Part X: Trauma

Chapter 71: Abdominal and Pelvic Injuries

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Abdominal and pelvic injuries are major contributors to morbidity and mortality after trauma. Blunt abdominal injuries account for less than 10% of total injuries, but contribute to about 25% of deaths. The incidence of penetrating abdominal injuries is higher, approaching 25%. Important considerations of abdominal and pelvic injuries are:

1. the severity of associated injuries (ie, to chest and head);

2. potential for severe haemorrhage;

3. difficulties in diagnosing organ damage; and

4. increased risk of post-traumatic sepsis and multi-organ failure.

Mechanisms of Injury

1. Penetrating Wounds

Stab and gunshot wounds account for most penetrating injuries to the abdomen.

(a) *Stab wounds* are immediately life-threatening when vessels are injured. Major vascular injury occurs in about 5% of all abdominal injuries, and is almost entirely due to penetrating injuries. Stab wounds are often multiple. Penetration of the thoracic cavity should be suspected when the wound is situated in the upper abdomen. About 25% of penetrating abdominal wounds involve thoracic structures. Conversely, 15% of stab wounds and 46% of gunshot wounds to the anterior chest involve abdominal structures. In one series of penetrating injuries to the anterior chest, 38% required combined thoracotomy and laparotomy, and 17% required laparotomy alone.

(b) *Gunshot wound* injuries depend on the calibre of the missile, its velocity and trajectory. Modern assault rifles and automatic weapons impart a tumbling action to the fired bullet, which causes extensive injury in its course through the body. On the other hand, small calibre, low velocity bullets from small hand guns result in minimal soft tissue injury, and may not exit the body of the victim.

2. Blunt Trauma

Motor vehicle accidents (MVAs) account for most blunt injuries to the abdomen and pelvis. Injuries may also occur as a result of falls and in industrial accidents. Associated injuries are common, with 50% sustaining one other injury, and 38% two or more injuries. Abdominal injuries accompany blunt chest injuries in 37% of cases. Neurological (brain and spinal cord), skeletal, and faciomaxillary injuries may also be commonly involved

Restraining devices (ie, seatbelts and child seats) in motor vehicles have reduced mortality and severity of head and faciomaxillary injuries in MVAs, but have resulted in a changing pattern of abdominal trauma. Poor seatbelt design and incorrect use are partly responsible. Seatbelt injuries are more severe in lateral impact collisions. Rotation and lateral movement of the belted victim focus impact and deceleration forces over narrow areas defined by the belt. Thus, trauma to the lower thoracic cage, liver, spleen and mesentery is common. Injuries to hollow viscera and retroperitoneal structures have increased markedly with the use of seatbelts. Loosely fitted lap belts may contribute to a higher incidence of pelvic and bladder injuries.

Initial Management

1. Resuscitation

Immediate resuscitation, early triage, diagnosis of severe injuries, and recovery of trauma victims to an area of definitive care are essential priorities. Active fluid resuscitation is essential during retrieval of severely injured patients with evidence of blood loss. On arrival at a trauma centre, resuscitation *should not* delay definitive surgery to control haemorrhage or manage other life-threatening complications.

Use of the pneumatic anti-shock garment (PASG, previously called MAST suit), is controversial, as it has not been shown to improve trauma mortality. Overall systemic arterial pressure is improved, but it may increase haemorrhage in thoracic cavity injuries, and worsen the respiratory status. Moreover, with prolonged use, risk of renal dysfunction may be increased. Nevertheless, the PASG deivce is useful to control haemorrhage in the pelvis and lower limbs, until more definitive therapy is available.

2. Clinical Diagnosis

A full and careful clinical examination by an experienced physician is most important. Eyewitness accounts of the accident may help to focus attention on particular anatomical areas. Areas of contusion and haemorrhage should be noted, and the relationship of superficial injury to underlying viscera observed. All body areas must be examined when penetrating injury is suspected, especially the back.

Penetrating injuries present few diagnostic problems, and the management dilemma relates to whether or not to explore the abdomen. Blunt abdominal trauma is more difficult to diagnose clinically, except when abdominal signs are obvious. When haemorrhage is suspected (eg, increasing abdominal distension) immediate surgery is warranted. However, usual features of intra-abdominal pathology, such as abdominal pain, tenderness, and guarding, may be absent or difficult to assess in multiple trauma. Patients with accompanying chest, head and skeletal injuries, may have more obvious symptoms and signs which mask an intra-abdominal condition. Life support therapy instituted for life-threatening injuries add to diagnostic difficulties. In these less obvious situations, diagnosis of (blunt) intra-abdominal injuries can be aided by screening procedures.

3. Diagnostic Procedures

(a) *Peritoneal lavage* is superior to abdominal paracentes and 4 quadrant taps. It has an accuracy over 95% in detecting intra-abdominal blood, with a low false-positive rate. However, if used in every case as the sole diagnostic criterion, it can lead to a "false laparotomy" rate of up to 14%, because of detection of bleeding from pelvic fracgtures and minor retroperitoneal injuries. Using the red blood cell count of lavage fluid increases diagnostic accuracy (Table 1), as gross inspection of the effluent alone may not be adequate. The general indications for peritoneal lavage are shown in Table 2.

Table 1. *Red Blood Cell Count for Positive Peritoneal Lavage in Abdominal Trauma*

Penetrating Injury	Red Blood Cells/mm^3	
Blunt Trauma	> 100.000	= positive findings
	50-100.000	= equivocal
Penetrating Injury		
Stab wound		
abdomen	> 100.000	= positive finding
	50-100.000	= equivocal
lower chest	> 5.000	= positive finding
Gunshot wound	> 5.000	= positive finding.

Table 2. *Indications for Peritoneal Lavage in Abdominal Trauma*

Blunt Abdominal Trauma
- Suspected trauma in unreliable examination, eg, coma
- Unexplained hypotension
- Multiple truma requiring general anaesthesia

Penetrating Injury
- Stab wounds known or suspected to infiltrate peritoneum
  - abdominal wounds
  - lower chest wounds
- Gunshot wounds suspected to infiltrate peritoneum.

(b) *Computerized tomography (CT)* is less sensitive than peritoneal lavage in detecting intraperitoneal blood. However, it is particularly useful when retroperitoneal injury and pelvic fractures are suspected, and may disclose the sourcwe of intra-abdominal bleeding or injury. Abdominal CT may also be useful to detect severe renal injury in patients with haematuria, and may be more sensitive than intravenous pyelography in this regard. Nevertheless, interpretation of CT scans in trauma patients requires considerable experience, and the scan is unsatisfactory in 20-30% for technical reasons. Moreover, the use of CT should be restricted to stable patients as it is time-consuming, uses considerable staff resources, and removed the patient from areas of definitive care.

(c) *Scintigraphy and ultrasonography* are also operator dependent and time-consuming, and are only useful in the stable patient.

(d) *Selective angiography* may be useful to detect the source of haemorrhage from pelvic fractures or retroperitoneal structures, and is obligatory when vascular injuries are suspected.

