Preventing fractures in the elderly

Fractures in the elderly are devastating, causing premature death and compromised independence among survivors. With demographic changes, their occurrence is expected to double. Given their crippling consequences, the identification and treatment of osteoporosis and prevention of falls are priorities.

Fractures constitute a major public health problem among older people and with demographic changes the burden is anticipated to double over the next 50 years. Approximately one third of women and one in five men aged over 50 years will suffer a fragility fracture during their remaining lifetime (Chrischilles et al, 1991). Fragility fractures typically occur at the distal forearm, vertebrae and proximal femur although they also occur at other sites including the proximal humerus, pelvis and ankle.

In the UK, osteoporosis results in more then 300 000 fractures per year, causing death, severe pain and disability to individuals at an annual cost of around £2 billion, approximately 2% of the total annual NHS budget (Harvey et al, 2010). Not only are fractures costly to society, but also to individuals: mortality is substantially increased following hip or vertebral fracture with highest rates in the 12 months following a hip fracture (21% for women and 36% for men) (Office of Technology Assessment, 1993). Given this heavy burden, any intervention that may reduce the risk of fracture at either the individual or population level warrants critical appraisal. This review will discuss fracture prevention in terms of identification and treatment of osteoporosis and reducing rates of falls.

Osteoporosis

Osteoporosis is the commonest bone disease, defined as: 'a progressive systemic skeletal disorder characterised by low bone mass and microarchitectural deterioration of bone tissue with a consequent increase in bone fragility and susceptibility to fracture' (Consensus Development Conference, 1993). Osteoporosis occurs among women more frequently than men, its prevalence increases with age and is treatable but often left untreated, not least because its onset is asymptomatic before the occurrence of fracture (British Orthopaedic Association, 2007).

The clinical significance of osteoporosis lies in this predisposition to fracture. Once an individual has experienced one fragility fracture, his/her risk of subsequent fragility fractures at the same or other anatomical sites is at least doubled. This increased risk is most dramatic in the spine, where the occurrence of one vertebral fracture is associated with a 5.0-fold increased risk of a subsequent vertebral fracture (Black et al, 1999). With the morbidity associated with osteoporotic fracture, the increased associated mortality and the unacceptable increase in subsequent fracture after the first fracture, the emphasis must be on prevention, rather than cure, of osteoporosis. In any patient who has experienced one fragility fracture, secondary prevention must be prioritized.

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Assessment of the risk of fracture

Prospective studies have shown that the risk of fracture increases progressively with decreasing bone mineral density measured by dual-energy X-ray absorptiometry. The risk of fracture approximately doubles for each standard deviation decrease in bone mineral density. The predictive value of bone mineral density is at least as good as blood pressure is of stroke (National Osteoporosis Guideline Group, 2010). The first attempt to define an at-risk population for targeting prevention was that of an expert panel convened by the World Health Organization in 1993 (Consensus Development Conference, 1993). Osteoporosis was defined as a bone mineral density measurement using dual-energy X-ray absorptiometry of less than 2.5 standard deviations below the young adult mean. Patients with a history of one or more proven fragility fractures, as well as bone mineral density ≤-2.5 standard deviations below the young adult mean (also known as a T score < -2.5) have 'severe' or 'established' osteoporosis.

Bone density measurement is currently the best available method of predicting future fracture but the bone mineral density-based definition can only capture the deterioration in bone mineralization. Many factors contribute to the risk of a bone breaking at a given level of force and therefore the use of bone mineral density to assess fracture risk has a high specificity but low sensitivity. This low sensitivity results in over 80% of low trauma fractures occurring in people who do not have a T score ≤ -2.5 (Jarvinen et al, 2008). However, it has been demonstrated that the performance characteristics of assessment can be improved by concurrent consideration of clinical risk factors, for example age and body mass index, which provide information on fracture risk independently of bone mineral density (National Osteoporosis Guideline Group, 2010).

