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In this article...

- Effects of prolonged bedrest on bones, skin and self-concept
- Pathophysiology of disuse osteoporosis and pressure ulcers
- Bedrest-induced alterations to body image, self-concept and self-esteem

Effects of bedrest 6: bones, skin, self-concept and self-esteem

Key points

Immobility may lead to reduced bone mass and density, bone demineralisation and bone loss

Patients confined to bed are at increased risk of kidney stones and disuse osteoporosis

Prolonged pressure on skin over bony prominences may lead to pressure ulcers

Pressure ulcers can be prevented by position changes, skin care, risk factor assessment and observation

Bedrest alters body function and appearance, so it influences patients' self-concept

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Abstract Although some patients require bedrest to recover from an illness or surgery, prolonged immobility has detrimental effects on the systems of the body. This article – the sixth and last in a series exploring the harmful consequences of bedrest on body and mind – describes how prolonged bedrest and immobility affect the bones, skin and self-concept.

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Bedrest is often necessary to recover from injury or disease but prolonged immobility is detrimental to all major organs and human body systems. This is the final article in our six-part series on the harmful effects of bedrest. It discusses how bedrest may affect bones and skin, increasing the risk of hypercalcaemia, kidney stones, disuse osteoporosis and pressure ulcers. It also explores how bedrest may lower self-esteem and alter patients' self-concept.

Effects on bone

The two primary functions of bones are to mechanically support tissues and muscles, and maintain mineral homoeostasis by acting as a reservoir of calcium, phosphorous and magnesium salts (VanPutte et al, 2017). In the skeleton, most of the calcium and phosphorus is present in the form of crystals of hydroxyapatite, the deposition and orientation of which are influenced by mechanical stresses on the bone (Montague et al, 2005). When there is little force acting on the body for a prolonged period, a drastic reduction in the mineral content of bone tissue is seen, leading to reduced bone density and strength. One study indicated that around 1% of bone density was lost in the vertebral column after only one week of immobility (LeBlanc et al, 1994).

Bone metabolism

It is a common misconception that bones are static tissues. In reality, bone, like muscle, is dynamic. Normal bone metabolism (remodelling) depends on two types of cells:

- Osteoblasts – bone-forming cells that are responsible for synthesising new bone tissue;
- Osteoclasts – bone-digesting cells that are responsible for breaking down (re-absorbing) existing bone tissue.

When the body is upright and engaging in regular exercise, osteoblasts and osteoclasts work at approximately the same rate; as a result, overall bone density is maintained and the skeleton is in a state of relative equilibrium.

Osteoblasts rely on the stress of body weight during normal daily activities to maintain their rate of bone formation (Takata and Yasui, 2001). During a period

of immobility, the mechanical loading of the skeleton is reduced so osteoblast activity declines, resulting in reduced bone synthesis. In the meantime, osteoclasts maintain their activity and continue to break down bone at a relatively stable rate; indeed, some studies suggest that reduced skeletal loading may even enhance osteoclast activity and, therefore, speed up bone demineralisation (Lau and Guo, 2011; Takata and Yasui, 2001). The end result is a gradual loss of bone density, leaving bones vulnerable to fracture after minor falls, during wheelchair transfers or even during physical therapy.

Bone is classified into two types:

- Compact (cortical) bone – dense and strong, this is primarily found in the shafts (diaphyses) of long bones such as those in the arms and legs;
- Cancellous (trabecular or spongy) bone – this is found at the epiphyses (enlarged, bulbous ends of long bones) and in the middle portions of flat bones such as the ribs and pelvis.

During periods of immobility, both compact and cancellous bone is lost, although the loss of bone occurs at a much slower rate than the loss of muscle (part 5). Cancellous bone appears to be particularly vulnerable to demineralisation.

Age can affect the rate of bone formation and resorption: the decreased production of sex hormones (see part 4) correlates with decreased bone turnover (total volume of bone re-absorbed and formed over a given period). However, surprisingly, a recent study examining bone loss after 16 days of bedrest showed that it was less pronounced in older men (those over the age of 60 years) than in their younger counterparts (Buehlmeier et al, 2017). The reasons for this have not been clearly established.

