

Multiple Injuries

Casualties with multiple injuries are more difficult to treat because of synergistic effects of the pathophysiological disturbances on more than one organ system, the increased frequency and severity of shock, and the competing priorities for immediate care of the various injuries. Wounds involving more than one organ are characteristically more frequently lethal.

ETIOLOGICAL CONSIDERATIONS

The patients in this group most often have sustained multiple missile wounds involving a number of organs or anatomical areas. In addition to missile wounds, these casualties frequently present with associated traumata of other kinds, as follows:

1. Thermal traumata (burns or cold injuries)
2. Physical traumata, including blast injuries, underwater compression as seen in submarine or ship crews, injuries following decompression (aviation or diving crews), crush injury, electrical injury, and rapid deceleration injuries as commonly seen in aircraft and vehicular accidents.
3. Chemical traumata such as phosphorus burns; exposure to organic fuels or propellants; injuries resulting from other chemical agents causing either cutaneous, respiratory or other systemic irritation, or depression of the nervous system.
4. Ionizing radiation injuries with either local or systemic effects.

These special injuries are seen with increasing frequency either in combination with the usual battle wounds or in combination with each other. Personnel working in various military specialties are subject to combinations of injuries which may be unique to their specialty or environment. Physiological disturbances secondary to multiple factors, such as climatic or environmental temperature extremes, dietary inadequacies, superimposed acute or chronic infectious diseases, and systemic poisoning, must also be considered and dealt with.

MANAGEMENT

Diagnosis, Triage, and Evacuation at the Division Level

A thorough examination is carried out at the battalion or division medical facility that initially receives the casualty. Additional problems to be countered are those imposed by the tactical situation and often the generally unfavorable condition of the combatant (dehydrated, grimy clothed, burdened with equipment, and frequently confused after blood loss or sedation given in the field). Under these circumstances, an accurate medical history is difficult to obtain. Unless great care is taken, it is easy to overlook injuries.

The best way to avoid a serious oversight is to remove the patient's clothing completely and consider systematically all the injuries that may have resulted either from a particular missile type and its trajectory, or from other trauma. In addition to obvious lower extremity trauma, a thoracoabdominal blast injury should be considered following a mine explosion. Carbon monoxide poisoning and burns of the respiratory tract must be considered in casualties with burns about the face and those involved in a fire in a closed environment, such as tank or armored personnel carrier. Shock, frequently present, is often severe. It is usually proportionate to the number and magnitude of injuries sustained.

After examination and identification of injuries, the degree of urgency and priorities of treatment must be established. Immediately, life-threatening problems must be corrected; general first aid measures, establishment of the airway, control of hemorrhage, and initiation of resuscitation are carried out according to usual routines. The patient with multiple injuries presents special problems for consideration during evacuation. If these casualties are to survive, essential care must continue en route to the definitive treatment center. Skilled medical attendants are needed to maintain the airway, support the respiration, control hemorrhage, and insure the adequacy of blood or fluid volume replacement. Rapid helicopter evacuation alone is not a substitute for adherence to the above principles, nor does it permit one to ignore the need for adequate fracture immobilization. Concise, accurate records of the injury, of the types of wounds, and of the treatments administered are mandatory to facilitate subsequent medical care.

Preparation for Initial Surgery

Casualties who are seen by medical personnel for the first time at a hospital are evaluated carefully in the manner already described. The accuracy of findings and the response to previous treatment are reassessed with each admission along the evacuation chain. Priorities for care of various wounds in the same patient must be established. While many patients with multiple, extensive wounds can be treated successfully, the potential lethality of certain wounds, such as a massive central nervous system injury or a 90% third-degree burn, must be realistically assessed as the lowest priority for treatment.

Although an oral airway may be adequate in some patients, an endotracheal tube is mandatory in others to assure an adequate airway. Where indicated, chest tubes are inserted and connected to closed drainage. Major bleeding must be controlled and blood volume replenished. Intravenous routes are established with due regard for the site of major injury; for example, a major abdominal injury is best managed with large-gauge cannulae placed in the upper extremities or neck. Other measures important in a patient with multiple injuries are evacuation of the stomach by nasogastric suction and the insertion of an indwelling catheter to measure the urinary output and determine the presence or absence of hematuria. Unstable fractures must be splinted either by conventional means or with the radiolucent inflatable splint before further transport. These splints are ill-suited for fractures of the femur or humerus. While inflatable splints may reduce blood loss, they can present a threat to the circulation if inflated other than by mouth, due to the expansion of air in the splint during evacuation at flight altitude.

Appropriate roentgenograms must be obtained and should **include** special studies when indicated, such as intravenous pyelograms or cystograms in abdominal and pelvic wounds. **Intra**-peritoneal injuries, produced by missiles entering through the thigh, buttock, or back, are easily overlooked. Abdominal radiographic studies in such wounds is particularly indicated.

The lack of response to vigorous resuscitation may necessitate immediate surgical intervention to control major internal blood loss. However, other causes, which produce or simulate shock must be considered (for example, drug overdose or other poisoning, cardiac tamponade, cerebral malaria, and other infectious diseases).

The complication of cardiac arrest usually is treated by closed cardiac compression; however, open cardiac compression may be required.

Operative Management

The order of priority of wound care is often difficult to establish. In general, those injuries most life threatening are treated initially; thereafter, good judgment must prevail. For example, a patient with both thoracic and abdominal injuries should have definitive operative correction of a lacerated bronchus before a repair of multiple intestinal injuries. Definitive care of intracranial, facial, ocular, and hand injuries frequently must be delayed until other more immediately threatening injuries have been dealt with. Usually, initial operative management of major chest, abdominal, and extremity wounds is performed at a forward hospital. After stabilization, the patient can be transferred to a larger supporting hospital for the appropriate care of remaining injuries. This staged approach, even though it requires a second anesthetic, is much safer than the evacuation of an unstable casualty.

Surgical staffing should provide **sufficient** personnel to insure appropriate care and to keep operating room and anesthesia time to a minimum. When the situation permits, this may best be accomplished by having separate teams operating on different regional injuries simultaneously. If the wounds are unrelated, it may be necessary to operate on various anatomical areas in successive procedures. Where possible, for example, a buttock wound should be debrided and bleeding controlled before exploring the abdomen. Patients in shock with continued blood loss are extremely unstable after lengthy operative procedures, and cardiac arrest is likely to occur if the procedures are performed in reverse order.

The simplest lifesaving surgical procedure consistent with established principles of combat surgery is all that should be attempted at this time. Unnecessary or meddling procedures, such as resection of an undiseased appendix or a Meckel's **diverticulum** during laparotomy and bowel repair, impose an unacceptable added risk to the patient.

Special Considerations

Despite optimal medical treatment by personnel at all echelons of care, the patients in the multiple injury category are at an extremely high risk. Respiratory support with mechanical ventilators is frequently the only way to counteract the pulmonary insufficiency and fatigue factor common to this group. This is particularly true in casualties with major blast injuries, hepatic wounds with concomitant pulmonary contusion, **thoracoabdominal** wounds or severe sepsis, and in patients who have required cardiorespiratory resuscitation.

A policy of restraint in intravenous crystalloid fluid administration during resuscitation and operations should be considered in cases where the development of posttraumatic pulmonary insufficiency is likely. This policy does not preclude the administration of large volumes of blood or colloid where indicated.

Experience has repetitively demonstrated that constant vigilance and an inquiring attitude will help to define confusing problems and provide practical solutions to what at first may have seemed an impossible problem.

TRAINING

Resuscitation and operations are performed by teams of medical officers, corpsmen, nurses, and other support personnel rather than by individuals. Each medical officer or surgeon has the responsibility of continually training his supporting personnel. The Advanced Trauma Life Support (ATLS) course of the American College of Surgeons provides a good starting point for training applicable to the evaluation and resuscitation of the casualty with multiple injuries. However, medical officers should not forget that ATLS was developed by civilian physicians for use by civilians in dealing with typically civilian trauma. It has not been tested in war and may not be entirely appropriate for combat casualty care. Certainly, a more problem-oriented approach may be necessary, especially during mass casualty situations in exposed field medical units.

ADVANCED TRAUMA LIFE SUPPORT

Primary Survey. During the primary survey, life-threatening conditions are identified and their simultaneous management is begun.

- (1) **A** — Airway maintenance with cervical spine (C-spine) control.
- (2) **B** — Breathing.
- (3) **C** — Circulation with hemorrhage control.
- (4) **D** — Disability; **neurologic** status.
- (5) **E** — Expose; completely undress the patient.

Resuscitation Phase During this phase, the oxygenation and ventilation are reassessed. Shock management is initiated and hemorrhage control is reevaluated. A urinary catheter and nasogastric tube may also be inserted during this phase if their use is not contraindicated.

Secondary Survey. The secondary survey does not begin until the primary survey (ABCDE) has been completed and the resuscitation phase (management of other life-threatening conditions) has begun. The secondary survey is a head-to-toe evaluation of the casualty. Each section of the body is examined in an organized fashion, utilizing look, listen, and feel techniques. Chest and C-spine X-rays may be obtained during this section, but only after the patient is stabilized.

Definitive Care Phase. In this phase, the patient's **less-life-threatening** injuries are managed (setting of fractures, **stabilization**, wound debridement, and transfer).

TREATMENT AND MANAGEMENT

Primary Survey:

- (1) **Airway and C-spine** Upper airway problems are not uncommon in the combat casualty arriving for definitive care. Initial attempts to establish a patent airway include the chin-lift, the jaw thrust maneuver, or simply the removal of foreign debris. Patients in whom blunt trauma has been a mechanism of injury, such as from a helicopter crash or blast displacement injury, should have a consideration for protection of the C-spine Excessive movement

of the C-spine can result in permanent injury. In any patient in whom a C-spine injury is suspected, a lateral C-spine X-ray should be taken. All seven vertebrae must be visually confirmed as **normal**. Pain, tenderness, swelling, and neurological exam are all unreliable indicators of C-spine injury.

(2) Breathing. Ventilatory exchange should be assessed by looking at the chest and listening with a stethoscope. Airway patency does not assure adequate ventilation. The three traumatic conditions that most often compromise ventilation are tension pneumothorax, open pneumothorax, and a large flail chest with pulmonary contusion. Ventilation may be accomplished with an oral or nasal airway and a bag valve device. Chemical injuries may create life-threatening breathing problems. Blast injuries can result in acute pulmonary dysfunction.

(3) Circulation. Adequate circulatory volume can be assessed by examining pulse, skin color, capillary refill, and blood pressure. If the radial pulse is palpable, the systolic pressure will be above **80mm** of mercury. If the femoral or carotid pulse is palpable, the systolic pressure will be above **70mm** of mercury. A quick and easy method of assessing the peripheral perfusion is the capillary blanch test, done on the hypothenar eminence, the thumb, or the toenail bed. In a normal **volemic** patient, the color returns to normal within two seconds. Extremity hemorrhage should be controlled by direct pressure. Tourniquets may be of value, but the use of clamps directly into the wound should not be employed. Pneumatic splints may be helpful in controlling bleeding as well. Occult bleeding into the major body cavities will result in shock if left unchecked, and bleeding around crush injuries and fractures will also contribute to hypovolemia. Blast injury can result in arrhythmias.

(4) Disability. A brief neurologic examination should be conducted to establish the level of consciousness and the status of the pupils. A more detailed neurologic examination will follow later in the secondary survey. Simply identifying the level of consciousness and the status of the pupils in the primary survey is **sufficient**.

(5) Expose. The patient should be completely undressed to facilitate thorough examination and assessment.

b. Resuscitation:

(1) Maintenance of airway, establishment of ventilatory

mechanism, and resuscitation of circulating volume should be initiated when the problem is identified rather than after completion of the entire primary survey.

(2) Supplemental oxygen therapy should be instituted. Nasal cannulae provide the simplest method of providing this; however, rebreathing masks provide a higher level of inspired oxygen.

(3) Two large-bore **IVs** should be started and a Ringer's lactate infusion begun. Percutaneous IV sites have the lowest incidents of complications. **Cutdowns** may be employed in the antecubital **fossa** or in the lower extremities. Central line placement in the internal jugular or subclavian veins may also be employed and are of value for central venous pressure monitoring. Resuscitation may also include type-specific whole blood or low-titer type 0 blood. Hypovolemic shock is not treated by vasopressors, steroids, or sodium bicarbonate. Adequate resuscitation is assessed by following pulse blood pressure and urinary output. Careful electrocardiogram (ECG) monitoring may be indicated by clinical circumstances, such as blunt chest trauma.

