Clinical Practice
Systems of life
Bedrest

In this article...
- Brief history of bedrest as a therapeutic intervention
- How immobility and a supine position affect the cardiac system
- Mechanism of orthostatic hypotension

Effects of bedrest 1: introduction and the cardiovascular system

SA

Nursing Times
Self-assessment

Key points

The therapeutic value of bedrest started to be questioned in the mid-1940s

Prolonged bedrest can have harmful effects on body, mind and function

Older patients may still spend between 71-83% of their time in hospital lying down

Cardiovascular effects of bedrest include dehydration, reduced venous return, increased heart rate and deconditioning

The risk of orthostatic hypotension is higher in people who have been confined to bed

Authors John Knight is senior lecturer in biomedical science; Yamni Nigam is professor in biomedical science; both at the College of Human Health and Science, Swansea University. Aled Jones is reader in patient safety and healthcare quality at Cardiff University.

Abstract Until the mid-20th century, bedrest was considered a beneficial intervention that helped people heal and recuperate – although Hippocrates had already noted the risk of loss of muscle, bone and tooth. Today there is greater recognition of bedrest's negative effects on body and mind, and of the need to minimise these. This series explores the effects of immobility and bedrest on the human body and the psychological wellbeing of patients. After outlining a brief history of the perception of bedrest, this first article looks at how it affects the cardiovascular system.

The human body has evolved to function optimally in the upright position for around 16 hours per day. The average adult sleeps 8-9 hours per day, during which the body is normally in a supine (recumbent) position. Research has revealed that consistently sleeping more than nine hours or less than eight hours per day negatively affects physiological, psychological and cognitive function (Van Dongen et al, 2003). This six-part series explores the effects of immobility and bedrest on the systems of the body and also on patients' psychological wellbeing. This first article summarises the history of therapeutic use of bedrest then discusses its effect on the cardiovascular syste

Shifting perceptions of bedrest
Periods of prolonged bedrest (lasting over 24 hours) have been prescribed to patients since the time of Hippocrates (circa 450BC). Bedrest was widely believed to facilitate the process of healing and help people recuperate. Hippocrates himself may have been one the first physicians to recognise the potential harm of confining patients to bed, noting the risk of progressive loss of muscle, bone and tooth (Chadwick and Mann, 1950).

Bed-rest as an aid to recuperation was rare before the 19th century, when larger families meant most people could not afford to take to their beds as they would risk destitution. There was also a fear (sometimes justified) that taking to bed often meant never getting up again (Sprague, 2004).

From the 1860s right through to the mid-1950s, the use of bedrest as an aid for recuperation increased. As late as the 1960s, it was still common practice for health professionals to routinely prescribe strict (sometimes enforced) bedrest. Standard periods of prescribed bedrest were four weeks following a myocardial infarction, three weeks after hernia surgery and two weeks following childbirth (Corcoran, 1991).

The value of bedrest as a therapeutic intervention started to be questioned in the mid-1940s. In the Second World War, it was noted that, when forced to mobilise

Clinical Practice
Systems of life

quickly (due to a lack of available bed space), soldiers recovered more quickly from injuries and infections. Around the same time, aeronautics research began to examine the effects of immobility and weightlessness on human physiology in preparation for space flights. These studies confirmed that prolonged immobility was detrimental to human health, adversely affecting all major organs (Sprague, 2004).

Further evidence of the beneficial effects of early mobilisation came in the 1960s from the forced rapid remobilisation of casualties during the Vietnam War. The importance of correctly positioning soldiers in bed and the benefits of range-of-motion and strengthening exercises also became apparent (Hertzman, 1968).

Surprisingly, although the potentially harmful effects of prolonged bedrest have been documented for centuries, current research is still relatively sparse and far from complete. Even today, patients aged 65 years and over can spend 71-83% of their time in hospital lying down (Fox et al, 2018). Long periods of bedrest can lead to 'bedrest dependency', which is characterised by limited upright activity, a compulsion to return to bed quickly and, sometimes, a refusal to get up (Fox et al, 2009). Recently, there have been calls and action to end the 'pyjama paralysis' in UK hospitals, and get inpatients out of their beds and nightwear and into their day clothes, in an effort to speed up their recovery (Oliver, 2017).

