The Complications Of Immobility In The Elderly Stroke Patient

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Abstract: ***Background:*** **Approximately 500,000 persons in the United States suffer a stroke each year; the majority of these individuals are 65 years of age or older. The neurological impairment occurring as the result of stroke can lead to both acute and chronic disability. Further medical complication and disability are often the result of immobility-related illness that occurs while the patient is still in the hospital.**

Methods: **A MEDLINE search for articles published from 1980 to 1990 was made using the key words immobilization and stroke rehabilitation. The bibliographies of these articles, key rehabilitation and geriatric textbooks, the bibliographies of these textbooks, and the authors' personal files were also sources of information.**

Results and Conclusions: **Immobility-related medical complication and disability can be substantially reduced by identifying risk factors and applying preventive measures. As long-term providers of medical care, family physicians are in a position to devise a preventive care plan for immobility-related disability and to appreciate the beneficial effects of such a plan on patient outcome. (J Am Board Fam Pract 1992; 5:389-97.)**

Most family physicians care for stroke patients and witness not only the acute neurologic event of stroke, with its physical and functional effects, but also the related chronic disability. The present US population of long-term disabled stroke survivors is large, estimated at about 2 million persons.[1,2] Because family physicians care for many of these individuals, methods of reducing stroke-related disability are important to the family practitioner.

Acute and chronic disability from stroke has several causes; the two most important are the functional effects of the neurologic impairment itself and the added functional decline related to immobility. Although in many cases modern science cannot substantially reduce the actual neurologic deficit, immobility-related decline is preventable.[3] This review focuses on the approaches designed to prevent the adverse consequences of immobility. It is important that this aspect of acute stroke management be considered early, as immobility-related functional decline can occur in an elderly stroke patient within a few days. As an additional factor in the older stroke patient, normal aging produces physiologic changes that put the elderly individual at increased risk from bed rest.[4]

The potential consequences of immobility in the stroke patient and appropriate preventive strategies are listed in Table 1. Early mobilization and an activity prescription are key points in each preventive intervention. In many cases clinicians are not attentive to activity orders. A recent study of hospitalized, elderly, medical-surgical patients in five New England hospitals revealed that of a total of 3500 patient days covering the first 7 days of admission, 13 percent of patient days had no activity order in effect and that a similar percentage of patient days had a bed rest order.[5] On average, 8 percent of patients had a bed rest order for the entire 7 days studied.[5] All too often the hospitalized elderly patient is kept in bed for ill-defined medical reasons, because the physician forgets to advance the activity order or because mobilization is thought to strain available manpower. In reality, there are very few medical illnesses that require strict bed rest, and the mobile, conditioned, aged patient requires less physical assistance in the long run.

Although the stroke patient is discussed specifically in this review, the information presented is generally relevant to any immobilized elderly person.

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Table 1. Preventive Strategies for Potential Complications of Immobility in the Elderly Stroke Patient.

Complication	Preventive Strategies
Skin	
Decubitus ulcer	Early mobilization Frequent body repositioning Daily skin inspection Pressure-relieving mattress surface Bowel and bladder management Adequate nutrition
Bowel	
Constipation Fecal impaction Fecal incontinence	Early mobilization Bowel clean-out, if necessary High-fiber diet Adequate fluid intake Stool softeners Bowel evacuation regimen
Bladder	
Urinary tract infection	Hydration
Urinary retention Urinary incontinence (overflow)	Early mobilization Investigate for secondary causes of retention Prompted voiding Intermittent catheterization, as necessary Avoid Foley catheter drainage, if possible
Cardiovascular	
Orthostatic hypotension	Early mobilization Elastic stockings Slow elevation from supine to sit to stand Daily "leg dangling" Adequate fluid intake
Deep venous thrombosis and pulmonary embolus	Early mobilization Elastic stockings Subcutaneous heparin Range-of-motion joint exercises
Pulmonary	
Atelectasis Aspiration pneumonia	Early mobilization Prompt assessment and management of dysphagia Bedside respiratory care Proper positioning
Metabolic and endocrine	
Osteoporosis	Early mobilization and weight bearing
Urinary tract stones	Early mobilization Adequate fluid intake Urinary acidification, if necessary
Musculoskeletal	
Deconditioning	Early mobilization Daily passive and, if tolerated, active assistive range-of-motion exercises Bed mobility training Daily weight bearing Encourage activities of daily living
Contractures	Early mobilization Proper bed positioning Range-of-motion exercises
Neuropsychiatric	
Sensory deprivation and depression	Early mobilization Appropriate communication system Increase patient interaction with staff and family Provide necessary adaptive aids Environmental orienting cues