Algorithms that integrate the weight of clinical risk factors for fracture risk, including and not including bone mineral density, have been developed by the WHO Collaborating Centre for Metabolic Bone Diseases in Sheffield (*Figure 1, Table 1*). The FRAX tool can be

accessed by the clinician from the internet in the clinic (www.shef.ac.uk/FRAX) and used to compute the 10-year probability of a hip and/or any major osteoporotic (defined as clinical vertebral, hip, wrist or humerus) fracture. The tool is simple and quick to use, providing that the clinical risk factor information is available, and is designed so that the clinician can share the fracture risk estimation with the patient in front of them.

It must be borne in mind that the algorithms cannot take account of all scenarios, in particular dose relationships: an individual with four previous fragility fractures is at higher risk than one with one prior fracture, a prior vertebral fracture carries a greater risk of future fracture than any other fragility fracture and glucocorticoid exposure has a dose–response relationship. The estimations derived using FRAX must be tempered by the clinician's knowledge of all relevant factors in the individual. However, with this proviso, the use of 10-year fracture probabilities is a major step forward in identifying those in whom intervention is needed and its use may well improve compliance.

Figure 1. The FRAX algorithm, available online at www.shef.ac.uk/FRAX.

Population-based public health strategies for the prevention of osteoporosis

Osteoporosis is associated with a sedentary lifestyle, tobacco smoking and alcohol excess. Physical exercise is an important determinant of bone mineral density as bone responds to mechanical stimuli such as weight bearing or resistance training. For this reason, prolonged bed rest is associated with rapid decrease in bone mass, sarcopenia (reduced muscle mass) and reduced physical fitness, especially in the elderly. Cross-sectional studies have shown a positive correlation between exercise levels and bone mass. A systematic review suggested that physical exercise programmes may have a modest positive effect on bone mineral density and there are studies suggesting some reduction in the risk of vertebral and hip fractures with such programmes (Bonaiuti et al, 2002) but to date there are no studies evaluating the uptake and compliance with these regimens to suggest that they can safely be recommended at a public health level. Potentially, advice to stop smoking and reduce levels of alcohol consumption are valuable public health approaches in osteoporosis as in diseases of other bodily systems but, again, there are no studies of the feasibility or effectiveness of this approach on fracture prevention (National Osteoporosis Guideline Group, 2010).

Nutrition, protein, calcium and vitamin D

There is a clear relationship between low body weight and body mass index (poor nutrition) and increased risk of hip fracture (British Orthopaedic Association, 2007).

Table 1. Clinical risk factors assessed in the FRAX algorithm

Risk factor	Comment
Age or date or birth	Age between 40–90 years (will accept entries outside this range but will provide an estimated fracture rate for closest available age)
Gender	Calculates gender-specific rates for men separately from women
Weight (kg)	Used to calculate body mass index
Height (cm)	Used to calculate body mass index
Previous fracture	Yes for a previous fracture occurring in adult life spontaneously or from low trauma (i.e. trauma that would not normally have caused a fracture in a healthy individual)
Parental hip fracture	Yes for a history of a hip fracture occurring in the individual's mother or father
Current smoking	Yes for a current smoker of tobacco
Exposure to glucocorticoids	Yes if currently taking oral glucocorticoids or has ever been exposed to prednisolone (or equivalent) >5 mg for more than 3 months
Rheumatoid arthritis	Yes if confirmed with rheumatoid arthritis
Secondary osteoporosis	Yes if the patient has another disease strongly associated with osteoporosis (type I diabetes mellitus, osteogenesis imperfecta, untreated long-standing hyperthyroidism, hypogonadism or premature menopause (<45 years), chronic malnutrition or malabsorption or chronic liver disease)
Alcohol 3 or more units/day	Yes if patient drinks >3 units alcohol/day
Femoral neck bone mineral density (g/cm^2)	Not required for estimate to be made. If available, clinician must select dual energy X-ray absorptiometry scanning equipment used and enter actual femoral neck bone mineral density in g/cm^2

Post-fracture outcomes may be improved after hip fracture with multi-nutrient supplementation. Housebound, frail, elderly adults, particularly those in nursing homes, have a very high prevalence of calcium and vitamin D insufficiency. It has been shown that calcium and vitamin D supplementation in nursing homes can decrease the risk of secondary hyperparathyroidism and reduce the risk of hip fracture. In these groups of individuals, supplementation with vitamin D 800 IU and calcium 1.0–1.2 g daily is recommended.