Effects on the cardiovascular system
After periods of prolonged immobility, the cardiovascular system undergoes dramatic and extensive changes. Water loss and a phenomenon called cardiac deconditioning are triggered by a shift and redistribution of fluids in the supine body. Fig 1 summarises the changes that take place in the cardiovascular system – as well as those in the respiratory and haematological systems, which will be covered in the second article in this series.

Gravity and fluid shift
When the body is upright, the fluids within it are continually exposed to the effects of gravity. This encourages lymph and blood to move downwards into the lower limbs. The human body has adapted to minimise these gravity-associated fluid shifts. Most large- and medium-size veins and lymphatic vessels possess reinforced valves that close to prevent the downward flow of blood and lymph (VanPutte et al, 2017). However, even with such adaptations, 75% of the total blood

Fig 1. **Effects of prolonged bedrest on the respiratory, cardiovascular and haematological systems**
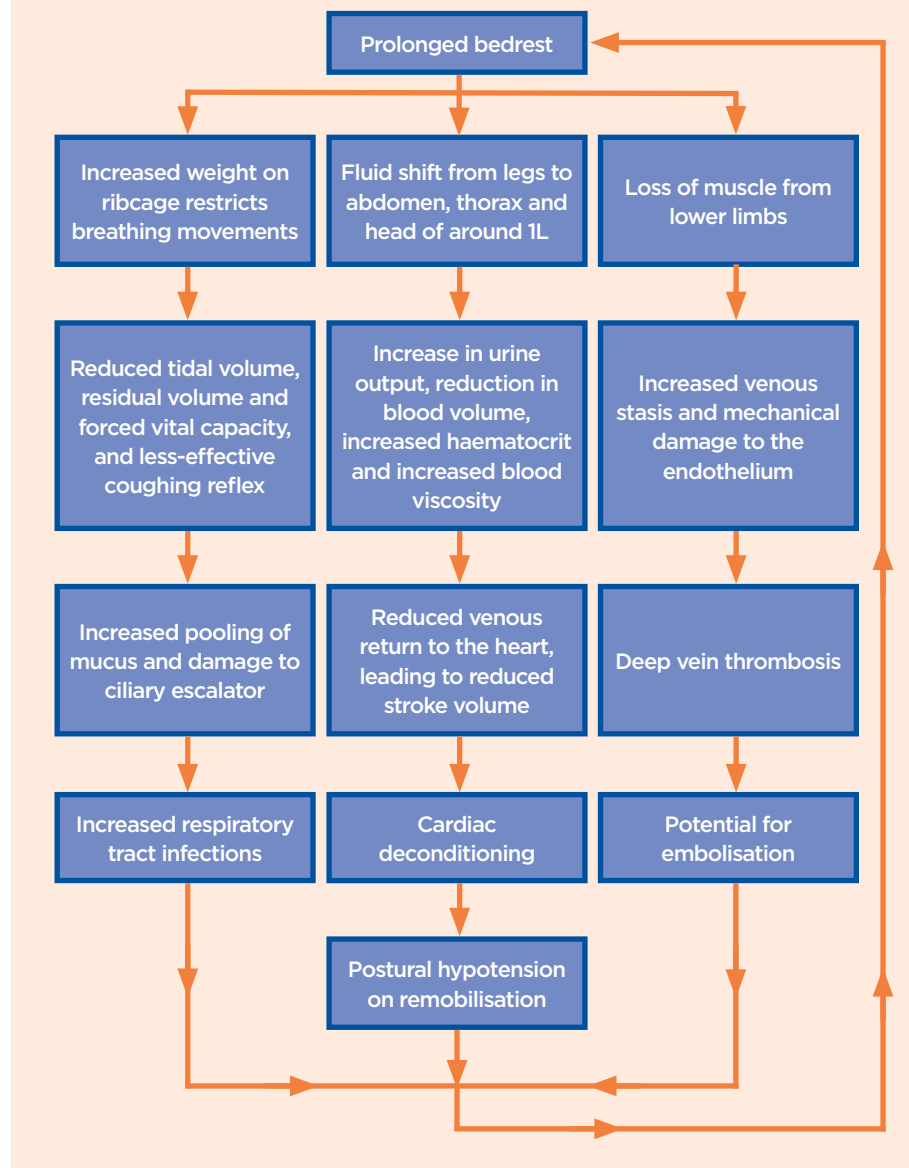
volume in an active person is found in the distensible veins below the level of the heart.