In most situations, peritoneal lavage can be used alone as the primary diagnostic modality. It can be performed easily in the emergency room without interrupting resuscitative measures. If the lavage findings are unhelpful or inconsistent with the clinical examination, CT scanning should follow. A CT scan may guide subsequent treatment (ie, surgery versus conservative management). However, CT detection of intraperitoneal blood may be spurious, if the examination follows peritoneal lavage and residual fluid is present.

Specific Injuries

Injury to the Spleen

The spleen is the organ most frequently injured by blunt abdominal trauma. The presentation of splenic injury varies according to the severity of injury, and diagnosis may be delayed in cases of mild trauma. When associated chest or neurological injuries are severe, minor splenic injury may not initially be detected. Fractures of the lower left ribs are found in about 40% of patients with splenic rupture. Minor trauma may cause splenic injury when the spleen is enlarged, eg, from malaria, lymphomas, and haemolytic anaemias. Peritoneal lavage is useful to detect splenic injury when diagnosis is unclear.

Splenic injury may be managed by:

1. splenectomy
2. laparotomy and preservation of splenic tissue, and
3. close observation.

The risks of overwhelming sepsis in infants and young adults who have undergone splenectomy (see Chapter 63, Severe Sepsis), have led to a more conservative approach. However, non-operative management has a high failure rate (approaching 70%). Hospital stay is prolonged, and subsequent operative splenic salvage is much more difficult. For these reasons, laparotomy and a splenic preservation procedure is the preferred management.

Operative procedures to conserve splenic tissue have included partial resection, use of local clotting substances (eg, gelfoam and ativine), mesh wrapping of the spleen, and splenic artery ligation. Splenectomy with autotransplantation of splenic tissue has also been advocated, but its efficacy in preventing overwhelming post-splenectomy infection (OPSI) is as yet unproven.

Splenectomy is reserved for patients with severe and uncontrolled haemorrhage, pulverized spleens, or severe multiple injuries, where delays due to repairing the spleen may compromise survival. In patients who have undergone splenectomy, polyvalent pneumococcal vaccine (Pneumovax) should be administered. The incidence of OPSI in an adult population is low (1 per 332 patient years of observation). Use of penicillin prophylaxis is primarily recommended for children and young adults, whose risk of OPSI is increased 50 times in later life.

Injury to the Liver

The liver is the second most commonly injured organ after blunt abdominal trauma, and the most common with penetrating abdominal injuries. Blunt injuries of the liver are usually diagnosed by peritoneal lavage, and other diagnostic tests (eg, CT scan) are of little additional value. Anatomical injuries include simple capsular tears and lacerations, multiple or stellate lacerations, avulsion or crush injuries, and hepatic venous injuries. In most cases (about 70%) liver injuries can be managed with little difficulty.

Surgical management may present problems when injury is severe and/or devitalized liver tissue is present, and bleeding is active. When bleeding is excessive, haemostasis can often be achieved by manual compression of the liver with gauze packs. Temporary packing of the liver is often sufficient control. The packs are removed in a repeat laparotomy 48-72 h later. If haemorrhage continues despite packing, occlusion of the porta hepatis (Pringle manoeuvre) may be tried. If this, in turn, is unsuccessful, exploration of the laceration to ligate injured vessels is required. Once bleeding is controlled, the liver edges can be approximated and the injury repaired. Avulsion and crush injuries require debridement to remove devitalized tissue. With liver surgery, adequate drainage is important to avoid accumulation of blood and liver secretions, and thus, secondary infection.

Occasionally, hepatic artery ligation may be necessary to control severe liver haemorrhage. The common right or left hepatic artery can be ligated, depending on the anatomical site of injury. The gall bladder must also be removed with right hepatic artery ligation, otherwise viscus necrosis will result. Hepatic artery ligation may not be helpful when bleeding is from the venous system. Hepatic venous injury with or without vena caval involvement, is a particularly troublesome problem. Early surgery is imperative. Surgery requires adequate exposure of the injured area, which may include a median sternotomy for access to the thoracic inferior vena cava. An intracaval shunt may be inserted to isolate the haemorrhage while repair is performed.

Postoperative care of patients with liver injuries involves continuing blood transfusion, correction of coagulation defects, and respiratory, cardiovascular, and nutritional support. Dilutional coagulopathy is common. Patients must be observed in the immediate postoperative period for hypoglycaemia, thrombocytopenia or hypoalbuminaemia. Early complications of liver injury relate to complications of hypoperfusion or massive blood transfusion. Late complications are usually associated with sepsis.

Injuries to Duodenum, Small Intestine, and Colon

Injuries to hollow viscera are commonly due to penetrating injuries rather than blunt trauma. The incidence of hollow viscus injury in abdominal gunshot wounds is approximately 95%. Viscus injury from abdominal stab wounds may be difficult to diagnose, unless peritonitis or unexplained hypovolaemia is present. A plain erect abdominal X-ray may show free air in the peritoneal cavity, but is not presumptive evidence of hollow viscus injury, as air may have entered through the abdominal wound. Diagnostic peritoneal lavage may reduce the incidence of unnecessary abdominal exploration of stab wounds. Instillation of contrast material into the wound tract has been used as a diagnostic aid, but with poor results.

Blunt abdominal injuries to mesentery, small intestine, and colon are increasing with the use of seatbelts, and are more difficult to evaluate. Colonic injuries usually present with obvious peritoneal signs. However, small bowel and duodenal perforations often have minimal signs on physical examination. Duodenal injuries have a high mortality, primarily due to delay in diagnosis. It is not unusual for the initial clinical examination to be normal, and established peritonitis presents several hours later. Diagnostic peritoneal lavage may be useful to provide an indication for laparotomy. CT is a very sensitive indicator of free intraperitoneal air, and when combined with an upper gastrointestinal contrast study (via a nasogastric tube), may be useful to identify retroperitoneal duodenal injuries or haematomas.

Preoperative antibiotics to cover enteric aerobic and anaerobic organisms should be administered to patients with bowel injury and continued for 48 hours postoperatively. Simple closure of lacerations, or primary repair and anastomosis, combined with bowel decompression are procedures for duodenal, small bowel and colon injuries. However, in extensive injury or intraperitoneal soiling, a faecal diversion procedure with delayed repair is indicated. Adequate abdominal drainage *must* be instituted. Intraperitoneal lavage with antibiotics may be useful to reduce abdominal sepsis. If surgery has been delayed, and established peritonitis, abscess formation or necrosis has occurred, mortality is high. "Open management" of intra-abdominal sepsis is currently being proposed and evaluated. The abdomen is not surgically closed, and repeated lavage and drainage is performed under direct vision - (an abdominal mesh zipper facilitates this). (See Chapter 63, Severe Sepsis.)

Diaphragm and Abdominal Wall Injuries

Diaphragmatic injury (see Chapter 69, Chest Injuries) occurs in less than 5% of cases of blunt injury and is commonly associated with injuries to abdominal organs. Diaphragm and abdominal wall injuries are more common with penetrating injuries. Herniation of abdominal viscera into the pleural cavity may result in respiratory difficulties and evisceration may occur with abdominal wounds. Injuries should be repaired to avoid postoperative herniation of abdominal contents. In many cases of blunt rupture of the diaphragm, repair is more readily performed via a thoracotomy incision. Blast injuries to the abdominal wall require debridement and a synthetic mesh implant may be required to close the defect.

