■ ASPECTS OF CURRENT MANAGEMENT

The prevention of osteoporotic fractures

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The World Health Organisation defined osteoporosis in 1994 as a condition in which the bone mineral density (BMD) is 2.5 SDs or more below the mean seen in young normal subjects.[1] By this definition, a quarter of all post-menopausal American Caucasians, in total 26 million white American women, are regarded as having osteopororis.[2] Fragility fractures, the major clinical problem in osteoporosis, have increased in recent decades, (Fig. 1)[3] so that in Western countries it is estimated that half of all women and one-third of all men will sustain a fragility fracture during their lifetime.[4] The incidence of osteoporotic fractures is expected to increase so that by 2050, it is thought that approximately 6.3 million hip fractures will occur globally (Fig. 2).[4] Why this increase has occurred is not fully understood but a higher proportion of the elderly in the population, changes in BMD and other risk factors may influence the rate of fracture.

The rising burden of these fractures imposes an enormous cost on society[5] and increases morbidity and mortality.[6] A fracture of the hip is associated with a reduction of 20% in expected survival.[7] Many patients become permanently disabled with the proportion who cannot walk rising from 20% before to 50% after the fracture.[8] One-third become totally dependent, necessitating institutionalisation.[9] It is, therefore, imperative to implement strategies for preventing such fractures in the community.[10]

The BMD is currently the best measure for assessing the strength of bone and therefore a predictor of fractures. It is usually used as a surrogate end-point for fractures in randomised, controlled trials (RCTs), but it must be remembered that the risk of fracture increases exponentially with a decrease in the BMD.[11] There is no specific level of BMD which indicates a 'fracture threshold'. A decrease in the BMD of 1 SD is usually regarded as increasing the risk of fracture by 1.5 times and by 2.5 times in the measured skeletal region.[12,13] However, all risk factors must be considered in addition to a low BMD (Table I; Fig. 3).[14] We have searched Medline from 1966 onwards to evaluate data which support different strategies for the prevention of fractures. We assessed prospective RCTs using fracture as the end-point. We also looked

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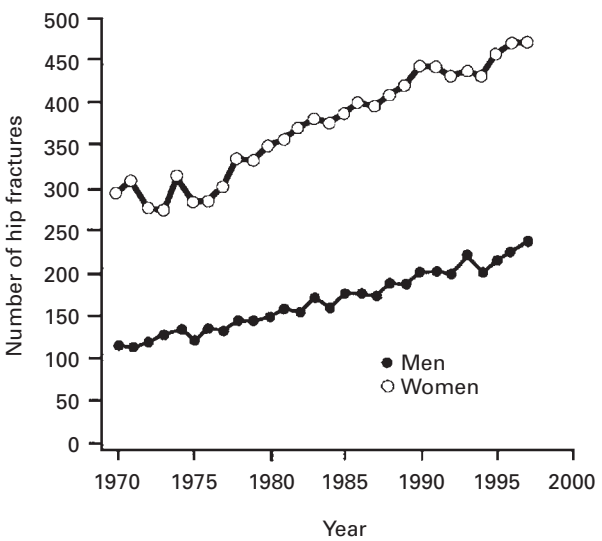

Fig. 1

A graph showing the age-adjusted incidence (per 100 000 persons) of hip fractures in Finland in women and men aged 50 years or more between 1970 and 1997 (reprinted with permission from Kannus et al).[111]

