17 Respiratory and cardiovascular support

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17.1 Introduction

Our survival depends on the production of energy in the form of adenosine triphosphate by the process of oxidative metabolism. This in turn depends on a continual supply of substrates, particularly oxygen and glucose, to vital tissue beds such as the brain and myocardium. In a critically ill patient, the supply of oxygen may be compromised while at the same time metabolic demand is increased. Inadequate oxygen delivery may become the factor mitigating against recovery or even survival of the patient (Schumacker and Cain, 1987). In a patient with brain injury, cerebral metabolic reserve is already reduced by the primary insult and subsequent oxygen lack is a potent cause of avoidable secondary brain injury (Chesnut et al., 1993).

This chapter will address fundamental physiological principles of oxygen delivery from the atmosphere to the cells, the pathophysiological effects of head injury on oxygen delivery and principles of resuscitation and the maintenance of cardiopulmonary homeostasis.

17.1.1 THE PATIENT AT RISK

Although severe head injury is most common in healthy young adults, particularly males, a proportion of patients may suffer from pre-existing medical conditions, which may be exacerbated by the stress of neurotrauma and complicate its management (Luce, 1993; Kaufman et al., 1993). Ischemic heart disease and hypertension mandate meticulous attention to mean perfusion pressures of brain and myocardium; these patients may require higher pressures than the normal population to ensure optimal oxygen delivery. Control of excessive centrally mediated sympathetic activity is important in reducing both cerebral and myocardial oxygen demand. Respiratory problems such as asthma or chronic obstructive pulmonary disease may affect mechanical ventilation.

Pre-existing nervous system disorders such as epilepsy, cerebrovascular or neuromuscular disease may make assessment of effects of trauma and prognosis more difficult.

Renal and hepatic dysfunction, particularly if there is pre-existing impairment, may alter pharmacokinetics and drug interactions, leading to either toxic or subtherapeutic effects. As an example, phenytoin, frequently administered for prophylaxis of post-traumatic seizures, is a potent inducer of hepatic enzymes and will alter the pharmacokinetic profiles of lipid soluble drugs such as β-adrenergic blockers and opiates (Runciman, Myburgh and Upton, 1990).

Operative intervention may potentiate the acute stress response, even in the young previously fit patient (section 15.8).

17.1.2 THE ROLE OF INTENSIVE CARE

Management of head-injured patients in the Intensive Care Unit (ICU) aims to optimize oxygen and substrate delivery to vital tissues and to detect events that may harm the injured brain before they do damage.

This requires prompt detection of potentially harmful changes by accurate monitoring and early intervention (Borel et al., 1990; Andrews, 1993). All aspects of the patient’s state must be taken into account, with particular attention to the cardiorespiratory, renal, metabolic and nutritional systems (Bloomfield, 1989).

Although ICUs were established to preserve life and improve the quality of survival, they are frequently the setting for complications that increase morbidity and mortality. Complications may result from intravascular catheters or from mechanical ventilators. Nosocomial infection (Rodriguez et al., 1991) is a major source of increased morbidity and prolonged ICU stay (Hsieh et al., 1992). The acute respiratory distress syndrome and multiple organ system failure may occur as a result of the initial insult (Piek et al., 1992).
It is therefore important that the risk of each procedure or drug administered is balanced by a net benefit to the patient. To minimize complications and to maximize patient benefit, all clinicians involved in management must understand cardiopulmonary and metabolic pathophysiology and the particular effects of head injury. This applies both to neurosurgeons and intensivists.

17.2 Physiology

Optimal cardiorespiratory function exists when oxygen delivery from the atmosphere to the cell matches oxygen demand (Figure 17.1; Nunn, 1987a).

After a severe head injury, the brain is very vulnerable to secondary insults, and remains so for a period of perhaps 2–3 weeks. The oxygen cascade may be threatened at a single link in the pathway, for example by airway obstruction, but more often several links are threatened simultaneously. The threat may be primarily to the respiratory system (e.g. by chest trauma or pneumonia) or to the circulatory system (e.g. by hypovolemia or myocardial failure). Continued impairment of oxygen delivery may lead to multiple organ failure, especially after traumatic injuries and sepsis.

Understanding the physiological determinants of the various stages of the oxygen cascade may provide guidance for optimizing cellular oxygen tension. (West, 1985; Nunn, 1987a).

17.2.1 DISTRIBUTION OF OXYGEN FROM THE ATMOSPHERE TO THE ALVEOLI

The main determinants of alveolar oxygen tension ($P_{A\text{O}_2}$) are inspired oxygen tension and alveolar ventilation.

(a) Inspired oxygen tension

Inspired oxygen tension ($P_{I\text{O}_2}$) is determined by the fractional concentration of oxygen ($F_{i\text{O}_2}$) and the ambient barometric pressure (BP) corrected for saturated water vapor pressure:

$$P_{I\text{O}_2} = F_{i\text{O}_2} \times (BP - P_{\text{wat}})$$

where $P_{\text{wat}}$ represents the saturated water vapor pressure (47 mmHg).

$P_{I\text{O}_2}$ is proportional to $F_{i\text{O}_2}$ if barometric pressure and saturated water vapor pressure remain constant. Ambient air has an oxygen concentration of 21%, so that by administering 100% oxygen with a closed circuit $P_{I\text{O}_2}$ may be raised from 150 mmHg in air to 700 mmHg. This is achieved by alveolar denitrogenation since ambient air contains 80% nitrogen.

Hence, since $P_{I\text{O}_2}$ can be effectively and markedly raised by the administration of supplemental oxygen, all severely injured patients should receive supplemental oxygen at the highest percentage available as early as possible, to maximize the supply of oxygen to the cells. (The effect of altitude on $P_{I\text{O}_2}$ is discussed in Chapter 15.)

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**Figure 17.1** Transfer of oxygen from ambient air to mitochondria and summary of factors that influence oxygenation at different levels in the oxygen cascade. $P_{E\text{O}_2}$ = end-expired gas. See text for details. (Source: modified from Nunn, 1987a, p. 243, with permission.)
Table 17.1 Capacities of available oxygen delivery systems.

<table>
<thead>
<tr>
<th>Apparatus/device</th>
<th>Oxygen flow (l/min)</th>
<th>Inspired oxygen concentration (%)</th>
</tr>
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<tbody>
<tr>
<td>Nasal catheters</td>
<td>2</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>35</td>
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<tr>
<td></td>
<td>6</td>
<td>45</td>
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<tr>
<td>Semi-rigid masks (e.g. Hudson, Edinburgh)</td>
<td>4</td>
<td>35</td>
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<tr>
<td>Semi-rigid masks (e.g. Harris, Mary Catterell)</td>
<td>8</td>
<td>50</td>
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<tr>
<td></td>
<td>10</td>
<td>60</td>
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<td></td>
<td>12</td>
<td>65</td>
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<tr>
<td>Venturi type mask (e.g. Ventimask, Accurox)</td>
<td>8</td>
<td>40, 60</td>
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<tr>
<td>Reservoir plastic masks</td>
<td>4–8</td>
<td>40–80</td>
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<td>(e.g. Pneumask, Polymask)</td>
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<tr>
<td>Anesthetic circuits</td>
<td>Variable</td>
<td>21–100</td>
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<tr>
<td>Ventilators</td>
<td>Variable</td>
<td>21–100</td>
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Table 17.1 shows the oxygen delivery capacities of available oxygen circuits.

(b) Alveolar ventilation

Adequacy of ventilation is reflected by the $P_aCO_2$, normally 40 mmHg at sea level. Hypercapnic respiratory failure is defined as a $P_aCO_2$ greater than 50 mmHg at sea level unless there is a compensatory metabolic alkalosis. (Serum bicarbonate concentration indicates whether hypercapnia is acute or chronic, as renal compensation for chronic hypercapnia takes 2–3 days.)

$P_AO_2$ is related directly to alveolar ventilation and inversely to arterial carbon dioxide tension ($P_aCO_2$). This relationship is expressed in the alveolar gas equation:

$$P_AO_2 = P_{E}O_2 - P_{a}CO_2 / RQ$$

where $RQ$ is the respiratory quotient and reflects the ratio of oxygen consumption to carbon dioxide production; normally 0.8.

The $P_AO_2$ in normal adults breathing air at sea level is 150 mmHg.

Hypercapnia may reduce $P_AO_2$ by displacing oxygen from the alveoli. Hypercapnia-induced respiratory acidosis may also reduce the consciousness level. However, this acidosis rapidly resolves on restitution of effective alveolar ventilation. The acute stress response to injury also increases basal metabolic rate and carbon dioxide production which may further reduce $P_AO_2$. The respiratory quotient usually remains within the normal range due to an associated increase in oxygen consumption ($V_O_2$).

The other determinant of $P_aCO_2$ is carbon dioxide production ($V_{CO_2}$); normally 200 ml/minute in a healthy person. An increase in carbon dioxide production or decrease in effective alveolar ventilation ($V_A$) will increase $P_aCO_2$ as expressed in the relationship:

$$P_aCO_2 = V_{CO_2} / V_A$$

Adequacy of ventilation is assessed by arterial blood gas analysis obtained from repeated arterial punctures or preferably from indwelling arterial catheters. A continuous approximation of $P_aCO_2$ may be gained from the end-tidal carbon dioxide tension measured by a capnograph. Continuous end-tidal CO$_2$ tension may not represent $P_aCO_2$ in ventilated patients, particularly if there is coexistent lung injury or disease, and should be checked frequently against $P_aCO_2$ (Liu, Lee and Bongard, 1992). End-tidal carbon dioxide is usually slightly less than $P_aCO_2$ but wide limits of agreement between these parameters have been demonstrated (Russell, Graybeal and Strout, 1990; Myburgh, 1991, Pansard et al., 1992).

Ventilatory variables such as respiratory rate ($f$), tidal volume ($V_T$) and their product, minute volume ($V_E$), may be accurately measured with most modern ventilators. Alveolar ventilation ($V_A$) is difficult to determine and cannot be measured directly, although alveolar ventilation may be inferred if $V_E$ and dead space ventilation ($V_D$) are calculated:

$$V_A = V_E + V_D$$

Dead space refers to those airways that are ventilated but take no part in gaseous exchange and consists of both conducting airways (anatomical dead space) and non-perfused alveoli (physiological dead space; Nunn, 1987a). The normal ratio of dead space to tidal volume ($V_D / V_T$ ratio) is 0.3. This may be calculated in patients whose $P_aCO_2$ and expired carbon dioxide tensions ($P_{E}CO_2$) are known, using the modified Bohr equation:

$$\frac{V_D}{V_T} = \frac{P_{E}CO_2 - P_{a}CO_2}{P_{a}CO_2}$$

This ratio is an index of effective ventilation and is helpful in determining the time to wean from mechanical ventilation. The $V_D / V_T$ ratio in mechanically ventilated patients with normal lungs is approximately 0.5. A ratio of 0.6 or greater is associated with inadequate alveolar ventilation and hypercapnia.

In acute head injury, impaired conscious state is associated with a loss of protective airway reflexes and hypoventilation. Abnormal breathing patterns may be exacerbated by alcohol, drugs, respiratory injury or disease, resulting in alveolar hypoventilation and a rise in $P_aCO_2$ (North and Jennett, 1974; Demling and Riessen, 1990). Hypoxia then rapidly
ensues and is potentiated by an increase in oxygen consumption (Pfenninger and Lindner, 1991). This reinforces the need for an adequate airway, ventilation and supplementary oxygen in these patients.

(c) Lung mechanics
In addition to respiratory rate, tidal and minute volume, other aspects of lung mechanics, in particular lung compliance and airways resistance, are important for effective alveolar ventilation in both spontaneously breathing and mechanically ventilated patients (Bone, 1985; Nunn, 1987b).

17.2.2 DETERMINATION OF THE ARTERIAL OXYGEN TENSION

Arterial oxygen tension ($P_aO_2$) is determined by the diffusion capacity of the alveolar–capillary membrane, pulmonary shunting and the relationship between ventilation and perfusion. $P_aO_2$ is frequently a major limiting step in the transfer of oxygen to the tissues.

Diffusion of oxygen across the short diffusion distances of the alveolar capillary membrane normally results in a negligible reduction in $P_aO_2$.

Ventilation–perfusion scatter or mismatching of ventilation and perfusion (normally by ±4 mmHg) occurs through shunt and increased dead space ventilation and reduces both oxygen and carbon dioxide transfer.

Shunt is defined as perfusion of unventilated areas of the lung. Normally a small shunt of approximately 4 mmHg exists as a result of the bronchial and cardiac venous (Thebesian) circulations. Important features of true shunting are that hypoxia due to shunting cannot be abolished by increasing the $F_iO_2$, and the degree of resulting hypoxia is proportional to the shunt fraction.

