Complications of Immobilization and Bed Rest

Part 2: Other complications

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SUMMARY

Prolonged immobilization affects almost every organ system. Respiratory complications include decreased ventilation, atelectasis, and pneumonia. Decreased basal metabolic rate, increased diuresis, natriuresis, and nitrogen and calcium depletion affect metabolism. Genitourinary problems include renal stones and more frequent urinary tract infections. Glucose intolerance, anorexia, constipation, and pressure sores might develop. Central nervous system changes could affect balance and coordination and lead to increasing dependence on caregivers.

RÉSUMÉ

L'immobilisation prolongée affecte pratiquement tous les organes. Les complications respiratoires comprennent une baisse de la ventilation, l'atélectasie et la pneumonie. Quant au métabolisme, il est affecté par la réduction du métabolisme basal, la diurèse accrue, la natriurèse et la déplétion azotée et calcique. Les problèmes génito-urinaires incluent les lithiases rénales et des infections urinaires plus fréquentes. Il peut également se développer une intolérance au glucose, une anorexie, une constipation et des plaies de pression. Les changements au niveau du système nerveux central peuvent affecter l'équilibre et la coordination et augmenter la dépendance envers les personnes soignantes.

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BED REST AND IMMOBILIZATION are time-honoured treatments for managing trauma and acute and chronic illnesses. Although bed rest and immobilization often benefit the affected part of the body, they sometimes harm the rest of the body. Complications can compound the primary disease or trauma and could actually become greater problems than the primary disorder. In Part 1 of this article we discussed musculoskeletal and cardiovascular complications of prolonged immobilization. In Part 2 we consider complications of other organ systems (*Table 1*).

Respiratory complications

Decreased ventilation. Immobilized patients show reduced tidal volume and minute ventilatory volume. Inactive, supine patients find it difficult to contract ventilatory muscles sufficiently to accomplish a full inspiration. A decrease in muscle strength during immobilization includes the respiratory muscles. A restrictive impairment, an overall decrease in muscle strength, deconditioning of respiratory muscles, and failure to fully expand the chest wall results in a 25% to 50% decrease in respiratory capacity.

Respiratory rate increases to compensate for decreased capacity. In dependent areas of the lung, the ventilation-to-perfusion ratio might alter: poor ventilation and overperfusion cause arteriovenous shunting and reduced arterial oxygenation.[1-4]

Atelectasis and pneumonia. Immobilization (often made worse by motor weakness) can result in a markedly impaired ability to clear secretions. Secretions then accumulate in the lower parts of the bronchial tree,[5] blocking airways and eventually causing atelectasis and hypostatic pneumonia. As well, atelectasis and pooled secretions form an ideal environment for the development of bacterial pneumonia. Treatment to prevent respiratory complications includes chest physiotherapy, such as deep breathing and coughing, vibration, postural drainage, and incentive spirometry.

Endocrine and renal complications

Basal metabolic rate is decreased during the entire period of immobilization.[6,7] A

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variety of hormonal and biochemical changes have been documented.

Increased diuresis, natriuresis, and extracellular fluid shifts. Diuresis, the consequence of suppression of the antidiuretic hormone, leads to natriuresis to maintain plasma osmolality at a normal level. A subsequent temporary rise in intravascular volume causes the eventual release of fluid-retaining hormones, such as antidiuretic hormone, aldosterone, and cortisol.

Negative nitrogen balance. Inactivity results in loss of nitrogen from the whole body. Average nitrogen loss through urine could reach 2 g/d.[1] This nitrogen loss is due to an increase in protein catabolism and a concurrent decrease in protein synthesis. Nitrogen loss peaks during the second week of immobilization.[8,9] A negative nitrogen balance might be accentuated by starvation, trauma, infection, or inflammation and reach up to 12 g/d.[10-12]

Glucose intolerance. Glucose intolerance is a frequent but often overlooked complication of bed rest. Glucose tolerance tests of immobilized subjects demonstrate hyperglycemic and hyperinsulinemic responses.[1] The glucose intolerance appears to be due to increased tissue resistance to endogenous insulin[1]; insulin levels can rise to twice normal. The number of insulin binding sites on muscles appears to decrease. Glucose intolerance sometimes mimics brittle diabetes in patients who undergo prolonged bed rest.[1]

