

MANAGEMENT OF THE RECUMBENT DOG
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Introduction

Prior to World War II, patients with abdominal surgeries were kept on bed rest one week or more. These patients often developed complications such as atelectasis, deep vein thrombosis, hypostatic pneumonia, pulmonary thromboembolism and a loss muscular conditioning. Through out the war attention was given to teaching effective breathing and coughing and leg exercises to maintain adequate venous return and early ambulation.¹⁷ Physical therapy is often overlooked early in the treatment of critically ill veterinary patients because the complications of prolonged immobility have not yet caused functional limitations.¹⁴

Constant recumbency is unnatural and has far-reaching consequences.¹³ The consequences of prolonged periods of recumbency are associated with neuromuscular and skeletal affects, cardiorespiratory effects and skin trauma, all of which can be directly affected by physical therapeutic management of the patient.^{5, 6, 13, 14, 15, 19} Other systems and functions of the body can be adversely affected by immobilization, such as the gastrointestinal, endocrine, immune, and vascular systems as well as the psychological.¹³ While physical therapy management may not address these systems directly, it may be of indirect benefit.

Recumbency and the Musculoskeletal System

It is well known that muscle atrophy is caused by disuse, such as bed rest or unilateral lower limb immobilization.¹ Twenty days of bed rest has been shown too induce up to 10% atrophy in the lower limb muscles of health men and women.¹ Total hind limb immobilization has been shown to cause as much as 40% loss of muscle mass to the soleus muscle in rats within seven days.^{3, 6, 7} The antigravity muscles are the ones most affected.⁶

Physiological changes occur in the muscles and bones due to immobility. Immobilized muscles show a significant decrease in weight, serial sarcomere number, fibre area and a decline in aerobic capacity of the muscle.^{6, 7} Immobility results in a loss of muscle protein due to an early decrease in protein synthesis rate which leads to an increase in protein degradation and hence a loss of muscle volume.⁴ When muscles are immobilized in a shortened position, this causes a loss of serial sarcomere number along the length of the muscle fibres with a consequent shortening of muscle length.⁷ As well, deconditioning results in the atrophying of slow twitch (oxidative) muscle fibres and a transformation of subtype IIa skeletal muscle fibres to convert into type IIb, thus further debilitating the oxidative (aerobic) capacity of the muscles.^{3, 6, 18} Additionally, in situations of non-use, bone exhibits demineralization (specifically, a loss of calcium) and protein wastage due to loss of gravitational forces and movement.^{6, 13}

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Physical therapy management of the musculoskeletal system in the recumbent dog relates to therapeutic activity with the goals of promoting physical fitness and preventing disability and the complications of bed rest. Standard human intensive care unit practice tend to incorporate passive range of motion (PROM), active range of motion (AROM), active-assisted range of motion (a-AROM), active-resisted range of motion a-rROM) and exercise.¹⁵

PROM is the passive movement of a limb through its available range. Continuous passive motion machines utilized on critically ill human patients have been shown to prevent muscle fibre atrophy and protein loss in the earliest stages of immobility.¹² While continuous passive motion may be unrealistic in the recumbent canine patient due to practicality of its application, regular intervals of PROM lasting 20 – 30 minutes per session are advised.^{7, 15} Muscle stretching has been shown to be effective in preventing the loss of serial sarcomeres, joint ROM, connective tissue proliferation and has been demonstrated to reduce the amount of muscle fibre atrophy.^{7, 10, 18} It is, in fact, recognized as a very powerful stimulant of muscle growth and protein synthesis.¹⁰ Of the many existing stretching protocols trialed on rats, the most effective has been the use of daily stretching for 30 minutes to the affected muscle group(s).⁷

AROM would be the volitional movement of a limb through its range of motion. Exercise that encourages movement or active repositioning is important and any patient that can stand or walk should be encouraged to do so.¹⁵ Slings, harnesses or manual support that enable standing practice or positional changes every 4 – 6 hours can be utilized for the canine patient. Active-assisted ROM may be accomplished by encouraging the animal to move its own limbs perhaps with the stimulus of a pin prick or tickling of a limb.¹⁵ Active-resisted ROM is accomplished by resisting a volitional limb movement. Practitioners may attempt to resist a flexor withdrawal or protective extension reflex of the limbs or may utilize the opposition reflex to engage trunk muscles.

