Falls and syncope

Introduction

Falls by elderly people primarily occur because increasing frailty and comorbidity make avoidance of extrinsic environmental hazards more difficult. The reported incidence of falls varies with the population studied (community dwelling, hospital patients, or in long-term institutions) and the methods of ascertaining falls. There are also important differences in the frequency of falls in relation to age and sex.

The most important problem concerning falls by elderly people is not simply the high incidence, since young children and athletes suffer a higher incidence of falls than all but the frailest older person, but rather the high incidence together with a high susceptibility to injury. This liability to fall-related injury is due to the high prevalence of clinical diseases such as osteoporosis and of age-associated physiological changes such as slow protective reflexes. Under certain circumstances these make even a mild fall dangerous. Other serious consequences include loss of confidence, functional decline, and institutionalization. If falls are to be prevented, attention must be paid to identifying and reducing fall risk factors as well as providing focused and multidisciplinary assessment and diagnosis.

Epidemiology

In a year, 30 per cent of people aged over 65 years in the community fall; 18 per cent fall once and 12 per cent fall twice or more. Incidence rates are up to 41.4 falls per 1000 person-months (O'Loughlin *et al*. 1993). However, annual incidence rates vary considerably in community-based series, ranging from 217 to 1630 per 1000 persons at risk. Similarly, hospital-based surveys report annual incidence rates varying from 380 to 2900 per 1000 persons at risk in acute and chronic medical and geriatric facilities. This figure increases up to annual rates of 3700 per 1000 persons at risk in psychiatric facilities for older people (Robbins *et al*.1989). Fall rates are similarly high in institutions, where 10 to 25 per cent will have a serious fall each year (Rubenstein *et al*.1990). The mean incidence is 1.5 falls per bed per year (range 0.2–3.6 falls) (Rubenstein *et al*. 1994).

The variation in fall frequency rates is primarily attributable to methods of ascertainment; for example, regular chart review in a nursing home setting identifies more fall events than appear in incident reports, and both methods give underestimates compared with self-reports of falls (Kantern *et al*. 1993). Self-reporting is also the most accurate method of data ascertainment for hip, wrist, and humeral fractures—the most important fractures caused by falling.

In comparisons of data from patient interviews, staff questionnaires, medical and nursing notes, and accident report forms, patient and staff versions of in-hospital falls often differ widely. In addition, up to 80 per cent of accidents in hospitals and institutions are unwitnessed. Therefore, where possible, the incidence of falls should be based on self-reported events. Even patient recall is not without error; patients recall falls and injuries during the previous 12 months well, but are less accurate for recall periods of 3 and 6 months.

Although the incidence of home accidents is high, few events are reported to medical services (Graham and Firth 1992). Family practitioners are the main contact for patients who report home accidents, and primary care workers have important opportunities for advising elderly people on the prevention of accidents in the home. Awareness of falls can be increased by asking the patient about falls in the previous year and by documenting all reported and recalled falls.

Most falls occur indoors. In institutions over half of falls occur in bedrooms or bathrooms. Apart from ground frost, there is no significant association between prevailing weather conditions and no seasonal variation. In a large series of over 60 000 consecutive adult attendances at an accident and emergency department (Richardson *et al*. 1997) there was no seasonal variation in the proportion presenting with falls; over 85 per cent of falls presented between 8.00 a.m. and 8.00 p.m.

Definition

A fall is an event whereby an individual comes to rest on the ground or another lower level with or without loss of consciousness. Falls have traditionally been described as extrinsic, due to environmental hazards, or intrinsic, due to age-associated physiological changes and/or clinical disorders. Most falls, particularly of very elderly people, are due to a combination of intrinsic and extrinsic factors. Because presyncope and syncope can cause 'unexplained' falls and because of the frequent overlap of falls and syncope, the presence or absence of loss of consciousness has now been incorporated into the definition.

In patients who are cognitively normal, falls can be classified according to their clinical characteristics. If a patient has tripped or slipped, the fall is 'accidental'. If a patient has fallen and/or lost consciousness for no apparent reason, the episode is described as an 'unexplained' fall. Recurrent falls are defined as three or more falls in the previous 12 months, and intrinsic causes of recurrent and unexplained falls are often similar. Table 1 defines the classification of falls in older adults who are not cognitively impaired.

Causes

There have been many attempts to determine the causes of falling by elderly people. Unfortunately, comparisons between studies are limited because of the differences noted above in the populations examined, the approaches to assessment and investigation, and the definitions of diagnoses.

Multiple pathology and comorbidity are common in later life. There is frequently more than one possible diagnosis for the cause of falls or for a tendency to fall. Therefore it is important to differentiate between an 'attributable diagnosis' and an 'associated diagnosis'. In our studies we have only attributed diagnosis if a specific procedure reproduces symptoms and/or clinical signs or if a specific intervention alleviates the symptoms. If a cause cannot be attributed to a fall, diagnoses are considered to be 'associated'. Rubenstein and Josephson (1996) have reviewed 12 studies, of which six were conducted in institutionalized populations

and six in community-dwelling populations. The fall incidence and the distribution of causes of falls clearly differed between the different populations. Frail high-risk populations had a higher incidence of all types of falls, particularly those related to disease, than was reported in healthier populations (Table 2).

Environment-related falls are more common in community-living populations than in institutions. The most common environmental factors resulting in falls include poor lighting, ill-fitting carpets and rugs, doorsteps, poorly arranged furniture, children's toys on the floor, and lack of support equipment on the stairs or at the bedside. Accidental falls, which account for 25 to 45 per cent of falls in the community, are due to environmental hazards, sometimes complicated by gait imbalance, poor vision, musculoskeletal abnormalities, impaired postural control, impaired postural reflexes, poor memory, or impaired hearing. Ageing can be associated with impaired responses for adapting to sudden changes in orientation. With advancing years there is a reduction in the ability to adapt to sudden horizontal displacement by rapid adjustment of the hip or by stepping backwards or forwards. Falls may be associated with orthostatic changes (getting out of bed, toileting at night, standing at a wash hand basin, or standing after meals), but it is likely that some of these falls are due to a combination of neurocardiovascular instability and environmental hazards.

Gait disorders can be attributed to age-associated physiological changes and various underlying disorders which increase with ageing (e.g. stroke, Parkinson's disease, arthritis). Inactivity can cause significant neuromuscular deconditioning. The prevalence of lower-limb weakness increases with advancing years, and has been reported to affect 48 per cent of elderly people in the community, 57 per cent of people in residential facilities (Tinetti *et al*. 1986), and 80 per cent of nursing home residents (Robbins *et al*. 1989). Most studies report an abnormality of gait or balance in 20 to 50 per cent of older patients, with a higher prevalence in fallers (Nevitt *et al*. 1989; Robbins *et al*. 1989).