Retroperitoneal Haematomas

Retroperitoneal haematomas can be classified into:

1. Pelvic haematomas, which are usually associated with pelvic and/or bladder injuries.

2. Flank haematomas, located lateral to the psoas muscles and rectus sheath, above the iliac crests, and which are usually associated with parenchymal renal injury, and occasionally, injuries of the vessels of the colon or lateral or posterior abdominal wall.

3. Central haematomas, primarily located in the central retroperitoneal area above the pelvic brim, which have 2 subgroups:

(a) The retroperitoneal haematom arises from injuries to major arteries or vein.

(b) The haematoma is associated with injuries to the pancreas, duodenum and local structures.

Retroperitoneal injury may also be of a combined type involving several of the above mentioned structures. There is a high incidence of associated injuries, including liver (22%), spleen (12%), and hollow viscera (20%). Pancreatic injuries mostly involve the capsule and a limited amount of pancreatic parenchyma, and need only drainage and possible debridement. When the pancreatic duct is involved, distal pancreatectomy is required to avoid fistula formation. Pancreaticoduodenectomy is only rarely required.

Management of retroperitoneal haematomas depends on the severity of injury and evidence of persistent bleeding. A patient with haemodynamic stability is managed by observation and simple measures, eg, fixation of pelvic fractures. When haemorrhage is persistent, diagnostic angiography and embolization may be the treatment of choice. If haemorrhage persists then laparotomy may be required. Management is similar, if retroperitoneal haematoma is an incidental finding at laparotomy, ie, if bleeding is not active, exploration of the haematoma is not indicated. Continuing bleeding associated with pelvic injuries cause more serious management problems.

Pelvic Injuries

Injuries to the pelvis can present difficult problems in trauma management. Immediate haemorrhage may be uncontrollable (see below). There is also potential long term disability due to damage of pelvic nerves controlling bladder, anorectal area and sexual function. Furthermore, the important structural role of the pelvis in transmitting weight from the body to the lower extremities may lead to permanent physical disability. Damage to the urethra also gives rise to significant long term disability.

Pelvic fractures can be classified into 3 types.

1. *Type I fractures* are comminuted (crush) injuries which involve 3 or more major components of the pelvis, and are often unstable.

2. *Type II fractures* are unstable injuries which are often associated with haemorrhage and fracture displacement. They can be classified into 4 subgroups:

(a) diametric fractures with cranial displacement of the hemipelvis (Malgaigne fracture),

(b) undisplaced diameteric fractures,

(c) open book (sprung pelvis), and

(d) acetabular fractures.

3. *Type III fractures* are stable injuries of 2 types:

(a) an isolated fracture, or

(b) a fracture of the pubic ramus.

Type III fractures are sgtable and may require no further treatment other than immobilization and symptomatic pain relief.

The majority of pelvic injuries (approximately 75%) become haemodynamically stable after initial resuscitation. Simple measures such as immobilization or internal/external fixation of fracture diastasis may be required to assist haemodynamic control, nursing, and early mobilization. Surgery is also indicated when genitourinary injury is suspected. Ruptured bladder or torn urethra occurs in approximately 30% of pelvic fractures. Suprapubic cystostomy and bladder repair or exploration of the lower genitourinary tract should be performed. About 5% of pelvic fractures are associated with a urethral injury, usually at the prostatomembranous junction. Patients with complete urethral disruption invariably develop stricture at the site of disruption. Elective repair can be attempted 2-3 months after injury. This approach reduces impotence to about 20%, which may be the lowest, unavoidable incidence. Secondary stricture formation occurs in 15-20%, and often can be managed by endoscopic urethrotomy and urethral dilatations.

Severe haemorrhage occurs in approximately 25% of pelvic fractures and requires immediate attention. Haemorrhage is primarily due to lacerations of veins in the posterior pelvis, and to small arterioles associated with fractures in cancellous bone. A PASG may be used initially during resuscitation. It is imperative to reduce fractures, and external fixation may help to control bleeding. If haemorrhage is severe, arteriography and embolization of identified bleeding vessels is warranted. Major bleeding from the common iliac, external iliac or hypogastric arteries can be treated by operative repair. In some cases ligation of the artery may be required. When the bleeding involves superior gluteal or pudendal arteries, embolization at the time of arteriography is more effective.

Compound fractures of the pelvis involving the rectum, intestines or vagina present particular problems in management. There is a high incidence of faecal contamination, even if the rectum is not injured. Diversion of the faecal stream is mandatory if severe pelvic sepsis is to be prevented. The mortality of patients treated with a faecal diversion procedure is halved at 25%. In addition, devitalized tissue should be excised and adequate drainage instituted.

Injury to the Kidney

Kidney injuries may result from blunt or penetrating injuries. Microscopic or gross haematuria is the first indication of kidney injury on initial assessment. Such patients should undergo peritoneal lavage, as there is a high incidence of associated injuries to other abdominal organs. Haematuria also requires investigation of the genitourinary tract. An excretory examination, intravenous pyelogram (IVP) is the primary diagnostic tool. CT scan is useful in defining anatomical renal injury. In severe kidney damage, IVP may not detect the extent of damage, and CT is strongly indicated.

Bed rest and observation successfully manages 95% isolated blunt injuries to the kidney. Once haematuria has cleared, ambulation is allowed. All renal injuries due to penetrating injuries should undergo surgical exploration, unless the injury is regarded as

minor. Excessive or persistent retroperitoneal bleeding, or the presence of pulsatile retroperitoneal haematomas, urinary extravasation, or vascular injury requires immediate surgery. Surgical repair of the injured kidney is successful in about 90% of cases. Total nephrectomy is seldom necessary, and should only be performed when the kidney is non-viable.

Complications

1. Coagulopathies

Abdominal and pelvic injuries are often associated with significant blood loss and blood transfusion replacement, with resultant dilutional coagulopathy. Disseminated intravascular coagulopathy (DIC) may also develop. (See Chapter 88, Haemostatic Failure.) Management is standard, including haemological/clotting studies and replacement of blood components.

2. Nutrition

Patients with abdominal and pelvic injuries often have delayed recovery of gastric and bowel function. Parenteral nutrition is instituted when acute fluid, blood, and blood component replacement is completed. A feeding jejunostomy feeding tube can be placed during laparotomy.

Patients with absent bowel activity should also receive some form of stress ulceration prophylaxis (eg, H_2 antagonists, Sulcralfate, oral antacids).

3. Infection

Abdominal and pelvic injuries may be more susceptible to infection for the following reasons:

(a) Contamination of peritoneal cavity from penetrating wounds.

(b) Depressed immune function mediated through neurohumoral mechanisms activated by trauma, pain and haemorrhage.

(c) Nosocomial infection from invasive procedures.

