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Clinical Practice
Systems of life
Bedrest

In this article...
- Harmful effects of prolonged bedrest on appetite, digestion and elimination
- Hormonal, metabolic and neural changes occurring in people confined to bed
- Consequences of sensory deprivation and learnt helplessness

Effects of bedrest 3: gastrointestinal, endocrine and nervous systems

Key points

Harmful effects of bedrest on the gastrointestinal system include gastric reflux and constipation

Inactivity and immobility gradually slow down the metabolic rate

Bedrest and immobility promote insulin resistance and impaired glucose tolerance

Prolonged bedrest is likely to lead to changes in brain tissue and brain biochemistry

Patients confined to bed may experience sensory deprivation leading to psychosocial symptoms

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Abstract Patients who spend prolonged periods in bed are at increased risk of a range of physical adverse effects, including gastric reflux, constipation, reduced metabolic rate, glucose intolerance, insulin resistance, sensorimotor dysfunction, reduced serotonin levels and structural changes to brain tissues. They may also experience sensory deprivation leading to psychological symptoms such as aggression, depression and hopelessness. This article – the third in a series exploring the harmful consequences of bedrest on the body and mind – describes changes to the gastrointestinal, endocrine and nervous systems induced by prolonged bedrest and immobility, as well as measures nurses can take to counteract them.

Until the mid-20th century bedrest was considered a beneficial intervention but today there is wide recognition of its negative effects. The first two articles in this series on the effects of bedrest on the body and mind covered the cardiovascular, respiratory and haematological systems as well as mental wellbeing. This third article explores how bedrest compromises the functioning of the gastrointestinal, endocrine and nervous systems.

Effects on the gastrointestinal system
Loss of appetite
Older studies have suggested that prolonged bedrest is linked to a reduced sense of taste and smell and a loss of appetite (Rousseau, 1993; Bortz, 1984). Food intake has been found to be significantly lower in healthy men with sedentary lifestyles compared with their more-active counterparts (Stubbs et al, 2004). However, some recent studies have shown minimal changes in the sensation of hunger during bedrest (Debevec et al, 2016), so the link between appetite and immobility remains unclear.

Gastric reflux
Swallowing is more difficult in the recumbent position, and food takes longer to pass through the stomach – the passage of food is 66% slower than when a person is in the upright position (Thomas et al, 2002). Gastric secretions may collect around, and press against, the lower oesophageal sphincter, causing irritation. Patients confined to bed may, therefore, experience symptoms of gastro-oesophageal reflux (Fig 1) disease, such as regurgitation and heartburn; they also appear to be at greater risk of gastric ulceration (Spellman, 2000). GORD can be alleviated by propping the person up with pillows after a meal, which will reduce the risk of reflux by encouraging gastric juices to collect in the lower part of the stomach.

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Constipation

In patients confined to bed, smaller food intake and slower peristaltic rate lead to reduced motility in the gut, which is associated with atrophy of the mucosal lining and shrinkage of glandular structures (Bortz, 1984). Increased transit times slow down the movement of faeces through the colon and rectum, increasing water reabsorption, and stools progressively harden.

In the upright position, peristaltic motility occurs at a normal rate and faecal material collects in the rectum; under the effect of gravity, stools exert pressure on the anal sphincter, initiating the urge to defecate. In the supine position, this does not happen and the urge to defecate is therefore reduced. As a consequence, people confined to bed are up to 16 times more likely to experience constipation than those who are mobile (Spellman, 2000). Constipation is a particular risk in patients taking opioid-based medication, as drugs such as morphine dramatically slow down gut motility (Holzer, 2014).

If constipation becomes chronic (Fig 2), the build-up of faecal material can exert significant pressure on the colon wall, increasing the risk of diverticulitis (inflammation or infection of the diverticula, small bulging pouches that commonly form in the lining of the intestine after the age of 40).