Gait and balance disorders are an associated diagnosis in 45 per cent of cognitively normal and almost all cognitively impaired adults attending an accident and emergency department because of a fall. Other associated causes are detailed in Fig. 1 (Shaw and Kenny 1997) and Table 3.

An important intrinsic cause of falls, particularly unexplained falls with or without loss of consciousness, is neurocardiovascular instability. The main clinical diagnoses included in this broad category are carotid sinus hypersensitivity, orthostatic hypotension, vasovagal or neurocardiogenic syncope, and postprandial hypotension.

Overlap with syncope

The traditional definitions of syncope and falls treat them as separate conditions with different aetiologies. More recently, evidence has accumulated of an overlap between them. Separation of falls and syncope into two distinct entities relies on an accurate history of the event and an eye-witness account. These are unavailable for at least one-third of older patients who have experienced falls or syncope. In one study of 354 community-dwelling elderly people, of whom 80 per cent were aged between 60 and 80 years and 92 per cent had normal cognitive function, over one-third did not recall having fallen 3 months after a documented fall event. This was despite the event having been reinforced by a health visitor calling shortly afterwards (Cummings *et al*. 1988). Similarly, witness accounts of syncopal events were only available in 40 to 60 per cent of elderly people attending a syncope clinic with recurrent symptoms (McIntosh *et al*. 1993*a*).

The overlap can be demonstrated in young as well as old subjects. In one study (Lempert *et al*. 1994), syncope was induced in 56 of 59 young healthy volunteers by a sequence of hyperventilation, ortho-static change, and Valsalva manoeuvre. Thirteen subjects (25 per cent) fell but preserved consciousness. One subject had amnesia and unresponsiveness without falling. The remainder lost consciousness, fell, had myoclonic activity, and recalled losing consciousness.

An experimental demonstration of the scope for overlap of falls and syncope can be seen in cardio-inhibitory carotid sinus syndrome. Asystole can be induced in patients with a hypersensitive response by carotid sinus massage during head-up tilting. One-third of patients lose consciousness (as witnessed by laboratory staff) during asystole but deny this afterwards, thus demonstrating amnesia for loss of consciousness (Kenny and Traynor 1991). From a review of three studies which included a total of 109 patients for whom cardio-inhibitory carotid sinus syndrome was an attributable cause of falls, 38 per cent of patients presented with falls alone or falls and dizziness but denied syncope. Of the fallers, 51 per cent demonstrated amnesia for loss of consciousness. Two-thirds of patients with orthostatic hypotension as the attributable cause of symptoms also presented with falls only or with falls and dizziness (Ward and Kenny 1996; Stout and Kenny 1998). Of 169 patients who attended a dedicated syncope and falls clinic, over one-third presented with a history of unexplained falls; two-thirds of these had an attributable cardiovascular diagnosis, of which the majority were carotid sinus hypersensitivity, orthostatic hypotension, cardiac arrhythmia, or vasovagal syncope (McIntosh *et al*. 1993*a*).

Drop attacks

Falls are termed drop attacks when the cause is not certain, the event is unexpected, and there is no apparent loss of consciousness. The initial accounts of drop attacks in elderly subjects were detailed by Sheldon (1960), who noted the dramatic suddenness of the event, without alteration in alertness, an inability to regain upright posture immediately afterwards, and a preponderance of women. Since then, most reports on the epidemiology of falls have included drop attacks as one of the important causes of falls by elderly people.

The incidence of drop attacks increases with age. A survey of 12 studies comprising 3684 falls (Rubenstein and Josephson 1996) estimated that 9 per cent (range 0–52 per cent) were due to drop attacks. In a study of

falls among a random sample of elderly subjects aged over 65 years, the proportion due to drop attacks increased from 2 per cent in those aged between 65 and 74 years to 15 per cent in subjects aged over 90 years (Campbell *et al.* 1981).

Brainstem ischaemia remains the most widely accepted likely pathology for drop attacks, although few studies have reproduced symptoms during ischaemic episodes. Various pathological states which implicate the vertebrobasilar circulation have been causally related to drop attacks. The most important of these are tumours compromising brainstem blood supply, compression of the vertebral arteries by cervical osteophytes in cervical spondylosis, and athero-sclerotic vertebrobasilar insufficiency (Kubala and Millikan 1964). In a large series of 1970 patients with cerebrovascular insufficiency, 8 per cent of 373 with intermittent vertebrobasilar disease presented with drop attacks (Meissner *et al*. 1986).

Amnesia for loss of consciousness may be partially or wholly responsible for syncope presenting as drop attacks in older subjects. The higher incidence with increasing age may be due to altered cerebral autoregulation, reduced cerebral perfusion, and lower thresholds for cerebral hypoperfusion. Absence of a prodrome prior to syncope may further complicate the clinical presentation (Ward and Kenny 1996). The fall in blood pressure may be too fast to produce a prodrome. Interventions which slow down the rate of decline in blood pressure have been shown to convert drop attacks to syncope with a well-recognized prodrome of dizziness (Ward and Kenny 1996).

Transient neuronal dysfunction has also been proposed as a causal factor in some series. Drop attacks in children are typically due to epilepsy, but atypical epilepsy is a rare cause of drop attacks in elderly subjects. Labyrinthine and vestibulo-ocular dysfunction leading to erroneous correction of posture has also been postulated as one of the less likely causes of drop attacks in adults. In the largest series on adult drop attacks (Kubala and Millikan 1964) 64 per cent of 108 cases (mean age 70 years) remained undiagnosed. Most patients who had epilepsy as an attributable cause were aged less than 40. A substantial number of undiagnosed patients had hypertension (41 per cent) and heart disease (25 per cent). This study predated recent publications on the importance of baroreflex control in unexplained falls and standardized tests of baroreflex assessment for patients with drop attacks.

More recently, episodic hypotension and/or bradycardia, mediated by altered baroreflexes, have been reported as common attributable causes of drop attacks in elderly people. These conditions (neuro-cardiogenic syncope, carotid sinus syndrome, and orthostatic hypotension) produce global cerebral ischaemia and may be manifest as drop attacks rather than as the more classical syncope. In a recent prospective series (Dey *et al*. 1996), 35 consecutive elderly patients with recurrent drop attacks were investigated in detail, including cardiovascular testing. A diagnosis was attributed to an abnormality only when symptoms were reproduced during investigation and alleviated after specific intervention. Episodic bradycardia or hypotension was diagnosed in 24 patients, carotid sinus syndrome in 18, orthostatic hypotension in five, and vasovagal syncope in one. Seven of the ten remaining patients for whom no attributable diagnosis was made also had underlying cardiovascular pathology.