Mismatch is increased in patients with underlying chronic airways disease or pulmonary hypertension. Neuromuscular ventilation–perfusion mismatch has been described as an uncommon complication of acute head injury (Schumacker et al., 1979). This may be exacerbated by acute intercurrent pulmonary pathology such as contusion, aspiration or infection, or simply by placing patients on mechanical ventilators (Hubmayr, Abel and Rehder, 1990). Pulmonary thromboembolism may cause severe hypoxia through both ventilation–perfusion mismatch and reduction in cardiac output (Demling and Riessen, 1990).

These three factors – diffusion gradient, shunting and ventilation perfusion mismatch – are responsible for a normal alveolar–arterial oxygen gradient of approximately 10 mmHg, determined by subtracting a measured $P_aO_2$ from $P_AO_2$ calculated by the alveolar gas equation.

Potentiation of any of these physiological causes of reduced arterial oxygen tension by pathological processes will result in hypoxia.

An increased alveolar–arterial oxygen gradient after head injury is most often due to alveolar obstruction or collapse caused by contusion, aspiration of gastric content or blood or consolidation from infection. Neuromuscular ventilation–perfusion mismatch is rare.

17.2.3 DELIVERY OF OXYGEN FROM THE ALVEOLI TO THE TISSUES

Oxygen delivery ($\dot{D}O_2$) to individual cells involves convective and diffusive oxygen transport (Leach and Treacher, 1992).

Convective oxygen transport refers to the bulk movement of oxygen in the blood. It depends primarily on the distribution of cardiac output to individual organs and the mechanisms regulating tissue microcirculation.

Diffusive oxygen transport is the process of uptake and extraction of oxygen from blood by the tissues (Leach and Treacher, 1992).

$\dot{D}O_2$ may be calculated as the product of arterial oxygen content ($C_aO_2$) and cardiac output ($\dot{Q}$):

$$\dot{D}O_2 = \dot{Q} \times C_aO_2.$$  

The normal value for $\dot{D}O_2$ is 900–1300 ml/min.

(a) Oxygen content

Arterial oxygen content ($C_aO_2$) is the total oxygen per milliliter of blood and includes both oxygen combined with hemoglobin and dissolved oxygen. $C_aO_2$ (normal value 16–20 ml $O_2$/100 ml blood) may be measured directly with oxygen content analyzers or calculated by the following equation:

$$C_aO_2 = (Hb \times 1.34 \times S_aO_2) + (P_aO_2 \times 0.003)$$

where $Hb$ is hemoglobin (g/dl); 1.34 is the oxygen carrying capacity of hemoglobin (ml/g); $S_aO_2$ is arterial oxygen saturation of hemoglobin (%); 0.003 represents the solubility of oxygen in blood in plasma at 37°C.

The chemical relationship between oxygen and hemoglobin is represented by the oxygen–hemoglobin dissociation curve. The idealized curve assumes a normal hemoglobin and $P_{50}$ (oxygen tension with 50% hemoglobin saturation). A number of logarithmic equations have been designed to determine oxygen saturation from a single $P_aO_2$ measurement (Breuer et al., 1989). However, $P_{50}$, which is influenced by pH and 2,3-diphosphoglycerate, a marker of intracellular metabolism and utilization of oxygen, is frequently reduced in critically ill patients (Myburgh, Webb and
Worthley, 1991). Wide limits of agreement between calculated and measured saturations have been demonstrated. Therefore oxygen saturations, either arterial or venous, should be measured directly by oximetric analysis when calculating oxygen delivery or consumption (Myburgh, 1992). This also applies to the measurement of jugular venous bulb oxygen saturation (see below).

The major determinant of arterial oxygen content is hemoglobin. Dissolved oxygen is a minor component of the total. Anemic or bleeding patients have a reduced oxygen transport capacity; hence transfusion of blood is an integral part of resuscitation in trauma patients.

There is contention on the optimal hemoglobin level in severely head-injured patients with coexisting extracranial injuries (Dhainaut et al., 1990; Greenberg, 1990) and consideration must be given to the risk and benefits of small blood transfusions intended to raise the hemoglobin level by only 1–2 g/dl (Dietrich et al., 1990). There is increasing evidence that a fall in hemoglobin is part of the early response to trauma (Crosby, 1992), and that single unit transfusion does not significantly increase tissue oxygen utilization and may cause splanchnic ischemia (Silverman and Tuma, 1992). Impairment of immune function and the risk of transmitting infection are additional arguments against single unit transfusion in critically ill patients.

Optimal tissue oxygen extraction from hemoglobin to the brain and myocardium occurs at a hematocrit of around 0.32 (Dantzker, 1989). It is not possible to recommend a specific hemoglobin or hematocrit for all clinical situations. However a hemoglobin of 8 g/dl is regarded as the transfusion limit for young and otherwise healthy trauma victims.

(b) Cardiac output

Cardiac output is the product of heart rate (HR) and stroke volume (SV). Any reduction in cardiac output will reduce oxygen delivery.

Heart rate, normally 70 beats per minute, is determined by autonomic influences on intrinsic cardiac pacemakers and is the principal factor maintaining cardiac output in the traumatized patient (Schumacker and Cain, 1987; Guyton, 1991). The heart rate response may be blunted in patients on beta-blockers or in patients with high spinal injuries causing cardiac denervation (Luce, 1985).

Stroke volume, normally 70 ml/beat, is determined by three factors:

- **preload**, defined as end diastolic cardiac muscle fiber length and representing the volume status of the patient;

- **contractility**, the inotropic state of the myocardium;

- **afterload**, the impedance to ventricular ejection.

Preload is the most important determinant of stroke volume after trauma (Dhainaut et al., 1990). Reduced preload, i.e. hypovolemia, must be aggressively treated to ensure adequate tissue oxygen delivery. Impaired contractility may result from direct myocardial injury or pre-existing ischemic cardiomyopathy. Drugs (inotropes) are being increasingly used to maintain adequate contractility once hypovolemia has been corrected, not only to promote oxygen delivery but also to maintain cerebral and myocardial perfusion (Schumacker and Samsel, 1989; Dhainaut et al., 1990).

Mean arterial pressure (MAP) is the product of cardiac output and total peripheral resistance, derived as systemic vascular resistance (SVR) from pulmonary artery catheter measurements.

17.2.4 PERIPHERAL EXTRACTION AND UTILIZATION OF OXYGEN

The uptake and extraction of oxygen from blood by the tissues are dependent primarily on diffusive oxygen transport (Leach and Treacher, 1992). This refers to the transfer of oxygen from blood through the extracellular matrix along a capillary–intracellular oxygen tension gradient; the process depends on arterial oxygen tension. Alterations in microvascular tone by neurohumoral (myogenic) and metabolic means adjust local oxygen delivery to changing metabolic demands. Microvascular beds in different tissues respond to increased oxygen requirements either by increasing flow or by recruiting additional vessels. For example, skeletal muscle can substantially increase the number of functioning capillaries in response to the increased metabolic demand, whereas the myocardium and brain are more dependent on changes in pressure and flow.

In acute neurotrauma, it is essential to maintain adequate cardiac output and cerebral perfusion pressure in order to sustain flow-dependent oxygen transport. Interstitial edema, by increasing the distance for diffusion, also jeopardizes oxygen transfer (Schumacker and Samsel, 1989).

Assessment of oxygen utilization by tissues is difficult and in general only global oxygen consumption and delivery can be measured easily. Global (i.e. whole body) oxygen consumption (normally 175–300 ml/min) is best measured by indirect calorimetry of expired respiratory gases using metabolic monitors. The accuracy of these monitors is reduced at high inspired oxygen concentrations ($F_{O_2} > 0.8$) and by air leaks (Takala et al., 1989). Oxygen consumption may be determined by the Fick equation, which...
expresses the relationship between cardiac output ($\dot{Q}$), oxygen consumption ($\dot{V}O_2$) and the arteriovenous oxygen content difference:

$$\dot{V}O_2 = \dot{Q} \times (C_aO_2 - C_vO_2).$$

The measurement and manipulation of oxygen delivery and extraction have been extensively debated (Vincent, 1990; Dantzker, Foresman and Gutierrez, 1991; Shoemaker, Appel and Kram, 1992; Reed, 1993). When oxygen delivery is reduced under physiological conditions, oxygen consumption is maintained until very low levels of oxygen delivery through increasing oxygen extraction. Below a critical oxygen delivery level oxygen consumption falls even though oxygen extraction continues to increase (Schumacker and Cain, 1987).

Under pathological conditions, such as trauma and sepsis, the relationship between oxygen delivery and consumption is more linear and a critical oxygen delivery level cannot be identified. Recent studies indicate that outcome in patients is improved if oxygen consumption matches delivery, if necessary through cardiovascular support (Shoemaker et al., 1988; Reinhart, Hannemann and Kuss, 1990).

The clinical measurement of both oxygen consumption and delivery remains difficult and is compounded by mathematical errors (Archie, 1987; Reed, 1993). Elevated serum lactate does not necessarily imply anaerobic metabolism, and its value as an index of regional oxygen demand is limited (Silverman, 1991; Mizock and Falk, 1992).

### 17.3 Effects of head injury upon oxygen delivery

Severe head injury may impair oxygen delivery through the development of abnormal breathing patterns, activation of a systemic inflammatory response and centrally mediated sympathetic overactivity.

#### 17.3.1 BREATHING PATTERNS IN HEAD-INJURED PATIENTS

Variable breathing patterns may result from head trauma and other insults to the central nervous system (North and Jennett, 1974). Cheyne–Stokes or periodic respiration is associated with bilateral hemispheric supratentorial lesions; central neurogenic hyperventilation or apneustic respiration with pontine lesions, and ataxic respiration or apnea with medullary or midbrain lesions. However, in practice, the diffuse nature of the primary and secondary effects of trauma upon central brain structures means that these patterns are seldom of diagnostic value.

Abdominal paradox, an inward movement of the abdominal wall during inspiration, indicates abnormal diaphragmatic contraction and may be seen in patients with high cervical spinal cord injury above or involving the phrenic nerve nuclei (level C3–C5; Luce, 1985). Chest trauma causing flail chest and pulmonary contusion results in tachypnea and intercostal muscle retraction (Demling and Riessen, 1990; Luce, 1993).

#### 17.3.2 SYSTEMIC PHYSIOLOGICAL RESPONSE TO ACUTE INJURY

The systemic physiological response to acute injury and infection causes a potent, mediator-induced, non-specific syndrome termed the systemic inflammatory response syndrome (SIRS). This syndrome has been extensively studied (Bone, 1991a, b; Bone et al., 1992a; Vincent and Bihari, 1992) and includes one or more of the following clinical manifestations:

- body temperature greater than 38°C or less than 36°C;
- heart rate greater than 90 beats/min;
- tachypnea, with a respiratory rate greater than 20 breaths/min or hyperventilation, as indicated by a $P_{aCO_2}$ less than 32 mmHg;
- white blood cell count greater than 12 000/mm$^3$ or less than 4000/mm$^3$, or containing more than 10% immature neutrophils (bands) representing an acute alteration from baseline in the absence of other known causes such as chemotherapy, induced neutropenia and leukopenia.

Infection may produce a systemic inflammatory response syndrome. Non-infectious pathological events such as multiple trauma, hemorrhagic shock, ischemia and pancreatitis result in an identical syndrome (Bone et al., 1992a).

**SIRS is mediated through a number of cytokines such as tumor necrosis factor (Strieter, Kunkel and Bone, 1993; Tracey and Cerami, 1993), interleukins (Dinarello, 1984; Plataniás and Vogelgang, 1990) and arachidonic acid metabolites (Lewis, Austen and Soberman, 1990).** The organ system responses, particularly the cardiovascular, respiratory and renal responses, may either regress or progress to well-defined clinical conditions such as acute lung injury, septic shock, acute renal failure and multiple organ dysfunction syndrome (Clarke, 1985; Cerra, 1992). Hemodynamic hallmarks of this response are a high cardiac output state due to reduced systemic vascular resistance and global blood flow redistribution. Cardiac output and stroke volume are usually well maintained during the initial acute stages of this response and may reflect a mediator-induced increase in oxygen delivery to vital tissue beds.

It is important to recognize the systemic inflammatory response syndrome and to actively seek infectious and/or non-infectious causes. If infection occurs,
SIRS may progress to septic shock, maldistribution of blood flow, myocardial depression and end organ failure. (Bersten and Sibbald, 1989).

17.3.3 NEUROHUMORAL RESPONSE

In addition to the acute systemic inflammatory response, sympathetically mediated responses following acute head injury result in three clinical symptom complexes: neurogenic hypertension, myocardial ischemia and neurogenic pulmonary edema.