Skin

Decubitus Ulcer

There are four physical factors involved in the development of decubiti: pressure, shearing force, friction, and moisture. The stroke patient is at increased risk of skin breakdown because of all four factors. Additionally, stroke patients are often immobile, incontinent, and confused. Edema, sensory impairment, malnutrition, poor circulation, and anemia also predispose to skin breakdown.[6,7] The consequences of skin breakdown in the stroke patient include local infection, pain, positioning problems, a delay in ambulation because of the location of the decubitus, and worsening spasticity, as the lesion is a nociceptive stimulus.[8]

In general, elderly patients are more prone to skin breakdown because of a higher rate of contributing comorbid illnesses, as well as intrinsic age-related cutaneous changes. Age-related skin changes that increase the risk of traumatic injury include delayed wound healing because of decreased epithelial cell turnover, thinning of subcutaneous tissue, loss of collagen elasticity, dermal atrophy, and flattening of rete pegs.[9]

Compounding these age-related risks, the elderly immobile stroke patient can be exposed to the four physical factors noted above. For example, pressure phenomena occur under bony prominences when an individual lies on a normal mattress.[10] As capillary pressure (32 mmHg) is exceeded, local tissue ischemia develops. As shown in animal studies, pressure effects are an essential, but not solitary, component of decubiti formation.[11] Substantial pressures (up to 290 mmHg) do not produce breakdown in pig skin.[11] Much lower pressures ($\geq$ 45 mmHg), however, result in decubiti when coupled with other adverse physical factors.[11] These additional factors are almost always present in a bedridden patient. One factor, shearing force, is particularly relevant for the sacral area. The sitting patient (including the patient sitting on a reclining chair) or the supine, bedfast patient whose head of the bed is raised greater than 30 degrees can slide forward, creating a strong shearing force on the sacrum.[10] This shearing force stretches dermal blood vessels and decreases cutaneous blood flow, thereby compounding the tissue ischemia from pressure phenomena.[11] Friction develops when patients are pulled across bed linens. Finally, moisture and skin maceration occur if there is inattention to incontinence and skin occlusion by bedding.[10]

From both a time and cost perspective, decubitus ulcers are much more easily prevented than cured.[12] Important points in prevention include prompt mobilization, bowel and bladder management, early assessment and attention to the provision of nutrition, daily skin inspection, a pressure-relieving mattress surface, and frequent repositioning.[13] Although the "turn every 2 hours rule" is quoted in general texts, the necessary frequency for turning is unclear and probably varies with the patient's risk status.[11,14] Regarding positioning, the right and left 30-degree oblique positions will relieve the five major pressure points of the sacrum, greater trochanters, ischial tuberosities, lateral malleoli, and heels.[6,10,14] These bony areas should be inspected for skin change with every repositioning; nonblanching erythema is a prelude to breakdown. When positioning a patient, limbs that rest against each other should be separated with a pillow. If erythema is present, heels can be elevated from the bed by placing a pillow under the calf. A sheepskin placed over the sheets can reduce skin friction and maceration.

Regarding mattress surface, the appropriate pressure-relieving device is unclear. Regular 2-inch eggcrate foam pads do not substantially decrease local pressure but can increase comfort and still help in decubitus prevention.[15] Other foam pads of different construction (Geo-matt™) have minimal cost and will decrease local pressure below capillary pressure.[15] Air fluidized beds (Clinitron™) and alternating pressure air mattresses (Clinicare™) work well but are expensive, and the former impair bed mobility and transfers.[11] No pressure-relieving mattress surface replaces the need for regular turning and skin inspection. Special beds are not needed for the typical stroke patient if a good program of prevention is instituted early.

