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BODILY RESPONSES TO IMMOBILIZATION

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One of the most prevalent syndromes encountered by rehabilitation specialists is that resulting from prolonged bed rest and immobilization. This is not surprising, since the syndrome occurs whenever illness or injury causes loss of mobility, or whenever a patient is on prolonged bed rest.

The basis of the immobilization syndrome is an imbalance of the normal relationship between rest and physical activity, two biologic processes that are essential to preserve man's optimal physical condition. The prescription of bed rest is a time-honored, common-sense therapeutic measure widely used by physicians in the presence of a serious illness. The value of physical exercise as an equally important treatment modality had been recognized by the Spanish physician Cristóbal Mendez, who in 1533 wrote a treatise on the physiologic responses to exercise and its therapeutic indications.[1] Yet physicians have often neglected exercise prescriptions in spite of the fact that cardiologists, physiatrists, and sports physicians have long documented the excellent results derived from exercise in a variety of clinical conditions.

The literature on the effects of immobilization clearly reflects the interest of numerous investigators in elucidating the pathophysiologic responses to various forms of immobilization. The first experimental study recorded in modern literature was that of Cuthbertson in 1929,[2] followed by the classic investigations conducted in the 1940's by Taylor et al.,[3] Deitrick et al.,[4] and Widdowson.[5] As a result of the interest in human space explorations, there was a new flurry of research in the early 1960's with the goal of measuring the adaptation of the human body to simulated weightlessness.[6,7] From the moment that men started making brief orbital flights until their recent prolonged stays in space laboratories, several investigators have systematically collected data on the performance of astronauts and cosmonauts in the zero-gravity (O-G) environment.[8,11] Simultaneously, rehabilitation researchers started documenting the pathophysiologic changes exhibited by patients with extensive paralysis[12–15] and found some striking similarities between the clinical manifestations of prolonged immobilization in paralyzed patients and in healthy subjects after bed rest or sustained weightlessness.

Modalities of Immobilization

There are four types of inactivity which by themselves may cause the syndrome of immobilization:

1. Prolonged bed rest prescribed for the treatment of an acute illness or injury
2. Restricted neuromuscular activity due to paralysis
3. Continuous stay in a given position (e.g., sitting or recumbent), which effectively reduces the influence of gravity forces
4. State of weightlessness experienced

during space travel (especially if the traveler does not perform isometric or isotonic exercises while in flight) or simulated through prolonged immersion in water

Each one of these modalities of inactivity may produce subtle physiologic changes within a short period of time. Obvious clinical manifestations (e.g., orthostatism) occur within five to seven days, while others (e.g., ankylosis, renal lithiasis) do not become apparent until the individual has remained inactive for 'weeks or months. The first two modalities are of greatest interest in rehabilitation medicine because they prevail in the majority of physically disabled persons.

Basic Physiologic Concepts

As a prerequisite to a discussion of the pathophysiology of immobilization, it is appropriate to define and discuss the concepts of functional capacity, physiologic maximal potential, and functional reserve.

1. **Functional Capacity** is the maximum metabolic rate achieved by a subject during exertion.
2. **Physiologic Maximal Potential** is the maximum metabolic rate that the same individual is capable of achieving after a systematic program of physical training.
3. **Functional Reserve** is the difference between the functional capacity and the physiologic maximal potential.

Kottke discussed these concepts in an article in which he reviewed the impact that bed rest, exercise, and illness may have on an individual's functional capacity, physiologic maximal potential, and functional reserve. [14] As shown in Figure 52-1, the average sedentary person has a certain functional capacity that is considerably lower than his/her physiologic maximal potential. If the individual undertakes a program of physical training, the functional capacity will gradually increase to a point where it almost equals the physiologic maximal potential. On the

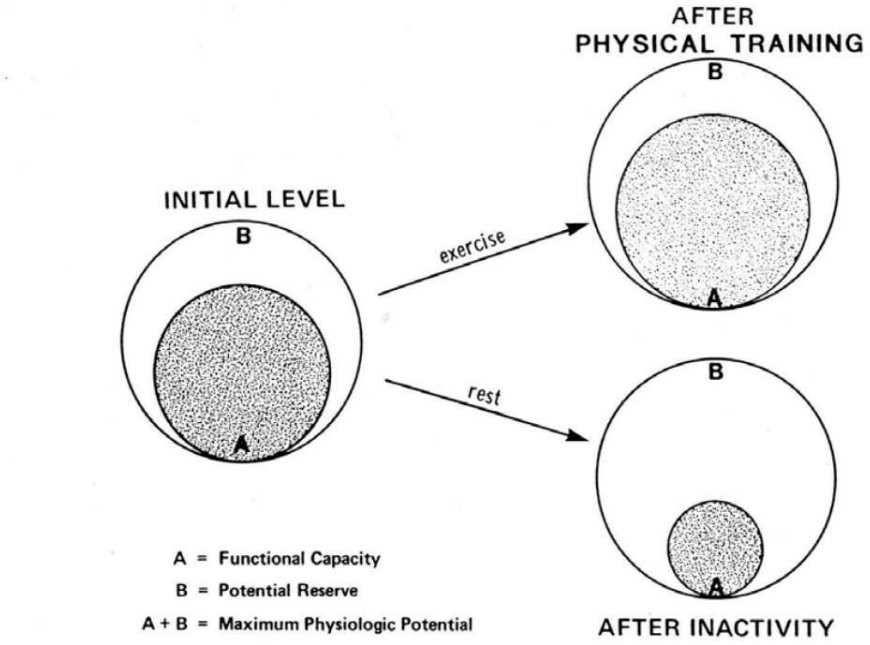

FIGURE 52-1. The effect of inactivity and training on a person's functional capacity, potential reserve, and maximum physiologic potential. Area A inside of the left circle shows the functional capacity of an average sedentary person. Area B in the same circle represents that person's potential reserve. Area A + B represents the person's maximum physiologic potential. After physical training, the individual's functional capacity, as shown by area A on the top circle on the right side, will increase considerably. Similarly, there will be a slight increase in the potential reserve as shown by area B. On the contrary, after prolonged inactivity, there will be a marked decrease in functional capacity, as shown by the Smaller Size of area A in the bottom circle on the right side. The potential reserve will be high, but the maximum physiologic potential will, at best, remain the same as before the period of inactivity. (Modified from Kottke, F. J.: The effects of limitation of activity on the human body. JAMA, 196:117-122, 1966.)