(a) Neurogenic hypertension

Neurogenic hypertension is common after head injury. It appears to be sympathetically mediated and is directly proportional to the degree of catecholamine release (Graf and Rossi, 1978). Brain areas implicated include the hypothalamus, the nucleus of the tractus solitarius, the area postrema and areas A1 and A5 of the medulla (Kirland and Wilson, 1991). Increased ICP, brain-stem compression or medullary ischemia may cause severe systemic hypertension, accompanied or preceded by vagally mediated bradycardia (Graf and Rossi, 1978). This is the classical Cushing response but in practice it is not always seen; the hypertensive response can be masked by coexisting hypotension due either to hypovolemia or to a hyperdynamic systemic inflammatory response.

In as many as 25% of patients with neurogenic arterial hypertension, there is no associated increase in ICP (Simard and Bellefleur, 1989).

Unlike essential hypertension, neurogenic hypertension may be quite labile, with varying cardiac output and systemic vascular resistance, probably due to mediator-induced vasoresponsiveness. When or whether to treat neurogenic hypertension is still controversial, since an elevated systemic arterial pressure may represent an intrinsic compensation for impaired cerebrovascular autoregulation and low cerebral perfusion pressure. Cerebral autoregulation may be abolished or impaired in the injured brain, either regionally or globally, so that the normal relationship between CPP and blood flow is disrupted (Obrist et al., 1979) and cerebral blood flow becomes perfusion pressure-dependent (Simard and Bellefleur, 1989). Theoretically, elevating systemic arterial pressure may benefit ischemic areas, but may also increase hydrostatic pressure and vasogenic cerebral edema. Conversely, low arterial pressures may increase ischemia, cellular hypoxia and cytotoxic cerebral edema (Kong et al., 1991).

Neurogenic hypertension should only be treated when mean arterial pressure approaches 150 mmHg and sedation or analgesia with opiates and/or benzodiazepines has already been employed without success. Beta-blocking drugs appear to be preferable, particularly when ICP is elevated (Robertson et al., 1983), but are seldom needed.

(b) Myocardial injury

Myocardial injury, in the absence of coronary artery disease, may occur in up to 50% of head-injured patients. The lesions are similar to those seen after acute myocardial infarction, subarachnoid hemorrhage, pheochromocytoma or prolonged catecholamine infusions, where subendocardial hemorrhages are commonly found at autopsy. Inhibition of both catecholamine release (Talman, 1985) with adequate sedation and beta-adrenergic receptor blockade (Neil-Dwyer et al., 1978; Cruikshank et al., 1987) have been shown to prevent or ameliorate myocardial injury after head injury.

Although the brain effector site is unknown, there may be an association between hypothalamic lesions and myocardial damage. In patients with myocardial injury, cardiac isoenzymes may be elevated and electrocardiogram changes consistent with an infarction pattern can appear. These changes may be potentiated by pre-existing ischemic heart disease or hypertension. It has been suggested that electrocardiogram changes may indicate patients with more serious brain injury and thus a higher mortality (Kirland and Wilson, 1991).

Similarly, a wide range of cardiac dysrhythmias may occur as a result of centrally mediated sympathetic and vagal discharge. The true incidence of dysrhythmias is unknown and all patients with a severe head injury should have electrocardiograph monitoring. Tachyarrhythmias are predominantly supraventricular and include atrial fibrillation or flutter and paroxysmal or multifocal atrial tachycardias (Cruikshank et al., 1987). Ventricular tachyarrhythmias are uncommon. Reducing sympathetic tone with adequate sedation or occasionally with beta-blockade is usually sufficient treatment for tachyarrhythmias. In patients with poor ventricular function persistent hemodynamically significant tachyarrhythmias are best treated with class 3 agents such as amiodarone. Nodal and sinus bradycardias are quite commonly associated with sustained intracranial hypertension and rarely require treatment. Atropine, beta-agonists or, rarely, transvenous pacing may be indicated if hemodynamic compromise results.

These cardiovascular conditions are seen frequently during the initial stages of neurotrauma. However, hypertension may persist into the rehabilitation phase. In a study of head-injured patients in a rehabilitation center, 11% had systemic hypertension yet only one had a history of essential hypertension and 68% were...
less than 30 years of age. At discharge, 63% still required antihypertensive medications. A total of 21% of these patients had non-specific ST–T wave changes on their electrocardiograms (Kalizky et al., 1985).

(c) Neurogenic pulmonary edema
The most dramatic of these sympathetically mediated processes is neurogenic pulmonary edema. It was reported many years ago to be present in most patients who died of intracranial hemorrhage (Weismann, 1939) and in patients with severe isolated head injury (Simmons et al., 1969), but was much less common in patients who survived (Singbartl, Cunlitz and Hamrouni, 1982). The common locus of damage is the brain stem. The mechanism is a large sympathetic discharge (Denling and Riessen, 1990), which leads to a redistribution of blood from the systemic to the pulmonary vasculature and a rapid increase in pulmonary vascular pressures. This is a transient phenomenon but it may damage the pulmonary endothelium, causing a persistent permeability defect (Theodore and Robin, 1976).

This mechanism alone does not explain all cases of neurogenic pulmonary edema. Other mechanisms that may be involved include: neurogenic depletion of pulmonary surfactant (Beckman, Bean and Baslock, 1974); direct sympathetic control of the number and size of pulmonary endothelial pores (Rosell, 1980); constriction of the pulmonary lymphatics by the sympathetic discharge (McHale and Roddie, 1983); lung microembolization from activation of the clotting cascade, possibly caused by release of brain tissue thromboplastin into the systemic circulation (Kaufman et al., 1980; Simpson, Speed and Blumbergs, 1991); and the direct effects of increased sympathetic activity on lung vasculature resulting in pulmonary hypoperfusion and hypoxia (Balk and Bone, 1983).

Typically, neurogenic pulmonary edema has an immediate onset and becomes clinically evident 2–12 hours after injury. Occasionally clinical onset may be delayed from 12 hours to several days. It is usually self-limiting and, if the patient survives, it will resolve in hours to days. A prolonged episode of pulmonary edema would suggest other causes of either cardiogenic or non-cardiogenic origin.

The clinical findings in patients with neurogenic pulmonary edema are tachypnea, hypoxia, decreased pulmonary compliance, diffuse alveolar and interstitial ‘fluffy’ infiltrates on chest X-ray and low filling pressures measured by right heart catheters (cf. acute respiratory distress syndrome).

Treatment is primarily supportive and aims at ensuring adequate oxygenation and ventilation. This usually requires endotracheal intubation and mechanical ventilation with positive end-expired pressure (PEEP).

Sympathetic overactivity is usually abolished with adequate sedation and beta-blockade is usually unnecessary. Diuretics are effective but must be titrated against the volume status of the patient so that cerebral perfusion pressure is not compromised.

17.4 Management: resuscitation
The resuscitation phase is defined as the period from on-site resuscitation and evacuation to stabilization. Priority must be given to the most life-threatening problems and this is the principle of basic and advanced trauma life support systems (Buss, Nel and van den Heever, 1990; Trauma Committee, Royal Australasian College of Surgeons, 1992).

17.4.1 AIRWAY MANAGEMENT
Loss of airway patency and hypoxia at any stage of head injury management is a life-threatening event. It is the main cause of secondary brain injury and significantly increases morbidity and mortality (Gentleman, 1992; Chesnut et al., 1993).

All severely head-injured patients are at risk from inadequate airway protection. The degree of airway intervention necessary will depend on the level of consciousness and adequacy of protective glottic reflexes.

The mouth and upper airway must be inspected for foreign bodies, bleeding or ill-fitting dentures and cleared under direct vision using a rigid sucker. Simple maneuvers such as chin lift and jaw thrust and the use of oropharyngeal airways may be enough to allow efficient oxygenation in self-ventilating patients. Nasopharyngeal airways must be used with caution if cribiform plate fracture is suspected since they have been passed directly into the cranial cavity. They may also cause trauma to the nasal mucosa with bleeding into the nasopharynx and upper airway. The same cautions apply to the use of nasotracheal and nasogastric tubes.

Emergency endotracheal intubation is indicated whenever the airway is threatened, especially in the comatose head-injured patient. It is also indicated in faciomaxillary trauma or upper airway obstruction from direct laryngeal trauma. This is best performed where there is expertise in anesthesia and specific airway techniques such as rapid sequence induction and intubation, blind nasal intubation, fiberoptic laryngoscopy, cricothyroidotomy and tracheostomy. However, urgency may dictate intubation in less optimal circumstances.

Indications for early endotracheal intubation after head injury are:
• coma;
• inability to obey commands with evidence of respiratory compromise:
  – tachypnea >30/min
  – sternal recession
  – upper respiratory bleeding;
• blood gas analysis – $P_aO_2 < 70$ mmHg; $P_aCO_2 < 45$ mmHg.

Indication for cricothyroidotomy or tracheostomy are:

• coma and inability to intubate;
• trauma, bleeding or fractures involving the upper airway.

Comatose patients (Glasgow Coma Score less than 8) should be intubated electively as soon as possible to prevent aspiration of gastric contents, to allow effective oxygenation, ventilation and control of $P_aCO_2$ and definitive imaging with computed tomography (CT; Pfennninger and Lindner, 1991). Similarly, head-injured patients who are agitated or combative may best be managed by early intubation and controlled ventilation until diagnostic and therapeutic interventions are completed (Redan et al., 1991). These patients should be intubated orally using a rapid sequence induction of anesthesia after preoxygenation and with the application of cricoid pressure as described in Table 17.2 (Walls, 1993). Patients with significant extracranial injuries causing ventilatory failure must also be intubated early. Patients with moderately depressed or fluctuating conscious states may be observed provided adequate glottic reflexes are present and the cardiorespiratory state is stable.

All unconscious head-injured patients must be regarded as having cervical spine injuries and intubation should be performed with in-line cervical spine immobilization with the head in the neutral position so that neck flexion and extension is avoided. Patients may be intubated with rigid collars in place and these should be left in situ until definitive radiological views of the cervical spine are obtained (Crosby and Lui, 1990).

The best method of assuring correct placement of an endotracheal tube is by directly visualizing the passage of the tube through the vocal cords (Szekely et al., 1993). End-tidal carbon dioxide and a respiratory waveform on capnography (Lillie and Roberts, 1988), auscultation of the chest and misting on the endotracheal tube during exhalation give further confirmation of correct placement, although false positives have been described following inadvertent esophageal intubation (Holland, Webb and Runciman, 1993). Advancing the tube down the right main bronchus is a common error especially in children. It is best prevented by repeated auscultation and chest X-ray examination. Tubes must be secured at the appropriate length (normally 21 cm at the teeth in women and 23 cm in men). Adhesive tape is the most effective means of securing endotracheal tubes.

**Table 17.2** Priorities of rapid sequence induction of anesthesia for emergency intubation in neurotrauma. See text for further details. $ETCO_2 = $ end-tidal carbon dioxide tension

<table>
<thead>
<tr>
<th>Optimize conditions for intubation</th>
<th>Equipment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skilled assistant</td>
<td>Oropharyngeal airways</td>
</tr>
<tr>
<td>Adequate light</td>
<td>Self inflating hand ventilating assembly and mask</td>
</tr>
<tr>
<td>Suction</td>
<td>100% oxygen</td>
</tr>
<tr>
<td>Neutral position</td>
<td>Two laryngoscopes</td>
</tr>
<tr>
<td></td>
<td>Magill forceps</td>
</tr>
<tr>
<td></td>
<td>Introducer/bougie</td>
</tr>
</tbody>
</table>
|                                   | Endotracheal tubes (mm): $\Omega - 7.0, 8.0$
|                                   | $\Omega - 8.0, 9.0$
| Cricothyroidotomy equipment:      | Scalpel |
|                                   | 6.0 mm cuffed endotracheal tube |
| Drugs                             | Induction agent |
| Suxamethonium (1–2 mg/kg)         | Atropine (0.6–1.2 mg) |
| Adrenaline (10 ml 1:10 000 solution) | Sedation (fentanyl, diazepam, morphine, midazolam) |
| Monitoring                        | Pulse oximetry |
| Capnography                       | Arterial blood pressure |
| Electrocardiograph                | Procedure |
| Preoxygenation 100% oxygen for 3–4 minutes | Preload with 250–500 ml colloid intravenously |
| Manual in-line cervical spine immobilization | Cricoid pressure applied |
| Induction agent + suxamethonium | Direct visualization of vocal cords |
| Oral endotracheal intubation      | Inflation of cuff until sealed |
| Confirmation of end-tidal $CO_2$ and chest auscultation | Cricoid pressure released |
| Secure tube at correct length     | Consider naso- or orogastric intubation |
| Ensure adequate sedation ± muscle relaxant | Chest X-ray |
| Confirm blood gas analysis        |}

**Tracheostomy** is only indicated acutely in patients who cannot be intubated; e.g. those with laryngeal or
tracheal trauma or some patients with high cervical spinal cord injury.

