>1400	□ Vomiting, diarrhea, shock, CNS impairment. Death within hours.					Patient already dead			>1400

The prodromal phase, onset and duration of symptoms according to dose received.

* Thick horizontal line is drawn at the LD_{50} level.

□ Onset of symptoms.

Duration of the latent period and presenting type of the manifest illness according to dose received.

Recovery — Stem cell survivors and/or marrow transplants replicate and produce normal blood elements.

The above signs and symptoms are clearly manifest during the period of overt clinical illness but are of little use early on when initial sorting and

Table 4. The acute radiation syndrome: the clinical picture at moderate, median lethal, and lethal dose levels

Time after exposure	Moderate dose (100–300 rems)	Median lethal dose (400 rems)	Lethal dose (600 rems)
1st week	Nausea and vomiting mild or absent	Nausea and vomiting after 2 h	Nausea and vomiting within 2 h
		Absence of symptoms	Diarrhea, inflammation of mouth and throat
2nd week	Absence of symptoms		Fever, rapid weight loss, death
			(Mortality probably 100%)
3rd week		Epilation, loss of appetite, malaise	
	Epilation	Fever and severe inflammations of mouth and throat	
4th week	Loss of appetite, malaise	Pallor, hemorrhages and petechiae, diarrhea	
	Sore throat	Rapid weight loss, death	
	Pallor, hemorrhages and petechiae, diarrhea	(Mortality probably 50%)	
	(Symptoms may be mild. Recovery likely)		

diagnosis are made since even low-dose radiation exposure produces acute nausea, vomiting, diarrhea, anorexia, apathy, and fatigue. However, careful observation of time of appearance and duration of early prodromal signs and symptoms and lymphocyte depression greatly facilitates the early diagnosis and prognosis of radiation injury (Table 4 and Fig. 1).
Rather than definitive diagnosis of radiation injury, attention should be focused early on sorting into three categories: radiation injury unlikely; radiation injury probable; and radiation injury severe. Table 5 illustrates the relationship of prodromal signs and symptoms to these categories as defined by Alter and Conklin (1984).

Radiation Injury Unlikely. No signs or symptoms of radiation sickness. Return to duty unless other injuries or medical problems exist.

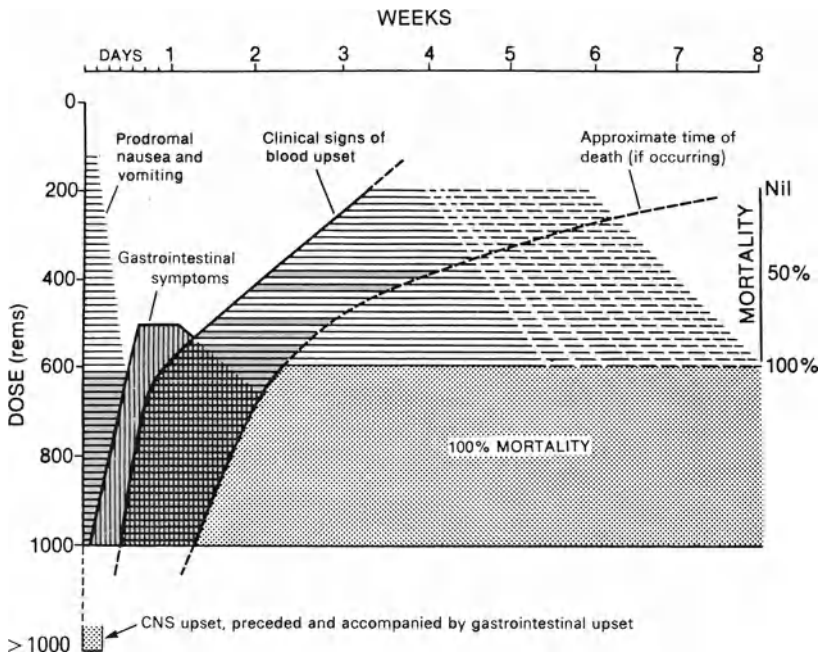


Fig. 1. The acute radiation syndrome: time sequence of the main events according to dose

Table 5. Preliminary triage of casualties with possible radiation injuries

Symptoms	Unlikely	Probable	Severe
Nausea	–	+ +	+ + +
Vomiting	–	+	+ + +
Diarrhea	–	±	± to + + +
Hyperthermia	–	±	+ to + + +
Hypotension	–	–	+ to + +
Erythema	–	–	– to + +
CNS dysfunction	–	–	– to + +

Radiation Injury Probable. Anorexia, nausea, and vomiting present. Lymphocyte counts indicated as follow-up. Should not require therapy for radiation injury in first few days. Management based on other injuries. Watch for gastrointestinal or hematopoietic syndromes.

Radiation Injury Severe. Very early onset of nausea, vomiting, anorexia, explosive diarrhea, hypotension, and neurologic disability. Dose is potentially fatal. Confirmed by lymphocyte counts. Receive symptomatic care as resources permit.

Signs and Symptoms During the Prodromal Period

Nausea and Vomiting. Onset within first hour with explosive bloody diarrhea signals fatal outcome. Appearance during first 2–3 h indicates a high dose. Onset between 6–12 h and termination within 24 h suggests sublethal (100–200 rads) dose. Must be documented at the initial and each subsequent examination and differentiated from a normal stress/anxiety response.

Hyperthermia. Significant rise in body temperature in first hours after exposure is associated with fatal outcome. Fever and chills in first 24 h have similar meaning.

Erythema. Doses of 1000–2000 rads cause erythema within first 24 h of exposed body surfaces. Less frequent and later appearance with lower doses (400 rads).

Hypotension. Associated with supralethal whole body irradiation. More than 10% drop in systolic blood pressure is significant. Difficult to document.

Neurologic Dysfunction. Mental confusion, ataxia, convulsions, and coma in first 2–6 h after exposure indicate a supralethal dose.
Careful attention to the time of onset and duration of these signs and symptoms enables the physician to do rapid early sorting of potential radiation casualties.

Lymphocyte Response to Radiation

Circulating lymphocytes are highly radiosensitive, and excellent correlations between radiation dose and lymphocyte count have been shown. All patients in the radiation injury probable and severe categories should have a lymphocyte count as soon as possible and again at 24 and 48 h if possible. Figure 2 relates changes in lymphocyte count over 48 h after exposure to radiation.

- Lymphocyte count greater than 1500 per mm^3: no significant exposure
- Lymphocyte count between 1000 and 1500 per mm^3: moderate exposure. Moderate marrow depression in 3 weeks. Prognosis good with treatment. Possible marrow transplant candidate.
- Lymphocyte count between 500 and 1000 per mm^3: severe radiation injury. Hemorrhage and infections within 2–3 weeks. Marrow transplant indicated within first week post-exposure.
- Lymphocyte count less than 500 per mm^3: probably lethal exposure. Gastrointestinal syndrome and inevitable pancytopenia. Marrow transplant of little use.

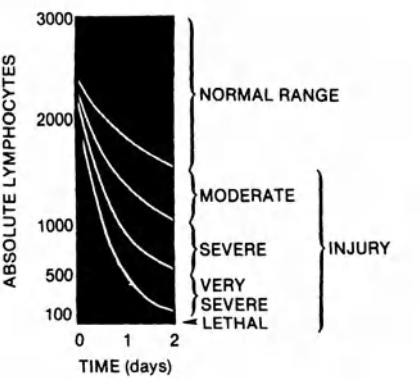


Fig. 2. Relationship between the decrease in lymphocytes in early counts as a function of radiation exposure

- Lymphocyte count less than 100 per mm^3: supralethal dose; survival unlikely.

Thus, the nonspecific prodromal signs and symptoms of radiation exposure can be combined with changes in circulating lymphocyte counts over the first 48 h post-exposure to accomplish initial sorting and provide a preliminary working diagnosis of radiation injury.

Combined Injury

In contrast to the types of injuries produced at Hiroshima and Nagasaki by high-yield strategic nuclear weapons, low-yield tactical nuclear weapons produce a high percentage of casualties with severe burn and blast injuries in addition to significant radiation injury — a phenomenon known as combined injuries. In these patients initial triage and treatment are based upon the conventional injuries alone. Later there may be a need for reclassification based upon the prodromal symptoms of the acute radiation syndrome and lymphocyte counts.
The following effects should be anticipated in combined injuries:

1. While initial healing is not affected, radiation injury adversely affects the subsequent healing process. Wounds and fractures heal more slowly and may break down.
2. There is profound immunoparalysis, depressed inflammatory response, and increased incidence of wound infection and sepsis which is extremely difficult to treat.
3. The potential for hemorrhage is increased, and shock is often refractory.
4. There is increased toxicity from many anesthetics and analgesics.
5. Injuries that are not life-threatening by themselves may become lethal in combination with otherwise potentially survivable whole-body irradiation.
6. The latent period before onset of frank radiation sickness is decreased.

In general, one should operate early on burns and wounds (within the first 36 h) to take advantage of the latent period in radiation injury. Severe burns, severe blast injury, and prodromal signs of radiation injury indicate a high probability of significant radiation injury. These patients should have surgery deferred. Patients with soft tissue or single penetrating wounds, no burns, and no prodromal signs should have surgery first since their radiation exposure is probably sublethal. Subsequent staging of procedures must await recovery of the hematopoietic system.

Acute Psychologic Reactions After Nuclear Weapon Attack

Human exposure to combat elicits a spectrum of psychological reactions beginning with "normal" anxiety, fear, and autonomic arousal, sometimes extending through minimal to severe, immediate, subacute or chronic incapacitation and disability. The bombings of Hiroshima and Nagasaki provide the only data on civilian and military groups exposed to nuclear weapon attack. Prior to these attacks there was no general understanding of the short- or long-term effects of exposure to nuclear weapons, and the psychological context was quite different to that produced by the current general knowledge of acute and chronic effects of radiation on human populations and global ecology. Thus, it may be anticipated that future nuclear weapon attacks will provoke a much greater degree of psychological reaction and incapacitation in survivors, even in those not directly exposed to the weapon's effects.

Animal studies have shown that even low doses of radiation (100 rads) alter neural physiology and brain function, and behavioral changes have been documented with exposures as low as 30 rads. Anecdotal accounts of survivor behavior in Japan described lethargy and fatigue with general reduction of motivation. Social order and military discipline seem to remain intact but are strongly influenced by rumor and misinformation. Direct experience with large numbers of horribly wounded casualties and intense physical destruction after a nuclear attack will increase the rates of combat stress reactions above those anticipated during conventional combat. Current animal and human data regarding radiation effects on neural physiology and function are inadequate to predict an additive component to these psychological reactions. However, concomitant effects should be anticipated that influence quality, degree and type of psychological reactions in the survivors.

Military unit cohesion, proper training and preparedness, and the provision of accurate information concerning the nuclear threat and the tactical situation following an attack will provide some degree of protection against increased rates of combat stress reactions. Combat unit commanders and medical personnel must plan to deal with these issues prior to combat, following the basic management principles for individual combat stress reactions and military unit reconstitution.

Management of Radiation Injury

Detailed consideration of medical management for radiation injury is beyond the stated scope of this chapter. However, some comments are warranted. Patients whose lymphocyte counts are below $1500/mm^3$ at 48 h

post-exposure may require specialized care, particularly if combined injuries are present. Susceptibility to both infection and hemorrhage is significantly increased due to marrow depression with pancytopenia and immunosuppression (Fig. 3).

Hematology-oncology services are best prepared to handle patients with the hematopoietic syndrome based on extensive experience in managing oncology and immunosuppressed patients with marrow depression. Previously healthy radiation casualties should be expected to do better than oncology patients. However, like immunosuppressed patients, radiation casualties will be highly susceptible to nosocomial infections and invasion by endogenous nonlethal organisms with a fulminating infectious process. In combined injury with open wounds, the risk of sepsis increases greatly.

Prior planning should designate centers to which patients meeting certain criteria will be evacuated. Treatment protocols should be developed covering care in a protective environment, prophylactic nonabsorbable oral antibiotics for intestinal decontamination, indications for marrow transplant within 7 days post-exposure, and possible subsequent granulocyte and/or platelet transfusion. Most of these measures will be impractical in nuclear warfare mass casualty situations.

Different criteria must be developed for mass casualties and for small numbers of patients from radiation accidents. As demonstrated by the Chernobyl experience, these measures are of limited efficacy for exposure above 400 rad with associated major burns.

Appendix 2 addresses the organization of a military field medical system to deal with mass casualties from the battlefield use of nuclear weapons. As would be true for a civilian nuclear disaster producing casualties which overwhelm the available medical system, there is no adequate solution. This must not be used as an excuse for avoiding necessary planning and training.

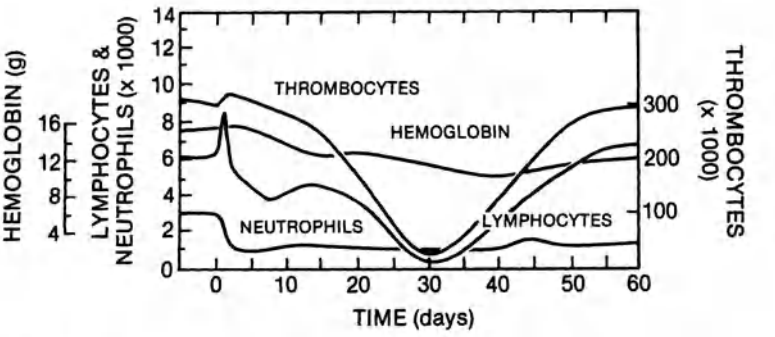


Fig. 3. Hematological response to 300 rads whole-body exposure

What can be done must be done, limited as it may be in terms of saving lives.

Medical Advice to Military Commanders Following Attack

Blast and thermal injury will kill most casualties with significant radiation exposure from high-yield strategic nuclear weapons. However, after attack by low-yield tactical weapons there may be large numbers of military personnel relatively protected from blast and thermal radiation who become casualties principally due to initial radiation or the cumulative effects of fallout and subsequent attacks. The effects from relatively small doses of nuclear radiation may be delayed. This, plus the asymptomatic latent period in the radiation syndrome, may permit some personnel to remain effective long enough to complete specific military tasks. Delayed effects may significantly impair the unit's combat effectiveness for a long period of time, requiring repeated command action to reorganize and reconstitute to maintain any degree of effectiveness.

To make proper decisions, commanders of military units or civil authorities in civilian settings need information on the present and future health of subordinate personnel and affected populations. Prediction of the future health of the command requires an experience factor which does not exist for radiation sickness after tactical nuclear weapon attack. The following information is provided for the medical officer to utilize in working with unit nuclear, biological, and chemical disaster (NBC) personnel in casualty and damage assessment, and to clarify the physician's role as staff medical advisor to command.

The time after exposure before a previously unexposed individual in good health becomes sick or dies depends upon total dose received, dose rate, shielding of parts of the body, and individual body tolerance. Due to the variance in individual tolerance to whole body radiation, the prediction of effect even from a known specific dose is extremely difficult. However, the average effect on a large group may be predicted with enough accuracy for military purposes. Table 6 shows the expected typical responses to radiation of previously unexposed military personnel. The predicted response ranges are based on ED50 (effective dose) casualty criteria: the dose at which 50% of the group will experience a specific effect. This table is equally useful for integrating medical care requirements for dealing with radiation casualties.

Table 7 shows five response categories used to describe reduced combat effectiveness after nuclear attack. Figures 4 and 5 show the expected response to various doses at times after exposure for physically demanding tasks (loading weapons systems) and physically undemanding tasks (fire control center). The dashed line in Fig. 4 shows that after exposure to 1500

Table 6. Biological effects of nuclear radiation

Dose range* cGy (rads)	Onset and duration of initial symptoms	Performance (mid-range dose)	Medical care and disposition
0 to 70	From 6 to 12 h: none to slight incidence of transient headache and nausea; vomiting in up to 5% of personnel in upper part of dose range.	Combat effective.	No medical care; return to duty.
70 to 150	From 2 to 20 h: transient mild nausea and vomiting in 5%–30% of personnel.	Combat effective.	No medical care; return to duty; no deaths anticipated.
150 to 300	From 2 h to 2 days: transient mild to moderate nausea and vomiting in 20%–70%, mild to moderate fatigability and weakness in 25%–60% of personnel.	DT: PD from 4 h until recovery. UT: PD from 6 to 19 h PD from 6 weeks until recovery.	At 3 to 5 weeks: medical care for 10%–50%. At low end of range, less than 5% deaths; at high end, death may occur for more than 10%; survivors return to duty.
300 to 530	From 2 h to 3 days: transient moderate nausea and vomiting in 50%–90%; moderate fatigability in 50%–90% of personnel.	DT: PD from 3 h until death or recovery. UT: PD from 4 to 40 h and from 2 weeks until death or recovery.	At 2 to 5 weeks: medical care for 10%–80%. At low end of range less than 10% deaths; at high end, death may occur for more than 50%; survivors return to duty.
530 to 830	From 2 h to 2 days: moderate to severe nausea and vomiting in 80%–100% of personnel. From 2 h to 6 weeks: moderate to severe fatigability and weakness in 90%–100% of personnel.	DT: PD from 2 h to 3 weeks; CI from 3 weeks until death. UT: PD from 2 h to 2 days and from 7 days to 4 weeks; CI from 4 weeks until death.	At 10 days to 5 weeks: medical care for 50%–100%. At low end of range, death may occur for more than 50% at 6 weeks; at high end, death may occur for 99% at 3½ weeks.

Table 6 (Continued)

Dose range* cGy (rads)	Onset and duration of initial symptoms	Performance (mid-range dose)	Medical care and disposition
830 to 3000	From 30 min to 2 days: severe nausea, vomiting, fatigability. weakness, dizziness, and disorientation; moderate to severe fluid imbalance and headache.	DT: PD from 45 min to 3 h; CI from 3 h until death. UT: PD from 1 to 7 h; CI from 7 h to 1 day; PD from 1 to 4 days; CI from 4 days until death.	1000 cGy: at 4–6 days, medical care for 100%; 100% deaths at 2–3 weeks. 3000 cGy: at 3–4 days, medical care for 100%; 100% deaths at 5–10 days.
3000 to 8000	From 30 min to 5 days: severe nausea, vomiting, fatigability, weakness, dizziness, disorientation, fluid imbalance, and headache.	DT and UT: CI from 3 to 30 min. PD from 30 to 90 min, CI from 90 min until death.	4500 cGy: at 6 h to 1–2 days, medical care for 100%; 100% deaths at 2–3 days.
Greater than 8000	From 30 min to 1 day: severe and prolonged nausea, vomiting, fatigability, weakness, dizziness, disorientation, fluid imbalance, and headache.	DT and UT: CI from 3 min until death.	8000 cGy: medical care needed immediately to 1 day; 100% deaths at 1 day.

* Free-in-air; CI, combat ineffective (less than 25% performance); PD, performance degraded (25%–75% performance); DT, demanding task; UT, undemanding task.

rads the unit will continue to be effective for about 50 min. It will then become performance degraded (PD) and over 4 h decline to the combat ineffective (CI) category. After 2 days most of the group will recover to the PD category but will then decline to CI about 4–5 days after exposure and remain so until death, which can be expected about 12 days after exposure.

On a nuclear battlefield units may have multiple exposures to nuclear radiation. Since the effects of radiation are cumulative, commanders must consider the consequences of using personnel previously exposed to radiation at an asymptomatic level. Table 8 is useful in designating the

Table 7. Responses to acute doses of radiation

Combat ineffective	Combat-ineffective (CI) personnel function at 25% or less of their preirradiation performance level. Combat ineffectiveness is manifested by shock and coma at the high-dose levels. At lower dose levels, combat ineffectiveness is manifested by a slowed rate of performance resulting from physical inability and/or mental disorientation.
Performance degraded	Performance-degraded (PD) personnel, while not CI, function at between 25% and 75% of their preirradiation performance level. They suffer acute radiation sickness of varying degrees of severity and at different times. Radiation sickness is manifested by various combinations of projectile vomiting, propulsive diarrhea, hypotension, dry heaving, nausea, lethargy, depression, and mental disorientation.
Immediate permanent ineffectiveness	IP ineffectiveness is the physiological response from a dose of 8000 cGy (rads). Personnel become ineffective about 3 min after exposure and remain ineffective for any task until death, which usually occurs within 1 day.
Immediate transient ineffectiveness	IT ineffectiveness is the physiological response from a dose of 3000 cGy (rads). Personnel become ineffective for any task about 3 min after exposure and remain so for approximately 7 min. Personnel recover to greater than 75% of their preexposure performance levels after about 10 min and remain so for about 30 min. Then their performance degrades for around 5 h for undemanding tasks or 2 h for demanding tasks, when radiation sickness becomes so severe that they are ineffective. They remain ineffective until death, which usually occurs in 5–6 days.
Latent lethality	LL is the physiological response from a dose of 650 cGy (rads). For physically undemanding tasks, performance degrades about 3 h after exposure and remains so for approximately 2 days, when personnel will recover to combat effectiveness for 6 days or so. Then they relapse into degraded performance and remain so until 4 weeks after exposure when radiation sickness becomes so severe that they are ineffective. They will remain ineffective until death approximately 6 weeks after exposure. For physically demanding tasks, personnel performance degrades about 2 h after exposure and remains so for 3 weeks, when radiation sickness becomes severe enough to render the personnel ineffective. They remain ineffective until death approximately 6 weeks after exposure.

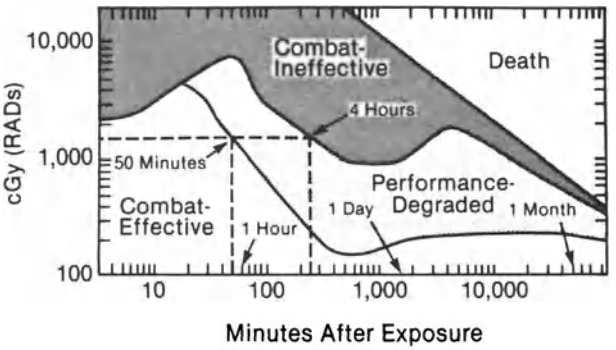


Fig. 4. Expected response to radiation for physically demanding tasks

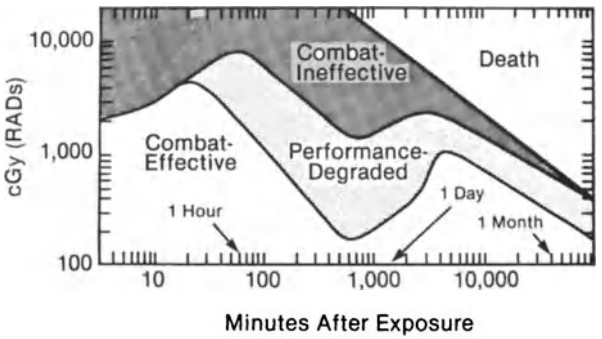


Fig. 5. Expected response to radiation for physically undemanding tasks

radiation exposure status of military units and provides a basis for advising command on the risk of further exposures.

Another topic requiring command policy decisions relates to the issue of the use of radioprotectant drugs such as WR2721 or potassium iodide and antiemetic drugs. While efficacy is claimed under laboratory conditions, adverse reactions can also be anticipated. Current data are insufficient for recommendations.

Table 8. Categories of radiation exposure

RES 0	A unit that has never been exposed to nuclear radiation, a unit which has received no dose.
RES 1	A unit that has received a dose greater than 0 but less than or equal to 70 cGy (rads).
RES 2	A unit that has received a significant but not dangerous dose of radiation, a dose greater than 70 cGy (rads) but less than or equal to 150 cGy (rads). If the situation permits, units in this category should be exposed less frequently and to smaller doses than the units in RES 1 or RES 0 categories.
RES 3	A unit that has already received a dose of radiation greater than 150 cGy (rads); consequently, further exposure is dangerous. This unit should be exposed only if unavoidable because additional exposure in the immediate future will result in sickness and the probability of some deaths.

Appendix 1: Radiation Accident Protocol for Emergency Departments

1. Team captain, or delegate, should obtain on-site information about the accident, including:
   A. Type of radiation accident: irradiation, contamination, and/or incorporation.
   B. Number of uncontaminated victims and their condition.
   C. Number of contaminated victims and their condition.
   D. Type of radioactive isotope(s) — if possible, get sample from site.
2. Prepare for arrival of victim(s):
   A. Evacuate the emergency department if isolated "radiation emergency area" (REA) is not available.
   B. Clear area from ambulance entrance to decontamination room, or REA, of unnecessary patients and personnel.
   C. Prepare pathway from ambulance to REA
      1. Cover route from ambulance entrance to decontamination room (REA) with plastic, paper, or bed sheets and secure the covering to floor with tape.
      2. Mark off above route with ropes and label it "radioactive" until it has been declared safe by the Radiation Safety Officer (RSO).
   D. Make ready the decontamination room or REA

1. If REA's ventilation system is separate from the rest of hospital, turn it off (or call hospital engineer); if not separate, no other precaution may be necessary.
2. Cover floor with plastic, paper, or bed sheets and secure to the floor with tape.
3. Place strip of tape on the floor at the entrance to REA so as to delineate contaminated side from noncontaminated side.
4. Remove all nonessential equipment from room or cover with plastic film.
5. Cover all light switches and handles on cabinets and doors with tape.
6. Provide a suitable decontamination tray for the patient, using an autopsy table, stretcher, or improvise.
7. Provide several large plastic or metal containers with plastic bags to receive discarded contaminated items, such as clothing, gauze, and disposable supplies.
8. Insert batteries in radiation survey instruments and check operation.
9. Survey and record background radiation level in REA.

E. Decontamination team duties
   1. Physician:
      a. Takes charge of medical needs of patient.
      b. Directs decontamination procedure.
   2. Head nurse:
      a. Designates persons who will remain outside REA and obtain supplies for medical and decontamination teams.
      b. Assists physician.
      c. Is responsible for collection of all specimens and swabs of contaminated areas.
      d. Monitors and records vital signs.
   3. Radiation safety officer (RSO):
      a. Designates person who will remain at REA entrance to monitor all personnel, equipment, and samples leaving REA
      b. Monitors patient, decontamination team, and other involved persons for radioactivity during treatment and care of patient
      c. Is responsible for radioassay of all samples of contamination
   4. Circulating nurse:
      a. Assists as needed
      b. Labels all specimens
      c. Picks up and passes on needed supplies that are delivered to the REA from the outside
      d. Records data on areas and levels of contamination as measured by RSO

F. Decontamination team preparations
   1. Use rest rooms in anticipation of prolonged attendance in REA.
   2. Each member labels a film badge with name and attaches it to street clothing.
   3. Each member dons full surgical dress:
      a. Surgical trousers and pullover shirt
      b. Surgical hood
      c. Waterproof shoe covers
      d. Surgical gown
      e. Surgical gloves — tape gloves to sleeves and cuffs to shoe covers
      f. Second pair of surgical gloves — do not tape and change whenever torn or contaminated
      g. Surgical mask
   4. Each member attaches personal dosimeter to outer gown at neck so it will not become contaminated easily; dosimeter is to be read periodically during decontamination and data reported to RSO.

3. Victim's arrival and evaluation.
   A. Stretcher with decontamination basin or improvised trough is brought to the ambulance to receive victim who may be contaminated.
   B. Victim should be given a preliminary examination by physician and RSO — preferably while still in or near the ambulance — for airway, breathing, and cardiovascular status, and to determine the extent of injuries and degree of radioactive contamination.
   C. Administer drugs, fluids, and medical or surgical procedures as needed for stabilization of patient.
      1. If critical, patient — even if contaminated — goes directly to the emergency department or REA without waiting for a radiation survey; do not bother to remove contaminated clothing at this stage.
      2. If not critical and contaminated, patient is removed from vehicle and placed on a stretcher, clothing is removed and left next to the ambulance, patient is covered with a plastic or cloth sheet and taken to the decontamination room or REA. Do not leave the patient wrapped in a plastic sheet.
      3. If not critical and not contaminated, the patient goes, still dressed, to the regular trauma section of the emergency department.
   D. Take swab samples of ear canals, nares, and mouth as soon as possible and before washing or showering.

E. Obtain all other requisite laboratory samples, electrocardiograms, and radiographs according to patient's status.
   1. Blood (for CBC, type crossmatch, and chromosomal analysis); urine (for routine urinalysis, BUN, and serum creatinine concentration); urine and other excreta (for radioassay)
   2. Place samples in separate stoppered containers; label with patient's name, sample site, and time.
   3. Store containers in lead receptacle or at a remote safe place until they can be tested for radioactivity.

F. RSO monitors entire patient, including back; areas and amounts of contamination are recorded on an anatomic chart.

G. Take swab samples of all contaminated areas and store in a similar manner to the above.

H. Ambulance attendants remain with the ambulance until they and the ambulance are monitored for contamination.
   1. If noncontaminated, both are returned to duty.
   2. If contaminated, RSO will give instructions for the decontamination of the persons and/or vehicle before they are returned to duty.

4. Decontamination treatment:
   A. Open wound(s) — attend to first.
      1. Wash wound(s) with normal saline for 3 min.
      2. Monitor for radioactivity — repeat saline wash and resurvey until near background level of radiation or steady state is reached.
      3. If contamination is persistent, wash wound(s) with 3% hydrogen peroxide solution or other appropriate agent.
      4. Cover wound(s) after decontamination if other areas require decontamination.
   B. Eye.
      1. Rinse with stream of water, in nose-to-temple direction away from medial canthus.
      2. Monitor and repeat rinse as needed.
   C. Ear canal.
      1. Swab first, then rinse gently with a small amount of water; suction frequently.
      2. Monitor and repeat rinse as needed.
   D. Nares or mouth.
      1. If possible, turn head to side or down.
      2. Rinse gently with small amounts of water; suction frequently.
      3. Urge patient to avoid swallowing.
      4. If contaminant is known or suspected to have been ingested, insert nasogastric tube into stomach; suction and monitor contents. If gastric contents indicate radioactivity:

a. Lavage stomach with small amounts of normal saline until washings are clear of contaminant.
b. Begin decorporation procedure (see Conklin et al. 1983; Hubner and Fry 1980; Leonard and Hicks 1980; Milroy 1984; NCRPM 1980).

E. Intact skin.
1. Using soap and tepid water, gently scrub with soft brush for a few minutes — do not redden or irritate skin. An ambulatory patient with widespread contamination can be showered.
2. Monitor and repeat washing as needed.
3. If contamination persists:
a. Use a mild, abrasive soap or a 1:1 mixture of liquid detergent and cornmeal or
b. Use Clorox (full strength for small areas, diluted for large areas and around face or wounds).

F. Hair.
1. Wash with mild shampoo for 3 min and rinse.
2. Monitor and repeat washing as needed.
3. If contamination persists, clip off hair — do not shave.

5. Physician should perform complete physical examination and obtain medical history as soon as feasible.

6. Exit of patient from decontamination room:
A. Dry patient.
B. RSO surveys patient's entire body for radioactivity.
C. If RSO is satisfied with results, a clean floor covering is placed from REA exit to patient and, if needed, from exit to a clean stretcher outside decontamination room.
1. Clean stretcher or wheelchair is brought in if patient is not ambulatory.
2. Transfer patient to clean stretcher or wheelchair; attendants should not be those who took part in the decontamination procedure.
3. RSO surveys stretcher and wheels of gurney or chair as it is about to leave decontamination room.

7. Exit of decontamination team:
A. Team members remove their protective clothing in the following sequence at the "clean line" and place it in a plastic container marked "contaminated":
1. Outer gloves are removed first; turn them inside out while pulling them off.
2. Dosimeters are given to RSO.
3. Remove apron.
4. Remove tape at trouser cuffs and sleeves.

5. Remove outer surgical gown; turn it inside out and avoid shaking.
6. Remove surgical shirt.
7. Remove head cover.
8. Pull surgical trousers off over shoe covers.
9. Remove mask.
10. Remove shoe cover from one foot; if shoe when surveyed by RSO is clean, step over "clean line" remove other shoe cover and have other shoe monitored. If either shoe is contaminated, remove it and leave it with contaminated articles of clothing.
11. Face the treatment room, then remove inner gloves and discard them into the labelled waste bag.

B. Survey feet and hands a final time; if still free of radioactive contamination, leave the area.

Appendix 2: The Field Medical System on a Nuclear Battlefield

A military force attacked by nuclear weapons will face a disaster situation which degrades the functional capabilities of all organizational elements (combat, combat support, combat service support). The response to this disaster must be a systematic organizational response based upon prior planning producing policies and standard operating procedures which have been taught and practised prior to the disaster. The military organization must be able to address its tactical mission, damage assessment and limitation, and unit reconstitution in the face of degraded communications and huge losses of personnel and material.

The medical response is one element of the organizational response and must be integrated into and coordinated with the overall response. It will depend heavily upon support from nonmedical elements for communication, transportation, initial first aid, collecting, and primary sorting. It is unlikely that combat would stop, thus enabling concentration of all unit resources to deal with the nuclear attack. It is equally unlikely that medical and other reinforcements will be readily available to assist on the nuclear battlefield. Therefore, very simple and realistic policies and procedures must be developed to utilize maximally all remaining medical capability.

The medical system will be rapidly overburdened by the large numbers of casualties, requiring a system for sorting and holding in addition to the normal procedures of evacuation and hospitalization. These procedures, *sorting* of casualties, often by inexperienced or untrained nonmedical personnel, and *holding* the excess numbers who are too seriously impaired to remain

with their units, but who do not need or cannot be admitted to hospital must be included as part of the essential organization and operations of the medical support system in a field army.
The following are the principal problems facing the field medical system:

Collecting. Unit dispersal on a nuclear battlefield plus degradation of communications systems after attack will be major obstacles to determining the location, number, and type of casualties. As with all other combat casualties, delay in initial first aid and subsequent treatment will increase morbidity and mortality. All personnel on the battlefield must know first aid and each unit must have a standard operating procedure for casualty collection and for restoration of communication to the rear.

Evacuation. Enemy action to exploit the nuclear attack may greatly delay evacuation in frontline areas. Rear areas are also likely to have been struck. Massive casualties would overwhelm existing evacuation means and force unit commanders to consider the use of nonmedical transportation assets which will introduce additional command, control, and patient-regulating problems.

Sorting. Prior to and during evacuation casulties must be sorted to insure that those who will benefit most from hospitalization receive it first. Preplanned functional teams should set up sorting stations along the evacuation routes on the periphery of the attack zone using simple criteria to determine priority of evacuation and destination: hospital or holding area. Presence or absence of radiation injury will be ignored in the primary sorting process since radiation injury is difficult to diagnose from prodromal signs and symptoms and is not a treatable emergency medical problem. The following three groups of patients should be identified:

1. Minimal injuries — neither incapacitating nor life threatening. With minimal or little treatment the patients can continue as partially effective troops. Return to duty or delay.
2. Severe multiple trauma — obviously requiring extensive, time-consuming care with doubtful outcome. These are delayed: time will triage many.
3. Simple injuries requiring immediate intervention — not extensive or multiple with probable favorable outcome if timely initial surgery is provided. These have first priority for evacuation.

Holding Facilities. These should be part of the expansion plan of all hospitals in the field, set up close to parent hospitals with limited equipment and a small number of medical personnel. A variety of patients — those requiring minimal treatment, those unfit for field duty but not requiring full hospital care, and the very severely injured — should be sent there. The following categories are included:

1. Minimal burns
2. Mild trauma cases
3. Severely injured patients not expected to survive who may be kept alive by supportive measures until treatment becomes available.

As nearby hospitals catch up with treatment demands, these patients should be transferred or returned to some level of duty.

Decontamination. Contaminated patients and their equipment are hazardous to themselves and to those around them. Decontamination procedures must be part of every facility's standard procedures, including disposal by burying of contaminated waste in small quantities to avoid hazardous accumulation of radioactive materials.
Decontamination should not delay resuscitation or emergency treatment. Simple removal of clothing and gentle soap and water washing of exposed surfaces will remove 95% or more contamination. Risk to attending personnel is negligible if surgical masks, gloves, smocks, and caps are worn.

Fallout. Subsequent and additional exposure to downwind spread of radioactive material must be limited for all personnel. This is a command action following forecasts of downwind spread, tactical, and logistic factors. Medical advice based upon estimates of additional radiation exposure is required. If evacuation in advance of fallout is impractical, personnel must be protected from the fallout field especially during the first 2–3 days. This protection involves not only shelter but also informal control of exposure while accomplishing tasks requiring exposure. Assignment to these tasks must be rotated among all personnel to minimize individual overexposure. Once the peak radiation as measured by appropriate RADIAC or other instruments has been reached, various tables are available to predict the rapid decay of the gamma radiation hazard.

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2.11 Hyperbaric Medicine

Y. Melamed and S. Bursztein

Introduction

Hyperbaric medicine is concerned with the therapeutic implications of exposure to barometric pressures above 1 atm. The hyperbaric chamber is the therapeutic tool, while oxygen breathed at a pressure greater than 1 ATA (one atmosphere absolute) is the principal drug. There are two patient population groups. The first consists of those who, as a result of their occupation, are exposed to varying ambient pressures, including divers, submariners, pilots, and compressed air workers. The main illnesses found in this group are decompression sickness, pressure injury (barotrauma), and air embolism. Diving medicine is concerned with this group; here the hyperbaric chamber as such is the principal therapeutic tool.
The second group consists of patients who have not been exposed to variations in ambient pressure: the indications are medical, the hyperbaric chamber being used for the therapeutic effect of oxygen at a pressure greater than 1 ATA. The indications include post-traumatic ischemia, gas gangrene due to anaerobic infection, air embolism as a result of a penetrating lung injury, carbon monoxide poisoning, and others. In this second group, the hyperbaric chamber is an adjunctive therapeutic tool to other medical procedures.

Hyperbaric Terminology — Definitions of Pressure

The accepted unit employed to measure pressure, which is force per unit surface area, is the atmosphere (atm). In a vacuum the pressure is 0 (zero) atm, while at sea level the pressure is defined as 1 ATA, equivalent to an air pressure of 1 kg per cm^2, which is 14.7 pounds per square inch or 760 mm Hg. The pressure gauge which serves to measure pressure is calibrated to zero at sea level, so that 1 ATA is equivalent to zero atmospheres on the pressure gauge (gauge pressure 0 gP), which is the relative pressure. Ten meters of sea water produce a pressure of 1 kg per cm^2, so that at a depth of 10 m (33 feet) of sea water, the pressure gauge will read 1 gP (relative) or 2

ATA, or 1520 mm Hg. The rise in pressure inside the hyperbaric chamber (where the pressure is raised by means of compressed air) is equivalent to the increase in pressure obtained by diving below sea level. Increasing the pressure inside the hyperbaric chamber to 20 m of sea water is equivalent to raising the pressure to 3 ATA or 2 gP (relative).
Most of the pressure gauges found in the pressure chamber define the pressure in depth of sea water: MSW (meters of sea water), FSW (feet of sea water), ATA, or in kg/cm^2.

Basic Physiological Principles in Hyperbaric Medicine

When air is breathed at atmospheric pressure, oxygen is transported in the blood bound to the hemoglobin and dissolved in the plasma. The oxygen which is physically dissolved in the plasma is in direct proportion to the partial pressure of oxygen (PO_2; Henry's law). Barometric pressure is composed of the total sum of the partial pressures (Dalton's law): $BP = PO_2 + PCO_2 + PN_2 + PH_2O$.
When air is breathed at normal barometric pressure (1 ATA), the PO_2 in the arterial blood (PaO_2) is 100 mmHg. When 100% oxygen is breathed at a pressure of 1 ATA, the oxygen partial pressure in the arterial blood (PaO_2) will be 675 mmHg (675 mmHg = 760 mmHg −40 mmHg PCO_2 −45 mmHg PH_2O). When 100% oxygen is breathed at an ambient pressure of 3 ATA, the PaO_2 will be 2195 mmHg. (2195 mmHg $PO_2 = 3 \times 760$ mmHg −40 mmHg PCO_2 −45 mmHg PH_2O).
(The values for PCO_2 and PH_2O remain the same, since PCO_2 is a function of alveolar ventilation and PH_2O is a function of body temperature). In a normal man this increase in oxygen content is not always achieved, since there is a certain amount of blood which does not reach the alveolar capillary bed during its passage from the pulmonary artery to the pulmonary vein (shunts). This fact is of the greatest importance when the patient in question has a large right to left shunt (pulmonary or cardiac). For this reason, the PaO_2 must be known in order to evaluate the efficacy of the treatment; it is not always sufficient to know the inspired partial pressure.
At normal body temperature, the solubility factor for physical solution in the blood is 0.022 ml O_2/ml blood per ATA. Therefore, when 100% oxygen is administered in hyperbaric conditions at 3 ATA, the quantity of oxygen dissolved will be about 6 vol%:

$$\frac{(3 \times 760) - 40 - 45}{760} \times 2.2 = 6.3\%$$

The difference in oxygen content of arterial and venous blood is usually about 6 vol %, so that in theory it is possible under these conditions (at rest) to supply the tissues' oxygen requirements even without hemoglobin.

The significant increase in the quantity of oxygen, and its availability to the tissues under high pressure conditions, is of great importance in those situations in which the oxygen-carrying capacity of hemoglobin is impaired (for example in CO poisoning), and in situations in which tissue oxygenation needs to be improved. This is the basis for utilizing hyperbaric oxygen (HBO), in anaerobic infections, in cases of trauma complicated by ischemia, and in diving accidents in which gas emboli obstruct blood vessels, causing local or systemic hypoxia. HBO also causes vasoconstriction in arterial bood vessels. This effect on cerebral blood vessels and the resulting reduction in intracranial pressure it represents is the rationale for the use of HBO in cases of cerebral edema, particularly when brain edema develops in CO poisoning, air embolism, or cerebral decompression sickness. In cases of extensive injury to the limbs and in crush injury, similar tissue edema reduction can be expected.

HBO increases phagocyte and fibroblast activity, and enhances the growth of new blood vessels. These effects are significant in the treatment of infections, in extensive injury with ischemic damage, and in refractory osteomyelitis.

In arterial air embolism resulting from pulmonary barotrauma, or in decompression sickness in which gas emboli in blood vessels and tissues cause the specific signs of the illness, the therapeutic effect is inherent in the very act of increasing the ambient pressure. In these cases, air is sometimes used at higher pressures than those used with oxygen.

Treatment Technique

There are two principal methods of administering HBO. The first is in a small, monoplace (one-person) hyperbaric chamber, where the entire chamber is pressurized with 100% oxygen, and the patient breathes the chamber atmosphere. Another type of small chamber serves as a pressure stretcher for transfer to a large chamber (to transfer diving accident casualties to a treatment center).

The second method utilizes a large chamber containing more than one compartment (the multiplace or walk-in chamber), which is compressed with air. The patient breathes 100% oxygen via a mask or a ventilator at the ambient treatment pressure. This is a spacious chamber, which makes it possible to have medical equipment and staff inside during treatment, and to carry out resuscitation and urgent surgical procedures. The chamber is equipped with communication systems, air conditioning, and the instrumentation required for intensive care.

In most cases HBO is given at pressures of between 2 and 3 ATA for about 90 min. When treating diving accidents, higher treatment pressures are used

and for longer periods of time. The number of treatment sessions and their duration vary with the indication for treatment and the condition of the patient.

Limitations and Complications of Treatment

In HBO one has to take into account technical and physiological factors in order not to endanger the medical team, the operator, and the patient. The principal physiological limitation to HBO is due to the toxic effects of high partial pressures of oxygen. Even within the range of accepted pressures and duration of treatment, toxic effects of oxygen on the respiratory and central nervous systems may appear. Exposure to oxygen pressure above as little as 0.5 ATA for prolonged periods of time is liable to result in the development of oxygen toxicity. The higher the partial pressure of oxygen, the shorter the duration of the exposure that will result in toxicity. The first signs of toxicity are irritation and inflammation of the upper airways and may be followed by the clinical picture of adult respiratory distress syndrome (ARDS). When oxygen is administered at pressures above 2.8–3 ATA, there is a real threat of oxygen toxicity to the CNS, the principal outward sign of which is convulsions of the "grand mal" type. Therefore, HBO is limited to pressures and periods of time below the threshold of CNS or pulmonary toxicity. Recovery periods of air breathing which alternate with the periods of oxygen breathing are included in the treatment profile in order to prevent oxygen toxicity.

Another group of pathophysiological problems likely to arise on exposure to sudden varying pressures is barotrauma (pressure injuries). The damage is mainly caused (according to Boyle's law: $P \times V = K$, where P = pressure, V = volume, and K = constant) in those organs which contain air spaces. The sinuses, the middle ear, the lungs, and the colon will be exposed to the squeeze or overpressure effect should the patient or medical team fail to equalize pressures as the ambient pressure changes.

By taking into account all of the above-mentioned points, hazards are prevented whilst providing effective treatment. From the physiological point of view, entering a hyperbaric chamber is equivalent to underwater diving. To prevent accidents the strict observance of diving procedure instructions and hyperbaric chamber operation procedure is mandatory.

Indications

Indications for treatment are divided into two groups. The first group contains those indications for which HBO is the treatment of choice (as, for example, diving accidents), and indications in which HBO is an adjunct to other conventional medical treatment (as, for example, gas gangrene). The

second group contains those indications for HBO which are still at an experimental stage (a theoretical basis exists, and clinical data have shown that the treatment is beneficial).

Currently accepted indications

Acute/emergency
- Decompression sickness
- Air embolism
- Intoxications (CO, CN)
- Gas gangrene
- Soft tissue infections (anaerobic, mixed anaerobic/aerobic, bacteroides)
- Crush injury with acute peripheral insufficiency (vascular axis, continuity intact)
- Cerebral edema due to head injury
- Thermal burns
- Anemia due to acute blood loss

Acute/subacute
- Compromised skin flaps and skin grafts

Chronic
- Osteomyelitis (chronic, refractory)
- Malignant otitis externa
- Radiation necrosis (osteoradionecrosis, soft tissue radiation necrosis)
- Actinomycosis (refractory)
- Mycoses (mucormycosis, Canibolus coronato)
- Problem, nonhealing wounds/ulcers induced by diabetes, stasis, vasculitis, thromboangiitis obliterans

Investigational indications

Acute
- Intoxications (H_2S, CCl_4)
- Spinal cord and cerebral trauma
- Intestinal obstruction
- Paralytic ileus
- Central retinal artery insufficiency, acute
- Cerebrovascular accident, acute
- Purpura fulminans, secondary to meningococcus or pneumococcus
- Sickle cell crisis

Subacute/chronic
- Ulcer, gastric and duodenal
- Pseudomembranous colitis (c. difficile)

- Radiation myelitis, cystitis, enteritis, proctitis
- Intra-abdominal and intracranial abscesses
- Pyoderma gangrenosum
- Lepromatous leprosy
- Vertigo

Chronic
- Multiple sclerosis (acute and chronic progressive phases)
- Retinopathy, adjunct to scleral buckling procedures in sickle cell peripheral retinopathy and retinal detachment
- Compromised bone grafts
- Fracture healing

Clinical Hyperbaric Military Medicine

The following are those indications which have direct implications for military medicine and traumatology (at base hospital or navy installation): absolute — gas gangrene, arterial air emboli, carbon monoxide poisoning; relative — acute peripheral ischemia, cerebral edema, skin grafts, problematic wounds, burns, osteomyelitis.

Gas Gangrene. The danger of infection in combat injuries is extremely high, particularly those in which definitive treatment in a base hospital is delayed. One of the most dangerous infections is by anaerobic bacteria of the *Clostridium* strain, present in large quantities in the soil and in the gastrointestinal tract. Compound injuries to the skeletal muscles, especially in the lower limbs, may cause a severe impairment of perfusion and tissue oxygenation.

Clostridium is an anaerobic, gram-positive rod bacterium which grows and multiplies very quickly in an environment in which the oxygen partial pressure is below 30 mm Hg. At higher oxygen pressures its growth is contained, as is its ability to produce the alpha exotoxin which damages cell walls and leads to the destruction of the tissues.

This toxin is also responsible for the systemic complications of the disease.

The rationale for HBO is the increase in the partial pressure of oxygen in the blood and the tissues. At oxygen pressures of 250 mm Hg, toxin production ceases. In the case of clostridial sepsis, it is sufficient to increase the partial pressure of oxygen in the arterial blood in order to halt the process. (Breathing oxygen at 3 ATA raises its partial pressure to about 1600 mm Hg, as was explained in the introductory section.)

In clostridial myositis due to a compound injury, myonecrosis, and impairment of the circulation are the prime factors. Shock, edema, and formation of gas from the muscle putrefaction aggravate the perfusion disturbance.

HBO follows upon urgent surgical treatment: wide radical debridement (occasionally life-saving amputation), 20–40 million units crystalline penicillin IV daily, and correction of shock. The HBO is given at 3 ATA for a period of 90 min, with three treatments being given in the first 24 h and two daily thereafter. Improvement is to be expected after the first treatment.
Gas gangrene is characterized by the extremely rapid progression of the infective process; HBO should be initiated as soon as possible, particularly when the clinical picture is dominated by the systemic complications.

Arterial Air Emboli. Pulmonary overpressure injury (barotrauma) and iatrogenic accidents are the two main causes of arterial air emboli. For example, air emboli are likely to be formed during uncontrolled ascent from a dive when there is insufficient exhalation of the contents of the lung. In this case, pulmonary overinflation produces a rupture of the lung parenchyma, and air emboli are released into the arterial circulation. Pulmonary overpressure injury is also liable to result from blast injury, and as a complication of positive pressure ventilation, particularly in cases where there is damage to the lung parenchyma (penetrating bullet wound in the chest or any penetrating lung injury).
In rupture of the pulmonary parenchyma, air may pass directly to the pulmonary veins, and from there to the left ventricle and the rest of the cardiovascular system. The main iatrogenic causes of air emboli are: surgery of the aorta or the arteries of the neck, lung surgery, arteriography, dialysis, and open heart surgery. Air emboli due to pulmonary barotrauma are generally arterial air emboli. Those which are due to iatrogenic accidents are generally venous emboli which are liable to pass into the arterial circulation. When the air embolism creates an occlusion, the main damage is to those organs which are most sensitive to hypoxia — the brain and the heart. The clinical picture depends on the anatomic position of the affected vessel. Occlusion of a cerebral artery will produce signs related to the occluded area, such as loss of consciousness, convulsions, hemiplegia, vision and speech impairment, and so on. Occlusion of the coronary artery will result in a clinical picture of acute myocardial infarction.
The contact between the surface of the air bubble and the erythrocytes causes a number of processes which aggravate the local hypoxia and result in systemic disturbances such as denaturation of protein, aggregation of thrombocytes, activation of the coagulation and fibrinolytic mechanism. It is quite possible that fat emboli may be formed. One of the complications that are almost certain to arise and which have been shown in animals exposed to blast injury, is disseminated air microemboli as a result of pulmonary overpressure injury, with systemic disturbance as mentioned above.
The treatment of air embolism in the hyperbaric chamber is based on two major principles. Exposure to a high ambient pressure causes a physical reduction in the volume of the bubble (Boyle's Law) and accelerates its

dissolution (Henry's Law). Exposure to HBO reduces the partial pressure of nitrogen in the blood, so that the pressure gradient between the nitrogen in the bubble and the blood increases, accelerating its dissolution.
In this indication more than in any other, it is of the greatest importance to administer the therapy with all possible speed. The sooner this treatment is applied after the event, the less irreversible will be the damage and the greater the chances of recovery. The time factor is so important here that a mere suspicion of the diagnosis is a sufficient reason for initiating therapy. The moment there is a suspicion of the diagnosis, it is recommended that the casualty be placed on his left side with the head lower than the body. This is more effective when dealing with an air embolism on the venous side, this position being used in an attempt to keep the air in the right atrium. Then, 100% oxygen must be given as soon as possible, and the nearest hyperbaric chamber should be contacted.
The recommended treatment in the hyperbaric chamber combines treatment at a pressure of 6 ATA for a short period with 100% oxygen starting at a pressure of 2.8 atm. (This schedule is called in hyperbaric terminology "Table 6A" and begins at 6 ATA for 30 min, followed by 75 min at 2.8 ATA, a 30-min ascent to 1.9 ATA, 2½ h at 1.9 ATA, and a 30-min ascent to the surface. This table is designed for air emboli.) In every penetrating chest injury complicated by neurological signs, particularly when positive pressure ventilation is employed, the military physician's first though must be of the possibility of air embolism.

Carbon Monoxide Poisoning. Incidents of carbon monoxide (CO) poisoning occur as a result of fire (smoke inhalation) and breathing in CO, which is to be found in coal gas and automobile exhaust fumes. CO is a gas produced by the incomplete combustion of carbons.
CO impairs tissue oxygenation by a number of mechanisms, competing with oxygen for hemoglobin-binding sites. Its affinity for hemoglobin is 200 times greater than oxygen, and it forms carboxyhemoglobin, a compound that is not available for oxygen. CO which is dissolved in the plasma passes into the tissues by diffusion and competes with the oxygen for cytochrome oxidase A_3, paralyzing the intracellular oxidative system. CO is cleared via the lungs, and only a small amount becomes CO_2.
CO poisoning causes headaches, nausea, vomiting, dizziness, dyspnea, impairment of vision, muscle weakness and convulsions, unconsciousness, and finally, death. A late neurological syndrome is also known to appear days and even weeks after what is apparently a complete recovery, and includes Parkinsonism, incontinence, and mental disturbances. HBO is the treatment of choice.
HBO considerably reduces the half-time for the elimination of carboxyhemoglobin, which normally, when breathing air at 1 ATA, is 5 h and 20 min. With 100% oxygen at 1 ATA, this is reduced to 1 h and 20 min. When breathing 100% oxygen at 3 ATA, this is further reduced to only 23 min.

This fact serves as the physiological basis for giving treatment whith HBO. At high pressure there is an increase in the amount of oxygen dissolved in the plasma, and with this it is possible to supply most of the tissues' oxygen requirements. HBO also accelarates the elimination of the CO bound to the intracellular respiratory enzymes.
The patient's clinical condition is usually a better guide to the severity of poisoning than the carboxyhemoglobin blood level, so that treatment is indicated by the level of consciousness, neurological signs, changes in the ECG, metabolic acidosis, or a carboxyhemoglobin level above 40%.
HBO is particularly effective in preventing the late nervous and mental disorders due to the poisoning. For this reason HBO should be given even after a prolonged delay, as long as the signs justify this.
On evacuation from the site of the accident 100% oxygen should be given until the patient is transferred to the hyperbaric chamber. Treatment is given at 3 ATA for 90 min, and it is repeated every 4 h until all signs disappear.

Acute Peripheral Ischemia. The principal causes of acute peripheral ischemia in military medicine are severe injury to the limbs with damage to a major blood vessel and/or crush injury. When ischemic damage already exists after injury, it is difficult to distinguish between necrotic tissue, partially damaged tissue, and healthy tissue. The purpose of HBO is to save the hypoxic, partially damaged tissue and to make it easier for the surgeon to distinguish between viable and necrotic tissue. Tissue hypoxia is detrimental to basal metabolic processes. The body's defense mechanisms against infection function less effectively, and the wound-healing process is impaired. In the majority of cases, small blood vessels are obstructed by the agglutination of erythrocytes. The plasma which passes the obstruction reaches the tissues beyond, and the tissue hypoxia aggravates itself by a vasodilation reaction distal to the obstruction and an increase in blood volume. Due to the impairment of capillarity and the increased permeability of the blood vessel membrane, there is a leakage of proteins and fluid to the extracellular tissue. Edema is formed and exerts pressure on the blood vessels, thus aggravating the hypoxia. At high partial pressures of oxygen, the accumulation of fluid in the extracellular tissue causes an increase in the oxygen diffusion distance. (When oxygen is administered at high pressure, the dissolved oxygen passes the arterial obstructions by diffusion and reaches the hypoxic tissues.) The diffusion capacity increases in direct proportion to the partial pressure of oxygen, and due to the pressure gradient between the oxygen in the blood vessels and that in the tissue, the function of the hypoxic tissue improves.
HBO also causes vasoconstriction and a subsequent decrease in the leakage of proteins and fluid to the extracellular tissue, and thus a reduction in the intracellular pressure and edema. Tissue oxygenation is maintained due to the high partial pressure and content of oxygen in the plasma. The high

level of tissue oxygenation improves the bactericidal effect of the leukocytes, increases the production of fibroblasts, helps create new blood vessels and speeds up the healing process in the damaged tissue.
It must be kept in mind that HBO is effective primarily in those cases in which the limb's main vascular axis is intact and where some form of collateral blood flow exists. The chances of success with this treatment are good in limbs in which the blood vessels were previously intact.
The earlier treatment is given, the greater will be the chances of success: ideally the treatment should be given within 6 h of the injury. Treatment is given at a pressure of 2.4 ATA for 90 min at least twice in the first 12 h. Effectiveness of the treatment is measured by a rise in limb temperature, changes in color, and a reduction of the edema. Treatment is continued for a long as improvement continues to be observed. When no further improvement is noted in two successive sessions, treatment is discontinued. In cases such as these, one should use tissue oxygen monitoring equipment in order to establish the efficacy of treatment.

Cerebral Edema. One of the serious complications of head trauma and penetrating cranial injury is an increase in intracranial pressure. Intracranial pressure is determined by the sum of the effects of the brain tissue volume, cerebrospinal fluid volume, and intracranial blood volume.
Changes resulting from trauma or severe head injury are liable to influence each of these factors and to increase the intracranial pressure, with all the accompanying complications. HBO causes cerebral vasoconstriction, reducing blood flow, and as a result intracranial pressure drops within a few minutes of initiation of treatment. (This effect can be eliminated, should there be justification for so doing, by adding CO_2 which causes cerebral vasodilation.) In addition to its vasoconstrictive effect which brings about a reduction in the edema, exposure to HBO also causes an increase in the total oxygen content of the arterial blood. It will be found that despite the reduction in cerebral blood flow due to vasoconstriction, tissue oxygenation is improved due to the high PO_2.
Unlike hyperventilation, which also reduces intracranial pressure, HBO does not cause an increase in the lactate concentration in the cerebrospinal fluid. There is as yet no clear-cut answer to the question of whether HBO in these cases is in fact of value when employed in conjunction with other modes of therapy. Patients with cerebral edema are in an extremely serious condition indeed, and there is a grave risk involved in transferring them. For that reason, each case must be examined very carefully. There is no doubt that cerebral edema is an added indication for treatment when an absolute indication such as CO poisoning or cerebral air embolism exists.

Skin Grafts, Problematic Wounds, and Burns. A wound that is in the process of healing requires greater quantities of oxygen than does healthy connective tissue. At the edges of the wound the connective tissue is the site of a great deal of intensive metabolic activity. In every wound there is an in-

flammatory reaction, intensified leukocyte activity in the face of local infection, production of collagen tissue, and the creation of new capillaries. As long as the oxygen supply to the site of the wound is intact, the healing process will proceed normally. In complex comminuted injuries in which there is perfusion impairment at the site of injury the oxygen supply to the area is generally impaired.

The basic process in wound healing is the production of collagen tissue, and in conditions of hypoxia, this process is impaired. The creation of new blood vessels is also an important stage in wound healing and in preventing the rejection of skin grafts. In hypoxic conditions, this process is impaired, whereas in hyperoxic conditions the process is accelerated. HBO accelerates healing in wounds whose blood supply is impaired but has no effect on wounds whose blood supply is intact.

HBO is recommended primarily for those wounds in which there has been a large amount of skin loss, in particular after extensive burns, and where there is little skin available for grafting. It assures maximum success of the graft as well as accelerating healing in the area from which the graft was taken, so that it might be available for further use. This therapy is also effective in cases of chronic wounds in which a vascular problem is involved, by accelerating the formation of granulation tissue at the base of the wound as a preparation for grafting, of as adjunctive therapy after grafting.

Treatment is given once or twice per day at 2.4 ATA, with as many as 20–40 treatments, or more, being given until there is clinical evidence of improvement.

Osteomyelitis. A late complication of bone injury is chronic osteomyelitis, mainly due to infection in areas of anoxia and hypoxia and the reaction of the surrounding tissue. Hypoxia, as already mentioned above, interferes with the healing process, with the production of collagen, and with the bone's capacity to withstand infection. An oxygen partial pressure of 23 mm Hg found in osteomyelitic bone, in contrast with 45 mm Hg in normal bone. When oxygen is administered at a pressure of 2 ATA, an oxygen partial pressure of 104 mm Hg is found in the medulla of the bone. The frequent failure of the usual forms of treatment for chronic post-traumatic osteomyelitis is due to the fact that the various components of the body's natural defence mechanisms and the antibiotics cannot reach the site of the infection in the bone. HBO accelerates the proliferation of blood vessels in the bone, which enables leukocytes and antibiotics to reach the site of infection, while the formation of new bone is also accelerated.

Treatment is given at 2 ATA for 90 min once or twice daily as required, with a total of between 40 and 60 treatment sessions.

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2.12 Infections in the Wounded

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Pathophysiology

The nature and severity of infections following trauma are largely determined by the nature of the trauma itself. The probability of wound infection may reflect the character of the wounding instrument; knife vs bullet, high velocity vs low velocity missile, etc.
Infection does not follow trauma merely because of the entry of infectious material into tissue. Trauma is accompanied by other factors such as a hypermetabolic state, tissue necrosis, shock, foreign bodies (including catheters), and hematomata which significantly interfere with host immunity.

1. The presence of necrotic muscle in contaminated wounds favors the growth of anaerobic bacteria, including *Clostridium perfringens* and *C. tetani.*
2. Injudicious use of silk sutures, drains, and cautery may promote growth of a variety of pathogens and limit local host response to infection.

The incidence of wound infection is related to the time which has elapsed before debridement and antibiotic administration. The incidence of gas gangrene increases dramatically with delays of more than 24 h.
Dehydration and hypovolemia from blood loss are common given the setting of the battlefield or disaster. Administration of large amounts of fluid is crucial not only to prevent and treat shock in such patients, but also to the prevent exacerbated infection due to low tissue perfusion.

Classification of Wound Cleanliness

The initial evaluation of wound infection should consider the wound itself: laceration or puncture, penetration of viscus, compound or simple fracture, etc. The incidence of wound infection is related to the "cleanliness" and circumstances of injury as follows:

1. *Clean.* Gastrointestinal, urinary or respiratory tract not entered. No inflammation, contamination, or break in aseptic technique. Infection incidence about 1.5%.

2. *Clean/contaminated.* Gastrointestinal or respiratory tract entered, without significant spillage. Infection incidence about 7.7%.
3. *Contaminated.* Without pus or gross spillage, or following lack of aseptic technique (most fresh traumatic wounds fall into this category). Infection incidence about 15.2%.
4. *Dirty.* Pus or contents of hollow viscus contaminate wound area. Delayed treatment of trauma (>4 h) should also lead to considering the wound as "dirty." Infection incidence about 40.0%.

The concept of "clean wounds" is restricted to the hospital environment. Open trauma is always "contaminated" or "dirty".

Bacteriology

Coliform bacteria, *Staphylococcus aureus,* and *Pseudomonas* account for 35%, 24%, and 13%, respectively, of the pathogens isolated from patients infected after major trauma. Anaerobic bacteria are common in the setting of colonic, orofacial, and vaginal injury.

Aerobic and facultative gram-negative bacilli have emerged as important battlefield wound pathogens in the antibiotic era. Such species are commonly resistant to traditional and standard forms of prophylaxis and therapy.

Clostridia (C. tetani, C. perfringens, etc.) and other "classic" surgical pathogens such as group A streptococci and *Staphylococcus aureus* should always be considered in wound infection.

Factors such as terrain and season influence the flora of battlefield wounds. For example, wound infection by *Pseudomonas pseudomallei* was encountered from the rice paddies of Viet Nam; *Vibrio vulnificus* and *Aeromonas hydrophila* often infect marine injuries.

Local diseases such as malaria, typhus and a variety of arthropod-borne (Arbor) virus diseases pose a threat in specific areas of the world.

Epidemiology

The most common sites of infection in severely traumatized patients are the urinary tract and lungs (20% and 25% of total infections, respectively). Infections of intravenous devices accounts for 12% and wound sepsis only for 10%. Ten percent of infections are characterized as "primary bacteremia"; i.e., bacteremia without an apparent source.

The flora of military wound infections often reflects the local flora of the battlefield.

Placing the traumatized patient into an intensive care environment is a "double-edged sword". Although intensive care provides for efficient monitoring and treatment of such patients, the risks for superinfection are often

extreme. As many as 75% of individuals in a surgical intensive care unit can be expected to acquire infection within 10 days; 90% will be found to carry nosocomial bacteria.
Infection begins with contamination by a potential pathogen, progressing to overt wound infection over a period of days. Infection due to gram-positive cocci is usually encountered 2–7 days following trauma, and gram-negative bacillary infection, 8–14 days. Infection by group A *Streptococcus* or *C. perfringens* may become evident within 12–24 h!
Local infection may evolve into a locally expanding process or enter the blood and lymphatic systems, seeding multiple organ systems.

Management

The *prevention* of infection involves debridement (surgical removal of hematomata, devascularized tissue, foreign bodies, etc.), sanitation, hygiene, antibiotic prophylaxis, and vaccination. The *treatment* of infection requires rapid diagnosis, antisepsis, sanitation, isolation if necessary, judicious disposal of infected materials, radical wound excision and drainage repeated as indicated, and appropriate antibiotic treatment.
The management of treating infection is determined by the nature and extent of the casualty situation, geography, economics, and the availability of resources and personnel.
The value of adequate debridement followed by delayed primary suturing cannot be overemphasized.
Shock may supervene in even the most minor of wounds and infections. Strict attention to adequate hydration and monitoring of vital signs are the key to the prevention of such a complication.
The use of antibiotic prophylaxis in some forms of trauma remains controversial. Most authorities agree that prophylaxis should be (a) directed at likely pathogens, (b) adminstered promptly, and (c) continued for as few days as possible. In patients with overtly contaminated wounds, the border between prophylaxis and therapy is indistinct. Indications for prophylaxis are based on the nature of the wound itself. In general, antibiotics should be routinely administered to patients with "contaminated" or "dirty" wounds (e.g. all war wounds).
Basic guidelines for preoperative prophylaxis can be summarized as follows: for cardiovascular, orthopedic, head and neck, gastric, biliary, vaginal, and clean soft tissue trauma, a single dose of cefazolin (1.0 g for an adult) may be administered; colorectal trauma requires additional coverage for anaerobic bacilli and enterococci, e.g. clindamycin (600 mg) *plus* gentamicin (1.5 mg/kg) *plus* ampicillin (2 g). Continued administration following surgery sould be tailored to operative findings and subsequent evidence of infection.

General Principles of Site Management

1. Prevention of battlefield wound infection is by good local care. The incidence of wound infection during the past 5 decades has not been reduced through the introduction of prophylactic antibiotics in the field.
2. Penetrating and crushing injuries should be considered as infected a priori. Burns, closed trauma, and superficial wounds are considered as potentially infected. In general, about 65% of wounds fall into the first category.
3. Planning should include the availability of adequate stocks of a small number of antibiotics which remain stable in spite of prolonged storage and extreme environmental conditions. We recommend three basic antibiotics for field use: penicillin G in vials of 5 million units, chloramphenicol in vials of 500 mg, and gentamicin in vials of 80 mg.

Immunization

The maintenance of stocks of tetanus toxoid and human antitoxin is a basic necessity. Certain disasters create the demand for other, often exotic, vaccines. Major emergencies are often accompanied by a breakdown in hygiene, refrigeration, and drinking water control. Epidemics are common in such settings, particularly in developing countries.

1. Tetanus toxoid (0.5 ml) and human tetanus immune globulin should be administered as in Table 1.

Table 1. Immunization plan in field trauma

Full toxoid series	Last booster	Condition of wound	Boost tetanus toxoid	Human tetanus immune globulin
Fully immune	Within 1 year	Any wound	No	No
Within 10 years	None	Clean, fresh	Yes	No
More than 10 years	Within 10 years	Clean, fresh	Yes	No
	≥ 10 years	Clean, fresh	Yes	No
	≥ 5 years	Unclean, old	Yes	250 µl
Questionable	Questionable	Clean, fresh	Yes[a]	No
		Unclean, old	Yes[a]	500 µl

[a] Administer complete toxoid series. Note that a toxoid series should also be administered following treatment of clinical tetanus. *Tetanus infection does not confer immunity!*

2. Pneumococcal vaccine is indicated following splenectomy for any reason.
3. Prevention of cholera, typhoid fever, typhus, and viral hepatitis is by immunization with specific vaccines and the use of pooled gamma globulin. Hygiene includes strict control of drinking water and food storage, establishment of latrines, application of insect repellent, and the prompt burial of the dead. These are essential measures to prevent the spread of food poisoning and other infections in the field.

Specific Forms of Trauma and Wound Infection

A guide to the choice of antimicrobial agents and duration of antimicrobial therapy for the various types of wounds and infection is given in Tables 2 and 3.

Orthopedic Injury. Infection develops in 11% of patients with open fractures and 3% of those with closed fractures subjected to open reduction. (Most of the latter are due to hospital acquired bacteria.) Careful debridement and irrigation are indicated; the wound should be left open if possible.

Table 2. Choice of antimicrobial agents for pathogens which cause wound infection

Organism	Therapy[a]	
	First choice	Second choice
Acinetobacter calcoaceticus	imipenem	aminoglycoside
Bacteroides (non fragilis)	penicillin G	clindamycin
Bacteroides fragilis	clindamycin	metronidazole
Clostridium perfringens	penicillin G	tetracycline
Escherichia coli	ampicillin	cephalosporin
Other *Enterobacteriaceae*	cephalosporin	piperacillin
Pasteurella multocida	penicillin G	tetracycline
Pseudomonas aeruginosa	tobramycin + piperacillin	amikacin + ceftazidime
Staphylococcus aureus	nafcillin, etc[b]	vancomycin
-methicillin-resistant	vancomycin	imipenem
Streptococcus pyogenes	penicillin G	erythromycin
Streptococcus faecalis	penicillin G	vancomycin

[a] Assuming in vitro susceptibility. Dosage and route of administration will largely depend on body weight, severity, and site of infection. Intravenous therapy is advised in initial stage of all major forms of wound infection.

[b] Penicillinase-resistant semisynthetic penicillin.

Table 3. Duration of antimicrobial therapy[a]

Infection type	Duration
Bacteremia, following removal of focus	14 Days
Osteomyelitis – acute	6 Weeks
– chronic	Months[b]
Gas gangrene	10 Days
Septic arthritis	3 Weeks
Skin and fascial infection	10 Days
Urinary tract infection related to catheter	10 Days

[a] Duration of antimicrobial therapy after resolution of acute anatomic abnormality, removal of catheters, debridement, drainage, etc.

[b] The therapy of chronic osteomyelitis is primarily surgical; i.e., removal of sequestra and pus.

Although *Staphylococcus aureus* and streptotocci continue to be major pathogens, facultative gram-negative bacilli are also increasingly isolated. *Pseudomonas aeruginosa* can be found, particularly following trauma to the lower extremities.

Abdominal Injury. The overall infection rate in penetrating wounds of the abdomen has been estimated at 22%; 46% if three or more organs are injured. Fifty percent of the mortality in penetrating colon injuries is ascribed to infection, with intestinal coliform and anaerobic bacteria predominating. Increased risk of infection is associated with shock on admission to hospital, or with injury to the left colon necessitating colostomy.

Head and Neck Injury. Trauma to the eyes or brain is prone to infection. In contrast, intraoral injury is extremely resistant to infection. Injuries which compromise respiration or deglutition may be complicated by severe, recurrent pulmonary infection.

Cardiothoracic Injury. When chest injuries are treated promptly, with drainage of hemothoraces, wide debridement, and delayed closure, an infection rate of less than 5% may be achieved. Primary thoracotomy is associated with an infection rate as high as 30%.

Burn Infection. The incidence and severity of infection following thermal burns is related to the extent and depth of the burn. Burn patients suffer from diminished T-lymphocyte responsiveness, chemotaxis, phagocytosis, and opsonization of bacteria. The prevention of burn infection requires careful maintenance of a clean environment, particularly during the many dressing changes, debridements, hydrotherapy, etc., to which the patient is exposed. The hands of medical personnel are an important source of infection. *There is no substitute for strict asepsis.*

Although penicillin and other antibiotics have been routinely administered to patients with extensive dermal burns for decades, the usefulness of such prophylaxis has been questioned in recent years. It may be prudent to restrict routine antibiotic use to the immediate period surrounding burn debridement and other manipulative procedures in such patients. Silver sulfadiazine and similar agents are effective in reducing the concentration of local bacteria. The effectiveness of mafenide is relatively low, and the agent is rarely used nowadays. Bacteriological cultures of small biopsies taken as frequently as every other day may be helpful in selecting specific antibiotic therapy.

Radiation Injury. Injury due to ionizing radiation may be complicated by major infection through several mechanisms, including thermal damage to skin and mucosae, gastrointestinal damage, and immune suppression due to death of immunocytes. Sepsis following irradiation is generally due to endogenous coliforms, *Pseudomonas aeruginosa,* and *Staphylococcus aureus.*

Crepitant Soft Tissue Wounds. Crepitation in soft tissues following injury may represent either a gas-forming infection or the presence of air from a contiguous wound, viscus, or lung. At least seven forms of wound infection can be described, all associated with local swelling and the presence of a foul or "sour" odor:

1. Clostridial cellulitis is characterized by gradual onset, mild pain, and slight skin discoloration. Systemic toxicity is minimal and a copious, thin discharge is present.
2. Anaerobic cellulitis is occasionally encountered in diabetics or superimposed on a chronic wound infection. *Bacteroides,* anaerobic streptococci, and enteric gram-negative bacteria may be isolated in pure or mixed culture.
3. Streptococcal myositis follows local trauma or surgery and is typified by marked pain and erythema associated with an abundant purulent discharge.
4. Necrotizing fasciitis often involves the abdominal wall or perineum and is characterized by severe swelling and pain with marked systemic illness. A mixture of aerobic and anaerobic bacteria is usually identified.
5. Infected vascular gangrene complicates vascular insufficiency of the lower extremities. The lesion is discolored and foul smelling; systemic signs are minimal.
6. Synergistic necrotizing cellulitis may complicate diabetes mellitus, perianal infection, and edema or obesity due to other diseases. The patient is extremely ill, and a foul, "dishwater" pus exudes from the wound. A mixture of anaerobic and enteric bacilli are usually present.
7. Clostridial myositis (gas gangrene) occurs after trauma or surgery. The onset is acute; pain, swelling, and systemic toxicity are marked. Fever is

not prominent. A thin discharge is noted, often with gas bubbles. Underlying muscle is nonviable; hemoglobinemia and shock may evolve within hours.

Crepitant soft tissue infections require wide incision and debridement. Antimicrobial therapy is directed by Gram's stain; initial coverage of facultative and anaerobic bacteria should be included pending culture. The therapy of gas gangrene consists of intravenous penicillin G (2 million units every 3 h) and early debridement, decompression, and amputation as necessary. The efficacy of antitoxin has not been established. Hyperbaric oxygen appears to be of most value for gas gangrene of the trunk, but may be employed for other forms of severe anaerobic infection if available.

Tetanus. The introduction of *Clostridium tetani* into a deep wound, often in a mixture of bacteria, leads to tetanus. After an incubation period of 3–14 days, the patient develops trismus followed by generalized spasms of skeletal muscle. Mild and localized forms are recognized. The diagnosis is based on clinical signs. Attempts to isolate the organism are generally unsuccessful. Therapy includes early institution of an airway (tracheostomy), intravenous hyperalimentation, sedation, and administration of a paralytic agent, such as tubocurarine. Careful attention to the nutritional and metabolic status of patients is crucial. The prevention of tetanus is discussed above.

Mass Injury — Military Versus Civilian

No sector is more experienced in the treatment of infection in a mass casualty situation than the military. The applicability of this experience to civilian casualties is somewhat limited by the unique nature of battlefield medicine.

In the field, a wounded soldier is likely to receive immediate attention and transport, including rapid cleansing and bandaging of the wound with appropriate administration of antibiotic prophylaxis, tetanus toxoid, etc. The soldier is most likely to be a young male previously in good health; however, he is likely to be dehydrated on presentation to a medical facility. Infectious disease considerations are primarily limited to traumatic wounds and/or burns.

The civilian may be of any age and of indeterminable nutritional and health status. The spectrum of possible infection will range from gas gangrene to cholera. Transport, wound care, and antibiotic administration largely reflect local resources and priorities which are heterogeneous. The situation is not helped by the unanticipatable nature of mass disasters.

2.13 Combat Stress Reaction

A. Shalev and H. Munitz

Definition

Combat stress reaction (CSR) is the prototype of psychological trauma. It is the immediate result of a failure to cope with combat stress and is typically characterized by an acute and severe reduction in the patient's functional capacity and by a subjective experience of overwhelming anxiety and inescapable threat. The incidence of CSR in modern wars has been 10%–22% of the total number of physical injuries. It has a typical course, the knowledge of which lends itself to primary prevention, early detection, and appropriate treatment. In 30%–45% CSR results in an incapacitating chronic syndrome: the posttraumatic stress disorder (PTSD). Specific attributes of battles that contribute to CSR are well recognized and may eventually be reduced or prevented. Early and prompt therapeutic intervention is the condition sine qua non for success in the treatment. CSR responds better to treatment which is conducted near the front. Evacuation of CSR patients to the rear should, as much as possible, be avoided.

Overview

Extreme behavioral and emotional reactions to combat have been known for centuries. They can be divided into two groups: one is an immediate reaction to combat events (CSR) and the other is a prolonged condition that continues for a long time after the battle (PTSD or stress response syndrome). The correlation between the two is not simple: CSR does not always lead to PTSD and, vice versa, PTSD can develop without being preceded by CSR (late onset PTSD). PTSD has been the subject of many descriptions and evaluations during the last decade, specifically with relation to its occurrence in post-Vietnam veterans. Acute CSR does not receive that amount of attention. As CSR is the condition which prevails on the battlefield, it is more appropriate to consider it in the framework of a manual on war and disaster.

During the 20th century CSR was successively named "shell shock" (World War I), a term that emphasized a hypothetical role of the blast effect of shells; "war neurosis" (World War II), a term that implied a causal role to intrapsychic conflicts activated by the situation of war; and "battle fatigue" or "combat exhaustion", a term that was introduced, for administrative rather than professional reasons, during the second half of World War II and relates to the pervasive role of exhaustion during prolonged operations.

The concept of combat stress reaction stems from the theory of stress. It refers to the effect of both physical and psychological pressures on the individual, emphasizes situational factors rather than psychological traits or liabilities, and assumes a phase of attrition before the onset of the acute syndrome. The theory of stress assumes, in addition, the presence of a specific activity aimed at reducing the impact of external pressures (coping).