(d) Post-splenectomy sepsis (see above).

(e) Delayed diagnosis of hollow viscus injuries.

Intra-abdominal sepsis remains an important preventable cause of death after trauma. Early diagnosis and institution of effective lavage and drainage procedures may reduce the incidence of intra-abdominal sepsis. Prophylactic antibiotics for 48 hours are warranted in penetrating injuries. The development of unexplained fever and/or neutrophil leucocytosis, or multiple organ failure point to intra-abdominal sepsis. (See Chapter 63, Severe Sepsis.) Prompt and definitive treatment of associated injuries may lessen the risk of infection.

4. Acalculous Cholecystitis

Acute post-traumatic acalculous cholecystitis is a serious and life-threatening complication, and mortality may approach 66%. Acalculous cholecystitis usually occurs 3-4 weeks after injury, and is associated with severe shock on admission, multiple transfusions, use of high doses of narcotics, sepsis, respiratory failure requiring mechanical ventilation, acute renal failure, and parenteral nutrition. Patients present with fever, leucocytosis, right upper quadrant pain and tenderness, with or without a mass. Liver function tests indicate hyperbilirubinaemia in about 60%, and increased alkaline phosphatase is detected in 22%. Untreated, the condition may lead to necrosis or gangrene of the gall bladder, and perforation with generalized peritonitis follows. Early surgery may reduce mortality, and consists of cholecystectomy and/or cholecystotomy. About half the patients have positive bile cultures at operation, due to aerobic and anaerobic enteric organisms. Prophylactic antibiotics should be given for the immediate operative and postoperative periods. Right upper quadrant tenderness is difficult to assess in abdominal and pelvic injuries. Therefore, any case of unexplained fever, leucocytosis or sepsis should be investigated by ultrasonography or abdominal CT scan.

5. Jaundice

Jaundice is a not uncommon complication of abdominal and pelvic injuries. Its aetiology is usually multifactorial. Factors implicated include massive blood transfusion, resorption of soft tissue haematomas, cholestasis, and hepatocellular injury from hypoperfusion, hypoxaemia and/or septicaemia. Improved screening procedures have lessened the risk of viral hepatitis from blood transfusion, but high-risk groups (eg, intravenous drug users or homosexuals) should be observed for concomitant viral infections. Precautions should be taken with the handling of blood and body fluids from such patients.

6. Acute Respiratory Failure

Acute respiratory failure may occur as an immediate or delayed complication of abdominal or pelvic injury. In the acute phase, respiratory failure is usually due to massive blood and fluid replacement. Laparotomy and surgical procedures in the upper abdomen may contribute. Abdominal pain, tenderness and distension splint the abdomen to limit deep inspiratory efforts and inhibit effective coughing. Respiratory failure in the later phase is usually due to sepsis (intrabdominal or nosocomial pneumonia), and will persist unless infection can be controlled.

7. Acute Renal Failure

Acute renal failure shows a similar pattern of disease as acute respiratory failure. The early phase of injury may be complicated by severe hypovolaemia and prolonged hypotension, which may lead to acute tubular necrosis. Effective pre-hospital retrieval and resuscitation have contributed to a decline in the incidence of acute renal failure from these causes. When renal failure develops as a late complication, its cause is invariably intra-abdominal sepsis. Recovery cannot be expected unless the sepsis is adequately controlled.

Chapter 72: Near-Drowning

T E Oh

Death by drowning claims over 700 lives each year in Britain, 500 in Australia, and 6000 in the USA; and the rate continues to rise in the last 2 countries. The incidence on a global scale is estimated to be 5.6 deaths per 100,000 population. In Queensland, drowning now accounts for most deaths in the under 5 years old, more than road accidents or congenital abnormalities. Alcohol consumption and epilepsy are prominent factors in deaths by drowning. Death may be caused by laryngeal spasm, lung reflexes, and vagal cardiac effects (ie, "immersion") rather than true drowning with aspiration of fluid. Drowning can occur in very shallow water, and the volume of water inhaled by a drowned victim may be relatively small.

Near-drowning may then be defined as survival, at least temporary, following asphyxia while immersed in a liquid medium. Useful figures of near-drowning cases in Australia are unknown.

Pathophysiology

Upon submersion, there is an initial period of voluntary apnea. The "diving reflex" (as induced by cold-water immersion of the face) consisting of apnoea, bradycardia, and intense peripheral vasoconstriction with preferential blood shunting to the heart and brain, occurs in infants and toddlers, and to a lesser extent in man. Initial voluntary apnoea reaches a "breakpoint" (determined by hypercarbic and hypoxic drives) when involuntary inspiration is made. Water then enters the lungs, and at the same time, gasping occurs. Laryngeal spasm may follow in some victims. Airway resistance is increased, reflex pulmonary vasoconstriction occurs, surfactant is diminished, and lung compliance is decreased. Water shifts from alveoli into the circulation. Swallowing, vomiting and aspiration of vomitus is likely. A phase of secondary apnoea follows within seconds of immersion, proceeded by further involuntary gasping and loss of consciousness. Respiratory arrest and cardiac arrhythmia occur several minutes later and precede death.

Hyperventilation before diving increases the risk of death by drowning. The resultant hypocarbia will suppress the central drive to breathe, even in the presence of severe hypoxaemia from the prolonged voluntary breath holding. Consciousness is lost before spontaneous central respiratory efforts resume.

Nature of Inhaled Fluid

In fresh water drowning, water is quickly absorbed into the circulation and may cause haemolysis. Pulmonary surfactant characteristics are altered (denatured) producing widespread atelectasis. Electrolyte changes are usually insignificant and transient. Haemolysis may unusually be significant, which may then produce haemoglobinuria and acute renal failure. Any chlorine and soap in fresh water does not appear to be of any adverse consequence to the lungs.

In sea water drowning, the hypertonic salt water promotes rapid fluxes of water and plasma protein into the alveoli and interstitium, dilutes or washes out surfactant, and disrupts

the alveolar-capillary membrane. Both inhaled fresh and sea water produce an inflammatory reaction in the alveolar-capillary membrane, leading to an outpouring of plasma-rich exudate into the alveoli. Inhaled gastric contents may contribute to this reaction.

Lung Injury

Regardless of whether fresh or salt water was the immersion medium, the above changes lead to widespread atelectasis, pulmonary oedema, severe intrapulmonary shunting, gross ventilation:perfusion mismatch, increased pulmonary vasoconstriction, decreased compliance, and marked hypoxaemia. Hypoxaemia and large increases in intrapulmonary shunting can occur with inhalation of as little as 2.5 mL/kg body weight.

Denaturization of surfactant can continue despire successful resuscitation. The term "secondary drowning" has been used to describe pulmonary insufficiency which may develop any time up to 72 hours after the event. This occurs after a period of improvement following resuscitation and is seen in about 5% of survivors. Hyaline membrane formation in small airways and alveoli has been demonstrated at autopsy in patients who have survived from 12-72 hours. Infection and theadult respiratory distress syndrome (ARDS) may follow a near-drowning incident.