Bowel

Constipation, Fecal Impaction, and Fecal Incontinence

The increased sympathetic nervous system response to bed rest results in inhibition of gastrointestinal peristalsis and consequent constipation and fecal impaction.[4,16,17] Additionally, many elders are chronically constipated and take medi-

cations with potential constipating effect. Drugs that cause constipation include nonsteroidal anti-inflammatory agents, calcium channel blockers, opioids, iron supplements, phosphate-containing antacids, and medications with anticholinergic activity.

Fecal impaction can be of two types, colonic and dyschezic. Colonic impaction occurs when there is prolonged transit time in the entire large intestine, which results in water absorption from the stool and the development of hard fecal masses in the colon and rectum. In dyschezic impaction, transport is normal until the sigmoid colon and rectum, where a large mass of soft feces is formed. Soft stool, therefore, does not rule out impaction but can point to its cause.[18]

Often fecal impaction presents clinically as fecal incontinence.[18] Leakage of stool occurs when feces overload the rectum, causing a decrease in sphincter tone and stool release. Other nonneurogenic causes of fecal incontinence include overuse of stool softeners and cathartics and the effects of functional limitations. Examples of the latter are impaired mobility and impaired communication that could limit access to the toilet and impaired cognition that could limit awareness of the need to use the toilet. In the stroke patient these nonneurogenic causes of fecal incontinence are much more common than is incontinence caused by a neurologically impaired bowel. In fact, because of the nature of bladder and bowel innervation, if there is no urinary incontinence, any fecal incontinence is almost exclusively from a nonneurogenic cause.[19] The presentation of the fecal incontinence can give a clue as to its cause. Patients with fecal impaction usually leak a small amount of loose stool throughout the day; patients with a neurologically impaired bowel will be incontinent of a small, formed stool, once or twice daily, coincident with their gastrocolic reflex.

The prevention and treatment of constipation, fecal impaction, and fecal incontinence include the same basic principles. Cathartics can be required initially to clean out the bowel. A moderately high-fiber diet, stool softeners, and adequate fluid intake are needed to maintain a soft, formed stool.

The final factor is an established regimen for bowel evacuation. It is recommended that the patient use the toilet daily, preferably in the morning, 15 to 30 minutes after breakfast, to take advantage of the gastrocolic reflex.[20] Also, a commode or toilet is preferable to a bedpan so intra-abdominal pressure can assist in complete evacuation. Finally, on a predetermined schedule based on the patient's premorbid bowel habits, a glycerin or bisacodyl suppository can be used if the patient does not pass a stool spontaneously. To work most effectively, the suppository must be placed next to the bowel wall, where its local irritant effect can stimulate the sacral evacuation reflex. Scheduled cathartics can also be used but can lead to incontinent episodes.

Bladder

Urinary Tract Infection, Urinary Retention, and Urinary Incontinence

The stroke patient is prone to developing a urinary tract infection because of many potential factors including a preexistent atonic bladder, the change in urogenital flora with hospitalization, possible comorbid conditions, such as malnutrition and prostate enlargement, poor perineal hygiene, and most importantly, the common use of a Foley catheter. Catheter drainage should be used for a limited number of indicated conditions that are discussed below, not as a matter of medical or nursing convenience.

Bed rest promotes urinary retention through several mechanisms. Postvoid residual urine is often increased because of incomplete relaxation of perineal muscles during supine voiding. Also, gravitational forces and intra-abdominal pressure have a lessened effect on supine voiding.[16] Outlet obstruction, medications, and comorbid disease also can be factors in increasing residual urine or causing retention. A patient with urinary retention should be examined for causes of outlet obstruction, such as fecal impaction, urethral stricture, or prostatic enlargement. Additionally, elderly patients often are receiving medications that can compound the effects of bed rest on residual urine. Medications with anticholinergic effect (e.g., tricyclic antidepressants, antihistamines, and neuroleptics) will cause detrusor relaxation, calcium channel blockers directly impair detrusor contractility, and adrenergic blockers (e.g., reserpine, ephedrine, and phenylpropanolamine) will increase sphincter tone and cause outlet obstruction.[20] Comorbid disease, such as dia-

betes, can be associated with an atonic bladder. A chronic neurogenic bladder can be occult if before being bedridden the patient has been successfully voiding despite having high postvoid residual urine volumes. Retention only develops with the added insult of immobility. Urinary retention can lead to overflow incontinence as urine output exceeds bladder capacity.