17.4.2 VENTILATION

Once the airway is secured, all injured patients must receive oxygen at the highest concentration available (Oh and Duncan, 1988). The decision to continue ventilation is primarily a clinical one and is based on the severity and pattern of head injury, associated injuries and pre-existing lung function. Arterial blood gas analysis of $P_aO_2$, $P_aCO_2$ and pH are important but should not delay this decision.

For self-ventilating patients, face mask oxygen using the delivery systems outlined in Table 17.1 are suitable. For intubated patients, manual or mechanical ventilating assemblies can reliably deliver an $F_iO_2$ of 1.0. Hand-held, self-inflating assemblies can effectively ventilate both intubated and non-intubated patients and also give an impression of lung compliance during inflation, thereby reducing the risk of disconnection from either the endotracheal tube or the face mask (Table 17.1).

Emergency assessment of oxygenation may be difficult when an impaired conscious state may be due to head injury, alcohol, drugs or sedatives. Pulse oximetry has greatly aided this assessment and is now mandatory in trauma patients. A saturation greater than 95% generally indicates a $P_aO_2$ of at least 75 mmHg and is recommended.

Pulse oximeters are less reliable in hypoperfused, hypothermic and agitated patients (Runciman et al., 1993). Nevertheless, even under conditions of poor perfusion, a recorded saturation of less than 95% with a pulse waveform indicates hypoxia (Clayton et al., 1991). This must be corrected by delivering the highest possible oxygen concentration, reassessing the airway and ventilation, and excluding potential causes of unexplained hypoxia such as tension pneumothorax or cardiac tamponade. Arterial blood gas analysis should be performed as soon as possible, as this is the only means of calibrating a pulse oximeter.

For 30–40 years hyperventilation has been advocated for the initial reduction of intracranial pressure. Global and regional cerebral blood flow may be reduced by as much as 50–60% during the first several hours following injury. Acute hypocapnia may further reduce cerebral blood flow in areas of the brain where vasoresponse to hypocapnia is relatively preserved. This may or may not reduce ICP, yet the reduction in blood flow may worsen ischemia. Early hyperventilation is now only recommended in patients with clinical signs of herniation or rapid neurological deterioration, prior to imaging or measurement of ICP and providing adequate circulatory resuscitation is complete. The level of $P_aCO_2$ required to reduce cerebral blood flow and achieve optimal ICP is not well defined but, on balance, moderate hypocapnia of $P_aCO_2$ to not less than 30 mmHg is recommended. In the patient without clinical evidence of intracranial hypertension or brain shift, ventilation should be set to achieve normocapnia.

17.4.3 CIRCULATION

Hypotension after severe head injury is independently associated with significant increases in morbidity and mortality. Resuscitation protocols for brain-injured patients should aim assiduously at avoiding hypovolemic shock. (Buss, Nel and van der Heever, 1990; Chesnut et al., 1993; Hill, Abraham and West, 1993).

Restoring circulating blood volume is the basis of hemodynamic resuscitation in trauma patients. The initial assessment of circulatory status may be difficult since blood pressure may be well maintained by sympathetic activity. Tachycardia and reduced capillary return are cardinal signs of hypovolemia. However tachycardia is common after trauma and peripheral perfusion may be difficult to assess in hypothermic patients. Hence, 1–2 liters of balanced salt solution (lactated Ringer’s or physiological saline solution) should be infused initially in all patients through large-bore peripheral venous access until definitive monitoring of blood pressure and volume status is established. Extracranial sources of hemorrhage must be identified and rapidly treated. This requires a coordinated ‘trauma team’ approach where priorities are given to the most life-threatening injuries in the shortest possible time (Trunkey, 1991; Trauma Committee, Royal Australasian College of Surgeons, 1992).

Hypotensive patients should be given colloid solutions to expand the intravascular volume and restore preload. Normal (5%) serum albumin solution or synthetic polygeline are suitable solutions. The use of hypertonic solutions in neurotrauma remains controversial and is discussed in Chapter 16. Blood transfusion must be administered to patients who have lost more than 20–30% blood volume or where significant hemorrhage is anticipated. Patients who require anesthesia for intubation should have adequate preload and arterial blood pressure monitoring in situ as sudden ablation of sympathetic tone by anesthetic agents may unmask a compensated hypovolemic state and induce hypotension.

As a general principle vasopressors should not be used in hypovolemic patients until volume resuscitation is completed.

Causes of refractory hypotension in these patients include acute spinal injury, tension pneumothorax, cardiac tamponade and severe myocardial contusion. These must be excluded early.
17.4.4 SEDATION AND ANALGESIA

In the acute phase, sedation for intubated and ventilated patients must be titrated against the hemodynamic stability. High-dose narcotics (15–25 μg/kg fentanyl) and non-depolarizing muscle relaxation (pancuronium 8–10 mg) provide sufficient sedation and allow control of ventilation for 1–2 hours while imaging and other investigations are performed. This combination provides hemodynamic stability; moreover tachycardias due to vagal blockade by pancuronium are reduced with high doses of fentanyl (Salmenperä et al., 1983). Frequent reassessment of conscious state is essential as excessive sympathetically activity may potentiate raised intracranial pressure or myocardial ischemia in a paralyzed but awake patient (a state that should be carefully avoided). Sedation may need to be supplemented with intermittent doses of opiates or benzodiazepines.

17.4.5 MONITORING

(a) Arterial pressure

During the initial resuscitation phase, a clinical impression of cardiac output and tissue perfusion can be gained from the strength of peripheral pulses and the time required for superficial capillaries to refill after they have been blanched (normally 2–3s). Accurate measurement of mean arterial blood pressure and heart rate is essential.

Non-invasive blood pressure devices are inaccurate outside the normal range, tending to over-read in states of hypotension and under-read in hypertension (Rutten et al., 1986; Cockings et al., 1993). Radial, brachial or femoral arterial catheterization is recommended. The arterial impulse may be connected via a fluid-filled catheter to a transducer. This is the standard method for measuring systemic blood pressure in the ICU and may also be used to obtain samples for blood gas analysis (Runciman, Ilsley and Rutten, 1988). Local vascular responses at the radial arterial level may result in false elevations of or reductions in blood pressure (Pauca et al., 1992).

Mean arterial pressure correlates more closely with organ perfusion than systolic pressure and is less susceptible to the errors of measurement that occur with suboptimally damped transduced measuring systems. Optimal mean arterial pressure is usually 60–80 mmHg, although patients with pre-existing hypertension or raised intracranial pressure may require higher mean arterial pressures.

Complications of arterial catheterization such as local hematoma formation, ischemia distal to the site of insertion or local infection may be minimized by slow continual flushing of these catheters with dilute solutions containing heparin and by sterile catheter insertion and maintenance techniques.

(b) Electrocardiography

Electrocardiography is routine, but since changes in heart rate in response to hypoxia and hypotension occur late, the ECG is a poor marker of vital organ oxygenation (Ludbrook et al., 1993).

(c) Central venous pressure

Central venous pressure monitoring is a useful guide to volume status, but the insertion of these catheters should not delay the restoration of volume. Catheters should be placed under sterile conditions by skilled operators on a semi-elective basis and pressures should be measured with electronic transducers (see section 17.5.2(b)).

The response to fluid resuscitation is best assessed by improvements in heart rate, capillary return and mean arterial blood pressure, and by a maintained urine output of 0.5–1 ml/kg/h.

17.4.6 NEUROSURGICAL DIAGNOSIS

Once patients have been adequately resuscitated the definitive diagnosis of intracranial pathology, usually by CT scanning, is essential. In a patient who shows signs of brain-stem compression, evacuation of an intracerebral hematoma is a matter of great urgency. Resuscitation should therefore be conducted pari passu with the CT scan, or the patient should be taken directly to the operating room. Following CT diagnosis and/or evacuation of intracerebral hematomas, ICP and other monitoring devices such as jugular bulb oxygen catheters should be inserted.

Accurate, lightweight and portable monitors allow continuous display of ECG, heart rate, oxygen saturation and arterial pressure and are recommended during transport. This phase of treatment is covered in more detail in Chapters 15 and 18.

17.5 Management: maintenance of cardiopulmonary–cerebral homeostasis

After acute resuscitation and any necessary operative procedures comes the phase of management in which the chief aim is to prevent secondary hypoxic–ischemic brain injury. This phase continues until the patient no longer requires homeostatic control and support. During this period patients are usually managed in the Intensive Care Unit, with mechanical and/or pharmacological cardiorespiratory and metabolic support. The period of vulnerability to secondary brain injury is not
clearly defined and varies from patient to patient, but it may last up to 10–14 days after injury and beyond the time when intracranial pressure is elevated. Management of the patient requires close liaison between intensivists, neurosurgeons and other involved specialists.

All observations should be accurately recorded on 24-hour charts that correlate all vital signs and cumulative hourly fluid balance. This facilitates early identification of trends, a more important indicator of progress than single isolated recordings.

17.5.1 RESPIRATORY MANAGEMENT

(a) Airway management

Patients should be intubated until ICP is controlled (i.e. consistently below 20 mmHg) and respiratory function indices are satisfactory (see below). Endotracheal intubation may be performed either translaryngeally, through the nose or mouth, or transtracheally, via a tracheostomy. The development of polyvinylchloride tubes with low-pressure, high-volume cuffs has reduced the incidence of tracheal mucosal damage and ulceration from nasal or oral endotracheal tubes. As a result, translaryngeal intubation may continue as long as the head injury and lung function require it (Stone and Bogdonoff, 1992).

Nasal intubation gives good support to the endotracheal tube and patients often tolerate nasal tubes with little sedation. These tubes are less prone to move in the trachea than oral tubes and therefore cause less tracheal mucosal damage. Patients are able to swallow their secretions and oral hygiene is more easily maintained than with an oral tube. However suction through nasal tubes is more difficult because of their narrower diameter and greater length. Nasal intubation is contraindicated in patients with basal skull fracture especially when the cribriform plate is damaged.

Oral intubation is preferred in an emergency. It allows passage of a tube with a larger diameter (8 mm or more) and easier bronchial toilet. However these tubes are more prone to movement and therefore cause more leaks as a result of changing cuff compliance, tracheal dilatation with prolonged intubation or tube movement (Kearl and Hooper, 1993). Excessive cuff pressure may cause tracheal ulceration and later stenosis. Frequent volumetric cuff checks and cuff pressure measurements should be performed to ensure an adequate and safe seal (Rischbieth, Blight and Myburgh, 1994).

(b) Principles of ventilation

Ventilation must provide adequate oxygenation and control of arterial carbon dioxide tension.

Morbidity and mortality of patients supported by mechanical ventilation, especially those receiving prolonged support, remain very high. There has been increasing concern that iatrogenic problems relating to mechanical ventilatory support may be important contributing factors (MacIntyre, 1993).

Accidental disconnection from mechanical ventilators is the most common and potentially lethal complication of mechanical ventilation. All ventilators must have a dedicated disconnection alarm.

Other complications of ventilation are patient–ventilator dyssynchrony, alveolar overdistention and adverse effects of intrinsic positive end-expiratory pressure (auto PEEP).

Patient–ventilator dyssynchrony occurs either when the breath delivered by the ventilator is out of cycle with the spontaneous effort by the patient or when the response of the ventilator is not adequate to
meet the flow demands of the patient’s effort. This results in an increase in the pressure load placed on the respiratory muscles and a substantial increase in oxygen consumption and carbon dioxide production. This may augment respiratory muscle fatigue and failure (Mador, 1991; Oh et al., 1991). This is frequently and incorrectly described as the patient ‘fighting the ventilator’. The reverse is more appropriate: in reality the ventilator is fighting the patient. The resulting sympathetic overactivity may elevate ICP (Ersson et al., 1990). Similar effects occur as a result of coughing induced by endotracheal suctioning or of Valsalva maneuvers in restless, agitated patients (Walsh et al., 1989).

Optimizing the patient–ventilator interface requires adequate sedation and the use of a mode of ventilation that reduces the work of breathing.

**Sedation** is best achieved initially with a rapid onset sedative/narcotic combined with a muscle relaxant, such as fentanyl with pancuronium. Thereafter, combined infusions of a narcotic with a benzodiazepine (e.g. morphine and midazolam) should be titrated against the conscious state of the patient (Fisher and Raper, 1991). The degree of sedation required may vary widely according to the severity of the underlying head injury and individual pharmacokinetic differences. The total dose of drug infused is not important, but rather the titrated effect.