(4) Placement of urinary and nasogastric catheters should now be considered. Urinary catheters are contraindicated in the presence of suspected urethral transection, and nasogastric tubes are contraindicated in the presence of cribriform plate fractures.

c. Secondary Survey:

(1) Head. The secondary survey begins with an evaluation of the head and proceeds downward. The scalp and bony structures of the head should be checked for evidence of blunt penetrating trauma. The eyes should be examined for chemical irritation, foreign bodies, and pupillary integrity.

(2) Maxillofacial trauma. Maxillofacial trauma is important because of its relationship to the airway, the central nervous system and the cervical spine. Maxillofacial trauma by itself can usually be managed at some later time. Patients with **midface** fractures may have fractures of the cribriform, plate and in these patients gastric intubation should be performed by the oral route.

(3) C-spine/neck. Patients with maxillofacial trauma produced by blunt injury should be presumed to have a C-spine fracture until proven otherwise. The absence of a **neurologic** deficit, pain, or deformity does not rule out a C-spine injury. A lateral C-spine **X**-ray is the only way to completely rule out a C-spine injury. Following blunt trauma to the head and neck, the C-spine should be mobilized utilizing sandbags and tape until such time as the injury

has been ruled out. Penetrating wounds of the neck should not be explored in the emergency area with probes or fingers, but should be evaluated in the operating room. Arteriography may be indicated prior to exploration.

(4) Chest. Visual examination of the chest, both front and back, will identify most penetrating trauma. Sucking chest wounds should be covered with Vaseline gauze or treated with chest tube insertion. Evaluation of ventilator-y function is best performed utilizing the stethoscope. A check for the status of the neck veins may be helpful in making an assessment of cardiac tamponade.

(5) Abdomen. All penetrating abdominal traumata should be explored in the operating room. Blunt trauma to the abdomen requires special assessment. Close observation and frequent reevaluation are important in the management of blunt abdominal trauma. Patients with **neurologic** injury resulting in an impaired sensorium may present special difficulties in evaluating blunt abdominal trauma. Peritoneal lavage may be of assistance in these instances.

(6) Rectum. A complete rectal exam is important in all trauma patients: look at the perineum, examine sphincter tone, check the integrity of the rectal wall, check the location and mobility of the prostate and look at the examining finger for the presence of gross blood. This is especially important in blunt trauma.

(7) Fractures. Extremities should be examined for contusions or deformity. Palpation and examining for tenderness, crepitation, or abnormal movements along with shafts will help identify fractures. A special check for fractures of the pelvis in blunt trauma is particularly important, because the identification of a fractured pelvis usually indicates the need for significant blood volume replacement. Pulses should be examined in each of the extremities in which there is blunt or penetrating trauma.

(8) **Neurologic.** An **indepth** neurological examination should be conducted in which the physician looks for reflexes, evaluates motor and sensory function, and reevaluates the level of consciousness. The Glasgow Coma Scale is important in assessing the patient with head trauma.

d. Definitive Care. The definitive care of each injury will be discussed in subsequent chapters.

Most combat casualties are young, healthy individuals; however, senior personnel and civilian combatants may provide the

opportunity to care for individuals with preexisting medical problems and possible medication complications. An “AMPLE” history is important.

A -Allergies

M -Medication

P -Past illnesses

L -Last meal

E -Events preceding the injury

Reevaluation of the patient is an essential part of all patient assessment, whether for blunt or penetrating trauma. Many injuries may not be evident when the patient first presents. As the patient remains in the health care system and is transported from one location to another, injuries and altered physiology may be evident. Continuous monitoring of vital signs is essential.

Meticulous recordkeeping is extremely important since more than one provider will be participating in the care of the patient along the evacuation chain. Precise records are essential in order to keep up with the patient’s clinical status. As the patient is transported along the evacuation chain, all records of laboratory tests, treatments, and X-ray evaluations should accompany him.

PART II

Response of the Body to Wounding

Shock and Resuscitation

One encounters multiple classifications of the shock syndromes. The common denominator in all forms of shock is inadequate capillary perfusion. This chapter concerns itself with the diagnosis and treatment of hemorrhagic shock, that clinical state in which the capillary perfusion is inadequate to satisfy tissue requirements as a result of the loss of blood. For the sake of completeness, we will briefly mention the other forms of shock:

(1) **Septic Shock** — This syndrome results from the absorption of bacterial toxins or toxic products from infected muscle or other tissues in which debridement has not been performed or was performed inadequately. Massive infection of serous cavities especially predisposes to this potentially catastrophic complication.

(2) **Neurogenic Shock** — Neurogenic shock results from autonomic nervous system stimulation, causing either widespread vasodilatation or the inhibition of vasoconstriction. This can result in vascular collapse. Neurogenic shock may occur after head injury, may be brought on by pain, or may occur on an emotional basis. The pulse is slow, usually around **60/minute**. The syndrome is most often encountered in the operating room in association with the use of certain pharmacologic agents.

(3) **Oligemic Shock** — Oligemic shock, like hemorrhagic shock, results from loss of circulation volume. The volume loss in this situation usually results from severe, unreplaced, nonhemic losses, such as those arising from severe vomiting or diarrhea, ileus, intestinal obstruction, or enteric **fistulas**. Loss of plasma by seepage, as occurs with burns, intestinal infarction, and crush injury, also results in external or extravascular "third spaces" losses.

The combat surgeon should bear in mind that the most common cause of death on the battlefield and during evacuation to the hospital is exsanguination. Hemorrhagic shock is far and away the most commonly encountered shock syndrome. Experience has also shown that the majority of casualties, presenting in advanced shock will require surgical intervention to achieve hemostasis

before stabilization and hemodynamic improvement can be achieved and maintained.

ORGANIZATION OF A TRIAGE AND RESUSCITATION FACILITY

Prompt, preoperative resuscitation saves lives. Careful preliminary preparations must be made and resuscitative measures instituted with the least possible delay once a casualty has been received in the resuscitation facility. The triage and resuscitation of casualties in shock require considerable clinical experience and acumen. An experienced medical officer, assisted by an experienced and well-trained staff, should be in charge of the triage and resuscitation facility. There must be coordination between the triage officer, the resuscitation personnel, and the operating room personnel. The triage surgeon not only sets individual resuscitation and operative priorities but also must be intimately involved in coordinating and facilitating casualty flow.

PHYSICAL SETTING

1. The facility should be a large, well-lighted expanse of uninterrupted space, allowing free movement of people and an unobstructed view of the entire room. Partitions or unnecessary structures which interfere with communication have no place. To effectively direct activities within the receiving area, the triage officer must be able to see and be seen throughout the area.

2. Such a facility should be capable of handling a large number of casualties. Its location is important in relation to the transportation which delivers the casualties, to the other supporting services, and to the overall internal patient flow. It should be immediately adjacent to the ambulance unloading area or the helicopter pad so that transfer into and out of secondary vehicles is not required. The area should be situated close to the operating room. Portable X-ray apparatus should be close at hand. These arrangements reduce the necessity of moving the patient, which is always deleterious in shock.

3. Supplies and equipment should be immediately visible and accessible without obstructing floorspace. A large number of open shelves lining the walls circumferentially about the triage area will be valuable for this purpose.

4. The blood bank and X-ray facility should adjoin the triage area. Laboratory tests other than cross-matching of blood and determination of arterial blood gases are not needed for initial resuscitation and can be set up in a laboratory closer to the wards and intensive care unit.

5. The facility should be arranged so that casualties can be moved easily and rapidly from the triage area or X-ray facility to the preoperative area and the operating rooms. After initial evaluation and treatment, the wounded should be separated according to priorities. Those most critically wounded are moved to an appropriate surgical stabilization area or, in dire circumstances, may require immediate movement into the operating room. Those that require general anesthesia and can be stabilized are managed in a preoperative area while awaiting their turn in the operating room. Those needing only debridement of minor wounds under local anesthesia may be cared for in a separate area.

EQUIPMENT AND SUPPLIES

1. The frames upon which stretchers will be placed should always be in position, carefully arranged to allow enough space between patients for easy movement. A minimum of other furnishing is necessary. Aside from a desk or countertop work space for record keeping, there should be no chairs or furniture about the working area. Stethoscopes, sphygmomanometers, intravenous administration sets, IV fluids, and devices for suspension of IV bottles or bags should be at every stretcher position.

2. Sterile prepacked sets for emergency procedures, such as cut-downs, tracheostomies, insertions of chest tubes, and control of bleeding, should be conveniently located. These sets must include all of the instruments, sutures, and fittings needed for the purpose and should be plainly marked.

3. Suction equipment must be immediately available for airway aspiration.

4. Laryngoscopes and endotracheal tubes with inflatable cuffs should be conveniently located in the resuscitation area. Oropharyngeal airways prevent the tongue from obstructing the oropharynx in the unconscious patient. Insertion of the endotracheal tube is a rapid means of assuring upper airway integrity and facilitates the later performance of a tracheostomy under more controlled circumstances. A ventilating bag with mask

and endotracheal tube fittings-for manual ventilation should be available at numerous locations.

5. Large bandage scissors should be in each corpsman's pocket and at numerous other places to allow quick removal of the casualty's clothing.

6. Intravenous fluids, in large quantities, should be immediately available in the triage area. One bottle of Ringer's lactate with tubing inserted should be hung in place over each set of litter frames. A blood filtration set should be at hand for those who require subsequent administration of blood.

7. Percutaneous venous catheters are preferable to needles in administering intravenous fluids. The intravenous pathway should be at least 18 gauge.

8. Large-bore catheters for chest drainage and sterile tubing for insertion of underwater drainage or suction should be available. Heimlich one-way valves attached to chest tubing are acceptable only for temporary purposes.

9. Quantities of prepackaged sterile dressings in various sizes should be in ample supply at every stretcher.

10. Prepackaged sterile syringes in 5, 10, and 20 ml sizes should be within reach. In addition, preheparinized 5 ml syringes should be available for blood gas determination samples.

11. Sterile prepackaged sets of urinary catheters will be needed and should be available. Only large balloon Foley catheters should be used.

PATHOPHYSIOLOGY

Early post-hemorrhage circulatory changes are compensatory, all serving to preserve perfusion of the vital organs. **Vasoconstriction**, shunting, and fluid shifts all contribute to the attempt to maintain perfusion of vital vascular beds. A more detailed account of these homeostatic mechanisms operative in the shock state is provided in Chapter X, dealing with the physiologic response to trauma. For our purposes here, suffice it to say that the response to hemorrhage is graded and complex. The circulating blood volume represents approximately 7% of body weight, or about 5 liters in the 70 kg man. In the young healthy individual, a **significant** blood loss can be tolerated without major changes of the blood pressure early on. The foregoing may not apply to the older casualty, to the depleted casualty, or even the younger casualty

as the interval between wounding and initiation of therapy lengthens. The following is offered as a guide in assessing the volume of acute blood loss:

(1) Up to 15% blood volume loss (Class I hemorrhage). Mild tachycardia is the only clinical sign in an uncomplicated situation. This represents a blood loss of 500 cc or less in the 70 kg person. The blood pressure, respiratory rate, urine output, and mental status are within normal limits. The capillary blanch test is normal, refilling occurring within two seconds. These casualties should be resuscitated with crystalloid solutions.

(2) **15-30%** blood volume loss (Class II hemorrhage). This degree of loss in the 70 kg soldier amounts to 750-1500 cc of blood. Clinical findings include a pulse greater than **100/minute**, a slight decrease in the blood pressure, an altered capillary blanch test response, and subtle central nervous system changes including inordinate anxiety or fright. The urine output is only minimally depressed. This class of patients can also be resuscitated with crystalloid alone.

(3) **30-40%** blood volume loss (Class III hemorrhage). This represents a **1,500-2,000** cc blood loss in the standard male. Tachycardia (usually at greater than **120**), tachypnea, diastolic and systolic hypotension, and scanty urine output are apparent. These casualties will require blood in addition to crystalloid for resuscitation.

(4) Over 40% blood volume loss (Class IV hemorrhage). This degree of hemorrhage is clearly life threatening. It amounts to a hemorrhage in excess of 2,000 cc. All of the classic signs of shock are present. The skin is cold, clammy, and pale, and the mental faculties are clearly depressed. These casualties not only require large-volume blood replacement in addition to crystalloid, but in addition to volume replacement often times require immediate surgical intervention if resuscitation is to be successful. That is to say, they require operation for resuscitation rather than resuscitation for operation.

