Part X: Trauma

Chapter 66: Management of Severe and Multiple Trauma

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Trauma can be defined as physical injury. It frequently leads to death, hospital admission, and use of ICU resources. Trauma is usually categorized as either blunt or penetrating. In Western countries, severe blunt trauma is common, and is most frequently caused by road crashes, falls, and less commonly, blows and assaults. Severe penetrating trauma, usually from gunshots and stabbings, is less common except in larger cities of USA. In some respects, blunt trauma is more difficult to manage than penetrating trauma. Assessment is harder, because injuries are frequently internal and not initially obvious. They are also often multiple.

Of the trauma admissions to hospital, only a minority have severe or multiple trauma. Their life-threatening injuries are, in order of frequency, to the head, abdomen, and chest. However, the hospital resources used by these patients are out of proportion to their numbers. Required services include resuscitation, imaging tests, laparotomy and major surgery, respiratory support and Intensive Care. Craniotomy and thoracotomy are needed less frequently.

Emergency Assessment and Setting Priorities

1. Triage

An important first step in the management of severe trauma is triage, ie, to sort patients with acute life-threatening injuries and complications, from those whose lives are not in danger. The severity of total body injury is related to the number of separate injuries present, and to the severity of the individual injuries. Assessment can be made either at the scene of injury, or on arrival at hospital. As in any emergency, assessment, diagnosis and treatment must be concurrent. There is no time for detailed histories, examinations, investigations, and well-considered diagnoses, before starting resuscitation and emergency care. Those with severe injury can usually be distinguished early by the presence of (a) a depressed level of consciousness, (b) abnormal breathing, or (c) signs of shock.

(a) *Coma* in the trauma patient can be related to brain injury, hypoxaemia, shock, alcohol, or other drugs which have been taken. Frequently, a combination of factors is present, and the degree of physical head trauma is not known precisely.

(b) *Breathing difficulties* are common in patients with trauma to the head, face, neck and chest. If rapid or distressed breathing is present, airway obstruction, laryngeal injury, pulmonary aspiration, and lung or chest wall injury (especially pneumothorax and lung contusion) should be considered.

(c) *Shock* always requires explanation and is almost always hypovolaemic. However, cardiogenic shock does occur in the trauma patient. The earliest, most constant and reliable signs of shock are seen in the peripheral circulation. A patient with cold, pale peripheries has

shock until proved otherwise. Hypotension is a later sign of shock, and tachycardia is not always present.

2. Priorities

A trauma patient often has multiple problems which require attention, and determining priorities is often not easy. In general terms, the priorities are:

(a) *Support life* - The patient is kept alive with whatever resuscitative techniques necessary, while various injuries and complications are attended to.

(b) *Locate and control bleeding* which may be external, internal or both.

(c) *Prevent brain stem compression and spinal cord damage.*

(d) *Diagnose, evaluate and treat* all other injuries and complications.

Basic Management Principles

A systematic approach to the management of severe and multiple trauma is important. A number of basic principles apply to the management of all patients with severe trauma:

1. Emergency Assessment

Before anything else, the following must be recognized and treated:

(a) *A - Airway obstruction* suggested by noisy (or silent) breathing with paradoxical respiration and respiratory distress.

(b) *B - Breathing difficulty* suggested by tachypnoea, mental confusion, cyanosis, and abnormal pattern of breathing.

(c) *C - Circulatory shock* manifested by cold peripheries with delayed capillary refill, low blood pressure, and rapid weak pulse.

2. Oxygen Therapy

High-flow oxygen by mask is given to any patient with fractured femur or a worse injury, because mild or moderate hypoxaemia is common. Patients with severe trauma frequently also require ventilatory support. A restless uncooperative patient may require anaesthesia with intubation and ventilation just to facilitate resuscitation.

3. Blood Cross-Match

Blood is cross-matched urgently. Six units should be requested in the first instance, instead of attempting to estimate the amount of blood loss beforehand. At the same time, blood is sent for baseline haematological, urea and electrolytes, and liver function tests.

4. Fluid Resuscitation

Resuscitation fluids are given. If necessary, 2 or 3 large 14 or 16-gauge intravenous cannulas are inserted in upper limb or neck veins.

5. Analgesia

Analgesia is often overlooked. Opioid drugs should be titrated intravenously, and never given intramuscularly or subcutaneously.

6. Urine Output

A urinary catheter is inserted, unless a ruptured urethra is suspected (because of blood at the urinary meatus or a severe fractured pelvis). Urine output monitoring is an important guide to resuscitation from shock.

7. Other Injuries

All injuries should be evaluated.

Evaluation of Injuries

Injuries are easily missed in an emergency, especially when one injury is obviously present. It is important to look systematically at all body regions, ie, head, face, neck, chest, abdomen, spine, pelvis, and extremities. The back of the patient, as well as the front should also be examined, and special attention is paid to regions with external lacerations, contusions, and abrasions.

1. *Head* - Neurological observations are made and the ears and nose are inspected for the presence of cerebrospinal fluid and blood.

2. *Face* - Bleeding into the airway should be excluded.

3. *Spine* - Signs of spinal cord injury should be looked for, ie, paralysis, diaphragmatic breathing, priapism, loss of vasomotor tone, and loss of anal tone. A cervical spine fracture or dislocation is assumed in all patients with head injury until proved otherwise.

4. *Chest* - Fractured ribs per se, are not usually important, but haemothorax, pneumothorax, lung contusion, and flail chest often require attention. Less common but very serious injuries occur to the heart and great vessels.

5. *Abdomen* - The spleen, liver, and mesenteries are often damaged. Retroperitoneal haemorrhage is common. Injuries to the pancreas, duodenum, and other viscera are less frequent, and are often missed until signs of peritonitis occurs. Renal injury with retroperitoneal haemorrhage is suggested by haematuria and loin pain.

6. *Pelvis* - Pelvic fractures may be difficult to detect clinically, especially in the unconscious patient. Blood loss may be massive, particularly with posterior fractures involving sacro-iliac dislocation. Ruptured bladder and ruptured urethra may be seen with anterior fractures.

7. *Extremities* - A litre of blood or more may be lost around a fractured femur. Long bone fractures are more serious if they are open, comminuted, or displaced, or if there is associated nerve or arterial damage.

8. *External* - Contusions nay be extensive and serious, especially in patients who jump or fall from high places, and may be overlooked if the back of the patient is not examined. Road crash victims may sustain serious burns.

Shock in the Trauma Patient

Cardiogenic Shock

If the trauma patient with shock has distended neck veins, possible injuries are cardiac tamponade, tension pneumothorax, myocardial contusion, and concurrent myocardial infarction.

Hypovolaemic Shock

If the neck veins are empty, hypovolaemic shock should be inferred. There are 5 possible sites of blood loss causing shock. Bleeding can be from one or more of these sites:

1. *External* - which is obvious from blood soaked clothing and the ambulance trolley.

2. *Major fractures* - which are obvious clinically (ie, femurs) or seen on a plain X-ray (ie, pelvis).

3. *Chest* - The chest X-ray will detect blood in the chest, and intrapleural drains will reveal the amount and rate of blood loss. The chest X-ray will also show signs of ruptured aorta, pneumo-haemothorax, lung contusion, and rib fractures.

4. *Peritoneal cavity* - as diagnosed by laparotomy, diagnostic peritoneal lavage or CT scanning.

5. *Retroperitoneum* - inferred, when all of the above are negative.

Diagnostic Peritoneal Lavage

Peritoneal lavage should be used to diagnose intra-abdominal bleeding, using 1 L of isotonic saline, particularly when the patient is unconscious or has multiple injuries. Clinical examination can be grossly misleading in these two situations. Caution is needed with pregnancy, previous abdominal surgery, or massive pelvic injury. A positive result is frank blood on incising peritoneum, or the return of pink lavage fluid. If lavage is not followed by

laparotomy, a specimen of fluid should be sent to the laboratory for red and white cell counts and amylase level. Positive peritoneal lavages inevitably result in some laparotomies which do not reveal any intra-abdominal bleeding. However, in severe trauma, the additional morbidity of a negative laparotomy is negligible.

Fluid Resuscitation

Initial Fluids

The main fluid lost in trauma is blood, and almost all patients who are hypotensive or noticeably vasoconstricted, will need blood transfusion. However, since cross-matched blood is not immediately available, other fluids are used first. Uncross-matched, group O Rh negative blood is occasionally indicated in the patient who is exsanguinating, but in general, it is wasteful of blood products to infuse large quantities while bleeding is uncontrolled.

The first intravenous fluid given to a trauma patient should be isotonic saline or a balanced salt solution. Patients with shock may need 2-3 L in the first few minutes. One litre bags or bottles and giving sets with in-line pumps should be used on all IV lines. A colloid plasma expander can be the second fluid used, and by 20-30 minutes, cross-matched blood should be available. Freeze dried plasma should be reserved for massive transfusion or suspected coagulopathy. In these situations platelets may also be needed.

All resuscitation fluids have a high sodium concentration similar to that of extracellular fluid. Few trauma patients require any other type of fluid in the first day. It is not possible to have a resuscitation fluid which is low in sodium. Glucose 5% and glucose-saline solutions are not effective resuscitation fluids.

Urine Output

Hourly urine output is a useful guide to resuscitation from shock. The absolute minimal acceptable urine output is 0.5 mL/kg/h, but 1-2 mL/kg/h is more adequate. Frusemide has no place in initial resuscitation.

Inadequate Resuscitation

Patients in shock have depletion of interstitial fluid and need resuscitation fluid volumes greater than the actual volume of blood lost. With blunt injury, volume losses often continue for 24-48 hours. Prolonged shock from delayed and inadequate resuscitation and inappropriate fluids, leads to oliguric and non-oliguric renal failure, adult respiratory distress syndrome (ARDS), sepsis, and multisystem failure.

Pulmonary Oedema

Pulmonary oedema during trauma resuscitation is not usually related to fluid overload. Direct lung trauma, pulmonary aspiration of gastric contents, pulmonary responses to non-thoracic trauma, and reactions to resuscitation fluids can all cause "leaky" capillaries and produce non-cardiogenic pulmonary oedema.

Head Trauma

Serious injuries to the head are common, although those requiring an urgent cranial operation, less so. Their assessment and management can be difficult especially when other injuries are present. Head injury is frequently only one part of multiple trauma. Although the head injury may initially be the most obvious injury, it may not be the most important.

Emergency Treatment

Patients with airway obstruction or inadequate airway protection should be immediately intubated and hyperventilated under anaesthesia, to ensure optimal cerebral oxygenation and blood flow, until full evaluation of cerebral status is possible. Those with one or two unreactive pupils should be given mannitol 1 g/kg IV in an attempt to relieve brainstem compression, until definitive diagnosis and treatment can be arranged.

Shock cannot be attributed to brain injury unless brain death has occurred or is imminent. Shocked patients with head injury require the same fluid resuscitation as those without head injury. Management of shock and maintenance of cerebral perfusion is a vital part of managing head injuries. Contrary to common belief, sodium containing fluids are not inherently dangerous in head trauma. However, oncre resuscitation is complete, further sodium administration is inappropriate. On the other hand, excessive water administration is also inappropriate. Free water is potentially dangerous as it can lead to hyponatraemia, hypo-osmolality, and brain swelling. After initial resuscitation, a patient with head trauma may need less than 500 mL of water to maintain normal serum biochemistry.

Neurological Evaluation

Factors such as hypoxaemia, shock, alcohol and other drugs all depress consciousness and worsen neurological signs. Analgesia and anaesthetic drugs and muscle relaxants also interfere with neurological assessment, but are often essential for effective resuscitation. Clinical neurological information to be recorded, if possible, on all trauma patients include the following clinical observations for the Glasgow Coma Scale:

1. Does the patient obey a simple command?

2. Does the patient open his eyes?

3. The vocal responses, ie, whether uses conversation, words, grunts and moans, or remains silent.

4. The motor responses of each limb, ie, whether localizing, flexion, extension, or no movement.

5. Spontaneous eye movements and position.

6. Pupillary responses.

The above information, and changes with time, will enable management decisions to be made. A deteriorating level of consciousness, or the presence of lateralizing motor or pupillary signs, are indications for CT scanning if available, or for emergency burr holes. CT scanning is indicated in all patients who are unresponsive to vocal command, especially if rendered neurologically "inaccessible" by sedative and relaxant drugs.

Radiology for Trauma Patients

Patients with depressed consciousness, breathing difficulties, or unstable circulation, should not be sent to a remote Radiology Department, away from skilled supervision and facilities. They need X-rays where they can be cared for at the same time. Similarly, extensive radiography of distressed shocked patients in the Emergency Department is unacceptable. Less urgent imaging examinations should be performed in the Operating Room or the ICU. Only 5 examinations should be requested as portable procedures in the Emergency Department:

1. *Chest* - This is the only X-ray ever justified in an unresuscitated patient. If a pneumothorax is obviously clinically present, it is unnecessary to await a chest X-ray before insertion of an intrapleural drain. A supine film is usually most practical in the first instance, although an erect film gives more information. An erect film is a better examination for showing intrapleular air or fluid, ruptured diaphragm, and for defining an abnormal mediastinum.

2. *Lateral cervical spine* - This should be done in all patients with head injury or multiple injuries, as cervical spine fractures are often missed. In a patient with head or facial injuries, a cervical fracture should be assumed initially, and a cervical collar applied. A lateral cervical spine X-ray is taken later, when the patient has been resuscitated.

3. *Pelvis* - A pelvic fracture which is not clinically obvious can be the site of unexplained blood loss. A dislocated hip can be missed in a patient with multiple injuries, especially if unconscious.

4. *"One shot" intravenous urogram (IVU)* - In suspected renal trauma, this is a useful procedure before laparotomy. It often avoids the need for a lengthy investigation in the Radiology Department.

5. *Skull* - Plain skull X-rays do not often guide immediate management unless there is a depressed skull fracture present. A CT scan is more useful.

Other X-rays should be deferred until after adequate resuscitation, to be performed in the Radiology Department or the ICU.

1. *Extremities* - X-rays of the extremities for assessing orthopaedic injuries are not urgent unless there is vascular injury. Fractures of the extremities are usually obvious. These films should, therefore, not be taken in the Emergency Department unless the patient is going directly to the Operating Room.

2. *Spine* - X-rays of thoracic or lumbo-sacral spine are also seldom indicated in the Emergency Department. Clinical examination is more important.

3. *Abdomen* - A plain abdominal X-ray is of limited value in the initial evaluation of trauma. Abdominal CT is valuable in evaluation of the patient who is haemodynamically stable.

4. *Aortography* - If aortic rupture is suspected either from the nature of the injury, symptoms and signs, or chest X-ray, the radiologist responsible for aortography should be consulted immediately. In general, diagnosis of ruptured aorta takes priority over all other injuries, except in the patient who needs immediate laparotomy or craniotomy on clinical grounds. This approach is a calculated risk because the incidence of positive aortography is low.

