PREVENTION OF HIP FRACTURES IN THE ELDERLY

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INTRODUCTION

Fracture of the proximal femur is a common, morbid, and costly health problem in the elderly. In 1986, 250,000 patients were admitted to U.S hospitals with hip fractures(1). Because our population is aging, hip fractures will remain a major health issue. It has been estimated that by the year 2040, the number of hip fractures will increase in persons 50 years or older to 512,000(2). The associated cost to care for patients with hip fractures will increase from about $7.2 billion (1984 dollars) to $16 billion in the year 2040(2).

TABLE 1. Annual Costs of Hip Fractures

Direct Costs	
Hospital inpatient services	$1,136,030,000
Outpatient and emergency room services	27,180,000
Physician inpatient services	739,310,000
Physician outpatient services	10,220,000
Other practitioner services	82,450,000
Drugs	3,440,000
Nursing home services	4,001,930,000
Prepayments and administration	270,030,000
Non-health sector goods and services	900,080,000
Total Direct Costs	$7,170,670,000

(Reproduced from Holbrook TL, Grazier K, Kelsey JL, Stauffer RN: The Frequency of Occurrence, Impact and Cost of Selected Musculoskeletal Conditions in the United States. American Academy of Orthopaedic Surgeons, Chicago, 1984, p. 165.)

The risk of hip fracture increases with age. The median age of fracture is 80 years old (3) and it is estimated that 33% of white women and 17% of white men 90 years old have suffered a hip fracture(4). However, many people will not live to such an extreme age and it is useful to consider the life-time fracture risk of a 50 year old white woman and man being 17.5% and 6.0%, respectively (3).

Hip fractures are associated with significant long-term disability(5-9). About 50% of patients have some impairment in physical function a year following fracture. About 9% of previously independent patients reside in a nursing home a year after sustaining a hip fracture(5,10,11). Premorbid physicaland mental function appear to predict post fracture physical function(12,13).

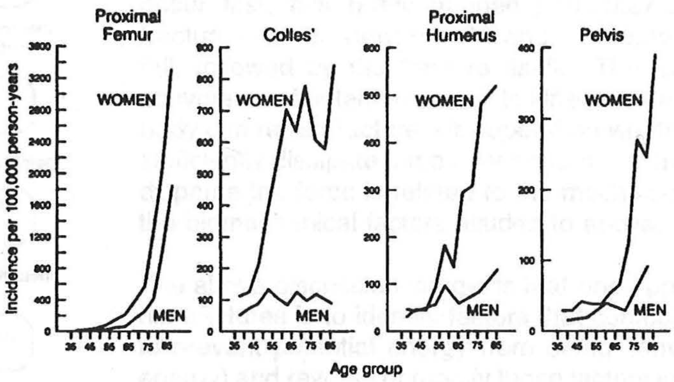


FIG. 1. Age- and sex-specific incidence rates among residents of Rochester, Minnesota, for four age-related fracture sites. (Note that the vertical scale varies for each fracture site.)

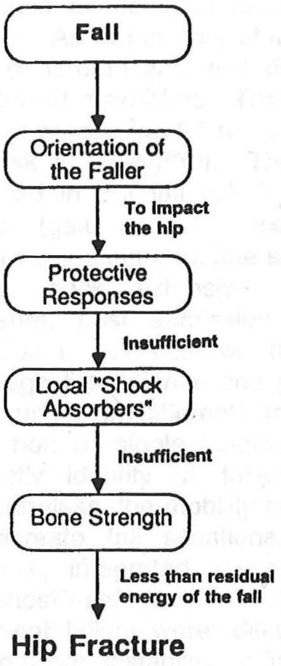
The excess mortality associated with hip fractures occurs during the first six to eight months after fracture before returning to age-and sex-matched norms(6,14). The one-year mortality rate has been reported between 17 to 29%(6-8). In-hospital mortality rates are generally 4 to 10%(6,8,9). Serious comorbid medical conditions and delirium are associated with higher mortality(8).

FACTORS ASSOCIATED WITH HIP FRACTURE

In general, hip fractures occur under circumstances in which a number of different factors, operating together, result in injury. The major proximate factors cited for increased fracture risk in the elderly are falls and reduced bone strength(15). Both factors are critical to understanding the cause and prevention of hip fractures. Moreover, it is important to recognize that falls constitute an important clinical entity by itself.

The aging process is associated with a number of changes that independently increase the vulnerability to fall as well as reducing bone strength (15-26). Such factors include changes in the mechanics of falling, increased number of comorbid conditions associated with risk of falling such as hearing and visual loss, orthostatic hypotension, neurologic diseases and receiving multiple medications. Although reduced bone mineral density at the hip is associated with increase risk of fracture, there is considerable overlap among elderly fallers with and without hip fracture(19,23) (see Fig. 3 on page 5).

In addition to low bone mass, other determinants of bone strength such as accumulated microdamage, age-related osteocyte cell death and architectural weakness such as perforated trabeculae render older individuals more susceptible to fracture(20,21,22).



Overwhelmingly (more than 90%), hip fractures occur following a fall (although some have argued that hip fractures occur first, this rarely happens)(19,20,27,28). Therefore, fractures can be viewed as having two components - first the fall, followed by the fracture itself. The fall results in the conversion of potential energy to kinetic energy. How well the body can resist fracture will depend on whether the body can sufficiently dissipate force over an area or time. The ability to disperse the force is related to the mechanics of the fall and the biomechanical factors alluded to above.

The above discussion suggests that one approach to reduce hip fractures is to identify factors that contribute to falls (how to prevent potential energy from being converted to kinetic energy) and reverse or modify those factors in order to reduce falls and subsequently hip fractures. A number of clinical investigations have been carried out with the goal of identifying fall risk factors.

IDENTIFYING FALL RISK FACTORS

Tinetti, et al. identified risk factors in two populations of older adults, community dwelling elders over 75 years old and residents (mean age 79 years) admitted to an intermediate care facility(16,29). In 336 elderly persons living in the community, 32 percent fell at least once during the year of follow-up with 24 percent of the falls resulting in injury. Table 2 lists the factors that were independently associated with increase risk of falls.

In addition, the risk of fall increased linearly with the number of risk factors. Furthermore, although the study design precluded the determination of environmental factors as risk factors, acute medical, activity-related, and environmental factors were identified with 82% of the falls. The most common environmental contributor was tripping over an object. Medical problems included pneumonia and dizziness.