A recent observational study reported an increased risk of myocardial infarction among patients receiving calcium supplementation without co-administered vitamin D (Bolland et al, 2011). The magnitude of the effect was small (odds ratio for myocardial infarction 1.27, 95% confidence interval 1.01–1.59) but the authors recommended a review of the guidance around calcium supplementation (without co-administration of vitamin D) in the prevention of osteoporosis, particularly as the benefit of calcium supplementation alone on fracture risk is at best modest.

Pharmacological therapies in osteoporosis

There have been rapid developments in pharmacological therapies for osteoporosis in the last three decades. Most of the available therapies act by prevention of bone resorption although some of the more modern therapies have effects on promotion of bone formation (*Table 2*).

Bisphosphonates

Etidronate, alendronate, risedronate, ibandronate, zoledronate and clodronate are all examples of bisphosphonates, the most ubiquitous pharmacological therapies for osteoporosis. These are antiresorptive therapies with their potency and skeletal retention determined by their side chain. The oral bisphosphonates are poorly absorbed in the intestine and are associated with gastrointestinal side effects, requiring that they are taken on an empty stomach after an overnight fast with a long glass of water and food avoided for at least half an hour. Although antifracture efficacy has been clearly demonstrated (*Table 2*), compliance with oral bisphosphonates tends to be poor with less than 50% of subjects remaining on therapy after 9 months. Zoledronate has been licensed for the treatment of osteoporosis annually by intravenous infusion (over 15 minutes) which may improve rates of adherence in the longer term but data are not yet available. The antifracture efficacy of bisphosphonates has only been tested when co-prescribed with calcium and vitamin D supplementation.

Recently, concerns have been raised about side effects of longer-term bisphosphonates, centring on three conditions: osteonecrosis of the jaw, oesophageal cancer and atypical femoral fractures (Compston, 2011). Osteonecrosis of the jaw, defined as exposed bone in the maxillofacial region for >8 weeks, was initially described in patients with bone metastases receiving high-dose bisphosphonates usually intravenously for malignant hypercalcaemia, among whom the incidence is 1–12%. The major risk factor is dental trauma, poor dental health and caries. To date, there have been very few cases worldwide reported among postmenopausal women receiving bisphosphonates in standard doses for idiopathic osteoporosis, with an estimated incidence of <1 in 100 000 person-years of exposure. It is unclear, however, whether this complication will be seen more frequently with increased use of intravenous bisphosphonate therapies such as zoledronate. It is advised that necessary oral or dental surgery is carried out before starting bisphosphonates and patients taking these drugs should have regular dental checks.

Table 2. Antifracture efficacy of pharmacological therapies among postmenopausal women with osteoporosis when co-prescribed with calcium and vitamin D

	Vertebral fracture	Non-vertebral fracture	Hip fracture
Alendronate	A	A	A
Etidronate	A	B	-
Ibandronate	A	A (subsets only)	-
Risedronate	A	A	A
Zoledronate	A	A	A
Denosumab	A	A	A
Calcitonin	A	B	B
Calcitriol	A	B	-
Raloxifene	A	-	-
Strontium ranelate	A	A	A (subsets only)
Teriparatide	A	A	-
Hormone replacement therapy*	A	A	A

* Hormone replacement therapy is no longer indicated in the prevention of osteoporosis because of its wider effects on other diseases. A = grade A evidence from systematic reviews or randomized controlled trials; B = grade B evidence from systematic reviews or randomized controlled trials

Recently, there have been a number of case reports of atypical femoral shaft fractures among patients who have been exposed to bisphosphonates for long periods. These atypical fractures also occur in patients never exposed to bisphosphonates. Other risk factors include glucocorticoids, vitamin D deficiency, co-treatment with other antiresorptive therapies, rheumatoid arthritis and diabetes (Shane et al, 2010). Although these fractures are rare (<1% of all hip and femoral fractures) they are associated with high morbidity and poor healing usually requiring surgical fixation.