PREDISPOSING AND AGGRAVATING FACTORS

Circulatory collapse is hastened or aggravated by a number of factors. Preexisting fluid or electrolyte imbalances resulting from excessive **sweating**, diarrhea, or vomiting all contribute. The same effect occurs when handling of the casualty during evacuation

is rough and traumatic, when injured extremities are not splinted, or when there is sudden shifting of the casualty's position.

Relative overdoses of morphine and certain operating room drugs can make matters worse, as can operation with inadequate anesthesia, prolonged operation, excessive mesenteric traction, or massive contamination of the peritoneum.

INITIAL HOSPITAL EVALUATION

The approach to the casualty in the shock state should be directed to the adequacy of the airway, control of bleeding, and the restoration of the blood volume. Simultaneously, with the institution of initial fluid administration, the surgeon ascertains the mechanism of injury, the wounding agent, the time elapsed since wounding, and, if possible, the position of the casualty when wounded, the estimated initial and enroute blood loss, the drugs administered prior to hospital arrival and the presence or absence of known allergies. Since most combat casualties are young and were previously healthy, history of past or preexisting diseases or chronic medication requirements is usually of little value. This may not be the case in older casualties, especially civilian casual ties.

On arrival, a rapid but thorough physical examination is performed to determine vital signs and to identify the number, location, and extent of wounds. The casualty should be completely undressed to allow head-to-toe front and back examination. Blood pressure, respiratory rate, mental status, skin color, capillary refill, and temperature are recorded in the abbreviated clinical record. The capillary refill test is performed by depressing the fingernail or tip of the finger. A normal response is refill of the capillary bed as manifested by the return of color within two seconds. Hidden blood loss into the chest, abdomen, fracture sites (pelvis and thigh) or crush injury sites may be present. These fractures can account for 15-2 liters of acute blood volume loss. In the presence of shock, with a chest wound or probable chest wound, a closed-tube thoracostomy should be performed without delay.

As the large-bore intravenous infusion lines are placed, blood is aspirated for type and crossmatch. If additional laboratory tests are indicated, blood is drawn at this time. Usually this amounts to a hematocrit determination for future comparison as therapy progresses. It should be emphasized that the hematocrit has no

place in the estimation of the **volume** of acute blood loss.

HEMOSTASIS

Early in the evaluation and resuscitation phase of the unstable casualty, extremity dressings should not be removed, as the ensuing bleeding will only serve to further deplete circulating volume and will impede therapy by diverting attention away from the business at hand. Direct pressure will adequately control most external hemorrhage. Blind clamping in deep wounds is usually time consuming and frustrating, and in general should be avoided. Tourniquets should be used only after other methods of control have failed. If indicated, the properly applied tourniquet can save the life, but endangers the limb. A common mistake with tourniquets is inadequate compression, which occludes the veins but fails to occlude the artery, resulting in an increased rate of blood loss. The tourniquet should be placed as distally as possible on the extremity, just proximal to the wound. Once in place and adequately controlling hemorrhage, it should not be released until the casualty reaches the operating room. When applied in the field or **enroute**, the time of tourniquet application should be recorded on the field medical card.

Expedient evacuation of the shock casualty to a definitive facility should not be delayed by application of military antishock trouser (MAST). Some controversy still surrounds their use. MAST trousers can produce ischemia and compartment syndromes if improperly used. A recent combat casualty with abdominal wounds and no lower extremity wounds was treated with MAST trousers in addition to fluid resuscitation and operation. Instability was such that the trousers were left inflated for 18 hours. This casualty subsequently required bilateral above knee amputations. The trousers should never be inflated beyond 100 mm Hg. If there has not been a hemodynamic response within 30 minutes, the inflation pressure should be reduced as the resuscitation continues.

Operation in the resuscitation area of the hospital is rarely necessary; however, the casualty that arrives with penetrating or perforating chest wounds and very recent loss of vital signs is an exception. Salvage may be attempted by immediately opening the unprepped chest of the unanesthetized casualty in an attempt to temporarily control hemorrhage, as fluids are pumped in and the casualty is ventilated via endotracheal tube. An occasional young

man will be salvaged in this manner. If some degree of stability is achieved, the casualty is moved to the operating room for definitive repair and closure.

Autotransfusion devices may be available in future wars. There are basically two types of such devices. Both add small amounts of anticoagulant to the collected blood. One simply collects the blood, filters it and reinfuses it. The other type collects the shed blood, washes and centrifugally separates out the red blood cells, and then reinfuses them. These devices may be practical in the resuscitation area for casualties with substantial and ongoing hemothorax. In the operating room, these devices may be applicable in extremity wounds or in cases of uncontaminated hemoperitoneum.

VENOUS ACCESS

Multiple sites of venous access, utilizing large-bore, relatively central catheters, provide both rapid infusion and venous pressure monitoring capability. The most commonly employed percutaneous approaches are the internal jugular, the subclavian, and the median basilic veins. If a **cutdown** is required to achieve **large-bore** venous access, the median basilic, the greater saphenous in the groin, or the distal saphenous vein at the median malleolus are all easily isolated. **Cutdowns** performed under emergency conditions are prone to infection and should be discontinued about 24 hours after the emergency. The magnitude and location of the casualty's wounds will influence the site selected for infusion. Except for the most emergent situations, such as cardiac arrest, one should avoid using the common femoral vein for direct access, as the incidence of injury of adjacent structures and deep vein thrombosis can significantly complicate the postoperative course.

REPLACEMENT THERAPY

Lactated Ringer's solution is the resuscitation fluid of choice. It has advantages over solutions such as Dextrone (5% dextrose in lactated Ringer's solution) since the glucose is poorly metabolized in the presence of the catecholamine response. The incremental elevation in blood glucose levels results in an osmotic diuresis, misleading urine output levels, and dehydration. Colloid solutions are expensive and do not equilibrate with the interstitial space as rapidly as Ringer's lactate. Even though smaller volumes

of colloid are required for initial resuscitation, the consensus is that colloid-containing fluids have no significant advantage over Ringer's lactate for resuscitation of the shock casualty.

The shock casualty should be given 1,000-2,000 cc of lactated Ringer's solution, infused as rapidly as possible. Another rule of thumb is an initial fluid challenge of 10-25 ml/kg given over a ten minute period. Some will respond promptly and remain stable with only this therapy. If the hemorrhage has been severe or is ongoing, the response will usually be only transient, but nevertheless may allow time for typing and crossmatching of blood. Lactated Ringer's solution, in addition to providing a rapid increase in circulating volume, will begin the correction of the reduced extracellular volume space resulting from compensatory fluid shifts induced by the shock state. Crystalloid solution rapidly equilibrates between the intravascular and interstitial compartments. For this reason, adequate restoration of hemostatic stability may require large volumes of Ringer's lactate. It has been empirically observed that approximately 300 cc of crystalloid is required to compensate for each 100 cc of blood loss. This 3:1 rule is a good beginning point for fluid resuscitation, but obviously is not a hard and fast rule for those with massive hemorrhage. If the 3:1 ratio were adhered to in a casualty requiring 5,000 cc of blood replacement, inundation would result. About 3,000-4,000 cc of Ringer's lactate seems reasonable.

Several clinical parameters are utilized by the medical officer in determining the casualty's response to the therapeutic intervention. Assessment of clinical response can be made on the basis of changes in blood pressure, pulse rate, capillary refill, urine output, and mental status. Where large volumes of fluid and blood are required, the progress of therapy is facilitated by central venous pressure monitoring. The centrally-placed catheter affords an accurate measure of the right heart's volume requirement and its ability to accept additional fluid loading. Serial measurements are clearly of greater value than a single determination. Sophisticated systems that measure cardiac output and the pulmonary artery wedge pressure do not add a great deal to the early treatment or treatment assessment of the combat casualty.

Blood transfusion is an integral part of the resuscitation of casualties presenting with Classes III and IV hemorrhages and in those with continuing hemorrhage. Whole blood is preferred due to its lower viscosity, faster infusibility and potential provision of

some of the clotting factors. Prior to hospital arrival, a more forward echelon may have already infused low-titer type 0 blood. Those casualties that have been started on type 0 blood should continue to receive type 0. Switching to type-specific blood, especially after several units of type 0 blood have been given, can result in a transfusion reaction secondary to the reaction between anti-A and anti-B introduced into the recipient by donor 0 blood and the antigens A and B in the patient's blood. As a general rule, if four units or less of low-titer 0 blood have been given, a change to type-specific blood is possible without producing ill effects. It is recommended that **type-specific** blood be withheld for **2-3** weeks or longer if more than four units of type 0 was initially administered. Female casualties who require the immediate use of type 0 blood should be given Rh-negative, if available, to avoid the potential of future problems associated with sensitization. Ideally, the casualty is given type-specific, cross-matched blood. This was the practice of American forces in Vietnam, where 80% of the blood administered was **type-specific**. In the Korean conflict, the practice was to use type 0, low-Rh titer blood.

Whole blood should be filtered during administration to remove small clots and other aggregations. A 160 micron macropore filter accomplishes this objective. Blood infusions should be warmed to prevent not only cardiac arrhythmias but also hypothermia. The incidence of cardiac arrhythmia is highest when almost-outdated, old blood with high potassium levels is infused, when the blood is not warmed prior to infusion, and when the infusion catheter rests in a cardiac chamber. When using packed cells, it is recommended that every fourth unit be followed by a unit of fresh frozen plasma. Banked blood in the combat zone, not uncommonly, is close to its expiration date. After an infusion of about ten units of this product, coagulation defects and bleeding diatheses often arise. They should be anticipated and may be avoided by interspersed transfusions of fresh frozen plasma and platelet packs, or by intermittently infusing freshly drawn local donor blood. The majority of those requiring blood transfusions do not require calcium supplementation; however, when infusion rates exceed 100 cc/minute, 250-500 mg of calcium chloride should be given as a slow **bolus** through a separate **infusion** line.

Adequate volume replacement is reflected by a normal central venous pressure and a urine output of 0.5-1 cc/kg/hour. This level of urinary output should be substantially increase, in cases of crush injury.

The tachypnea of trauma tends to produce a state of respiratory alkalosis; however, this effect is more than overcome by the metabolic acidosis resulting from the perfusion deficit. **Persistence** of the shock state results in shifts to anaerobic metabolism, and further worsens the acidosis. Bicarbonate should be administered in those whose **pH** approaches 7.2. Serum potassium levels may rise to dangerously high levels as a result of acidosis-triggered potassium shifts. Hyperkalemia can in turn evoke cardiac arrest.

In situations in which infusion therapy fails to initiate a favorable response, conditions other than hypovolemia should be suspected. Cardiac tamponade, tension pneumothorax, **myocardial injury**, **nerogenic shock**, and acute gastric dilation may be responsible or contributory. Continued and unrecognized hemorrhage into the chest or abdomen is the most common cause of poor response to fluid therapy. In this sort of situation, the surgeon must operate to resuscitate rather than **resuscitate** to operate

The following chart outlines the classes of shock, their presenting signs and symptoms, and the guidelines for resuscitation. These are guidelines only. The amount of blood lost is estimated only as a starting point for resuscitation. Clinical parameters must guide the response to therapy.

TABLE 6.—Estimated Fluid and Blood Requirements in Shock

(Based on Patient's Initial Presentation)

	Class I	Class II	Class III	Class IV
Blood Loss (ml)	up to 750	750-1500	1500-2000	2000 or more
Blood Loss (%BV)	up to 15%	15-30%	30-40%	40% or more
Pulse Rate	100	100	120	140 or higher
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Capillary Blanch Test	Normal	Positive	Positive	Positive
Respiratory Rate	14-20	20-30	30-40	> 35
Urine Output (Ml/hr)	30 or more	20-30	5-15	Negligible
CNS-Mental status	Slightly anxious	Mildly anxious	Anxious & confused	Confused-lethargic
Fluid Replacement (3:1 Rule)	Crystalloid	Crystalloid	Crystalloid & blood	Crystalloid & blood

Adequate volume replacement can be guided by urinary output. Fifty cc per hour is a minimum objective of resuscitation for an adult. This figure should be doubled in cases of crush injury.