Dry Drowning

An estimated 10-20% of drowning are "dry", ie, little or no fluid is found in the lungs at autopsy. It has been suggested that in this group, the initial entry of water into the larynx may produce a (vagal) reflex laryngospasm which persists until asphyxial death supervenes. The laryngospasm is followed immediately by an outpouring of thick mucus, which with bronchospasm, may prevent entry of water when the spasm relaxes shortly before death. "Dry drowning" appears to be more common in adults, and facilitation of such pulmonary reflexes by raised blood alcohol levels has been suggested.

Superimposed Hypothermia

If the environment is cold, cases of drowning may be complicated by acute hypothermia. Cold water impairs motor activity and movement. Even stron swimers with life-jackets drown within minutes if the water is very cold (eg, 4°C). Uncontrolled involuntary hyperventilation occurs in immersion in a cold medium, and consciousness may be impaired in hypothermia. Drowning will result when there is no control of respiration. Hence submersion is not essential for drowning and life-jackets will not always prevent drowning.

Cardiovascular Effects

The cardiovascular system of most near-drowned victims shows remarkable stability. A wide variety of ECG changes have been reported, but early reports of ventricular fibrillation secondary to fresh water aspiration were probably overemphasized. Blood pressure changes seem to be secondary to the state of oxygenation. Blood volume changes secondary to fresh or salt water aspiration are rarely significant to be life-threatening. Consequently, changes in haemoglobin and haematocrit are usually not marked.

Management

The basic pathophysiological problems of fresh and salt water drownings are similar, ie, hypoxaemia, pulmonary oedema, metabolic acidosis and circulatory dysfunction. Initial management of the critically ill survivor is thus similar, discounting whether fresh or salt water was the immersion medium. Therapy is directed towards restoring adequate oxygenation and circulation, correcting acid-base imbalance, and cerebral resuscitation and protection. Many regimens such as HYPER which aggressively treats over *hy*dration, *p*yrexia, *e*xcitability, and *r*igid motor posturing use empirical and controversial treatment methods discussed below.

1. Immediate First Aid Treatment

Cardiopulmonary resuscitation is initiated. Lung drainage procedures are controversial. There is a likelihood of inducing vomiting, since over half the immersion victims vomit during resuscitation. Portable oxygen-powered suckers are inadequate for aspiration of vomitus. Mouth-to-mouth ventilation with external cardiac massage where indicated, should be instituted immediately. Oesophageal obturators are useful in experienced hands, but the mask and bag resuscitators are generally unsuitable outside the hospital environment. Oxygen should be given and the victim kept warm while en route to hospital. The possibility of spinal injury, especially in diving or surfing accidents, must be remembered during resuscitation.

2. Hospital and Intensive Care Treatment

It is important to know the time and place of the immersion, the immersion medium and its temperature and degree of contamination, the resuscitation details including duration of apnoea or asystole, the level of consciousness at the time, whether head or neck injuries were sustained, and the past health of the victim (eg, whether an epileptic, asthmatic, or alcoholic).

(a) *Restoring Ventilation and Oxygenation*

Oxygen is given by a semi-rigid mask if the patient is breathing spontaneously. Bronchospasm if present, is relieved by aminophylline and beta-2 adrenergic agents. Comatose patients are intubated. Mechanical ventilation is instituted in patients with severe hypoxaemia and pulmonary oedema. Positive end expiratory pressure (PEEP) improves pulmonary oedema as well as ventilation:perfusion mismatch. The level of PEEP varies with the clinical situation. Intermittent mandatory ventilation (IMV) with PEEP may be used to control $PaCO_2$ if the patient is able to tolerate the ventilation pattern. If the patient is able to spontaneously maintain a normal $PaCO_2$ without too much effort, continuous positive airway pressure (CPAP) may be used instead of controlled ventilation. (See Chapter 22, Mechanical Ventilatory Support.) Awake patients seldom require endotracheal intubation. CPAP, by means of a tight-fitting CPAP facemask, is applied to these patients. Treatment of ARDS is described in Chapter 25, Adult Respiratory Distress Syndrome.

(b) *Restoring Circulation*

Low cardiac output is corrected by positive inotropic agents (eg, adrenaline, dopamine

or dobutamine infusion) and fluid replacement. Isotonic fluids are usually all that is required, but plasma and blood may be needed if haemolysis is severe. Fluid replacement is guided by central venous pressure and pulmonary capillary wedge pressure measurements in case of shock. The regimen of fluid restriction with IV frusemide, aimed at lowering raised intracranial pressure in near-drowned vistims is controversial.

Arrhythmias from acidosis, hypoxia, hypothermia and electrolyte abnormalities are treated conventionally. (See Chapter 7, Cardiac Arrhythmias.)

(c) *Correcting Acidosis*

Intravenous sodium bicarbonate (50-100 mmol) is given if the metabolic acidosis is significant (eg, pH < 7.0).

(d) *Rewarming*

Core temperature must be kept above 28°C, as temperature below that may give rise to spontaneous ventricular fibrillation and coma may be anticipated at temperatures below 30°C. Induced hypothermia for brain protection in near-drowning victims (eg, using surface ice packs) is controversial and its effectiveness unknown. Normothermia should probably be maintained. However, it is pointless to rewarm the immersed victim rapidly is his temperature is above 30°C. Rewarming can be accomplished over about 6 hours by warmed intravenous fluids, humidification of inspired gases, and heated blankets. Hot baths are difficult to carry out in practice. More aggressive forms of treatment include warm peritoneal lavage and cardiopulmonary bypass but are rarely indicated.

(e) *Cerebral Protection*

Attempts at brain resuscitation and protection are probably important. (See Chapter 41, Cerebral Protection for full discussion.) Treatment protocols include intracranial pressure monitoring, hyperventilation (to maintain a $PaCO_2$ of approximately 30 mmHg or 4 kPa), lowering raised body temperature, maintaining adequate oxygenation and circulation, and controlling intracranial hypertension, hypertension, hyperglycaemia and fits. Steroids and barbiturate therapy are controversial - their benefits remain unproven and they should probably not be given.

(f) *Other Treatment*

In general, prophylactic antibiotics are not useful. A broad spectrum antibiotic (eg, amoxicillin or cephalosporin) is indicated if there are signs of infection. It may be necessary to prescribe against Gram-negative and anaerobic bowel organisms (eg, with gentamicin and metronidazole). The site of immersion may have some influence on the type of inhaled organism and thus antibiotics. A nasogastric tube should be inserted to decompress the stomach and drain possible large volumes of water.

Investigations and Monitoring

1. Cardiovascular Monitoring

This includes ECG, arterial and central venous pressure, and (if indicated) a Swan-Ganz catheter for pulmonary artery pressure.

2. Oxygenation

Monitoring of arterial blood gases, saturation (by pulse oximetry) and lung shunting (eg, alverolar-arterial oxygen gradient) will indicate progress and guide therapy.

3. Body Temperature

4. Serum Biochemistry

Theoretically, serum electrolyte levels fall in fresh water drowning and rise in salt water drowning. However, gross changes are rarely seen in human victims because a very large water volume would need to be aspirated to produce persistent changes in serum concentrations. Serum osmolality estimation on admission may be useful.