Coping includes all the ways by which a subject attempts to reduce personal distress, preserve psychological integrity, and pursue organized activity. It includes intrapsychic mechanisms (e.g., denial of danger, rationalization of suffering, control of emotions); external activities (e.g., reaching out for help, information, and support), and complex internal and external dispositions (e.g., magic faith in a leader, passive surrender). Passive surrender, which in this context means an inhibition of aggressive responses to external offenses, is one of the most typical strategies of coping with personal discomfort and distress in a military setting. Effective coping is evaluated by its efficacy in achieving the following four goals: relief of personal distress, maintenance of a sense of personal worth, conservation of the ability for rewarding social contacts, and sustained capability to meet the requirements of the task.

CSR expresses itself as a dysfunction in these above-mentioned areas: an increase in distress, a subjective sense of inadequacy, loneliness, and guilt, social detachment and isolation, and increasing difficulties in task accomplishment. In addition CSR includes a massive decrease in the patient's ability to adapt and regulate his behavior. This deficit manifests itself in decreased stimulus tolerance, inappropriate response to social clues, and disregulation of affect. The presence of "paradoxical reaction" is an example of this deficit: the patient, who seems indifferent to massive stimulation (e.g., shelling or shooting), may show strong and sudden reactions to minimal stimuli such as minor social frictions or minimal noises.

Pathology of Combat Stress Reaction

Pathophysiology

The pathophysiology of CSR resembles a homeostatic system under excessive demands. In such a system the acute failure is preceded by a phase of formal equilibrium and functional resilience that masks a progressive exhaustion of buffers and resources. Such a premonitory stress reaction is often present to some degree before the externalization of CSR — sometimes even before the onset of combat.

Another point is the subjective nature of the appraisal of stressful situations. Beyond its similarity to a homeostatic system, the impact of a psychological stressor on the patient depends largely on the way in which it is perceived and evaluated (i.e., its appraisal). Similar situations can be very stressful for one person and much less so for another according to the way in which they are perceived and analyzed.

Appraisal has, in fact, two directions: one is the evaluation of the threat involved in the situation, and the other is the evaluation of available resources. This process leads to a global evaluation made of the chances of success in coping with the situation. In extreme stress this would result in an overall "fight or flight" reaction (or, in modern terms, challenge versus avoidance). The quality of appraisal depends on the integrity of the subject's cognitive function (i.e., his capacity to think clearly, concentrate, shift attention, scan possible alternatives, and plan in advance). These cognitive functions are largely dependent on the subject's state of arousal which, itself, depends on the existing physiological (e.g., dehydration, insomnia) and psychological distress. The result is the vicious circle of stress, high arousal state, diminished cognitive resources, and distorted evaluation of the situation presented in Fig. 1.

The end result of this vicious circle can be a state in which even minor events related to the combat are appraised as conveying an imminent, ines-

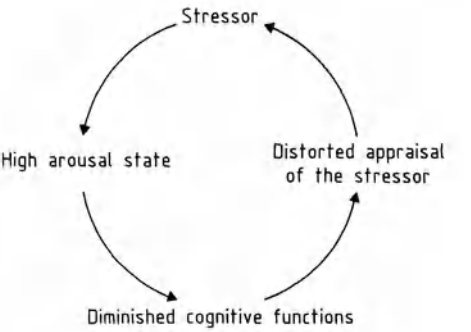

Fig. 1. The vicious circle of stress and appraisal in combat stress reaction

capable, and intolerable threat against which no effective action can be conceived. This is the case of acute CSR.
If such a condition lasts for a long time (that is, if the subject is left in a state of extreme fear and arousal) a traumatic reappraisal occurs, in which the entire coping capability of the subject is discredited. This results in the installation of stimulus avoidance (flight) as the dominant and permanent response to stress. This is the case in prolonged CSR and the beginning of PTSD. The process of traumatic reappraisal can take from a few hours to several weeks. During this critical time an external intervention can reverse the process. Later on, one can expect only partial results. After 6 months, the resulting PTSD is a chronic condition.
Social support has a great role in reversing the process of traumatization. The soldier in war is, in fact, involved in a massive group activity on which depend all his resources (information, orientation, food, mode of activity, protection from threat, etc.). Group factors, therefore, have a cardinal role in modulating the level of external demands on the individual and that of his subjective appraisal and distress. Often one individual's appraisal, mode of coping, and personal distress reflect those of his reference group.
Once CSR has started, the same group factors, by validating his self value, accepting him as peer, cutting short his isolation, etc., can support the individual in his attempts to reverse the process of traumatic reappraisal.

Etiological Factors

In evaluating the etiology of CSR, one should consider the characteristics of the agent and those of the host. CSR may start either after a unique, intensive and unpredictable event or follow the progressive accumulation of pressure. Some conditions were shown to favor the development of CSR. They include the intensity of the combat, the number of deaths and physical injuries, the unpredictability of a specific event, the lack of social support, lack of clarity of information, and enforced passivity:

1. Factors related to the conditions of combat:
   Intensity
   Unpredictability of stressor
   Lack of clarity in information
   Failure of leadership, death, or replacement of a leader
2. Psychological factors related to the subject's task:
   Isolation from the basic unit
   A new soldier in a unit
   Passive role (drivers, technicians)
   Lack of adequate military training for the actual role
   Inability to sustain denial: overexposure to casualties, to atrocities, death of friend or relative

3. Physiological conditions:
   Deprivations: sleep, food, water
   Exhaustion due to weather conditions

The role of the military unit as a buffer and protector from stress can hardly be overemphasized. Adequate training, high motivation, cohesiveness within the group (esprit de corps), and effective leadership are the best protectors of the individual against personal trauma. Predisposing factors related to the personality of the soldier are less important then those related to the conditions of the battle. The preventive elimination from the army of hypothetically predisposed subjects failed to reduce significantly the incidence of CSR. Predisposed subjects are more prone to develop chronic PTSD, but in acute CSR actual pressure plays the dominant role.
Acute CSR can occur in any person exposed to combat. Consequently, no soldier with CSR should be considered as presenting a trait of mental inadequacy. Similarly there is no solid ground to look for a record of psychopathology in CSR patients: in the vast majority the result of such an inquiry will be negative. Factors from the subject's history that have been shown to contribute to CSR are recent major life events and CSR in a previous war.

Principles of Treatment

The object of the treatment of CSR is to prevent or reverse the traumatic reappraisal and the resulting stimulus hypersensitivity and avoidance. The treatment of CRS is designed to stop the transformation of a stressful event into a traumatic event. It follows two successive stages:

1. An initial withdrawal from the stressful situation, allowing a decrease in arousal and an improvement in the cognitive ability.
2. A deliberate effort to help the subject to evaluate the situation and his own coping resources in a positive way.

An attitude of rejection and denigration towards a soldier with CSR will increase his sense of failure and will diminish, or destroy, his attempts to regain his capacity to accomplish his combat role. Many still tend to consider CSR as a trait of permanent weakness or, worse, as a form of madness; consequently, they develop rejecting attitudes which are fueled by the pressure of urgent tasks and the sight of the physically wounded as opposed to CSR patients. However, the correct approach is a readiness to reintegrate a soldier into his unit.

Clinical Presentation

The natural course of CSR includes a premonitory phase, an acute phase, a stabilization phase, and a final common pathway known as PTSD. The intensity, duration and severity of the stressor determine the length or even the presence of each of these phenotypic reactions.

Premonitory Phase

The typical signs of the premonitory phase are:

High arousal
- Restriction of field of interest
- Inability to relax
- Inability to shift attention
- Inability to concentrate
- Disrupted decision making

Lack of emotional control
- Irritability
- Impulsive responses to stimuli
- Uncontrolled emotional discharge

Diminished social interaction
- Withdrawal, isolation
- Loss of sense of humor
- Loss of affective adaptation to others
- Sustained criticism and mistrust

Physiological manifestations of anxiety
- Diarrhea, nausea, tremulousness
- Weakness, cold sweating
- Headaches, palpitations

Unexplained physical complaints serving as a pretext for any consultation.

At this phase it is sometimes difficult to distinguish between CSR and reactions of fear and anxiety that are common during combat. Many soldiers are, at times, tense and restless and experience anxiety and fear. There are, however, clinical signs that allow a distinction between the "normal" reaction to combat and CSR in *status nascendi*. The principal indicators are the following:

1. Emotions are strong enough to interfere with task accomplishment.
2. The level of distress of the individual is significantly more pronounced than that of others who are exposed to the same conditions.
3. Tension is beyond the subject's control and, very typically, does not lessen during periods of relaxation in combat.

4. The subject's behavior or responses seem to others different from his usual character.
5. The subject himself becomes detached and isolated. He "loses contact", can no longer adjust his emotional tone and his level of activity to those of others (e.g., does not laugh with others, wanders around when everyone is at rest, stays apart while others are together, etc.).

Other unit members are, often, those who recognize the change and bring the soldier to the attention of the medical staff. The identification and treatment at that stage are of the utmost importance and their efficiency is maximal. Most of the time soldiers in this phase will recover after having the opportunity to rest and restore any physiological deficit (in sleep, food, water, protection from extreme weather conditions).
The shift from this phase to the acute phase often follows an additional event to which the subject fails to respond and, therefore, develops symptomatic behavior. This last event is often described as the cause of CSR, but a careful inquiry often reveals a premonitory phase before the onset of the acute phase.
Spontaneous recovery from this phase is also possible — if the battle is over. If not, every additional stress can revert the individual into the acute reaction described below.

Acute Phase

In the acute phase the functional deficit is total, and the subject is overwhelmed by a sense of inescapable catastrophe. The presence of gross psychiatric symptoms is the rule:

Cognitive impairment
  Dissociative states
  Confusion and disorientation
Impaired stimulus response
  Overreactiveness to stimuli
  Inappropriate response to minor events
Psychomotor symptoms
  Restlessness and agitation or
  Stupor and motor retardation
Affective symptoms
  Anxiety, panic, terror
  Sadness, guilt, shame
  Perplexity, stupefaction, shock
Conversion symptoms
  Paralysis, blindness, muteness

Stupor, panic, various conversions, or dissociative states can occupy the scene. Sadness, withdrawal, or on the contrary agitation and restlessness are

frequent. Overreactiveness to minimal stimuli is often present. Confusional states merit particular attention: they often include both disorientation and overreactiveness which can lead to indiscriminate exposure to real danger. Psychosis rarely occurs, and distortion of reality testing is rare. This should be remembered because it means that even at that phase the subject is responsive to external contact and can be reassured, oriented, and encouraged. The subject is often unable to process, use, or answer adequately to verbal efforts made in order to reach him but preserves his capacity for contact and for recognition of the affective tone of the contact. Subjects are particularly responsive to the warmth of human touch and can be easily handled that way. Violent body treatment is, on the contrary, of no help and increases the subject's fear and withdrawal. Vigorous maneuvers such as slapping, beating, or shaking the subject should be avoided.

Stabilization Phase

The stabilization phase occurs within several days or weeks. It is often seen by the primary physician at the end of military operations as the first manifestation of CSR either in subjects who could handle the acute reaction without medical help or in those whose CSR developed insidiously. This is often the case of people in positions of command who "cannot afford" to breakdown as long as active operations continue. Another circumstance which favors this type of reaction is the first contact with the family at home, particularly the first leave.

The symptomatology of this late reaction is midway between acute CSR and PTSD. It is characterized by the presence of affective symptoms (depression, guilt, shame) along with intrusive thoughts or vivid imageries of an event or scenes from battle. Sleep disturbances are frequent with ensuing fatigue and nervousness.

This condition should be distinguished from normal grief reactions that accompany at that phase the realization of losses. Good indicators of CSR are the persistence of intrusive memories and imageries over time and the presence of nightmares.

Final Common Pathway: The Posttraumatic Stress Disorder

In 45% of the subjects CSR results in PTSD. All the clinical varieties of CSR, irrespective of their initial form, converge into that "final common pathway". Neither the clinical picture of acute CSR nor the intensity of the disorder involved predict PTSD. PTSD, however, occurs with a higher incidence in soldiers with previous psychopathology (including CSR from previous war) and in those who are evacuated and treated in the rear. The significance of the last finding is difficult to evaluate because the evac-

uation to the rear is often a result of intractable CSR and not its cause. There are, nevertheless, strong indications that evacuation contributes, by itself, to the development of PTSD.
PTSD is a pervasive and chronic condition which is associated with a massive disruption of the social, familial, and professional life of the patient. It is considered incurable after 6 months of evolution (chronic PTSD according to American Psychiatric Association's Classification of Mental Disorders, 3rd ed). The description of PTSD is beyond the scope of this manual. It is, nevertheless, important to remember that PTSD is the potential end result of CSR and that the real challenge of treating CSR is the prevention of PTSD.

Diagnosis and Treatment

The treatment of CSR can be defined as a deliberate effort to reestablish preexisting psychological homeostasis of the subject by providing temporary relief from stress along with biological and social support. The following principles should be observed:

1. Compensate physiological needs
2. Achieve temporary relief from external sources of stress
3. Use human contact in order to prevent the soldier from engaging in the process of psychological trauma, i.e., in a reappraisal of the situation as catastrophic and in a massive devaluation of his own resources and values. Reassure, clarify, allow sharing of emotions, humanize and legitimize fears, allow expression of guilt for actions or omissions, confront self-depreciation but respect personal values and beliefs
4. Share with the patient an expectation for full recovery and return to duty
5. Promote social support that will allow reintegration of the subject in the same unit and, if possible, the same role
6. The sooner one treats CSR, the better are the chances to reverse the process of traumatization
7. Do not evacuate unless absolutely necessary

CSR involves a massive decrease in the subject's ability to adapt to new situations and to new roles. Any change in the subject's status (such as evacuation to the rear or changes in his military role) should, therefore, be considered as potentially harmful. The benefit of withdrawal from the direct source of stress should be weighed against the severely disturbing effects of such a change. As a rule, the first station at which temporary protection from the direct source of stress can be achieved is the one at which treatment should take place. This first station may be among the soldier's buddies in the field, in the battalion first aid station or in the field hospital. The

status of the soldier as a member of a combat team should not be changed unless the proper efforts designed to reverse the traumatic effects of stress have been carried out.

Prevention and Early Intervention

Primary physicians and medics can have an active role in reducing the risk factors for CSR. Preventive interventions on the group level include counselling commanders on the psychological impact of specific combat conditions and identification and prevention of physiological deficits inflicted to the group. On the individual level those who present the premonitory syndrome should be identified and treated. The treatment follows the above mentioned principles.

1. Start as soon as possible
2. Compensate physiological needs (the "chicken soup" treatment)
3. Allow stress relief by having the subjects rest for a limited time
4. Provide personal support by allowing the expression of fears and anxieties and showing their common nature (many soldiers at this point are afraid of "losing face", feel ashamed and tend to conceal such emotions)
5. Encourage the subject, his peers and his commanders to consider the temporary failure as acceptable and plan full return to duty. Direct communication with commanders and fellow soldiers is an extremely important aspect of the treatment program.

The specific goal of the treatment is to prevent the subject from isolating himself from others and from experiencing loneliness and detachment. Tranquilizers should be avoided. Subjects respond to suggestion and can easily be handled by the competence and authority of a medical officer. Some authors report, however, that the administration of mild doses of minor tranquilizers (e.g., diazepam 5–10 mg) has proven successful in promoting sleep in some soldiers with prolonged sleep deprivation.

First Aid in the Field

When CSR occurs during active combat, particular attention should be paid to potentially dangerous behavior due to dissociative states, disorientation, or agitation. The soldier should be contained and protected from exposing himself to firing or from losing contact with his unit. This is the principal role of the medic, during the battle, in the field. It should be emphasized here again that not every emotional reaction is CSR — most of them are normal reactions that are well contained by the subject's friends.

The following triad can help to distinguish CSR from normal emotional reaction:

1. Task accomplishment is disturbed
2. Social contacts are impaired
3. Gross emotions or modifications of behavior are present and do not improve with rest

Furthermore, it should not be forgotten that anxiety can result from bleeding and that stuporous states are easily confounded with brain commotion. The patient should have a thorough physical examination.

The Battalion Medical Aid Station

The battalion medical aid station is the last station in which the subject is still within his unit and can enjoy support from peers and rapidly integrate his functions. Whenever the battle conditions allow it, CSR patients should remain in the battalion medical aid station for 24–48 h. The treatment follows the following schedule:

1. Assess for medical and surgical trauma
2. Record accurately elements of the trauma. The information obtained at that phase is invaluable: memories of the battle are often repressed or distorted by the subject and cannot be obtained in the following stations.
3. Orient the subject in time, space, events, and treatment plan
4. Compensate physiological needs
5. Reassure. Use empathy to promote self-value and dignity
6. Have the soldier rest for as much as needed (6–24 h)
7. Reactivate, starting by tasks related to the activity of the medical aid station.
8. Return to duties, having arranged for proper acceptance by peers and commanders

The Field Hospital

The principles discussed above should be observed in the field hospital with the following differences. The field hospital is the first station in which the soldier is disconnected from his group. The psychological impact of this separation is significant: not only does he lose contact with comrades and commanders, but he is often deprived of his belongings, his personal weapon is taken away, and he finds himself coping within circumstances to which he was never trained and handled by professionals who have yet to gain his confidence. At the same time he gains an official status of a "patient" — and not less important, of a mental patient. All these changes add to his preexisting difficulties.

On the other hand, the field hospital is the first place in which professional help can be provided, and, where necessary, time and sheltered space are available. The psychiatric unit in the field hospital must accomplish a double task: military atmosphere and discipline should be maintained in order to convey a message of potential return to duties, while rest and individual relaxation should be encouraged in order to promote psychological reintegration. The professional team should be specifically trained for this type of intervention. Soldiers should remain dressed in their uniforms and maintain military discipline. They should be prevented from regressing to the sick role implied by lying on stretchers or beds. Evacuation to the rear should be avoided, and the general expectation will be to return the soldiers to their units.

The time allowed in a field hospital will vary from 48 h to 7 days according to the conditions of combat and the availability of staff. Professional treatment, planned for as long as a week, will include personal and group therapy and progressive but active confrontation with objects of avoidance (e.g., weapons, tanks, etc.). It is unwise to attempt, at this stage, an explorative therapy of the trauma or of any of the subject's basic conflicts. The overall goal of treatment is still the reinforcement of previous defenses and coping mechanisms with respect to the subject as he was before. No changes in personality or in habits should be hoped for at this time.

All CSR patients in the field hospital are expected to return to duty, and such an expectation has to be explicitly shared with the soldiers. In fact, 70%–93% can be returned to their units from here (as opposed to 30% rate of full return to duty from the base hospital).

Differential diagnosis is with psychosis and malingering. The rare cases of psychosis occur most often in patients with a previous history of mental illness. Delusions, hallucinations, and impaired reality testing and judgment distinguish psychosis from CSR. Psychotic patients should be evacuated immediately and, if needed, treated with major tranquilizers (chlorpromazine 50–300 mg a day in progressive doses).

Malingerers are rare during active warfare. Malingering and the purposeful simulation of symptoms are generally seen after the cessation of active warfare and among noncombatant soldiers. They can be recognized by the absence of typical signs of anxiety and arousal, of insomnia, of hypersensitivity to stimuli, or of specific avoidances and by their hostile attitude towards group activities.

Medical officers who are not specialized in the treatment of CSR can provide adequate help for 48 h by compensating physiological needs and allowing withdrawal from threat. They will succeed in a large proportion of CSR patients. This should be preferred over immediate evacuation.

The decision to reintegrate a soldier into his unit is made on the basis of renewed ability to function and a decrease in anxiety attested by the ability to relax, to concentrate, and to be emotionally "tuned" to the group.

The Base Hospital

The base hospital receives CSR patients from three different sources: soldiers who failed to improve in previous stations, soldiers who have been evacuated without previous intervention (mostly by air), and soldiers whose CSR manifested itself for the first time or was aggravated during periods spent in the rear. The base hospital should not function as a primary care facility. Its role in the last two groups is to identify cases of CSR and arrange for their integration in to a more advanced treatment facility.

As to the treatment of soldiers who failed to respond to previous treatments, it should be assumed that such a failure often resulted from the absence of adequate conditions in the field (e.g., absence of professional help in a particular field hospital, battle conditions that imposed rapid evacuation, hasty decisions to evacuate, etc.). The resulting therapeutic attitude is a renewed effort to reverse the process of traumatization. Military atmosphere and discipline should, therefore, be maintained, and the expectation for return to duties explicitly conveyed.

The advantage of the base hospital is in the possibility of providing skilled professional help and attempting an exploration of the traumatic experience and an elaboration of its significance for the patient. Individual and group psychotherapy are the main tools for achieving these goals.

Attempts at recalling events and expressing emotions related to them (abreaction) can be done if highly skilled professionals are present. Suggestion or hypnotic induction are the basic techniques. Barbiturates and sedatives were never shown to function better than suggestion. Repressed memories are not the unique cause of CSR and PTSD. This old assumption has been challenged, and abreaction is not considered as mandatory. However, in those of the soldiers who do abreact at recalling repressed memories, this is often a turning point in their treatment.

Behavioral psychotherapy has an important role in attempting to end specific avoidance.

Pharmacotherapy, including essentially antidepressants (imipramine: 150 mg a day in a single daily dose at bedtime) should be attempted in seriously depressed soldiers and in those manifesting panic attacks.

As a whole the base hospital allows a more individualized approach to CSR patients including controlled repression, exploration of specific areas of conflicts, and specific configuration of the trauma. The therapy is, nevertheless, time limited, focused, and basically supportive.

Conclusions

CSR is an universal phenomenon which constantly accompanies warfare. It depends largely on variables of the combat and the way units are trained and prepared for it. The number of definite casualties from CSR and their

severity depend on the availability of adequate help in time and on the firm and supportive attitude of treating personnel and commanding staff. The policy of prompt treatment reduces definite casualties in a proportion of 1 to 5. The reason for such a difference resides in the fact that the trauma related to CSR is a relatively long process which can be reversed by proper intervention in time.

CSR can be recognized in early stages by the presence of irreducible anxiety, impaired social function and failing stimulus regulation. In the acute phase, CSR comprises severe cognitive and emotional dysfunction with inability to follow task-oriented activity. Acute psychiatric symptoms including conversions, stupor, dissociative states, and acute anxiety states are present. This clinical picture can revert into PTSD when intrusive memories and systematic avoidance of stimuli progressively replace the signs of the acute phase. This "final common pathway" makes the individual the victim of an endless repetition of his traumatic experience of the war.

The treatment of CSR is basically the same in all the stations and includes protection from external sources of stress, compensation of physiological losses, firm supportive and humanizing intervention, reactivation, and reintegration. Rejecting and segregative attitudes are frequently encountered and should be avoided. One can expect up to 70%–90% recovery rate from early intervention conducted within the military corps of the soldier, and a 30%–50% rate of return to duty from treatment carried out later.

Acknowledgement. The authors wish to express their gratitude to Dr. Reuven Bar-On from the IDF Dept. of mental health for his valuable and skillful help in editing this chapter.

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Part 3

Treatment of Wounds: General

3.1 Trauma Scoring

M. Michaelson

The variety and complexity of injuries in disasters or modern battlefields create the need for an objective system to categorize their severity.
The trauma score should be used as a helpful tool in establishing priorities of evacuation to hospital. It is an objective tool for passing on information on the severity of the condition of the casualties and allows an objective assessment of the quality of treatment at various echelons.
Any system of trauma scoring in the field should be based on easily available data, be simple, accurate and reproducible by trained personnel and correlate with the probability of survival.
The Champion trauma score is such a system. This score consists of the Glasgow coma scale plus four other physiological variables: respiratory rate, respiratory effort, systolic blood pressure and capillary refill; each variable receives an individual score. These are totaled together, resulting in a number from 1 to 16: the lower the score, the greater the severity of injury.
The trauma score (see p. 186) should be assessed at every echelon on the way to the hospital and recorded on the chart.

Further Reading

Champion HR, Sacco WJ, Carnazzo AJ, et al. (1981) Trauma score. Crit Care Med 9:672–676

Date: _____ Name: _____

Hour: _____ No: _____

Signed: _____

Trauma Score

	Value	Points	Score
A. Respiratory rate/min	10–24	4	
	25–35	3	
	>35	2	
	<10	1	
	0	0	A [ ]
B. Respiratory effort	normal	1	
	rapid, shallow, or retractive	0	B [ ]
C. Systolic blood pressure	>90	4	
	70–90	3	
	50–69	2	
	<50	1	
	no carotid pulse 0	0	C [ ]
D. Capillary refill			
refill in 2 s	normal	2	
delayed >2 s	delayed	1	
no capillary refill	none	0	D [ ]

E. Glasgow coma score

(I) Eye opening			
spontaneous	4		
to voice	3		
to pain	2		
none	1		
(II) Verbal response		Total points GCS	
oriented	5		
confused	4	14–15 = 5	
inappropriate words	3	11–13 = 4	
incomprehensible words	2	8–10 = 3	E [ ]
none	1	5– 7 = 2	
		3– 4 = 1	
(III) Motor response			
obeys commands	6		
purposeful movements	5 (pain)		
flexion (pain)	4		
withdraw (pain)	3		
extension (pain)	2		
none	1		

(I)+(II)+(III) total GCS points [ ]

Trauma score total points A+B+C+D+E [ ]

3.2 Mass Casualties and Triage

N. D. Reis

"Triage" means "sorting out of casualties". Sorting is needed whenever more casualties present themselves than can be dealt with definitively and simultaneously by the medical staff at hand. The term "mass casualties" is used variously. Ten serious casualties caused by a disaster or in battle with one medical officer on the scene is a mass casualty situation for that physician. Thermonuclear war or a major earthquake with the resultant flooding of medical facilities with vast number of casualties is another kind of mass casualty situation. In each case, the available medical personnel must triage the injured in order to attempt to do the maximum good to the greatest number.
The aim of sorting is to decide on:

1. What to do?
2. For whom?
3. When?

Triage is an essential repetitive duty at all echelons. It is the first duty on site, at the medical aid station, again in the receiving area of the field hospital, and, once again, in this age of helicopter evacuation direct from the scene to hospital, in the emergency room of the base hospital.

Primary Triage On Site and at the Medical Aid Station

Four distinct groups of casualties are sorted out (Fig. 1):

1. Trivial injuries: these are immediately discharged or returned to their unit.
2. Minor injuries which do not require evacuation or hospital treatment. After treatment at the medical aid station these casualties are discharged or returned to their unit.
3. Serious casualties in need of urgent treatment in the field and evacuation to a field or base hospital.
4. Dead or dying from fatal injuries.

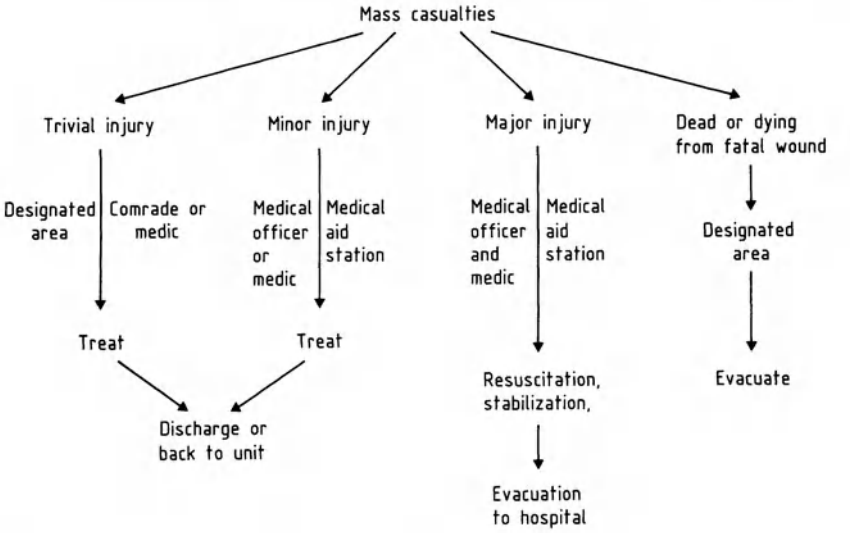

Fig. 1. Mass casualty triage on site by medical officer

Having completed his triage, the medical officer must concentrate his efforts on *group 3*. In this group he must apply the principles, methods, and priorities described in the chapter on the primary treatment of wounds. Following resuscitation, the stabilization of the vital functions, and all the other steps in primary wound care, priorities for evacuation are determined.

Triage in the Field or Base Hospital

When rapid evacuation of the wounded is available, large numbers of casualties will arrive in the hospital without prior triage. The initial sorting described for the medical aid post echelon is then indicated at the hospital echelon.

Sorting before first wound surgery should be done by the most experienced surgeon available. He must decide for each case whether:

1. Immediate surgery is imperative (e.g., in cases with uncontrolled internal or external hemorrhage, or asphyxia requiring tracheostomy).
2. Delay for further vital function stabilization and/or diagnostic investigation is indicated.
3. Surgery can safely be postponed.

3.3 Multiple Injuries

N. D. Reis

Multiple injuries may occur in one individual or present as mass casualties, i.e., a large number of individuals who have suffered one or more injuries. In the latter case triage of the wounded precedes the actual treatment of each individual (see chapter on mass casualties and triage).
Multiple injuries in a single casualty are characterized by:

- High mortality and morbidity
- Tendency to severe shock
- Difficulty in determining the priorities of treatment
- Difficulty in optimizing the treatment to suit the pathophysiology of more than one organ system

Etiology. Alone or in any combination, the causative agents are: (a) missiles penetrating at multiple anatomical sites, (b) burns, (c) blast, (d) crushing, (e) deceleration (vehicular crashes), (f) chemical trauma, and (g) ionizing radiation.
Environmental factors such as cold, heat, exposure, starvation, and infection complicate an already complex problem.

Treatment

On Site and Medical Aid Station

For explanation of resuscitation, see chapter on primary care in the field.
Clinical examination begins with history taking. Try to obtain information as to how the injury occurred, e.g., blast, etc. However, the patient is often exhausted, dirty, fully clothed, and cluttered with equipment. He may be confused by the circumstances of his injury or by having received on-site sedation. Thus, strip off all clothes and examine the entire body (including the back!) systematically:

- Assess airway and respiration
- Observe for open wounds made by missiles
- Palpate chest wall: feel for emphysema, crepitus and observe chest wall movement; auscultate for air entry and heart sounds

- Observe abdomen for bruising, abrasion and movement; palpate for tenderness and rigidity
- Look at urethra (milk it in the male) to exclude blood
- Palpate body contours of head, neck, spine, pelvis, and limbs; move major joints passively; do not move neck
- Assess state of consciousness: order active movement of all four limbs; demand answer to simple questions of identification; examine pupillary reaction to light

When resuscitation has been initiated and the almost contemporaneous rapid clinical examination has been completed, treatment should continue, based on the clinical findings as described in the appropriate chapters. Brief records must be kept of the injuries, the treatment, and the response.

The multiply injured patient is frequently a high priority for evacuation. Skilled and close attention to his airway, respiration, control of bleeding, and IV fluid administration is required whilst waiting for and during evacuation. The attending physician makes a continuous reassessment of the condition, until the casualty is delivered to a hospital.

A painstaking attempt to stabilize the patient's condition on site or at the medical aid station is always superior to "scoop and run" evacuation. This should be done even when at some risk from a hostile environment.

Field and Base Hospital

The general condition of the casualty is reassessed (taking into account his on-site and evacuation medical record) under the following headings which are in order of priority and are managed as described in the chapter on resuscitation.

1. Airway control
2. Respiration-ventilation: in case of ineffective ventilation, think of: progressive pneumo- or hemothorax, aspiration, cardiac tamponade, rupture of a major blood vessel, rupture of diaphragm
3. Arrest of bleeding
4. Volume replacement: replace blood loss as soon as possible.
   A rough estimation of blood loss for various common injuries is as follows: pelvic injury (2–20 liters), chest and abdominal injury (2 liters), Fracture of femur (2 liters), fracture of arm or leg (1 liter), severe head, scalp, face lacerations (½–1 liter), fracture of forearm bone, one rib, foot (150 ml). A healthy person must lose more than 25% of his blood volume for shock to set in; age and environmental factors (dehydration, starvation, infection) greatly reduce this figure. If shock does not respond to adequate ventilation and volume replacement, think of occult bleeding in the chest, abdomen, or pelvis.

The patient is now systematically examined to diagnose the various local injuries:

- Follow once again (but taking more time and in greater detail) the schedule of examination detailed in the previous section.
- Establish priorities in treatment: first, pay attention to the *chest* and rule out or treat pneumothorax, hemothorax, cardiac tamponade. Next, carefully reassess the *abdomen* (in particular review the abdomen once again if the chest condition has been treated and the casualty is not improving). Peritoneal lavage (diagnostic) is a useful routine when in doubt. Also take a plain X-ray for free gas. Feel for tenderness and guarding (in the conscious) and absence of bowel sounds and distension (serial measurement of abdominal girth).
- Catheterization is routinely performed to monitor urine output. In obvious urethral injury (bleeding from urethra) transurethral catheterization is contraindicated. Drainage by suprapubic puncture is then employed, once the bladder is distended, or by an operative method.
- Insert nasogastric suction to empty the stomach and prevent aspiration.

Surgical Treatment

The priorities of operative treatment of the multiply-injured patient are difficult to determine. Follow these principles of urgent multitrauma surgery:

- Operate urgently only to save life and organ function and to excise wounds
- Avoid all unnecessary procedures during primary surgery
- Transfer from field hospital and base hospital to specialized units for neurosurgery, maxillofacial and eye surgery, plastic and hand surgery, etc., whenever possible, once the life-threatening conditions have been dealt with

Immediately life-threatening treatable injuries must be seen to first:

- Chest drainage
- Urgent thoracotomy for uncontrolled intrathoracic bleeding (ruptured aorta, lacerated bronchus, or obstinate cardiac tamponade)
- Exploration of the neck for life-threatening hemorrhage
- Urgent laparotomy for uncontrolled intra-abdominal bleeding
- Laparotomy for abdominal organ ruptures or lacerations

Of lesser urgency and priority are:

- Neck wounds without severe hemorrhage
- Intracranial wounds
- Face wounds

- Hand wounds
- Eye wounds
- Ear wounds
- Fractures of the axial and limb skeleton

(Frequently surgery is performed simultaneously, e.g., craniotomy and laparotomy, excision of limb wounds and thoracotomy, etc.) The definitive surgical treatment of many types of wounds is better delayed, providing that the wounds are excised (see chapters on the different kinds of wounds) in order to avoid wound infection. Optimally, excision of soft tissue wounds should be within 8 h of injury and penetrating wounds into body cavities or open fractures, within 6 h.

Postoperative Care

- Multitrauma patients carry a very high risk of mortality and morbidity.
- Many require postoperative respiratory support by mechanical respirators in an intensive care unit.
- Multiple blast injuries, patients after cardiorespiratory resuscitation, thoracoabdominal injuries, severe head injuries, all of which may be associated with multiple fractures, are the highest-risk groups.
- There is an ever constant danger of severe sepsis.

3.4 The Physiological Metabolic Response of the Body to Injury

S. Bursztein and N. P. d'Attellis

The pathophysiology of mechanical injury, burns, and sepsis includes marked alterations in hormonal secretions, in metabolism, and in response to nutrients.
Two phases are defined in the response to injury: the ebb or shock phase, followed by the flow phase. The flow phase has been further divided into a catabolic and an anabolic phase. Two types of flow phases are distinguished, the flow phase proper in which oxidation is adequate, leading to recovery, and a necrobiosis phase leading to death. The duration of these phases depends on both the type and severity of the insult.

Ebb Phase

The ebb phase may last a few hours after uncomplicated surgery, but 2–3 days after severe injury, burns, or sepsis. The ebb or shock phase is characterized by hemorrhage or loss of intravascular fluids to interstitial fluid and transcellular spaces. Blood pressure and blood flow fall; there is greatly increased sympathetic activity, although energy expenditure falls. There is marked initial hyperglycemia, deriving mainly from glycogen stores. Subsequent development of hypoglycemia with exhaustion of glycogen gives a poor prognosis. Reduced energy expenditure is accompanied by lowered body temperature and seems to be related to changes in thermoregulation in the hypothalamus.

Flow Phase

The flow phase may last from a few days to several weeks or longer with severe burns or sepsis. This is the hypermetabolic (or increased energy expenditure) and the hypercatabolic (or increased protein breakdown and protein loss) stress state which is generally associated with trauma.
Energy expenditure that is increased by about 10%–20% after elective surgery, increases by 10%–40% with injury, 30%–50% with sepsis, and by up to 100% with severe burns. Starvation may reduce energy expenditure by up to

40%. Since injured or septic patients are often starved to some extent, their change in energy expenditure will be the algebraic sum of the two effects. Indeed, energy expenditure of patients under intensive care can range from about 40% below to 60% above normal values.
The hypermetabolism of injured and septic patients is associated with increased body temperature. This increase in temperature in the flow phase, like the decrease in the ebb phase, is related to thermoregulation in the hypothalamus, which results in a change in the thermoneutral temperature range, i.e., the ambient temperature at which the nude subject feels most comfortable and expends the minimum energy at rest. In the flow phase the thermoneutral zone is increased several degrees above normal values of 27°–29°C.
In severe injury, sepsis, and burns there will be major alterations in substrate metabolism.

Protein Metabolism

The injured, septic, or burned patient is hypercatabolic as well as hypermetabolic. As in fasting normal subjects, the bulk of N loss comes from skeletal muscle. The extent of the N loss is dependent on the severity of the stress. Since N losses are markedly affected by diet, quantitative comparison to normals can be made only at constant diet intake. Rates of N loss in various pathological states during 5% dextrose administration range from 2 g/day (or 12.5 g protein) in normal fasted subjects to up to 27 g/day (or 168 G protein) in severe by burned patients, with in between about 15 g/day (or 94 g protein) for major surgery and sepsis and 17 g/day (or 106 g protein) for multiple injury.
For the abovementioned reasons, injured and septic patients have markedly increased requirements for protein compared with normal subjects. If not previously malnourished, they respond to increased N intake similarly to normal subjects, in that at zero energy balance they reach a state where additional N intake causes no further increase in N balance. With normal subjects this occurs at zero N balance; with stressed patients it occurs at different levels of negative N balance. When N intake is zero in this kind of patients, they are in very negative balance; when N intake reaches 200 mg/day, N balance is less negative, but increases of N intake above this amount have no further effect on N balance. We can recommend for such patients a daily N intake of about 200 mg/day, which is about three times the minimum requirement for normal subjects. If multiply injured patients are kept on 5% dextrose they may in 10 days lose more than 1.5 kg of protein, representing more than 20% of the body cell mass. By adequate nutrional management the protein loss can easily be reduced over the same period of time to less than 200 g, which will not cause serious malnutrition.

However, patients with sepsis and multiple organ failure are usually resistant to all nutrients and they will remain in severe negative N balance.

Carbohydrate Metabolismn

A characteristic feature of injury, sepsis, and burns is hyperglycemia. Blood glucose levels may range from just above normal after elective surgery to as high as 800 mg% in severe cases. The hyperosmolarity of blood with these very high glucose concentrations can have severe clinical consequences. These high blood glucose concentrations have been termed the "diabetes of injury." However, unlike diabetes mellitus, they are associated with increased rather than decreased concentrations of insulin. Long and colleagues demonstrated with tracer techniques that hyperglycemia was associated with increased production of glucose and that gluconeogenesis in septic patients was much higher than in normal subjects. This abnormally high rate of glucose production is associated with a decreased rate of oxidation of glucose. At each level of intake, glucose oxidation is lower in the stressed patients than in normal subjects, while fat and protein oxidation is higher.

Glucose turnover increased even more than glucose oxidation in trauma and sepsis, due to an increase in glycolysis (or anaerobic glucose metabolism) which is a requirement of the wound or septic region. In sepsis there is at the site of infection an increase in white blood cells, which use glucose for glycolysis rather than oxidation. In injury or burn the healing tissue also uses glucose for glycolysis rather than oxidation. Wound requirements have been measured (by Wilmore et al.) in burn patients in whom one leg was either severely burned (50%) or lightly burned (10%). Blood flow and O_2 consumption were measured for the whole body and for a single leg. Glucose consumption and lactate production were also measured in the leg. There were no significant differences in whole body percent burn, blood flow, or O_2 consumption between the patients with the large or the small leg burns; leg blood flow, glucose consumption, and lactate production were much higher with the large than the small leg burn, but there was little difference in O_2 consumption. Almost all the glucose consumed was recovered as lactate. Thus the major difference between the legs with large and small burns was due to a difference in the amount of glucose used for glycolysis, not for oxidation. This difference, of 0.30 mg glucose/min per 100 ml leg, may be attributed to the burn, amounting to 40% of the leg area (50% minus 10%). Extrapolated to the whole body, the amount of glucose glycolized by the burn wound in a patient with a 40% burn is about 200–300 g per day. The energy derived from glycolysis of glucose is about one-twentieth that obtained from oxidation or 0.2 kcal/g. Thus, although the glucose requirements of the wound are very large, the energy requirements of the

wound itself are low, about 60 kcal day, much less than the increase in energy expenditure in the body generally due to the presence of the wound, which is closer to 500–1000 kcal day.
At any given level of carbohydrate intake, glycogen storage is increased and glycogenolysis is decreased in stressed patients compared to normal subjects.

Fat Metabolism

Fat oxidation tends to be higher in hypermetabolic patients than in normal subjects or than in malnourished patients. Lipolysis of triglycerides found in adipose tissue increases to a much greater extent than fat oxidation. This increase in fat mobilization is accompanied by marked increases in plasma concentrations of glycerol but not of fatty acids. Thus there is a dissociation of the normal relations between fatty acid turnover and concentration. These increases in fat mobilization and glycerol concentration and maintenance of normal concentrations of fatty acids occur despite the increase in insulin concentrations associated with trauma.
Although fat metabolism and oxidation are increased with stress, ketogenesis is decreased compared to normal fasted subjects. The metabolic rate will vary with the severity of the stress, being much greater after a 75% third-degree burn than after an abdominal operation. A major difference from normal patients is a three-fold increase in glucose production, mainly used for glycolysis in the wound. However, glycogenolysis is suppressed and all the glucose comes from glycogenolysis. Since ketogenesis is partly inhibited by high glucose and insulin concentrations, all the brain requirements are met by glucose, and in addition, under these conditions, other tissues oxidize some glucose. This increased glucose oxidation is derived almost entirely from dregradation of muscle protein.

Hormonal Changes in Trauma and Sepsis

The metabolic pattern in the stressed patient does not undergo the same adaptation with time as is seen in the normal fasting patient. This is because the normal regulatory mechanisms, which are very sensitive to insulin concentration and glucose intake, are overwhelmed by increases in counterregulatory hormones.
There are many hormone changes which accompany injury, burns, or sepsis. The best understood and most important changes are the increases in epinephrine and norepinephrine, cortisol, and glucagon. Increases in norepinephrine mainly reflect increases in sympathetic activity. They occur im-

mediately after the insult, are very high in the ebb phase, and continue at high but declining rates during the catabolic part of the flow phase. Increases in cortisol occur more gradually but are evident throughout the flow phase. Infusion of cortisol, glucagon, and epinephrine into normal subjects at rates which maintain blood concentrations in the range seen in stressed patients causes metabolic changes which mimic those seen in injury and sepsis. These include hypermetabolism, hypercatabolism, hyperglycemia, and hyperinsulinemia, together with increased glucose and insulin resistance. The hyperglycemia results from both increased production and decreased clearance of glucose.

There are two main pathways by which the wound or septic insult causes this change in the neuroendocrine milieu. One of these involves stimulations of a variety of receptors which send impulses via the afferent nervous system to be coordinated in the hypothalamus. Nerve endings, including pain receptors at the site of injury, are stimulated. Epidural analgesia prevents transmission of the stimuli and blocks the development of the stress response immediately after injury. Baroreceptors respond to the low volume and flow in the ebb phase and chemoreceptors respond to variations in O_2, CO_2, pH, and K concentrations. Fear, anxiety, temperature, and other physiologic and environmental factors also have input into the metabolic response to injury. These various signals are coordinated or integrated in the hypothalamus and appropriate efferent signals are transmitted through the sympathetic system and adrenal medulla, which respond rapidly, and the pituitary gland, which responds more slowly.

The other pathway mediating the effects of injury or sepsis on metabolism involves secretion of a number of polypeptide hormones or groups of hormones by white blood cells at the site of injury or infection. Some of these polypeptides have been isolated and characterized and there may be others. Endogenous pyrogen, leukocyte endogenous mediator, and lymphocyte activating factor constitute either one or a family of polypeptides of 13000–16000 daltons secreted by a wide variety of phagocytic cell types under appropriate stimulation.

One of these is the same as interleukin-1, a peptide of 16000 daltons. Once released, this peptide or peptides act directly on the hypothalamus, augmenting and supplementing the effects of afferent nervous stimuli. In addition, they influence the activity of many tissues, including lymphocytes, granulocytes, macrophages, bone marrow, the reticuloendothelial system, liver, muscle, and endocrine organs. Among their actions are stimulation of fever, granulopoiesis, synthesis of acute phase proteins, hyperinsulinemia, and uptake of zinc and iron by the liver. Interleukin-1 acts directly on muscle to increase proteolysis and net breakdown of protein, providing an increased supply of amino acids for use by the liver and hematopoietic tissues. This action appears to be mediated by prostaglandin E_2. A second peptide of about 4000 daltons has been isolated from plasma of septic patients. This also directly stimulates proteolysis in muscle and may be a de-

gradation product of interleukin-1. A third peptide, cachectin or tumor necrosis factor, is released by stimulated macrophages. It has many pathophysiologic effects which overlap those of interleukin-1; it also stimulates release of interleukin-1, and appears to be the major factor mediating the lethal effects of endotoxin.
Although afferent neurogenic pathways and these various peptides can account for the ability of injury and sepsis to effect neuroendocrine and metabolic changes, the detailed mechanisms of their actions, the relative importance of each, and whether other factors play a role remain to be determined.

Mechanism of Response to Injury

These metabolic changes, which are shared by humans with many other species, seem to be adaptive, at least in the short term of days to weeks. The injured organism has increased nutrient requirements in order to cope with wound healing and infection, but, particularly in the wild state, must obtain these nutrients from its own body stores. Some authors have suggested that the increased muscle protein breakdown is needed as an energy source per se. However, even in most severe stress, endogenous protein accounts for only 25% of total energy requirements. Furthermore, mobilization of fat, which is the major endogenous source of fuel, is greatly increased with stress. It seems likely that increased muscle proteolysis is needed to supply specific nutrients, glucose and amino acids, which cannot be derived from fat.
An increased supply of amino acids is required by the liver and hematopoietic tissues for synthesis of acute phase proteins and for increased production of white blood cells. These are needed to control infection, to clear up necrotic tissues, and to take part in wound healing. In addition, in the absence of carbohydrate, amino acids are the main source of synthesis of the glucose needed for the brain and other glucose-requiring tissues and, particularly, for the wound itself.
Perhaps the most significant metabolic change due to injury is the occurrence of hyperglycemia in the absence of carbohydrate intake. In fasting normal subjects, blood glucose concentrations are decreased and extensive ketogenesis greatly reduces the amount of glucose required by the brain. Excretion of N will drop below 5 g/day (80 mg/kg/day) in prolonged fasting.
As discussed above, the wound may require 200 g or more of glucose per day which is needed for glycolysis rather than oxidation. It thus seems very likely that hyperglycemia is an effective adaptive response to meet the glu-

cose requirements of the wound or septic site. If this is true, then in treating injured or septic critically ill patients, efforts should not be made to lower glucose concentrations down to normal values.

While the requirements of the wound appear to be responsible for the hyperglycemia, they are not directly responsible for increased glucose consumption, since the wound converts glucose to lactate and pyruvate which are used by the liver to resynthesize glucose. Normally, hyperglycemia, particularly when accompanied by hyperinsulinemia, would suppress fat mobilization and oxidation. In the fasting, hyperglycemic, hyperinsulinemic, stressed patient, fat mobilization is above normal, and 75%–90% of energy is supplied by oxidation of fat. Thus tissues such as skeletal muscle, heart, and liver utilize fatty acids preferentially, despite high glucose concentrations. The high concentrations of glucose and insulin also decrease ketogenesis as compared to normal fasting. With less ketones available, the brain requires much more glucose in the fasted stressed patients than in fasted controls. It is this high net requirement of the brain for glucose which is oxidized, much more than the wound requirement for glucose for glycolysis, which determines the amount of protein that must be broken down for gluconeogenic purposes in the stressed patient.

Thus the need of the wound for hyperglycemia is seen as the main etiological cause of the neuroendocrine changes in injury and sepsis. This disrupts the usual relations by which glucose intake or production and insulin concentration regulate fuel utilization. Brain requirement for glucose remains high and some glucose is oxidized by other tissues. This amount of oxidized glucose must be synthesized from muscle protein, producing a hypercatabolic state.

The increased substrate cycles discussed above, lipolysis and triglyceride synthesis, and glycolysis and gluconeogenesis, which are energy producing processes and an obligatory part to the changed neuroendocrine and metabolic conditions, cause increases in energy expenditure. In addition, increase in protein turnover also increases energy expenditure. These changes can account for much if not all of the increase in energy expenditure in injury and sepsis. We may speculate that the rise in core temperature and the thermoneutral zone, which accompany the increase in energy expenditure, provide, like hyperglycemia, a more favorable environment for tissue regeneration and host defense.

Effects of Nutrition in Injury and Sepsis

The changed neuroendocrine milieu in stressed patients changes the response to nutrients. Since fuel utilization patterns are now largely deter-

mined by the increased sympathetic activity and counterregulatory hormones, the ability of exogenous nutrients, particularly glucose, to change these patterns is attenuated.

In septic or injured patients receiving 3 liters 5% dextrose, gluconeogenesis is twice as high as in fasting normal subjects, and is not competely suppressed until at least 600 g glucose is given per day. Injury or sepsis also attenuates the effect of administered glucose to decrease fat oxidation and increase glucose oxidation. Even with glucose intakes in excess of energy expenditure, the injured and septic patients derive one-third of their energy from endogenous fat.

These effects of injury and sepsis, attenuating the effects of glucose intake on gluconeogenesis and on fat and glucose oxidation, occur despite the fact that blood glucose and insulin concentrations are consistently higher in response to glucose intake than in normal subjects. A consequence of decreased glucose oxidation and increased fat oxidation in stressed patients, at any given level of glucose intake, is that glycogen storage is much greater than normal and can reach levels as high as 2000 g.

The effect of protein intake to improve N balance is also attenuated in injured, septic, or burned patients. Normal subjects attain zero N balance at zero energy balance with an intake of 80 mg N/kg/day, whereas severely stressed patients are in negative N balance of 3–4 g/day with N intakes of 200–300 mg/kg/day and energy intake in excess of expenditure.

Many investigations have still to be performed to improve our knowledge concerning the disorders induced by severe injury. Only recently, many pathophysiologic aspects of response to injury previously considered as harmful, appeared in fact to be adaptive mechanisms of the body to overcome the damage caused by the wound and the stress. This is probably true not only for increased gluconeogenesis and high levels of blood sugar, but for many other alterations that we sometimes try to correct without any evidence that we are not disturbing the normal and favorable response to injury.

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3.5 Posttraumatic Shock

U. Taitelman

Introduction

Death from post-traumatic shock occurs mainly in three periods: immediately (within minutes) after the trauma, within 2 h after the trauma, or days after the trauma. Analysis of the results of the treatment of cases of post-traumatic shock from the Korean and Vietnam wars, from other military conflicts, and from trauma centers in the United States of America shows that the best results are obtained if the patient receives first aid within minutes after the trauma and is then transferred to a hospital at which definitive care can be administered.

If first aid and transfer are completed in less than 1 h, many of the deaths occurring 2 h after the trauma can be prevented. However, in war or disaster, immediate transfer to a trauma center from the scene of the incident is often not feasible. Transfer is then from the site of the incident to a first-aid station after variable periods of time. Hence the management of post-traumatic shock is described in three situations: on site, at the first-aid station, and in the hospital.

Early post-traumatic death is caused by the isolated or combined deterioration of the functions of three vital systems: the circulatory, respiratory, and central nervous systems. Unless proven otherwise, the cause of post-traumatic circulatory failure is hypovolemic shock due to hemorrhage.

Pathophysiology

The natural history of untreated or partially treated hemorrhagic shock in experimental animals has three phases: hypotension, pending shock, irreversible shock.

Phase 1. During the first phase (hypotension), the compensatory mechanism includes central and peripheral sympathetic stimulation and catecholamine discharge as well as activation of the renin-angiotensin system. The results of these reflexes are tachycardia and peripheral vasoconstriction.

Vasoconstriction in the skin, muscles, kidneys, and splanchnic organs is maximal, and blood flow is diverted from these organs to perfuse the heart and brain. During this phase extracellular fluid shifts to the intravascular space. *This phase is fully reversible by intravenous blood or fluid infusion.* The duration of this reversible phase depends on the amount and rate of blood loss and the age of the animal: about 2 h after bleeding loss of 30%–40% of the estimated blood volume in young animals, and only 15 min after rapidly losing 75% of estimated blood volume. In young animals adequate volume replacement without red cells results in survival, even when the hematocrit drops to 11%.

Phase 2. The second phase — pending shock — *is not reversible by simple reinfusion of the shed blood or a corresponding amount of fluid, but is reversible by continuous large amounts of fluid infusion.* The duration of this phase depends on the severity of tissue and cell damage caused by the low perfusion state, and the functional reserve of the heart and lungs.
The following phenomena explain the harmful effects of the ischemic peripheral tissues on circulatory homeostasis:

1. Anaerobic metabolism produces lactic acid, causing local and systemic metabolic acidosis (during acidemia, catecholamines are ineffective).
2. Release of histamine causes local vasodilatation in the peripheral microcirculation and vasoconstriction in the pulmonary circulation.
3. Cellular swelling is associated with penetration of sodium and water into and leakage of potassium out of the cells. These ionic changes modify the excitability of neurons and muscles (especially the myocardium).
4. Progressive rupture and destruction of cellular membranous structures, i.e., cell, mitochondrial, and lysosomal membranes, result in leakage of intracellular components into the extracellular and intravascular space.
5. Cell death is associated with excess penetration of calcium ions into the cytosol.
6. The leakage of lysosomal enzymes causes local and systemic proteolysis and lipolysis. Myocardial depressant factor is a peptide liberated from the ischemic pancreas and from lysosomes in other tissues. Its main action is decreasing the contractility of the myocardium.
7. Metabolites of arachidonic acid are liberated: Trombexane and prostaglandin $F\alpha_2$ are procoagulant and potent vasoconstrictors, while other prostaglandins are anticoagulant and vasodilators. Their anarchic production contributes to an increased capillary permeability in the systemic microcirculation and to an increased pulmonary vascular resistance and alveolar capillary permeability.
8. The slow blood flow in the microcirculation, the increase in vascular permeability associated with the unbalanced activity of arachidonic acid metabolites, and the activation of complement and kallikrein, promote intravascular coagulation and coagulopathy due to the consumption of coagulation factors and enhancement of fibrinolysis.

In young, healthy animals the breakdown of circulatory homeostasis is attributed to the hypoperfusion of organs other than the heart and brain. As a result of cellular and tissue ischemia, the precapillary arteriole becomes refractory to catecholamine and dilates. *Fluid from the intravascular compartment moves in to the interstitial space and into the intracellular space.* Lymphatic clearance decreases, and an increasing amount of blood is stagnant and pools in the microcirculation, thereby lessening the venous return and decreasing the cardiac output.

The young, healthy animal is able to step up its cardiac output by a multiple of four or more, and to increase the respiratory minute volume in order to maintain the flow of oxygenated blood and eliminate carbon dioxide. But if cardiac or pulmonary disease is present, or cardiac and respiratory reserves are decreased by age, the duration of this phase of pending shock becomes shorter.

During this phase important pathological changes take place in specific organs:

1. Blood flow to the pancreas may be decreased up to 93%. The ischemic pancreas becomes the source of toxic mediators which aggravate cardiopulmonary dysfunction.
2. During severe prolonged hypotension, flow to the small intestine decreases by 70%. The ischemic bowel is the site of severe engorgement and stasis. The permeability of the mucosa and the microcirculation increases, enabling endotoxins and enteric pathogens to penetrate into the portal circulation: ischemic gut is also a major source of lactic acid and lysosomal enzymes.
3. Blood flow to the kidney decreases by 75% during severe hypotension. Hypotension alone may be the cause of acute renal failure. When hypotension is associated with systemic accumulation of myoglobin, bile salts, and bilirubin, the incidence of acute renal failure increases.
4. In the lungs prolonged and severe hypotension causes: increase in pulmonary vascular resistance, increase in lung water content, increase in alveolar capillary permeability, leakage of water and proteins to the interstitial and alveolar space, washout and decreased production of surfactants, decreased lung compliance, decreased functional residual capacity, increased right to left shunt, and increase in work of breathing. All nine factors lead to progressive, hypoxemic respiratory failure.
5. Myocardial oxygen extraction from the blood is maximal under normal resting conditions. The only way to increase the oxygen supply is by increasing myocardial arterial blood flow. Myocardial arterial blood flow is effective only during diastole and depends mainly on the aortic diastolic pressure. During tachycardia and hypotension, the duration of diastole shortens, and myocardial perfusion pressure decreases, while the oxygen demand of the myocardium is increased due to the tachycardia and the catecholamine effect. Ischemic cardiac failure may result

especially when coronary arteriosclerosis and a very low hematocrit coexist.

Phase 3. The third phase is irreversible shock characterized by refractoriness to all kinds of therapy. During this phase the terminal vascular bed behaves as a passive structure with stasis in the pre- and postcapillary vessels and the development of disseminated intravascular coagulation and fibrinolysis.

Treatment

Team Training

The medical teams on duty on site and at the first-aid station must be trained and have experience in the following therapeutic interventions:

1. Cardiopulmonary resuscitation.
2. Cannulation of peripheral and central veins by percutaneous puncture and cut-down techniques. The appropriate peripheral veins are the forearm, dorsal hand, and antecubital fossa veins. They are usually collapsed, and percutaneous cannulation is therefore very difficult in severe shock. The central veins recommended are the femorals, the external and internal jugulars, and as a last choice, the subclavian veins. Many patients will need cannulation of several veins.
3. Endotrachial intubation and cannulation of the trachea via the thyrocricoid membrane.
4. Hemostasis of external bleeding sites by local pressure or tourniquet.
5. Closed chest tube drainage for hemo- or pneumothorax.
6. Closed percutaneous paracentesis of the pericardium.
7. Splints and temporary fixation or traction of fractures, especially those of the cervical spine.

The team should be able to perform all of these therapeutic procedures in less than 15 min.

On Site

Start respiratory and circulatory support immediately. Simultaneously, calmly and quickly examine the patient after removing all clothing.

Diagnosis. The classical signs of shock are: low blood pressure, rapid heart rate, anxiety and mental obtundation, pale cold skin, cold perspiration, and cold extremities, and tachypnea is frequently present. The absence of tachy-

cardia or the presence of bradycardia together with the clinical signs of shock may be caused by: severe hypoxemia (or extreme anemia), increased intracranial pressure, spinal cord injury, hypothermia, life-threatening tension pneumothorax or pericardial tamponade, severe hyperkalemia, expecially if associated with hypocalcemia and hyperphosphatemia which are seen mainly after the release of crushed limbs, severe vagal stimulation as seen in cases of visceral distention and mediastinal displacement, and cardiac arrhythmia, especially after cardiac contusion.
Atropine and/or isoprenaline are administered and the cause promptly treated in order to achieve a pulse rate above 50/min.

Management

1. *Fluid administration* is the therapeutic mainstay of post-traumatic shock. The management of post-traumatic shock is based on the principle that *too much fluid infusion is better than not enough*. Thereby, more patients reach hospital alive without irreversible shock or acute renal failure, but the incidence of acute respiratory failure is increased (ARDS, adult respiratory distress syndrome). The volume of fluid administration depends on the amount and rate of bleeding. Both crystalloids and colloids can be used for fluid resuscitation. On site, 1 liter of colloids followed by or together with 3 liters of crystalloids are given as a starting load. The rate of fluid administration is determined by the patient's hemodynamic response: Fluid administration is inadequate if the systolic blood pressure is below 90 mmHg and heart rate above 120 in adults.
2. In most cases *hemostasis* of external wounds is adequately performed by a compressive bandage. The use of arterial tourniquet is indicated only if other methods to stop arterial bleeding are ineffective. An arterial tourniquet should not be applied if the arterial bleeding is in the groin or the axilla, because it will be ineffective. If an arterial tourniquet has been placed, the site should be bared for constant inspection and never covered by blankets or sheets. Arterial bleeding in the neck, axilla, and groin should be treated by direct compression and/or surgical hemostasis.
   The pneumatic antishock garment is effective in reducing or stopping bleeding and maintaining central blood pressure. It increases metabolic acidosis, increases the incidence of acute renal failure, may cause a compartment syndrome, and is contraindicated in trauma of the diaphragm and in heart and lung failure. Deflation in the hospital should be performed progressively and only when surgical hemostasis is feasible immediately, and under appropriate blood and fluid resuscitation. Its optimal indication is severe internal bleeding due to isolated trauma or penetrating injuries of the pelvis and lower abdominal cavity. This is the only unequivocal indication I recommend at the time of writing.
3. *Endotrachial intubation* is indicated in:
   - Coma due to head injury
   - Airway obstruction due to oromaxillofacial injuries

- Upper airway obstruction due to inhalation of hot or corrosive vapors
- Presence of clinical symptoms of respiratory failure, manifested in adults by respiratory rate lower than 8 breaths per minute, or higher than 35 breaths per minute, cyanosis, labored breathing, use of respiratory accessory muscles, presence of pulmonary edema or evidence of respiratory muscle fatigue (paradoxical breathing, alternate breathing, asynchronous breathing)
- Persistence of circulatory shock in spite of massive fluid therapy
- Flail chest, with a large unstable segment

4. *Closed chest tube drainage* should be performed if hemo- or pneumothorax is evident or suspected on clinical examination. A sucking wound of the thorax cavity should be closed hermetically with vaseline gauze and the pleural cavity drained at another site. Tension pneumothorax is life threatening and if suspected, is immediately drained.
5. *Percutaneous pericardial drainage* is rarely indicated on site. However, if heart sounds are very distant or absent on direct chest auscultation whilst venous engorgement is evident in the neck and face and a thin peripheral pulse is present, a direct puncture below the xyphoid directed towards the left shoulder is justified.

First-Aid Station

Reevaluation. The vital signs of the patient are reevaluated: circulatory function, respiratory function, central nervous system status. The patient is then fully reexamined. The indications for the various therapeutic interventions described above are reestablished and the treatment given accordingly.

Priorities for evacuation to hospital. These include: persistent internal bleeding in the abdominal or thoracic cavity, craniocerebral trauma with suspected intercranial hematoma and/or increased intracranial pressure, arterial tourniquet, pneumatic antishock garment, progressive respiratory failure.
If the evacuation of the post-traumatic shock patient is delayed for more than half an hour, institute the following measures if possible:

1. Ventilate with pure oxygen during spontaneous or mechanical respiration and use a positive end expiratory pressure of 5 cmH_2O in all patients with respiratory failure.
2. Measure the central venous pressure (CVP), and use a fluid challenge protocol as follows: Infuse fluids rapidly until the CVP is 5 cmH_2O. If the patient is still in circulatory failure in spite of a CVP of 5 cm H_2O, continue fluid challenge by infusing aliquots of 200 ml of fluid every 10 min whilst measuring the CVP before and immediately after each infusion. The challenges should continue until the patient regains an acceptable blood pressure, pulse rate, and urine output, or until the CVP reaches 15 cmH_2O and does not increase by more than 2 cmH_2O for

each fluid aliquot. If the CVP increases by 5 cmH_2O increments or reaches 20 cmH_2O before the patient acquires hemodynamic stability, the fluid challenge is temporarily withheld, and an intravenous dopamine infusion is started (2 μg/kg per min) and titrated up to 10 μg/kg per min. If the CVP drops, the fluid challenges may be restarted together with the dopamine infusion until the patient achieves an acceptable hemodynamic status.

3. Perform nasogastric and urinary catheterization. Urinary output above 1 ml/kg per h is one of the best indicators of adequate fluid therapy for circulatory shock.
4. Consider blood transfusion for massive bleeding and/or patients with arteriosclerosis and a decreased cardiac reserve. (The use of typed packed cells is safe without crossmatching if the typing of the blood was performed in a blood bank using modern screening procedures.)

The Hospital

Only those patients arriving in circulatory shock due to persistent intra-abdominal or intrathoracic bleeding in spite of appropriate fluid and respiratory therapy are taken directly to the operating room. After ascertaining blood type, blood is given rapidly via a large-diameter intravascular cannula. In all other cases take the following steps:

Readjustment of Emergency Procedures and Hemodynamic Stabilization.

1. As necessary, perform endotracheal intubation and apply mechanical ventilation; introduce closed intercostal drainage, cannulate supplementary intravenous lines; perform hemodynamic stabilization by using the fluid challenge protocol.
2. Abdominal tap, computerized tomography with contrast pyelography and ultrasound aid in the assessment of intra-abdominal injury (perform computerized tomography only on hemodynamically stable patients).
3. Perform urgent laboratory tests: administer packed cells to achieve a hematocrit of 30% and platelet concentrate to achieve a thrombocyte count above 40000 and/or a normal bleeding time (fresh frozen plasma and cryoprecipitate are administered according to the coagulation profile).

Confirm the Diagnoses and Patient's Problem List.
Perform a complete physical examination and order:

1. Mandatory X-rays: upright or at least semiupright chest, lateral cervical, and PA pelvis (limbs, and other vertebra as necessary).
2. Computerized tomography, if available, for craniocerebral trauma wide mediastinum, and intra-abdominal unjuries.
3. Tests for blood type and match, hematocrit, arterial blood gases, electrolytes, blood sugar, coagulation profile. Record 12-lead electrocardiogram.

Surgical Priorities

The patient is then taken to the operation room for surgical treatment, with the aim of completing all major surgical procedures in less than 24 h. During anesthesia, it is imperative to monitor hemodynamic parameters, urine output, blood gases, hematocrit, coagulation factors, body temperature, and ECG. Laparotomy and/or thoracotomy, when indicated, should be completed as soon as possible. Vascular reconstruction and reperfusion of arteries should be performed by the 6th h after injury to ensure the best result. Neurosurgical treatments for intracranial bleeding, hematoma, or depressed fractures have a better outcome if performed within 5 h after injury. Insertion of an intracranial pressure monitoring device is frequently helpful for the patient's management in the ICU.
Internal or external fixation of bones should be completed before the 24th h after injury. After shock has been treated and surgery completed, the patient requires continuing treatment in an ICU wherever possible. Optimally the surgical procedures chosen are so planned as to lead to a patient in the ICU who does not need any further major surgical procedure in the following 12 days.

Management in the Intensive Care Unit

Hemodynamically Stable. After surgery, CVP and urinary output are adequate indicators for all patients who are not in cardiac or respiratory failure and whose pulse rate and blood pressure are within the normal range. The first goal of therapy for patients after recovery from circulatory failure is to eliminate as soon as possible any fluid excess which has accumulated from the emergency fluid therapy. If the CVP is high, a test dose of intravenous furosemide (20–40 mg) is used to initiate a high urine output.

Hemodynamically Unstable. If a postoperative patient is hemodynamically unstable and/or in *acute respiratory failure,* complete intravascular monitoring is necessary for optimization of therapy (pulmonary artery line and arterial line).

The ICU Fluid Challenge Protocol. Infuse fluids in aliquots of 500 ml rapidly until pulmonary wedge pressure reaches 8 mm Hg. Then continue with units of 200 ml at 10-min intervals and measure the wedge pressure before and immediately after each volume unit. Fluid challenge continues if the wedge pressure does not increase after a unit volume by more than 3 mm Hg and does not reach a wedge pressure of 18 mm Hg. If wedge pressure increases by 7 mm Hg after a single volume unit infusion, or reaches 18 mm Hg, fluid therapy is temporarily withheld and pharmacological support with dopamine and/or dobutamine is started.

If systemic vascular resistance is high and the mean arterial pressure is within or above normal range, intravenously applied sodium nitroprusside or nitroglycerin are indicated for preload and afterload reduction. (Patients with respiratory failure are ventilated with a volume cycle respirator and positive end expiratory pressure.)
A good response to fluid therapy is the best indicator of a favorable prognosis. The unfavorable prognostic markers are:

1. Persistence of lactic acidemia
2. Coexistence of very low systemic vascular resistance with high pulmonary vascular resistance
3. Persistence of a low cardiac index in spite of fluid and drug therapy
4. Persistence and aggravation of coagulopathy

In the early postoperative period, close monitoring is required. Prompt therapy for lung and kidney failure (the major target organs of post-traumatic shock) is instituted:

The Respiratory System. ARDS can result from hemorrhagic shock; its incidence increases if the shock is associated with major trauma, surgery, and/or massive blood and fluid infusion. Its signs are:

1. Hypoxemia in spite of increased oxygen concentration in the inspired gas (high FIO_2)
2. Diffuse alveolar infiltrate on chest X-ray film
3. Decreased lung compliance

Mostly it develops slowly within 12–96 h after the trauma, but in some patients it follows rapidly upon acute pulmonary edema after severe head trauma and massive fluid therapy for shock.
The treatment is:

1. Early use of mechanical ventilation with a volume cycle respirator and positive end expiratory pressure (PEEP).
2. Diuresis in order to achieve a negative fluid balance. Hemofiltration or plasmapheresis, if the diuresis is inadequate, are indicated.
3. Decreasing the pulmonary intravascular pressure by the negative fluid balance and pharmacological agents.

The signs of a good prognosis are: the ability to maintain arterial PaO_2 and $PaCO_2$ within the normal range and a concomitant decrease in FIO_2 and PEEP; and an increase towards normal value of lung compliance.

Acute Renal Failure. Early solute diuresis can prevent in many cases acute renal failure. The use of furosemide and solute diuresis may maintain diuresis in spite of reduced renal clearance and prevent anuric renal failure — nonoliguric renal failure is safer and easier to manage. Suspect acute renal failure when urinary volume is low, fractional excretion of sodium is higher

than 1%, and urine osmolality close to that of plasma. Confirm by finding an increase in serum creatinine and a decrease of creatinine clearance.
The therapy of oligo-anuric or progressively uremic patients is:

1. Early and frequent dialysis: Survival is enhanced by daily dialysis, by keeping the serum creatinine below 5 mg% (0.5 mmol/l) and the serum urea below 100 mg% (16 mmol/l). Hemofiltration is effective in removing fluid excess before and between hemodialysis. The use of bicarbonate in the dialysis fluid and the adjustment of the dialysis fluid osmolality aid hemodynamic stability during hemodialysis.
2. Adequate nutrition.
3. Eradication of infections: The eradication of existing infection and the prevention of sepsis and septic shock, becomes the prime preoccupation after the stabilization of vital functions has been achieved. The common sources of infection are: intra-abdominal abscess after abdominal surgery, leakage from a bowel anastomosis, infected hematoma and surgical wounds, contamination of intravenous and intra-arterial lines, and lung infection.
   Septic shock after major trauma surgery is the main cause of multiorgan failure. In the trauma patient without head injuries, multiorgan failure is responsible for most of the delayed fatalities after trauma.

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3.6 Primary Treatment of the Wounded: On-Site and Medical Aid Station

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The first phase of treatment is the stabilization of the vital functions and preparation for evacuation.

Stabilization of Vital Functions

The *most urgent* evaluation and treatment are needed for (in order of priority):

1. Cardiac arrrest and airway obstruction
2. Bleeding
3. Tension pneumothorax and open sucking chest wound
4. Hypovolemic shock

Spot Diagnosis: Check *rapidly* but *carefully* for external bleeding and look for the signs shown in Table 1.

Table 1. Checklist of signs to look for in primary spot diagnosis

Signs	Cardiac arrest	Hypovolemic shock	Tension pneumothorax
Alertness	Absent	Reduced	Agitation
Spontaneous breathing	Absent	Shallow, rapid	Extreme air hunger
Extremity movement	Absent	Reduced	Variable
Peripheral pulse	Absent	Weak, rapid	Weak (bradycardia or tachycardia)
Breath sounds	Absent	Present	Unilateral
Pupils	Dilated, fixed, no reaction to light – a sign of brain damage	Mostly normal (contracted if morphine was given)	Mostly normal

Cardiac Arrest

Cardiopulmonary resuscitation (CPR) is started by medical orderlies and continued by the medical officer as soon as he reaches the casualty. Since the cerebral cortex may be irreversibly damaged within 4–5 min of hypoxia, CPR must be started immediately. If respiratory arrest occurs (airway obstruction, head trauma), but circulation continues, the onset of brain damage is delayed by 2–3 min. In the field it is impossible to differentiate asystole from ventricular fibrillation or electromechanical dissociation. Thus CPR technique is the same for every case.

Traumatic Etiology

Airway obstruction: blood; secretions; foreign bodies (vomitus)
Central nervous system (CNS) depression: head trauma
Chest injury accompanied by: tension or open pneumothorax; rupture of a major bronchus; heart trauma
Exsanguination, especially intra-abdominal bleeding or injury to a big vessel Lung burns
Carbon monoxide poisoning (e.g., tank crews)
Severe and prolonged hypothermia

Prevention

Unless cardiac arrest is a sudden event, there is always a chance that one may prevent the patient's deterioration into cardiorespiratory arrest, if these rules are followed:

Keep the airways free by suction and intubation.
Maintain an adequate circulatory volume by arresting external bleeding (preferably by means of a pressure bandage or, failing this, a tourniquet) and infusion of fluids.
Perform intercostal drainage.

Diagnosis

The signs of cardiac arrest are:

- Absence of carotid or femoral pulse (or if the femoral pulse is present, blood pressure (BP) lower than 60 mmHg).
- No audible heart sounds. In obese patients this is not a reliable sign.
- Respiratory arrest or gasping.
- Loss of consciousness; no reaction to pain (the presence of convulsions means that the brain is not anoxic — yet !).
- Pupillary dilatation (an indication of brain anoxia).
- No spontaneous movement of limbs.

Treatment

Treat the cause of cardiac arrest if it is evident whilst simultaneously performing CPR. The proper order of treatment is: airway, breathing, circulation, drugs.

Airway

1. Tilt the patient's head backward.
2. Displace the lower jaw forward (jaw thrust).
3. Check rapidly for face injury; the origin of blood in the mouth or pharynx; the accumulation of secretions; teeth avulsions or foreign bodies.
4. Clean the upper airways immediately by using: gauzes, fingers, or a portable suction instrument (driven by a foot mechanism), if available.

Breathing

1. Check the victim's spontaneous respiration. If respiration is superficial or the patient is apneic, *start artificial ventilation* by mouth-to-mouth breathing (16% oxygen), or, better, using an Ambu-mask bag (a self-filling, non-rebreathing system, 20.9% oxygen). Attach available oxygen source to the Ambu if available.
   Only then:
2. Intubate. Don't waste time — intubate orally; check that the distal tip of the tube lies above the carina tracheae (the distance between the teeth and the tracheal bifurcation is not more than 21–22 cm). Use your stethoscope to verify air entry into both lungs. *NB:* The first place for auscultation is the epigastric area (in order to ascertain the tube is not in the esophagus!).
3. Alternatives to intubation are:
   a) *Tracheostomy:* Indicated when it is impossible to intubate the trachea through the larynx, i.e. when there is severe maxillofacial injury or severe oropharyngeal bleeding. The disadvantages of this method are that is time consuming and demands special skill.
   b) *Cricothyreotomy:* the introduction of a small artifical metallic airway into the larynx through the crico-thyroid membrane. Indications are the same as for tracheostomy. The disadvantage is that the airway is of small diameter and may therefore became obstructed by blood and secretions.

Circulation

External cardiac massage should be started as soon as possible. Position the victim on his back, on as firm a surface as can be found. Apply pressure to the lower half of the sternum, at a rate of 80–90 compressions per minute. If there is only one rescuer, he should alternate 15 sternal compressions and two lung inflations (mouth-to-mouth or bag ventilation). If two rescuers are

available, they should work at a 5:1 ratio (with no interruption of cardiac massage. The attempt to assess efficacy should be made by palpating the victim's peripheral pulse (preferably the femoral, since the carotid pulse is difficult to feel during resuscitation maneuvers)

Drugs

Rules

1. *Do not* use the intracardiac route. If there is "no open vein", use the endotracheal route: intracardial puncture may cause pneumothorax, bleeding, and pericardial tamponade or myocardial lacerations; besides, it would necessitate interruption of the cardiac massage.
2. Establish an intravenous route as soon as possible by percutaneous peripheral puncture, percutaneous central vein cannulation see below) or venesection of the long saphenous vein.

Specific Drugs

1. Epinephrine: 1 mg intravenously or through the endotracheal tube. Epinephrine improves the perfusion pressure by increasing myocardial contractility.
2. Sodium bicarbonate: 1.5 mg/kg body wt. as a loading dose, then 20%–30% of this dose every 5–10 min. Too much bicarbonate may lead to dangerous metabolic alkalosis.
3. Atropine: 1 mg intravenously in case of bradycardia. This dose can be repeated every few minutes.

Administration of Fluids

In trauma, cardiac arrest is provoked by exsanguination. Fluid replacement is therefore one of the major steps in CPR.

Table 2. Signs indicating outcome of CPR

Signs	Successful resuscitation	Successful resuscitation with brain damage	Unsuccessful resuscitation
Consciousness	+	−	−
Peripheral pulse	+	+	−
Spontaneous respiration	+	+	−
Pupils	Small, quick response to light	Fixed, dilated unresponsive to light	Fixed, dilated unresponsive to light
Skin color	Normal	Normal or cyanotic	Cadaveric

When to stop CPR?
The victim in a war or a disaster is usually a young healthy person. Therefore, consider the CPR unsuccessful *only* if signs of death are present after 1 hour of continuous resuscitation (see Table 2). Even when CPR is only partially successful there is still some hope of reversing brain damage or preventing further CNS deterioration.

Tension Pneumothorax

The priority of treatment of tension pneumothorax is after CPR, equal to the staunching of major bleeding, and *before* volume replacement. *Definition:* Tension pneumothorax is pneumothorax associated with progressive accumulation of air trapped in the pleural space.

Pathophysiology

The pleural pressure is converted from a negative to an increasingly high positive pressure and mediastinal shift compresses the inferior vena cava as it passes through its diaphragmatic hiatus, impeding venous return to the heart. Contralateral lung compression along with pulmonary collapse on the affected side results in severe hypoventilation.
Hypoperfusion and hypoventilation lead to hypoxia which, if not corrected, will result in rapid death.