Electrical muscle stimulation (e-stim) may also have some value in preventing muscle atrophy. E-stim at 2.5 Hz has been shown to create muscles that are faster and stronger and possess a better capability to fatigue resistance than normal muscles.⁹ The animals (rats) used in this study were subjected to e-stim for either 2 hours or ten hours a day, with the group receiving 10 hours of e-stim realizing the greater benefits.⁹ It has yet to be studied to determine if animals that receive less than this amount of stimulation would also benefit from e-stim.

Contraindications to mobilization therapy include any cases where movement could be detrimental (i.e. unstable fractures).

Recumbency and Decubitus Ulcers

Decubitus ulcers, pressure ulcers or dermal ulcers are described as an area of localized damage to the skin and underlying tissue caused by pressure, shear, friction and/or a combination of these.^{11, 16} They are characterized by a local breakdown of soft tissue as a result of compression between a bony prominence and an external surface.¹¹ Normally, when pressure of short duration is relieved, tissues demonstrate reactive hyperaemia, reflecting increased blood flow to the area, however, sustained high pressure leads to decreased capillary blood flow, occlusion of blood vessels and lymphatic vessels and tissue ischaemia.^{2, 11} The impaired blood supply and tissue malnutrition, which allows for toxic metabolites to accumulate locally, increasing the rate of cell death, leading to ulceration and necrosis of the skin and underlying tissue.² The process starts first with deep tissue destruction (muscles and subcutaneous tissues) before dermal and epidermal changes.^{2, 11} Epidermal necrosis occurs late in the course because epidermal cells are better able to withstand prolonged absence of oxygen than deeper cells both in vivo and in vitro, thus when the skin eventually ruptures, a deep draining wound is revealed.² The risk factors precipitating decubitus ulcers include excessively moist environments (due to urinary or fecal incontinence, excessive wound drainage or perspiration in humans), immobility or lack of activity, sensory deficits, malnutrition, dehydration, hypotension, prolonged anesthesia and advanced age.^{2, 11, 13} The classifications of dermal ulcers can be viewed in table 1.

Table 1: Classification of Decubitus Ulcers (Bansal et al. 2005)²

Stage 1	Skin is intact with signs of impending ulceration: blanching and/or non-blanching erythema, warmth and induration. These clinical signs can be resolved in 5 – 10 days with care.
Stage 2	The area clinically shows as a shallow ulcer of the epidermis and dermis, with pigmentation changes. The ulcer may first show as an abrasion, blister or superficial ulcer. This stage may also be reversible with care.
Stage 3	There is a full-thickness loss of skin with extension through the subcutaneous tissue but not the underlying fascia. There is a necrotic, foul-smelling crater with altered light and dark pigmentation.
Stage 4	There is full-thickness skin and subcutaneous tissue loss, with ulcer penetration into the deep fascia, resulting in involvement of muscle, bone, tendon or joint capsule. Osteomyelitis or even a fracture may be present.

The best management of pressure ulcers is in prevention. This requires frequent repositioning (every 2 hours), regular inspection of the skin overlying bony prominences, and a reduction in excessive moisture (i.e. use of a padding that wicks away moisture).^{2, 11, 13} Pressure relief devices can be utilized such as pillows, foam, mattresses and gel protectors, paying special attention to avoid direct ‘kissing’ contact between bony prominences (i.e. between knees/stifles and ankles/hocks).^{2, 11} Nutritional management to avoid malnutrition is essential and the creation of a strict fecal and urine voiding schedule may be useful as well.^{2, 13} Active or passive movement of the limbs or exercise such as

walking may assist to increase circulation and lymphatic drainage of the limbs and relieve prolonged period of pressure and hence aid in preventing dermal ulcers.^{13, 15} Massage has also been suggested as a preventative measure to reduce fluid in interstitial or joint spaces in order to improve arterial, venous and lymphatic flow in paralyzed or weak muscle, hence improving the condition of the skin and underlying tissues.¹⁵

Recumbency and the Cardiorespiratory System

Recumbency can also have deleterious effects on the cardiorespiratory system. Some of these effects include a reduction in functional residual capacity of the lungs, changes in lung volume, and atelectasis.¹³ As well, a pooling of respiratory secretions in the dependent lung, lung consolidation, and a depressed cough reflex are also complications.^{8,14,17} These problems can lead to infection, and pneumonia.^{8,14,17}

The goals of chest physiotherapy are to hasten the elimination of secretions from the airways, re-expand atelectatic lung segments and reduce the incidence of pneumonia.¹⁴ Techniques utilized to accomplish these goals include postural drainage, percussion, vibration and passive forced expiration and cough stimulation.^{8,14,17}