After US Food and Drug Administration reports of cases of oesophageal cancer occurring among patients treated with bisphosphonates, several observational studies have been carried out, the results of which are conflicting. Even the two largest, both using data from the UK General practice research database, found opposing results such that one found no significant association but the other found a significant increase in rates of occurrence of oesophageal cancer among bisphosphonates users exposed >3 years (Cardwell et al, 2010; Green et al, 2010). At present, the data are inconclusive but caution is needed if considering using long-term bisphosphonates in individuals with Barrett's oesophagus.

The causal role of bisphosphonates in all three of these complications is therefore controversial. Overall, the risk/benefit balance still overwhelmingly favours bisphosphonates in patients at high risk of osteoporotic fracture. However, clinicians should be aware of the risks of long-term bisphosphonates and the ongoing need for the treatment should be reviewed in all patients periodically. Currently, many clinicians suggest bisphosphonates for a period of 5 years with a 'drug holiday' for a period thereafter but these intervals are arbitrary, not backed up by available evidence at this time and individual judgement will be required in patients with an unacceptably high risk of fracture (Compston, 2011).

Strontium ranelate

The mode of action of strontium ranelate is not fully understood although it has been claimed to maintain bone formation while being antiresorptive. It is approved at a dose of 2 g daily for the treatment of postmenopausal women with osteoporosis among whom it has been shown to prevent vertebral fractures. In a post-hoc analysis of women aged >74 years, it has also been shown to prevent hip fractures (*Table 2*). Taken as a daily sachet of granules, which are dissolved and drunk at least 2 hours after the last meal, it is usually taken at bedtime. It is contraindicated in patients with renal impairment and side effects include gastrointestinal symptoms such as nausea and diarrhoea as well as headaches and eczema.

Selective estrogen receptor modulators

Selective estrogen receptor modulators selectively inhibit transformation of the oestrogen receptor exerting a variable profile on tissues with oestrogen receptors such as breast, myocardium and bone. The first clinically-used selective estrogen receptor modulator was tamoxifen, which was extremely effective in the secondary prevention of breast cancer but had limited skeletal effects. Raloxifene is an antiresorptive therapy that has been shown to significantly decrease the rate of new vertebral fractures among postmenopausal women with previous vertebral fractures but does not convey a decreased risk of non-vertebral or hip fractures. It is licensed for the prevention and treatment of vertebral osteoporosis but also showed efficacy in the primary prevention of breast cancer among low-risk women. It cannot be used in the early post-menopause as it exacerbates vasomotor symptoms and it carries the same increased risk of venous thromboembolism as hormone replacement therapy.

Parathyroid hormone peptides

Parathyroid hormone is an essential regulator of calcium metabolism. Its physiological actions are on the skeleton, where it promotes bone resorption to normalize calcium, but it also acts upon the kidney to promote renal conservation of calcium and phosphate and indirectly acts upon intestinal calcium absorption through its actions to promote renal production of the active vitamin D metabolite 1,25 (OH)2 vitamin D. When parathyroid hormone peptides are administered intermittently, they exert anabolic skeletal effects increasing bone formation.

Teriparatide (recombinant human parathyroid hormone 1,34) is licensed for treatment of osteoporosis in postmenopausal women and men at high risk of fracture and severe glucocorticoid-induced osteoporosis, administered by daily subcutaneous self-injection (20 µg) once daily for a maximum of 24 months. Its effects on serum calcium levels must be monitored and it is contraindicated in hypercalcaemia, severe renal failure, malignancy and/or radiotherapy affecting the skeleton. In the UK, the National Institute for Health and Clinical Excellence approved its use only in patients with severe osteoporosis who are unable to take bisphosphonates and strontium ranelate or have had an unsatisfactory response and who are aged >65 years with a T score < -4.0 standard deviations or a score -3.5 standard deviations with two or more fragility fractures and in patients aged 55–64 years with a T score < -4.0 and more than two fragility fractures (National Institute for Health and Clinical Excellence, 2011a).