In 79 consecutive elders residing in an intermediate care facility, nine risk factors were identified as being associated with falling ($p < 0.05$). As in the prior study, a fall risk index was generated and was depen- dent on the number of disabilities. The fall risk index was able to predict the 25 recurrent fallers (but not the risk of injury)(30). The nine risk factors included in the fall risk index were: mobility score (gait and balance), distant vision (Snellen), hearing, morale score, mental status score, back extension, orthostatic blood pressure, post admission medications, and admission activities of daily living score. Although the balance and gait tests were the most useful in differentiating recurrent fallers from non or single fallers, it was unable to correctly identify all fallers (see Table 3). Nevertheless, the mobility test offers the ability to recreate fall situations, and provides a dynamic, integrated assessment of mobility. The most useful items on the test to identify recurrent fallers were: difficulty with rising and sitting down, instability on first standing,

TABLE 2. Risk Factors for Falls*

Risk Factor	Adjusted Odds Ratio	95% CI
Use of sedatives	28.3	3.4–239.4
Cognitive impairment	5.0	1.8–13.7
Lower-extremity disability	3.8	2.2–6.7
Palmomental reflex	3.0	1.5–6.1
Foot problems	1.8	1.0–3.1
No. of balance-and-gait abnormalities		
0–2	1.0	—
3–5	1.4	0.7–2.8
6–7	1.9	1.0–3.7

*CI denotes confidence interval. Adjusted odds ratios were obtained from multiple logistic-regression analysis.

(Taken from Tinetti, et al., N Eng J Med, vol. 319, pg. 1705)

TABLE 3. Balance and Gait Problems Associated with Falling

	Recurrent Fallers (n = 25)	Those Who Fell Once or Not at All (n = 54)	Percent with Disability Who Fell/ Percent without Disability Who Fell	
Total mobility score less than 19/28	19 (76)*	9 (17)	68/12†	(5.7)‡
Balance				
Difficulty arising from chair	5 (20)	0	100/27§	(3.7)
Unsafe or unsteady sitting down	14 (56)	9 (17)	61/20**	(3.0)
Balance score less than 10/15	20 (80)	14 (26)	59/11†	(5.4)
Unsteady turning	19 (76)	13 (24)	59/13**	(4.5)
Unstable on first standing	17 (68)	12 (22)	59/16**	(3.7)
Gait				
Step length less than foot length	19 (76)	14 (26)	58/13†	(4.4)
Steps discontinuous	13 (52)	10 (19)	57/21§	(2.7)
Gait score less than 9/13	17 (68)	20 (37)	46/19††	(2.4)
Poor endurance	17 (68)	20 (37)	46/19††	(2.4)

* Numbers in parentheses are percentages.
† $p \leq 0.0001$, chi-square.
‡ Relative risks are in parentheses.
§ $p \leq 0.005$, chi-square.
** $p \leq 0.001$, chi-square.
†† $p \leq 0.05$, chi-square.

staggering on turning, and short discontinuous steps.

These studies support the general notion that the etiology of falls in the elderly is multifactorial and the more disabilities present, the greater the risk of falling. It follows that if risk increases with the number of risk factors, by modifying and eliminating as many risk factors as possible, we may be able to decrease falls and possibly fractures.

FALL AND FRACTURE RISK

Since only 5% of falls result in hip fracture, several investigators have tried to determine the fall factors which contribute to fracture risk. In a case-control study(287) of 174 patients (mean age 80) admitted for hip fracture after a fall, cases were more likely to have impaired neuromuscular function (specifically lower-extremity dysfunction), neurological conditions (stroke and Parkinson's disease), impaired distant vision, and used long-acting barbiturates. Increased body mass was associated with a significant reduction in the relative risk for hip fracture.

The same investigators(31) carried out a case-control study of hip fracture in black women. Lower-limb dysfunction and need for ambulatory aids, history of stroke, and heavy daily alcohol consumption (at least 7 drinks a day) were associated with increased risk of fracture. Long-acting psychotropic drugs showed a trend toward increased risk (95% confidence interval 0.8 to 9.4), but fell out under multivariate analysis. Reduced body mass index was strongly associated with fracture risk. Consistent with other case-control studies, it appeared that longer duration of estrogen use and younger age of usage was associated with a reduced risk for hip fracture.

A large case-control study(32) found an increased risk of hip fracture associated with long acting hypnotics-anxiolytics, tricyclic antidepressants and antipsychotics but not for short-half life hypnotics-anxiolytics. The authors suggested that psychomotor impairment due to sedative effects and the alpha-adrenergic blockade, which may increase the likelihood of orthostatic hypotension, is the mechanism of increasing the possibility of a fall resulting in hip fracture. No details regarding the frequency of falls were reported for either the case or control group. However, the association of hypnotics with hip fractures has not been consistent across all studies(33-35).

Nevitt et al(36) reported falling to the side or straight down significantly increased the risk of hip fracture. The report suggests that the direction of the fall influenced the type of fracture but that bone density and age-related postural responses to falls determine whether a fracture occurs. Reduced bone mass decreases bone strength whereas the diminished reflexes or limb strength may impair the ability to break a fall and thereby reduce the impact of a force on the hip. These factors are particularly important since a fall from a standing height has the potential energy to fracture even a normal hip(37).

The importance of fall mechanics was further supported by Hayes et al(38) by finding that hip fracture was more likely when the fall impact was near on the hip. Body mass index was inversely related to fracture. These findings suggest that the kinetic energy of a fall is less likely to be adequately dissipated if there is the force of impact is directly over the hip and there is reduced soft tissue at the site of impact. Moreover, Greenspan et al.(19) compared the femoral neck bone density and fall characteristics in fallers with hip fractures to fallers without hip fractures. They found the fall direction, height of fall (potential energy), body mass index and femoral bone mineral density were all independent risk factors for hip fracture (see Table 4).

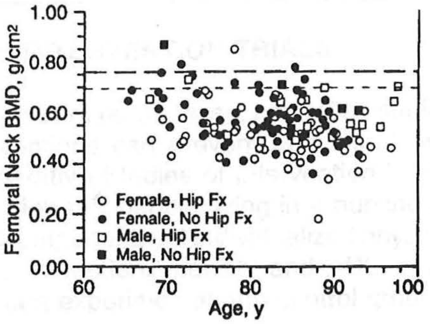


FIG. 2 –Femoral neck bone mineral density (BMD) (g/cm^2) vs age (years) in fallers with and without hip fracture (Fx). The lines represent 2 SDs less than peak bone mass (theoretical fracture threshold) for women (dots and lower dashed line) and men (boxes and upper dashed line). Mean (±SD) femoral neck peak bone mass for women, 0.895 (±0.100) and for men, 0.979 (±0.110) g/cm^2.

TABLE 4. Multiple Logistic Regression of Factors Associated With Hip Fracture

Factor	Adjusted Odds Ratio	95% Confidence Interval	β (SE)	P
Fall to side	5.7	2.3-14	1.75 (0.46)	<.001
Femoral neck bone mineral density, g/cm²*	2.7	1.6-4.6	−10.0 (2.76)	<.001
Fall energy, J†	2.8	1.5-5.2	0.0062 (0.0020)	<.001
Body mass index, kg/m²*	2.2	1.2-3.8	−0.171 (0.063)	.003

*Calculated for a decrease of 1 SD.
†Calculated for an increase of 1 SD.