Propofol, a short-acting anesthetic induction agent given by infusion, is becoming popular for the treatment of head-injured patients. It has the theoretical advantage of a rapid and potent titrated sedation without prolonging ventilation. Consciousness returns rapidly on cessation of the drug because of its rapid distribution and metabolism. (Farling, Johnston and Coppel, 1989a, b). Propofol is a potent myocardial depressant and cerebral perfusion should be monitored carefully. Definitive studies analyzing the long-term use of this agent as a sedative have yet to be carried out.

**Neuromuscular blockade**

The use of non-depolarizing muscle relaxants in critically ill, ventilated patients should be questioned for two reasons. Firstly a paralyzed but awake patient is more susceptible to autonomic sympathetic swings which will exacerbating raised ICP and predispose to increased oxygen consumption. Secondly, a polyneuropathy has been demonstrated in ventilated patients who have received regular non-depolarizing muscle relaxants with or without steroids. This axonal degeneration significantly prolongs ventilation and is associated with increased extracranial complications and morbidity (Gooch et al., 1991; Hansen-Flaschen, Cowen and Raps, 1993; Hsiang et al., 1994).

**Alveolar overdistention**

There is now a clearer understanding of the pathophysiological consequences of alveolar overdistention in mechanically ventilated, critically ill patients. Previously, patients were ventilated with tidal volumes much larger (10–15 ml/kg) than those occurring in spontaneously breathing individuals. This is the standard method of ventilating patients during general anesthesia and was advised in order to prevent atelectasis by promoting recruitment of collapsed alveoli, to negate the adverse effects of increased dead space and to increase functional residual capacity. There is increasing evidence that this technique is dangerous in critically ill patients with injured lungs or in those who require prolonged (>24 h) ventilation. The consequences include initiating or exacerbating an acute lung injury (Tsumo, Prato and Kolobow, 1990; MacIntyre, 1993), barotrauma to the lung and impairment of cardiac output (Dreyfuss and Saumon, 1992). Pulmonary edema due to increased permeability has also been demonstrated in human and animal studies after several hours of ventilation with high tidal volumes (Dreyfuss and Saumon, 1993). This phenomenon, termed ‘volutrauma’, is related to the degree of alveolar volume and to distention rather than to peak airway pressure.

In patients and animals with an acute lung injury, large tidal volumes increase edema accumulation (and extravascular lung water), leading to increased shunt, impairment of oxygenation and a significant reduction in pulmonary compliance (Carlton et al., 1990; Sohma et al., 1992; Tsumo, Prato and Kolobow, 1990).

The inflating airway pressure is transmitted along interstitial bronchovascular planes and may result in pneumothorax, pneumomediastinum, pneumopericardium or pneumoperitoneum and increase in intrathoracic pressure. This will reduce venous return to the heart, increase the impedance to ventricular ejection and reduce cardiac output (Marini, 1990).

(c) Modes of mechanical ventilation

**Positive pressure ventilation** is the routine form of mechanical ventilation in current practice. Gas is delivered under positive pressure to either a preset pressure or preset volume. The mode selected for any patient should be based upon the lung mechanics of that patient (Bone and Eubanks, 1991; Sassoon, 1991; Burchardi, Sydow and Criece, 1994). For most patients with normal lungs a volume control mode will suffice.

**Volume-controlled ventilation** delivers a preset tidal volume at a preset rate. This mode of ventilation is suitable in patients with normal lung mechanics but may cause barotrauma and alveolar overdistention in patients with poorly compliant lungs. The mean
inspiratory pressure may be reduced (pressure-limited volume control ventilation) to prevent barotrauma, but at the expense of the tidal volume. This mode ignores spontaneous respiratory efforts by the patient and should be used only in paralyzed, deeply unconscious or anesthetized and sedated patients. Most portable transport ventilators are of this type and patients being transported usually need muscle paralysis and increased sedation to optimize ventilation (Phillips and Skowronski, 1986).

**Assisted mechanical ventilation** of various types is designed to optimize the patient–ventilator interface by using a volume control mode and allowing the patient to trigger the ventilator to deliver a preset tidal volume. This is accomplished by generating a small negative airway pressure or flow that activates the inspiratory cycle, the duration of which is determined either by the ratio of inspiration to expiration (I:E ratio) or by reaching a pressure limit. The sensitivity of the triggering mechanism must be such that the inspiratory efforts of the patient are detected without imposing an increased work of breathing and oxygen consumption, but not so sensitive that the ventilator will cycle in response to fluctuations in airway pressure.

**Synchronized intermittent mandatory ventilation (SIMV)** is the standard form of assisted volume controlled ventilation. This mode sets the mandatory minute volume delivered to the patient while reducing dyssynchrony by only delivering ventilated breaths at the end of a spontaneous expiration. The degree of mandatory ventilation is adjusted according to the spontaneous minute volume of the patient. Initially the ventilator respiratory rate may be set high enough to provide most, if not all, of the patient’s minute volume and then reduced as sedation is reduced or the underlying pathology improves. The potential benefits of SIMV include less patient ventilator dyssynchrony, lower sedation requirements, reduced mean airway pressure by combining spontaneous and ventilated breaths, and improved respiratory muscle function by allowing patients to breathe spontaneously (Bone and Eubanks, 1991). Disadvantages may include insufficient mandatory minute volume in unstable patients and the possibility of respiratory muscle fatigue in patients with inappropriately low ventilator rates. As with any volume control mode of ventilation, alveolar overdistention is a potential hazard; this can be minimized by using an upper peak inspiratory pressure limit above which the ventilator will not deliver a breath (usually 30–40 cmH₂O).

It has become routine to combine **inspiratory pressure support ventilation (PSV)** with SIMV in order to reduce the work of breathing through ventilator and endotracheal tubing during the spontaneous breaths of the patient (Bersten et al., 1989). In this mode, spontaneous inspirations are augmented with a level of positive airway pressure that is preset (usually to 20 cmH₂O) to achieve a desired tidal volume. As patients are weaned from SIMV by reducing the ventilator rate and/or tidal volume, the inspiratory pressure support will continue so that patients set their own minute volume without incurring increased respiratory effort. Pressure support ventilation alone is used chiefly to ensure an adequate minute volume during the weaning phase and in patients with adequate respiratory drive and minimal sedation. It is not suitable for the acute phase of injury, where control of minute ventilation and particularly of $P_{aCO_2}$ is desired (Brochard et al., 1989; Santak et al., 1991; Sassoon et al., 1991).

**Pressure controlled ventilation** delivers gas until a preset peak inspiratory pressure is reached at a given ventilator rate (Blanch et al., 1993a, b). The tidal volume is determined by the patient’s compliance and the I:E ratio. Increasing the inspiratory time in this mode has been advocated so that the I:E ratio is reduced (pressure control inverse ratio ventilation – PC-IRV) to progressively recruit collapsed alveoli. Inspiratory pressure therefore remains constant in order to maximize alveolar ventilation at the lowest inflation pressure, not only through the whole period of inspiration, but also when lung compliance improves. The shortened expiratory time is intended to prevent alveoli collapsing again and usually induces a higher auto PEEP. This mode of ventilation remains controversial and manipulations of the I:E ratio are best combined with frequent reassessment of both lung mechanics and auto PEEP and the effects on cardiac output and oxygen transport (Marcy and Marini, 1991; Mercat et al., 1993).

(d) **Positive end-expiratory pressure (PEEP)**

*Auto PEEP*

In addition to the factors outlined above, a subtle yet potentially hazardous effect is the presence of an elevated positive pressure on exhalation known as intrinsic or auto PEEP (Schmidt and Wood, 1993). Auto PEEP is seen typically in patients with airflow obstruction, but it can also occur without obstruction when minute ventilation is very high. Patients susceptible to the development of auto PEEP are those with asthma, acute on chronic respiratory failure or acute lung injury, where a prolonged expiratory time is necessary.

Auto PEEP was first described in ventilated patients (Pepe and Marini, 1982), but it can also occur in spontaneously breathing patients as a result of exhalation against the glottis, varying in magnitude from
1–10 cmH2O. These patients usually have degrees of airflow obstruction and the physiological effects of auto PEEP are those of extrinsically applied PEEP, namely alveolar recruitment to reduce shunt and increase functional residual capacity. The work of breathing to overcome auto PEEP may be significant. When such patients are ventilated, auto PEEP together with extrinsically applied PEEP may result in air trapping at end expiration and the risk of barotrauma, reduced cardiac output and increased physiological dead space (Fessler et al., 1991; Pinsky, Desmet and Vincent, 1992). The level of extrinsic PEEP applied must be adjusted in accordance with the level of auto PEEP so that alveolar overdistention and increased work of breathing do not occur.

Auto PEEP can be dangerous if undetected. It is therefore essential that auto PEEP be identified and measured in ventilated patients by stopping air flow at end expiration just before the next breath, allowing the pressure in the airways and the ventilator tubing to equilibrate and reading the pressure from a ventilator pressure gauge. Many new ventilators have the facility to monitor and display the level of auto PEEP (Gottfried, Reissman and Ranieri, 1992).

Extrinsic PEEP can be used to maintain alveolar recruitment and functional residual capacity throughout the ventilatory cycle in order to improve arterial oxygenation (Schmidt and Wood, 1993). Low levels of PEEP have also been shown to significantly reduce the work of breathing imposed by ventilators and endotracheal tubes, particularly when used with inspiratory pressure support (Bersten et al., 1989; Banner, Blanch and Kirby, 1993). Consequently, PEEP of 5–10 cmH2O is usually added to most ventilatory modes. Higher levels (more than 10 cmH2O) may reduce cardiac output and cause barotrauma; they are only used in patients with severe hypoxia who need high levels of inspired oxygen.

The application of PEEP above 10 cmH2O should be titrated against oxygen delivery to provide ‘optimal PEEP’ (Lin and Oh, 1990). Although the efficacy of PEEP in preventing atelectasis is not conclusively proven, it may be of some benefit in neurosurgical patients who cannot be positioned with head elevation. PEEP can also be used in spontaneously breathing patients, either through a tightly fitting face mask or an endotracheal tube, in which case it is called continuous positive airway pressure (CPAP). CPAP is useful in weaning patients with critical levels of auto PEEP or with reduced ventilricular function. The withdrawal or reduction of positive pressure ventilation or PEEP from these patients may worsen oxygenation and require hours or even days of therapy to reverse (Zwissler et al., 1991).

The use of PEEP in the head-injured patient has been controversial. The rationale for avoiding PEEP was that the increased intrathoracic pressure, transmitted via the venous system to the sagittal sinus and cerebral veins, would raise intracranial pressure. However there is evidence that PEEP only increases intracranial pressure if it reduces systemic arterial pressure (Frost, 1977; Shapiro and Marshall, 1978). The mechanism suggested is arteriolar vasodilatation induced by reduced cerebral perfusion pressure. On balance, PEEP probably does not adversely effect cerebrovascular dynamics unless there is an associated reduction in cerebral perfusion pressure, and PEEP should be used primarily to improve oxygenation in the head-injured patient.

(e) Control of carbon dioxide

Hyperventilation is of great value when ICP rises acutely and requires urgent reduction such as during resuscitation or prior to evacuation of intracranial hematomas.

Urgent hyperventilation must be employed carefully because vigorous manual inflation (‘banging’) or marked elevation of the rate of mechanical ventilation may decrease mean arterial pressure or increase mean airway pressures (Ersson et al., 1990). The temporary benefits derived from hypocapnia-induced vasoconstriction may be lost if systemic arterial pressure and hence cerebral perfusion pressure are reduced. Furthermore excessive vasoconstriction due to hyperventilation may result in a reduction of oxygen delivery to ischemic levels. Hyperventilation during early diagnosis and management should be used for as short a time as possible. If diagnostic studies show no mass lesion, and intracranial pressure is low, P$_{aCO2}$ should gradually be returned to normal.

Prolonged reduction of P$_{aCO2}$ to 25 mmHg or lower has been advocated for the last two decades without specific evidence that it reduces mortality or morbidity. Indeed, clinical studies have suggested that prolonged hyperventilation may be associated with a worse outcome (Jennett et al., 1980; Proctor et al., 1984; Muizelaar et al., 1991). Potential detrimental effects of prolonged hyperventilation include increased cerebral lactic acidosis and uncoupling of oxidative phosphorylation. Profound reduction in the cytochrome a$_{1}$–a$_{3}$ system has been attributed to cerebral oligemia induced by prolonged hypocapnia (Proctor et al., 1984; Rosner, 1987). Furthermore the vasoconstriction induced by hypocapnia is temporary. Animal and human studies have shown that despite continuous hyperventilation to maintain a P$_{aCO2}$ of 25 mmHg there is a steady loss of pial arteriolar constriction and a return of vessel diameters to baseline by 20 hours. During this period, arterial and cerebrospinal fluid pH return to normal. Return to normocapnia will now result in vasodilatation, an increase in cerebral blood
volume and rise in intracranial pressure (Muizelaar et al., 1988; 1991; Bouma and Muizelaar, 1992).