Elderly people are particularly prone to syncope because of age-associated physiological changes in cerebral autoregulation, baroreflex sensitivity, intravascular volume regulation, and neurohumoral control. Why elderly patients have amnesia for loss of consciousness is unclear; it may be due to mild cognitive impairment, age-associated impairment of cerebral autoregulation, paradoxical cerebral vasoconstriction during provocative testing, impaired postural control of balance control during mild haemodynamic changes, or other reasons (Lipsitz *et al*. 1985*b*). Syncope and falls are often indistinguishable and are manifestations of similar pathophysiological processes.

Syncope

The epidemiology of syncope in old age has not been well studied and accurate estimates of prevalence and incidence are not available. The elucidation of syncope is often complicated by the presence of multiple disorders that may cause it synergistically and by difficulties in determining a relationship between circumstances, medications, and symptoms. Available data suggest that syncope accounts for 3 per cent of emergency room attendances and 1 per cent of medical admissions to a general hospital (Day *et al*. 1982). In a study of 711 elderly subjects (mean age 87 years) living in a chronic care facility, the prevalence of syncope was reported to be 23 per cent over a 10-year period, with an annual incidence of 6 per cent and a recurrence rate of 30 per cent over a 2-year prospective follow-up (Lipsitz *et al*. 1985*b*). This is undoubtedly an underestimate because falls were excluded.

A cause of syncope could not be determined for over 40 per cent of 210 community-dwelling elderly patients (Kapoor *et al*. 1986). Syncope due to a cardiac cause was associated with higher mortalities irrespective of age. In patients with a non-cardiac or unknown cause of syncope, older age, a history of congestive cardiac failure, and male sex were important predictors of mortality. This study predated the establishment of standardized tests for baroreflex sensitivity, and did not specifically investigate all patients for neurocardiovascular abnormalities.

The temporary cessation of cerebral function that causes syncope results from transient and sudden reduction of blood flow to parts of the brain, such as the reticular activating system of the brainstem, responsible for consciousness. Age-associated physiological impairments in heart rate, blood pressure, and cerebral blood flow, combined with comorbid conditions and concurrent medications, account for the increased prevalence of syncope in elderly subjects. Baroreflex sensitivity is blunted with ageing, manifesting as a reduction in the heart rate response to hypotensive stimuli. Elderly people are prone to reduced blood volume due to excessive salt loss through the kidneys as a result of a decline in plasma renin and

aldosterone, a rise in atrial natriuretic peptide, and diuretic therapy. Low blood volume together with age-associated diastolic dysfunction can lead to a low cardiac output which increases susceptibility to orthostatic hypotension and vasovagal syncope. Cerebral autoregulation, which maintains a constant cerebral circulation over a wide range of blood pressure, is altered in the presence of hypertension and possibly by ageing. As a result, sudden mild to moderate declines in peripheral blood pressure can affect cerebral blood flow markedly. Causes of syncope are detailed in Table 4.

Carotid sinus syndrome

Carotid sinus syndrome is an important but frequently overlooked cause of syncope and presyncope in elderly people. It is characterized by episodic bradycardia and/or hypotension resulting from exaggerated baroreceptor-mediated reflexes or carotid sinus hyper-sensitivity. The syndrome is diagnosed in subjects with unexplained symptoms when 5 s of carotid sinus massage produces asystole exceeding 3 s (cardio-inhibitory), or a fall in systolic blood pressure exceeding 50 mmHg (6.6 kPa) in the absence of cardio-inhibition (vasodepressor), or a combination of the two (mixed).

The afferent limb of the carotid sinus reflex terminates at the nucleus of the tractus solitarius in the medulla. The efferent limb comprises the sympathetic nerves supplying the heart, the vasculature, and the cardiac vagus nerve. Physiological rises in arterial blood pressure generate the stretch necessary to activate the reflex. In health, the carotid baroreceptors in conjunction with those of the aortic arch play a major role in the neural control of blood pressure. In patients with carotid sinus syndrome, baroreflex sensitivity, which normally declines with increasing age, is enhanced compared with age-matched controls (Dehn *et al*. 1984). The site and mechanism of this hyper-sensitivity are not known, but may be central (O'Mahony 1995).

Atropine in a dose up to 700 μg can abolish bradycardia in all patients. Carotid sinus syndrome and sick sinus syndrome are two separate diagnostic entities, although up to 5 per cent of patients with carotid sinus syndrome can have abnormal intrinsic sinus node function (Morley *et al*. 1983). It is possible that carotid sinus syndrome results from a central abnormality of baroreflex gain. The frequent association of carotid sinus syndrome with atherosclerotic comorbidities has led to speculation that cerebral and/or cardiac ischaemia may play an important role in its pathogenesis (Morley *et al*. 1983).

The prevalence of carotid sinus reflex hypersensitivity in asymptomatic individuals is not known. However, it definitely increases with age and is rare in patients with syncope or falls who are aged less than 50 years. Recent studies suggest that it is not a feature of normal ageing. In a study of 25 healthy elderly subjects, none developed diagnostic or symptomatic cardio-inhibition or vasodepression during carotid sinus massage (McIntosh *et al*. 1994*a*). In another series, abnormal cardio-inhibition was reported in 2 per cent of 288 healthy subjects (age range 17–84 years) (Brignole *et al*. 1985). Carotid sinus hypersensitivity was demonstrated in 19 per cent of 1000 patients (aged over 50 years) who presented to an accident and emergency department with 'unexplained falls', and the prevalence increased to 21 per cent and to 35 per cent in fallers aged over 65 and over 80 respectively (Fig. 2) (Richardson *et al*. 1997). Abnormal responses to carotid sinus massage are more likely to be observed in asymptomatic individuals with coronary artery disease and those on vasoactive drugs known to influence reflex sensitivity (digoxin, β-blockers, and methyldopa). The prevalence of drug-induced carotid sinus hypersensitivity among elderly fallers is 11 per cent (Richardson *et al*. 1997). Although it is still generally considered as a rare condition, referral centres which routinely perform carotid sinus massage in all older patients presenting with syncope and unexplained falls diagnose carotid sinus syndrome in up to 45 per cent (McIntosh *et al*. 1993*a*).

