reduced muscle activity, as thyroid hormones do not seem to change much during bedrest. The metabolic rate continues to fall when the body remains sedentary, probably reflecting the decline in lean muscle mass from disuse. Interestingly, a reduced metabolism does not usually lead to weight gain, and most patients confined to bed maintain a fairly stable weight. Any gain expected because of reduced basal metabolism may be offset by reduced lean muscle mass and lower calorie intake because appetite is poor (Rousseau, 1993).

Several studies have shown that, while lean mass decreases, there is a simultaneous rise in fat storage in adipose tissues (Krebs et al, 1990).

Glucose intolerance and the insulin response

Immobility, or just a sedentary lifestyle, has been linked to insulin resistance, impaired glucose tolerance and consequent development of type 2 diabetes (Blanc et al, 2000).

The body’s ability to regulate blood glucose is adversely affected by long periods of bedrest. Studies have shown a progressive development of glucose intolerance that correlates directly to the length of time that patients remain in bed (Takayama et al, 1974; Rousseau, 1993). The loss of appetite and reduced caloric intake associated with prolonged bedrest can trigger a condition called starvation diabetes.

The number of insulin receptors expressed in skeletal muscles increases in proportion to physical activity. When a person is active and exercising regularly, expression of insulin receptors is high.

When active people eat a meal rich in carbohydrates, their blood glucose levels will rise, triggering insulin release. Insulin will bind to the abundant receptors within the skeletal muscles, promoting rapid glucose uptake, returning the blood glucose levels to normal.

Immobility and reduced food intake are associated with a reduction in the expression of insulin receptors in the skeletal muscles (Rousseau, 1993).

When patients confined to bed eat carbohydrate-rich meals, the sensitivity of the skeletal muscles to the effects of insulin is much lower, resulting in lower glucose uptake and a higher blood glucose concentration. This reduced sensitivity also typically results in overproduction and secretion of insulin by the pancreatic islets, leading to hyperinsulinaemia (Blanc et al, 2000).

Glucose intolerance may be alleviated by encouraging patients to undertake light exercise. This increases the number of insulin receptors within skeletal muscle (Soman et al, 1979), enhancing the effects of insulin.

Renin-angiotensin-aldosterone cascade

The renin-angiotensin-aldosterone cascade plays a key part in the long-term control of blood pressure (Montague et al, 2005). When blood pressure drops, the kidneys release the enzyme renin, which catalyses the conversion of the plasma protein angiotensinogen into angiotensin I. Angiotensin I is rapidly converted into angiotensin II by the angiotensin-converting enzymes (ACE) in the lungs. Angiotensin II is a potent vasoconstrictor, increasing blood pressure and stimulating the adrenal glands to release the hormone aldosterone. Aldosterone increases sodium reabsorption in the kidney, increasing blood sodium levels, blood volume and pressure. In those confined to bed, plasma volume falls significantly, largely as a result of increased urine output (see part 1). This loss in blood volume, together with sodium loss during diuresis, initiates the renin-angiotensin-aldosterone cascade, which can be seen in increased plasma renin activity and increased plasma aldosterone levels (Annat et al, 1986; Gharib et al, 1985).

This hyperaldosteronism stimulates the kidneys to reabsorb more sodium, helping to maintain blood volume and arterial pressure.

Mineral and electrolyte concentrations

Diuresis associated with long periods of bedrest has been shown to increase the loss of sodium, potassium, zinc, phosphorus, sulphur and magnesium (Rousseau, 1993). Sodium loss occurs rapidly in the early stages of bedrest, primarily due to reductions in anti-diuretic hormone levels (see part 1), which trigger increased urine output and lead to a drop in total body sodium (Rousseau, 1993). Sodium levels tend to stabilise as hyponatraemia (low blood sodium) and reduced blood volume trigger aldosterone release (see above).

Although increased aldosterone secretion is effective at limiting further sodium losses, at the same time it causes a progressive loss of potassium in the urine (Chobanian et al, 1974). Plasma calcium levels increase in patients confined to bed, largely because of bone demineralisation (see part 3).

Losses in bone density are exacerbated by an increase in the level of parathyroid hormone, which is known to stimulate the activity of osteoclasts – specialised cells that break down bone (Montague et al, 2005).

RENAL SYSTEM

Urine distribution

In the upright body, gravity plays a major role in draining urine from the kidneys through the ureters to the bladder. In patients in a supine position, urine is still transported from the kidneys into the bladder by peristaltic waves generated within the walls of the ureters.

However, the renal calices rely entirely on gravity to drain fully and, when the body is in a recumbent position, urine collects in the lower portions of the renal calices, where it may form small static pools (Fig 2).

Urinary retention

In the upright position, urine collects in the lower portion of the bladder under the influence of gravity.