Diagnosis

The signs of tension pneumothorax are:

- Acute air hunger with very rapid, shallow respiration
- No air entry and very resonant tympany to percussion on the affected side
- Engorged neck veins
- Fall in BP
- Subcutaneous crepitation in the neck
- Cyanosis
- Acute anxiety

Treatment

To save life, the pleural space must be decompressed *immediately*. Use the device with the largest lumen available on the spot: a trocar, a large intravenous cannula, a 14 or 16 gauge needle. Insert it into the fifth intercostal space, in the midaxillary line, not more than 2 cm beyond the rib (in order to avoid lung puncture), thereby converting the tension pneumothorax into a simple one.

An improvement of the clinical state: palpable peripheral pulse, improved skin color, normal BP, reduction in respiratory distress and cyanosis, and disappearance of engorged veins, is evidence that the emergency decompression has been effective. Insert a chest tube. The best device in field conditions is the *one-way valve intercostal drain,* and this must be available at all levels and echelons. Its advantages are that it is a simple device which permits a rapid decompression and does not need an underwater seal.
In hemopneumothorax, make a rapid assessment of the amount of blood lost in order to replace it by an appropriate volume of the available solution.

Hypovolemic Shock

The two major aims in treatment of hypovolemic shock are cessation of bleeding and correction of hypovolemia.

Cessation of Bleeding

Identify the source of bleeding. If hemorrhage from a major artery or vein is clearly visualized, the vessel should be clamped both proximally and distally.
External hemorrhage can usually be controlled by pressure on the site of the bleeding vessel or at one of the "pressure points" by applying a pressure bandage or by direct digital pressure. This should be the sole occupation and responsibility of one attendant.
If arterial bleeding from a limb is not controlled by pressure apply a tourniquet. The tourniquet to be used is the simple Esmarch broad rubber elastic bandage. It is applied proximally as close to the wound as possible and tied and strapped to the limb so that it cannot become loose and so that the casualty himself cannot untie or loosen it! Inspection of the wound should ascertain complete cessation of bleeding other than an ooze from the bone itself which cannot be staunched by a tourniquet.
Write on the skin next to the tourniquet or on a piece of tape applied to the limb the time and date of application of the tourniquet. The tourniquet may be released only by an authorized experienced medic or physician and reapplied as required if the bleeding recurs. If no experienced personnel are available the tourniquet must be left in place until the casualty reaches primary wound surgery in the field hospital. Poor tourniquet application is extremely dangerous — better to lose a limb than a life!
Immobilize a fractured limb. Blood loss is increased by instability of the fracture and associated vascular injury.

Administration of Fluids

Peripheral intravenous infusion must be started immediately, if possible. In cardiac arrest or shock, however (e.g., severe bleeding, tension pneumotho-

rax, etc.), it is very difficult to find a suitable peripheral vein, since the veins are collapsed due to hypovolemia and compensatory peripheral vasoconstriction. If this is the case, do not waste time in searching for a "nonexistent" peripheral vein but decide on the *central venous approach.*
Conditions in the field are unsuited for an accurate measurement of the central venous pressure; thus the indication for introducing a central vein catheter is only the impossibility or failure of cannulation of peripheral vein. The order of choice of vein is: (1) internal jugular vein, (2) femoral vein.

Rules
1. Respect sterility as much as field conditions permit.
2. Try first the *right* internal jugular vein puncture: the distance to the superior vena cava is shorter and the right pleural dome is lower than the left.
3. Keep the patient in the head-down position: this will fill the vein lumen with blood even in the presence of hypovolemia.
4. Use only an "over-the-needle" catheter which reduces or prevents such complications as bleeding, cutting the distal tip, or misplacement.

Internal Jugular Vein
Technique. Extend the neck and turn the head toward the opposite side. Keep the fingers of the left hand on the carotid artery. The skin puncture point is along the lateral border of the sternocleidomastoid muscle, 0.5 mm lateral to the carotid artery and 4–5 cm above the clavicle.
Search for the vein by *first* using a large intracatheter. Direct the needle distally at 45° to the skin plane and parallel to the median line of the sternum. Advance it slowly and progressively. Once blood returns, leave the needle in place and insert the plastic catheter. Attach the infusion, then lower the infusion bag to observe backflow of blood.
Since a chest X-ray cannot be done in the field, make sure that the distal tip of the catheter is in the right position by: (a) lowering the infusion bottle below the right atrium level and looking for the blood syphoning into the infusion set, (b) observing the respiratory oscillations of the fluid in the catheter *before* connecting it to the infusion set, and (c) ensuring the absence of "ventricular pulsations" of the fluid level: if these are present, withdraw the distal tip of the catheter, which is in the right ventricle. Apply a sterile dressing to the skin.
For additional security the catheter should be sutured to the skin at two points, and the tubing looped behind and over the patient's ear and taped.

Femoral Vein
The advantages of choosing the femoral vein for administration of fluids are that it is an easy approach and the only anatomical landmark you need is the femoral pulse (it is present even in shock). In addition, there is no need to interrupt CPR in order to cannulate the vein. However, since femo-

ral vein catheterization is associated with a high rate of thrombophlebitis and misplacement, the femoral vein should be chosen as an *easy* and *urgent* approach rather than a long term route of infusion.

Technique. Use the femoral vein on either side. Do not shave the skin unless hair is causing an obstruction. The skin puncture point is just below the inguinal ligament, 0.5–1 cm medial to the arterial pulsation.
Use an 18 gauge needle and only an "over-the-needle" set (see above). Because of the risk of thrombophlebitis and misplacement, *do not insert* long catheters (50 cm) such as are used for measurement of central venous pressure.
Use the same sequence as for the internal jugular vein: 18 gauge needle, wire guide, 16 gauge catheter. Suture the catheter to the skin and apply an occlusive dressing.
As soon as circulatory disturbances are corrected, try to cannulate one of the peripheral veins or, failing this, the internal jugular, and then remove the femoral catheter.

Subclavian Vein
A subclavian cannulation may cause pneumothorax and is not recommended on site or in the medical aid station.

Solutions
Ringer Lactate: An electrolyte solution containing Na^+, K^+, Ca^{++}, Cl^- and lactate.
Advantages: An almost physiological osmolality (275 mosmol/l); a relatively high concentration of sodium (130 mEq/l); a pH of 5. The lactate, transformed by a normal liver into bicarbonate, contributes to the treatment of metabolic acidosis, which usually accompanies hypovolemic shock. It is inexpensive and available in plastic bottles of 1000 ml.
Drawbacks: Ringer lactate leaves the intravascular space a short time after administration and reduces (by dilution) the colloid-osmotic pressure of plasma.

Hemacel: A plasma expander, containing gelatin (molecular weight 30000).
Advantages: A half-life 6 h; a longer presence of the molecule in the circulation; no interference with blood clotting; no side effects.
Drawbacks: Hemacel is more expensive than Ringer lactate.

Alternatives
Dextrans: Molecular weight 40000–72000; interfere with clotting and blood group determination; very rarely provoke anaphylactic reactions.
Saline: Crystalloid solution containing Na^+ and Cl^- in equal amounts (155 mEQ/l). No lactate; osmolality 310 mosmol/l).

Note: Under field conditions blood and its products are not easily available. Do not waste time! The first choice for bleeding is either Ringer lactate or a plasma expander. Use them in the first urgent stage to expand the blood volume: the red cell and plasma deficit can be dealt with later.

Amount and Rate of Infusion

The amount and rate of infusion are decided by the rough estimate of the amount of blood lost, which depends on the type of the injuries (see Table 3) and the clinical signs of shock: signs of shock indicate that at least 30% of the blood volume has already been lost and that this amount must be infused at maximum speed at once (normal blood volume is 80 ml/kg). *Example:* a casualty estimated to weigh 70 kg has a blood volume of $80 \times 70 = 5600$ ml. The signs of shock indicate that at least nearly 2000 ml have been lost to the circulation. This amount is therefore infused at maximum speed.

The blood pressure is not always a reliable sign. Compensatory mechanisms (tachycardia and peripheral vasoconstriction) can delay the drop in BP and mask the preshock and early shock phases.

If the field conditions permit, test orthostatic hypotension: a head down and then up tilt is done and BP and heart rate (HR) are measured. A drop of more than 20 mmHg in systolic BP and/or an increase of more than 20 heart beats/min speaks for at least 25% volemic deficit.

The clearcut clinical signs in shock are: tachycardia, thin pulse due to hypotension; obnubilation or agitation (signs of cerebral hypoxia),oligoanuria, tachypnea, cyanosis, pallor.

If field conditions permit an aseptic technique, insert an urinary catheter. An hourly urine production of 30 ml or more indicates adequate fluid replacement of hypovolemia.

Results of treatment which evidence hemodynamic stabilization are: improvement in consciousness, a stable BP, 30–60 ml/h urinary output, a strong peripheral pulse, and normal skin color (warm, dry, pink).

Table 3. Estimated blood loss by injury type (adults)

Neck, face, throat	From 500 cc up to many liters
Intra-abdominal bleeding	From 500 cc up to many liters
Intra-thoracic	From 500 cc up to many liters
Fractured pelvis	From 500 cc up to many liters
Fractured femur	1–2 liters
Fractured tibia	½–1 liter
Fractured fibula, radius, ulna	¼–½ liter
Hand wounds, severe scalp wounds	½–2 liters
Fractured ribs	200 ml per rib

Analgesia

Many wounds are not extremely painful, and often, after dressing and splinting, only gross movements cause pain. Analgesics are therefore often not needed before transportation and should be given only for severe pain and after the examination of the casualty has ruled out an acute abdomen, which would be masked by morphine.

Indications

Analgesics may be given to relieve severe pain, to eliminate the contribution of severe pain to shock, and to facilitate transport.
Morphine is the drug of choice, as:

1. It has the desired triple effect: analgesia–sedation–amnesia.
2. The onset of analgesia is rapid.
3. The analgetic effects last a long time.
4. It may be administered intravenously or intramuscularly.
5. It causes almost no cardiovascular depression in a supine, normovolemic patient.
6. In clinical doses it provokes hardly any histamine release.
7. There is a direct relationship between the dose and respiratory depression effects.

Administer morphine at a dose of 10–16 mg intramuscularly or 4–8 mg intravenously. Repeat the dose according to the efficacy of the first dose and the patient's respiratory state.
Exercise caution in regard to the following points:

Table 4. Dosage and duration of action of opiates

Generic name	Equipotent dose (mg)	Duration of action (h)
Natural opiates:		
Morphine	10	4–5
Semisynthetic opiates:		
Oxymorphone	1–1.5	4–5
Hydromorphone synthetic deriv.	1.5	4–5
Meperidine	80–100	2–4
Pentazocine	45–60	3
Nalbuphine	10	3–6

1. In a hypovolemic patient, morphine administration could further lower the cardiac output, so use morphine sparingly in shock cases. Morphine should *never be given intramuscularly* in shock: *only intravenously*. Poor absorption after intramuscular administration in a hypovolemic patient may be suddenly increased as the patient comes out of shock, and there is a danger of respiratory depression during this "rebound flushing" phenomenon.
2. Large doses can produce marked respiratory depression. Do not administer a fresh dose unless the respiratory rate is above 12/min.
3. Morphine can have unpredictable effects on patient behavior.

Dressings and Splinting On Site

Dressings

A most urgent wound dressing is that for an open sucking chest wound. The deficit in the chest wall should be blocked up by a voluminous dressing — preferably with a fatty petroleum jelly gauze adjacent to the wound — and partially stuffed into the pleural cavity, in order to seal the air leak. The dressing is then bandaged and/or strapped to the chest by firm circumferential bandaging or taping.

The second very urgent dressing is the pressure bandage to stop bleeding. This is the only indication for circumferential compression bandaging on a limb. The wound is packed with sterile gauze pads which should project out 2–3 cm above the level of the skin and a circular bandage is applied with sufficient force to stop the local bleeding while ensuring that the peripheral blood flow (pulse and/or capilliary return) remains intact.

If hemostasis fails because, in a bulky or — especially — fat limb, the application of local pressure to depth of a wound by bandaging is difficult, a hard object such as a stone wrapped in a thin layer of sterile gauze should be placed over the wound and included in the bandage, thereby increasing the local pressure exerted into the wound. Failure to stop the bleeding by pressure bandaging indicates a need for pressure-point digital compression or for a tourniquet.

General wound dressing is never by circumferential compression. Sterile gauze pads should cover the wounds with a generous margin to spare. Large "abdominal" pads are used to cover large wounds, but the "personal" dressing with its tapes for tying is the most useful for most types of wounds. The dressings are meant as a cover to prevent further contamination from the environment: the tapes are tied around *lightly*, only tight enough to stop the dressing from slipping. They must never embarrass the blood supply to the limb or block the venous drainage.

For burns and special dressings, see the appropriate chapters.

At the *medical aid station* the dressings are inspected. If they are adequate and there is no external bleeding, *they should be left alone:* every unnecessary change of dressings is painful and increases the risk of infection. No purpose is served at this stage by inspecting the wound.
Dressings should not be removed in the emergency reception area of the *field hospital or base hospital.* Unless there is a specific indication to inspect a wound for bleeding or a need to renew a faulty dressing, all wounds should remain covered by their original dressings until the casualty is anesthetized and ready for primary wound surgery in the operating theater.

Splinting (On-Site and Medical Aid Station)

Splinting is carried out to immobilize unstable skeletal parts so as to prevent further serious injury (e.g., to spinal cord in spinal fractures), to aid vital function (e.g., in flail chest), and to prevent shock and reduce pain (e.g., in limb fractures), and also to rest the soft tissue wound.

Spine: Whenever a cervical spine injury is suspected, place the *neck* of the supine patient in a hard collar or support it by sandbags or heavy objects placed on both sides of the head. Support the neck in the neutral position at all times. For *thoracic* and *lumbar* spine injuries, avoid spinal movement: roll the casualty onto a door or hard stretcher or, preferably, use the four-man technique (Chapter 4.3) for transport.

Flail Chest: Breathing is improved by positional splinting of the chest wall. When the flail segment is unilateral lay the patient down (preferably half-sitting) on the injured side. When the flail segment is anterior, other injuries allowing, place him prone with a pillow under the chest. These positions will reduce the paradoxical movements of the unstable segments of the chest wall, thereby enhancing ventilation.

Limbs: Upper arm: with the elbow bent to a right angle, bandage or tape the upper limb firmly (but not tightly) to the chest in the Velpeau position with the addition of a collar and cuff (Fig. 1). Elbow and forearm: use the Velpeau bandage or a padded wire Cramer splint lightly bandaged on posteriorly from axilla to knuckles.
Hand: see Chapter 4.11.
Thigh and knee: fractures of the femur and around the knee are best splinted on site by tying the two lower limbs together above and below the knees and at the ankles. Thomas splints are applied at the medical aid station or field hospital, with traction applied to the boots.
Leg: apply a padded wire dorsal Cramer splint from the upper thigh to the tips of the toes with the knee bent 20° and the foot plantigrade.

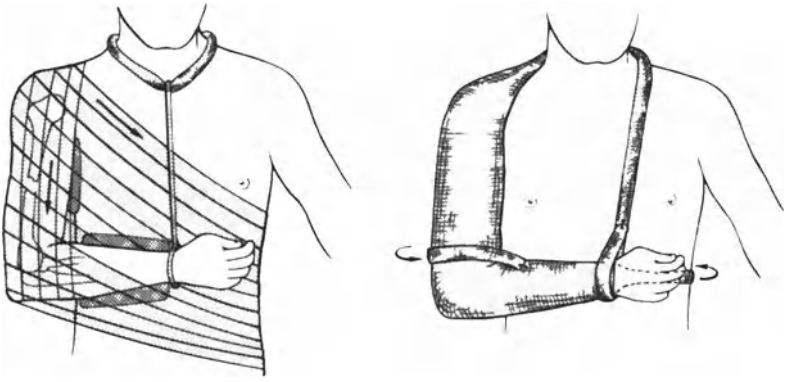

Fig. 1

Ankle and foot: apply a padded wire Cramer splint placed dorsally from below the knee to beyound the toes with the foot plantigrade.

Fluids

At the scene of disaster or on the battlefield casualties are usually underhydrated. If there is no contraindication, water should be given by mouth ad lib.

If the casualty is unable to drink or there exists any contraindication to oral imbibition, an intravenous infusion of Ringer lactate is indicated as routine, at the rate of 1 l in the 1st h and then 3 l in the ensuing 24 h; these quantities should be adjusted according to climatic conditions, urine output, the previous general medical and cardiologic state of the patient, and ensuing developments associated with the injury.

Antibiotics

In all open wounds it is desirable to commence intravenous antibiotic therapy, by giving 5 million units of crystalline penicillin every 6 h and 80 mg garamycin every 8 h at the medical aid station.

Medical Records

Medical records should be kept on the body of the casualty at the medical aid station. The following points are to be stressed:

1. Record the injury score at the time of primary treatment and prior to evacuation.
2. Ensure that the record contains a list of all the injuries and treatments given.
3. Record tourniquet application and time in a prominent place.
4. Record the time, type, and agent of injury.
5. Include all the information available concerning the casualty's identity.

Regional Wounds

For the on-site and medical aid station care see the appropriate chapters.

Hypothermia

Hypothermia is an insidious stealthy danger in the field. Routinely record the temperature and ensure that casualties are adequately covered whilst awaiting evacuation. For the treatment of established hypothermia see Chapter 2.4, p. 84.

Evacuation

The priorities of evacuation are closely related to the priorities of treatment. The primary treatment in the field described in this chapter *should always be performed prior to evacuation:* i.e., evacuation must be delayed until the primary treatment has been scrupulously completed. Evacuation of a stabilized casualty gives a much better prognosis than a "scoop and run" evacuation. However, should circumstances prevent primary treatment on site or at the medical aid station, then the priorities for evacuation are according to the need for primary treatment: i.e., resuscitation (CPR), asphyxia, hemostasis, tension pneumothorax, volume replacement for shock — in short, all those steps previously described in this chapter.
A useful list of priorities for evacuation is as follows:

1. Uncontrolled asphyxia, respiratory obstruction, tension pneumothorax, open pneumothorax, and shock due to any cause, including external and internal bleeding and other grave injuries.
2. All cases in which CPR was successful and all conditions listed under point 1 which have been stabilized by primary treatment.
3. All internal injuries of the chest and abdomen without major internal bleeding and asphyxia. Burns of more than 30% total body surface. Vascular injuries needing repair: all casualties on whom a tourniquet has been applied. Head injury with a worsening Glasgow Coma Scale score.
4. Multiple fractures. Open fractures. Soft tissue wounds with muscle damage. Open head injuries and head injuries with altered consciousness. Spinal injuries.
5. Burns of 30% or less. Closed fractures. Soft tissue wounds without much destruction. Eye injuries. Ear-nose-throat and maxillofacial injury without airway disturbance.

Final Check before Evacuation

1. Make sure that
   - Cardiovascular status is stable
   - Alveolar ventilation is efficient
   - Pleural drainage is working
   - Intravenous lines are patent
   - Stomach is empty (nasogastric tube in place)
   - Fractures are immobilized
   - Tracheotomy wound does not bleed
   - Patient is comfortable and pain-free
   - Urinary catheter is functioning
2. Put everything you have done in writing.
3. Transmit all the necessary information to the team in charge of evacuation.
4. Accompany the wounded to the heliport or parking lot and keep an eye on vital signs during this last stage of the management.

3.7 Primary Care of Burn Victims

R. Shafir and J. Weiss

The ideal initial care of the burn victim will be described here, but compromise may be dictated at the time by the magnitude of the disaster and the degree of availability of medical aid. The initial care is divided into immediate and delayed primary acts on site and/or at the primary medical facility. Immediate care involves the following steps:

1. Remove the cause of the burn
2. Triage — select the treatable from the dead or doomed and the severely wounded from less urgent cases
3. Assure a free airway
4. Search for associated injuries (arrest bleeding)
5. Estimate rapidly the severity of the burn
6. Cool heat-inflicted burns

Removal of Burn Causes. The first and most urgent step to be taken is removal of the cause. Fire victims must be moved from confined spaces into the open air. A running person in flames is stopped and flung to the ground (so that the flames will not engulf his head). The flames are extinguished by rolling the victim on the ground, by sand, blankets, or cloth. With chemical burns the need is for an instantaneous rinse, preferably with copious water, for several minutes, or buffering with diluted alkaline (bicarbonate soda) or acid (acetic acid). Phosphorus burns (garlic smell and continuous fuming of the tissues) are treated by immersion in water or covering with a water-soaked cloth, which must be kept soaked until the arrival of the burn unit. In the case of electric burns, if possible, the electricity should be switched off. If not, the patient's hand or any other part in contact with the source should be struck off with cushions or shoes.

The Suffocating Patient. Burn victims from closed environments (rooms, vehicles) which delayed evacuation may suffer the inhalation of carbon monoxide or other carbonaceous or toxic fumes, resulting in suffocation. Initial treatment is by giving 100% oxygen by mask or intubation, if necessary. Inhalation of very hot air, especially hot moist air (steam), causes laryngeal spasm and oedema, requiring immediate intubation.

Triage and Search for Associated Injuries. The burn victim is not a first priority emergency, even if first appearances suggest the worst. Chest, abdom-

inal, head and vascular injuries take precedence for evaluation and treatment. However, the burn may well distract attention from another more urgent injury, and this must be borne in mind constantly. Semi-urgent airway problems will be discussed later.

Rapid Estimation of the Severity of the Burn. A quick evaluation of the extent and degree of the burn can only be determined after the patient has been completely divested of removable, non-adherent clothing. The entire body surface of patients should be examined, not forgetting to roll them on their side in order to view the back.

Cooling. By cooling heat and chemical burns, dermal temperature and vasoconstriction are reduced (thereby limiting the depth of the burn, the severity of tissues damage and oedema). Cooling is most effective if started at once by immersion in cold water, or the application of ice bags (avoiding direct contact between the ice and skin). It is an important step in treatment of heat burns but is logistically hard to perform and time-consuming. On the other hand, it is not a life-saving procedure. The possibility of its use should be kept in mind, but it should not take the place of other emergency procedures.
Cooling at $-3°C$ is limited to 30 min and only to isolated body parts, otherwise hypothermia develops. At 15 °C, 60 min of cooling is necessary.

Delayed Primary Emergency Care

The next stage of treatment in order to priority is:

1. Analgesia
2. Fluid replacement
3. Estimation of the severity of the burn and associated injuries
4. Respiratory care
5. Urinary catheterisation, gastric tube insertion
6. Warming of the patient
7. Elevation of the limbs

Analgesia. The burn victim may be severely agitated, stressed and in pain. Analgesics must be administered intravenously, never intramuscularly, or subcutaneously, since medications thus administered accumulate in the oedema fluids and are thus rendered ineffective, only to be later absorbed in large toxic quantities. Morphine can be given cautiously.

Fluid Replacement. Fluid is administered through large bore superficial veins. If none are available, then a central vein is cannulated. Burned areas should not be punctured if possible, and the use of a peripheral vein in the burned limbs should be avoided. Every burn victim losing more fluids than can be taken in needs fluid replacement, and this begins to occur in a sec-

ond degree burn over 20% of the body surface area (in infants and the aged, even smaller burns). The standard first-aid fluid, most readily available and easiest to store, is Ringer's lactate solution.
Fluid requirements are calculated according to the estimated extent of the burn and the patient's weight. The team should be acquainted with one formula. The modified Brook formula, 2 cc (3 cc in children) of Ringer's lactate for each per cent of burn area multiplied by the kg body weight is administered in the first 24 h. At least half of that amount should be infused within the first 8 h. Effectiveness of fluid replacement is best evaluated by urine output — the optimal amount is 50 cc/h in adults. Diuretics should be avoided if at all possible, unless large amounts of fluid do not initiate urine flow, and the catheter has been checked for patency. Blood transfusion should only be given when blood loss is causing haemorrhagic shock.

Estimation of the Severity of the Burn and Associated Injuries. Treatment and grading of evacuation priorities depend upon this vital step. Anamnesis includes the cause, for example, the composition of the chemical causing a burn; the site, whether it was a closed or open-air environment; the length of time the victim was trapped in the enclosed fuming atmosphere; and any pre-existing disease such as heart, lung or kidney conditions, diabetes, medication such as steroids etc. If the environment was closed, the composition of the burning materials must be ascertained, giving information concerning the inhalation of toxic materials. A rough estimate of the burn surface area was made initially. Now defining more exactly the burn surface area is necessary for calculating the fluid replacement formula and assessing the severity of the injury. The *rule of nines* is employed (Fig. 1) (in children the head comprises 20% of body surface area as compared with 9% in the adult). The victim must be rolled onto one side to look for burns or injuries of the back. Burn depth estimation at this stage will only be very rough, differentiating between superficial (i.e. first and very superficial second degree) and deep burns. The former are characterised by areas of either reddened intact skin, or a very thin layer of peeled-off skin, with red healthy dermis underneath. These areas are very sensitive. Deep burns have white dead tissue under an easily peeling carbonised layer and are anaesthetic. The severity of the burn increases with old age and in infants.
The grading of the victim's condition depends mainly on the area of second or third degree burns. Medium severity injury is a burn of 20%–40% body surface area of second degree or 20% of third degree. Severe injury concerns a second and third degree burn of over 40%. The condition is critical if over 60% body surface area is involved by deep burns. Concomitant injuries, inhalation injuries, and age upgrade the severity.

Catheterisation of Urinary Bladder. Monitoring of fluid replacement is accomplished by hourly measurements via a catheter. A gastric tube is inserted in severely burned cases to avoid acute dilatation and treat paralytic ileus.

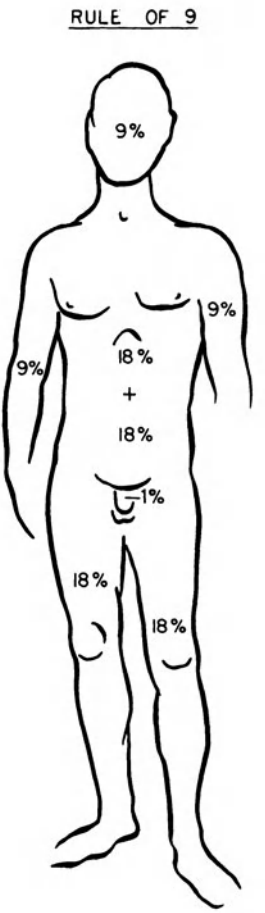

Fig. 1

Respiratory Care. While immediate respiratory problems may not have been encountered, they can nevertheless develop later, either in the upper or lower airway. Severe facial and neck burns are prone to develop laryngeal oedema (stridor, hoarseness and tachypnoea). Early intubation may obviate a later urgent tracheostomy through burned tissue, which in itself can lead to lethal complications. Tracheal and/or bronchial occlusion by oedema or retained mucous secretions appear at a later stage (within some hours of injury), usually as part of an inhaltion injury. The clinical signs of inhalation injury are a history of acquiring the burn in an enclosed area, facial and especially lip burns, burned nasal vibrissae, wheezes, erythema

and carbonaceous material in the oral cavity and carbonaceous soot in the sputum. Urgent evacuation to an intensive care unit is warranted.

Remember: if in doubt, it is safer to introduce a laryngeal tube prior to evacuation, rather than having to intubate under even more difficult conditions, or perhaps miss the chance for intubation, and have to perform a tracheostomy.

Warming. Cooling has an important local effect on the burned skin itself, which is not contradicted by warming. Warming reduces heat loss from the burned areas, thus reducing shivering and energy loss. Warming is achieved by covering the victim with thin metal sheets or special burn dressings, under which warm humid air accumulates, reducing heat loss and fluid evaporation.

Elevation of the Limbs. By reducing oedema accumulation, escharotomies may be delayed or avoided.

The following "do nots" will avoid common errors:
1. Do not give painkillers intramuscularly or subcutaneously.
2. Do not try to reverse burn shock with blood transfusion.
3. Do not give diuretics unless the fluid load has been thoroughly implemented.
4. Do not give oral fluids in severe burns.
5. Do not give steroids for treatment of burn shock or inhalation injury.
6. Do not use vasopressors in place of large amounts of fluids.
7. One shot of penicillin may be given, but do not start sophisticated antibiotics.
8. Do not apply topical ointment therapy unless the patient is to be retained for a prolonged period, as it may cause difficulties in accurate estimation and treatment at the burn unit.

Evacuation

First priority evacuation is afforded to wounded with haemorrhages, penetrating abdominal or chest wounds and brain injuries. Second priority evacuation is for infants and patients with inhalation injuries. Third priority is for chemical burns of the eyes, phosphorus or electrical burns. It should be noted that the superficial examination of an electrical injury may be misleading, since electrocardiographic changes have to be excluded as well. The adult burn patient with no other injuries can safely be kept at the site of trauma, until the previously mentioned priority categories have been safely evacuated.

3.8 Treatment of Burns in Hospital

R. Moscona and B. Hirshowitz

Primary Evaluation and Emergency Management

The priorities of attention are: airway, shock, associated injuries, the burn wound itself and pain.

Airway

Respiratory distress is not due to the burn unless the victim has suffered an inhalation injury or has severe restriction of the chest wall movement due to circumferential burns. First look for other causes of respiratory embarrassment and then pay attention to the early signs of inhalation injury: burns around the mouth, scorched nasal hair, soot in the oropharynx, hoarseness, cyanosis, tachypnea. On admission obtain blood gases and perform a chest X-ray. Fiberoptic endoscopy is a useful diagnostic tool in some patients. During the shock phase (36–48 h), blood gases are checked every 3–6 h, and attention is paid to any early appearance of hoarseness or change in respiratory rate or sounds. If the PO_2 level is above 65 mmHg and respiratory distress is not severe, nebulized oxygen should be administered through a face mask. If PO_2 level drops under 65 mmHg and severe tachypnea or hoarseness develops, endotracheal intubation is the treatment of choice. This allows adequate toilet of the respiratory tract and, with proper management, can be used for long periods without causing permanent damage to the larynx or trachea.

Tracheostomy is a simple surgical procedure which gives immediate respiratory relief but is complicated by an increased rate of pulmonary infections and the late development of laryngotracheal stenosis. Its use is restricted to those cases in which intubation is not possible or contraindicated.

Shock Status

Monitoring. Blood pressure, pulse rate, respiratory rate, and body temperature are measured hourly. Other clinical signs are: temperature of the extremities and nasal tip, the state of consciousness, and restlessness.

Obtain blood samples for hemoglobin and hematocrit levels, serum electrolytes, crossmatching, and order plasma as required (see below). Ask the patient to evacuate the bladder: urine output is measured henceforth. (The insertion of a catheter is contraindicated as a routine practice unless the victim is definitely in shock, unable to micturate, unconscious, has a pelvic injury, or is paraplegic.) The central venous pressure (CVP) is not measured routinely because infection may enter the blood stream along the CVP catheter tract (hemodynamic changes in burned patients occur gradually, and satisfactory control is possible without its use).

Treatment. A peripheral IV line is introduced as far as possible away from the burn site using large-size cannulas; whilst central lines, if not mandatory, should be avoided, a saphenous venous cutdown at the ankle is often useful. An infusion of 1000 ml lactated Ringer's solution is commenced until a crossmatch is performed and plasma becomes available (see later).

Associated Injuries

A detailed history is taken and a complete physical examination is made to search for associated injuries. Priorities are established accordingly.

Burn Wound

Expose the patient completely. Evaluate the burn size according to the "rule of nines" or the Lund and Browder Chart (Fig. 1), in which modifications are made for age. Determine burn wound depth (partial or full thickness) and fill in the chart.
The patient is covered with a sterile sheet and sent to the burn unit.

Pain

Administer IV narcotics in sufficient dosage. Avoid the subcutaneous, intramuscular, and per os routes from which absorption is uncertain due to shock and/or edematous fluid accumulation; this may create a dangerous "backlash" phenomenon if multiple doses are absorbed simultaneously at a later stage.

BURN ESTIMATE AND DIAGRAM

AGE vs. AREA

Area	Birth 1 yr.	1–4 yr.	5–9 yr.	10–14 yr.	15 yr.	Adult	B 2°	R 3°	Total
Head	19	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Ant. Trunk	13	13	13	13	13	13			
Post. Trunk	13	13	13	13	13	13			
R. Buttock	2½	2½	2½	2½	2½	2½			
L. Buttock	2½	2½	2½	2½	2½	2½			
Genitalia	1	1	1	1	1	1			
R.U. Arm	4	4	4	4	4	4			
L.U. Arm	4	4	4	4	4	4			
R.L. Arm	3	3	3	3	3	3			
L.L. Arm	3	3	3	3	3	3			
R. Hand	2½	2½	2½	2½	2½	2½			
L. Hand	2½	2½	2½	2½	2½	2½			
R. Thigh	5½	6½	8	8½	9	9½			
L. Thigh	5½	6½	8	8½	9	9½			
R. Leg	5	5	5½	6	6½	7			
L. Leg	5	5	5½	6	6½	7			
R. Foot	3½	3½	3½	3½	3½	3½			
L. Foot	3½	3½	3½	3½	3½	3½			
					TOTAL				

NAME_____

AGE_____

SEX_____

WEIGHT_____

DATE_____

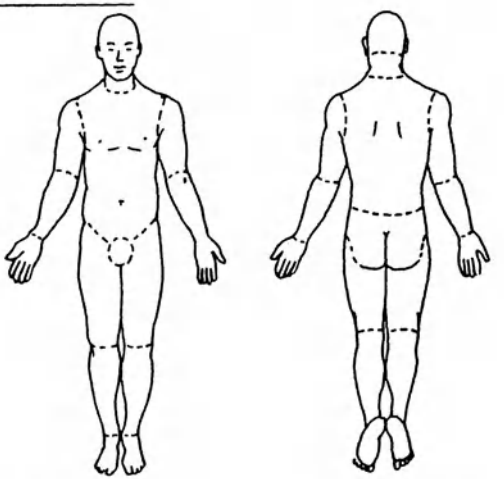

Fig. 1. Lund and Browder chart for estimating and recording the burn area (as percentage of total body surface) and the burn depth. *B*, Blue; *R*, Red

Management of Burn

Shock Phase (first 36–48 h)

An understanding of the burn shock mechanism is necessary (see also chapter on the pathophysiology of burns):

Low circulating volume → Organ hypoperfusion

1. Reduction of red blood cell mass during the shock phase is only about 15%.
2. Increased capillary permeability causes plasma constituents to move freely from the intravascular into the extracellular space.
3. Interstitial fluid accumulation (edema) in burns of up to 30% of total body surface area (TBSA) is limited to the burn area itself, but in more extensive injuries, increased capillary permeability will be found in all body tissues.
4. The increased capillary leak is maximal in the first 8 h after the burn and then gradually improves towards the end of the first 24 h.
5. Damage to the epidermal barrier will result in an increase of evaporative water loss. This process also continues after the shock phase.
6. Cardiac output is decreased during the shock phase due to an undefined myocardial depressant factor circulating in the plasma. This is a significant factor mainly in burns over 50% TBSA.

Fluid replacement

The goal is to prevent the onset of uncompensated shock. Controversy exists regarding the type and amount of fluids to be administered for optimal resuscitation.

Resuscitation Formulas. Such formulas are used as a guideline and not as rigid regimens. Continuous monitoring of the patient's condition leads to appropriate adjustments in the fluid program.

Burn patients can be resuscitated by any formula, but the various formulas influence differently the extent of post-burn edema and subsequent patient morbidity and late mortality.

There is a consensus that edema in unburned tissues as well as in the burn should be minimized. Therefore, a formula in which less water is given during resuscitation (but increases the load of sodium ions) is to be preferred.

The Haifa formula (blased on the Odstock formula) uses mainly hypertonic plasma which contains: Na^+ (165–170 mEq/l), K^+ (4–5 mg%), glucose (300 mg%), protein (6–7 g%), thus combining free water restriction with a high concentration of sodium ions. Every unit of plasma is examined for

hepatitis B and AIDS viruses. If this essential screening is not available, do not use plasma; rather, use an alternative formula.
The Haifa formula consists mainly of plasma (75 ml/kg) supplemented by Ringer's lactate (RL) (30 ml/kg). Its rate of administration IV is one-third in first 8 h, one-third in next 12 h, one-third in next 16 h. This is supplemented during the 1st day by RL 30 ml/kg.
In order to meet the differing demands of various clinical conditions, this formula is adjusted by multiplying the amount of plasma given by a factor:

of 0.75 for burns smaller than 35% TBSA
of 1.5 for electrical injury
for inhalation injuries
for very young or very old patients with major burns
for delayed arrival in hospital
for patients who do not respond immediately to resuscitation

Advantages of Hypertonic Plasma Resuscitation (Haifa formula). Less respiratory tract edema (less free water in the lung), less need for assisted respiration, less frequent indications for intubation or for tracheostomy, fewer lung infections, and improved respiratory function (Fig. 2). Reduced burn wound edema means improved oxygen delivery to the wound; hence the extent of escharotomy is reduced, there is less deepening of 2nd degree burns, reduction of burn wound infection, and wound healing is improved. Reduced gastrointestinal edema leads to less vomiting, gastric dilation, and paralytic ileus, thus permitting earlier enteral feeding, improved absorption from the gut, improved nutrition, and decreased incidence of gastrointestinal bleeding. Reduced cerebral edema means that the patient is more alert and has less nausea and vomiting.
Blood transfusions should not be used during the shock phase unless indicated for hemorrhage caused by associated trauma.
Cessation of IV transfusions should occur no later than 72 h post burn in order to avoid bacterial invasion via venepunctures and venesections.

Alternative formula [Parkland (Baxter) formula (modified)] when plasma is not available or contraindicated: In the first 24 h lactated Ringer's solution (3–4 ml x kg x % burn) is administered, one-half in first 8 h, one-quarter in second 8 h, one-quarter in next 8 h. In the second 24 h dextrose 5% in water (D5W) and colloids are given, one-third to one-half 1st day's requirements according to the patient's need. Enteral hyperalimentation commences on admission.

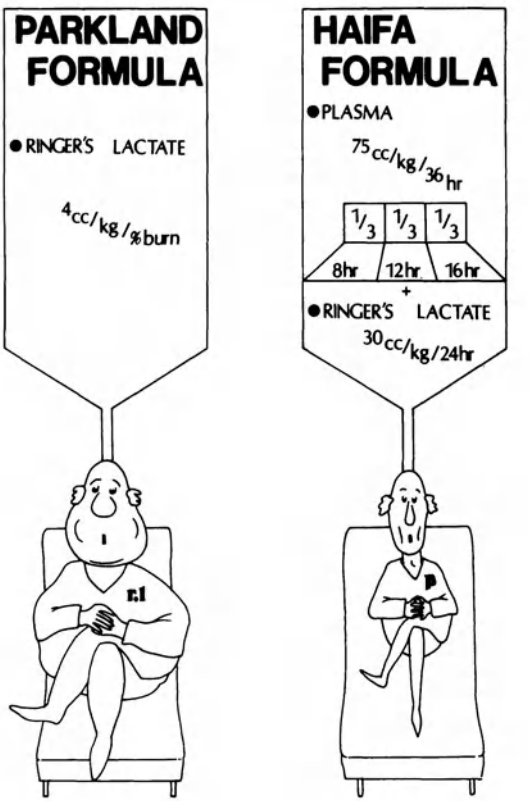

Fig. 2. Comparison between Parkland and Haifa formulas

Summary of Monitoring Routine on Admission

1. Vital signs — Hourly BP, pulse, and respiration; temperature (every 8 h)
2. Cardiac monitoring — Important in patients with electrical injury or preexisting cardiopulmonary disease, and in old patients with extensive burns.
3. Urine — Evacuate bladder on admission and measure urine output. Urine output should range between 20–30 ml per for children up to 10 years of age and between 30–50 ml per h for patients over 10 years old. Foley catheterization is not necessary in every patient. Urine output can be measured by spontaneous micturation every 3–5 h. A dark urine color indicates the presence of hemoglobin and myoglobin due to extensive red cell destruction and deep muscle burn. Fluid replacement should be increased together with the administration of mannitol in order to prevent acute tubular necrosis.

4. Weight — Measurement on admission, and daily thereafter, is a parameter needed to calculate resuscitation requirements during the shock phase and is a guide to fluid accumulation and nutritional status in the post-resuscitation phase.
5. Respiratory function:
   - Look for signs of inhalation injury, and/or carbon monoxide poisoning (headache, dizziness, nausea, cherry red color of skin and mucosa)
   - Check carboxyhemoglobin levels in blood
   - Take blood gases, chest X-ray
6. Gastrointestinal tract — Look for gastric dilatation and disappearance of bowel sounds. Insert nasogastric tube if necessary as indicated by nausea and/or vomiting.
7. Level of consciousness — If burn patients are not fully conscious, look for:
   - head injury
   - inhalation of toxic products
   - drug overdose
   - severe shock
8. Laboratory tests — Hematocrit, hemoglobin, white blood count, platelets, serum electrolytes, BUN, glucose, urinary sodium, and urinary specific gravity, to monitor hydration and as baseline data. Examine 2–3 times daily during the shock phase, once daily during the first week, and 2–3 times a week until discharge.
9. Invasive hemodynamic monitoring (only for special indications, to avoid infections) — Pulmonary artery wedge pressure measurement is indicated in shocked, unresponsive patients requiring intermittent positive pressure ventilation (since the CVP may be misleading).

Post-resuscitation Phase (48 h and thereafter)

Fluid mobilization is judged by: diuresis, weight reduction, levels of hematocrit, clearance of hemoglobinuria.
Pulmonary edema or adult respiratory distress syndrome may develop (chest X-ray and blood gases as well as a daily clinical assessment are important for early detection).

Laboratory Tests

- Renal function tests (BUN, creatinine, serum and urine sodium).
- Hyponatremia is encountered with the use of hypotonic solutions like silver nitrate 0.5% as topical agent, or in the case of inappropriate antidiuretic hormone (ADH) secretion.
- Hypernatremia occurs in osmotic diuresis (diabetes), inadequate replacement of insensible water loss.

- Hypokalemia may be due to renal losses following the use of silver nitrate 0.5% as topical agent, or mafenide acetate as topical agent, or in case of diarrhea.
- Acid-base balance: Metabolic acidosis is frequently met with during the shock phase but adequate resuscitation improves blood pH without any need to add sodium bicarbonate. It may develop later if mafenide acetate cream is used as a topical agent, due to its inhibitory effect on carbonic anhydrase in the kidney.

Management of Burn Wounds

Attention is paid to the burn wound only after hemodynamic stability has been achieved. Clean the burn wound with sterile saline and hexachloridine solution. Then dress as follows:
Limbs, chest, and abdomen — occlusive dressings are employed. The internal layer of the dressing in contact with the surface of the burn is soaked in 0.1% sodium hypochlorite solution and is irrigated every 8 h with the same solution.
Back — 1% silver sulfadiazine cream is applied 3 times daily directly to the burn site and covered by a thick, occlusive dressing. The victim lies on the dressings which have previously been spread on the bed.
Ears — wet gauze pads soaked in 5% mafenide acetate solution are applied twice daily.
Face — left exposed.
Dressings are changed daily in the burn unit using heavy sedation under strict asepsis. The burn wound is examined daily, and the topical treatment is altered depending on the state of the burn wound (which is matter of clinical judgement) and the microbial colonization.

Surgical Treatment

Escharotomy

Escharatomy is a longitudinal incision of the burn eschar in circumferential deep burns of the limbs when evidence of circulatory impairment is present, and of the chest wall when respiratory movements are restricted, allowing interstitial pressure release. (It is not a routine procedure, as it provides an additional portal of entry for infection.)

Technique. A full-thickness eschar incision is made without anesthesia in areas of deep burns from the shoulder, over the elbow on the lateral aspect of the upper limb, and into the palm of the hand between the thenar and hypothenar eminences. The dorsal aspect of the hand is also incised, as is

the full length of each finger on its medial and lateral aspects. The chest wall is incised along the anterior axillary line on both sides and if necessary, transverse cuts are added above the umbilicus and below the clavicles. In the legs, longitudinal cuts are made on the lateral aspect from the hips to the little toe.

Excisional Therapy

Early surgical excision of the burn wound is associated with a reduced mortality and morbidity and a shorter stay in hospital. Excisional therapy is a complicated and difficult surgical procedure and should be performed only in suitably equipped and staffed medical centers. The goal of any excisional procedure is to reduce the extent of the burn eschar and to cover the excised area as early as possible with a skin graft. The operative time should not exceed 1 h, and no more than 20% of total body surface area may be excised in one session.

Timing. Small deep burns are excised and grafted primarily on admission. Extensive burns are excised when the general condition is stable (optimally after 5–8 days). If the hands are involved, excision should be performed as early as possible. Deep burns of the face or back are not routinely excised: Only after the depth of the burn is definitely known may excision be undertaken.

Depth. Tangential excision, sequential removal of thin layers of necrotic burn eschar down to healthy viable tissue until punctate bleeding is encountered, requires skill and experience. This kind of excision is performed on the 3rd or 4th post-burn day in deep partial thickness injuries. It can prevent transformation of a partial thickness into a full thickness burn, and by immediate skin grafting gives good functional and cosmetic results (the method is especially indicated in the hands).
Full thickness excision, extending down to the underlying fascia, is a rapid, simple method causing only limited blood loss.

Skin Grafting

The excised areas should be grafted as early as possible. Autogenous skin cover is the ultimate goal: If it is not available in adequate quantities (major burns), it is expanded by meshing to a ratio of 1½:1 or, less preferably, 3:1.
Deep burns of the face are grafted according to cosmetic units (meshed grafts are not used). Donor sites are dressed. Donor sites can be reused after healing (8–10 days).
Allografts are rejected after 2–3 weeks: They are useful as a temporary skin cover and in combination with autografts.

Skin substitutes are indicated when neither autologous nor homologous skin is available.
Skin cultures are still under investigation. Early clinical trials show only 25%–50% take.
After skin grafting 0.05% sodium hypochlorite solution or 1% neomycin-streptomycin solution are employed together with nitrofurazone-impregnated gauze dressings.

Management of Infection

Sepsis still accounts for more than 50% of deaths of patients with burns. Every burn eschar is heavily colonized with bacteria from the 4th post-burn day.

Diagnosis

Clinical Findings. In general, temperature, pulse rate, and, respiratory rate are increased, ileus or diarrhea is present, and drowsiness. In the wound, a purulent discharge is obvious.
- Change in color of granulation tissue: from bright red to dull red, grey, or purple.
- Transformation of partial to full thickness burn.
- Loss of skin grafts.
- Deepening of donor sites.

Laboratory Investigations. From the burn wound, wet swab cultures are taken every other day, and biopsy cultures with quantitative counts are taken twice a week. Daily blood tests, including complete blood count and thrombocyte count, are taken. Blood cultures are taken when clinical signs of septicemia are present.

Septicemia

The features described for infection with the addition of a positive blood culture indicate sepsis. However, in view of the high incidence of negative blood cultures in burn patients, the diagnosis may be made purely on the grounds of the clinical indications. The clinical indications of sepsis are:
- Rigors
- Decreased level of consciousness
- Appearance of ileus
- Sudden change in pulse and respiratory rate

Laboratory data indicating sepsis are:
- Leukopenia after leukocytosis
- Sudden drop of hemoglobin
- Thrombocytopenia
- Constant rise of immature neutrophils (shift to the left)

Prevention of infection:
- Limit quantity of electrolyte fluids during the shock phase to reduce edema.
- Withhold prophylactic antibiotics.
- Restrict invasive monitoring lines.
- Discontinue IV lines no later than 48–72 hours post-injury.
- Excise the burn wound early.

Antibiotics. Systemic administration of antibiotics does not affect the flora in the burn eschar, since full thickness burns are avascular. Prophylactic antibiotics are not used routinely (see Fig. 3). Administer them to patients with burns who have in addition: inhalation injuries or open fractures, or perioperatively to patients undergoing surgical interventions which might induce bacteremia. Do not administer them for partial thickness burns, or deep burns without associated injuries.

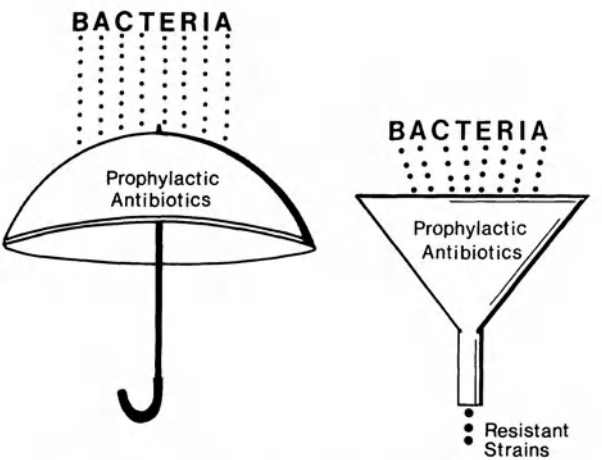

Fig. 3. The administration of prophylactic antibiotics does *not* have an umbrella effect, but rather acts like a funnel, permitting resistant strains to develop in the burn

Nutrition

An aggressive, enteral, nutritional approach based on a supplemental egg-rich diet is employed to support the severely burnt patient with injuries more extensive than 25% TBSA: this diet consists of five different flavors of milk shakes enriched with eggs, chocolate, vanilla, strawberry, banana, and orange. The content of each 250 ml bottle is 3 eggs, milk-based ice cream, milk powder, and glucose. This provides about 30 g of proteins, 30 g of fat, and about 50 g of carbohydrates. The total caloric intake for a patient of 70 kg of body weight ingesting 10 bottles is 6000 kcal per day.

Enteral feeding is initiated immediately after admission and gradually reaches the full formula within 7–8 days (Fig. 4). It is carried out orally or through a nasogastric drip or a combination of both, depending on the patient's condition.

This high load of proteins, cholesterol, and the various lipoproteins contained in eggs enhances plasma protein levels within 3 weeks of initiating the egg-rich diet. Plasma lipids remain within normal levels during and

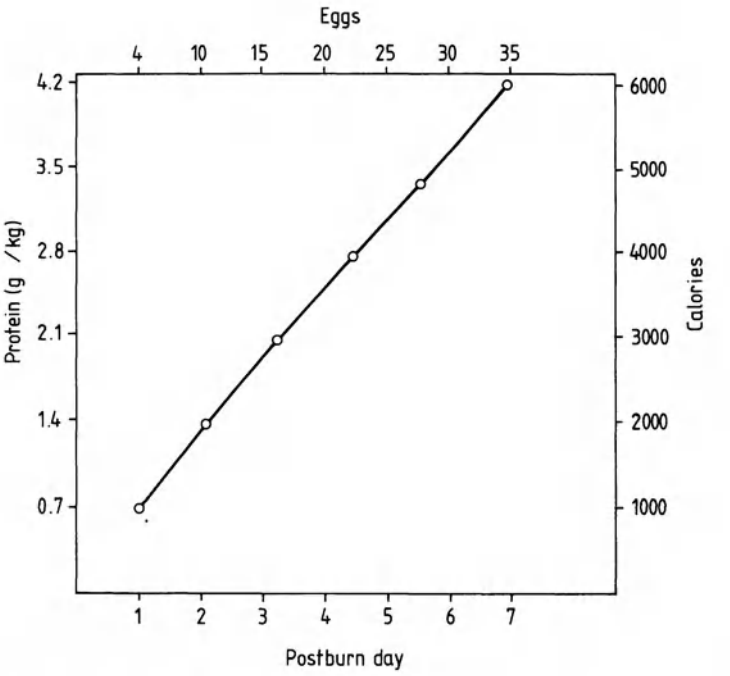

Fig. 4. Interrelationship between egg intake and the calculated total calorie and protein requirements

after the cessation of the diet, not withstanding the high cholesterol content of the egg yolks.

Physical Therapy

Goal. To assist the patient to regain his preburn level of function is the goal. The physical therapist participates in every stage of patient care in the ward and during the changing of dressings in the operating room. Patients with burns are encouraged to perform their exercises independently since the time spent with the therapist is never sufficient in itself.

Positioning. Placement of different body parts should be such as to oppose the contracting forces; for example, in burns of the axillae and shoulder area the arm should be placed and held in 90° abduction.

Exercise:

1. Active (in the cooperative patient) to maintain range of movement and muscle strength, and regain function.
2. Passive to maintain range of motion when active movement is absent.
3. Stretching to achieve an optimal position before splinting.

Occupational Therapy

The occupational therapist should provide a therapeutic regime to minimize burn scar deformities, increase independence in daily activities, and finally rehabilitate to work. This goal is achieved by the use of: occupational exercise, splints, both static to maintain position and dynamic to exert traction, and pressure garments (Jobst outfits) for the treatment of hypertrophic scarring.

3.9 Radiology in the Management of Wounds

O. B. Adler

The mainstay of the radiological workup of the acute trauma patient are plain radiographs, complemented when available by computed tomography examinations. Fluoroscopy and angiography are used under specific circumstances. Nuclear medicine and ultrasonography can be employed in blunt injuries to the abdomen and in the follow-up of these patients.
After the wounded patient's condition has been evaluated in the triage area and the cardiorespiratory and hemodynamic conditions stabilized, the next step in care is the assessment of the character and extent of the injuries. In the acute stage the radiological workup for this purpose encompasses a search for fractures, foreign bodies, organ injuries, blood vessel injuries, iatrogenic injuries, and complications.

Fractures. The diagnosis of fractures is made by radiographs in at least two projections at an angle of 90° to each other and oblique views when deemed necessary. Fractures involving the spine and the pelvis should be additionally evaluated by computed tomography. These complex archlike bony structures are best displayed in the transverse plane of computed tomography, which permits visualization of anteroposterior and lateromedial relationships often projected over each other in the conventional radiograph. In the spine, soft tissue involvement and impingement of bone fragments on the content of the neural canal (dural sac and nerve roots) can be evaluated simultaneously.

Foreign Bodies. The search for the localization of retained metallic foreign bodies was one of the first goals in the early days of radiological examinations. Fluoroscopy in various obliquities and less often radiographs have been used for this purpose to the present day. Various palpating sticks to protect the hands of the examining radiologist, complex techniques, devices, and mathematical formulas were devised in order to overcome a problem inherent in the radiological examination: the three dimensions of the body are displayed only in two dimensions. Whether a foreign body is located inside the body and what its relationship is to the vessels and organs are the questions addressed by the radiological examination. (In the extremities radiographs in two views are satisfactory for detection and localization of the foreign bodies.) The skull, orbit, spine, mediastinum, and abdomen (harboring the vital organs), none of which have perfect geomet-

rical shape, are the problematic areas. The exact location of the foreign body is impeded by the difficulty in performing all the necessary views, either by fluoroscopy or by radiography, in a severely injured patient who is often surrounded by many monitoring devices. In contrast, the use of computed tomography scanning permits easy and precise localization of foreign bodies in the least disturbing way for the wounded.

Organ Injuries. With the exception of the lungs, conventional radiographs furnish minimal information about injuries to the brain, orbits, mediastinum, and abdomen. The addition of peroral or i.v. contrast medium to study the gastrointestinal and urinary tracts widens the realm of conventional radiological examination but only in a limited way. The advent of angiography meant great progress. Angiography serves a double purpose: it demonstrates disruption of great vessels and thus the source of bleeding, and by outlining solid, parenchymatous organs, like the spleen, liver, or kidneys, it furnishes information about their integrity or degree of injury.

Radioactive isotopes. The assessment of organ integrity is facilitated by radioactive isotypes, and they can be used successfully to diagnose injury of the liver or spleen. Dynamic isotope angiography is a less invasive method than angiography to detect bleeding vessels.

Ultrasound equally may be used to evaluate abdominal injury, mainly in the liver, spleen, and kidney; unlike isotope methods it also assesses the presence of fluid, e.g., blood in the abdomen. However, the pressure of the transducer may not be tolerated by a patient with an acute abdomen or penetrating wounds; in addition, the examination of the spleen and liver high up beneath the ribs may be precluded in these patients. Ultrasound is very useful in the follow-up of abdominal trauma patients, especially for complications such as abscess formation and survey of hematoma resorption.

Computed tomography scanning is very convenient, relatively fast, and well-tolerated. In addition to the relative comfort and convenience for the casualty, examination of the brain and body in the multitrauma patient can be performed in the same session. The examination results in a wealth of information in every area of the body, often eliminating the necessity for other types of radiological examinations.

Blood Vessel Injury. Angiography is the examination of choice for detecting blood vessel injury. Bleeding vessels can be occluded by embolization in the radiology department. Tears, occlusion, or intimal flap formation are treated surgically. Mapping out the injury is done by angiography. The consequences of bleeding, e.g., hematoma formation, are usually revealed by computed tomography examination.

Iatrogenic Injury. Modern care of trauma patients entails monitoring the respiratory and cardiovascular systems. Catheters, tubes, wires and drains are introduced into the pleural space, large airways, large vessels of the

neck and thorax, or into the heart. Their proper placement is controlled by chest radiographs.

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3.10 Anesthesiology, Monitoring, and Intensive Care

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Introduction

Anesthesia and related problems during war and disaster are logistical rather than medical. The challenge is to achieve proper management from the site of wounding along the echolons to a rear hospital. The correct use of resuscitation techniques, the prevention of shock, the rapid stabilization of vital signs, and smooth evacuation are the ideals of the prehospital activity (see chapter on Primary Treatment of Wounds).

The critical point in the field hospital activity is correct triage and decision making, surgical operation being undertaken only in those cases in which a life or a limb are in danger. All other patients should be promptly treated, their condition stabilized and sent to a rear hospital (unless evacuation is impossible or very much delayed).

In the base hospital the main difficulties are organizational. The on-going overload on the anesthesia team may go on for days, weeks, and sometimes months after the disaster of battle is over, creating a chronic manpower shortage and a long delay in elective surgery.

The postoperative management of the critically ill requires good organization, facilities, and equipment.

The role played by the anesthesiology team in the field and base hospital is a critical one in dealing with emergencies, in triage, in making surgery as safe as possible, in the intensive care of the critically ill (preoperatively and postoperatively), and in the relief of acute and chronic pain.

Field Hospital

The field hospital is a temporary, mobile structure placed somewhere between the front line or disaster area and the base hospital. Its tasks are:

- Triage
- Preparation for further evacuation
- Performance of surgical procedures which cannot be postponed *by any means.*

The anesthesiology team will be directly involved in all three main activities.

Triage of Wounded

A general surgeon and an anesthesiologist make up an ideal triage team. Surgical specialists should be on stand-by call. The anesthesiologist is usually the most expert at both recognizing and treating dire emergencies.
The injured are sorted out into one of the following categories:

1. Ready for evacuation to a base hospital
2. In need of additional treatment to stabilize the vital signs prior to evacuation
3. Requiring urgent surgical intervention to save life or a limb
4. Ready for discharge back to front or home

The triage team must identify the most severely wounded who need further treatment on the spot and perform lifesaving interventions: cardiopulmonary resuscitation (CPR), artificial ventilation, treatment of airway obstruction, treatment of shock.
Triage is facilitated by X-ray and laboratory investigations. Urgent X-ray upon arrival is indicated on suspicion of tension pneumothorax, cervical spine injury, or one lung intubation. The vital functions must be monitored at all times on the way to, in, and on the way back from the department of radiology.
Evaluation of *acute anemia* (Hb, Hct) is the most useful and available laboratory investigation at the field hospital echelon. The initial hematocrit is often not low if the wounded are seen early after the injury as the diminished red cell mass has then not had time to become diluted by interstitial fluid which has moved into the vascular tree or by intravenously infused fluids.

Stabilization of vital functions for evacuation

An orderly and meticulous examination is carried out in order to assess the casualty's condition and establish priorities of treatment.

Head Injury

Check:
- State of consciousness
- Orientation

- Pupil's reaction to light
- Cranial nerves
- Corneal reflex
- Spontaneous respiration
- Temperature

Treatment:
- Place the patient in the side lying position
- Cover the body with warm blankets in case of hypothermia
- Decide *between:* suction/oral airway/tracheal intubation + artificial ventilation.
- Insert an nasogastric tube. Be sure it is in the stomach and that the stomach is not full.

Spinal Cord Injury

Check:
- Movement of limbs (each one separately)
- Penetrating wounds in the vicinity of the spine
- Spontaneous respiration (a lesion above C_4 will paralyze the diaphragm as well as the intercostal muscles).

The following wounded should be considered to have a spinal cord injury until proved otherwise:
- A patient with any kind of head injury
- An alert patient who cannot move his extremities upon command
- An unconscious patient who fails to move an extremity when sternal pressure is applied
- A person who has suffered penetrating trauma to the neck or torso and is unable to move an extremity upon command or sternal compression
- A patient with maxillofacial trauma

Treatment for cervical spine injury:
- Apply a hard collar or "sandbags"
- *Don't* flex the head if you have to intubate the patient. (One simple head–neck movement can transform the wounded into a quadriplegic).

Treatment for thoracic and lumbar spine injury:
- Do not move the patient without adequate help
- Do not allow him to elevate himself

Upper Airway Injury

For treatment of injuries to the upper airway, see Table 1.

Table 1. Treatment protocol for injuries to the upper airway

Lesion	Check	Treatment
Maxillofacial	Intraoral bleeding	Suction
	Absence of teeth	Extract the foreign bodies from the oral cavity
		Direct laryngoscopy, in order to decide between: simple oral airway, orotracheal intubation or tracheotomy
		Pack the oropharynx (after intubation) in case of continuing bleeding
Laryngeal	Voice change	Direct laryngoscopy
	Sign of burn	Tracheotomy
	Hematoma formation	
	False passages	

Thoracic Injury

Check for the presence of hemothorax or pneumothorax (open or tension) and record the spontaneous respiration (rate and depth). Check for:

- Unilateral chest motion
- Cardiovascular status
- Subcutaneous emphysema
- Hemoptysis
- Mediastinal emphysema (X-ray sign; speaks for a tracheal or large bronchial injury).

Ensure respiratory function by:

- Creating an artificial airway: oral intubation; tracheotomy
- Providing efficient pleural drainage in hemo- and/or pneumothorax. Optimally none of these procedures should be done during transportation (especially not during air evacuation!). Thus, they should have been done correctly before allowing the wounded to leave the field hospital premises.
- Promoting alveolar ventilation and normal gas exchange:
  a) Administer oxygen by mask, tube, or tracheotomy in case of spontaneous breathing.

b) Ventilate the patient, if spontaneous respiration is inefficient or absent, by using an Ambu bag, or a portable ventilator (Pneumopack, Takaoka).
c) Before evacuating the patient, be sure that bilateral chest expansion is efficient. Perform a chest X-ray in order to verify the efficiency of pleural drainage and the position of the artificial airway.

Hypovolemic Shock

Check:
- Source of bleeding
- Blood and pressure heart rate
- Skin color
- Respiratory status
- Alertness
- Urinary output
- Central venous pressure (CVP)

Treatment:
- Stop the external bleeding (if chest or abdominal bleeding is not controlled by conservative treatment, rapid surgical intervention may be indicated).
- Make sure there are at least two intravenous cannulations open for administrating fluid.
- Look for signs of cardiovascular stability: blood pressure close to normal, quiet spontaneous breathing, urinary output >30 ml/h.

Administration of Blood

Up until this point, the patient's volume deficit will have been corrected by crystalloid solutions and/or synthetic plasma expanders. The field hospital should host a blood bank.

Goals. Blood is given to restore the intravascular volume, maintain an adequate O_2 carrying capacity of the blood, and preserve normal clotting activity.

Indications. Massive hemorrhage or continuous bleeding which cannot be compensated for by administration of other fluids are the indications for administering blood. Massive hemorrhage demands effective surgical hemostasis. Do not evacuate a patient who needs more than 1 unit of blood every 2 h in order to maintain a normal blood pressure.

Procedure

1. Determine patient's blood group.

2. Order packed cells and plasma if the bleeding is not massive; order packed cells, plasma *and* whole blood if more than 5 units of blood are to be administered (1 unit of plasma for every 4 units packed cells).
3. Check the CVP frequently according to patient's condition – if necessary, every few minutes.
4. Use a peripheral vein for blood transfusion, in order to have a CVP line for ongoing evaluation.
5. Observe the patient during administration of the first 50 ml of *each* new unit in order to detect signs of incompatibility: flushing, chills, tachypnea, tachycardia, flank and back pain, sudden oozing from wounds, hemoglobinuria, and oliguria. If these signs appear:
   - Terminate the blood transfusion *immediately*
   - Repeat cross-matching and obtain a Coombs test
   - Detect whether there is free hemoglobin in the patient's blood
   - Force diuresis: administer fluids and diuretics
   - Administer heparin (1000–2000 units i.v. in case of disseminated intravascular coagulopathy)
   - Administer fresh frozen plasma, cryoprecipitate and/or platelets as dictated by coagulation parameters.

Full Stomach

A full stomach is potentially very dangerous, especially in the comatose. Silent regurgitation or vomiting of gastric contents may cause obstruction of airways by solid food or inundation of airways by fluid contents; or Mendelson's syndrome due to chemical lung injury by acid gastric contents.

Management. Whenever the danger of regurgitation is evident (coma, maxillofacial injury with no control of swallowing, etc.) use the crash induction technique:
Administer metoclopramide 20 mg intravenously (to speed emptying of the stomach).
Administer atropine 1 mg intravenously (this doubles the pressure the cordia is able to withstand from the gastric aspect).
Prepare the laryngoscope, a suitable endotracheal tube and a good suction device.
Tilt the patient foot-down 40°.
Administer 100% O_2 for 5 min (by mask).
Have assistant ready to apply pressure on the cricoid cartilage (Sellick maneuver) until the tracheal tube is inserted.
Administer 3 mg curarine intravenously (this will prevent fasciculations produced by succinylcholine and thus will avoid increase of intragastric pressure).
If the patient is comatose, omit thiopental; otherwise, administer 1 ml 2.5% thiopental solution every 2 s and observe the patient. Once he is asleep, rapidly administer succinylcholine 1.5 mg/kg intravenously.

Do not ventilate the patient; rather leave him to breathe spontaneously up to the moment the relaxant *apneic* effect of succinylcholine is evident. (During the previous 5 min, 100% oxygenation will fill the lungs with enough O_2 to keep PaO_2 at 80 mmHg during the apnea; the $PaCO_2$ will increase by only 3 mmHg/min).
Insert the laryngoscope into the oral cavity, reveal the glottis, and introduce the tracheal tube.
Inflate the cuff of the tube immediately and auscultate the tracheal area to ensue that the airways are sealed off.
Relieve the pressure on the cricoid (too heavy a pressure can produce esophageal rupture).
Introduce a large nasogastric tube and evacuate the gastric content.
An alternative method for barbiturate-relaxant administration is:
a) Topical analgesia for upper airways: lidocaine spray for mucosa; injection of 2 ml lidocaine 2% through the cricothyroid membrane, directing the needle upward; or
b) Bilateral superior laryngeal nerve block.

Final Check Before Evacuation

Make sure that:
- Cardiovascular status is stable
- Alveolar ventilation is efficient
- Pleural drainage is working
- Intravenous lines are patent
- Stomach is empty
- Fractures are immobilized
- Tracheotomy wound does not bleed
- Patient is comfortable, pain free

Put everything you have done in writing, record the trauma score, and transmit all the necessary information to the team in charge of evacuation. Accompany the wounded to the heliport or parking lot and keep an eye on vital signs during this last stage of management.

Summary of Anesthesia Team's Tasks in the Triage and Preoperative Areas

1. Assessment and correction of vital functions (ventilation and circulation):
   - Check the correct position of tubes, drains, and intravenous lines.
   - Adjust the ventilator parameters.
   - Connect the patient to ECG monitor.
   - Stabilize the blood pressure (fluids, drugs).
   - Check hemoglobin, hematocrit, blood gas partial pressures, and electrolyte balance.

2. Prevention and treatment of cerebral edema. Secondary brain trauma is the "second trauma" (usually produced by hypoxia, hypercapnia, hypotension) and is as dangerous as the primary injury itself.
   - Hyperventilate the patient (keep the $PaCO_2$ between 30–35 mmHg)
   - Look for unilateral signs
   - Administer: barbiturates, diazepam, mannitol if indicated
   - Use Glasgow coma scale for assessment.
3. To provide analgesia, especially in case of burns, fractures, and penetrating abdominal wounds, cautiously give small, repeated 2- to 5-mg doses of opiates intravenously, whilst assessing their influence on the respiratory function.
4. Reassurance (especially for those with mutilating injury). Inform the conscious casualty about his condition, the planned surgery, and the technique of anesthesia to be used. Premedication is not mandatory: good analgesia and small doses of benzodiazepines on the operating table often cover the basic need before surgery.

Surgery

In a field hospital in war or disaster, nothing can be taken for granted: electricity, the necessary drugs, the equipment, tanks of fresh gases may not be available. Therefore only those surgical procedures which cannot be postponed under any circumstances are performed. These include surgery to save limbs (e.g. after severe arterial injury) or lives (e.g. where there is massive bleeding). Various other urgent operations may be carried out when evacuation to a base hospital is delayed.

Preparations

Use the short time before surgery to prepare the patient for anesthesia.

1. Check hemoglobin and hematocrit and order a chest X-ray.
2. If the patient has a *full stomach*, evacuate the gastric content (see above if patient comatose) by inserting a large nasogastric tube, not forgetting to remove the tube afterwards.
3. If the patient has *hypovolemia:*
   - Ensure at least two free flowing intravenous cannulations.
   - Insert a CVP catheter.
   - Insert a urinary catheter.
   - Order blood and blood products.
4. In a *comatose* patient, an epidural or subdural hematoma must be excluded by clinical examination. Anesthetizing a patient with an unidentified hematoma can prove fatal.

5. Check the airways: check air entry and efficiency of the artificial airway. Look for signs of lung trauma: subcutaneous emphysema, mediastinal emphysema, hemoptysis. Interpret the X-ray.
6. Many wounded are frightened and worried about their condition. Reassure them, explaining the anesthetic and surgical procedures and emphasizing postoperative analgesia.

There is no place for regional anesthesia in a field hospital; there is an absolute indication for keeping the patient well ventilated and correctly monitored. Also, since it is not at the beginning evident how extensive surgery is going to be, anesthesia must be versatile, giving the surgeon the conditions for wide exploration.

Premedication is not essential (never use the intramuscular route). Administer small doses of sedatives intravenously as required (e.g., diazepam 2.5–5 mg), but avoid diminishing the patient's control of swallowing up to the moment of endotracheal intubation.

Equipment

1. *Anesthesia machine:* check the circuit, sources of fresh gases (especially O_2), laryngoscope, tubes, and soda lime before bringing the patient to the operating room.
2. *Resuscitation equipment:* defibrillator, blood transfusion pumps, blood heater.
3. *A ventilator:* frees your hands during anesthesia.
4. *Monitoring:* a blood pressure manometer, ECG monitor, tidal volume spirometer and a core temperature probe (esophageal thermometer) are essential for basic monitoring.
5. *Suction equipment.*

Manpower

The period of induction just before surgery is critical: it includes induction of anesthesia and the administration of drugs which affect the compensatory mechanisms. Optimally at this stage three assistants are needed: one for the Sellik manoeuver, one for administering fluids and blood rapidly, and one for simultaneous monitoring of the vital signs (blood pressure, pulse, ECG).

Induction of Anesthesia

In an *unconscious patient*, avoid hypnotic drugs. If the patient is not already intubated, administer curarine *before* succinylcholine, in order to prevent a sudden rise in intragastric pressure. (The intubation can sometimes be per-

formed without using any relaxant). Suspicion of a full stomach is an indication to use the crash induction technique (see p. 253).

In a *conscious and hemodynamically stable* patient:
- Give a small dose of diazepam (2.5–5 mg) intravenously; repeat it if necessary.
- Oxygenate the patient by mask for 3–5 min.
- Administer a small dose of curarine (3 mg) (see above).
- Give thiopental 2.5%, only just as much as is needed to induce unconsciousness (usually the patient will need not more than 2–3 mg/kg).
- Administer succinylcholine 1.5 mg/kg.
- If there is no suspicion of a full stomach (i.e., the casualty is fasting), gently ventilate the patient for 1–2 min. If patient is not fasting omit this step: air will enter the stomach and regurgitation and aspiration is very possible.
- When the patient is asleep and relaxed, insert the tube transorally into the trachea, inflate the cuff, and start ventilating the lungs first by administering 100% oxygen for 2–3 breaths, then a N_2O/O_2 mixture (60%:40%).

In a hypovolemic unstable patient:
- Try to restore blood volume before induction.
- Give a small dose of diazepam (2.5–5 mg) intravenously.
- Then administer *ketamine* 1.5–2 mg/kg intravenously in 15–30 s. Ketamine is the only anesthetic drug with an adrenergic effect on the blood pressure and the heart rate. Rule in using ketamine:
  a) Do not rely upon so called "protective laryngeal reflexes". Ketamine cannot prevent aspiration. To avoid sudden regurgitation ensure that the stomach is empty.
  b) Do not use ketamine if there is suspicion of increased intracranial pressure.
  c) Use diazepam to reduce the incidence of hallucinations produced by ketamine.

Maintenance of Anesthesia

There is no special indication for the use of one drug or another. In a field hospital the technical and logistic facilities are limited, therefore intravenous drugs can be used instead of N_2O for obtaining analgesia and pharmacological hypnosis; the inhalatory anesthetics such as halothane or isoflurane will only rarely be available.

The goals of maintenance of anesthesia are:
1. To provide analgesia by administering opiates (morphine 5–10 mg, pethidine 30–50 mg, fentanyl 0.1 mg) intermittently, intravenously.

2. To maintain pharmacological sleep by intravenously administering repeated doses of diazepam (2 mg) and/or ketamine (0.5 mg/kg) and/or thiopental (150 mg).
3. To give muscular relaxation by using curarine (it may produce hypotension), pancuronium (secondary effects: tachycardia and hypertension), or atracurium.
4. To ensure efficient alveolar gas exchange by ventilating the patient manually or mechanically. For safety, keep the minute volume over 100 ml/kg/min in order to maintain the $PaCO_2$ between 30 and 35 mmHg (capnography is usually not available).

As many parameters as possible are monitored meanwhile, using every available device together with a precordial or esophageal stethoscope.

Ending Anesthesia

The need for further evacuation must always be borne in mind. At the end of surgery the casualty may *need to be ventilated.* If so: don't reverse muscle relaxant; don't use narcotic antagonist; don't extubate!
The casualty may *need a further period of stabilization:* recheck hemoglobin, hematocrit, urinary output. Decide if he needs more fluids. Hypovolemia is the most frequently encountered cause of intraoperative and postoperative *oliguria.* Therefore, give more fluids before you decide to administer diuretics. Above all, check the patency of the urinary catheter.
Check the place of the endotracheal tube, the need for pleural drainage, input, and the *temperature.* In case of hypothermia, use blankets. Administer fluids through heating devices, if available. *Hypothermia* is one of the common causes for a delayed recovery from anesthesia.
Only when the casualty's general condition is stable enough to permit extubation should the muscle relaxant action be reversed, and naloxone or, better, nalorphine be used as a narcotic antagonist. Nalorphine is preferred because the secondary effects of naloxone (due to symphathetic discharge: tachycardia, hypertension) could jeopardize cardiovascular stability.

Recovery Area

The recovery area functions as an improvized intensive care unit. Here the patient's general condition is further monitored and stabilized prior to evacuation (delay in evacuation of wounded is to be expected).
The recovery area needs ECG monitors, mechanical ventilators, a defibrillator, and suction equipment.

Records

Recording of data, measurements of vital functions, and quantities of fluids or drugs administered during anesthesia is mandatory. The anesthesia

chart must be kept up to date with explanations for any special decisions taken. Before evacuation, summarize the casualty's condition, including the treatment received to date. Record the trauma score. This information is essential for the flight surgeon and the next echelon.

Base Hospital

In a base hospital, surgical procedures aim at solving the various problems posed by multiple trauma. *Surgery is frequently prolonged and complicated:* the choice has to be made between concomitant and sequential surgery (e.g., a patient has eye trauma and limb fractures: can both teams work together, or must they operate one after the other?).
The prolonged exposure of the patient on the operating table demands continuous monitoring of his temperature. For hypothermia, use an electrical blanket and heated fluids, heated inspired gases. The patient's vital functions may suddenly deteriorate. In this case, stop the surgeon if this will help efforts to restabilize the patient's status. Postpone all nonvital procedures, add monitoring parameters if necessary (e.g., CVP, arterial blood gases, pulmonary artery pressure; see below).
Complicated and prolonged surgery may need a *complete anesthesia team*, which will include one senior anesthesist, one or two residents or general practitioners trained in anesthesia, and one nurse.
As in the field hospital the role of regional anesthesia is limited by the factors of major complicated surgery, long procedures, and the patient's psychological stress.

Monitoring

The possibilities in a base hospital are more versatile than in a field hospital.
Use an arterial catheter when variations in blood pressure or frequent checks of arterial blood gases are anticipated.
Cannulate the *radial artery*. Use the percutaneous technique, inserting a cannula not larger than 21 gauge. The advantages of cannulating the radial artery for intraoperative monitoring are the simple technique and the easy approach for the anesthesiologist.
For patients who need continuous invasive monitoring of blood pressure, change the cannulation at the end of the surgical intervention to the femoral or axillary artery, as these vessels are less prone to thrombosis and give more accurate data.
After craniotomy in a patient with head injuries who is to undergo more surgery, insertion of an intraventricular (or subdural) catheter for monitor-

ing the intracranial pressure is an advantage. In such cases, the neurosurgical patient, instead of being transferred to an intensive care unit, remains on the operating table for another surgical procedure. Monitoring of intracranial pressure is compulsory in addition to clinical monitoring: pupil size, blood pressure, heart rate. The neurosurgeon must be informed about any change in the patient's neurogical status during other surgical procedures.

Various conditions change normal pharmacokinetics and the metabolism of muscle relaxants: hypothermia, alkalosis/acidosis, use of aminoglycoside antibiotics, electrolyte imbalance, and diuretics.

A train-of-four neurostimulator test will help in deciding on whether to administer more relaxants as well as in decisions on reversal of anesthesia and extubation.

In massive trauma, with myocardial contusion present or suspected, use PEEP >10 cm H_2O in order to maintain a PaO_2 >70 mmHg at a FiO_2 >0.40. The need for more data on cardiovascular function is the indication for inserting a pulmonary artery catheter. The pulmonary artery catheter adds precious information on the cardiac index, capillary wedge pressure, and systemic and pulmonary vascular resistance.

