Osteoporosis

Version 2 Final

Document control

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1. Introduction

Description[1]

A World Health Organisation (WHO) working group and consensus conference have defined osteoporosis as "A disease characterised by low bone mass and micro-architectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk".
Osteoporosis is a systemic skeletal disease and osteoporotic fractures can occur at any site, though the fractures classically associated with this disorder are those involving the thoracic and lumbar spine, distal radius, and proximal femur. The definition does not imply that all fractures at sites associated with osteoporosis are due to the disorder. The interaction between bone geometry and the dynamics of the fall or the traumatic event, happening in a given environment, are also important factors in causing fracture. These can happen independently of, or in association with, low bone density.

Prevalence

Estimates have been high e.g. the figure of over 2 million women in England and Wales has been quoted frequently although recent epidemiological data indicate that the figure may be closer to 1.2 million[2].

The prevalence of osteoporosis in women increases markedly with age after the menopause, because bone loss is accelerated by the loss of oestrogen production[3]. 70% of women over the age of 70 are affected.[4]

The lifetime risk of osteoporosis in men is approximately half that of women[5]. Secondary causes of osteoporosis are more common in men, accounting for 40% of cases[6].

About 180,000 symptomatic osteoporotic fractures occur each year in England and Wales.

One in two women and one in five men will suffer a fracture after the age of 50 years[7].

The number of hip fractures each year in the UK is over 60,000 and the cost to the NHS and social care services is at least £1.73 billion per year .

Fragility fracture definition

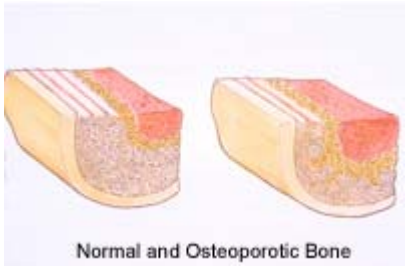
A fragility fracture occurs as a result of mechanical forces that would not ordinarily cause a fracture.
The World Health Organization has quantified this as a force equivalent to a fall from standing height or less.

About half of all fragility fractures occur in people with osteopenia (mild thinning of the bone mass, but not as severe as osteoporosis). This is because although the risk of fracture is higher in people with osteoporosis, osteopenia is more common than osteoporosis[8].
Fragility fractures occur most commonly in the vertebrae, hips, and wrists.

2. Pathology/Aetiology

Bone consists of a framework of collagen strengthened with mineral (calcium phosphate) and living cells. Normally these cells, osteoclasts and osteoblasts, resorb and reform bone in dynamic equilibrium in terms of space, degree and time. Between 10% and 30% of the adult skeleton is remodelled every year. This remodelling of bone is influenced by mechanical and electrical forces, hormones and local regulatory factors, e.g. parathyroid hormone, calcitonin.

There are two major forms of bone; compact cortical bone and trabecular medullary bone. Vertebral bodies consist predominantly of cortical bone. These two types of bone have different responses to metabolic influences and different susceptibility to fracture. Trabecular bone accounts for 80% of bone turnover.


Normal and Osteoporotic Bone

During childhood and teenage years, new bone is formed faster than bone is resorbed resulting in larger, heavier and denser bones.

Peak bone mass is achieved at about 35 years of age for cortical bone and earlier for trabecular.

After reaching its peak, bone mass decreases with age due to loss of both mineral and organic matrix but basic organisation of bone tissue is maintained.

The density of bone in the adult depends on peak bone mass and subsequent alteration of bone density due to genetic, mechanical, nutritional and endocrine factors.
Negroid race, load-bearing exercise, sex hormones (oestrogen), adequate dietary calcium tend to increases bone mass.
Collagen gene mutations, lack of exercise, early menopause (natural or iatrogenic), anorexia nervosa, increasing age, smoking, alcohol, amenorrhoea in early life and thinness tend to decrease bone mass.

2.1 Classification

Primary

Idiopathic: Occurs in children or young adults with normal gonadal function.