5. *Interventional Radiology* - Percutaneous transcatheter embolization can be a life-saving haemostatic procedure in massive retroperitoneal haemorrhage associated with pelvic fracture. However, the logistics of caring for such haemodynamically unstable patients in the Radiology Department are formidable.

Severity and Morbidity of Trauma

An important development in trauma care has been systemic grading of the severity of injury. This is the Abbreviated Injury Scale (AIS), which can provide a basis for research, audit and allocation of resources. In concept, the AIS divides the body into 6 regions: heand and neck, face, thorax, abdomen, pelvis and extremities, and external. Criteria are laid down to grade specific injuries, and the severity within each body region is graded from 1-5. The AIS works best for blunt trauma, and is specifically designed for motor vehicle accidents. Multiple injuries are catered for by the Injury Severity Score (ISS), which is an empirical system based on the AIS grades for the various body regions. It gives a score between 0-75 for total body injury. Total severity of trauma is related not just to the severity of individual injuries, but also to the combined effects of multiple injuries.

Severity of injury measured by ISS corresponds with the need for therapeutic modalities like ventilatory support, duration of stay in ICU, and with mortality. An ISS of 16 or more is indicative of major trauma. Death with an ISS of 24 or less ir rare, and is usually related to management error. Mortality rises abruptly with a scale of 25 and over, mostly from the severe head injuries. Mortality is even higher at a score of 30 or more, particularly from exsanguination, but also from complications of respiratory failure and sepsis. With a rating of 50 or more, survival is not common, but is increasing, especially in young patients. There is very little margin for management error with injury of this severity.

AIS and ISS, unfortunately study only the anatomy of injury. Obviously other factors influence trauma mortality and morbidity. These include age, pre-existing health, pre-hospital and early hospital care, and complications. The degree and duration of shock is particularly important, proportionate with the probability of complications. Complications from trauma include aspiration, thromboembolism, renal failure, ARDS, sepsis, liver failure, and multi-organ failure.

In Western countries, trauma is a leading cause of death and disability under the age of 40, with the majority of deaths occurring at the scene of injury. Reduction of mortality depends on public education, on-site advanced care, rapid evacuation, trauma expertise (ie, trauma centres), and coordination of services.

Chapter 67: Severe Head Injuries

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Head injuries are a major medical and social problem in developed countries. Neurotrauma is the leading cause of mortality and morbidity in the 15-24 year age group. The death rate per 100,000 from neurotrauma in New South Wales in 1977 was 28, and in South Australia, 25. Statistics are often difficult to compare, because of differences in definition, coding, and data collection. Mortality from head injuries in the USA in the late 1970s was estimated to be 22-25 per 100,000, while in Britain it was 9 per 100,000. Motor vehicle accidents are responsible for approximately 60% of the deaths due to head injuries.

Hospital admission rates for head injuries in the USA and Britain are approximately 200-300 per 100,000 (ie, over 400,000 admissions in the USA per year). Most of these are admitted only for observation over 24-48 hours.

In general, only patients with severe head injuries, or who have associated multiple trauma or medical complications are admitted to the ICU. Severe head injuries form approximately 9-11% of the total head injury population. This is the group of hospital patients exposed to the highest mortality and morbidity. Aggressive ICU treatment has been shown to improve the outcome of severe head injuries, without increasing the number of severely disabled or vegetative survivors.

Initial Assessment

The severely injured patient must have a rapid but complete initial assessment. General measures are instituted to reduce the incidence of secondary insults, particularly hypoxia, hypercarbia and hypotension. These include:

1. Establishing Airway and Ventilation

Intubation and continuous mechanical ventilation (CMV) are required if ventilation or gas exchange are inadequate, and/or if the patient is incapable of protecting his airway. About 30% of severe head injuries, particularly those associated with multiple trauma, are hypoxaemic and should be intubated promptly. Adequate ventilation should produce a $PaCO_2$ of 25-30 mmHg (3.3-4.0 kPa) and PaO_2 greater than 70 mmHg (9.3 kPa). Until proven otherwise with cervical X-rays, these patients should be considered to have a cervical spine injury and handled appropriately.

2. Treatment of Shock

Hypovolaemia is a common finding in multiple trauma. However, shock is uncommon in isolated head injuries, except those involving young children, medullary injuries or large scalp lacerations. Prompt and effective resuscitation is required.

3. Clinical Examination

A full examination helps to determine priorities of treatment and ascertain other injuries.

4. Complete Neurological Examination

(a) *Conscious state* - The Glasgow Coma Scale (GCS) is advocated for all head injuries and has some prognostic significance by itself and in conjunction with other clinical and laboratory findings. It is useful for comparing treatment regimens from different centres, but is not a complete neurological assessment. Drug or alcohol intoxication will make assessment of conscious state difficult. Unfortunately, intoxicated patients often have an associated head injury.

(b) *Pupils* - The pupil size and reactivity are especially important. Abnormalities indicate compression and compromise of the third cranial nerve. This may help to localize supratentorial lesions. Signs of third nerve compression, depression of conscious state plus asymmetrical motor responses, are the triad of signs for transtentorial herniation. Extraocular movements (doll's eyes and reponse to aural caloric testing) are also important in the assessment of the mid brain and pons. The doll's eye manouevre should not be performed until fractures of the cervical spine have been excluded. Papilloedema is uncommon in the acute phase of head injuries.

(c) *Motor function* - Evidence of motor function, especially decerebrate rigidity, hemiplegia or other localizing signs are sought.

(d) *Other assessment* - These include the gag and cough reflex, cardiac status, particularly arrhythmias, ventilatory patterns, and examination of the remaining cranial nerves.

If the patient is repsonsive to commands or questions then a more detailed neurological assessment must be performed.

5. Further History

This should be obtained with particular reference to the circumstances of the injury and retrieval, seizures, intoxications, pre-existing medical problems and medications.

Patients with severe head injuries should be seen early by a neurosurgeon. Those with a GCS less than 8 or with progressive deterioration of neurological status, should be seen immediately. Early CT scanning should be performed. Intracranial mass lesions occur in only 6-7% of total head injury admissions, but in 40-60% of severe head injuries.

Further Diagnostic Measures

1. Computerised Tomography (CT)

The CT scan is the procedure of choice to determine the presence or absence of mass lesions. It will also indicate areas of oedema, infarction, contusion, intracranial air, and size of the ventricular system. In addition, the CT scan may help to decide on intracranial pressure (ICP) monitoring and may be of some prognostic value. It has replaced angiography, ventriculography and the exploratory burr hole in the emergency assessment and management of head injuries. If time permits and there are indications, views of the cervical spines are also obtained.

It is important to plan for all the logistical problems involved in moving an acutely injured patient to the CT scanner. The patient is required to lie completely still during the scan, otherwise an inadequate scan will result. Early generation scanners took approximately 20 minutes for a complete scan but newer scanners are much faster. Thus, many patients, if not already intubated at this stage, will require intubation and anaesthesia for the CT scan.

All routine preparations should be made for emergency neuroanaesthesia in cases with a potential intracranial collection. If such a collection shows on CT, the patient is kep anaesthetized and ventilated, prior to transfer to the operating theatre. Should the patient's neurological status deteriorate rapidly, a single cut CT scan through the mid-ventricular region may provide sufficient information for surgery. Such a patient should also receive mannitol in an IV dose of 0.5-1 g/kg before surgery.

2. Skull X-Rays

The presence of a fracture increases the likelihood of complications (ie, intracranial haematoma), and may help to localize an extradural haemorrhage. The position of a calcified pineal gland, if seen, may indicate shift of intracranial contents relative to the midline. Fractures of the base of the skull may be seen on X-ray without prior clinical indication of their presence. Skull X-rays are also important in the assessment and future planning of reconstructive surgery. However, skull X-rays are not generally as helpful as CT scanning, and their routine use in the emergency evaluation of head injured patients has been questioned.

The importance of adequate X-rays, if a fracture of the cervical spine is suspected has been stated.

3. Cerebral Angiography

This is now rarely indicated when there is ready access to a CT scanner. However, if an isodense traumatic lesion is seen on the CT scan, the clinical condition is not consistent with the CT finding, or a vascular lesion is suspected, then cerebral angiography should be performed. In the absence of a CT scanner, cerebral angiography can be used in the diagnosis of intracranial haematoma.

4. Ventriculography

Ventriculography has been to a large extent superseded by CT scanning. It will provide information on the degree of midline shift and allow measurement of the intracranial pressure.

5. Echo Encephalography

This has been superseded by the CT scanner.

6. Radio Isotope Scan

This is of little benfit compared to a CT, but it does give some information on cerebral vascularity. Newer imaging techniques, ie, positron emission tomography (PET) will provide additional information on cerebral blood flow and neuronal function.

7. Magnetic Resonance Imaging (MRI)

MRI of head injuries may have some advantages over CT scanning in:

(a) diagnosing and estimating the size of extracerebral fluid (especially small) collections;

(b) distinguishing chronic subdural haematomas from hygromas;

(c) displaying non haemorrhagic contusions.

However, there are many logistical problems in supporting and monitoring injured patients during MRI, and CT scanning remains superior for diagnosis of acute parenchymal and subarachnoid haemorrhages. CT thus remains the procedure of choice for acute head injuries and MRI has yet to find its place, particularly for severe head injuries. It has been suggested that MRI may be of prognostic value in the management of mild and moderate head injuries.

8. Intracranial Pressure (ICP) Monitoring

Surgically amenable intracranial mass lesions should be diagnosed and treated early in head injured patients. The continuous measurement of ICP is of great value, particularly in a patient who is comatose or on a ventilator, when assessment of neurological function is difficult. The value of ICP monitoring has been established. Prolonged levels of ICP over 25 mmHg (3.3 kPa) are associated with a very poor prognosis. It is reported that the outcome of head injuries may be improved if ICP over 15-20 mmHg (2.0-2.7 kPa) is treated with aggressive therapy. However, it has also been suggested that early evacuation of intracranial haematoma without ICP measurement can produce comparable results.

9. Recordings of Cerebral Activity, ie, Multimodality Evoked Potentials (MEP) and the Electroencephalogram (EEG)

These may be useful after the initial stabilization of the patient. They can aid in the monitoring of the patient's clinical course and specific neurological function.

Management

The aims of ICU management for head injuries are:

1. Early detection of changes in neurological status through constant observation and monitoring.

2. Prevention of secondary cerebral insults, especially those related to hyponatraemia, hypotension, hypoxaemia, hypercarbia and raised intracranial pressure.

3. Early diagnosis and treatment of medical and surgical problems, particularly intracranial mass lesions, cerebral oedema and epilepsy, which may be intercurrent or in the process of developing.

The main principles of management are as follows:

1. Constant Observation

Nursing observations as per GCS is extremely important. If deterioration occurs the cause must be sought.

2. Patient Position

If possible, the patient should be nursed in the head-up position (approximately 30-45°) with the head in a neutral plane relative to the body, in order to facilitate ventilation and reduce ICP.

3. Respiratory Care

Hypoxia, hypercarbia or respiratory obstruction must be avoided. The inspired oxygen should be adjusted to maintain a PaO_2 greater than 70 mmHg (9.3 kPa) and the ventilation adjusted to produce a $PaCO_2$ of 25-30 mmHg (3.3-4.0 kPa). Endotracheal suction and physiotherapy will increase the ICP, and that such manoeuvres should be preceded by adequate sedation and analgesia.

4. Blood Pressure Control

Control of the blood pressure (BP) to keep it within its normal limits (ie, a systolic BP of 100-160 mmHg (13.3-21.3 kPa). There are varying degrees of loss of autoregulation following head injuries. It is therefore important to prevent BP being in the ranges where the cerebral blood flow is pressure dependent.

In acute severe head injuries, vascular factors probably account for a greater proportion of the increase in ICP than cerebrospinal fluid (CSF) factors.

5. Surgery

Operative treatment is indicated for complications such as intracranial mass lesions, hydrocephalus and depressed skull fractures.

6. Treatment of Raised Intracranial Pressure

If intracranial mass lesions and hydrocephalus are excluded, then raised ICP in head injuries is either due to cerebral vasodilation, cerebral oedema or varying combinations of the two. In the acute stages, the vascular dilatation may be more important in the genesis of raised ICP, whereas in the later stages cerebral oedema may become more important. To date, it has been clinically difficult to separate these two and hence varying combinations of the following are used:

(a) *Controlled Ventilation*

It is well accepted that reducing the arterial $PaCO_2$ will result in a reduction in ICP. Hyperventilation is used extensively for this purpose. The arterial $PaCO_2$ should be maintained around 30 mmHg (4.0 kPa). In addition to reducing the arterial $PaCO_2$, controlled ventilation will facilitate optimal airway management and oxygenation. Duration of ventilation is generally 48-72 hours in the first instance, followed by an attempt to wean the patient off the ventilator provided ICP is controlled. If ICP rises during weaning, then ventilation is continued for a further 24-48 hours. Increases in ICP during controlled ventilation necessitate the checking of arterial blood gases, re-assessment of ventilation and CT scanning.

(b) *Osmotic Diuretics*

If the blood-brain barrier is intact, osmotic diuretics such as mannitol and urea will lower ICP by drawing fluid across the blood-brain barrier (thus reducing the bulk of the normal brain). Mannitol is generally used, as there is less rebound with mannitol than urea. If a patient deteriorates rapidly in the acute stage of a head injury, mannitol in a dose of 0.3-1.0 g/kg is used. The subsequent dose of mannitol is 0.25-0.5 g/kg every 6 hours. The osmotic diuresis should not be pursued at the expense of cardiovascular stability. If a diuresis does not occur, mannitol should not be continued.

Serum osmolality is used as a guide to mannitol therapy. It should not rise above 310 mosm/kg as mannitol itself will enter the brain and interfere with the efficacy of the dehydration therapy. If the serum osmolality exceeds 350 mosm/kg, serious cellular damage may occur. Treatment with mannitol is continued for only 24-48 hours, as eventually, mannitol will cross into the brain and cause an increase in brain volume, ie, "rebound phenomenon". In one study, empirical mannitol therapy without ICP monitoring produced similar results to mannitol given for ICP elevation greater than 25 mmHg (3.3 kPa).

(c) *Steroids*

The value of steroids to treat cerebral oedema associated with intracranial tumours is well documented and accepted, but remains unproven for cerebral oedema associated with head injuries. Saul et al have suggested that there may be a subgroup of head injured patients, who are early responders to overall treatment, whose outcome may be improved by steroids. However, several prospective double-blind studies have indicated that steroids do not significantly alter morbidity, mortality or ICP. As a result, the use of steroids in the management of head injuries has declined markedly.

(d) *Diuretics*

The reduction in brain oedema with frusemide may be due to mechanisms other than the diuresis per se. Use of both dexamethasone and diuretics has been shown to produce a greater reduction of brain oedema than the use of either agent alone. Frusemide is the diuretic of choice in patients with congestive heart failure plus cerebral oedema, and may produce less marked changes in serum electrolytes and osmolality than mannitol. Experimentally, frusemide can act synergistically with mannitol, thereby sustaining the osmotic gradient established with mannitol. Once-diuretic therapy (albumin plus frusemide) has similar cerebral effects to mannitol or frusemide.