When a person is confined to bed, the effects of gravity are negated, resulting in a gradual shift of fluids away from the legs towards the abdomen, thorax and head. Research has revealed that bedrest for longer than 24 hours results in a shift of around one litre of fluid from the legs to the chest. This temporarily increases venous return to the heart and elevates intracardiac pressure (Perhonen et al, 2001a).

Diuresis and dehydration
Fluid balance in the human body is regulated by several hormones. Increases in blood volume and venous return stretch the right atrium of the heart, which

stimulates the release of the hormone atrial natriuretic peptide (ANP). This is a powerful diuretic and its effects are to increase urine output and decrease blood volume. Conversely, decreases in blood volume and pressure are detected as reduced stretch within the aortic arch and carotid sinus baroreceptors; this stimulates the release of antidiuretic hormone (ADH), which stimulates water reabsorption in the kidneys, thereby reducing urine output and increasing blood volume.

In a healthy, mobile person, the antagonistic effects of ANP and ADH (together with the actions of other hormones) are effective at maintaining fluid balance. However, in prolonged bedrest, the delicate interplay between these two hormones is disrupted.

Clinical Practice
Systems of life

In a person confined in a supine position, the shift of blood from the legs into the thorax increases atrial stretch, which stimulates the release of ANP. This initiates diuresis, leading to significant water loss. The same shift of blood also stretches the aortic arch and carotid sinus baroreceptors, which reduce the release of ADH. As the levels of plasma ADH fall, less water is reabsorbed in the kidneys, thereby amplifying the diuretic effect of ANP.

Bedrest is also associated with an increase in sodium excretion by the kidneys and, as water tends to follow sodium, this leads to sodium diuresis that contributes to the reduction in plasma volume (Chobanian et al, 1974). These changes result in a significant increase in urine output and a progressive reduction in blood volume, which can lead to dehydration and contribute to orthostatic hypotension (OH, also known as postural hypotension).

Health professionals can ensure patients in bed for prolonged periods stay well hydrated by monitoring fluid intake and urine output, and adjusting fluid intake based on these observations as well as on blood results and any pre-existing medical conditions that may affect fluid status. Patients will often need help to sit up in bed and drink; many will find adapted drinking cups helpful although recently there has been a campaign to end the use of beakers because of view that their use can infantalise patients and may increase the risk of aspiration pneumonia (De Castella, 2018). Older patients who are in poor health or forgetful may need to be reminded to drink regularly; some will require intravenous infusion to stay adequately hydrated.

Reduced muscle pump function

The skeletal muscles of the legs, particularly the calf muscles, play an important role in compressing the major veins in the legs. This helps to force blood upwards against the natural pull of gravity, ensuring adequate venous return to the heart (VanPutte et al, 2017).

Prolonged bedrest rapidly leads to skeletal muscle atrophy (sarcopenia) throughout the body (Fig 2). Insulin promotes the uptake of glucose by the muscles, providing energy for muscle contraction. With prolonged bedrest, muscle fibres become less sensitive to insulin and, as a result, there is less glucose to power muscular contraction (Dirks et al, 2016).

