

In this article...

- Effects of prolonged bedrest on muscles, tendons, ligaments and cartilage
- Mechanisms of muscle disuse, weakness and atrophy
- Immobility-induced alteration of collagen structure in tendons and ligaments

Effects of bedrest 5: the muscles, joints and mobility

Key points

Disused muscles lose mass and strength, become weaker and undergo atrophy

Sarcopenia is associated with reductions in the size of muscle fibres

Loss of muscle strength during prolonged bedrest is also due to metabolic and neural changes

Immobility can cause contractures severe enough to restrict the range of movement in major joints

Exercise and mobilisation, when feasible, help avoid muscle disuse and joint contractures

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Abstract This article – the fifth in a series exploring the harmful consequences of bedrest on body and mind – describes how prolonged bedrest and immobility affect muscles and joints, two of the main components of the musculoskeletal system. The sixth and final article in the series will cover the skeletal system.

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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. Muscles and joints allow the body to move and carry out physical activities, and muscle weakness or joint stiffness resulting from immobility may limit patients' physical activity and reduce their quality of life. Prolonged bedrest often leads to reduced muscle mass and increased risk of fracture. This fifth article in our six-part series explores how bedrest affects muscles and joints, two components of the musculoskeletal system. Effects on bones will be discussed in part 6.

Effects on muscles

Due to its heavy demand for oxygen and glucose, muscle tissue is metabolically expensive for the body to build and maintain. Muscles rapidly undergo disuse atrophy and lose mass after only a short period of immobility. The principle of 'use it or lose it' applies perfectly to them.

Atrophy and sarcopenia

Disuse of skeletal muscles rapidly leads to a loss of lean muscle mass (sarcopenia) as individual muscle groups atrophy. This is

accompanied by a decline in skeletal muscle strength at a rate of around 12% a week (Jiricka, 2009) or even up to 40% within the first week of immobility (Topp et al, 2002).

In patients who have had a stroke, are paralysed or have limbs immobilised by a splint, muscles atrophy with a loss in size and mass of 30–40%. People with peripheral nerve injury that leads to flaccid paralysis can lose as much as 95% of lean muscle mass in affected muscles, where fibres are replaced by fat and connective tissue (Dittmer and Teasell, 1993).

One study found that 72 hours of limb immobilisation could cause atrophy of slow- and fast-twitch muscle fibres by 14% and 17% respectively (Lindboe and Platou, 1984). The larger and better trained the muscle, the faster the loss of strength and the quicker the deconditioning (Jiricka, 2009). Muscle fibre atrophy quickly leads to a loss of strength and mass in the postural muscles of the back, legs and arms.

Among the first muscles to atrophy and weaken are those in the lower limbs, because they resist gravitational forces in the upright position (Parry and Puthucherry, 2015). Extensor muscles (such as the quadriceps femoris), which have a

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prime role in posture, tend to atrophy more than flexor muscles (such as hamstrings). Backache and fatigue during convalescence are often due to disuse atrophy of the underlying core muscle groups, rather than the condition that necessitated bedrest. Postural and locomotive muscles lose their tension-generating capacity, while paraspinal and abdominal muscles become weak if not used.

Slow- and fast-twitch fibres

Skeletal voluntary muscle consists of two types of fibres:

- Slow-twitch (type 1);
- Fast-twitch (type 2).

Slow-twitch muscle fibres contract slowly and produce large amounts of energy so they can keep moving for long periods. They are rich in blood capillaries, mitochondria (the organelles that release energy) and myoglobin, a protein pigment similar to haemoglobin that binds to and releases oxygen during muscular contraction. These features make slow-twitch muscles resistant to fatigue (VanPutte et al, 2017). Slow-twitch fibres are abundant in the muscles of the neck and back, where they help maintain posture while sitting or standing. They are also abundant in many muscles of the lower leg, such as the soleus, where they support endurance activities such as long-distance running and cycling.

Fast-twitch muscle fibres contract quickly but contain little myoglobin and fewer mitochondria. They are found at high density in the muscles of the arms, where they allow rapid movements. Fast-twitch fibres are not able to generate a steady supply of adenosine triphosphate (ATP) to power muscle contraction so, although they contract more rapidly, they also tire more quickly.

Long periods of immobility affect the two types of muscle fibres differently. Studies conflict as to whether one type atrophies faster than the other (Topp et al, 2002; Kannus et al, 1998). The consensus today is that sarcopenia occurring as a result of immobility is associated with an overall reduction in size in both slow- and fast-twitch fibres, with a slightly more rapid loss in the fast-twitch type (Parry and Puthucherry, 2015).