The risk of constipation can be reduced by ensuring patients eat enough fibre and regularly drink water. The fibre will soak up the water, increasing faecal bulk and softening stools.
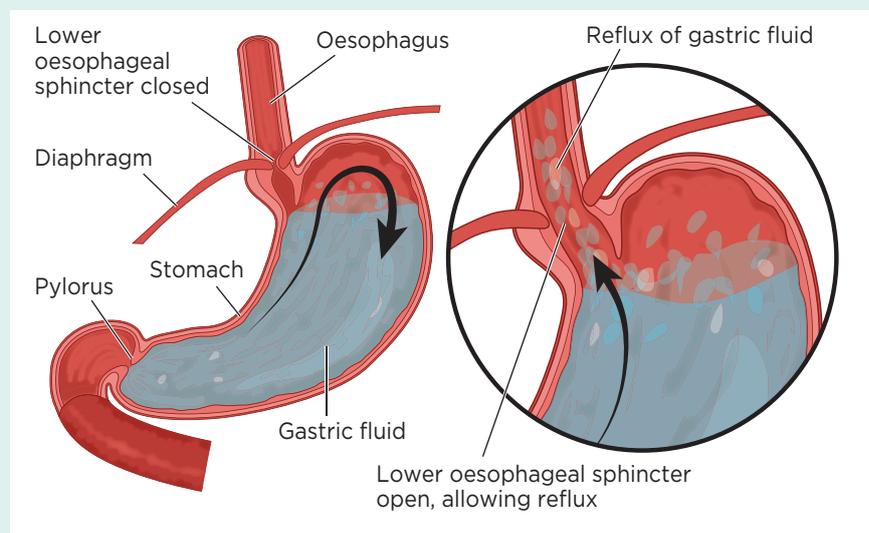
Decreased food intake

Changes in the gut and the reduced appetite that is often reported in patients who are confined to bed can lead to decreased food intake, potentially causing:
- Reduced calorific intake;
- Vitamin and mineral deficiencies.

As tissue healing and recovery from infection require an adequate intake of calories and macro- and micro-nutrients, the detrimental effects of bedrest on the gastrointestinal system can delay recovery. It is essential that nurses ensure adequate nutrition; reduced food intake is now recognised as an independent risk factor for

"People confined to bed are up to 16 times more likely to experience constipation than those who are mobile"

Fig 1. **Gastro-oesophageal reflux**

Lower oesophageal sphincter closed
Oesophagus
Reflux of gastric fluid
Diaphragm
Pylorus
Stomach
Gastric fluid
Lower oesophageal sphincter open, allowing reflux

When a person is in the supine position, gastric fluid may collect around, and press against, the lower oesophageal sphincter, causing irritation; this can lead to gastro-oesophageal reflux

mortality in patients who are hospitalised (Hiesmayr et al, 2009).

Effects on the endocrine system
Increased cortisol secretion

One of the major complications of prolonged bedrest is a progressive loss of muscle mass, known as sarcopenia (see part 5). This is exacerbated by changes in the levels of adrenal glucocorticoid hormones.

Physical injury or starvation prompts the release of the stress hormone cortisol, a natural anti-inflammatory that also promotes gluconeogenesis (generation of glucose derivatives from proteins and fat) (VanPutte et al, 2017). In people confined to bed after an injury and/or surgery, cortisol secretion increases (hypercortisolemia). This leads to skeletal muscle breakdown and the release of amino acids into the blood. Prolonged bedrest also sensitises skeletal muscles to the catabolic effects of cortisol, thereby accelerating muscle atrophy (Fitts et al, 2007; Ferrando et al, 1999).

It remains unclear whether bedrest alone is responsible for increased cortisol levels; some studies report a continuous increase in cortisol secretion (Liang et al, 2012), while others report little change in cortisol levels (Trudel et al, 2009). Based on the current research, it seems likely that sarcopenia associated with prolonged bedrest is predominantly due to a combination of disuse atrophy and increased sensitivity of muscles to the effects of cortisol. Supplementing the diet of patients

confined to bed with a mix of essential amino acids and carbohydrate (sucrose) helps to preserve muscle function and strength (Fitts et al, 2007).

Metabolic changes

Inactivity and immobility lead to a progressive drop in the metabolic rate (Withers et al, 1998). The basal metabolic rate begins to fall after as little as 10 hours of immobility; after 10-24 hours it will have fallen by around 6.9% (Rousseau, 1993). It continues to fall while the body remains sedentary, probably reflecting the decline in lean muscle mass as a result of disuse.

Older studies examining thyroid function during bedrest have reported little change in the hormones that regulate metabolic rate. Plasma concentrations of the hormones triiodothyronine (T_3) – which increases metabolism – and tetraiodothyronine (T_4), also known as thyroxine, remain stable (Balsam and Leppo, 1975). However, more-recent research has shown decreases in plasma T concentrations after eight weeks of bedrest, which is suggested to be due to reduced energy expenditure (Belavý et al, 2012).