Except during the acute flaccid muscle phase, stroke does not cause urinary retention or overflow incontinence. When either of these two conditions occurs, it is more likely caused by immobility compounded by the factors or diseases discussed above. In a true neurogenic bladder caused by stroke, incontinence is due to loss of central inhibition over bladder contraction.[21] Bladder emptying and, therefore, postvoid residual urine are normal (less than 50 to 70 mL), but uninhibited bladder contractions result in urge incontinence. Even with urge incontinence, functional issues rather than true neurologic damage could be the cause. Again, as in fecal incontinence, the physician should consider functional causes, such as impaired mobility, communication, and cognition.

The patient who has experienced several days or more of bladder distention may require temporary bladder rest by catheter draining. Causes contributing to retention should always be sought. For acute retention, for example, as occurs during the acute flaccid muscle stage of stroke, a short period of regular, intermittent (every 6 to 8 hours) bladder catheterizations might be all that is necessary. The goal of frequent catheterizations is to maintain bladder volume at no greater than 400 mL. Greater volumes will cause overdistention and lesser ones might not provide an adequate stimulus for voiding. As detrusor tone recovers, voiding again occurs, postvoid residual urine decreases, and catheterization becomes unnecessary. In all cases, manual bladder pressure (Credé method) techniques can be taught to increase emptying. A program of timed voiding (every 2 to 4 hours) on a commode or toilet should also be initiated to encourage a schedule of bladder emptying. Prompted voiding is also important in the prevention of immobility-related urinary retention. Persistently high postvoid residual urine or urinary retention should prompt referral to a urologist.

Cardiovascular Effects

Orthostatic Hypotension

Orthostatic hypotension is a relatively common event in the aged, occurring in 20 to 25 percent of unselected elderly patients.[4,22] Postural hypotension is due to physiologic changes of aging along with coincident factors of disease and drug use.[4,22] The ill effects of orthostatic hypotension in the stroke patient can be serious and include stroke extension and falls resulting in fracture.

Age-related changes affecting postural mechanisms include a decrease in baroreceptor and β-adrenergic receptor sensitivity, a decrease in resting ventricular stroke volume, and a blunted response of stroke volume and cardiac output to stress.[4,22] Disease states that can contribute or lead to orthostatic hypotension include diabetes mellitus, vitamin B deficiencies, hypothyroidism, Parkinson disease, and any process leading to hypovolemia.[22] Medications that can result in orthostatic hypotension include diuretics, anti-hypertensives, antidepressants, and major tranquilizers.[22]

Bed rest further increases the rate of orthostatic hypotension in the already predisposed aged patient. The normal postural compensatory changes in heart rate, stroke volume, and cardiac output are lost after only 1 or 2 weeks of bed rest.[4,16,17,23] Shorter periods of inactivity will still have a negative effect on postural mechanisms, especially in the predisposed elderly patient.[16] Additionally, the diuresis precipitated by bed rest increases the tendency toward orthostatic hypotension.[24] Subsequent reconditioning of postural reflexes takes time. In young persons placed at bed rest for 3 weeks, recovery required 1.5 to 2.5 months.[25] It is reasonable to assume postural recovery would take at least as long in older persons.

Several simple measures will decrease the occurrence of orthostatic hypotension. The use of thigh-high elastic stockings minimizes postural hypotension by decreasing venous pooling when the legs are in a dependent position.[17,22] Slowly elevating the patient to a sitting position and pausing before standing are also recommended. Maintaining proper fluid intake, getting the patient out of bed early in the hospital course, or at least having the patient sit upright daily with legs dangling will also prevent the loss of postural reflexes.[4,22,24]

Deep Venous Thrombosis and Pulmonary Embolism

Stroke patients have a high occurrence of deep venous thrombosis.[26] Pulmonary embolism is responsible for 12 percent of deaths in the first week after stroke.[27] There are several reasons for the high rate of thromboembolic complications after stroke. The diuresis associated with bed rest leads to increased blood viscosity through a decrease in plasma volume. A reduction in venous blood flow and increased venous pooling occur in the paretic limbs of stroke patients and can account for the higher rate of deep venous thrombosis in the weak versus nonparalyzed extremity.[4] Bed rest can also lead to stasis through mechanical compression of leg veins.