5. Haematological

Haemoconcentration may disguise the presence of anaemia. Tests for haemolysis are:

(a) Free haemoglobin in urine.

(b) Free haemoglobin in plasma.

(c) Decreased serum haptoglobin. (Free plasma haemoglobin combines with serum haptoglobins and the resultant complex is taken up by the liver).

(d) Increased serum methaemoglobin. (Free plasma haemoglobin divides into globin and haem. The haem moiety is oxidized into methaem, which combines with serum albumin.)

6. Radiology and Imaging

A chest X-ray may show infiltrates and pulmonary oedema. Patients with normal X-rays on admission generally survive with therapy. Skull and cervical X-rays are required if the possibility of spinal injury exists. Head CT scans are indicated for comatose patients. Where there is suspicion of child abuse (eg, bath tub immersion), consideration is given to a skeletal survey examination.

7. Neurological

Evoked brain potential tests and electroencephalograms may be useful additional investigations to head CT scans. Psychometric assessments are recommended in survivors with suspected intellectual damage.

8. Drug Assays

Blood alcohol estimations and those of serum levels of anti-convulsant and sedative drugs may be indicated on admission.

9. Microbiological

Cultures of aspirated water, tracheal swabs and sputum may be indicated in severely polluted water immersion.

Complications

Complications of near-drowning after rescue which may be seen in the ICU include secondary drowning (see above), fits, hyperpyrexia, pneumonia, septicaemia, gastrointestinal bleeding, ARDS, and multi-organ failure.

Prognosis

In near-drowning without aspiration of water, complete recovery usually results if resuscitation is commenced early. With aspiration, the outlook is less predictable. While the severity of hypoxaemia and metabolic acidosis frequently correlates with the extent of pulmonary injury, blood gases on admission should not be used as prognostic factors of survival. Surf immersion, cold water, short immersion times, and skilled administration of cardiopulmonary resuscitation are favourable factors. Triage classification following successful resuscitation have been reported to be prognostically useful:

A = awake patients (ie, conscious and alert) and
B = blunted patients (ie, obtunded but rousable and responds purposefully to painful stimuli) had a better than 90% chance of recovery without neurological deficit.
C = comatose patients (ie, unrousable, with abnormal respiration and no purposeful response to pain stimuli) had a 34% mortality, and a fifth of survivors had neurological impairment.

Time of first respiratory efforts after rescue is believed to be important. Prognosis is good if the first gasp is within 30 min of rescue and there is continuing clinical improvement, especially in children. The presence of fixed dilated pupils on admission to ICU, and an arterial pH below 7.0 are bad prognostic sign.

Of all children rescued lifeless, about 1/2 from fresh water and over 2/3 from surf drownings will survive. About 3% of all child survivors will exist in the vegetative stage, and 2% have major chronic neurological problems. A third of apparent normal child survivors show minimal cerebral dysfunction on psychometric testing, but sequential recovery is possible. Resuscitation and treatment should not be abandoned early, especially in young victims, since survival after immersion for minutes has been reported, particularly in cold water immersion.

Chapter 73: Burns

C Aun, T E Oh

Severe burns produce devastating physical and psychological effects. Management involves a wide spectrum of activities ranging from initial resuscitation to eventual surgery and rehabilitation. Burn patients are most effectively treated by specially trained staff in an environment controlled in temperature, humidity and perhaps by laminar flow isolation. This chapter will limit its scope to resuscitation and early care of burn patient.

Pathophysiology

Burns may be inflicted by heat (wet or dry), chemicals, electricity or radiation. The severity and extent of injury is usually classified as:

1. *First degree burn* which involves only the epithelial layer. It is often very painful, but resolves with no residual scarring.

2. *Second degree burn* which involves epithelium and to a varying degree of dermis. Pain and scarring vary according to the depth of the dermal injury.

3. *Third degree burn* which involves the full skin thickness. It is usually painless due to destruction of cutaneous innervation and will lead to scarring.

Cardiovascular and Circulatory Effects

There is a marked, immediate increase of vascular permeability in the area of burn. Transvascular leakage becomes generalized in burns involving greater than 20% of total body surface area (TBSA). The transvascular fluid is similar to plasma in protein and ionic content. Sequestration and oedema formation is maximal in the first 24 hours. The increased vascular permeability is probably due to the direct effect of heat on the vascular tissue, and to vasoactive substances such as the leukotrienes, prostaglandins, oxygen radicals and histamine that are released from the burned tissues.

Cardiac output falls significantly due to reduced plasma volume. The contribution of a circulating myocardial depressant factor to the reduction in cardiac output remains controversial. Burn shock is most likely to occur in the first 48 hours in patients with more than 20% TBSA burn, leading to hypoperfusion of vital organs.

Red cells loss due to haemolysis and intravascular coagulation is likely to occur in patients with extensive burns (over 20% TBSA) and may be as high as 1% of red cell mass per 1% of full skin thickness burn. The increased red cell destruction is thought to be due to some agents in plasma which modify red cells making them more liable to destruction.

Coagulopathy may occur in the resuscitation phase. Apart from dilution, there is also a rapid activation of clotting factors leading to shortening of clotting times and acute reduction of various clotting factors including prothrombin, Factors V, VIII and IX. Disseminated intravascular coagulation (DIC) is common in extensive burns. Intravascular

platelet aggregation with thrombocytopenia and thrombocytopathy may necessitate platelet transfusion. Thereafter a phase of normal coagulation parameters is usually present. A hypercoagulable state may develop 2-4 weeks post burn.

Respiratory Effects

Pulmonary dysfunction is a major cause of mortality and morbidity. The incidence and causes are related to numerous factors. Thermal injury ni a closed environment may not only cause pulmonary inhalation injury, but also intoxication from combustion products. Of these, the important ones are carbon monoxide and cyanide. Upper airway damage is due to heat and inhalation of hot particles or noxious chemicals. The pathophysiology of parenchymal lund damage is still uncertain. It may be caused directly by thermal injury and chemical irritation or indirectly by fluid overloading, secondary infection, the adult respiratory distress syndrome, and pulmonary embolism.

The work of breathing increases significantly from increased airway resistance and reduced lung compliance. Arterial hypoxaemia with hypocarbia is the usual finding on blood gas analysis. These changes are most marked during the 10 to 14 days following burns. Bronchopneumonia is usually caused by airborne organisms rather than by haematogenous spread from the infected burn wound.

Metabolic Effects

Increase in metabolic rate associated with nitrogen loss following thermal injury, is greater than after any other forms of trauma. There is a proportion increase in oxygen consumption. The stress response is manifested by persistent hyperpyrexia, tachycardia, hyperventilation and hyperglycaemia. The mediators of the response are humoral; primarily an increased release of catecholamines, along with other anti-insulin hormones are responsible for the glucose intolerance noted during the early postburn period. Plasma insulin level is low immediately after thermal injury, but it is usually followed by a prolonged "insulin resistance" phase. Hypermetabolism increases with cooling, pain and sepsis. Hence efforts to limit the metabolism are directed to raising the environmental temperature, reducing evaporative loss by covering the burn areas with heterograft, and dressing the wound with pain-free dressing.