(e) *Cerebral Metabolic Depression*

Treatment aims to reduce cerebral metabolic demand in tandem with reducing cerebral blood flow, thus preserving neuronal function. The agents generally used are the barbiturates, althesin (no longer available) or lignocaine. These agents may be used in patients with raised ICP, who are not responding to the above treatment, and who do not have surgically correctible mass lesion on repeat CT scanning. High doses of these agents are often required, and extensive cardiovascular monitoring and support are required together with ICP monitoring.

Barbiturate therapy is generally commenced at an ICP over 20-25 mmHg (2.7-3.3 kPa) with a closed skull, and over 15 mmHg (2.0 kPa) with a craniectomy. It reduces ICP, however, Ward failed to show that prophylactic pentobarbital improved outcome. Eisenberg recently reported that while high-dose barbiturates are only indicated in a small subset of patients, barbiturates are an effective adjuvant to "conventional therapy" for the control of ICP. In addition, there was a marked difference in the one-month survival between responders and non-responders to therapy directed at ICP control (ie, "conventional ± pentobarbital).

7. Fluid Balance

Following initial resusctiation and stabilization, strict countrol of fluid balance will help control cerebral oedema. However, fluid restriction should not be pursued at the expense of cardiovascular stability or renal function.

8. Electrolytes

Electrolyte disturbances are frequently seen in patients with head injuries as a result of the head injury, stress responses, osmotic diuresis, diabetes insipidus, fluid restriction, feeding regimens and medications. Regular monitoring of electrolytes, urea, creatinine, blood sugar and osmolalities are important in determining fluid and electrolyte therapy. It is important to prevent hyponatraemia and water overload.

9. Physiotherapy

Physiotherapy is important to remove lung secretions, prevent contractures, and in rehabilitation. Adequate sedation and blood pressure control prior to chest physiotherapy are required in order to prevent ICP elevation.

10. Antibiotics

These are used if there is a base of skull or compound fracture, or a fracture into a sinus. Antibiotics may also be used prophylactically following the insertion of an intracranial pressure monitoring device.

11. Treatment of Epilepsy

Epileptic seizures will markedly increase cerebral metabolic demands, and hence it appears logical to minimize the incidence of seizures. The routine use of phenytoin has been recommended for postoperative neurosurgical patients, including head injuries. However, the efficacy of prophylactic phenytoin to prevent early post traumatic seizures has yet to be firmly established. Acute seizures should be treated with a barbiturate or a benzodiazepine, and phenytoin commenced for longer term therapy.

12. Prophylaxis Against Gastric Ulceration

Gastroduodenal lesions, particularly erosive gastritis, are frequently seen on endoscopy in patients with severe head injuries, but significant haemorrhage only occurs in 10-14% of cases. Prophylaxis using antacids ± H2 receptor blockers should be considered pending the introduction of enteral feeding.

13. Feeding

Severe head injuries demonstrate markedly increased energy requirements, a negative nitrogen balance, weight loss and hypoalbuminaemia. Clifton recommended early enteral feeding while others have suggested that early parenteral feeding will improve outcome.

14. Other Additional Treatment

Other aspects of the management of head injuries deserve comment:

(a) *Temperature control* - Fever increases the metabolic demands of the brain and thus may exacerbate neuronal injury. It is therefore important to determine the cause and to treat

appropriately. Although hypothermia has been shown experimentally to be protective to the brain in head injury and cerebral oedema, it has not become routine therapy.

(b) *Syndrome of inappropriate ADH* - This syndrome may be seen following a head injury and is managed as described in Chapter 79, Fluid and Electrolyte Therapy.

(c) *Diabetes insipidus* - may also follow a head injury.

(d) *Coagulopathy* - Coagulopathies are not uncommon in patients with severe head injuries, and must be looked for and treated promptly in order to reduce the occurrence of intracranial haemorrhage.

Prognosis of Head Injuries

A number of factors are important including the age of the patient, time-lag between injury and treatment, type of injury, Glasgow Coma Scale and severity of neurological deficit, plus the occurrence of complications, particularly hypoxaemia and hypotension. In general, poor motor function indicates a poor outcome, especially in the older age groups. Patients under 30 years of age have a better prognosis than those older with the same degree of head injury. However, it is important to avoid making a rash prognostic decision too early, as many head injured patients, particularly the very young, show a remarkable improvement with time.

Brain death, which is an indication for cessation of all active treatment is described in Chapter 42, Brain Stem Death.

Chapter 68: Faciomaxillary and Upper Airway Injuries

P G Moore

Faciomaxillary and upper airway injuries are common and pose problems in airway management. In severe cases, there are often associated injuries to the cranial fossae and brain, cervical spine, skeleton and chest. Management often involves many specialty disciplines (ie, otolaryngologists, oral surgeons and dentists, plastic surgeons, ophthalmologists, neurosurgeons, anaesthetists and trauma surgeons). Fragmented care is to be avoided and the intensivist may play an important role in coordinating the various services.

Mechanisms of Injury

Faciomaxillary and upper airway trauma are due to sharp or blunt injuries to the head or neck. Sharp injuries usually result in lacerations and penetrating injuries, whilst blunt injuries result in fractures to the facial skeleton. Over 50% of facial trauma are the result of motor vehicle accidents, most of the remainder are due to physical violence or sporting injuries, which tend to be less severe, and a small number occur as falls and work-related accidents. The severity of facial fractures are directly related to the degree of force applied and the velocity of injury. Over 50% of severe faciomaxillary injury are accompanied by other associated injuries.

Penetrating neck wounds are commonly due to knife and gunshot wounds. They may result in injury to the air passages (ie, pharynx, larynx, trachea, and lung), nervous system (ie, spinal cord, brachial plexus, cranial nerves, or peripheral nerves), blood vessels (ie, aortic arch, innominate vessels, carotid, jugular and subclavian vessels) and the gastrointestinal tract (ie, pharynx and oesophagus). Blunt injuries to the neck are rare and are commonly due to motor vehicle accidents or physical violence. They cause damage to the supraglottic airspaces, larynx and trachea and may lead to severe airway problems.

The pattern of injury has changed in recent years. Passenger restraining devices and improved motor vehicle design (ie, windscreen glass, dashboards and steering columns) have reduced the incidence and severity of facial injuries, and the use of helmets has decreased mortality and the incidence of facial and neurological injury in motorcyclists.

Acute Management

The acute management of faciomaxillary and neck injuries focus on airway patency, which is the major priority. Once the problems of airway management have been addressed, management of other life-threatening injuries and trauma-related major system failure will follow. Thus, treatment priorities are to clear and secure the airway, control haemorrhage, treat hypovolaemia, and evaluate for associated life-threatening injuries. When these are satisfied, management is directed towards the facial, neck and other injuries.

1. Airway Management.

The airway must be assessed in the early triage, to exclude airway obstruction, rupture, or bleeding, and determine conscious level and presence of a full stomach and cervical, skull, or associated injuries. Simple measures to clear the airway by suction, posture and insertion of an oropharyngeal airway wil suffice in many cases. An oropharyngeal airway is best avoided in the conscious patient, as discomfort or vomiting may result. Nasopharyngeal airways should be avoided in all injuries to the middle third of the face or with basal skull fractures. The airway should be observed regularly in the acute phase of injury, as increasing oedema, swelling and haematoma may compromise a previously patent airway. Stridor, voice changes, dysphagia, dyspnoea, haemoptysis or surgical emphysema following blunt injury to the neck, may indicate serious injury to the larynx, pharyngeal airspaces, or extrathoracic trachea.

Immediate orotracheal intubation by a *skilled* person under direct vision is indicated in cases of:

(a) severe obstruction;

(b) respiratory depression; and when

(c) general anaesthesia is required to manage concomitant injuries.

Thus, foreign material can be removed and correct tube placement ascertained. When anatomical disruption makes intubation difficult or impossible, an emergency cricothyroidotomy may be life-saving.

Nasotracheal intubation should not be attempted when midfacial injuries are present, and is absolutely contraindicated when basal skull fractures are suspected. The use of a fibreoptic laryngoscope may be difficult for anatomical reasons, and because blood may obscure lens vision. Furthermore, suctioning through the fibreoptic instrument may be inadequate to remove secretions, blood and foreign material. Blind intubation techniques are contraindicated in the acute phase of injury, because a disrupted anatomy may be present. If intubation difficulties are anticipated, tracheostomy under local anaesthesia should be considered. Formal tracheostomy is best performed as a planned procedure in the operating room under local or general anaesthesia provided airway patency and protection can be maintained.

Soft tissue injuried and facial lacerations may mitigate against the use of an air-tight face mask for oxygen therapy or general anaesthesia. The presence of an associated cervical spine injury should be confirmed when possible, and appropriate measures (ie, a cervical collar or neck traction) taken to minimize movement if intubation or tracheostomy is planned. In some cases, cervical injury is suspected but not confirmed, and urgent airway intervention is required. Under these circumstances, while the airway takes priority, care should be taken to minimize neck movement when intubation is performed.

2. Control of Haemorrhage.

Haemorrhage from the midface or base of skull may occasionally be massive, and in severe cases, difficult to successfully control. Provided the airway is secured, the use of topical vasoconstrictors, nasopharyngeal packs, or a Foley balloon catheter inflated in the nasopharynx, may control or reduce blood loss. If bleeding persists, coagulation studies should be performed and appropriate replacement therapy given. Operative reduction of fractures and direct ligation of bleeding vessels may be attempted when simple measures fail to control bleeding. When these measures are unsuccessful, more radical measures, including ligation of the external carotid artery or intra-arterial embolization performed under angiographic control, should be considered.

3. Clinical Evaluation of Injuries.

A history of how the injury occurred should be taken from the patient, bystanders, police, or paramedics. Physical examination includes inspection for facial deformity or asymmetry, malocclusion of dentition, palpation of facial bones (including orbital margins), instability and movement of facial fragments, motor and sensory function, visual disturbances (ie, diplopia, limitation of eye movement, and loss of vision) and presence of cerebrospinal fluid (CSF) rhinorrhoea.

4. Specific Investigations.

Most facial fractures can be easily diagnosed with a minimum of X-ray studies. Useful studies include stereo Water's view, stereo Caldwell's view, postero-anterior, lateral oblique and Panorex views. Two-dimensional and 3-dimensional CT provide additional information about specific patterns of fracture, and may facilitate surgical care. CT may also be useful when laryngeal injury is suspected.

General Management

Patients without airway obstruction are more comfortably nurse in 30° head-up position, to encourage drainage of blood, saliva and CSF away from the airway, while preventing obstruction by disrupted tissue. Once airway patency is secured or confirmed, maxillary and mandibular fragments can be repositioned and a headwrap applied to maintain stabilization. Any other associated injuries are then assessed.

After life-threatening matters have been addressed, definitive reduction of faciomaxillary fractures can be planned. There is a "grace period" of up to 10 days for such surgery. While there is no strong evidence that early fixation may be beneficial, patient comfort might be best served by such an approach. In some cases, particularly orbital injuries when ocular function is at risk, early surgery is mandatory. When gross facial swelling occurs, definitive surgery should be delayed while measures are instituted to reduce swelling. These include debridement of open wounds, removal of foreign bodies, closure of facial lacerations, initial non-definitive stabilization measures, use of ice packs, and head-up nursing of the patient to reduce venous pressure and encourage fluid resorption. Prophylactic antibiotics should be used for patients with CSF rhinorrhoea, compound wounds and when operative fixation of fractures is performed.

Specific Injuries

Fractures

The most common fractures of facial bones are the nasal bones (37%), zygoma and zygomatic arch (15%), mandible (11%), orbital floor (11%), and maxilla (8%).

1. Mandibular fractures.

The mandible occupies a prominent facial position, and therefore is easily fractured. Multiple mandibular fractures are common, and common fracture sites are the condylar neck, angle of mandible, alveolar process, symphysis, and body. Coronoid process fractures are uncommon, as the bone is protected by the zygomatic arch. Ramus fractures are rare, because the strength of the bone in this area transmits impact forces to other areas of the mandible. Body of mandible fractures are oftena accompanied by fractures of the opposite angle or condylar neck, due to transmitted forces. Direct symphyseal impact may result in parasymphyseal or bilateral condylar neck fracture. High speed injuries often result in compound or comminuted fractures at the impact point.

Mandibular fragments are often distracted due to the action of the muscle of the lower jaw. Respiratory obstruction may occur following bilateral mandibular angle or body fractures (Andy Gump fractures) due to posterior displacement of the tongue. In emergencies, respiratory obstruction can be relieved by forward traction of the tongue or by placement of an oropharyngeal airway. Mandibular fractures are definitively managed by internal wiring or plating of fractures, and when teeth are present, by intermaxillary fixation by wiring of upper and lower jaws together, using upper and lower arch bars.

2. Maxillary Fractures.

LeFort described a classification of maxillary fractures in 1901 which is still used today, although fractures are usually of mixed types. Airway obstruction often accompanies LeFort injuries. The soft palate may sag against the tongue, posterior pharynx, or a haematoma or oedema in the pharyngeal wall. Foreign debris may be present in the mouth, and nasal obstruction may occur due to septal dislocation, swelling, or blood clots and foreign material.

Isolated maxillary fractures are rare, because the impact needed to cause fractures is usually sufficiently severe to break other facial bones. Mandibular fractures may be present in as many as 55% of cases of maxillary fracture, depending on the type of LeFort injury. Fractures of nasal bones, zygoma, orbit and skull (particularly base of skull fractures) and soft tissue injuries, including ocular injuries, are often associated with maxillary fractures.

(a) LeFort I Fracture.

This is the least severe fracture and occurs in about 30% of maxillary fractures. It is a dentoalveolar fracture which follows a horizontal plane above the floor of the nose. The fracture (sometimes called Guerin's fracture) separates the palate from the remainder of the facial skeleton, and is usually caused by direct low-maxillary blows or by a lateral blow to the maxilla.

(b) LeFort II Fracture.

LeFort II fractures, also referred to as pyramidal fracturesw, are the most common maxillary fractures (42%). They extend from the lower nasal bridge through the medial wall of the orbit, and cross the zygomatico-maxillary process. They are caused by direct blows to the mid-alveolar area, or by lateral impacts and inferior blows to the mandible when the mouth is open, and consist of a freely mobile pyramidal-shaped portion of the maxilla.

(c) LeFort Fractures.

These are known as craniofacial dysjunctions because the fractures completely separate the midfacial skeleton from the base of the cranium, resulting in the characteristic "dish-face" deformity. The fracture extends through the upper nasal bridge and most of the orbit and across the zygomatic arch. The fracture involves the ethmoid bone, and thus may affect the cribriform plate at the base of the skull. LeFort III fractures usually occur as a consequence of superiorly-directed blows to the nasal bones, and occur in about 28% of patients with maxillary fractures.