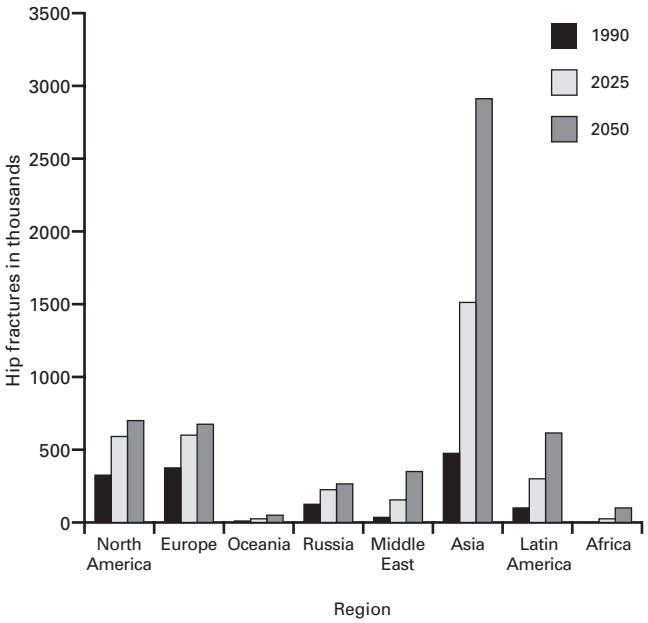

Fig. 2

Bar chart showing the estimated numbers of hip fractures in eight geographic regions in 1990, 2025 and 2050 (reprinted with permission from Cooper et al).[4]

Table I. Risk factors for osteoporosis, falls and fractures

Risk factor	Osteoporosis	Fall	Fracture
Low BMD			+
High age	+	+	+
Female gender	+	+	+
Primary or secondary amenorrhoea	+		+
Primary or secondary hypogonadism in men	+		+
Premature menopause	+		+
Post-menopausal status	+	+	+
Tallness		+	+
Low body weight	+		+
Long hip axis length			+
Previous fragility fracture	+	+	+
Family history of fracture			+
White or Asian ethnic origin			+
Immobility/low physical activity	+	+	+
Current smoking	+	+	+
High caffeine intake			+
Alcohol abuse	+	+	+
High bone turnover	+		+
Osteomalacia/vitamin-D deficiency	+	+	+
Low dietary calcium intake	+		+
Chronic illnesses	+	+	+
Glucocorticoid therapy	+		+
Sedative medications		+	+
Visual impairment		+	+
Cognitive impairment		+	+
Neurological diseases		+	+
Lower limb disability	+	+	+
Hyperthyroidism	+		+
Hyperparathyroidism	+		+
Malabsorption	+		+
Coeliac disease	+		+
Gastrectomy	+		+
Chronic arthritides	+	+	+
Chronic renal/liver diseases	+		+
Cushing's syndrome	+		+
Malignancies	+		+
Organ transplantations	+		+
Living in a nursing home		+	+

at prospective and retrospective observational studies with fracture as the end-point and RCTs with BMD or a frequency of a fall as the surrogate end-point for fractures.

Non-pharmacological prevention of osteoporotic fractures

Nutrition. The role of calcium in attaining an adequate peak bone mass and reducing age-related bone loss has been