To summarize, although hip fractures in the elderly are related to low bone density (osteoporosis), there are other important factors that are associated with fracture. This is supported by a number of observations. Epidemiological studies demonstrate a disparity between the peak age of radial and hip fractures (both considered osteoporotic fractures) (see Fig. 3). Furthermore, over 90% of fractures are associated with falls yet only about 5% of falls result in hip fractures. In vitro data suggests that the potential energy from a standing height is adequate to fracture even normal bone. Lastly, there is evidence that direction of fall is associated with hip fracture independent of bone mass. These factors suggest, in addition to osteoporosis and falls, that age-related changes in fall mechanics such as decrease in gait

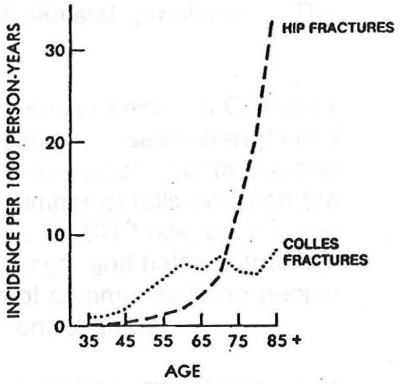


FIG. 3 Age-specific incidence of hip and Colles' (distal forearm fractures among white women. Adapted from Riggs & Melton.

speed, step height, reduced righting reflexes, decreased ability to break a fall such as with an outstretched arm or grabbing a wall all significantly contribute to the propensity to fracture.

TABLE 5. Factors Associated with Hip Fractures

Lower extremity dysfunction	Sedative agents - particularly long-acting
Neurologic disease	Inverse relationship to BMI
Impaired distant vision	Direction of fall
Low bone mass	

FALL INTERVENTION TRIALS

A number of randomized, controlled studies have been carried out to determine if specific interventions can prevent falls. Mulrow et al(39), as part of the Frailty and Injuries: Cooperative Studies of Intervention Techniques (FICSIT trials) studied a group of very frail, high risk people living in a nursing home. The experimental intervention utilized a multicomponent, yet individualized physical therapy regimen aimed at improving strength, range of motion, mobility and ADL. There was no difference in the number of falls between experimental and control groups (79 and 60 falls, respectively, P=.11).

In a randomized, controlled design, Rubenstein(40) and colleagues provided a post-fall assessment of ambulatory elderly persons who lived in a residential care facility and had fallen. The assessment was given to the patients' primary care physician. Included in the assessment were the probable cause of the fall, identified risk factors, and therapeutic recommendations were made such as physical therapy and medication adjustment. Patients receiving the falls assessment had no difference in subsequent falls compared to the control group. Because recommendations were given to the primary care providers, it is could not be determined if the recommendations were actually followed.

A study conducted in England assessed the effect of a multidimensional home assessment of people over the age of 70 years(41). The intervention included an assessment of nutritional, environmental, and medical problems as well as the use of a physiotherapist for assessment and therapy for musculoskeletal problems. The intervention had no effect on fall or fracture rate.

A community-based trial of relatively healthy attendants of a senior center in California received a standard program of low-intensity exercises intended to increase strength and balance and a class on preventing falls(42). Although the 1-year incidence of falls in this population was high (39%), there was no difference in the number of falls between the control and experimental group. Hornbrook(43), as part of the FICSIT trials, determined that a program of low intensity exercises such as walking, strength and balance training, and home safety improvements in a basically healthy group of community living people over 65 years of age did not decrease falls compared to a control group.

Two recent studies have shown a reduction in falls following specific interventions. Both were part of the FICSIT trials. The Atlanta FICSIT study compared the effectiveness of balance training by comparing three groups of relatively healthy community dwelling elders(44). One group used a balance platform, a second group was instructed in Tai Chi (a Chinese exercise regimen to train balance)(45) and a third control group simply attended an educational discussion group. The results of the study showed that subjects in the balance platform and control groups had the same rate of falls during a one-year follow-up. However, the Tai Chi group experienced statistically significant fewer falls with an adjusted risk reduction of about 40%.

The Yale FICSIT trial(46) compared a targeted multifactorial intervention to a control group receiving usual health care plus social visits. Subjects were members of a large HMO. The major outcomes were to find if the experimental intervention would reduce both the proportion of people who fell and the rate of those that did fall.

Their hypothesis was generated from epidemiologic studies which found that the risk of falling increases almost linearly with the number of risk factors that people possess. This relationship is probably because risk is related to a variety of impairments that impedes the person's ability to compensate. Therefore, if the risk of falling increases with the number of risk factors, can the overall risk be reduced if these factors are identified, and interventions instituted to modify and/or eliminate as many risk factors as possible?

The subjects were over 70 years of age, non-institutionalized, cognitively intact, (-MMSE > 22 needed to carry out these exercises independently,) not terminally ill, not too physically active (defined as no regular exercise) and possessed at least one targeted risk factor.

The intervention took place in the patient's home. A study nurse assessed postural blood pressure, and reviewed medications and a list of environmental hazards. A physical therapist assessed the patient's arm and leg strength, balance and transfer impairments, gait and foot problems. The specific intervention given was related to the individual's identified risk factors. Possible interventions included medication adjustments, recommended behavioral changes, gait and transfer training. All medication changes were done in concert with their primary care physician.

EXAMPLES OF YALE FICSIT INTERVENTIONS:

- For those on sedatives, they tried to taper off the sedatives
- Postural hypotension - tried to eliminate contributing drugs
- Behavioral strategies included sleep hygiene, sleep restriction therapy (attempt to substitute behavioral treatments for sedatives to manage sleep problems
- Postural adjustments for people with postural hypotension
  hand and dorsiflexion exercises before they get up, raising the head of the bed, and slow position changes.
- Those with gait and transfer problems were trained with more effective gait and transfer patterns appropriate assistive devices.
- Other components of intervention included:
- Progressive exercises for those with balance and strength problems, exercises were competency based, as they achieved a certain level, they were given a set of exercises that were progressively more difficult. Balance exercises included progressively doing movements that challenged there support system and required them to do maneuvers with decreasing base of support.

After one year, there was a statistically significant reduction in the number of falls in those receiving the intervention. For the 147 subjects randomized to the targeted intervention, 35% suffered at least one fall compared to 47% in the control group (144 subjects). The fall rate (or number of falls per person year) was .63 for the experimental group compared to 1.3 for controls. This computes to a 25% reduction in the proportion of people who fell or a 44% reduction in the rate of falling. The largest change in specific risk factors was in reducing those with more than 3 prescriptions, unsafe tub/toilet transfers, gait impairment, and balance.

