securing the airway, ensuring adequate oxygenation and mechanical ventilation when indicated, is therefore essential.

When there are no symptoms conservative treatment, consisting of eliminating the precipitating cause, including correction of any hormone deficiency (e.g. cortisol, aldosterone, thyroid hormone), and simple water deprivation is the preferred course of action. Normal saline should be given to those who are volume-depleted. Symptomatic hyponatraemia can be treated with intravenous hypertonic sodium chloride (3%, 514 mmol/L) sometimes combined with administration of a loop diuretic such as furosemide.

Although conventionally too rapid correction of profound, chronic (>2 days) hyponatraemia is thought to increase the risk of precipitating severe shrinkage of brain cells and central pontine myelinolysis (Sterns et al., 1986), it seems that prompt judicious therapy with intravenous sodium chloride before the development of respiratory insufficiency is associated with better outcomes than fluid restriction alone (Ayus and Arieff, 1999). Certainly chronic symptomatic hyponatraemia in postmenopausal women can be associated with serious morbidity and mortality (Ayus and Arieff, 1999) and the risk of not correcting cerebral oedema exceeds the small risk of osmotic demyelination as a result of treatment. Nevertheless, in cases of symptomatic hyponatraemia the rise in plasma sodium should not usually exceed 1–2 mmol/h. The plasma sodium concentration should be monitored frequently (e.g. every 2 hours) and should not be allowed to increase to above about 133 mmol/L. In those with seizures and/or respiratory distress the sodium concentration should be increased more rapidly, for example by about 8–10 mmol/L over the first 4 hours in profound hyponatraemia or until cessation of seizure activity. This rate of correction can only be accomplished using hypertonic saline. Treatment with hypertonic saline should be discontinued when either the patient becomes asymptomatic or the plasma sodium reaches a concentration of between 124 and 132 mmol/L. The plasma sodium should not normally be increased by more than about 20 mmol during the initial 48 hours of treatment and as a general principle should not be rapidly increased to normal or high levels. As a guide, and assuming that water comprises 50% of total bodyweight, 1 mL/kg of 3% sodium chloride will raise the plasma sodium by 1 mmol/L.

A recent multicentre trial demonstrated that in patients with euvolaemic or hypervolaemic hyponatraemia caused by chronic heart failure, cirrhosis or the syndrome of inappropriate ADH an oral vasopressin V_2-receptor antagonist was effective in increasing serum sodium concentrations (Schrier et al., 2006).

**HYPERNATRAEMIA** (Pavlevsky et al., 1996)

**Causes**

Hyponatraemia (a plasma sodium >145 mmol/L) is one of the most commonly encountered electrolyte abnormalities in the hospital population and is seen most often in situations of predominant water depletion (e.g. diabetic coma, diabetes insipidus, fever, elevated ambient temperature, infirmity). It may also occur in association with salt retention in acute renal failure and can be exacerbated by the excessive administration of sodium ions, often as isotonic saline, artificial colloids or 8.4% sodium bicarbonate.

**Clinical features**

Conscious level is frequently impaired and seizures are common. Associated laboratory abnormalities may include metabolic acidosis, azotaemia, hypophosphataemia and hyperglycaemia. Most patients have severe concomitant disease and hypernatraemia is associated with substantial long-term morbidity and mortality.

**Treatment**

Associated medical conditions must be treated as a matter of urgency. Pure water depletion should be treated with hypotonic intravenous fluids or dialysis, but rapid falls in plasma sodium greater than 2 mmol/h may precipitate cerebral oedema and must be avoided. Too rapid correction of hypernatraemia has been implicated in the morbidity and mortality of diabetic ketoacidosis (see Chapter 17). When required (e.g. following excessive administration of isotonic saline) solute removal should be accomplished by diuretic therapy or dialysis.

**HYPOKALAEMIA**

**Causes**

The normal daily intake of potassium is 50–150 mmol (1–1.5 mmol/kg per day). Hypokalaemia is usually related to inadequate replacement of excessive urinary or gastrointestinal losses (e.g. diarrhoea). For a number of reasons, critically ill patients are particularly prone to the development of hypokalaemia. Elevated levels of aldosterone are found in both cardiac and liver failure, as well as in response to stress, while steroids and diuretics also increase urinary potassium losses. Movement of potassium into cells is promoted by β-agonsists, insulin, mineralocorticoids and theophyllines. Also potassium may be lost in the urine during the recovery phases of acute renal failure, and severe hypokalaemia may itself lead to tubular dysfunction. Severe vomiting may be accompanied by a hypochloraemic alkalosis which leads to a shift of potassium into the cells and increased potassium losses from the kidney. Conversely, acidosis favours the movement of potassium out of cells in exchange for hydrogen ions. These changes in plasma potassium are more marked with metabolic than with respiratory acid–base disturbances.