Calcitonin

Calcitonin is an antiresorptive endogenous polypeptide hormone. Nasal salmon calcitonin 200 IU daily is approved for use in postmenopausal women with established osteoporosis to reduce the risk of vertebral fracture but it has not been shown effective for prevention of non-vertebral or hip fracture. It cannot be used in patients with hypocalcaemia or nasal ulceration.

Calcitriol

1,25 dihydroxyvitamin D is the activated form of vitamin D and is an antiresorptive therapy. It is approved at a dose of 25 μg twice daily in established postmenopausal osteoporosis for the prevention of new vertebral fracture but its efficacy in prevention of non-vertebral and hip fracture is not established. The main complication of calcitriol is hypercalcaemia and serum calcium levels must be monitored at 1, 3 and 6 months.

Hormone replacement therapy

Hormone replacement therapy is available in several preparations consisting of oestrogen with or without progestogen. Hormone replacement therapy is efficacious in the prevention of peri-menopausal symptoms such as flushing, mood disturbances and night sweats provoked by the menopause. During the peri-menopause, women undergo rapid bone loss as a consequence of the endocrine changes. Some hormone replacement therapy preparations have been demonstrated to prevent the bone loss associated with the menopause and to reduce the risk of fractures in the spine and non-vertebral sites. However, the risk:benefit ratio of hormone replacement therapy for fractures was impacted by data showing an increased risk of breast cancer associated with long-term hormone replacement therapy use as well as other complications such as venous thromboembolic disease, cardiovascular disease and gall bladder disease.

In the light of the co-morbidities and the advent of other more bone-specific therapies, hormone replacement therapy is now limited to the treatment of women in the early post-menopause with symptoms of severe oestrogen deficiency and its use is not indicated in the long-term prevention of osteoporosis or fracture.

Denosumab

Denosumab is a humanized monoclonal antibody directed against the receptor activator of nuclear factor kappa-B ligand (RANKL). The role of the RANK/RANKL pathway has been relatively recently elucidated in the promotion of differentiation of pre-osteoclasts to osteoclasts (the predominant bone resorptive cells). In normal health, this pathway is tightly regulated by osteoprotegerin and RANKL, both of which are secreted by osteoblasts. Therefore, denosumab inhibits the maturation of osteoclasts, resulting in suppressed bone turnover and increased bone mineral density (9.2% at the lumbar spine and 6.0% at the total hip) (Cummings et al, 2009). The reduced bone turnover resulted in a 68% reduction in vertebral fracture and 40% reduction in hip fracture. Denosumab has been approved for administration subcutaneously (60 mg) every 6 months over 3 years among postmenopausal women with osteoporosis, in whom it has been shown to be effective in reduction of vertebral and non-vertebral fractures (National Institute for Health and Clinical Excellence, 2011b). Data from studies of use in prostate cancer patients suggest that it has similar efficacy among men (Smith et al, 2009).

Falls

As people age, they fall over more often, largely as a result of deteriorating eyesight, poor balance and dementia. It has been estimated that almost one in three adults aged >65 years living in the community fall each year (Gillespie et al, 2009). Although most falls do not result in serious injury, the consequences for an individual of falling or of not being able to get up after a fall can include fear of falling and loss of confidence in being able to move about safely, loss of mobility, leading to social isolation and depression, increase in dependency and disability, hypothermia, pressure-related injury and infection. Approximately 1 in 5 falls require medical attention but only 1 in 10 result in a fracture. Although only 1% of falls result in a hip fracture, 90% of all hip fractures result from a fall of standing height or less. Therefore, any strategy to prevent hip fracture must focus on fall prevention as well as other factors.