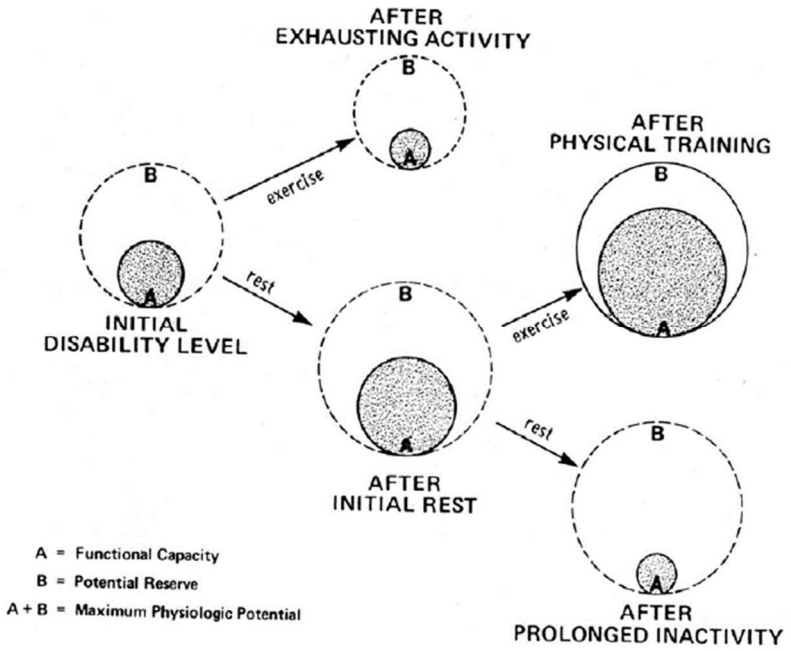

FIGURE 52-2. The impact of illness or injury on a person's functional capacity, potential reserve, and maximal physiologic potential. As a result of disability, both functional capacity and potential reserve (as shown by areas A and B in the circle on the left side) are much smaller than in the healthy state. The maximum physiologic potential (area A + B) is also smaller. A period of initial rest is necessary to achieve partial recovery. This leads to an increase in areas A and B, as shown in the bottom circle of the middle section. Exhausting activity rather than rest during convalescence would cause further losses, as shown by the smaller sizes of areas A and B in the top circle of the middle section. Judicious physical training is necessary beyond the period of convalescence in order to facilitate recovery, as shown by the larger areas A and B in the top circle on the right side of the graph. Prolonged inactivity would result in a serious decrease in functional capacity, as shown by the smaller area A in the bottom circle on the right. (Modified from Kottke, F. J.: The effects of limitation of activity upon the human body. JAMA,196;117−122,1966.)

other hand, continuing lack of exercise or prolonged inactivity will lead to a decline in functional capacity. Figure 52-2 depicts the sudden drop in functional capacity, as well as in functional reserve, which was experienced by the disabled person at the onset of a severe acute illness or injury. The prescription of exhaustive physical activity to an acutely ill or injured person would cause a further drop in functional capacity. At that stage, it is necessary to institute a program of bed rest in order to allow for a gradual recovery of the functional capacity and functional reserve. However, prolongation of bed rest beyond the initial convalescence would cause a new drop in functional capacity and functional reserve. It is for this reason that the patient must start a gradual program of physical activity with the goal of regaining the predisease levels of functional capacity and functional reserve (assuming, of course, that the initial illness or injury has not caused permanent damage).

The magnitude of these changes in functional capacity, functional reserve, and maximal potential depends on the severity of the disease or injury, the duration of bed rest, and the intensity of exercise. They are also dependent on the individual's age, since children recover at faster rates than young adults, [16,17] and these, in turn, at faster rates than the elderly. [18]

Individuals who participate in a regular program of physical exercise reach levels of functional capacity that are close to physiologic maximal potential. By contrast, deconditioned individuals have a marked reduction of functional capacity and eventually may lose the major part of their functional reserve. The process of aging also results in a gradual and irreversible decrease of an individual's functional capacity and functional reserve. However, the

process may be slowed down considerably if the aged individual participates in a well-regulated program of physical activity and exercise.

Clinical Manifestations

The clinical manifestations of the immobilization syndrome are multiple and reflect the fact that prolonged inactivity causes profound physiologic and biochemical changes in practically all organs and systems of the body. The primary involvement in any of them may, in turn, affect the others, thereby setting up a pathophysiologic vicious circle of deleterious consequences. Often, the immobilization syndrome leads to a greater degree of disability than that caused by the initial illness or injury, jeopardizes the rehabilitation process, and significantly increases the cost of care. [19]

Table 52-1 summarizes the manifestations of the immobilization syndrome in each one of the body systems. A summary review of the pathophysiology of these manifestations is provided in the following paragraphs. The interested reader can find a detailed description of the functional pathology of immobilization in a recent monograph by Steinberg. [20]

Central Nervous System

Prolonged immobilization causes disturbances in the central nervous system. Their number and intensity depend, of course, on the primary illness that caused the inactivity. The major manifestations include the following:

Altered Sensation. This occurs because of a general decrease in sensory input. The problem is readily apparent in the paralyzed individual with involvement of the sensory afferent pathways and resulting anesthesia or hypesthesia below the level of the lesion. In some patients, prolonged inactivity in accompanied by paresthesias and a low threshold for pain.

