Clinical review

Osteoporosis and its management

Kenneth E S Poole, Juliet E Compston

Fractures caused by osteoporosis affect one in two women and one in five men over the age of 50, resulting in an estimated annual cost to the health services of around £1.8bn (€2.7bn; $3.5bn) in the United Kingdom and €30bn in all of Europe.[1 2] Most patients with osteoporosis are managed in primary care, but a minority will benefit from referral to specialised centres. In recent years considerable advances have been made both in the identification of people at high risk of fracture and in therapeutic options to reduce the risk of fracture. This review focuses on these areas and also on the partnership that is required between primary and secondary care to optimise the management of patients with osteoporosis.

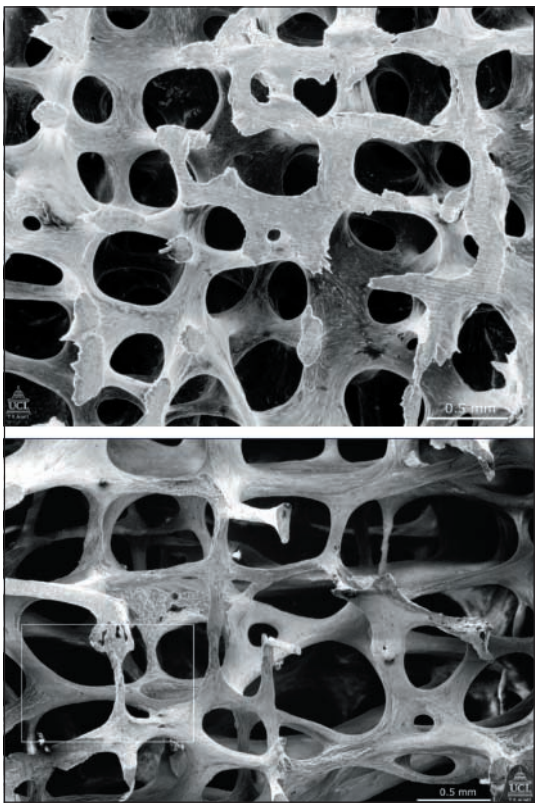

Fig 1 Scanning electron micrographs to show the structure of L3 vertebra in a 31 year old woman (top) and in a 70 year old woman (bottom). Note that many of the plate-like structures have become converted to thin rods

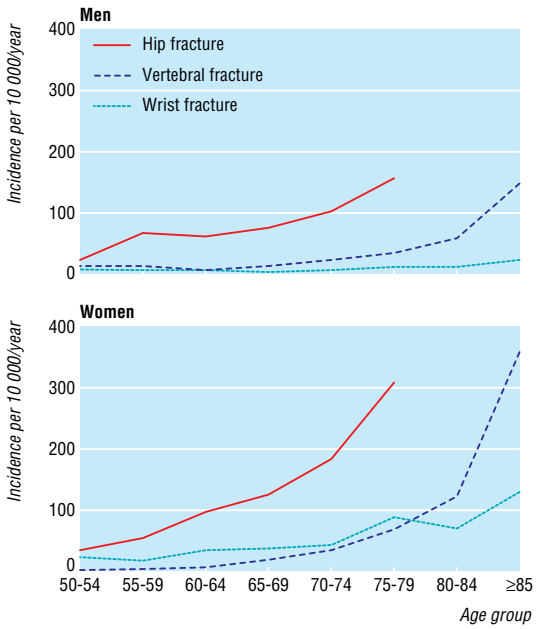

Fig 2 Epidemiology of osteoporotic fractures in men and women. Reprinted with permission[28]

What is osteoporosis?

Osteoporosis results from reduced bone mass and disruption of the micro-architecture of bone (fig 1), giving decreased bone strength and increased risk of fracture, particularly of the spine, hip, wrist, humerus, and pelvis. The risk of fractures increases steeply with age (fig 2) and most of those affected are over 75.[1 2] Globally, osteoporotic fractures caused an estimated 5.8 million disability adjusted life years in the year 2000[w1] and are also associated with increased mortality. Hip fractures (fig 3) result in loss of independence for at least a third of people with osteoporosis, and vertebral fractures (fig 4) cause height loss, chronic pain, and difficulty with normal daily activities.

What causes osteoporosis?