Postural drainage is the use of positioning that allows for a gravitational drainage of tracheobronchial secretions. Placing a healthy dog head-down by 20 degrees from horizontal can increase the velocity of tracheal mucus drainage by 40%.¹⁴ Positioning alterations can be varied and adapted to enhance drainage from each lobe. See table 2. Contraindications to head-down postural drainage positions include tachycardia, arrhythmia, cerebral vascular infarcts or aneurisms, high intracranial pressure, nausea / vomiting, danger of regurgitation, eye surgery or nose bleeds, hypertension, coughing blood within the last two days, pneumothorax, pulmonary edema, cerebral spinal fluid lead, acute abdominal problems or facial swelling.⁸

Table 2: Description of Postural Drainage Positions for the Canine Lungs (from Manning et al 1997)¹⁴

Lateral segment of the left caudal lung lobe.	The patient is placed in left lateral recumbency with the hind end elevated 40°.
Left and right caudal dorsal lung fields.	The patient is in sternal recumbency with hind end elevated 40°.
Left and right caudal ventral lung fields.	The patient is in dorsal recumbency with hind end elevated 40°.
Left and right cranial ventral lung fields.	The patient is in dorsal recumbency with front end elevated 40°.
Left and right cranial dorsal lung fields.	The patient is in sternal recumbency, with the front end elevated 40°.
Right middle lung lobe.	The patient is in dorsal recumbency. A pillow has been placed under the right side of the thorax so that the right side is higher than the left side. The hind end is elevated 40°, and the front end is rotated one quarter turn to the right.
Lateral segment of the right caudal lung lobe.	The patient is in left lateral recumbency with the hind end elevated 40°.

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Percussion is the application of a pressure wave (via a cupped hand clap) to the chest wall which transmits to the lungs and mechanically dislodges secretions from bronchial walls. Percussions should be confined to the affected lung segments and consist of 3 – 4 sessions of 3 minutes each.^{14, 17} Contraindications to percussion include rib fractures, pneumothorax, coagulopathy, low platelet count, subcutaneous emphysema of neck or thorax, unstable cardiovascular condition, recent skin grafts or flaps in the area, areas of open wounds or burns, thoracotomy within the previous 24 hours, severe pain, malignancy in lungs unless lung function is of vital concern, bronchial spasm, osteoporosis, pleural effusion and labile blood pressure.^{8,17}

Vibration and passive forced expiration utilizes shaking of the chest wall during expiration in order to move the secretions towards larger airways.^{8, 17} The practitioner utilizes their hands to provide intermittent coarse compression or increasing pressure against the chest wall during exhalation.^{8, 17} As well a finer vibratory motion can be attempted using the practitioner's own hands or fingertips depending upon the size of the dog.¹⁴ Contraindications would be the same as for those pertaining to percussion.

The cough reflex is often suppressed in traumatic, post-surgical or chronically recumbent patients. Stimulating a cough aims to eliminate secretions from the level of the trachea down to the 4th generation segmental bronchi.¹⁴ Sometimes placing the animal in sternal recumbency is enough to stimulate a cough. Other manual techniques can include a tracheal tickle by applying a gentle pressure to the trachea at the level of the 3rd tracheal ring.¹⁴ Alternately, a pressure in the epigastric region (aiming towards the diaphragm) with the practitioners other hand on the lateral chest wall to compress downward.¹⁴ Contraindications to stimulated coughing would be the same as those for percussion.

Other Systems Affected by Recumbency

Other bodily systems can be adversely affected by prolonged recumbency. These systems include the cardiovascular system, the endocrine system, the gastrointestinal system, the immune system and fluid and electrolyte balance.¹³ The cardiovascular system is impacted by an increase in procoagulation factor synthesis, an increase in fibrinolysis, shortened thromboplastin time and a decrease in the formation of red blood cells.¹³ The endocrine system experiences a disruption of circadian rhythms which are dependent upon a diurnal cycle.¹³ With convalescence, there are changes in insulin cycles, pancreas activity decline, glucose intolerance, an increase in thyroid hormone activity and a fall in the levels of androgen.¹³ The gastrointestinal system suffers from anorexia, a suppression of gastric secretions, and a reduction of peristalsis when in the presence of recumbency.¹³ The immune system becomes compromised, with an impairment of T-cell activity, slowing of neutrophilic phagocytosis activity, and the pooling of secretions in recumbent cavities that may allow colonization of bacteria.¹³ Finally, there is an impact on fluid and electrolyte regulation. Eighty percent of the blood volume migrates to the thorax, there is a reduces excretion cycle of sodium, calcium leaching from bone, impaired renal excretion of calcium and blood volume to the

cranium increases.¹³ All of these systems may be bolstered indirectly by physical therapeutic management, as described above, of the recumbent animal.