This study was too small to have the statistical power to assess the impact of the intervention on fractures or injurious falls, although the trend was in the predicted direction. It was estimated that 5000 subjects would be required to address these outcomes.

In summary, there have been 7 interventional trials of which 2 were shown to be effective in reducing falls. In a study of healthy community dwelling elderly, Tai Chi has shown promise in reducing falls. This rather eloquent means of balance training was superior in reducing falls compared to a group using a balance platform or placebo group. The second (Yale FICSIT) successful intervention utilized the multiple risk factor reduction strategy in a less healthy community group. Interventions for nursing home residents have been unsuccessful in reducing falls. The reason for this may be that the patients are simply too frail or the right approach has yet to be determined. In the community setting, a falls assessment and consultation without assuring that recommendations are carried out is ineffective as is non-targeted, low intensity exercise, and behavioral programs.

ASSESSING FALL RISK AS PART OF THE PRIMARY CARE OF ELDERLY PATIENTS

A number of formal assessment tools are available for the assessment of mobility and fall risk(47). The non-selective use of such tools for the busy clinician would be too time consuming. However, a brief history and abbreviated performance oriented assessment of mobility is an effective way to evaluate and screen patients who are at risk for falling(48).

The medical history and exam should include questions/examination that help identify patients at risk for falling such as: a history of falls, visual or hearing loss, sedative medication use, postural hypotension, number of medications (particularly more than 3) and a simple and rapid screen of mobility and balance. The latter can be performed by observing the patient getting up from a chair, standing, walking ten feet, turning and returning to the chair and sitting down. For those who have problems or instability in carrying out these tasks, they will require a more targeted exam to help establish a diagnosis(49)(See Table 6). In addition to intrinsic factors related to falling, the patient and/or family should be counseled regarding home safety(49-51) (see Table 7 and Appendix 1).

TABLE 6. Elements in the Assessment of Balance and Gait

Abnormality	Possible Diagnoses	Rehabilitative or Environmental Interventions
Balance		
Difficulty in getting up from and sitting down in chair	Myopathy; arthritis; Parkinson's syndrome; postural hypotension; deconditioning	Exercises to strengthen lower extremities; transfer training; high firm chairs with arms; raised toilet seats
Unsteadiness during neck turning and extension	Cervical degenerative disorder (e.g., arthritis spondylosis)	Neck exercises; cervical collar; appropriate storage of items in kitchen and bedroom
Unsteadiness after nudge on sternum	Parkinson's syndrome; normal-pressure hydrocephalus; other central nervous system disease; back problems	Balance training; back exercises; obstacle-free environment; appropriate walking aid; night light
Gait		
Decreases step height	Central nervous system disease; multiple sensory deficits (visual, vestibular, proprioceptive); fear of falling	Careful sensory evaluation; gait training; proper footwear; appropriate walking aid; low pile carpet or nonskid floor without throw rugs
Unsteadiness on uneven surfaces	Decreased proprioception; ankle weakness	Gait training; appropriate footwear; appropriate walking aid; avoidance of thick carpet
Unsteadiness while turning	Parkinson's syndrome; multiple sensory deficits; cerebellar disease; hemiparesis; loss of visual field	Gait training; proprioceptive exercises; appropriate walking aid; obstacle-free environment
Increased path deviation	Cerebellar disease; multiple sensory deficits; sensory or motor ataxia	Gait training; appropriate walking aid

(From Tinetti and Speechley, NEJM, April 20, 1989, pg. 1058)

TABLE 7. Environmental Factors Affecting the Risk of Falling in the Home

Environmental Area or Factor	Objective and Recommendations
All areas	
Lighting	Absence of glare and shadows; accessible switches at room entrances; night light in bedroom, hall, bathroom
Floors	Nonskid backing for throw rugs; carpet edges tacked down; carpets with shallow pile; nonskid wax on floors; cords out of walking path; small objects (e.g., clothes, shoes) off floor
Stairs	Lighting sufficient, with switches at top and bottom of stairs; securely fastened bilateral handrails that stand out from wall; top and bottom steps marked with bright, contrasting tape; stair rises of no more than 6 in.; steps in good repair; no objects stored on steps
Kitchen	Items stored so that reaching up and bending over are not necessary; secure step stool available if climbing is necessary; firm, nonmovable table
Bathroom	Grab bars for tub, shower, and toilet; nonskid decals or rubber mat in tub or shower; shower chair with hand-held shower; nonskid rugs; raised toilet seat; door locks removed to ensure access in an emergency
Yard and Entrances	Repair of cracks in pavement, holes in lawn; removal of rocks, tools, and other tripping hazards; well-lit walkways, free of ice and wet leaves; stairs and steps as above
Institutions	All the above; bed at proper height (not too high or low); spills on floor cleaned up promptly; appropriate use of walking aids and wheelchairs
Footwear	Shoes with firm, nonskid, nonfriction soles; low heels (unless person is accustomed to high heels); avoidance of walking in stocking feet or loose slippers

(From Tinetti and Speechley, NEJM, April 20, 1989, pg. 1058)

AGING AND BONE QUALITY

Osteoporosis has been described as a disease in which low bone mass results in reduced bone strength rendering bone susceptible to fracture (mechanical failure) under normal or minimal forces. The level of bone mass at which susceptibility to fracture increases has been referred to as the fracture threshold. The remaining bone, although reduced in quantity, has typically been described as normal. However, this model of osteoporosis has a number of shortcomings. For example, a review of case-control studies(23) comparing bone mass and hip fracture found that patients with fractures did not appear to be distinctly more osteopenic than persons of similar age without fracture. In another study, reduced bone density remained a statistically significant factor for hip fracture but considerable overlap existed in patients without fracture(19), similar findings are found when lumbar bone mineral density is compared with age- and sex-matched controls with and without fracture. In addition, some interventional studies have found that favorable increases in bone mass failed to reduce fracture rate(52). Furthermore, epidemiological studies show a long latency period between the age-related decline in bone mass and the increase in hip fracture rates. Although some of this disparity can be attributed to falls and other non-osseous age-related changes, the increase in hip fractures after age 80 years does not seem to be solely accounted for by falls or declining bone mass(53). These observations imply that a variety of factors contribute to bone strength. The term bone quality has been operationally defined as the material, architectural and mechanical characteristics which in addition to bone mass, contribute to bone strength(54). The following is a brief discussion of the relation between bone mass, bone quality, aging and hip fracture.

Bone Mineral Density

Low bone mass is an important contributing cause of hip fractures(19,55,56). Although reduced radial and calcaneal bone density is associated with increased risk of hip fracture, the bone density at the hip is superior to measurements from other sites(57). The greater association of hip density with fracture risk compared to other sites is demonstrated by estimating the lifetime risk of hip fracture in 50 year old women with a radial bone density at the 10th and 90th percentile as 19 and 12 percent, respectively, whereas the same determinations made at the hip would mean a lifetime risk of 25 and 8%(57,58).