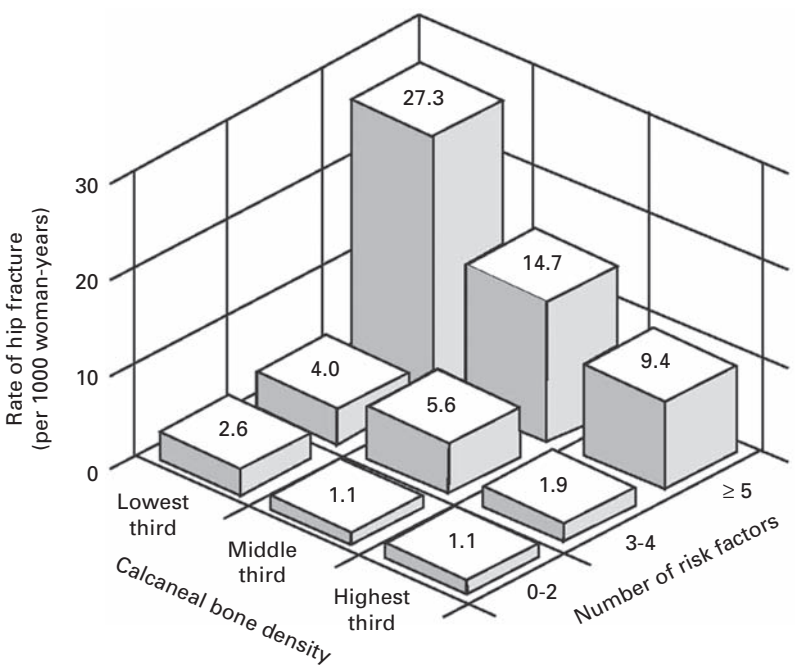

Fig. 3

The annual risks of hip fracture according to the number of risk factors and the age-specific calcaneal bone density (reprinted with permission from Cummings et al).[14]

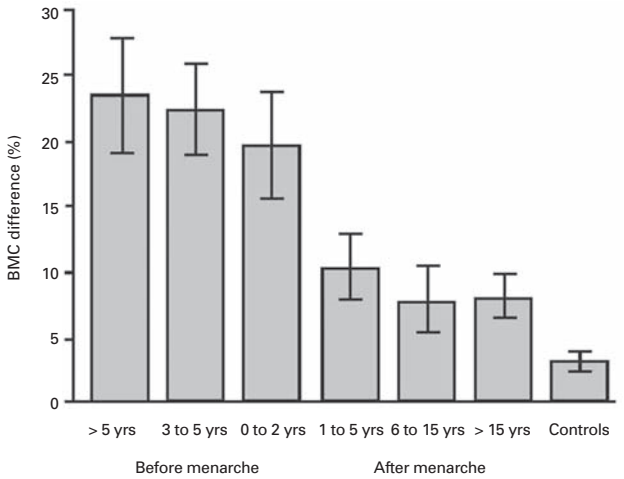

Fig. 4

Bar chart showing the mean and 95% confidence intervals difference in bone mineral content (BMC) of the humeral shaft of the playing and the nonplaying arm in 105 female tennis and squash players and their 50 control subjects according to biological age at which training was started (i.e. starting age of playing relative to age at menarche) (reprinted with permission from Kannus et al).[35]

thoroughly investigated. The positive correlation between dietary calcium and the BMD has been established in observational studies in children,[15] adolescents,[16] and young women,[17] but the relationship between the dietary intake of calcium and risk of fracture of the hip is less clear.[18-20] In spite of all the published data there is no universal agreement as to the required daily intake of calcium. In 1994, a consensus conference recommended a daily intake of 1200 to 1500 mg for adolescents, 1000 mg for adults up to the age of 65 years, and 1500 mg for post-menopausal women not receiving oestrogen and for the elderly.[21] It has also been reported from observational studies that an intake below 400 mg per day requires increasing supplements of calcium in order to reach an adequate BMD.[22]

Absorption of calcium is dependent on the level of vitamin D. The current recommendation is that the daily intake of vitamin D should be between 400 and 800 IU if exposure to sunlight is low, especially in the elderly, but this recommendation is based on a low level of evidence. In frail elderly people, it is difficult to achieve an adequate intake of protein, total energy and the necessary amount of other nutritional components such as phosphorus, magnesium, zinc, copper, iron, fluoride, sodium and vitamins D, A, C, and K, all important for skeletal health. Currently, there is no consensus as to the recommended levels of these nutritional elements. Consumption of alcohol and caffeine and smoking may lower the BMD, but no RCT with fracture as the end-point has been carried out which addresses adequate or inadequate intake of the different components. The time influence of these elements is not known.