Hypercalcemia and calcium loss. Immobilized patients lose calcium. Maximum calcium loss from bones occurs during the fourth and fifth weeks. Hypercalcemia is commonly seen in patients with high bone turnover, such as children,[13] adolescents,[13] and those with Paget's disease of bone.[14] Up to 50% of healthy children immobilized with lower extremity fractures will experience hypercalcemia.[13]

In some cases, such as young quadriplegics, hypercalcemia is symptomatic. It appears as anorexia, abdominal pain, nausea, malaise, headache, polydipsia, polyuria, lethargy, and even coma 4 to 8 weeks after bed rest begins.[13,15-17] Symptomatic hypercalcemia is treated with aggressive intravenous saline rehydration. Other medications that could be useful include calcitonin,[18] etidronate,[19] clodronate,[20] and pamidronate. Muscle tension on bone is important to prevent calcium loss. Prolonged immobilization leads to osteoporosis.

Renal stones. The triad of hypercalciuria, urinary stasis, and a urinary tract infection often causes stones to form in the kidneys or bladder. One series showed that urolithiasis developed in more than 50% of children with hypercalcemia due to immobilization after spinal cord injury.[21] Catheters further increase the risk of bladder stones by forming a nidus for stone formation. Renal stones can harbour bacteria, making it more difficult to treat associated urinary tract infections with antibiotics.

Gastrointestinal complications

Anorexia. Decreased caloric demands, endocrine changes, anxiety, and depression all contribute to a loss of appetite. However, weight gain is common due to inactivity.

Constipation. Constipation, common in immobilized patients, results from decreased peristalsis and constrictive sphincters. Low-fibre diets and decreased fluid intake also contribute to constipation. Constipation is best treated with a high-fibre diet, stool softeners, irritant laxatives, increasing fluids, and a regular bowel routine. We have found oral lactulose effective for our patients, decreasing the need for regular enemas. (We aim to achieve a bowel movement on average once every 2 days.)

Pressure sores

Pressure sores or decubitus ulcers are localized areas of cellular necrosis. They are usually found over bony prominences subjected to external pressure greater than capillary pressure for prolonged periods. Pressure sores occur most often in two populations who are frequently immobilized: patients with spinal cord injuries and elderly patients. Pressure sores develop in 25% to 80% of all patients with spinal cord injuries, and resulting complications account for up to 8% of deaths in this group. Up to 4.5% of all patients develop pressure sores in hospital. Most

Table 1. **Potential complications of immobilization**

RESPIRATORY
- Decreased overall ventilation
- Regional changes in ventilation and perfusion
- Difficulty coughing
- Atelectasis

ENDOCRINE AND RENAL
- Decreased basal metabolism
- Increased diuresis, natriuresis, and extracellular fluid shift
- Negative nitrogen balance
- Glucose intolerance
- Hypercalcemia and calcium loss
- Renal stones

GASTROINTESTINAL
- Anorexia
- Constipation

SKIN
- Pressure sores

CENTRAL NERVOUS SYSTEM
- Altered sensation
- Decreased motor activity
- Autonomic instability
- Emotional and behavioural disturbances
- Intellectual deficit
- Poor coordination

Table 2. **Possible complications of pressure sores**

INFECTION
- Osteomyelitis
- Sinus formation
- Septic joint
- Septicemia
- Amyloidosis

DRAINAGE
- Protein and water loss

DAMAGE TO NERVES, TENDONS, AND MUSCLES

INCREASE OF EXISTING SPASTICITY

LOSS OF FUNCTIONAL ABILITIES
- Transfers
- Wheelchair

FRAGILE SCAR

commonly they occur in patients with loss of normal sensation, especially loss of deep pressure sensitivity and proprioception. Patients at particular risk for pressure sores are those who are comatose, obese, or have burns or ill-fitting casts.