Associated basal skull fractures occur in about 20% of LeFort III and some LeFort II fractures, and may lead to CSF leakage, meningitis and pneumocranium. Nasal intubation may result in passage of the endotracheal tube through the cribriform plate and into the cranial cavity. The maxillary antrums are often opaque due to the presence of blood clot.

Despite the LeFort classification, maxillary fractures may often be a mixed variety. Similarly, facial fractures may be comminuted and may not be symmetrically distributed.

Nevertheless, comminuted fractures usually follow the LeFort fracture lines. LeFort II and III fractures involve the orbit and are frequently associated with orbital blowout fractures through which ocular muscles may herniate. Definitive surgery may involve internal fixation with wiring and plating, and intermaxillary fixation. Often, external fixation is required with intermaxillary fixation and cranial suspension to wire fixation at frontal bones.

3. Fractures of Zygoma and Orbit.

The malar region absorbs lateral and oblique blows to the mid-face. The zygoma is uncommonly fractures, but its attachments to the maxilla, frontal, and temporal bones are vulnerable and may be disrupted. When the zygoma is displaced, disruption of the lateral wall and floor of the orbit may ensue. The eye and its function must be carefully examined when fractures involving the orbit are suspected.

Isolated zygomatic arch fractures are often stable after operative reduction, and may require no other active management other than "protective" measures to ensure the area is not accidently bumped. Unstable and comminuted fractures require internal or external fixation. "Tripod" fracture of the zygoma require open reduction. Herniation of orbital contents and entrapment of ocular muscles must be relieved by distraction of the fractures which, in turn, are stabilized by wiring. Autogenous bone grafts and use of alloplastic materials may be required to reconstruct the orbital floor, if the fractures are severely comminuted and if there is bone loss. Orbital blowout fractures are managed in the same manner.

4. Nasal Fractures.

These are the most common fractures of the facial skeleton. Bleeding may be copious, particularly in patients with underlying hypertension or bleeding tendency. Vasoconstrictor agents, such as adrenaline, may be useful in controlling bleeding; in most cases the bleeding will settle. In some cases, nasal packing or inflation and traction of a Foley catheter balloon into the nasopharynx may be required. Closed reduction and external splinting is required to manage nasal fractures and must be performed within 10 days of injury.

Soft Tissue Injuries

Basic tenets of wound management apply to all soft tissue injuries to the face and neck. The region is anatomically complex, and contains many important structures, and extensive wound debridement should be avoided. The rich regional vascular supply, to some extent, protects against nutrient devitalization. Minimal debridement and delayed wound closure provides the best approach to management of the heavily contaminated wound. Where there is extensive tissue loss, once the wound is clean, myocutaneous or osteocutaneous grafts may be performed by microsurgery. Penetrating neck injuries, particularly those due to knife and gunshot wounds, may produce life-threatening exsanguination injuries and require careful evaluation and early surgical exploration.

1. Facial Nerve Injuries.

Any wound around the anatomical vicinity of the facial nerve must be carefully assessed to exclude facial nerve injury. A thorough neurological test of facial nerve function

should be performed in the conscious patient. However, because of extensive interneural connections between buccal and zygomatic branches of the facial nerve, a simple laceration of one branch may not produce any obvious clinical signs. Wound exploration should be carefully performed with the aid of a nerve stimulator before wound closure is performed.

Nerve lacerations medial to the orbit are not repaired due to extensive nerve arborization in these areas (before entering the facial musculature). Nerve lacerations lateral to the pupil should be repaired by a primary procedure before wound closure is performed. The amount of recovery of function, even in the best circumstances, is less than 50%. Dyskinesis is a frequent complication of repair, particularly when a major division of the nerve trunk is involved. Lacerations of marginal mandibular branch have poor recovery after repair. There are a number of procedures, including cross-facial nerve grafts and vascularized muscle transplants, which have been used with mixed results to improve outcome in long established facial palsy.

2. Parotid Injuries.

These injuries are characterized by a penetrating wound which lacerates the capsule and separates the parenchyma or parotid ducts. There is an intimate association between the parotid gland and Stensen's duct with the facial nerve, and an injury to one should draw suspicion to injury of the other. Simple lacerations to the parotid capsule are repaired by closure with absorbable suture. Minor collecting duct injuries require no specific surgical repair and any leakage of saliva soon ceases when the wound heals. The occasional formation of a sialocoele is resolved by serial aspirations. Major duct injury requires microsurgical reconstruction. With an injury to the extraparenchymal portion of Stensen's duct, it may be preferable to manage a parotid fistul to the oral cavity, and perform a delayed surgical repair weeks or months later.

3. Laryngeal Trauma.

Blunt or penetrating injuries to the larynx require immediate attention to the airway. If the airway is acutely compromised, an emergency tracheotomy or cricothyroidotomy may be preferable to blind or hasty intubation, which may misplace the endotracheal tube or extend the injury. Gaping wounds of the larynx can be intubated under direct vision, pending subsequent surgery. Simple contusions of the anterior neck may not cause serious laryngeal injury, and the patient can be managed by neck stabilization, head-up posture, and humidified oxygen therapy by face mask. Swallowing may be painful and may be accompanied by spasm or tracheal aspiration, and oral fluids or solids should not be given for 48 hours.

Blunt trauna to the larynx causing a compromised airway requires immediate attention, as laryngeal fracture or collapse is suspected. After the airway is secured by tracheostomy, a detailed examination including direct laryngoscopy should be performed. When the laryngeal skeleton is disrupted, surgical exploration and repair is indicated. Laryngotomy is required when fracture or disarticulation is present and definitive surgical repair can be performed. Post-traumatic fibrosis of the cricoarytenoid joint leading to impaired vocalization, can be minimized by careful surgical techniques. In some cases, particularly injuries to children, an internal stent may be required to minimise airway narrowing.

Following surgical repair, the tracheostomy is kept open for 7-10 days. After this time, the tracheostomy is plugged to test airway patency, and if satisfactory, the tracheostomy tube is removed. In extreme injuries, laryngeal function may not adequately recover and a permanent tracheostomy may ensue. In less severe cases, delayed reconstructive surgery should be attempted. Permanent voice alteration may remain as the minimum disability after laryngeal trauma.

Outcome

Management of the airway and other life-threatening injuries are priorities in the care of the traumatized patient. Mortality will be influenced by the care in the acute phase. However, appropriate and prompt definitive care of facial and neck injuries may significantly affect morbidity. In most cases, definitive treatment while the patient is receiving life support, may avoid complications which will significantly affect the patient's quality of life after recovery.

Chapter 69: Chest Injuries

G M Clarke

The commonest form of chest trauma in Australia is closed chest injury secondary to motor vehicle accident. Associated extrathoracic injuries, which themselves may be life-threatening, are often present.

If morbidity and mortality are to be minimized, swift assessment and resuscitation are carried out simultaneously. A team approach is necessary. The team leader must know his priorities as, in almost every case, respiratory and circulatory resuscitation will take precedence over everything else.

Initial management is directed toward detection and correction of life-threatening disorders. When these have been dealth with, a secondary assessment of the patient is made. It is during this secondary assessment that detailed radiological investigations are usually undertaken. However, the chest X-ray is an integral part of initial assessment and should be obtained as soon as possible.

Immediate Management

Obvious external bleeding is controlled. An intravenous cannula is inserted and basic circulatory resuscitation is initiated. Blood is sampled for cross-match, biochemistry and haematological tests. At the same time the respiratory and general measures listed in Table 1 are undertaken.

Table 1. *Immediate Management of Chest Trauma*

Assure patent airway, oxygenation and ventilation.
Exclude or treat:
- pneumothorax
- haemothorax
- cardiac tamponade

Assess for extrathoracic injuries.
Decompress stomach.
Provide pain relief.
Reconsider endotracheal intubation, ventilation.

1. Oxygenation

A clear airway must be assured. Oxygen is administered by face mask, and ventilation assessed. Immediate endotracheal intubation and controlled ventilation is indicated in compromised airways, severe head injuries, and gross hypoventilation and/or hypoxaemia unrelated to pneumothorax. Intubation and ventilation in the presence of tension pneumothorax carries the risk of a fatal outcome. Emergency cricothyroidotomy or tracheostomy is only rarely required when an upper respiratory tract obstruction cannot be bypassed by translaryngeal intubation. (See Chapter 20, Acute Upper Respiratory Tract Obstruction.)

2. Pneumothorax and Haemothorax

Pneumothorax and significant haemothorax are treated if present. A 12 or 14FG intravenous cannula may be inserted percutaneously to relieve tension pneumothorax in dire emergencies. Usually, however, there is time to insert a wide bore intercostal catheter under sterile conditions. A tube directed superiorly through the second anterior intercostal space will adequately drain a pneumothorax. Insertion through the mid-axillary line at the level of the nipple or above is recommended if a more lateral position is required. If a haemothorax is to be drained, the tube should then be directed posteriorly.

3. Cardiac Tamponade

Cardiac tamponade is suspected in any patient with thoracic trauma who exhibitis a low blood pressure and raised venous pressure. In this setting, the differential diagnoses are tension pneumothorax (the most likely) and severe heart failure (usually due to gross myocardial contusion, or prolonged and inadequately treated shock).

Emergency treatment of cardiac tamponade due to pericardial effusion is aspiration of the pericardial sac, preferably under continuous ECG control. The limb leads of the ECG are attached to the patient, and the chest lead connected to the metal hub of the 16FG aspirating needle by a sterile wire. The needle with plastic cannula is advanced towards the left shoulder at a 35 degree angle to the skin from a point 2 cm below the apex of an angle formed between the xiphoid process and the left 7th costal cartilage. Aspiration is made as the needle is slowly advanced. Remarkable improvement may follow the removal of as little as 30 mL of blood. Contact with the myocardium is denoted by ST elevation on the ECG or ectopic

beats. When a positive tap is obtained, the plastic cannula is left in situ for continued drainage. Subsequent thoracotomy and full exploration will usually be necessary.

In penetrating injuriesz with suspected tamponade, many centres emply prompt thoracotomy with pericardial decompression, bypassing attempts at aspiration.

4. Extrathoracic Injuries

In assessing extrathoracic trauma, head, neck and abdominal injuries, and significant concealed blood loss must be excluded. This initial rapid assessment should be made before potent analgesics are administered.

5. Gastric Decompression

Gastric distension with attendant risks of regurgitation, vomiting and aspiration, especially in patients with associated head injury, is extremely common in cases of severe chest trauma. The stomach should be decompressed by a nasogastric tube. If urgent endotracheal intubation is necessary, a rapid sequence ("crash") intubation is recommended, including the use of cricoid pressure.

6. Pain Relief

Pain relief is usually obtained at this early stage with IV narcotics. This will frequently relieve respiratory distress in patients with fractures of the ribs and/or sternum.

7. Reconsideration of Mechanical Ventilatory Support

After the initial management, mechanical ventilatory support should then be reconsidered. Major indications are listed in Table 2. Ventilation should also be considered for patients with borderline respiratory distress associated with:

(a) gross obesity;
(b) significant pre-existing lung disease;
(c) severe pulmonary contusion or aspiration; and
(d) severe abdominal injuries requiring surgery.

Table 2. *Major Indications for Endotracheal Intubation and Ventilation*

Dangerous hypoxaemia and/or hypercarbia.
Significant head injury.
Gross flail segment and contusion and respiratory distress.

Specific Thoracic Injuries

Specific thoracic injuries should be systematically excluded.

1. Ruptured Aorta

A widened mediastinum should always arouse suspicion of a ruptured aorta. In one series, a mediastinal width greater than 8 cm was present in all 10 patients with ruptured thoracic aorta. Suspicion of aorta rupture in the presence of an widened mediastinum is further heightened if associated with one or more of the following:

(a) left haemothorax;
(b) depressed left main bronchus;
(c) blurred outline of the arch or descending aorta;
(d) fractured 1st rib or left apical haematoma; and
(e) displacement of the mid oesophagus to the right (easily detected when a nasogastric tube is in situ).

Aortography should then be undertaken. The classic site of traumatic rupture of the aorta is at the junction of the mobile arch and fixed descending aorta. This is immediately beyond the origin of the left subclavian artery. Rupture at this site is attributed to forward movement of the mobile arch against the tethered descending aorta in a deceleration situation (eg, motor vehicle accident). In about 10% of cases, the tear is in the ascending aorta or near the origin of the other great vessels. These tears are usually due to direct trauma. Treatment is prompt surgery and often necessitates cardiopulmonary or left atrio-femoral bypass. These techniques or local shunmts do not necessarily protect against consequent paraplegia.

2. Ruptured Diaphragm

The usual cause of a ruptured diaphragm is gross abdominal compression, and the incidence may have risen since seat belts were made compulsory. Rupture of the left diaphragm is more common. A haemo-pneumothorax is commonly misdiagnosed when the dilated stomach gives a horizontal air-fluid interface on the erect chest X-ray. A ruptured diaphragm, as an isolated injury, is often surprisingly well tolerated by the patient. Nevertheless, with a left diaphragmatic rupture, there is significant risk of gut strangulation, and surgical repair should follow basic resuscitation.

Rupture of the right diaphragm is more difficult to diagnose due to the presence of the liver. The radiographic appearance is similar to a paralysed right diaphragm. In the absence of right sided rib fractures, a small pneumothorax together with a "high right diaphragm" is suggestive evidence.

3. Disruption of Major Airways

Although signs and symptoms may vary according to the level of the rupture, the clinical picture is frequently that of respiratory distress, subcutaneous emphysema and haemoptysis. A pneumothorax, which may be under tension, is invariably present in those with ruptured bronchus. Mediastinal emphysema is commonly seen on the chest X-ray. With tracheal injuries, immediate management involves endotracheal intubation beyond the tear to ensure an adequate airway, prevent aspiration of blood, and to abate the air leak. A pneumothorax if present, must be drained. Suction to the intercostal catheter may be necessary to keep the lung expanded. After this is achieved, bronchoscopy and early primary repair is

undertaken. Intubation with a double-lumen tube may be necessary if air leak from a disrupted bronchus is significant, to enable adequate ventilation of the patient as well as operative repair of the injury.

4. Massive Haemothorax

Immediate management involves insertion of a wide bore intercostal catheter and adequate resuscitation. Common causes include disruption of intercostal and/or internal mammary arteries. If the cause is massive bleeding from the aorta or major pulmonary arteries, the condition is usually fatal. Continued significant blood loss is an indication for early thoracotomy. Inadequate drainage of a haemothorax may require a thoracotomy and "decortication" at a later date. However, such decortication is rarely necessary.

5. Pulmonary Contusion

This is due to bruising of the lung and, as with any bruised tissue, becomes more oedematous over the following 48 hours. In the management, overhydration must be avoided. When associated with severe flail segments and respiratory distress, assisted ventilation is required, although this need not usually be prolonged.

