Paper:
Until the mid-20th century bedrest was considered beneficial but its detrimental effects on the human body and mind are now widely recognised. Sometimes bedrest is unavoidable, but its negative consequences should be minimised. This article, the second in a six-part series on the effects of bedrest, explains how prolonged bedrest impairs respiratory function, increases the risk of deep vein thrombosis (DVT) and embolisation, and affects mood and wellbeing. It also outlines what nurses – along with other health professionals – can do to reduce the detrimental effects of bedrest and highlights the benefits of early and gradual remobilisation.

In this article...
- Mechanisms through which bedrest alters lung volumes and affects airway structures
- Virchow’s triad and risk of deep vein thrombosis as a consequence of bedrest and immobility
- Nursing interventions that help reduce the detrimental effects of bedrest

Key points
- Prolonged bedrest impairs respiratory function and increases the risk of deep vein thrombosis and embolisation
- Prolonged bedrest can lead to depression, anxiety, forgetfulness and confusion
- The longer a patient is confined to bed, the longer the recovery period
- Patients confined to bed need turning and repositioning, cough exercises, range-of-motion exercises, relaxation and communication
- Early and gradual remobilisation is key to improve physical function and mental wellbeing

Effects of bedrest 2: respiratory and haematological systems

Effects on the respiratory system
Lung volume
Prolonged bedrest causes several changes to lung volumes, including:
- Tidal volume – volume of air exchanged during normal breathing
- Residual volume – air remaining in the lungs after a full forced expiration
- Forced vital capacity (FVC) – amount of air that can be forced out of the lungs after a maximal inspiration

Normal tidal volume is typically around 500ml (Montague et al, 2005). In the upright position, 78% of tidal exchange is due to the motion of the ribcage, but this is reduced to around 32% in the supine position. According to Manning et al (1999), this reduction in residual volume is due to:
- Blood moving away from the lower limbs into the abdomen and thorax

A figure published in the first article in this series (bit.ly/NTBedrest1) summarises the effects of bedrest on the respiratory, cardiovascular and haematological systems.
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causing an increase in pulmonary blood volume;
- Abdominal organs shifting towards the thorax, pressing on the diaphragm and compressing the lungs.

Normal FVC is typically around 4.5L (Montague et al, 2005). The supine position reduces both FVC and forced expiration in one second (FEV1). Manning et al (1999) hypothesised that this is due to a combination of airway obstruction (potentially by pooled mucus), increased resistance in the airways and loss of elastic recoil due to structural changes in the lungs.

**Structural changes to the respiratory tract**
The airways of the respiratory tract are coated with a thin layer of mucus, which keeps them moist and traps inhaled foreign particles. The mucus film is propelled by microscopic hair-like structures called cilia. Contaminated mucus is continually swept upwards by this ‘muco-ciliary escalator’ and, on reaching the pharynx, is swallowed to be sterilised in the acidic environment of the stomach.

When a patient is confined to bed, mucus tends to pool, under the influence of gravity, in the lower portions of the airway (Corcoran, 1981). This pooled mucus can swamp the lower portion of the muco-ciliary escalator, impeding its function. This is compounded by the fact that patients confined to bed are often dehydrated, which causes mucus to dry and become extremely sticky and difficult to shift.

The diameter of the airways, particularly the bronchioles, decreases after a period of immobility in the supine position. The supine position causes airway narrowing even in healthy people, but is most notable in older people, overweight people and smokers (Dean, 1985). This reduction in airway size, together with pooled mucus and the weight placed on the ribcage, makes breathing more laboured with fewer deep breaths. This can lead to airways and small areas of lung tissue collapsing (atelectasis), which reduces the area available for gaseous exchange (Corcoran, 1981).

Prolonged bedrest dramatically increases the risk of respiratory tract infection (RTI) – for example, patients with stroke confined to bed for 213 days are two-to-three times more likely to have an RTI than healthy, mobile individuals (Halar, 1994). The ability to cough is greatly diminished, and pooled mucus is allowed to stagnate, thereby reducing the clearance of pathogens and irritants.