Early mobilization, the use of elastic or intermittent compression stockings, institution of range of motion exercises, and the appropriate use of subcutaneous heparin are thought to help prevent deep venous thrombosis.[26] The successful deep venous thrombosis prevention trials with prophylactic administration of subcutaneous heparin and graded compression stockings have been conducted in postoperative patients and in those with nonstroke medical conditions (congestive heart failure, myocardial infarction), but not specifically in stroke patients.[28-30] Nonetheless, it is probable that a similar positive effect of such treatment would result in stroke patients.[26]

Pulmonary Disorders

Atelectasis and Aspiration Pneumonia

Six percent of deaths in the first 2 to 4 weeks after stroke are caused by aspiration pneumonia.[27,31] The stroke patient is at particular risk for this complication because of the common occurrence of many factors: dysphagia, difficulty handling secretions, altered level of consciousness, decreased or absent cough reflex, decreased chest movements on the hemiplegic side, and hospital-acquired changes in oral flora. Bed rest worsens aspiration risk because the supine position results in a less effective cough, impaired clearance of secretions, ventilation-perfusion mismatch, and basilar atelectasis.[4,16] These changes are particularly important in the elderly patient because of an age-related increase in residual volume and decreases in lung recoil and respiratory muscle strength.[32]

Prevention of atelectasis and aspiration pneumonia is achieved by prompt assessment and appropriate management of dysphagia, bedside respiratory care (e.g., incentive spirometry), and attention to proper positioning. Moving the patient to an upright chair position will improve diaphragmatic movement, increase aeration of basal lung segments, and increase arterial oxygen tension (PaO_2).[4] The family physician must also be vigilant about looking for aspiration pneumonia, as its symptoms can be insidious and nonspecific. The first indication of a problem could be a change in cognitive or physical function. Mild tachypnea, without fever, could be the only change in vital signs.

Metabolic and Endocrine Effects

Osteoporosis

Although usually not recognized as clinically important, there are major metabolic and endocrine changes with bed rest and immobility. These changes occur slowly and insidiously and require a prolonged recovery period for reversal.[16] Only the major effects are discussed here.

In studies done on normal men confined to bed, researchers found a 0.9 percent weekly loss of bone density from lumbar vertebrae.[4] Immobility leads to osteoporosis through several mechanisms. First, bony growth is encouraged along lines of bone stress from weight bearing and the normal movement of tendons and ligaments. This fact might account for a greater loss of calcium in weight-bearing bones. Second, immobility is associated with decreased osteoblast and increased osteoclast activity.[4] Elderly patients are particularly at risk for disuse osteoporosis because of an age-related decrease in osteoblast function and gastrointestinal calcium absorption. In women, osteoporosis potential is further heightened by the effects of estrogen loss.[33] For prevention, weight-bearing exercise (e.g., walking) done several times a day could be sufficient to decrease disuse osteoporosis.[4]

Urinary Tract Stones

Hypercalciuria and phosphaturia also occur after 2 to 3 days of bed rest and lead to an increased risk of renal and bladder calculi.[4,8,16] The supine position will decrease urinary drainage from the renal pelvis and ureters, and the combination of hypercalciuria, phosphaturia, and urinary stasis can promote stone formation, which then can serve as a nidus for infection.[16] Prevention of

calculi is through maintenance of adequate fluid intake and, if necessary, urinary acidification.

Musculoskeletal Effects

Deconditioning

Deconditioning has two components: a decrease in muscle strength and endurance and a decrease in cardiopulmonary endurance and exercise tolerance. Bed rest adversely affects both components. The elderly stroke patient confined to bed must combat the effects of the neurologic deficit compounded by the deconditioning effect on impaired and normal limbs and the cardiopulmonary system. After a stroke, the additional disability conferred by deconditioning can produce further loss of ambulation and independence in activities of daily living skills (Table 2) and limit the patient's ability to tolerate rehabilitation exercises.