Physical activity. The frequency of falls is the most potent risk for fractures. The same factors which influence falls also apply to fractures (Table I).[23-25] Falls are commonly used in RCTs as a surrogate end-point for fractures and recent prospective RCTs have shown that exercise can reduce the risk of falling in the elderly and frail.[26-31] Exercises which include balance training have been shown to reduce the risk of falling,[32-34] but the best outcome has been achieved by training in Tai Chi which reduced falls by about 50%.[26,27]

Physical activity may increase the BMD and strength of bone by 30% to 50%[35,36] when training is initiated before puberty since bone seems to be most adaptive to mechanical load during periods of fast growth (Fig. 4).[35,36] This has been shown in several short-term RCTs with BMD as the end-point.[37,38] Regular impact-loading activities which create strains of high magnitude which may be distributed throughout the bone structure improve bone strength best.[36,39-41] Squash, tennis, badminton, aerobics, step exercises, volleyball, basketball, soccer, gymnastics and weight and power training can increase the BMD.[36] By contrast, endurance training, such as long-distance running, swimming and cycling are not as efficient.[36]

Bone responds to exercise less in adulthood than during growth. In the adult and after the menopause, physical activity should be regarded as bone-preserving rather than bone-building. Several studies and RCTs have shown that in adults, exercise increases the BMD by a few percentage points at best.[39,42] However, exercise may reduce the risk of fracture by increasing muscle strength and improving balance.[43-47] Exercise should be carried out throughout life since cessation is followed by a rapid decline in the BMD and in muscle benefits.[40,48]

There is no RCT which evaluates the effect of physical activity with fracture as the end-point. We have to rely on prospective and retrospective observational and case-control studies. These consistently show that both past and current physical activity is associated with a reduction of up to 50% in the risk of hip fracture in both men and women.[36,43,44] The studies focusing on physical activity and other fractures are few and have conflicting results,[36] but they suggest that lifetime physical activity is associated with a low incidence of fracture.[43,44,49] However, vigorous activity in frail elderly people may increase the risk of falling. Their activity programme must be individually designed based on the physical abilities of the individual and then undertaken with caution.[49]

Modification of environmental risk factors. The effectiveness of modifications of a variety of environmental risk factors for falls and fractures (Table I) has not been demonstrated in a RCT. In spite of this, it seems sensible to modify and eliminate all possible elements of risk which could lead to falls in the home, since these are easy to achieve. Previous falls are an independent risk factor for subsequent falls and it is especially important to evaluate each elderly person who has fallen for risk factors in the home environment. This approach has been successfully used in the 'Prevention of Falls in the Elderly' trial, in which intervention decreased

Table II. Randomised controlled trials (RCTs) with the incidence of vertebral and hip fractures over three years, if not otherwise stated, with the percentage of patients and relative risk (95% confidence interval or p value) in trials with calcium and vitamin D, hormone replacement therapy (HRT), raloxifene, alendronate, risedronate, nasal calcitonin, 1-34 fragment of recombinant human parathyroid hormone (rh 1-34 PTH) and strontium ranelate in the treatment of post-menopausal osteoporosis