TABLE 8. Association Between Bone Density and Hip Fractures

BMD (g/cm²)	Relative risk for hip fracture* (95% CL) Unadjusted	Relative risk for hip fracture* (95% CL) Age-adjusted	Area under ROC curve
Proximal femur			
Total	3·0 (2·3, 4·0)	2·7 (2·0, 3·6)	0·76
Femoral neck	3·0 (2·2, 4·0)	2·6 (1·9, 3·6)	0·76
Intertrochanteric	2·8 (2·1, 3·7)	2·5 (1·9, 3·3)	0·75
Trochanter	3·0 (2·3, 3·9)	2·7 (2·0, 3·6)	0·77
Ward's triangle	3·0 (2·4, 3·9)	2·8 (2·1, 3·6)	0·78
Lumbar spine	1·7 (1·3, 2·3)	1·6 (1·2, 2·2)	0·62
Distal radius	1·8 (1·4, 2·4)	1·6 (1·2, 2·1)	0·66
Mid radius	1·7 (1·4, 2·2)	1·5 (1·2, 1·9)	0·66
Calcaneus	2·3 (1·8, 3·1)	2·0 (1·5, 2·7)	0·70

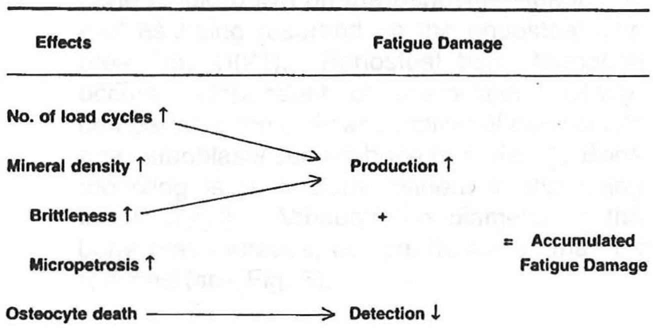
*Relative hazards per SD decrease in bone mineral density (BMD).
All results based on 65 hip fractures that occurred after measurements of hip and spine bone density.

(From Cummings et al, Lancet, vol 341, pg. 73)

Fatigue Damage

All materials subjected to repeated loading are susceptible to fatigue damage. With age, bone is exposed to an increasing number of load-unload cycles which can result in microfractures(21). Because of bone remodeling, microfractures can be repaired and thereby bone strength is maintained. If microfractures accumulate, the structure (bone) could weaken and develop a macrofracture. It has been suggested that with age, microfracture repair becomes impaired perhaps by osteocyte cell death(21). Osteocytes may play a critical role in the influence and control of bone modeling and remodeling. Because osteocytes are directly connected to bone lining cells and other osteocytes via cytoplasmic extensions that pass through morphologic tunnels or canaliculi, they are anatomically well suited to both detect strain and control mechanically related bone modeling/remodeling as well as communicate with other osteocytes and osteoblasts to regulate cellular activity and transformation(22). In addition, there is an increase in the proportion of highly mineralized bone with age leading to increased brittleness, making microfractures more prevalent (high density bone may be more difficult to resorb and therefore repair)(60)(see Fig. 9). Parfitt has suggested that bone age, osteocyte death and fatigue failure are more likely relevant to the pathogenesis of hip fracture than vertebral fractures(61,62). There is some evidence to support an age-related reduction in osteocyte viability in cancellous bone in the femoral head but not lumbar vertebrae(63,64).

TABLE 9. MECHANISMS WHEREBY INCREASED BONE AGE COULD LEAD TO ACCUMULATION OF FATIGUE DAMAGE



(Adapted from Parfitt, *Calc. Tiss. Int*, vol. 53(Suppl)., pg. S83)

Architectural Weakness

Parfitt(65) has described age-related structural changes in bone. Early postmenopausal bone loss is associated with rapid loss characterized by excessive depth of osteoclastic resorption cavities that lead to focal perforation of the trabecular plate leading to discontinuity of the bone structure. In this scenario the bone is likely to be weaker than predicted from bone density measurements because of the trabecular disconnectivity. The therapeutic implications are that if the trabecular lattice is not reconnected in some way, significant increments in bone strength are not likely to take place. A similar process takes place in cortical bone on the endosteal surface by subendosteal perforation and subsequent cortical thinning. In contrast, in late menopause there is reduced bone formation (osteoblast dependent) with inadequate filling of bone cavities causing general thinning of cancellous and cortical structures which is associated with a proportional reduction in bone mass and strength.

With aging there is also an increase in intracortical porosity due to an increase number and mean diameter of haversian systems. The inner cortex (subendosteum) is affected more than the outer cortex, explained on the basis of *Wolff's law which states that the skeleton attempts to adapt its structure to the loads it experiences*(66). How this occurs is not known but working hypotheses are based on bone cells being sensitive to either elastic deformations of bone or some related factor such as local pressures.

Bone Modeling

As we age, there is an increase in the diameter of the diaphysis of long bones as an adaptive response to loads applied to the skeleton. The bone is laid down on the periosteal surface as well as being resorbed on the endosteal side (see Fig. 4)(21). Periosteal bone formation occurs independent of osteoclastic activity, compared to the cellular coupling of osteoclasts and osteoblasts seen in bone remodeling. Bone modeling is particularly evident in the aging femur(67,68). Although the diameter of the bone may increase, cortical thickness may be reduced (see Fig. 5).

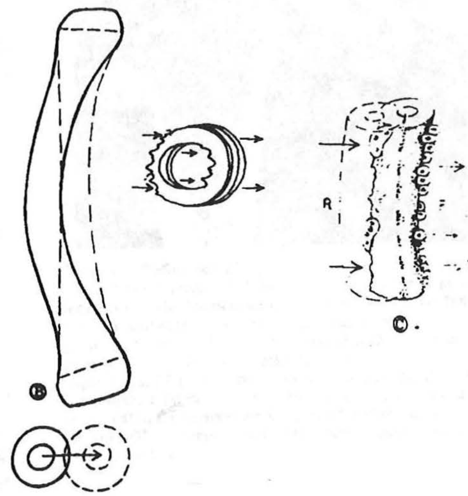
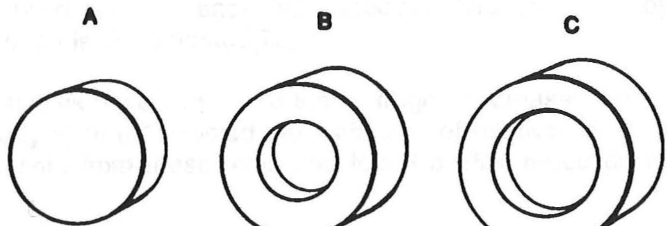


FIG. 4



	A	B	C
AREA (cm^2)	2.77	2.77	2.84
MOMENT OF INERTIA(cm^4)	.61	1.06	1.54
BENDING STRENGTH(%)	100%	149%	193%

FIG. 5 Effect of changes in distribution of cross-sectional area on the area, moments of inertia and bending strength of some regular cylindrical geometries. These idealized geometries reflect the approximate dimensions of the femoral midshaft of the Pecos Pueblo population. The changes between B and C are thus comparable to the geometric remodeling that occurred with aging in that population. From Hayes and Gerhart.