Type I (postmenopausal): Occurs between the ages of 51 years and 70 years and is six times more common in women than in men.
In the immediate peri-menopausal years increased bone absorption (bone loss) decreases bone mass and in later years decreased new bone formation decreases bone mass.
The rate of postmenopausal bone loss varies not only from site to site but also from woman to woman[9].

Bone loss affects trabecular (cancellous) bone much more than cortical bone due to its greater surface area available to osteoclasts and osteoblasts. Vertebral crush fractures and distal radius fractures predominate in Type I osteoporosis because bone at these sites is mainly trabecular.

Type II (involutional or senile): Occurs mainly in people over 70 years of age and is twice as common in women as it is in men. Affects both cortical and trabecular bone often resulting in fractures of the femoral neck and other fractures e.g. of the vertebrae, proximal tibia and pelvis. In women Type I and Type II may occur together.

Secondary

Accounts for less that 5% of all cases. Secondary causes are present in almost half the men presenting with osteoporosis[10]

Many clinical conditions and some drugs are associated with osteoporosis.
The most common secondary causes of osteoporosis are:

- **Endocrine:** poorly controlled thyrotoxicosis, male hypogonadism, and primary hyperparathyroidism.
- **Malabsorptive or nutritional:** inflammatory bowel disease, chronic liver disease, coeliac disease, anorexia nervosa, vitamin D deficiency.
- **Drugs:** phenytoin, phenobarbital, over-treatment with thyroxin.
- **Oral and inhaled corticosteroids:** most studies on corticosteroids have been in people taking oral treatment for more than 6 months — the effects of high-dose, short-term treatment or intermittent courses over a long period of time are less well known.
    - However, as the rate of bone loss is greatest in the first few months of administration, treatment for as short a period as 3 months may result in increased fracture risk.
    - There is evidence that fracture risk is related to the cumulative dose of corticosteroid taken (important for people taking intermittent courses of treatment).
    - There are concerns that inhaled corticosteroids may cause effects on bone mineral density (BMD), particularly when given in high doses for long periods, but there is no convincing evidence of this.
    - A Cochrane review found that in people with asthma or mild chronic obstructive pulmonary disease, inhaled corticosteroids at conventional doses, given for 2–3 years,

had no effect on BMD or vertebral fractures. Higher doses were associated with biochemical markers of increased bone turnover but data on BMD and fractures at these doses are not available[11].

- **Medroxyprogesterone acetate** (Depo-Provera®) causes a reduction in bone mineral density in many women that use it, but it is unclear whether there is an increased risk of osteoporosis and fractures in later life.

- **Others:** rheumatoid arthritis, myeloma, renal disease (including renal tubular acidosis).[12, 6, 2]

2.2 Risk factors for primary osteoporosis[6]

The risk factors that have best evidence for increasing risk for this group are shown in Table 1. Although many are not modifiable, these factors contribute to a threshold for diagnostic testing, which helps prioritise which patients should be sent for a DXA scan.

Table 1: Risk factors for osteoporosis (when no history of fracture)

Strongest risk factors	Other significant risk factors
Female sex	Caucasian origin
Age > 60 years	Early menopause
Family history of osteoporosis	Low BMI
	Smoking
	Sedentary lifestyle
	Long term (≥3 months) corticosteroid use

It is difficult to offer evidence based advice about particular combinations of risk factors which justify further investigation since the evidence is lacking, but there seems to be an additive effect of risk factors – more present means greater risk. A systematic approach to offering osteoporosis assessment to all such patients should be developed, though scarce resources should be targeted at those at highest risk to ensure the most efficient use of these resources.

It must be clear which risk is being considered: the risk of osteoporosis, or the risk of falling or fracturing. Each of these is linked and osteoporosis is only one risk factor for fracture.

Priority should be given to finding and managing patients at the highest risk of falling and experiencing a fracture. Those who have already experienced a fracture are at high risk of a further fracture. Thus patients with a previous fracture are a key target group.