	Study	Risk profile of patients	Gender	Mean age (yrs)	Numbers of patients included	Incidence of fracture (%)*		
						Placebo	Drug	Relative risk (95% CI or p value)
Vertebral fracture								
Drug								
HRT	WHI[75]	Healthy post-menopausal women	F	63	16 608	0.74	0.48	0.66 (0.44 to 0.98)
Raloxifene 60 mg	MORE-1[79]	No vertebral fracture	F	65	3012	5.0	2.0	0.50 (0.4 to 0.8)
Raloxifene 60 mg	MORE-2[79]	Vertebral fractures	F	68	1539	21.0	15.0	0.70 (0.6 to 0.9)
Alendronate 5 to 10 mg	FIT-1[88]	Vertebral fractures	F	71	2027	15.0	8.0	0.53 (0.41 to 0.68)
Alendronate 5 to 10 mg	FIT-2[89]	No vertebral fracture	F	68	4432	3.0	2.0	0.56 (0.39 to 0.8)
		Subgroup T-score < -2.5	F	--	1631	4.0	2.0	0.50 (0.31 to 0.82)
Alendronate	Orwoll et al[92]	FN T-score < -2 or > -1 and a fragility fracture	M	63	241	7.1	0.8	0.11 (p = 0.02)
Risedronate 5 mg	VERT-US[94]	Vertebral fractures	F	69	1628	16.0	11.0	0.51 (0.36 to 0.73)
Risedronate 5 mg	VERT-MN[95]	Vertebral fractures	F	71	815	29.0	18.0	0.59 (0.43 to 0.82)
Calcitonin 200 IU	PROOF[98]	Vertebral fractures	F	69	557†	16.0	11.0	0.67 (0.47 to 0.97)
Rh 1-34 PTH 20 μg	Neer et al[101]	Vertebral fractures	F	69	892	14.0	5.0	0.35 (0.22 to 0.55)
Strontium 2 g	Menuier et al[104]	T-score < -2.5 and vertebral fractures	F	69	1649	24.4	17.7	0.59 (0.48 to 0.73)
Hip fracture								
Drug								
Calcium 1.2 g/vit D 800 IU	Chapuy et al[63,64]	Living in care home	F	84	3270	4.2	2.4	0.73 (p = 0.043)
HRT	WHI[75]	Healthy post-menopausal women	F	63	16 608	0.77	0.52	0.66 (0.45 to 0.98)
Raloxifene 60 and 120 mg	MORE[79]	Osteoporosis (T-score < -2.5) with or without vertebral fractures	F	67	7705	0.7	0.8	1.1 (0.61 to 1.9)
Alendronate 5 to 10 mg	FIT-1[88]	Vertebral fractures	F	71	2027	2.2	1.1	0.49 (0.23 to 0.99)
Alendronate 5 and 10 mg	FIT-2[89]	T-score < -2.5	F	--	1631	1.6	0.72	0.44 (0.18 to 0.97)
		T-score < -1.6	F	68	4432	0.8	0.65	0.79 (0.43 to 1.44)
Risedronate 5 mg	VERT-US[94]	Vertebral fractures	F	69	1628	1.8	1.4	NA
Risedronate 5 mg	VERT-MN[95]	Vertebral fractures	F	71	815	2.7	2.2	NA
Risodronate 2.5 and 5 mg	HIP[96]	70 to 80 years with osteoporosis	F	74	5445	3.2	1.9	0.6 (0.4 to 0.9)
		Subgroup prevalent vertebral fracture	F	--	--	5.7	2.3	0.4 (0.2 to 0.8)
		> 80 years with or without osteoporosis	F	83	3886	5.1	4.2	0.8 (0.6 to 1.2)
Calcitonin 200 IU	PROOF[98]	Vertebral fractures	F	69	557	1.8	1.2	0.5 (0.2 to 1.6)
rh 1-34 PTH 20 μm	Neer et al[101]	Vertebral fractures	F	69	892‡	0.74	0.0037	NA

* follow-up period when calculating incidence and relative risk: WHI[75] 5.2 years; FIT-2[89] 4.2 years extrapolated to three years; PROOF[98] five years' data extrapolated to three years; Chapuy et al[63,64] 18 months; Trivedi et al[66] five years and Neer et al[101] data 21 months. NA, not available

† total study included 1255 women of which 557 received 200 IU calcitonin

‡ total study included 1637 women of which 892 received 20 μg recombinant human parathyroid hormone

the frequency of falling by 70% among patients who had attended an emergency department with a fall-related injury.[28]

Hip protectors. Energy absorption in the soft tissue around the hip has been shown to protect against fractures of the hip,[50] partly explaining why the overweight have fewer such injuries.[51] As a result, a variety of hip-padding systems have been devised such as the energy-shunting "horseshoe",[52] crash-helmet,[53] energy-absorptive[54] and airbag types.[55] All aim to reduce the impact on the hip in a fall.[56,57] There have now been several independent RCTs published on residents of nursing homes and on the frail and elderly with visual impairment living at home, which indicate that hip protectors reduce the incidence of fracture by 34% when using pooled data.[56-58] So far, no studies have convincingly shown a general protective effect by hip protectors in home dwellers. The most obvious problem with their use appears to be that of compliance.[56,59]