Frequently turning and repositioning patients can help prevent the abnormal distribution and pooling of mucus in the respiratory tract. Those who are immobile or confined to bed should try doing cough exercises to help shift pooled mucus.

**Effects on the haematological system**

**Loss of plasma volume and increased blood viscosity**
The diuresis associated with prolonged bedrest leads to a gradual reduction in plasma volume (see part 1). After one week in bed, approximately 10% of plasma volume is lost and, after four weeks, the loss is around 15%. In the early stages of bedrest, the total red cell mass remains relatively constant, but the progressive loss of plasma volume increases the haematocrit (packed red cell volume), which increases blood viscosity (Kaplan, 2006).

**Decreased red cell mass and haemoglobin**

As bedrest is associated with skeletal muscle atrophy (discussed in part 5), there is a gradual reduction in oxygen demand. This is reflected by reduced erythropoiesis (generation of erythrocytes) in the red marrow, which eventually causes erythrocyte numbers to drop, gradually reducing the total red cell mass and total amount of haemoglobin (Kaplan, 2006).

**Reduced oxygen transport**

Reductions in lung function, plasma volume and erythrocyte numbers lead to a decrease in arterial oxygen saturation. Conversely, blood carbon dioxide concentrations increase (Trappe et al, 2006; Manning et al, 1999). These changes in blood gases can have serious consequences for many organ systems, particularly the skin (discussed in part 6).

Hypoxia is a low concentration of oxygen at cellular level. Many older people show signs of it after being in a recumbent position, even for as short a period as a night’s rest (Heath and Schofield, 1999). Hypoxia can cause acute confusion and other changes in cognition such as poor memory, concentration and judgement.

**Reduced maximum oxygen consumption**
The maximum oxygen consumption (VO2 max) – the maximum amount of oxygen used per kilogram of body weight per minute during exercise – is a good general measure of cardiorespiratory fitness. In a healthy individual leading a sedentary lifestyle, a VO2 max of around 35ml/kg/min is typical. Bedrest dramatically decreases VO2 max, with the reduction correlating with the length of bedrest. A loss of around 0.9% VO2 max per day has been reported with 30 days of bedrest (Convertino, 1997). Reductions in VO2 max seem to occur regardless of age or gender. They almost certainly reflect the cumulative negative effects of bedrest on the heart, vasculature, musculature, respiratory tract and oxygen transport.

**Virchow’s triad**

Virchow’s triad refers to three factors that, when present together, dramatically increase the risk of DVT (Montague et al,
Prolonged bedrest activates all three factors and, consequently, up to 13% of patients undergoing prolonged bedrest may develop DVT (Kierkegaard et al, 1987). The three factors of Virchow’s triad (Fig 1) are:

- Venous stasis – reduced efficiency of the skeletal muscle pump (discussed in part 1) leads to sluggish blood flow in the veins of the lower limbs; in some veins, blood flow may cease, leading to blood pooling and venous stasis;
- Hypercoagulability – poor blood flow and pooling of blood in the veins of the lower limbs leads to a reduced clearance of clotting factors by the liver; this, together with reduced plasma volume and elevated haematocrit count, increases the viscosity of blood;
- Blood vessel damage – the endothelial lining of arteries and veins is only one layer of cells thick and, therefore, extremely delicate (Montague et al, 2005); it rests on top of a layer of collagen-rich connective tissue and is incredibly smooth, ensuring a free flow of blood with minimal drag and resistance; the continual weight of the supine body (particularly if the patient is not turned regularly) compresses blood vessels, which can damage the endothelium.

**Risk of DVT**

A study examining the effects of prolonged bedrest on small blood vessels demonstrated impairment of endothelial function and significant endothelial damage (Demiot et al, 2007). Injury to the endothelium is often exacerbated by the pooling of blood and venous stasis. Endothelial cell death and detachment can expose the underlying collagen-rich tissue. Platelets rapidly stick to the exposed collagen fibres and become activated, leading to the formation of blood clots (Kaplan, 2006; Halar, 1994). This pattern of DVT is common, not only after prolonged bedrest but also after immobility of any kind. Most famously, the constraints of economy seats on long-haul flights predisposes passengers to DVT formation. This is often referred to as ‘economy class syndrome’.