The next group to target are those with osteoporosis risk who have not yet sustained a fracture.

Falls that result in a fracture

- The Royal Society for the Prevention of Accidents estimates that about 30% of people aged 65 years and over have a fall each year, increasing to 50% in people aged 80 years and older[13]. A fifth of incidents require medical attention[14].
- Fractures commonly occur in older people following a fall:
    - About 5% of older people in community-dwelling settings who fall will sustain a fracture or will need hospitalization.
    - Between 10% and 25% of falls in nursing homes and hospitals result in a fracture.
- The incidence of hip fractures in the UK is 86,000 per year, and 95% of these are the result of a fall. The cost to the NHS is £1.7 billion a year.

Non-Modifiable Risk Factors[6]

Age

BMD decreases, and consequently the risk of osteoporosis increases with age. A significant increase in prevalence with each decade after age 60 has been demonstrated. The United States National Health and Nutrition Survey (NHANES) III survey of postmenopausal women showed that the prevalence of osteoporosis in non-Hispanic white American women was 27% (50-59 years), 32% (60-69 years) and 41% for those ≥70 years. A previous estimate based on data from Rochester, Minnesota indicated a lower (though still high) prevalence – 14.8% (age 50-59 years), 21.6% (aged 60-69 years), 38.5% (70-79 years) and 70% (≥80 years.) A Yorkshire based study showed a prevalence of 24% in women aged 60-69 years.

Sex

Women are at greater risk of osteoporosis as they have smaller bones and hence lower total bone mass. Additionally, women lose bone more quickly following the menopause, and typically live longer. Osteoporosis is less common in men but is still a significant problem.

The rate of bone loss in men is less than that in women. In the Framingham Osteoporosis Study annualised percent bone loss for women was 0.86% to 1.21% at different sites and for men, 0.04% to 0.90%. Secondary causes of osteoporosis are, however, more common in men, affecting approximately 40% of cases. Excepting reproductive factors and taking into account the increased influence of secondary factors in men, the risk factors in women also apply to men.

Ethnicity

Afro-Caribbean women have a higher BMD than white women at all ages due to a higher peak bone mass and slower rate of loss. White women have a 2.5 fold greater risk of getting osteoporosis.

Reproductive Factors

A late menopause or short time from menopause to BMD measurement is associated with higher BMD. There is consistent evidence that low BMD is associated with early menopause. Consequently, women with an early menopause should be considered at higher risk of osteoporosis than others at a similar age.

BMD decreases most rapidly in the early postmenopausal years.

There is no consistent evidence that tubal ligation, parity, number of previous miscarriages, or breast feeding affects BMD.

Current use of oestrogen replacement therapy is associated with a higher BMD. Those currently taking oestrogen therapy should therefore be considered as being at lower risk than others at a similar age, unless the therapy was prescribed for osteoporosis.

Family History of Osteoporosis

Lower BMD is found in women and men with a family history of osteoporosis, a family history being defined as a history of osteoporosis or brittle bones, kyphosis ("dowager's hump"), or low trauma fracture after age 50 years as reported by the offspring.

Individual BMD decreases as the number of family members with osteoporosis increases. Overall family history is a more sensitive predictor of osteoporosis risk than maternal or paternal history alone. Prevalence of a positive history in sisters is similar to prevalence reported for mothers.

In one epidemiological study the greatest risk of categoric osteopaenia was in patients whose father had a history of osteoporosis.

Modifiable Risk Factors[6]

Weight

Weight loss or low body mass index (BMI) is an indicator of lower BMD. In addition, those in the lowest tertile of BMI have a two-fold greater bone loss than those in the highest tertile over two years. Post menopausal women with below average BMI should be considered as being at increased risk of osteoporosis.