Pharmacological prevention of osteoporotic fractures

Calcium and vitamin D. Calcium supplement, generally in a dose of 500 to 1000 mg daily, is known to slow the rate of loss of BMD in elderly patients with a low calcium intake.[15-17] There are studies which suggest that a calcium supplement may reduce the incidence of fractures. This regime is regarded as useful but not sufficient for the treatment of osteoporosis and fractures due to fragility.[60-62]

However, calcium in conjunction with vitamin D has been shown to reduce the incidence of hip fracture in eld-

erly dwellers in nursing homes. An RCT carried out in France included women living in care homes who were treated daily for three years with 1200 mg of calcium and 800 IU of vitamin D. They had a reduction in fractures of the hip of 29% and fewer non-vertebral fractures (24%) compared with the placebo group (Table II).[63,64] The value of calcium supplementation in elderly patients living in the community with or without a low calcium intake is uncertain. An RCT involving 389 men and women over the age of 65 years and living in the community found a reduction of more than 50% in non-vertebral fractures during daily supplementation with calcium and vitamin D over three years.[65] A British study of 2686 men and women between 65 and 85 years of age living in their own homes found that treatment with calcium and vitamin D during a five-year period reduced the risk of fracture by 22% and of fractures of the hip, forearm and spine by 33%.[66] However, when 2578 elderly healthy Dutch women were treated daily with 400 IU of vitamin D for 3.5 years, there was no effect on the risk of a fracture of the hip.[67]

One recent meta-analysis included 21 studies, of which 20 were RCTs in elderly patients. It showed that treatment with vitamin D alone did not reduce the risk of fractures, but in combination with calcium, the risk was reduced by 26% in elderly patients living in care homes.[68] This survey also found that there was no reduction in the incidence of fracture of the hip in healthy individuals living in their own home, but the risk of vertebral fractures in this group was reduced by 54%.[68] Similar results were published in another meta-analysis of 25 RCTs including 8124 individuals.[69] It is clear from these studies that supplementations of calcium and vitamin D reduce the risk of fracture in elderly people living in care homes, but that in healthy elderly people with an adequate intake of calcium the results of this regime are not convincing.[66,68,69]

Hormone replacement therapy (HRT). Several small RCTs have supported the view that oestrogen increases the BMD over a period of one to three years by a few percentage points and reduces the risk of fractures in the spine by about 50%.[70-72] One meta-analysis of 13 RCTs found a reduction of 33% in vertebral fractures after HRT[73] and other analyses, including 22 RCTs, suggested that there was a reduction of 27% in non-vertebral fractures, specifically of 40% in both the hip and wrist.[74] The Women's Health Initiative Study (WHI)[75,76] of oestrogen in combination with progestin included 161 809 healthy post-menopausal women within the different study arms. Evaluation of the risk of fracture was carried out in 16 608 over a period of 5.2 years.[75,76] At this point, the planned follow-up of eight years was cancelled when the adverse negative effects outweighed the positive features, but this length of follow-up was sufficient to show that oestrogen reduced the incidence of fracture of the hip by 34%, vertebral fractures by 34%, fragility fractures by 23%, and all types of fracture by 24% (Table II).[75] One recent meta-analysis, including four RCTs with more than 20 000 women followed for a mean 4.9 years, supported this view when noting a general reduction in fractures of 28% after HRT.[77] The Million Women Study, a prospective observational study in which 138 737 post-menopausal women were followed for 1.9 to 3.9 years, noted a reduction in the risk of fracture in current HRT users of 38%, but not in former users.[78]

However, HRT has many serious adverse effects, including vaginal bleeding, breast tenderness, deep-vein thrombosis and pulmonary embolism, stroke, heart disease and gallbladder disease, and an increased risk of breast, endometrial and ovarian cancer after long-term use.[75,76] On this basis, oestrogen is not recommended currently as the primary prevention of osteoporosis in most countries.