PROCEDURES

(Adopted with permission from the ATLS Providers Manual, ACS, 1984)

Internal Jugular Venipuncture

1. Place the patient in a supine position, at least 15° with the head-down to distend the neck veins and to prevent an air embolism. Turn the patient's head away from the venipuncture site.
2. Cleanse and prep the skin around the venipuncture site and drape the area. Sterile **gloves should** be worn when performing this procedure.
3. Introduce a large-caliber needle, attached to a 6 ml syringe, into the center of the triangle formed by the two lower heads of the sternomastoid and the clavicle.
4. After the skin has been punctured, turn the bevel of the needle upward, and expel the skin plug that may occlude the needle.
5. Direct the needle **caudally**, parallel to the sagittal plane, at a 30° posterior angle with the frontal plane.
6. Slowly advance the needle while gently withdrawing the plunger of the syringe.
7. When a free flow of blood appears in the syringe, remove the syringe and occlude the needle with a finger to prevent an air embolism. If the vein is not entered on the first attempt, the needle generally is too medial. Withdraw the needle and direct it $5-10^\circ$ more laterally.
8. Quickly insert the catheter to a predetermined depth (such that the catheter is above rather than within the right atrium).
9. Remove the needle and connect the catheter to the IV tubing.
10. Suture the catheter in place, apply antibiotic ointment, dress the area, and tape the tubing in place. Label the adhesive with the date of the procedure.
11. Obtain a chest film to check the position of the IV line and to rule out pneumothorax.

Infraclavicular Subclavian Catheterization

1. Place the patient in a supine position, at least 15° head down

to distend the neck veins and to prevent an air embolism. Turn the patient's head away from the venipuncture site.

2. Cleanse and prep the skin around the venipuncture site and drape the area. Sterile gloves should be worn when performing this procedure.

3. Introduce a large-caliber needle, attached to a 5 ml syringe, 1 cm inferior to the junction of the middle and medial thirds of the clavicle.

4. After the skin has been punctured, with the bevel of the needle upward, expel the skin plug that may occlude the needle.

5. Direct the needle medially, slightly **cephalad**, and posteriorly behind the clavicle towards the posterior, superior angle of the sternal end of the clavicle (toward a finger placed in the **supra**-sternal notch).

6. Slowly advance the needle while gently withdrawing the plunger of the syringe.

7. When a free flow of blood appears in the syringe, remove the syringe and occlude the needle with a finger to prevent an air embolism.

8. Quickly insert the catheter to a predetermined depth such that the catheter does not rest within a cardiac chamber.

9. Remove the needle and connect the catheter to the IV tubing.

10. Suture the catheter in place, apply antibiotic ointment, dress the area, tape the tubing in place, and label the adhesive with the date of insertion.

11. Obtain a chest film to check the position of the IV line and to rule out pneumothorax.

Saphenous Vein **Cutdown** at the Ankle

1. One site for a peripheral venous **cutdown** is the greater saphenous vein at the ankle, just anterior to the medial malleolus. Another secondary site is the antecubital median basilic vein, 2 cm lateral to the medial epicondyle of the humerus at the **flexion** crease of the elbow. Another site is the proximal greater saphenous vein caudad to the **fossa ovalis**.

2. Cleanse and prep the skin of the ankle, and drape the area.

3. Infiltrate the skin over the saphenous vein with local anesthetic

4. A full-thickness transverse skin incision is made through the area of anesthesia to a length of about 2 cm.

5. By blunt dissection, using a curved hemostat, the saphenous vein is **identified** and dissected free from the saphenous nerve, which is attached to the anterior wall of the vein.

6. Dissect the vein from its bed and elevate the vein for a distance of approximately 2 cm.

7. Ligate the distal mobilized vein leaving the suture in place for traction.

8. Pass a tie about the vein, proximally.

9. Make a small transverse venotomy and gently dilate the venotomy with the tip of a closed hemostat.

10. Introduce a plastic cannula through the venotomy and secure it in place with the upper ligature about the vein and **cannula**. The cannula should be inserted an adequate distance to prevent dislodging.

11. Attach the IV tubing to the cannula and close the incision with interrupted sutures.

12. Apply a sterile dressing with a topical antibiotic ointment. Label the adhesive with the date of the insertion.

Needle Thoracentesis

NOTE: This procedure is applicable to the rapidly deteriorating casualty with a life-threatening tension pneumothorax.

1. Identify the second intercostal space, in the midclavicular line on the side of the pneumothorax.

2. Insert a 14 or 16 gauge needle into the skin and direct the needle just over (*i.e.*, superior to) the top of the rib into the intercostal space.

3. Puncture the parietal pleura. If the patient has a tension pneumothorax, a rush of air will exit from the hub of the needle.

4. Prepare for a chest-tube insertion. The chest tube should be inserted at the nipple level anterior to the midaxillary line of the affected side.

5. Connect the chest tube to an underwater seal device or a flutter-type valve apparatus.

6. Obtain a chest X-ray.

Chest Tube Insertion

1. Fluid resuscitation via a large-caliber IV, and monitoring of vital signs should be in process.

2. Determine the insertion site, usually the nipple level (5th intercostal space) anterior to the midaxillary line on the affected side. A second chest tube may be required for a hemothorax.

3. Prep and drape the chest at the predetermined site of the tube insertion.

4. Locally anesthetize the skin and rib periosteum.

5. Make a 2-3 cm transverse (horizontal) incision at the predetermined site and bluntly dissect through the subcutaneous tissues, just over the top of the rib.

6. Puncture the parietal pleura with the tip of a clamp and put a gloved finger into the incision to insure that the pleural space has been entered and the area is free of adhesions.

7. Clamp the end of the thoracostomy tube and advance the thoracostomy tube into the pleural space to the desired length.

8. Look for "fogging" of the chest tube with expiration, or listen for air movement.

9. Connect the end of the thoracostomy tube to an underwater-seal apparatus or flutter valve.

10. Suture the tube in place.

11. Apply a dressing and tape the tube to the chest.

12. Obtain a chest X-ray.

Pericardiocentesis

1. Monitor the patient's vital signs, central nervous pressure (CVP), and ECG before, during, and after the procedure.

2. Prep the xiphoid and subxiphoid areas, if time allows.

3. Using a #16-18 gauge, 6-inch or longer over-the-needle catheter, attach a 35 ml empty syringe with a three-way stopcock.

4. Assess the patient for any mediastinal shift that may have caused the heart to shift significantly. This is best determined by noting the position of the palpable trachea and the point of maximal intensity of the apical heart beat.

5. Puncture the skin 1-2 cm inferior to the left of the xiphichondral junction, at a 45° angle to the skin.

6. Carefully advance the needle cephalad and aim toward the tip of the left scapula.

7. If the needle is advanced too far (into the ventricular muscle) an injury pattern (eg., extreme STT wave changes, or widened and enlarged QRS complex) will appear on the ECG monitor. This

pattern indicates that the pericardiocentesis needle should be withdrawn until the previous baseline ECG tracing reappears. Premature ventricular contractions may also occur, indicating undesired needle contact with the ventricular myocardium.

8. When the needle tip enters the blood-filled pericardial sac, withdraw as much **unclotted** blood as possible.

9. In a simple tamponade, the aspiration of pericardial blood will cause a rapid drop in the CVP and a slower improvement in the blood pressure.

10. As aspiration progresses and blood is withdrawn, the **sur-**face of the heart will reapproach the pericardial surface and the tip of the needle. An ECG injury pattern may reappear. This indicates that the pericardiocentesis needle should be withdrawn slightly. Should this injury pattern persist, withdraw the needle completely.

11. After aspiration is completed, leave the pericardiocentesis catheter in place with the stopcock closed. Secure the catheter in place.

12. Reassess all vital signs and the CVP. A full **12-lead** ECG should also be done upon completion of this procedure.

13. Should cardiac tamponade persist, the stopcock may be reopened and the pericardial sac reaspirated. The plastic **pericar-**diocentesis needle can be sutured or taped in place, and covered with a small dressing to allow for continued decompression en route to the hospital or the operating room.

CHAPTER X

Compensatory and Pathophysiological Responses to Trauma

Major combat wounds initiate sudden and intense physiological and metabolic responses. The magnitude and duration of the response are directly proportional to the extent of injury and the interval between wounding and treatment. The development of post-traumatic complications also influences the duration and magnitude of the response. The combat surgeon who has an understanding of the pathophysiological responses to trauma is better able to provide both acute and chronic care to these casual ties.

SYSTEMIC PATHOPHYSIOLOGIC RESPONSE

The magnitude of the systemic response to trauma is proportional to the extent of the injury and the local changes at the site of injury. The response is biphasic, with early post-injury hypofunction followed by later hyperfunction in most organ systems. The acute phase is characterized by progressive circulatory insufficiency, decreasing cardiac output, decreasing oxygen consumption, developing acidosis, and discharge of the adrenergic nervous system. If adequate resuscitation is provided, a chronic hyperdynamic, hypermetabolic state persists until resolution of the traumatic injury and any post-traumatic complications. This phase is characterized by an increase in cardiac output and oxygen consumption, tachycardia, and negative nitrogen balance with depletion of lean body mass. This response occurs following a variety of injuries and is modified by any pre-existing metabolic disorder or post-traumatic complications that may arise.

CARDIOVASCULAR RESPONSE

Loss of blood triggers a compensatory vasoconstriction and tachycardia, which permit a reduction of blood volume of 20-30% while maintaining the blood pressure at nearly normal levels. If hypovolemia persists or rapidly progresses below these levels, hypotension results, and, if not corrected promptly, may cause death. Changes that are characteristic of the hypovolemic shock state are decreased cardiac filling pressures, decreased systemic arterial pressure, tachycardia, and increased systemic vascular resistance secondary to catecholamine release.

Liberation of histamine, serotonin, and prostaglandins, leukocytosis; the activation products of complement and coagulation systems; and neutrophils liberated from injured tissue all contribute to a local state of increased vascular permeability. This response can aggravate the intravascular volume deficit. The acute discharge of catecholamines from the sympathetic nervous system and the adrenal glands serves to maintain tissue perfusion in the face of acute intravascular volume loss.

The catecholamines exert an inotropic influence on the heart tending to increase cardiac output which is falling secondary to decreasing preload. Peripherally, there is a redistribution of blood flow, which is in part secondary to a graded autonomic innervation in the vascular beds of various organ systems. Blood flow to vital organs, such as the brain and heart, is maintained at the expense of decreasing flow to skin, muscle, renal, and enteric beds in a prioritized fashion. This response is regulated by the density of alpha receptors responding to the vasoconstrictive influence of circulating catecholamines and by that tissue's inherent local sympathetic nervous system innervation. Hence, we see a casualty with rapid, thready pulse, and pale cool skin, before development of hypotension. If this casualty is administered anesthetic agents that depress these compensatory autonomic responses in the hypovolemic patient, hypotension and shock may result.

Following these acute reflexes, which tend to maintain perfusion, a series of endocrine responses occurs, which serves to replenish the intravascular volume. As an initial response, vasopressin (ADH) is released from the posterior pituitary. ADH exerts a direct action on the renal collecting tubule to increase passive diffusion of water across the cell and back into the peritubular vessels. Under normal conditions, ADH is primarily

regulated by hypothalamic osmoreceptors; however, in the response to acute blood loss, volume stretch receptors, **hypothalamic** osmoreceptors, and a neural pain/stress response appear to play an important role. Subsequently, the release of aldosterone, mediated through the renin-angiotensin system, following stimulation of the juxtaglomerular apparatus of the kidney, acts to maintain extra-cellular fluid volume. Aldosterone, acting at the proximal renal tubular level, causes the reabsorption of sodium and the conservation of water.

RESPONSE TO THERAPY

The hemodynamic response to the initial fluid infusion falls into one of three categories. A small number of patients will respond to the initial fluid bolus with a prompt normalization of blood pressure and will maintain hemodynamic stability. Further therapy is directed at replacing ongoing losses. This response is usually seen in patients with volume deficits of less than 20%. The majority of patients will show a transient response to the fluid bolus. Over time, the initial improvement dissipates, requiring further administration of volume to restore and maintain hemodynamic stability. Most of these patients have experienced a 20-40% volume loss, and may have ongoing bleeding necessitating surgical intervention for control. The third category consists of that small number of patients who show minimal or no response to fluid boluses and usually have an exsanguinating hemorrhage, requiring immediate surgical control. The clinical picture of this subset of patients may be compounded by myocardial dysfunction, necessitating invasive assessment of volume status and myocardial function.