A reduced metabolism does not usually lead to weight gain and most patients confined to bed maintain a fairly stable weight. Any weight gain expected because of reduced basal metabolism is probably offset by the reduced lean muscle mass and lower calorie intake (Rousseau, 1993). Unfortunately, while lean muscle mass

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decreases, there is a simultaneous increase in fat stored in the adipose tissues (Krebs et al, 1990).

Glucose intolerance and insulin resistance

Immobility – or simply a sedentary lifestyle – has been linked to insulin resistance, impaired glucose tolerance and, in some people, the subsequent 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, with a progressive development of glucose intolerance that correlates directly with the length of time spent in bed (Rousseau, 1993; Takayama et al, 1974).

The number of insulin receptors expressed in skeletal muscles increases in proportion to physical activity. In people who are active and exercise regularly, the expression of insulin receptors is high. When they eat a meal that is rich in carbohydrates, their blood-glucose levels rise, which triggers the release of insulin. The insulin binds to the abundant receptors in the skeletal muscles, which promotes rapid glucose uptake by the muscles, following which blood-glucose levels return to normal.
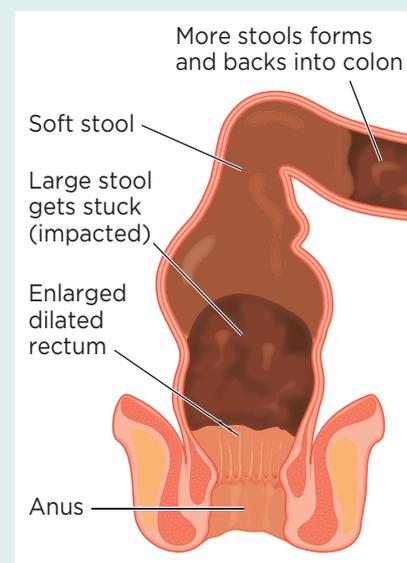
Conversely, immobility and reduced food intake are associated with a reduced expression of insulin receptors in the skeletal muscles (Rousseau, 1993). In people who are confined to bed, the sensitivity of the skeletal muscles to the effects of insulin is lower, so eating a meal that is rich in carbohydrates will result in lower glucose uptake by the muscles and higher blood-glucose concentration.

With prolonged bedrest and immobility, insulin resistance – the key feature of type 2 diabetes – progressively increases. Fasting blood-glucose levels have been reported to be significantly increased in patients who have been confined to bed for 10 days (Coker et al, 2014).

Reduced sensitivity to insulin typically results in the overproduction of insulin by the pancreatic islets, leading to hyperinsulinaemia (Blanc et al, 2000), which is known to trigger fat deposition and thought to be linked to obesity (Erion and Corkey, 2017). It seems likely that insulin resistance and the associated hyperinsulinaemia are linked to changes in blood-lipid profile and fat deposition.

Immobility and bedrest elevate blood triglyceride and low-density lipoprotein cholesterol levels, and promote lipid accumulation in liver and muscle – although the exact mechanisms through which this

Fig 2. **Chronic constipation**

- More stools forms and backs into colon
- Soft stool
- Large stool gets stuck (impacted)
- Enlarged dilated rectum
- Anus

Patients who are confined to bed are at increased risk of chronic constipation as a result of:
- Reduced motility in the gut
- Increased transit times
- Increased water reabsorption
- Increased hardening of stools
- Reduced urge to defecate

occurs remain unclear (Coker et al, 2014). Such changes to blood-lipid profiles would increase the rate of atherosclerotic occlusion in patients who are confined to bed (see part 1). Increases in fat deposition in the liver may also be linked to non-alcoholic fatty liver disease, which itself appears to be linked to reduced physical activity (Bergouignan et al, 2011).

Glucose intolerance and insulin resistance can be alleviated by light exercise, as this increases the number of insulin receptors in skeletal muscle, thereby enhancing the effects of insulin. Although it can be difficult to persuade patients who are confined to bed to exercise, nurses should try to motivate them to do so. Increased muscular activity is associated with reduced glucose intolerance, so even light exercises undertaken in bed would be beneficial. Reversing bedrest-induced impaired glucose tolerance via light exercise typically takes 5-14 days, even in young healthy adults (Heer et al, 2014).