Age related bone loss starts in the fourth or fifth decade of life (fig 5). It occurs as a result of increased bone breakdown by osteoclasts (fig 6) and decreased

References w1-w17 are on bmj.com

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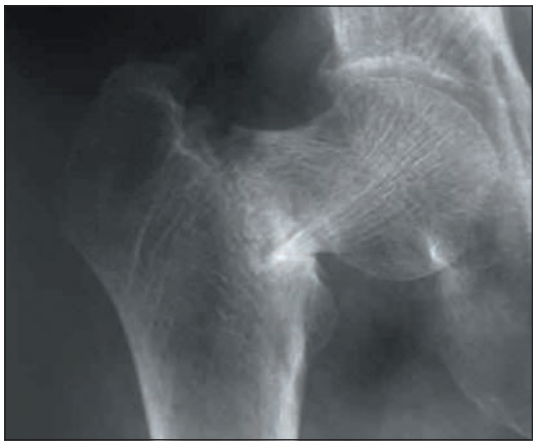


Fig 3 Fracture of the femoral neck

bone formation by osteoblasts.[3] The role of oestrogen deficiency in menopausal and age related bone loss in women is well documented, and bone mass in elderly men is also positively related to oestrogen levels. Vitamin D insufficiency and secondary hyperparathyroidism are common in elderly people and may contribute. Other possible factors are reduced physical activity with ageing and decreased production of insulin-like growth factors.

Genetic factors have a strong influence on peak bone mass, which is attained during the third decade of life and is an important determinant of bone mass later in life. Nutrition, particularly calcium and vitamin D intake, hormonal status, and physical activity also influence peak bone mass.

Although low bone mass has a major role in the pathogenesis of fracture, factors related to falling—risk of falling, protective response, and energy absorption—make an important contribution. In addition, aspects of bone composition and structure that may not be captured by bone mineral density measurements, such as bone size and geometry, and bone structure and material, contribute to increased bone fragility.

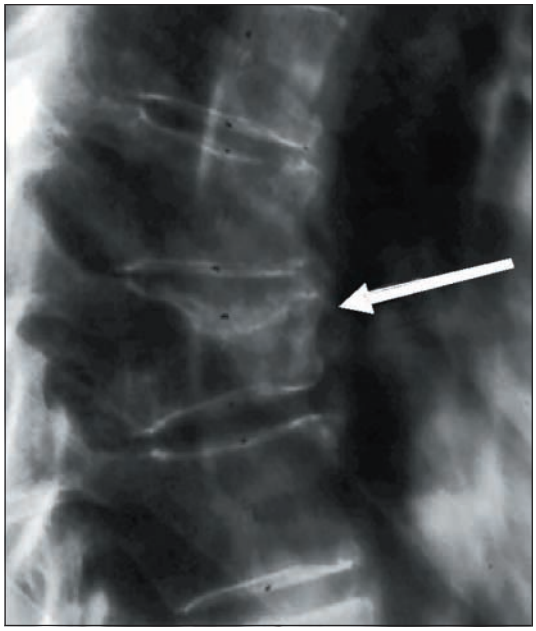


Fig 4 Vertebral fracture

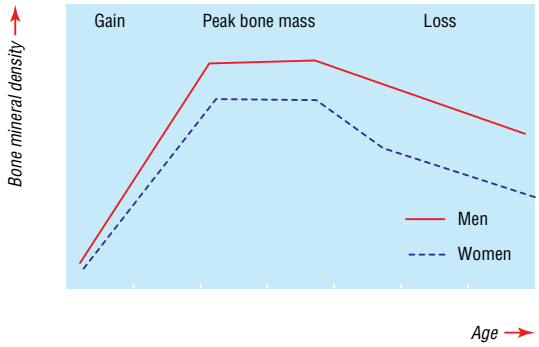


Fig 5 Age related changes in bone mass throughout life in women and men. Peak bone mass is attained in the third decade of life and age related bone loss probably starts around the age of 40 in both men and women. In women, bone loss accelerates around the time of the menopause

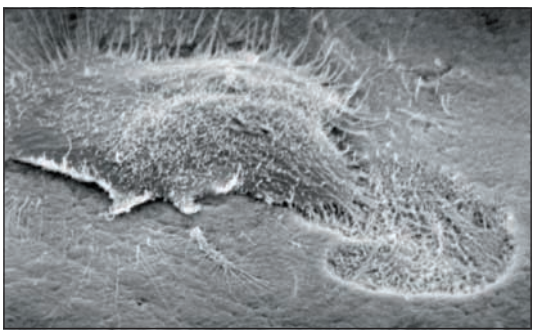
Who is at risk of osteoporosis?