The classic studies on the cardiovascular effects of bed rest immobility involved conditioned young men.[25] After 4 to 6 weeks of bed rest, these men had an increase in basal heart rate, a decrease in maximal oxygen consumption (VO_2 max) and exercise tolerance, and a blunted stroke volume and cardiac output response to exercise.[4,16,17,19,25] Regarding muscle strength, Müller's studies, which involved bed rest and cast immobilization of limbs, measured a 15 to 20 percent loss of initial muscle strength after the first week of immobilization.[17,23,34,35] Bed rest with limited activity produced a less profound, but still important, loss of strength and endurance, especially in the large weight-bearing lower extremity muscles.[23] Even a 10 percent loss of strength after 1 week of bed rest can be enough to make a marginally independent elderly person dependent in activities of daily living.[24]

Table 2. Functional Status Measures.

Measures of Physical Functioning	Items Included
Activities of daily living	Bathing
	Dressing
	Toileting
	Transfer
	Continence
	Bladder
	Bowel
	Feeding
Mobility	Walks
	Propels wheelchair (as applicable)

Data on the deconditioning effect of bed rest in elders are limited. Studies have shown that as little as 3 to 5 days of bed rest will lead to cardiovascular deconditioning in the aged.[19] This rapid cardiovascular deconditioning is in part due to known age-related decreases in stroke volume and cardiac output response to exercise.[4] Deconditioning is obviously easier to prevent than treat. Patients should sit up in bed as soon as they are neurologically stable, usually within 24 to 48 hours.[35,36] The simple act of daily range-of-motion exercises, both passive and, if tolerated, active assistive, done 5 to 10 minutes a day, can be helpful.[17] Exercise while sitting or standing upright will decrease the loss of aerobic capacity.[4] Bed mobility training and daily weight bearing, through transfers to a chair or commode, can occur as soon as sitting balance is sufficient for an upright posture.[24] Basic activities of daily living should be encouraged.[24]

Contractures

Many factors are responsible for the development of joint contractures in the early poststroke phase, including immobility, weakness, cognitive and perceptual deficits, and neglect of range-of-motion joint maintenance. In addition to cosmetic alterations, joint limitations are painful and time consuming to reverse and interfere with positioning, thereby increasing the risk of skin breakdown. Contractures also affect a patient's independence by limiting activities of daily living and mobility skills.[23,37]

An immobile joint will develop a contracture within 3 to 4 weeks; if the joint is injured, important loss of range can occur in as little as 2 weeks.[38] From both a time and cost perspective, prevention of contractures is effective. The key to prevention is proper, varied bed positioning and prompt institution of range-of-motion exercises. Stroke rehabilitation texts and articles contain clear pictures of the various bed positions that should be used with the stroke patient during the flaccid muscle phase.[8,37] Proper positioning in the spastic stroke phase is more complex; Bobath's text or hospital physical therapists should be consulted for information.[39] Regarding range-of-motion exercises, Mossman's stroke rehabilitation text has good instructional illustrations.[23,40] Three repetitions of range-of-motion movement at each joint, done

twice a day, should be sufficient to prevent contractures.[8,38]

Neuropsychiatric Effects

Sensory Deprivation and Depression

The effects of sensory deprivation are well described in a variety of populations, including prisoners in solitary confinement, healthy young subjects in experimental settings, and hospitalized patients.[4] Affective changes and perceptual distortions have been recognized with social isolation alone; when immobilization is combined with social isolation, cognition is also affected.[19] Hospitalized elderly stroke patients are at particularly high risk for sensory deprivation, as they are often immobile and cognitively impaired and could have premorbid visual impairment, auditory deprivation, and other pre-existing immobilizing conditions. Because of the stroke, the patient's communication abilities might be impaired, and staff could inappropriately limit attempts at conversation. Additionally, the sterile hospital environment provides little sensory stimulation. As a group, therefore, aged stroke patients are at great risk for the effects of sensory deprivation. Sensory deprivation will precipitate or worsen depression and cause anxiety, confusion, and noncompliant behavior.[19] Each could lead to iatrogenic complications as the physician attempts to treat these behaviors.