Renin-angiotensin-aldosterone cascade

The renin-angiotensin-aldosterone cascade plays a key part in the long-term control of blood pressure (VanPutte et al, 2017).

When blood pressure drops, the kidneys release renin, an enzyme that catalyses the conversion of the plasma protein angiotensinogen into angiotensin I; this is rapidly converted into angiotensin II by angiotensin-converting enzymes in the lungs. Angiotensin II, a potent vasoconstrictor, increases blood pressure and stimulates the adrenal glands to release the hormone aldosterone, which increases sodium reabsorption in the kidneys, increasing blood-sodium levels, blood volume and blood pressure.

In people who are 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 (see part 2), initiates the renin-angiotensin-aldosterone cascade, which has the effect of increasing plasma renin activity and plasma aldosterone levels (Annat et al, 1986; Gharib et al, 1985). This stimulates the kidneys to reabsorb more sodium, helping to maintain blood volume and arterial pressure."

Effects on the nervous system
Blunting of baroreceptor response

To date, there have been few studies examining how prolonged bedrest affects the nervous system. Among existing studies, most have explored the effects of immobility on the autonomic nervous system, particularly the blunting of baroreceptor responses, which increases the risk of orthostatic hypotension (see part 1). Prolonged bedrest is associated with sensorimotor dysfunction that commonly manifests as postural instability and a dysregulated sense of balance (Yuan et al, 2018). This, together with reduced muscle mass and strength, increases the risk of falls (see parts 1 and 5).

Changes in brain tissue

Magnetic resonance imaging studies have revealed that prolonged bedrest alters the structure of brain tissue, with losses of grey matter in the temporal and frontal lobes thought to be linked to reduced synaptogenesis (synapse formation), as well as negative effects on locomotion, coordination and cognition (Li et al, 2015). However, research in that domain is still sparse.

Neurochemistry

There is also a paucity of information on how brain biochemistry is affected by bedrest. Older studies have revealed that levels of major neurotransmitters – including dopamine, noradrenaline and

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serotonin – tend to decrease after periods of inactivity (Corcoran, 1991). Serotonin is known to play a key role in mood, cognition and appetite (Jenkins et al, 2016), so reduced serotonin levels reported during immobility may be linked to the depressed mood, reduced cognitive skills and loss of appetite commonly seen in patients who are confined to bed.

Sensory deprivation and learnt helplessness

Patients who are confined to bed in hospital often experience a reduction in environmental and psychosocial stimuli, because of limited opportunities for interactions outside of their immediate environment. This restriction, sometimes called sensory deprivation (Hayes, 2000), can have a knock-on effect on behaviour. It is often associated with:

● Restlessness;
● Increased aggression;
● Insomnia;
● Reduced pain threshold (Fletcher, 2005; Rousseau, 1993).

Information comes to the brain both from outside and within the body. External and internal information constantly 'compete' for the individual's attention. When the external environment is relatively 'quiet', increased attention is paid to information coming from within the body.

Forced bedrest often leads to sensations of uncertainty and unpredictability, which may, in turn, lead to anxiety. Lack of control or hopelessness are associated with depression (Walker et al, 2012). This effect of bedrest on mental state is exacerbated by changes in neurochemistry, such as the decrease in serotonin levels, outlined above.

Hospital patients confined to bed may become increasingly dependent, relying on staff to help even with simple tasks; this 'learnt helplessness' (Corcoran, 1991) is often reinforced by the well-meaning support of staff. According to the theory of learnt helplessness outlined by Seligman (1975), people exposed to uncontrollable

events initially react against the stressor with anger and frustration.

Nurses need to keep in mind that perceptions and sensations are based on personal experiences and will vary between patients. Two patients experiencing the same, prolonged period of bedrest may react in completely different ways. It is also worth remembering that anxiety and depression are normal and reversible responses to stressful situations, but also that they can have serious disabling effects when experienced intensely or for long periods of time.

The complications of bedrest-related sensory deprivation can be partly alleviated by encouraging social interaction, exercise and early mobility (Rousseau, 1993). Providing medium- and long-stay hospital patients with information about the physical and psychological effects they are likely to experience during prolonged bedrest may help to reduce their anxiety. **NT**

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