6. Myocardial Contusion

Myocardial contusion is common in blunt chest trauma and may result in arrhythmias and cardiac failure. Both of these complications should be managed as in myocardial infarction. A standard 12 lead ECG may show a variety of abnormalities ranging from non-specific T wave changes to pathological Q waves. Abnormalities can also be demonstrated by myocardial nuclear scanning (not normally undertaken in the acutely injured patient). Serious damage to virtually every cardiac structure has, at some time, been reported. Cardiac injuries such as rupture of the ventricular free wall, interventricular septum, valvular apparatus, and disruption of major coronary arteries have usually been associated with penetrating injuries. However, many such complications have also been reported in non-penetrating chest trauma.

7. Systemic Air Embolism

This is more commonly seen in penetrating injuries and is immediately life-threatening. Though uncommon, it is probablyunderdiagnosed as it is unlikely to be proven at conventional autopsy. Air embolism is caused by a broncho-pulmonary vein fistula. It is suspected in the chest injured patient if:

(a) focal neurological signs exist in the absence of head injury;

(b) circulatory collapse immediately follows the institution of intermittent positive pressure ventilation (IPPV) in the absence of tension pneumothorax; and

(c) froth is obtained when arterial blood is sampled from a collapsed patient.

8. Oesophageal Perforation

Though usually due to penetrating injury, it can occur rarely with closed chest trauma. The patient may complain of retrosternal pain and difficulty in swallowing, and exhibit haematemesis and cervical emphysema. A chest X-ray may show mediastinal emphysema, widened mediastinum, pneumothorax, hydrothorax or hydropneumothorax. If suspected, a gastrografin swallow and/or endoscopy is performed. Treatment is immediate surgical repair. A gastrostomy and feeding jejunostomy are usually performed at the same time.

Management of a Flail Chest

The management of a flail chest remains unresolved. The concept of Pedulluft (the to and fro movement of air between the flail and non-affected sides of the thorax) has been shown to be incorrect. With a flail chest, overall ventilation may be reduced, but it is distributed to both lungs because the mediastinal shift equalizes the pleural pressures. Nevertheless, there is poor expansion in contused, low compliant lung areas, impairment of coughing and serious reduction in overall ventilation in gross cases. Moreover, gross mediastinal shifts may impair systemic circulation. While several basic approaches to managing a flail chest have emerged (Table 3), the ultimate choice of approach is determined by the severity of the chest injury, associated injuries and the method of pain relief.

Table 3. *Management of a Flail Segment*

Conservative
Assisted ventilation
- (a) Continuous positive airway pressure (CPAP)
- (b) Intermittent mandatory ventilation (IMV)

Controlled ventilation ± PEEP
- (a) Conventional IPPV
- (b) Independent lung ventilation

Surgical stabilization ± above measures.

1. Conservative Therapy

Conservative treatment involves oxygen by mask, adequate pain relief, and physiotherapy. It is the treatment required in a mild injury (ie, an isolated thoracic injury with fractured ribs, but without significant flail or disturbed blood gases). Similarly, it may be employed in the patients with a moderate injury (ie, a significant flail but with adequate blood gases and the ability to cough). "Prophylactic" ventilation in both of these groups has been deemed inappropriate, with possible disadvantages of barotrauma, infection, tracheostomy complications, and prolongation of hospitalization.

2. Mechanical Ventilatory Support

However, in a severe injury, ie, a gross flail segment with gross pulmonary contusion ± aspiration, and in a patient with associated head injury, endotracheal intubation and assisted ventilation is necessary. The early use of intermittent mandatory ventilation (IMV) has been

claimed to result in a shorter duration of assisted ventilation. As with conservative therapy, increased residual deformity may well be the price for unrestrained used of this technique. Continuous positive airway pressure (CPAP) alone has not been fully evaluated. Independent lung ventilation, a technique of selectively ventilating each lung separately, using a double lumen tube, may be used to treat a unilateral pulmonary contusion and/or flail. (See Chapter 22, Mechanical Ventilatory Support.)

3. Surgery

There is revived interest in surgical stabilization of the chest wall. Advantage claimed are either a shorter period of assisted ventilation being required or a shorter hospital stay. Internal surgical stabilization undoubtedly reduces deformity, and a stable chest wall will help a patient to cope with an underlying lung problem. However, except for a broken sternum, rupture of the diaphragm, and in the course of an otherwise necessary thoracotomy, the case for surgical repair has yet to be established.

Complications

Following resuscitation and initial management, complications may follow which usually require treatment.

1. Sputum Retention

Adequate pain relief is the major determinant of whether sputum retention will occur in the spontaneously breathing chest injured patient as efficient coughing must be maintained. Similarly, assisted ventilation may be avoided in many moderately injured cases if the method of pain relief is carefully selected and significant respiratory depression is avoided. Options (see also Chapter 48, Pain Relief in Intensive Care) include:

(a) intravenous narcotics given by frequent small dose intermittently or by continuous infusion;

(b) entonox inhalation during physiotherapy;

(c) intercostal nerve block either:

(i) multiple individual nerve blocks (repeated as necessary) or

(ii) single large volume (eg, 20 mL 0.5% bupivacaine) into one intercostap space (uni- or bilaterally), spreading to block nerves above and below the site injected.

(iii) intrapleural bupivacaine (0.25-0.5%) via uni- or bilaterally placed intercostal catheters. Epidural catheters have been used for this purpose. Either intermittent or continuous infusion of bupivacaine may be employed.

(d) conventional epidural analgesia using agents such as bupivacaine; and

(e) epidural or spinal opioids.

For ventilated patients adequate humidification and frequent endotracheal and endobronchial suctioning must be employed. Frequent change in position is important. In

ventilated patients, analgesic techniques producing respiratory depression are not a problem except during weaning or if IMV is used.

2. Bronchospasm

Bronchospasm suggests aspiration and is treated conventionally.

3. Tension Pneumothorax

The possibility of a late tension pneumothorax is ever present, especially if the patient is being ventilated with IPPV and positive end expiratory pressure (PEEP).

4. Acute Respiratory Failure

This is commonly seen in these patients. Causes include aspiration, pulmonary contusion, previous shock with delayed resuscitation, and fat embolism. If a classical adult respiratory distress syndrome (ARDS) occurs late after injury, then the most common cause is sepsis. However, humoral factors may play a role in the massively injured. Supportive treatment is instituted and, where possible, the underlying cause is treated. (See Chapter 25, Adult Respiratory Distress Syndrome.)

5. Infection

Sepsis remains a major cause of death in patients with severe chest and other injuries. The source of such infection is invariably endogenous, mainly coming from bacteria colonising the patient oropharynx and alimentary tract.

This has led to the use of parenteral antibiotic prophylaxis (eg, cefotaxime) active against community bacteria (eg, *S pneumoniae, H influenzae, B catarrhalis, S aureus,* or *E coli*) from the time of admission for 4 days. At the same time, oral and intragastric non-absorbable combinations of polymycin E, tobramycin and amphotericin B are administered, to prevent colonization and infection by *enterobacteria, pseudomonas* and fungi such as *candida.*

6. Thromboembolism

Preventative measures include frequent movement, full length leg stockings, avoidance of pressure on limbs, and low dose heparin (5000 units bd or tds) subcutaneously.

7. Inadequate Nutrition

Gastric atony and stasis are common. In many cases adequate enteral feeding is possible by appropriate posturing (eg, positioning on right side during feeding). In others, especially those with associated abdominal trauma, parenteral nutrition is necessary.

8. Coagulopathies

Prompt resuscitation, control of haemorrhage, and possibly, use of blood filters for massive blood transfusion help in this regard.

Prognosis

Reported mortality rates in chest injured patients vary greatly, often reflecting the severity of the chest injury and the extent of extrathoracic injuries. In one Australian series, of 1119 patients with chest and other injuries, the overall mortality rate was 5.3%. The 3 commonest causes of death were respiratory tract sepsis (35.6%), severe head injury (33.9%) and exsanguination (18.6%). Mortality rate was 37.5% for patients over 60 years of age who had respiratory failure, and for all age groups requiring mechanical ventilation, was 22.8%. Trunkey reported a 16% mortality in patients with isolated pulmonary contusion. When combined with a significant flail chest, the mortality rose to 42%.

Chapter 70: Spinal Injuries

M M Fisher

Introduction

The human spine is an engineering masterpiece designed to allow movement, maintain stability, and protect the spinal cord. Major trauma to the spine, especially when the underlying spinal cord is damaged, produces devastating injuries leading to economic, personal, and social tragedies. This situation is compounded by the development of paramedic services, which have lead to better survival of patients with high spinal injuries who in the past would not have reached hospital alive. The incidence of spinal injuries in Australia is 20-25 per million population per annum.

While significant advances have lead to improvements in both the rate and quality of survival of paraplegic and quadriplegic patients, the major thrust is in prevention and education - the use of appropriate safety helmets and car restraints; adequate preparation for contact sports, ie, football; and teaching of safety in water sports.

Aetiology

Motor vehicle (both car and motor cycle) trauma provide the major source of spinal injuries in Australia. The next commonest causes are football injuries and water accidents, especially those caused by diving into shallow water. Accidents related to hang gliding and ultralight aircrafts, falls and gunshot wounds comprise other common causes.

Spinal injuries are therefore likely to be associated with other injuries, particularly head and abdominal injuries, and aspiration of salt or fresh water. A careful check for other injuries should be mandatory in the patient with spinal injuries. Conversely, the multiple trauma patient should be assumed to have spinal injuries until proven otherwise.

Mechanism of Injury

The magnitude and type of injury depends of the ability of the spine to withstand and absorb various forces. Flexion injuries usually compress the vertebral bodies and disrupt the posterior longitudinal ligaments, leading to herniation of the intervertebral disc. Extension injuries disrupt the anterior ligaments and fracture posterior segments of the vertebral column. Compression injuries produce explosive fractures of the bodies and ligamentous rupture. Rotational injuries either cause fracture dislocations of the facets, which damage the midsection of the bodies, or disrupt the ligaments producing fractures. The combined rotation flexion and rotation extension injuries in the cervical region severely damage ligaments, bone and the underlying spinal cord.

Injury to the cord leads to the bruising or mechanical destruction of nerve, haemorrhage, reduced perfusion, oedema and necrosis. An element of the cord damage can be reversible, and up to 4 weeks may be required to assess the final degree of damage.

Initial Treatment

The initial management is rapid immobilization of the fracture area and evacuation to a centre dealing in spinal injuries. Paramedic and ambulance services usually have devices such as backboards, scoop stretchers (eg, a Jordan frame) and extricators which permit safe movement of the patient. The traditional "log rolling" technique is used, but movement of unstable thoracolumbar segments is possible. Nevertheless, it is still necessary in many first aid situations. Hard and soft cervical collars are inadequate to stabilize patients with suspected spinal injury who are awaiting radiological examination. The classic technique of sandbags at each side of the head should be used. As movement of either neck or body may extent the spinal injury, the body should be immobilized on the bed/trolley as well, by means of straps or sheets. Suction equipment should be immediately available at all times.

In high spinal injuries, intubation may be necessary to provide ventilation. The preferred method of stabilizing the neck during intubation is controversial. In one recommended technique, the patient is intubated after administration of IV thiopentine and muscle relaxants, with cervical traction applied longitudinally by means of skull tongs. Care is taken not to extend the neck, although a small degree of flexion is permissible. However, in a study performed on dead patients with cervical cord injuries, longitudinal traction during orotracheal intubation may produce subluxation at C6-C7 fracture dislocations. In practice, extreme care must be used. An array of accessories for difficult intubation should be ready prior to intubation. Consideration is given to intubating with a fibreoptic laryngoscope/bronchoscope or carrying out a cricothyrotomy in very unstable high spinal injuries.

During transport, regular assessment of ventilation and level of neurological deficit is essential, as both may deteriorate. The patients are vulnerable to hypothermia, becoming poikilothermic after high spinal injury. A transient hypertension and bradycardia may occur, but the usual cardiovascular feature is hypotension, which is difficult to treat. Patients are unusually sensitive to both volume loading and drugs because of loss of vascular tone, muscle activity and thoracic effects of breathing on venous return. Treatment should be cautious and titrated with repeated assessments.

Hospital Management

1. Initial Assessment

Upon arrival in hospital or ICU, the neck should be initially stabilized as described above. The next priority is radiological diagnosis/assessment, especially PA, lateral and through-the-mouth oblique x-rays, and CT scanning where possible. At the same time, ventilation is assessed and other injuries treated. Following neurological and radiological assessments, more definitive (surgical) stabilization is considered, which usually involves the use of skull tongs, halopelvic traction or anterior or posterior spinal fusion.

2. Circulation

Haemodynamic stability may be extremely difficult to achieve. Sympathetic outflow disturbances lead to hypotension, and the spinal injury associated injuries may both produce significant hypovolaemia. The radiological appearance of a widened mediastinum in thoracic spine injuries often leads to concern about aortic damage, and if associated signs are present angiography may be required. (See Chapter 69, Chest Injuries.) In high spinal injuries, a low blood pressure is tolerated in the early period provided that urine output, peripheral perfusion and acid base states are maintained.

3. Respiratory

Fractures above C5 lead to the loss of diaphragmatic function and those above C8, loss of intercostal function. Initial and repeated assessment of lung volumes is mandatory. Patients with forced vital capacities of less than 2 L will usually require mechanical ventilatory support. Atelectasis, particularly of the upper lobes, is common and bronchoscopic clearance may be necessary. Sitting patients more upright will increase functional residual capacity. Abdominal binding may assist ventilation. In patients who will require tracheostomy, it is good policy to perform tracheostomy early, and to use a speaking tube which provides obvious advantages especially in improving morale.

4. Nursing

Patients should be nursed supine on a bed capable of lateral tilting and allowing them to be lifted for skin care. Suprapubic catheterization or ureteric catheterization are necessary, and are replaced in males with uridome drainage after urodynamic assessment at 4-6 weeks. Prophylactic antibiotics are not indicated in early treatment.

5. Social Consideration

In injuries such as C1-C2 total quadriplegics, the wishes of the patient and family regarding continuation of treatment should be sought when the complete nature of the lesion is established.

Specific Treatment

Various modalities of treatment have been tried experimentally in animals and humans. Such treatments have included use of hyperbaric oxygen, direct cord hypothermia, osmotic agents, steroids and naloxone. The clinical evaluation of these therapies is extremely difficult and none have any proven value from controlled trials. An isolated case of a dramatic response to local hypothermia, and anecdotal responses to hyperbaric oxygen have been reported.

Complications of Spinal Cord Injury

1. Cardiovascular

Postural hypotension is common during rehabilitation. The cardiovascular system may be unstable for periods of up to a week, particularly during anaesthesia. Thromboembolic disease is also common. Although there is no controlled study on the prophylaxis of thromboembolism in spinal injured patients, available studies suggest that venous thrombosis occurs early, and that conventional prophylactic measures such as low dose subcutaneous heparin may reduce the incidence.

2. Skin

Pressure sores are a major problem and meticulous attention to turning, lifting and pressure areas is mandatory. Such areas may be complicated by soft tissue infections and osteomyelities which require surgical debridement.