The anatomy of the proximal femur may have particular importance in determining the vulnerability of the femoral neck to fracture(69). This is because bone modeling is dependent on periosteal tissue, yet the femoral neck lacks a periosteum because it stops at the trochanteric line which is where the hip capsule inserts. Therefore, the mechanism for compensatory periosteal appositions is lost at the femoral neck reducing its ability to compensate to loads.

Finally, in addition to the above considerations, a number of recent studies suggest that age-related changes in female femoral neck geometry may be important determinants of hip fracture susceptibility(70-71). For example, Faulkner et al found that hip axis length predicted hip fractures independently of age and bone mineral density in elderly women.

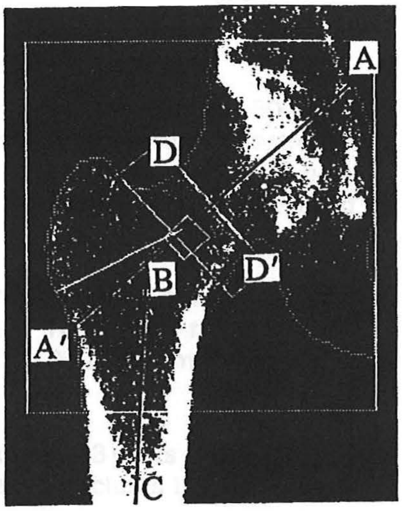


FIG. 6. Definition of geometric measurements from femoral DXA scans. *AA'*, hip axis length, defined as the length along the femoral neck axis as defined by the DXA analysis software, from below the lateral aspect of the greater trochanter, through the femoral neck, to the inner pelvic brim. Angle *ABC*, neck/shaft angle, defined as the angle formed between the femoral neck and the shaft of the femur. *DD'*, neck width, defined as the shortest distance within the femoral neck region of interest, as defined by the DXA analysis software, perpendicular to the femoral neck axis.

DRUG INTERVENTIONS FOR THE PREVENTION OF HIP FRACTURES

Estrogen

The antiresorptive action of estrogen on bone in postmenopausal women is well established(72-73). Estrogen replacement therapy prevents bone loss following menopause and the effect seems to persist as long as therapy continues. The minimum anti-resorptive dose of estrogen is 0.625 mg of conjugated estrogen daily. Transdermal estrogen (estradiol) at a dose of 0.05 mg daily(74,75) also effectively inhibits postmenopausal bone loss. There is no evidence that a specified duration of therapy will prevent bone loss once estrogen is discontinued(76).

A number of epidemiologic studies have suggested that estrogen decreases the risk of hip fracture (33,77-86). Grady et al (87) pooled the estimates of relative risk in ever-users of estrogen with nonusers from these trials and found a 25% reduction in hip fracture.

However, there are no prospective randomized trials to help us determine if estrogen started in late menopause has a significant impact on reducing hip fractures. Most of the studies that suggest estrogen has a protective effect against hip fractures includes primarily women less than 75 years (79,81,84-86) or 80 years (33,77,80). There are no studies that have shown that estrogens reduce fracture risk in patients over 75 years.

Weiss et al(85) carried out a case-control study of postmenopausal women between 50 to 74 years of age and found that women who had taken estrogens for at least six years and current users had a 50% lower risk of hip and forearm fractures (combined) than those who had not taken estrogens. Less than five years of use was not protective. It is not known which patients may have received estrogen for osteoporosis. The authors calculated that the added incidence of endometrial cancer would be greater than the reduced risk in hip fracture. However, the added risk of uterine cancer should be eliminated by progesterone therapy.

A retrospective case-control study by Johnson(80) in women between 52 and 80 years (30% between 77 and 80) found a trend toward a reduction in hip fracture in estrogen users. They did not control for hysterectomy or prior hip fracture, (cases had 7.1% prior fractures compared to 0.6% in the control group) and the small size of the study limited the statistical power of the investigation.

A population-based cohort study from Sweden(86) found that estrogen reduced the risk of hip fractures within the first ten years of menopause. There was no significant reduction in fracture after 65 years of age and only 6% of the 23,246 women were over 70 years old.

Ettinger and associates(82) determined that estrogen started within 3 years of menopause and used for at least 5 years resulted in a decline in vertebral fractures but there were only two hip fractures in each group.

Hammond et al(84) in a 1979 case-control study of 301 patients treated with estrogen and 309 controls found fewer fractures in estrogen users but fracture type was not specified, the average age at the end of follow-up was 56 years, and estrogen users were all white while a third of the control group was black.

Hutchinson(77) compared cases (mean age about 70) admitted to an orthopedic service with hip fracture or distal radial fracture to controls admitted to an orthopedic service for other reasons. Case patients had significantly greater frequency (11.5 vs. 3.2) of alcoholism, and lower mean weight. There was no difference by reviewing the record in estrogen use and hip fracture in controls and cases (7/157 and 10/157 case and controls). However, in a subgroup analysis of only patients interviewed (rather than information solely obtained from the chart) estrogen exposure that began within 5 years of menopause was protective.

Paganinni-Hill and colleagues(33) in a case-controlled study of women (mean age 72 years) living in a retirement community found that estrogen use was associated with a lower hip fracture rate but the protective effect was seen primarily in those who were status post oophorectomy and with usage greater than 60 months (relative risk ratio of .14). Ten years later the same group(78) published their findings from a cohort prospective study involving 8600 women with a mean age of 79 years residing in a retirement community. The rate of hip fractures almost doubled every 5 years between 70 and 90 years. There was a trend for fewer hip fractures in recent estrogen users (RR=0.8, confidence interval crossed 1.0) with declining benefit noted for users who stopped estrogen use 15 or more years ago (RR=1.).

The retrospective cohort study(83) from the Framingham Heart Study found estrogen therapy in women 65 to 74 years old was associated with a 63 percent reduction in hip fracture but only 18% reduction for those over 75 years old. As with the above studies, there was no determination of reason for estrogen use or circumstances of the fracture. Only about a third of women who took estrogen during the first 4 years after menopause continued to take them. Current users had greatest reduction in fractures. The number of women taking estrogen long term was insufficient to determine an effect from prolonged use.