There has been much research on the link between length of bedrest and risk of DVT. One study estimated that prolonged immobility of >14 days was associated with a five-fold increase in the risk of DVT (Weill-Engerer et al, 2004). People over the age of 70 years are particularly at risk of DVT on anticoagulation therapy did not increase the risk of PE and was associated with better outcomes in the remission of pain in the affected limb (Liu et al, 2015).

**Risk of embolisation**

Following DVT there is a danger of embolisation. Clots most often develop close to the cusps of venous valves in the calf area. When the patient moves, muscle contraction increases venous blood flow and clots may detach and form emboli, which can travel to distant areas, become trapped in small vessels and cut off blood flow. Emboli commonly locate in the:

- Lungs – where they cause pulmonary embolism (PE);
- Brain – where they cause cerebrovascular accident (stroke);
- Heart – where they cause myocardial infarction.

These three types of embolism are often fatal. Even with modern anticoagulation, PE is still one of the most common causes of unexpected death in hospital patients – although its incidence is decreasing (Kopcke et al, 2011).

The risk of thrombosis and embolisation can be reduced through regular physical therapy. Nurses can also help by encouraging regular leg exercises to keep venous blood flowing in the lower limbs – especially as many physiotherapy services are under resourced. Fig 2 shows simple foot and ankle exercises that encourage venous return (calf pump exercises). Patients in high-risk groups may also need support stockings and/or anticoagulant therapy.

Strict bedrest was long considered a cornerstone of DVT management, but it is now accepted that early mobilisation and exercise in patients on modern anticoagulation therapy does not increase the risk of embolisation (Aschwanden et al, 2001). A recent meta-analysis of 13 studies showed that early mobilisation of patients with DVT on anticoagulation therapy did not increase the risk of PE and was associated with better outcomes in the remission of pain in the affected limb (Liu et al, 2015).

**Effects on mental health**

The second half of the 20th century has brought improved understanding of the link between physical and mental wellbeing; this has been particularly well established in studies on the effects of bedrest. Several studies have ascertained that extended periods of bedrest have a negative effect on the psychological states of patients and relatives (Molfit et al, 2008; Ishizaki et al, 2002; Maloni et al, 2001).

Negative psychological effects of extended bedrest include:

- Depression;
- Anxiety;
- Forgetfulness;
- Confusion.

They may arise due to lack of control, as bedrest restricts the ability to undertake activities usually taken for granted, such as walking to the toilet or stretching the legs. Lack of control over personal environment has long been linked to increased
stress and the release of stress hormones such as corticosteroids (Ogden, 2007). The increased levels of corticosteroids, such as cortisol, that are seen during periods of immobility have multiple detrimental effects on human physiology; these are discussed in part 3.

Being out of the home environment, together with worry about their medical condition, and separation from family and friends, contribute to the low mood and anxiety commonly experienced by patients confined to bed.

Unavoidable bedrest

Some periods of bedrest – such as after major surgery or when in intensive care – are unavoidable. When patients must remain confined to bed, nursing staff need to ensure they are regularly repositioned and, if possible, undergo active or passive range-of-motion exercises. Relaxation and communication with other patients on the ward or volunteers may make a positive contribution to their wellbeing.

Listening to music while confined to bed has been shown to increase levels of the hormone oxytocin, which increases feelings of relaxation (Nilsson, 2009). Music may, therefore, negate some of the negative psychological effects associated with prolonged bedrest. However, the provision of ‘entertainment stations’ at patients’ bedsides is controversial; sometimes promoted as providing TV, phone and radio ‘without having to leave your bed’ they have been shown to result in increased immobility (Papasparys et al, 2008).

Recovery on remobilisation

Most adverse effects of prolonged bedrest on the cardiovascular system (described in part 1) and on the respiratory and haematological systems and mental wellbeing (described in this article) will resolve with physical activity. A prospectively randomised clinical trial. Journal of Clinical Nursing; 18: 15, 2153-2161.

References