Calcium levels

Within just a few days of immobility, bone demineralisation leads to loss of calcium from bone and, therefore, a rise in plasma calcium levels; after five weeks of bedrest there is a measurable increase of up to 50ml in the amount of calcium excreted in the urine (Zerwekh et al, 2007). If immobility and bone demineralisation continue, this significantly increases the risk of renal precipitation, potentially encouraging the formation of kidney stones (see part 4). Longer periods of bedrest result in much greater reductions in bone density.

A high-calcium diet will not necessarily improve bone uptake of the mineral; instead, it may add to the excess calcium already excreted in the urine and further increase the risk of kidney stones.

In patients with heterotopic calcification (also called myositis ossificans), excess calcium is deposited in soft tissues such as muscles, blood vessel walls and cardiac valves, where it may interfere with joint and/or muscle function, or even cause valve stenosis. This will be exacerbated by any immobility-related increase in calcium excretion.

High levels of plasma calcium (hypercalcaemia) can interfere with synaptic transmissions in the nervous system, leading to:

- Confusion;
- Muscle pain;
- Fasciculation (twitching);
- Nausea, possibly associated with reduced appetite and anorexia (see part 4).

"When patients with osteoporosis resume weight-bearing activities, they may experience pain"

Bone mass and bone density

Loss of bone mass and density may lead to osteoporosis, which renders bones increasingly fragile and liable to fracture. Fractures associated with osteoporosis, called fragility fractures, occur following skeletal stress that would not normally be expected to result in fracture, such as after a fall from standing height or lower (National Institute for Health and Care Excellence, 2012).

Many factors increase the risk of osteoporosis, including age, smoking, being female, poor diet that lacks vitamin D and/or calcium, a family history of the disease, low body weight (and therefore reduced skeletal loading) and reduced mobility (Bit.ly/NOFOsteoporosisRisk). Prolonged bedrest reduces skeletal loading and stress, and the demineralisation and reduced skeletal mass associated with immobility are referred to as disuse osteoporosis (Lau and Guo, 2011).

When patients with osteoporosis resume weight-bearing activities after a period of bedrest, they may experience pain, most often in the back. This may be caused by:

- Vertebral fractures;
- Bone deformation;
- Increased muscular tension and/or joint imbalances.

A recent review has outlined the nature, origins and treatment of pain associated with osteoporosis (Catalano et al, 2017).

Any patient experiencing pain on remobilisation requires careful assessment, as the risk of fracture is known to be significantly higher after prolonged immobility. Lost bone mass is not regained for some weeks after muscle mass and strength have returned to normal (Bloomfield, 1997).

Bone marrow

There are two types of bone marrow:

- Yellow marrow – this is composed predominantly of adipose tissue (fat) and, in adults, found predominantly in the cavity that runs through the shaft of long bones such as the femur and humerus (medullary cavity);
- Red marrow – the major haematopoietic tissue, this is responsible for producing erythrocytes (red blood cells), leukocytes (white blood cells) and thrombocytes (platelets). Most of it is found in the cancellous bone located in the central portion of flat bones such as the pelvis, ribs and vertebrae; smaller amounts are found in the epiphyses of major long bones (VanPutte et al, 2017).

There have been few studies examining the effects of immobility on bone marrow. However, the decreased mechanical stress on bone seen during periods of immobility appears to be associated with an increase in the amounts of yellow bone marrow (David et al, 2007).

Trudel et al (2009) proposed that extended bedrest increases the deposition of adipose tissue in regions of bones that play a key role in generating blood cells. In their study, women confined to bed for 60 days showed an increase in fat deposition of around 9% in the lumbar vertebrae, and fat levels had not returned to normal even a year after remobilisation. Immobility also appeared to foster activity in the red bone marrow, with increased production of some major leukocytes, including neutrophils and antibody-producing lymphocytes. The researchers speculated that the increase in fat deposition in bone may trigger increased red bone marrow activity.