Decreased Motor Activity. Unless the individual carries out frequent isometric or isotonic exercises while in bed (or in a weightless state), the overall motor output of the immobilized person will be consistently lower than that of a sedentary person performing activities of daily living. This problem is especially serious in patients whose restricted physical activity is the result of flaccid paralysis. Patients with spastic paralysis maintain a significant level of muscle activity, thereby preventing atrophy (see below).

Autonomic Lability. The autonomic nervous system of immobilized persons becomes either hyperactive or hypoactive and, as a result, it is difficult to maintain a stable level of autonomic activity and the individual cannot adapt readily to daily stresses (e.g., changes in posture).The effect of autonomic lability on the cardiovascular system is described below.

TABLE 52-1. THE IMMOBILIZATION SYNDROME: CLINICAL MANIFESTATIONS

Central Nervous System
Altered sensation
Decreased motor activity
Autonomic lability
Emotional and behavioral disturbances
Intellectual deficit
Muscular System
Decreased muscle strength
Decreased endurance
Muscle atrophy
Poor coordination
Skeletal System
Osteoporosis
Fibrosis and ankylosis of joints
Cardiovascular System
Increased heart rate (adrenergic state)
Decreased cardiac reserve
Orthostatic hypotension
Phlebothrombosis
Respiratory System
Decreased vital capacity (restrictive impairment)
Decreased maximal voluntary ventilation (restrictive impairment)
Regional changes in ventilation/perfusion
Impairment of coughing mechanism
Digestive System
Anorexia
Constipation
Endocrine and Renal Systems
Increased diuresis and extracellular fluid shifts
Increased natriuresis
Hypercalciuria
Renal lithiasis
Integumentary System
Atrophy of the skin
Bed sores

Emotional Disturbances. Limited sensory input combined with a personal awareness of

unproductivity is the major contributor to the anxiety-depression syndrome that occurs so frequently in the immobilized person.[21,22]
The underlying pathology may be the major factor leading to depression. Contrariwise, a prolonged stay in bed may be a manifestation of depression, since oftentimes a depressed person feels that by staying in bed and sleeping through a chronic illness all the worries will go away and the depression will disappear.

Intellectual Deficit. The capability of an individual to perform intellectual activities may decrease significantly. as a result of prolonged inactivity and confinement. [23] Indeed, immobilized persons often experience difficulty in performing arithmetic or other complex tasks. The resulting frustrations contribute to the state of anxiety-depression previously mentioned.

Muscular System

The most obvious signs of prolonged immobilization occur in the muscular system. This is particularly true in patients who have suffered a paralytic condition. The most common manifestations are the following:

Decreased Muscle Strength. It is a well-known fact that whenever an individual does not carry out a program of moderate physical activity, his/her muscle strength decreases significantly. Studies conducted by Hettinger and Mueller[24] showed that after one week of bed rest there may be up to 20 per cent loss of the initial strength level and another 20 per cent decline in residual strength for each week of bed rest. For example, in the absence of any motor neuron lesion, a man whose grip strength on the dominant side is 50 kg will have a strength of 40 kg after one week of immobilization, 32 kg after two weeks, 25 kg after three weeks, etc. This has major implications in rehabilitation, because the rate of recovery is much slower; i.e., there is only a 10 per cent increase of initial strength per week if the individual participates in a program of daily exercise at 100 per cent of maximal muscle strength.

Decreased Endurance. The loss of endurance after prolonged immobilization is a consequence of the decrease in muscle strength and occurs at a similar rate. Physical exercise, on the contrary, improves muscular endurance in proportion to the overall increase in functional capacity.

Muscle Atrophy. Undoubtedly, the loss of muscle mass is one of the most visible manifestations of prolonged immobilization and it, in turn, accounts for the decrease in muscle strength and endurance. The degree of atrophy varies according to the modality of immobilization. It is quite evident in cases of flaccid paralysis, where for each involved lower motor unit there is abolition of its neuron's action potentials and loss of contraction of all the muscle fibers (tens or hundreds) innervated by that neuron. A totally denervated muscle may lose its normal bulk by as much as 90 to 95 per cent. [25] If denervation is not reversible, the muscle fibers undergo permanent degeneration and are replaced by fat and connective tissue. In patients with spastic paralysis as a result of an upper motor neuron lesion, or in patients immobilized by splinting, the degree of muscle atrophy is only 30 to 35 per cent of normal.

Poor Coordination. The combination of atrophy, decreased strength, and limited endurance leads to poor coordination of movements. It is manifest in both upper and lower extremities, and it seriously jeopardizes the individual's capacity to perform activities of daily living and causes severe frustration. In patients with CNS lesions the major factor leading to incoordination is the pathologic process affecting the motor units or higher centers, but the immobilization per se will also be a contributory factor.