Smoking

A meta-analysis of studies looking at the effect of smoking found that BMD in smokers was 2% lower with each increasing decade after the menopause than in non-smokers, with a 6% difference at 80 years. Men who smoke show greater bone loss at the trochanter. Female smokers have been shown to be at greater risk of hip fracture than non-smokers, with the risk increasing in line with cigarette consumption. The level of risk declines on giving up smoking, but is not significantly reduced until 10 years after cessation.

Alcohol

Evidence for alcohol as a risk factor for low BMD is inconsistent, as the majority of studies do not include subjects with excessive alcohol intake. Anecdotally, however, alcohol abusers' bone quality is poor, coupled with an increased tendency to fall and fracture.[15]

Exercise

A positive relationship between both current physical activity, physical activity in adolescence and BMD has been shown in young female Canadians (18-35 years) and in Italian middle aged women. Current exercise has been associated with higher bone density in postmenopausal English women and in Norwegian women aged 50-75 years with fractures.

However a study of an Australian population has shown that current physical activity was positively associated with BMD but that after adjustment for age, BMI, calcium intake, and quadriceps strength the relationship did not remain statistically significant. Consequently, individuals with a sedentary adolescent lifestyle should be considered at higher risk of osteoporosis. Those who currently have a sedentary lifestyle may also be at higher risk.

Diet

Past dietary intake of milk in adult pre-menopausal women (45-49 years) has been positively associated with BMD. Evidence of association between current calcium intake and low BMD is inconsistent. Vitamin D levels have been shown to be positively correlated with BMD in independent living men and women aged >80 years in Stockholm. No consistent association has been found between other dietary factors and BMD.

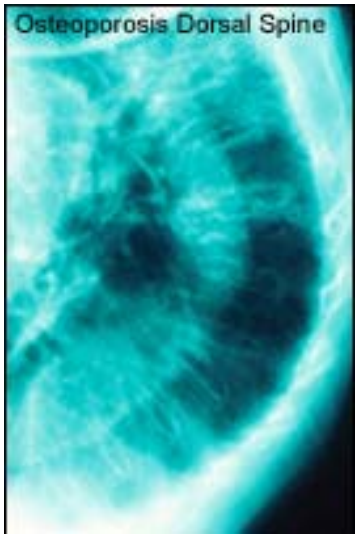
3. Diagnosis

Bone mineral density (BMD) is the major criterion used for the diagnosis and monitoring of osteoporosis. BMD differs between different sites around the body and there is only a moderate correlation between BMD at different sites. BMD of a specific site is the best predictor of fracture at that particular site.
Techniques available for measuring BMD are shown below:

Technique	Appropriate sites
Dual-energy X-ray Absorptiometry (DEXA or DXA)	Antero-posterior (AP) spine, lateral spine, proximal femur, total body, forearm, heel
Quantitative Computed Tomography (QCT)	Spine
Peripheral Dual-energy X-ray Absorptiometry (pDXA)	Forearm
Peripheral Quantitative Computed Tomography (pQCT)	Forearm
Single Photon Absorptiometry (SPA)	Forearm
Single-energy X-ray Absorptiometry (SEXA or SXA)	Forearm
Radiographic Absorptiometry (RA)	Phalanges

Other techniques are available that measure properties related to bone density. Quantitative Ultrasound (QUS) can be used to measure properties of the calcaneus related to bone quality and structure, though it cannot be used to diagnose osteoporosis or to target treatment. Biochemical markers such as resorption markers can be used to assess bone turnover.

The risk of falls and the resultant trauma are difficult to assess and predict. The WHO definition of osteoporosis therefore captures only the bone-specific estimate of fracture risk. This is best captured by bone mineral density. The WHO working group used this technique to stratify risk as follows:



Normal Bone mineral density less than 1 standard deviation below the young normal mean (T > 1)

Osteopaenia Bone mineral density between 1 standard deviation and 2.5 standard deviations below the young normal mean (T between 1 and 2.5)

Osteoporosis Bone mineral density more than 2.5 standard deviations below the young normal mean (T < 2.5)

This definition only applies to women. Recent reviews have suggested that applying the same definition to men, based on a male normative range, would have the same utility although this is not universally accepted.