In another study(88) from the Framingham study cohort, women on estrogen therapy for > 7 years had higher hip densities compared to the no therapy group but this effect was no longer present for women over the age of 75 (see Table 10). Only 32% of women in the study took estrogen for this long and information on fractures was not reported.

TABLE 10. Adjusted Bone Mineral Density in Postmenopausal Women, According to the Presence or Absence and Duration of Estrogen Therapy.*

Site	Therapy for ≥7 yr	No Therapy	P Value	Difference in BMD (%)†
Age <75 yr				
No. of women	45	209	—	—
		g/cm²		
Shaft of radius	0.56	0.50	<0.001	11.3
Ultradistal radius	0.28	0.23	<0.001	18.8
Femoral neck	0.78	0.72	0.004	7.6
Ward's triangle	0.64	0.57	<0.001	13.0
Trochanter	0.69	0.62	<0.001	11.5
Lumbar spine	1.15	1.07	0.018	8.0
Age ≥75 yr				
No. of women	24	249	—	—
		g/cm²		
Shaft of radius	0.50	0.46	<0.02	8.5
Ultradistal radius	0.25	0.24	0.44	4.4
Femoral neck	0.70	0.69	0.87	0.1
Ward's triangle	0.56	0.54	0.33	4.4
Trochanter	0.62	0.62	0.75	1.4
Lumbar spine	1.10	1.06	0.32	3.9

*Values have been adjusted for age, age at menopause, weight, height, and cigarette smoking. Women who received therapy for one to six years are not included.

†BMD denotes bone mineral density. The difference shown is between women with seven or more years of therapy and those with no therapy.

Kanis(89) and colleagues conducted a population based case-controlled trial by using a questionnaire to examine drugs affecting bone metabolism and risk of hip fracture in women over 50 years old. Women taking estrogen had a 45% reduction in hip fracture risk. When analyzed according to age, the risk reduction for estrogen was no longer present in women above 80 years old. In the recent study by Greenspan and associates(19) which evaluated the risk of fall severity and bone mass on hip fracture, there was no difference noted in estrogen use in those who fractured a hip compared to controls (mean age 83). In the study by Grisso et al(31) looking at risk factors for hip fracture in black women, estrogen therapy (less than 10% of study participants received ERT) was associated with reduced risk in women below 75 years but not above.

These studies have almost uniformly found that current or recent usage and long duration of therapy correlates with fracture reduction. These findings support the action of estrogen as an antiresorptive agent which is effective in reducing osteoclastic mediated bone resorption thereby reducing bone loss (at all sites) which contributes to the maintenance of bone strength and a reduction in fracture risk. The apparent lack of risk reduction after 75 years is likely because bone loss at this age is estrogen independent(90). Some investigators have suggested that estrogens may reduce hip fractures by having a favorable effect on the processing of sensory information in the central nervous system(91). The findings from the above studies do not refute this theory but they may suggest that beginning estrogen therapy late after menopause may not be able to affect increases in information processing sufficient to have an impact on injurious falls.

The present data suggests that those individuals on estrogen therapy should continue therapy regardless of age. However, there is no good evidence that starting an 80 year old women on estrogen for the first time will reduce her fracture risk.

Calcium, Vitamin D, and Thiazides

There are few well controlled trials that assess the effectiveness of calcium supplementation in the prevention of hip fractures in the elderly. A review of findings from a number of studies, however, suggest that calcium supplementation may have greater beneficial effect on cortical than trabecular bone, a finding that could be important for reducing hip fractures since the femoral neck is made of approximately 70% cortical bone and the intertrochanteric area of the hip is 50% cortical bone compared to 30% cortical bone at vertebral sites(92). Dawson-Hughes et al(93) found that calcium citrate malate reduced bone loss at the spine, femoral neck and radius in normal elderly patients with a low dietary intake of calcium and tended to lower losses at the hip and radius in those with higher dietary calcium. The difference in the latter group was not statistically significant. Calcium carbonate reduced bone loss at the femoral neck and radius in those with low dietary calcium intake. The control group of a recently reported trial using slow-release sodium fluoride were given calcium citrate and showed no change in femoral neck bone density from baseline(94). Chapuy(95) demonstrated that the biochemical evidence of bone abnormalities in elderly patients on a low calcium and vitamin D diet could be reversed by providing adequate calcium and vitamin D supplementation. The same group reported(96) their findings from a randomized trial that fewer hip fractures occurred in elderly women taking calcium and vitamin D supplementation compared to a control group. The patients had low dietary calcium intakes and were probably vitamin D deficient before the trial began. Hip bone density measurements were performed in a subgroup of patients and controls. The placebo group had a larger than predicted fall in hip density than predicted over the 18 month study (4.6%) and the treatment group had a 2.7% rise. Although the patients studied appeared to be frankly calcium and vitamin D deficient (probably osteomalacic), both problems are frequent findings especially in institutionalized elderly. In addition, age-related bone loss is in part mediated via inadequate dietary intake of calcium and/or impaired absorption which results in a negative calcium balance and secondary hyperparathyroidism. The adequate provision of calcium will decrease parathyroid hormone (PTH) release with a subsequent decline in bone resorption. Furthermore, since PTH preferentially effects areas high in cortical bone such as the hip, reducing circulating PTH may lead to a reduction in hip fracture.

In summary, calcium supplementation is an antiresorptive agent which in the elderly acts by primarily reversing the processes which promote secondary hyperparathyroidism. Calcium seems to be more effective in maintaining cortical bone mass and specifically hip density than vertebral bone mass. Calcium citrate may also be useful in maintaining vertebral bone mass in late postmenopausal women with low dietary calcium intake. There is little evidence in elderly women with low dietary intake of calcium and vitamin D, that supplementation with these agents may reduce subsequent hip fracture.

A number of studies have suggested that thiazide diuretics may reduce hip fractures. It is well known that these agents reduce urinary calcium and thereby may improve calcium balance. A retrospective study by Wasnich et al(97) found that hypertensive men (50 to 80 yrs) taking 25 mg of hydrochlorothiazide had higher bone mineral content than hypertensive patients not on thiazides or individuals without hypertension. The groups were matched for age, body-mass index and activity level, and all were comparable. Transbol and associates(98) studied normal early postmenopausal women during a three year placebo controlled trial. Subjects received either 5 mg a day of bendroflumethiazide or placebo. Bone mineral content in the distal forearm initially was maintained in the treatment group but after six months there was no difference in the rate of decline of bone mass in treatment or controls. LaCroix et al(99) prospectively studied the incidence of hip fractures in people over the age of 65 years in three communities. They found that thiazide use was associated with a reduction in the risk of hip fracture during a four year follow-up period. The association held up even after consideration of other risk factors including impaired mobility, low body-mass index and older age. More recently a report from the Study of Osteoporotic Fractures Research Group(100) found that thiazide diuretics had no statistically significant effect on either fall risk or nonspinal fractures. A recent report on risk factors for hip fractures in black women found thiazide to have no protective effect(31).

