Written report: prolonged immobilization

Written Report: Prolonged Immobilization I. A. Definition Defined as prolonged inactivity, bed rest and immobilization were widely used in the early days of rehabilitative science in the management of trauma and acute and chronic illness, before the physiologic effects were well understood. It was generally assumed that rest fostered healing of the affected part of the body. However, while it has beneficial effects to soft tissue healing and to bone healing after fractures among a myriad of other conditions, immobility and inactivity could also be harmful to the unaffected parts of the body. Problems arising from immobilization can complicate a primary disease or trauma and might actually become greater problems than the primary disorder. I. B. Definition of terms * Deconditioning — the most deleterious effects of inactivity can which is defined as reduced functional capacity of musculoskeletal and other body systems. * Disuse atrophy — an alteration of metabolism and muscle cell homeostasis in response to muscle inactivity * PCSA — physiologic cross-sectional area. PCSA increases with pennation angle, and with muscle length. In a pennate muscle, PCSA is always larger than ACSA. In a non-pennate muscle, it coincides with ACSA. It is (Muscle Volume/Fiber Length). * Titin — a myofibrillar protein, appears to have a major role in providing resistance to passive elongation, increased in immobility * Myostatin — growth factor-beta protein that inhibits muscle synthesis and is increased during bed rest. * Sarcopenia — Muscle mass loss associated with aging * VO2max — maximum oxygen consumption(L/min) * Fitness — general term indicating a level of cardiovascular functioning that results in heightened energy reserves for optimum performance and well-being * Endurance — ability to resist fatigue * Local endurance — aka “ muscle endurance", is the ability of a muscle to contract repeatedly against

a load and resist a fatigue over an extended period of time * Total body endurance — aka “ cardiopulmonary endurance", associated with repetitive, dynamic motor activities which involves use of large muscles of the body * Interleukin-1 — inhibit production of proteoglycans necessary in the protection of cartilage * Wolff’s law — constant gravity loading facilitates bone remodeling and formation I. C. Clinical Features A. Musculoskeletal system; a. Disuse atrophy i. Generalized or localized to the immobilized limbs and more prominent in anti-gravity muscle ii. Whole body protein production significantly reduced and is considered the main contributor to muscle atrophy iii. Number of sarcomeres is reduced as a result of diminished chronic stretch and adaptation of new muscle length and vice versa c elongation iv. Number of sarcomeres in parallel is reduced contributing to the reduction of muscle fiber PCSA v. Affects type I and type IIa muscle fibers more prominently than type IIb vi. MRI reveals greater extent of atrophy at the lower limbs than the upper limbs vii. Synthesis of collagen fiber is reduced, resulting to increase in muscle collagen content and changes in mechanical elastic properties viii. Titin is increased, which increases resistance to passive stretch ix. Serum creatine kinase (CK) isomer and fibrolast growth factor release are both reduced during bed rest and is proportional to the reduction in muscle fiber size x. Myostatin is increased, which inhibits protein synthesis xi. Sarcopenia occurs to elderly patients, increasing risk to functional decline, falls, and increased dependency xii. Increased breakdown of nitrogen from the normal value of 2g/day to 12g/day for nutritionally depleted patients xiii. Excretion of creatine and creatinine, the mechanism is not well understood, however b. Loss of strength xiv. The loss of strength is rapid after the first day of immobilization and reaches its

maximum 10 to 14 days later xv. Loss os strength is more prominent in the lower limbs than in the upper limbs xvi. Proportionally greater than reduction in size of muscles xvii. Major contributors of strength loss are the reduced number of mitochondria, muscle fiber nuclei, and reduction of sarcomeres in parallel xviii. Maximal instantaneous power is reduced and a decline in muscle twitch and titanic tension xix. Changes in contractile forces resulting from diminished levels of myofibrillar proteins and reduction of sarcoplasmic reticulum calcium ion uptake, but not the rate of its release c. Loss of endurance xx. Reduction of adenosine triphosphate and glycogen storage sites and rapid depletion of them after resumption of activity xxi. Reduction of muscle protein synthesis and oxidative enzyme function, and premature anaerobic energy production with rapid accumulation of lactic acid lead to fatigability and reduced endurance xxii. Decreased ability to utilize fatty acids xxiii. Sequence of events that transpires during deconditioning; 1. Prolonged reduction of muscle repetitive contractions below 50% of maximum alters muscle protein synthesis and decreases glycogen and ATP storage 2. Reduction of oxidative enzymes, mitochondrial function, and microvascular circulation, impacting muscle metabolic activity 3. Oxygen supply is attenuated, extraction of oxygen from blood is diminished, negatively affecting VO2max 4. Loss of muscle mass leads to reduction of muscle strength and endurance, reducing muscle blood flow, red blood cell delivery, oxidative enzyme activity, and oxygen utilization in the muscle 5. Precipitated further loss of musculoskeletal and cardiovascular functional reserve to low or dangerous levels 6. Specific muscle gene activation and expression are altered as well 7. Decreased formation of oxidative muscle fiber types I and IIa, the main factors in reduction of endurance and fitness d.