Falls have a multifactorial aetiology. The principal recognized risk factors for falls are detailed in *Table 3*. There is evidence to suggest that rate of falls and risk of falling can be reduced (Gillespie et al, 2009) by multi-faceted exercise interventions in groups (rate of falls ratio 0.78, 95% confidence interval 0.71–0.86; risk of falls ratio 0.83, 95% confidence interval 0.72–0.97), and Tai Chi (rate of falls ratio 0.63, 95% confidence interval 0.52–0.78; risk of falls ratio 0.65, 95% confidence interval 0.51–0.82).

When multiple-component home-based exercise was individually prescribed, it was effective on both the rate of falls and the risk of falling (rate of falls ratio 0.66, 95% confidence interval 0.53–0.82; risk of falls ratio 0.77, 95% confidence interval 0.61–0.97). In contrast, assessment and multifactorial intervention reduced the overall rate of falls (rate of falls ratio 0.75, 95% confidence interval 0.65–0.86), but not the risk of falling. Overall, population-based vitamin D supplementation did not reduce falls, but vitamin D supplementation targeted at those with insufficiency or deficiency may be effective.

Equally, home safety interventions did not reduce falls except in people with severe visual impairment and other individuals at high risk of falling. An anti-slip shoe device reduced rate of falls in icy conditions (rate of falls ratio 0.42, 95% confidence interval 0.22–0.78). Gradual withdrawal of psychotropic medication reduced the rate of falls (rate of falls ratio 0.34, 95% confidence interval 0.16–0.73), but not the risk of falling. A prescribing modification programme for primary care physicians significantly reduced risk of falling (risk of falls ratio 0.61, 95% confidence interval 0.41–0.91). Pacemakers reduce the rate of falls in people with carotid sinus hypersensitivity (rate of falls ratio 0.42, 95% confidence interval 0.23–0.75). First eye cataract surgery was also shown to reduce the rate of falls (rate of falls ratio 0.66, 95% confidence interval 0.45–0.95).

Conclusions

The prevention of fractures in older people is a priority particularly with the rapidly ageing population. Prevention strategies should be aimed at identification of those at highest risk, using clinical risk factor algorithms with or without bone mineral density assessment and targeting pharmacological therapies appropriately but also must encompass an assessment of the risk of falling and adjustment of the risk where possible. BJHM

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Table 3. Risk factors for falls in elderly people

Personal factors	Balance, gait, mobility	Musculoskeletal, e.g. arthritis, proximal myopathy
		Neurological, e.g. Parkinson's disease, peripheral neuropathy, reduced proprioception, cerebrovascular disease, weakness
		Amputation, slow walking speed
		Comorbidities: diabetes
	Frailty	Delayed reaction time
		Slower neuromuscular coordination
	Visual impairment	Age-related acuity changes
		Cataracts
		Glaucoma
		Macular degeneration
	Delayed cognition	Alzheimer's disease or dementia of other cause
		Cerebrovascular disease
		Alcohol, drugs
	Causes of 'blackouts'	Cardiovascular: syncope, dysrhythmia, heart block, postural hypotension, vertebro-basilar insufficiency
		Metabolic: hypoglycaemia, dehydration, vitamin D deficiency
		Neurological: epilepsy, cerebrovascular disease
Extrinsic factors	Environmental	Poor lighting
		Cluttered furniture
		Rugs
		Slippery floors, baths, showers
		Pets, toys
		Electrical wires
		Steep staircases
		No handrails
	Personal	Unsupportive footwear
		Poor clothing (e.g. long skirts)
		Medications: sedatives, hypotensives, antidepressants, anticonvulsants
	Hazards	Icy pavements
		Uneven or broken paving, potholes

KEY POINTS

- Fractures are common in the elderly and are both life threatening and life changing when they occur.
- It is possible to estimate an individual's 10-year risk of an osteoporotic fracture using bone mineral density measurements in combination with clinical risk factors.
- There have been rapid developments in pharmacological therapies over the past two decades. Therapies can be administered to patients at the highest risk of osteoporotic fracture in tailored regimens producing effective prevention of fracture.
- Any strategy to prevent fractures in individuals must also consider their risk of falls and address risk factors for falling.