Carotid sinus reflex sensitivity is assessed by measuring heart rate and blood pressure responses to carotid sinus massage. In asymptomatic elderly subjects, the normal response is as follows: cardio-inhibition, 1038 ± 195 ms; vasodepression, of 21 ± 14 mmHg (2.8 ± 1.8 kPa) (McIntosh *et al*. 1994*a*).

Carotid sinus massage is a crude and unquantifiable technique and is prone to both intra- and interobserver variation. More scientific diagnostic methods employing neck chamber suction or drug-induced changes in blood pressure can be used for carotid baroreceptor activation, but neither of these techniques is suitable for routine use. A standardized 5- to 10-s stimulus has been accepted by most current investigators as safe and effective. Over 75 per cent of patients with cardio-inhibitory carotid sinus syndrome respond positively to right-sided carotid sinus massage either alone or combined with left-sided carotid sinus massage (McIntosh *et al*. 1993*b*).

Complications resulting from carotid sinus massage include cardiac arrhythmias and neurological sequelae. Fatal asystole or ventricular arrhythmias are extremely uncommon and have generally occurred in patients with underlying heart disease undergoing therapeutic rather than diagnostic massage. Digoxin toxicity has been implicated in most cases of ventricular fibrillation. Neurological complications are also uncommon (0.14 per cent) (Munro *et al*. 1994). In a retrospective analysis of 16 000 episodes of carotid sinus massage (Davies and Kenny 1998), only 12 patients developed neurological complications. These resolved within 24 h in eight patients and within 1 week in two patients; neurological deficits persisted in two patients.

It has been suggested that carotid sinus massage should not be performed in patients with known cerebrovascular disease or carotid bruits unless there is a strong indication. It should also be avoided immediately after myocardial infarction when reflex sensitivity may be increased. If there is a significant hypotensive response during the stimulus, it is prudent to rest patients for at least 10 min before allowing them to stand up for other tests. It is not possible to predict from either clinical characteristics or carotid Doppler studies which patients will develop neurological sequelae.

Carotid sinus syndrome should be diagnosed when carotid sinus hypersensitivity is documented in a patient with otherwise unexplained dizziness, falls, or syncope and in whom carotid sinus massage reproduces

symptoms. As discussed previously, three subtypes of carotid sinus hypersensitivity or carotid sinus syndrome are currently recognized. The independent vasodepressor response can be confirmed by repeating carotid sinus massage after abolishing significant cardio-inhibition using either atrioventricular sequential pacing or intravenous atropine (Walter *et al*. 1978). Asystole exceeding 1.5 s should be considered 'significant' in this regard.

Symptom reproduction during carotid sinus massage was regarded by early investigators as essential in the diagnosis of carotid sinus syndrome, but it is not always justified in patients with reproducibly abnormal responses. Up to 15 to 30 per cent of patients with unexplained falls only have an abnormal response during upright carotid sinus massage (Kenny and Traynor 1991).

In carotid sinus hypersensitivity, recognized triggers for symptoms are head movement, prolonged standing, postprandial state, straining, looking or stretching upwards, exertion, defecation, and micturition. In a significant number of patients no triggering event can be identified. An abnormal response to carotid sinus massage may not always be reproducible necessitating repetition of the procedure if the diagnosis is strongly suspected.

Carotid sinus hypersensitivity is frequently associated with other hypotensive disorders such as vasovagal syncope and orthostatic hypotension (McIntosh *et al*. 1993*b*), indicating a common pathogenic process. Overlap of hypotensive disorders can make an attributable diagnosis difficult to identify, but every effort should be made as interventions may vary and assessment of response depends on the initial diagnosis. Approximately half of patients sustain an injury during symptomatic episodes; fractures, particularly of the femoral neck, had been sustained by 25 per cent of patients in one series (McIntosh *et al*. 1993*b*). In a prospective study of falls in nursing home residents, a threefold increase in the fracture rate was observed in those with carotid sinus hypersensitivity (Murphy *et al*. 1986). Indeed, it can be considered as a modifiable risk factor for fractures of the femoral neck (Ward *et al*., in press). Carotid sinus syndrome is not associated with an increased risk of death. The mortality in patients with carotid sinus syndrome is similar to that in patients with unexplained syncope and the general population matched for age and sex (Brignole *et al*. 1992). Mortality rates are similar for the three subtypes of the syndrome (Brignole *et al*. 1992).

Atrioventricular sequential pacing is the treatment of choice for patients with symptomatic cardio-inhibition (Morley *et al*. 1982). With appropriate pacing, syncope and unexplained falls are abolished in 75 to 90 per cent of patients with cardio-inhibition (Almquist *et al*. 1985). Treatment of vasodepressor carotid sinus syndrome is less successful owing to poor understanding of its pathophysiology. Fludrocortisone, a mineralocorticoid widely employed to treat orthostatic hypotension, is used with good results in the treatment of vasodepressor carotid sinus syndrome, but its longer-term use is limited by adverse effects (da Costa *et al*. 1993).

Orthostatic hypotension

Orthostatic or postural hypotension is arbitrarily defined as a fall of 20 mmHg (2.6 kPa) in systolic blood pressure or 10 mmHg (1.3 kPa) in diastolic blood pressure on assuming an upright posture from a supine position (Mathias and Bannister 1992). Orthostatic hypotension implies abnormal blood pressure homoeostasis and increases in frequency with advancing age. Depending on the methodology used, the prevalence of postural hypotension is found to be between 4 per cent (Lipsitz *et al*. 1985*a*) and 33 per cent (Palmer 1983) in community-dwelling older people. The prevalence and magnitude of falls in systolic blood pressure increase with age and are associated with general physical frailty and excessive mortality (Masaki *et al*. 1995).

The compensatory response to orthostasis involves activation of the sympathetic nervous system and inhibition of the parasympathetic nervous system, and reflects the integrity of the total arterial baroreflex arc. Orthostatic hypotension results from failure of the arterial baroreflex, which is most commonly due to atherosclerosis plus arterial stiffness or disorders of the autonomic nervous system among community-living elderly people (Bannister and Mathias 1992).