Negative impact on ADLs and specific muscle groups xxiv. Vastus intermedius, which is composed predominantly of type I muscle fibers (which are active in slow ambulation and standing), shows accelerated atrophy compared to the other muscles comprising the quadriceps xxv. Such accelerated rate of atrophy and weakness were also noted in the; 8. Hip and back extensors 9. Hip abductors 10. Ankle plantar flexors and dorsiflexors xxvi. Reduced personal independence e. Muscle pain and stiffness xxvii. Occur especially in the presence of limb swelling xxviii. Speculated that localized, prolonged, low-intensity isometric muscle contractions cause the pain B. Joint Contracture f. Factors that affect rate of contracture development xxix. Limb position xxx. Duration of immobilization xxxi. Pre-existing joint pathology g. Factors that can precipitate fibrosis xxxii. Edema xxxiii. Ischemia xxxiv. Bleeding h. Myogenic contracture xxxv. Shortening of resting muscle length that is due to intrinsic or extrinsic causes, limiting full ROM and causing abnormal positioning of the limbs or body xxxvi. Intrinsic changes are structural and may be associated with the inflammatory, degenerative, or traumatic processes 11. Muscle dystrophy a. Muscle fiber loss b. Segmental necrosis c. Increased numbers of lipocytes d. Fibrosis 12. Heterotopic ossification e. Common after trauma, joint surgery (esp. of the hip), SCI, or other CNS injuries f. Alteration in calcium metabolism and local metabolism or blood flow may be responsible for initiating this process g. Surgery is recommended xxxvii. Extrinsic muscle contracture is secondary, resultin from neurologic conditions or mechanical factors 13. Multiple injuries 14. Chronic illness 15. Sedentary individuals i. Arthrogenic contracture xxxviii. Degeneration of cartilage xxxix. Congenital incongruency of joint surfaces xl. Synovial inflammation or effusion xli. Accompanied by pain that

fosters limited joint mobility j. Soft tissue contracture xlii. Cutaneous, subcutaneous, and loose connective tissue around the joint may become contracted during immobility xliii. Collagen fibers proliferate and are laid down in random fashion xliv. Limit movement in only one plane or axis xlv. Burned skin is particularly susceptible Duration of Immobilization | Physiological Effects | 2 days | Slow muscle wasting begins, rapid progression | 10 days | 50% of eventual muscle weight loss | 14 days | 50% reduction of muscle protein synthesis | 20 days | 9. 4% reduction of muscle volume of the gastrocnemius10. 3% reduction of muscle volume of the soleus5. 1% reduction of muscle volume of knee extensors8. 0% reduction of muscle volume of knee flexors46% reduction of mean cross-sectional area of dark ATPase fibers69% reduction of mean cross-sectional are of light ATPase fibers2. 1% to 4. 4% reduction of muscle thickness | 42 days | 16% reduction of cardiac output30% reduction of oxygen delivery17% reduction of muscle cross-sectional area16% reduction of volume density of mitochondria28% reduction of total mitochondria volume11% reduction of oxidative enzyme activity | 45 days | 24% reduction of instantaneous muscle power | 1 week | 10% to 15% reduction of muscle strength | 2 -3 weeks | 25% to 40% reduction of muscle strength | 4 weeks | 50% reduction of muscle strength | Overall onset | 20% reduction of strength of knee flexors44% reduction of strength of knee extensors5% reduction of strength of upper limbs | II. Signs and Symptoms * Musculoskeletal system * Weakness, fatigue, atrophy, poor endurance * Mm. and joint contractures * Stiffness and pain * Hypercalcemia * Cardiovascular and pulmonary system * Degradation of the functional capacity * Dehydration * Orthostatic intolerance * Reduction of VO2max * Difficulty in elimination of bronchial secretions * Genitourinary and