Skeleton System

There are several changes in the bones that result mainly from the muscular disturbances described above. The integrity of man's bone metabolism and the equilibrium between accretion and resorption of skeletal mass depend in great part on the daily stresses and strains imposed by the pulling action of the tendons and by the force of gravity during the standing position. Paralysis and a prolonged stay in the horizontal position will cause profound changes in the skeletal system. The most common manifestations are the following:

Osteoporosis. It results not only from decreased muscular activity but from complex endocrine and metabolic reactions that take place as a result of bed rest (see below). Both organic and inorganic constituents of bone suffer the consequences of immobilization. The well-documented increased excretion of hydroxyproline and calcium after immobili-

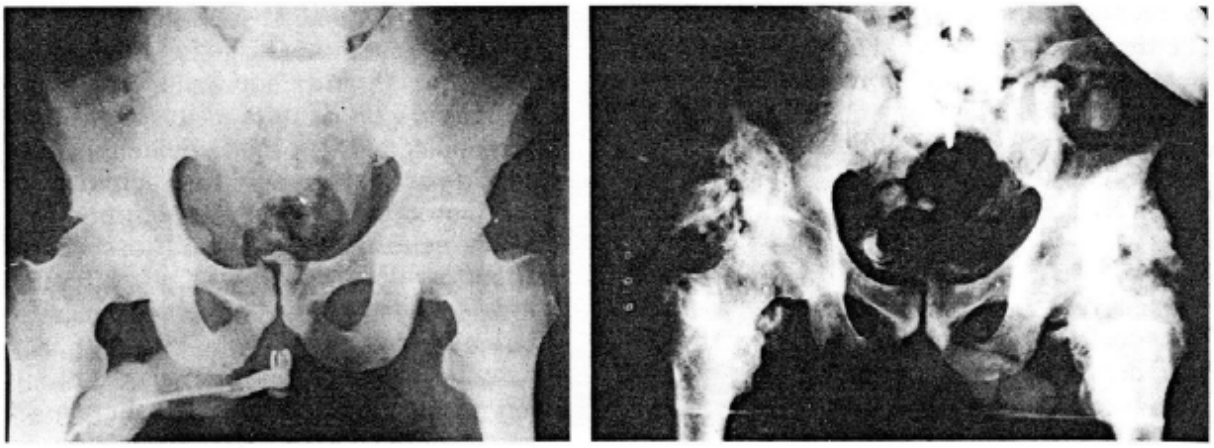

FIGURE 52–3. Radiographs of the pelvis of a 30-year-old paraplegic man. The x-ray on the left was taken within a few days after the onset of a spinal cord injury. It shows an indwelling catheter in place, but the bony structures are intact. The radiograph on the right was obtained at the time of admission to a rehabilitation center, 10 months after the injury. The patient had remained in bed during all this time. The radiograph shows abundant soft tissue calcification around the head of both femurs.

zation clearly shows that there is a depletion of organic and inorganic compounds.[12, 26, 27] The net result is a substantial decrease in total bone mass. The profound metabolic changes that occur in the skeletal system have repercussions in other systems mainly because of the mobilization of bone calcium that causes transient slight hypercalcemia, protracted hypercalciuria, and frequent deposition of calcium in injured soft tissues (ectopic calcification). The x-ray films of Figure 52—3 show clearly the development of ectopic calcification in a paraplegic patient subjected to prolonged immobilization.

Fibrosis and Ankylosis of the Joints. These two impairments are also a major manifestation of the syndrome of immobilization. They occur in varying degrees whenever a joint is not subjected to active or passive motion. Eventually, the overlying muscles are replaced by connective tissue. The joint then becomes stiff, unable to go through a full range of motion, and may become irreversibly deformed. As an example, oftentimes the patient who has suffered a stroke cannot walk well, not only because of paresis and spasticity but also because of ankyloses of the hip and ankle which occur if the patient remains in a recumbent position with the hip slightly flexed and the ankle in plantar flexion.[14] Ectopic calcification of the soft tissues around a joint may cause permanent ankylosis of that joint.

Cardiovascular System

As a result of prolonged immobilization, the cardiovascular system suffers major impairments and cannot respond adequately to metabolic demands above the basal state. The clinical manifestations are as follows:

Increased Heart Rate. Several clinical investigators have pointed out that physical deconditioning leads to a preponderance of the sympathetic or adrenergic system over the cholinergic or vagal system. This accounts for an increase in the basal heart rate. Raab, [28] using two hypothetical examples, contrasted the process of chronic adjustment of the heart rate to daily exercise and to prolonged inactivity:

> Professor A. D. , an eminent scientist and noted Alpinist, former head of the Department of Physiology at the University of Vienna (eighty seven years old) has lived alone for the past twenty years with his paralyzed wife and a library of several thousand volumes in a 15th century isolated log cabin above Schruns, Vorarlberg, at an altitude of 5,000 feet. He cuts trees for firewood, raises his own vegetables, does all household chores, digs a path through deep snow on a precipitous slope, fetches mail and groceries twice weekly from a village in the valley and carries them up one and one-half hours to his abode. He makes extensive climbing excursions alone to high peaks (up to eighteen hours in a stretch) nearly every weekend, he does daily calisthenics at home. He still follows the scientific literature, writes articles and lectures occasionally. In recent years he had a few episodes of auricular fibrillation, but no signs of cardiac failure. His heart rate at rest is 48 to 60, at arrival on mountain peaks, about 90.
>
> Miss B. C. , a sixty-nine year old spinster, former piano teacher, decided at the age of thirty-seven,

when her father died, to spend her life in bed. She stayed in bed for thirty years without interruption (except for one walk around the house after two years), wrote a large book about the courtship of her father, numerous poems and musical compositions. At the age of sixty-seven, when her older sister and house keeper died, she learned again slowly to walk and now spends a few hours out of bed every day. She does not do any housework. She has a small pliable thyroid nodule, but there are no signs of thyrotoxicosis. Her protein-bound iodine is low. Her heart rate, which has been over 100 for many years, is now up to 140 despite digitalization.

It is not clear why inactivity leads to a preponderance of the sympathetic (adrenergic) over the parasympathetic (cholinergic) system, but it is clear that a constellation of metabolic, endocrine, and mechanical factors interact to produce an adrenergic state with an increased cardiac rate as one of its manifestations. Increased basal heart rates (e.g., greater than 80 per minute) are indeed very common in sedentary persons and in those who remain in bed for long periods of time (several weeks). They are also common in paralyzed patients except in cases of high spinal cord injury, which causes complete denervation below the C4 level and which interrupts the transmission of sympathetic impulses to the sinoauricular pacemaker.