Use of PEEP

Sometimes the use of a device (connected to the anesthesia circuit) which provides positive pressure during expiration is necessary. This is the case where there is a fluid overload (the casualty having received too much fluid on his way from the site of wounding to the rear hospital), or severe pulmonary contusions with subsequent atelectasis and increased intrapulmonary shunt. Another indication is early adult respiratory distress syndrome, caused by:

- Administration of a large amount of blood containing amorphous substances and debris
- Early sepsis
- Fat emboli (fractures)
- Head trauma (leading to neurogenic pulmonary edema)
- Prolonged hypovolemic shock
- Carbon monoxide poisoning
- Gastric juice aspiration
- Smoke inhalation

PEEP will increase the functional residual capacity and improve oxygenation. Low PEEP (<10 cm H_2O), whilst having a beneficial effect, produces only a minimal cardiac disturbance which could be treated by further administration of fluids and inotropic drugs (like dopamine).

Inhalatory Anesthetics

Many injured will need further surgery and anesthesia. The best inhalatory anesthetic is isoflurane; repeated exposure to halothane may cause rare cases of postoperative hepatoxicity. Since the kidneys are at risk in hypovolemic shock (with a possibility of renal shut-down), in dehydration due to field conditions and prolonged fasting, and in rabdomyohemoglobinuria due to crush injury, the use of methoxyflurane has been abandoned because of its nephrotoxicity.
Halothane should be avoided when increased intracranial pressure is evident.
Never add halothane to the inspired gases unless you are sure the patient is moderately hyperventilated.

Dilutional Coagulopathy

This condition may appear after the administration of 6 or more units of old blood. Contributory factors are that 50% of platelets in blood disappear after only 24 h of storage, and levels of factors V, VII, and VIII in stored blood diminish proportionally to the period of storage. Prolonged shock and/or sepsis could also be a cause.
To prevent dilutional coagulopathy arising, use fresh (<24 h storage) blood, if possible. Administer 2 units of fresh frozen plasma for every 5 units of packed cells or whole blood, and inject 6–12 units of platelets if the platelet count is lower than 50000. If hypotension and hypocalcemic EKG changes are present after multiple transfusions, administer 0.5–1 g calcium chloride intravenously, slowly.

Laboratory Tests

Send blood and urine to the laboratory for examination as follows:
- Hemoglobin and hematocrit in cases with massive bleeding.
- Blood gases in cases with pulmonary contusion, prolonged surgery, suspicion of hypoxia, massive blood replacement, oligoanuria, any kind of suspected respiratory insufficiency.
- Urinary Na^+ and osmolality, in case there is a difficulty in defining the origin of oliguria (a very low level of urinary Na^+ together with a high urinary osmolality would be specific for dehydration).
- Coagulation studies: prothrombin time, partial thromboplastin time, fibrinogen and fibrinogen split products in cases with massive transfusion or suspicion of dilutional coagulopathy.
- Free myoglobin in plasma: in cases of crush injury.

Postanesthetic Phase: Recovery Room

Logistics

A large postoperative area is needed, since many casualties will need continuous monitoring and the intensive care facilities will be over-crowded. Instead of the usual 1.5–2 beds per operating table, 2.5–3 beds per operating table are required in disaster or war. Manpower must be adjusted accordingly.
Ventilatory and monitoring equipment will be required as follows:

Pressure ventilator, 1/4 beds
Volume ventilator, 1/3 beds
Simple ECG display monitor, 1/1.5 beds
ECG + pressure display, 1/3 beds
Blood warming device, 1/3 beds
Automatic infusion pumps, 1/1 bed
Warming/cooling blanket, 1/4 beds

Recording of Vital Data

Record every event and/or treatment. A very simple marking system of vital functions is a multiple choice list with three options for each system:

CNS:	a) conscious
	b) sleepy but arousable
	c) unarousable
Blood pressure:	a) normal
	b) hypotension
	c) hypertension
Pulse:	a) normal, strong, regular
	b) feeble or slow
	c) rapid
Respiration:	a) spontaneous, deep
	b) shallow or bradypnea
	c) artificial ventilation
Skin color:	a) pink, dry
	b) pale, moist
	c) blue

All the 'b' and 'c' conditions require close observation and/or treatment. When all parameters are graded 'a' the casualty is discharged from intensive care.

Complications

The complications which appear in the recovery room are often a natural continuation of the difficulties encountered in the operating room.

Bleeding may be from incomplete surgical hemostasis or due to disseminated intravascular coagulation. Management is by surgical reintervention and/or infusion of fresh frozen plasma, platelets, and volume replacement, preferably blood transfusion.

Respiratory insufficiency may be due to incomplete muscle relaxant reversal, prolonged influence of narcotics, inefficient pleural drainage, post-traumatic pulmonary insufficiency, aspiration, or an obstructed airway. The management is by reversing the drug effects, intubation with or without artificial ventilation, use of PEEP, continuous use of respiratory monitoring — oxymeter, capnograph, blood gases, peak inspiratory pressure, chest X-ray — and endotracheal suction.
It is better to keep the patient intubated and ventilated when you have no explanation for his poor respiratory performance, a repeat surgical intervention is anticipated, the patient has had prolonged surgery and anesthesia, and/or the patient is to be transferred to an intensive care unit.

Hypertension may be due to pain and anxiety, hypoxia, fluid overload, increased intracranial pressure, hypothermia (due to vasoconstriction), or, commonly, a full bladder. Depending on the cause, management is as follows:
Administer morphine 2–4 mg intravenously.
Repeat if necessary.
Give larger doses if the patient is ventilated.
Correct the respiratory impairment.
Treat increased intracranial pressure by barbiturates, hyperventilation and diuretics.
Warm the patient.
Void the bladder.

Delirium may be caused by pain, hypoxia/hypercapnia, prolonged influence of muscle relaxants, hyponatremia, or use of benzodiazepines. Give sedatives, reverse muscle relaxants, administer NaCl and physostigmine 1–2 mg intravenously.

Shivering is the most frequent and troublesome complication. Warm the patient. Give small doses of pethidine or ritaline (phenylphenidate) intravenously. (Shivering increases O_2 consumption by 500% and therefore needs immediate therapy).

Ongoing contact with the surgical team in the operating theatre is necessary for scheduling urgent repeat interventions.

Transfer to an Intensive Care Facility

First ensure that the intensive care team is ready to take over the case. The transportation must be carried out under careful supervision. the following equipment should be at the ready during transport:
- A manual/mechanical device for artificial ventilation (which would include a PEEP option)
- A portable ECG monitor
- A battery-powered defibrillator
- Infusion pumps

Postoperative Intensive Care

The treatment of disturbance of vital organs starts on site and at the medical aid station. It continues in the field hospital as part of the preparation for evacuation or as postoperative management after emergency surgery.
A disaster situation will require a 10% bed allocation for intensive care in addition to special allocations for burns and neurosurgical intensive care. The manpower shortage is solved by switching from 8-h to 12-h shifts for nurses, medical staff and paramedical professionals (physiotherapists, respiratory technicians, laboratory technicans).

Respiratory Problems

Prolonged postoperative artificial ventilation may be indicated in multiple trauma, prolonged surgery (thoracoabdominal procedures), prolonged shock, fluid overloading, flail chest, pulmonary contusion, high spinal cord injury, and adult respiratory distress syndrome (ARDS). ARDS may be associated with fat emboli, head trauma, disseminated intravascular coagulopathy, carbon monoxide poisoning, aspiration of materials, sepsis, or any of the previously mentioned indications for prolonged artificial ventilation.
Treatment is by intubation (see Table 2) or tracheotomy and artificial ventilation. *Tracheotomy* is indicated when it becomes apparent that prolonged intubation would be required or when it is clear from the beginning that artificial ventilation will be needed for more than 10–14 days, or when severe facial or neck injury makes intubation impracticable.
Respiratory performance is improved by efficient endotracheal suction, lung posture and physiotherapy, change of position (sitting position increases the functional residual capacity), and aerosol therapy, such as using mucolytics wisth humidification and bronchodilators.

Table 2. Advantages and drawbacks of oral and nasal intubation

	Nasal	Oral
Advantages	Better fixation of tube Better tolerated by patient	Less traumatic
Drawbacks	Traumatic maneuver May produce otitis, sinusitis	Less effective fixation of tube Difficult oral hygiene Less tolerated by patient

Indications for weaning from respiratory support are:
Absence of sepsis
Normal cardiac index
Absence of acute pulmonary disease
Full consciousness
Patient able to keep airways free of secretions
A useful step-by-step method is: controlled ventilation → intermittent mandatory ventilation (IMV) → reduce FiO_2 to <0.4 → reduce PEEP to <5 cm H_2O → reduce IMV rate → reach CPAP point (spontaneous breathing + continuous positive air pressure) → extubation. For every step, use adequate on-line monitoring of saturation SO_2, PCO_2, cardiac index, blood pressure, heart rate, spontaneous respiratory rate.

Pneumothorax

Etiology
Pneumothorax in the postoperative period may be due to anaerobic or staphylococcal pneumonia, or it may be traumatic: there may be a blunt injury, a penetrating injury, barotrauma (due to positive pressure used for artificial ventilation) or an iatrogenic injury (e.g., on catheter insertion for CVP measurements).
Goals of treatment are to re-expand the lungs and prevent further collapse.

Treatment
1. Observation only, without treatment, if:
   - The pneumothorax is less than 50%
   - There is no active leak (chest X-ray stable over 24–48 h)
   - There is no evidence of significant underlying lung disease
   - The patient is ventilated with small PEEP and normal tidal volume
   - Continuous monitoring is available and in use

2. Needle aspiration: only as an urgent temporizing method in a case of tension pneumothorax.
3. Chest tube insertion (see also chapter on Chest Injury):
   - Insert the tube into the 5th intercostal space, midaxillary line
   - Suture tube to skin
   - Connect it to underwater seal or to suction
   - Asses tube patency 8–12 h after insertion

A chest tube may be complicated by the development of a pleural space infection (empyema). Hence the chest tube should not be left in place longer than necessary. Use large tubes in cases of hemothorax.

4. Open thoracotomy: in case of persistent leak and/or bleeding.

Aspiration

The aspired material could be food, blood, or gastric juice. Aspiration may occur after injury in a comatose patient or during intubation.

Prophylaxis
During artificial ventilation:
- Keep the tracheal tube cuff inflated.
- Before deflating, suction the oral secretion.
- When the cuff is deflated, be sure the stomach is empty and place the patient in a sitting position.
- Use a continuous drip for enteral feeding (rather than by a large bolus).
- Keep the gastric pH above 3.5 when the patient is not receiving enteral feeding [this prevents chemical pneumonitis (Mendelson syndrome) in case of aspiration of gastric juice].

Treatment
- If the patient is not already intubated, isert an endotracheal tube.
- Ventilate the patient with or without PEEP.
- Remove fluid aspirate by vigorous tracheal suction and solid materials under the guidance of bronchoscopy.
- Administer bronchodilators if there is a sign of bronchospasm (aminophylline intravenously and/or salbutamol spray).
- Look for signs of infection in chest X-ray and sputum cultures.
- Do not give steroids or prophylactic antibiotic therapy.

Cardiac Problems

A danger of cardiac disturbances always exists, especially in the middle aged and elderly.

Cardiac Failure

Etiology

Decreased preload:	hypovolemia, high PEEP.
Impairment of contractility:	hypoxia caused by pulmonary contusion, aspiration, pneumothorax, hemorrhagic shock, smoke inhalation, cardiac contusion, tamponade, sepsis, or propane inhalation.
Increased afterload:	postresuscitation hypertension.
Tachycardia/bradycardia:	quadriplegia or high intracranial pressure.

Diagnosis

Clinical signs of hypoperfusion include oliguria, pallor, cold extremities, decreased sensation and sweating. Other indicative measurements are CVP, pulmonary wedge pressure, cardiac output and systemic vascular resistance, which require pulmonary artery catheterization. Monitors should include a pulmonary artery catheter for CVP and an arterial line for arterial blood gases.

Treatment

1. Artificial ventilation (to reduce the work of breathing and to avoid increasing that part of cardiac output which goes to respiratory muscles).
2. Inotropic support therapy: dopamine or dobutamine (see Table 3).

The best approach is to use dopamine in rather small doses (< 5 μg/kg/min). Thus you will obtain a good combined inotropic effect with renal and mesenteric vasodilation but almost no tachycardia.

Cardiac Arrhythmias

1. Sinus bradycardia may be caused by increased intracranial pressure or high spinal cord injury. *Treatment:* try atropine, isoprenaline.
2. Sinus tachycardia is caused by pain, hypovolemia/hypervolemia, hypoxia/hypercapnia, heat loss, burns, and sepsis. *Treat* the cause itself. In hypertension give 1/4–1/2 mg β-blocker (propranolol) at 5 min intervals and a calcium blocker (verapamil) in a 5-mg bolus very slowly, intravenously.
3. Ventricular premature beats are caused by hypoxia, raised K^+ or reduced Ca^{++}, dobutamine/dopamine, sepsis, intracardiac lines, cardiac contusion, pulmonary emboli, and myocardial ischemia. *Treatment:* Treat the causative factors and administer lidocaine 1-mg/kg in a bolus intravenously, followed by the same dose again after 15 min if necessary, and then continous intravenous infusion of 2 mg/min for 24 h.

Cardiac Contusion

Some cases of cardiac contusion with or without tamponade are diagnosed *only* in the postoperative period (especially asymptomatic forms).

Table 3. Advantages and drawbacks of dopamine and dobutamine in treatment of cardiac failure

	Dopamine	Dobutamine
Advantages	Renal vasodilation Mesenteric vasodilation No vasoconstriction effects up to 10 μg/kg/min	Effects stop short of tachycardia
Drawbacks	Tends to induce tachycardia	No renal and/or mesenteric vasodilation
β Agonist	+	+
α Stimulant effect	> 10 μg/kg/min	> 10 μg/kg/min

Diagnosis:
In tamponade:
- The patient may be hypotensive with a low pulse pressure.
- CVP is always elevated, neck veins are distended.
- If a pulmonary artery catheter is inserted, then CVP = pulmonary wedge pressure = pulmonary arterial pressure.
- The chest X-ray shows a "tear drop" heart but may be normal (if the amount of pericardial fluid is small). An echocardiogram will help the diagnosis, but the definite diagnosis can be established only by pericardial puncture and aspiration of blood.
- Heart sounds are weak und distant.

In case of *contusion:*
- ECG is usually nonspecific but sometimes shows ischemia, conduction blocks, or arrythmias.
- Creatinine phosphokinase and myoglobin may be elevated.
- There may be an angina-like pain which is not relieved by nitrites.

Management
- Cardiac monitoring in every case of blunt chest trauma.
- Daily ECG for the next 3–4 days.
- Appropriate treatment of cardiac failure and arrythmias (see above).

Acute Renal Failure

Main causes in the post-operative period:
- Pre- and intraoperative shock accompanied by "renal shut-down": massive bleeding; peritonitis; ileus; burns.
- Sepsis.
- Drug toxicity: antibiotics; contrast medium.
- Hemoglobinuria (massive and/or mismatched blood transfusion).
- Postrenal obstruction: trauma.
- Rhabdomyolysis skeletal muscle in crush injury: cell contents escape into the extracellular fluid and into the blood stream (see also chapter on Crush Injury).

Diagnosis

Oliguric Renal Failure
Postrenal obstruction usually causes anuria: an ultrasonographic examination can reveal or exclude a bilateral ureteral obstruction, whilst renal arteriography excludes an arterial traumatic occlusion. In case of oliguria (less than 20 ml) urine/hour, a fluid challenge may be tried (a 250 ml bolus of saline is administered intravenously within 10–15 min. Concomitantly, samples of urine and blood are sent to laboratory for testing (see Table 4).

Nonoliguric Renal Failure
Nonoliguric failure is the clinical form with a better prognosis: a near-normal urinary output is combined with a steady rise in serum creatinine and fall in creatinine clearance. The clinical course is milder since there is little fluid overloading, hyperkalemia, or metabolic acidosis.

Table 4. Measurements for differential diagnosis in oliguric acute renal failure

	Pre-renal failure	Acute tubular necrosis
Urinary Na^+ (mEq/l)	< 25	> 25
Urinary specific gravity	> 1025	< 1025
Urine/plasma urea (units)	> 1.8	< 1.1
Urine/plasma creatinine (units)	> 30	< 10

Treatment

1. Fluid balance:
   - In nonoliguric failure, according to urinary output.
   - In oliguric failure, a strict regime of fluid restricted intake (insensible loss minus endogenous water daily fluid needs), and add any extra-renal loss (nasogastric tube, drains, etc.).
2. Potassium:
   - Continuous cardiac monitoring (since the most dangerous effect of hyperkalemia is arrhythmia, including ventricular fibrillation).
   - No intake of K^+.
   - Infusion of dextrose + insulin in order to facilitate entry of K^+ into cells.
   - Administration of exchange resins (orally, rectally, by nasogastric tube).
   - Administration of bicarbonate for acidosis.
   - Dialysis if these measures fail.
3. Calcium. Hypocalcemia is common: administer Ca^{++}, especially if bicarbonate is injected (an alkaline pH exacerbates hypocalcemia).
4. Magnesium. Mg^{++} administration is not indicated; in oligoanuria hypermagnesemia could lead to cardiac block.
5. Nutrition. The goal is to maintain the essential proteins without exacerbating azotemia. Hence, for protein, 6–7 g N should be given daily (essential aminoacids), and 150–200 calories/1 g N administered (in the form of hypertonic glucose).
6. Dialysis (see Table 5). Indications for dialysis are: pulmonary edema, refractory hyperkalemia, severe metabolic acidosis, uremic encephalopathy (seizures), coagulopathy (platelet defect).

The mortality in acute "traumatic" renal failure is still more than 60%.

Administration of intravenous fluids on site, during evacuation, and on the operating table in sufficient amounts can prevent the development of acute renal failure in many cases.

By giving diuretics there exists a chance of transforming an oliguric into a nonoliguric form of renal failure. However, diuretics could accentuate ex-

Table 5. Peritoneal dialysis vs. hemodialysis

	Peritoneal dialysis	Hemodialysis
Hemodynamic instability	Less	Frequent
Efficiency	Less	More
Need for equipment	Less	More
Need for continuous monitoring	Less	More
Need for regional anticoagulation	No	Yes

isting hypovolemia or may cause a further nephrotoxic insult if furosemide is administered. An abrupt increase in intravascular volume and further cardiovascular deterioration could be produced by rapid administration of an osmotic diuretic such as mannitol (20%, 350–500 nl).

Brain Injury

Brain dysfunction in the postoperative period can be a result of:
- Penetrating or blunt acute head injury associated with hematomas or fractures.
- Brain concussion in combination with injury to other systems.
- "Second brain insult" produced by hypoxia, hypercapnia, or hypotension, resulting in brain ischemia and permanent neuronal damage (see Fig. 1).

Aggresive medical therapy and invasive monitoring help to minimize intracranial hypertension and subsequent neuronal damage.

Intracranial Pressure Monitoring

Indications:
- Comatose patient after head injury or neurosurgical procedure.
- A patient after multiple trauma who has an altered level of consciousness and must undergo various surgical procedures for other lesions.
- Any patient who has had an intracranial hematoma evacuated.

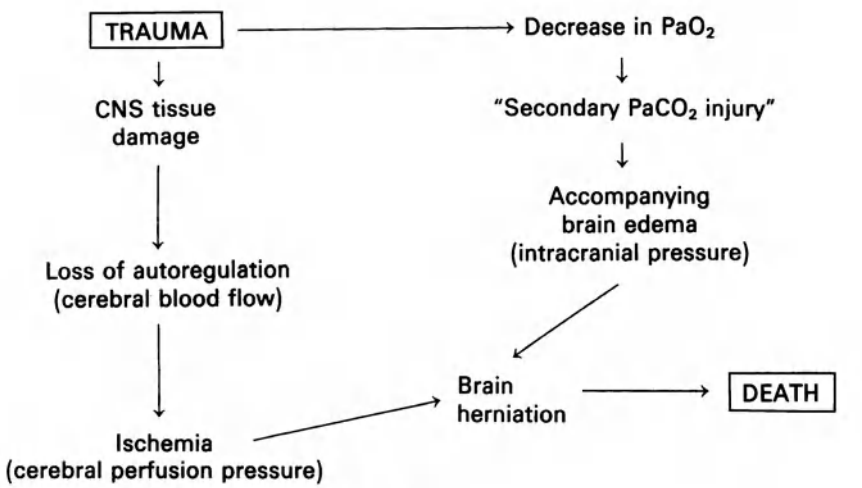

Fig. 1. Algorithm of "second brain insult"

Technique:
- Continuous measurement of pressure.
- Drainage of CSF in case of hypertension.

The main danger of intracranial monitoring is *infection!* Remove the intraventricular device as early as possible. (See also chapter on Head Injury.)

Intracerebral Hypertension

In the prevention and treatment of intracerebral hypertension (including postcardiac arrest brain damage), the goal is to keep the intracranial pressure lower than 15 mmHg.

1. Hyperventilation:
   - $PaCO_2$ 25–30 mmHg.
   - Use a capnograph for obtaining "on-line" data.
2. Sedation: barbiturates. If the intracranial pressure is higher than 20 mmHg, give a high dose of pentobarbital (rather than thiopental, which depresses cardiac function). Loading dose: 10 mg/kg/h for 4 h; then 100–200 mg/h in order to keep a constant blood level of 2.5–4 μg/ml.
3. Keep serum osmolality <300 >280 mosmol/l.
4. Oxygenation: PaO_2 must be kept to 100 mmHg. The use of PEEP at 10 cm H_2O hardly increases the intracranial pressure, so do not hesitate to use moderate levels of PEEP if the pulmonary condition demands it.

Continuous Clinical Surveillance

Brain injury patients should be continuously monitored to detect:
- Deterioration in the level of alertness.
- Development of lateralizing signs.
- Pupillary changes.
- Changes in vital signs.
- Progressive deterioration in motor response.

Notify the neurosurgeon whenever there is a significant change in the patient's condition.

Measure the intracranial pressure.

Order a computer tomographic scan on suspicion of a space-occupying intracranial lesion.

Use the Glasgow Coma Scale for continuous evaluation and assessment of neurological condition and progress.

Sepsis

The origins of sepsis may be as follows:
- Infected wounds: peritonitis; empyema; open fractures; burns.
- Invasive monitoring: peripheral intravenous lines; CVP lines; pulmonary artery catheter; arterial line; urinary catheter; intracranial pressure catheter.
- Respiratory: prolonged intubation; tracheostomy wound; atelectasis; aspiration pneumonia.

Tetanus

Intubate and ventilate the patient. Perform tracheostomy (since the patient will need to be ventilated for weeks). Administer neuromuscular blocking agents for the first period, together with large doses of intravenous diazepam, 10 mg, according to blood pressure and state of awareness.

Aspiration Pneumonia

The danger of aspiring gastric contents is ever present at all echelons from the site of injury to the intensive care unit of the base hospital. There is no evidence that prophylactic antibiotic therapy is of benefit. Nevertheless, suspect pneumonia in the first 24–72 h after aspiration has occurred, and ensure that a daily chest X-ray and sputum cultures are carried out. Antibiotics effective against anaerobes (the majority of the germs present in the oral cavity) are given only if clinical and radiological signs confirm pneumonia.

Septic Invasive Procedures

Nonsterile procedures may be performed on the battlefield or at any echelon on the way to the base hospital, for instance: peripheral intravenous infusion, central vein cannulation, chest drainage, tracheostomy. Suspicion of an infection in such a nonsterile procedure dictates that the line be changed as soon as possible and the former catheter be cultured. In case of positive cultures, antibiotics must be administered accordingly and the patient be observed for signs of local infection.

Postoperative Nutritional Support

Postoperative nutritional support is needed in the hypercatabolic phase in the postoperative period following major surgery, burns, and sepsis, or during interruption of the function and/or anatomical integrity of the gastroin-

testinal tract which prevents normal alimentation (postoperative ileus, fistulas, diversions).

Daily Needs

Nutritional support must be individualized. The amount of N and calories as well as the route of alimentation should be decided upon according to the catabolic state and the integrity of the gastrointestinal tract. The protein needs are calcuated as follows: daily loss of N = urinary urea(g)/2 + 2 g; 1 g N = 6.25 g proteins.
The caloric needs are calculated according to the severity of catabolism and daily N loss. For every 1 g N lost one should administer:

- In mild hypercatabolism (e.g., postoperative period), 150 calories
- In moderate hypercatabolism (after major surgery), 175 calories
- In severe hypercatabolism (sepsis) 200–250 calories

Beside proteins and calories (carbohydrates and lipids), trace elements and vitamins should be added and water and electrolytes given in accordance to daily renal and extrarenal loss.

Routes of Administration

Enteral

This is always the route of choice because it is natural and has few complications.

Mouth
In feeding by mouth, the only concern is to be able to provide enough calories and proteins to cover the daily needs.

Nasogastric Tube
Indications:
- The patient is unable to tolerate mouth feeding.
- Liquid supplements are indicated in addition to regular meals.
- The patient is not alert enough to feed by mouth.

The basic required qualities of the nutrient solution are a relatively low osmolality and an energy content of 1–1.5 cal/ml.
Techniques: Normal nasogastric tubes should be used when there is a suspicion concerning the absorption and gastric suction must be performed every couple of hours. Thin silicone tubes have the advantage of being better tolerated and cause fewer complications (otitis, sinusitis, etc.).

- Initiate the feeding using 0.5 cal/ml.
- Drip at a constant rate (50 ml/h).
- Watch out for gastric retention and diarrhea, then
- Increase rate to 75 → 100 → 125 ml/h.
- Subsequently, increase the caloric density to 0.75 cal/ml → 1 cal/ml.

Gastrostomy
Indications: After chest or abdominal surgery when there is an anticipated need for prolonged enteral feeding.
Drawbacks: Surgical procedure; does not prevent aspiration.
Techniques and rules: As for nasogastric tube.

Jejunostomy
Indications: At the end of surgery when aspiration is anticipated (impairment of the swallowing mechanism).
Advantages: Interposes the pyloric sphincter between the food and pharynx.
Drawbacks: Surgical procedure, accompanied by relatively high rate of diarrhea.
Techniques and rules: As for nasogastric tube.

Total Parenteral Nutrition

Indications:
- Loss of integrity of gastrointestinal tract
- High risk of aspiration
- Complications of enteral feeding
- Repeated surgical procedures (e.g., for burns) which require long periods of fasting

Approach:
- For relatively short periods (<2 weeks) of total parenteral nutrition (TPN), choose the internal jugular vein.
- For prolonged TPN, choose the subclavian vein, with or without subcutaneous funnelization.

Rules:
- The main danger of TPN is *line sepsis*. Insertion must be under strict asepsis.
- The TPN line must never be used for any other purpose (CVP measurement, drug or fluid administration, withdrawal of blood, etc.).
- The protein needs have to be covered by a solution of essential/nonessential aminoacids.
- Caloric needs should be provided by carbohydrates (2/3) and lipids (1/3).

- The available 3-l bags permit mixing of aminoacids, dextrose and soya lipids: this mixture avoids multiple punctures of bottles and bags.
- Add a special bottle containing electrolytes, vitamins, and trace elements.
- Change intravenous lines every 7–10 days if the daily inspection shows no signs of local infection, or immediately on discovery of
- Signs of local skin infection of sepsis of unknown origin.
- A special nutritional calculation should be done in renal failure or hepatic failure.

Stress Ulceration

The 10 risk factors for developing stress ulcer are:

1. Shock
2. Sepsis
3. Burns
4. Respiratory failure
5. Cardiac arrhythmia
6. Hepatic failure
7. Polytrauma
8. Renal failure
9. Major surgical procedures
10. Fat embolism

Recommended Protocols of Prevention

Feeding by Mouth
- Check the gastric pH and make sure that it is kept above 3.5
- If pH <3.5, add antacids — sodium citrate 0.3 mol 20 nl

Parenteral Feeding
- Insert a nasogastric tube
- Suction gastric contents
- Administer antacids 20 ml
- Check gastric pH after 60 min

Cimetidine or ranitidine intravenously administered elevates pH slowly and should be administered at least 0.5 h before surgery.

Management of Upper Gastrointestinal Tract Bleeding

Diagnosis: hematemesis; melena; coffee grounds

Treatment:
- Assure wide-bore intravenous cannulation
- Order blood
- Measure hemoglobin and coagulation factors
- Check the orthostatic hypotension:
  A fall in blood pressure of more than 20 mmHg shows a loss of 25% of blood volume.
  An increase in the heart rate of more than 20 beats/min is regarded as a sign of active bleeding and calls for a blood transfusion.
  A large nasogastric tube should be inserted and kept in place for at least 24 h.
  The bleeding will stop in 75% of cases, but if it continues endoscopy should be recommended.

Emotional Factors in the Postoperative Period

Causes of Emotional Stress

Battlefield events and their memory.
Long-term intensive therapy.
Mutilation, uncertainty, fear of the future, despair.
The family: worry about those at home, or antagonism between the wounded and the family.

Alleviation of Psychic Injury

Keep the patient fully informed.
Instill confidence by constant reassurance.
Make contact with the family if possible.
Expose the patient to the media: TV, radio, newspapers.
Reduce stress factors such as noise, light, pain, tubes, and catheters.
Use psychiatric consultation.

Repeated Anesthesia

Indications:
- Burns (change of dressings); skin graft; cosmetic surgery.
- Reconstructive surgery to limbs and face.
- Complications of surgery such as bleeding, breakdown of repairs, or septic foci.

Hepatotoxicity. There still is a controversy concerning the role of repeated anesthesia in producing hepatotoxicity. Take the following precautions:

1. Do not use halogenated inhalatory drugs if you plan further exposure to anesthesic drugs.
2. In case of postoperative unexplained jaundice or unexplained fever, do not use halogenated inhalatory drugs for the next surgical procedures.
3. Isoflurance can be used repeatedly and is the drug of choice.

Chronic Pain

Chronic pain is associated with damage to peripheral nerves in various parts of the body (causalgia) and the development of pain syndromes such as phantom limb.
The treatment of chronic pain is difficult:

1. The cause of the pain may be impossible to diagnose.
2. The cause of the pain may be correctly diagnosed but impossible to treat.
3. The cause may be correctly diagnosed and treatment given, but without bringing relief.

A complex aspect of chronic pain is the inter-relationship between the patient's psychology and behavior and the organic cause of pain. The distinction between organic pain and the psychic manifestations of a former injury is almost impossible to make.

General Rules of Treatment

1. Reassurance and psychotherapy.
2. Moderate use of strong analgetics (remember the dangers of secondary effects and addiction).
3. Use of an electrical nerve simulator — an innocent method if effective.
4. Repeated reversible nerve blocks for painful areas corresponding anatomically to a region under the influence of a particular peripheral nerve, nerve root, or ganglion.
5. Intrathecal administration of phenol (or alcohol) — indicated only when other more conservative methods have failed to give relief.

Although acupuncture is not used in China for relief of chronic pain, since a patient is referred for this kind of treatment after all previous "orthodox" methods have failed, it is worth trying, and any benefit is very acceptable indeed.
Maximal daily physical activity reduces pain perception, probably due to secretion of endorphins.

Further Reading

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3.11 Wounds of Soft Tissues and Limbs

N. D. Reis

General Principles

Wounds of the soft tissues and limbs comprise about two-thirds of all war and disaster wounds. The on-site principles of management follow the order described in the chapter on primary treatment of wounds in the field. All wounds are left open after the primary treatment in hospital except for special ones, such as sucking wounds of the chest, wounds of the peritoneal cavity, and wounds exposing the meninges or the brain, all of which require primary closure.

Hospital Management

Routine on Admission

The surgical team receiving the casualties must have a clear-cut concept and *routine* for the examination, resuscitation, and priority assessment in the multitrauma case. The proper sequence of action is thus determined. Commonly, mistakes are made due to failure to strip off all clothes and to turn the casualty to examine the entire dorsal body surface. At this stage, urgent attention to the limb is concerned only with the arrest of hemorrhage, temporary sterile wound cover, and splinting. Locally applied manual pressure is the method of choice for the arrest of bleeding, and when necessary this should be the sole occupation of one of the attendants.
On arrival at the emergency triage area tourniquets are immediately removed if present, and after ensuring that there is no significant bleeding, and other injuries permitting, the casualty is X-rayed on the way to the operating theatre. Instructions are given not to expose the wounds unnecessarily and to restrain clinical curiosity until the wounds are inspected thoroughly in the relatively sterile environment of the theatre. Meanwhile, the prophylactic antibiotic routine of 5 million units of crystalline penicillin IV 6-hourly and 80 mg gentamicin 8-hourly is instituted or continued.

A final clinical examination of the state of the wound or limb should now be made. A good light and a gentle pair of hands are all that is needed for the detailed clinical examination which is mandatory for decision making and for the later evaluation of progress and complications. Record the state of all the tissues and functions of the limb: the pulses, venous and capillary filling, the neurological findings, the precise description of wounds (type, location, extent), and damage or loss of bone, muscle, skin, and joint. If the casualty is unconscious, shocked, or drugged, the wounds and limbs should be re-examined as soon as the patient is able to co-operate.
Radiographic examination is essential for the evaluation of fractures and identification of foreign bodies. Angiography is indicated only when the multiplicity of the wounds handicap the localization of the site of vascular injury by physical examination alone.
Prior to the induction of anesthesia the general condition of the casualty is reassessed, taking into account the proper priorities of multiple injuries.

Anesthesia

Local anesthesia is not practical except for the most minor wounds. Regional blocks are ideal for distal limb injury. General anesthesia is required for injuries of the trunk and proximal limbs.

Wound Lavage

The routine of wound lavage is as follows: The entire limb (or wound area) is thoroughly scrubbed using a soapy antiseptic solution, and the wounds are flushed out with jets of Ringer's lactate solution. The whole process is then repeated, flushing out with copious amounts of fluid whilst the gloved hand gently opens up the tissue spaces of the wound to allow jets of fluid to reach the depths.
A very wide area around the wounds or the skin of the entire limb is then prepared by painting with an antiseptic solution, including the limb girdle when the wound is proximal, and the patient is towelled up, leaving a large area around the wound exposed and the entire limb free for manipulation as required. When a fracture is present in a limb wound suited for the application of an external fixation device, the next step before debridement is wound stabilization.

External Fixator Wound Stabilization

The correct sequence of the operative steps is important. The external fixator screws or pins are first introduced, whenever possible, through normal skin (always apply Schanz screw fixation from one side only in preference

to a through and through configuration; this is often not possible when pins must be placed near joints or when the frames must vault over joints, and in these cases through pins are used).
Rigid fixation is not the objective, rather stability which gives rest to the wound and allows movement of the limb joints. If necessary, the apparatus spans a joint. The external fixator can often be usefully combined with one or several interfragmentary lag screws (so-called minimal internal fixation).
The image intensifier is very helpful in the precise placement of the screws and pins, but X-ray control is not essential. In planning the placement of the pins and/or screws, future procedures must be taken into consideration (vascular surgery, peripheral nerve surgery, muscle and/or skin pedicle flaps, microsurgical vascular pedicle bone, and/or soft tissue transfers), so that the apparatus will not prevent or complicate essential delayed primary or secondary operations. Having rigged the scaffolding, a near-normal limb anatomy can now be restored by distracting to normal limb length, this greatly facilitating the subsequent painstaking excision of the wound and the exhaustive exploration of its depths and the missile tracts. When the wound area is collapsed down and unstable, it is most difficult to navigate and chart the disrupted anatomy: Stabilization and distraction makes this task easier and quicker.

Wound Debridement

Excision of the soft tissues which are judged to be avascular or impregnated with foreign material is performed next. The tracts of superficial through and through wounds are laid open along their entire length. Deep penetrating wounds are best excised by approaching the entry and exit wounds separately.
Excision of skin edges and necrotic subcutaneous and fascial tissues is followed by excision of the non-viable muscle. Viable muscle is reddish rather than blue, bleeds when cut, and contracts when stimulated, the latter being the most reliable sign. If necessary, incisions in the skin, fascia, and muscle planes are extended for access to deeper structures to remove foreign material, facilitate drainage, and allow the exposure required for proper exploration of the wound. For wounds of limbs distal to the upper arm and thigh, a pneumatic tourniquet is always applied at the ready, but it is seldom used as it tends to obscure judgement of tissue vascularity; inflation is necessary when identifying fine neurovascular structures. *The wound is always left open.* When bone is exposed and is dirt impregnated, it is scrubbed with a brush, or when brushing fails to clean, decorticated with a sharp osteotome or rongeur.
Free cortical fragments, if required for stability, are retained at this stage. High velocity missiles often cause very severe damage, the extent of which

is not apparent at the first session; nevertheless, the aim should always be a complete primary wound debridement. Wound exploration and excision are not finished until the entire wound is seen to be composed of living tissue and until the total extent and all the details of the structures in the wound, including vessels and nerves, have been observed.
Wound excision is repeated as indicated every 48 h under anesthesia until all non-viable tissues have been removed.

Wound Coverage

Delayed primary suture or *delayed primary split-skin grafting* is performed on the 5th day, providing the wound is free of all necrotic tissue. Suturing is done for all wounds in which the skin edges can be approximated without tension. Split-skin grafting is performed when skin loss prevents suture without tension and when the wound contains healthy vascular soft tissues.
When bones, joints, or tendons lie exposed in the wound (the bone and tendon are usually avascular), healing is promoted, and infection is prevented by bringing vascular soft tissue to cover the wound. If local skin can be maneuvered without tension to cover them, then this should be done. Sometimes this will require a releasing incision in skin over soft tissue on the opposite side of the limb (care must be taken not to reduce the vascularity of the skin at the wound edges). If skin cover is not available, the following techniques are considered.

Local Muscle Pedicle Transfer (used mainly in the lower limb). The function of the normal muscle or the part of it used in the transfer is sacrificed for the sake of wound cover, viability, and resistance to infection. The principle is to isolate and mobilize the donor muscle, if necessary freeing its origin, insertion, or both, being careful to preserve its blood supply. In transferring the belly, care is taken not to over-stretch or twist the vascular pedicle to the muscle in order to ensure that it remains viable in its relocated position. Frequently, the fascia covering the muscle, its aponeurosis, or tendon may be sutured to the edges of the defect to fix the transfer in position. It may not be necessary to detach the donor muscle at the origin or the insertion; freeing part or all of the belly and swinging its middle over an adjacent bony defect may suffice, e.g., tibialis anterior to the middle one-third of the tibia. The muscle transfer is in turn covered with a split-skin graft either immediately or, if there is any doubt of the vascularity of its outer surface, as a delayed procedure.
When the wound bed does not contain avascular bony fragments or tendons and it has been adequately excised, then cover is no longer a problem since vascular granulations will soon fill the wound, and it can then be covered with a split-skin graft. Only when avascular bony fragments are present in the wound (or tendons or an open joint are exposed) or there are

defects in the chest or abdominal wall does the problem of wound cover become complex. If the entire avascular bone mass is excised, the bone defect and the bulk of the bone graft that will be required to fill the defect are increased and stability reduced. Having decided under such circumstances that excision of the avascular bone or discarding large avascular cortical fragments will produce too large a defect, it is then that we are faced with the problem of how to obtain wound healing and wound coverage without infection of the avascular tissues in the wound. This situation occurs very frequently in massive wounds of the limbs, and then vascular pedicle transfer to cover the avascular bone becomes an important technique. Such a muscle transfer may also be indicated to cover a cancellous bone graft in a massive defect.

Vascular Skin Pedicle Flap Transfer (used mainly in the upper limb). At the delayed primary stage the pedicle skin flap is indicated precisely for the same reasons as the pedicle muscle transfer. Whereas the pedicle muscle transfer is used for the lower limb, the pedicle skin flap is used in the upper limb, from various sites on the chest wall, opposite upper limb, groin, and abdomen. Transfer of skin pedicles from the trunk or opposite upper limb to the injured upper limb are simple and convenient, with plenty of good vascular skin available. However, in the lower limb cross-leg skin flap techniques necessitate whole body immobilization and "waltzing" from trunk to lower limb flap procedures are lengthy and complex, both procedures being undesirable in patients suffering from multiple injuries. Local transposition and rotation skin flaps are not recommended at this stage: The local skin tissue is likely to be much less viable than its external appearance may suggest.

The Open Wound Method

When no vascular soft tissue is available to cover the exposed defect and the wound cannot be covered by split skin for lack of a vascular base for the graft, the wound is left exposed until granulations creep over it (usually within 2–4 weeks), after which a split-skin graft may be applied, but often the wound has contracted to such an extent that grafting is no longer needed. An autologous cancellous bone graft can be similarly left exposed in the wound.

Quality of Skin Cover. A split-skin graft is adequate for sound wound healing. A pedicle graft for definitive high quality vascular skin cover (over a joint or friction area) is ideally performed as a secondary procedure.

Timing of Wound Coverage. Definitive wound coverage can only be performed when the wound is clean of all necrotic soft tissue. This can usually be achieved within the first 10 days. When it is absolutely certain that a primary definitive excision of the wound has been achieved, then the mus-

cle or skin flap transfer over a defect or avascular bone, tendon, or joint can be performed at the delayed primary stage with little risk of failure. Usually one cannot be certain that all soft tissue remaining in the wound is viable, and therefore suspicion of wound non-viability and danger of infection exists, and the skin flap or muscle transfer should be delayed. However, a race against time exists to achieve living vascular tissue cover over exposed avascular bone and/or tendons if these are to be saved, since once infected they will have to be excised radically. This means that repeated excisions must be pursued aggressively for a maximum of about 12 days, after which infection is usually well-established in dead bone. Exposed tendons and aponeuroses usually become infected and necrose by the 5th day.
Exposed nerve and vessel repairs should, whenever possible, be covered primarily by mobilization of adjacent, living, soft tissue elements. Repeat excision of remaining necrotic tissue should be performed every 48 h and all potential dead space drained as required.
Crush wounds and wounds caused by high velocity missiles are particularly prone to need repeated excision because the extent of tissue necrosis cannot be ascertained at first.

Nerve Injury

When a nerve lesion is diagnosed, primary excision of the wound is not complete without a formal exploration of the nerve from normal tissue into the wound area and out again into normal tissue so that the precise state of the nerve has been observed. It is quite wrong to delay exploration since without an exact diagnosis, optimal treatment cannot be planned. Doubt as to the prognosis of the nerve injury prevents correction decision making.
It is accepted teaching that lacerated or crushed nerves in severe wounds are repaired secondarily. At excision the nerve ends are stitched down to adjacent soft tissue to prevent retraction. However, secondary nerve suture following severe destructive injury is often impractical because of scar distortion of the anatomy and gross nerve tissue loss. Microtechniques in nerve grafting have not proved to be conspicuously more successful than the older methods.
When limb function will depend on the repair of a major nerve injury, the best chance of success in massive wounds is achieved by radical excision of the damaged nerve ends and, when necessary, limb shortening and end-to-end suture without tension as a delayed primary procedure.

Wound Stabilization

Following primary, delayed primary, or secondary procedures, the wound should have "perfect rest" whilst joints and muscles should be exercised

as soon as possible. This is best achieved by using external fixation. When not possible or not available, padded plaster of Paris casts and splints are used instead: Circumferential casts are immediately bivalved after application. Joints are placed in a neutral functional position, or in whatever position relieves tension on soft tissue coverage and nerve, vessel, or tendon repairs.

Preventing Wound Infection

The primary principle for the prevention of infection of the wound is radical excision of the necrotic non-viable tissue.
The open wound is dressed twice daily by gently applied gauze soaked in dilute antiseptic. All circumferential dressings are strictly forbidden. The purpose of wound dressing is to prevent desiccation and secondary contamination from the environment. In addition, if a dead space is present, a tube is placed in the depths of the wound for constant drip irrigation, and a dependent counter incision is made. The purpose of irrigation is to maintain moisture, mechanically flush out, and promote lavage of those pockets not drainable by dependency.
Five million units of crystalline penicillin IV every 6 h are routinely given for 10 days. Gentamicin 80 mg every 8 h is given IV or intramuscularly for 5 days only. If penicillin sensitivity develops during treatment, use 4–6 g cephaloridine daily parenterally for 10 days.
Cultures are taken from the wound at primary surgery and at 2-day intervals until wound healing. The antibiotic treatment is changed according to sensitivity tests performed on the bacterial cultures. Long-term antibiotic therapy is reserved for the treatment of traumatic osteomyelitis.
A tetanus toxoid boost is given routinely unless the casualty proves to be up-to-date with tetanus shots. Tetanus antitoxin and anti-gas gangrene sera are not used, as their efficacy remains in doubt: The combination of radical surgical excision and massive dose penicillin excludes anerobic infection.

Wound Infection

When primary wound infection develops (usually clinically obvious on the 4 or 5 post-injury day), the cause is necrotic tissue left in the wound, inadequate drainage of dead space, premature wound closure, or retained foreign bodies including dirt, earth, clothes, missiles, etc.
It is treated by widely opening the entire wound, repeating radical excision, ascertaining efficient drainage of spaces in the wound and/or eliminating dead space, taking cultures, giving parental antibiotics, and where necessary instilling local irrigation suction treatment.
The prevention of late infection is achieved by meticulous execution of the principles of wound excision and drainage, by stabilization of the bony el-

ements, and by vascular soft tissue coverage of avascular bone. Late infection developing after 2–3 weeks or more is due to osteomyelitis, abscess formation in dead space, and retained contaminated foreigns bodies. The treatment is to lay open the wound, excise all infected dead bone and remaining dead soft tissues and foreign bodies, eliminate dead space, and drain and instill open constant irrigation or closed irrigation suction treatment for 3 weeks.

3.12 Injuries to the Bones and Joints

N. D. Reis and C. Zinman

The on site and medical aid post management is described in Chap. 3.6 and the primary hospital management of wounds of the limbs in Chap. 3.11. This chapter is devoted to methods of skeletal stabilisation, the treatment of bone stock loss, post-traumatic bone infection and joint injuries in the field or base hospital.

Skeletal Stabilisation

Biplane radiographs are made and then the appropriate method of stabilisation is chosen.

Casts and Splints

Plaster of Paris casts and splints are the primary method of choice for open and closed long bone shaft fractures when external fixation is not available and for most fractures in and around joints, providing that the wound is not a massive complex one requiring repeated attention and revision surgery. The casts are well padded and fenestrated in order to allow inspection of wounds, change of dressings, and the taking of bacterial cultures. The windows are very large and extend beyond the limits of the wounds. The extent of the wounds is marked on the cast immediately on completion. Once dry, circular casts are always bivalved and the fenestra are cut; the cut-outs are kept and bandaged back into place to prevent local swelling. All the details of the underlying injury are then drawn and written onto the cast.
The following are the recommended casts and splints for the various anatomical locations.

Foot. The cast is from below the knee to beyond the toes on the sole; the toes are left free on their dorsal surface, however, to allow for toe extension and inspection. The foot is plantigrade.

Ankle, Leg and Knee. The cast extends from the groin to the toes, with the foot plantigrade or in up to 20° plantar flexion and the knee in 20° flexion.

For severe fractures of the tibia a Steinmann crosspin through the proximal tibia can be incorporated into the cast.

Thigh and Hip Joint Region. A spica cast-bandage extends from the nipples to the toes on the injured side and to above the knee on the uninjured side. The ipsilateral knee and hip are positioned as dictated by the fracture and the foot is plantigrade. The spica is reinforced by staves of wood across the hip on the affected side and connecting the legs (from the front of the knee on the injured side to the front of the thigh on the uninjured side).

Hand. (See Chap. 4.11).

Wrist, Elbow and Forearm. The cast extends from the axilla to the proximal palmar crease and dorsally to the knuckles. The fingers are free and the thumb is opposed but abducted. The elbow is at 90°, the forearm is pronated, and the wrist is in 20° extension and in neutral deviation. For proximal forearm fractures the forearm is supinated.

Arm and Shoulder Joint. A shoulder spica in the Velpeau position is applied with copious padding in the axilla and between the elbow and the chest wall. The wrist and hand are free.

Skeletal Traction

Traction is the best conservative treatment for fractures of the *femur* but has the disadvantage of confining the casualty to bed, making it more difficult to manage the intensive care, and may interfere with proper wound management. External fixation is therefore to be preferred for the primary treatment of open fractures (see Fig. 2) and internal fixation for closed fractures. For open fractures too close to the hip joint to be treated by external fixation, traction is better than a spica cast, unless the casualty must be transported.
When external fixation is not possible or not available for fracture of the *humerus* and a shoulder spica cast-bandage is contraindicated because of the need to have the chest free, bone traction is applied through the olecranon.

Internal Fixation by Osteosynthesis

Closed fractures may be treated by primary osteosynthesis but only by a surgeon experienced in the method. Open fractures are rarely internally fixed primarily, and the indications can only be determined by a specialist. External fixation is much safer, occasionally in combination with minimal internal fixation, which can be added at a delayed primary or secondary stage when the wound is clean. However, in the hand or foot, or within a

joint, internal fixation of open fractures by transfixing pins or screws is often the method of choice as a delayed primary procedure.
Osteosynthesis, often in combination with a bone graft, is extensively indicated as a secondary method of choice for open fractures after primary management by external fixation, traction or plaster cast until the major wound problems have been solved.

External Fixation of Fractures

External fixation is the optimal method for the management of severe open fractures and involves the use of percutaneous transfixing pins, transversing the bony fragments perpendicular to the long axis of the bone. The protruding pins are connected outside the limb by a rigid but adjustable scaffolding: it is this scaffolding that is "external". In this way, there is a minimum of metal inside the tissues. The fracture elements can be realigned, distracted or compressed at will.
External fixation makes it possible to salvage limbs with extensive open fractures and soft tissue damage that might otherwise require amputation. Its application is simple, quick and safe as long as one adheres to the strict rules for pin insertion, to avoid pin tract infection and ring necrosis in the bone, and has sufficient knowledge of the anatomy of the limbs to avoid further damage to vessels and nerves.

Indications

1. Open fracture with severe soft tissue injuries or large soft tissue defects.
2. Badly comminuted closed fracture, especially in a patient suffering from multiple injuries.
3. Polytrauma patient requiring intensive care, in whom fracture stability is needed immediately at minimal risk.
4. Fracture in a burn area.
5. Closed fracture which develops a compartment syndrome and fasciotomy is indicated.
6. Failed infected internal fixation and post-traumatic osteomyelitis.
7. Limb stabilisation required for major vessel and/or nerve repair.

Advantages

1. The wound area is easily exposed: dressings and repeated surgical procedures are convenient and cause the patient minimal discomfort (often a difficult problem where there are large soft tissue defects).
2. Limb elevation is facilitated: this reduces swelling and makes dressing and wound irrigation convenient.
3. Early movements of adjacent joints are possible with little pain.

4. Stability of the skeleton and soft tissue is achieved despite bone loss and/or comminution.
5. The procedure is rapid and non-shocking.
6. Limb length is preserved.
7. Transportation and evacuation to the rear are then easy.

Principles

1. Assemble the external fixation frame before wound excision: use any frame type you are trained to use providing that it is quick and simple to apply. Restore limb length.
2. Inspect the bone ends and excise avascular, loose splinters of cortical bone and foreign bodies.
3. Retain cortical bone attached to soft tissue.
4. Now perform soft tissue wound excision (see Chap. 3.11).
5. Reduce the fracture and perform a minimal internal fixation, whenever possible, by applying interfragmentary screws.
6. Compress whenever possible. However, most frames are used where there is bone loss or a comminuted unstable fracture — these are called neutralization frames; in such cases compression is appropriate only much later (see point 7).
7. Keep a comminuted fracture distracted at first, to preserve limb length; compress it only weeks or months later, when the local conditions are suitable.
8. Ensure fracture end contact: this is very important to prevent non-union.
9. When bone mass is lost, the external fixation apparatus assures limb length and axial malignment. Treat the defect by one of several bone grafting techniques, repeated as required.
10. Leave the external fixation in situ until bony union has occurred, except in cases of pin tract infections or delayed union. In cases of the latter, once the wound is soundly healed, perform internal fixation or use the cast brace technique with bone grafting as required.
11. External fixation frames: use unilateral Schanz pins in most cases in the upper limb and the femur (insert lateral pins in the humerus always with direct vision in the lower half, to avoid damaging the radial nerve) (Figs. 2, 3). In the leg, unilateral pins inserted into the medial subcutaneous area of the tibia or anteroposteriorly through the tibia crest, penetrating both cortices, provide adequate stability. This method does not penetrate muscles and tendons in the leg, and the risk of infection in the pin tract is negligible (Fig. 1). Pins can be put through damaged skin and damaged soft tissue, if necessary (e.g. in burns).
12. For wounds near or involving joints, have the frames cross over the joint (Fig. 4).

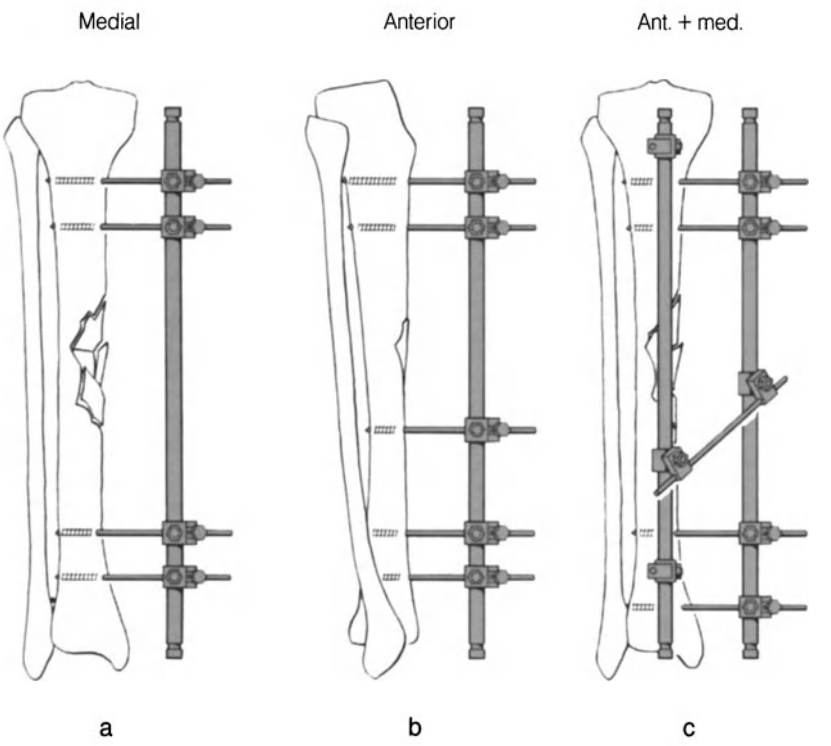

Fig. 1

13. When changes in limb length are anticipated during the treatment (e.g. primary bone shortening for vessel or nerve repair followed by secondary lengthening), use a Wagner or similar lengthening apparatus (Fig. 5).

Technique

The authors are familiar with the AO tubular system and the Wagner lengthening apparatus, the use of which is described here. The application of the AO system is simple, even for the general surgeon, but attention to detail is important. Perform each step as follows:

1. Prepare the skin with povidone-iodine solution.
2. Incise 1 cm in skin and fascia.
3. Assemble trocar in sleeve and introduce down to bone.

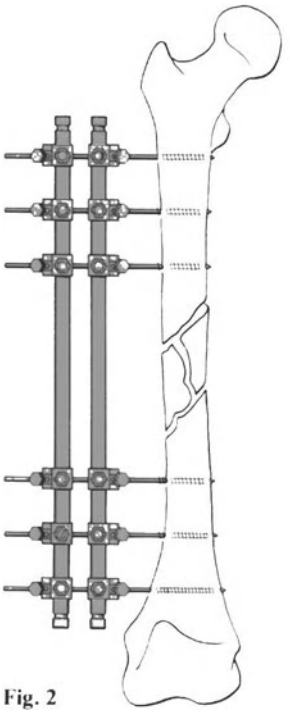

Fig. 2

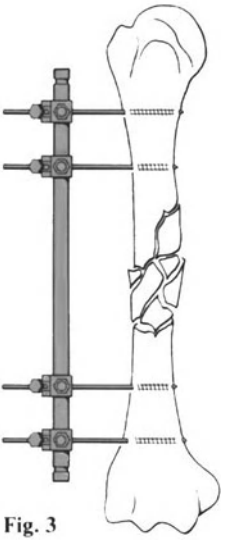

Fig. 3

4. Withdraw trocar and leave sleeve, holding it firmly against the bone at the point you wish to drill.
5. Predrill the tract gently (slow speed) using a 3.5-mm drill, then remove the drill and sleeve.
6. Introduce 5-mm Schanz screw using a hand chuck (never the power instrument) for the AO tubular system; for the large Wagner apparatus use a 6-mm Schanz screw and for the small a 4-mm Schanz screw (3.2-mm drill).
7. Make generous releasing cuts in the skin and fascia around the protruding pins, to avoid pain, soft tissue pressure necrosis and infection.
8. Image intensification is useful especially in fractures near joints, but is not essential.
9. a) The "half frame" or "single frame" technique is preferred for most fractures (Figs. 1–5).
   b) The "double frame" ("through and through") technique is used, rarely, in the vicinity of joints: use threaded 5-mm Steinmann pins in spongy bone (Fig. 6).

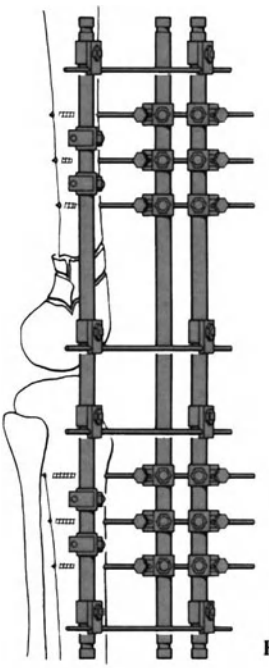

Fig. 4

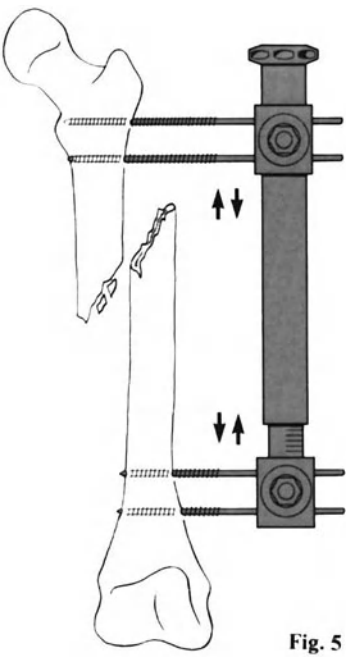

Fig. 5

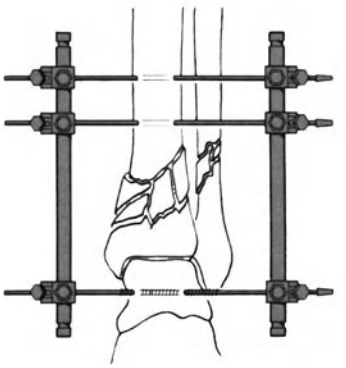

Fig. 6

10. Realign and reduce the fracture as best possible before inserting the pins.
11. In fractures with major vascular injuries, if surgery has commenced within 6 h of injury, first stabilize the fracture and then correct the arterial lesions. If surgery starts later, repair of the arterial injury takes

precedence over stabilisation of the fracture (unless a temporary bypass shunt has been installed, in which case skeletal stabilisation precedes the vessel repairs).

Complications

1. "Ring necrosis" is the name given to a tube sequestrum around the pin. The cause is introduction of a pin by high speed power drill without predrilling. Infection often occurs in addition. Avoid ring necrosis by using the proper technique; treat it by radical curettage of the pin tract.
2. Infection of the pin tract is usually due to an error in the technique of pin insertion. Prevention is by incising the skin to avoid skin tension around the pin, daily cleaning by gentle removal of skin scales around the pins, hydrogen peroxide lavage around the pin, and painting with povidone-iodine solution. The risk of pin tract infection is greatest when pins work loose.
3. Pin breakage is an infrequent complication: replace the pin.
4. Refracture can occur after removal of the external fixation. If the quality of union is in doubt, support the fracture in plaster of Paris until union is sound.
5. When the major soft tissue problems of the wound have been solved, do not hesitate to abandon external fixation for another method if indicated, e.g. by malalignment, pin infection or delayed union.

When costly instrumentation is not available, cheap improvised external fixation can be performed. Only the Schanz screws or Steinmann pins are needed: the external frames can be made of wood or, better, iron or aluminium rods or pipers, bent, if necessary, to lie snugly alongside the pins. These rods can then be fixed to the pins using dental acrylic resin, plaster of Paris, or any available method.

Joint Injuries

Whether to leave an *open joint injury* open to drain like any other destructive wound, or to strive for primary closure in order to prevent cartilage dessication and invasion by organisms, is still controversial. The following routine is recommended:

1. Primary closure is performed if excision of damaged tissue is meticulously complete and the joint can be easily closed by healthy soft tissue. Continuous in and out lines for irrigation are inserted if the joint is a large one, and the joint is irrigated constantly (3 l daily) for 3 days, antibiotics

being added to the Ringer's lactate fluid as indicated by daily bacterial studies.

2. Delayed primary closure is performed if the joint cannot be easily covered by healthy soft tissues or primary wound excision is incomplete. This is done within the 1st week after injury when the wound is judged to be clean. Closure by healthy soft tissue is achieved either by mobilisation and suture of local tissue or by muscle pedicle, skin pedicle or even microvascular free transfers. Before closure the wound is excised repeatedly as required and kept permanently moist by wet dressings.
3. Post-traumatic septic arthritis is treated by wide arthrotomy, repeat wound excision, radical synovectomy, continuous irrigation and intravenous administration of antibiotics as dictated by the cultures.
4. Severely damaged joints are best treated by primary or delayed primary arthrodesis. In the hip, secondary total joint replacement can be considered. In the knee, total joint replacement should be reserved for the elderly or those whose ambulatory function is very restricted due to other injuries or disease. Excision arthroplasty is best for some joints, e.g. metacarpophalangeal, temporomandibular.
5. Movement of the damaged joint should be as early as healing of the fracture and soft tissues allow. Careful use of continuous passive motion machines, if available, is to be recommended.
6. Residual instability and permanent local joint damage are treated by late secondary reconstructive techniques after wounds and fractures are soundly healed.

A *closed joint injury* is treated by rest and immobilisation. Instability is dealth with conservatively or by operation as indicated, primarily or secondarily, depending on the circumstances and availability of surgical expertise. Intra-articular displaced fractures should ideally be reduced and internally fixed in order to achieve near-perfect reconstruction of the joint surface.

Bone Loss

After a wound in which bone has been lost has been initially stabilised, and when the wound is judged viable throughout with no doubtful or necrotic tissue remaining (this is usually within the first 10 days), the gap that remains is filled with autogenous cancellous bone chips. An iliac cancellous bone graft is also applied generously in cases in which the fracture area is judged to be avascular (the timing for this is similar). Cancellous bone grafting may have to be repeated as necessary.

A cortico-cancellous or cortical bone strut graft may be very useful when there is a persistent large cortical bone defect. An avascular cortical strut

cannot resist infection and must therefore be used only as a late procedure after the wound has closed completely and there have been no signs of infection for many months. Ideally, cortical bone grafts should be of the vascular pedicle transfer type, if an expert in microvascular techniques is available.

In the leg, the fibula-pro-tibia method is very useful for the common problem of bone stock loss of the tibia.

Bone stock loss surgery must always aim at an ideal of vascular soft tissue coverage for all the bony elements. This means using local muscle pedicle transfers over the exposed bone and graft (or — if the expertise is available — free muscle and skin transfers using microsurgical anastomotic techniques). In the lower limb, muscle transfers are needed, whilst in the upper limb, a full thickness pedicle skin flap is usually available. The only bone graft which may utilise an open technique is cancellous bone (the Papineau technique): this excellent simple technique, which may need to be repeated several times, needs no specialised knowledge.

Allograft bone from a bone bank is another technique which is applicable only at a late secondary stage when infection from some local source in the wound has been totally ruled out. At this late secondary stage it seems likely that Ilizarov's principle and technique of controlled mechanical distraction osteogenesis will replace other methods of repairing major bone stock loss in the shafts of long bones.

Post-traumatic Osteomyelitis

This stubborn complication can be prevented by scrupulously applying the principles of primary wound treatment described in Chap. 3.11. It is not an acute osteomyelitis: its signs are delay of wound healing, discharge from the wound and non-union of the fracture. The cause is dead infected bone retained in the wound. Treatment consists of: radical re-debridement of the wound (repeated as necessary) including the excision of all dead bone; stabilising the bony elements, ideally by external fixation; leaving the wound open to drain freely; giving antibiotics according to the sensitivity of cultures for at least 3 months (for the first 6 weeks intravenously). Finally, once the wound is "clean" clinically (even though cultures may still be positive), cancellous bone grafting around the non-union or into the gap of the bone stock loss is beneficial to the healing of the infection as well as promoting bony union. Cancellous bone grafting should be repeated every 6 weeks as required.

Further Reading

1. Uhthoff H (Ed) (1982) Current concepts of external fixation of fractures. Springer, Berlin Heidelberg New York
2. Hierholzer G, Rüedi T, Allgöwer M, Schatzker J (1985) Manual of the AO/ASIF tubular external fixator. Springer, Berlin Heidelberg New York

3.13 Amputations

N. D. Reis

Introduction

One of the most important and problematic aspects of limb injury is the decision-making process which leads to amputation: surgeons will save the patient long-term suffering and morbidity if they face up to their duty unflinchingly in the timely recognition of the need to amputate primarily or for the failure of treatment when it occurs, thereby avoiding heroic but useless surgery to save a doomed limb.

Traumatic Causes of Limb Severence and Destruction. Direct immediate limb amputation may be caused by blast, crushing, avulsion, or mutilation by missiles, charring, frostbite. Limb artery occlusion by any one of the aforementioned agents will present a dead limb with or without a clearcut demarkation.

On Site and Medical Aid Station

Management of an acute traumatic severance includes the urgent arrest of bleeding and the treatment of hypovolemic shock. A total transection of the major limb artery may bleed only a little due to retraction and spasm, whereas a mutilation and incomplete severance of the limb may bleed catastrophically due to an incomplete laceration of the limb artery and/or vein which prevents retraction, whilst spasm is ineffective.

When bleeding is slight, a firm bandage is applied to cover the stump. However, the stump may suddenly begin to bleed as a result of a rise in blood pressure in a shocked casualty after resuscitation, or due to the abolition of vasospasm or the loosening of a clot. Hence there is a need for close inspection and measurement of vital signs every quarter hour. A lightly wounded comrade is best designated for this purpose.

If bandaging does not control the bleeding, digital pressure is applied in the groin for the leg and on the upper humerus in the armpit for the upper limb. This pressure point method requires one person to be concerned entirely and only with this single function.

Failure to control bleeding by local pressure, the absence of a suitable therapist, or the need to evacuate or transport the casualty are indications to apply a tourniquet. Once again its efficacy should be checked every quarter hour and released once every hour: if brisk bleeding ensues, the tourniquet is immediately reapplied; if there is no bleeding or only some oozing, the stump is bandaged and closely watched as previously mentioned.

Hospital Management

A casualty presenting with a *totally severed limb,* after reassessment of his general condition and associated injuries, is taken to the operating theatre for radical stump wound debridement and hemostasis. The stump is left open and a definitive formal reamputation is performed at a delayed primary or secondary stage when the wound is perfectly clean.

The indications for performing a *primary amputation* are: when there is no hope of achieving a functioning limb due to irreparable damage to the vessels and/or nerves of the limb; extensive destruction of bone, joint, and muscle; loss of critical skin such as the sole of the foot and heel pad; life-threatening gas gangrene or necrotizing fasciitis; uncontrollable hemorrhage; and extrication. There are no absolute parameters, and the judgement of the surgeon based on a meticulous examination and his personal experience are decisive. The amputation stump is always left open for revision as required and for delayed or late elective reamputation.

Delayed primary or *secondary amputation* is necessary if: an adequate blood supply to the limb has not been realized; life-threatening, uncontrollable, generalized sepsis (including gas gangrene) sets in; life-threatening secondary hemorrhage starts; the extent of tissue loss and damage was greater than first realized; amputation and limb fitting will give a better functional result, thus making limb salvage untenable. The amputation performed is definitive except if done for sepsis, when it is left open as for a primary amputation.

Late amputation is required when: vessel and nerve repairs have failed to give adequate result; chronic sepsis is present, and the treatment to eradicate it has failed; an acceptable functional result is no longer possible, and/or the general condition of the patient is deteriorating, or there is evidence of amyloid disease; the functional result of limb salvage is unacceptable. The amputation is elective and definitive.

Surgical Techniques

Primary amputation for all indications is done by simple excisional or circular techniques. The level is the most distal level at which the amputation can be performed through healthy tissues. The stump is left open to drain. Limbs destroyed by explosions and high velocity missiles are often extensively damaged proximal to the site of obvious tissue mutilation. Repeated debridement or more proximal reamputation may be required in the ensuing days. Amputation for gas gangrene or other sepsis must be proximal to the invasive "front" of the infection.

Excisional Technique

This is the simplest and best technique for amputations and incomplete amputations caused by explosions and missile mutilation. The amputation is treated as any other wound: excision of all nonviable tissues is undertaken methodically commencing with the skin and subcutaneous tissue, then the fascia and the muscle (the latter most meticuously and radically), and finally excess protruding bone is sawn off and/or the shattered bone fragments are excised. Often an incomplete amuptation will require the severance of the remaining tissues in continuity with the dead and/or destroyed distal part. However, bone fragments with soft tissue attachments required for stump length are retained. As much skin as possible is retained. No attention is paid to ideal amputation levels or optimal skin flaps. The principle governing this method is maximal viable tissue preservation.

Circular Technique

This is best for amputations for sepsis or closed limb death. The skin, muscle, and vessels are cut circumferentially at the same level. The vessels are double tied as they are encountered. Nerves are pulled gently taut distally and cut proximally to ensure deep proximal retraction. The bone is cut 2 cm shorter than the soft tissues (just enough to avoid its projection from the end of the stump).

Stump Dressing and Management

A bulky, loose, absorbtive dressing is lightly bandaged onto the open stump (figure-of-8 elastic stump bandage) and retained in place by a stockinette or net stocking which is stuck down proximally by painting the skin with an adhesive glue.

Compressive dressing at this stage is contraindicated since it is painful and reduces the already tenuous blood supply to the stump tissues. Traction on the skin via the adherent stocking is likewise contraindicated for the same reasons.

The dressings are changed daily under morphine analgesia. The stump wound is inspected. If necrotic tissue has been retained, repeat excision is performed under general, spinal, or regional anesthesia, as often as is necessary to achieve a completely clean, living stump.

Joint contractures (especially flexion at the hip and knee) are avoided by proper positioning and vigorous physiotherapy.

Stump Wound Closure and Definitive Amputation

There is *no need for hurry:* The stump should be closed when the wound is clean, there is little or no discharge, the swelling has gone down, and the skin edges are no longer edematous and look healthy. Premature closure leads to delayed healing, infection, and often loss of length because of the need to revise, excise more skin, and shorten the bone. The inexperienced surgeon does not attempt a definite amputation at this stage. He simply performs stump wound closure with maximal preservation of limb length.

When in doubt, wait with stump closure. Much surgical experience and acumen is needed. The experienced surgeon performs the definite amputation at the time of closure, which will usually be beyond the delayed primary period. As much muscle bulk is excised as is necessary for skin approximation without tension at optimal limb length. In the leg the fibula can be totally excised if needed for skin closure over the tibial stump.

3.14 Peripheral Nerve Injuries

N. D. Reis

Diagnosis

In disaster or war with the likelihood of mass casualties and/or multi-trauma wounded, peripheral nerve injury must nevertheless be diagnosed, even though this will not be a high priority function. In the unconscious or drugged patient diagnosis may be very difficult, but as soon as the casualty is able to cooperate a rapid neurological examination is done and recorded starting at the medical aid station echelon.

Diagnosis *before primary wound surgery* is of great advantage to establish a clear-cut indication for primary nerve exploration. The examination consists of asking the casualty to gently move his limbs at all the joints in all directions as much as his wounds allow and rapidly check the limbs for loss of light touch.

The findings are interpreted in the light of a knowledge of peripheral nerve anatomy, the dermatomes, the myotomes (see chapter on spinal and spinal cord injury), and the site of penetrating or closed injuries. In the presence of an open wound a very detailed neurological examination of the limb is necessary.

Management

Closed Nerve Injury

The management at all echelons is conservative: splinting of the paralyzed parts, avoidance of pressure over anesthetic or hypoanesthetic skin. Gentle passive movement, if possible, to prevent joint stiffness.

Open Nerve Injuries

In all open wounds in which a nerve injury has been diagnosed, *exploration of the nerve at primary wound surgery is mandatory!* This is the only way to glean information for correct management and to determine the prognosis.

The presence of an intact or lightly contused nerve is noted: neurapraxia, or, if no recovery is evident within 3 weeks, axonotmesis can confidently be predicted and the appropriate waiting periods determined.
Crushed, shredded, or severed nerves are diagnosed as neurotmesis. No recovery can be expected without surgical intervention. These injuries when caused by missiles are always contaminated and the ends of the lacerated, shredded, or crushed nerve must be sliced serially proximally and distally until healthy fascicles pout out of the epineural envelope. The gaps to be overcome may be formidable and not amenable to nerve mobilization and joint positioning: cable grafts will then be needed unless the problem can be solved by *primary bone shortening*, enabling end-to-end suture without any tension. Primary bone shortening in an open fracture (or by an osteotomy when no fracture is present) combined with primary external (or, very rarely, internal) skeletal fixation will give a better result than cable grafting, especially in catastrophic (often multiple) proximal major nerve defects.

Timing of Peripheral Nerve Surgery

Primary Wound Surgery

If a nerve lesion has been diagnosed prior to surgery, then the appropriate nerve is explored to well proximal and distal to the wound. If no nerve injury has been diagnosed prior to primary wound surgery (or the patient is unconscious or uncooperative or there was no proper examination before anesthesia), major nerves in the proximity of the wound are routinely explored. If a neurotmesis is discovered then there are two possibilities. If the wound is clean and suitable for primary closure (e.g., clean-cut wound due to flying glass), a primary end-to-end epineural nerve suture is performed. The limb is immobilized in a position which avoids tension at the site of suture for 6 weeks. However, usually the wound is not clean and has to be excised radically: then the nerve ends are tethered down in situ and marked by a prominent stitch, or a crushed or shredded segment of nerve in continuity is similarly marked by a stitch.

Delayed Primary Wound Surgery

At this stage (5–10 days after wounding) wounds are closed if judged clean: delayed primary nerve suture should be performed on *all* cases of neurotmesis at this stage of wound closure. In a war or disaster situation definitive nerve repair is not delayed as is generally taught for contaminated and lacerated nerve wounds. The nerve ends are sliced serially proximally and distally until healthy fascicles pout out. The gap between the nerve ends is overcome by mobilizing the nerve proximally and distally gently with the finger (never by sharp dissection, which further damages the blood supply to the nerve), and then positioning the appropriate joints to allow approxi-

mation of the nerve ends without tension by stay sutures. A standard epineural suture is made and the limb is immobilized to maintain that position, ensuring absence of tension at the suture line for at least 6 weeks: after this the joints are gradually allowed to regain their full range of motion by the application of serial plasters (or use of hinge-lock splints) over the ensuing 8 weeks.

When a gap cannot be overcome by nerve mobilization and joint positioning two options remain. Nerve grafting by various techniques is usually impractical at this early stage and the results are likely to be even worse than the poor results achieved with secondary nerve grafting: a specialist in nerve microsurgery is required.

Delayed primary bone shortening should always be considered in severe multiple nerve injury, especially when proximal at the root of a limb. End-to-end nerve sutures are possible at the delayed primary stage, with results which are far superior to secondary cable grafts deposited in severely scarred tissue. If necessary, gradual limb relengthening can be commenced 6 weeks after nerve suture, using an external fixator method of skeletal stabilization (ideally, using the Ilizarov technique).

Secondary Wound Surgery

Secondary nerve repair can be undertaken not earlier than 3 weeks following complete wound healing for all those neurotmeses not repaired at delayed primary stage.

Late secondary revision of primary or delayed primary repairs may be necessary after some months if there are signs of axonal arrest at the repair site: no functional recovery, no progression of a Tinel's sign distally, but rather a maximal Tinel's sign at the "neuroma" of the anastomosis. A neurolysis in an area of scar tissue with rebedding of the nerve in healthy soft tissue may be indicated, or an excision of the "neuroma" and resuture, according to the local findings at exploration.

Part 4

Treatment of Wounds: Regional

4.1 Head Injuries

M. Feinsod

The percentage of head injuries in war is steadily increasing, and the victims' survival prospects are gloomy owing to the growing use of high velocity missiles.

Primary Treatment

On Site

Head injuries on the battlefield are usually seen within minutes by a medic. Brain protection measures can be initiated even before transfer to the battalion aid station or its equivalents:

1. *The airway* — In stuporous and comatose patients the mouth is cleared by digital inspection of any obstructive material, and an oropharyngeal airway is inserted.
2. *Volume resuscitation* is to be started as soon as possible by an intravenous peripheral line, at a rate of 120 ml/h or as the patient's hemodynamic condition requires. Isotonic Ringer's lactate is the fluid of choice. Hypotonic solutions should be avoided.
3. *Position of patient* — The head is positioned in a 30° head-up position, without neck flexion. For stuporous or comatose patients the position of

Fig. 1. The stable side position for stuporous or comatose patients

choice is the stable side position (Fig. 1). In this position the airway is kept open by head and neck extension: the tongue does not fall back, and vomit is not aspirated.

4. *Dressing* — For open head wounds a sterile dressing is applied. Extruding brain tissue is covered, if possible by wet gauze in order to prevent adherence of brain tissue and further bleeding when the dressing is removed.