The three phases of heart rate and blood pressure responses to orthostasis—an initial heart rate and blood pressure response, an early phase of stabilization, and a phase of prolonged standing—are all influenced by ageing (Wieling *et al*. 1992). The maximum rise in heart rate and the ratio of the maximum to the minimum heart rate in the initial phase decline with age, implying a relatively fixed heart rate unaffected by posture (Wieling *et al*. 1992). Despite a blunted heart rate response, blood pressure and cardiac output are adequately maintained on standing in active, healthy, well-hydrated, and normo-tensive elderly subjects. The underlying mechanism involves decreased vasodilatation and reduced venous pooling during the initial phases and increased peripheral vascular resistance after prolonged standing. However, in elderly subjects with hypertension and cardiovascular disease, who are receiving vasoactive drugs, the circulatory adjustments to orthostatic stress are disturbed, predisposing to postural hypotension.

Hypertension and orthostatic hypotension

Ageing is associated with an increased prevalence of hypertension as well as hypotension. Hypertension itself increases the risk of hypotension by impairing baroreflex sensitivity and reducing ventricular compliance. A strong relationship between supine hypertension and orthostatic hypotension has been reported amongst unmedicated institutionalized elderly subjects (Lipsitz *et al*. 1985*b*) (Fig. 3). Hypertension increases the risk of cerebral ischaemia from sudden declines in blood pressure. Elderly hypertensives are more vulnerable to cerebral ischaemic symptoms, even with modest and short-term postural hypotension, as their range of cerebral autoregulation is set at a higher mean level (Strandgaard 1976). In addition,

antihypertensive agents impair cardiovascular reflexes and further increase the risk of orthostatic hypotension.

Several pathological conditions are associated with orthostatic hypotension. Autonomic failure (Mathias 1995) (Table 5) and drugs (Table 6) are important causes. In a consecutive series of 70 patients with orthostatic hypotension referred to a dedicated syncope facility, the most common attributable cause of hypotension was medication (Stout and Kenny 1998). Establishing a causal relationship between a drug and orthostatic hypotension requires identification of the responsible medicine, abolition of symptoms by withdrawal of the drug, and rechallenge with the drug to reproduce symptoms. Rechallenge is an important step in the diagnosis but is often omitted in clinical practice in view of potential serious consequences. When several drugs are being taken, the situation is complicated by inter-actions. A number of non-neurogenic conditions are also associated with postural hypotension. They include myocarditis, atrial myxoma, aortic stenosis, constrictive pericarditis, haemorrhage, diarrhoea, vomiting, ileostomy, burns, haemodialysis, salt-losing nephropathy, diabetes insipidus, adrenal insufficiency, fever, and extensive varicose veins. In the absence of well-recognized conditions causing primary and secondary autonomic failure (Mathias 1995), ageing can also be considered as a cause.

Primary autonomic failure syndromes

These are discussed more fully in Chapter 18.12. The three main clinical entities are pure autonomic failure, multiple-system atrophy or Shy–Drager syndrome, and autonomic failure associated with idiopathic Parkinson's disease. Pure autonomic failure, a relatively benign entity, was previously known as idiopathic orthostatic hypotension. This condition presents with orthostatic hypotension, defective sweating, impotence, and bowel disturbances. No other neurological deficits are found and resting plasma noradrenaline (norepinephrine) levels are low. Multiple-system atrophy carries the poorest prognosis. Clinical manifestations include features of dys-autonomia and motor disturbances due to striatonigral degeneration, cerebellar atrophy, or pyramidal lesions. Resting plasma noradrenaline levels are usually within the normal range but fail to rise on standing or tilting. The prevalence of autonomic failure in Parkinson's disease is not precisely known. Cerebellar and pyramidal signs are not seen. Orthostatic hypotension in Parkinson's disease may be due to factors other than dysautonomia, for example side-effects of drugs, autonomic neuropathy complicating coexisting diabetes mellitus, and confusion with early multiple-system atrophy with predominant Parkinsonian features (Mathias 1995).

The clinical manifestations of orthostatic hypotension are due to hypoperfusion of the brain and other organs. Depending on the degree of fall in blood pressure and cerebral hypoperfusion, symptoms can vary from dizziness to falls to syncope associated with a variety of visual defects ranging from blurred vision to blackout. Other reported ischaemic symptoms of orthostatic hypotension are non-specific lethargy and weakness, suboccipital and paravertebral muscle pain, low backache, calf claudication, and angina (Bleasdale-Barr and Mathias 1994). Several precipitating factors for orthostatic hypotension have been identified, namely speed of positional change, prolonged recumbency, warm environment, raised intrathoracic pressure (coughing, defecation, micturition), physical exertion, and vasoactive drugs.

Orthostatic hypotension is an important cause of unexplained falls and syncope, accounting for 14 per cent of all diagnosed cases in a large series (Kapoor *et al*. 1986). In a tertiary referral clinic dealing with unexplained syncope, dizziness, and falls, 32 per cent of elderly patients had orthostatic hypotension (McIntosh *et al*. 1993*a*). Irrespective of symptoms, orthostatic hypotension increases the risk of falls in the elderly patients. Nineteen per cent of elderly fallers attending an accident and emergency department had a diagnosis of orthostatic hypotension (Moss *et al*. 1980). Orthostatic hypotension was the cause of drop attacks in 15 per cent of 35 patients aged over 50 years and probably contributed to unexplained falls in a similar proportion of patients.

The diagnosis of orthostatic hypotension involves a demonstration of a postural fall in blood pressure after standing up. Reproducibility of orthostatic hypotension depends on the time of measurement and on autonomic function (Ward and Kenny 1996). The diagnosis may be missed on casual measurement during the afternoon. The procedure should be repeated during the morning after maintaining a supine posture for an adequate period (10 min). Sphygmomanometer measurement is not as sensitive as sophisticated phasic blood pressure measurements, and standing up is as diagnostic as head-up tilting (Ward and Kenny 1996). In patients with unexplained syncope or falls, an attributable diagnosis of orthostatic hypotension depends on reproduction of symptoms where possible.

The aim of therapy for symptomatic orthostatic hypotension is to improve cerebral perfusion. There are several non-pharmacological interventions including avoidance of precipitating factors, elevation of the head of the bed at night, and application of graduated pressure from a support garment to the lower limbs to reduce venous pooling. There are reports suggesting benefit, in a small number of patients, from implantation of cardiac pacemakers to increase heart rate during postural change (Moss *et al*. 1980; Mathias 1995). However, the effects of tachypacing on improving cardiac output in patients with maximal vasodilatation remains conjectural. Many drugs have been used to raise blood pressure in orthostatic hypotension. The most commonly used of these agents are fludrocortisone, midodrine (an α-agonist), and desmopressin. Patients receiving drug treatment for orthostatic hypotension require frequent monitoring for supine hypertension, electrolyte imbalance, and congestive heart failure.