T-SCORES AND Z-SCORES

Measurements of bone mineral density are often cited in terms of a ***T-score***, which is the number of standard deviations by which the patient's BMD differs from the mean peak BMD for young normal subjects of the same gender. Another measure of BMD is the ***Z-score***, which is the number of standard deviations by which the patient's BMD differs from the mean BMD for subjects of the same age.

Conventional radiographs should not be used for the diagnosis or exclusion of osteoporosis.
When plain films are interpreted as ''severe osteopaenia'' it is appropriate to suggest referral for DXA.

Differential diagnosis

- Cushing's syndrome
- Thyroid bone disease: thyrotoxicosis causes increased bone turnover with resorption exceeding formation. After many years osteoporosis may occur.
- Osteogenesis imperfecta: in the young, mild osteogenesis imperfecta and rarely osteoporosis glioma syndrome (absent family history and blue sclera absent) may be confused with osteoporosis.

4. Treatment

The aims of treatment[6]

- Reduction in the incidence of fractures
- Alleviation of fracture related morbidity

4.1 Prevention

The goals of prevention are to preserve bone mass and prevent fractures. Preventive measures are indicated in postmenopausal women and older men, patients taking long-term systemic corticosteroids, and patients at high risk (e.g. osteopenia with multiple risk factors or secondary causes).

Prevention measures for osteoporosis include exercise, nutrition and avoidance of risk factors e.g. smoking and alcohol. However there is little evidence that osteoporosis can usefully be tackled by a public health policy to influence risk factors such as smoking, exercise and nutrition. Measures to prevent falls will also reduce the incidence of fractures.

Exercise and Physiotherapy[6]

There is mounting evidence to suggest that physical exercise reduces the risk of falling in older people. Gait training, appropriate use of assistive devices, and exercise programmes with balance training have emerged as key components of exercise programmes for community dwelling older people.

A number of systematic reviews and meta-analyses have suggested that an exercise programme combining low impact weight bearing exercise and high-intensity strength training maintains bone density in men and postmenopausal women.

DIETARY DERIVED CALCIUM[6]

Two systematic reviews suggest that dietary derived calcium is as effective as pharmacologically derived sources at maintaining adequate calcium balance in postmenopausal Caucasian women.

A well conducted meta-analysis suggests that 1000 mg per day of dietary calcium leads to a 24% reduction in hip fractures.

CALCIUM AND VITAMIN D SUPPLEMENTATION[6]

Calcium supplementation using tablets is one means of ensuring an adequate calcium intake in those unwilling or unable to do so by dietary means. A daily calcium intake of 1,000 mg or more taken in tablet form is likely to reduce fracture rates by a similar rate to that seen with dietary derived sources of calcium. There is no evidence that a vitamin D supplement is needed for active people under 65 years of age. However, everyone over 65 years of age should aim to take 10µg (400 IU) daily of vitamin D. For the majority of people this can only be achieved by vitamin D supplementation. Where vitamin D deficiency has been confirmed or is likely, such as in the case of housebound individuals, a vitamin D supplement of 20µg (800 IU) is the recommended dose.

Other dietary interventions

There is no evidence that the following treatments have any beneficial effect:

- Water fluoridation
- Natural progesterone, magnesium, boron, and homeopathic remedies
- Ipriflavone - Ipriflavone is a flavinoid found in large amounts in soy-rich foods.
- Caffeine

Use of HRT in the management of osteoporosis

It is simply not known whether data from primary prevention studies can be extrapolated to treatment of osteoporosis.

4.2 Pharmacological management

Bisphosphonates are first-line drug therapy. By inhibiting bone resorption, bisphosphonates preserve bone mass and can decrease vertebral and hip fractures by 50%. To treat osteoporosis, bisphosphonates can be given orally[16]. All increase bone mineral density and decrease risk of at least vertebral fractures. Oral bisphosphonates must be taken on an empty stomach with a full glass of water, and the patient must remain upright for ≥ 30 min. They can cause oesophageal irritation. The latest bisphosphonates may be prescribed in a once-weekly regime.