Medical Aid Station

The goal of treatment is to prevent secondary damage and restore optimal physiologic conditions. Patients are treated according to the severity of their neurological condition (this is not the place for a detailed neurological examination: a rough estimate of the state of consciousness suffices). Dramatic changes in the neurologic symptomatology can be seen after proper resuscitation (evaluation and triage should be carried out only later):

1. The *airway* of all comatose and stuporous patients is secured, preferably by endotracheal intubation. The endotracheal tube protects against aspiration, or airway obstruction by head and neck position, and allows oxygenation by mechanical ventilation. After intubation a nasogastric tube should be inserted for proper drainage, lowering of intra-abdominal pressure, and prevention of aspiration.
   Intubation is spared in unconscious patients whose respiration is regular, effective, and nonlaborious, and if the response to pain is purposeful or the level of consciousness is increasing. These patients are watched closely.
2. *Hyperventilation* — It is wise to assume that a comatose patient is suffering from increased intracranial pressure (ICP): hyperventilation is therefore employed to reduce pCO_2 to 25–30 torr. Between 16 and 20 respirations per min with the AMBU device will produce this result until the patient is connected to mechanical respiration.
   Hyperventilation ranks first in effectiveness and speed in lowering ICP.
3. *Associated injury* — The casualty is now evaluated for other injuries, especially for any problem that hampers proper respiration and oxygenation, such as hemopneumothorax.
4. *Elevating the head* (without neck flexion) to 30°–40° reduces intracranial pressure and prevents intracranial venous engorgement.
5. *Shock* — Ensure volume resuscitation. Shock is treated vigorously and should never be underestimated. The prognosis of patients with a head injury in a severe neurological condition and partially treated hypovolemic shock is worse than for those in the same neurological condition but normotensive.

The concept of "central" neurogenic shock should be abandoned, and all shock is to be regarded as hypovolemic and treated accordingly. Large scalp wounds and lacerations may cause up to 1000 ml blood loss. Bleeding from injury to the dural sinuses can be profuse (20% of the cardiac output is directed to the brain and most of it is drained by the dural sinuses) and require large amounts of blood to achieve blood volume stabilization.

6. *Stabilization before evacuation* — Rapid evacuation of neurosurgical patients to the hospital is important, but not until the proper establishment of the airway and volume replacement have been carried out. "Scooping and running" with poorly ventilated and hypovolemic patients with head injuries is disastrous.
7. *Intracranial pressure* — If after the above measures have been taken there is a further deterioration in the patient's neurological condition, it must be assumed that the ICP is on the rise and an IV bolus of 200–300 ml of 20% mannitol over a 15–20 min period should be given. A second dose is administered after 6 h, if the condition has not improved. Furosemide may be added in an attempt to reduce ICP, but its use in the acute stage in addition to hyperventilation and mannitol seems to be superfluous.

   Dexamethazone and other steroids were found by most studies to be ineffective in the traumatic situation for controlling ICP, reducing cerebral edema, or influencing long-term results. Present knowledge negates the use of steroids.
8. *Convulsions* — Diazepam 10 mg IV will stop most acute epileptic seizures. If possible, start prophylactic anticonvulsants: 250 mg of phenytoin is given slowly IV or as a single IM injection. Do not give barbiturates at the battalion level to control ICP or to overcome epileptic seizures: they may cause a fall in the blood pressure.
9. *Restlessness* — The lightly stuporous but restless patient may create a difficult situation. Resist the temptation to sedate the patient immediately by promethazine, chlorpromazine, and especially diazepam or morphine. Restlessness of the unconscious patient is due to anoxia, pain from a broken rib or limb, or even a distended bladder. Attention to these conditions will obviate the need for sedation. Morphine should be restricted to patients who are mechanically ventilated and thus protected from respiratory depression.
10. *Assessment of neurologic state* — The patient's neurologic condition is evaluated and recorded only after resuscitation is completed. The Glasgow Coma Scale (Appendix 1) serves as a basis for repeated examinations of the level of consciousness. The respiratory rate, pulse, blood pressure, papillary abnormalities, appearance of hemiplegia, or anisocoria are recorded in addition every half hour to aid the early recognition of the development of space-occupying collections of blood and brain stem herniation.

Evacuation priority to central hospitals varies according to the severity of the neurological condition (unless the patient is in a state of brain death, that is: flaccid paralysis; areflexia; wide, dilated, unresponsive pupils; and no spontaneous respiration).
Blunt head injury is more prone to cause delayed, increased ICP (intracranial hematomas or edema) than penetrating head injury.

11. *Prophylactic antibiotic* treatment is started for all patients with open wounds: crystalline penicillin 5 million units IV 6 hourly, and later in hospital gentamicin 80 mg 8 hourly.

Treatment in Hospital

1. *Re-evaluation* — On arrival the patient is re-evaluated by checking all previously described steps. Even if the casualty seems to be suffering from a neurosurgical problem only, he must be re-evaluated for associated hidden injuries.
2. *Arterial line monitoring* — An arterial line is established and blood gases and electrolytes are monitored frequently. Pulse and blood pressure are monitored (by transducers and monitors, if available).
3. *Priorities in the multiply injured patient:*
   - Securing a patent airway and effective blood oxygenation
   - Hemostasis: volume replacement
   - Treatment of the specific injuries

   It is for the general surgeon to decide upon the priorities of treatment: laparotomy and craniotomy can be carried out simultaneously.

Diagnosis

1. Repeat evaluation of the patient's neurological state.
2. CT scan (if available) of the head should be performed in all patients with a head injury: it gives the fastest accurate diagnosis of cranial and intracranial acute traumatic lesions. This facilitates the surgical treatment of depressed skull fractures or penetrating injuries, and leads to the recognition of space-occupying lesions and to treatment before deterioration sets in; unnecessary surgical explorations are prevented. Neurosurgical patients should be evacuated to a hospital with CT facilities.
   Proper evaluation of the CT scan renders the plain skull X-ray superfluous: linear skull fractures are usually of no clinical significance, the treatment of fractures of the base of the skull is not dependent on precise radiological diagnosis, whilst depressed skull fractures are better demonstrated by CT.
3. Plain X-rays are to be done if no CT scanner is available. Having made an anatomical diagnosis the treatment policy can be planned.

Conservative Treatment

Patients who do not suffer from penetrating injuries and do not harbor a space-occupying lesion are treated conservatively. Patients who score 9 and more in the Glasgow Coma Scale (GCS) are followed up by repeated examinations: the best parameter is the level of consciousness as assessed by the GCS. These patients are seldom intubated and do not require special treatment for increased intracranial pressure (ICP).
If the patient's condition deteriorates, a CT scan is done as soon as possible; in the meantime, the patients are treated as if they have an increased ICP, i.e., a bolus of mannitol 20% IV and, if necessary, if coma is threatening or deepening, intubation and hyperventilation. This turn of events is most often due to poor respiratory care and ensuing hypoxia.
Patients who score 9 or less in the CGS should have an additional monitoring parameter, the ICP (see Appendix 2). The clinical evaluation in these patients is more difficult the less they score in the GCS: a change in the ICP reading may predict the development of a space-occupying lesion or brain edema well before a deterioration in the patient's condition is noticed. (In this context it is important to bear in mind that the hypoxic brain by itself may increase the ICP and that lesions in the lower regions of the temporal lobe may cause herniation without a rise in the ICP.)
In patients who have evidence of brain injury (hematomas, lacerations, and contusions), anticonvulsive treatment is continued or commenced by giving a loading dose of phenytoin 250 mg t.i.d. followed by phenytoin 300 mg/day.

Surgical Treatment

With penetrating craniocerebral injuries, a CT scan will reveal the extent of the injury much better than a plain skull X-ray. It will disclose all metal fragments and bone fragments that may be missed by the plain skull X-ray. Some of these fragments are driven into much deeper regions than can be reached by even aggressive surgery. Experience has shown that the rate of complications caused by these "left-behind" fragments is very low. In a follow-up period of 4 years, none of 175 patients developed a brain abscess or uncontrollable epilepsy. The amount of brain tissue lost in their surgical retrieval will greatly increase the neurological deficit. The same is true for relatively superficial bone fragments. Therefore, only the most superficial bone and metal fragments are removed, in order to preserve as much brain tissue as possible.

1. Shave the scalp and paint it with povidone-iodine.
2. Minimally debride skin and soft tissues (the blood supply to the scalp is very rich, and the risk of wound infection is very low). This conservative debridement reduces the need for skin flaps or grafts.

3. The cut is extended to expose the bone for 2–3 cm around the wound edges.
4. A burr hole is drilled into stable bone close to the bone wound.
5. A circumferential craniectomy is performed until normal dura is exposed for 1–2 cm around the dura wound.
6. The dura wound is carefully enlarged and extruding brain tissue, hair, blood clots, and metal and bone fragments are removed.
7. Brain tissue is carefully debrided and any foreign material is removed. Thorough irrigation of the wound track will flush out all significant hematomas, devitalized brain tissues and free fragments.
8. Hemostasis is usually easy and best done by gentle pressure through cotton pledgets and bipolar coagulation.
9. The dura is closed usually by the use of a graft obtained from the temporal muscle fascia, or pericranium. The scalp is closed in two layers.
10. If the patient's preoperative GCS was 9 or less, or if the brain injury is extensive, it is recommended to drill a burr hole several centimeters away from the wound and to insert a subdural, thin catheter for ICP monitoring.

Nonpenetrating head injuries are best diagnosed and localized by CT, enabling the best placement of the craniotomy flap, and avoidance of multiple exploratory burr holes. If CT is not available the diagnostic burr holes should be placed over the hemisphere contralateral to the developing hemiplegia or ipsilateral to the unresponsive, dilated pupil. The first exploratory burr hole is placed over the temporal region and if negative is followed by frontal and parietal ones.
The acute lesions that may be found and their surgical treatment are summarized as follows:

Acute Subdural Hematoma. Following a fall, road accident, or assault, there is rapid development of unconsciousness and hemiplegia, and epileptic seizures are common. A decerebrate state may develop with pupillary abnormalities. Unless rapidly treated by craniotomy evacuation of hematoma and hemostasis of bleeding cortical vessels, malignant edema develops, and mortality may reach 80%.

Acute Epidural Hematoma. Following all kinds of blunt head injury, consciousness is lost with or without a lucid interval, and lateralizing signs develop as pressure on the hemisphere builds up. Herniation is evidenced by unequal pupils, decorticate and decerebrate rigidity, and respiratory disturbances. Mortality is much lower than in acute subdural hematoma because the pressure transmission is mitigated by the strong dura.
Operative treatment consists of craniotomy, evacuation of the hematoma, coagulation or ligature of the bleeding vessels, tacking the dura to the periosteum, and wound closure over epidural drains.

Acute Cerebral Contusion and Intracerebral Hematomas. The history and clinical findings may mimic those of acute sub- or epidural hematoma. Small or moderate contusions do not require surgical treatment. An initial trial of medical therapy, in some cases assisted by ICP monitoring, should be attempted. In patients with a marked mass effect and ventricular compression, craniotomy is performed, and the injured tissue is resected.
Cerebral contusions are hemorrhagic in nature and may form secondary hematomas. They should be evacuated if they produce a mass effect, cause a high ICP not controlled by medication, or lead to a progressive neurological deficit.

Postoperative Care

1. Respiratory care is (as in the initial phases of the treatment) one of the most important aspects of treatment. Hyperventilation is continued until the patient's condition stabilizes and the ICP does not rise with cessation of assisted respiration. Repeated examinations of the arterial blood gases monitor the efficacy of respiration. The decision to extubate the patient is based on an assessment of the improvement in his state of consciousness.
2. The fluid and electrolyte balances are ensured by the IV route and later by mouth. There is no need to dehydrate the patient: an adequate blood volume for proper perfusion of vital tissues is paramount.
3. Feeding is started as soon as possible. Calorific requirements are high, and protein and calorie depletion will promote infections. Twenty-four hours after surgery clear fluids are given through the nasogastric tube and if absorbed, nutrient solutions are gradually added.
4. Penicillin and gentamicin are continued IV for at least 7 days. Then, if there is no fever or signs of wound infection, the treatment is discontinued. Cultures of blood, CSF, sputum, and urine are taken, and the treatment is adjusted accordingly.
5. In every case in which evidence of brain tissue injury exists, anticonvulsive treatment is continued for several months: 300 mg phenytoin daily are given by mouth, through a nasogastric tube, or parenterally.

Appendix 1: The Glasgow coma scale

Verbal response	Oriented	5
	Confused conversation	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Best motor response	Obeys	6
	Localizes	5
	Withdrawal	4
	Abnormal flexion	3
	Extension	2
	None	1
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1

Appendix 2: Technique for monitoring intracranial pressure

The intracranial pressure (ICP) can be measured from any location. The preferred one, however, is through a right frontal burr hole. If the skull has been opened in a surgical procedure it is possible to use that skull defect, but a separate burr hole will reduce the chances of infection.

The chosen area is shaved and cleansed. A 3–4 cm skin incision reaching the periost is done, and the wound edges are separated with an automatic retractor. A burr hole is drilled and the dura exposed. A 3–4 cm cruciate incision of the dura enables the introduction of a thin polyethylene feeding tube (5 Fr) into the subdural space for a length of 4–5 cm. The catheter can emerge through the skin or better at a distance through a subcutaneous tunnel. The catheter should be properly secured. It has to be connected to a pressure transducer and a monitor; when these are not available, a connection to a simple water manometer will provide all the necessary information.

As a rule, ICP above 20 mmHg (280 mmH_2O) should treated by mannitol or hyperventilation. When ICP is on the rise, treatment should start even earlier.

4.2 Spinal Injuries

L. Z. Shifrin

Part I: Emergency Care

General Principles

- Preservation of life takes precedence over treatment of spinal injuries.
- Every casualty should be suspected of having a spinal injury until proven otherwise.
- The purpose of emergency care, after life life-saving procedures, is to prevent neurologic damage or deterioration.
- Accurate medical record documentation is essential to insure that correct transportation means are used and to facilitate decision-making at all echelons of medical care.
- Definitive care should be delayed until the patient arrives at a medical facility with qualified specialists and ancillary support.

Preliminary Classification of Spinal Injuries

Path of injury

Closed: blunt trauma, blast, deceleration
Open: missiles by direct contact, bone fragments, or shock wave

Location of spinal column injury

Describe according to anatomical regions: cervical, cervicothoracic, thoracic, thoracolumbar, lumbar, lumbosacral, sacral

Neurologic injury description

Spinal cord: complete, incomplete (quadri- or paraplegia; quadri- or paraparesis)
Cauda equina: complete, incomplete (paraplegia, paraparesis)
Nerve root: complete, incomplete

Example of diagnosis that might be recorded by the first physician to examine the victim: "Suspect closed cervical spine and cord injury, quadriparesis"; "Open missile thoracic spine and cord injury, paraplegia."

Note: A detailed diagnosis is not needed or, indeed, possible to make under "field" conditions. But this general description will be sufficient for triage and transportation handling.

On-Site Rescue of the Spinal-Injured Patient

The patient should be immediately extracted from the site of injury if the location is dangerous, such as on the battlefield or in a collapsing or burning building. A stretcher, door, large flat board, etc. should be brought to the victim.
While moving the victim, keep the axis of the head and spine in a straight line.
Two people can slide or roll the patient onto the stretcher, one supporting the head in line with the spine. Three or four people can lift the patient, with one supporting the head.
Securely strap patient to the stretcher and convey to the casualty station.

Clinical Assessment at Medical Aid Station

Immediately establish whether there is need for cardiopulmonary resuscitation or control of hemorrhage. Look for external evidence of spine injury: torticollis, tenderness or swelling over spinous processes, gibbus, signs of contusion, penetrating wound. To inspect the back, turn patient on to one side while maintaing a straight axis of the spine.
Conscious patient:

- Inquire whether there is neck or back pain, sensory or motor disturbance.
- Test voluntary motion of the extremities by examining motor groups. This is best done by asking the patient to flex and extend each joint individually, noting whether motion is absent or present.
- Test superficial pain sensation by dermatome distribution, preferably with a needle.
- Record highest level of motor and sensory deficit for each side of the trunk and extremities, and distal findings in incomplete lesions.
- Test anal sphincter tone, voluntary contraction, perianal sensation, bulbocavernosus reflex.

Unconscious Patient:

- Assume that the unconscious patient has a spinal injury until proven otherwise. Look for external signs of injury.

Emergency Treatment

Immobilization. Warn conscious patient not to allow passive or active motion of spine if injury is suspected. Secure patient to firm support. For cervical spine injuries there should be supports on either side of the head such as sand bags, infusion sacs, rolled-up cloths. Cervical collars and other orthoses should be applied only by trained medical personnel. Pad the hollows of the normal cervical and lumbar lordosis. Sedate agitated patient with narcotic or tranquilizer unless contraindicated by other injuries.

Respiratory Assistance. Respiratory insufficiency is common in high cervical cord injuries, and in low cervical cord ones with associated chest and abdominal injuries. Look for tachypnea, cyanosis, use of accessory breathing muscles. Clear the air passage of any obstruction. Insert oral airway and begin Ambu ventilation. Endotracheal intubation should be done with the cervical axis neutral at all times in cervical injuries and in unconscious patients. Motion should only by permitted at the temporomandibular articulation. The hollow of the cervical lordosis can be supported by padding. Tracheostomy may be necessary when facial injuries prevent intubation. A nasogastric tube is inserted to decompress the stomach, relieving pressure on the diaphragm, and to lessen the risk of aspiration.

Urinary Bladder. Catheterization is performed by sterile technique if the bladder is distended, as determined by palpation or percussion, or if large amounts of fluids are to be given. All spinal cord and cauda equina-injured patients are to be catheterized.

Penetrating Wounds of the Spine. Cover the wounds with a sterile dressing. Administer intravenous antibiotics (crystalline penicillin) and antitetanus prophylaxis.

Transportation (Evacuation) to Advanced Medical Facility. The victim can be transferred on a standard stretcher, special spine frame, door, or long board that provides firm support. For cervical injuries, place support on either side of the head. Special traction devices and orthoses should be applied only by trained medical personnel. Pad the hollows of the normal cervical and lumbar lordosis.

Skin Pressure Protection. Pad pressure points, particularly the elbow, sacrum, and heel. Give instructions to quarter-turn the patient every 2 h while maintaining a straight spinal axis. Remove all hard objects from under the patient. Splints for extremity fractures should be well padded. Avoid plaster casts.

Part II: Hospital Management

Evaluation

Documentation

On arrival of the patient, medical records should be rapidly reviewed. Additional information can be obtained from medical personnel accompanying the patient. Inquire from the patient or others about the time and mechanism of injury.

Examination

- Attention must be directed immediately to all other injuries that may be life-threatening.
- If the patient is conscious, inquire whether there was or is any numbness, "electric shock", or burning sensation in the extremities.
- Inquire whether or not weakness exists in the extremities; transient, static, progressive?
- Does the patient complain of pain along the spine?
- Inspect patient after removing clothes. Look for external signs of injury: torticollis, swelling and tenderness over spinous processes, gibbus, penetrating wounds, abrasions, and contusions.
- Note that lack of pain does not exclude spinal injury in patients with multiple injuries, in shock, or after narcotic administration.

Neurologic Examination

The paralysis from a cord injury is characterized by myotome distribution, so that joint motion rather than individual muscles should be tested. The opposite is true for cauda equina and nerve root injuries. Record the highest level of motor and sensory deficit on both sides of the trunk and extremities, and distal distribution in incomplete lesions.

Grading of motor strength:

I No active motion
II Trace of motion
III Active motion against gravity but not against resistance
IV Motion against resistance, but weak
V Normal strength

Sensory grading:

Absent
Altered (diminished or hypoesthesia, hypersensitive, or hyperesthesia)
Normal

Movement and segment of cord:
- Shoulder abduction and lateral rotation C5
- Elbow flexion C5, C6
- Elbow extension C7, C8
- Forearm supination and pronation C6, C7
- Wrist flexion and extension C6, C7
- Finger muscles, long C7, C8
- Finger intrinsics C8, T1
- Hip flexors L2, L3
- Hip adductors L2, L3
- Knee extensors L3, L4
- Ankle extensor L4, L5
- Ankle flexors L5, S1
- Anal sphincter S2, S3, S4

Dermatome cord segment:
- Anterolateral shoulder C5
- Thumb C6
- Middle finger C7
- Little finger C8
- Medial arm T1
- 3rd, 4th interspace T3
- Nipple line T5
- Xyphoid T6
- Navel (umbilicus) T10
- Pubis T12
- Inguinal L1
- Medial thigh L2
- Medial knee L3
- Medial calf to great toe L4
- Dorsum of foot L5
- Lateral foot S1
- Posteromedial thigh and calf S2
- Perianal S2, S3, S4

Peripheral reflexes cord segment:
- Biceps C5, C6
- Radial C5, C6
- Triceps C7, C8
- Superficial abdominal T6–T12
- Cremasteric L1, L2
- Knee L3, L4
- Ankle S1, S2
- Bulbocavernosus S3, S4
- Anal ("wink, blink") S3, S4

Neurologic Syndromes

Central Cord Syndrome. Damage to the cervical cord that results in greater weakness of the upper extremities than the lower, sacral sparing.

Anterior Cord Syndrome. Motor, pain, and temperature sensation lost in the extremities, light touch, position, and vibration proprioception intact.

Brown-Sequard Cord Syndrome. Loss of motor function, light touch, and position sense on one side (ipsilateral), loss of pain and temperature sense on the opposite side (contralateral).

Dorsal Column Cord Syndrome. Position and vibratory sense lost, motor, pain, and temperature intact.

Mixed or Unclassifiable Cord Injuries
Conus Medullaris Syndrome. Isolated lesion at first lumbar vertebra level. Disturbances in spincters, micturition, sacral anesthesia. Bulbocavernosus and anal reflexes absent. Extremities normal, no pyramidal signs.

Cauda Equina Syndrome. Lesion below L1 vertebral level. Flaccid paralysis of legs with sensory loss, saddle (perianal) anesthesia, sphincter paralysis, absent bulbocavernosus and anal reflexes.

Mixed Conus and Cauda Lesions
Spinal Cord Shock Syndrome. Immediately after sudden and severe spinal cord injury, there is a temporary cessation of all cord neuronal activity, a state that is called spinal shock. During this state there is flaccid paralysis below the lesion, sensory loss, absent sphincters, bulbocavernosus and anal reflexes, and it is not possible at this time to determine whether there is an incomplete or complete cord lesion. Spinal shock rarely lasts more than 24 h and is heralded by the return of the bulbocavernosus and anal reflexes (except in a conus lesion). If only saddle (perianal, sacral sparing) sensation appears, then there is still hope of functional motor recovery.
It is important to remember that, especially during the period of spinal shock, quadriplegics and high paraplegics may not show the normal physiologic response to a hypovolemic state. Tachycardia may be masked by the bradycardia effect of spinal shock. A fall in blood pressure can be due to spinal shock, hypovolemia, or both. The classic signs of peritoneal irritation in intra-abdominal bleeding (guarding, rebound) are not present because there is lack of sensation and muscle tone. Peritoneal lavage (or CT) should be done to exclude abdominal injury when there is hypotension, when the mechanism of injury is compatible with abdominal injury, and there are external signs of abdominal trauma. Remember, also, that extremity injuries can be easily overlooked because of the lack of pain response during examination.

Radiologic Examination

Indications for radiographs:

- Neurologic lesion
- Painful spinal column
- Head and facial injuries may have associated cervical spine injuries
- Unconscious patient. Specifically a cervical spine radiograph in case intubation is needed for resuscitation or anesthesia
- Blast and severe blunt trauma victims should have their entire spine radiographed
- Penetrating injuries

Technique

The patient is kept supine, head and spine in neutral axis. Supervision by a physician or trained medical personnel is desirable. Biplane anteroposterior and lateral views of the spine are the minimum needed for diagnosis.

Cervical Spine. Anteroposterior and cross-table views are routine. Additional views:

- AP of atlantoaxial (C1-C2) by open mouth or dens view in conscious patient
- If there is torticollis, an additional lateral radiograph is taken perpendicular to the skull to see C1-C2 better
- It is essential that the entire spine up to the C7-T1 interval be seen, which may be difficult in short-necked individuals. Gentle traction on the arms to depress the shoulders may allow sight of the lower cervical area. If not, employ the "swimmer's view"
- Right and left oblique projections for seeing the pedicles, facets, and neural foramina in more detail
- Right and left pillar views for facets
- Lateral view in flexion and extension to demonstrate instability. This can be done only in a fully conscious patient with no neurologic deficit and normal AP, open mouth, and oblique projections.

Thoracic Spine. Anteroposterior and lateral views are routine. Use "swimmer's view" to see the cervicothoracic junction.

Lumbar Spine. Anteroposterior and lateral views are routine. Right and left obliques allow better visualization of the facets, pars interarticularis.

Special Radiographic Studies

Plane tomography in the anteroposterior view may be useful in studying the atlantoaxial junction and os dens. Lateral tomography can demonstrate narrowing of the canal by bony fragments.

It is often opportune to examine the spine by CT when other areas of the body must be studied (chest, brain, abdomen). This is particularly true when the condition of the patient will not allow additional radiographs. CT is helpful in studying the cervicothoracic region when conventional methods fail. Missile injuries with neurologic damage may not leave a clearly seen vertebral lesion on radiograph. CT will demonstrate whether the path of the missile crosses the vertebral column. CT will show whether there is compromise of the spinal canal by bone fragments and the position of foreign bodies.
If surgery of the spine is contemplated for unstable fractures, CT is useful in determining the extent of bony injury both anteriorly and posteriorly, which is important in planning the operation.

Myelography. When a neurologic lesion exists in the absence of a radiographically proven spine injury, or if there is no correlation between the neurologic level and the radiographic findings, myelography is indicated.

Orthopedic Management

General Principles and Goals of Treatment

- Prevent neurologic injury or its worsening, and establish optimum conditions for neurologic recovery
- Restore normal anatomic relationships of skeletal and neural elements and obtain spine stability
- Create conditions that will facilitate rehabilitation and the best possible functional result
- In multiply injured patients, treatment must be based on methods that will facilitate the care of other injuries and the patient in toto and not just the spinal lesion.

Open Wounds

If the entry wound is remote from the spine, local debridement is all that is necessary, followed by systemic antibiotics and delayed closure of the wound after a few days. This course of treatment is to be pursued even if there are bullet or shrapnel fragments in the spinal canal. Attempts to remove them will not improve the neurologic status and may be complicated by meningitis and cerebrospinal fluid fistulae.
Direct entry wounds that expose skeletal or neural elements require extensive and meticulous debridement. This procedure may be delayed for up to 24 h if transfer to a surgical spine center is possible. All devitalized tissue, loose bone fragments, and accessible foreign bodies are removed while carefully decompressing the cord. The dura should be closed by fascia lata or local fascial graft. Soft tissue coverage of the cord is necessary and may require the use of local muscle flaps. Isolate the spine from contamination

by intestinal and other injuries and make sure that the retroperitoneal and peritoneal spaces are drained adequately.

Treatment of Injuries to the Vertebral Column

Methods

In order to restore normal anatomic relationships, healing, and stability of the spine, a variety of methods are available. When dealing with multiple casualties, the simplest and most expedient methods should be employed initially. Later, when conditions are more favorable, sophisticated and complex methods can be initiated. Often, this requires transfer to a surgical spine injury center.

Nonoperative Treatment

In the majority of cases, nonoperative treatment is sufficient. The degree of mechanical instability of the spine and neurologic damage determines treatment. Spinal stability exists when, under normal physiologic loads, the spine retains its normal alignment without embarrassment of neural elements. Spinal instability is present if normal physiologic loads lead to loss of normal spinal alignment and compromise of the neural elements or disabling pain. Any vertebral column injury associated with neurologic signs is unstable until proven otherwise. Radiographic signs of instability are:

- Displacement of a vertebra in relationship to its neighbor
- Abnormal angulation between spinal segments
- 50% loss of vertebral body height
- Widening of interpedicular distance on the AP view
- Widening of the interspinous distance between two segments on AP or lateral views
- Intrusion of bone fragments into the spinal canal
- Fractures of pedicles, facets
- Dislocation of facets
- Fractures of the axis (dens)
- A distance greater than 3 mm in children and 4 mm in adults between the anterior arch of atlas and dens process of the axis on the lateral view
- Widening of the distance between the lateral masses of the atlas on the open mouth view
- Fracture of vertebra body and transverse process at the same level
- Fracture of thoracic spine in the presence of a sternal fracture
- Increased intervertebral distance
- Significant structural damage to both the anterior and posterior parts of the vertebra

Stable, Undisplaced Fractures Withouth Neurologic Damage

Cervical Spine. Cervical orthoses can be used with the cooperative patient to relieve pain, provide a modest degree of immobilization, serve as a visible warning to all that a spine injury exists, and allow the easy movement and transportation of the patient. A rigid plastic collar is best, but stockinette-covered firm felt or other material is also suitable. The collar is applied in such a way that slight extension is obtained. The patient must remain recumbent until a final decision is made on treatment. An orthosis (cervicothoracic, four-poster, Somi, Yale) for definite ambulatory treatment is the treatment of choice in most instances and should extend from the chest to occiput.

Halter (chin strap, Glisson) traction may be used in recumbent, cooperative patients. However, this requires traction equipment and interferes with moving or transporting the patient. About 2–3 kg is the maximum weight that will be tolerated. More than that causes pressure sores on the chin and pain at the temporomandibular joints. This is usually temporary treatment.

Cast immobilization should never be used if it is possible to treat with an orthosis and only in ambulatory patients. Minerva casts are heavy, impede nursing care, require constant turning of bed-ridden patients, and expertise in cast application. Radiographic control is more difficult through the cast.

Thoracic and Lumbar Spine. For many patients it is sufficient to treat by recumbency until acute pain has subsided, followed by ambulation without external support. Injuries around the thoracolumbar junction are most likely to benefit from immobilization by spinal orthoses that reduce painful motion. In the thoracic and thoracolumbar areas, a hyperextension or three-point type of brace is used. In the lumbosacral spine a chair-back type of brace is preferred.

Plaster body jackets may also provide sufficient support to allow early, pain-free ambulation with thoracolumbar fractures. Usually this is of the hyperextension variety and can best be applied on a thin patient. Whenever, possible, a spinal orthosis is preferable.

Unstable, Displaced Spine or Neurologic Injury

Cervical Spine. Skeletal skull traction is the preferred treatment when injury to the cervical spine is associated with neurologic deficit, spinal column instability, or malalignment. Apply skull traction under local anesthesia (skull calipers of the cone or Gardner-Wells type are preferable because of the simplicity of application). The hair in the area immediately above the ear is shaved or cut as short as possible and then cleansed with antiseptic solution. In a conscious patient local anesthesia is infiltrated from skin to the outer table of the skull. A 0.5-cm skin incision is made 1.0–1.5 cm above

the pinna in line with the external meatus and mastoid process. The pins are inserted according to the particular method used.
Traction is by a rope-pulley system with the line of pull in the long axis of the neck. The weights should be free-hanging with no danger of reaching the floor or being caught on the bed frame. The bed should have an anti-Trendelenburg slope of 10°–20° to provide countertraction. Check that there is a free length of rope between the caliper and the pulley to ensure continous traction.
The pin sites should be cleaned three times per day with H_2O_2 followed by antiseptic solution. The patient should be quarter-turned every 2 h. In most cases 6 weeks are required before sufficient stabilization is achieved to allow ambulation in an orthosis. The amount of traction weight to apply depends on the age and size of the patient and type of spinal injury. As a general rule (according to the level of injury): children, start with 1 kg for C1 and add an additional 0.5–1 kg for each level caudally; adults, start with 2 kg for C1 and add 1.5–2 kg for each additional level.
Immediately after the application of traction a lateral radiograph must be obtained. Check that there is sufficient traction, that there is no overdistraction, and whether alignment can be improved by changing the direction of pull.
When bilateral facet dislocation occurs, greater traction weight for reduction is required than usual, with continuous radiographic monitoring. Unilateral facet dislocation can sometimes be reduced by closed manipulation. Both of these procedures should be attempted only by an experienced spinal surgeon.
Halo skull traction is appropriate when it is anticipated that early ambulation in a halo-cast or vest is possible. Because of the risk of pressure sores, a cast is contraindicated when there is no trunk sensation, but the padded vest may be used if frequent examinations of the skin are made. The ambulatory halo should be used cautiously with lower level fractures, extreme instability, multiple level cervical fractures, and if less than 2 weeks has passed after closed reduction of dislocation or subluxation.

Thoracic and Lumbar. Prolonged recumbency is the definitive treatment until fracture healing and stability can be considered if the patient is young and otherwise healthy, or when concomitant problems (age, other medical illnesses) prevent consideration of surgical intervention. "Conservative" treatment can be successful in those institutions that provide skilled nursing care and are familiar with the methods of postural reduction of fractures.

Surgical Treatment

General Principles (Cervical, Thoracic, Lumbar). The objectives of any operative procedure are to relieve compression on the neural elements and to restore normal spinal alignment and long-term stability. Usually, immediate stability is required for optimum management of the patient.

Surgical procedures should be performed only by experienced spine surgeons. If none are available, treat nonoperatively until transfer to a spine center is feasible.

Indications:
- Progressive neurologic deficit and evidence of neural compression
- Failure to reduce by nonoperative means a dislocation, major displacement, and some subluxations or minor displacement associated with neurologic deficit.
- Unstable spine injury when early ambulation of the patient is necessary (multiply injured, uncooperative, elderly)

Methods:
- Either the anterior or posterior approach is utilized
- Restoration of normal spine alignment may be sufficient to decompress the canal and its contents.
- Bone or disc fragments causing compression may require removal and will influence the choice of surgical approach
- Internal fixation is used whenever possible to maintain reduction and provide immediate stability
- Supplemental autogenous bone graft to obtain bony fusion of the injured vertebral segments is employed in most instances

4.3 Spinal Cord Injury

M. E. Brooks and A. Ohry

Introduction

Until World War II, the prognosis in cases of severe spinal cord lesions was extremely poor, and the mortality rate was high. Statistics from the American Civil War, the Balkan Wars and World War I show a mortality rate of over 95%. World War II marked the turning point in the treatment of spinal cord injuries. The use of antibiotics greatly reduced the early mortality, and the introduction of multidisciplinary rehabilitation by Donald Munro, Sir Ludwig Guttmann, Howard Rusk and others in special cord injury centres reduced the mortality and morbidity due to complications of long-term recumbency and hospitalisation. The overall mortality for the American combat forces was 3.6% in the Korean and 2.3% in the Vietnamese conflict.

In this chapter we present guidelines for the treatment of spinal cord-injured patients as they apply to combat and civilian disasters. Most of the spinal cord injuries in combat are caused by projectile penetration with its accompanying shock wave greatly increasing the probability of additional organ injury. In contrast, the non-penetrating closed spinal cord injuries seen in the civilian population result in trauma to the vertebrae and associated ligamentous structure.

These injuries cause direct physical damage to the cord with partial or complete transection, or result in damage to neural structures through direct pressure, oedema, vascular compromise, catecholamine release, thecal injury or autoimmune response.

The spinal cord, an extension of the brain, has very little capacity for repair and none for regeneration. Damage is permanent. Spinal cord injuries can be divided into two groups: complete and incomplete. In the incomplete lesion, there remains some neurological function caudal to the level of injury, be it sacral sensation or weak volitional muscle movement in any muscle below the injury level. Approximately 85% of the initially complete spinal cord injuries remain complete. Those injuries which are incomplete at the time of trauma have the potential for full recovery. The term paraplegia refers to complete paralysis of the lower limbs or parts thereof with complete sensory loss, whereas the term quadriplegia or tetraplegia refers to complete paralysis of the legs in addition to complete paralysis of both up-

per extremities or parts thereof with attendant sensory loss. The recording of the spinal cord level with its vertebral number is that last distal level at which normal function exists (Fig 1).

On-Site Management

The most important considerations for first aid are: (a) the establishment of an airway (caution: Do not hyperextend the neck during airway insertion in

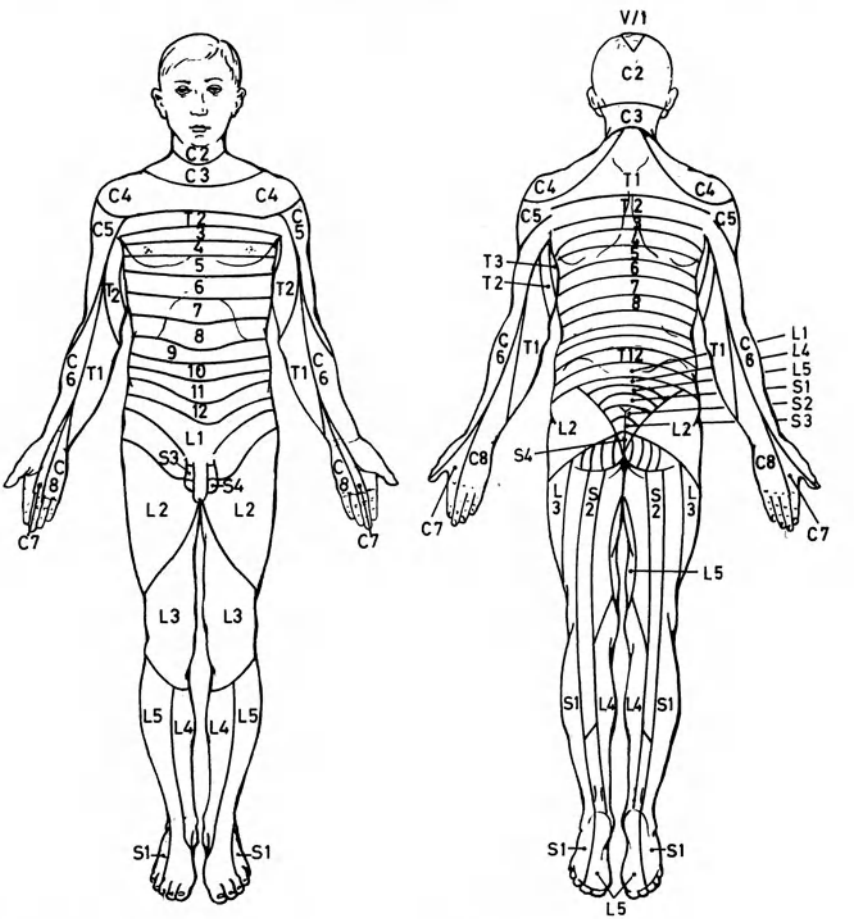

Fig. 1 Dermatomes corresponding to spinal cord levels

patients with cervical spine injury), (b) wound tamponade and (c) intravenous fluids.

In the combat situation one must constantly bear the possibility of spinal cord injury in mind. The on-site assessment of a spinal cord injury includes: (a) level of consciousness, (b) respiratory function, (c) bleeding, (d) circulatory and cardiac function, and (e) examination to establish the level and extent of the spinal cord injury.

The wounded patients are divided into conscious and unconscious groups. The conscious patient aids diagnosis by telling the examiner what he cannot move or feel thus suggesting the presence of a spinal cord injury. In the unconscious, semi-conscious, or hysterical patient, the diagnosis is more difficult. Here the examiner must rely on external signs of physical injury to the vertebral area, penetrating wounds, the posture of the patient when found, absence of reflex movements, and the consistency of the signs and symptoms. Whenever a suspicion of spinal injury exists, that patient must be assumed to be suffering from cord damage and treated accordingly until proven otherwise.

The initial treatment of the spinal cord-injured patient is based on the principles of stabilisation for transfer and prevention of further neurological loss. Complete and permanent "secondary spinal cord injuries" are caused by the improper initial management of an incomplete spinal cord injury at the time of the first or subsequent transfer. A basic knowledge of the anatomy of the spine and its neurological structures and the physiology of nervous tissue is assumed. The initial assessment must include a quick history of what happened, what moves, what does not move, what is felt and what is not felt. A brief neurological examination to confirm these findings will quickly differentiate the cervical from the thoracic and lumbar lesions. A lesion above the C4 level is not compatible with life unless ventilatory assistance is provided. At the C4 level intact spontaneous diaphragmatic respiration is present. Abduction of the shoulder is a C5 function, flexion of the elbow with or without the presence of weak wirst extension is mainly from the C6 level, extension of the elbow and flexion of the wrist and fingers requires an intact C7 level, opposition of the thumb is a function of C8, and finger abduction and abduction require T1 function.

The sensory level can be quickly assessed by examining the C5 dermatome at the area of the deltoid bursa, the C6 level on the dorsal aspect of the metacarpal joint of the thumb, the C7 level on the lateral surface (opposite the thumb) of the index finger, the C8 level on the medial surface (opposite the little finger) of the ring finger, and the T1 level at the base of the fifth metacarpal. The thoracic motor level corresponds to the sensory level and can be quickly estimated by examining sensation of the chest and abdomen *bilaterally* and remembering that the T5 level is the nipple line, the T10 level is the umbilicus, and the T12 level is the pubis. The inbetween levels are estimated in relation to the proximity of one of these landmark levels.

With each thoracic level, intercostal musculature aiding respiratory function is added. An adequate vital capacity accrues below the T8 level. The abdominal muscles begin to gain innervation at this level. allowing for the stabilisation of the abdominal wall, coughing and the performance of the Valsalva's manoeuvre. The area just below the clavicle is supplied by cervical segments which overlap the clavicle and not by dorsal segments. The lumbar levels control leg motions, with the L1 level innervating the hip flexor, the L2, 3, 4 levels the quadriceps (knee extension), the L4, 5 level the ankle dorsiflexors, and the S1, 2 levels the triceps surae (ankle plantar flexion). Sacral innervation is determined by the rectal examination of the tone and voluntary contraction of the anal sphincter, anal and perianal sensation, and the bulbocavernosus reflex, which is elicited by pulling or pinching the penis or the clitoris and noting the presence or absence of the anal sphincter reflex contraction on the examining finger. All tendon reflexes are examined to determine the level of tone in the muscles. The sensory level is delineated on the patient's skin with an indelible marker to enable the next examiner to determine whether any change has taken place. In the unconscious patient the examination is difficult and depends upon the absence or presence of tendon reflexes and paradoxical respiration (as a result of intercostal muscle paralysis).

Once a spinal cord injury has been diagnosed or is strongly suspected, the patient must be moved to a center for definitive care (e.g. vertebral dislocation reduction, surgery) as soon as possible, without adding to the neurologic damage already noted, since a delay of more than 4 h will render temporary reversible changes permanent and irreversible. In dire peril (fire, further enemy action, drowning, etc.) rapid transport by any means and under any circumstances to save the patient's life is called for regardless of the danger of causing further damage. However, if the situation allows for a controlled evacuation, then the basic principle is to avoid causing further neural damage.

The transfer onto the stretcher involves the following gentle and delicate steps, assuming that there is no other life-threatening wound which requires immediate and definitive treatment and that an adequate airway has been ensured. If patient is conscious, he is told not to move. Using at least three but better four or five people in unison, the patient is lifted without twisting and bending the spine (Fig.2). One supports the head and directs the team in moving the patient to a prepared stretcher, plank or door. All movements are to be as a team with the objective of moving along one axis at a time. If the injury is a cervical injury, a small cushion is placed under the neck (not under the head!) to maintain a position of extension, and sandbags should be placed alongside of the neck to prevent rotation. Prolonged lying on a hard surface causes pressure sores. No hard objects are placed on the patient, as in the absence of sensation they cause a pressure sore. The patient is covered to keep him warm.

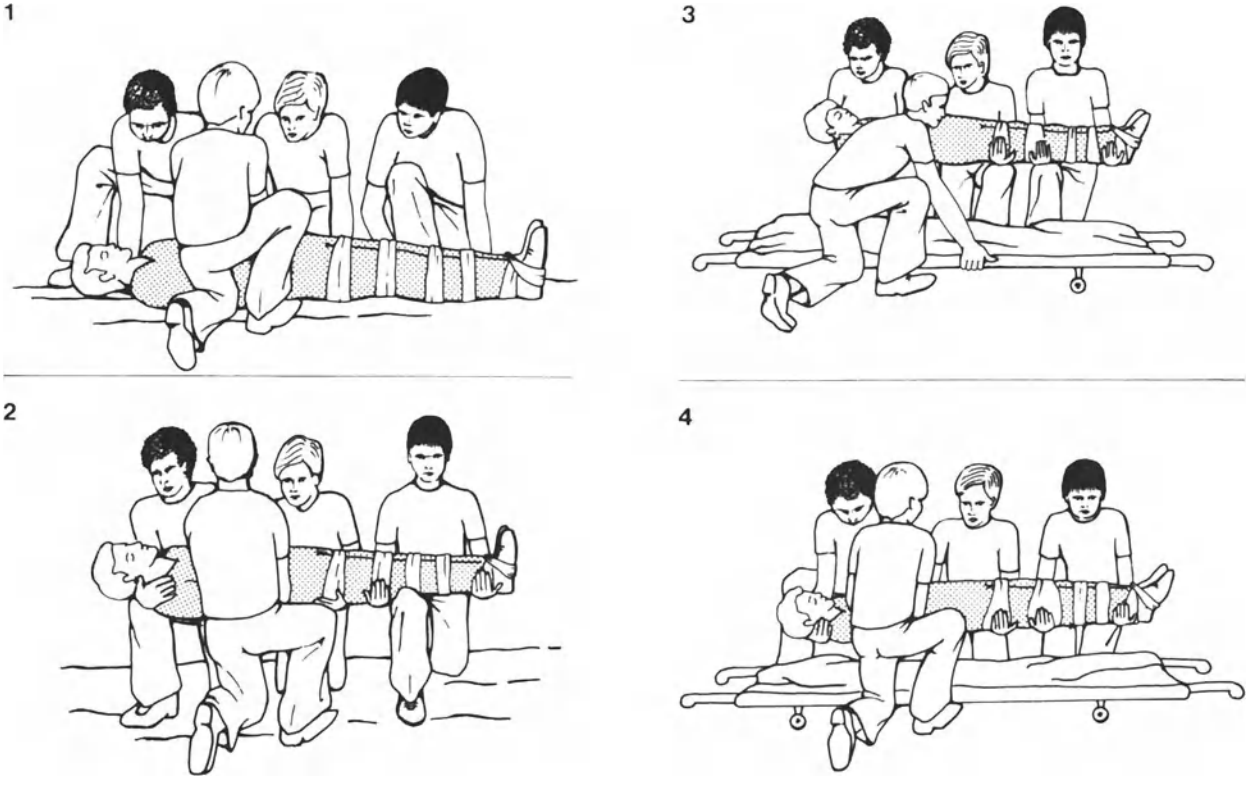

Fig. 2 Correct procedure for transferring a patient with a suspected spinal cord injury

First Aid Station

In the evacuation of the spinal cord-injured patient the fewer the number of transfers the less likely is further cord damage. If the first aid station is a terminal for air evacuation, then this is the first stop. However, if immediate on-site air evacuation is available, it is the preferred method, bypassing the first aid station. During evacuation and upon arrival at the first aid station, the following points must be remembered.

Spinal shock, the immediate cessation of all reflex activity in the spinal cord below the level of the injury, brings about reduction in the blood pressure. Intravenous fluids are administered using Hartmann's solution. Selective vasoconstriction and dilatation is abolished caudal to the spinal cord injury, thus predisposing the casualty to pulmonary oedema. If the drop in blood pressure does not correct itself within 2 h after elevating the foot of the stretcher, 100 mg metarminol is added to the Hartmann's solution, and the blood pressure is titrated to a level of 90–95 systolic. *Do not give morphine.* There is *no* need to catheterise the patient, unless the bladder is distended. In that case a catheter is introduced on a one-time basis to drain the bladder and is removed. There is no evidence that steroids in large doses prevent or diminish spinal cord damage; indeed, they may cause greater morbidity. Limb fractures are treated in the usual way, but casting of anaesthetic limbs must be avoided. Paralytic ileus with accompanying distention is a frequent immediate complication of spinal cord injury: a gastric tube should be inserted.

The Field and Base Hospital

Thoracic or abdominal wounds which require urgent surgery dictate evacuation to the nearest field hospital. In the absence of such concomitant injuries, the preferred evacuation route is direct to the nearest spinal cord injury centre. If this requires an air ambulance, then another transfer from the helicopter to the fixed-wing aircraft occurs. During all this time the spinal cord-injured patient, who has no sensation, is lying on a hard surface and may be developing pressure sores.

When treatment in a spinal cord injury centre is not available, management in hospital should be along the lines described for the spinal cord injury centre.

Spinal cord injury centre. A complete physical and neurological examination is performed. Routine laboratory examinations of blood and urine are carried out at the spinal cord injury centre. X-ray studies in addition to those of the spine include chest, pelvis and long bones if there is evidence

of further trauma. An electrocardiogram is also taken. Further investigation of the spinal cord injury involves X-rays and, if available, a CT scan or magnetic resonance imaging to determine the extent of the bony and soft tissue damage to the spine. The prognostic evaluation of the completeness of the neurological damage and assessment of the chances of recovery depend upon serial neurological examinations and the use of spinal and cerebral evoked potentials from peripheral nerves stimulated above and below the level of the spinal lesion.

Any open wound of the spine will require debridement and delayed primary closure. The decision to operate on the spinal injury is based only on the presence of *instability of the skeletal injury or on the worsening of the neurolocal deficit*. For instability alone there is no need for a decompression procedure, but with worsening there may be need for stabilisation as well as decompression. There is no difference in the recovery rate or in the final neurological state in those patients who have had exploratory laminectomies or any other therapy, excluding thoses cases in which a worsening neurological deficit was the indication for surgery. Postural reduction of any dislocation in the thoracic or lumber area can be accomplished by extension of the spine with pillows at the level of the dislocation.

Conservative treatment involves complete bed rest and turning the patient every 2 h. Proper limb positioning and cushioning is also important to prevent the formation of contractures and pressure sores. This is most easily accomplished in the Egerton bed (an electric tilting-turning bed) but can also be accomplished in an ordinary bed by devoted, trained nursing staff. If the skeletal injury is to the cervical spine, then skull traction using cone, Crutchfield or tongs equipment is indicated. In the presence of a dislocation, up to 30–40 kg of traction may be necessary to effect reduction. Without a dislocation, 7 kg of traction is sufficient to maintain the alignment of the cervical spine. A small sausage-shaped cushion is placed under the neck to maintain extension. After 7–10 days the traction is reduced to 4 kg. The patient with a thoracic or lumbar spine injury is also kept at bed rest and in hyperextension of the spine with a small pillow to prevent gibbus formation. Total bed rest is from 10 to 12 weeks with periodic X-ray studies to determine the presence of bone union and alignment. Where operative stabilisation has been performed, bed rest may be shortened to 6–8 weeks. In the presence of a total cord lesion, where a stable thoracic fracture is the only bony injury or no fracture at all is present, the period of bed rest can be further shortened to 10–21 days.

All patients are given prophylactic anticoagulant therapy (5000 units of heparin subcutaneously, twice daily) until mobilisation begins, unless contraindications exist connected with the initial wound. For thrombophlebitis, 15000 units are given intravenously in two divided doses for 8 weeks to maintain the partial prothrombin time at double the normal level. Once this is achieved, the patient is converted to Coumadin maintenance at sufficient dosage to keep the level of the prothrombin time at 30% for 3 months in the

case of a single episode. For recurring episodes, the total anticoagulant time is 6 months. Cimetidine 200 mg–1 daily, is given for 8–10 weeks to prevent the occurrence of stress ulcers.

Management of the urinary bladder is by sterile intermittent catheterisation using the no-touch technique. Patients are catheterised every 6 h (more frequently if necessary) with the number of catheterisations decreasing according to the residual urine: with a residual less than 300 ml the patient is catheterised twice daily; with a residual urine less than 100 ml, once daily; and with a residual less than 40 ml, catheterisations are halted. Patients are given vitamin C, 1 g twice daily, and hippuric acid, 1 g twice daily, to acidify the urine. Urine cultures are obtained at weekly intervals, and infections are treated with the appropriate antibiotics. Oral fluid intake is not restricted, and the patient is encouraged to drink at least 3.5 l daily.

Bowel movements are regulated with laxatives and suppositories every other day. Enemas are discouraged except as a one time procedure to empty a non-responsive bowel or to prepare the patient for a special examination such as pyelography. A well-balanced high protein diet is encouraged.

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4.4 Maxillofacial Wounds

D. Laufer and Z. Tulchinsky

Introduction

Facial injuries due to modern warfare are frequent. The final aim of treatment is to restore shape and function. Unless associated with skull fractures, even severe injuries to the bones of the face are not life threatening. The first consideration should not be the facial bone injury, but the patient's general condition. Patients who have suffered extensive maxillofacial injuries will require long-term hospitalization in a specialized department of oral and maxillofacial surgery for reconstructive surgery of soft and hard tissues.

Treatment on Site and at Field Medical Aid Station

Immediate treatment should consist in the diagnosis and management of airway obstruction, shock, and hemorrhage, both locally and of associated life-threatening wounds.

Prevention and Treatment of Airway Obstruction

The causes of airway obstruction are:

- Clot, vomit, saliva, fragments of bone, teeth, and dentures
- Aspiration of any of above
- Collapse of the tongue backwards against the posterior pharyngeal wall
- Occlusion of the oronasopharynx by the downward and backward displacement of the fractured maxilla
- Edema of the base of the tongue, oropharynx, and larynx

In order to remedy such conditions without delay, proceed as follows:

1. Remove all visible foreign bodies from the mouth and nasopharynx (if possible, apply suction)

2. Place the patient in the semiprone or face-down position so that blood and vomit can flow out
3. Insert an oral or nasal airway especially in the unconsious patient, or
4. Perform endotracheal intubation, which is the most effective way of clearing and preserving the airway, especially in patients with massive destruction of the lower face and a depressed level of consciousness. An intravenous diazepam injection facilitates the passage of a tube in the conscious patient

Assume cervical spine injury until proved otherwise.

Control of Hemorrhage

Blood loss from facial injuries is rarely sufficient to cause hypovolemic shock. No attempt should be made to explore wounds; they should be dressed or packed and immobilized by placement of external bandages. This also affords some support to fractured bones. Blood loss from an open wound can be controlled by direct clamping and ligation of the vessels in the wound, if locally applied pressure is ineffective. In the presence or suspicion of shock, an intrathoracic or intra-abdominal source of bleeding should be sought.

Control of Pain

Maxillofacial injuries cause little pain, but the discomfort is considerable, so in the majority of cases analgesia may still be necessary at the scene of injury. It is important to avoid powerful analgesics which depress the level of consciousness and respiration.

Evacuation

Following the establishment of the airway and after controlling bleeding, evacuation of the patient from the place of injury is carried out by any means available. During evacuation medical personnel are responsible for the uninterrupted continuation of the treatment initiated on site: establishing and maintaining airway, controlling hemorrhage, and treatment of shock.

Treatment at Field or Base Hospital

Clinical Examination

Following a general physical examination of the patient and the institution of resuscitative measures, attention is directed to the local injury in the maxillofacial region. Examination may disclose minor injuries such as superficial lacerations or contusions of the soft tissues, or more serious and deep wounds and fractures resulting in disfigurement and functional disability.
The signs of a fracture of the facial bones are asymmetry, abnormal mobility, failure of transmitted movement, clicking, crepitus, derangement of occlusion, irregularity in the line of the teeth, bleeding, swelling, salivation, and discharge of CSF due to fracture of the middle cranial fossa or cribriform plate area. Edema of the eyelids, subconjunctival ecchymosis, diplopia, and uneven pupillary levels are signs of orbital wall fractures.
The patient is examined for cranial nerve damage (facial palsy occurs usually due to damage to the peripheral branches of the facial nerve).

Radiological Examination

Radiographs are taken to confirm the presence of a fracture, to determine the direction of the fracture line, the severity of the bone damage, the relationship of the teeth to the fracture line. The radiographic projections to be used are:

- Lateral view of the face
- Orbital walls
- Occipito-mental 30° (for facial bones)
- Submento-vertical projection (zygomatic arch)
- Mandible-postero-anterior 10° and lateral oblique views
- Panoramic tomograph of the mandible
- Occlusal view
- Tomography (for severe maxillofacial injuries and orbital blow-out fracture)

Primary Wound Surgery

The gunshot injury of the face is characterized by a small entry and a large exit wound. Loss of tissue is usually not extensive. The extensive wound is due to retraction of the edges by the pull of severed muscles, the elasticity of the surrounding skin, local edema, and displacement of fractured bone segments.

After the general condition of the patient has stabilized, the plan of treatment is drawn up.

Wound Cleaning

After the administration of general anesthesia, the surgical field is carefully prepared. The wound is washed by gentle scrubbing with a soft brush to remove dirt, soil, grease, and other foreign bodies. The cleansing may be accomplished by soap and saline. In addition, 1% hydrogen peroxide is pumped and sprayed throughout the wound using a Waterpik. The effervescence floats small pieces of dirt and foreign bodies to the surface. Towels and gloves are changed, and then the wound is exposed and cleaned again in a normal manner.

Debridement

The blood supply of the facial tissues is so plentiful and resistance to infection so high, that only minimal excision of skin is necessary. Only the crushed tissue is removed from the wound, using a sharp scalpel or scissors. In the face, skin edges are trimmed only when they are irreparably damaged. Bone fragments are removed only if they are completely loose, and if needed for reconstruction, they are stored in saline solution.

Principles of Fracture Treatment

Treatment of fractures of the jaw is successful only when bony continuity has been established, and the normal masticatory mechanism has been restored. The best guide to the correct position of the fragments is the occlusion of the teeth, and therefore the teeth are used in the immobilization of fractured jaws. The stabilization of the dislocated segments and the fractured bones always precedes soft tissue closure.

Soft Tissue Closure

Facial injuries are one of the exceptions to the general rule that soft tissue wounds should not be closed at the time of primary surgery of contaminated wounds. For optimal cosmetic results, primary wound closure is to be preferred to delayed primary suture: this can be done due to the rich blood supply of the facial tissues. An inner lining should be provided as well as an out covering in suturing soft tissue wounds. In injuries penetrating into the oral cavity the mucous membrane must be sutured first.

Repair of wound proceeds from the depth towards the surface. No pockets must be left in the depth. If such a pocket cannot be closed by suture, a drainage tube must be inserted into it. On repairing the wound, care must be taken in reconstructing correctly the damaged muscle of facial expression. The ends of severed branches of the facial nerve must be recovered using binocular lenses and sutured with very fine stitches. Stensen's duct, if

damaged, must be reconstructed over a fine drainage tube (diameter 2 mm) introduced retrograde from its outlet in the mouth.
Wounds which are likely to be complicated by secondary hemorrhage or the development of a hematoma or seroma are drained. A capillary drain is adequate for a small wound; it is inserted between the wound edges and reaches into the subcutaneous tissues. Large and deep wounds are drained by tubes with side openings; they are introduced through a separate stab incision into the deepest part of the wound. Suction drainage is used in wounds where there has been extensive mobilization of bone — the tubes are connected to a suction pump. Drainage is removed within 24–48 h.
In severe penetrating wounds with extensive loss of tissue, opening widely into the oral cavity, and in the presence of poor general condition, primary reconstruction is not attempted. After careful wound toilet and the reduction of the displaced parts, the skin edges are sutured to the mucosal margin around the entire defect. Particular care must be taken to cover the exposed bone. In such cases, reconstruction is carried out by mucosal or skin flaps as soon as the general condition of the patient allows.

Bone Fracture Management

Diagnosis of fractures is based on the history of the injury, the presence of deformity, abnormal mobility, crepitation while in motion, functional disability, as well as adequate roentgenograms. The treatment consists of reduction and immobilization of the mobile segments.
A fractured tooth, or a tooth with an exposed root in the fracture line should be extracted in order to prevent infection. However, this is not axiomatic, and each case should be considered separately. For instance, should the extraction of the tooth result in the displacement of the bony segment, it is preferable to retain the tooth and perform the extraction at a later stage when the fracture line is stable.
Several methods of immobilization of the jaws are used:

- Prefabricated arch bars
- Interdental wiring
- For edentulous patients: fixation of lower denture to the mandible by circum-mandibular wires and immobilization of the mandible to the maxilla
- Immobilization of the maxilla by suspension wires from the zygomatic arch, inferior orbital rim, or frontal bone
- Extraoral skeletal fixation or cast metal cap splints

Prefabricated Arch. Various types of prefabricated arch bars are available: small hooks serve to receive the tie wires. The bar is adapted to the teeth and is fixed with 0.4- or 0.5-mm soft stainless steel ligature wires (Fig. 1).

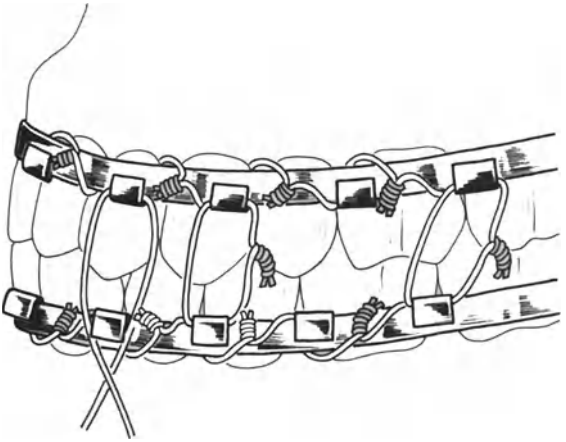

Fig. 1. Application of prefabricated arch bars

Interdental Eyelet Wiring. An eyelet is formed in the middle of a wire by twisting the ends. The two ends of the eyelet wire are pulled from the buccal side through the interdental space of the two teeth to which the eyelet is to be applied. One wire end is then brought through the neighboring distal interdental space and another through the mesial one in a buccal direction.

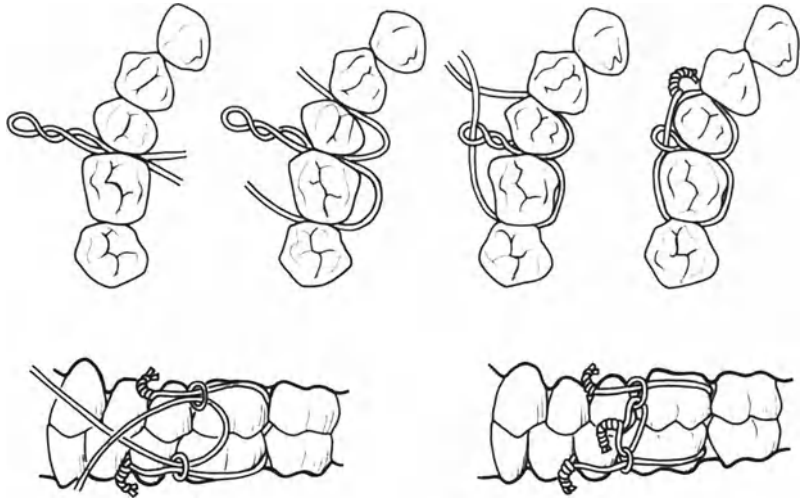

Fig. 2. Method of application of eyelet wires

The distal end is pulled through the eyelet and twisted with the mesial end. Tie wires are drawn through the eyelets, and the jaws are wired together. A minimum of three sets of eyelets is used on each jaw (Fig. 2).

Mandibular Fractures. Mandibular fractures occur at the angle, the condylar neck, the body, the symphysis, the ascending ramus, the coronoid process, or a combination of those. There is a great variety depending on the causative factors. Gunshot wounds are destructive and frequently cause comminution. When the fracture line is in the body of the mandible, and there are enough teeth for immobilization, the simplest methods of immobilization can be applied. In fractures of the angle the posterior segment of bone may be displaced upward, and to hold it down a bite block is placed over the retromolar tissues. In the presence of gross dislocation of segments, open reduction of the fracture is necessary: this implies an incision for the exposure of the fragments and the drilling of holes for the passage of fixation wires through the bone (Fig. 3). Osteosynthesis by compression plates has the advantage of functional stability, so that postoperative intermaxillary fixation is not necessary. Plating is indicated in:

- Fractures in edentulous patients
- Fractures of the dentulous mandible in association with fractures of the condyle (which require early postoperative functional therapy)
- Displaced fractures of the angle or ramus of the mandible
- Comminuted fractures with a bony defect

The plates have two horizontal, inner holes and two oblique, oval, outer holes. In the inner holes the drilling is eccentric, and when the screw is tightened dynamic compression at the fracture line is achieved along the lower border of the mandible (Fig. 4). In order to obtain adaptation of the occlusal plane, drilling is performed on the lower part of the oblique, oval outer holes. Insertion of the screws then causes compression along the alveolar line of fracture (Fig. 5).

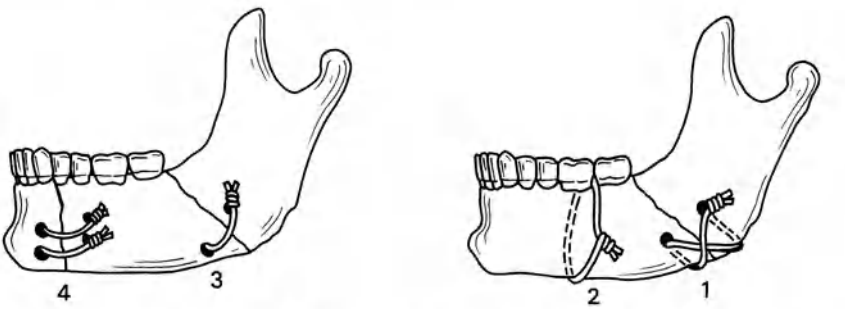

Fig. 3. Various wire ligature shapes used in open reduction. *1*, figure-of-eight; *2*, circumferential; *3*, simple; *4*, double

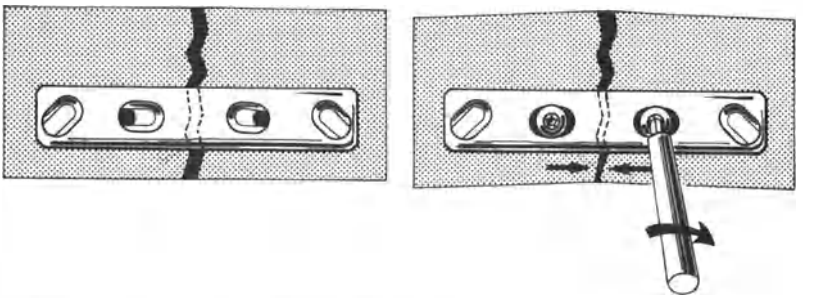

Fig. 4. Osteosynthesis by compression plating.

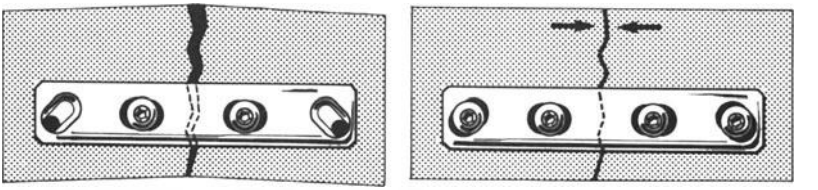

Fig. 5. Osteosynthesis by compression plating.

In the edentulous patient the mandibulomaxillary splint can be made from his own dentures fixed together, utilizing circum-mandibular wires and suspension wires from the zygomatic arches.
In condylar fractures with no disturbance of occlusion, immobilization is not required. If the occlusion is disturbed, manual reposition for normal closure of the teeth is done, and immobilization is necessary for 2 or 3 weeks, followed by intensive physiotherapy. If the occlusion is not perfect, elastic traction is useful for the fixation period in order to achieve intercuspidation of teeth.

Zygomatic Bone Fractures. Displaced fracture of the zygoma may be overlooked at the time of injury because of swelling and ecchymosis. The reduction of an uncomminuted fracture can be performed by using a steel hook with which the inferior margin of zygomatic bone is grasped; by traction upward and forward, the bone is elevated into the normal position. Usually no fixation is required. Another approach can be performed through an incision in the temporal region, close to the hair line, and tunneling downwards between the temporal muscle and fascia, using for elevation a Gillis instrument. Another method is the intraoral approach, using a strong dull instrument introduced under the zygoma through an intraoral incision in the vestibule. The elevation of the fragments in comminuted fractures of the

zygoma, especially when associated with fracture of the floor of the orbit, is done via the Caldwell Luc approach, and additional support is achieved by an inflated Foley catheter or with iodoform gauze. When these means of reduction fail, direct interosseous wiring can be performed to stabilize the frontozygomatic area and restore the continuity of the orbital rim.

Fracture of the Zygomatic Arch. These fractures may be reduced by a temporal approach, as described above, by incision over the arch at the site of depression, or by an intraoral approach. Usually no fixation is necessary after the reposition.

Fracture of the Maxilla. There are three types of fractures which produce partial or complete separation of the maxilla from the rest of the skull. They are classified as Le Fort I, II, and III fractures. They may occur singly or in combination.
Treatment is by reduction with restoration of normal occlusion and immobilization. In Le Fort I and II fractures, intermaxillary fixation and circumzygomatic suspension wires are required. In severe deformity in Le Fort II and III, the reposition of the impacted maxilla is achieved by Rowe's disimpaction forceps. Further traction and immobilization can be maintained by extraoral devices or suspension wires. The displaced bone fragments can be fixed by using an intraosseous wire ligature, by application of plates, or monocortical screws. The suspension wires for the mandibulomaxillary complex can run from the zygomatic arches, if they are intact, or from the lateral orbital wall. In patients with an edentulous maxilla, after manual reduction, the fragments can be immobilized by replacing the patient's upper denture and by fixation with circumzygomatic suspension wires.
In palatal fracture, reduction and immobilization by interdental wiring is sufficient.

Postoperative Care

A maxillofacial injury with intermaxillary fixation (where no tracheostomy has been performed) requires 48 h of close supervision to ensure a free airway. A wire cutter must be immediately available to release intermaxillary fixation in case airway obstruction develops (e.g., vomiting). A fluid and semifluid diet is required (a feeding tube may be necessary for the first few days). The patient is weighed weekly to check his nutrition. The mucosa of the lips and cheeks is protected from wires by the application of peripac material (produced by De Trey, Switzerland) of a consistency like chewing-gum. The intermaxillary fixation must be checked daily. Mouth hygiene is extremely important as part of the postoperative treatment. Mouth irrigation with a solution of 0.2% chlorhexidine, hydrogen peroxide 1%, or normal saline is performed after each meal using a syringe or Waterpik.

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4.5 Eye Injury

M. Belkin

Introduction

In spite of its being such a small organ, injuries of the eye comprise up to 10% of all injuries requiring hospitalization in modern wars, because the eye is exceedingly vulnerable. A metal fragment a fraction of a millimeter in size, which will hardly be noticed anywhere else, causes a severe injury in the eye. Such trauma always requires hospitalization and frequently hazardous surgery, will occasionally lead to the loss of the injured eye and on very rare occasions the loss of the other eye as well through sympathetic ophthalmia. This specific sensitivity of the eye to mechanical injury and other forms of physical trauma is hardly surprising when one considers that this small ball, 2.4 cm in diameter, contains many delicate tissues each vital to vision.

Classification of Eye Injuries

1. Injuries of the ocular adnexa
   (a) The eyelids
   (b) The lacrimal system
   (c) Orbital injuries:
      - Orbital hemorrhage
      - Orbital and facial fractures
2. Injuries to the eye ball
   (a) Blunt:
      - Cornea and sclera
      - Anterior chamber, iris, and ciliary body
      - Lens and vitreous body
      - Retina and choroid
      - Optic nerve
   (b) Superficial:
      - Conjunctival abrasions and foreign bodies
      - Corneal abrasions and foreign bodies

(c) Perforating:
- Proliferative vitreoretinopathy
- Intraocular foreign bodies

3. Nonmechanical injuries
(a) Ocular burns
(b) Radiation injuries

Injuries of the Ocular Adnexa

The ocular adnexa are the eyelids, the lacrimal system, the orbit, and its contents, excluding the eyeball itself.

The Eyelids

The function of the eyelid is to shield the eyeball from injury and to keep the cornea moist and transparent. Eyelid injuries can be sharp or blunt. Blunt trauma to the edge of the orbit may press the soft tissues against the bones and cause a well-demarcated tear along the eyebrow down to the bone. Laceration of the levator palpebrae superioris, its tendon, or nerve results in ptosis. If the injury is to the tendon, primary surgical repair is indicated to prevent retraction of the tendon into the orbit.
The main complication of eyelid injuries is that the cornea is exposed and unprotected when the eyelid is avulsed completely or partially. If the casualty is to be evacuated to a base hospital shortly after the injury, it is sufficient to bandage the wound, give systemic antibiotics and keep the eye moist during the evacuation process. If the evacuation is delayed for many hours the cornea must be covered by suturing the remaining eyelid to the orbital rim. This is the only occasion when a physician in the field is allowed to treat surgically an injury in the ocular area.
In the base hospital all eyelid lacerations will be carefully sutured using microsurgical techniques to ensure the best possible postoperational and cosmetic results.
An injury typical of combat is one that results from an explosion which causes multiple, relatively superficial injuries from the penetration of small pieces of earth, soil, and gunpowder into the cornea, eyelids, and face. Casualties suffering from this type of injury look as if their normal appearance cannot be salvaged. The swollen face and eyelids are peppered with multiple foreign bodies covered by blood clots. However, the final outcome is often surprisingly good. The reason is that the particles which penetrated the skin and cornea are usually small and thus not energetic enough to penetrate deeply. Furthermore, most of the particles driven into the face were sterilized by the heat of the explosion.

On site treatment includes bandaging the injuries and giving systemic antibiotics. In the base hospital all the large fragments such as pieces of stone or glass will be removed with a pair of forceps, and the area will be cleaned by washing in saline and soap once the casualty is anesthetized. If necessary a brush can be used to wipe away most of the explosion debris. The area is then smeared with antibiotic ointment and covered. Always remember to examine the eyeball properly in the base hospital since the skin wounds may mask eyeball injuries such as intracorneal and intraocular foreign bodies.

The Lacrimal System

Injury to the lacrimal gland is rare and does not constitute a clinical problem. Injuries to the tear drainage system however are very common, especially a tear of the lower canaliculus.
On site treatment includes bandaging the injury and giving systemic antibiotics. As with all ocular injuries, this type of tissue should be handled only in hospitals at which microsurgical facilities and an experienced ophthalmic surgeon are available. The operation required is a careful suture of the canaliculus around a polyethylene splint which will remain in place for a long time to prevent intraluminal scarring, consequently ensuring the patency of the canaliculus.
Injuries to the nasolacrimal sac and duct are common in facial injuries. They often lead to epiphora and dacryocystitis which may require late surgical treatment.

Orbital Injuries

Orbital hemorrhage is a common result of periocular trauma. The resulting "black eye" or "shiner", if not accompanied by any injury of the eyeball or other soft tissue or bone injuries, does not require treatment. It always requires, however, examination by an ophthalmologist.
The commonest cause of orbital and facial fractures is a head-on injury in road or aircraft accidents or assault by blunt weapons. Isolated orbital fractures occur when a force acts directly on the orbital bones and only these are broken. Otherwise the fractures are a continuation of skull or face fractures. They may be usefully classified into: (a) fractures of the upper third of the skull resulting from direct and frontal area trauma; (b) fractures of the middle third of the skull resulting from direct trauma to face; (c) inner orbital fractures resulting from direct impact on the orbit; and (d) indirect orbital fracture accompanying head trauma. A detailed description of these injuries is outside the scope of this chapter.
In all cases of orbital fracture, the eyeball itself, the extraocular muscles, the optic nerves, and the other sensory and motor nerves transversing the orbit

may be injured. A fracture peculiar to the orbit is a blow-out fracture, which is the result of a sudden increase of intraorbital pressure consequent to violent, sudden contact with the eyeball by a blunt object whose diameter is smaller than the orbital opening. The increased pressure in the eyeball is transferred to the orbital contents and thereby to the orbital walls, whose weaker parts, mainly the floor and sometimes the nasal wall, are broken and pushed outward. Orbital fat and extraocular muscles, commonly the inferior rectus, may become entrapped in the fracture.

The characteristic syndrome includes limitation in the elevation of the eye, diplopia, enophthalmus, and superficial anesthesia in the area of the second division of the trigeminal nerve. The roentgenographic picture is pathognomonic: the orbital contents bulge into the maxillary sinus like a "hanging drop." In some of these cases the eyeball itself escapes injury but the soft tissues are always affected by edema, hemorrhage, and sometimes emphysema of the eyelids, orbit, or both (evidence of a direct connection with a nasal sinus). If massive orbital hemorrhage occurs, the eye will be exophthalmic but will become enophthalmic after the absorption of the hemorrhage since some of the orbital contents are now in the nasal sinus.

All orbital fractures must be evaluated in a hospital. Not all of them have to be treated surgically, but if surgery is indicated, it should be performed early, since orbital and facial fractures heal quickly and permanent deformation results if malalignment is perpetuated by the healing process. These deformations are notoriously difficult to treat at a later stage.

Injuries to the Eyeball

Blunt Injury

Blunt injury is caused by the application of sudden pressure on the eyeball. The extent of damage sustained by the eye depends chiefly on the kinetic energy of the injurious agent. The pathogenesis of blunt ocular injury is from:

1. The direct impact by the causative agent, the maximum effect of which is at the site of impact
2. The energy imparted to the eye which creates a pressure wave moving, sometimes explosively, through the eye's fluid contents and causing damage especially in areas distant to the point of impact by a contrecoup mechanism
3. Indirect force transmitted through neighboring structures to the eye

Cornea and Sclera. The most sensitive layer of the cornea to blunt trauma is the nonregenerative endothelium which is responsible for the relative detumescence and consequently the transparency of the cornea. Damage to the

endothelium leads to edema of the corneal stroma and thus to corneal opacity, which usually clears spontaneously if not too extensive. A sudden blunt force acting on the eye can cause tears of the sclera directly or indirectly by increasing the intraocular pressure or by causing the sclera to come into contact with the orbital wall. A typical rupture is in the limbal area (the transition zone between the cornea and sclera). Sometimes, however, the rupture is in the posterior segment of the eye. Such cases may be taken at first glance to be mild injuries with subconjunctival and vitreous hemorrhage only. Hypotony, however, points to the diagnosis of rupture; therefore, after blunt trauma all eyes should undergo tonometry and be evaluated and examined by an ophthalmologist. If the intraocular pressure is low, computerized tomography should be performed to diagnose and delineate the scleral tear. Occasionally operative exploration of the sclera is indicated. It cannot be overemphasized that after blunt trauma even with no obvious symptoms and signs the patient has to be examined at a base hospital by a well-equipped, experienced ophthalmologist.

Anterior Chamber, Iris, and Ciliary Body. The most common outcome of blunt ocular trauma is hyphema or hemorrhage into the anterior chamber, where the blood can be seen as a red fluid level in the erect patient. The blood usually resorbs spontaneously within a few days. Numerous methods have been suggested for the treatment of primary hyphema, but none is very effective. A secondary hemorrhage may occur a few days after the injury, associated with a sudden increase of intraocular pressure. If this pressure does not fall within a few days there is a clear indication to drain the hemorrhage surgically. If left untreated, a hemorrhagic glaucoma sets in, and hematogenic pigmentation of the cornea may permanently impair vision.