Prevention of sensory deprivation is aimed at increasing patient interaction with staff and family through attempts at communication and participation in activities of daily living. It is imperative to maintain communication with the patient, whether it be verbal, gestural, or through pictures. Also, the placement of orienting information, such as calendars, clocks, and family pictures, and the provision of necessary glasses and hearing aids are helpful.[23]

Rehabilitation Potential

A common therapeutic consideration in the aged stroke patient is discharge to a hospital-level rehabilitation program. For the appropriate patient, the comprehensive, interdisciplinary team approach of the rehabilitation unit provides the best chance of functional improvement. Immobility-related disability can be so important that an otherwise appropriate stroke patient might become unable to take advantage of a hospital-level rehabilitation program. For example, bed-rest–related deconditioning can limit exercise tolerance to such an extent that a stroke patient might not tolerate the level of exercise required in a hospital-level rehabilitation program. Also, immobility-related medical problems, such as deep venous thrombosis and decubiti, can prolong the acute hospitalization and delay rehabilitation unit admission. Because the majority of neurologic recovery and maximal functional improvement generally occur in the first few months after stroke,[8,19,41] a delay in intensive rehabilitation could result in irretrievably lost function. Consequently, although a formal rehabilitation program begins after acute hospital discharge, rehabilitation potential can be maximized through prompt development of a preventive plan for the adverse consequences of immobility.

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References

1. Hayes SH, Carroll SR. Early intervention care in the acute stroke patient. Arch Phys Med Rehabil 1986; 67:319-21.
2. Feigenson JS. Stroke rehabilitation: effectiveness, benefits, and costs. Some practical considerations. Stroke 1979; 10:1-4.
3. Hunt TE. Rehabilitation of the elderly. In: Reichel W, editor. The geriatric patient. New York: HP Publishing, 1978:172-80.
4. Harper CM, Lyles YM. Physiology and complications of bed rest. J Am Geriatr Soc 1988; 36: 1047-54.
5. Lazarus BA, Murphy JB, Coletta EM, McQuade WH, Culpepper L. The delivery of physical activity to hospitalized elderly patients. Arch Intern Med 1991; 151:2452-6.
6. Sine RD. Pressure sores: development, pathogenesis, prevention and treatment. In: Sine Robert D, Liss SE, Roush RE, Holcomb JD, editors. Basic rehabilitation techniques. Germantown, MD: Aspen System, 1977:191-202.
7. Kelly JF, Winograd CH. A functional approach to stroke management in elderly patients. J Am Geriatr Soc 1985; 33:48-60.
8. DeLisa JA, Mikulic MA, Melnick RR, Miller RM. Stroke rehabilitation: part II. Recovery and complications. Am Fam Physician 1982; 26:143-51.
9. Fenske NA, Lober CW. Skin changes of aging: pathological implications. Geriatrics 1990; 45: 27-35.