A study of men who underwent 17 weeks of bedrest showed significant loss of muscle tissue from the major muscle groups of the legs (thigh and calf), which resulted in reduced strength in the lower limbs (Leblanc et al, 1992). Loss of muscle tissue in the legs reduces the muscle mass available to squeeze the walls of the veins, thereby impairing the skeletal muscle pump function and significantly reducing venous return. Patients confined to bed have an impaired skeletal muscle pump until they remobilise, so they should be encouraged to do simple foot and ankle exercises, such as rotating the foot in a circular motion, to encourage venous return.

Sarcopenia associated with bedrest contributes to significant reductions in lower-limb muscle strength, compounding the patient's inactivity (Kortebein et al, 2008). On mobilisation, those capable of exercising can find short-duration, high-intensity jumping (jump training) effective at restoring and maintaining lower-limb strength (Kramer et al, 2017). Patients' ability to exercise should be carefully assessed; those who are frail, have pronounced OH or neurological conditions that may affect coordination will need a gradual introduction of exercise.

Changes in stroke volume

According to the Frank-Starling principle, the greater the volume of blood entering the heart during diastole (when the ventricles are relaxed), the greater the volume ejected during systolic contraction (stroke volume). As prolonged bedrest leads to progressive reductions in blood volume and impairs venous return, there is a gradual reduction in the diastolic volume and stroke volume declines. To counteract this decrease in stroke volume and maintain sufficient cardiac output, a gradual increase in heart rate normally occurs in patients confined to bed. After four weeks of bedrest, the resting heart rate has typically increased by around 10 beats per minute (bpm). Similarly, the heart rate after exercise is up to 40bpm faster in patients who have just had a period of bedrest, and their tolerance of exercise will not fully normalise until 5-10 weeks after remobilisation (Corcoran, 1991).
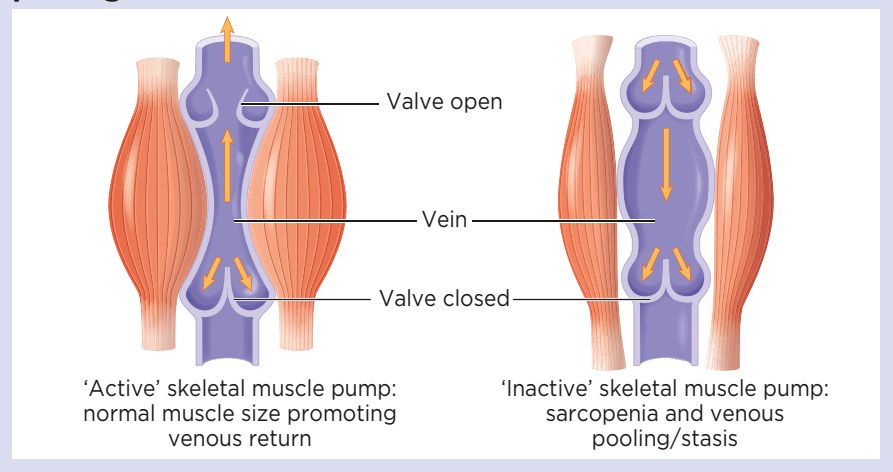
Cardiac deconditioning

Like skeletal muscle fibres, cardiac muscle fibres need the stress of physical work to be maintained; the principle of 'use it or lose it' applies. As stroke volume decreases, the myocardium (heart muscle) has less and less work to do and starts to atrophy. Myocardial thinning, particularly in the ventricular regions, is common in patients confined to bed and has been recorded in both men and women (Dorfman et al, 2007).

Magnetic resonance imaging has shown that after six weeks of bedrest, the total mass of the left ventricle decreased by around 8% and the thickness of the left ventricular wall reduced by around 4% (Perhonen et al, 2001b). As the left ventricle is responsible for ejecting blood into the systemic circulation, it is reasonable to assume that myocardial thinning associated with immobility will greatly reduce the effectiveness of the heart as a pump. However, cardiac deconditioning can be reduced by encouraging immobilised patients to do exercises that aid venous return and increase stroke volume – provided it is safe for them to do so.