Decreased Cardiac Reserve. Under predominantly adrenergic influences there is an increased heart rate, which accounts for less diastolic filling time than at slow heart rates, a smaller systolic ejection, and a greatly diminished capacity of the heart to respond to metabolic demands above the basal level. The importance of maintaining low basal heart rates is. evident if we take into consideration that at low rates the diastolic phase of the cardiac cycle is longer than the systolic phase, whereas at high rates the systolic phase is longer than the diastolic phase (e.g.,at a heart rate of 60/min the electro-mechanical systole lasts 0.40 second and diastole 0.60 second, whereas at a rate of 150/min the systole time is 0.25 second and diastole 0.15 second. Since coronary blood flow occurs mainly during diastole, it is clear that at low rates there is greater blood flow through the coronary arteries per minute than at high cardiac rates. A greatly diminished cardiac reserve accounts for the patient's inability to carry out limited physical efforts. Such efforts may cause marked tachycardia and anginal pain, which often is masked in the quadriplegic individual.

Orthostatic Hypotension. This is one of the most common manifestations of cardiovascular deconditioning following bed rest. Whenever a deconditioned individual attempts to sit up or stand up there is marked pooling of blood in the lower extremities, thereby decreasing the circulating blood volume and the venous return, preventing adequate ventricular filling during diastole, and causing the ventricle to eject a stroke volume that may be too small to achieve effective cerebral perfusion. In some instances, the blood pressure reaches levels as low as 60/30 mm Hg within 10 to 20 seconds of sitting with the legs hanging unsupported at the side of the bed. The pooling of blood in the lower extremities occurs because, in spite of an increased adrenergic state with concomitant stimulation of the vascular alpha-receptors, the arterioles and venules of the legs do not constrict sufficiently well to offset the effect of gravity on the column of blood that falls from the heart to the feet. As a result, there is increased hydrostatic pressure in the capillary bed, extravasation of fluid in the interstitial tissue, and marked dependent edema. The orthostatism that is so evident after prolonged immobilization contributes to the endocrine and metabolic disturbances that are also part of the immobilization syndrome[29] (see below).

Phlebothrombosis. One would expect that during prolonged bed rest there would be considerable venous stasis in the legs if we consider that in the horizontal position there is infrequent intermittent constriction of the arterioles and venules as well as a reduced or absent pumping action by the skeletal muscle. Yet prolonged bed rest in healthy subjects does not slow down significantly the ankle-to-groin venous flow time.[30] In spite of this, it is well known that paralyzed patients have a greater tendency to develop phlebothrombosis than the general population. The incidence of phlebothrombosis in patients with spinal cord injuries is considerably higher than in the normal population.[31,32] In hemiplegic patients, phlebothrombosis is more frequent in the paralyzed than in the nonparalyzed side. It is evident that some hemodynamic changes in the venous return do occur in all paralyzed persons. These changes, coupled with a disturbance in the clotting mechanism, constitute a major determinant factor in the pathogenesis of phlebothrombosis. Possible changes in the production of prostaglandins or other disturbances of the platelet-aggregating mechanisms may play a role in the phlebothrombosis of immobilized persons, but this has not been clearly elucidated. The presence of sepsis or of infection in the wall of a major vein may precipitate the clotting process.

Respiratory System

The changes in respiratory function that occur in immobilized persons may also contribute to the severe disability that occurs as a result of immobilization, especially in patients with CNS lesions. The typical picture is that of a restrictive respiratory impairment which is manifested by the following:

Decreased Vital Capacity. Although healthy subjects subjected to prolonged bed rest do not show any significant reduction in the total lung capacity and its subdivisions (vital capacity, inspiratory capacity, expiratory reserve volume, functional residual capacity, and residual volume),[33] most immobilized patients, especially those with quadriplegia, show a greater reduction in vital capacity than one would expect from their underlying musculoskeletal pathology. Indeed, the inactive patient while in the supine position will seldom contract the intercostals, diaphragms, and abdominal muscles in order to accomplish a maximum inspiration or a forceful expiration. The overall decrease in muscle strength that was mentioned previously (see above) may eventually affect the respiratory muscles. If, in addition, the costovertebral and costochondral joints are not submitted to a full range of motion, they may become fixed in an expiratory position, thereby decreasing even further the capacity of the chest to achieve maximum inspiration. Thus, in the immobilized paralytic patients there can be a 25 to 50 per cent decrease in vital capacity and in functional respiratory capacity.

Decreased Maximal Voluntary Ventilation. The same mechanisms that account for a decrease in vital capacity lead to inability to sustain a maximal ventilatory effort and to decreased respiratory endurance. The observed values of maximal voluntary ventilation in the inactive person are significantly lower than predicted (by as much as 25 to 50 per cent below that of a nonparalyzed person).

Regional Changes in the Ventilation/ Perfusion Ratio. As a result of the restrictive impairment described above and of the effect of the horizontal posture on the pulmonary circulation, there are marked regional differences in the ventilation/perfusion ratio.[20] Normally, these differences are not significant because the individual consciously or unconsciously mobilizes the thorax sufficiently to prevent underventilation and overperfusion of major parts of the lung. This does not happen in the immobilized person and, consequently, the dependent areas become poorly ventilated and overperfused. As a result, there is a significant arteriovenous shunt that lowers the arterial oxygen tension (Pa02). Although this may not be significant while at rest, if the patient has an increased metabolic demand (because of an infection or exercise), hypoxia becomes apparent.