Lower peak bone mass, increased bone loss at the menopause, and greater longevity all confer a greater risk of osteoporosis in women than in men, and the disease is most commonly seen in postmenopausal women. Some of the risk factors for osteoporosis (box 1) are at least partially independent of bone mineral density,[w2-w6] whereas the effect of others on fracture risk is mediated solely through reduced bone mineral density. Oral glucocorticoids, which are taken by about 1% of the population and 2.5% of those aged over 75, are a common cause of osteoporosis, and there are specific national guidelines for the prevention and management of glucocorticoid induced osteoporosis (see additional educational resources box).

How does osteoporosis present?

Osteoporosis often presents as a clinically evident fracture. A low trauma fracture (following a fall from standing height or less) in someone aged over 45 should trigger the suspicion of osteoporosis. In other cases, osteoporosis may present as backache, height loss, spinal deformity, or radiological osteopenia.

Although most fractures due to osteoporosis present clinically, vertebral fractures may be asymptomatic in as many as two thirds of patients.[4] It is important to detect these fractures since they carry a high risk of further fractures in the spine and elsewhere.[5] Lateral x rays of the spine should be considered in patients with height loss or spinal deformity.



T R ARNETT

Fig 6 Scanning electron micrograph of an osteoclast resorbing bone

Methods

We searched PubMed with the terms osteoporosis plus randomised controlled trials (124 hits), systematic reviews (118), meta-analyses (218), and Cochrane database (15). We also searched using the terms risk factors plus osteoporosis and systematic reviews (34) or meta-analyses (58). We searched the "epub ahead of print" sections of the relevant specialist journals.

Who should be treated?

The World Health Organization's definition of osteoporosis is based on bone mineral density in the spine and proximal femur measured with dual energy x ray absorptiometry (DXA). Osteoporosis is classified as a bone mineral density 2.5 or more standard deviations below normal peak bone mass—that is, a T score ≤ -2.5.[6] Other techniques—for example, ultrasound of the calcaneus and peripheral DXA measurements—cannot be used in the same way to diagnose osteoporosis but are useful as a preliminary assessment of risk where access to axial DXA is inadequate.

Box 1: Risk factors for osteoporosis

Independent of bone mineral density

Age
Previous fragility fracture
Maternal history of hip fracture
Oral glucocorticoid therapy
Current smoking
Alcohol intake ≥3 units/day
Rheumatoid arthritis
Body mass index ≤19
Falls

Depending on bone mineral density

Untreated hypogonadism
Malabsorption
Endocrine disease
Chronic renal disease
Chronic liver disease
Chronic obstructive pulmonary disease
Immobility
Drugs (aromatase inhibitors, androgen deprivation therapy)

Ongoing research

- Evaluation of new treatments, including:
  - A human monoclonal antibody to the receptor activator of NFkB ligand (RANKL), which is given subcutaneously once every six months
  - Oral calciomimetic drugs that stimulate intermittent production of parathyroid hormone
  - Selective oestrogen receptor modulators with mixed oestrogenic and anti-oestrogenic effects
  - Inhibitors of sclerostin, a protein produced by bone that is a negative regulator of bone formation, and its signalling pathway
- Investigation of the causes and management of poor compliance and persistence
- Assessment of the long term effects of anti-resorptive treatments on bone strength

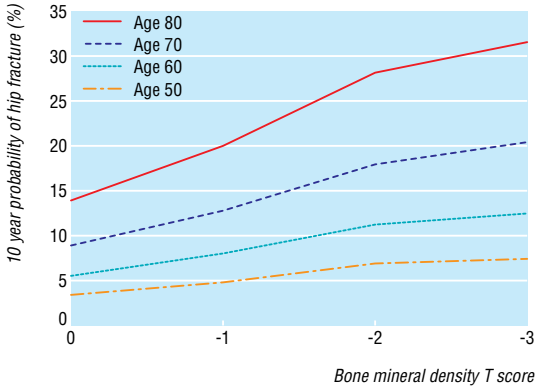


Fig 7 Age affects fracture risk independently of bone mineral density. For any given bone density, the fracture probability increases with age. For example, at a T score of –2, the 10 year hip fracture probability at the age of 50 is around 5% but at the age of 80 it is around 30%[29]