The use of blood transfusions should be limited to cases of severe and ongoing hemorrhage where blood loss exceeds 30% of the total blood volume (i.e. 1500-2000 cc). Red blood cell concentration (hematocrit) determines the blood's viscosity and oxygen-carrying capacity. The goal in blood transfusion is to optimize oxygen delivery to the cells. While an increasing hematocrit allows for a greater oxygen-carrying capacity, the concomitant elevation in viscosity can cause a decreased cardiac output secondary to increased vascular resistance, which impedes the delivery of oxygen to the cell. Viscosity varies little between hematocrits of 20-35%, however, it rapidly increases above this level. In patients who are

hypermetabolic and able to elevate their cardiac output, a hematocrit of 30–35% is adequate to ensure sufficient oxygen transport in the systemic circulation. However, in the maximally stressed patient, there may be no further reserve to increase cardiac output to meet the fixed elevated peripheral oxygen needs. Under these circumstances, an infusion of red cells will increase the hematocrit and may increase delivery of oxygen to the tissues.

Transfusions may be associated with complications, including transfusion reactions, transmission of disease (donor pool dependent), and coagulopathy (in patients receiving massive transfusions) secondary to either dilution or a disseminated intravascular coagulation (DIC)-like state. Transfusion related transmission of an immunosuppressing virus is but one of many transfusion-related infectious complications. Transfusion of massive quantities of blood may result in hypothermia, which may be partially avoided through the use of a blood-warming apparatus.

METABOLIC/ENDOCRINE RESPONSE

Trauma produces a sympatheticoadrenal response which partially initiates a hypermetabolic state. Following resuscitation, oxygen consumption increases to supranormal levels. The extent of hypermetabolism is proportional to the severity of injury. The hyperdynamic response is mediated by elevated levels of the counter regulatory hormones: catecholamines, glucagon, and cortisol, which acutely maintain blood glucose levels and later maintain an accelerated body catabolism while opposing the anabolic functions of insulin. In the early post-injury period, insulin levels are low, contributing to hyperglycemia. With time, insulin levels rise toward normal, even in the presence of persistent hyperglycemia. There appears to be an altered tissue receptor sensitivity to insulin in peripheral tissues. Additionally, hepatic glucose production from peripheral precursors is elevated proportionately to the extent of injury. Epinephrine promotes glycogenolysis, also contributing to the hyperglycemia; high concentrations of epinephrine may even inhibit the production of insulin.

Anaerobic glucose utilization at the injury site represents up to 80% of the consumed glucose. The byproducts produced by the wound, lactate and pyruvate, are recycled to the liver where gluconeogenesis occurs. Accelerated peripheral proteolysis occurs

during the hypermetabolic state, resulting in an erosion of lean body mass and an increased nitrogen excretion. Amino acids from skeletal muscle are mobilized and serve as additional substrates for hepatic gluconeogenesis. In order to prevent the depletion of lean body mass in the hypermetabolic injured patient, nutritional support should be initiated following resuscitation. Nutritional support must provide sufficient protein and carbohydrate to match the elevated energy demands of the patient. The hypermetabolic response is exaggerated by post-traumatic complications such as sepsis, and is especially detrimental in casualties who are already at the limits of their metabolic reserves.

PULMONARY SUBSYSTEM

Pulmonary vascular changes parallel the systemic circulatory response to trauma. The increase in pulmonary vascular resistance is proportionately greater and more persistent than that seen in systemic vascular beds. Although the etiology of the increase in pulmonary vascular resistance is not fully understood, studies of burn patients suggest that release of vasoactive agents, primarily thromboxane, may play an important role. There appears to be little, if any, change in pulmonary capillary permeability. As a component of the hypermetabolic response to injury, minute ventilation increases significantly as a result of increases in both tidal volume and the respiratory rate. This increase in minute ventilation results in a respiratory alkalosis. Post-injury respiratory alkalosis is appropriate under these circumstances and attempts should not be made to correct it pharmacologically or to suppress the respiratory drive. Hyperventilation can be further aggravated by post-traumatic fever, anemia, or sepsis.

Post-traumatic pulmonary insufficiency can result from penetrating or perforating pulmonary injury, pulmonary contusion secondary to blunt or blast trauma, and smoke inhalation. Aspiration of gastric content is another common cause, especially in the unconscious casualty. Aspiration can result in chemical and/or bacterial pneumonitis. Respiratory insufficiency may also result from the pulmonary edema of excessive fluid resuscitation. Massive blood transfusion, usually greater than ten units over 24 hours, also predisposes to pulmonary insufficiency. The common end result of these divergent pulmonary insults can be the adult respiratory distress syndrome (ARDS). Although the specific

pathogenesis of ARDS remains undefined, it has been postulated that activation of the complement system via an alternative pathway causes aggregation and activation of neutrophils, which in turn damage the pulmonary microvasculature resulting in increased vascular permeability.

Clinically relevant ARDS manifests itself by tachypnea and an increased respiratory effort. Pulmonary secretions may be minimal and the breath sounds dry. Pulmonary compliance decreases and pulmonary arteriovenous shunting increases, with a resultant decrease in the PaO_2 . Characteristically, the decreased PaO_2 is relatively unresponsive to increases in the inspired oxygen content (F IO_2).

Chest X-ray changes may lag 12-24 hours behind pathophysiological changes. When they appear, one sees diffuse alveolar infiltrates, which commonly progress to complete consolidation.

ARDS therapy usually requires endotracheal intubation, mechanical ventilation, and the maintenance of positive end expiratory pressure (PEEP). Failure to respond to treatment is often related to pulmonary or remote infection. In those cases where treatment fails and the process progresses, the lungs become less compliant and more difficult to ventilate, even with inordinately high inspiratory pressures. In these casualties, the PaO_2 progressively falls and the PaCO_2 progressively rises, in spite of maximal F IO_2 , maximal levels of PEEP and maximal inspiratory pressures and rates. Ultimately, the hypoxemia, hypercarbia, and acidosis can result in death; however, the majority of these patients die of sepsis.

Because of the lethal problems associated with ARDS, efforts should be directed at preventing the development of the full-blown syndrome. Prophylactic pulmonary care should include avoidance of overly zealous fluid resuscitation, prevention of aspiration, and frequent pulmonary toilet. In the presence of progressively worsening ARDS requiring very high ventilatory pressures, the surgeon should consider the placement of prophylactic chest tubes. Prompt identification and treatment of both local and remote infections decreases the likelihood of sepsis-related ARDS. It may be appropriate to choose a more appropriate or effective antibiotic in some cases. Humidification of inspired oxygen and, if possible, the avoidance of prolonged utilization of high inspired oxygen concentrations are also major considerations.

The early use of diuretics and parenteral albumin, may reduce pulmonary fluid.

GASTROINTESTINAL SUBSYSTEM

Preferential redistribution of blood flow in the shock state results in splanchnic ischemia. The ischemic mucosal insult can subsequently result in gastric stress ulceration, especially in the presence of associated sepsis. Gastrointestinal hemorrhage of significant degree is usually the presenting symptom. The onset of bleeding usually presents about ten days post injury. These gastric ulcerations are frequently multiple. Perforation can occur. Prophylactic therapy consists of antacid buffering of the gastric content, and administration of a histamine hydrogen receptor antagonist, such as cimetidine. **Enteral** alimentation is also thought to provide gastric mucosal protection and should be instituted when feasible.

Intractable upper gastrointestinal hemorrhage from stress ulceration may require gastric resection or vagotomy and pyloroplasty. Perforation is another indication for operative intervention.

Acalculous cholecystitis may occur in trauma victims at a time when it is most difficult to diagnose. Presumably, it develops under the conditions of dehydration or lack of stimulation by oral intake, or from the effects of drugs. All of the foregoing occur in trauma casualties, oftentimes in association with abdominal wounds. It may mimic other more common conditions following trauma, and may progress to gangrenous cholecystitis and rupture before it is suspected.

The generalized ileus usually seen in the shock state necessitates nasogastric decompression to prevent emesis and possible aspiration.

HEMATOLOGIC AND CLOTTING SUBSYSTEMS

Certain casualties, such as those with heart or liver wounds and those with pelvic crush injuries, require very substantial infusions of whole blood. Very often, ten units of blood will have been infused before operative control of the source of hemorrhage is controlled. In the combat zone, it is not uncommon for bank blood to be nearing its expiration date. This combination of **circum-**

stances set the stage for catastrophic cardiac arrhythmia. The elevated potassium concentration of old bank blood, when infused directly into a cardiac chamber, can precipitate fatal arrhythmias. The same complication can result from infusion of large quantities of cold blood. The blood should be warmed, and infusion directly into the right atrium should be avoided.

Another common and very serious complication in this sort of circumstance is the development of a diffuse bleeding diathesis. Some degree of coagulopathy occurs routinely after about ten units of infusion and worsens as the blood requirement increases. The diathesis can be avoided, lessened, or corrected with infusions of fresh frozen plasma and platelet packs. If these components are not available, freshly drawn blood, less than 24 hours old and procured within the facility from the walking donor pool, should be employed. If the hemorrhage or diathesis persists, requiring massive transfusion, about every fourth unit should be freshly drawn. Bank blood becomes progressively platelet- and clotting factor-deficient from the third day on. Citrate in banked blood aggravates the situation. When available to the surgeon, therapy is based on the results of the platelet count, partial thromboplastin time, prothrombin time, and the fibrinogen level. With lesser laboratory capability, the surgeon must anticipate the diathesis and resort to empiricism.

Anemia will develop in those casualties where large volumes of asanguineous fluids were utilized to treat hemorrhagic shock. Reticuloendothelial system removal of damaged bank red cells and the excessive drawing off of blood for laboratory tests will contribute to the anemia.

Disseminated intravascular coagulation (**DIC**) may develop in association with shock, tissue injury, or sepsis. Consumption of clotting factors by disseminated intravascular microthrombi give rise to the consumptive coagulopathy. The casualty with DIC may present a clinical spectrum ranging from a simple **hyper**-coagulability state to fulminant consumptive coagulopathy resulting in massive diffuse bleeding. Therapy includes correction of the shock state, appropriate wound debridement, and treatment of sepsis. In the presence of laboratory evidence of DIC and elevated levels of circulating fibrin degradation products, patients with a bleeding diathesis may be treated with repeated small doses of heparin.

RENAL SUBSYSTEM

Urinary output: The decrease in urinary output that occurs as a physiological response to wounding is the result of both metabolic and vascular changes. Normally, the urinary output is in excess of 500 ml per 24 hours when the blood pressure is within the normal range and the urinary flow is not mechanically obstructed. As the systolic blood pressure is lowered by hemorrhage to a level of 60-80 mm Hg or even lower, the urinary flow decreases and may progress to oliguria; that is, to a volume of urine less than 20 ml per hour, or less than 400 ml per 24 hours.

Obstruction of a urinary catheter is a particularly likely cause of absence of detectable urinary output. Most patients, even those with acute renal failure, excrete 50 ml or more of urine per day. When anuria develops, a mechanical reason for it should be suspected. Frequent causes are obstruction or actual destruction of the urethra or ureters by wounds in the pelvic region, spasm of the urethral sphincter, and atony of the bladder. Careful physical examination; catheterization of the urinary bladder; and intravenous pyelography, cystoscopy, or exploration, as indicated, will establish the presence or absence of adequate urinary flow from the kidneys. If a urethral catheter has been inserted, it may be obstructed by mucous plugs or blood clots. These are such obvious causes of oliguria and anuria that, paradoxically, they are sometimes overlooked.

Acute renal insufficiency: Acute renal insufficiency or acute renal failure indicates sudden and essentially complete failure of the excretory function of the kidneys. This complication, in which the pathological process is acute tubular necrosis (lower nephron nephrosis), must be suspected if less than 400 ml of urine is excreted in a 24 hour period. It is important to recognize, however, that some casualties who develop the syndrome of acute renal failure do not have oliguria but may become uremic nonetheless (high-output renal failure). Although urine volumes may be normal to high, a lack of concentration indicates failure to clear solutes. Failure to recognize this fact and to monitor the patient's fluid administration may result in overhydration and fatal circulatory embarrassment of the nonoliguric as well as the oliguric patient with acute renal failure. Failure to recognize high-output nonoliguric renal failure can result in worsening the hypovolemic state, further compounding the renal insult. Paradoxical polyuria

should be replaced at 05 cc per cc of urine output, but care should be taken to avoid "chasing" the urine output and causing overhydration. Excessive urine output may be associated with significant urinary potassium losses, requiring frequent monitoring of the serum potassium level and replacement as indicated.