Calcitonin 200 IU calcitonin intranasally in association with 1000 mg calcium plus 400 IU vitamin D per day has been shown to reduce the incidence of vertebral fractures. Unusually, a dose response relationship was not seen: neither 100 IU per day nor 400 IU per day were associated with a change in the incidence of morphometric vertebral fractures. Calcitonin has not been shown to have efficacy in reducing the incidence of non-vertebral fractures in well conducted RCTs.[6]

4.3 Duration of treatment[6]

After initiating therapy on the basis of assessment of fracture risk defined using fracture history, usually together with axial DXA measurement in the context of the patient's age, it is likely that treatment would be required on a lifelong basis. Fracture efficacy data, however, exist only for between 1-4 years, the duration of the double-blinded randomised placebo-controlled trials. Safety data do, however, exist for several years thereafter for bisphosphonates and suggest that there is unlikely to be any cumulative disadvantage to the skeleton even though they are likely to be retained in the skeleton for years. Few data exist regarding BMD or fracture risk after cessation of bisphosphonates, although one study reported increases in markers of bone turnover, without changes in BMD two years after stopping alendronate and this may indicate reactivation of processes that may ultimately result in bone loss.

5. Prognosis

20-25% of patients who suffer femoral neck fractures are dead within twelve months and the majority are functionally disabled.[17]
6.7% of women become dependent for basic activities of daily living during their lifetimes because of osteoporotic fractures.[18]

After osteoporotic hip fracture only 50% of patients aged 65 years or more return to their pre-fracture ambulatory health state.
Studies of ambulatory status and ability to perform activities of daily living following a hip fracture indicate that most physical recovery occurs during the first four months with additional but gradual improvement extending through the first year[19].

Internal fixation has become the treatment of choice for trochanteric fractures and either internal fixation or the use of a cemented or a non-cemented prosthesis is now indicated for an intracapsular fracture of the neck of femur. These improvements have not however resulted in a significant decrease in the amount of residual disability following hip fracture.

One study showed that more intensive rehabilitation (than usual as routine) post operatively did not alter recovery whereas another study of a multidisciplinary case programme post operatively showed fewer complications, fewer necessary transfers to intensive therapy units, improved ambulatory ability and fewer discharges to nursing homes.

Other studies emphasise that psychosocial intervention in the health care setting may improve patient wellbeing and the quality of care.

Recent data from Sweden suggests that hospital stay is less than 16 days for hip fracture, non weight bearing status has decreased to one day in some cases, and return home within the month has increased to 75%.

In US, 40 to 50% are discharged to their previous accommodation directly from acute stay hospital.
Bones often return to near normal within a few years but vertebral deformity of some degree often persists.

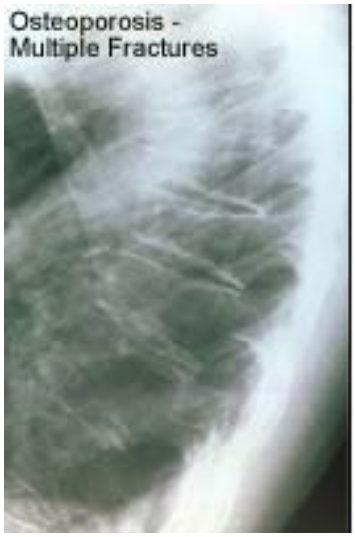
5.1 Specific Manifestations

Accelerated bone loss occurs (rarely) in childhood, adolescence, young adults and in pregnancy.

Around puberty, idiopathic juvenile osteoporosis may become manifest. This condition is usually self limiting, usually associated with periods of rapid growth of adolescence, and results in reduced growth rate, trunk shortness and possible kyphosis if there is a vertebral fracture.