Blunt trauma can affect the iris and ciliary body, causing a tear of the sphincter pupillae, tear of the base of the iris, or disinsertion of the ciliary body from its base. Commonly, blunt trauma results in a temporary or permanent paralysis of the iris muscles (iridoplegia) or of the ciliary body (cycloplegia). Immediately after the injury one may observe miosis and ciliary spasm, but these usually disappear. A more severe result of blunt trauma is ciliary body tears; these lead to deepening of the anterior chamber angle and its recession. This type of injury may lead to traumatic glaucoma after months or years; thus the examination of the anterior chamber angle (gonioscopy) is mandatory after blunt trauma. Damage to the anterior chamber angle is an indication for repeated intraocular pressure measurements over many years.

Lens and Vitreous Body. Tearing of the suspensory ligament of the lens is a relatively common result of blunt ocular trauma. In mild cases it manifests itself as a tremor of the lens on eye movements (phakodonesis). In more severe cases the lens can be subluxed or dislocated into the anterior chamber of the vitreous body. These alterations in the lens position lead to opti-

cally induced visual impairment, glaucoma, and uveitis. The lens, being a most sensitive organ, is very liable to lose its transparency and becomes cataractous as a result of blunt trauma, even after apparently mild injuries.

Blunt trauma usually causes the relatively unimportant complication of liquifaction of the vitreous. More severe trauma results in vitreous hemorrhage from the retina or ciliary body blood vessels. Blood absorption from the vitreous body is slow, but it is usually complete, and normal vision is regained, especially in young people. Occasionally, however, the hemorrhage becomes organized and scarred. The scar is not only nontransparent, it also retracts and may lead to retinal detachment. A late complication of many vitreous hemorrhages is hemosiderosis which may be accompanied by uveitis and glaucoma. The treatment of a massive vitreous hemorrhage or one that is not absorbed within a few weeks is vitrectomy — an operation which entails removal of the hemorrhage together with the vitreous substance.

Retina and Choroid. Blunt trauma may cause hemorrhages, laceration of neural tissues, and atrophy of retinal cells, producing permanent visual incapacitation. Traumatic retinal edema is common in blunt trauma — it manifests itself ophthalmoscopically as a milky-white zone with indefinite borders in the posterior pole of the eye. Inside this zone the macula is seen as a cherry-red spot similar to its appearance after central retinal artery occlusion. In mild cases normal vision is restored. In most cases, however, atrophic retinal zones with pigmentary changes, cystoid degeneration, or a hole in the macula appear. Another possible consequence of blunt trauma is ischemic necrosis of the retina, which may lead to atrophic changes and fibrovascular proliferation.

Retinal tears may appear after blunt trauma as a result of the impact shortening the anteroposterior diameter of the eye and of equatorial spread. Such tears do not commonly occur in young people, yet even mild or indirect trauma may lead to retinal tears in the eyes of myopes and the elderly. Such tears may be asymptomatic for years and then suddenly manifest themselves as retinal detachment. Therefore, all cases of blunt trauma to the eye must undergo a thorough fundus examination to exclude retinal tears. If such tears are found, retinal detachment may be prevented by laser photocoagulation. The same forces acting on the retina during blunt trauma may affect the choroid. Intrachoroidal, subchoroidal, or subretinal hemorrhages are common. These tend to be absorbed and remain as atrophic pigmentary scars in the fundus. More severe injuries may lead to choroidal detachment as a consequence of a tear and hemorrhage from one of the posterior ciliary vessels. Another result of severe blunt trauma is the appearance of tears in the choroid near the optic disc, usually on the nasal side but occasionally encircling the disc; this site is the location of the maximal contrecoup force when the impact on the eye is from the front. In most

cases of choroidal tear the retina remains intact. Tears of both choroid and retina occur when the posterior part of the eye is struck but not torn by a missile entering the orbit.

Optic Nerve. The optic nerve may be torn from the eyeball by a missile transversing the orbit or paraorbital structures. It may also be avulsed as a result of increased intraocular pressure which tends to blow out the weakest portion of the sclera, the lamina cribrosa, through which the optic nerve exits the eyeball. The injury of the optic nerve may be partial or complete and is permanent in most cases since the optic nerve, being an extension of the brain, has negligible regenerative capacity.

Superficial Ocular Injury

Superficial ocular injuries include abrasions and nonperforating lacerations of the conjunctiva, cornea, and sclera with and without small foreign bodies; they form the only ocular injuries for which the physician in the field can provide definitive treatment.

Conjunctival Abrasions and Foreign Bodies. A small grain of sand or similar foreign body which impinges on and remains in the conjunctiva of the eyeball or eyelid causes a "foreign body in the eye" sensation, especially when the lid moves. The eye is red and waters. To examine the external eye a good light and, if possible, magnification are used. The physician should determine whether or not the eye has been perforated: If any suspicion exists, the casualty must be seen by an ophthalmologist. The physician first examines the inner surface of the lower lid which can be everted by lightly pressing with a finger on the lower orbital rim. This maneuver must be performed very gently with pressure exerted downwards on the bony orbital rim only and not in the direction of the eyeball since ocular contents might be extruded if pressure is applied to a perforated eye. It should be reemphasized that this maneuver should be performed only if the physician on site or at the first aid station is sure that the eye is not perforated. Next the upper lid is everted without applying pressure to the eyeball. A physician unfamiliar with this maneuver had better not perform it unless convinced that the eye is not perforated. Then the conjunctiva on all sides of the cornea should be examined. A foreign body found in any of these locations is washed away by irrigation with sterile normal saline. Alternatively, it is removed by gently wiping it off with a cotton tipped wooden stick. If the foreign body cannot be removed, if the symptoms and signs do not disappear within 24 h after removal, or if the physician suspects that the superficial foreign body is not the only lesion in that eye, he should refer the patient promptly to an ophthalmologist in a base hospital. Whether or not the foreign body can be successfully removed, broad spectrum local antibiotics should given for a few days.

Examination and treatment can be facilitated by a drop of local anesthetic solution; but the patient should not be allowed these solutions for personal use, since he may cause further damage to an anesthetised eye. Pain and discomfort are best overcome by systemic analgesics.

Corneal Abrasions and Foreign Bodies. This is a very common occurrence, and every physician should be familiar with its treatment. The symptoms are an intense "foreign body in the eye" feeling which is due to the rich sensory innervation of the cornea. Copious watering and redness are present as well as a decrease in visual acuity if the abrasion or the foreign body is in the center of the cornea. The cornea should be examined under good illumination, preferably with magnification. When the cornea is illuminated by a penlight the white reflex returned from the cornea is irregular, being distorted by the lesion or the foreign body. This leads to the diagnosis of the corneal foreign body in most cases.

Caution in the use of local anesthetic drops is also pertinent in the handling of corneal foreign bodies. The treatment is to wash out the foreign body or remove it with a cotton-tipped stick. However, the physician administering first aid should never attempt to remove the foreign body forcefully since this maneuver might lead to perforation of the eye. If the foreign body is not easily removed the physician should refer the casualty to an ophthalmologist. If the foreign body was removed or if only an abrasion was found, ophthalmic antibiotic ointment should be applied and the eye patched. Ointment should never be applied to eyes with a suspected or actual perforation since it tends to become an intraocular foreign body which is very hard to remove.

Pain is treated by oral analgesics. The casualty should be re-examined after 24 h and if the eye is still irritated referral should be made to an ophthalmologist. The ophthalmologist will remove the foreign body atraumatically using surgical instruments under magnification with a microscope and proper illumination. A long-standing, ironcontaining foreign body may rust in the cornea and stain it brown. The treatment of this stain should also be left to the ophthalmologist who will scrape it away.

Any uncertainty about the ocular condition or its treatment (even in cases which seem trivial) is an indication for referral of the patient. An unnecessary evacuation is preferable to the loss of an eye.

Perforating Injury

The great variety of mechanical damage caused by a perforation injury agent is due to the close proximity of the many delicate tissues existing within the small volume of the eyeball. The extent and severity of the damage depends on the location of the perforation and the size and energy of the injurious agent. Small objects tend to become intraocular foreign bodies

or leave the eyeball to end up in the orbit (double perforation). Large objects may destroy the eye completely.
Most perforations are from the front, piercing the cornea and the sclera or both. The wound may be small and well demarcated and seal spontaneously in an orderly or disorderly fashion; the latter occurs especially if some tissue is missing. Lack of closure can lead to the proliferation of the corneal epithelium inwards, to cover the walls of the chamber of the eye (a complication which is very hard to treat). Large and gaping injuries can entrap intraocular contents in the opening, thus becoming closed. The entrapped tissues may be the iris (most commonly), the ciliary body, the lens, the vitreous, or even the retina: if untreated, healing is disturbed and adhesions of the tissues to the cornea or sclera result in a opaque scar (leukoma adherens). After the healing of very large wounds, the intraocular pressure may cause an outward evagination of the cornea or sclerae, owing to the weakness of the scar. This staphyloma distorts the eye and usually reduces vision. A less common complication is corneal fistula. An injury which perforates deeper than the cornea and the sclera leads to a variety of clinical and pathological presentations, resembling those caused by blunt trauma despite the different mechanisms of injury. In the iris one finds a hole or disinsertion (iridodialysis) with the resulting hemorrhage. The injured lens develops a cataract sooner or later according to the size of the wound. Injuries to the ciliary body, retina, and choroid lead to vitreous hemorrhage.
Holes in the retina, unless large, do not usually cause retinal detachment since the hole becomes blocked by connective tissue proliferation. Retinal detachment following perforating injuries is usually caused by contracture of fibrovascular scars in the vitreous (proliferative vitreoretinopathy).

Treatment. On site and at the first aid station all the physician can do is to avoid meddlesome handling and manipulation of the perforated eye, to bandage the eye without exerting pressure on it, to give systemic antibiotics and systemic analgesics as required and evacuate the casualty as soon as possible.
The hospital treatment of most severe perforating eye injuries in the not too distant past was enucleation. There were three main reasons for removing the severely injured eye: (1) the danger of sympathetic ophthalmia which is a severe intraocular inflammation caused in both eyes by an injury of one them; (2) the danger of intraocular infection; and (3) the lack of effective surgical techniques to manage severe ocular injuries (especially those of the posterior segment of the eye). However, these reasons no longer have such significance due recent advances in medicine and ophthalmology.
The incidence of sympathetic ophthalmia has decreased considerably, although it is still potentially a blinding complication. This potential danger is not an indication for enucleating an injured eye. Enucleation should be performed only if the disease is suspected of already being present in the

uninjured eye. The disease is now treatable by corticosteroids and cytotoxic drugs. Likewise, the potential for severe intraocular infection and inflammation (endophthalmitis or panophthalmitis) is not an indication for enucleation. Antibiotic treatment including intraocular applications combined with removal of abcesses from the eye by vitrectomy facilitate the salvage of eyes previously considered hopeless. Our inability to restore the eye morphologically and functionally is still a cause for enucleation in very severe cases, but only when the injured eye is painful, since even when sight is lost, it is better to fit a cosmetic appliance over the eyeball than in to the empty socket.

The armamentarium required by the ophthalmic surgeon to enable him to avoid enucleation and restore vision in many traumatized eyes previously considered hopeless consists of the techniques of microscopic surgery with appropriate sutures made of noninflammatory materials and of vitrectomy, which enables the ophthalmic surgeon to operate in the previously forbidden territory of the vitreous cavity, allowing for the safe removal of blood, fibrovascular tissue, and even the vitreous substance itself.

In most cases these techniques enable the surgeon to restore the near-normal anatomy of all injured ocular tissues. In addition to the primary microsurgical suture of the ocular wound, the surgeon removes the lens when it is damaged, usually through a separate incision. Vitreous in the anterior chamber along with lens material, especially when blood is also present, is an indication to remove the lens, vitreous, and blood since they promote proliferative vitreoretinopathy, the intraocular scarring process which clouds the eye's refractive media, distorts the globe, and leads to retinal detachment.

When the posterior part of the eye is affected, the scleral lesions must be sutured and the retina reattached (if detached). When the globe is severely disorganized the anatomical integrity of the eye should be restored even if preserved sclera has to be used. Functional recoveries have been recorded in seemingly hopeless cases, and the prospect of maintaining minimal vision (the distinction between light and dark) justifies all surgical attempts.

Even eyes which did not sustain severe injuries and in which the primary medical and surgical handling was succesful and infection did not ensue may be lost as a result of the two main complications of ocular injuries: proliferative vitreoretinopathy and intraocular foreign bodies.

Proliferative Vitreoretinopathy

Scarring is a beneficial process in wound healing. However, over days or weeks, scarring in the eye may become the destructive, progressive process of proliferative vitreoretinopathy. The fibrovascular tissue affects vision not only by being opaque but also by the tendency to contract, leading to trac-

tion on the retina and its susequent detachment or to deformation of the eye, causing impairment of vision and even blindness. The transformation of beneficial scarring to the destructive process of proliferative vitreoretinopathy is insidious: It may go undetected at an early stage if not suspected and sought after.
The clincal symptoms and signs indicating the development of proliferative vitreoretinopathy are: decrease in visual acuity, sudden changes in intraocular pressure, ocular hypotony (which may escape detection if the drainage of the aqueous humor was impaired by the injury), pupillary membrane, irregular or eccentric pupil, and lens dislocation. If treated properly, most injured eyes need not reach the stage at which they exhibit any of the above-mentioned signs. Proliferative vitreoretinopathy should be prevented by removing destroyed tissues which tend to promote its appearance. Removal of the disrupted lens has been mentioned previously: many ophthalmic surgeons tend to perform vitrectomy in severe posterior segment injuries some days after the initial repair of the eye. This maneuver not only removes the vitreous cavity hemorrhage but the vitreous body itself, since the latter acts as a scaffolding to fibroblasts and thus favors proliferation. Thus, the replacement of the vitreous body with saline prevents generalized proliferative vitreoretinopathy. Since this applies to the vitreous cavity only, some surgeons also carry out preventative buckling of the eye, to avoid retinal detachment due to the contraction of scar tissue inside the retina and on its surface.

Intraocular Foreign Bodies. When a missile enters the eyes and remains there as a retained intraocular foreign body, the ophthalmologist is faced with diagnostic, therapeutic, and surgical problems. Even small foreign bodies can lead to severe visual problems which culminate in blindness. The foreign bodies concerned are small, ranging in size from less than 0.1 mm to 3 mm in their largest dimension, their weight not exceeding 500 mg. Larger foreign bodies damage the eye so severely upon penetration, that they leave no chance of surgically restoring the integrity of the eye, although very rarely such cases have been recorded.
The prognosis of eyes with retained intraocular foreign bodies depends mainly on the metallic content and composition. Nonmetallic foreign bodies are usually inert, and if they did not mechanically destroy the eye upon penetration, are not exceedingly large, or lie in the optic path, they may generally be left in the eye (if they are not a source of infection). The effects of retained metallic foreign bodies on the eye can be divided into three phaşes.

1. In the first stage, the type and severity of the injury is determined by the mechanical damage, and the clinical effects do not differ essentially from other penetrating injuries. It must be mentioned that contamination is not commonly caused by military metallic foreign bodies, which tend

to become sterilized by the heat generated by the explosion and by their friction with the air.

2. In many eyes with retained metallic intraocular foreign bodies, even if the mechanical injury is small, the eye becomes inflamed a few hours or days after the injury, even if no infection is present. This sterile inflammatory reaction varies according to the nature of the foreign body and the tissues in which is settles. If it lies in the anterior chamber or its angle, it can remain inert for a long time. Also, a small foreign body in the lens does not elicit a reaction if the hole in the capsule created by its entry is small and seals spontaneously. The cataract caused by such an injury can be minimal and remain so for many years. The uveal tissues, especially the ciliary body, are very sensitive to any injury, even if the agent is composed of inert material. In such cases, chronic persistent uveitis comes on hours or days after the injury and may end in atrophy of the eyeball or intractable shrinkage of the eye. If the foreign body is composed of pure copper or one of the alloys containing a high percentage of copper, the inflammation my be violent and detrimental to vision. It leads to the development of a sterile abscess within the posterior and anterior segments of the eye. In such cases only immediate removal of the foreign body can save the eye. Some metals other than copper lead to less violent reactions.
3. An eye which harbors a metallic intraocular foreign body and has overcome the mechanical damage caused by the penetration and the immediate inflammatory reaction is not safe yet. At a later stage it is threatened by the chemical action of the foreign body which may dissolve in the eye. Soluble metals react with ocular tissues, mainly the retina, and damage them irreversibly. The severity of this process, known as metallosis, and the rapidity of its development depend on the nature of the foreign body, its location in the eye, and the degree of encapsulation. The metals which are particularly liable to cause metallosis are iron and copper, which are unfortunately the most common components of foreign bodies in both peace and war times.

The diagnosis of intraocular foreign bodies may be difficult especially when the foreign body is small and not noticed by the victim. Consequently, any eye which sustained a penetrating injury is suspected of harboring a foreign body until proven otherwise. In military trauma, especially when the injured soldier has sustained multiple injuries, both eyes must be carefully examined for the presence of intraocular foreign bodies and other injuries. The most important clinical examinations are by a slit lamp biomicroscopy and indirect binocular ophthalmoscopy. All injured and potentially injured eyes must be carefully and meticulously examined using these instruments even if the patient is not comfortable. A corneal or scleral wound or a hole in the iris or lens immediately raises the suspicion of an intraocular foreign body. A sign which indicates the presence of a foreign

body is the lack of prolapse of uveal tissue through the corneal hole — the uvea having been pushed inward by the missile. However, uveal prolapse does rule out an intraocular foreign body. The clinical visual examination mentioned above must always be complemented by other tests, since not all foreign bodies can be so diagnosed, especially the small ones or those found in tissues such as the ciliary body. Therefore, all eyes suspected of having a retained intraocular foreign body, which essentially means all eyes with perforating injuries or suspicion of such an injury, must undergo computerized tomographic examination of the eye and orbit. Conventional roentgenological examination is useful, but is should not be used if computerized tomography is available because it is not sensitive enough and does not show the exact topographical relationship of the foreign body to the ocular walls.

Ophthalmic ultrasound is used as a complementary examination to computerized tomography. The advantage of ultrasound over the roentgenological method is its ability to detect nonmetallic foreign bodies and its portability which allows its use in the emergency room and operating theatre. However, one should not accept a negative result from an ultrasonographic examination as final.

Another method for diagnosing the presence of metallic intraocular foreign bodies is based on electrical induction system detectors.

The treatment of an intraocular foreign body is surgical. All attempts to use chelating agents to remove dissolved metals from the eye have failed. All intraocular foreign bodies should be removed unless after long-term observation their inertness has been assured, or if they are small and encapsulated and the operation for their removal is considered more hazardous than their retention in the eye.

In cases in which the decision is made to leave the foreign body in the eye, the patient should be closely followed up for the rest of his or her life for delayed complications of the injury and the foreign body. The examinations which are particularly useful for this purpose, in addition to direct observation, are perimetry and electroretinography, both of which measure functions which tend to be affected gradually by metallosis.

The operation to remove the foreign body in the anterior part of the eye is usually straightforward since the operation can be performed under direct visualization. Two-thirds of the foreign bodies, however, are in the retina or vitreous, and they are often masked by hemorrhage and organization. The prognosis of such cases has been considerably improved in recent years with the development of vitrectomy and other methods of surgery of the posterior segment of the eye. The success of this type of surgery has so much improved the prognosis of intraocular foreign bodies that in many centers, all foreign bodies of the posterior segment are removed by forceps and not by the uncontrollable magnets. Thus, the previously essential question of whether question of whether the intraocular foreign body is magnetic or not has ceased to be meaningful.

Nonmechanical Injuries

Ocular Burns

Chemical burns are the only instance in ocular traumatology in which the treatment in the field clearly affects the prognosis. Burns caused by chemical substances splashed into the conjunctival sac and on to the cornea (be they acids or bases) and thermal burns are difficult to treat. These accidents denature the corneal and conjunctival proteins and consequently lead to corneal opacification, conjunctival scarring, blockage of the lacrimal secreting system, and the formation of adhesions between the palpebral conjunctiva, the bulbar conjunctiva, and the cornea. All these factors indicate a poor prognosis for vision.

In contrast to all other types of ocular injuries, the most important factors in treatment to improve the prognosis of chemical burns is the rapidity and effectiveness of first aid. This consists of thorough rinsing of the eyeball, the conjunctival sac, including the folds between the eyelids, and the eyeball conjunctiva (the upper and lower fornix). The extent of damage caused by the chemical depends on the duration of its contact with the tissues. The first person treating the casualty should subject the burned eyes to a stream of water which will remove as much as possible of the chemical. For this purpose he or she must hold the eyelids open and evert them while continuing the washout. This should be done properly even against the casualty's resistance which is caused by the great pain being experienced. This washing of the eye should take precedence over all other measures including evacuation. Only when the treating person is satisfied that no chemical remains in the conjunctival sac should systemic antibiotics be started, the eye patched and the casualty evacuated to a hospital with an eye department.

Radiation Injuries

The eye is the body organ most sensitive to all types of electromagnetic ionizing and nonionizing radiations, from gamma rays to microwaves. Exposure of the eye to radiation in modern society is common, and ocular accidents involving radiation injuries, especially those caused by lasers, are becoming quite frequent. The ocular damage induced depends on the intensity and duration of the radiation exposure, but the main factor which determines the particular type of damage is the wavelength. The wavelength determines the depth of penetration into the eye and the mechanism by which the tissues are damaged. Ionizing radiation (gamma and X-rays) traverses the eye and causes damage to all the tissues when the quantity of irradiation exceeds a certain threshold. The lens is the most sensitive tissue in the body, and cataracts occur after relatively low-level irradiation. Fractionated doses have a higher damage threshold than single exposures.

Ultraviolet irradiation is mostly absorbed by the cornea and destroys its epithelium, causing "welder's keratitis," a disease typically consequent on watching ultraviolet sources, such as a welding arc, with unprotected eyes. After a latent period of a few hours the corneal epithelium starts to shed, and the patient experiences intense pain and a foreign body sensation in the eye. The lesion and its symptoms heal spontaneously after a day or two and should be treated by patching the eyes and administering systemic analgesics. Local anesthetics should never be given for self-application.
Some of the more intense ultraviolet radiation enters the eye and plays a role in cataractogenesis; even a retinal lesion has been recorded. The visible electromagnetic radiation, of course, penetrates the eye fully and is focused in the retina. The intense energy concentration within the retina, if above the damage threshold, is liable to cause retinal burns. In the past, such accidents were due mainly to intrabeam viewing of searchlights and sun gazing, notably during solar eclipses and drug trips. Currently such lesions are caused by the intense radiation produced by lasers, either intentionally as part of ophthalmological treatment or unintentionally by accidental viewing of a laser beam. The typical lesion consists of a coagulated zone surrounded by edematous inflammation; the latter may subside, but the coagulated area leaves a blind spot in the field of vision. If it occurs in the macula the person may remain permanently blind. Quite a few such cases were recorded recently, all of them due to misuse of laser instruments. It is certain that more laser accidents will occur in the future due to the increased use of laser instruments (military and/or civilian).
Short infrared radiation behaves like the visible spectrum; its energy is concentrated on the retina and produces lesions there. Most accidents in this region of the spectrum are sustained by the use of near infrared lasers. Long infrared radiation does not penetrate the eye and affects the cornea only. Acute and chronic exposure to microwaves, which penetrate the eye, is complicated by the formation of cataracts.
The longest electromagnetic irradiation, radio waves, is not know to cause any acute occular effects. Whilst not part of the electromagnetic spectrum ultrasound irradiation must be mentioned in this context, since it is known to cause ocular damage, particularly cataracts, if it exceeds a certain threshold.

Ocular Protection

The vast majority of both mechanical and nonmechanical ocular injuries can be prevented by the avoidance of hazardous situations and the adoption of protective methods and devices in the appropriate circumstances. The prevention of ballistic injuries illustrates the relative ease of achieving

adequate, almost complete, eye protection. Since most injuries are caused by relatively small objects, a transparent sheet of impact resistant material placed in front of the eyes will stop almost all objects which are liable to produce an ophthalmologically significant injury. This material should not shatter if penetrated and should interfere minimally with all visual functions. This material should also be of stable optical properties, rigid, scratch resistant, of low weight, easy to manufacture, and able to adapt to the protective system which will be placed in front of the eye.

The potential materials for this purpose are thermally or chemically tempered glass and the plastics cellyl resin (CR-39) and polycarbonate (Lexan). The plastic materials are usually preferable to glass since they are more protective, do not lose their impact resistance because of minor surface scratches, and do not shatter upon penetration. Other advantages are their lightness and their lesser tendency to accumulate water vapor on their surface. Their disadvantage is their proneness to scratching — a problem which has been largely overcome by modern surfacing technology. Polycarbonate is far superior to CR-39 in its impact resistance.

These materials can be incorporated either in spectacles, goggles, or eye shields (visors), the precise shape of which and the protective factors of which can be tailored to the requirements of the particular occupation of the wearer. For general use, most of the developed nations adopted the laudable standard of requiring the lenses of all eyewear, whether worn for optical or cosmetic purposes, to be made of one of the above-mentioned impact-resistant materials. This standard is of major importance for ocular protection from ballistic injuries. All soldiers should be issued protective goggles to wear during military exercises and combat.

Additionally, all potential occupational hazards should be minimized through environmental controls such as placing shields over the potential sources of flying objects, e.g., grinding wheels. Protection from radiation injuries should also be mandatory in all locations and occupations in which such an eye injury is possible.

Summary of On Site and First Aid Station Management

Diagnosis and Treatment

The optimal modern treatment of ocular injuries involves experienced ophthalmic surgeons and a well-equipped hospital. A patient with a severe eye injury should be treated only in a department in which, in addition to the proper staff versed in microsurgery and vitrectomy, there is access to diagnostic and surgical instruments such as a slit lamp, indirect ophthalmoscope, ophthalmic ultrasound, computerized tomography, operating microscope, microsurgical instruments, and vitrectomy apparatus. Consequently,

patients with severe eye injuries should never be treated in the field. There, all that can be usefully done is to bandage the injured eye without exerting pressure on the globe itself, give systemic antibiotics such as chloramphenicol (1.0 g) and crystalline penicillin 5×10^6 units intravenously and evacuate the patient to a hospital which fits the above description as soon as possible. The antibiotic dosage should be repeated every 6 h until modified according to the results of the bacterial culture.

Conjunctival Foreign Body

Symptoms and Signs. Feeling of a foreign body, redness, excessive lacrimation.

Treatment. Check cornea, conjunctivae, and behind the eyelids using a light source and if possible magnification. Remove foreign body by rinsing or with a cotton-wool-tipped stick. Give local antibiotics. Do not give local anesthetic except for the examination. Treat pain with oral analgesics.

Corneal Abrasion and Foreign Body

Symptoms and Signs. Pain and the feeling of foreign body especially in the upper eyelid, irregular corneal light reflex, conjunctival redness, and occasionally decreased visual acuity.

Treatment. A superficial foreign body can be removed by rinsing or with a cotton-wool-tipped stick. If a foreign body cannot be taken out easily, the patient should be referred to an ophthalmologist. The eye is bandaged after instilling antibiotic drops.

Eyelid Laceration

Bandage, give systemic antibiotics, and refer the patient to an ophthalmologist. If the cornea is exposed, the area should be kept moist during the evacuation. If corneal exposure is extensive and evacuation considerably delayed, the eyelids may be sutured together to cover the cornea.

Blunt Ocular Trauma

This type of injury may cause intraocular damage which is sometimes difficult to detect and occasionally may not cause an obvious visual disturbance but nevertheless requires treatment. These lesions are hemorrhages into the anterior chamber (hyphema), tears of the iris, changes in pupil diameter and pupillary paralysis, damage to the anterior chamber angle (which may lead to delayed glaucoma), cataract, lens dislocation, vitreal or retinal he-

morrhage, retinal tears and detachment, macular edema, choroidal rupture, and even scleral rupture. All cases of severe ocular injuries require evacuation to an ophthalmologist.

Perforating Injury

Symptoms and Signs. Reduction in visual acuity, soft eye, flat anterior chamber, and the appearance of ocular contents, such as the iris, in the wound.

Treatment. Bandage, give systemic antibiotics, and evacuate urgently.

Intraocular Foreign Body

Suspect an intraocular foreign body in any case of perforating injury of the eye and the surrounding area or when the patient complains of a sudden decrease in visual acuity or a sudden pain in the eye.

Treatment. Bandage, give systemic antibiotics, and evacuate urgently.

Orbital Fracture and Hemorrhage

Facial asymmetry, enophthalmos (eye deep in the orbit), exophthalmos, pain, swelling, disturbances in ocular movement, diplopia, hypoesthesia, or anesthesia in the skin around the eye: any of the above may indicate injury to the orbital contents or bones. The fractures often cannot be diagnosed without an X-ray examination. Bandage and evacuate.

Burn of the Eye

Symptoms and Signs. The eye is red, irritated, swollen, and painful. There is damage to the tissues, notably the cornea.

Treatment. In cases of chemical burn — exhaustive washing of the eye, preferably with sterile saline, but tap water will do. Systemic antibiotics should be given and the patient evacuated while irrigation continues, until all traces of the chemical are removed from the eye. For a thermal burn, apply an eye patch only.

4.6.1 Ear, Nose, Throat and Neck Injury: Part I

I. Eliachar and J. Hayes

Nasal Injuries

Introduction

Although disfiguring and often associated with other injuries, only certain aspects of nasal injuries require prompt attention.

Incidence

The nose is fractured more than any other facial organ. Fracture should be suspected when blunt trauma produces epistaxis and internal or external nasal deformity. Not only the ascending portion of the maxilla and the nasal bones but also the septum and turbinates can be fractured.

Classification

Nasal fractures are classified just like all fractures:

1. Simple vs comminuted
2. Closed vs compound
3. Displaced vs nondisplaced

Diagnosis/Evaluation

Rule out pre-existing deformities. Physical examination is the mainstay of diagnosis. Palpation of the external nose and intranasal examination with good lighting reveals the necessary data for diagnosis and treatment planning. A septal hematoma may become infected and produce cartilage necrosis, purulence, and, through venous drainage, cavernous sinus thrombosis. If swelling of the external nose precludes palpation, diagnosis and definitive treatment are delayed until the swelling resolves. X-rays are of limited value. Nasal fractures heal by fibrous union, so the age of the fracture cannot be determined radiologically.

Signs of a complicated nasal fracture include overflow tearing (epiphora), telecanthus, CSF leak, or meningitis. The nasal airway should be checked for patency bilaterally. Vision and eye movements should be carefully evaluated.

Treatment

On Site Hospital and Medical Aid Station. Epistaxis should be treated as on p. 369. Examination of the nose should be carried out with proper illumination and instruments under topical anesthesia and vasoconstriction (e.g., lidocaine 1% spray and 2% ephedrine or 0.5% Neo-Synephrine drops). Remove foreign bodies and debride only completely devitalized tissue. Septal hematomas are incised and drained and the nose packed. Displaced bones are elevated back into place and the nose packed to control bleeding and support them in position. Antibiotics should be given according to the nature of the injury and whenever nasal obstruction is produced by the injury or by packing in the course of treatment.
Displaced fractures may require an osteotomy 7–10 days after injury to achieve good reduction. External and/or internal splints, nasolacrimal duct stenting, and wiring of fragments in place may be required. Splint the nasal dorsum with tape and plaster cast or prefabricated splint for 7–10 days.

Paranasal Sinus Injuries

Introduction

The paranasal sinuses are air-filled cavities lined with thin mucosa; few injuries to them are urgent. They are, however, surrounded by vital structures:

Maxillary sinus: orbits above, internal maxillary artery posterior
Ethmoid sinus: orbits laterally, brain superior
Frontal sinus: orbit inferior, brain posterior and superior
Sphenoid sinus: carotid artery, optic nerves lateral, pituitary and brain above

The sinuses are a common site of infection under field conditions. Untreated sinusitis may lead to cavernous sinus thrombosis, orbital cellulitis, meningitis, and brain abscess.

Incidence

Sinusitis is common and often follows upper respiratory infection. Penetrating wounds between the upper lip and the eyebrow will frequently encounter a sinus cavity. A sinus cavity which has been penetrated will become infected unless treated.

Classification

Sinus injuries are classified as to the site, etiology, and extent of tissue damage.

Diagnosis

Purulent rhinorrhea indicates which sinus is infected, if the source can be localized. Swelling, tenderness, and erythema over the affected sinus are good localizing signs. Epistaxis can be a sign of injury to any sinus. Clear fluid from the nose may indicate a cerebrospinal fluid leak.

Evaluation

X-rays must be taken to evaluate the sinuses reliably. Opacification of a sinus can be due to infection, blood, mucosal thickening, polyps, cysts, or pus. Air/fluid levels are often found in acute injuries or infections.

Treatment

On Site. Oral antibiotics for sinusitis (e.g., penicillin G or V, 250 mg p.o. 6-hourly, or erythromycin, 250 mg p.o. 6-hourly) along with local treatment to the nose with irrigation and decongestion. For epistaxis secondary to sinus injuries, see p. 369.

Medical Aid Station. Continue antibiotics and local treatment such as vasoconstriction of nasal mucous membranes, nasal lavage, or sinus irrigation. Evaluate the patient for signs of complications, such as ethmoid sinusitis extending into the orbit or meningitis.

Hospital. Trephine, evacuate, and drain or obliterate sinuses as appropriate. Explore surgically, scrape off infected mucoperiosteum, and check for damage to dura mater, cribriform plate, and orbit. Realign fractures, ensure drainage of involved cavities, and pack for hemostasis and support. Antibiotic treatment may be appropriate. Examine teeth. Remove foreign bodies and bone fragments from infected sinuses. Conservatively debride skin and bone, attempt preservation of periosteum, suture lacerations, and re-

align anatomical features, particularly around the eyes, inner and outer canthi, nostrils, and lips. *Consult* ophthalmologist, dentist, and neurosurgeon.

Epistaxis

Introduction

Epistaxis can be simple or complicated. Simple bleeding arises from the anterior septal area and responds to a few minutes of pressure in the sitting position. Complicated bleeding is the main subject of this section.

Incidence

Trauma causes only about 10% of nose bleeds but may have the most serious blood loss. Predisposing conditions include low humidity, septal perforations, sinusitis, blood dyscrasias, advanced age and atherosclerosis, hypertension, and tumors.

Classification

Epistaxis should be classified as:

1. Unilateral vs bilateral
2. Anterior or posterior or both
3. Volume and rate of blood loss: minor, moderate, severe, massive
4. Etiology, e.g., nasal fracture, wound, base-of-skull fractures

Diagnosis

The patient's head is held elevated if other injuries and vital signs permit. Using adequate lighting, secretions and blood are cleared as well as possible. The nasopharynx is examined for the presence, laterality, and quantity of blood issuing from behind the nose.
Note the side of anterior blood loss. If possible, the mucosa should be vasoconstricted and suctioned free of blood and secretions and the bleeding points identified directly. This is often impossible in a brisk nose bleed.
Other injuries, if present, will be obvious from inspection of the skull, face, head, and neck. Arteriography can be used to localize the larger blood vessels for direct treatment if ligation is needed. The nose is supplied by numerous small vessels from both the external and internal carotid arteries with communicating anastomoses.

Evaluation

The critical elements in triaging epistaxis are:

1. Volume and rate of blood loss
2. Accessibility of bleeding points to cautery, tamponade by packing, and indication for ligation of vessels

Severe, massive, or unrelenting moderate epistaxis requires an anterior and posterior nasal pack to tamponade the bleeding. Lesser epistaxis usually responds to an anterior pack alone.
Major blood loss can be occult if the patient swallows the blood pouring down the nasopharynx. Tracheotomy or endotracheal intubation may be required to keep the patient from "drowning" in his own blood in cases of massive bleeding epistaxis, as in base-of-skull fractures. Epistaxis may lead to shock and exsanguination.

Treatment

On Site. Minor epistaxis is usually self-limited. Keep patient sitting with his head erect. Apply digital pressure laterally from the level of nasal bones to nostrils for 5–10 min. Reapply as necessary. Pack nose with cotton or gauze strips and do not remove for 24–36 h. Saline or decongesting nose drops are helpful (e.g., ephedrine 2% or Neo-Synephrine 0.5%).
For moderate epistaxis, the initial treatment is the same as for minor epistaxis. If unsuccessful, vasoconstrict with 1% ephedrine or oxymetazoline nose drops, then place an anterior nasal pack of strip gauze soaked in an antibiotic ointment (e.g., bacitracin). Use nasal forceps to layer the gauze from the floor of the nose upward. The gauze must be placed at least 5 cm deep to the nostril in an adult, and a meter or more may be required to achieve a tight anterior nasal pack. Desiccated sponges or small vaginal tampons swell when they imbibe fluid and may be used as anterior packs.
In severe epistaxis with high flow rates and large volume loss, a very tight anterior pack must be placed to tamponade the bleeding effectively. To achieve the required tension, a posterior nasal pack is placed to provide a barrier against which to build a tight anterior pack. This procedure is usually performed in the medical aid facility. Evacuate the patient with the head elevated or extended according to his condition and other injuries; replace fluids.

Medical Aid Station. Minor epistaxis will have resolved by the time it is seen at the primary care facility. The nose should be cleared of secretions and blood, examined under good lighting, and treated as appropriate. Cautery, either electric or chemical (silver nitrate), may be applied (Fig. 1).

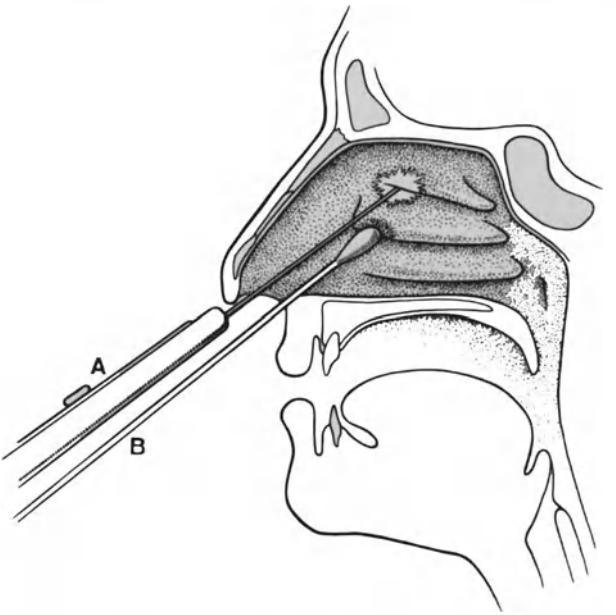


Fig. 1. Lateral view of the nasal cavity, demonstrating electrocautery (*A*) and application of silver nitrate (*B*) to control bleeding. The same may be applied to the nasal septum

For moderate epistaxis leave packs in for 48 h or more. When removing packs topically, decongest the nose with ephedrine 2%, cocaine, oxymetazoline, etc. Replace blood loss; monitor pulse and blood pressure.
In severe epistaxis, if bleeding persits despite posterior nasal packs, remove all packs and replace them with a new, tight, anterior and posterior nasal pack. Two readily available techniques can be used to form this barrier:

1. Slide a large Foley catheter through the affected nostril until it enters the nasopharynx. Fill the balloon with 15–30 cc of water. Pull the catheter back through the nares until the balloon is firmly seated in the posterior nasal opening (choana). While holding gentle tension on the catheter, place a tight anterior pack. Wrap a 4×4 gauze around the catheter at the nostril, and clamp the catheter with a flow-control wheel valve or a hemostat. The gauze cushion protects the nose from pressure trauma by the valve or the hemostat (Fig. 2).
   When available, use a special nasal inflatable balloon designed to adapt to the inner nasal anatomy when injected with water (Fig. 3). Such sim-

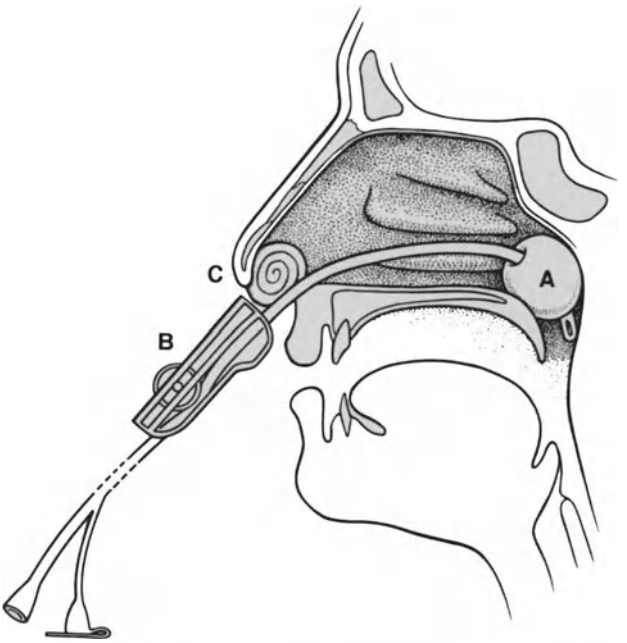


Fig. 2. The Foley catheter shown in position, with the balloon inflated within the choana. The anterior pack may be either limited to the nasal vestibule or extended deeper; it protects the nostrils against pressure trauma. When using the flow-control wheel lock, thread the catheter through it prior to its insertion into the nose

ple balloons placed under field conditions will most often tamponade the bleeding enough for transport to more sophisticated facilities.

2. Fold two pieces of 4×4 gauze cotton or lamb's wool into a square or sphere, then tie them into this shape on the end of a 100-cm heavy thread (#1 or #2 silk). Pass a narrow catheter through the nose until it is visible in the nasopharynx. Grasp it with an instrument, and bring the end through the mouth just enough to tie the free end of the heavy thread to the catheter. Pull the catheter back through the nose. This delivers the free end of the thread through the mouth and out of the nostril. Repeat on the other side.

   Guide the pack through the mouth, posterior to the soft palate, then upwards into the choana. While holding tension on the thread, place a tight anterior nasal pack as above. When the nose is anteriorly packed, a rolled gauze sponge can be tied to the thread at the nostrils to prevent the posterior packs from slipping back into the pharynx and obstructing the airway (Fig. 4).

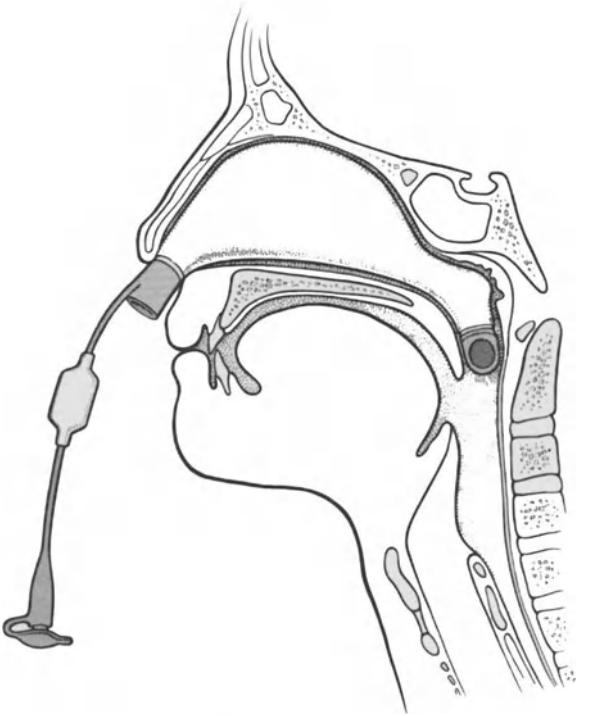


Fig. 3. Lubricate and insert the balloon on the floor of the nose until the tip shows up in the nasopharynx. Inflate partially, preferably with water; pull back slightly for proper adjustment; add more pressure and secure

Individuals requiring a posterior nasal pack should be evacuated to a hospital or regular medical facility for monitoring. Nasal packs should be left for 48 h or more. Provide oxygen and humidification.

If bleeding continues, urgently evacuate the patient to a hospital for angiography, embolization, or ligation of the appropriate vessels. Monitor and replace blood loss.

Hospital Care. Minor epistaxis rarely requires hospital care. Moderate epistaxis is treated as above. With severe epistaxis, if the expertise is available, the internal maxillary artery and the anterior and posterior ethmoid arteries can be embolized or ligated separately if an arteriogram demonstrates an isolated bleeding point; otherwise, uncontrolled bleeding may require an external carotid ligation.

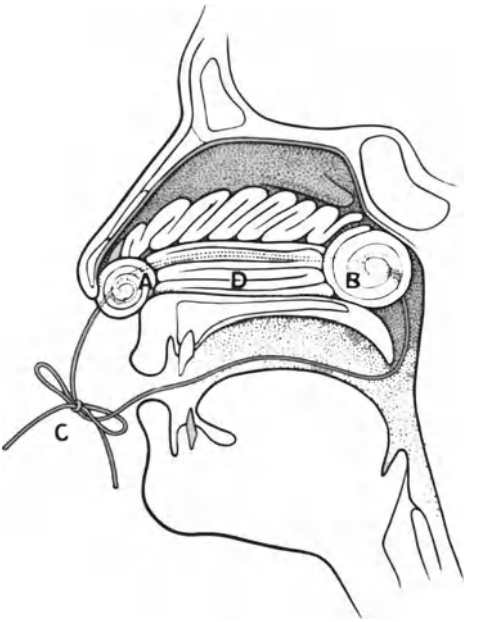


Fig. 4. The final position of the anterior and posterior nasal packs (see text)

Guidelines

1. Patients with nasal packs for more than 24 h should be given antibiotics such as penicillin or erythromycin due to the rapid development of otitis media and/or sinusitis when the ostia and eustachian tubes are occluded.
2. When posterior packs are used, attention must be paid to potential pressure necrosis of the nasal alae due to traction by the device which is pulling the posterior pack anteriorly and, therefore, riding against the skin of the nasal rim. Skin necrosis at the area can occur rapidly and is difficult to repair. Excessive pressure on the alae should be diffused by the use of soft cushioning between the thread or catheter and the skin. The cushion should be inspected daily, and the skin integrity under it should be assessed.
3. Packs should be removed no sooner than 24 h and almost always require at least 48 h to be fully effective. Packs should be removed in stages beginning with about one-half of the anterior pack, followed by several hours of observation for resumption of bleeding, and then removal of the remaining anterior pack. Gentle pulling on the strip gauze at 90° from the plane of the face will remove the gauze. If bleeding resumes

briskly, the nose is repacked and another 48 h are allowed for effective tamponade. The posterior pack is removed last since it is the most difficult to replace. To remove it, the string is freed from the anterior retaining gauze, and the posterior pack is visualized in the nasopharynx or oropharynx and removed with forceps. Slight bleeding will often accompany removal of the posterior pack due to irritation of the nasopharyngeal mucosa. This bleeding almost always stops spontaneously.

4. Note that the patients with nasal packing in place may become hypoxic due to suppression of the "nasopulmonary reflex", by which nasal obstruction and absence of nasal ventilation acts through the central nervous system to interfere with pulmonary functions. Patients with CNS trauma and under the effect of narcotics must be closely observed for potential respiratory arrest when packed. The same is true with regard to the aged and patients with chronic obstructive pulmonary disease. Oxygen and humidification must be administered.

Ear Injuries

The ear is very susceptible to injury. The vascular and nervous supply to the ear is abundant. Bleeding may be profuse, and anesthesia (regional or local) is required. The perichondrium provides blood supply to the cartilage and may revitalize severely damaged tissue. Debridement ought to be minimal.

External Ear and Ear Canal

Introduction

Injuries include damage to the pinna, external autitory canal (EAC), otitis externa, auricular hematoma, and perichondritis. The ear is particularly exposed to the elements. Thermal trauma is divided into frostbite and high-temperature burns.

Cold exposure. Tissue fluids may crystalize very quickly in the exposed ear. The condition is deceiving, as only pallor and numbness may be apparent. Full extent of the damage should be assessed after the ear has been rewarmed.

High-temperature burns. Fire, gasoline, chemical, electrical, combustion, and steam injuries may cause various degrees of burns. First- and second-degree burns usually heal well when conservatively treated. Third-degree burns with devascularization of the cartilage often result in perichondritis and severe deformity.

Incidence

The external ear is often injured in combat; trauma may be mechanical or thermal. The wound is often contaminated.

Classification

1. *Injury to the pinna:* Assess by percentage still attached, depth of laceration (i.e., through which layer), depth of burn, location of injury on pinna
2. *EAC injury:* Determine nature of obstruction, debris, swelling, etc.
3. *Otitis externa:* Determine patency of canal, drainage, spread of erythema, and infection beyond ear
4. *Auricular hematoma:* Describe location, size, tension, and secondary infection

Diagnosis/Evaluation

Most external ear injuries are obvious. The EAC should be cleaned and suctioned under vision (but not irrigated) to rule out or assess middle ear injuries. Antibiotics should be considered in keeping with the nature of the injury. Particular attention should be given to auricular hematoma. This subperichondrial collection of blood, which dissects the perichondrium off one or both surfaces of the cartilage, can lead to cartilage necrosis and external ear deformity ("cauliflower ear") if not treated promptly.

Treatment

On Site

Avulsion of the pinna: If the ear is still attached by more than 10% of its base, place it in a natural position, apply a mastoid dressing, and evacuate to a field hospital. If the ear is completely amputated, wrap the amputated part in saline-soaked gauze and evacuate the patient to a facility where the ear can be buried in a subcutaneous packet to nourish the cartilage before reattachment is attempted.

Laceration: Clean wound adequately. Suture skin without suturing through cartilage. If perichondrium is lacerated, reapproximate the layers and allow the skin sutures to hold them in place.

Burns: Clean wound well. Apply burn ointment.

Frostbites: Rewarm with moist compresses, 37°–45°, for 15 to 30 min. Apply burn dressing with moderate pressure.

Medical Aid Station
Avulsion: Suture partially avulsed pinna back into place. Splint in place with moist cotton molded to ear shape, then apply mastoid dressing.
Laceration: As above.
Burns: Debride minimally. Monitor for development of chondritis which can rapidly dissolve the cartilage.
Auricular hematoma: If sterile conditions are available, including needle evacuation of the blood, then place moist cotton balls or dental rolls in the crevices of the pinna molded to the natural shape of the ear. A tight mastoid dressing is then applied to reapproximate the perichondrium and the cartilage (Fig. 5).

Hospital
Avulsion: Examine and clean the ear under the surgical microscope. If pinna is completely avulsed or is nonviable, detach it. Dermabrade off the epithelium, and bury it under a neck skin flap. Elevate buried pinna in 7–10 days.
Laceration: Primary closure may still be obtained of full-thickness loss of portions of the pinna (up to one-third) by rotating the defect edges and suturing together after careful debridement. This should be attempted only when the wound is fresh (up to 24 h); otherwise, the edges should be allowed to heal, then rotation accomplished on a delayed basis.
Burns: Incise and drain areas of chondritis. Frequent warm soaks in antibiotic solutions (e.g., neomycin 0.05%) may prevent spread of chondritis.
Auricular hematoma: Sterile incision and curettage of any remaining or recurrent blood clots followed by suturing a molded bolster on each side of the pinna to maintain the layers together more forcefully than with a mastoid dressing. Systemic antibiotics active against *Staphylococcus* should be given (Fig. 5).

External Canal Injuries

On Site. Foreign bodies can be removed carefully by passing a small right-angle ear hook medial to the object and "raking" it out. Irrigation should be avoided since water may enter the middle ear via perforations, or hydrophilic material may swell completely, occluding the canal. Insects may be removed by paralyzing them with alcohol, lidocaine, or ether dropped into the EAC, then removing them with a hook as above.
Lacerations of the EAC should be gently cleaned and kept dry, and antibiotic drops containing neomycin and polymyxin B should be applied three times a day.

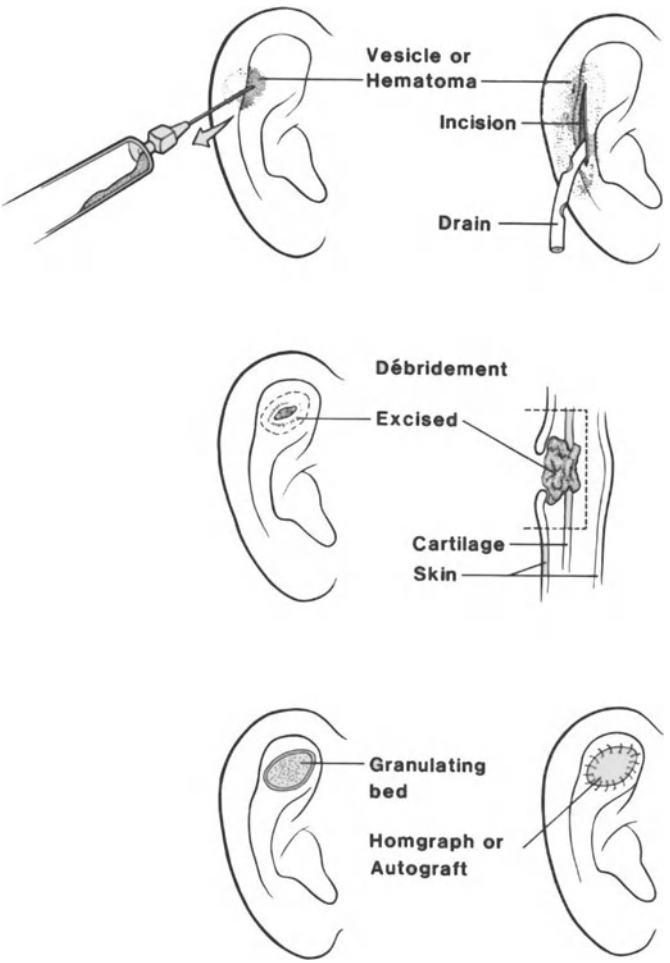


Fig. 5. Management of auricular vesicles or hematoma. Aspirate contents. If incomplete, incise the overlying skin or excise it; curette blood clots and necrotic tissue; drain and pack the ear with molded dressing. When the wound has granulated, graft the defect

Medical Aid Station. Obstructions of the external canal should be removed and the ear canal cleaned under direct vision to prevent external otitis.

Hospital. Only fracture of the anterior canal wall (glenoid fossa) requires hospital treatment. This is usually associated with mandibular trauma.

Carefully check for an intercranial injury associated with a canal fracture and assess the facial nerve. Clean the canal under the microscope and apply antibiotic solutions or ointments as necessary. An ear wick soaked in antibiotic and corticosteroid ointment or cream, introduced gently, under vision, may be applied to reduce local obstructing edema and swelling. It should be changed every 24 h (Fig. 6).

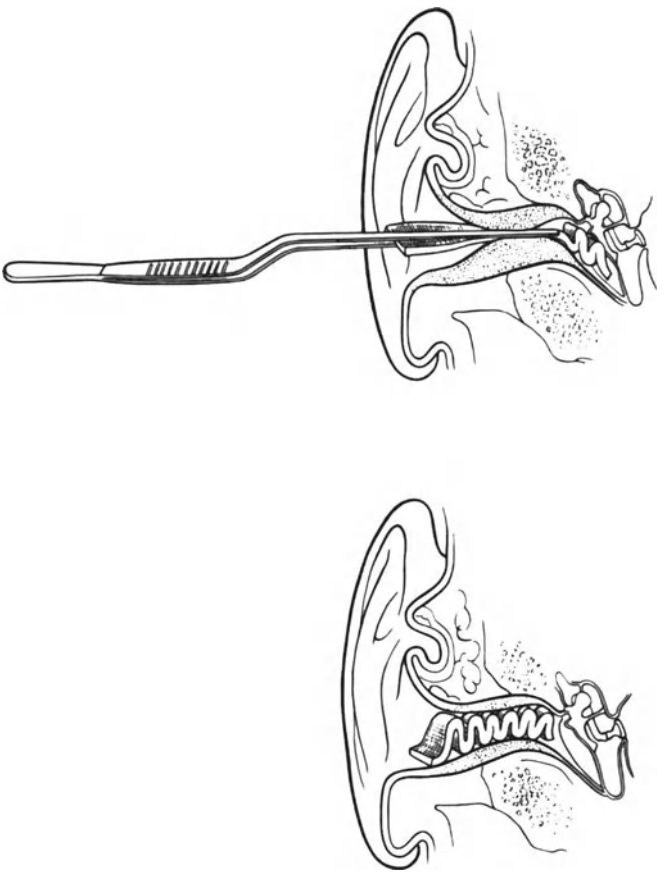


Fig. 6. Insertion of an ear wick into the external ear canal. When soaked with antibiotics and corticosteroids, it reduces inflammation and controls infection

Middle Ear Injuries

Introduction

The middle ear is less accessible to injury that the external ear but can be a source of debilitating injury. Barotitis is common in paratroopers and airmen. Otitis media often accompanies upper respiratory infection; blast injuries can produce tympanic membrane (TM) perforations; and penetrating objects, shrapnel, and blasts can injure the delicate ossicles.

Classification

Injuries to the middle ear are described by:

1. Site and size of perforation of TM
2. Presence of fluid in middle ear (e.g., clear, blood, pus)
3. Degree of hearing loss

Diagnosis

After the EAC is cleared of blood, debris, or pus, the TM and middle ear are examined by otoscopy. A hearing test is done when available. Tinnitus and otalgia are common symptoms. Check for signs of intracranial injury and facial nerve abnormality. Dysequilibrium, in the form of unsteadiness, dizziness, or vertigo (in ascending order), may be associated with vomiting and immobilize the patient.

Treatment

On Site

Barotitis: Have patient "clear" ears by gently blowing nose with the nostrils pinched or by repeated swallowing maneuvers. Topically decongest nose (ephedrine 2%, Neo-Synephrine 0.5%, or oxymetazoline 1% drops). If the patient is debilitated, the TM can be pierced with a small needle in the anterior-inferior quadrant to relieve the negative pressure in the middle ear.

Otitis media: Antibiotics given orally require 24 h to provide relief. If the TM is bulging, it may be incised with a myringotomy knife in the anterior-inferior quadrant for immediate relief of pain; evacuate the pus and ventilate the middle ear.

Perforation of TM (traumatic): Clean EAC of any debris. Remove foreign bodies. Keep water out of ears until TM has healed.

Ossicular injuries: No field therapy other than as for TM perforations. If associated with vertigo, treat as for inner ear trauma.

Medical Aid Station. As above, except patient should be evaluated for complications such as meningitis, facial nerve injury, mastoiditis, perilymph fistula, and inner ear injury.

Hospital. Examine and treat ear under the surgical microscope; carry out hearing tests.
Barotitis or otitis media: Consider myringotomy with ventilating tube if problem is persistent or recurrent.
TM perforation: Most traumatic perforations close spontaneously, but the success rate may be improved with a "cigarette paper patch" placed on the TM and impregnated with ointment to align and provide an adhesive framework for the mucosal fragments which heal more rapidly.
Ossicular injuries: These require a restorative operation (tympanoplasty) at a later date and after adequate hearing tests. Exclude water from the ears and treat any other conditions such as secondary infection.

Inner Ear Injuries

Introduction

Blast injuries, chronic or sudden exposure to loud noise, otitis media, and head injuries can cause damage to the inner ear function of hearing and balance. Both functions can be disturbed in an acute injury, although noise exposure rarely produces marked balance loss. Severe blast injury may deafen the ears, perforate the TM, and cause debilitating vertigo, pain, and tinnitus.

Incidence

Hearing loss is common in war veterans. Initially, most patients with noise-induced acute injuries will recover part of their hearing (temporary threshold shift) but will eventually develop persistent hearing loss at the higher frequencies (>2 KHz), a permanent threshold shift. Penetrating injuries of the inner ear are caused by small or large particles of high-velocity shrapnel, and a CT scan is useful in tracking their path.

Classification

Inner ear injury is classified by site and severity of injury:

1. Unilateral vs. bilateral
2. Hearing affected, balance affected, both
3. Degree of hearing loss

Diagnosis

Inner ear injury is initially associated with symptoms of hearing loss, dizziness (spinning vertigo), or tinnitus. Nystagmus is the only physical sign. Hearing tests need not be sophisticated to be reliable, but formal audiology and vestibular function testing are required to quantify losses.
Otoscopy should be carried out. The presence of a hemotympanum, TM perforations, blood, or CSF otorrhea are suggestive of inner ear injury when associated with symptoms. Evaluate all patients for facial nerve dysfunction, temporal bone fracture, and perilymph fistula.

Treatment

On Site. Patients with noise-induced hearing loss of more than a minor degree should be removed from noisy environments or wear hearing protection if practical. Patients with nystagmus, tinnitus, and hearing loss from blast injury should be evacuated (especially if vomiting and prostrated).

Medical Aid Station. Look for otorrhea, nystagmus, and hearing loss. Give vestibular suppressants (diazepam, 5 mg p.o. or i.m., meclizine, 25 mg p.o.) and antibiotics parenterally as appropriate.

Hospital. Perform complete neurological evaluation with X-ray studies (CT) and hearing and balance tests. Patients suspected of having injuries to the oval or round windows (inner ear fluid system) should be placed on bed rest, with the bed flat, and given vestibular suppressants. Most fistulae of the inner ear fluid system close within 5–7 days. Surgical exploration of the ear may be undertaken if signs of meningeal irritation appear.

Trauma to the Facial Nerve

Introduction

Facial paralysis is a most deforming motor-neural injury resulting in ocular and oral complications with severe social, psychologic, and emotional im-

plications. Prompt treatment may promote rehabilitation. Delay in diagnosis and management may result in an irreversible paralysis. This chapter is concerned with facial nerve injuries distal to the brain stem. Adequate knowledge of the anatomy contributes to successful management (Fig. 7)

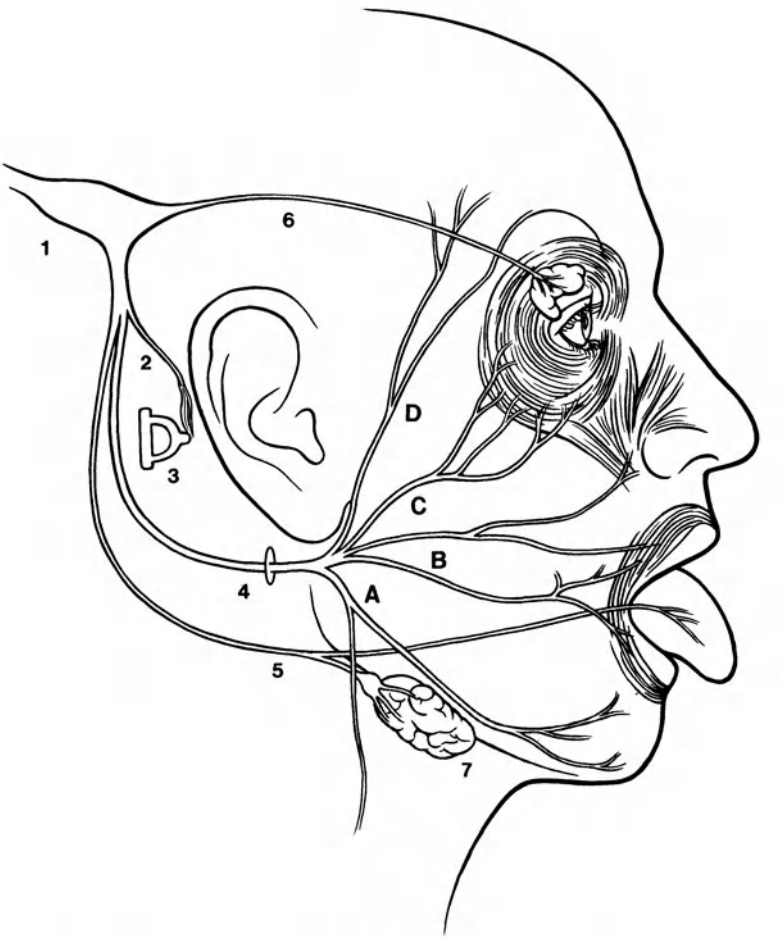


Fig. 7. Schematic anatomy of the facial nerve: *A*, mandibular branch; *B*, buccal branch; *C*, zygomatic branch; *D*, frontotemporal branch; *1*, geniculate ganglion; *2*, branch to stapedius muscle; *3*, stapes; *4*, stylomastoid foramen; *5*, chorda tympani; *6*, greater petrosal nerve; *7*, submandibular ganglion

Incidence

Intracranial trauma to the 7th nerve within the temporal bone is more common than extracranial.
Longitudinal fractures of the petrous bone are more common than transverse.

Classification

1. Open penetrating wounds of ear and temporal bone
2. Closed fractures of the temporal bone (base-of-skull fractures) (Fig. 8)
   Longitudinal (facial paralysis in 20%–25%): due to parietal and temporal blows. Fracture line runs parallel and anterior to petrous ridge and traverses the tympanic annulus, producing hematorrhea and conductive hearing loss
   Transverse (facial paralysis in 50%): blows to occipital area; fractures perpendicular to petrous ridge. Traverses otic capsule causing unilateral sensorineural hearing loss. Tympanic membrane intact with hemotympanum
   Mixed (paralysis in over 50%)
3. Extratemporal, penetrating, facial or head and neck wounds, especially lacerations and penetrating wounds over cheek and parotid gland

Diagnosis

Hemifacial paralysis without sparing of ipsilateral forehead. Eye open, tearing, Bell's phenomenon, corner of mouth droops, drooling. Do not relate lack of movement to local swelling and hematoma. Facial movements are best examined when the patient is awake and conscious. Records of initial post-traumatic condition are critical for management. X-ray and CT examinations provide localization of site of lesion and tract of trajectory.
Note blood and/or CSF drainage from ear. Look for ecchymosis over mastoid process, suggesting fracture of skull base — "Battle sign."
Electrodiagnostic techniques, CT scans, audiology and balance tests must be employed in the hospital for timing of surgery and for topographic diagnosis.
The majority of intracranial facial paralyses recover spontaneously when not associated with actual direct laceration or avulsion of the nerve, especially if onset of paralysis is gradual. Paralysis following blunt tissue trauma often recovers spontaneously; it must be monitored by otologic, neurologic, and electrodiagnostic tests.

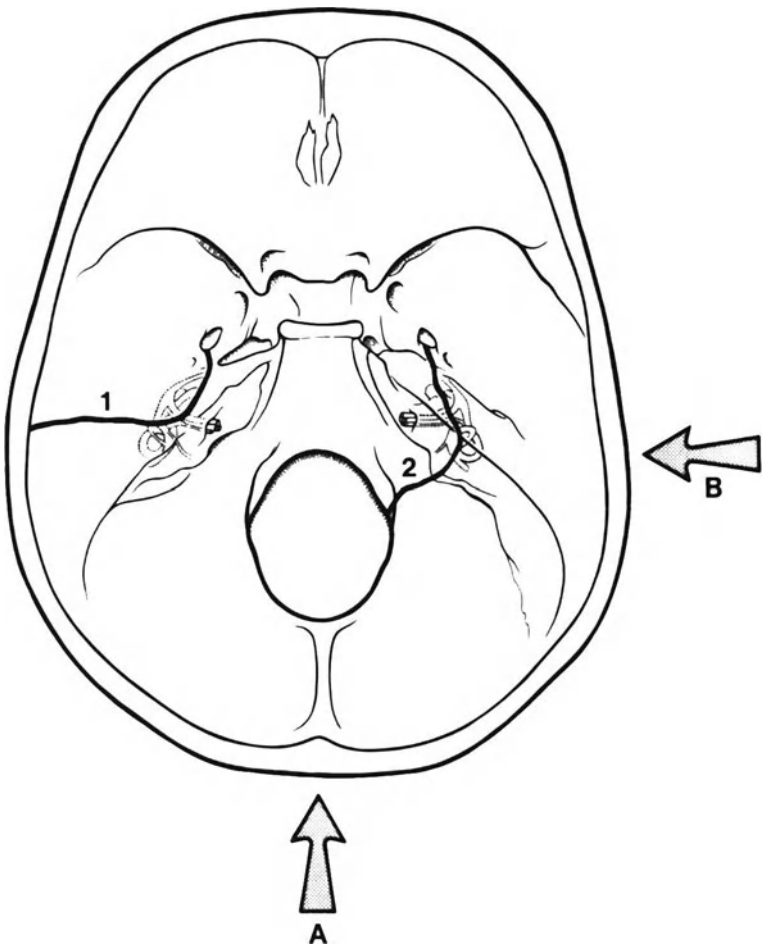


Fig. 8. Transverse fracture of the temporal (*2*) bone caused by a blow (*A*) on the occipital area. Longitudinal fracture (*1*) caused by a blow to the parietotemporal area (*b*)

Treatment

Microsurgical restoration of continuity by end-to-end anastomosis or grafting is preferred over substitution surgery.

On Site. Prompt diagnosis (if patient loses consciousness, it may be impossible). Stop bleeding by pressure — do not pack ear. Record paralysis or paresis. Moisten, cover, and protect the eye.

Primary Medical Facility. Do not probe wound or clamp vessels, even to stop bleeding, unless life-threatening. Do not debride tissue in parotid region. Gently dress wound, keep well moistened. Hydrate and protect eye with patch.

Hospital. Do not inject local anesthesia into wound before the main stem and branches of the facial nerve are located. Ask anesthetist not to paralyze the patient for general anesthesia.
Repair by end-to-end anastomosis or by nerve grafting is best performed within 36 h after injury, before Wallerian degeneration occurs. If repair is impossible, tag the distal segment of the nerve within the open wound with silk sutures to aid in localizing the nerve at a later date.
Immediate attempts to perform end-to-end anastomosis or grafting should be carried out once the patient is stable. All nerve branches 1 mm in diameter and greater should be repaired or grafted. Smaller branches will regenerate. Ophthalmologic consultation should be obtained to protect the eye from acute or chronic injury.

4.6.2 Ear, Nose, Throat and Neck Injury: Part II

I. Eliachar and J. Hayes

Injuries to the Neck

Introduction

This chapter deals with trauma of the anterior and lateral cervical triangles (Fig. 1). Note the following points:

1. The neck is separated into compartments by strong fascial envelopes. The critical organs in the anterior and lateral triangles are mobile and protected by strong muscles and fibroelastic connective tissue; thus, approximately half of the patients with penetrating neck trauma have no significant injury.
2. Blunt neck trauma may result in severe tissue injury. The primary consideration is the maintenance of an adequate airway.
3. Open or penetrating wounds of the neck are frequently associated with significant vascular involvement. An expanding mass within the neck may be a sign of vascular injury.
4. Airway patency can be jeopardized by a progressive soft-tissue swelling due to hemorrhage, edema, infection, or subcutaneous emphysema.
5. Bleeding from the mouth may indicate mid-neck vascular (e.g., carotid) disruption.
6. Gunshot wounds produce a wide track of damaged tissue, often requiring segmental resection and end-to-end anastomosis or grafting of the injured vessel or organ.
7. An obstructed airway or hypovolemic shock requires immediate surgical exploration.
8. Stable patients should be constantly observed and examined carefully for evidence of trauma to the vascular, neural, airway, or digestive tracts.

Multidisciplinary cooperation is imperative. Angiography, contrast studies of the hypopharynx and the esophagus, and CT with endoscopy examinations are performed. If these diagnostic facilities are lacking, it is prudent to explore the neck whenever there is suspicion of a deep visceral injury.
The ballistic properties of the missile and its tract are important, but they may be misleading. Close clinical observation with a high index of suspi-

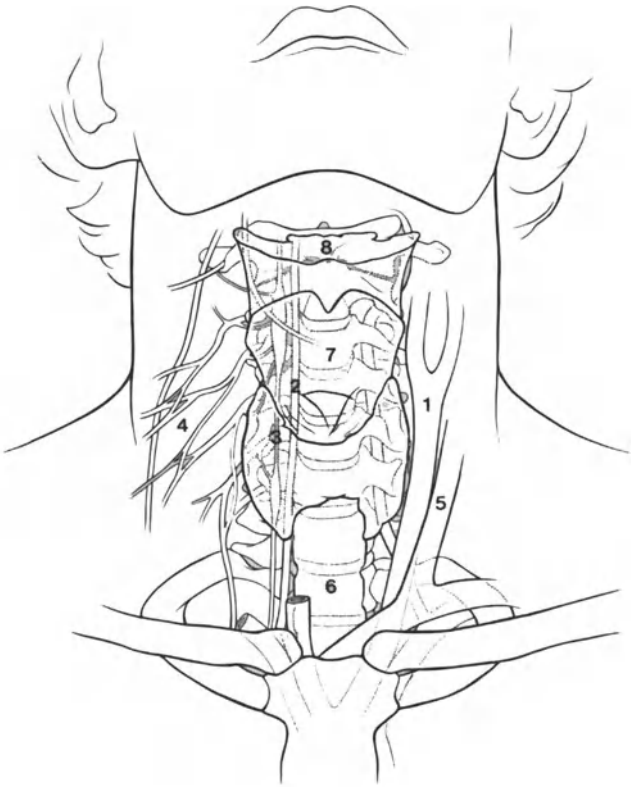


Fig. 1. Vital structures in the anterior and lateral cervical triangles: *1*, carotid artery bifurcating into its internal and external branches; *2*, vagus nerve; *3*, cervical sympathetic ganglion chain; *4*, cervicobranchial plexus; *5*, internal jugular vein; *6*, trachea; *7*, thyroid cartilage — note thyroid gland below it; *8*, hyoid bone

cion, together with sophisticated diagnostic techniques, determines the management.

Incidence

Over 95% of injuries to the great cervical and thoracic vessels are due to penetrating wounds. Judicious exploration, modern diagnostic capabilities, rapid transportation, and the availability of well-trained personnel have currently reduced the mortality of these wounds to less than 5%.

Classification

Classification is by anatomic compartment, depth of penetration, and the organ system involved (vascular, neural, muscular, spinal, air, and digestive tracts).

Diagnosis

On Site and First Aid Station. Clinical signs and symptoms:
Airway injury: hoarseness, stridor, respiratory distress, coughing, hemoptysis, subcutaneous emphysema, cyanosis
Pharynx and esophageal trauma: odynophagia, dysphagia, hematemesis, food and saliva in wound, subcutaneous emphysema
Nerve injuries: cervical or brachial plexus lesions, Horner's syndrome, deviation of the tongue, pharyngeal "curtain" sign; superior or recurrent laryngeal nerve injuries; facial nerve paralysis; diaphragmatic paralysis — phrenic nerve
Cervical spine injuries: Baseline neurologic examination followed by periodic checkups should detect deteriorating neurologic status (see chapter on spinal cord trauma)
Vascular injuries: may present as shock, active local bleeding, thrills or bruits, pulse deficits, expanding hematoma, and chylous leak
One-third of patients with damage to major neck vessels do not have distinct clinical signs; hence, the presence of normal pulses may be misleading.

Hospital. When time and conditions allow, plain films of the neck reveal organ and air column displacements and the track of the missile. A chest X-ray may demonstrate a widened mediastinum, dissecting emphysema, pneumothorax, hemothorax, and spinal column fractures. Arteriography, contrast studies, and endoscopy examinations, when available, are rapidly performed in every patient with equivocal signs and symptoms suggesting spinal, vascular, airway, or pharyngoesophageal injuries.
Fracture of the hyoid bone suggests trauma to the larynx or hypopharynx.
The esophagus is examined radiologically and/or endoscopically for lacerations.
Sternal, clavicular, and rib fractures may indicate the presence of crush injuries with severe vascular and visceral injuries.
Potential cervical spine injuries and trauma to the spinal cord must always be kept in mind. Until these are ruled out, the head and neck must be immobilized in the neutral intermediate position. Cross-table lateral views of the cervical spine should be performed prior to any change in neck position, such as for elective tracheostomy.

Treatment

On Site and First Aid Station. Cradle head and neck (Fig. 2). Ensure unobstructed airway (Fig. 3). If the airway is obstructed, intubate or perform cricothyroidotomy or tracheostomy as indicated. Insert one or more IV infusions (in lower extremity if you suspect subclavian transection). External bleeding should be controlled by digital pressure and tamponade. Avoid blind probing or clamping. Promptly transport directly to hospital, if possible with competent and well-equipped medical attendant.
If pneumothorax is coexistent, insert chest tubes.
Do not debride wound. Administer antibiotics and oxygen as indicated.

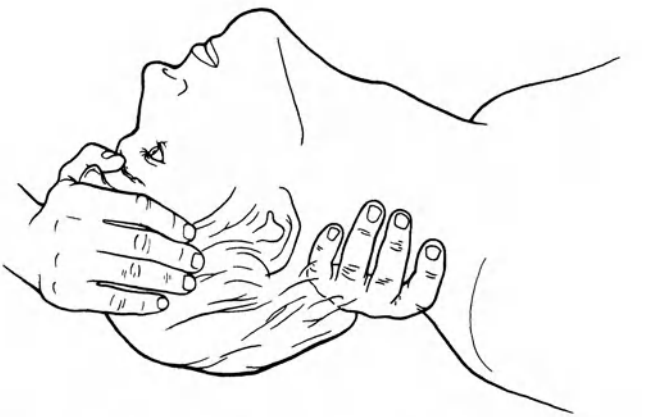


Fig. 2. Carefully support and stabilize the neck using doughnut-shaped headrests and bolster bilaterally

Fig. 3. In the absence of airway obstruction, of significant cervical bleeding and in the supine position with the head turned sideways, the victim may be prepared for transportation. This position prevents aspiration in the event of vomiting

Hospital. Secure airway; perform chest and cervical X-ray examinations to rule out vertebral fractures; provide oxygen, blood, fluids, and antibiotics.

In the presence of obvious airway obstruction and/or massive bleeding, rush to the operating room; explore the neck and repair major vessels as indicated. With hemothorax, anterolateral thoracotomy must be performed to control retrosternal or subclavian bleeding. Otherwise, perform arteriography, contrast esophageal studies, and endoscopy. Observe or intervene as indicated. Observe patient closely until completely stable from the ventilatory, vascular, and neurologic points of view. Be ready to repeat studies or take immediate action as may become indicated.

Definite or suspected airway, vascular, or visceral injuries should be surgically explored.

Transoral bleeding can be controlled by pharyngeal packing; however, intubation or tracheotomy is mandatory prior to placement of the pack. Vaginal packing with a long instrument (e.g., a Kelly clamp) can be used.

A team approach by neuro-, vascular, plastic, and maxillofacial surgeons together with the otolaryngologist is preferred.