Hence continuous hyperventilation is probably only effective for 20–24 hours and subsequent attempts to return to normal \( P_{\text{aCO}_2} \) may increase intracranial pressure. Therefore if hyperventilation is used initially, it should only be continued until the level of intracranial pressure can be determined. Thereafter it should be used to treat acute elevations in intracranial pressure while other measures such as osmotherapy, ventricular drainage and the evacuation of mass lesions are being initiated (Pitts and Andrews, 1992). Routine prolonged hyperventilation, still practiced in many units, is difficult to justify.

(f) Management of ventilated patients

Once mechanical ventilation is established, meticulous attention to oxygenation and ventilation is essential. A nurse–patient ratio of 1:1 is desirable. In an unstable patient with multiple injuries, or multiple organ failure, a ratio of up to 2:1 may be justified. The methods and principles of respiratory function monitoring described in section 17.2.1 should continue for the duration of ventilation.

Humidification

In all ventilated patients, inspired gases must be artificially humidified, since the humidifying functions of the nasopharynx are bypassed by endotracheal intubation. Normal respiratory humidification is important for conserving heat and water. Humidification and warming continue along the airways so that alveolar gas is 100% saturated at 37°C. Effective mucociliary function depends on a relative humidity of 75–100% irrespective of temperature. In ventilated patients, the relative humidity of inspired gases falls to 50% or less and rapid heat loss can result in hypothermia. Cold, dry gases in the trachea and bronchi increase mucus viscosity and inspissation, leading to mucosal ulceration, depressed ciliary function and microatelectasis due to obstruction of small airways. Humidification should provide 75–100% saturated gas in the trachea at a constant temperature (32–36°C) without increasing the work of breathing, dead space or resistance to either spontaneous or controlled ventilation. Water-bath humidifiers and aerosol nebulizers or atomizers are commonly used but they may lead to infection from bacterial contamination of water reservoirs, to overhydration, overheating and electrical hazards. Most of these complications can be avoided by using heat and moisture exchangers attached to the endotracheal tube. These give safe and efficient humidification and may be combined with a bacterial filter to maintain sterile gases (Shelly, Lloyd and Park, 1988).

Patients with reactive airways due to pre-existing asthma, chronic airway disease or acute bronchospasm from infection or pulmonary edema may benefit from nebulization of \( \beta_2 \)-agonists (e.g. salbutamol) to improved gaseous exchange and reduced inspired airway pressure. The addition of nebulized anticholinergics (e.g. ipratropium bromide) may benefit patients with severe airflow obstruction, particularly in the weaning phase. The routine use of these agents in ventilated patients, however, has not been shown to be of benefit (O’Driscoll et al., 1989).

(g) Weaning from positive-pressure ventilation

In the head-injured patient, mechanical ventilatory support can generally be withdrawn when intracranial pressure is consistently less than 25 mmHg and there is no significant mass effect on CT scan. Extracranial injuries should also be stable and should not need inotropic support or preload augmentation. Lung function, gaseous exchange and lung compliance should approach that of the patient’s presumed premorbid status. The patient should be placed on assisted ventilation (either synchronized intermittent mechanical ventilation or pressure support) so that spontaneous efforts can eventually take over respiration. Sedation and analgesia should be minimal so that cough and glottic reflexes are present.

As a general rule, weaning is not commenced until \( P_{\text{aO}_2} \) is 70–80 mmHg with a \( F_{\text{O}_2} \) not greater than 0.4. The \( P_{\text{aCO}_2} / F_{\text{O}_2} \) ratio is a useful measure of the alveolar arterial oxygen gradient (Table 17.3). Similarly, \( P_{\text{aCO}_2} \) should be normal, as discussed in section 17.5.1(e). Pulse oximetry has greatly facilitated assessment of oxygenation, although the limitations outlined in section 17.4.2 must be considered; frequent blood gas analyses should be performed, particularly after a change in ventilator settings or \( F_{\text{O}_2} \) (Linton, 1993). Pulse oximetry may be used to predict success in weaning patients from mechanical ventilation but these should be interpreted within the specific clinical context. They are accurate guides, providing the work of breathing through the endotracheal tube and ventilator tubing is compensated by inspiratory pressure support of approximately 10–15 cmH\(_2\)O (Banner et al., 1993b). Alternatively, lung volumes can be assessed by connecting the endotracheal tube to a Wright’s respirometer. Of the indices of lung mechanics, the ratio of respiratory rate to expired tidal volume (\( f/V_T \)) is easily determined and values greater than 100 may accurately predict successful weaning (Yang and Tobin, 1991). Lung mechanics can also be assessed by the simple bedside demonstration of a strong expulsive cough on tracheal suction. This implies adequate forced vital capacity, cough and glottic reflexes.
Intermittent T-piece weaning is suitable for patients with underlying airflow obstruction or mechanical muscle weakness. The patient is connected to a T-piece for trial periods, of perhaps 5–30 minutes every 30–120 minutes. Each trial period is increased by 5–10 minutes until the patient maintains adequate mechanics and oxygenation without mechanical ventilation. A balance must be achieved between allowing the patient to exercise and causing respiratory muscle fatigue with increased oxygen consumption. T-piece weaning has been largely superseded by pressure support ventilation using newer ventilators such as the Siemens Servo 900C, Engstrom Erica or Puritan Bennett 7200a (Tomlinson et al., 1989; Bone and Eubanks, 1991; Oh et al., 1991).

Weaning from synchronized intermittent mandatory ventilation may be accomplished by progressively reducing the ventilator rate and tidal volume until the patient can maintain an adequate minute volume with spontaneous respiration. Inspiratory pressure support should be added as soon as the patient begins spontaneous efforts and then maintained throughout the weaning period. Once the patient is breathing adequately on pressure support ventilation alone this may be gradually reduced to 10 cmH2O and the patient extubated or, if tracheostomized, placed on a humidified T-piece.

Exubation may be considered if the level of consciousness has improved and the pharyngeal and tracheal reflexes are able to protect the airway from aspiration of secretions or gastric contents.

With recovery of physical and mental status, a patient with a tracheostomy may progress from a cuffed to a non-cuffed or fenestrated tube and then be extubated.

(h) Complications: nosocomial pneumonia

Ventilated patients are susceptible to respiratory infections. Early pneumonia, defined as respiratory infection already present or developing soon after intubation, may arise from aspiration of gastric contents or infection of collapsed lobes. Late or true ventilator-associated (nosocomial) pneumonia is defined as infection occurring 48 hours after intubation and not present or incubating at time of intubation (Meduri, 1993).

True ventilator-associated pneumonia is associated with a significant morbidity and mortality, particularly in those patients who progress to develop an acute lung injury (section 17.5.1). In patients with severe head injury who require positive pressure mechanical ventilation for more than 3 days, the incidence of pneumonia has been reported to be 35–70%. Nosocomial pneumonia significantly prolongs ventilation and ICU stay and incurs a reported mortality of up to 50% (Rodriguez, 1991).
Management of ventilator-associated pneumonia is twofold. The first essential step is aggressive pulmonary toilet, postural drainage and prevention of gastric aspiration. Increased sedation may be necessary during tracheal suctioning to prevent sympathetically mediated swings in blood pressure and intracranial pressure (Ersson et al., 1990). There is increasing evidence that ventilated patients nursed at 30° head elevation have a lower incidence of pulmonary aspiration and improvement in $P_{a}O_{2}$ as a result of reduced shunting. The positioning of head-injured patients has been the topic of some controversy. This is discussed further in section 17.5.2(c). The second step is appropriate antibiotic cover, using the least toxic bactericidal agent in appropriate doses, guided by blood level monitoring if necessary. Antibiotics should only be used when the criteria for nosocomial pneumonia are fulfilled and the bacteriological cultures are positive. Sensitivity testing is the best guide to antibiotic choice.

(i) Complications: acute respiratory distress syndrome/acute lung injury

The acute respiratory distress syndrome (ARDS) has been defined as ‘a sudden clinical pathophysiological state characterized by severe dyspnea, hypoxia, diffuse bilateral pulmonary infiltrations and stiff lungs following massive acute lung injury, usually in persons with no previous lung injury’ (Ashbaugh et al., 1967). The hallmark of this syndrome is increased permeability of the alveolar–capillary endothelium.

The constellation of clinical, radiological and pathological findings, resulting from diffuse injury to the lung parenchyma, that defines this syndrome has been the subject of intense debate (Petty, 1990; Oh, 1992; Repine, 1992; McNaughton and Evans, 1992; Kolleff and Schuster, 1995). The terms acute respiratory distress syndrome or acute lung injury are generally used interchangeably, but they have recently been defined in terms of the severity of insult, specifically the degree of hypoxia; ARDS is now regarded as the more severe form of acute lung injury (Bernard et al., 1994).

The insult is not uniform and spares some areas of lung parenchyma (Figure 17.2). In damaged areas, the lung is atelectatic, edematous and hemorrhagic. Hypoxia occurs as a result of increased intrapulmonary shunting of blood caused by the loss of functional alveoli and hence reduction in functional residual capacity. Lung compliance is reduced (<30 ml/cmH$_2$O) and physiological dead space is increased, leading to hypercapnia. Diffuse alveolar and interstitial infiltrates appear progressively on chest radiographs and may be quantified in accordance with the severity of injury (Murray et al., 1988). Microscopic examination reveals intra-alveolar collections of proteinaceous fluid, red blood cells and inflammatory cells. Microthrombi or white cell aggregates may be seen in small vessels. After 24–48 hours, hyaline membranes line the alveoli. These are formed by fibrin that has escaped
through the capillary walls. Subsequently, as repair of the injury occurs, fibrosis may ensue.

Some but not all patients with ARDS develop dysfunction or failure of one or more organ systems, either sequentially or simultaneously. By contrast, other patients develop multiple organ failure without ARDS, although they may have less severe degrees of parenchymal lung injury. This suggests that ARDS is the respiratory manifestation of multiple organ failure syndrome, just as septic shock is the cardiovascular manifestation (Bone et al., 1992b).

The patient with head injury is at risk of developing ARDS as a result of neurotrauma per se – neurogenic pulmonary edema is a form of ARDS – or of extracranial factors such as lung contusion, multiple transfusion, burns, pneumonia, pancreatitis, aspiration of gastric contents and sepsis.

Treatment of ARDS

Treatment of patients with ARDS is primarily supportive and aims to maintain oxygen delivery to all organ systems. Most patients with ARDS who die do so from multiple organ failure or sepsis rather than respiratory impairment (MacNaughton and Evans, 1992). Hence the underlying cause must be treated and suspected sites of sepsis need to be managed aggressively with appropriate antibiotics and surgical drainage. Careful fluid management is necessary to maintain euvolemma without increasing lung water (Humphrey et al., 1990; Hudson, 1992). Pulmonary artery catheters may be used to facilitate the measurement of oxygen delivery (Russell, Graybeal and Strout, 1990; Mitchell, 1992). Pressure-limited ventilation and PEEP, as outlined in section 17.5.1(d), form the mainstay of respiratory support.

Permissive hypercapnia has been shown to reduce mortality in ARDS (Hickling, 1990). This employs pressure-limited ventilation at the expense of tidal volume and a rise in $P_{\text{a}CO_2}$. In these studies $P_{\text{a}CO_2}$ was allowed to rise to greater than 80 mmHg, without adverse effects, despite incurring a moderate respiratory acidosis. However, this strategy may not be feasible in head-injured patients, where control of $P_{\text{a}CO_2}$ is essential for controlling ICP and CPP (section 17.5.1(e)).

The prognosis of ARDS depends mainly on the cause and the presence of multiple organ failure. Fat embolism and neurogenic pulmonary edema may cause a severe but transient ARDS, which often has a good outcome. Gram-negative sepsis and shock states have a higher incidence of multiple organ failure and a higher mortality.

Despite improvements in the understanding of pathophysiological pathways and in mechanical support for patients with ARDS and the multiple organ failure syndrome, no definitive ‘magic bullet’ treatment is available and mortality remains high at 40–70% (Kraus et al., 1993).

(j) Complication: pulmonary thromboembolism

Fatal pulmonary embolus occurs in 2–3% of patients with head and multiple injuries. It is usually caused by thrombosis of the iliofemoral veins. Deep venous thrombosis in the legs may occur in 30–40% of these patients but has a lower incidence of embolism.