Factors which frequently cause acute renal insufficiency are long periods of hypotension, crushing injuries, burns, hemolytic reactions (most frequently from blood transfusions); drug nephrotoxicity, sepsis, and hypersensitivity phenomena.

At first, the urine is pale and dilute unless blood or hemoglobin is present. If hemolysis has occurred, it is characteristically dark brownish red. Proteinuria may be conspicuous for a day or two. Granular and heme-pigment casts soon appear. The specific gravity falls rapidly, and by the third day it may be as low as 1.010 and fixed.

The BUN (blood urea nitrogen) level rises rapidly. The rate of increase is closely related to the extent of trauma or to factors which influence the catabolic rate. In a massively wounded and catabolic patient with renal failure, the BUN may rise as much as 120 mg percent per day. Hyponatremia is a frequent finding and is usually attributed to an excessive administration of water rather than to an actual sodium deficit. With the development of metabolic acidosis, the serum bicarbonate falls. Hypocalcemia is frequently present. Anemia and leukocytosis are usually present, even in the absence of infection. Because infection is a leading contributory cause of death in acute renal failure, search for foci of infection is mandatory. Diarrhea, sometimes with bloody stools, may develop if uremia persists. Abdominal distention may be marked. Drowsiness, disorientation, muscular twitchings, and even convulsions may occur. Diastolic hypertension of considerable degree is not unusual. Acute pulmonary edema and congestive heart failure are more likely to develop when hypertension is marked, especially if an excess of fluid has been given. Weight loss and hypoproteinemia, progressing to emaciation, reflect the catabolic state, and dependent edema may occur even when the fluid allowances are less than conventional. The clinical course may be complicated by extensive and progressive infection, impairment of wound healing, and a distinct tendency to bleed.

Many of the abnormalities observed in acute renal insufficiency are the result of potassium intoxication. Because of catabolism,

the potassium ion shifts from its normal intracellular location to the extracellular fluid compartments. The process may be more rapid in the presence of necrotic tissue or hematoma formation and should be suspected whenever major injury to muscles is present. In the presence of acidosis and uremia, the plasma potassium levels are abnormally high, and potassium intoxication can occur on the first day after wounding in casualties who are oliguric. Frequently, physical signs or symptoms do not reflect the gravity of the situation until death is imminent. Neuromuscular and cardiac changes are manifestations of potassium intoxication. Tendon reflexes are diminished to absent, and complete paralysis may follow. Potassium intoxication causes certain electrocardiographic changes, such as high-peaked T waves in the precordial leads, a widening of the **QRS** complex, a depression of the P waves, and a sloping ST segment in the limb leads. Conduction disturbances can lead to ventricular arrhythmia and death. Fatalities from cardiac arrest secondary to potassium intoxication have been observed as early as the fourth day after wounding.

The earliest sign of acute renal failure is usually the appearance of oliguria with no other obvious cause for a decreased urinary output. Volume expansion, monitored by the CVP, will help in identifying and treating cases of prerenal oliguria. This is accomplished by rapidly administering a test load (**500-1,000 ml**) of intravenous fluid, rapidly followed by a single dose of diuretic agent. The urine specific gravity is usually 1.010 in the syndrome of acute tubular necrosis, and the urine sodium concentration is relatively high (60 to 100 **meq/l**). The **UUN/BUN** (urine to serum urea ratio) is usually less than **10:1**. Electrocardiographic and chemical determinations may confirm the presence of hyperkalemia.

The clinical manifestations of sepsis, shock, and necrosis of undebrided tissue are quite similar to those of uremia, but the differential diagnosis is seldom difficult because these manifestations appear considerably earlier than those of uremia. Even though both oliguria and azotemia may be present in the first few days after wounding, the nausea, vomiting, disorientation, and convulsions which occur at this time are not likely to be of uremic origin.

Since renal insufficiency usually is not diagnosed in its incipency, treatment during this phase, when the only manifestation is oliguria, is vascular volume expansion using blood and other

suitable electrolyte solutions,' with monitoring of the central venous pressure, adequate debridement of any wounds, a trial of mannitol intravenously injected in a 12.5-25gm bolus, and the administration of antibiotics as indicated. The concern of the medical officer in a forward unit should be the correction of potentially reversible renal failure by prompt restoration and preservation of adequate blood volume and urinary flow.

In a temperate climate, the total fluid intake for 24 hours, exclusive of blood, plasma, or plasma expanders, should be 500 ml to cover insensible loss, plus the measured output. The measured output is the total of urinary excretion, vomitus, diarrhea, fluid removed by gastric suction, and fluid lost from burned surfaces. Allowance must also be made for increased insensible fluid losses. These vary accordingly to climatic conditions and body temperature. In humid tropical regions and febrile states, these losses may be 2,000 ml per day or more.

Maintenance of the proper relationship of fluid intake to fluid output is important, for increasing the fluid intake will not increase the urinary output in acute renal insufficiency. An excessive intake, in fact, will endanger the patient's life. The responsible medical officer must give his personal attention to the calculations. A careful record must be kept, and nurses and aidmen must be instructed specifically about how to keep it. A warning notice to keep the fluid intake-output chart must be displayed prominently on the patient's bed.

The patient's thirst must not be allowed to influence the volume of intake, and close supervision is necessary to insure that he does not overhydrate himself. A daily weight record should be maintained if practical. An increase in weight implies water retention and, therefore, overhydration. A useful general rule is the maintenance of 0.5 pound daily weight loss under usual catabolic conditions.

Administration of fluids should be oral if tolerated and feasible. When parenteral administration is required, as it often is, it should be a continuous intravenous infusion at a constant rate. It is technically simple to pass a polyethylene catheter into the superior vena cava via a peripheral vein, and little trouble need be expected if the tube is allowed to remain in situ for no longer than five days. This technique minimizes the risk of thrombosis, which would be associated with infusion by a needle or cannula in a peripheral vein for this period of time. It also makes movement of the patient

simpler and allows CVP monitoring through the same catheter. Although wound management is essentially the same as in patients without renal failure, early debridement is even more critical in that damaged tissue aggravates the effects of renal failure. Hypoxia and respiratory acidosis during anesthesia should be particularly avoided, since they may promote the release of intracellular potassium into the plasma.

Caloric intake should be maintained by the use of carbohydrates and fats, with the complete elimination of protein-containing foods. Hypertonic glucose can be given effectively through a central venous catheter. Potassium should not be administered to the oliguric patient unless the concentration of the ion is deficient.

The early use of mannitol as an osmotic diuretic has been mentioned. If diuresis results from a **12.5–25g** bolus, a sustained infusion of 20% mannitol may be used to titrate an adequate urine volume. Furosemide and ethacrynic acid should be used as an initial diuretic. Caution is required in their use, however; serious adverse reactions have been reported, including deafness and death. The treatment of **hyperkalemia** with cation exchange resins, such as Kayexalate, has decreased the requirement of dialysis for the sole purpose of treating hyperkalemia. Usual doses are **10–50 gm** by mouth or enema every two to six hours. Sorbitol, as an osmotic cathartic (5–10 ml) by mouth or by enema, also promotes diarrhea and intestinal potassium losses. Since many drugs are **excreted** through the kidneys, decreased renal function requires decreased doses of most antibiotics and other drugs such as digitalis. Magnesium-containing compounds, such as antacids, should be used sparingly in the oliguric patient because of the possibility of magnesium toxicity.

An oliguric patient should not be kept in the forward area any longer than necessary. Instead, he should be evacuated as expeditiously as possible to a center that possesses an artificial kidney and that is otherwise specially equipped to treat acute renal insufficiency. If he cannot be evacuated, a patient who remains oliguric for 72 hours should be treated by the following emergency measures, designed to reduce or counterbalance an excess of serum potassium:

1. Intravenous glucose is given in 10% concentration through the superior vena **cava**. This measure will cause potassium to be reincorporated into intracellular glycogen and will lower the

serum concentration. The 'concomitant use of insulin may facilitate this process.

2. Since calcium is a specific antagonist of potassium, a continuous infusion of 10% calcium gluconate will counterbalance excess potassium if it is not extreme. Although sodium is also an antagonist of potassium, large amounts of this ion should not be used during the first days of acute renal insufficiency. Sodium, in this setting, is used sparingly and only to replace that lost by urinary excretion, gastric suction, or diarrhea. Generally, **one-third** normal saline is used to replace urine output, and normal saline to replace gastric fluid loss.

3. Fluid balance is maintained by the use of carefully calculated amounts of the required fluids. Consider, for example, a patient who has excreted 50 ml of urine and who has lost 150 ml of fluid in **vomitus** or by gastric suction within 24 hours. His measured output is thus 200 ml. This amount should be added to the basic allowance of 500 ml to give a total intake for 24 hours of 700 ml. Of this, 100 ml may be 10% calcium gluconate, 400 ml should be 10% glucose in water with 10 units of regular insulin, and 200 ml should be isotonic saline. Sodium bicarbonate in 7.5% solution may replace saline if the **pH** determination reveals metabolic acidosis. Since some wounded patients develop respiratory alkalosis, monitoring of serum **pH** is necessary to determine appropriate fluid replacement. Peritoneal and extracorporeal dialyses are effective techniques of treatment but are not usually feasible outside a special center staffed by personnel trained in these techniques.

Infection

GENERAL PRINCIPLES

War wounds are characterized by lacerated, contused, and devitalized tissue; extravasated blood; disruption of the local blood supply; presence of foreign bodies; and contamination with various microorganisms, all of which predispose to the development of subsequent infection. The devitalized tissue and extravasated blood provide an excellent culture medium to support the growth of microorganisms and thus are conducive to the development of wound infections. Injury-related edema may produce tension within a fascial compartment that compromises the capillary circulation of the tissues within the compartment, resulting in local tissue anoxia. Additionally, the anaerobic character of hypoxic tissue may inhibit leukocyte phagocytosis or limit the function of leukocytes. The time lag between wounding and treatment represents an incubation period during which bacteria may proliferate and initiate infection. Early adequate surgery is therefore the most important step in prophylaxis against wound infection. A wound, debrided of nonviable contaminated tissue and left with an excellent blood supply, is best able to resist infection.

Although early antibiotic therapy plays an important role in the prevention and treatment of wound infections, antibiotics do not take the place of early surgical therapy. Antibiotic therapy should be based upon a knowledge of the likely causative organism and the antibiotic or antibiotics most suitable for controlling the organism.

Prophylaxis and early treatment are of the greatest importance. Once infection is established, it may be lethal and it is always costly in terms of further destruction of tissue, persistence of disturbed body physiology, delayed wound healing, and prolonged morbidity. Underlying medical problems, such as malignant disease, diabetes, malnutrition, and metabolic disease, may reduce an

individual's resistance to microorganisms. These factors, however, are uncommon in the typical active duty military casualty.

ETIOLOGIC FACTORS

The development of a wound infection is generally associated with one or more of the following factors:

- (1) Delay in surgical treatment.
- (2) Inadequate wound debridement.
- (3) Associated vascular injury resulting in regional tissue ischemia.
- (4) Inadequate hemostasis at the initial wound operation, resulting in subsequent hematoma formation.
- (5) Retention of foreign bodies within the wound.
- (6) Failure to provide adequate drainage.
- (7) Tight packing of the wound or the use of tight circular dressings or casts.
- (8) Primary closure of war wounds.
- (9) Failure to recognize and treat a perforated hollow **viscus**.
- (10) Wound contamination with bacteria that are resistant to antibiotics.
- (11) Secondary contamination from fomites, or exposure to personnel who are carriers of pyogenic bacteria.
- (12) Presence of metabolic diseases, such as diabetes, which predispose to the development and spread of infection.

DIAGNOSIS OF INFECTION

The classic signs and symptoms of infection are redness, swelling, heat, and pain. Redness of the skin is due to intense hyperemia and is seen in infections which involve the skin or subcutaneous tissue and, in some patients, in the skin overlying foci of **sup**-purative thrombophlebitis. The hyperemia is responsible for the local increase in temperature. Fever and tachycardia are additional but nonspecific signs of infection. Rigors and chills are suggestive of septicemia.

Leukocytosis commonly accompanies acute bacterial infection. Generally, the more severe the infection, the greater the leukocytosis. The leukocytosis is characteristically accompanied by an increase in the proportion of immature granulocytes, the so-called "left shift."