The prevalence of pressure sores tends to increase significantly with age. Patients older than age 70 not only have more than 70% of all pressure sores but get them within 2 weeks of admission to hospital. Once decubitus ulcers occur, nursing costs can increase by as much as 50%. Total cost of treatment per ulcer has been conservatively estimated at between $15 000 and $20 000.

Prolonged pressure greater than capillary pressure of 32 mm Hg can result in ischemia of underlying tissues. The longer the duration and the greater the magnitude of pressure, the greater the chance of necrosis. Microscopic changes have been observed with pressures of 70 mm Hg after only 2 hours. About 95% of decubitus ulcers occur at five sites: the sacrum, ischial tuberosities, greater trochanters, heels, and ankles. Supine patients get sores on the sacrum and heels; sitting patients risk sores on the ischial tuberosity; and patients who lie on their sides put pressure on their hips and ankles. Fat and other subcutaneous tissues have a poorer blood supply than the skin and are affected first. Consequently, many decubitus ulcers have an inverted cone shape.

Complications often develop with pressure sores (*Table 2, Figure 1*). Complications of grade 3 and 4 pressure sores can be life threatening. However, the most common problem is infection. It is perhaps best to assume that all decubitus ulcers are infected. The organisms are commonly polymycrobial and include both aerobic and anaerobic bacteria. Deeper tissue and bone infection can result in periostitis, sinus formation, osteomyelitis, and septic arthritis. Septicemia occurs, especially in the early stages. Deep infections sometimes lead to heterotopic ossification providing the foci for new pressure sores and increasing susceptibility to tetanus. Chronically infected decubitus ulcers could lead to secondary amyloidosis or chronic anemia. A draining ulcer can discharge as much as 50 g of body protein daily (in addition to the protein lost due to the bed rest). Anemia secondary to bleeding and water loss also occur.

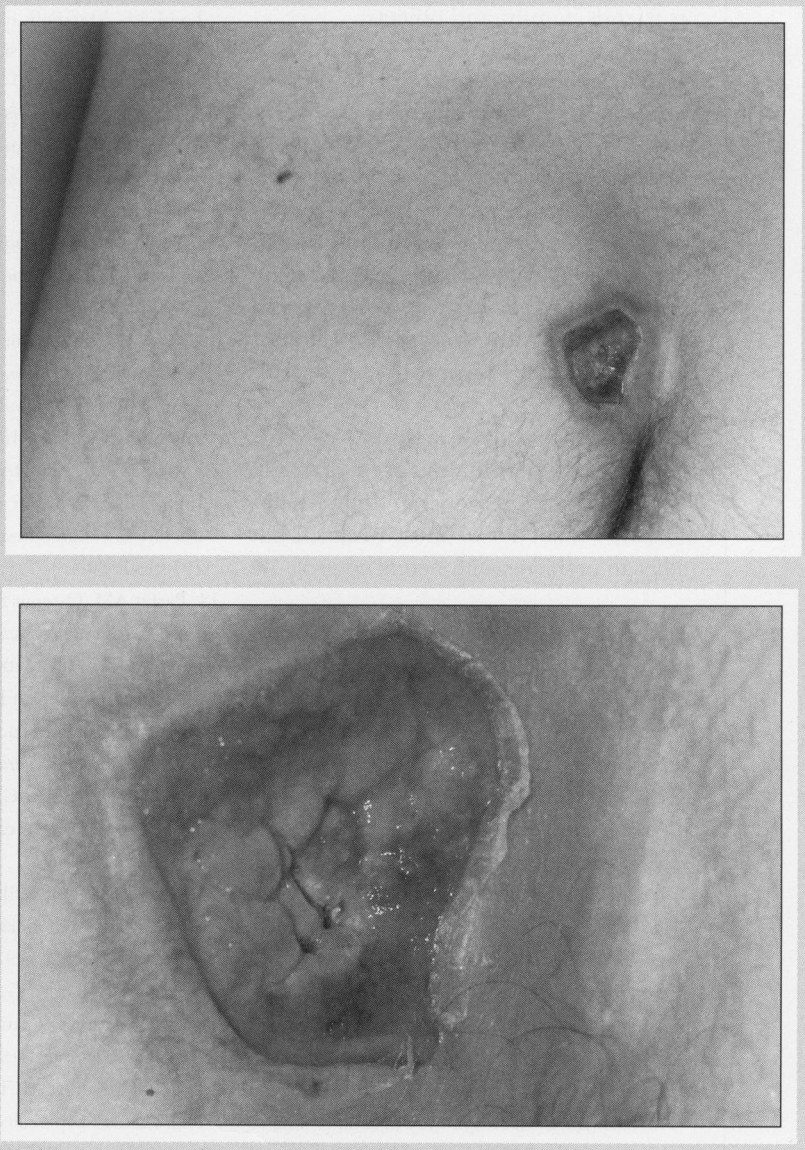
To prevent pressure sores, relieve the pressure. Patients should be repositioned every 2 hours and observed for reddened areas. Lying flat spreads the weight over the entire body; 2 to 4 hours daily should be spent in the prone position. Try to avoid the semirecumbent position because it concentrates weight on the sacrum and heels: patients tend to slide down, creating shear forces over the sacrum. The 90° lateral position (lying on the side) is somewhat controversial because bridging allows suspension of bony prominences. Doughnut cushions should not be used because they distribute pressure in undesirable ways that can worsen ischemia.