Neurocardiogenic syncope

The hallmark of neurocardiogenic or vasovagal syncope is hypotension and/or bradycardia sufficiently profound to produce cerebral ischaemia and loss of neural function. Vasovagal syncope has been classified

into cardio-inhibitory (bradycardia), vasodepressor (hypotension), and mixed (both) subtypes depending on the blood pressure and heart rate response (Sutton *et al*. 1992). In most patients, the manifestations occur in three distinct phases: a prodrome or aura, loss of consciousness, and a postsyncopal phase. A precipitating factor or situation is identifiable for most patients. These include emotional stress, anxiety, trauma, physical pain or anticipation of physical pain (e.g. before venesection), sight of blood, accident, warm environment, air travel, and prolonged standing. The most common triggers in older individuals are prolonged standing and vasodilator medication (McIntosh *et al*. 1993*b*). In our experience drug-induced falls and syncope in elderly people are more often due to vasovagal syncope than to orthostatic hypotension. Some patients experience symptoms in association with micturition, defecation, or coughing. Prodromal symptoms include extreme fatigue, weakness, sweating, nausea, visual defects, visual and auditory hallucinations, dizziness, vertigo, headache, abdominal discomfort, dysarthria, and paraesthesiae. The duration of prodrome varies from seconds to several minutes, during which some patients take action, such as lying down, to avoid an episode. The syncopal period, during which some patients develop involuntary movements ranging from tonic–clonic movements to myoclonic jerks, is usually brief. Recovery is generally rapid but some patients can experience protracted symptoms such as confusion, disorientation, nausea, headache, dizziness, and a general sense of ill health. The clinical characteristics of neurocardiogenic syncope are similar for young and elderly patients.

The sequence of events leading to neurocardiogenic syncope are not fully understood. The possible mechanism involves a sudden fall in venous return to heart, a rapid fall in ventricular volume, and virtual collapse of the ventricle due to vigorous ventricular contraction (Samoil and Grubb 1992). The net result of these events is stimulation of mechanoreceptors leading to peripheral vasodilatation (hypotension) and bradycardia (Samoil and Grubb 1992).

In comparison with younger adults, healthy elderly subjects are not particularly prone to neurocardiogenic syncope. Owing to an age-associated decline in baroreceptor sensitivity, the paradoxical responses to orthostasis (as in neurocardiogenic syncope) are possibly less marked in elderly subjects. Thus situational syncope is less common in old age. However, in the presence of hypertension and atherosclerotic cerebrovascular disease, excessive loss of baroreflex sensitivity leads to dysautonomic responses to prolonged orthostasis (in which blood pressure and heart rate decline steadily over time) and patients become susceptible to neurocardiogenic syncope. Diuretic- or age-associated contraction of blood volume further increases the risk of syncope.

By using the strong orthostatic stimulus of head-up tilting and maximal venous pooling, reflex neurocardiogenic syncope can be reproduced in a susceptible individual in the laboratory (Kenny *et al.* 1986). The sensitivity of head-up tilting can be further improved by provocative agents, such as isoprenaline (isoproterenol) and glyceryl trinitrate (nitroglycerin), which accentuate the physiological events leading to vasovagal syncope. Because of the decline in β-receptor sensitivity with age, isoprenaline is less useful as a provocative agent and has a higher incidence of adverse effects. The positivity of head-up tilting can also be enhanced by intravenous cannulation (McIntosh *et al.* 1994*b*).

Patient education, involving avoidance of precipitating factors and vasodilator drugs and lying down during prodromal symptoms, has great value in preventing episodes of vasovagal syncope. However, some patients experience symptoms without warning and so need drug therapy. A number of drugs, including β-blockers, disopyramide, transdermal scopolamine, fludrocortisone, fluoxetine, and midodrine, are reported to be useful in alleviating symptoms. Permanent cardiac pacing has been used in some patients, with the dual-chamber mode being preferred (Grubb *et al.* 1993). However, pacing influences the bradycardia component of the response, but has no effect on the vasodilatation and hypotension which frequently dominate. Its utility is limited in some instances to prolongation of the prodrome in order to allow other evasive action (Grubb *et al.* 1993).

Postprandial hypotension

In healthy elderly subjects, systolic blood pressure falls by 11 to 16 mmHg (1.4–2.1 kPa) and heart rate rises by 5 to 7 beats/min 60 min after meals (Lipsitz and Fullerton 1986). The change in diastolic blood pressure is not as consistent. In elderly subjects with hypertension, orthostatic hypotension, and autonomic failure, the postprandial blood pressure fall is much greater and without a corresponding rise in heart rate (Jansen *et al.* 1987). These responses are marked if the energy and simple carbohydrate content of the meal is high. In the majority of elderly subjects, most of these hypotensive episodes go unnoticed. Postprandial physiological changes include increased splanchnic and superior mesenteric artery blood flow at the expense of peripheral circulation, and a rise in plasma insulin levels (Potter *et al.* 1989) without corresponding rises in sympathetic nervous system activity. The vasodilator effects of insulin (Potter *et al.* 1989) and other gut peptides such as neurotensin and vasoactive intestinal peptide are thought to be responsible for postprandial hypotension, although the precise mechanism remains uncertain. The clinical significance of a fall in blood pressure after meals is difficult to quantify. However, postprandial hypotension is causally related to recurrent syncope and falls in elderly subjects (Jonsson *et al.* 1990). In our experience postprandial hypotension occurs in at least 20 per cent of patients with orthostatic hypotension. A reduction in the simple carbohydrate content of food, its replacement with complex carbohydrates, and frequent small meals are effective interventions for postprandial hypotension. Drugs useful in the treatment are indomethacin, octreotide, and caffeine. When given with food, caffeine prevents hypotensive symptoms in both fit and frail elderly subjects (Potter 1996).

Risk factors

A number of risk factors have been identified for falls. They include lower-limb weakness, peripheral neuromuscular dysfunction, gait and balance disturbances, assistive devices, visual defects, stroke disease, self-reported limitations, cognitive impairment, inability to perform activities of daily living, orthostatic hypotension, arthritis, incontinence, and medications. Table 7 lists the major risk factors for falls identified from 16 studies which compared fallers with non-fallers. Risk factors for falls vary according to whether a community, hospital-based, or nursing home population is studied (Rubenstein and Josephson 1996).

Falls during hospital stays are more common in confused patients and those with greater comorbidity. This profile differs from that of fallers in the community, possibly because hospital patients are more ill. Injurious falls are associated with a substantially increased resource utilization, and independently correlated with increased length of stay and, in the United States, with total health-care charges. After adjustment for confounders, fallers in one series stayed in hospital on average 12 days longer and incurred charges over $4000 higher than controls (Bates *et al.* 1995).