6. Main Disabling Effects

Functional disability encompasses psychological and social recovery as well as physical recovery. The four main domains of functional disability are physical, mental, emotional and social disability.



Osteoporosis does not cause aches and pains except when it results in fractures.

6.1 Vertebral Fractures

Only a third of osteoporotic vertebral fractures are symptomatic[21]. Loss of body height and/or kyphosis may be the only signs of multiple vertebral fractures[20].

Back pain can persist at moderate levels for several years after spinal fracture. Additional studies are needed to determine the incidence and severity of back pain and disability soon after fractures occur and to examine the duration in greater detail[21]. Patients who suffer one vertebral fracture are likely to suffer more. Associations with back pain and disability are greater in magnitude for new vertebral fractures than for prevalent fractures.

In osteoporosis, acute compression fracture usually occurs during some relatively ordinary activity, e.g. lifting or getting out of chair. Fracture usually affects one vertebra (T8-L3) and is associated with onset of pain (in two thirds of cases) which limits coughing, respiration, activities and spinal mobility. The spine is tender at the level of the fracture and X-rays usually show fracture immediately, but in some cases compression may develop over a few months (Kummel's disease).

Severe local back pain usually subsides within a few months but persistent pain, often in the low back, may also occur due to the altered spinal dynamics of an increase in the normal lumbar lordosis which compensates for the kyphosis caused by the compression fracture and enables the patient to stand erect.

Over twenty five years ago it was recognised that 5-10% of patients present with bone pain along the whole length of the dorso-lumbar spine. The pain is present when erect and there is tenderness to pressure over affected levels of the spine, the ribs, metaphases of large bones and around the knee of the foot. No explanation for this pain was available though there was speculation about its cause. It was then commented that this tends to spontaneously resolve within four or five years[22].

Recent treatments for kyphosis and back pain due to osteoporotic wedge fractures include vertebroplasty and balloon kyphoplasty. Vertebroplasty is a minimally-invasive procedure whereby a needle is inserted from the back, along the pedicle into the body of the vertebra. Bone cement is then injected which strengthens the bone. In balloon kyphoplasty, a ballon is first inserted via the needle and inflated, reducing the kyphosis.

The balloon is then removed and cement injected, strengthening the bone and maintaining the corrected position. Both procedures have their potential complications but pain relief has been reported in many studies, with vertebroplasty alone possibly giving better results.[23]

6.2 Hip Fractures

Half of osteoporotic hip fractures cause some loss of independence[24].

Walking – it is reported that 22% of patients with a hip fracture returned to pre-fracture ambulation at six months post fracture and 76% of patients walked in some manner (independently, walking stick, Zimmer frame, crunches or two sticks) at six months post fracture.

Another study indicated that 93% of post hip fracture patients walk in some manner at six months post fracture but suggested limitation of walking distance and the presence of significant limp.

Activities of daily life – one study showed that 19% require long term care in a nursing home as a direct result of a hip fracture.

Studies show that 10-20% of women who sustain a hip fracture are functionally dependent in some activities of daily life as a result of the fracture.

One study shows that of patients living at home prior to hip fracture, 80% returned home within the first year and 82% of these achieved independence in the activities of daily living.

Another study which included patients who were in institutions shows that 55% of patients did return to pre-fracture ability to perform basic life activities. Functional recovery is greatest in the first year and additional recovery is not likely beyond two years post-fracture.

Factors affecting rehabilitation of hip fracture

Measures of disability including psychosocial measures of performance indicate that far less patients with fracture of hip return to pre-fracture physical, mental, emotional and social functional status.
Institutionalisation and loss of function are most likely in patients who have pre-existing impairment of mental status, coexisting medical conditions or functional disability prior to fracture.

Institutionalisation and loss of function are most likely in patients who have pre-exiting impairment of mental status, coexisting medical conditions or functional disability prior to fracture.

In the very elderly the relationship between mental status and the recovery following hip fracture is clearly indicated. Depression impedes recovery processes if a person is not motivated to obtain adequate rehabilitation.