Metabolic changes

A small bedrest study of six men found that, after 14 days of immobility, there was a significant decrease in their leg and whole-body lean muscle mass (Ferrando et al, 1996). This sarcopenia coincided with a 50%

drop in protein synthesis within muscle, which suggests that immobility not only causes muscle atrophy, but also reduces the biosynthesis of new muscle tissue.

Reduction in muscle strength is not only due to the physical loss of muscle fibres, but also to metabolic changes within muscle tissue. The primary source of fuel for muscle contraction is glucose, which is delivered to muscle fibres under the control of the hormone insulin. Glucose is then stored in the muscles in the form of an animal starch called glycogen (VanPutte et al, 2017).

Periods of immobility have been linked with decreased glycogen stores and a reduced ability of muscle to mobilise fatty acids. Simultaneously, the activity of oxidative enzymes within contractile muscle fibres drops with disuse, leading to a reduction in the use of oxygen by muscle tissue. This reduced oxidative capacity of mitochondria contributes to muscles tiring more easily in patients who are immobile. Furthermore, the detrimental effects of bedrest on the cardiovascular and respiratory systems (see parts 1 and 2) result in reduced blood flow in, and oxygen supply to, muscle tissue.

“The risk of contracture can be reduced through good positioning and body alignment in bed”

Protein synthesis and catabolic breakdown

Muscle is a dynamic tissue that is broken down and rebuilt when required. Atrophy and sarcopenia associated with prolonged bedrest typically occur when there is an imbalance between protein synthesis and the catabolic breakdown of muscle. The loss of lean muscle mass occurs primarily through disuse, but many researchers have highlighted increases in the long-term stress hormone cortisol during prolonged bedrest – and cortisol is known to stimulate the catabolic breakdown of muscle (see part 3).

Prolonged immobility has also been linked to an increased production of various inflammatory mediators and damaging superoxide anions, both of which are associated with reduced protein synthesis and increased muscle breakdown (Puthucherry et al, 2010).

Fitts et al (2007) showed that the negative effects of bedrest on human skeletal muscle fibres could be partially offset by

dietary supplementation with amino acids and sucrose. They also demonstrated that simulating an increase in the levels of plasma cortisol to mimic those seen in a hospital inpatient caused an increase in muscle protein catabolism, which resulted in muscle breakdown exceeding protein synthesis.

Neurological changes

Bedrest-induced physiological changes in neural control (see part 3) contribute to the deterioration of muscle strength and endurance. In patients who are inactive, motor unit recruitment (the progressive activation of a muscle by successive recruitment of contractile units) is diminished, as is the ability to activate all motor units during contractions.

Changes in electrical activity within muscles and a loss of integrity of the neuromuscular junction have also been reported following immobility (Blotner and Salanova, 2015), potentially contributing to the fatigue seen with muscle disuse.

Avoiding disuse weakness

A recent study showed that, during 60 days of bedrest, participants who did three minutes of light jumping exercises (consisting of three series of 12 jumps repeated six or seven times a week) maintained leg muscle strength, whereas control groups (who did not take part in the exercise) lost around 40% of their leg muscle strength (Kramer et al, 2017).

Similar results have been reported with the use of flywheel exercise machines to provide resistance training for patients confined to bed for 90 days; the exercise was effective in reducing leg muscle atrophy and exercising participants retained a higher leg muscle mass than their non-exercising counterparts (Belavý et al, 2017).

Clearly, not all patients confined to bed will be able to exercise, but Belavý et al (2017) highlight the importance of exercise in maintaining lower limb strength in those who are able and willing to do so. As highlighted in part 1 of this series, maintaining muscle mass in the legs is particularly important: when these muscles contract, they squeeze the veins of the legs, encouraging venous return to the heart, which helps prevent the pooling of blood in the legs, thereby reducing the risk of venous stasis and clots.

Remobilisation

On remobilisation, disuse weakness is typically reversed at a rate of around 6% per week with exercise. It typically takes about

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four weeks to recover from the disuse atrophy caused by immobility, which is slower than the recovery from direct muscle trauma (Halar, 1994).

Loss of muscle mass and strength can have negative psychological effects on patients, contributing to fatigue and low mood. This may affect their motivation to undertake exercise and lead to a vicious cycle of immobility and inactivity.

The significant loss of strength in the major muscle groups involved in maintaining posture may partially explain why patients may be unsteady on their feet when they start to remobilise; it certainly contributes to an increased risk of falls.