Summary of Indications for Surgical Exploration of Neck Wounds

1. Airway obstruction:
   Acute or progressive respiratory distress
   Laryngotracheal separation
   Laryngeal fracture, open or closed
2. Vascular injury:
   Immediate indications:
      Airway obstruction due to hematoma
      Hemorrhage from neck wound
      Expanding cervical hematoma
      Active transoral bleeding without visible source
      Widened superior mediastinum
      Positive angiogram
      Absence of main cervical arterial pulses
      Progressive CNS deficit (hypoperfusion of the brain suspected)
   Delayed indications:
      Major vessel thrombosis
      Occult hemorrhage
      Arteriovenous fistula
      Aneurysm or pseudoaneurysm
      Vertebral artery injury
      Thoracic inlet injury
      Chyle leak
      Bruit

3. Neurologic (indications delayed unless associated with airway or vascular injuries)
   Jugular foramen syndrome (deficit of 9th, 10th, and 11th cranial nerves)
   Parotid and submandibular space injury with deficit to 7th and lingual nerves
   Horner's syndrome
   Brachial plexus deficit
   Hemiplegia
   Diminished visual acuity or contraction of visual fields
4. Respiratory and digestive tract injury
   Late indications:
   Progressive respiratory distress
   Displacement, disfigurement, and crepitance of hyoid, thyroid, and cricoid cartilages
   Progressive aphonia, dysphonia, or dysphagia
   Neck infection/abscess formation
   Mediastinitis
   Positive endoscopy — indicating perforation of the hypopharynx or esophagus
   Positive contrast study of aerodigestive tract

Injuries of the Larynx and Cervical Trachea

Introduction

The larynx is the vulnerable bottleneck of the airway, serving as a protective valve for the lungs. Airway obstruction must be assessed and addressed immediately. Coincident hemorrhage, hematoma, subcutaneous emphysema, and edema should be monitored and managed promptly. Sensory and motor dysfunction of the vocal cords is as dangerous as mechanical occlusion. Respiratory distress may progress to sudden obstruction and respiratory failure.

Blunt or penetrating trauma, gunshot or shrapnel wounds, inhalation of smoke or toxic fumes, and ingestion of caustic agents may cause severe damage of the airway.

Patients with multiple diverse injuries may require prolonged surgical procedures and medical care after establishment of the airway. Do not forget or overlook laryngotracheal reconstruction, especially in blunt cervical injuries (Fig. 4). Laryngotracheal stenosis is an ominous sequel of bad or unsuccessful management with lifelong implications. Its extent may not be recognized until decannulation is attempted.

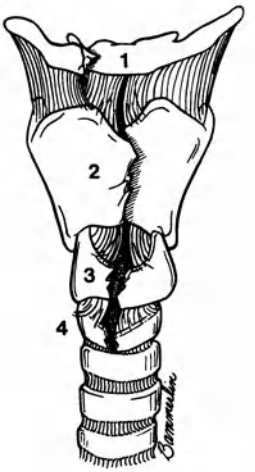


Fig. 4. Fractures at the various levels of the larynx and the trachea: *1*, fracture of the hyoid bone often associated with pharyngeal perforation and/or lacerations, supraglottic laryngeal trauma; *2*, fracture of the thyroid cartilage, usually involving the vocal cords at the glottic level — the voice will be hoarse and the airway markedly obstructed; *3*, fracture of the cricoid cartilage, often leading to severe laryngeal stenosis; *4*, cricotracheal fractures — may result in laryngotracheal separation

Classification

1. Level of injury: supraglottic, glottic, infraglottic; concomitant
2. Injuring agent: penetrating or blunt; gunshot wounds and/or shrapnel; burns and caustic injuries
3. Laryngotracheal separation
4. Damage to superior and/or inferior laryngeal nerves may occur in combination with other injuries

Diagnosis

1. Laryngeal trauma
   Symptoms: respiratory distress, pain, cough, dysphonia, aphonia, aspiration, dysphagia, and odynophagia
   Signs: local ecchymosis, abrasion, laceration, hematoma, edema, hoarseness, stridor, hemoptysis, and cyanosis
   Palpation: loss or deformity of anatomical landmarks (Adam's apple, cricothyroid notch), crepitus, and subcutaneous emphysema
2. Tracheal trauma
   Upper tracheal injuries have diagnostic characteristics similar to those of the larynx. The pain may be located lower in the neck or in the upper chest. Laryngotracheal separation may be encountered in open cervical injuries; however, rapid flexion-extension of the neck in sudden deceleration may lead to similar damage (whiplash injury).

Evaluation

Blunt trauma may be overlooked and/or underestimated: There is often severe soft tissue and/or skeletal damage. Symptoms may evolve slowly. Dislocation of the cricoarytenoid joint must be considered, but in the acute stage, edema may mask this relatively subtle finding.
The following injuries may coincide with laryngotracheal trauma: fractures of the mandible and hyoid bone; lacerations and perforations of the pharynx and esophagus; vascular and spinal trauma.
The aphonic patient may not be able to express his pain and discomfort, so provide him with a writing pad and bell for communication.

Examination

Indirect laryngoscopy may be attempted initially but may be difficult to perform in patients with multiple injuries.
Direct rigid or flexible fiberoptic hypopharyngoscopy, laryngoscopy, tracheoscopy, and bronchoscopy are dependable diagnostic methods. These frequently clarify the nature and magnitude of the injury. When doubt exists, direct rigid endoscopy should be performed in the operating room with the airway secure, usually together with tracheostomy and reconstructive surgery. Disruption of normal anatomy, the presence of mucosal lacerations, foreign material, and cartilage injury should be documented. Vocal cord mobility and position must be assessed and the arytenoids palpated. In massive trauma, injuries to the trachea or esophagus often coexist. Esophagoscopy should be included.
Soft-tissue X-rays of the neck are valuable for identifying subcutaneous or retropharyngeal emphysema, locating foreign bodies, and following the track of a penetrating bullet or shrapnel. CT scans are valuable for the assessment of osseous, cartilaginous, and vascular injuries, and spinal involvement. *Note:* Chest films should be included, but only if the time and conditions allow; a physician competent and equipped in establishing the airway must accompany the patient at all times until the patient is stable.

Treatment

On Site. Monitor vital signs constantly. Clear mouth and pharynx. Introduce oral or nasopharyngeal airway. Perform low tracheostomy or cricothyroidotomy, or intubate through the open wound if necessary. Administer oxygen. Secure cervical spine. Evacuate promptly.

Primary Medical Facility or First Aid Station. Re-examine and monitor. Take steps to clean, ensure, or improve airway. Cervical X-rays should be taken to rule out spinal fracture and injury. Examine for associated vascu-

lar trauma and esophageal lacerations, fractures of mandible and hyoid bone. Look out for vomiting and aspiration. Start broad-spectrum antibiotics. Provide oxygen and humidification. Designate high transportation priority with competent, well-equipped attendant.

Hospital. Complete endoscopic examination under anesthesia. If injury is mild, limited to edema, hematoma, and superficial lacerations, abrasions, or contusions, monitor for 24–48 h. Otherwise, exploration and repair should be performed within 24 h. Repair lacerations and defects with flaps or grafts. If the wound is contaminated, meticulous wound excisions are required along with good judgement and experience. Local infection and chondritis are serious complications. Suprahyoid release may be indicated to aid in re-anastomosing the larynx and trachea.
If necessary, revise and reposition the tracheotomy to the most appropriate site, low on trachea. *Cricothyroidotomy is a laryngotomy, not a tracheotomy, and must be relocated within a few hours to the lower trachea.* Stent airway if indicated.
Suture or graft, at least one sectioned or avulsed recurrent laryngeal nerve. Reposition arytenoid-cricoid dislocation. Introduce nasogastric tube to prevent vomiting and aspiration. Treat the esophagus, pharynx, and mediastinal emphysema; drain hypopharyngeal fistulae or attempt primary closure if appropriate.
Monitor continuously until stable. Evaluate aspiration, burn, and caustic injuries and treat expectantly.

The Obstructed Airway (and Ventilatory Failure)

Introduction

Trauma causing respiratory obstruction may occur at any point from the anterior oropharynx to the trachea. There may be more than one site of obstruction. (Refer also to chapters on endotracheal intubation and injury to the larynx and trachea.)
Management principles and guidelines:

1. Primary management is by simple measures such as positioning of the head and neck, head tilt, jaw thrust, and lift and tongue support or pull. These maneuvers must be performed with care because cervical spine injury should always be suspected. Restrict motion of the neck until injury of the cervical spine has been excluded (Fig. 5).
2. Foreign bodies must be removed manually or instrumentally and the oropharynx cleared of blood, food, secretions, and teeth fragments, etc. Suction and good lighting are helpful (Fig. 6).

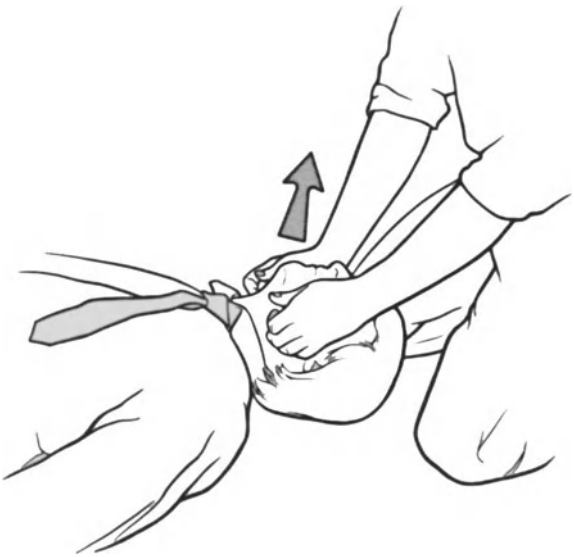


Fig. 5. Jaw thrust, cervical support, and head tilt often stabilize and open the upper airway

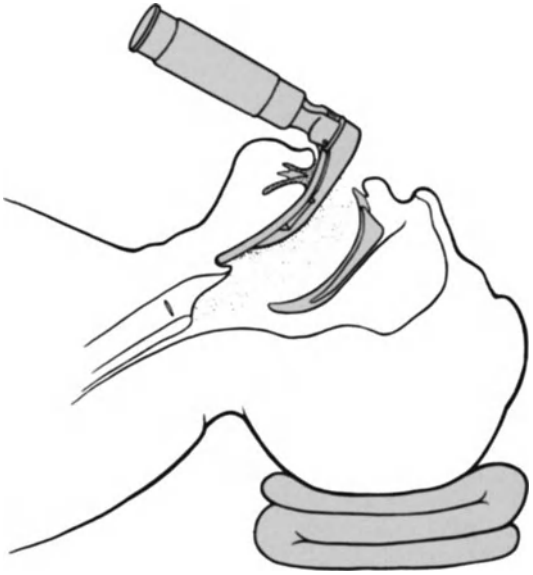


Fig. 6. Probe open the mouth; light suction and proper instrumentation are helpful; remove secretions, foreign bodies, food, dentures, bone fragments, etc.

3. Be aware and look for signs of exposure to fire, smoke, and noxious irritating gases with bronchoalveolar or parenchymatous lung involvement.
4. Introduce an oral or nasopharyngeal airway to maintain air flow until definitive establishment of the airway is performed (Fig. 7, 8).
5. Every effort should be made to avoid emergency tracheotomy.

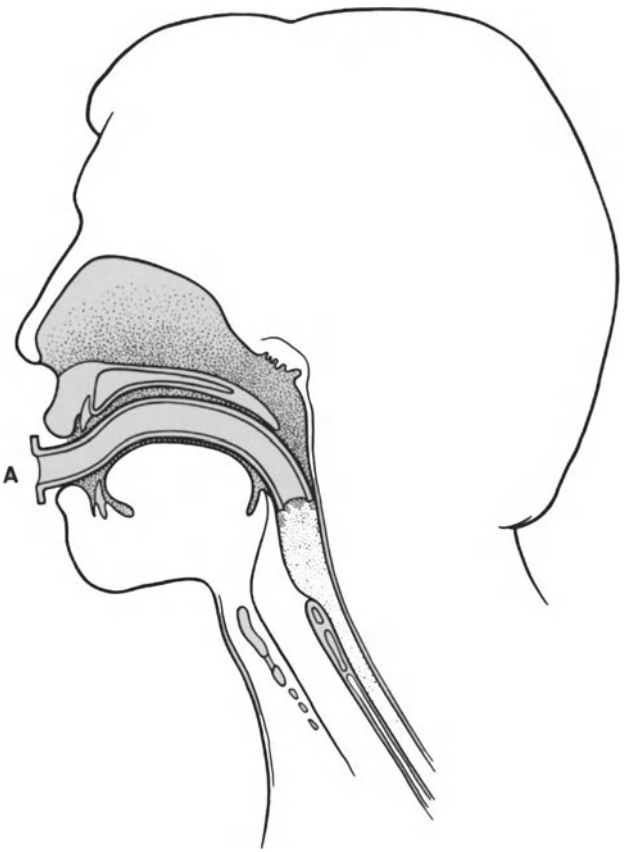


Fig. 7. Pull the tongue out using a tongue or a towel clip or insert an oral airway (*A*)

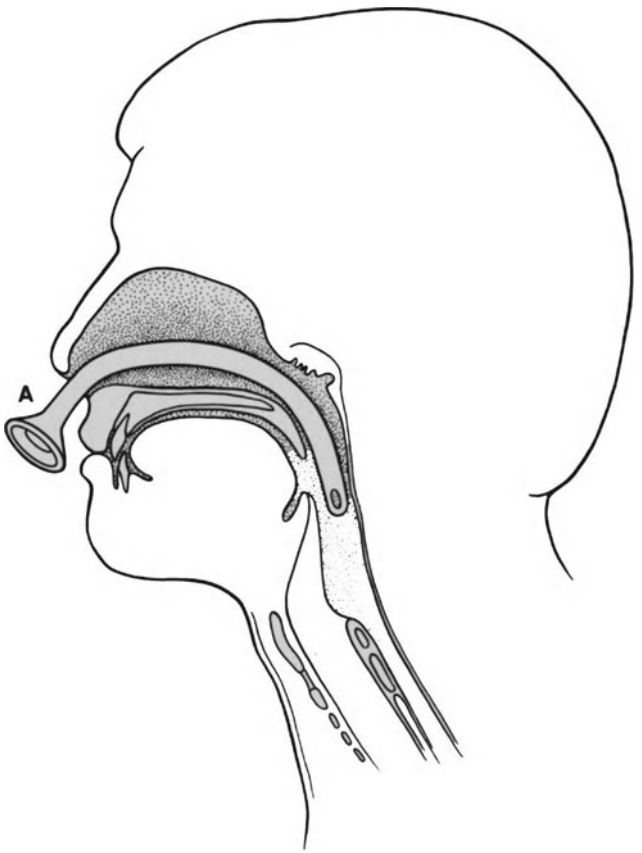
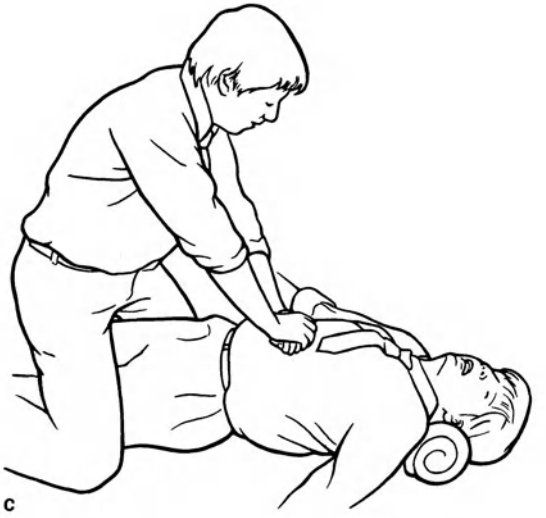


Fig. 8. A nasopharyngeal airway (*A*) is preferred in mandibular and oral trauma or in the agitated patient

6. Tracheotomy is preferably performed over an orotracheal or nasotracheal tube, bronchoscope, or a temporary airway inserted through the cricothyroid membrane.
7. Unsuccessful, traumatic attempts at intubation may prompt obstruction of the airway.
8. Time should not be wasted in first obtaining X-rays or ancillary studies (e.g., blood gases).
9. The Heimlich maneuver may be effective in clearing foreign bodies from the airway (Fig. 9).

a

b



c

Fig. 9a. The Heimlich maneuver — this emergency procedure may be effective in clearing the upper trachea, larynx, and hypopharynx of unlodged foreign bodies. **b** With the hands grasped under the xiphoid below the rib cage, apply quick forceful upward thrusts (not simply a squeeze or bear hug); repeat six to eight times. **c** With the patient lying down (if unconscious or heavy), press heel of hands abruptly over upper abdomen, just below the xiphoid. If patient vomits, quickly turn on side; then deliver four to six, sharp, hard, palm-of-hand blows to the interscapular region

10. Whatever method is employed, the airway must be established without delay and ventilation provided promptly and effectively to prevent irreversible brain damage.

Definition of Surgical Terms

Intubation is the introduction of a tube via the mouth or nose into the larynx and trachea to keep the airway open or to restore its patency if obstructed.
Tracheotomy is a temporary opening of the trachea.
Tracheostomy is a long-term or permanent opening with establishment of a mucocutaneous junction.
Cricothyroidotomy is an opening into the larynx (not the trachea) between the thyroid and cricoid cartilages through the cricothyroid membrane. It is indicated in acute emergencies and provides an effective opening into the airway with a relatively low risk of bleeding. A proper tracheotomy is done as soon as the patient's condition permits. Cricothyroidotomy is unsuitable for a long-term airway.
Emergency tracheotomy is indicated for (a) mechanical obstruction at or above the level of the larynx, and for (b) patients who cannot raise secretions and clear their airway.
Elective tracheotomy is indicated when respiratory insufficiency is anticipated in patients with thoracic, abdominal, head, and neck trauma, or in postoperative periods. Pulmonary insufficiency may also be an indication, as are lack of effective cough, coma states, aspiration, and gastroesophageal reflux.
Therapeutic tracheotomy is performed in cases with alveolar hypoventilation or tracheobronchial hypersecretion with or without bronchial spastic phenomena, to remove secretions and provide for safe prolonged use of assisted mechanical ventilation with decrease of the dead space. Long-term or permanent tracheostomy is an elective procedure utilizing tracheal and cutaneous flaps to form a self-sustained patent tract between the trachea and the skin, with a circumferential mucocutaneous junction.

Diagnosis

Signs and symptoms:
Air hunger — restlessness, retained secretions, ineffective cough
Stridor, especially inspiratory
Retractions — intercostal, suprasternal, supraclavicular
Rising pulse and respiratory rate
Apprehension, disorientation, coma
Pallor, cyanosis, fatigue, and exhaustion

Evaluation. Never wait for late clinical signs before establishing a reliable airway. Perform intubation or surgery whenever the indication is considered. Hypoxemic patients are prone to cardiac arrest. Exhaustion is a sign of imminent danger. Monitor respiratory and pulse rates. Do not wait for cyanosis.

Treatment

Placement of an endotracheal tube should be the preferred mode of airway management. However, intubation may not always be feasible (e.g., massive injury to the lower midface, hemorrhage, cervical spine injury, laryngeal fracture, or an acutely obstructing foreign body). Morbidity and mortality increase when a tracheotomy is performed in a struggling patient without initial airway control. An endotracheal tube or bronchoscope should be introduced first whenever available and possible, especially in children.
Indications for intubation:
Lack of respiratory drive

CNS trauma — drug overdosage
Cardiac failure or arrest
Spinal injury
Chest splinting — phrenic nerve injury, abdominal injury
Loss of ventilatory muscular activity — neuromuscular reaction to drugs, toxins, poison, venom

Obstruction of airway

Trauma: bleeding, edema, rupture
Infection: croup, epiglottitis
Tumors
Coma
Stenosis
Malacia — skeletal collapse of the airway
Bilateral vocal cord paralysis or spasm
Foreign body — bronchial spasm

Inadequate bellows mechanism

Penetrating chest wounds
Pneumothorax or pneumomediastinum
Flail chest
Pleural effusion — hemothorax

Parenchymatous-pulmonary failure
Lung contusion — hematoma
Atelectasis
Pulmonary embolus

Fat emboli
Drowning
Carbon monoxide poisoning
Pulmonary edema — diffusion abnormality
Smoke and fume inhalation, toxic gases

Uncontrolled aspiration

Stroke
Head trauma, CNS or neck injury
Drug overdose
Coma
Massive oropharyngeal bleeding and base-of-skull fractures

Indications for Tracheotomy

Obstruction

1. Trauma, blunt or penetrating injuries of the larynx or proximal to it, including laryngotracheal separation causing:
   Partial or complete obstruction of the airway by internal or external injury
   Edema of the endolaryngeal or tracheal structures
   Blood, secretions, or foreign material in the airway
   Maxillofacial injury with aspiration and local edema, internal bleeding, or retroplacement of the tongue and bone fragments
   Collapse of airway
2. Foreign body
3. Inflammation
   Angioedema
   Allergic response
   Secondary to caustic or thermal injury
4. Paralysis or paresis of the vocal cords
   Bilateral
   Due to spine or brain injury
   Loss of laryngeal sensation due to superior laryngeal nerve section — uncontrolled aspiration

Ventilatory failure (nonobstructive)

1. Retained secretions
   Depression of cough (postsurgical, pneumonitis)
   Aspiration of gastric contents
2. Inefficient ventilation due to coma, CNS injury, chest wall instability or paralysis, pretraumatic chronic lung disease

Surgical Techniques

Cricothyroidotomy

This is an alternative to tracheotomy, being easier and more rapid. In difficult conditions it can be performed with fewer instruments and less experience. No anesthesia or sterility are required in an emergency situation. Place the patient in a comfortable, head-high position with the neck extended if possible and not contraindicated. Identify the cricoid cartilage; normally it is the first rigid structure the finger encounters when moving upward from the suprasternal notch (Fig. 10). In the conscious patient and when time permits, obtain local anesthesia with 1% lidocaine. Make a 1–2 cm horizontal skin incision over the notch and repalpate through it to confirm your position (Fig. 11). Locate the soft tissue depression between the cricoid arch and the lower margins of the thyroid cartilage. Hold the blade horizontally like a pencil, limiting its potential penetration to 0.5–1.0 cm. Point it backward and caudally over the upper rim of the cricoid arch, and pierce the cricothyroid membrane (Fig. 12). Insert any tube 0.5–1 cm in

Fig. 10. Proper position for cricothyroidotomy and tracheotomy — note palpatory identification of cricothyroid membrane (see text)

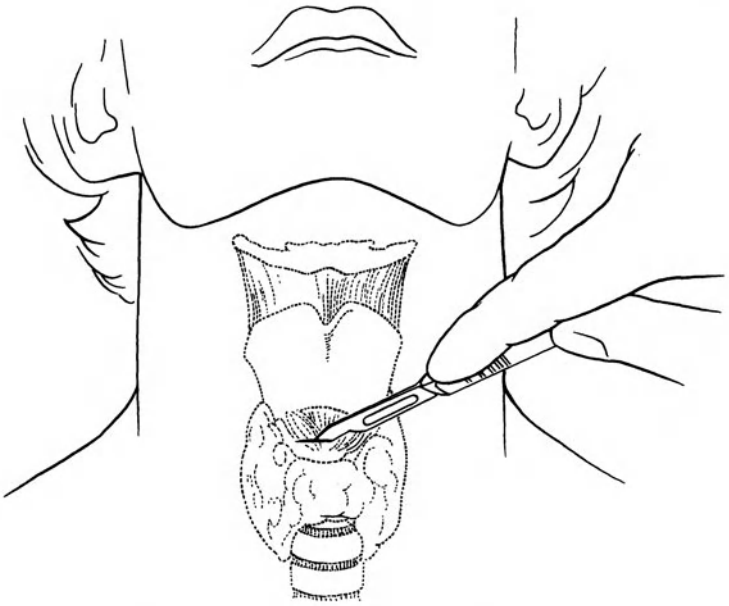


Fig. 11. Skin incision over the cricothyroid membrane

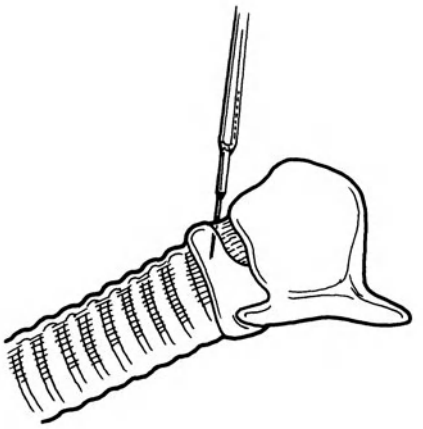


Fig. 12. Angle of the piercing knife in cricothyroidotomy

diameter in a caudad direction over and behind the cricoid arch into the trachea. The hollow cylinder of a pen or other tube may serve as a temporary cannula. A number 4 or 6 tracheostomy cannula or endotracheal tube may be used, when available. Tape or suture it to the neck.

This procedure may be performed with special trocars designed for this purpose. These are dangerous in untrained hands. The open-staged method requiring a blade and any cannula or tube is preferred.

Make sure to note on the patient's records in a very clear manner that you performed a cricothyroidotomy and not a tracheotomy to ensure that the latter operation may supplement your procedure as soon as conditions permit. Subglottic stenosis is an inevitable complication of cricothyroidotomy whenever it is not changed to a tracheotomy within 24–48 h.

Tracheal oxygenation, by means of a 14- or 16-gauge needle or catheter introduced through the cricothyroid membrane, may work by creating a diffusion gradient through the tracheobronchial tree down to the alveoli. This method may be used to buy time until more adequate means of oxygenation are instituted. (Review the anatomy presented in Fig. 13).

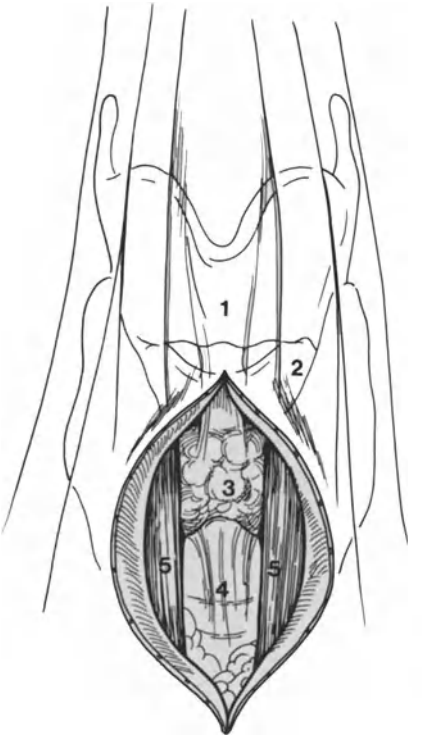


Fig. 13. Anatomic landmarks in tracheotomy: *1*, thyroid cartilage; *2*, cricoid cartilage; *3*, thyroid isthmus; *4*, trachea deep and posterior in suprasternal notch; *5*, strap muscles

Tracheotomy

Tracheotomy is only occasionally performed as an emergency procedure. The complications, morbidity, and mortality increase with suboptimal conditions. This procedure is best performed under general or local anesthesia, with an established temporary airway, such as an endotracheal tube, bronchoscope, cannula, or catheter introduced through a cricothyroidotomy. Under good lighting and with a sandbag or rolled towels between the shoulder blades, the neck is extended as much as is deemed safe for the cervical vertebrae, then prepped and draped. A 2–3 cm vertical incision (preferred in emergencies) is made from the suprasternal notch to the cricoid cartilage (Fig. 14). The anterior cervical veins are clamped or retracted laterally. The strap muscles are identified and separated in the midline, exposing the thyroid isthmus and the underlying trachea. The upper trachea is palpated and bluntly exposed. A tracheal hook is used to elevate the trachea towards the surface. The hook is placed through the first or second tracheal rings. The trachea is exposed and cleared of vessels, fascia, and fat.

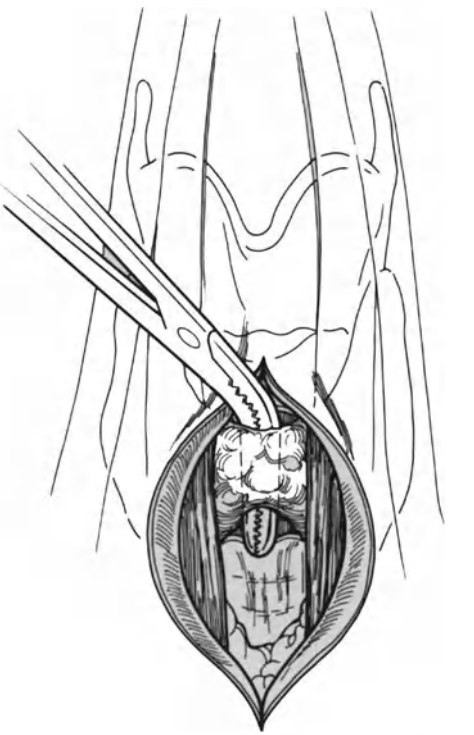


Fig. 14. The vertical skin excision and lateralization of the strap muscles allow exposure of the thyroid isthmus. The latter may be elevated, ligated, incised, and suture-ligated or simply elevated to expose the underlying cervical trachea (note the thyreoidea ima artery; it may cause severe bleeding if severed)

A round window, 1 cm in diameter, is made through rings 2 and 3 or 3 and 4 (never through the first ring); (Fig. 15). (In infants, a single vertical incision is advocated through rings 3–4. Two silk ties are passed on either side of the tracheal incision to help identify its position). A cannula of suitable diameter is introduced with the aid of a special tracheal dilator (when available). A plastic double tracheal cannula with an inflatable cuff is preferred when assisted respiration is indicated or anticipated (Fig. 16). Hemorrhage is controlled by ligation and/or electrocautery. The incision is only partially closed with layered sutures to prevent subcutaneous emphysema and tension pneumomediastinum. It is always wise to suture the cannula to the skin, in addition to the tape tied around the neck.

A post-tracheostomy X-ray of the neck and chest is warranted (especially in children and when performed by inexperienced hands) to verify the position of the tube and rule out complications.

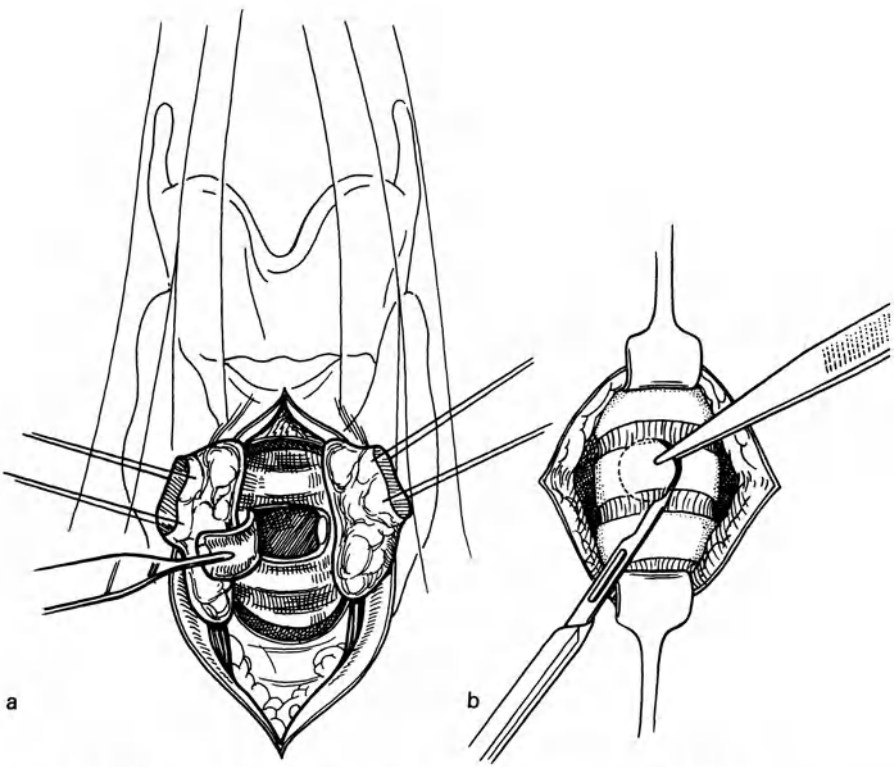


Fig. 15a, b. With the thyroid isthmus out of the way, carve out a circular window into the tracheal lumen through tracheal rings 2 and 3

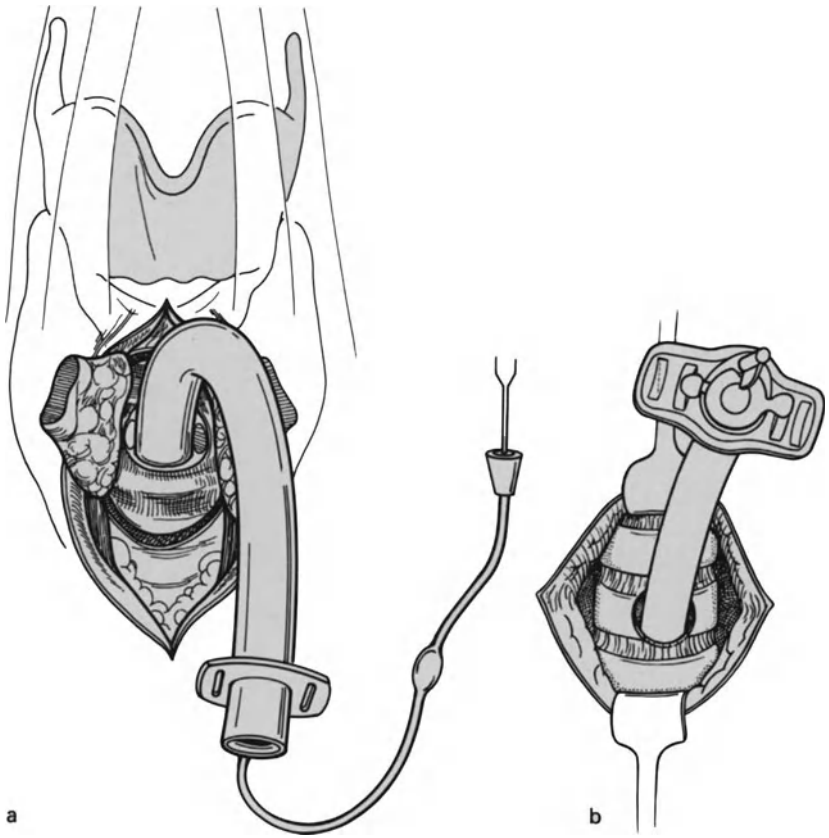


Fig. 16a, b. An endotracheal tube or tracheotomy cannula

The following complications may occur within 24 h after the operation:

Hemorrhage

Pneumothorax and pneumomediastinum

Immediate apnea, following reinstitution of the airway, occurs in patients in whom the hypoxic drive has been eliminated. Controlled respiration may be needed in this situation.

Subcutaneous emphysema sometimes results from tight incisional closure, aggravated by mechanical ventilation

Atelectasis

A poorly positioned or dislodged tube

Aerophagia

Tracheoesophageal fistula

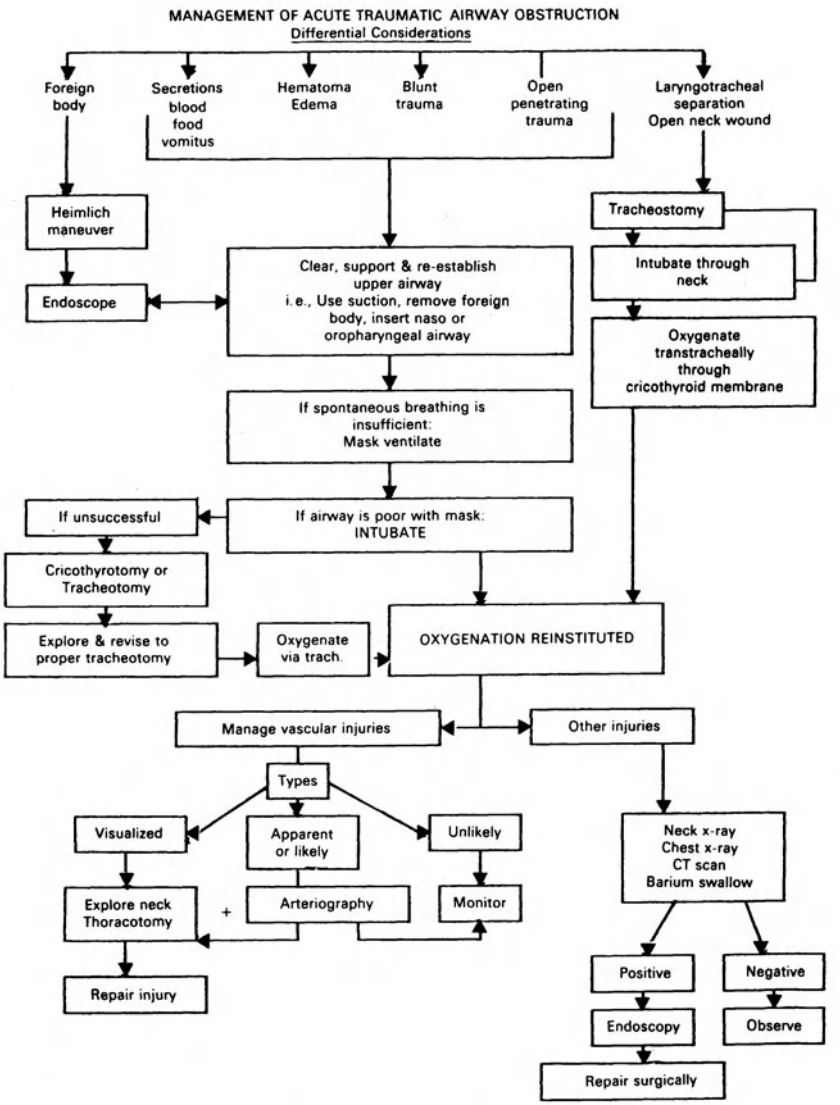


Fig. 17. Management of acute traumatic airway obstruction

Summary of Steps for Airway Obstruction (Fig. 17):

1. Clear and reestablish airway
2. If there is no or poor spontaneous breathing: mask ventilate
3. If the airway is not clear: intubate
4. If intubation is not possible or not available: perform cricothyroidotomy or, better, tracheotomy
5. When airway and ventilation have been reinstated: (a) CPR hemostasis and volume replacement and (b) treat underlying causes of airway obstruction, etc.

4.7 Cardiovascular Injury

G. Merin

Since the pathophysiology, clinical course, and treatment of cardiac injury are different from those of vascular trauma, it is appropriate to consider them separately.

Cardiac Injuries

The incidence of penetrating cardiac injuries in military practice is much higher than blunt cardiac trauma, whereas the reverse is true for civilian disasters.

Blunt Injuries. The most common blunt cardiac injury is myocardial contusion (found in up to 25% of all blunt thoracic injuries). Blunt trauma may cause direct coronary artery injury, e.g., intimal tear or subintimal dissection and subsequent myocardial infarction, simulating severe myocardial contusion. Traumatic valve rupture, secondary to a sudden rise in intrathoracic pressure, has also been described. An excessive rise in intrathoracic pressure may result in cardiac rupture. Myocardial laceration may be associated with rib or sternal fractures.

Penetrating Injuries. Stabbings and gunshot wounds are the main causes for penetrating cardiac injuries in civilian practice. In military practice, survival following a high-velocity missile cardiac injury is extremely rare. Shrapnel wounds may result in survival only when the size of the shrapnel is small or when the injury is in a low-pressure system (e.g. right atrium), and ensuing low-pressure tamponade may lead to survival. The most common clinical manifestation is cardiac tamponade, which usually presents as cardiovascular collapse accompanied by signs of obstructed venous drainage. Myocardial contusion may mimic the whole spectrum of symptoms and signs of myocardial infarction.

Diagnosis

The diagnosis of blunt cardiac trauma in the presence of other thoracic injuries is made more frequently when a heightened awareness of this possibility exists. Frequently, minimal electrocardiographic changes, mainly ST-segment depression and T-wave inversion, are observed. The level of creatine phosphokinase (CPK) is usually elevated, and special attention should be given to its myocardial fraction. In severe cases of myocardial contusion, electrocardiographic changes suggestive of myocardial infarction may be present. This is especially true in cases of blunt trauma causing coronary artery thrombosis and may be accompanied by echocardiographic evidence of regional reduction of myocardial function.

Cardiac tamponade is a grave complication and is a potentially lethal condition. Prompt diagnosis should therefore be made. Depending on its severity, pericardial tamponade leads to restriction of ventricular filling, resulting in venous engorgement, reduction of stroke volume, and resultant circulatory collapse.

Clinically, the patient is restless, has a grey complexion, sweats and fights for every breath. The pulse rate is rapid and typically with a narrow pulse pressure which disappears with every deep inspiration; simultaneously, neck vein distention may occur. In milder forms, circulatory collapse is not present, and other physical signs may not be prominent. A typical chest X-ray demonstrating globular enlargement of the cardiac silhouette or straightening of both cardiac borders on the PA chest X-ray should prompt the examiner to perform an echocardiogram: this will demonstrate the presence of pericardial effusion, which in acute cases may not exceed 200–300 ml, as opposed to chronic cardiac tamponade, where the pericardium distends gradually to accommodate over 1000 ml of fluid.

Intramyocardial lesions, such as traumatic valve incompetence or septal rupture, are diagnosed according to the specific lesion.

Treatment

Treatment of cardiac injuries is aimed at correction of the pathophysiological disturbance, repair of the basic injury when indicated, and prevention of subsequent complications. Apart from general supportive and resuscitative measures, there are no specific measures to be taken on site.

Bed rest is usually recommended for myocardial contusion, especially if there is concomitant electrocardiographic and echocardiographic evidence of myocardial injury. Severe contusion should be regarded as equivalent to myocardial infarction and treated as such. Although the initial injury involves direct damage to the coronary arteries, the place of coronary angiography and subsequent revascularization in this subset of patients has not yet been elucidated.

The degree of gravity of injury to intracardiac structures such as valves is the main indication for specific treatment.
Cardiac tamponade should always be relieved, and if caused by myocardial laceration, this should be repaired. The timing of the intervention depends again on the gravity of the situation, and on the availability of adequate facilities and qualified personnel. In the absence of these, open thoracotomy should be reserved only for those who do not respond to supportive treatment, such as increasing the venous filling pressure and general resuscitative efforts, and in whom, in spite of these measures, vital signs continue to deteriorate. Otherwise, it may be worthwhile to transfer even a gravely ill patient who can be kept alive to a facility where not only relief of the tamponade but also adequate control of its cause can be achieved.
Pericardiocentesis is a valuable adjunct, and sometimes a definitive treatment for such a condition. Usually, a large bore, 15-cm long needle may be inserted at the left xiphocostal angle, aimed at the right shoulder of the patient at an angle of 45°–30° to the frontal plane. The needle should be advanced cautiously with gentle continuous negative pressure applied to it with a syringe. Unless bleeding is excessive, the withdrawn blood should not clot. If the aspirated blood clots readily, further drainage should be stopped immediately, and the needle withdrawn. When successful, pericardiocentesis results in restoration of circulatory stability and provides relief. Even when relieved, pericardial tamponade should still be regarded as a life-threatening condition, and the patient should therefore be urgently transferred to the nearest appropriate facility for further observation and possible definitive treatment which, in this case, involves control of hemorrhage and adequate drainage, accompanied by volume replacement and general supportive measures.

Complications. Apart from immediate complications related to the trauma itself or to the basic injury (such as wound infection, etc.) or to resultant loss of segmental myocardial function, complete recovery should be expected. Rarely, late pericardial constriction may follow the drainage of a hemopericardium.

Summary. On site, general supportive and resuscitative measures (see chapter on shock, p. 206) should be carried out. At the medical aid station, supportive and resuscitative measures should be continued and monitored, In the field and base hospitals, treatment is as above. Pericardial paracentesis and/or thoracotomy by a trained surgeon should be performed as indicated.

Vascular Injuries

Vascular injuries may result, if untreated, in the loss of an organ, a limb, or a life. An understanding of basic pathophysiological processes associated with vascular injuries helps to prevent such disasters. Rapid evacuation is vitally important since the rate of organ salvage is inversely related to the time interval between injury and treatment. The availability of adequate facilities for treatment is another factor necessary for success.

Classification

Arterial Injury. The primary classification should distinguish between arterial and venous injuries. Blunt arterial injuries may lead to a different outcome than penetrating injuries. Whereas in both kinds ischemia may be present distal to the site of damage, blunt injury can be accompanied by much less blood loss than penetrating injury. One must distinguish between penetrating injuries that cause complete transection of an artery and a laceration with preservation of continuity.

Venous Injury. Isolated venous injuries are seldom a cause for concern, and while they may result in considerable blood loss, they are limited by tissue tamponade.

Pathophysiology

Blunt arterial trauma may rarely cause the transection of a vessel (e.g. deceleration injuries of the thoracic aorta). In most cases, however, when complete or incomplete transection of a vessel occurs secondary to blunt trauma, it is usually associated with a bone fracture and should therefore be regarded as a penetrating injury. Blunt vascular trauma alone may result in blood vessel wall damage, intimal dissection, and subsequent arterial thrombosis without blood loss. Such is the case when a high velocity missile strikes in the vicinity of an artery. Its outer wall is not penetrated and immediate arterial bleeding does not occur, but the high kinetic energy delivered by the missile, followed by cavitation and a negative pressure wave, cause immediate thrombosis with resultant ischemia. Later on, arterial wall necrosis and rupture may occur as a result of damage to the vasa vasorum by the initial trauma. High and low velocity missiles alike may cause damage by direct injury to the vessel wall, disrupting its continuity. Complete arterial transection may result in a copious initial blood loss which usually stops when the vasoconstrictory mechanism secondary to blood loss takes effect, in addition to the local spasm of the injured vessel wall. Thus, in such cases, loss of continuity results both in blood loss and distal ischemia.

When arterial continuity is preserved and there is only a partial tear, the local vasospasm may not be sustained, and further bleeding may occur, but on the other hand distal ischemia may not be as severe or, sometimes, not present at all.
Solitary venous injuries do not need separate attention unless they involve a major vein, such as the common femoral, iliac, renal, or other veins of that magnitude. The main danger is that of uncontrolled hemorrhage to an open wound, but when the injured vein is surrounded by other tissues, bleeding usually stops as local tamponade occurs.

Diagnosis

Peripheral arterial injury is readily diagnosed when brisk arterial bleeding from an open wound occurs. Often this may not be the case. Expanding hematomas should create a high degree of suspicion even in the presence of a peripheral pulse and in the absence of ischemia, as is the case in a partial tear of an artery.
Signs of acute limb ischemia may be present even with preserved arterial continuity because of shock, exposure to cold, crush injury, vessel spasm secondary to contusion, or outside pressure from displaced adjacent structures. In some cases, accurate diagnosis may require exploration. The use of ultrasonography and Doppler flow detectors is valuable in detecting ischemia with preserved vessel continuity and also to detect the approximate site of injury. As a rule, one should suspect major vascular injury even if the only indicative fact is the proximity of the wound to a major vessel.
The diagnosis of arterial injury is made by the following clinical findings: brisk hemorrhage from the wound; expanding hematoma, which may become pulsatile; cold pulseless, numb limb with various degrees of function loss.

Treatment

The aims of the treatment of arterial injuries are the control of hemorrhage and the preservation of organs and limbs. Since anoxic tissue damage is irreversible after a few hours, urgent surgical treatment is indicated even in the absence of hemorrhage. Reparative surgery should be attempted at all times as long as the signs of frank necrosis are not present, since the time lag between injury and irreversible damage is unpredictable.

On Site and Medical Aid Station. Control of hemorrhage is an urgent life-saving measure. This is obtained best by immediate direct pressure or, if possible, clamping of the vessel. A tourniquet should be applied with restraint, with the minimal tension sufficient for control of blood loss. The tourniquet should be cautiously released after 15 min; in many instances

the bleeding does not resume. If it does, the tourniquet is reapplied. This intermittent release of circular pressure is helpful in restoring some collateral flow to the distal limb and therefore should be attempted. General supportive treatment, i.e. volume replacement and immediate administration of i.v. penicillin, is also given. Once hemorrhage is controlled, the patient should be transferred to a facility at which definitive treatment can be administered.

Field and Base Hospitals. Often upon arrival there is no bleeding when the tourniquet is released again, and the magnitude of the blood loss is questioned. When organ viability is not evident, exploration is immediately undertaken. In some instances, if hemorrhage does not occur after release of the tourniquet and organ viability is preserved, one may be tempted to procrastinate only to face secondary, unexpected hemorrhage or to have to treat later a traumatic pseudo-aneurysm or arteriovenous fistula. It is therefore recommended to explore all wounds that are near major vessels and in which hemorrhage was observed. Reconstruction of vascular continuity is then undertaken. The value of an urgent temporary bypass is dubious, since it calls for an available anticoagulated conduit or systemic anticoagulation, with a preceding embolectomy and irrigation of the distal vessel, with the necessary tourniquets to hold the bypass in place, and may take almost as much time as the actual repair itself. Basic surgical principles regarding treatment of trauma are observed. When dealing with a high velocity missile injury, wide debridement of adjacent tissue is performed. The injured vessel itself should be resected both distally and proximally, much more extensively than the outer appearance of the vessel may suggest, as the extent of the damage caused by a high velocity missile is much wider than its actual trajectory. When dealing with low velocity objects, a more conservative approach can be applied. All portions of arteries showing mural hematomas are also resected. The absence of backflow through the distal orifice is ominous, and distal thrombosis or sequential laceration should be ruled out by the introduction of an embolectomy catheter and by distal irrigation with heparinized saline solution. Only after establishment of backflow should vessel continuity be restored. In most cases after debridement, end to end anastomosis may be difficult to achieve without tension, and interposition of a vascular graft should be employed. When dealing with blunt trauma where there is no open wound and sterility of the operative field is unquestioned, a vascular prosthesis may be used. In all other instances, a reversed autologous saphenous vein graft is used. It is recommended that when the injury to a leg is widespread and there is suspicion of deep vein damage, the saphenous vein should be harvested from the other leg.

When concomitant arterial and venous injury exists, special attention should be given to the adequacy of venous drainage of the injured limb. Many successful arterial repairs are performed only to fail later because of limb edema and deep vein thrombosis. This is especially true for the com-

mon femoral vein, ligation of which should be avoided, and repair should be performed even when low velocity flow may cause gradual thrombosis later on, as this time lag should enable the development of a venous collateral circulation.

After adequate flow has been established and its efficacy proven by limb viability and the presence of a distal pulse (not only adjacent to the repair), immediate coverage of a vascular repair is mandatory. The usual practice of debridement and secondary closure may be disastrous in the case of a bare vascular anastomosis. After establishing adequate drainage, the surrounding viable muscles and skin are utilized to cover the widely debrided area of vascular anastomosis. Failure to do so renders a successful repair useless, as ensuing infection will destroy it.

When long bone fracture occurs concomitant with vascular injury, fixation of bony structures is essential as excessive motion at the site of vascular repair may endanger it. When the danger of potential infection is minimal, internal bone fixation is safer and should be done prior to vascular repair, as manipulation of the fractured bone may cause damage to the repaired vessel. External fixation is employed in all other instances. When the arterial injury is accompanied by a considerable loss of surrounding tissues, bone shortening should be contemplated so as to reduce the gap, and facilitate the vascular anastomosis. When a cast is used, it should be bivalved to accommodate the ensuing limb edema that usually occurs a few hours after vascular repair and which is related to prior ischemia, tissue damage, and venous injury. When applying a cast, one should also expose distal points for close repeated examination of limb viability.

Postoperatively, the usual supportive measures and antibiotic treatment are given (see chapter on the general management of wounds). As a rule, in contaminated wounds this should include administration of antibiotics as part of the resuscitative measures immediately medical treatment is available.

When limb edema endangers an otherwise successful arterial repair, liberal fasciotomy of all compartments is undertaken under local anesthesia. It is advisable to perform fasciotomy at the time of vascular repair whenever the venous drainage is questionable and when arterial repair is performed after a prolonged period of ischemia, which can be attested to by loss of voluntary fine motion distal to the site of vascular trauma at the time of initial preoperative assessment.

The postoperative use of systemic anticoagulation should be avoided as danger of secondary hemorrhage from traumatized tissues is imminent. Low dose heparin (5000 units b.i.d. subcutaneously) for prophylaxis of deep vein thrombosis is recommended, as the incidence of this complication in vascular trauma is high.

Surgical sympathectomy cannot replace a vascular repair, and, when vascular repair fails, cannot alter the outcome. Pharmacological sympathectomy

by paravertebral or epidural blockade may be of use in differentiating between vascular spasm and vessel injury as a cause for limb ischemia.

Summary. There are two principles of surgical treatment. Exploration of all suspected or known major arterial or venous closed or open injuries is mandatory. Establishment of vascular continuity will result in organ salvage, whereas control of hemorrhage alone may save life but cause permanent damage. The sequence of surgical procedures is: internal skeletal fixation (when possible and not contraindicated — see text), otherwise external skeletal fixation; establishment of vascular continuity; wide debridement of traumatized tissue; use of viable tissue to cover vascular repair site; and fasciotomy when indicated (see text).

4.8 Thoracic Injuries

H. Peleg

General Principles

An apparently "minor" thoracic injury may start a fatal chain of pathophysiological changes (Fig. 1). A thorough examination may have to be postponed in favor of urgent lifesaving therapeutic steps: CPR, establishment of the airway, blood volume replacement, chest drainage, and rarely, urgent thoracotomy.

History

The type of injury, the direction of missiles, and the position of the patient in the field or inside a colliding vehicle may help in evaluating the injury.

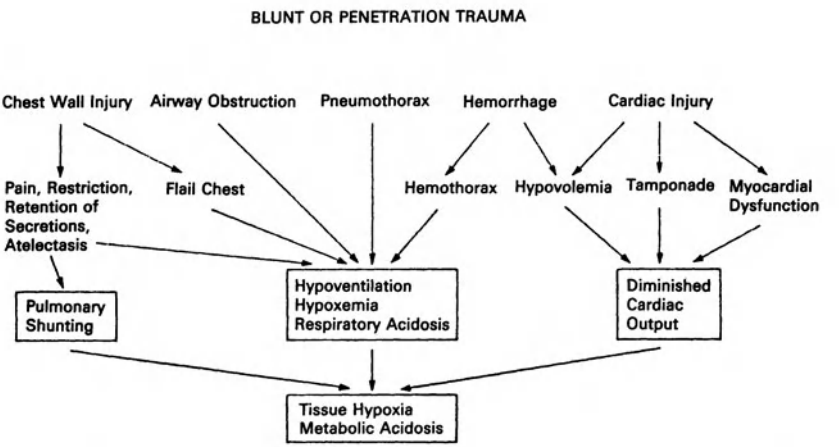


Fig. 1. Pathophysiological changes in chest trauma, all leading to tissue hypoxia and metabolic acidosis with eventual fatal outcome. [From Hood (1983), Fig. 13-1]

Examination

1. Observation — note the location of entry and exit wounds, abrasions, contusions, or any other external evidence of injury, and the movement of the chest wall.
2. Palpation — subcutaneous emphysema, percussion, and vocal fremitus.
3. Auscultation (a common mistake is not to examine the back, thereby missing important findings).
4. Associated injuries — in particular abdominal and spinal injuries which may be caused by the same wounding agent. An upright chest roentgenogram should be done as soon as possible in order to demonstrate pneumo- or hemothorax, cardiac tamponade, and widening of the mediastinum, which are often missed on supine X-rays.

Treatment

Less than 20% of thoracic injuries will require a surgical procedure other than closed intercostal drainage (CID) and/or excision of the wound. CID is a life-saving procedure which every physician must be capable of performing.

The indication for CID is evidence of thoracic injury, pneumothorax or hemothorax, in the presence of: respiratory distress, delay in evacuation to hospital, airborne evacuation, or anticipation of need for mechanical ventilation.

When in doubt whether or not pneumothorax or hemothorax is present, and a roentgenogram is not available, do a diagnostic pleural puncture, with a 10–20 ml syringe and a no. or 18 or 19 needle.

The indications for thoracotomy are:

1. Imminent loss of life from wounds of the heart and great vessels requires immediate life-saving thoracotomy upon arrival in hospital.
2. After CID, if bleeding continues at more than 200–300 ml per h for several hours.
3. After CID, when massive air leak continues, the lung does not expand, and it is difficult to maintain adequate ventilation.
4. Injuries to the esophagus.
5. Large chest wall wounds needing excision and closure.

Complications

Wet Lung. A dangerous complication of chest trauma is wet lung. It appears as a result of: contusion of the lung, intrabronchial bleeding, reduced ventilation after rib fracture, and aspiration. An accumulation of secretion, blood, and exudate in the parenchyma of the lung obstructs, completely or

partially, small or medium bronchi and causes multiple minute or large areas of atelectasis with impaired ventilation, which may be widespread in both lungs or confined to limited areas only. Some degree of hypoxia is always caused. This process may spill over into neighboring areas and to the contralateral lung, creating a fatal vicious circle, unless proper treatment is instituted in time. Prevention is achieved mainly by encouraging patients to breathe deeply and to clear the airway by coughing, as long as they are able to cooperate. Otherwise it may become necessary to intubate or to do a tracheostomy: then it is necessary to maintain bronchial toilet by repeated suction. Antibiotic cover should be provided.

Antibiotics. Infection frequently complicates thoracic injuries. Optimal antibiotic treatment is in accordance with sensitivity studies of bacteria cultured from infected wounds and secretions. Nevertheless, start prophylactic antibiotic therapy in the field: crystalline penicillin, 5 million units IV every 6 h. This high dose of penicillin is effective against most gram-positive bacteria, and gram-negative bacteria including anaerobes found in chest infections. If the patient is known to suffer from hypersensitivity to penicillin or its derivatives, a wide spectrum antibiotic should be used. A culture should be taken of every wound as early as possible. If infection ensues, penicillin should be exchanged – according to the sensitivity studies, preferably for two antibiotics from two different groups, such as a cephalosporin derivative and an aminoglycoside (monitoring of renal function is necessary).

Injuries to the Thoracic Wall

The thoracic wall is not only the container of the intrathoracic organs but plays an important part in the respiratory mechanism. It takes part in practically every thoracic injury and sometimes is the only injured part. A small external injury may direct our attention to severe intrathoracic damage within.
Major thoracic wall injuries may be life threatening (Fig. 1), even if there is no direct injury to the underlying lung.

Flail Chest

This is the most severe form of injury to the chest wall. It occurs when four or more adjacent ribs are fractured at two points, either on one or both sides of the sternum (Fig. 2). The area of the thoracic wall between the points of fracture loses its stability as related to the rest of the chest wall. This results in "paradoxical respiration". This unstable area is sucked inwards on inspiration whilst the rest of the normal thoracic wall moves out-

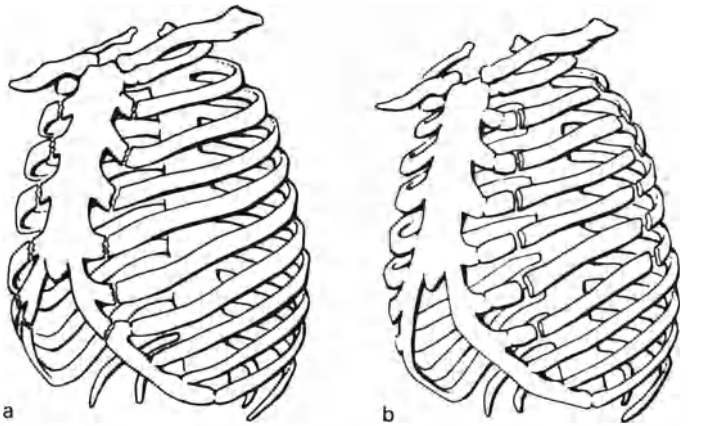


Fig. 2 a, b. The two principal forms of flail chest. **a** Anterior type is usually accompanied by a fracture of the sternum; **b** lateral type. [From Glinz (1981), Fig. 48]

wards. On expiration this area returns to its normal position or may even be pushed outwards. The paradoxical movement counteracts part of the respiratory activity of the stable part of the chest wall, and in order to compensate the patient has to increase his effort. In addition, most patients with flail chests have also suffered a contusion of the lung: the vicious circle of wet lung will tend to start. Coughing is less effective, and secretions accumulate in the airway. The result is hypoventilation and hypoxia. Often flail chest is obvious when first seen, though it may take some days to become apparent.

Contusion of the heart, hemothorax, pneumothorax, and extrathoracic injuries sometimes accompany flail chest.

Diagnosis. The history is of a blunt injury. Observation will reveal a part of the thoracic wall that moves paradoxically. The respiratory distress may not always be apparent at first, but deterioration will occur within hours or days. On palpation, crepitation of the rib fragments can often be felt. The auscultatory sounds of retained secretions in the bronchial tree appear soon and increase. Chest X-rays reveal the double row of fractures but when costal cartilages on both sides of the sternum are broken, no fractures will be seen. Within hours or a day or two spontaneous ventilation will become more and more difficult due to development of wet lung.

The signs and symptoms of flail chest may become apparent only gradually. This occurs possibly because some of the rib fractures are at first impacted or stabilized by remnants of periosteum and tend to loosen up later. The state of wet lung also takes some time to develop.

Treatment. On site and at the medical aid station, position the casualty with the intact side of the thorax upward; if the lesion is of the anterior type, the supine position is best. Raise the upper part of the body 30°. Encourage deep breathing and coughing. Treat pain with an adequate dose of morphine or its derivatives: the pain depresses ventilation and coughing more than a reasonable dose of morphine. Evacuate to hospital as early as possible. If evacuation is delayed and spontaneous ventilation becomes inadequate, endotracheal intubation and artificial ventilation may become necessary. IV fluids and antibiotics are started.

In *hospital* the treatment alternatives are: mechanical ventilation and the conservative method. Mechanical ventilation is complemented by part of the conservative protocol, and some patients, treated at first with the conservative method, will have to be ventilated if they deteriorate. Both require intensive care facilities but if necessary can be treated under less than optimal conditions, in a field hospital. Some surgeons prefer to use mechanical ventilation as soon as fail chest causes respiratory distress. Others prefer to try the conservative method first. Mechanical ventilation has a very significant rate of complications, sometimes with a fatal outcome.

On admission the casualty is reassessed for all types of chest and associated injuries.

The conservative method: (a) Restrict crystaloid fluid intake to a maximum of 1000 ml for resuscitation and then do not exceed 50 ml per h. (b) Give frusemide 40 mg IV on admission and then daily for 3 or 4 days. (c) If available, give methylprednisolone 500 mg IV and repeat every 6 h for 48–72 h. (Pharmacological doses of other steroids may be used if methylprednisolone is not available.) (d) Replace blood loss with blood or plasma and not with crystaloid solutions. (e) Give salt-poor albumin 25 g (100 ml) daily to maintain plasma oncotic pressure. (f) Analgesia: reduce pain with morphine. (g) Physiotherapy: keep airways clean by nasotracheal suction and use incentive spirometer, if available. (h) Oxygen: administer nasal oxygen if PO_2 is less than 80 mmHg. (i) Follow up: daily chest roentgenograms for the first few days. Look for pleural effusion, pneumothorax, and atelectasis, and treat appropriately.

Table 1. Indications for mechanical ventilation and weaning

	Support	Weaning
Respiratory rate/min	>35–30	<30–35
Arterial blood gases:		
PaO_2 mmHg	<60	>60
$PaCO_2$ mmHg	≥55	≤40

Note: For concomitant brain injury, maintain $PaO_2 > 80$ mmHg

Mechanical ventilation will become necessary if the patient's respiratory rate and blood gases indicate the need for support (Table 1). These criteria apply equally to a patient receiving an unsuccessful conservative treatment or for starting mechanical ventilation primarily.
Mechanical ventilation can sometimes be discontinued after 4 or 5 days, but often 2 or 3 weeks are necessary. The decision to wean the patient depends on his respiratory stability. This is examined by letting the patient breathe humidified oxygen spontaneously for 10–20 min under supervision. If no shortness of breath appears and the arterial blood PO_2 remains within acceptable limits on repeated examinations, weaning may be started (Table 1). An endotracheal tube should be used for the first few days but when it becomes obvious that mechanical ventilation be prolonged (over 5–7 days), perform a tracheostomy.
After weaning, conservative management continues until the clinical signs and chest roentgenogram show improvement sufficient for it to be gradually discontinued.
Physiotherapy should be continued all through convalescence (usually 4–8 weeks).

Fracture of Ribs

Blunt trauma is the frequent cause; the importance of fractured ribs is mainly in the concomitant damage done to the organs beneath them (lungs, diaphragm, liver, spleen). This refers not only to the damage that occurs immediately (contusion by compression or laceration by penetrating fractured ribs) but also to the secondary damage that may develop in the lung underlying the fractured ribs; because of the pain patients unconsciously reduce their respiratory movements in the injured areas. They inhibit the cough reflex. This leads to hypoventilation and accumulation of secretions in the part of the lung adjacent to the fractured ribs, setting the stage for the vicious circle described under "wet lung". The older the patient, the higher the risk of wet lung.

Diagnosis. Fractured ribs can often be diagnosed clinically without radiograms. The history of the injury will suggest the possibility. Local pain even when coincident with respiratory movements is not a certain sign of fracture. Often the crepitation caused by the movement of the fractured ribs can be felt on palpation or heard on auscultation. If this is not elicited, mild pressure on the part of the rib which is far from the point of fracture will cause pain at the site of fracture.
When available, a roentgenogram confirms the diagnosis. A standard, erect, chest roentgenogram is done in addition to the rib roentgenogram to exclude hemothorax or pneumothorax as well as contusion of the lung.

If the lower ribs are involved, damage to the liver and spleen should be excluded. If a fracture of the first rib exists, suspect damage to great vessels in the upper mediastinum.

Treatment: Whether on site, at the medical aid station and in hospital the treatment is similar. (Hospital facilities are necessary for the radiograms to confirm the diagnosis and to exclude underlying hemo- or pneumothorax.) Once damage to intrathoracic and intra-abdominal organs has been excluded, the most important consideration is to avoid development of wet lung. Though the patient cannot go on with his usual activity because of the pain caused by the fractured ribs, he is encouraged to be as active as possible. To relieve his pain he should receive morphine or its derivatives. Deep breathing and coughing exercises should be practised every waking hour. Intercostal block is not used routinely as it lasts for only a few hours and is not devoid of complications. Only patients who cannot be managed by the usual analgesics and who cannot be mobilized without it will need an intercostal nerve block.

If only a few ribs are fractured and the patient is not elderly or debilitated, hospitalization will not be necessary. If more than two or three ribs are broken, the underlying trauma may at first not be evident, and the patient should be admitted for observation and control radiograms. Elderly patients tend to withdraw into their beds at home, whereas in hospital physiotherapy and mobilization are initiated more effectively. As soon as those objectives are reached, patients can be discharged.

The pain of a rib fracture lasts from 2 to 4 weeks, depending on the degree of displacement.

Fractures of the Sternum

These are rare as compared with rib fractures. A fracture of the thoracic spine often accompanies sternal fracture when the injurying force flexes the thoracic trunk. It is important as a clue to internal damage (e.g., contusion of the heart) and thus should be monitored by recording vital signs and the central venous pressure every hour for 24–48 h: an ECG (and when possible ultrasound scans of the heart) is done to exclude such injury. Once cardiac damage has been excluded, the management is as for fractures.

Wounds of the Thoracic Wall

Wounds of the thoracic wall are important if they derange respiratory physiology by allowing the penetration of air through the wound into the pleural cavity. Often such a "sucking wound" declares itself by the sucking sound heard at examination. Other wounds admit air intermittently and then the leak may be missed. The arteries of the chest wall bleed more than the

pulmonary vessels. Bleeding from this source is a potent cause of hemothorax.
Even the smallest thoracic wound may overlie a tension pneumothorax or hemothorax: hence these must be excluded or treated promptly. If in doubt it is often wise to insert an intercostal tube (see indications for intercostal tube drainage).

Treatment. On site and at the medical aid station, if the wound is small and not an obvious sucking wound, it is enough to cover it with vaseline gauze, which in turn is covered by a larger gauze pack. The dressing is secured to the chest wall with tape without restricting respiratory movements. This fatty dressing acts as a valve: the vaseline gauze is drawn tight onto the skin on inspiration and prevents air from entering the pleural space. When coughing or straining occurs — and the intrathoracic pressure rises — the air is expelled between the skin and the vaseline gauze. A sucking wound (open pneumothorax) is managed by closed intercostal tube drainage (see Figs. 6–8) and dressing the wound in a similar manner. If the respiratory distress continues, immediate endotracheal intubation and manual mechanical respiration are indicated.
Prophylactic antibiotic therapy is commenced as early as possible.
In *hospital* exclude pneumothorax and hemothorax; if present, treat them first. Ensure that antitetanus and antibiotic coverage have been taken care of. Small wounds with clean edges need no local treatment.
Large and lacerated wounds are excised under general anesthesia, using mechanical ventilation, along the entire depth of the thoracic wall wound. If the pleura is open, its cavity is checked through the opening, and blood and debris removed by suction. If an intercostal drain has not yet been inserted, it should be done before closing the wound, always through a separate stab wound.
If damage to the lung is judged to be severe, the wound is enlarged to treat it but often a formal thoracotomy is more advisable (see later).
The debrided wound is closed by suturing the muscular layer using long-term absorbable suture material to close the pleural space. (The pleura itself, unlike the peritoneum, cannot be sutured). Sometimes it is necessary to mobilize a local muscle flap to achieve an airtight closure. The subcutaneous layer and the skin are left open and packed with vaseline gauze. The skin is closed by delayed primary suture 5–7 days later. If it is not possible to close the pleura hermetically a pack of paraffin gauze is used to seal the pleural space. It is removed (preferably under general anesthesia) 5 days later: by then, adhesions between the lung and thoracic wall will have formed. The wound is closed later by secondary suture or can be left to heal spontaneously by granulation if secondary suture is contraindicated by the general state of the patient.

Pneumothorax and Hemothorax

Accumulation of air (pneumothorax) or blood (hemothorax) in the pleural cavity is the most common disturbance resulting from thoracic trauma. There are three forms of pneumothorax: simple, tension, and open.

Simple pneumothorax results if air leaks from an injury in to the lung parenchyma (which may be spontaneous or traumatic). If the injury seals off before much air has accumulated in the pleural space, a simple pneumothorax will result (Fig. 3). The lung is partially expanded and occupies most of the hemithorax. The casualty will suffer moderately from pain, but there will be no appreciable respiratory distress. It must be remembered that the same injury may start leaking again and progress to a tension pneumothorax.

In tension pneumothorax (Fig. 4) the injury in the parenchyma persists and acts like a valve mechanism which admits air into the pleural space during inspiration but prevents its escape during expiration: the injured lung collapses, and the mediastinum is forcible shifted to the healthy side. The casualty suffers increasing respiratory distress, which, if not treated promptly, leads to a fatal outcome.

Open pneumothorax (Fig. 5) indicates that the pleural space communicates freely with atmospheric air. This results in collapse of the ipsilateral lung, shift of the mediastinum to the other side, and reduction in the volume of the contralateral healthy lung: severe respiratory distress ensues, and the outcome is fatal unless treated early.

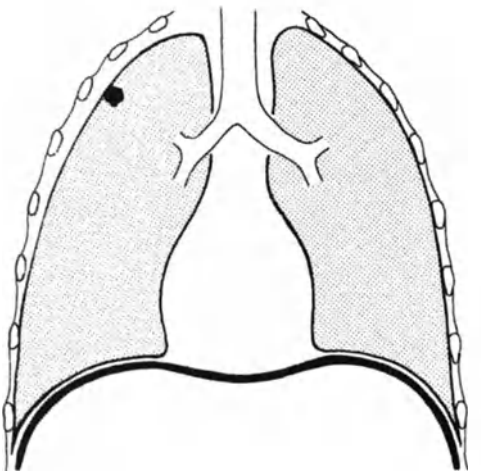


Fig. 3. Simple pneumothorax: small amount of air in pleural space, no atelectasis, no shift of mediastinum

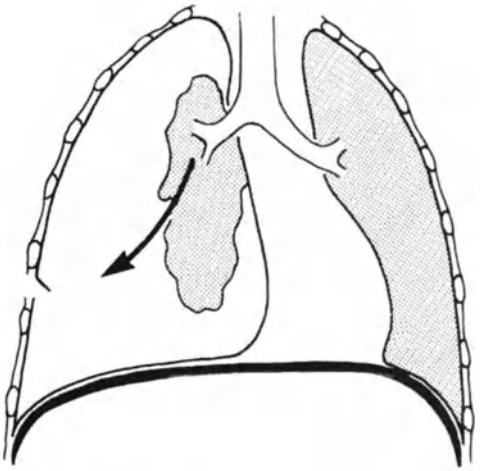


Fig. 4. Tension pneumothorax: valve-type laceration of lung, or thoracic wall, collapsed lung, shift of mediastinum, reduced volume of contralateral lung, flattened diaphragm

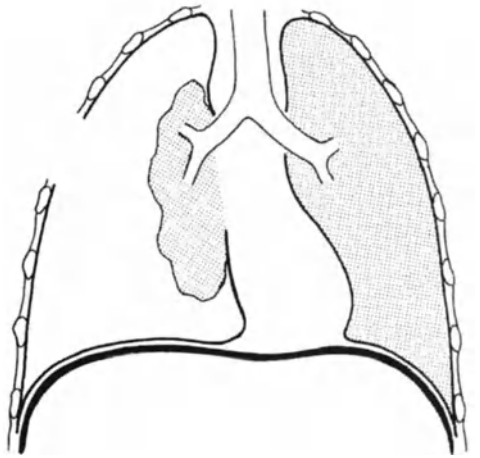


Fig. 5. Open pneumothorax: free communication between pleural space and atmospheric air, collapsed lung, shift of mediastinum, reduced volume of contralateral lung

With a hemothorax, the blood is lost to the patient's circulation, and this may be sufficient to cause hypovolemic shock. The volume taken up by the blood reduces the lung volume and can cause respiratory distress.
Hemothorax and pneumothorax often coexist in trauma patients and are then referred to as a hemopneumothorax. Hemothorax and pneumothorax are caused by both blunt and penetrating trauma: the management is the same.

Diagnosis. The history of wounds and abrasions on the chest wall direct attention to the thorax. Sometimes the only external mark is on the shoulder or abdomen (contusion or gunshot wound). A minimal simple pneumothorax can easily be missed. There is little or no respiratory distress. The respiratory movement of the thorax and the breath sounds are only slightly reduced. A chest roentgenogram is diagnostic. (NB: In thoracic trauma all chest roentgenograms are made in the erect position, standing or sitting up straight to demonstrate a fluid level, or air above the lung and fluid below it. If the roentgenogram is taken supine, air and fluid are easily missed; however, should an erect roentgenogram be contraindicated, a supine one is taken. If this does not result in a clear diagnosis, a "lateral decubitus" picture with horizontal X-rays is taken. This is done by turning the patient on his side (preferably the injured side up) and taking a roentgenogram with the plate at the back of the patient and the X-ray tube in front (the X-rays coursing in the horizontal plane). This will demonstrate any air above the lung parenchyma and any fluid below. If both are present, a fluid level will be seen on the radiograph. Only in casualties who cannot be moved at all, such as those with spinal fractures, will it be necessary to rely on a supine roentgenogram.

Tension pneumothorax presents with acute respiratory distress, an immobile hemithorax, hyperresonance, and reduced or absent breath sounds. Often venous engorgement can be seen in the neck. Open pneumothorax presents with similar findings in addition to an open wound communicating with the pleural space. Air is heard to pass through this wound as with a sucking wound.

Most patients with traumatic pneumothorax (and some with spontaneous pneumothorax) present with subcutaneous emphysema.

A patient with hemothorax will have less respiratory distress but will show the general signs of acute blood loss. The hemithorax will be immobile, dull to percussion on the dependent part, and the breath sounds reduced.

Chest roentgenograms are made in full inspiration. An expiration roentgenogram is indicated to demonstrate a small pneumothorax (during expiration there is a decrease in the total volume of the thorax, but no decrease in the volume of air in the pneumothorax; this relative increase of the volume of the pneumothorax can be seen on the roentgenogram: thus a small pneumothorax will be demonstrated better, but a simple pneumothorax can be transformed to look like a tension pneumothorax on an expiration film).

Treatment. On site and at a medical aid station, a simple pneumothorax needs no treatment, provided that surface evacuation to a hospital is available within half an hour: if the anticipated evacuation time is longer, closed intercostal drainage is performed. If the patient is to be evacuated by air or if he will require mechanical ventilation during transportation, *an intercostal drain must be inserted.*

Simple and open pneumothoraces are drained with a chest tube as soon as possible. A sucking wound is covered as previously described.
Tension pneumothorax is very often an extremely urgent situation. If equipment for intercostal tube drainage is not available immediately and the patient is in distress, insert a large-bore needle or a thick, plastic IV cannula through the chest wall into the pleura. The air will be heard to rush out and the patient will be relieved, at least for awhile, during which time proper drainage must be inserted. A word of warning! Such needles will not remain open for very long and can be relied upon for a short time only.
Hemothorax should also be drained as early as possible, and an IV infusion started to prevent or treat hypovolemic shock.
All casualties with all kinds of pneumothorax or hemothorax should be evacuated to a hospital as soon as possible. If intercostal drainage is not available in the field, the evacuation becomes even more urgent.
Summary of steps for treating pneumothorax and hemothorax, singly or in combination in the field:

1. Examine and assess patient.
2. Cover sucking wound (if present) with "valve type" dressing.
3. Drain hemo- or pneumothorax with intercostal tube (if specific equipment not available improvise with needles or cannulas as mentioned in text).
4. If not relieved: endotracheal intubation and mechanical ventilation. (NB: never ventilate a patient with tension pneumothorax before establishing intercostal tube drainage. An open pneumothorax may be ventilated before drainage or dressing of wound.)
5. Start IV infusion. (If hemorrhagic shock is more marked than respiratory distress, rapid fluid infusion should precede the previous steps.)
6. Evacuate to hospital.

Hospital treatment is essentially the same as that in the field, whilst utilizing the additional facilities available. An erect chest roentgenogram should be made as early as possible if a chest tube has already been placed before arrival. If the patient has not been drained and is in respiratory distress, then intercostal drainage should precede the roentgenogram; otherwise, the roentgenogram should be done first.
Arterial blood gases should be examined as early as possible to evaluate better the respiratory state of the patient. For tension pneumothorax with respiratory distress, perform intercostal drainage first. If hypovolemic shock is the dominant feature, start with IV cannulation and fluid administration. If ventilation is diminished by an obstructed airway or central apnea, endotracheal intubation takes precedence.