Impairment of the Coughing Mechanism. As a result of immobilization, even in healthy subjects, there is a significant decrease in the normal ciliary efficiency. [12] Because of this, the respiratory mucus secretions tend to accumulate in the dependent bronchioles, become more viscous than usual, interfere with the normal ciliary motion, and adhere to the airway epithelium. Coughing may fail to clear the bronchial tree, the problem being compounded if there is abdominal muscle weakness due to a motor lesion. Under these conditions, a mild upper respiratory infection may cause severe secondary infection of the lower airway and of the lung tissue.

Digestive Apparatus

An often overlooked aspect of the immobilization syndrome is an overall decrease in gastrointestinal activity, which affects not only the motility but also the secretory functions of the digestive glands (salivary, pancreas). The major manifestations are the following:

Anorexia. It is not surprising that the decreased caloric demands of the inactive individual result in significant loss of appetite. Often the anorexia is also a manifestation of the anxiety-depression that accompanies the immobilized State. The profound endocrine changes that occur in this state may further decrease the person's appetite.

Constipation. The adrenergic preponderance that occurs in the immobilized state inhibits peristalsis and constricts the sphincters. The overall result is a decrease in gastrointestinal motility. Contributing factors may be an increased intestinal absorption of water and a low dietary intake of liquids and/or fiber. In some instances, the protracted constipation causes severe fecal impactions.

Endocrine and Renal Systems

The profound metabolic and renal changes that occur in the immobilization syndrome result from the interaction of the endocrine system with others. There is an abundant literature on the nature of the endocrine and metabolic alterations that occur after bed rest [12] and in paralyzed persons.[34] Claus Walker and Halstead have done a thorough review of the

literature and have compiled the results of the most significant studies in a series of articles . [35] The most important endocrine and metabolic manifestations of the immobilization syndrome are the following:

Increased Diuresis. It occurs predominantly in the early phase of bed rest because the placement of the body in a horizontal position causes a temporary increase in circulating blood volume. This is due to a shift of some extracellular fluid to the venous side of the capillary bed with the subsequent increase in venous return. As a result, there is a stimulation of the volume receptors of the right atrium and a reflex inhibition of the antidiuretic hormone (ADH). The initial diuresis of bed rest does not pose a problem in the patient who receives intravenous fluid therapy.

Increased Natriuresis. This is also a temporary occurrence concomitant with the initial diuresis and it represents an attempt to maintain the plasma osmolality at a normal level.

Hypercalciuria. As mentioned previously (see above) osteoporosis is one of the major complications of prolonged inactivity. From a clinical standpoint, an important consequence is the constant mobilization of calcium from the bone matrix into the blood and eventual urinary excretion of the excess calcium. In addition to the previously mentioned mechanical factors that intervene in the development of osteoporosis, one has to consider the contributory role of the adrenal corticosteroids that may be released in excessive amounts because of the stress that accompanies the immobilizing disease or injury as well as the transient stress that occurs when the immobilized person starts sitting up or standing and develops orthostatic hypotensi[29] Under these conditions, and in an attempt to compensate for the decrease in circulating blood volume, there is a reflex release of fluid-retaining hormones (ADH, aldosterone, cortisol). Although the compensatory effect of these substances is not too efficacious in preventing orthostatism, it nevertheless has a lasting metabolic influence and eventually facilitates gradual adaptation to the upright posture.

Renal Lithiasis. The triad of hypercalciuria, urinary stasis, and urinary tract infection, when present, is dangerous because it leads to the production of calculi in the renal pelvis or in the lower urinary tract. The problem is of greater magnitude in paralyzed persons because of their marked hypercalciuria, their impaired bladder function, and the inevitable urinary tract infection that occurs in catheterized patients. Staghorn calculi develop in the renal pelvis (Fig. 52—4), and stones of various sizes may settle in the urinary bladder. Urinary obstruction poses serious problems, especially in the anesthetized spinal cord injury patient who does not feel the usual pain of renal colic. Repeated episodes of urinary tract infection and calculi may cause gradual impairment of renal function and eventually lead to a clear-cut picture of renal insufficiency.

Integumentary System

The skin and adnexa are not immune to the pathophysiologic changes that result from prolonged immobilization. The most common manifestations are the following:

Atrophy of the Skin. The subtle changes in extracellular fluid volume that occur in the dependent parts of the body affect the consistency of the subcutaneous tissues and dermis and lead to a gradual loss of skin turgor. Insufficient appetite and inadequate nutrition result in loss of subcutaneous fat and contribute to changes in skin turgor. Inadequate hygiene may worsen the problem and lead to skin breakdowns as well as to paronychia and ingrown toenails.

Bed Sores. They are common manifestations of prolonged immobilization and account for a great part of the cost of the rehabilitation of inadequately treated paralyzed individuals. [19] Chapter 46 discusses the problem of decubitus ulcers in detail. [36] It is important to state here that extensive bed sores produce considerable loss of protein, especially albumin. At the capillary level, the decrease in serum protein results in a drop in oncotic pressure. This facilitates extravasation of fluid to the extracellular space when the individual sits up or gets up from bed. The extravasation aggravates the decreased circulating blood volume that occurs under the influence of gravity and contributes .to the orthostatic hypotension that is so common in immobilized individuals (see above).