Selective ostrogen receptor modulator (SERM). A selective oestrogen receptor modulator (SERM), such as raloxifene, is an antagonist of oestrogen in the breast and the endometrium but an agonist in bone and lipid metabolism. Evaluation of the incidence of fracture is based on only one large RCT, the Multiple Outcomes of Raloxifene Evaluation (MORE), study involving 7705 women with osteoporosis (Table II). It reported a reduction of vertebral fractures of 30% in women without and a reduction of 50% in those with a vertebral fracture at the start of treatment, but it had no effect on non-vertebral fractures.[79] Raloxifene also lowers the frequency of breast cancer by 70%,[80] but increases the incidence of venous thrombosis and pulmonary embolism at a similar rate as HRT.[81] The ongoing Raloxifene Use for the Heart trial study will provide more data regarding the effects of raloxifene. As new SERMs are under development in phase-III trials their number will probably expand in the future.

Bisphosphonates. Etidronate was the first bisphosphonate used for the treatment of osteoporosis in doses of 400 mg daily for two weeks every three months. The increase in BMD was reported to be about 4%, with a reduction in the rate of vertebral fracture after treatment for two years,[82,83] but not after three years.[84] A recent meta-analysis, which included 13 RCTs of etidronate with a follow-up of more than one year, with nine studies including 1010 patients evaluated for vertebral fractures, reported a reduction of 40%, whereas there was no effect on other fractures.[85]

Alendronate is another bisphosphonate in which RCTs have shown a reduction in fractures.[86-88] The Fracture Investigation Trial (FIT-1) of 2027 women with osteoporosis and at least one vertebral fracture, found that the incidence of vertebral, wrist, and hip fractures was reduced by about 47%, with hip fractures by 51% and vertebral fractures by 47% compared with placebo treatment. The dose was 5 mg daily for two years followed by 10 mg daily in the third year (Table II).[88] The FIT-2 study,[88,89] which was an RCT over four years in women with a low BMD but without vertebral fractures, supported this view showing a non-significant decrease in clinical fractures ($p = 0.07$) and a reduction of 45% in new vertebral fractures during the period of study (Table II).[89] Pooled data from both studies including women with osteoporosis, strengthened this view,

concluding that treatment with alendronate for 12 to 18 months did reduce the incidence of fracture.[88-90] Another RCT of 1908 post-menopausal women with a BMD below -2 SD found that the use of 10 mg of alendronate daily reduced the risk of sustaining a non-vertebral fracture after one year by 47%.[91] The incidence of radiographically-verified vertebral fracture in men was reduced by 89% after treatment with alendronate for two years.[92] A recent meta-analysis of 11 RCTs of alendronate (10 mg daily) and with a follow-up of more than one year in which eight studies evaluated vertebral and non-vertebral fractures, concluded that the risk of sustaining vertebral fractures was reduced by 48% and of non-vertebral fractures by 49% in those who were treated with 10 mg of alendronate daily.[93]

Risedronate is a third bisphosphonate. In the Vertebral Efficacy with Risedronate Therapy Study (VERT) of 2400 women with vertebral fractures at the start of the investigation, who were given 5 mg of risedronate per day, the cumulative incidence of patients with new vertebral fractures was reduced by 65% after the first year and by 41% over three years (Table II).[94] In another report from the VERT study group treatment with risedronate (2.5 or 5 mg daily) in 1226 patients with at least two vertebral fractures reduced the incidence of this fracture by 49% over three years (Table II).[95] However, these two series did not record a reduction in fractures of the hip, but this has been shown in other RCTs with risedronate. One study of 5445 women aged between 70 and 79 years with osteoporosis found a reduction of 40% in fractures of the hip over a period of three years, reaching 60% in those with an additional vertebral fracture at the start of the trial.[96] Another meta-analysis of eight RCTs with risedronate contained five studies which evaluated vertebral fractures and seven of non-vertebral injuries. It stated that risedronate reduced the risk of vertebral fractures by 36% and of non-vertebral fractures by 27%.[97]

There are other bisphosphonates such as clodronate, tiludronate and pamidronate, but no details are available as to their influence on fractures. Ibandronate and zoledronate are two other potent bisphosphonates now undergoing clinical trials.