**Clinical features and diagnosis**

Plasma potassium levels are not always a good guide to total body potassium, which may be significantly depleted even when the patient is normokalaemic. In this situation the diagnosis is suggested by finding a metabolic alkalosis with a paradoxically acid urine, caused by renal conservation of
potassium ions in exchange for hydrogen ions, with renal retention of sodium and bicarbonate.

Hypokalaemia may also be detected by recognizing the associated electrocardiographic (ECG) changes of ST-segment depression, decreased T-wave amplitude and prominent U waves (Fig. 11.2). Subsequently there may be widening of the QRS complex and atrioventricular block. Hypokalaemia can also cause supraventricular tachycardias, particularly in the presence of digoxin, as well as more serious ventricular arrhythmias. Profound potassium depletion may be associated with skeletal muscle dysfunction, severe weakness, hyporeflexia and, in extreme cases, paralysis (e.g. hypokalaemic periodic paralysis). Muscle weakness may be exacerbated by accompanying hypophosphataemia as potassium depletion leads to renal tubular phosphate wasting. Weaning from mechanical ventilation may be prevented, while gut motility and vascular responsiveness to pressor agents may be reduced. Acute respiratory failure due to severe hypokalaemia in the presence of a hyperchloremic acidosis has been reported (Dunn et al., 1999).

Metabolic effects of hypokalaemia include reduced protein and carbohydrate synthesis and glucose intolerance.

**Treatment**

Underlying causes should be corrected when possible. Treatment is with potassium chloride by infusion. Concentrated solutions of potassium are extremely irritant and must therefore be administered via a centrally placed catheter. If potassium is given too rapidly, plasma concentrations may reach dangerous levels before equilibration between extracellular and intracellular compartments has occurred. Except in exceptional circumstances, intravenous administration of potassium should not exceed 40 mmol/h. When there is a coexisting metabolic acidosis potassium should be replaced before correcting the acidosis in order to avoid further dangerous reductions in plasma potassium levels. Resolution of metabolic alkalosis can be a useful guide to correction of the whole-body potassium deficit. Magnesium is required for cellular potassium uptake and to prevent continued renal potassium losses.

**HYPERKALAEMIA**

**Causes**

Hyperkalaemia can be defined as a plasma potassium level of more than 5 mmol/L and may be associated with a high, normal or low total body potassium. Regulation of the distribution of potassium between compartments has to be extremely efficient since the extracellular movement of as little as 2% of the intracellular potassium could result in potentially fatal hyperkalaemia.

Hyperkalaemia is most often caused by renal insufficiency but is also frequently iatrogenic as a consequence of excessive potassium administration. Administration of suxamethonium to patients with diffuse tissue damage (e.g. muscle trauma, burns, tetanus or paralysis) can release large quantities of potassium into the circulation. There is a risk of this complication 5–15 days after the injury and the danger persists for 2–3 months in those who have sustained burns or trauma, and for perhaps 3–6 months in those with upper motor neurone lesions (Gronert and Theye, 1975). Other causes of hyperkalaemia include hypercatabolism, diabetic ketoacidosis, rhabdomyolysis, tumour lysis syndrome, severe burns, haemolysis and reperfusion of ischaemic tissues. Metabolic acidosis may be associated with, or exacerbate hyperkalaemia. It is important to exclude causes of pseudo-hyperkalaemia such as faulty venesection technique, in vitro haemolysis, extreme leukocytosis or thrombocytosis.

**Diagnosis and treatment**

Signs and symptoms may be absent. The ECG changes associated with hyperkalaemia are peaked T waves and widening of the QRS complexes, followed by bradycardia and asystole (Fig. 11.3).

Treatment is therefore urgent. Obviously potassium administration, and drugs that promote potassium retention, should be stopped. In extreme emergencies 10 mL 10% calcium gluconate injected intravenously will temporarily antagonize the cardiac effects of hyperkalaemia. An intravenous injection of 50 mL 50% dextrose containing 15 units soluble insulin given over 15–20 minutes will drive potassium into the cells. The effect may last for up to 6 hours, after which the treatment may be repeated. Alkalinization with sodium bicarbonate and, if possible, hyperventilation will also shift potassium into the intracellular compartment, as well as enhancing potassium excretion via the kidneys. The former has by far the greater effect on plasma potassium levels. Sodium bicarbonate should be avoided in patients prone to sodium overload (e.g. those with renal failure). Promotion of a diuresis with loop diuretics or a thiazide will increase potassium excretion, in part by increasing sodium delivery to the distal tubule. In cell lysis syndromes an osmotic diuretic may be more appropriate. Potassium levels may be further reduced by the slow intravenous administration of a β, adrenoreceptor agonist (e.g. salbutamol 500 μg), which will promote cellular

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