Orthostatic hypotension

When moving from a sitting or supine position to a standing position, blood and lymph naturally tend to rush downwards into the lower limbs under the influence of

Fig 2. **Reduced efficiency of skeletal muscle pump after prolonged bedrest**

Valve open
Vein
Valve closed

'Active' skeletal muscle pump: normal muscle size promoting venous return

'Inactive' skeletal muscle pump: sarcopenia and venous pooling/stasis

PETER LAMB

Clinical Practice
Systems of life

Nursing Times.net

For more articles
on cardiology, go to
nursingtimes.net/cardiology

gravity. To reduce this fluid shift, valves in the veins and lymphatic vessels close. Arteries do not have valves so, on standing, there is often a rapid drop in arterial blood pressure. Unless this pressure drop is quickly corrected, there is a risk that blood flow to the brain will be reduced, potentially leading to the dizziness and fainting characteristic of OH.

In healthy, mobile individuals, the rapid drop in blood pressure experienced on standing is immediately detected by the aortic arch and carotid sinus baroreceptors; these quickly relay this information to the:

- Cardiac centre, which responds by increasing sympathetic stimulation of the heart, in turn increasing cardiac output and raising blood pressure;
- Vasomotor centre, which then increases sympathetic stimulation of the blood vessels in the lower limbs, leading to partial vasoconstriction that minimises the downward movement of blood.

These responses help maintain blood pressure and cerebral circulation, which reduces the risk of OH. In patients confined to bed, these mechanisms are impeded by:

- Reduced blood volume due to increased diuresis, which may lead to a greater drop in blood pressure on standing;
- Blunting of baroreceptor reflexes, mainly due to reduced blood volume, which affords less of a stretch stimulus and leads to a progressive decrease in the sensitivity of the stretch receptors;
- Reduced venous return and stroke volume;
- Cardiac deconditioning and associated myocardial thinning, which limits the effectiveness of the heart as a pump.

OH is one of the first problems observed in patients confined to bed and has been recorded after as little as 20 hours of bedrest (Gaffney et al, 1985). Most research indicates that reductions in plasma volume play a key role in OH, but cardiac deconditioning exacerbates the problem (Dorfman et al, 2007). This predisposes patients to OH, which often becomes apparent when they first remobilise. It is not uncommon for older patients to be admitted to hospital with a hip fracture (for example), spend some time confined to bed then, on remobilising, experience a fall (and potentially another fracture) due to OH.

Fainting or dizziness when first mobilising after bedrest can make patients anxious. Extreme symptoms of OH can give rise to panic attacks and subsequently lead patients to develop a fear response to similar situations in the future (Walker et al, 2007). Such 'classically conditioned' fear or anxiety is difficult to treat. In hospital settings, better preparation for moving and handling procedures – such as transferring a previously recumbent patient from a bed to a chair – can help reduce anxiety. Before the manoeuvre, the nurse should explain what they are about to do and the sensations the patient is likely to experience. This can help the patient anticipate 'faint feelings' and overcome their fear.

Recovering sufficient orthostatic function to eliminate susceptibility to OH is a slow process, particularly in older people, but even young, fit and healthy adults may take several weeks after they start mobilising again to fully recover (Fletcher, 2005).

Increased cardiovascular risk

C-reactive protein (CRP) and cystatin C are biomarkers associated with general inflammation in the body, including the inflammatory events involved in atherosclerotic occlusion (where fatty plaque is deposited on the arterial walls). Bedrest has been shown to increase the levels of both CRP and cystatin C, suggesting that longer periods of bedrest may augment the risk of atherosclerosis (Arinell et al, 2011).