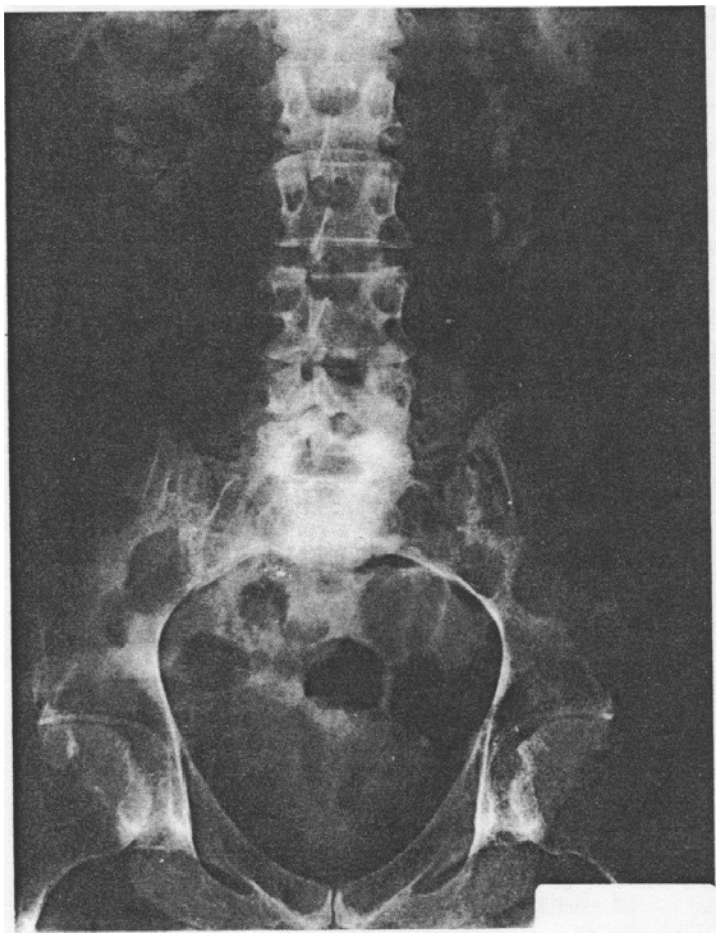

FIGURE 52-4. Radiograph of the abdomen of a quadriplegic person eight months after a cervical spinal cord injury. The patient had been inactive most of the time. Notice the kidney stones in the right and left kidney areas. There is extensive osteoporosis of the spine and pelvis

Prevention and Treatment

In order to prevent or to minimize the physiologic changes that occur as a result of immobilization, it is necessary to institute the following measures:

Sensory Stimulation. The institution of a program of sensory stimulation is the most effective means of preventing or treating the CNS manifestations of immobilization. A good comprehensive rehabilitation program must provide for environmental stimuli in order to ensure adequate sensory input to high cerebral areas and to compensate for whatever losses of sensation have occurred because of a CNS lesion. Participation in occupational and recreational therapy while in the hospital and stimulation by the patient's family to participate in discussions, watching of television, etc., may provide an antidote to sensory deprivation and may help overcome the emotional disturbances that occur frequently in the immobilized patient. Similarly, it is necessary to provide intellectual challenges to these patients and make them perform specific tasks (e.g., arithmetic, comment on news) in order to preserve intellectual function at a level similar to that before immobilization.

Active Muscle Exercise. A nonparalyzed person who must remain in bed should keep all muscle groups active to prevent muscular deconditioning. If there are no cardiovascular contraindications, the patient should be able to perform a variety of isotonic exercises such as the leg movements necessary to activate bicycle pedals mounted on a board that can lie on the patient's bed.[37] The exercise should take place at least once daily for 5 to 10 minutes.

Another useful way to preserve the integrity of the musculoskeletal and circulatory systems while in bed is the performance of simple isometric exercises. A particularly good one consists in making the supine patient apply pressure with the feet against a board placed perpendicular to the surface of the bed. This requires active isometric contraction of practically all muscle groups, especially those of the back and legs. Initally, the patient should do this once or twice a day by applying sustained pressure against the board for 5 seconds, relaxing for 10 seconds, and repeating the isometric contraction-relaxation three or four times. This type of exercise is obviously contraindicated in patients with spinal problems. In order to achieve effective isometric contractions of the arm muscles the patient should make a bilateral strong grip action for a few seconds with the arms

extended. This exercise should take place at the same rate and frequency as the trunk and leg isometric exercises.

Adequate Positioning and Range of Motion Exercises. Both are necessary to prevent the skeletal changes that occur in immobilized, heavily sedated, or debilitated patients. The extremities should be properly positioned by maintaining each joint in its functional position to avoid ankylosis and deformities. Furthermore, the physician should prescribe range-of-motion exercises to be performed by a physical therapist, a nurse, or a member of the patient's family after proper training. A judicious program of range-of-motion exercises consists of making three to five consecutive full-range movements of each joint at least once (preferably twice) daily. The bathing of the patient offers an excellent opportunity for the nursing staff (or the family) to achieve passive range of motion exercise of practically all the joints.

Cardiovascular Reconditioning, Passive Tilt. The programs of muscular exercise previously described are helpful in preventing severe cardiovascular deconditioning. It is important, however, to avoid the patient's exhaustion by imposing excessive metabolic demands. During bed rest it is also prudent to maintain a heart rate at less than 120/min while performing isotonic or isometric exercises.

Individuals who are not paralyzed should assume the sitting and the standing postures as soon as feasible. This should take place gradually, and as stated previously, the first attempts to sit up should consist in propping up the head of the bed at gradually increasing angles while keeping the legs horizontal. As the patient begins to sit at the edge of the bed and to stand up it is useful to prevent orthostatic hypotension by covering the legs with elastic stockings.

If the patient is unable to sit up because of paralysis or other disabling condition, it is highly advisable to impose the effect of gravity upon the body through passive assumption of the upright posture on a passive tilt table. Initially, the patient should reach a slight degree of tilt (e.g., 30 degrees), remain there for one minute, and gradually increase the duration of tilt to 30 minutes twice a day. As the patient's tolerance improves, the degree of tilt is increased by 5 to 10 degrees every week until the patient tolerates the 70 degree position (equivalent to IG force) for about half an hour twice a day.