Although osteoporosis indicates a high likelihood of fracture, many fragility fractures occur in people with bone density values above the defined level.[7] Fractures can be better predicted by adding clinical risk factors that contribute to fracture risk independently of bone mineral density (fig 7; box 1).[8] This approach is being developed under the auspices of the WHO and will be delivered in the form of an algorithm that enables the probability of a fracture to be calculated from clinical risk factors with or without bone mineral density values. Intervention thresholds based on cost effectiveness[w7] can be used to make a decision about treatment.[9]

People who have already had a fragility fracture are at greatly increased risk of sustaining a further fracture,[5] and pharmacological intervention should be started promptly in such cases. Bone density measurement is not always required to confirm the diagnosis of osteoporosis, particularly in older patients, but is useful where the trauma preceding the fracture is uncertain or where other causes of fracture are suspected. Secondary causes of osteoporosis should be excluded (box 2).

Management of osteoporosis

Non-pharmacological measures

Falls have an important role in the pathogenesis of fragility fractures, particularly in frail and elderly people. Multiple medical and environmental factors increase

Box 2: Routine investigations to exclude secondary causes of osteoporosis

- Full blood count and erythrocyte sedimentation rate
- Liver and renal function tests
- Bone function tests (calcium, phosphate, and alkaline phosphatase)
- Serum immunoglobulins and paraproteins, urinary Bence-Jones proteins
- Thyroid function tests

Pharmacological interventions for osteoporosis

Intervention	Dosing regimen	Route of administration	Licensed indication
Alendronate	70 mg once weekly, or 5 mg or 10 mg once daily	Oral	Postmenopausal osteoporosis; glucocorticoid-induced osteoporosis; osteoporosis in men
Etidronate	400 mg daily for 2 weeks every 3 months	Oral	Postmenopausal osteoporosis; glucocorticoid-induced osteoporosis
Ibandronate	150 mg once monthly	Oral	Postmenopausal osteoporosis
	3 mg once every 3 months	Intravenous injection	Postmenopausal osteoporosis
Risedronate	35 mg once weekly, or 5 mg once daily	Oral	Postmenopausal osteoporosis; glucocorticoid-induced osteoporosis
Raloxifene	60 mg once daily	Oral	Postmenopausal osteoporosis
Strontium ranelate	2 g once daily	Oral	Postmenopausal osteoporosis
Teriparatide	20 μg once daily	Subcutaneous injection	Postmenopausal osteoporosis
Parathyroid hormone 1-84	100 μg once daily	Subcutaneous injection	Postmenopausal osteoporosis

the risk of falling and many of these are modifiable. Multifaceted interventions have been shown to reduce the frequency of falling but not fractures.[10]

Lifestyle measures to improve bone health include maintaining adequate dietary calcium intake and normal vitamin D status.[w8 w9] Appropriate levels of exercise should be recommended and smoking and alcohol abuse discouraged.[11] Physiotherapy and pain relief are important in managing fractures.

Pharmacological interventions

Therapeutic options for osteoporosis have increased considerably over recent years. Although most patients with osteoporosis can be managed in primary care, some patients benefit from specialist assessment: younger men and women, patients who continue to fracture despite treatment, and those who require assessment for anabolic treatments. Anabolic and intravenous treatments are generally instigated by hospital specialists; thereafter, shared care between primary and secondary organisations is appropriate.

Currently licensed treatments (table) include the bisphosphonates, raloxifene and hormone replacement therapy (which prevent bone resorption), strontium ranelate (uncertain mechanism of action), and parathyroid hormone peptides (anabolic). Without head to head comparison trials with fracture end points, the efficacy of these drugs cannot be directly compared. Some, but not all, have proved efficacy against vertebral and non-vertebral fractures, including hip fractures,[w10] and this is an important factor influencing choice. Safety, tolerability, and cost are important considerations, and NICE (the National Institute for Health and Clinical Excellence) is currently assessing the cost effectiveness of different interventions in the primary and secondary prevention of osteoporotic fractures.[w11]

Bisphosphonates

Alendronate, etidronate, ibandronate, and risedronate are approved for treating postmenopausal osteoporosis. Alendronate, etidronate, and risedronate are approved for glucocorticoid induced osteoporosis, and alendronate is approved for osteoporosis in men. Because alendronate and risedronate have been shown to reduce vertebral and non-vertebral fractures, including hip fractures,[12–15 w12 w13] they are considered first line options for preventing postmenopausal osteoporosis. Oral bisphosphonates must be taken fasting, with a full glass of water, and the individual must be upright and stay sitting or standing without taking food or drink for the next 30-60 minutes. Bisphosphonates are generally well tolerated but may be associated with upper gastrointestinal side effects, particularly if the dosing regimen is not adhered to.