Immunological Effects

Both immune and nonspecific inflammatory host defense systems are generally depressed in severe burn injuries. Complement and immunoglobulin levels are initially depressed, but swiftly return to normal. A plasma inhibitor of chemotaxis is probably responsible for the depression of chemotaxis. Despite advances in burn wound care and in topical and systemic antibiotics, sepsis still accounts for more than 50% of the mortality in burns.

Other Systemic Effects

Renal failure, as oliguric and non-oliguric failure, may occur as a complication of renal hypoperfusion, haemoglobinuria, myoglobinuria or septicaemia. It is associated with a high mortality even after dialytic therapy. There is progressive azotaemia, acidosis and

hyperkalaemia although the symptoms is non-oliguric failure are less prominent. After thermal injury, acute ulceration of the stomach or duodenum, known as Curling's ulcer, has been shown to occur in approximately 11% of the total burn population. Major bleeding episodes from this ulceration may require surgical intervention.

Management

Calculation of the area of skin with second or third degree burn is made in the adult by the "Rule of 9". Each arm carries 9% of body skin. Front and back of the trunk are 18% each, head 9%, legs 18% each and perineum 1%. At the time of admission body weight is measured or estimated.

1. Fluid Resuscitation

Intravenous fluid resuscitation is indicated in adults if the burn involved more than 20% TBSA or 15% with inhalation injury. There is disagreement regarding the quantity and choice of fluids to administer. Hypertonic salt solutions have been shown to achieve the same resuscitation with less volumes than isotonic saline, but serious side effects (eg, hypernatraemia, hyperosmolality, and intracellular dehydration) are possible. Colloids (as albumin or plasma) cause concern, because of the increase in vascular permeability induced by burns, with consequent extravasation of plasma proteins. However, more recent studies suggest that this increased vascular permeability is probably transient and significant only in the first 3-6 hours post burn. Hence, withholding colloidal proteins for too long after the injury could be detrimental, with possible decreased oncotic pressure, hypovolaemia, and peripheral and pulmonary oedema. Convincing, consistent clinical evidence of the best type of fluid to use are yet unavailable, but a mixture of Ringer's lactate with albumin may be superior. Consensus conference recommendations for supportive therapy in burn care are:

(a) In the initial 24 hours postburn, only balanced salt solutions (eg, Ringer's lactate) is given at a rate of 2-4 mL/kg/% burn, the higher volume for larger burns. Half of the estimated quantity is to be given in the first 8 hours.

(b) Colloids are administered as required after the initial 24 hours, when the capillary leak has most probably been sealed. Recent work (see above) tend to favour earlier use of colloids, eg, after the first 6 hours. Various formulae have been recommended (Table 1), but they serve only as quidelines, and the quantity and composition of the resuscitation fluids should be titrated against individual patient response. The use of glucose containing solutions is probably inadvisable, because of an early glucose intolerance in the immediate postburn period.

Sodium requirement is about 0.5 mmol/kg/% burn. However, frequent monitoring of patient response and biochemical investigations are necessary to adjust the fluid replacement regimen. Blood transfusions are indicated in patients with massive haemolysis or blood loss.

Haematocrit between 0.30-0.35 is optimal for oxygen carrying capacity. If haemolysis or muscle damage is significant, mannitol is given to produce a urine output of 1-2 mL/kg/h to facilitate elimination of nephrotoxic haem pigments.

Table 1. *Various Fluid Resuscitation Formulae in Adult Burns*

0-24 hours

Formula	**Crystalloids**	**Colloid**
Evans	Lactated Ringer's 1.0 mL/kg/% burn Dextrose water 2000 mL	1.0 ml/kg/% burn
Parkland	Lactated Ringer's 4.0 mL/kg/% burn	None
Brooke	Lactated Ringer's 1.5 mL/kg/% burn Dextrose water 2000 mL	0.5 ml/kg/% burn
Modified	Lactated Ringer's 2 mL/kg/% burn	None

24-48 hours

Formula	**Crystalloids**	**Colloid**
Evans	Lactated Ringer's 0.5 mL/kg/% burn Dextrose water 2000 mL	0.5 ml/kg/% burn
Parkland	None	20-60% of calculated plasma volume
Brooke	Lactated Ringer's 0.75 mL/kg/% burn Dextrose water 2000 mL	0.25 ml/kg/% burn
Modified burn.	None	0.3-0.5 mL/kg/%

2. Respiration

Careful assessment of patency of the upper airway with a high index of suspicion is vital. Upper airway obstruction may develop suddenly and catastrophically. If any doubt exists, the airway should be secured immediately with an endotracheal tube. Tracheostomy is associated with high mortality and morbidity, but may occasionally be the only option. Intubation and ventilatory support, with or without positive end expiratory pressure are required if respiratory failure and/or the adult respiratory distress syndrome (ARDS) develops. Suxamethonium should be avoided in the first 60 postburn days, as severe hyperkalaemia may follow its administration. These patients are relatively insensitive to non-depolarizing drugs and may require large doses for paralysis. The lungs are particularly sensitive to barotrauma and may rupture easily with excessive inspiratory pressure.

Carbon monoxide poisoning should be suspected if patients show signs of mental disturbances. Oxygen should be administered immediately by a device capable of delivering

100%. (See Chapter 18, Oxygen Therapy.) This will decrease the half life of COHb from 4 hours on air to about 80 minutes. Blood COHb level can be measured by co-oximeter. Symptoms appearing at a COHb level of 15% include headache, nausea and angina pain in patients with ischaemic heart disease. At 25% COHb, ST depression on ECG may appear and sensorium is depressed. Arterial PO_2 may be normal during significant CO poisoning. A valuable diagnostic clue is the measured oxyhaemoglobin saturation being much lower than expected in relation to PaO_2. Cyanide poisoning is very dangerous, and unfortunately difficult to detect. Anticyanide chemotherapy includes sodium nitrate, sodium thiosulfate and hydroxycobalamine.

3. Electrolyte Disturbances

Electrolyte disturbances are commonly encountered following the initial resuscitation phase. Hypernatraemia is usually due to salt loading and inadequate replacement of insensible fluid losses. Salt restriction and dextrose in water will correct this abnormality. Hyponatraemia and hypokalaemia may have arisen from frequent bathing of burned patients in water rather than in an isotonic solution. Therapy with a mixture of sodium and potassium chlorides will restore the electrolyte concentrations. Hyperkalaemia may be severe in the acute phase due to tissue and red cell destruction. Dextrose and insulin may be required if hyperkalaemia is marked. Later renal losses of potassium may be high and potassium supplement with 80-200 mmol/day is then indicated. Hypocalcaemia is usually due to albumin depletion, and correction of hyponatraemia is often all that is required.