gastrointestinal system * Urinary stasis * Loss of appetite * Constipation * Metabolic and endocrine system * Glucose intolerance * Electrolyte alterations * Increased parathyroid hormone production * Other hormone alterations * Immune system * Impaired wound healing * Reduction in cellular Immunity * Resistance to infection reduced * Anti-inflammatory suppression reduced * Cognitive and behavioral * Sensory deprivation * Confusion and disorientation * Anxiety and depression * Decrease in intellectual capacity * Impaired balance and coordination * Cellular/Genetic * Diminished gene expression * Mitochondrial dysfunction * Compromised immune system III. Etiology Prolonged immobilization is a secondary condition to the ff; * Chronically ill, aged, disabled * Paralysis (Stroke, NMD, SCI, BI/coma) * LBP * Post-op/complications * Poly-trauma, CAD, Obstetrical complications IV. Pathophysiology A. Paralysis B. Aging C. Secondary complications (Most common and case-related) a. DVT i. Virchow’s triad 1. Hypercoagulability 2. Venous stasis 3. Vessel wall injury ii. In stroke patients, more common in the involved extremities iii. Frequency has direct relationship with length of bed rest iv. Mechanism; 4. Stasis is present, thrombus formation at the valve cusp of the deep veins 5. Stasis may contribute to anoxia and damage of the endothelial cells in the valve pocket 6. May lead to formation of thrombin 7. Platelet aggregation, then thrombosis v. Most common in the veins of the calf vi. 20% of calf thrombi extend to popliteal and thigh veins, half of which would embolize to the lungs with fatal consequences b. Ulcerations vii. Pressure sores form within 1-2 weeks of immobilization viii. Occurs at bony prominences ix. Untreated ulcerations may result to sepsis c. Orthostatic intolerance x. Impairment of the cardiovascular system to adjust to the upright position 8. After several

days of immobilization, standing up from supine position would result in a shift of 500 ml of blood from the thorax and abdomen into the legs, causing blood pooling 9. Blood pooling occurs because of impaired venous compliance and response to increased intravenous pressure 10. Venous return to the heart is reduced due to diminished cenous compliance, an increase in venous pooling and intravascular volume depletion 11. Decreased stroke volume and cardiac output 12. Significant decrease in the systolic blood pressure response on rising xi. Normal orthostatic response after prolonged recumbency is significantly compromised xii. Circulatory system is unable to maintain a stable blood pressure and mount an adequate sympathetic vasopressive response xiii. Adaptation to the upright position may be completely lost after 3 weeks of bed rest in healthy individuals d. Hypostatic pneumonia xiv. Clearance of secretions is more difficult in recumbent position 13. Dependent lobes (usually posterior) accumulate more secretions 14. Whereas the upper parts (anterior) become dry 15. Ciliary lining is rendered ineffective for clearing secretions 16. Secretions pool in the lower bronchial tree 17. Impaired coughing due to ciliary malfunction and abdominal muscle weakness 18. Regional changes in the ventilation-perfusion ratio in dependent areas occur when ventilation is reduced and perfusion is increased 19. Significant arteriovenous shunting with lowered arterial oxygenation occurs 20. Atelectasis and hypostatic pneumonia are the ultimate result e. Immobilization osteoporosis xv. Loss of bone mass d/t absence of muscle activity xvi. Aged patients are more prone and at risk xvii. Mechanism; 21. Non-weight bearing over several weeks can cause trabecular and endosteal (and later cortical) mineral bone loss in the tibia (requires 1 to 1. 5 years to return to baseline levels) 22. Loss of calcium

and hydroxyproline from the cancellous portion of long bone, epiphyses, metaphyses, and cortical bone near the bone marrow cavity leads to disuse osteopenia, which develops into disuse osteoporosis 23. Longer duration of immobility increases the time required to restore bone density to pre-immobility levels V. Epidemiology Prolonged immobilization occurs mostly to elderly patients who are prone to stroke and orthopedic accidents regardless of gender. VI. Medications * Calcitonin- for hypercalcemia * Aspirin- for inflammation * Calcium blocker/ beta blockers- * Enoxaparin- for pulmonary emboli * Warfarin(Coumadin)- chronic DVT VII. PT Treatment A. For muscle weakness * Apply PRE’s, stretching, aerobic exercise * Do exercises for flexibility, strength, endurance & fitness * Provide progressive mobility training B. For joint contractures * PROM * Prolonged stretching c low passive tension & heat modality * Resistance exercise to opposing muscles

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