An intravenous formulation of ibandronate is approved for postmenopausal osteoporosis. It is given as an injection over 15-30 seconds every three months. Antifracture efficacy has not been directly shown for this formulation or for the oral 150 mg once monthly regimen, but it is assumed from a bridging study based on changes in bone mineral density.[16 17]

Strontium ranelate

Strontium ranelate (a sachet mixed with water and taken daily) reduces vertebral and non-vertebral (including hip) fractures in postmenopausal women with osteoporosis.[18 19] Adverse events are generally mild and include diarrhoea and headache. The spectrum of anti-fracture efficacy of strontium ranelate makes it an alternative front line option to alendronate or risedronate,[w14] particularly in people for whom these drugs are contraindicated or are not tolerated.

Patient's story

My name is Jean Marsh and I am 73 years old. I had "sailed" through the menopause by the age of 45 and led an active life. My health was excellent until I was 58 years old, when one morning I noticed a dull ache between my shoulder blades. This got progressively worse and I saw my GP a few days later. He couldn't identify the problem and gave me painkillers. Later that evening I was in terrible pain. The next day I asked for an x ray and this showed collapse of the T7 vertebra.

My GP gave me a leaflet from the National Osteoporosis Society, which I joined immediately. Of the free booklets that I received, *How to Cope* was wonderful and I remember crying with joy that someone understood what I was going through.

The pain lasted for about 10 weeks, lessening a little as the weeks went by. I was terrified of falling, so going out was difficult. I couldn't lie down in bed and had to sleep propped up by lots of pillows. Clothes were difficult because of my new shape.

I was prescribed etidronate followed by HRT. My bone density increased during this time, and my fear of falling gradually disappeared. I now lead a very busy, normal life. I do have a weakness in my spine, which aches when I do too much gardening, but other than that I am now fine.

Tips for non-specialists

Patients at risk of osteoporosis can be identified by using clinical risk factors

Treatment should be started as rapidly as possible in patients presenting with a fragility fracture

The patient's preference is an important consideration in choosing treatment options

Compliance and persistence with treatments for osteoporosis are poor but can be improved by better patient education

Audit should focus on high risk groups, such as patients taking glucocorticoids and patients with previous fragility fracture

Raloxifene

Raloxifene reduces the risk of vertebral fractures, but has not been shown to prevent fractures at other sites.[20 w15] Side effects include hot flushes, leg cramps, and a threefold increase in the relative risk of venous thromboembolism. Raloxifene also protects against breast cancer.[21] It can be regarded as a second line option in younger postmenopausal women with vertebral osteoporosis.

Parathyroid hormone peptides

Teriparatide (recombinant 1-34 parathyroid hormone), given as a subcutaneous daily injection of 20 µg, reduces vertebral and non-vertebral fractures in postmenopausal women with osteoporosis.[22 w16] Preotact, the full 1-84 parathyroid hormone peptide, has recently been approved and is given in the same way in a daily dose of 100 µg. Neither of these interventions has been shown to reduce hip fractures. Because they cost more than other options, they are reserved for patients with severe osteoporosis who are unable to tolerate or seem to be unresponsive to other treatments.