Risk factors for falls in hospitals differ little from those in the nursing home population. Major in-hospital risk factors include congestive cardiac failure, digoxin therapy, benzodiazepine use, and psychoactive agents. Additional risk factors in hospital falls include a history of recent falls, depression, dizziness, and acute confusion.

The presence of impaired orientation, psychoactive drug use, stroke disease, and impaired performance in the ‘get up and go’ test correctly classified 80 per cent of inpatients into fallers and non-fallers. This is a simple and quick screening test which could be used to identify those at risk of falling in hospital (Lord *et al.* 1992). Home hazards are frequently cited as risk factors for falls, but, although associated with a higher incidence of hip fracture, they are no more frequent for fallers than for non-fallers (Clemson *et al.* 1996).

Mobility impairment, including poor gait, poor balance, reduced leg extension strength, abnormal gait and stride length, and abnormal stepping height, is a risk factor for falls, particularly recurrent falls (Graafmans *et al.* 1996). The loss of postural balance and the inability to recover from an impending fall may be attributed to both abnormal muscle physiology and age-associated delays in central processing.

Four independent predisposing factors for falls (and for incontinence and functional dependence) are slow timed chair-stand (lower extremity impairment), decreased arm strength, decreased vision and hearing, and a high anxiety and depression score. Falls, incontinence, and functional dependence increase as the number of these predisposing factors increases (Tinetti *et al.* 1995).

Several studies have linked peripheral nerve dysfunction with postural instability and falls by older people. Although the comorbid causes of peripheral neuropathy are themselves often risk factors for falls, peripheral neuropathy appears to stand alone as a risk factor irrespective of associated comorbidity (Richardson and Hurbitz 1995). Sensitivity to foot position declines with age, mainly because of loss of plantar tactile sensation (Robbins *et al.* 1995). Tripping over an obstacle is a common cause of falls, and both young and old subjects are more likely to fail to avoid an obstacle if their attention is divided, but the effect is much greater in older persons (Chen *et al.* 1996). Although muscle strength declines with age, and in women begins to decline around the time of the menopause, the use of oestrogen replacement therapy does not appear to influence muscle strength, neuromuscular function, or the incidence of falls (Seeley *et al.* 1995).

Postural instability is a key feature of many specific neurological disorders such as Parkinson’s disease and stroke. Postural instability in Parkinson’s disease includes disturbed postural reflexes, poor control of voluntary movement, side-effects of medications (dys-kinesias), orthostatic hypotension, gait abnormalities, muscle weakness, and superimposed age-associated changes in peripheral sensation (Bloom 1992).

Falls are the most common single complication following stroke (Dennis *et al.* 1996), and fall-prevention strategies should be incorporated into stroke rehabilitation programmes (Nyberg and Gustafson 1995). The fear of falling affects both patients and carers (Forster and Young 1995). Falls occur with similar frequency in anticoagulated and non-anticoagulated patients; in hospital patients, the risk of minor injury is similar in the two groups (Stein *et al.* 1995).

Although posturography measurements are useful when elucidating balance problems (Baloh *et al.* 1995), they do not distinguish patients who fall and they do not correlate with other clinical risk factors for falls (Baloh *et al.* 1994). Medications are an often cited risk factor for falls. Common culprit medications include psychotropic drugs, benzodiazepines, antiparkinsonian treatment, and cardiovascular drugs. Psychoactive medication may predispose older patients to falling by impairing important sensorimotor systems which contribute to postural stability (Lord *et al.* 1995). Medication was a possible risk factor for falls in 40 per cent of cognitively normal and 60 per cent of patients with dementia attending an accident and emergency department because of falls (Davies and Kenny 1996; Shaw and Kenny 1997) (Fig. 1 and Table 3).

Cardiovascular medication has been cited as a cause of falls, but there does not appear to be a clear-cut relationship between drug-related orthostatic hypotension and falls (Liu *et al.* 1995). In a cross-sectional study of women using thiazide diuretics over a 10-year period, cases had a higher bone mass but an incidence of falls similar to that of women who had never used thiazide diuretics. The risk of non-spinal osteoporotic fractures was also similar in the two groups, but thiazide users had a trend towards a lower risk of fractures of the hip and the wrist (Cauley *et al.* 1993).

Visual impairment is cited as a major risk factor for falls and recurrent falls. Almost half of all fallers have evidence of visual impairment. In one series (Jack *et al.* 1995), 79 per cent of patients admitted to hospital after a fall had visual impairment including correctable refractory errors (40 per cent), cataract (37 per cent), and senile macular degeneration (14 per cent). The prevalence of visual impairment is higher in fallers admitted to hospital because of a fall than in community-dwelling fallers.

Most falls do not result in serious injury, and even more important than identifying risk factors for falling is identifying risk factors for injurious falls. In nursing home residents, lower-limb weakness, female sex, poor vision and hearing, disorientation, number of previous falls, impaired balance, dizziness, lower body mass and use of mechanical restraints, psychotropic medication, and functional dependence are the major risk factors for injurious falls (Tinetti 1987). Tinnetti *et al.* (1986) found that the risk of injurious falls increased from 27 per cent in patients with one risk factor to 78 per cent in patients with four or more risk factors. Similarly, Nevitt *et al.* (1989) found that the risk of recurrent falls in community-living persons increased from 10 to 69 per cent as the number of risk factors increased from one to four or more.

Investigation of a large series of institutionalized and outpatient populations identified many risk factors associated with falls (Robbins *et al.* 1989). Multivariate analysis enabled simplification of the model so that maximum predictive accuracy could be obtained using only three risk factors: hip weakness, unstable balance, and four or more prescribed medications. With this model the predicted 1-year risk of falling ranged from 12 per cent for persons with no risk factors to 100 per cent for persons with all three.

Use of restraints is uncommon in the United Kingdom. Cotsides (bed rails) were used for 8.4 per cent of one series of acute medical and geriatric patients. For almost all, the reason given for use was prevention of falls (O'Keefe *et al.* 1996). By comparison, in one United States series (Tinetti *et al.* 1992), 31 per cent of nursing home residents who were mobile and unrestrained at baseline had been restrained after 1 year of follow-up, two-thirds intermittently and a third continually. Serious fall-related injuries were more common in the restrained subjects (17 versus 5 per cent). Removing or changing restraints in nursing home patients who are not ambulatory will not increase their mobility. Increased mobility for patients will only be realized if restraint reduction programmes are combined with interventions to improve both mobility and behavioural performance.