Acute confusional state, whether produced by organic or psychosocial factors, has a significant impact on the recovery of older patients with hip fracture.

6.3 Wrist Fractures

Wrist fractures (distal radial fractures) tended to occur in thin oestrogen deficient women and limit the use of the extremity for four to eight weeks. The morbidity of wrist fracture is less than for vertebral, hip or proximal humerus fracture, but not negligible There is a 14.2% risk of further fractures within the 10 years following a wrist fracture. This is less than for other fractures- 25.7% after spine fracture, 24.9% after hip fracture, and 23.7% further fracture in the 10 years following humerus fracture. It may be, however, the first indication of osteoporosis and the instigator for further screening. These are the most common fractures in women referred for DEXA scanning.[25]

6.4 Ankle Fractures

Osteoporotic ankle fractures tended to occur in the obese and smokers.

Coexisting health disorders are associated with fractures other than of the wrist or ankle[26].

7. References

[1] Management of Osteoporosis. A National Clinical Guideline, Scottish Intercollegiate Guidelines Network, June 2003
[2] NICE, 2005a
[3] Stevenson et al, 2005
[4] Atik O, Gunal I, Korkusuz F. Burden of osteoporosis. Clinical Orthopaedics & Related Research. 443:19-24, 2006
[5] Royal College of Physicians and the Bone and Tooth Society of Great Britain, 2000
[6] SIGN, 2003
[7] National Osteoporosis Society, 2006
[8] Schuit et al, 2004; Raisz, 2005
[9] Kanis J A and the WHO study group. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis: synopsis of a WHO report. 1994
[10] Woolf Treating Osteoporotic Fractures. GP Oct 1997
[11] [Jones et al, 2002].
[12] National Osteoporosis Society, 2002a
[13] National Collaborating Centre for Nursing and Supportive Care, 2004
[14] Gillespie et al, 2003.
[15] Papaioannou A et al 2009. Risk factors for low BMD in healthy men age 50 years or older: a systematic review. Osteoporosis International. 20(4):507-18
[16] The Merck Manual for Healthcare Practitioners
[17] Oxford Textbook of Medicine (CD) Oxford University Press. Published in pring form by PasTest Third Edition 1996 Editors: DJ Weatherall, JGG Ledingham and DA Warrell Osteoporosis
[18] Tosteson A Quality of life in the economic evaluation of osteoporosis prevention and treatment. Spine. Vol. 22, No. 245, pp 58s - 62s.
[19] Craik R I Disability following hip fracture. Physical therapy. Vol. 74. No 5, May 1994.
[20] Osteoporosis. NIH Consens Dev Conf Consens Statement 1984 Apr 2-4;5(3):1-6.
[21] Ross P D et al. Pain and disability associated with new vertebral fractures and other spinal conditions. J Clin. Epidemiol.Vol. 47, N03, pp 231 - 239, 1994
[22] Frost H M Managing the Skeletal Pain and Disability of Osteoporosis. Orthopaedic Clinics of North America. Vol. 3, No 3. November 1972.

[23] Eck JC, Nachtigall D, Humphreys SC, (al, 2009) Hodges SD. Comparison of vertebroplasty and balloon kyphoplasty for treatment of vertebral compression fractures: a meta-analysis of the literature. Spine Journal: Official Journal of the North American Spine Society. 8(3):488-97; 2008
[24] Selby P 3rd Bandolier conference on osteoporosis 1997
[25] Hodsman AB, Leslie WD, Tsang JF, Gamble GD. 10-year probability of recurrent fractures following wrist and other osteoporotic fractures in a large clinical cohort: an analysis from the Manitoba Bone Density Program. Archives of Internal Medicine. 168(20):2261-7, 2008
[26] Honaken R et al. Relationships between risk factors and fractures differ by type of fracture: A population-based study of 12192 peri-menopausal women. Osteoporosis international (1998) 8:25-31.