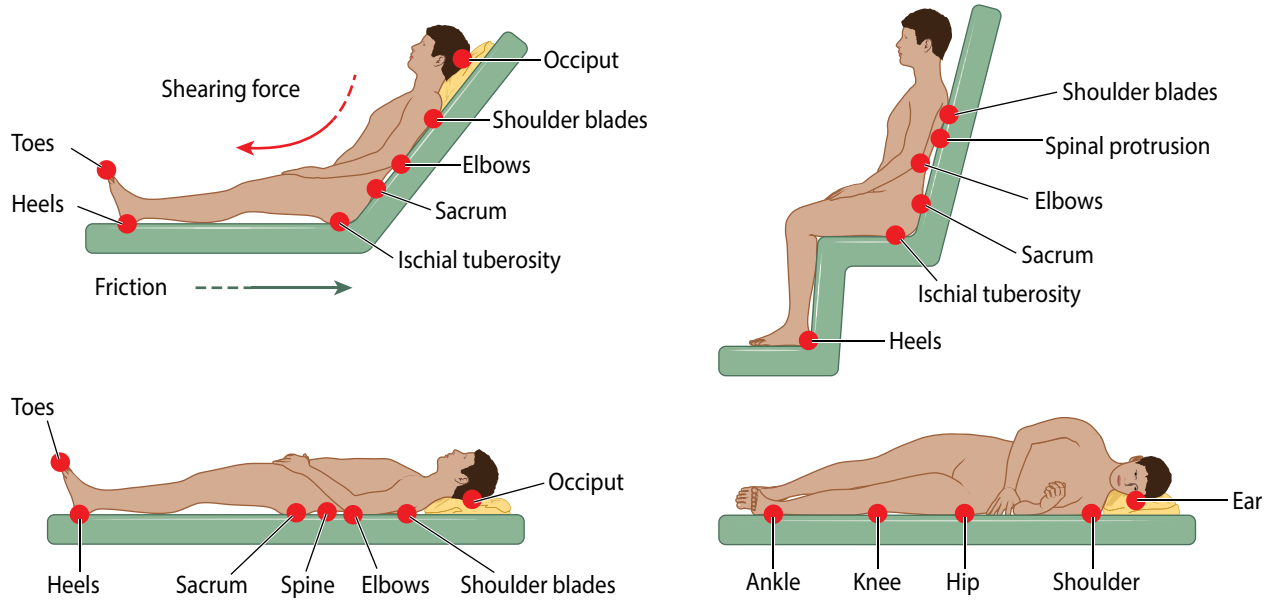
Effects on skin

The role of the skin includes:

- Protecting underlying muscles, bones and organs;
- Regulating body temperature;
- Contributing to the sensory system.

The only areas of skin adapted to bear weight for long periods are the soles of the feet, where the outer layer (epidermis) contains an additional layer of spongy cells

Fig 1. **Body sites at greatest risk of pressure ulcers in different positions**



(stratum lucidum). During bedrest, a large surface area of skin bears the weight of the body and is in constant contact with the bed. Immobility is the factor most likely to put an individual at risk of altered skin integrity.

Pressure ulcers

Aetiology. Usually, to relieve discomfort, people automatically shift their weight off pressure areas every few minutes, even during sleep. Patients confined to bed for long periods may be unable to do this because they have lost muscle mass and are not physically strong enough. Those with decreased sensation (for example, patients with sensory neuropathy from diabetes) may not feel the painful stimuli associated with skin damage that would normally prompt a change of position.

Without regular changes of position in bed, the prolonged pressure on the skin compresses blood vessels in the dermis, depriving both dermal and epidermal layers of blood (ischaemia), which ultimately results in the death of skin tissue (necrosis). Areas where skin is stretched tautly over bony prominences are at the highest risk of breakdown. Here, the risk of ischaemia is at its highest because blood vessels are easily compressed between the bone and a hard surface such as a bed or chair (Gulanick and Myers, 2007).

Impaired flow of lymph and blood may cause ischaemic lesions (commonly referred to as pressure ulcers), decubitous ulcers or bedsores. Extended periods of pressure on the skin (capillary pressure >33mmHg) may result in damage and necrosis of the skin and underlying tissues as a result of ischaemia (Agrawal and Chauhan, 2012). The longer the period of bedrest or immobility and the greater the pressure exerted, the higher the risk of a pressure ulcer developing.

At-risk patients and body sites. Pressure ulcers occur most frequently in:

- Older patients who are immobilised;
- Patients in critical care settings;
- Patients with spinal cord injuries.

Research has shown a significant reduction in blood flow in the sacral skin of nursing home residents when in the supine position and, therefore, an increased risk of pressure ulcer formation (Källman et al, 2016). A large study of 3,233 patients aged ≥65 years showed that:

- Around 6.2% developed pressure ulcers within two days of hospital admission;
- A majority of pressure ulcers were assessed as stage 2 (affecting the epidermis and dermal layers of the skin);
- Ulcers were most frequently located in the sacral area and on the heels (Baumgarten et al, 2006).