Additional educational resources

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Bone Research Society website picture gallery (www.brsoc.org.uk/gallery/default.htm)

Information resources for patients

National Osteoporosis Society (www.nos.org.uk)—UK charity that raises awareness of osteoporosis and provides patient support and education. The website contains links to the *BMJ*, several professional and patient societies, and the Department of Health and a number of educational resources for patients and professionals

International Osteoporosis Foundation (www.iofbonehealth.org)—a non-profit organisation that advocates the early prevention, diagnosis, and treatment of osteoporosis and related bone diseases and provides educational material for patients and healthcare professionals. A comprehensive teaching package on vertebral fractures is provided on this website

Summary points

Fragility fractures caused by osteoporosis affect one in two women and one in five men over the age of 50

Vertebral fractures in particular remain under-recognised and under-treated and are associated with poor health outcomes

A range of pharmacological treatments is effective in reducing the risk of fracture

Poor patient compliance and persistence with prescribed treatments for osteoporosis are common but may be improved by better patient education

A minority of patients will benefit from assessment in secondary care for starting anabolic treatments or interventional techniques, particularly when other treatments have failed

Hormone replacement therapy

Because the risk-benefit balance of hormone replacement therapy is generally unfavourable in older postmenopausal women, it is regarded as a second line treatment option.[23] It is an appropriate option in younger postmenopausal women at high risk of fracture, particularly those with vasomotor symptoms.

Calcium and vitamin D

The available evidence does not support a role for calcium and vitamin D alone in preventing osteoporotic fractures, except in institutionalised elderly people.[24 25 w17] Calcium and vitamin D supplements should be prescribed with other treatments for osteoporosis since the evidence base for their efficacy in preventing fractures is derived from studies in which calcium and vitamin D were routinely administered.

Monitoring of treatment

Whether treatment response should be monitored and, if so, whether bone density measurements or biochemical markers should be used, is unclear. Compliance and persistence with osteoporosis treatments need to be improved[26]; possible approaches include better patient education and the use of intermittent dosing regimens, such as once weekly or once monthly oral bisphosphonate therapy and three monthly intravenous ibandronate. Even longer dosing intervals are expected in the near future, with the likely approval of once yearly intravenous zoledronic acid.[27]

We thank T R Arnett, University College London, for the scanning electron micrographs (figures 1 and 6).

Competing interests: KESP has been reimbursed by the Alliance for Better Bone Health for attending a scientific conference. JEC has received payment for consultancy work and speaking

engagements from Procter & Gamble, Eli Lilly, GSK/Roche, Amgen, Pfizer, Servier, Shire, Novartis and Nycomed and has been reimbursed for attending scientific conferences by Procter & Gamble, Eli Lilly, and Servier. She receives funding for a grant from Procter & Gamble and has acted as an expert witness in several cases of glucocorticoid-induced osteoporosis and in an alendronate patent dispute.

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Another inconvenient truth?

Al Gore flew into New Zealand for half a day this week amid protests that he should have come by bicycle or, at the very least, by train. But it did make me think—which, after all, is the point.

I recently attended the European Respiratory Society's 16th annual congress in Munich, along with 17 239 others. I travelled further than most, and it cost 3700 kg of CO_2 just for me. Thankfully, then, fewer than 5% of attendees at the Munich meeting were from my part of the world. But what of the carbon costs of the meeting? Using the statistics from the society's website, one can determine the country of origin of most attendees. Assuming 170 g of CO_2 per km travelled for plane passengers (140 g/km for those who drove, and only 52 g/km for train passengers), the total carbon footprint for the travel is 3.92×10^6 kg CO_2. A mature tree absorbs about 5 kg CO_2 a year, and so 784 000 trees or 784 hectares at 1000 trees per hectare for a year will be needed to offset the travel.

To make matters worse the 4469 abstracts presented at the congress required a 2.1 kg book to hold them. The 17 240 attendees required 36.2 metric tonnes of tree in the form of paper, not to mention the kilos of rubbish in the complementary satchel. And assuming they were all taken home, that represents another 36.2 tons or 517 people equivalents (at the pre-obesity epidemic average of 70 kg per person) that had to be hoisted aloft.

I've planted a tree, which will take 740 years to offset my trip this year.

Mr Gore is right that this isn't sustainable; nor is it really necessary. Let's be honest, when did you last learn anything really important at a large meeting? Of course, it's the corridors and bars that are important. So my suggestion for next year is that we all get together, preferably by bicycle or at the worst train, somewhere rural with cheap accommodation and a large bar and chat about matters respiratory while planting trees. Let's see, 18 000 delegates for 4.5 days, 50 trees per person per day. . . .

Julian Crane *professor (heavy carbon footprint, reforming) (crane@wnmeds.ac.nz),* Brent Caldwell *research fellow (light carbon footprint, did all the work), Department of Medicine, Otago University, Wellington, New Zealand.*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. Please submit the article on http://submit.bmj.com Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.