Note the initial volume of blood collecting in the drainage system. Put a mark on the bottle. The drainage of a large additional volume may become an indication for an urgent thoracotomy.
If the patient has arrived with a Heimlich valve attached to his drain, it should be replaced by a water seal system upon arrival in the ward. For the time spent in the emergency room, radiology, etc., if is preferable to leave the Heimlich valve in place.
The water seal system should be connected to a properly regulated suction source (negative pressure of 10–20 cm H_2O). When available this will shorten the time required to expand the lung, and stop the bleeding and the air leak sooner. When suction is not available, a good result can still be expected in most patients, but full recovery may take longer.
Remove the drain after 48 h have elapsed without drainage of air or fluids. This requires marking of the fluid level in the collecting bottle at least once every 24 h, and more frequent observation for an air leak while the patient is instructed to cough. Follow-up chest roentgenograms should be taken after introducing the chest tube and daily thereafter until the drain has been removed. These are best done with a mobile unit in the ward since transporting patients with underwater seal drainage within the hospital is hazardous. The patient sits upright in bed, supported, if necessary, by an attendant or with folded blankets or pillows.
Findings on follow-up roentgenograms may be: collections of air or fluid which may indicate the need for additional drains, and atelectasis, which should be treated with nasotracheal suction and physiotherapy. If the atelectasis does not disappear promptly with those measures, bronchoscopy with suction is indicated. On the morning after the removal of the chest tube, an additional roentgenogram should be done to make certain than no air or fluid has reaccumulated.

Closed Intercostal Drainage (CID) Location and Technique

Figure 6 shows the locations for intercostal drains. For trauma, the best location is in the midaxillary line at the level of the male nipple (5 cm below the angle of Louis). This rather high location avoids injury to the diaphragm and the organs beneath it. In some trauma patients, not relieved by the midaxillary drain, it may be necessary to place an additional anterior drain at the midclavicular line in the second intercostal space.

Method A — Routine:

1. Locate and mark the skin at the point for drainage (Fig. 6).
2. Prepare surgical field.
3. Local anesthesia, and pleural puncture: Inject 20 ml of lignocaine 0.5%–1% or similar local anesthetic along the lines shown in Fig. 7 (A is better, B is easier). The skin and the pleura are the most sensitive parts of the tract and should receive the bulk of the anesthetic. Use a fine

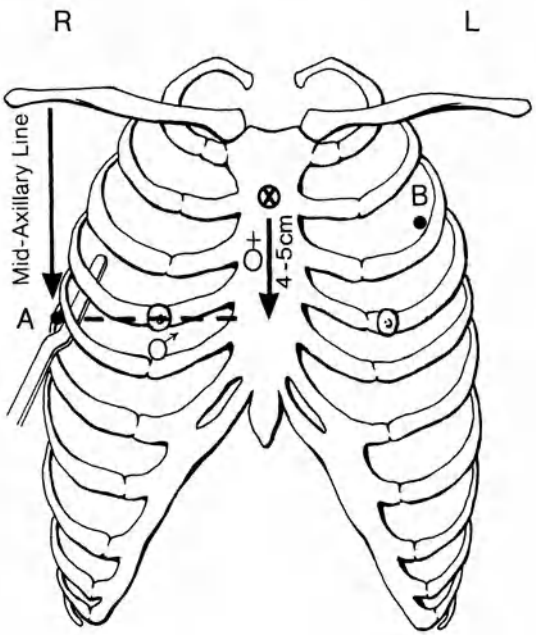


Fig. 6. Landmarks for placing an intercostal drain: for traumatic pneumothorax or hemothorax (*A*) in the midaxillary line at the level of the male nipple. In women this level is 5–6 cm below the level of the angle of Louis. For spontaneous pneumothorax, in the midclavicular line in the second intercostal space (*B*)

needle for the skin and change to a thicker needle (no. 18 or 19) for the deeper layers. When you reach the pleura (about 0.5 cm beyond the upper border of the rib) advance another 0.5–1 cm while pulling on the plunger of the syringe, thus performing a pleurocentesis.

If fluid or air return, it proves that there is either a pneumo- or hemothorax present, and you may proceed with the procedure. If neither air nor fluid appear, think again: are you performing the procedure on the wrong side? Is there another explanation for the findings that made you think that a hemo- or pneumothorax are present? Is it really necessary to drain? Only in the presence of compelling indications will it be wise to go on with the procedure.

4. Make a skin incision parallel to the rib and long enough for passing your gloved finger through it.
5. Using a regular hemostat clamp (preferably curved), spread the subcutaneous tissue and the muscle along the anesthetised tract until you reach and tear the pleura. The spreading should be almost as wide as the length of the skin incision. Pass a gloved finger through the tract and ascertain that an air or fluid space is present at the point of entry or at least that the pleura is free of adhesions at that point.
6. Grasp the drainage tube (without a trocar) with a large hemostatic clamp, near its tip (Fig. 8). Use the clamp to advance the tube into the pleura through the tract. When the tip is well within the pleura, stabilize

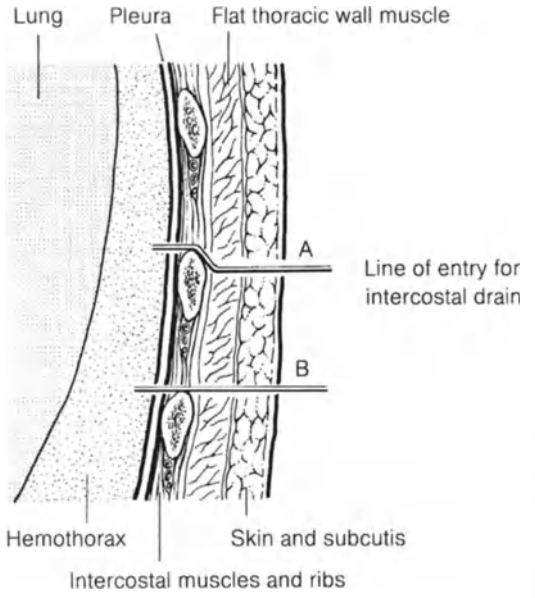


Fig. 7. Course for injecting local anesthetic and for passage of intercostal drain: *A*, preferred line; *B*, simpler line. Keeping close to the upper margin of the rib will prevent damage to the intercostal neurovascular bundle

Fig. 8. The tip of the chest tube held in a long clamp for guiding into the pleura

the tube with one hand and release the clamp with the other. Immediately occlude the tube with the same clamp to prevent air entering the pleura through the tube.

7. Advance the tube to the proper depth: 15 cm for an axillary line tube, 10 cm for a subclavicular one.
8. Put two strong sutures through the skin at the incision. Avoid passing the needle through the drain. The first suture, nearest the drain, is to be tied to the skin and then around the drain to prevent it from slipping out. The second, which is optional, should be a U stitch left untied for

closing the opening when the chest tube is withdrawn. Wrap it around the drainage tube.

9. After the drain has been tied, connect the tube to the Heimlich valve or to an underwater seal. Only then remove the large clamp from the tube and observe air and/or blood escaping.
10. Cover with a sterile dressing and tape to secure the chest tube to the skin.

Method B:
With pointed trocar, catheter drain. (May be used when presence of air or fluid space is certain. Requires more expertise than method A.) Follow the same steps as in method A, except for:

4. Incision in skin: almost twice the diameter of the tube to be used.
5. Using a regular hemostat clamp (preferably curved) spread the subcutaneous tissue and muscles along the anesthesized tract until you reach and tear the pleura. The spreading should be almost as wide as the length of the skin incision.

6a. Hold the trocar drain in one hand with its round end resting in the palm of your hand, for pushing. With your other hand guide the trocar by holding it near its tip.

6b. Insert the tip into the passage you have created and push gently but firmly while searching for the passage with the tip. If you cannot advance, repeat step 5, until a sufficiently wide passage is created. When the tip of the trocar has entered the pleura for 2–3 cm, withdraw the metal part of the trocar by the same length leaving the softer PVC tube leading inside the pleural cavity by 2–3 cm; now repeat the advancement of the trochar again until it projects 2–3 cm beyond the pleura and again withdraw the same length thereby leaving the PVC tube projecting into the pleural cavity 4–5 cm.

6c. Now prepare a large hemostat clamp and withdraw the metal part of the trocar rapidly and put the large clamp on the tube, to prevent air entering the pleura through the tube.

Proceed with steps 7–10 as in method A.
In an emergency, when proper trocar catheters are not available, sterile rectal tubes or urethral catheters of similar sizes can be used.

Valve Systems for Connection to Intercostal Drains

In the field, the Heimlich valve is the most useful appliance. It is a short, transparent, plastic tube with a connector at each side. On the inside is a soft flat rubber tube which acts as the valve mechanism. Care must be taken to connet this valve in the proper direction, which is marked with an arrow on the outer tube. In the original Heimlich valve the connector intended for

the side of the drain had a blue color, but other valves may be encountered.

The connection of the Heimlich valve to the intercostal tube will prevent air entering the pleural space. In order to avoid spillage of blood from a hemothorax a collection bag should be connected to the other end of the valve. Excellent bags are manufactured for this purpose but are usually not available. A sterile urine collection bag (2 l) can be used instead. Since the tubes of those bags have rigid connectors on them, a short, sterile connecting tube (PVC or latex) 8 mm in diameter and 5 cm in length should also be available. It is absolutely necessary to cut a 1–2 cm opening into the uppermost part of the bag, so that air from a tension pneumothorax can escape continuously.

In the hospital environment so-called underwater seal systems are usually employed. Many such systems are available, from improvised bottle arrangements to sophisticated disposable factory products. All of them have two functions: a one-way valve to prevent air from entering into the pleura, and a collecting vessel for blood or other fluids drained from other pleura. The third part is optional and sometimes provided separately from the other two: a suction regulator which enables continuous suction to be applied, to improve drainage at a controlled pressure (15–30 cm H_2O, negative pressure). The strong negative pressure from hospital wall systems or a mobile suction pump is dangerous when applied to an intercostal drain.

The simplest unit is the one-bottle system (Fig. 9). The tube should always be submerged in at least 2–3 cm of solution. It can function by itself without the additional components of the three-bottle system (Fig. 10). A two-bottle system can also function properly by combining the water seal bottle with either the first or the third bottle of the three-bottle system. In our

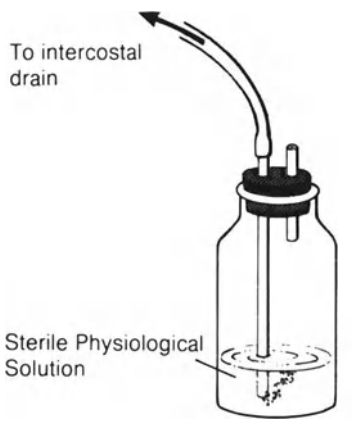


Fig. 9. One-bottle underwater drainage unit; it serves as a one-way valve and collecting vessel at the same time

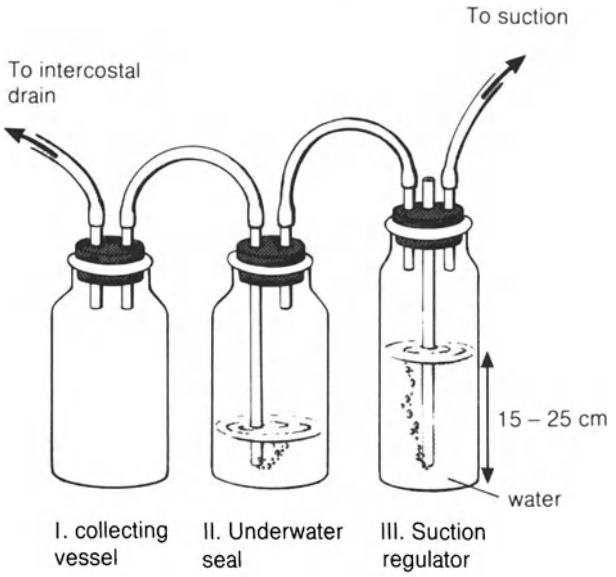


Fig. 10. Three-bottle system: The first bottle serves as a collecting vessel, the second as a one-way valve, and the third, which may be disconnected, serves to regulate the suction pressure

hospital we use the single-bottle system and usually attach a non-sterile suction control unit to it.

The water level in the underwater seal should alwags be at least 50 cm below the level at which the patient lies. If it becomes necessary to raise the bottle, this should be done only for the shortest possible time, during which the drain should be occluded. This should not exceed 1–2 min. If the bottle is full and has to be exchanged, another bottle with sterile physiological solution should be prepared in advance and the chest tube clamped just for the moment it is disconnected from the first bottle system until it is connected to the new one.

If the patient is being ventilated with an intermittent positive pressure respirator, the chest tube should not be clamped even for the above-mentioned maneuvers.

Sophisticated disposable factory products are very convenient, if available. They all operate on similar principles.

Subcutaneous and Mediastinal Emphysema

The presence of air in the soft tissues of the chest wall or in the mediastinum is always pathological. The air itself is usually not dangerous to the patient, but its presence means that something is wrong with the thorax. The sources of subcutaneous and mediastinal emphysema can be air from a pneumothorax that has leaked through the lacerated parietal pleura into the chest wall or perforation of the trachea, large bronchi, or esophagus. Subcutaneous emphysema is sometimes secondary to mediastinal emphysema since the air from the mediastinum can migrate to the neck and from there spread out in the subcutaneous plane.

Diagnosis. The diagnosis of subcutaneous emphysema is by palpation. The typical crepitation can be felt by the fingers on the skin on the patient. Crepitations can also be heard with the stethoscope. Gross subcutaneous emphysema is diagnosed by observation of swelling involving the face, neck, chest, abdomen, and scrotum.
Mediastinal emphysema can sometimes be diagnosed by auscultation: the crepitations can be heard in the precordium with a rhythm coinciding with the heart beat (Hamman's sign). On chest roentgenograms the air can be seen as dark spots in the subcutaneous area, and it often outlines the contours of the muscles of the chest wall; this renders the interpretation of the roentgenograms more difficult by obscuring the outlines of an underlying pneumothorax. Mediastinal air can be seen as dark lines following the borders of the mediastinum, pericardium, esophagus, and other mediastinal structures.

Treatment. Usually the only treatment necessary is to reassure an anxious patient. Occasionally the volume of air is so large as to cause respiratory distress: the air should then be let out. For subcutaneous emphysema incise the skin under local anesthesia beneath and parallel to the clavicles for about 3–5 cm. Spread the subcutaneous tissue with a hemostat down to the muscular fascia (the air bubbles out). Insert a small Penrose drain, fixed to the skin with a single suture. Cover the unsutured incision with a light dressing only. The air can be massaged towards the incision even from the face and abdomen.
For mediastinal emphysema a small cervical mediastinotomy is necessary. Under local anesthesia incise the skin 0.5 cm above the upper sternal border, for 3–4 cm. With a hemostat spread the tissues (subcutaneous fat and superficial cervical fascia). Keep very close to the upper border of the sternum. Avoid going deeper than the plane of the posterior plate of the sternum. To do this palpate with your finger that you have not gone too far. On reaching the posterior surface of the sternum, change the direction of your hemostat and try to proceed caudally along the posterior plate of the sternum, checking with your finger; when you can feel the posterior plate of

the sternum you have opened the mediastinal space and you will see air bubbles escaping. A soft rubber drain (Penrose) is introduced into the mediastinal space and secured to the skin with a single suture. The skin is left open. It can be removed after 2 or 3 days. The wound will heal spontaneously.
Both procedures should be performed with the usual aseptic surgical precautions.

Injuries to the Lung, Trachea, and Bronchi

Injuries to the lung, both penetrating and blunt, result mainly in hemothorax, pneumothorax, and contused or wet lung. Several situations may arise which will have to be managed specifically:

1. Hemothorax and pneumothorax should be managed as described above. In less than 20% of patients will it be necessary to perform a thoracotomy. After drainage of the pleural space, the lung will usually expand, and hemostasis will occur spontaneously. This is due to the relatively low pressure in the pulmonary vasculature. The coagulated blood will also stop most of the air leak.
2. Persistent bleeding through the drain of more than 200–300 ml per h for several hours will be an indication for early thoracotomy. Such bleeding may result from laceration of a large branch of the pulmonary artery, but more often it will be caused by a laceration of a systemic artery such as an intercostal or internal mammary artery. (Do not confuse this situation with that of massive bleeding, which indicates immediate thoracotomy.)
3. A large air leak may also be an indication for early thoracotomy. It causes much air to flow through the intercostal drain and on check roentgenograms the lung is not at complete expansion. Many of these patients will not be adequately ventilated even with a well-functioning and properly adjusted respirator. Such a situation signifies that a large bronchus or the trachea is lacerated. It is usually accompanied by mediastinal and subcutaneous emphysema. If possible, a bronchoscopy should be performed before operation, to visualize and evaluate the damage to the trachea and bronchi. Lacerations of the large bronchi or trachea should be sutured with fine (3/0; 4/0), long-term, absorbable, synthetic suture material.
4. Lacerated lung tissue, when found on thoracotomy, should be spared excision as far as possible. It has an excellent capability for recovery. Many air leaks and bleeding points from lacerated parenchyma can be stopped by simple suture (long-term, absorbable, synthetic, suture material). Areas of parenchyma suffused with blood or contused need not be excised. Only when most of the lobe is comminuted or when there is

severe, unrepairable damage near its hilum is a lobectomy indicated. High velocity bullets cause much more tissue trauma than just the passage of the bullet through the tissue, but they do not constitute an indication for operation or resection as such.

5. Foreign bodies (bullets, shell fragments, rocks, etc.) embedded in the lung parenchyma are not an indication in themselves for thoracotomy. When easily accessible during an operation performed for the given indications, they may of course be removed. It should be remembered that by dislodging such a foreign body hemorrhage may be started, which may necessitate lobectomy. Most foreign bodies in the lung will not have to be removed at all (for the indications for delayed operations, see below).
6. Contused lung tissue or parenchymal hematomas will appear on chest roentgenograms as patchy or solid radio-opaque areas of lung tissue. They are never an indication for operation. Antibiotics should always be administered, except for minimal lesions. Active physiotherapy and early ambulation will prevent the vicious wet lung. If the damage is widespread, respiratory distress will occur. In such situations the treatment is similar to the one described for flail chest and wet lung including, sometimes, mechanical ventilation. If thoracotomy is performed for one of the other indications and such contused or suffused parenchyma is encountered, it should not be excised.

   There are different criteria for removal of foreign bodies at a delayed operation, after recovery from the primary wound has occurred (at least 2 weeks after injury):

   - An object whose longest diameter exceeds 1.0–1.5 cm
   - An object in contact with the pleura
   - An object very close to a large pulmonary vessel or bronchus
   - An object with a sharp point
   - An object that changes position on serial chest roentgenograms
   - An object that is the cause of hemoptysis, cough, or recurrent infections

   It is usually possible to extract the foreign body at thoracotomy through a small incision from the nearest point to the pleura. In some patients a full posterolateral thoracotomy will be necessary. Care should be taken to avoid the large pulmonary vessels or bronchi.

Injuries of the Heart and Great Vessels

Most injuries of the heart and great vessels are fatal within minutes and are rarely seen by a physician before death. Amongst those who reach hospital

alive and receive immediate treatment, a large proportion may survive. Pericardial tamponade, wounds of the heart chambers or great vessels, and contusion of the heart are included. More complicated lesions such as wounds of the heart valves and some special injuries of the great vessels are beyond the scope of this book.

Treatment. On site the diagnosis cannot be confirmed, and the decision to afford priority of evacuation will depend on suspicion only. Treatment is supportive: start an IV infusion, establish an airway, and drain concomitant hemopneumothorax while waiting for evacuation. The main concern is to rush the patient to a hospital as soon as possible.

In hospital pericardial tamponade it is be prevented: Blood collects in the pericardial space and impedes the filling of the heart during diastole. This results in a drop in cardiac output, a fall in blood pressure, and if not treated promptly in a fatal outcome.

The source of the blood may be wounds of the heart itself and of the intrapericardial part of the ascending aorta or vena cava. Tamponade can occur both in penetrating and blunt injuries. The classical triad of congested veins (high venous pressure), distant heart sounds, and fall in arterial blood pressure is found in less than half the patients. A high degree of awareness is necessary in order not to miss the diagnosis. Chest roentgenograms and ECG will often be misleading. Echocardiography, if available, will detect the presence of blood in the pericardial space and give the most accurate diagnosis. Measuring and monitoring the central venous pressure is also reliable. Pericardiocentesis is both a diagnostic tool and has therapeutic value: the presence of blood in the pericardium will confirm the diagnosis and aspiration of even a relatively small volume will improve the patient's condition, at least temporarily, allowing time for orderly transfer to the operating room. All these methods of examination require time and cannot be employed in rapidly deteriorating patients. If there are signs or history of injury in the area of the heart, and other sources of arterial hypotension have been excluded or taken care of, it may become necessary to start a thoracotomy, even in the emergency room. This should be done if cardiac arrest has occurred or is imminent in order to resuscitate the heart before the advent of irreversible brain damage, as well as to control the bleeding which is causing the tamponade. Suspected wounds of the heart are one of the few remaining indications for open heart massage.

Procedure

1. Pericardiocentesis (Fig. 11): Use a long needle (10–15 cm) with a short bevel, like a no. 18 gauge spinal needle or, preferably, a special pleural puncture needle (even thicker), which has a short bevel and blunt point, to avoid injuries to the myocardium and coronary vessels.

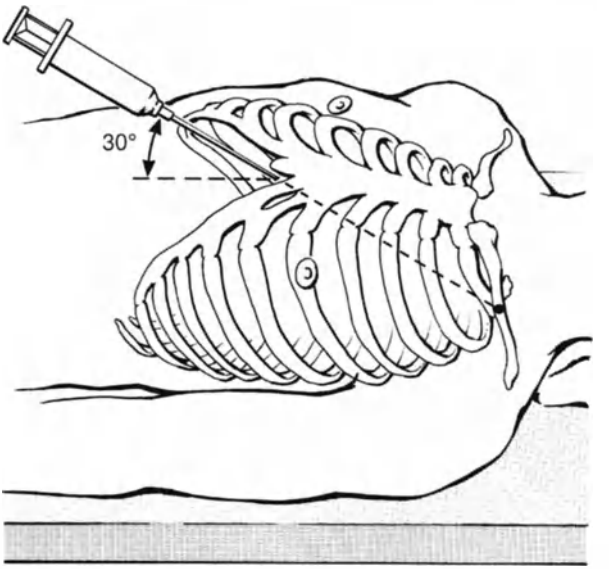


Fig. 11. Technique of pericardiocentesis (see text). [From Glinz (1983)]

For such a needle precede the puncture with a scalpel stab wound through the skin. Have the needle mounted on a 10 - 20 ml syringe containing a few ml of saline to lubricate the plunger. The point of puncture is inside the angle between the xyphoid process and the left costal arch. The needle should be held at an angle of 30° to the horizontal plane and point in the direction of the middle of the clavicle. If a blunt needle is used, it should be rotated while advancing the point. On reaching the pericardium, some increase of resistance will be felt and a sudden decrease upon entering it, especially if a hemopericardium is present. If possible, it is advisable to perform the pericardiocentesis with an ECG monitor: connect the hub of the needle to the V lead of the ECG monitor with the help of an alligator clip. On contact of the needle point with the epicardium the ECG pattern will change markedly, mainly by upward shift of the ST segment. This will be a warning to avoid myocardial damage and a sign that the needle has penetrated the pericardium. When the pericardium has been entered as much blood as possible is aspirated. Even a relatively small volume of blood can give significant, if temporary, relief of the tamponade: the patient is then taken to the operating room in a better condition. A positive puncture is always an indication for operation; a negative puncture does not exclude hemopericardium, as much of the blood may be clotted and obstruct the needle.

In such a case the decision to operate will depend on the general state of the patient.

2. Surgical approach: For wounds of the heart the supine position is always preferred. Endotracheal intubation and ventilation should always precede the incision. An anterolateral thoracotomy in the fifth intercostal space on the left side is best. It is the quickest approach to reach the heart for open massage. If necessary, the incision can be enlarged across the sternum for more exposure. The pericardium should be opened longitudinally anterior to the phrenic nerve. Clots and blood are evacuated and massage started if the heart has arrested. Even while massaging one can start looking for the lesions which bled into the pericardium. Most of them will be found in the anterior aspect of the heart, most often in the right ventricle.
   While massaging, spreading of the incision is achieved with appropriate available automatic retractors. Direct electrical defibrillation is used when indicated, and the same medication given as in closed cardiac massage. If the cause of cardiac arrest has been the tamponade, normal sinus rhythm will often be achieved.
   Wounds of the heart should be occluded by finger pressure until suture becomes possible.
3. Intrapericardial suture: Wounds of the atria or the ascending aorta can be occluded with a curved vascular clamp and sutured with a continous, fine, vascular suture. Wounds of the ventricular myocardium should be sutured with 1/0–2/0 silk, preferably buttressed with teflon-felt pledgets, avoiding the coronary arteries. The suture should not penetrate the endocardium.

If the coronary arteries are very close to the wound, vertical mattress sutures should be passed underneath the artery.

Injuries to the Esophagus

Esophageal injuries are rare compared with most other thoracic injuries, and especially rare in war and disaster situations. When caused by penetrating trauma, esophageal injuries are often accompanied by injuries to the great vessels or the trachea. They can usually be diagnosed and taken care of only in a hospital environment. The contamination of the mediastinum will cause cellulitis and sepsis. A mediastinal abscess can develop and perforate into one or both pleural spaces, causing empyema and sepsis.

Diagnosis. Whatever the cause, a perforation of the esophagus will cause spillage of esophageal contents, containing virulent flora, into the mediastinum or neck. If the patient is conscious he will suffer from severe pain. In rupture of the cervical esophagus the pain is located in the neck. For the

thoracic esophagus location is in the back, at a level roughly corresponding to the level of the rupture. In the lower thoracic esophagus pain may be referred to the epigastrium (which may be misleading and result in the performance of a laparotomy).

On physical examination, subcutaneous emphysema may appear in the neck some time after the injury. On chest roentgenograms, mediastinal or cervical air will be seen, and the retrotracheal space will appear widened on the lateral view. Contrast studies of the esophagus are the most important diagnostic tool and should be done as early as possible when an esophageal injury is suspected. Unfortunately, a negative examination does not exclude esophageal perforation, and if clinical suspicion is strong, it should be repeated early, so as to diagnose the perforation as quickly as possible.

Treatment. On site and at the medical aid station treatment is supportive only. *Hospital:* Perforations of the cervical esophagus are less serious than those of the thoracic esophagus, and easier to treat. All perforations (if diagnosed early enough) should be operated upon. If the operation is performed within 8–12 h of the perforation, it will usually be possible to suture it. Within 24 h some perforations may still be manageable by suture, but in many patients the tissues at the site of perforation will be necrotic and will not hold sutures. If pyopneumothorax has been established and more than 24 h have passed, drainage procedures only should be performed.

If the perforation is in the cervical esophagus, an incision at the anterior border of the sternocleidomastoid muscle is used from that side which is nearer to the perforation (as demonstrated radiologically). The esophagus is reached by dissecting between the trachea and carotid sheath. Care must be taken to identify and divide between ligatures and the middle thyroid vein and not to injure the recurrent laryngeal nerve. For the upper thoracic esophageal injury a right posterolateral incision is performed and for a lower thoracic esophageal wound, a left posterolateral thoracotomy.

The esophagus should be exposed well above and below the perforation to ensure drainage from the contaminated tissues. If the tissues at the site of the perforation are not too friable to hold sutures, it should be repaired with two rows of sutures. In the thoracic esophagus, the suture line can be reinforced with a pleural flap. Drainage should be provided for: in the neck, two Penrose drains should be passed through a stab wound near the incision pointing up and down along the esophagus. For thoracic lesions, two pleural drains should be placed as near as possible to the exposed esophagus. If the tissues are too friable for suturing, the same drainage procedures should be employed without suturing.

Cervical and upper mediastinal abscesses should be drained through the neck, even if the diagnosis was late. In established pyopneumothorax in a septic patient, thoracotomy is better avoided. Closed intercostal tube drainage is performed. *In all patients diagnosed too late for suturing, a gastrostomy should be performed,* at first for drainage of the stomach to avoid re-

flux and later for feeding. If the lesion is low in the esophagus, it is advisable to perform a feeding jejunostomy as it will be necessary to drain the stomach for a longer period through the gastrostomy. In some patients it may become necessary to perform also a cervical esophagostomy, by dividing the esophagus, and diverting the upper part to the skin and suturing the lower part. This will stop contamination of the fistula. Repair of the continuity of the GI tract will be necessary later on. It is essential to administer IV broad spectrum antibiotics as early as possible and vary the antibiotics according to culture sensitivity studies. Anaeorobic pathogens should also be considered even if they are not identified in cultures.
Before gastrostomy or jejunostomy feedings can be started, it is advisable to support the patient by total parenteral nutrition.

Injuries to the Diaphragm

Ruptures of the diaphragm are rare. They occur on the left about twice as frequently as on the right. Most of them are blunt injuries, resulting from sudden compression of the abdomen and thorax, exerting pressure on the diaphragm. In war, penetrating injuries will be more frequent. Another mechanism of injury in blunt trauma is a laceration of the diaphragm by the point of a fractured rib. Associated injuries of abdominal viscera are common and of great significance. Intrathoracic associated injuries are less frequent and usually manageable without operation. The consequence of such a tear is the displacement of abdominal organs into the chest, due to the physiological pressure gradient. Such displacement can be very voluminous and interfere with respiration, by pressure on the lung and displacement of the mediastinum. The displaced viscera are always in danger of incarceration, even years after the injury.

Diagnosis. The diagnosis is often overlooked, due to the grave condition of these casualties. The physical signs of decreased breath sounds, dullness, or hyperresonance can occur in other conditions. Only bowel sounds heard on auscultation of the thorax lead to the diagnosis. On roentgenograms of the chest the displaced viscera can be seen, sometimes as a homogeneous opacity, differing in contour from that expected in hemothorax or atelectasis, sometimes as multiple rings with a translucent center, representing bubbles in the viscera, and sometimes as a large bubble, caused by a dilated stomach or large intestine.
Another reason for missing the diagnosis is the delay of herniation of the abdominal organs into the chest, which may occur only a day or two after the injury.

Treatment. It is exceptional to diagnose this situation in the field, and even if it is diagnosed practically nothing can be done to relieve it. One practical conclusion of the diagnosis is to avoid pushing a trocar catheter into the displaced organs. A nasogastric tube is passed as soon as the diagnosis is suspected to avoid additional distension of the stomach or intestines inside the chest.

In the hospital, surgical repair should be performed as early as possible, bearing in mind that other associated injuries may be more important. The approach for acute ruptured diaphragm is usually through a laparotomy, the main reason being that associated intraabdominal lesions usually require urgent attention. It also happens that the rupture is first discovered during such laparatomy; the diaphragm should be inspected in all laparotomies performed for blunt trauma of force severe enough to cause such a rupture and for penetrating trauma of the upper abdomen. Only ruptures of the right diaphragm in which the liver is displaced should be approached through a right posterolateral thoracotomy in the fifth or sixth intercostal space. The diaphragm is sutured with two rows of strong, nonabsorbable sutures. Before starting this suture the pleural cavity must always be drained with a proper thoracic drain, connected to underwater drainage.

If the herniated viscera are discovered in the thorax at a time when adhesions have already formed, the approach should be through a thoracotomy, as this is the only way to deal with those adhesions.

The mortality associated with diaphragmatic rupture is high. A large part of this mortality is due to the accompanying injuries, but some patients will die due to the unrelieved respiratory disturbance, if not diagnosed and treated early enough.

Equipment Required for Treatment of Thoracic Injuries in the Field

1. Respiratory resuscitation: A laryngoscope and endotracheal tubes (sizes 7-8-9) are necessary as well as an Ambu respirator. For children, small size blades for the laryngoscope and small endotracheal tubes must be available. A spreader for the teeth, an "airway" tube, and a manually powered suction pump should also be available.
2. Intercostal drains: The best known is the Argyle Trocar Catheter, and its use has been described. For adults the sizes 32F and 28F are the most practical. Smaller tubes tend to clot, even when used for pneumothorax. For children, smaller catheters are needed (sizes F24, F20, F16 and F12 form a practical range).
3. Valves: Heimlich valves, a sterile urine collecting bag (12 l capacity), and a shorter connection tube (PBC or latex) with an internal diameter of

8 mm and length of 5 cm (to connect the urine bag to the Heimlich valve).

4. Instruments for closed intercostal tube drainage: The instruments listed may be part of other specific sets included in the medical officer's pack, or comprise a separate set for this purpose:

   1 Scalpel handle (to fit no. 11 blade)
   1 Regular hemostat (curved)
   1 Large hemostat (curved)
   1 Pair of scissors
   4 Sterile towels
   1 Pair of sterile gloves (appropriate size)
   1 No. 1 silk suture with cutting needle
   1 No. 1 surgical blade
   2 Syringes (10 ml)
   2 No. 20 needles
   2 No. 18 needles
   2 Sterile gauze pads (5 × 5 cm)
   Surgical adhesive tape 7.5 cm large
   Skin disinfectant
   20 ml Lignocaine 1% (sterile)

5. Dressing for thoracic wounds:
   Sterile vaseline gauze packs (sizes 5 × 5 cm and 10 × 10 cm)
   Sterile gauze pads (sizes 5 × 5 cm and 10 × 10 cm)
   Elastic adhesive tape

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4.9 Management of Abdominal Injuries

R. R. Rozin and R. Pfefferman

Introduction

Frequency of Abdominal Injury

In modern warfare, abdominal wounded constitute 4%–10% of all casualties and approximately 10% of those killed in action. In limited military conflicts, particularly guerilla incursions and hand to hand fighting, the frequency of abdominal wounds increases. During the Vietnam War, where light weapons were mainly used by the Vietcong, 14% of all American casualties had abdominal wounds. The incidence is slightly higher among the infantry than the armored corps and artillery.
Most abdominal wounded also suffer injuries to other body systems, especially to the chest, limbs, and soft tissues; only 15%–20% are injured solely in the abdomen.

Wounding Agents

In peacetime most abdominal injuries are caused by blunt trauma. In war the majority of abdominal injuries are due to penetrating wounds from bullets and fragments. Artillery has been the main wounding agent during all modern military conflicts. During terrorist activities and limited incursions, most abdominal injuries are caused by bullets.
Wartime blunt injuries to the abdomen are caused by road accidents, falls, or explosions.

Types of Injury

Penetrating Wounds

Damage caused by bullets penetrating the abdominal cavity at a high velocity is much more extensive than that caused by the low-velocity bullets used in the past.

The severity of the wound is affected by the density and elasticity of the injured tissue: the denser the tissue and the lower its elasticity, the greater the damage. Dense organs such as the liver, spleen, and kidneys are more extensively damaged by high-velocity bullets than fatty areas or hollow organs.
The damage caused by bullets fired from low-velocity weapons is usually limited to the area of the bullet's path. A single fragment of shrapnel usually causes less damage than a bullet. Extensive intraabdominal damage can be caused by simultaneous injury by numerous fragments of shrapnel, when widespread local damage and an extensive loss of the abdominal wall may occur.
The wounding track of shrapnel and bullets may be straight and simple, but at times it can be complex, changing its route to an entirely different direction and injuring additional organs lying in its new path. It is therefore wrong to assess internal injuries solely from the site of entrance and exit wounds.
Most injuries occurring in the abdominal cavity are multiple, especially in critically injured abdominal casualties who require immediate surgery in a field hospital.
Specific intraabdominal organs are injured in direct proportion to the volume they occupy within the abdominal cavity. Therefore, the large and small intestines and the liver are the most frequently injured.

Blunt Injury

Blunt injuries incurred during wartime or civilian disasters are caused by road accidents, blast from explosion which throws casualties against hard surfaces, impact of flying objects, or crush injuries. Blunt injuries can be caused by the blast itself in air or — especially — under water.
In blunt injuries the injuring force can cause internal organs to be thrown against the hard spinal column or the rib cage. A special type of injury is caused by sudden deceleration from high speed.

Pathophysiology

Loss of Blood

The immediate life-threatening danger due to abdominal injuries is loss of blood. Hemorrhage is the almost inevitable result of any injury to intraabdominal organs. Damage to the large blood vessels or to parenchymatous organs such as the spleen and kidneys can cause exsanguination.

Some injuries cause almost immediate death before medical aid can reach the casualty. Most abdominal casualties will require blood transfusions, and those in need of urgent surgery will need massive transfusion.

Loss of Fluids

Intravascular and extracellular fluid is always lost in abdominal injury. The fluid is lost by the escape of fluids into the peritoneal cavity with peritonitis, and paralysis of the stomach and the intestines causes a rapid influx of fluid into the intestinal lumen. In addition, fluid accumulates in the area of the injury in the form of edema and extravasation. This loss of fluid causes a rapid contraction of the intravascular space and a decrease in the circulating volume. All abdominal casualties should be considered hypovolemic and requiring volume substitution as soon as possible.

Acute Dilatation of the Stomach

The stomach almost invariably dilates rapidly after intraabdominal injury. A large amount of air, fluids, and, later, digested blood accumulates within the stomach's lumen. The blood gives a "coffee ground" appearance to the stomach contents. The dilatation of the stomach stimulates vomiting, which may cause aspiration of the stomach contents into the lungs. The extreme distension of the abdomen causes great discomfort to the wounded and pressure on the diaphragm by the dilated stomach can impair pulmonary function and cause respiratory distress.
The peritonitis or peritoneal irritation also causes paralytic ileus along the entire intestinal tract and this in turn causes abdominal distension. In the presence of gastric dilatation it is very hard to evaluate abdominal injury and any evaluation should be delayed until after the stomach is properly evacuated and the distension relieved.

Peritonitis

Any violation of the integrity of the peritoneal cavity causes irritation of its surfaces and pain and tenderness. Blood by itself rarely causes gross irritation but when mixed with intestinal contents or fragments of parenchymal organs gives rise to the classic signs of peritonitis. Perforation of the hollow organs causes spillage of food, intestinal contents, and fecal contents and peritonitis develops rapidly. Foreign bodies may be impaled into the peritoneal cavity and they too will cause peritonitis. It is therefore mandatory to assume that any intraabdominal injury is accompanied by peritonitis.

Diagnosis

Abdominal injury may be diagnosed very easily when there is damage to the abdominal wall and abdominal contents are protruding or are obviously injured. It should therefore be assumed to exist whenever there are penetrating injuries to the abdominal area. The injuries to its contents are much less obvious when there is blunt injury or when penetration is above or below the confines of the abdomen. Chest injury below the nipple must be considered also as abdominal injury. Trauma to the buttocks and perineum should also arouse suspicion that the peritoneal cavity has been breached.

Wounded from blast injuries, casualties evacuated from under heavy objects, and unconscious wounded should be suspected of having incurred intraabdominal injury and the injuries should be ruled out. *Hypovolemic shock without an obvious source of bleeding should alert the examiner to the possibility of intraabdominal injury.* Abdominal distension and vomiting may also indicate injury to abdominal organs. As a rule there is no difficulty in diagnosing penetrating abdominal injuries as there is accompanying abdominal pain, tenderness, and rigidity.

All casualties should be stripped of their clothing completely and searched for signs of penetrating injuries. Occasionally these are hidden under blood stains, dirt, or hairy areas.

Blunt injury to the abdomen can be more difficult to diagnose. It is easier to diagnose when there is trauma to the abdominal wall in the form of abrasion or surface bleeding. However, the signs of peritoneal irritation do not always develop during the initial hours following injury. Sometimes the only indication of intraabdominal bleeding will be shock or postural hypotension. Only later on will local or generalized sensitivity develop in the abdomen, accompanied often by pain referred to the shoulder.

Wounded who have suffered blunt injuries or trauma to the pelvis should be examined for abdominal injury. When pressure on both sides of the iliac crest causes pain or when there is pelvic instability one should suspect fractures and/or dislocations of the pelvis. Associated injuries are the rule in this type of trauma, and retroperitoneal hemorrhage and injuries to the bladder and urethra are common.

The inability to pass urine and a drop of blood at the tip of the penis indicate a tear at the base of the bladder or the proximal part of the penis.

Peripheral lower limb pulses should be examined and documented. The disappearance of a pulse in one of the lower limbs can indicate injury to the intraabdominal arteries.

Injury to adjacent body systems: signs of pneumothorax and hemothorax should be searched for in all casualties with injuries to the abdomen. Injury to the back should be checked for and a neurological examination conducted. Penetrating wounds often damage vertebrae, the spinal cord,

the cauda equina, and/or large nerves. It is difficult to make a diagnosis in comatose or restless, uncooperative patients.
In combat or mass casualty situations, no diagnostic procedure should be performed unless it has an immediate bearing on treatment. No diagnostic tap of the abdomen should be performed unless a surgeon and operating theater are available.
The key to diagnosing intraabdominal injury is a high degree of suspicion.

Treatment

On Site

The abdominal casualty should be treated according to the same order of priorities as other wounded. First, the airway should be secured and respiration started. The chest injuries which often accompany abdominal injuries should be treated first. The chest wall should be properly dressed and intercostal drainage must be performed where indicated.

Fluid Infusions

Most abdominal casualties are dehydrated or suffering from a degree of hypovolemia. The severity of their volume depletion depends on the severity of the abdominal wound, on accompanying injuries, and on the time elapsed since wounding. Some of the wounded are seen when they are in deep shock and have a very distended abdomen due to blood accumulation. These wounded need rapid volume repletion through several intravenous routes.
Every abdominal casualty requires fluid infusions. For the very severely wounded, central veins are required; for the less severely wounded, one or two peripheral veins may suffice. The infusion should be established in one of the veins of the upper half of the body: infusions into the femoral vein or the inferior vena cava will dissipate in the abdominal cavity or the retroperitoneum without increasing the blood volume. Volume repletion is carried out with the aid of Hartman's solution. The infusion should be given very rapidly. Within a few minutes, or after the infusion of about 2000 ml, the hemodynamic state usually begins to stabilize. The infusion is continued and calibrated by frequent examinations of the pulse, blood pressure, urine output, and general condition of the casualty.
Blood is not usually available in the field and volume replacement is done mainly with crystalloids. Experience has shown that volume replacement even with crystalloid solution alone is very effective during the first stages of resuscitation. The majority of wounded can be stabilized for a period of

a few hours without colloids or blood. Some of them will have extreme hemodilution (Hct of about 10%) and still survive.

Gastric Drainage

A nasogastric tube should be introduced in every abdominal casualty or any casualty suspected of having an abdominal injury. Drainage of the stomach will prevent gastric dilatation, improve respiration, and calm peristaltic activity. In the unconscious casualty, in order to prevent aspiration of vomitus, a tracheal tube should be introduced first and the cuff inflated, and only then should the nasogastric tube be introduced. The nasogastric tube can be introduced more easily when the casualty sits up and swallows some water. The cooperation of the casualty is very important, and the tube should be introduced gently. The stomach should be emptied of all traces of food and it should be flushed clean.

Catheterization

Abdominal wounded tend to develop reflex urinary retention due to pain, sympathetic stimulation, and administration of narcotics. The distended bladder adds to the abdominal pain and to the patient's distress, and catheterization should therefore be performed at an early stage. The catheterization allows monitoring of the urinary output, a parameter which is most helpful in monitoring volume replacement. The urine should be observed as hematuria may be present, signifying injury to the urinary system.
Whenever there is a likelihood or suspicion of a tear in the urethra, a catheter *should not* be introduced. In cases of fractures of the pelvis or bleeding from the urethra, catheterization should be delayed until arrival at a medical facility where a surgeon is available.
In cases where catheterization is contraindicated but urinary retention with distention of the bladder is present, suprapubic drainage is established with the aid of a needle or a plastic tube.

Local Treatment

Wounds of the abdominal wall should be flushed with normal saline solution to wash away all dirt and foreign bodies and covered with sterile dressings.

Analgesics

After the definitive diagnosis of abdominal injury is made, analgesics can be given freely. The indication for operation is absolute and it is no longer important not to mask the signs of peritoneal irritation. On the other hand, when the diagnosis is only tentative and there are no obvious signs of intrabdominal injury, analgesics should be withheld.

No analgesic is needed in abdominal injuries accompanied by little pain or for casualties in hypovolemic shock who experience no pain at all. However, for pain due to the abdominal or associated injury, morphine (10–15 mg) or meperidine (75–100 mg) may be administered intravenously (never into the muscle, since absorption is unpredictable and, especially in hypovolemia, the drugs may remain in the tissues without being transferred to the bloodstream and then, when perfusion improves, may be absorbed suddenly and cause respiratory depression).

Antibiotics

Every abdominal injury will cause bacterial spread. It is imperative to start the antibiotic therapy as soon after the wounding event as possible. Therefore antibiotic treatment should be started in the field and continued throughout the supportive treatment until surgery. Because the bacterial contamination in intestinal injury is very varied, broad-spectrum antibiotics covering the full range of gram-negative bacteria and anaerobic bacteria should be utilized. A practical combination is a high dose of crystalline penicillin (5 million units) administered intravenously and either chloramphenicol (1 g) or gentamicin (240 mg) also administered intravenously. These antibiotics should be added to the first infusion bag administered. Additional crystalline penicillin (5 million units in each bag, to a total of 20 million units) should be maintained during evacuation.

Follow-Up and Evacuation

The abdominal casualty requires frequent examinations for evaluation of vital signs and the state of the abdomen. These frequent examinations are an essential part of the treatment of abdominal casualties and help in establishing the priorities of evacuation and treatment.

Medical Aid Station

At the medical aid station, wounded can be treated under better conditions than on the site. They are reevaluated and examined without their clothing. Whenever the treatment described above for on-site management has already been instigated, it should be continued: fluid infusions in the form of crystalloid solutions and, when available, colloid solutions should be administered as required.

Many wounded, however, will arrive at the medical aid station without having been treated fully on site. These casualties should be given intravenous fluids, and have gastric drainage, catheterization, local treatment, and antibiotic treatment as outlined for on-site treatment. Some of the casualties will only at this stage be diagnosed as having abdominal wounds, and therefore it is important to note their symptoms on the accompanying medi-

cal chart. At this stage the blood pressure, pulse rate, nature of the contents of the gastric washings, and urine output should be noted.
The medical aid station provides better conditions for the insertion of lines into central veins for more efficient infusion and ease of evacuation. At this stage, the infusion rate is better calibrated.
Whenever there is blood available, transfusion should be started early. About 80% of abdominal casualties will require blood transfusions during their initial treatment. The average blood requirement for an abdominal casualty is about 2500 ml.
If the casualty arrives at a medical facility already with wound dressings, these should be removed and the wounds examined. It is important not to poke at the wounds in order to assess their depth or to attempt to remove shell fragments or foreign bodies.
If intestine or omentum protrude through the abdominal wall, they should not be replaced inside the abdominal cavity. The protruding organs should be moistened with saline solution and covered with large soaked pads.
The size of penetrating wounds should be noted and documented. Injuries to the lower chest, buttocks, and perineum should be considered as intra-abdominal injuries until proven otherwise.
The main aim of the treatment given at the aid station is to prepare the abdominal casualty for further evacuation. The time should be utilized for improving hypovolemia, for further decompression of the intestinal tract, and for the introduction of high levels of antibiotics.

Field/Base Hospital

The hospital should be capable of offering the abdominal casualty surgical options and better conditions for resuscitation.

Triage

Sorting is carried out in the hospital under much better conditions and by experienced surgeons. The casualties are completely undressed and, when necessary, radiography and CT scanning can be done and abdominal taps performed.
In mass casualty situations when surgical options are available but limited, the surgical priority of most abdominal wounded can be lowered and they can wait for operation for long periods of time or they can be safely transferred to other hospitals. Almost 80% of abdominal wounded can safely tolerate a delay of 6–8 h in their operative treatment, if proper supportive treatment is initiated early and maintained throughout. This treatment includes anticipatory volume replacement, nasogastric suction, proper oxygenation, administration of antibiotics, and monitoring.

Operative Priorities

Clinical experience is the key to the selection of casualties capable of withstanding delay when operative facilities are limited and decisions must be made as to who has priority to the operating room. The clinical judgment and the experience of the triage officer are of prime importance.

The abdominal casualties that *cannot withstand delay* are those in whom the vital signs deteriorate despite vigorous hemodynamic resuscitation: rapid introduction of blood and fluids in large quantities fails to stabilize the vital signs, or stability is obtained but is very brittle. Other indications for immediate surgery are extensive abdominal wall defects with evisceration, and a combination of thoracic and abdominal wall injury which demands thoracic exploration.

Abdominal wounded who can withstand further evacuation are those who are stabilized with the aid of a few liters of crystalloid solution and with the administration of 800–1200 ml whole blood over a period of 1–2 h. The group which can be further evacuated is characterized by the fact that they respond to supportive therapy, and their blood pressure and urinary output can be maintained adequately with a steady rate of volume replacement.

Operative Procedures

Casualties in need of immediate surgery will require long operations, tying up several medical officers and operating facilities. An average of 6000 ml blood is needed for such abdominal casualties before and during surgery. The operative procedures include the entire spectrum of surgical solutions for trauma of the organs involved. Injury to the liver requires hepatic lobectomy. Partial resection and suturing of the liver will be required in some patients.

Nephrectomies are performed in most cases of renal injury. It is rarely possible to do a partial resection although surgeons endeavor to preserve the injured kidney if at all possible. The large number of resections are due to the shattering effects of high-velocity bullets.

The majority of colon perforations and lacerations are treated according to their location by exteriorization, proximal colostomy, resection, and colostomy and right hemicolectomy. Primary suture of the colon is performed only for minor perforations or serosal injuries.

Splenectomy is performed for all splenic injuries. No conservative spleen-preserving procedures should be attempted.

Small-bowel perforations and lacerations are sutured when possible, but otherwise a bowel segment is resected whenever a large segment of bowel is involved. Gastric, duodenal, pancreatic, and retroperitoneal injuries are handled in the appropriate manner.

Postoperative Care

Abdominal casualties require intensive care after surgery. They will have many drainage tubes, catheters, and venous and arterial lines and require very close attention and observation. With this provided, they can tolerate further evacuation by air within hours of surgery, as long as they are well hydrated and properly oxygenated.

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4.10 Genitourinary Wounds

N. D. Reis

In a war or disaster setting, penetrating or blunt wounds of the kidneys, ureter, bladder and prostatic urethra are mostly associated with other visceral, vascular or bony injuries, making up part of a multi-trauma injury (particularly of the abdomen and chest).
The principles of management are not different from all other injuries: arrest of haemorrhage, debridement and drainage, followed by repair or reconstruction. Following resuscitation, preoperative diagnosis can be simple, repid and exact by using urographic and CT radiological techniques; these reveal at the same time anomalies, hidden injuries and the presence or absence of a functioning kidney. NB Intravenous pyelography is harmful and useless in shock.

On Site and Medical Aid Station

An urinary tract injury is suspected by the site of missile wounds, contusions, a mass and tenderness or by diagnosing a pelvic fracture. Frank blood issuing from the urethral meatus confirms an injury of the urethra or the bladder neck. Haematuria is evidence of injury to the kidney, ureter or bladder.
The general on-site management is as described in the chapter on primary treatment in the field. Specifically, *no attempt* should be made at catheterisation if frank blood is observed at the external meatus. If the bladder dilates with retention of urine, suprapubic drainage of the bladder is indicated: in the field this can be done by intermittent paracentesis with a long, wide-bore needle.

In Hospital

The casualty's injuries are reassessed and priorities are determined.

Kidney Wounds

If intra-abdominal bleeding from other injuries is diagnosed and urgent laparotomy is imperative, no special investigations are done. The kidneys are explored via the transperitoneal route (always making sure that a second kidney is present before undertaking any surgical treatment). If the general condition permits delay, urinalysis (to prove or exclude the presence of blood), intravenous pyelography and CT scanning are performed (confirming the presence and functioning status of the opposite kidney). Rupture or thrombosis of the major vessels of the renal pedicle can be confirmed by selective angiography.
The management is different for open and closed wounds.
Open kidney wounds must always be explored surgically in order to perform wound debridement. The approach to the kidney is transabdominal.
The vascular pedicle must be under vision prior to opening the perirenal fascia (the latter's closed space acts as a haemostatic tamponade) in order to control the catastrophic bleeding which may occur. Then kidney debridement and suturing are performed, and partial nephrectomy is indicated when one-third or more of the kidney is still intact. Total nephrectomy is performed for total or sub-total kidney destruction. The renal bed is always drained and a nephrostomy is used after kidney debridement and partial nephrectomy.
Closed kidney injury is managed by rest, observation and investigation by the aforementioned methods. Early indications for surgical intervention are uncontrolled bleeding in the urine or into the retroperitoneum (seen as a tendency to slip back into shock after resuscitation), extravasation of urine, renal vascular pedicle injury, severe damage to renal tissues as seen on CT scan or at laparotomy for an acute traumatic abdomen.
Late indications are infection and hypertension.

Ureteric Wounds

Injury to the ureters is rare but should be suspected when the tract of a penetrating unjury is in the vicinity of the ureter; whenever the casualty complains of pain in the flank, and there is gross or microscopic haematuria (although in total ureteric disruption there may not be any blood in the urine); in all cases of injuries to the liver, pancreas, colon, duodenum, and spleen and always in the presence of a retroperitoneal haematoma. Under these conditions the integrity of the ureters should be ascertained.
Within hours or days a tender swelling in the loin develops, often accompanied by fever, abdominal pain and an aqueous discharge (extravasated urine).

The ureter may not have been injured directly but be within the area of cavitation of a high energy missile: as a result a segment becomes necrotic and sloughs within a few days. A delayed but similar syndrome then develops (the initial pyelogram having been normal).
Whenever possible a high-dose intravenous pyelogram is performed prior to laparotomy (at least 1.5 ml/kg of 30% dye) This will usually show up spillage of the dye, confirming the diagnosis and pinpointing the site of the ureteric injury. (Intravenous pyelography depends on normal kidney function; therefore, it is contraindicated and indeed without value in a shocked patient!)

Surgical Treatment. For complete transection of a necrotic segment the ureter ends are debrided to healthy tissue and sutured with fine absorbable sutures, not penetrating the mucosa. Water-tight anastomosis is done obliquely in order to make the lumen as wide as possible to avoid late stenosis. There must be no tension at the suture line: proximally the kidney and distally the ureter are mobilised as required to ensure a tension-free anastomosis. Nephropexy in the distally shifted position is recommended. Proximal drainage by a nephrostomy or ureterostomy is absolutely essential to defunctionalise the anastomosis for 21 days; the anastomostic bed is drained.
Lower ureteric injury is treated by reimplantation of the ureter into the bladder: the ureter is brought through a submucosal tunnel, and its distal end is sutured to the trigonal area.

Bladder Wounds

The bladder is commonly wounded in pelvic trauma (open, penetrating and closed), especially when the bladder is full at the moment of injury. The tear in the bladder wall can be intraperitoneal and/or extraperitoneal. Associated wounds of the rectum or sigmoid colon are common in perforating missile wounds, especially when entry or exit is via the buttocks. Open or closed fractures or disruptions of the anterior pelvic ring are frequently present, and a bladder laceration may be caused by a bony fragment.
Diagnosis: Macroscopic (usually massive haematuria) is the rule. Intraperitoneal laceration may present as anuria, followed after some hours by abdominal tenderness and guarding. First an intravenous pyelogram is done to exclude a more proximal injury: it mostly fails to demonstrate bladder tears. The specific investigation is the retrograde cystogram done by instilling 300 ml of dye via the urethra by gravitation from a height of only 30 cm above the abdomen: anteroposterior and post-evacuation radiographs usually reveal the spillage from the laceration, although a false negative result can result from the plugging of the hole in the bladder by omentum or some other adjacent tissue such as a loop of small bowel, particularly when this examination is delayed for many hours or days after injury.

It is wise to explore the pelvis through the hypogastrium in all penetrating wounds of the lower abdomen, pelvis and buttocks with signs of peritoneal irritation (abdominal tenderness, guarding, tenderness per rectum, ileus).

Treatment. Suturing of the tears is done in two layers not including the mucosa with an absorbable suture. The bladder is drained by a wide-bore suprapubic catheter (preferably a Malecot catheter), and the space of Retzius is drained. The catheter should have a large bore so as to allow the evacuation of blood clots. Open wounds and missile tracts are excised and left open unless they are intraperitoneal. The suprapubic drain is usually required for 3 weeks to ensure healing of the bladder wall.

Urethral Wounds

In the male, the urethra is divided into the anterior (penile) urethra and the posterior (prostatic) urethra by the urogenital diaphragm between the descending rami of the pubic bones.
Injuries of both parts may be open and penetrating, closed or crushing. Late strictures cause chronic disability, hence the importance of the correct primary care of these injuries. Severe perineal wounds require a temporary colostomy for faecal diversion until healing is complete.

Posterior Urethra

Posterior urethral injury is usually a complication of pelvic fractures but may be caused by missiles.

Diagnosis: Suspicion is aroused by the presence of a fractured pelvis or a penetrating wound of the perineum or gluteal regions. The casualty is unable to void urine. Drops of frank blood are seen issuing, or can be massaged, from the penile meatus. Per rectum the prostate is palpated high up and mobile ("floating") with a tender gap instead of lying in its normal bed, if it has been totally avulsed from the urogenital diaphragm (this is the most common type of injury). A urethogram is performed by injecting 200 ml of dye up the urethra from the external meatus using an irrigating or simple syringe and placing a clamp on the distal penis. Radiographs are performed (anteroposterior and oblique at 45° from each side). The dye extravasated from the urethral wound is then observed extraperitoneally above the urogenital diaphragm if the injury is at the junction of the prostate and membranous urethra. If the tear involves the urogenital diaphragm and reaches into the root of the penis, dye is also seen in the perineum. No leakage of contrast indicates an intact urethra which is contused or stretched.

Treatment: The attempt to pass a catheter should not be made unless the urethrogram shows the absence of extravasation of dye. In the latter case, a small bore Foley catheter is passed into the bladder and no further therapy is required. The catheter is withdrawn after 2 days.
In all other cases operative treatment is the treatment of choice: The bladder and the retropubic space are explored. A Foley catheter is passed from the external meatus, and a second catheter is passed via the open bladder through the bladder neck, the ends of both of which appear in the retropubic space; once tied together, traction on the proximal catheter delivers the penile Foley catheter into the bladder. Then the balloon can be inflated to 30 ml or more and gentle traction applied to reduce the bladder neck and prostate to their anatomical position. The prostate is drown down in addition by sutures passing out through the skin of the perineum and tied over a button. A wide bore suprapubic bladder drain is left in place which also allows the passage of a strong non-absorbable suture through the tips of the Foley catheter, in case the latter requires exchanging at a later date; the space of Retzius is drained.
The uretheral catheter is withdrawn after 14 days and the suprapubic catheter after 21 days.

Anterior Urethra

Injury to the penile urethra is by penetrating missiles (commonly from below such as in antipersonnel mine explosions) or other direct violence.

Diagnosis: Blood at the external meatus is always present. An external wound or contusion is observed in the perineum or on the penis. Retrograde urethrography confirms the breach in the urethra.
Treatment: A contusion is dealt with by passing a size 16 catheter as a splint and to defunctionalise the urethra during healing. The catheter is withdrawn after 12 days.
A partial laceration is treated by a suprapubic cystostomy drain to defunctionalise the wounded urethra. The torn edges of the urethra are excised minimally and sutured to skin (marsupialisation). When the wound is clean and the swelling has gone down, the continuity of the marsupialised urethra is reconstructed.
For complete transection; after debridement the severed ends are sutured end-to-end by a fine absorbable suture. To avoid tension at the suture site the urethra is mobilised proximally and distally as much as required by dissecting out the corpus spongiosum together with the urethra from the corpora cavernosa, if necessary from the bulb to the corona. The repair is drained, and the skin left partially open. Urine voiding is diverted to a suprapubic cystostomy.

Wounds of the Testes, Scrotum and Penis

The blood supply to the male external genetalia is very rich, and therefore tissue vitality is great, similar to the skin of the face and scalp.

Treatment: Following minimal debridement wounds are left open for delayed primary suture. Tears in the penile Buck's fascia and tunica albuginea of the testis are sutured prior to skin closure.
After extensive loss of scrotal skin the testes are buried in thigh pouches. Denudation of the penis is treated by fashioning a temporary tube of scrotal skin. Orchidectomy is performed when the testis is necrotic owing to loss of its blood supply.

Further reading

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4.11 Wounds of the Hand

N. D. Reis

Introduction

The general principles of war surgery apply equally to all parts of the body. The application of these principles to the hand must allow, however, for some special factors. An orderly and thoughtful approach is particularly required owing to the potentially serious consequences of any hand injury, although apparently trivial.

The care and initial treatment will ultimately determine the final result. As hand injuries are potentially very disabling, and the danger of iatrogenic damage so real, all hand surgery should ideally be performed by an experienced hand surgeon. Unfortunately, this is not often practicable in war, and therefore a brief description of the principles of war surgery of the hand should be available in all field surgical stations and primary care hospitals, so that less experienced surgeons may have a reference readily available to refresh their minds.

Treatment

The surgeon operating on a hand wounded by a high velocity missile is faced with extensive soft tissue damage and comminuted fractured bone. He is often not able to perform a definitive primary excision because tissue death is more extensive than the primary appearance would suggest.

On Site and Medical Aid Station

First Aid and Resuscitation

A hand injury by itself seldom causes shock. When a hand injury is associated with shock and no external injuries are apparent, internal injuries must be sought and definitive treatment delayed until the other causes of shock have been excluded. Head, neck, chest, and abdominal injuries require attention, according to the proper priorities. Once the casualty has

been resuscitated and major injuries have been dealt with, the definitive primary treatment of the hand should be commenced forthwith.

Primary Care of the Hand

All rings, watches, and other constricting objects are removed. Sterile dry dressings are applied to the wounds. Sterile gauze pads are placed between adjacent fingers. A ball of cotton fluff is placed in the palm and copious cotton wrapped lightly around the hand up to the elbow. The hand is now splinted with the aid of a malleable wire mesh splint or with a board. The splint is affixed with a simple gauze bandage. On no account should any pressure be placed on the wounded limb. Bandaging is therefore very light. It is impractical in the field to splint the hand in an ideal (intrinsic plus) position. Any comfortable position is satisfactory. The wounded limb is supported in a sling. If evacuation is delayed, the hand should be elevated: the patient is placed in a recumbent position, with the elbow resting on the ground and bent at 90° with the hand vertically upwards, supported on some convenient object. Emergency tourniquets, which are frequently applied by the corpsman, are removed. They have been responsible for much damage in the past and are not necessary. A tourniquet is reapplied only if firm local pressure does not stop hemorrhage. Once the splint has been satisfactorily applied, and the injured part is at rest, no major analgesia is required except in crush injuries and severe burns. The next stage of treatment is delayed until the casualty reaches the hospital.

Field or Base Hospital

Reassessment — General and Local

In the hospital general reassessment of the casualty and reaffirmation of the priorities are performed (resuscitation is continued as required). Now a meticulous clinical examination to check the viability and state of the skin, bone, tendons, and nerves is performed and recorded. Intravenous administration of 5 million units penicillin or 1 g cephamycin is commenced. A dose of 0.5 ml of tetanus toxoid alone or with the addition of 250 units of antitetanus immunoglobulin should be administered in accordance with the casualty's history of immunization. Then X-rays are taken in order to provide detailed information of the fractures, dislocations, and embedded missile fragments. The patient is now prepared for surgery.

Anesthesia

All patients with injuries to the hand should be considered as incompletely prepared for anesthesia. In view of this, if the hand injury is not associated with injury to other parts of the body, regional anesthesia should be considered the anesthetic of choice. The sympathetic nerve block which occurs

with the peripheral nerve blocks improves the blood flow, which is of advantage in the promotion of healing, the reduction of edema, and the control of infection. Interscalene, brachial plexus, and axillary blocks are preferable to local infiltrations. Modern local anesthetics (bupivacaine hydrochloride) may provide long-lasting anesthesia. During wartime regional anesthesia is also preferable as the casualty can be monitored by junior staff. Supplementary agents such as narcotics and tranquillizers (diazepam) are effective in suppressing the stress of regional block and raise the threshold at which local anesthetics produce CNS complications. When an axillary block is used, tourniquet pain is abolished by infiltration of the subcutaneous tissues around the upper third of the arm.

If general anesthesia must be used (because of concomitant injuries), intubation by "crash" or "awake" techniques is indicated.

The *technical requirements* for the primary excision of the wound are: (1) good light, (2) set of fine instruments, (3) an assistant to afford adequate retraction for thorough exploration and excision of the wound, and (4) a tourniquet (preferably pneumatic).

Wound Lavage

After achieving anesthesia, the tourniquet is inflated to 250–300 mmHg to avoid further blood loss when clots are disturbed. Then comes the scrubbing and flushing out of the wound; the wounds are invariably contaminated, and the limb is usually filthy. The skin of the entire limb is scrubbed with a soapy antiseptic solution. The hair of the extremity is shaved only when it constitutes a mechanical nuisance at the wound edges.

The limb and the wound are now flushed with copious amounts of sterile balanced salt solution (Ringer's lactate). Hyperemia and other signs of tissue irritation have been demonstrated following irrigation with unbuffered saline. The purpose of irrigation is to remove loose debris, blood clots, and pieces of foreign materials. Flushing should be performed three times and under pressure. This may be achieved by squeezing out a jet from a plastic bag container of sterile solution, by pouring out from bottles at a height, or by the use of a special jet-flush instrument (we have not found the latter to be of any great advantage).

The tourniquet application is necessary for good vision, identification of vessels, nerves, and tendons, accurate excision, and avoidance of damage to intact tissues in the wound. It is released as required to test the vascularity of retained tissues. Maximal constant tourniquet time in primary wound excision is 1 h.

Excision of all nonviable tissue to prevent infection and avoid later toxicity from tissue necrosis is mandatory. Only living tissue can defend itself from bacterial invasion and multiplication.

Excision

Skin. The surgeon must always keep in mind that the hand possesses no spare tissues. The wound edges are not routinely excised, and only those parts of the skin edge which are definitely dead are excised. Factors which can serve as guides in making the decision are the texture and color of the skin, bleeding from the edges, and capillary return after release of the tourniquet. Very often the skin edge is ingrained with gunpowder or dirt, and it is not possible to decide at the primary excision whether the skin edge is alive or not. Elsewhere in the body there is spare skin and spare tissue so that primary excision can be wide and definitive, helping to achieve a rapid healing of the wound. Although the aim is the same in the hand, one dare not excise healthy tissues from this complex organ that has no spare tissue! The principle of wound excision in the hand is that of minimal excision. The hand wound (particularly when caused by high velocity missiles) is therefore excised repeatedly at 48-h intervals as necessary until a clean wound is achieved. This is normally possible in 4–6 days. The exceptions are crush wounds and some high velocity missile wounds in which it may take much longer to delineate the viable from the nonviable tissues.

Subcutaneous Tissues and Muscles. Next, the excision of deeper soft tissues which are judged to be avascular or impregnated with foreign material is performed. The principle of minimal excision applies equally to these structures. Excision of obviously destroyed subcutaneous fat and fascial tissues is followed by excision of nonviable muscle. Viable muscle is pink, bleeds when cut, and contracts when stimulated (the latter being the most reliable test). Totally shredded, crushed, or avulsed tendons are removed.

Nerves. Nerves are identified and, when torn, are approximated, fixed down, and marked by a stitch whenever possible.

Bone. Loose cortical bone fragments not essential for stability are removed. If bone fragments have the flimsiest soft tissue attachment, they are preserved. No attempt is made at this stage towards accurate reduction of fractures either by internal fixation or external splinting: shrapnel is removed but no effort is made to clear the wound of small embedded pieces. When necessary the skin wounds are extended, and separate incisions are made to facilitate access to deeper structures, remove foreign material, and promote wound drainage.

Destroyed fingers are amputated (whenever possible filleting out the finger and preserving as much skin as possible for future use to fill palmar or dorsal defects).

Additional Incisions. It is often prudent to further open wounds by extending incisions. Decompression of the carpal tunnel is commonly performed in severe hand injuries to avoid compression of contained structures. Open crush injuries or destructive high velocity missile injuries may require relaxing incisions in skin and fascial compartments.

Amputation. A finger or any part of the hand is destroyed when it has lost its blood supply and/or the sum of tissue losses is such as to pose no chance of useful future function and retention of the digit or hand will cause a functional embarrassment to residual tissues. The viable portion of amputated tissues is preserved for later use.
On termination of the excision, the tourniquet is released, and hemostasis is achieved; this should be meticulous. The potential danger inherent in the careless application of hemostats cannot be overemphasized. The wound is always left open. There is no place for primary wound closure in these injuries.

Dressing

The dressing should absorb exudates, protect the open wound from further injury and contamination, provide some very gentle support, and be bulky enough to immobilize the hand. The wound is best covered with nonadherent gauze, putting sterile dry gauze between the fingers, and a ball of wool in the palm of the hand, and lightly wrapping the whole from the fingers to the elbow in layers of cotton wool, bandage, cotton wool, and bandage (Jones bandage). The whole is splinted as required by using a volar or dorsal plaster slab or a Kramer wire splint from the hand to above the elbow, applied externally over the layers of bandaging and bringing the hand roughly into the intrinsic plus position. This position creates maximal stretching of the collateral ligaments of the finger joints and consequently minimizes later finger joint contracture. The injured hand is kept elevated above heart level to minimize swelling (less edema means less scarring).
After 48 h of constant hand elevation, the wounds are inspected (under anesthesia when necessary), and repeat excision is performed if necrotic tissues remain in the wound.

Summary

The primary management of the injured hand combines the following principles:

1. Detailed examination of the injury before and during surgery in order to establish that minimal vascular, neurological, and potential tissue functions exist or can be reestablished for useful function; otherwise, the destroyed part is amputated
2. Radical wound excision repeated every 48 h as necessary
3. Primary stabilization of fractures only in cases of major vascular repair
4. Antibiotic cover

Delayed Primary and Secondary Surgical Management

Wound Closure: General Principles

Hand wounds incurred in war are never primarily closed. The full extent of tissue damage cannot be assessed at the primary treatment phase. Wound secretion, edema fluids, retained foreign bodies, and retained nonvital tissues all require free drainage.

Wound closure is performed when the wound is clean of all nonvital tissues. This is commonly achieved between the 4th and 7th days. With these considerations constantly in mind, it is clearly desirable to close the wound as early as possible, particularly to cover exposed tendon, fascia, and bone. Exposed tendon and fascia tend to necrose after the 4th day. Skin loss may prevent simple closure; in this case a split-skin graft or skin flap cover will have to be considered. At the time of wound closure definitive treatment is also given to the fractures. Nerve and tendon repair procedures are best delayed until sound healing has been present for 12 weeks. Whenever wound closure creates a dead space, delicate drains should be left in for several days.

Principles of Skin Cover

The principles of skin cover in the hand are embodied in the desire to achieve wound healing as rapidly as possible, to have high quality sensation in the skin covering all important tactile and prehensile surfaces, and to cover successfully all exposed tendons and bones. Early coverage without infection gives the best chance for the return of function. Coverage of the wound can be achieved by split-thickness skin grafts, local flaps, pedicle flaps, and free flaps.

Defects covered by healthy granulations are satisfactorily covered by a split-skin graft. Such a graft is not good coverage for exposed tendon, bone, or joint; here flap tissue is mandatory. The use of the skin of a filleted, destroyed finger is an important technique; such skin is an ideal cover for a large volar defect and often retains good sensation. A similar technique is advantageous for covering a major dorsal defect which leaves the metacarpal bones exposed. Many techniques exist for full-thickness pedicle skin flap cover to the hand and fingers from the abdomen, groin, buttock, chest, opposite arm, etc. (Free flaps, made possible by microsurgical techniques, may be used in late reconstruction procedures and should not be considered in delayed primary wound coverage.)

Principles of Wound Stabilization and Fracture Treatment

Definitive fracture treatment must be undertaken at the latest before the fracture elements become adherent to each other. This normally means before the end of the 2nd week after injury, but ideally wound stabilization

must be contemporaneous with delayed primary wound closure. Wound stabilization precedes all other delayed primary procedures. Many fractures of the shafts of the phalanges and metacarpals with little or no displacement do not require any special treatment. Splinting the fractured finger to its intact neighbor is all that is required, allowing the finger to move when wound and fracture pain abate. The management of the severe hand wound is facilitated by stabilization of the fractures, thereby creating optimal conditions for the treatment of the complex soft tissue injury of the hand. Internal or external stabilization of open fractures acts as a deterrent to infection. The type of fixation used should be that which is most efficient and most readily applicable under the circumstances. The Kirschner wire technique is the most easily used method. Screws and small plates are satisfactory in stabilizing displaced metacarpal fractures; when there is bone loss a small plate or an external fixator on the metacarpal preserves length, and a fresh cancellous bone graft can be placed into the defect. Maintenance of length allows the remaining musculature to function under normal tension. In the fingers the fractured phalanges are best treated by multiple Kirschner wires or more rarely by small interfragmentary screws.

Fractures involving joint surfaces should always be reduced and internally fixed whenever this is at all technically feasible. There are a variety of techniques depending on the size and location of the fragments (delicate Kirschner wires, tiny interfragmentary screws, or pull-out wires). The use of plates in the phalanges often gives rise to stiffness and is not recommended. In my experience, various traction techniques through the phalanges using rubber bands or other devices to align, reduce, and immobilize finger fractures give rise to stiffness and are not recommended. There is little place for plaster of Paris in reducing and immobilizing fractures of metacarpals and phalanges. Plaster of Paris is very useful as an external support for the dressing, splinting the hand in the desired position and adding to the patient's comfort.

Whenever a joint has been irreparably destroyed, then in all interphalangeal joints and the thumb metacarpophalangeal joint a delayed primary arthrodesis should be performed, whereas at the metacarpophalangeal joints of the fingers delayed primary excision arthroplasty should be performed.

Open fractures of the carpal bones are treated by plaster of Paris or external fixation splinting. There is no indication for internal fixation. Arthrodesis as a late procedure may be required.

Management of Vascular Injuries

In life-threatening situations involving associated injuries, an ulnar or radial artery may be ligated. However, both arteries cannot be ligated without danger of ischemic necrosis in the hand. Since in about 5% of the population the radial or the ulnar artery is absent, an attempt should be made to

reestablish continuity, especially if both arteries are injured. It is best to suture them end-to-end after excision of the lacerated ends. Other methods available are vein patch, angioplasty, interposition of a short segment of saphenous vein, or primary bone shortening and end-to-end anastomosis. Severe injuries of the dorsum of the hand, particularly in the wrist region, may cause destruction of all the major dorsal vein elements of the cephalic and basilic systems, giving rise to persistent chronic edema of the hand. When most of the major dorsal veins draining the hand have been damaged, an attempt should be made to repair at least two major veins. If there are gaps, a vein graft is used. Microsurgical techniques for repairing small vessels are seldom necessary: loop magnification is all that is required.

Management of Nerve Injuries

The war wound is always contaminated, and the type of nerve injury is always a tearing or crushing one. Conditions are therefore not good for primary or delayed primary nerve repair. Secondary nerve repair at 8–12 weeks after skin healing is to be recommended. Nerve ends that have been tacked together at the primary or delayed primary procedure are easy to find and are excised and resutured. Digital nerves treated this way do extremely well, the ulnar and median nerves much less so. When gaps exist which cannot be overcome by mobilization of the nerve ends or by joint positioning, nerve grafting is indicated (the sural nerve, lateral cutaneous nerve of the thigh, saphenous nerve, or the lateral and medial cutaneous nerves of the forearm are used as donors). However, bone shortening allowing end-to-end suture is likely to give better results provided that the shortening itself does not do further functional damage.
The results of nerve grafts to the digital nerves are relatively good (good protective sensation can be expected with confidence). Grafting to the ulnar and median nerves gives poor results; it is here that bone shortening should be considered.

Management of the Tendon Injury

Delayed primary tendon repair can be considered (in rare cases) when the injury to the tendon is not of a crushing or lacerating type; even then tenolysis will be required later in most cases. Extensor tendons should be repaired as a delayed primary procedure whenever possible. Commonly the tendons have been crushed, avulsed, or shredded and are best excised to make place for a future secondary grafting procedure which may require placement of silastic rods in the first stage, to assist organization of a new tendon bed, whenever severe tissue damage with scarring has occurred. The tendon grafting is performed at the late secondary stage. Only fingers with an adequate circulation, protective skin coverage, proper alignment of bones, and good passive movement of the joints are candidates for secondary two-stage tendon reconstruction.

Amputation

As stressed previously, if an adequate blood supply exists in a destroyed finger (or any part of it), it is best to salvage all viable parts and use them in delayed primary or secondary reconstructive procedures. Delayed primary or secondary amputation is necessary if an adequate circulation is not realized or if uncontrollable infection exists. Late amputation is required if the end result is a stiff, useless, painful finger which interferes with the function of the hand.

Late Surgical Management

For 1 month after injury and until many months or years later, many reconstructive procedures are available. As a general principle, a hand must be well healed and covered and have regained supple healthy tissues before any complex reconstructive surgery can be undertaken. Such late secondary surgery includes pedicle skin skaps or free flaps to improve skin quality, nerve repair or grafting, bone grafting, osteotomy, tendon transfer, treatment of chronic infection of bone, arthrodesis, finger transplantation, etc., and follows the accepted principles of reconstruction and improvement of function (and sometimes cosmesis) of hand surgery. It is at this late stage that the modern microsurgical techniques of vascular pedicle skin, muscle, and bone grafting hold the most promise for the salvage of the severe war injury.

Microsurgery

In wartime there occur, on occasion, injuries which are not necessarily associated with missiles. Such sharp or clean-cut types of complete or partial finger or hand amputations and nerve injuries may require microsurgical techniques. These are seldom practicable in a war zone. The amputated part is often missing, irretrievable, or difficult to preserve or transport if still present, so microsurgical replantation is not feasible. High velocity gunshot causes serious hand wounds: severe damage, an extensive area of compromised blood supply, grossly contaminated fractures, and avulsed, crushed nerves which preclude this type of treatment. Associated head, chest, and abdominal injuries take precedence. Under such conditions lengthy microsurgical operative procedures are contraindicated even if the equipment and an expert team are available. Microsurgical procedures are therefore still mainly confined in this context to late reconstructive surgery.

Physiotherapy and Early Motion

Elevation, early motion, and tensing the muscles prevent swelling. One of the main causes of stiffness is chronic swelling due to lack of active muscle function and immobilization of joints leading to joint capsule and ligament contractures. Motion is also important psychologically to persuade the wounded that recovery of function is possible. Primary or delayed primary tendon and nerve suture and external splinting of fractures militate against early motion; this is an additional reason to avoid tendon and nerve repair in the early critical phase when motion (even if only passive) is so important. Similarly, the internal stabilization of fractures is of great advantage in this respect. The physiotherapist and occupational therapist have a vital role in encouraging and supervising the earliest possible active and passive movements and the recovery of hand function.

Further Reading

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