Effects on tendons, ligaments and cartilage

Tendons, ligaments and cartilage (Fig 1) require motion to stay healthy and will, therefore, deteriorate in patients who are confined to bed. Changes to their structure and functioning start to become apparent after 4-6 days of immobility and can remain, even after normal activity has been resumed. Most of these changes appear to be due to the altered structure of collagen fibres.

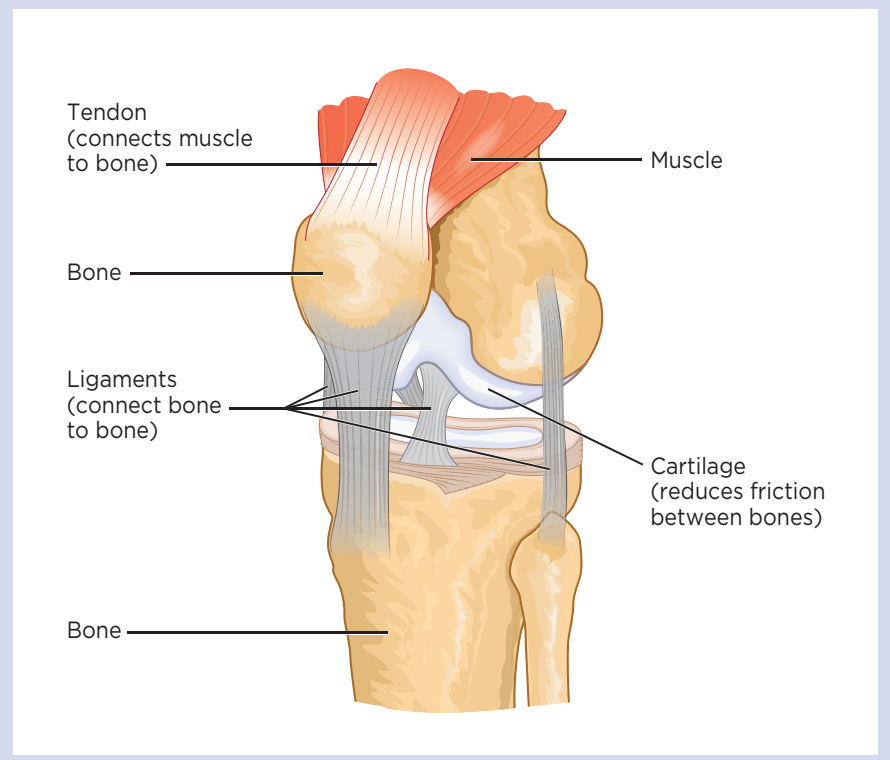
Tendons are stiff, cord-like structures connecting muscle to bone; 20 days of bedrest reduces their stiffness and increases their viscosity (Kubo et al, 2004), which negatively affects the transmission of energy from muscle to bone and reduces the ability of muscle groups to produce dynamic force. This may manifest as increased weakness and exhaustion.

Ligaments are elastic structures connecting bone to bone; their elasticity allows joints to maintain mobility while ensuring they are held together and not easily dislocated. Articular cartilage is the smooth, translucent or transparent tissue that covers the ends of bones where they come together to form joints; healthy cartilage allows bones to glide over each other with little friction. Ligaments and articular cartilage are both negatively affected by prolonged immobility.

Joint contracture

A contracture is a permanent shortening of tissue – such as muscle, tendon or skin tissue – resulting from disuse, injury or disease. It can occur, for example, as a result of changes to the collagen composition of tendons and ligaments caused by disuse. Although contractures are extremely common, their aetiology is still poorly understood (Wong et al, 2015). Muscle atrophy plays a part in their

Fig 1. **Accessory structures of the musculoskeletal system**



development because of the shortening and weakening of the muscles.

Contractures can develop over joints, often when there is an imbalance in the strength of opposing muscle groups. If allowed to progress, a joint contracture may develop to involve muscles, tendons, ligaments and internal structure of the joint capsule, resulting in a stiffening joint that is increasingly limited in its range of motion. A common example of joint contracture caused by immobilisation is contracture of a knee that has been plastered to treat a fractured tibia.

Joint contractures may begin to form within as little as eight hours of immobility (Corcoran, 1991). This may partially explain the morning stiffness many people experience after a night's sleep. Most morning stiffness is transient as, after activity is resumed, the joint tissues are stretched again and stiffness dissipates. However, 2-3 weeks of immobilisation will produce a much more severe form of joint contracture.