Exudate from the area of infection should be examined for color, odor, and consistency. A Gram stain of the exudate should be performed immediately to facilitate prompt institution of appropriate antimicrobial therapy. For each bacterial cell observed under microscopic oil immersion lens examination, there are approximately 10^5 similar organisms in each milliliter of exudate from which the smear was prepared.

A wound biopsy is a useful method of confirming the presence of infection in a wound, particularly in a burn wound or wounds of the subcutaneous and soft tissues. Areas of the wound that appear purulent or reveal new focal areas of discoloration should be biopsied. If the technical capability exists, a portion of the specimen should be sent to the microbiology laboratory for quantitative culture. The recovery of 10^5 or more organisms per gram of tissue from a quantitative culture is suggestive but not necessarily diagnostic of infection. This finding is highly sensitive but not specific for infection, since proliferation of colonizing organisms may account for such bacterial densities. The remaining portion of the specimen is forwarded to the pathologist for histologic examination. The histologic finding of microorganisms in viable tissue is highly specific and is diagnostic of infection. Consequently, the examination of histologic sections prepared from a biopsy specimen is the most reliable means of differentiating contamination or colonization of nonviable tissue from infection of viable tissue.

BACTERIOLOGY

Bacterial contamination of a war wound is certain. The wounds are contaminated at the time of injury and secondary contamination may occur at any time during the course of treatment. Clostridium species are commonly introduced at the time of injury. Hemolytic Staphylococci and Streptococci may also be introduced at the time of wounding or by later contamination with such organisms in the hospital. Animal studies have shown heavy growth of Gram-positive cocci and Clostridium species in experimental missile wounds after delayed debridement. **Gram-negative bacilli** are typically encountered later and are often hospital acquired. Patients with abdominal injuries are also at risk of developing Gram-negative infection, particularly those with an injury to a hollow **viscus**. Many of these bacteria produce toxins

and enzymes to facilitate their spread through tissues within wounds. Coagulase, fibrinolysin, proteinase, collagenase, and hyaluronidase favor the development and spread of wound infection.

The results of cultures taken from wound walls after debridement in animal studies indicate that, even though the degree of contamination or colonization can be significantly reduced by prompt debridement, the wound is not sterilized. Persistence of microorganisms in the wound following mechanical cleansing and removal of damaged tissue justifies the use of prophylactic antibiotics.

SURGICAL THERAPY

Prompt, adequate surgical debridement is the cornerstone of therapy of war wounds, particularly with respect to prevention of infection. In addition to adequate debridement and excision of crushed and lacerated tissue, the removal of foreign bodies and reduction of microbial density are important considerations. The current recommendation is that war wounds be debrided within six hours of injury.

Although such classic signs as impaired contractility, altered consistency, and lack of capillary bleeding have been shown to correlate poorly with tissue viability, they have a useful function. If there is any question about the adequacy of debridement, the wound is dressed and reexplored three to five days later. If there is no residual nonviable tissue and no evidence of infection, the delayed primary closure is performed. Delayed primary closure effects timely closure of an initially heavily-contaminated wound while minimizing the risk of infection. An even longer delay in wound closure may be indicated in some wounds, as was supported by the recent-albeit limited-experience with septic complications in limb wounds during the Falkland's campaign. This study showed that no septic complications developed in those patients undergoing delayed closure eight days or later from time of injury (none of five patients). Fifteen percent developed septic complications when closed at 5-7 days (six of 40), and 75% (three of four) when closed within four days. If at the time of inspection, 3-5 days post injury, nonviable tissue remains or infection is present, further debridement is performed and the infection is treated before closure is attempted.

ANTIBIOTIC THERAPY

The primary emphasis of antibiotic treatment of wounds is early administration before an infection becomes established. During the Yom Kippur War, medical personnel were instructed to administer antibiotics routinely to all wounded. A recent review of infections following soft-tissue limb wounds in soldiers injured during the Falkland Campaign indicated that a delay in surgery and a delay in antibiotic administration were the most important factors related to the subsequent development of infection. When surgical delay was unavoidable, the delay in antibiotic administration assumed an even greater importance. That study showed a greater incidence of septic complications when debridement was delayed more than six hours, as well as an increased incidence of infectious complications when the time from wounding to antibiotic administration exceeded six hours.

An animal study of **.223-caliber** high-velocity projectiles in a porcine model demonstrated that bacterial proliferation could be prevented with early institution of intravenous penicillin therapy. Another study in wounded pigs suggested that the growth of mixed flora in a contaminated missile wound predisposed the wound to infection with other more pathogenic strains and impaired the ability of reversibly-injured tissue to recover. The mixed flora in that study consisted of bacterial strains usually sensitive to penicillin. Yet another study in wounded pigs demonstrated a decrease in the amount of devitalized tissue during debridement at 12 hours in penicillin-treated animals as opposed to animals not treated with penicillin.

Selection of antibiotic therapy is based upon a knowledge of likely causative organisms, examination of the Gram stain of the wound exudate, and culture and sensitivity studies of the wound. The characteristics of antibiotics useful against various organisms commonly encountered in surgical infections are described in the table at the end of this chapter (Table 7).

HYPERBARIC THERAPY

In 1963, Brummelkamp and associates in Amsterdam reported the first use of hyperbaric oxygen in the treatment of infections caused by gas-producing microorganisms. The patients were placed in a room-sized chamber in which the air pressure was

raised to three atmospheres. During the course of three days, the patient inhaled 100% oxygen from a face mask for one-and-a-half hours on seven occasions. This increased the oxygen tension in plasma, lymph, and tissue fluids by 15-20 fold. Dramatic clinical improvement was described for most patients within the first day. Large pressure chambers are available at only a few medical centers in the world and at special military and marine industrial facilities. Much less expensive single patient chambers are now available. Therapy with hyperbaric oxygen, antibiotic administration, and surgical debridement has been reported as effective in patients with clostridial myonecrosis who evidenced toxicity. Hyperbaric oxygen appears to reduce toxemia and diminish the amount of tissue requiring excision. However, patients with gas-producing infections due to anaerobic Streptococci, *Escherichia coli*, and Klebsiella species showed no improvement after exposure to high-pressure oxygen. All of the foregoing notwithstanding, the use of hyperbaric oxygen is not feasible in the theater of operations. Even in referral centers, it is advocated only as an adjunct to the surgical treatment of clostridial infections, and not as a substitute for conventional modes of therapy, including early surgical debridement and the administration of antibiotics.

CLOSTRIDIAL INFECTIONS

Three types of clostridial infections of ascending severity have been described: simple contamination, clostridial cellulitis, and clostridial myonecrosis. Simple contamination of a wound by clostridia is common. It causes no discomfort to the patient and should be of little concern to the surgeon. A thin seropurulent exudate may be present. If the necrotic tissue harboring the microorganisms is debrided, there will be no subsequent invasion of surrounding tissue. The frequent contamination of war wounds with clostridia is due to the ubiquitous nature of this organism. A high oxygen tension in the surrounding healthy tissues prevents invasions in these areas.

Clostridial cellulitis is characterized by the presence of gas in necrotic and viable subcutaneous tissue that produces crepitus on palpation. Intact healthy muscle is not invaded. The cellulitis produces a foul-smelling **seropurulent** discharge from the depths and crevices of a wound. There are often local extensions along fascial planes, but involvement of healthy muscle and marked

toxemia are absent. The predominant organisms are proteolytic and nontoxigenic clostridia, such as *Clostridium sporogenes* and *Clostridium tedium*. Clostridial cellulitis generally has a gradual onset. The incubation period is from 3-5 days; systemic effects are usually mild, there is no toxemia; the skin is rarely discolored; and there is little or no edema. These characteristics distinguish the infection from gas gangrene.

Clostridial myonecrosis, or gas gangrene, is the most serious of the clostridial infections. This infection occurs most often in association with severe wounds involving large masses of muscle that have been contaminated with pathogenic clostridia, especially *Clostridium perfringens*. Such wounds are commonly caused by the high-velocity missiles of modern warfare and by crush injuries in which the skin is broken. Clostridial myonecrosis principally (although not exclusively) occurs in the lower limbs, buttocks, and upper limbs. In association with the muscle injury, the arterial supply to the limb may be impaired and the damaged tissues may be contaminated by soil, clothing, and other foreign bodies. Glycolysis continues in the anoxic wound with a drop in the oxygen tension, accumulation of lactate, and fall in pH providing an ideal environment for the growth of clostridia. Once bacterial growth is established and toxins and other products of bacterial metabolism accumulate, invasion of uninjured tissues is promoted and the anaerobic infection is established. Resistance to the infection and its spread is compromised by the avascularity of the necrotic tissue that prevents entry of phagocytes, antibodies, or systemically-administered antibiotics into that tissue.

Culture of sites of clostridial myositis usually yields several species of toxigenic clostridia, particularly *Clostridium perfringens*, *Clostridium novyi*, and *Clostridium septicum*. The common habitat of these species is the soil, but they also are found in the intestines of many animals, including man. The toxic metabolites elaborated by the anaerobes, together with other substances produced by their actions on the muscle, are responsible for the local pathological changes in the muscle and the associated toxemia and anemia.

The diagnosis of gas gangrene can often be made on the basis of clinical findings alone. The usual onset occurs one to four days after injury; however, onset can vary from 8-10 hours at one extreme to five or six days at the other. The most striking feature is a rapid deterioration of a casualty who had previously been

progressing satisfactorily. Pain is frequently the earliest symptom of clostridial myonecrosis and is frequently disproportionate to the apparent severity of the wound. Fever is common and blood pressure falls as the infection advances. Anemia and dehydration are common late findings. Examination of the wound may reveal profuse serous or serosanguineous discharge sufficient to soak through massive dressings. The discharge may contain gas bubbles, and it occasionally yields large Gram-positive rods evident on microscopic examination.

Although clostridial myonecrosis often is described as emitting a characteristic rotten meat odor, this is not always the case. The odor emitted from the wound is variable, ranging from sweet and pungent to foul and fetid, depending upon the species of bacterial present. Gas production is more marked with *Clostridium perfringens* infections than with other types of clostridia. Gas bubbles may be seen dissecting along fascial planes on roentgenograms; however, the absence of tissue gas does not exclude clostridial infection.

Several other conditions must be differentiated from clostridial myonecrosis. Anaerobic cellulitis is characteristically limited to the subcutaneous tissue and fascia, and does not involve muscle. Gas formation is far greater than in gas gangrene. The brownish and purulent discharge is profuse. Pain and toxemia are not prominent. Local changes include cutaneous erythema and swelling. This redness distinguishes it from clostridial myonecrosis. Anoxic gangrene results from ligation or failure to repair a damaged major extremity artery. It is often differentiated from clostridial myonecrosis by the history and absence of toxemia and other evidence of infection.

Although animal experimental data exist showing that penicillin alone will prevent gas gangrene, there are no data from humans to confirm this. Early adequate surgical debridement of war wounds remains the primary means of preventing gas gangrene, its threat to life, and the mutilating effects of the management required when it becomes established.

Preoperative antibiotic therapy consists of penicillin G, three million units IV followed by a total of 10-24 million units over the 24-hour preoperative period. Appropriate volume restoration measures should also be used. Antibiotic and fluid therapy should not significantly delay surgical intervention. In vitro studies have shown that both clindamycin and metronidazole, utilized as single agent therapy, are equally effective if penicillin cannot be used.

Ample exposure of the wound is necessary and rapid removal of the affected tissue is essential. When the infection is confined to a single fascial compartment, surgical excision of the affected muscle or muscle groups may be **sufficient**. Excision, however, must be as radical as is necessary to remove all discolored muscle and any muscle that does not bleed or contract when it is incised. This may mean removal of an entire muscle from origin to insertion, complete removal of a whole muscle group, or (if the whole limb is involved) amputation of the limb. When infection has extended beyond the practical limits of amputation or disarticulation, the fascial planes and muscle sheaths are incised to relieve tension and promote drainage. If septic shock develops, placement of a **Swan-Ganz** catheter will permit monitoring of cardiac function and the patient's intravascular volume status. Postoperatively, intravenous fluids should be infused to maintain an adequate hourly urine output between 30-50 cc. Intravenous penicillin is also administered in the postoperative phase.

In World War I, 5% of wounded patients developed gas gangrene with a fatality rate of 28%. In World War II, 0.7% developed gas gangrene with a 31% fatality rate. In Korea, 0.08% developed gas gangrene with no mortality recorded. Its incidence in Vietnam was even lower. This may be attributed to prompt adequate **debridement** and vascular repairs, attention to casts, and good surgical technique rather than lack of organisms.