4. Acid Base Status

Acidosis develops quickly (within hours) after severe thermal injury exceeding 30% TBSA. It has both metabolic and respiratory components. The metabolic part arises from the products of heat-damaged tissues and relative hypoxia, while respiratory acidosis is most commonly due to inhalation of smoke. Prompt correction is desirable because acidosis reduces cardiac output, increases total systemic vascular resistance, decreases oxygen carrying capacity of haemoglobin, and reduces renal blood flow.

5. Nutritional Support

Assessment of injury and nutrition should be made within 48 hours of admission. Nutritional support should be commenced when resuscitative phase is complete, usually within 72 hours. The oral and nasogastric route is favoured. In the presence of impaired gastrointestinal function, total parenteral nutrition (TPN) is then indicated. There are many formulae to calculate caloric and protein requirement. The Curreri formula is simple and applicable, where daily energy requirements:

25 kcal (105 kJ)/kg + 40 kcal (168 kJ)/% TBSA.

There is little reason to increase caloric intake beyond that calculated for a 50% TBSA. A calorie-to-nitrogen ratio of 100:1 and amino-acid solutions rich in the branched-chain aminoacids have been recommended for facilitating anabolism. A daily protein regime of 1.5 to 2.5 g/kg is given. The energy requirement is provided by glucose and lipids. Insulin may be required to control hyperglycaemia. (See Chapter 82, Parenteral Nutrition.)

6. The Burn Wound

Early excision and grafting is widely accepted for the treatment of burns, because it minimizes infection and hastens wound healing. Excision is carried out only after fluid resuscitation and the patient's condition is stable, usually 24-36 hours post burn. However, mortality is high in patients with extremes of age and more than 60% body surface burn. Contributing factors include massive blood loss and hypothermia. The excised wound must be grafted or temporarily covered using human skin (ie, autograft, allograft), pigskin (xenograft), collagen sheet, or synthetic membranes.

Topical antimicrobial therapy after wound cleaning and the deroofing of blisters, may lower the rate of infection, but not by itself, the treatment of choice.

(a) *Silver sulphadiazine* (SSD) 1% is the agent most widely used. Dermal hypersensitivity reactions and transient leucopenia occur in up to 5% of patients.

(b) *Mafenide acetate* 10% is effective, but can produce pain on application, and may cause metabolic acidosis by carbonic anhydrase inhibition.

(c) *Silver nitrate solution* 0.5% is effective and safe but being hypotonic, may cause hyponatraemia. Methaemoglobinaemia is occasionally encountered.

7. Systemic Antimicrobial Therapy

Systemic antibiotics are valuable in burn patients when properly used. However, injudicious use may be harmful either through direct toxicity or by contributing to the emergence of resistant strains of micro-organisms. General guidelines and principles for systemic antibiotic use are:

(a) The burned patient, despite all efforts, will be exposed to micro-organisms, and no single agent or combination of agents can destroy all organisms.

(b) Treatment is indicated after identification of the responsible organism.

(c) Appropriate antimicrobial agents should be chosen.

(d) Once chosen, the antibiotic should be used for a long enough period to achieve clinical effect (usually 5-7 days), but not long enough to allow for emergence of opportunistic or resistant organisms.

(e) Dosage must be adjusted based on serum concentration, because the pathophysiological changes occurring in burn patients may have a profound effect on the pharmacokinetics of the drugs administered.

(f) Routine prophylactic administration of penicillin in the immediate postburn period is no longer recommended.

(g) In general, prophylactic systemic antibiotics are indicated in only a few clinical situations. These include immediate perioperative periods associated with excision and autografting, and possibly in the early phases of burns in children. Choice of agent is based on wound culture or prior knowledge of the bacterial colonization pattern in the burn unit. Gram positive coverage using first generation cephalosporin is commonly used in autografting.

(h) Constant review of the agents and route of administration is mandatory.

The penetration of systemic antibiotic into burn eschar is unreliable because this aspect is not yet fully studied. *Staphylococcus aureus,* Group A *Streptococci, Pseudomonas, Klebsiella,* and *Escherichia coli* are common wound pathogens. Early burn excision and removal of infection nidus, nutritional support, and avoidance of invasive monitoring are important factors to be observed in controlling clinical sepsis.

Use of immunomodulation therapy such as *Corynebacterium parvum* vaccine, fibronectin replacement therapy, cryoprecipitate infusion are currently under investigation.

8. Tetanus Toxoid

Tetanus toxoid is given on admission if indicated by immune status. Patients not previously immunized should receive 250 units of tetanus human immune globulin and the first of a series of active immunization with tetanus toxoid (See Chapter 45, Tetanus.)

9. Analgesia

A partial thickness burn is often very painful. Immersion or showering of the affected area with cool water reduces the extent of thermal damage and provides pain relief. A small dose of continuous intravenous infusion of pethidine or morphine titrated against the patient's response is the method of choice to relieve pain. Ketamine is a useful anaesthetic and analgesic agent for burn patients.

10. Renal Failure

Renal failure may require dialytic therapy. Early dialysis, using the criteria of a blood urea nitrogen in excess of 6.6 mmol/L or a plasma creatinine concentration exceeding 180 micromol/L, regardless of volume or quality of the urine has been advocated. Bacterial infection is a major hazard, and many dialysed patients die from the results of infection rather than renal failure.

Investigations and Monitoring

The following recommendations are modified according to the severity of the burn.

Cardiovascular Function

Monitoring should include measurement of pulse, arterial blood pressure, central venous pressure and continuous ECG. Pulmonary capillary wedge pressure and cardiac output

estimations are indicated only in difficult cases, as the complications of insertion of Swan-Ganz catheters are greater in the burn patient than in the critically ill, non-burn patient. Urine output and measurement of acid-base balance are useful in monitoring the perfusion.

Respiratory Function

This is assessed clinically with the aid of chest X-ray, pulse oximetry, arterial blood gases and carboxyhaemoglobin levels. The upper respiratory tract can be evaluated by fibreoptic bronchoscopy. Xenon 133 lung scanning is used to identify areas of air trapping caused by small airway obstructions. Pulmonary function tests may be useful in predicting the extent of damage.

Metabolic and Fluid Balance

These are monitored by daily weighing and strict fluid balance charting. A urinary catheter is indicated when burns in excess of 20% BSA are present. Daily serum electrolyte and osmolality estimations are indicated in the early post-burn stage. Blood sugar, serum proteins and parameters of hepatic and renal function, should be assessed as indicated. Haemoglobin, packed cell volume, white cell, platelet counts and clotting profile should be closely followed.

Bacteriological Studies

Bacteriological studies of wound swabs and tissue biopsies are performed with isolation, quantification, and determination of antibiotic sensitivity of colonizing organisms. Sputum and urine should be cultured regularly.

Tissue Status

Tissue status is constantly observed. Viability is estimated by clinical observation. Ultrasonic flow meters and xenon flow studies have been reported to be useful.

Prognosis

Serious burns constitute a devastating injury. Burns of more than 20% TBSA plus severe smoke inhalation are fatal in 50-80% of patients. Major contributing factors to the improved survival of burn patients are adequate nutritional support, control of sepsis and early skin grafting.