Immobility can cause contractures that are severe enough to restrict the range of movement in major joints; this is one of the most frequent complications associated with prolonged bedrest. Among 155 patients who stayed in an intensive care unit (ICU) for two weeks or more, over a third developed a movement-limiting joint contracture; the joints most often

affected were the elbow, ankle, knee, hip and shoulder (Clavet et al, 2008). A follow-up study in the same population showed that those who had developed joint contractures had difficulties with mobility three years later (Clavet et al, 2015). The authors concluded that joint contractures could cause irreversible disability and that identifying and treating them in the ICU could prevent long-term functional limitations.

Furthermore, during bedrest, opposing folds of the synovial membrane (connective tissue that lines the inner surface of the capsules of synovial joints) may come into contact with each other and form abnormal adhesions that further limit joint movement (Trudel et al, 2003).

Alteration in collagen structure

The main component of tendons and ligaments is the fibroblast-derived protein collagen. In joints that frequently move, collagen fibres are in a loosely coiled arrangement that allows stretching and normal activity. In a patient who is immobile, the collagen structure changes into a mass of shortened, straighter and more densely packed fibres within one day. Within two or three weeks, this change in collagen structure can compound a joint contracture. After two or three months of immobility, contracture and stiffness may

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have become so severe that surgical correction will be needed to restore the full mobility of the joint.

Foot drop contracture

A common problem associated with prolonged bedrest and immobility is foot drop contracture or deformity (Fig 2), which results in the inability to place the heel in its correct position on the ground when standing or sitting. This is usually caused by entrapment of the common peroneal nerve at the neck of the fibula at the top of the calf.

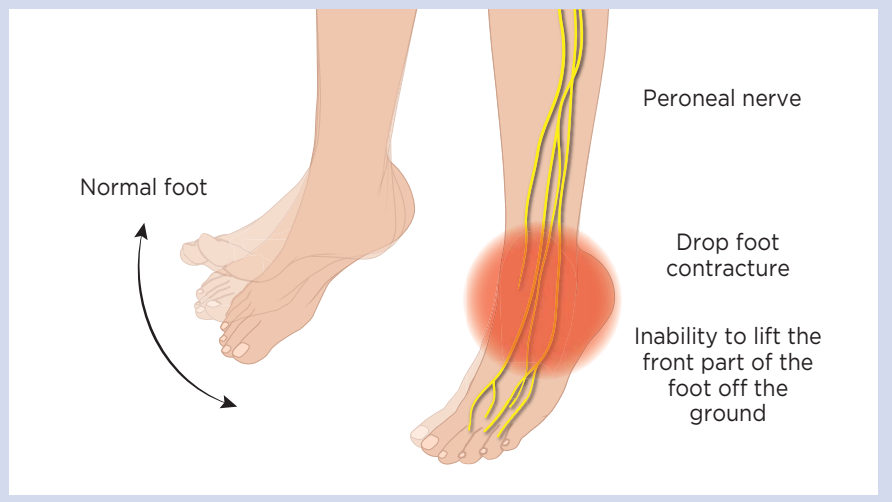
Foot drop contracture is compounded by a lack of passive exercise stretching the ankle joint or by inadequate joint support, both of which reduce tension on the Achilles tendon and lead to its shortening (Lippincott Williams and Wilkins, 2006). Similarly, a lack of stretching of the gastrocnemius muscle (one of the two major muscles of the calf) when the body is supine can lead to a tightening of the calf, thereby contributing to foot drop contracture (Amis, 2014).

Once the patient regains mobility, a shortened Achilles tendon can result in the toes pointing further forward than normal and make it difficult to place the foot in its usual position. This makes walking difficult and places undue strain on the Achilles tendon, causing pain and increasing the risk of tendon rupture.

Avoiding contractures

The risk of contracture can be reduced through appropriate positioning and body alignment in bed. Moving each joint through its full range of motion at least once every eight hours, whether actively or passively, also appears to help prevent contractures. Nursing staff can help by checking and correcting a patient's position in bed as well as their posture while sitting. A physiotherapist can undertake passive joint mobilisation exercises, as can appropriately trained nurses or healthcare assistants.

Fig 2. Foot drop contracture



Conclusion

As discussed here and in previous articles in this series, exercise and mobilisation, when feasible, will help counteract the negative physical and psychological effects of prolonged bedrest. In particular, they will help avoid muscle disuse weakness and joint contractures. **NT**

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