3. Alimentary System

In the early stages, paralytic ileus and gastric distention are common, making enteral feeding difficult and predisposing to aspiration. Although with nasogastric decompression, the ileus usually improves, it may recur or become protracted. Abdominal pain may be a feature and pancreatitis should be excluded. Non specific changes in liver function tests occur in 50%. After neurological and cardiovascular stabilization, faecal impaction is a common problem requiring regular enemas and evacuation. Spinal injured patients are at risk from stress ulceration. Control of gastric pH is difficult in quadriplegics due to the loss of sympathetic stimulation of the stomach. H_2 blockers and antacids should be used and gastric pH monitored 4 hourly. Higher doses than usual of antacids and H_2 blockers may be necessary to maintain a pH less than 4.0.

4. Metabolic

Although the metabolic rate is low, muscle wasting and hypoalbuminaemia rapidly occur. Calcium excretion is increased and glucose intolerance may occur.

5. Genitourinary

Infection of the urinary tract is a major problem. In the early phase of spinal injury, a paralysed overdistended bladder is a danger and drainage is necessary. Reflex activity

returns in 2-8 weeks and retraining, often with the help of self-catheterization can occur. Regular monitoring of renal function is mandatory in the early stages.

6. Neuromuscular

In the early phase, autonomic dysreflexa occurs in 50% of patients with lesions above T6. Symptoms include hypotension, sweating, blanching, headache, bradycardia and fever, and the episodes are often triggered by bowel or bladder distension. If prevention of the latter does not control the autonomic dysreflexia, alpha-adrenergic blockers may be necessary.

7. Other Complications

Causalgic pain and reactive depression are common. Non infective fevers frequently occur in the early phase of injury, but the majority of fevers are harbingers of infection.

Chapter 71: Abdominal and Pelvic Injuries

P. G. Moore

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 the adult 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.

Chapter 74: Thermal Syndromes

C Aun

An abnormal body temperature is a common finding in Intensive Care patients, and accurate monitoring with attention to thermal environment is routine practice. From a therapeutic viewpoint, the conditions of greatest interest are hypotheramia, heat stress, malignant hyperthermia, and the neuroleptic malignant syndrome.

Regulation of Body Temperature

Temperature varies throughout the body, but a suitable model is the "Core and Shell" hypothesis. In health, the core or central temperature, is normally kept within the range 36.0-37.5°C, by a balance between heat production and loss. Heat is produced from obligatory metabolism, assimilation of food, and muscle activity. It is dissipated by radiation, conduction, convection, and evaporation of sweat and respiratory water. The interplay of heat production and loss is regulated by a complex, highly sensitive feedback system based on sensing afferents, central integration with set-point, and reflex efferents.

Sensory Systems

Cutaneous thermoreceptors, abundant on exposed areas of the body, are the most sensitive to detect a change in environmental temperature. There are 2 types of receptors: cold-sensitive and warm-sensitive receptors. The cold receptors are stimulated by temperatures below 24°, whereas the warm receptors are stimulated by temperatures above 44°. Deep thermoreceptors in the spinal cord and intra-abdominal viscera are found in animals and

probably in man. Information from the peripheral receptors is relayed via the lateral spinothalamic tract, the reticular system of the medulla, and the thalamus to the anterior hypothalamus. Warm and cold receptors which can respond directly to the blood temperature bathing themm, are located within the anterior hypothalamus itself. Whether central or peripheral input plays the dominant role has been a matter of continuing debate. However, it is now believed from experiments that an interaction occurs between the cutaneous and the central regulating systems.

Central Integration

The anterior and posterior hypothalamus together make up the temperature regulating centre. The centre sets a reference temperature which is influenced by diurnal rhythm, age, exercise, hormones, neurotransmitters, pyrogens, and drugs. When a difference occurs between the body temperature and set-point, the posterior hypothalamus initiates the appropriate thermoregulatory response. The effector responses, which are controlled either neurally or hormonally, act either to conerve or to generate heat accordingly.

Effector Systems

1. *Higher cortical centres* - These are sensitive to signals from the hypothalamus, and can alter behaviour to conserve or to lose heat, such as posture, activity, appetite and clothing.

2. *Shivering thermogenesis* - This is a centrally mediated neural response which consists of involuntary rhythmic contractions of skeletal muscle. Shivering, although independent of sympathetic nervous system, is facilitated by catecholamines.

3. *Non-shivering thermogenesis (NST)* - This refers to an increase in combustion of fatty acids and glucose, both exothermic reactions. In adults, the most active metabolic areas are the liver and skeletal muscles. In the neonate, the highly vascular and richly innervated brown fat is the potent source for NST. The non-shivering thermogenesis is mediated by sympatho-adrenal (rapid response) and thyroid (slow response) function. On acute exposure to cold, noradrenaline is secreted from the peripheral nerve terminals to stimulate calorigenesis in fat and muscle cells. Adrenaline and thyroxine are important in long term cold adaptation, but play a minor role in the initial response to cold stress.

4. *Sweating* - Increase in eccrine sweat secretion is produced by direct or reflex stimulation of the centres of the spinal cord, medulla, hypothalamus or cerebral cortex. Shivering and sweating provide fairly coarse control of heat balance.

5. *Cutaneous blood flow* - This allows fine control of heat balance. These reflex changes in cutaneous blood flow are mediated by the sympathetic nervous system and occurs within seconds.

Abnormal body temperature may occur when the system is over-stressed (as in cold immersion or malignant hyperthermia), or as a result of failure of the thermoregulatory mechanism. The latter may be due to defective central regulation (eg, caused by pyrogens or head injury), or to depression of sensory and effector mechanisms, as occurs in anaesthesia, drug overdose or hypothyroidism.

Measurement of Body Temperature

The most useful instruments are the mercury thermometer (including the low reading type) and the thermistor. Sites for temperature monitoring are the mouth, nasopharynx, aural canal, oesophagus, rectum, and axilla. Rectal temperature is traditionally used to indicate the core temperature despite several disadvantages. It can be affected by heat-producing organisms in the bowel, cool blood returning from the legs, and insulation effect of faeces. Oesophageal probe should be positioned accurately in the lower end of the oesophagus, and it is the best single monitor of core temperature for hypothermic patients. Nasopharyngeal measurements can be affected by leakage of air around the endotracheal tube cuff. Axillary reading is the best monitor of muscle temperature, and therefore, the most sensitive in malignant hyperthermia. However, it is a poor measure of core temperature. Aural canal probe is traumatic and can be rendered inaccurate by cerumen. Skin temperature in correlation with core temperature is used clinically to assess tissue perfusion. Monitoring of readings from 4 or more sites to determine mean skin temperature is generally used in research.

Hypothermia

Hypothermia is defined by a core temperature below 35°C, requiring emergency and intensive management. It is frequently underdiagnosed and has a high mortality.

Classification

Many classifications of hypothermia have been proposed. The practical ones are based on either aetiology or severity of the hypothermia as determined by body temperature measurements. Clinically, hypothermia is classified according to the core body temperature.

1. *Mild hypothermia* - 32-35°C

2. *Moderate hypothermia* - 28-32°C

3. *Severe hypothermia* - below 28°C.

Aetiologically, hypothermia can be divided into:

1. *Induced hypothermia* - The body temperature has been lowered deliberately as part of a therapeutic regimen.

2. *Accidental hypothermia* - This occurs without deliberate intention, and it is useful to further differenitate into:

(a) *Primary* accidental hypothermia, in which the body possesses normal thermoregulation, but the exposure to cold is overwhelming (eg, from immersion or exposure).

(b) *Secondary* accidental hypothermia in which mild to moderate cold exposure leads to hypothermia because of abnormal thermogenesis. This division is helpful in the assessment and management.

Causes

There are numerous causes for hypothermia (Table 1). It is more common in the elderly and infants, where thermal liability and poor thermoregulation contribute to increased vulnerability to cold. Many underlying diseases and drugs predispose them to hypothermia, and increase mortality.

Table 1. *Causes and Predisposing Conditions of Hypothermia*

Accidental	Immersion
	Environmental exposure
	Immobility
	Poor living conditions
Drug induced	Anaesthetic agents (prolonged anaesthesia)
	Phenothiazines
	Barbiturates
	Antidepressants
	Phenformin
	Ethanol
Infections	Pneumonia
	Sepsis
Nutrition	Protein-calorie deficiency/malnutrition
Cardiovascular	Cerebrovascular accidents
System	Head injury
	Cerebral neoplasm
	Progressive mental deterioration
Renal	Uraemia
Endocrine	Hypopituitarism
	Myxoedema
	Hypoglycaemia
	Diabetic ketosis
	Hypoadrenalism.

Pathophysiology

As body temperature decreases, the following changes occur:

1. *Cardiovascular System* - Sympathetic activity is initially stimulated with elevated plasma levels of noradrenaline and free fatty acids. There is peripheral vasoconstriction, tachycardia, and increased cardiac output in mild hypothermia. However, a progressive fall in heart rate, cardiac output and blood pressure then follows as hypothermia progresses. This reduces tissue perfusion, ECG changes include bradycardia, prolongation of all phases of the cardiac cycle, conduction abnormalities, T-wave invession, and the occurrence of "J" ofr "Osborn" wave, an extra deflection at the QRS-ST junction. Supraventricular arrhythmias, in particular atrial fibrillation (AF) are common below 30°C and ventricular fibrillation (VF) may occur below 28°C. This form of VF has been reported to be refractory to electrical or pharmacologic cardioversion, unless core temperature is brought above 28°C. Fibrillation can occur earlier, in the presence of diseased or ischaemic myocardium, or with stimuli such as insertion of central venous line or endotracheal intubation.

Cardiac creatinine phosphokinase and lactate dehydrogenase levels are frequently elevated. This may be due to leakage from cells with impaired membrane integrity or microinfarction. Unnecessary manipulation should be avoided in these patients, because of the potential for cardiac arrhythmias. Cardiac ischaemia together with acidosis may partly be responsible for the ECG changes, arrhythmias and enzymatic changes.

There is an increased haematocrit caused by dehydration, and blood viscosity is increased significantly from cooling of blood per se and peripheral fluid sequestration.

2. *Respiratory System* - Following initial reflex stimulation from which tetany may result, ventilation is progressively depressed and ceases at 24°C. Hypoventilation results from a reduction in respiratory rate and minute ventilation volume. There is an impairment in hypoxic pulmonary vasoconstriction, and decreased gaseous diffusion capacity. Impaired cough reflex associated with hypothermia, together with diminished consciousness and dehydration, render the aged more susceptible to pulmonary infections and to aspiration.

The solubility of respiratory gases (including anaesthetic agents) in plasma increases with hypothermia. The oxygen-haemoglobin dissociation curve is shifted to the left, primarily due to increased affinity of haemoglobin for oxygen. Blood gas analysis measured at a temperature of 37°C are traditionally corrected by factors from normogram or formulae, or automatically corrected by modern blood gas analysers.

3. *Metabolic System* - Grave acidosis develops in the severely hypothermic patient which eventually shifts the oxygen-haemoglobin curve to the right. The acidosis contains both a respiratory and a metabolic component. CO_2 retention is due to inadequate ventilation, and the metabolic component is caused by reduced tissue perfusion with accumulation of lactate and other acid metabolites. A renal component due to a reduced tubular H+ ion secretory capacity, contributes to the acidosis in long standing hypothermia. Measurements of pH made at 37°C should be corrected by addition of 0.0147 pH unit for each degree *below* this temperature. It has, however, been suggested that correction offers no practical management benefits.

Metabolic rate is raised during the shivering phase. However, it eventually falls and the basal metabolic rate (BMR) declines at a rate of 5-7% per °C. Hyperglycaemia is a

common feature. This is thought to be due to decreased insulin release and impaired peripheral utilization of glucose, or related to mild pancreatitis. Insulin administration has little effect and the condition reverts on rewarming. However, in long standing hypothermia, the glycogen stores may be exhausted and hypoglycaemia will ensue. Liver function is depressed, affecting most enzymatic and detoxifying processes. Plasma cortisol levels are usually elevated despite a reduced ACTH secretion. This is probably due to a reduced hepatic hormone clearance rate.

4. *Renal System* - The decrease in renal perfusion reduces the glomerular filtration rate (GFR) and clearance of multiple drugs. Cold-induced diuresis is secondary to the volume load imposed on capacitance vessels by peripheral vasoconstriction, and also results from a depression of tubular reabsorptive function despite a diminished GFR. Glycosuria may add an osmotic contribution to the polyuria. Serum potassium concentration is variable. Hypokalaemia has been observed in hypothermic patients which is in contrast to the expected hyperkalaemia from a concomitant metabolic acidosis.

5. *Neurological System* - There is generalized cerebral depression. Loss of consciousness and pupillary dilation supervene at 30°C. Cerebral blood flow is reduced at a rate of 7% per °C drop. There is a corresponding reduction in cerebral metabolic rate so that demand does not outstrip supply. Shivering is gradually replaced by muscular rigidity at 33°C and a rigor mortis like appearance develops at 24°C.

6. *Gastrointestinal System* - Intestinal motility decreases below 34°C, with ileus occurring frequently.

Diagnosis

The diagnosis of hypothermia rests upon reliable temperature measurement. It may be difficult to differentiate from death especially in the immersion victim. Death should not be assumed until resuscitation attempts have failed in an adequately rewarmed patient. (See Chapter 72, Near-Drowning.) Investigations are indicated to monitor progress, clarify and underlying cause and detect complications. Useful tests include a full blood count, serum electrolytes, urea, creatinine, glucose and amylase, blood gases and pH, liver function studies, blood culture, drug and alcohol screen, chest X-ray; ECG, and thyroid function tests.

Management

1. General Measures

The hypothermic patient should be managed with careful monitoring of temperature, vital signs and fluid balance. Excessive handling may precipitate VF. Antiarrhythmic drugs and electrical deribrillation are ineffective at low temperatures. Adequate intravenous access and a patent airway are essential. Optimal oxygenation, if necessary by controlled ventilation, should be ensured. Hypoglycaemia should be corrected. All intravenous fluids must be prewarmed. Bradycardia may require the use of atropine and/or isoprenaline. If these drugs fail, cardiac pacing may be necessary until rewarming methods have stabilized the cardiovascular system. Bicarbonate may be required to correct acidosis. Electrolyte imbalances

should be corrected with care. Intravenous corticosteroids are no longer used as part of initial treatment. Infection which usually involves the lower respiratory or urinary tracts, requires broad-spectrum, bactericidal antibiotics (after appropriate cultures).

2. Specific Treatment

The mainstay of treatment is rewarming. Various methods of rewarming have been employed (Table 2). The choice is best based on the cause and degree of hypothermia.

Table 2. *Rewarming Methods*

Passive
- Warm environment (> 25°C)
- Insulating material (warm blanket)

Active
- (a) External
  - Immersion in hot water bath (40-45°C)
  - Electric blanket
  - Hyperthermia mattress
- (b) Core
  - Inhalation of heated inspired gasses
  - Dialysis
    - haemodialysis
    - peritoneal
  - Irrigation
    - mediastinal via thoracotomy
    - intragastric
  - Oesophageal thermal tube
  - Extracorporeal blood rewarming.