POST FALL PROTECTION - HIP PROTECTORS

Even with successful strategies to reduce falls and increase bone density, some patients will continue to fall and sustain hip fractures. A number of centers have been developing hip protectors that are designed to attenuate the force the hip is exposed to during a fall and thereby reduce the chance of fracture(101-103). There has been one promising report from a study in Copenhagen(101) in which a group of nursing home residents received a pair of hip protectors (supported by undergarments) to wear over the greater trochanter. Use of the protectors resulted in a 54% reduction in hip fracture risk. During the 11 month study period 8 fractures occurred in the treatment group while the control group had 31. A common problem in this and other studies is maintaining patient compliance(103) which was only 25%, but even with an intention-to-treat analysis they showed significant reduction in fracture incidence. No fractures occurred in the experimental group while they were wearing protectors. Comparable rates of falls were noted in both groups.

CONCLUSIONS

Hip fracture is a common and serious problem in the elderly that will become more prevalent as our population ages. The causes of hip fractures are multifactorial and are related to falls and reduced bone quality. Risk factors for falls have been identified and some evidence now exists that the identification of risk factors and attempts at reversing them may reduce the frequency of falls. The use of estrogen early after menopause helps maintain bone mass and reduces the risk of hip fracture. There is no evidence that initiating estrogen for the first time at an age of highest risk for fractures (over 75 or 80 years) will reduce fractures. Certainly, women at risk for osteoporosis (e.g. low body mass index) should strongly consider initiating estrogen replacement therapy at menopause and they should probably be maintained on replacement therapy indefinitely. Adequate calcium and vitamin D intake is also essential although there is limited evidence at this time that these agents can reduce hip fractures. Calcium seems particularly effective in helping to maintain cortical bone mass which may be especially relevant to the prevention of hip fractures. Woman over the age of 65 years on estrogen replacement therapy should consume at least 1000 mg of elemental calcium daily and those not taking estrogen 1500 mg.

The lack of randomized controlled trials limits our ability to determine what pharmacologic strategies may be effective in reducing hip fractures in high risk groups. In the future, the use of agents that stimulate bone formation may have a role in reducing hip fractures but this will need to be established. The use of hip protectors as a strategy to reduce forces imposed on the hip is an encouraging new strategy particularly for those at greatest risk of fall and hip fracture and where existing interventions have failed. Finally, because the underlying causes of hip fracture are multifactorial, the prevention of these events will ultimately require a variety of solutions, both traditional pharmacologic approaches to improve bone strength and strategies to prevent falls and limit injury.

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APPENDIX 1

HOME HAZARDS

General Household	Correction	Rationale
Lighting		
Too dim	Provide ample lighting in all areas	Increased illumination improves visual acuity
Too direct, creating glare	Reduce glare with evenly distributed light, indirect lighting, translucent shades	
Light switches inaccessible	Should be immediately accessible on entering room	Reduces risk of falling when walking across darkened room
Carpets, Rugs		
Torn	Repair or replace torn carpet	Prevents trips and slips in persons with decreased steppage height
Slippery	Rugs should have nonskid backs; should be tacked down to prevent curling	
Furniture		
Obstructs path	Arrange furnishings so that pathways are not obstructed; avoid cluttered hallways	Aids mobility in persons with impaired peripheral vision
Chairs, Tables		
Unstable	Must be stable enough to support weight of person leaning on table edges or chair arms and backs	Balance-impaired persons use furniture for support
Lack of arm rests	Arm rests should extend forward enough for leverage in getting up or sitting down	Assists persons with proximal muscle weakness
Low-back chairs	High backs provide support for neck and while transferring weight	Parkinson's disease patients often begin rocking movement to assist in getting up; high chair back prevents falling backward

Kitchen	Correction	Rationale
Cabinets, Shelves		
Too high	Keep frequently used items at waist level Install shelves, cupboards at easy-to-reach height	Reduces risk of falling due to frequent reaching or standing on unstable ladders or chairs
Floor		
Wet or waxed	Place rubber mat in sink area Wear rubber-soled shoes in kitchen Use nonslip wax or buff paste wax thoroughly	Prevents slipping, especially if gait-impaired
Gas Range		
Dial difficult to see	Mark "on" and "off" positions on dials clearly	Prevents a fall from being first sign of gas asphyxiation, especially if sense of smell is impaired
Chairs		
Arm rests lacking	Should have arm rests and study legs	Arm rests assist in transfer
Legs unsound	Avoid chairs with wheels	Sturdy, stable chairs do not slide away when transferring
Table		
Wobbly, unstable	Table should have four study of legs of even length Avoid tripod or pedestal tables	Gait-impaired persons often use table for support

HOME HAZARDS (Cont.)

Bathroom	Correction	Rationale
Bathtub		
Slippery tub floor	Install skid-resistant strips or rubber mat Use shower shoes or bath seat	Prevents sliding on wet tub floor If balance is impaired, sitting while showering prevents falls
Side of bathtub used for support or transfer	Use portable grab bar on side of tub	Aids transfers Portable grab bar can be taken along on travels
Towel Racks, Sink Tops		
Unstable for use as support while transferring from toilet	Fix grab rails into wall studs next to toilet	Aids on-off transfer from toilet
Toilet Seat		
Too low	Use elevated toilet seat	Aids on-off transfer from toilet
Medicine Cabinet		
Inadequate lighting Medications improperly labeled	Install brighter lighting All medications should be labeled for internal and external use Keep magnifying glass in or near cabinet	Helps avoid incorrect administration of medication, especially in visually impaired
Door		
Locks	Avoid locks on bathroom doors - or use only locks that can be opened from both sides of door	Permits access by others if fall occurs

Stairways	Correction	Rationale
Height		
Rise between steps is too high	Should be maximum of 6 inches	Reduces risk of tripping for persons with decreased steppage height
Handrails		
Missing	Should be installed and anchored well on both sides of stairway Rail should be cylindrical, and should be placed 1 to 2 inches away from wall	Ease of grasping with either hand
Improper length	Should extend beyond top and bottom step, and ends should turn inward	Signals that top or bottom step has been reached
Configuration		
Too steep Too long	Stairways with intermediate landings are best	Rest stop especially convenient for cardiac or pulmonary patients
Condition		
Slippery	Place nonskid treads securely on all steps	Prevents slipping
Lighting		
Inadequate	Install adequate lighting at both top and bottom of stairway Night lights or bright-colored adhesive strips can be used to clearly mark steps	Outlines location of steps, especially for persons with vision or perception impairments

(Adapted from Tideiksaar, Geriatrics, Vol.41, pp. 27-28)