Calcitonin. The Prevent Recurrence of Osteoporitic Fractures (PROOF) study, an RCT over five years of 1255 post-menopausal women with osteoporosis receiving 100, 200 or 400 IU calcitonin daily, reported that 200 IU of intranasal salmon calcitonin per day reduced the risk of vertebral fracture by 31%, but that no effect was found on peripheral fractures (Table II).[98] This study must, however, be interpreted with care since 60% of individuals were lost to follow-up; doses of 100 and 400 IU had no effect and no consistent effect on the BMD and markers of bone turnover was noted.[98] A recent meta-analysis evaluating calcitonin, including 30 RCTs, contained four studies which analysed vertebral fractures and three non-vertebral fractures. It concluded that calcitonin reduced the incidence of vertebral fracture by 54%.[99]

Parathyroid hormone (PTH). Intermittent injection of PTH in individuals with osteoporosis stimulates the formation of bone, increases the BMD and reduces the risk of fracture.[100,101] In one RCT of 1637 post-menopausal women with a vertebral fracture receiving 20 or 40 μg daily, 20 μg of subcutaneous recombinant human PTH daily for a median of 19 months reduced the incidence of a new vertebral fracture by 65%, and 40 μg daily by 69% (Table II).[101] The reduction in the incidence of non-vertebral fragility fractures was 53% with both doses during the same period, while after 21 months of treatment the BMD had increased by 9% and 13% in the spine and by 3% and 6% at the femoral neck with the two doses, respectively.

Strontium. Strontium ranelate is associated with reduced bone resorption and possibly an increase in the formation of bone.[102] A reduction in the incidence of vertebral fractures has been suggested in phase II trials.[103] A recent RCT of 1649 post-menopausal women with osteoporosis and at least one vertebral fracture found that 2 g of strontium ranelate per day over three years reduced the risk of sustaining a new vertebral fracture by 49% during the first year and by 41% during the entire period (Table II).[104]

Other drugs. Fluoride is incorporated into the hydroxyapatite crystal of bone. It stimulates recruitment and activity of osteoblasts, increasing the BMD in the spine and to a less extent in the hip.[105] However, no reduction in fractures has been found and indeed these studies show an increase in the incidence of non-vertebral fractures.[105] Several other drugs are also used as a prevention for osteoporosis and fractures. Alfacalcidol and calcitriol are vitamin-D analogues which have been shown to increase the BMD.[106] A low level of vitamin K is associated with a high risk of hip fracture.[107] Menatetrenone, a vitamin K2 compound, has been shown to improve the BMD.[108] Growth hormone could hypothetically increase muscle strength and BMD. Ipriflavone seems to prevent bone loss[109] and statins have been shown in animal studies to increase the BMD.[110] None of these drugs has been subjected to a RCT with fracture as the end-point.

The role of the orthopaedic surgeon

Orthopaedic surgeons see many patients with osteoporosis. It is a silent disease without any preceding symptoms until the first fracture occurs. The diagnosis is usually made after a low-energy fracture. However, the surgeon may miss the diagnosis while concentrating on the technical aspects of the treatment of the fracture. It can no longer be regarded as acceptable clinical practice to fail to initiate the investigation of osteoporosis in a patient presenting with a low-energy fracture.

A fragility fracture is one of the strongest predictors of forthcoming fractures (Table I) and measurement of the BMD has a better predictive ability for future fractures than blood pressure has for a future stroke. Evaluation of the patient must therefore include a history of risk factors and a BMD scan. It is the responsibility of the orthopaedic sur-

geon to arrange for patients to be properly advised and investigated for osteoporosis. This does not necessarily mean that the investigation and treatment of osteoporosis are the responsibility of the orthopaedic surgeon, but referral of the patient to the appropriate physician for such evaluation is his or her responsibility.

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