(a) *Passive External Rewarming (PER)* - This method is recommended for the elderly and for those with mild hypothermia. The patient is placed in a warm room covered with warm blankets and allowed to warm at his/her own pace. Rewarming is gradual at 0.5°C/h.

(b) *Active External Rewarming (AER)* - This method has been used successfully in young patients with accidental or environmentally induced hypothermia. The limbs should be excluded during immersion in hot water (40-45°C) to reduce the after drop in core temperature, and to protect against "rewarming shock" secondary to peripheral vasodilation. Other techniques include the use of electric blanket or hot water bottles. Hypotension, decreased coronary perfusion, and a shunting of cool blood to the myocardium leading to arrhythmia, are potential hazards. These may be too dangerous for elderly patients especially those with fragile cardiovascular systems.

(c) *Active Core Rewarming (ACR)* - This method may be used for moderate to severe hypothermia, and may have advantages over other methods. It produces a preferential warming of the myocardium, leading to an increase in cardiac output, less cardiac irritability,

and a rapid return to a near normal core temperature. The necessary equipment for ACR is usually sophisticated and may not be readily available. Recently, an oesophageal thermal tube has been developed. This is a non-invasive closed system using a disposable thermal tube (with water at 42°C at a flow rate of 3 L/h) in the oesophagus. The rewarming rate is approximately 1.5°C/h. It is a simple method, suitable even in the field situation, but the patient has to be intubated. It is now officially recommended as the treatment of choice for severe accidental hypothermia in Denmark.

After Effects

Many elderly patients tend to have a residual hypothalamic dysfunction, rendering them susceptible to recurrent hypothermia.

Mortality

The mortality among hospitalized patients with hypothermia varies from 20-85%. Mortality has been related to several factors namely the severity of hypothermia, the duration prior to treatment, the degree of hypotension and associated underlying conditions, in particular, the cardiovascular disease. Although rewarming technique is important, careful and meticulous supportive care may be responsible for the large differences in mortality report.

Heat Stroke

Heat stroke is a condition caused by excessive heat storage brought about by either "overloading" or failure of the thermoregulatory system during exposure to heat stress. It is the greatest threat to life among the heat disorders (ie, heat syncope, heat exhaustion and heat stroke). Overloading occurs when the rate of heat production or storage exceed the rate of heat loss. This is characteristically seen in young athletes or military recruits. Thermoregulatory failure refers to dysfunction in either central control or peripheral responses of heat loss mechanisms (sweating and vasodilation or skin blood flow). This is commonly found in the elderly, living without airconditioning in a hot and humid environment. Predisposing factors are dehydration, lack of acclimatization, obesity, infection, alcoholismmm, mental illness, and drugs impairing heat response (eg, phenothiazines, diuretics, and anticholinergics).

Pathophysiology and Clinical Presentation

The 3 cardinal signs of heat stroke are:

1. severe central nervous disturbance;

2. hyperpyrexia (core temperature 41-43°C); and

3. hot, dry skin, which is pink or ashen, depending on the circulatory state. Sweating may be profuse or absent.

Neurologic System - Confusion, aggressive behaviour, delirium, convulsions and pupillary abnormalities may progress rapidly to coma. There may be decorticate posturing, faecal incontinence, flaccidity or hemiplegia. Cerebellar symptoms including ataxia and dysarthria may be permanent in a small percentage of patients. The degree of damage is related to the duration and degree of hyperpyrexia as well as circulatory insufficiency.

Cardiovascular System - Hear rate increases at 10 beats/min for each 1°C rise in body temperature. The ECG may show ST segment depression and T wave changes, as well as supraventricular tachycardia. Cardiac output is initially elevated with low peripheral vascular resistance and high skin and skeletal muscle blood flow. Cardiac output eventually drops. Myocardial injury or an increased pulmonary vascular resistance, rather than peripheral pooling of blood, have been suggested to be the cause of circulatory failure.

Respiratory System - Initial respiratory alkalosis is usually followed by a mixed acid-base disorder in the presence of metabolic acidosis. Complications include aspiration, acute respiratory distress syndrome, and congestive heart failure with pulmonary oedema.

Renal, Fluid and Electrolyte Abnormalities - Dehydration and electrolyte (sodium, potassium, calcium, phosphorus and magnesium) deficiencies follow excessive sweating. Acute tubular necrosis is common and is related to thermal parenchymal injury. Subsequent hypotension and/or rhabdomyolysis can precipitate renal failure.

Coagulation Abnormalities - Bleeding diathesis observed is possibly contributed by thrombocytopenia, disseminated intravascular coagulation and liver cell damage.

Management

Rapid and effective cooling and support of vital organ systems are the two principal therapeutic objectives. Clothing should be removed and the patient rapidly cooled externally by ice packs, air conditioning, ice water bath, or internally by peritoneal lavage or gastric lavage cooling. However, recently it has been shown that wetting the skin and using warm air to induce evaporation is superior to the conventional methods.

Hypovolaemia, dehydration and electrolyte imbalance must be corrected cautiously with crystalloids guided by urine output, central venous pressure, serum electrolyte values, and haematocrit. A Swan-Ganz catheter may be of value in fragile patients. Mannitol may be required to promote renal blood flow. Anuria, uraemia and hyperkalaemia are indications for early dialysis. Oxygen therapy and controlled ventilation may be indicated. Anticonvulsants (eg, diazepam) may be required. Phenothiazines should be used to suppress shivering. Prophylactic steroids or antibiotics are not recommended. Hypoglycaemia may be present and should be treated. Evidence for underlying illness should be sought and treated accordingly. Cooling should be stopped at 39°C, since core temperature will continue to decrease and hypothermia should be avoided. Dantrolene, appears promising, but needs further evaluation.

Malignant Hyperthermia (Hyperpyrexia)

Malignant hyperthermia (MH) is a pharmacogenetic disorder characterized by acute hypercatabolic reactions in skeletal muscles, triggered by certain drugs (mainly used in anaesthesia) and stresses. Triggering drugs include any of the potent inhalational agents, any depolarizing muscle relaxant (mainly suxamethonium), amide local anaesthetics, caffeine, and, rarely, halogenated X-ray contrast media such as Diodrast. The true incidence is difficult to determine. However, a figure around 1:40.000 of the unselected anaesthetized population has been suggested. Stress reactions may be triggered by strenuous exercise or massive skeletal injury, especially during hot and humid climate or under emotionally stressful conditions.

Aetiology and Pathophysiology

The aetiology of MH is still not entirely clear despite years of intensive research. However, the most acceptable explanation appears to be a sudden increase in myoplasmic calcium concentration. This probably is secondary to an increase in calcium released from the malfunctioned sarcoplasmic reticulum. The mechanisms proposed for the rise in myoplasmic calcium concentration include defective sarcoplasmic reticulum membrane itself, or some other part of the muscle, eg, mitochondria, excitation-contraction coupling, calmodulin (main intracellular calcium receptor), sarcolemma and adrenergic innervation pathway.

MH has a spectrum ranging from mild to severe manifestation. A modest increase of myoplasmic calcium in mild MH activates enough energy metabolic processes to stimulate the production of heat, CO_2, lactic acid and oxygen consumption. In severe (classic) MH, the high level of myoplasmic calcium induces the following processes:

1. Myosin ATPase and sarcoplasmic reticulum ATPase is activated, thereby increasing heat production by accelerating the hydrolysis of ATP.

2. The configuration of troponin is changed, which allows contact of myosin to actin receptors, thus forming short and rigid actomyosin.

3. Calcium is taken up into the mitochondria, where it uncouples oxidative phosphorylation, leading to cessation of ATP production with further production of energy, CO_2, and lactic acid. This reduces the substrate for the active pumping of cellular and intracellular membrane, leading to increased permeability of ion and molecules in the direction of their natural concentration gradient. This causes further increases in cytoplasmic calcium. There is a leakage of potassium, phosphate, enzyme and myoglobin to the extracellular fluid, and entry of sodium and water intracellularly. Once ATP is depleted in the muscle, further treatment is probably futile.

MH appears to be a widespread membrane defect. The most adverse expression is in the skeletal muscles. Heart muscle, smooth muscle, brain cells, platelets and many other cell types may also be affected. The inheritance pattern is variable, and may be autosomal dominant, recessive and perhaps multigenic.

Clinical Presentation

Many MH susceptible patients lack obvious preoperative diagnostic clues, and an initial acute crisis may appear without warning, following a commonly used anaesthetic agent (such as suxamethonium and halothane). Early signs of MH are:

1. *Rigidity of jaw muscles* which may later become generalized.

2. *Tachycardia and cardiac arrhythmias* which include frequent ventricular extrasystoles, bigeminy and ventricular tachycardia. ECG may show tall peaked T waves characteristics of hyperkalaemia.

3. *Unstable blood pressure* - hypertension during the early stage, and hypotension is a late sign as the cardiac function becomes progressively impaired.

4. *Marked increase in CO_2 producting* leading to tachypnoea if patient is breathing spontaneously. A rising end-tidal CO_2 concentration despite an increase in ventilation is an early observable sign. Hence end-tidal CO_2 monitoring is a valuable adjunct to diagnosis. If a CO_2 absorber is in use, the temperature of the canister may rise, due to the accelerated chemical reaction within the soda-lime.

5. *Cyanotic mottling* of the skin, especially over the head, neck and upper chest.

Later signs are:

6. *Pyrexia* although the hallmark of MH is usually a later sign, and is influenced by the agents used. Suxamethonium in conjunction with halothane, induces an earlier and more rapid rise. The rate of rise in core temperature is variable (from 1°C/h to 1°C every 5 minutes).

7. *Acute pulmonary oedema* as a result of left ventricular failure commonly occurs in the late stage.

8. *Bleeding diathesis* may become apparent from disseminated intravascular coagulopathy.

9. *Other complications* include muscle swelling and pains, severe neurological damage, haemolysis, myoglobinaemia, myoglobinuria, renal failure from tubular necrosis.

Biochemical Changes

Laboratory investigations for the diagnosis and guidance of therapy include:

1. *Arterial Blood Gas Analyses* - A rise in CO_2 tension and a fall in oxygen tension in the mixed venous blood is an early finding during acute crisis, followed by a similar change in arterial blood, with a classical picture of mixed respiratory and metabolic acidosis (from increased lactate production).

2. *Serum Electrolytes* - There are major fluctuations in serum electrolyte levels. Serum potassium is usually elevated early, followed by a marked and prolonged fall. Variation in the serum calcium, phosphorus and magnesium levels may be observed.

3. *Serum Enzymes* - Creatinine kinase level increases markedly during the crisis, but in survivors, may reach their highest levels 24-48 hours later (as high as 1.000.000 units). Lactate dehydrogenase and aspartate aminotransferase are also very high during the first 72 hours in survivors.

4. *Haematological Studies* - Signs of haemolysis in blood and urine samples may be detected. Myoglobinaemia and myoglobinuria are common. Coagulation screen studies should be obtained, because lowered levels of Factor VIII and fibrinogen, together with thromocytopenia result in impaired coagulation.

5. *Blood Glucose, Lactate and Urea Nitrogen* - Accurate diagnosis requires skeletal muscle biopsy. Tests performed on the excised muscle include the caffeine halothane contracture (CHC) test, the caffeine skinned fibre tension test, the ATP depletion test, radioactive calcium uptake, and microscopy. Quin-2 test on lymphocytes is currently under investigation to evaluate its value as a non-invasive screening test.

Susceptibility to malignant hyperthermia is associated with clinical and subclinical myopathies. Early recognition is vital for the successful treatment of malignant hyperthermia. Routine monitoring of all patients undergoing anaesthesia must include heart rate, blood pressure, ECG, end tidal CO_2, pulse oximetry, airway oxygen monitor, tidal volume, airway pressure, respiratory rate and body core temperature.

Management

1. *Termination of anaesthesia* and surgery if possible.

2. *Hyperventilation with oxygen* through a vapour free circuit.

3. *Dantrolene sodium* is partly responsible for the marked improvement in MH survival rate, and is currently accepted as the drug of choice. It must be given early while muscle perfusion is still adequate. An infusion of 1 mg/kg/min is given until reduction of heart rate, muscle tone and temperature occur, or a total dose of 10 mg/kg has been given. A dose exceeding 4 mg/kg is seldom required, although the optimal dose in humans is not yet known. After being controlled, MH may recur in the post-anaesthetic period. Dantrolene administration is immediately repeated at the first sign of such occurrence. A repeat prophylactic dose of 2.4 mg/kg after 10-12 h (which is the elimination half-life of Dantrolene) has been advocated.

Dantrolene is believed to relax skeletal muscle. It causes dissociation of muscle excitation-contraction coupling by interfering directly with sarcoplasmic reticulum calcium release or the preceding "trigger calcium", or both. However, its mode of action is not yet fully understood. It is metabolized in the liver, both by oxidation and reduction, followed by acetylation to form the reduced acetylated derivative of dantrolene (RAD)-metabolites, which

themselves manifest some muscle relaxant properties. Dantrolene sodium is supplied in a vial containing 20 mg (together with 3.0 g of mannitol and enough sodium hydroxide to raise the pH to 9.5). The solution is irritant to veins and should be injected to a fast running drip or large vein.

While dantrolene itself has no myocardial effect, marked myocardial depression in animals has been reported after administration with verapamil. Simultaneous administration of these 2 drugs is contraindicated. Other side effects include drowsiness, nausea and vomiting, and synergism with neuromuscular blocking agents. Hepatic dysfunction, although documented in chronic oral administration, has not been shown following acute intravenous administration.

Prophylactic oral administration of dantrolene prior to anaesthesia in susceptible individuals is not been questioned, because an effective blood level cannot be guaranteed, and there are too many unpleasant side-effects. However, 2.4 mg/kg IV during induction of anaesthesia is recommended if there is a strong indication.

4. *Correction of acidosis* is achieved with sodium bicarbonate. Care should be taken against giving excessive alkali (noting the sodium hydroxide content in dantrolene). Over-correction of acidosis hjas been shown to lower the survival rate.

5. *Control of serum potassium,* if necessary, using glucose and insulin. Hypokalaemia may supervene.

6. *Mannitol and frusemide* is given to help reduce cerebral and muscle oedema, and prevent acute renal failure. The dose of mannitol is adjusted according to the mannitol given in conjunction with dantrolene.

7. *Control of arrhythmias* is undertaken with procaine or proacinamide. The dose should not exceed 7 mg/kg during crisis, as the myocardium is already compromised. Verapamil can be used, but never simultaneous with dantrolene. Repeated doses of 1.0 mg (0.015 mg/kg) propranolol IV (to a maximum of 0.15 mg/kg/6 h) may assist in lower the heart rate.