Elastic stockings help to minimize the effects of gravity because they prevent stasis of blood and edema in the lower extremities. The patient should wear the stockings most of the time while lying in bed, but especially in the periods of sitting or standing. In instances of severe orthostatic hypotension, the stockings should be extra firm in order to counteract the hydrostatic pressure exerted by the blood which pools in the lower extremities. Spinal cord-injury patients who cannot tolerate a gradual program of passive tilt in spite of wearing extra firm stockings may benefit from an especially tailored garment fashioned after a lower body G-suit.[38]

Respiratory and Coughing Exercises. The prescription of respiratory exercises is an essential component of the management of immobilized patients. While in bed, the inactive person must take three to five slow, deep breaths at least every hour while awake. A forced expiration should accompany each maximum inspiratory effort to maintain all lung compartments at a normal level. In order to reach a full inspiration, the person must learn to use equally the intercostals and the diaphragms. A useful approach is to concentrate on expanding the abdomen during inspiration. As the individual contracts both diaphragms, he lowers their position and pushes the viscera against the abdominal wall. If the abdominal muscles are weak a good contraction of the diaphragms results in marked expansion of the abdomen because of the limited resistance offered by the flabby abdominal wall. If the patient has respiratory muscle paralysis with a decrease in vital capacity below 60 per cent of normal, it is useful to carry out three to five passive lung inflations twice daily with a positive pressure apparatus at high settings of pressure, flow, and volume.

The nonparalyzed immobilized person should make a conscious effort to cough forcefully several times a day to prevent accumulation of tracheobronchial secretions and to facilitate periodic ventilation of all alveolar areas. Paralyzed persons can achieve effective coughing with weak expiratory abdominal muscles by applying pressure in the abdominal cavity at the end of a deep active inspiration or a passive inflation with a positive pressure apparatus.

Appropriate Nutrition and Fluid Intake. They are the mainstays of the prophylaxis of severe digestive and metabolic complications of immobilization. The caloric intake should be commensurate to the metabolic needs of the patient while at bed rest (these needs are greater if the patient is on an exercise program or has fever). Under most circumstances, there seldom is

a problem of excessive caloric intake because the patient's inactive state leads to loss of appetite. The diet, of course, must be adequately balanced and should have a high fiber content to facilitate bowel movements. If the diet is nutritious, there is no need for vitamin supplements.

A stool softener (e.g., dioctyl sodium sulfosuccinate) may be useful in preventing constipation and fecal impaction.

A diet of approximately I gram of protein per kilogram of body weight and I gram of calcium per day seems to prevent osteoporosis and hypoproteinemia in the nonparalyzed individual. In cases of hypoproteinemia, the protein content should be approximately 1.5 grams per kilogram of body weight. Paralyzed patients who have experienced a severe loss of lean body mass develop a significant degree of osteoporosis that is impossible to prevent. [39,40] These patients do not require a calcium supplement, since an excessive calcium intake would contribute to hypercalciuria and to renal lithiasis (assuming the presence of a concomitant urinary tract infection and renal stasis).

An adequate fluid intake is always necessary to prevent urinary complications unless the patient's cardiac condition is such that an increased fluid intake would create an unnecessary load to the heart muscle.

Sodium intake must be the usual one without the need to administer sodium supplements, since increased natriuresis is a transient occurrence and often inadvertent. Hypertensive patients should have a restricted sodium diet as required to maintain the blood pressure under control and to potentiate the effects of antihypertensive drugs, especially the thiazides.

Skin Hygiene. The immobilized patient must receive adequate skin hygiene. It is necessary to cleanse the skin thoroughly and to massage it adequately to maintain good turgor and avoid infections. 'This is particularly important in the skin of the dependent areas, since they are under constant pressure against the lying surface. Frequent trimming of all fingernails and toenails is a most important preventive measure.

In cases of immobilization due to paralysis, to heavy sedation, or to general debilitation, it is necessary to change the patient's position periodically in order to prevent prolonged, excessive pressure on the skin of the dependent areas. An accepted schedule is to change the body from one side to the back and from the back to the other side every two to four hours.

Summary

Rehabilitation physicians and allied health professionals who constitute the rehabilitation team must be aware of the pathophysiologic changes brought about by prolonged immobilization. These changes affect practically all systems of the body and may cause a greater degree of disability than the one produced by the illness or injury that caused the patient to remain in bed. The most salient manifestations of the immobilization syndrome are (1) in the central nervous system, changes in sensation, decreased motor output, autonomic lability, emotional disturbances, and intellectual deficit; (2) in the muscular system, decreased strength and endurance, atrophy, and poor coordination; (3) in the skeletal system, osteoporosis, joint fibrosis, and ankylosis; (4) in the cardiovascular system, increased heart rate, decreased cardiac reserve, orthostatic hypotension, and phlebothrombosis; (5) in the respiratory apparatus, decreased vital capacity, decreased maximum voluntary ventilation, loss of respiratory endurance, regional changes in ventilation/perfusion, and impaired coughing; (6) in the digestive system, anorexia and constipation; (7) in the metabolic and renal systems, increased diuresis, hypernatriuresis, hypercalciuria, and renal lithiasis; and (8) in the integumentary system, atrophy of the skin and bed sores.

The prevention and treatment of the immobilization syndrome require (1) provision of environmental stimuli and intellectual challenges, (2) active isotonic or isometric exercises, (3) adequate position and passive mobilization of all joints, (4) gradual cardiovascular reconditioning through physical exercise and passive tilt, (5) respiratory and coughing exercises, (6) adequate nutrition and fluid intake with protein and calcium supplements when indicated, and (7) skin hygiene.

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