About 95% of all pressure ulcers occur at five sites:

- Sacrum;
- Ischial tuberosity (sitting bones);
- Greater trochanter (the bony prominence over the side of the hip);
- Heel;
- Ankle.

In patients who are supine, pressure ulcers tend to occur mainly on the sacrum and heels; in patients who are sitting or reclining, they tend to develop on the ischial tuberosity. Fig 1 shows the body sites at greatest risk of pressure ulcers in the reclining, sitting and lying positions.

Risk factors and prevention. As well as age and pre-existing medical conditions, such as diabetes, many of the predisposing factors for pressure ulcer formation can be modified with lifestyle changes. These modifiable risk factors include:

- Extended immobility – recognised as the main risk factor for developing pressure ulcers, particularly in those patients who are frail and/or unable to regularly change position in bed (Anders et al, 2010);
- Poor nutrition;
- High body mass index;
- Smoking.

Prevention is better than cure for pressure ulcers and this can be achieved by:

- Frequent changes of position;
- Meticulous skin care;
- Early assessment of risk factors;
- Careful ongoing observation.

The risk of pressure ulcers can also be greatly reduced by ensuring that patients

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who are confined to bed receive adequate nutrition and remain hydrated; this includes careful monitoring of total calories consumed and of macro- and micronutrient intake (Posthauer et al, 2015). Patients should be assessed individually according to their needs. A risk assessment for pressure ulcers should explore a variety of factors – the main ones are listed in Box 1.

Patients found to be at risk of developing pressure ulcers need regular turning or repositioning. Repositioning a supine patient in bed is a skilled procedure: if undertaken incorrectly, it can cause additional friction and shearing, and pull weakened skin over muscles and bony ridges. There is a variety of specialised devices, including modified beds and rolling equipment, that can reduce the risk of skin damage when repositioning patients in bed. Devices that can help relieve skin pressure over bony prominences include air mattresses and specially designed cushions and seating.

Patients should be encouraged to keep mobile whenever possible. Getting out of bed and mobilising as soon as possible is recognised as the best strategy for the prevention of pressure ulcers.

Effects on self-concept

Immobility and the numerous associated physiological changes described in this series can affect patients' self-concept. The self is a central concept in psychology; self-concept is described as a stable set of beliefs about one's qualities and attributes (Marks et al, 2018). Related to this is self-esteem, which is the feeling of self-worth and a central component of psychological wellbeing (Walker et al, 2007). Self-concept and self-esteem are made up of an individual's body image, achievement, social functioning and self-identification.

Although levels of self-esteem and self-concept tend to remain stable, events such as sudden or chronic illness can produce drastic changes. Prolonged bedrest, which causes both a decrease in body function and an altered appearance, can force patients to re-evaluate their self-concept.

Studies of patients who were hospitalised indicated that body image plummets during illness (Fingeret et al, 2014). Hospitalisation may affect:

- The achieving self – prolonged bedrest threatens the achievement felt from work or hobbies. Many people draw a sense of achievement from their jobs, interests and activities, which is threatened if they are unable to carry them out;
- The social self – interactions with friends and family are a vital source of self-esteem and emotional support. A breakdown in this support system can have harmful consequences for the patient's sense of identity and security;
- The private self – bedrest creates a dependency on others; the resulting loss of independence and strain of imposing on others can diminish the private self.

Conclusion

This six-part series has explored the detrimental effects of prolonged bedrest and immobility on the functioning of the major organs and body systems, as well as on patients' mental wellbeing. Most research unequivocally indicates that, whenever possible, patients should remain as active and mobile as they can, both while in hospital (even when confined to bed) and when recovering at home. Patients who can take regular light exercise should do so, as this has been proven to reverse or prevent many of the adverse effects of prolonged immobility discussed in this series, and speed up recovery. **NT**

Box 1. **Pressure ulcer risk assessments: what to consider**

- Reduced mobility
- Current skin condition
- Previous history of pressure ulcers
- Reduced sensation (neuropathy)
- Nutritional status
- Cognitive status
- Patient's ability to reposition themselves

Source: National Institute for Health and Care Excellence (2014)

"Patients who can take regular light exercise should be encouraged to do so"

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