STREPTOCOCCAL MYONECROSIS

Anaerobic streptococci may cause necrosis of tissue in association with gas formation. Streptococcal myonecrosis, originally described in the **1940s**, resembles subacute clostridial gas gangrene. After an incubation period of **3-4** days, there is swelling, edema, and a purulent exudation from the wound. The infected muscle initially appears pale and soft but as the infection progresses, it becomes bright red and then finally purple and gangrenous. These signs are followed by pain, which rapidly becomes severe. Gas is present in the infected tissue and the involved muscle becomes gangrenous. The seropurulent discharge has a sour odor. The management of streptococcal myonecrosis includes surgery, combined with the antibiotic regimen outlined for gas gangrene. Surgery consists of relaxing incisions, extending through the deep fascia and into muscle, that will provide adequate drainage and relieve tension. Care

must be taken to extend the excision beyond the area of obvious infection into the neighboring or adjacent viable tissue.

TETANUS

Tetanus is a severe infection caused by *Clostridium tetani* and its toxin's effects on the nervous system. It carries a mortality rate of approximately 50 %. In an analysis of Vietnam War wound infections, no cases of tetanus were reported. The infection is characterized by local and general convulsive spasms of the voluntary muscles. *Clostridium tetani* is a strict anaerobe which exists in spore form in the soil and in the intestines of animals and man. Local necrosis and ischemia provide the conditions necessary for contaminating spores to evolve into their vegetative form and multiply rapidly at the site of infection. Once the vegetative forms have begun to multiply, large amounts of tetanus toxin are produced.

The incubation period is usually 6-12 days but may vary from 4-21 days or longer. In any event, the incubation period is sufficiently long to prevent the development of tetanus in war wounds if proper prophylaxis is employed within a day or two of injury.

Small, deep puncture wounds that often appear trivial are important sources of this infection and must be considered prone to tetanus. Early clinical manifestations may be of a general nature, such as irritability, insomnia, muscular tremors, local spasm, or rigidity in the muscle near the wound. Trismus is usually the first symptom. Sore throat, painful dysphagia, stiff neck, and difficulty in beginning micturition may be early evidence of muscular irritability. The dental officer may be the first to see the patient if trismus has been mistaken for some oral condition.

Trismus and risus sardonicus resulting from spasm of the masseters and muscles of the face are signs of established tetanus. Arching of the spine (opisthotonos) and respiratory difficulty from laryngeal and intercostal muscle spasm may also be present. The contractions are aggravated by additional spasms whenever any sensory excitation occurs. Usually reflex spasms are brought on by external stimuli, such as moving the patient or striking the bed, but later they occur spontaneously at regular and increasingly frequent intervals until the height of disease is reached. Spasms often begin with a sudden jerk. Every muscle in the body is thrown into intense tonic contraction; the jaws are tightly clenched, the head is

retracted, the back is arched, the chest and abdomen are fixed, and the limbs are usually extended. A severe spasm may result in respiratory arrest, Spasms may last a few seconds or several minutes. When spasms occur frequently, they lead to rapid exhaustion and sometimes to death from asphyxiation. Without spasms, mortality is low. Few patients with severe spasms survive

Cephalic tetanus is a form of tetanus in which irritation or paralysis of cranial nerves appears early and dominates the picture The facial nerve is affected most often. Ophthalmoplegia from involvement of the ocular nerves may develop. Trismus and dysphagia may also follow wounds of the head and face, and the symptoms often appear first on the injured side

Severe tetanus is often fatal, but those who recover do so completely without sequelae The patient who has survived tetanus is not immune and, unless immunized, is susceptible to a second attack. Recurrent tetanus in the same patient has been reported. Apparently a sublethal amount of tetanus toxin is not sufficient to provide an adequate antigenic stimulus for production of active immunity.

The diagnosis of tetanus is a clinical one, with bacteriological confirmation sometimes possible The morphologic appearance of the organism in stained smears (the so-called tennis racket terminal spore in Gram-positive bacillus) usually is not sufficient to differentiate *Clostridium tetuni* from other anaerobes with terminal spores The disease proceeds with fever, sweating and oliguria while the mind remains clear. Death usually occurs as a result of respiratory arrest during painful generalized convulsions. Toxemia, pneumonia secondary to aspiration, hyperpyrexia, and cardiac failure are other causes of death.

SURGICAL THERAPY

The surgical care of wounds should be immediate The most important features of surgical wound care are thorough cleansing and debridement. Foreign bodies and necrotic tissue can be contaminated massively with *Clostridium tetuni* and establish wound conditions promoting growth and exotoxin production. The wounds should be left open until the patient has recovered from the convulsion stage of disease Antibiotic therapy with penicillin is effective against the vegetative cells of *Clostridium tetuni*. Treatment of patients with severe tetanus involves the use of muscle

relaxants and sedation, as well as maintenance of fluid and electrolyte balance. Pulmonary toilet is necessary, as is elimination of visceral stimuli such as distention of the urinary bladder and fecal impaction. Careful nursing care is required. Translaryngeal intubation or even tracheostomy may be useful in maintaining a patent airway in patients who undergo frequent episodes of respiratory failure

TETANUS IMMUNIZATION

In 1984, the Committee on Trauma of the American College of Surgeons published recommendations concerning prophylaxis against tetanus and the management of wounds. Immunization in adults requires at least three injections of toxoid. A routine booster of absorbed toxoid is indicated every 10 years thereafter. Combined tetanus and diphtheria toxoid is recommended for routine or post-wounding boosters.

In individuals not adequately immunized (that is, the patient who has received only one or no prior injections of toxoid or the immunization history is unknown), 0.5 ml absorbed tetanus toxoid should be given for nontetanus-prone wounds. For tetanus-prone wounds, 0.5 ml absorbed toxoid and 250 units or more of human tetanus immune globulin should be given, using different syringes, needles, and sites of injection. Completion of the series of toxoid immunizations should then follow.

When the medical officer has determined that the casualty has been previously fully immunized and the last dose of toxoid was given within 10 years, no booster of toxoid is indicated for nontetanus-prone wounds. For tetanus-prone wounds and if more than five years have elapsed since the last dose, 0.5 ml absorbed toxoid should be given. When the patient has had three prior injections of toxoid and received the last dose more than 10 years previously, 0.5 ml absorbed toxoid for both tetanus-prone and non-tetanus-prone wounds should be given.

Passive immunization with tetanus immune globulin must be considered individually for each patient. Characteristics of the wound, the conditions under which it was incurred, its treatment, and the patient's age should all be considered. Immunization with human immune globulin is not indicated if the patient has ever received two or more injections of toxoid and the wound is less than 24 hours old. An injection of human immune globulin is indicated

if the wound is felt to be a **tetanus-prone** wound more than 24 hours old and only two prior toxoid injections have been administered. An injection of human immune globulin is also indicated for patients with tetanus-prone wounds who have not received any prior toxoid injections or only one prior injection.

ABDOMINAL WOUNDS

Sepsis is the most common cause of death in patients who sustain penetrating abdominal trauma and survive initial surgical therapy. Prophylactic antibiotic therapy for such patients should be directed toward pathogens encountered in the lower gastrointestinal tract and should be administered perioperatively for 24 hours. A generally accepted regimen of combination antibiotic therapy consists of an agent effective against the anaerobes (clindamycin or metronidazole) and an aminoglycoside (**gentamicin**) effective against Gram-negative rods. Recent studies of antibiotic therapy following penetrating abdominal trauma suggest, however, that single agent therapy with cefoxitin is equally effective. Given the lack of nephrotoxicity with cefoxitin and considering that the battlefield casualty likely exhibits some degree of dehydration, this regimen represents an attractive alternative. A review of wounded patients in the Vietnam War revealed that abdominal wounds were the wounds that most frequently became infected (6.89% following initial treatment). Penetrating abdominal wounds accounted for 24% of all wound infections but only 13% of all wounds.

MANAGEMENT OF SEPTIC SHOCK

Shock due to uncontrolled infection in a surgical patient requires prompt identification and **treatment of** the septic process. Control of infection by surgical debridement or drainage and the use of specific antibiotics represents definitive therapy. An attempt to identify the primary site of infection should be made upon diagnosis of this condition. If the source of infection is amenable to surgical control, this should be carried out expeditiously as soon as the patient's condition is sufficiently stable. Broad spectrum antibiotic therapy is initiated and based upon likely infectious organisms. A typical treatment regimen consists of triple antibiotics, such as ampicillin, gentamicin, and clindamycin. **Reple-**

tion of the intravascular volume with a physiologic crystalloid solution is generally recommended. Some authors advocate infusion of colloid-containing fluid to replace intravascular volume deficits. Since an increase in pulmonary capillary permeability accompanies septic shock, attempts to replete volume with **colloid**-containing fluid in this condition may result in a detrimental increase in extravascular pulmonary water.

Fluid therapy is best managed with the use of Swan-Ganz catheter monitoring of pulmonary artery wedge pressures and cardiac output. Insertion of a Foley catheter for measurement of the hourly urine output is also necessary. Many patients with sepsis and shock will develop pulmonary insufficiency necessitating endotracheal intubation and assisted ventilation. Inadequate tissue oxygenation is a consistent factor in shock, and therefore efforts to maintain a normal oxygen hemoglobin dissociation curve should be undertaken. Alkalosis, decreased pCO_2 , decreased hemoglobin concentration, decreased **2,3-diphosphoglycerate**, and the presence of **carboxyhemoglobin** are all factors which increase the affinity of the hemoglobin molecule for oxygen and thereby inhibit delivery of oxygen to tissue.

Vasoconstrictive drugs are seldom used to raise blood pressure as they have a deleterious effect upon tissue blood flow. Agents such as epinephrine and norepinephrine support the circulation by a combination of a beta 1 adrenergic cardiac effect and alpha 1 adrenergic peripheral vasoconstrictive effect. The usual dose of epinephrine is 0.5 mg IV. Norepinephrine is usually administered in the form of a continuous intravenous infusion of **D5W** containing 8 mg per liter at a rate of 2-3 cc per minute or higher if needed to achieve the desired hemodynamic effect. These agents are used only when volume-restorative measures have failed to provide adequate blood pressure- to perfuse vital organs. When **volume**-restorative measures are ineffective, low dose dopamine infusion may be helpful in maintaining renal perfusion, but only as an adjunct to fluid infusion. Dopamine is thought to dilate renal and splanchnic vasculature by its action on the dopaminergic receptors. The usual intravenous "renal" dose of dopamine is 3-5 $\mu g/kg/min$ given as **D5W** containing 200 **mg/250** ml. This dosage can be increased for beta 1 adrenergic cardiac stimulation, and when given in doses greater than 10 $\mu g/kg/min$, commonly causes alpha stimulation and vasoconstriction that provide additional hemodynamic support in a deteriorating patient.

TABLE 7.—*Choice, Mode of Action, Spectrum, and Dosage of Antibiotic Agents*

Agent	Mode of Action	Antibacterial Spectrum of Clinical Importance	Dosage
Penicillin G	Bactericidal; interferes with bacterial cell wall synthesis	Streptococci, Pneumococci Clostridia, Neisseriae, Corynebacteria, Pasteurella multocida, Actinomyces, Treponema , Listeria	30 mil units IV/day every 2-4 hours
Ampicillin	Bactericidal; same as above	Hemophilus influenzae, Proteus mirabilis, Salmonellae, Shigellae and some E. coli. Grampositive organisms as with Penicillin G	8 gm IV/day every 6 hours
Gentamicin	Bacteriostatic; inhibition of bacterial protein synthesis	Klebsiella sp., Aerobacter sp., Pseudomonas aeruginosa, Serratia, indole positive Proteus sp., some Methicillin-resistant Staphylococci	5 mg/kg/day every 8-12 hours
Metronidazole	Bacteriocidal	Gram-negative anaerobes especially Bacteriodes fragilis. Also effective against several protozoa.	2 gm IV/day every 4 hours
Cefoxitin (2nd generation cephalosporin)	Cell wall synthesis inhibitors; stable to staphylococcal beta lactamases	Sames as above plus Bacteroides fragilis; not active against Enterobacter spp.	12 gm IV/day every 6 hours
Clindamycin	Bacteriostatic; inhibits protein synthesis	Gram-positive and Gram-negative anaerobes, Gram-positive aerobic cocci, Streptococcus faecalis. Clostridia	1200-2700 mg IV/day every 6-8 hours