High-risk factors include a previous history of thromboembolism, major orthopedic (especially pelvic) injuries, other major trauma, prolonged coma and hemiparesis. Prophylactic measures includes early mobilization, graduated compression stockings, pneumatic calf stimulators and low-dose anticoagulants such as heparin, low-molecular-weight heparin or warfarin. Although bleeding is an uncommon complication of low-dose anticoagulants, it is prudent to monitor coagulation profiles in patients with neurotrauma, giving anticoagulants only during the convalescent phase because of the risk of intracranial bleeding (Moser, 1990; Sudlow et al., 1992).
In high-risk patients or those with proven thromboembolism in whom anticoagulation is contraindicated, early placement of vena caval filters has been advocated (Persson, Davis and Villavicencio, 1991).

(k) Effects of pre-existing physical limitations of the chest on weaning

Patients with massive obesity and kyphoscoliosis may require pressure-limited ventilation. These patients and those with respiratory muscle weakness due to myopathies, neuropathies or paralysis may need prolonged weaning. Pleural and parenchymal pathology limits alveolar ventilation by restricting lung expansion and by increasing dead space. These disorders also increase the work of breathing, thereby increasing oxygen consumption and respiratory muscle fatigue.

Drainage of the pleural space, usually by means of tube thoracostomy, is indicated in patients compromised by pneumothorax, hemothorax or pleural effusion.

Nutrition, introduced early and preferably by the enteral route, will improve respiratory muscle function to a limited extent. It is associated with a reduction in nosocomial pneumonia and preservation of gut integrity (Chapter 16).

17.5.2 HEMODYNAMIC MANAGEMENT OF THE ACUTE HEAD-INJURED PATIENT IN THE ICU

Cardiovascular stability in patients with neurotrauma is of particular importance because of the need to maintain an adequate cerebral perfusion as well as myocardial, renal and splanchnic perfusion (Fessler and Diaz, 1993).

Vital organ perfusion depends on an adequate vascular volume, hence accurate monitoring of vascular volume and systemic blood pressure is essential. As discussed in section 17.4.5(a), mean arterial pressure should be monitored continuously, usually with an intra-arterial catheter.

(a) Volume status monitoring

Right atrial pressure (central venous pressure) will suffice to monitor volume status in most patients but more specific monitoring of cardiac output may sometimes be indicated. As with arterial pressure measurement, this is most reliable with electronic transducers.

Right ventricular preload is measured by inserting a catheter into the superior vena cava and measuring mean right atrial pressure when the tricuspid valve is open. The pressure measurement provides an approximation of right ventricular preload if ventricular compliance is normal, as it usually is in young patients.

The response of right atrial pressure to a fluid challenge will yield useful information regarding the volume status. This is particularly helpful when right atrial pressure is less than normal (approximately 5 mmHg) as pressure in the right ventricle is seldom much higher than in the left. In this context, a low right atrial pressure suggests decreased intravascular volume.

Factors that influence the accuracy of right atrial pressure measurements include right heart dysfunction, superior vena caval obstruction, tricuspid valve disease, cardiac tamponade and raised intrathoracic pressure (right atrial pressure may be elevated when left ventricular pressure is normal).

Left ventricular end-diastolic pressure and volume may be assessed with right atrial pressures if right and left ventricular pressures are similar in diastole. This is usually the case in patients with normal left ventricular compliance. However, in patients with poor left ventricular compliance or mitral valve disease, left ventricular pressure may be elevated when right ventricular pressure is normal. In these patients, in older patients and in those with multiple organ dysfunction, direct left-sided measurements may be appropriate for assessing left ventricular pressure.

Pulmonary artery pressure monitoring is widely used in the management of critically ill patients. However there has been strong opinion expressed against its routine use (Robin, 1985). Conversely, protagonists claim improved outcome in selected patients (Shoemaker et al., 1988; Mitchell, 1992).

Left ventricular preload may be estimated by passing a balloon-tipped catheter into the right-sided pulmonary circulation. With the balloon inflated, the catheter traverses the right atrium, right ventricle and main pulmonary artery until it occludes a branch of the pulmonary artery (Figure 17.2). Flow distal to the balloon ceases and the pressure measured at the catheter tip distal to the balloon reflects the pulmonary artery occlusion or ‘wedge’ pressure downstream. This approximates to left atrial pressure and is equivalent to left ventricular end-diastolic pressure when the mitral valve is open, assuming that pulmonary venous pressure is not elevated and that the mitral valve is normal. The left ventricular end-diastolic pressure is assumed to approximate to left ventricular end-diastolic volume and therefore to left ventricular preload. With the balloon deflated, a pulmonary arterial pressure waveform is continually displayed. When the catheter is correctly placed right atrial pressure is measured simultaneously from a port in the right atrium. The pulmonary artery
catheter may also be used to measure cardiac output by the thermodilution method using a thermistor at the distal end. New rapid-response catheters are able to determine continuous right ventricular ejection fraction and cardiac output (Dorman et al., 1992). A wide range of cardiorespiratory variables (Table 17.4) can be derived from these measurements, some of which have been discussed in section 17.2.3, and they play an essential part in providing optimal oxygen delivery.

Table 17.4 Hemodynamic indices obtained from pulmonary artery catheters. MAP = mean arterial pressure, SBP = systolic blood pressure, DBP = diastolic blood pressure, CO = cardiac output, BSA = body surface area, RAP = right atrial pressure, MPAP = mean pulmonary artery blood pressure, PAOP = pulmonary artery occlusion pressure.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Formula</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure (MAP)</td>
<td>MAP = SBP + DBP ( \frac{3}{3} )</td>
<td>60–90 mmHg</td>
</tr>
<tr>
<td>Cardiac index (CI)</td>
<td>CI = ( \frac{CO}{BSA} )</td>
<td>2.5–3.5 l/min/m²</td>
</tr>
<tr>
<td>Stroke volume index (SVI)</td>
<td>SVI = ( \frac{CO}{HR \times BSA} )</td>
<td>33–47 ml/beat</td>
</tr>
<tr>
<td>Systemic vascular resistance index (SVRI)</td>
<td>SVRI = MAP – RAP ( \frac{CI}{79.98} \times 79.98 )</td>
<td>1700–2400 dyn.s/cm²/m²</td>
</tr>
<tr>
<td>Pulmonary vascular resistance index (PVRI)</td>
<td>PVRI = MAP – PAOP ( \frac{CI}{79.98} \times 79.98 )</td>
<td>150–250 dyn.s/cm²/m²</td>
</tr>
<tr>
<td>Left ventricular stroke work index (LVSWI)</td>
<td>LVSWI = (MAP – PAOP) ( \times \frac{SVI \times 0.0136}{45–60\text{ g.m/m²/beat}} )</td>
<td></td>
</tr>
<tr>
<td>Right ventricular stroke work index (RVSWI)</td>
<td>RVSWI = (MPAP – RAP) ( \times \frac{SVI \times 0.0136}{7–12\text{ g.m/m²/beat}} )</td>
<td></td>
</tr>
</tbody>
</table>

Indications for pulmonary artery catheterization in a head-injured patient (Table 17.4)

There are no specific indications for pulmonary artery catheters in managing patients with head injury. They are most often used for acute intercurrent problems. Clearly they should only be used when the information derived from them will significantly improve management and the benefits outweigh their risks. Situations in which they may be used include:

- measurement of right heart pressures (right atrial pressure, pulmonary artery pressure)
  - ARDS
  - pulmonary hypertension
  - pulmonary embolism
  - cardiac tamponade;
- estimation of left heart filling (pulmonary artery occlusion pressure)
  - left ventricular failure
  - response to fluid loading;
- hemodynamic measurement (cardiac output, stroke volume, systemic vascular resistance)

- quantification of shock states (cardiogenic, septic, hypovolemic)
- assessment of response to treatment in the above;
- derivation of oxygen variables (\( \dot{V}O_2, \dot{D}O_2 \));
- measurement of intracardiac shunt (acute ventricular septal defect).

(b) Clinical use of central venous catheters

In interpreting central venous and pulmonary artery catheter measurements, account must take of the factors which alter the gradient between the pulmonary artery occlusion pressure and left ventricular end diastolic pressure (Nadeau and Noble, 1986). Intra-vascular pressures must be referenced to the extravascular pressures around them.

Pulmonary artery occlusion pressure is lower than true ventricular filling pressure in a spontaneously breathing patient during inspiration when pleural pressure may be greatly negative. Conversely, pulmonary artery occlusion pressure is higher than the true filling pressure in a mechanically ventilated patient during an inspiration with positive pressure. To avoid erroneous interpretation, the pulmonary artery occlusion pressure should be measured in end-expiration. Ventilated patients should remain on the ventilator during the measurement.

Other pulmonary factors that affect the pressure measurements include catheter-tip location in regions of the lung where alveolar pressure exceeds arterial pressure, PEEP and raised intrapleural pressure. Cardiac factors include mitral or aortic valve disease and poor left ventricular compliance.
(c) Contraindications to central venous catheterization

These include:

- lack of suitable vascular access;
- untreated bleeding disorders;
- patient instability preventing adequate time for the procedure.

(d) Complications of central venous catheters

These include:

- trauma to structures at insertion (pneumothorax, arterial laceration, hematoma, hemothorax, chylothorax);
- arrhythmias on passage of the catheter through the heart;
- perforation of great vessels and atrium;
- migration into a distal vessel, which may cause vessel rupture or lung infarction.

These complications may be avoided by determining the position of the catheter tip on the chest radiograph (Figure 17.2), by continuous monitoring of the pulmonary artery pressure waveform and by inflating the balloon with only 1–1.5 ml of air to obtain a pulmonary artery occlusion pressure. (Hagley et al., 1992). The optimal placement for right atrial pressure catheters is within the superior vena cava 2 cm above the pericardial reflection (McGee et al., 1993).

Central venous catheters are a major source of nosocomial infection and carry a significant morbidity and mortality. In general catheters inserted during resuscitation should be changed as soon as possible unless strict asepsis was used in their insertion. The incidence of infection increases markedly after 5 days and varies with the site of the catheter, increasing from subclavian through to internal jugular to femoral venous catheters (Norwood et al., 1991; Cobb et al., 1992; Mermel and Maki, 1994).

A persistent or new pyrexia, leukocytosis and an inflamed insertion site strongly suggest catheter sepsis and warrant a new catheter. Culture of the intradermal portion and tip of the catheter may confirm an infection, although removal of the catheter may be all that is necessary for treatment (Goldmann and Pier, 1993).

(e) Effects of therapy on cerebrovascular function

Until recently, head injury management was focused on reducing ICP and cerebral blood volume, particularly by osmotherapy, hyperventilation and barbiturate coma. Each of these methods may in part reduce ICP by reducing mean arterial pressure and therefore CPP. Recent studies have questioned their efficacy and emphasis is now being placed more on maintaining CPP (Stone, 1989; Paulson et al., 1990; Bouma et al., 1992; Miller et al., 1992; Fessler and Diaz, 1993; Myburgh and Lewis, 1996).

The hemodynamic effects of ventilation on ICP and CPP must be considered. Increased intrathoracic pressure may reduce venous return and hence preload. At the same time reduction of ventricular transmural pressure may reduce afterload. Thus in hypovolemic patients ventilation may increase hypotension and reduce cerebral perfusion. Conversely, patients with cardiogenic or neurogenic pulmonary edema often improve dramatically with ventilation because of the mechanical reduction in preload and afterload and increase in cardiac output and cerebral perfusion.

Impedance to right ventricular ejection may be increased by high intrathoracic pressures or by pre-existing or acute pulmonary hypertension, resulting in interventricular septal shift, increased left ventricular end diastolic volume and reduced stroke volume (Zwissler et al., 1991; Pinsky, Desmet and Vincent, 1992). As discussed in section 17.5.1(b), the effects of different modes of ventilation on auto PEEP must also be considered.

CPP-based algorithms are discussed in Chapter 19; they have altered the role of osmotherapy and elective dehydration. When the blood–brain barrier is intact, mannitol, a six-carbon non-metabolic sugar similar to glucose, remains within the intravascular space. It may have several modes of action. These include reduction in cerebrospinal fluid formation, reduction in brain tissue volume (Donato et al., 1994), an immediate increase in circulating blood volume and arterial blood pressure (Roberts et al., 1987), reduction of blood viscosity by hemodilution (Muizelaar, Lutz and Becker, 1984), increase in red blood cell deformability (Burke et al., 1981) and scavenging of potentially toxic free radicals (Fisher and Raper, 1988). Experimental studies have shown that cerebral vasoconstriction occurs in direct response to the decrease in blood viscosity after mannitol infusion (Muizelaar et al., 1983). In other words, a constant CBF is maintained by vasoconstriction if autoregulation is intact. Other studies have supported the vasoconstrictor action of mannitol. Intracranial pressure responded better to mannitol in patients with a low initial cerebral perfusion pressure, who are likely to be vasodilated, than those with a high cerebral perfusion pressure where the cerebral vasculature is likely to be already fully vasoconstricted (Rosner and Coley, 1987).