10. Goode PS, Allman RM. The prevention and management of pressure ulcers. Med Clin North Am 1989; 73:1511-24.
11. Kosiak M, Kottke FJ. Prevention and rehabilitation of ischemic ulcers. In: Kottke FJ, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 4th ed. Philadelphia: W.B. Saunders, 1990:976-87.
12. Itoh M, Lee MH. The epidemiology of disability as related to rehabilitation medicine. In: Kottke FJ, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 4th ed. Philadelphia: W.B. Saunders, 1990:215-33.
13. Allman RM. Pressure ulcers. In: Hazzard WR, Andres R, Bierman EL, Blass JP, editors. Principles of geriatric medicine and gerontology. 2nd ed. New York: McGraw Hill, 1990:1204-11.
14. Smith DM, Winsemius DK, Besdine RW. Pressure sores in the elderly: can this outcome be improved? J Gen Intern Med 1991; 6:81-93.
15. Treatment of pressure ulcers. Med Lett Drugs Ther 1990; 32:17.
16. Halar EM, Bell KR. Rehabilitation's relationship to inactivity. In: Kottke FJ, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 4th ed. Philadelphia: W.B. Saunders, 1990:1113-33.
17. Vallbona C. Bodily responses to immobilization. In: Kottke FJ, Stillwell GK, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 3rd ed. Philadelphia: W.B. Saunders, 1982:963-76.
18. Smith RG. Fecal incontinence. J Am Geriatr Soc 1983; 31:694-7.
19. Anderson TP. Rehabilitation of patients with completed stroke. In: Kottke FJ, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 4th ed. Philadelphia: W.B. Saunders, 1990:656-78.
20. Perkash I. Management of neurogenic dysfunction of the bladder and bowel. In: Kottke FJ, Stillwell GK, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 3rd ed. Philadelphia: W.B. Saunders, 1982:724-45.
21. Borrie MJ, Campbell AJ, Caradoc-Davies TH, Spears GF. Urinary incontinence after stroke: a prospective study. Age Ageing 1986; 15:177-81.
22. Rosenthal MJ, Maliboff B. Postural hypotension: its meaning and management in the elderly. Geriatrics 1988; 43:31-4,39-42.
23. Mossman PL. A problem oriented approach to stroke rehabilitation. Springfield, IL: Charles C Thomas, 1976.
24. Hoenig HM, Rubenstein LF. Hospital associated deconditioning and dysfunction. J Am Geriatr Soc 1991; 39:220-2.
25. Taylor HL, Henschel A, Brozek J, Keys A. Effects of bed rest on cardiovascular function and work performance. J Appl Physiol 1949; 2:223-39.
26. Prevention of venous thrombosis and pulmonary embolism. NIH Consensus Conference. JAMA 1986; 256:744-9.
27. Caplan LR, Stein RW. Stroke, a clinical approach. Stoneham, MA: Butterworth Publishers, 1986.
28. Fasting H, Andersen K, Kraemmer-Nielsen H, Husted SE, Koopmann HD, Simonsen O, et al. Prevention of post operative deep venous thrombosis. Low-dose heparin versus graded pressure stockings. Acta Chir Scand 1985; 151:245-8.
29. Allan A, Williams JT, Bolton JP, LeQuesne LP. The use of graduated compression stockings in the prevention of postoperative deep venous thrombosis. Br J Surg 1983; 70:172-4.
30. Lawrence D, Kakkar VV. Graduated, static, external compression of the lower limb: a physiological assessment. Br J Surg 1980; 67:119-21.
31. Elliot JL. Swallowing disorders in the elderly: a guide to diagnosis and treatment. Geriatrics 1988; 43:95-100,104,113.
32. Tockman MS. Aging of the respiratory system. In: Hazzard WR, Andres R, Bierman EL, Blass JP, editors. Principles of geriatric medicine and gerontology. 2nd ed. New York: McGraw Hill, 1990: 499-508.
33. Kraenzlin ME. Calcium and bone hemostasis with aging. In: Hazzard WR, Andres R, Bierman EL, Blass JP, editors. Principles of geriatric medicine and gerontology. 2nd ed. New York: McGraw Hill, 1990:799-812.
34. Müller EA. Influence of training and of inactivity on muscle strength. Arch Phys Med Rehabil 1970; 51:449-62.
35. Anderson TP. Rehabilitation of patients with completed stroke. In: Kottke FJ, Stillwell GK, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 3rd ed. Philadelphia: W.B. Saunders, 1982:583-603.
36. Rusk HA. Rehabilitation medicine. St. Louis: CV Mosby, 1977.
37. Ellwood PM Jr. Bed positioning. In: Kottke FJ, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 4th ed. Philadelphia: W.B. Saunders, 1990:520-8.
38. Kottke FJ. Therapeutic exercise to maintain mobility. In: Kottke FJ, Stillwell GK, Lehmann JF, editors. Krusen's handbook of physical medicine and rehabilitation. 3rd ed. Philadelphia: W.B. Saunders, 1982:389-402.
39. Bobath B. Adult hemiplegia: evaluation and treatment. 2nd ed. London: William Heinemann Medical Books, 1978.
40. Sharpless JW. Mossman's problem oriented approach to stroke rehabilitation. 2nd ed. Springfield, IL: Charles C Thomas, 1982.
41. Hewer RL. Rehabilitation of stroke. In: Illis LS, Sedgwick EM, Glanville HJ, editors. Rehabilitation of the neurological patient. Oxford: Blackwell Scientific, 1982:153-79.