Elevated cystatin C is also associated with major cardiovascular diseases such as coronary artery disease, myocardial infarction, hypertension and heart failure, as well as with non-cardiovascular conditions such as diabetes and chronic renal disease (Cepeda et al, 2010). **NT**

References
Arinell K et al (2011) Effect of prolonged standardized bed rest on cystatin C and other markers of cardiovascular risk. *BMC Physiology;* 11: 17.
Cepeda J et al (2010) Cystatin C and cardiovascular risk in the general population. *Revista Española de Cardiología;* 63: 4, 415-422.
Chadwick J, Mann WN (1950) *The Medical Works of Hippocrates.* Oxford: Blackwell Scientific Publications.
Chobanian AV et al (1974) The metabolic and hemodynamic effects of prolonged bed rest in normal subjects. *Circulation;* 49: 3, 551-559.
Corcoran PJ (1991) Use it or lose it – the hazards of bed rest and inactivity. *Western Journal of Medicine;* 154: 5, 536-538.
De Castella T (2018) Plastic spouted beakers 'infantilise' older patients. *Nursing Times;* 114: 8, 6-7.
Dirks ML et al (2016) One week of bed rest leads to substantial muscle atrophy and induces whole-body insulin resistance in the absence of skeletal muscle lipid accumulation. *Diabetes;* 65: 10, 2862-2875.
Dorfman TA et al (2007) Cardiac atrophy in women following bed rest. *Journal of Applied Physiology;* 103: 1, 8-16.
Fletcher K (2005) Immobility: geriatric self-learning module. *MEDSURG Nursing;* 14: 1, 35-37.
Fox MT et al (2018) Perceived acceptability and preferences for low-intensity early activity interventions of older hospitalized medical patients exposed to bed rest: a cross sectional study. *BMC Geriatrics;* 18: 1, 53.
Fox MT et al (2009) Perceptions of bed days for individuals with chronic illness in extended care facilities. *Research in Nursing and Health;* 32: 3, 335-344.
Gaffney FA et al (1985) Cardiovascular deconditioning produced by 20 hours of bedrest with head-down tilt (-5 degrees) in middle-aged healthy men. *American Journal of Cardiology;* 56: 10, 634-638.
Hertzman CA (1968) Rehabilitation of casualties in a combat theater. *American Journal of Physical Medicine;* 47: 3, 113-117.
Kortebein P et al (2008) Functional impact of 10 days of bed rest in healthy older adults. *Journals of Gerontology: Series A, Biological Sciences and Medical Sciences;* 63: 10, 1076-1081.
Kramer A et al (2017) How to prevent the detrimental effects of two months of bed-rest on muscle, bone and cardiovascular system: an RCT. *Scientific Reports;* 7: 1, 13177.
LeBlanc AD et al (1992) Regional changes in muscle mass following 17 weeks of bed rest. *Journal of Applied Physiology;* 73: 5, 2172-2178.
Oliver D (2017) Fighting pyjama paralysis in hospital wards. *British Medical Journal;* 357: j2096.
Perhonen MA et al (2001a) Deterioration of left ventricular chamber performance after bed rest: 'cardiovascular deconditioning' or hypovolemia? *Circulation;* 103: 14, 1851-1857.
Perhonen MA et al (2001b) Cardiac atrophy after bed rest and spaceflight. *Journal of Applied Physiology;* 91: 2, 645-653.
Sprague AE (2004) The evolution of bed rest as a clinical intervention. *Journal of Obstetric, Gynecologic, and Neonatal Nursing;* 33: 5, 542-549.
Van Dongen HP et al (2003) The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep;* 26: 2, 117-126.
VanPutte CL et al (2017) *Seeley's Anatomy and Physiology.* New York, NY: McGraw-Hill Education.
Walker J et al (2007) *Psychology for Nurses and the Caring Professions.* Maidenhead: Open University Press.

Nursing Times Self-assessment online

Test your knowledge with Nursing Times Self-assessment after reading this article. If you score 80% or more, you will receive a personalised certificate that you can download and store in your **NT Portfolio** as CPD or revalidation evidence.

Visit **nursingtimes.net/NTSABedRest** to take the test.

CLINICAL SERIES

Effects of bedrest series

Part 1:	introduction and cardiovascular system	Dec
Part 2:	respiratory system, haematological system	Jan
Part 3:	gastrointestinal, endocrine and nervous systems	Feb
Part 4:	renal, reproductive and immune systems	Mar
Part 5:	musculoskeletal system	Apr
Part 6:	bones, skin and self-perception	May