When the clinical goal is euvolemia and adequate cerebral perfusion, osmotherapy may be seen as a method of selectively reducing brain volume while avoiding dehydration (Miller, Piper and Dearden, 1993).
Repeated administration of mannitol, especially in combination with frusemide (furosamide), may result in a systemic hyperosmolar state and dehydration (Schettini, Stahurski and Young, 1982; Roberts et al., 1987). Mannitol will eventually cross the blood–brain barrier into the extracellular space, increasing free water and cerebral edema. The hemoconcentration caused by dehydration may falsely suggest that red cell mass is adequate. Hyperviscosity and microvascular shunting may adversely affect tissue oxygen delivery.

A substantial part of the action of mannitol depends on its ability to draw extravascular water into the vascular space. If extravascular water is markedly reduced mannitol will have only a minimal effect and its toxicity will be increased. When dehydration is well established, the patient will become refractory to mannitol therapy.

The renal toxicity of virtually all drugs commonly used in the neurosurgical patient, such as aminoglycoside antibiotics, mannitol, frusemide and contrast media, as well as transfusion reactions, are increased by dehydration. Dehydration or hypovolemia makes patients more liable to unstable and inadequate systemic and cerebral perfusion and to orthostatic changes in the blood pressure.

Serum sodium should be kept at 145–150 mmol/l and serum osmolality should be no greater than 300 mosmol/l (although many authors will accept 320 mosmol/l). Osmolality should be measured directly, as osmolalities calculated from sodium, urea and glucose will underestimate true osmolality when mannitol is used.

Mannitol doses should be limited to the minimum amount needed to control ICP and CPP and should not be given routinely with frusemide because of the risk of excessive dehydration. Dehydrating agents— not only mannitol— given appropriately can at times be life-saving, but chronic dehydration is potentially harmful.

(f) Head elevation

Head elevation to about 30° is widely used in nursing critically ill patients with pulmonary congestion from cardiac failure or pulmonary edema. Oxygenation is improved by increasing functional residual capacity and chest wall compliance and by reducing cephalad movement of the diaphragm. It also reduces the risk of aspiration of gastric contents.

In patients with head injury, head elevation may reduce ICP by reducing cerebral venous pressure. Rosner and Coley (1986) found that ICP was 7–10 mmHg higher in 50–70% of patients nursed flat compared with 30–50° head elevation. However, if there is an equivalent fall in arterial pressure measured at head level then CPP will not increase despite the fall in ICP. When severe intracranial hypertension exists, there is little cerebral venous volume available for displacement and CPP may be higher with patients nursed flat (Rosner, 1993). In one study, one-third of patients demonstrated an increase in ICP with head elevation (Feldman et al., 1992). This may be due to vasodilatation in response to decreased CPP (Rosner, 1993).

Fluctuations in intracranial pressure and cerebral perfusion pressure with head elevation may be minimizing by maintaining euvoelma and the cardiopulmonary benefits of slight head elevation can then be utilized.

Barbiturate therapy

There are few options available for treating patients with raised ICP refractory to the above measures.

Sodium pentobarbital at doses causing ‘burst suppression’ on the EEG will lower intracranial pressure. However, these doses of barbiturates cause profound myocardial depression and hypotension and reduce cerebral perfusion pressure. Theoretically, the reduction in cerebral perfusion pressure and blood flow may be offset by the reduction in cerebral oxygen consumption induced by barbiturates. However two randomized clinical trials have found no improvement in morbidity, mortality, or intracranial pressure control when barbiturates where given (Ward et al., 1985; Eisenberg et al., 1988). Barbiturates are still sometimes used when all other measures have failed.

A recent concept of aggressively maintaining cerebral perfusion pressure with inotropes and vasopressors and using barbiturates to lower intracranial pressure has yet to validated (Miller et al., 1992).

Barbiturate therapy has not been shown definitively to alter outcome in neurotrauma. In the group of patients refractory to other means of treatment, the persistent intracranial hypertension probably reflects the severity of the primary injury (Chapter 19).

(g) Hemodynamic manipulation

In managing systemic arterial pressure it is important to consider that systemic hypertension may be a response to intracranial hypertension and therefore necessary to maintain an adequate cerebral perfusion pressure (Bouma et al., 1992).

Patient manipulations such as endotracheal and nasotracheal suction, neurological examination, painful stimulation and even family visits, may increase ICP via sympathetic stimulation. This is unlikely to be harmful unless ICP is already raised. In this situation the increases can be prevented by increasing sedation and analgesia.
In current clinical practice optimal cerebral perfusion pressure is 60–70 mmHg (Saul and Ducker, 1982; Tsutsui et al., 1985), although levels of 80–100 mmHg have recently been proposed as providing better control of raised ICP. These levels of CPP may be achieved by manipulating volume status and by the use of vasoressors (Rosner, 1993).

Fluid management of head-injured patients has focused for the last 20 years upon reducing intracranial pressure via strategies of elective dehydration and osmotherapy. This was based on the concept that less water available in the body meant less water available to potentiate cerebral edema. There is, however, no evidence to substantiate this hypothesis and indeed the practice may caused more harm than good. Attention has now shifted to maintaining brain perfusion.

The primary goal of cerebral perfusion pressure fluid management is euvolemia, but avoiding systemic overhydration (Marmarou, 1992). A normally hydrated, euvolemic patient is more hemodynamically stable and has fewer blood pressure changes associated with ventilator manipulation and less need for vasopressor support. Furthermore, adequate perfusion of the kidneys minimizes the potentially nephrotoxic effects of mannitol, fruseamide and sepsis.

Establishing euvolemia, however, requires great care. The assessment of volume status depends on clinical markers such as blood pressure, heart rate, fluid balance and a measurement of preload (as discussed in section 17.5.2(a)). Biochemical markers such as serum sodium, urea, creatinine and osmolality have been advocated but usually change late with evolving volume depletion and are frequently a poor guide to volume status. Reaching and maintaining euvolemia depends on a number of factors, which should be considered in concert.

Maintenance fluids

Balanced salt solutions maintain normal hydration and replace insensible losses from pyrexia and ventilation. When osmotic diuretics and inotropes are used (see below), urine output may increase to 100–200 ml/h, resulting in dehydration. Fluids must be adjusted to maintain a normal volume status as apparently adequate fluid intake alone does not guarantee adequate vascular volume. It is possible for a patient to have interstitial overhydration with an inadequate intravascular volume.

Hypovolemic patients generally require colloid solutions. The best vascular volume expander in an anemic patient is red blood cells. Serum albumin as either normal (5%) or concentrated albumin is also useful. Synthetic colloids such as polygeline are suitable, although they have a short half-life and may interfere with clotting. (The fluid management of head-injured patients is discussed further in Chapter 16.)

Vasoressors

Augmenting mean arterial pressure and cardiac output by inotropes and vasoressors is standard practice in managing critically ill patients with sepsis and cardiac failure and is now more widely used to maintain adequate cerebral perfusion in patients with head injury (Chapter 19).

Euvolemia is an essential prerequisite for the use of these agents. As discussed above, a hypovolemic patient is susceptible to wide swings in blood pressure and these may be accentuated by vasoressors. The ideal inotrope and vasoressor is one that acts rapidly and has a short half-life, thereby allowing titration of dose against a desired end-point, which is usually mean arterial pressure and/or oxygen delivery.

Inotropes increase ventricular contractility by acting on myocardial alpha- and beta-receptors. They include adrenalin (epinephrine), noradrenalin (norepinephrine), dopamine, dobutamine and isoprenaline (Prichard et al., 1991; Runciman and Morris, 1993a). The principal hemodynamic effect of inotropes is an increase in cardiac output. Effects on peripheral vasculature are variable and may be either vasoconstriction or vasodilatation. The catecholamines (adrenalin, noradrenalin and dopamine) are increasingly regarded as first-line inotropes. They exert beta-adrenergic effects predominantly at low doses (i.e. increased heart rate, vasodilatation, inotropy and bronchodilation) and alpha-adrenergic effects (vasoconstriction, inotropy) at higher doses (Moran et al., 1993). Tachycardia and dysrhythmias are infrequent (Runciman and Morris, 1993a).

Coronary perfusion and myocardial oxygen delivery depend on aortic diastolic pressure and are at risk in states of low systemic vascular resistance such as sepsis (Schreuder et al., 1989). Patients with low cardiac output and high systemic vascular resistance, as in cardiogenic shock, are best treated initially with a catecholamine to improve inotropy and maintain coronary perfusion. The use of dobutamine and isoprenaline to treat cardiogenic shock has been increasingly questioned because of a high incidence of tachycardia and reduction of aortic diastolic pressure.

The effects of ‘low-dose’ dopamine, frequently advocated to improve renal blood flow, are unsubstantiated. Any such effects are probably not specific but mediated via increased cardiac output (Duke and Bersten, 1992).

Vasoactive drugs include vasoconstrictors (phenylephrine, metaraminol) and vasodilators (sodium
may potentiate cardiac failure and bronchospasm and to 300 mg/kg/min (Hansson, 1991). All beta-blockers accurate titration by intravenous infusion at doses up this. It has an extremely short half-life which allows (Robertson et al., 1983). Esmolol is ideally suited for. This has an extremely short half-life which allows accurate titration by intravenous infusion at doses up to 300 mg/kg/min (Hansson, 1991). All beta-blockers may potentiate cardiac failure and bronchospasm and

Although the role of vasopressors in neurotrauma is not fully established, the rationale for their use is the loss of the capacity to maintain cerebral perfusion pressure by cerebral autoregulation.

In severe brain injury cerebral perfusion may become 'pressure-passive', i.e. directly dependent on systemic arterial pressure. There may be marked regional differences so that the relationship is difficult to quantify. Vasoactive drugs should be titrated against a selected cerebral perfusion pressure. The degree to which the cerebral vasculature is directly affected by these vasoactive agents is unclear.

Beta-adrenergic antagonists are effective in controlling sympathetically-mediated release of renin from the juxtaglomerular cells of the kidney, and this also assists in lowering blood pressure. Thus beta-blockers may be indicated when hypertension is due to increased sympathetic tone with tachycardia and increased cardiac output, as in severe head injury (Robertson et al., 1983). Esmolol is ideally suited for this. It has an extremely short half-life which allows accurate titration by intravenous infusion at doses up to 300 mg/kg/min (Hansson, 1991). All beta-blockers may potentiate cardiac failure and bronchospasm and must be used carefully in susceptible patients (Runciman and Morris, 1993b). Vasodilators are seldom indicated in neurogenic hypertension.

Although glyceryl trinitrate and sodium nitroprusside are commonly used in other neurosurgical situations to control blood pressure, their propensity to increase cerebral blood volume and intracranial pressure, and to cause preferential peripheral vasodilatation over cerebral vasodilatation (cerebral steal), probably contraindicates their use in head-injured patients, who may have increased intracranial pressure (Anile et al., 1981; Davis et al., 1981; Michenfelder and Milde, 1988).

17.5.3 FUTURE ADVANCES IN NEUROMONITORING

Neuromonitoring after head injury aims to provide information about intracranial compliance and cerebral oxygen utilization (Lewis, Reilly and Myburgh, 1994). Measuring intracranial pressure alone does not give any information regarding cerebral oxygen utilization (Miller et al., 1992), cerebral blood flow or cerebral metabolic rate (Bouma et al., 1992).

Frequently, intracranial hypertension is present without any mass lesion on CT scan, when the patient has diffuse cerebral swelling. Hyperventilation and other methods of reducing intracranial pressure have been used empirically without there being any conclusive evidence that they improve outcome. Indeed, they may cause harm.

Jugular venous oxygen saturation, measured by fiberoptic catheters inserted into the jugular bulb, allows calculation of arteriovenous differences and oxygen uptake by the brain. Cerebral venous desaturation may occur as a result of intracranial hypertension, excessive hypocapnia, arterial hypoxia and systemic hypotension. Preliminary studies suggest that jugular bulb oximetry used in conjunction with intracranial pressure and cerebral perfusion pressure monitoring may allow more precise management of cardiorespiratory variables so as to optimize cerebral oxygen utilization (Sheinberg et al., 1992; Lewis, Reilly and Myburgh, 1995).

17.6 Conclusion

At present little can be done to influence the outcome from the primary brain injury. The duration of the period of increased vulnerability to secondary ischaemic and hypoxic insults remains unclear but may be up to 10 days to 2 weeks in some patients. Secondary insults must be prevented or rapidly detected and treated. Adequate substrate delivery is the key to preventing secondary insults. This requires assiduous attention to cardiopulmonary homeostasis, cerebral perfusion pressure and oxygen utilization. The interaction of these parameters must be recognized.


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