Consequences

Forty to eighty per cent of falls by community-dwelling elderly people result in injuries. Most are mild or superficial, but 5 per cent of falls result in fractures and 1 per cent in fractured neck of femur. Eighty per cent of falls in nursing homes result in some injury.

Hip fractures represent one of the most important consequences of falls. Approximately half of previously independent elderly hip-fracture patients become partially dependent and a third ultimately become totally dependent. Hip fractures are associated with a reduction of 12 to 20 per cent in expected survival, with 5 to 20 per cent excess mortality within a year of injury. Age-specific incidence rates have increased in recent years, and in many countries the rise is continuing. Highest incidences have been described in Scandinavia and North America. The average lifetime risk of a hip fracture is 17 per cent in white women and 5 per cent in white men. At the age of 80 one woman in five, and at the age of 90 one woman in two, has suffered a hip fracture. Men also have poor outcomes from fractures; in a population-based series of 131 men in Rochester, only 41 per cent had recovered prefracture functional level and more than half were discharged to nursing homes (Lauritzen 1996).

Half of all attendances by adults aged over 50 years at an inner-city accident and emergency department in the United Kingdom followed a fall, and half of the patients were admitted to hospital (Davies and Kenny 1996; Richardson *et al.* 1997). Up to 50 per cent of the fall-related injuries that required hospital admission resulted in the elderly person being discharged to a nursing home.

Falls are the single most common cause of restricted activity days experienced by community-dwelling older adults and are twice as common a cause of restricted activity as heart disease, arthritis, or hypertension (Kosorok *et al.* 1992). A major consequence of falls is 'fear of falling' and 'post-falling anxiety syndrome'. Being moderately fearful of falling is associated with a decrease of satisfaction with life, increased frailty, and depressed mood, and is further associated with decreased mobility and social activities. This reduction in mobility includes an increase of wheelchair use by ambulant nursing home and residential care subjects (Kutner *et al.* 1994). The negative impact of falls on quality of life, mood, and functional capacity is even more apparent for subjects who experience 'unexplained' or recurrent falls than than for those who have accidental falls (Davies *et al.*, in press).

Intervention

Intervention programmes for falls require detailed assessment and screening for modifiable risk factors. The assessment should include cognitive function, full history of the type and frequency of falls, including whether or not there is associated loss of consciousness and dizziness, and, where possible, a witness account of falls. Details should include associated injuries, comorbidity, and medication use including the timing of doses. A full neurological examination should include assessment of muscle strength in upper and lower limbs, evidence of sensorimotor dysfunction, including tactile sensation and reflexes in the lower limbs, assessment of gait and balance, including posturography, and a search for evidence of Parkinson's disease, stroke, and supranuclear palsy. Cardiovascular assessment should include examination for peripheral vascular disease, cardiac lesions, and arrhythmias. Orthostatic hypotension should be sought in morning supine and upright blood pressures, and postprandial blood pressures should be measured repeatedly.

If falls are unexplained or recurrent, or if some episodes have been associated with loss of consciousness, the heart rate and blood pressure response to both supine and upright carotid sinus stimulation should be determined. If falls remain unexplained or the history suggests neurocardiogenic syncope, prolonged head-up tilt testing is recommended.

Visual assessment should include measurement of visual acuity and examination for cataracts and senile macular degeneration. Other investigations include routine blood screen, incorporating urea and electrolytes,

liver function tests, haematology screen, and acute phase protein activity, in addition to chest radiography, and 12-lead surface ECG. Where indicated, patients may require head CT or magnetic resonance imaging scans and ambulatory blood pressure if hypotensive episodes are suspected. Ambulatory ECG monitoring for arrhythmias may be relevant, particularly if the routine ECG is abnormal or there is a history of palpitations associated with falls. Caloric tests and hearing assessment may be necessary if the history suggests vestibular dysfunction.

Post-fall intervention programmes have been developed in many institutions, but possible benefits require further validation in randomized controlled trials. In one nursing home study, a randomized post-fall intervention programme was administered by a nurse practitioner. The group experienced a reduction in hospital admission (26 per cent) and length of hospital stay (62 per cent) compared with controls, but the number of falls was not significantly reduced (Wolf-Klein *et al.* 1988).

Intervention programmes focusing on specific risk factors (weakness, balance impairments, gait abnormality) are currently under way in the United States (Frailty and Injuries: Co-operative Studies of Intervention Techniques (FICSIT)) and some evidence has emerged of benefits from exercise (Province *et al.* 1995). Several studies have shown that healthier older adults who engage in intensive exercise can increase lower-limb muscle power; this is so even in frail nursing home patients. Similarly, balance is improved by walking programmes and low-impact aerobic programmes.

Modification of medication can contribute to a reduction in fall rates. Manipulation of cardiovascular medication reduced the incidence of unexplained falls and syncope in a select population of symptomatic patients referred to a specialist syncope facility (McIntosh *et al.* 1993*a*). Cardiac pacing intervention is clearly beneficial in reducing syncopal events in older patients with bradyarrhythmia, in particular sinus node disease, atrioventricular conduction disturbances, and carotid sinus syndrome. In one series of patients with cardio-inhibitory carotid sinus syndrome one-third who presented with unexplained falls in addition to syncope were asymptomatic for falls and syncope 1 year after dual-chamber pacing. Randomized control trials of cardiac pacing for unexplained falls are under way.

Both cognitively normal and demented patients comply with environmental home hazard modifications (Chapman *et al.* 1997). Randomized controlled trials incorporating environmental hazard modification programmes are in progress.

Many fall-assessment tools have been developed for use in hospitals and nursing homes to identify patients at ‘high risk of falling’. The ‘fall risk’ status of a patient is determined by the number of identified risk factors or by a summary score. A nursing care plan is then implemented which includes interventions aimed at injury prevention. Few data support the validity of these assessment tools or the effectiveness of most prevention programmes.

Many devices have been marketed to alert caregivers to patient activity and falls. Alarm systems can deliver earlier assistance systems and may even reduce fall rates. Hip protector pads are currently advocated for use in patients at risk of hip fracture from falls and have been shown to reduce fracture rates (Lauritzen *et al.* 1993).

Because of the frequent presence of more than one risk factor in patients at most risk of injurious falls, randomized controlled trials of post-fall multi-intervention packages, tailored to individual needs, are required. In our experience, more careful assessment of cardiovascular causes of falls is an important and underutilized component of intervention programmes, particularly for patients with unexplained or recurrent falls.

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