Conclusion

The impairments and complications associated with recumbency can be just as serious as the pathology that led to the recumbency. It is imperative that physical management of the patient be considered on several levels to avoid these complications, reduce the time of hospitalization and speed recovery time to resumption of normal activities.

References:

1. Akima H, Kubo K, Imai M, Kanehisa H, Suzuki Y, Gunji A, Fukunaga T. (2001) 'Inactivity and muscle: effect of resistance training during best rest on muscle size in the lower limb.' *Acta Physiologica Scandinavica*. 172: pp 269-278.
2. Bansal C, Scott R, Stewart D, Cockerell CJ. (2005) 'Decubitus ulcers: A review of the literature.' *Intern J Derm*. 44: pp 805 – 810.
3. Barton E, Morris C. (2005) 'Mechanism and strategies to counter muscle atrophy.' *J Gerontol*. 58A (10): pp 923 – 926.
4. Booth FW, Criswell DS. (1997) 'Molecular events underlying skeletal muscle atrophy and the development of effective countermeasures.' *Int J Sports Med*. 18 (suppl 4): pp S265 – 269.
5. Bose D, Tejwani NC. (2006) 'Evolving trends in the care of polytrauma patients.' *Injury*. 37: pp 20 – 28.
6. Clini E, Ambrosino N. (2005) 'Early physiotherapy in the respiratory intensive care unit.' *Resp Med*. 99: pp 1096 – 1104.
7. Coutinho EL, Gomes ARS, Franca CN, Oishi J, Salvini TF. (2004) 'Effect of passive stretching on the immobilized soleus muscle fiber morphology.' *Braz J Med Biol Res*. 37 (12): pp 1853 – 1861.
8. Downie, Patricia A. (1987) *Cash's Textbook of Chest, Heart and Vascular Disorders for Physiotherapists. Fourth Edition*. (J.B. Lippincott Company: Philadelphia, PA).
9. Dupont Salter AC, Richmond FJR, Loeb G. (2003) 'Prevention of muscle disuse atrophy by low-frequency electrical stimulation in rats.' *IEEE Trans Neur Sys Rehabil Eng*. 11 (3): pp 218 – 226.
10. Gomes ARS, Coutinho EL, Franca CN, Polonio J, Salvini TF. (2004) 'Effect of one stretch a week applied to the immobilized soleus muscle on rat muscle fiber morphology.' *Braz J Med Biol Res*. 37 (10): pp 1473 – 1480.
11. Grey JE, Harding KG, Enoch S. (2006) 'Pressure ulcers.' *BMJ*. 332: pp 472 – 475.
12. Griffiths RD, Palmer TE, Helliwell T, MacLennan P, MacMillan RR. (1995) 'Effect of passive stretching on the wasting of muscle in the critically ill.' *Nutrition*. 11 (5): pp 428 – 432.
13. Looney AL. (2002) 'Rehabilitation considerations for critical patients.' In *Proceedings of the 2nd International Symposium on Rehabilitation and Physical Therapy in Veterinary Medicine*. pp 167. (Knoxville, Tennessee, USA).

14. Manning AM, Rush J, Rudnick Ellis D. (1997) 'Physical therapy for critically ill veterinary patients. Part I. Chest physical therapy.' *The Compendium*. 19 (6): pp 675 – 688.
15. Manning AM, Rush J, Rudnick Ellis D. (1997) 'Physical therapy for critically ill veterinary patients. Part II. The musculoskeletal system.' *The Compendium*. 19 (7): pp 803 – 807.
16. Nixon J, Cranny G, Bond S. (2005) 'Pathology, diagnosis, and classification of pressure ulcers: comparing clinical and imaging techniques.' *Wound Repair and Regeneration*. 13: pp 365 – 372.
17. Parker, Moyna J. (1998) *Physiotherapy in Thoracic conditions*. 7th Ed. (Faculty of Rehab Medicine: University of Alberta.) Copyright M. Parker 1988.
18. Sasa T, Sairyo K, Yoshida N, Fukunaga M, Koga K, Ishikawa M, Yasui N. (2004) 'Continuous muscle stretch prevents disuse muscle atrophy and deterioration of its oxidative capacity in rat tail-suspension models.' *Am J Phys Med Rehabil*. 83 (11): pp 851- 856
19. Winkelman C, Higgins PA, Chen YJK. (2005) 'Activity in the chronically critically ill.' *Res Dim*. 24 (6): pp 281 – 290.