Friction can be reduced with loose bedclothes. Patients should be lifted and not dragged across the bed. Spasticity, which might interfere with positioning and increase friction, can be minimized with medications and other measures. Shear forces can be avoided by raising the head of the bed or adding a footboard. Sheepskin has shear-resistant properties. Good skin care and prevention of soiling are very important not only for preventing, but also for helping to heal, pressure sores. Patients sometimes require an indwelling catheter.

Factors that delay healing of pressure sores include tissue hypoxia, a necrotic ulcer surface, local infection, inappropriate local wound care, and general debilitation (especially from malnutrition). Standard treatment for established ulcers is to restore blood supply to ulcer tissue by relieving localized pressure. Removing necrotic tissue by surgical or enzymatic débridement is essential. Local infections can be treated with local disinfectant agents. Wound dressings that allow airflow foster better granulation. Anemia and protein depletion should be corrected.

Surgery is sometimes indicated for deep pressure sores (ie, grade 3 and 4), sinus tracts, ulcers 4 to 5 cm in diameter, and bone, artery, or nerve exposures. Education and instituting preventive measures are essential to prevent recurrence. Infection must be controlled, and the wound should be granulating with the edges epithelializing. Skin grafts and direct closure are rarely indicated (a skin graft has no underlying padding, and direct closure places a scar right over the

Figure 1. **Grade 3 pressure sore left of the sacrum in a young man hospitalized for severe complicated sepsis:** *The ulcer extends well into the subcutaneous fat with granulation and epithelization at its margins. The ulcer healed with mobilization and pressure relief.*



pressure area). Current surgical practice involves excising the ulcer, smoothing the underlying bone, and then covering the area with a flap (skin or myocutaneous) with the suture line off the pressure area.

Central nervous system complications

Central nervous system complications include sensory deprivation, mental deterioration, and behaviour disturbances.

Sensory deprivation. Immobilized patients, especially those who are cognitively impaired (stroke, head injury, dementia) and the elderly, are prone to complications of sensory deprivation. These complications include intellectual regression, depression, a short attention span, and poor motivation. Social isolation in association with regular physical activity will not itself result in intellectual deterioration[7,22]; however, social isolation in combination with physical inactivity results in intellectual deterioration.[1,7] Prevention of social isolation excludes placing the patient in a private room. Patients should face the entrance of their rooms. Professionals are reminded to avoid speaking of patients in the

third person in their presence. Radio and television can provide orientation. Staff should attempt to keep patients oriented and encourage exposure to familiar friends, family, and objects.

Dependency. Dependency is an important problem especially for elderly patients. Patients tend to conform to expected roles: when caregivers overprotect, patients become increasingly passive, dependent, and immobile. Patients should not be helped to do something they can do for themselves. Educating patients and families about the harmful effects of dependency and immobility is useful.

Balance and coordination

Balance and coordination are negatively affected by immobilization.[23] Impairment of balance is not due to disuse weakness but rather to problems with neural control. ■

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