8. *Cooling* is started (although early diagnosis and dantrolene treatmetn may avoid aggressive cooling) with the following methods:

(a) ice packs and cooling blankets;

(b) ice water bath;

(c) cold intravenous solutions:

(d) internal cooling with cold solutions in the stomach and rectum;

(e) peritoneal dialysis using cold dialysate; and

(f) extracorporeal cooling if facility is available.

9. *Drugs* of marginal value are barbiturates, narcotics and antipyretics. Chlorpromazine increases heat loss by correcting peripheral vasoconstrictioon, and reduces heat production by inhibiting shivering, and may offer substantial benefit. Steroids have been recommended but their benefits are uncertain. Drugs which should *never* be given during a reaction include amide local anaesthetics, cardiac glycosides, calcium chloride, belladonna alkaloids and vasopressors.

10. *Movement* and handling of the patient should be minimized as they may precipitate ventricular arrhythmias.

11. *Monitoring* should include temperature, oximetry, end tidal CO_2, arterial and central venous pressures, urine output, ECG, arterial blood gases and pH, coagulation studies, and serum biochemistry such as electrolytes, glucose and enzymes levels.

The mortality rate from MH has declined from 86% in the 1960s to about 7% in the 1980s. This is attributed to the better understanding of the syndrome, advanced monitoring devices leading to early diagnosis, and vigorous treatment with early use of dantrolene.

Neuroleptic Malignant Syndrome

The neuroleptic malignant syndrome (NMS) is a relatively rare, but potentially fatal idiosyncratic response to neuroleptic drugs (eg, phenothiazines, butyrophenones, thioxanthenes and miscellaneous antipsychotic agents such as loxapine). The syndrome is characterized by hyperthermia, muscular rigidity, akinesia, impaired consciousness and autonomic dysfunction. The estimated incidence is 0.5-1.0% of all patients exposed to neuroleptics. NMS affects all ages; 80% are under 40 years of age, and there is a male to female ratio of 2:1. The pathogenesis is believed to be related to dopamine - receptor blockade in basal ganglia and hypothalamus. Hyperthermia in NMS is probably due to impaired hypothalamic temperature regulation, and sustained muscle contraction.

Clinical Presentation

The syndrome may occur at any time during treatment with neuroleptic medication, frequently within 2 weeks of institution of therapy or when the dosage is increased. Serum levels are commonly within the normal range. The symptoms of NMS usually progress rapidly over 1-3 days, with hyperthermia (around 40°C), muscle rigidity, and resting tremor. Autonomic dysfunction (eg, tachycardia, labile blood pressure, and sweating) may be premonitory signs. Mental status varies from confusion to coma, and a variety of associated neurological signs, with disorders of speech and swallowing being most common, are present. Predisposing factors include functional psychoses and organic brain diseases.

Complications include acute renal failure from dehydration and/or myoglobinuria, pulmonary embolism with cardiac arrest, and acute myocardial infarction with pulmonary oedema. Persisting neurological sequelae has been reported in 10% of patients with parkinsonism, dyskinesia, dementia and ataxia. Laboratory findings are non specific. These include increased creatinine phosphokinase (CPK) level, non specific leucocytosis, abnormal liver function tests, acidosis, hypoxia, hypercarbia, and raised plasma and urinary

catecholamine levels. Examination of cerebrospinal fluid are unremarkable. CT scans usually show no abnormalities. Those who survive recover in about 1-2 weeks (or longer with depot drug preparation).

A mortality rate of 20-30% has been reported. The association between malignant hyperthermia and NMS appears tenuous, and the 2 syndromes are probably distinct entities. NMS is differentiated from MH by its slow onset, moderate CPK rise, few associated metabolic changes, lack of family history, different triggering agents, and patient tolerance to anaesthetic agents.

Management

Treatment of NMS involves the following:

1. Immediate withdrawal of offending agent.

2. Supportive therapy:

(a) Cooling procedures as in MH.

(b) Treatment of cardiovascular instability.

(c) Mechanical ventilatory support if necessary.

(d) Treatment of dehydration with fluid therapy to maintain renal function.

3. Specific therapy:

(a) L-dopa with carbidopa (a dopa-decarboxylase inhibitor) mediate an increase in presynaptic synthesis and release of dopamine, and have been reported to be beneficial.

(b) Bromocryptine, a direct-acting dopamine agonist, has been successfully used with dantrolene.

(c) Dantrolene has been reported to alleviate manifestations of the syndrome at a dose as small as 100 mg/day either orally or IV. Until more is known about the pathophysiology of NMS, it is advisable to use a combination of dantrolene and dopaminergic drugs in its treatment.

Chapter 75: Electrical Injuries

T E Oh

The widespread use of electrical appliances and electronic equipment in homes and hospitals, exposes people, patients, and staff to the hazard of electric shock. The ICU is an area of special risk to patients, due to the use of invasive monitoring equipment and transvenous pacemakers, which not only bypass the resistance of the skin, but may provide a direct route to the heart for electric currents.

Electrical injuries may be in the form of:

1. Electrical flash burns resulting from radiant heat released when an arc forms between an energized source and ground.

2. Flame burns secondary to the ignition of clothing from high voltage injuries.

3. Electrocution.

4. High tension and lightning injuries.

Electricity

Electricity is produced by electrons which carry negative charges. This electricity may be static, such as that seen with some synthetic clothing, or dynamic, where there is a flow of current. An electric current is a flow of electrons through a conductor, from a point of higher concentration or potential, to a point of lower potential. This flow may be a direct current (DC) such as that obtained from a car battery, or alternating current (AC) from mains supply. With direct current, the electrons always flow in one direction, whereas with alternating current, the electrons flow back and forth at a frequency, ie, cycles per second, or hertz (Hz).

Current flow (the volume or number of flowing electrons) is measured in amperes (A). An ampere is a flow of one coulomb per second. One coulomb (C), the unit of electrical charge, is 6.28×10^{18} electrons. In order for current to flow, there must be a difference in the "electrical pressure" (ie, potential) from one higher potential point to a lower point. The unit of electrical pressure or potential is the volt (V). Mains supply in Australia is 240 V at 50 Hz. In USA and parts of Europe, the household current is 110 V at 60 Hz. Resistance to current flow is measured in ohms. The relationship of voltage, current and resistance is signified by Ohm's Law:

$$\mathbf{V = I \times R}$$

$$\mathbf{or\ I = V / R.}$$

where I = current in amperes (A)
R = resistance in ohms (Ω)
V = voltage in volts (V).

An electric current will flow if a circuit is completed either from positive to neutral wires, or from positive to earth. From Ohm's Law, amperage increases with larger voltages and lower resistances.

Physiological Considerations

For a current to flow through the body, the body must complete an circuit, and the current must overcome the resistance of the body. Most of this resistance is in the skin. If the skin is dry, there is a resistance in excess of 100.000 Ohms. Skin resistance will be markedly

reduced if the skin is wet, or if conductive jelly has been applied. The resistance of the current pathway from one extremity to another with intact, but moist skin, is approximately 1000 Ohms. Hence, a current of 240 milliamps (mA) from household and hospital electrical supply at 240 V, can easily flow through the body from electrical source to ground, whenever a circuit is completed, if the skin is moist or wet.

Electrocution

The pathophysiological processes of true electrical injuries are poorly understood. Risk of electrocution depends on any of the following:

1. the amount of current passing through the body;

2. the duration of the current; and

3. the tissues traversed by the current.

Obviously, amperage is most directly related to the extent of injury, but usually only the voltage is known. In general, lower voltages cause less injury, although voltages as low as 50 V have caused fatalities.

The electric current passing through the body produces these main effects.

1. Tissue Heat Injury

Body tissues in decreasing resistance are skin, bone, muscle, nerves, and vessels. It is not known whether electricity flows through the body homogenously, or follows pathways of lower tissue resistance. Skin entry burns depend on the area of contact and skin resistance. Oral commissure burns in toddlers biting through electrical cables, and burns at (lower resistance) wrist and antecubital areas, are other typical patterns of skin electrical injury. Organs may be injured by generated heat if they lie in the path of the current from entry to ground. Other electrical heat generated tissue injuries include coagulation ischaemia and necrosis, and vascular thrombosis.

2. Depolarization of Muscle Cells

An alternating current of 30-200 mA will cause ventricular fibrillation. Currents in excess of 5 A cause sustained cardiac systole. (This is the principle used a defibrillator.) Apart from ventricular fibrillation, other arrhythmias may occur. ST and T-wave changes may be seen on ECG. Global left ventricular dysfunction may occur hours or days later despite initial minimal ECG changes. Myocardial infarction has been reported, and the diagnosis is often difficult, because of the elevated creatine phosphokinase levels (even the MB isoenzyme levels), due to the extensive muscle injury.

Skeletal muscle is responsive to tetanic depolarization at household frequencies of 50-60 Hz and with currents greater than 15-20 mA. Hence, voluntary release from the electrical source may be prevented during electrocution.

3. Neurological Injuries

These injuries may involve the central nervous system, spinal cord, and/or peripheral nerves, and clinical manifestations may range from confusion and agitation to paresis and paralysis.

4. Renal Failure

Acute renal failure may follow due to myoglobinuria secondary to extensive muscle necrosis.

5. Other Injuries

Fractures and dislocations caused by falls, and damage to eyes, lungs, gastrointestinal tract as well as other organs may result from electrical injury. Intrauterine foetal death has been known.

Microshock

The above domestic/industrial electrocution is known as macroshock, when current flowing through the body passes through the heart. In the ICU there is potential for another form of electrocution, microshock. Microshock occurs when there is a direct current path to the heart muscle. The pathway may be provided by a saline-filled monitoring catheter or transvenous pacemaker wires. A current required to produce ventricular fibrillation in microshock is extremely small, in the order of 60 microA. Currents of 1-2 mA produce tingling of the skin and is barely perceptible. Hence a lethal macroshock current may be transmitted to a patient via a staff member who may be unaware of the conducted current. Such a small current is potentially lethal because a high current density is produced at the heart.

Tension and Lightning Injuries

High tension electricity is a supply greater than domestic supply. The voltage is usually many thousand volts. Tissue damage is mainly due to generation of heat. The heat generated, according to Joule's Law, is proportional to the amperage squared times the resistance (A^2 x Ω). Thus, tissues traversed by the current, but which are poor conductors, will suffer the greatest degree of thermal insult. Witnesses have described tissues actually exploding.

Lightning carries 12.000-200.000 A and voltage in the millions. Exposure to lightning is essentially the same as exposure to any other high tension electricity. Entry skin burns are described as having spidery, arborescent, and pine tree patterns. Asystole is more likely to be prodcued from myocardial injury than ventricular fibrillation. However, good recovery has been reported despite presenting signs of hopeless neurological function (eg, fixed dilated pupils).

Management of Electrical Injuries

1. *First aid and resuscitation* - It is imperative to make the immediate environment safe for rescuers. Power sources should be switched off and wet areas should be avoided wherever possible. Instinctive attempts to grab the electrocuted victim must be avoided until it is safe to do so. Cardiopulmonary resuscitation is carried out when indicated.

2. *Investigations* - are indicated to detect organs damaged and the degree of damage, and include serial ECG, echocardiography, biochemistry, X-ray, and CT examinations. Arteriograms may be indicated.

3. *Hospital and ICU management* - are directed towards treatment of burns, ischaemic and necrotic tissues, and injured organs. The principle of management of the electrical burn is early complete excision. Fasciotomies and amputations may be necessary. The outcome in approximately 50% of high tension injuries is amputation of affected limbs. Tetanus toxoid and antibiotics, especially penicillin are given if indicated.

Electrical Hazards in ICU

The ICU has the potential to inflict both macroshock and microshock injuries to staff and patients. Potential sources of these electrical hazards are:

1. Major Electrical Faults

If the earthwire is disconnected from a piece of electrical equipment, the active wire shorting of the casing will produce mains voltage at the casing. Mains current can then flow through any person who comes in contact with the faulty equipment. Obviously, any handling of exposed "live" electric wires also exposed the person concerned to mains voltage. As mentioned above, the outcome of the resulting electric shock will depend largely on whether the skin in contact is dry, and whether the body offers a low resistance path to earth.

2. Microshock Currents

(a) *Earth leakage*

There exists capacitance between the power supply of equipment and the casing. The capacitance allows a current to flow to earth. This earth leakage current can be very important if the earth wire is not of low enough impedance. Such a leakage current may still occur even with the apparatus' electrical mains supply switched off. In the microshock situation, an earth leakage current of 100 microA can be fatal. As mentioned above, a staff member may unknowingly provide the completion of a circuit from the equipment to the patient.

(b) *Different earth potentials*

When different pieces of equipment are earthed in different receptacles, there may be a significant potential difference between the earth terminals. A current may then pass from one earth terminal to another. The path to earth is still present with the instrument switched off. Again, as a current of only 60 microA may be fatal, the risk of microshock is very real.

Protection Against Electrical Hazards in ICU

All ICUs should take every necessary precautionary measures against macroshock and microshock injuries to both patients and staff. Some such measures are:

1. Electrical Systems

(a) *Core balance relay*

This monitors the current in the active and neutral wires. A short circuit will create a difference between the two wires, and activate a circuit breaker. The sensitivity of this system is not totally adequate for microshock protection, as the activating current is in mA rather than microA. However, this device can be designed to trigger after only 10 milliseconds, which limits the duration of a potentially lethal current, thus providing protection against macroshock.

(b) *Earthing*

All electrical equipment should be earthed at the same potential. A very low resistance wire should be used to achieve this. External equi-potential earth points can be used to earth equipment.

(c) *Mains isolated equipment*

Battery operated equipment, or equipment with a transformer in the patient circuit separate from the mains circuit, offer increased safety.

(d) *Isolation transformers*

The use of a 1:1 transformer to convert the supply of the ICU (or a bank of outlets) into a floating supply, with the incorporation of an earth-linkage alarm system, offers the safest protection.

2. Equipment Checks

The purchase of new equipment should be strictly controlled by a qualified person, and circuit diagrams should be provided with all new equipment. All pieces of apparatus should be checked for both function and current leaks prior to being used in the ICU. There should be regular checks on all equipment as machines which may have complied with safety regulations can deteriorate with age. Dated stickers should be applied on all equipment, and no piece of electrical apparatus without a current sticker should be used.

3. Personnel

Staff must take care when using any electrical or electronic equipment. Cords with frayed wires should never be used. Plugs should not be disconnected by pulling on the cords. All plugs should be clear plastic so that wire connections can be viewed. Extension cords should be avoided. Trolleys should not be wheeled over electrical cords. All staff involved

in using equipment should be aware of the principle of microshock. Two pieces of equipment should not be handled at the same time, nor should equipment be adjusted while in touch with the patient or the bed.

All hospitals should have an electrical safety committee which meets regularly to make hospital pollicies. There should be a qualified electrical safety officer who should regularly check all equipment, including leakage current when the apparatus is switched off. The requirements for equipment in the ICU should be strictly enforced. Australian Standards (AS 3003 and ASS 3200) set minimum requirements for Australian hospitals. AS 2500 has much useful information regarding the safe use of electricity in patient areas. Other countries have their own standards and safety regulations.