Osteoporosis

THE number of fractures of the hip, the wrist and the spine increase every year. People live longer and the age specific incidence of fractures has increased. As is made clear in this review, the reason for this is unknown. Fractures of the neck of the femur have almost doubled in incidence at every age in the past 30 years.

The incidence of osteoporosis steadily increases by about 0.5% annually from around the age of 40 years, but in women there is an accelerated increase to about 3% a year after the menopause. This is especially rapid after oophorectomy. Inadequate activity, corticosteroids, anticonvulsants, and a gastrectomy increase the rate of bone loss, as do hyperthyroidism and a parathyroid adenoma. A reliable method of estimating the level of parathyroid hormone has become available recently. Thiazide diuretics tend to protect against loss of calcium, but loop diuretics have the opposite effect. Tobacco and alcohol are thought to be deleterious to the osteoblasts, thus making bones fracture more easily. Thin women are particularly liable to suffer fractures, as they are 'not well padded'; in fatter women adipose tissue converts adrenal hormones into oestrogens, thus reducing the bone loss associated with the menopause.

During the past three years effective methods of assessing bone density have been established, using dual contrast photon absorptiometry. This is useful for research, but is not a practical method of assessing the risk of fracture in a patient. Apart from mineral content, the architecture of bones also affects their strength.

In 80% of patients with osteoporosis no cause can be found and no treatment is available. Oestrogen therapy, combined with progestogens, undoubtedly prevents the rapid post-menopausal bone changes, but it does not induce recalcification. Long term effects on the breasts and the vascular system still require elucidation. It has been suggested that patients should add calcium supplements to their diets, but studies of this have produced conflicting evidence. Diets grossly deficient in calcium and vitamin D undoubtedly induce osteomalacia, but normal diets contain adequate amounts of these substances. Excessive vitamin D undoubtedly induces renal damage. Fluoride salts (in very much larger quantities than required to prevent dental caries) increase bone density, but are very toxic. Calcitonin stimulates the osteoblasts, but would require several daily injections, and this is clearly not practical.

The most important practical advice we can give to our patients is to ensure they take reasonable precautions to avoid falls, such as having adequate light in passages, and no loose carpets or mats.

(G P)

Source: Cooper C. Osteoporosis — an epidemiological perspective: a review. *J R Soc Med* 1989; **82**: 753-757.

Contributors: Frank Sullivan, Glasgow; Jonathan Anderson, Glasgow; J Holden, St Helens; Moya Kelly, Glasgow; Gus Plaut, York.

INFECTIOUS DISEASES UPDATE

Pneumococcal vaccination

Pneumococcal vaccine is not widely used but it should be considered in certain specific situations. The most important is for those patients who are undergoing splenectomy. Following splenectomy, pneumococcal pneumonia, septicaemia and meningitis are more common and vaccination prior to splenectomy is recommended. Further booster doses are not normally given but chemoprophylaxis with penicillin should be considered, perhaps indefinitely. It is especially important to inform these patients of the danger of serious pneumococcal infection and to advise them to seek prompt medical care when febrile illnesses occur. The place of pneumococcal vaccine in other conditions is less well defined but it can be used for example in elderly or debilitated people or in the immuno-compromised, such as those with the acquired immune deficiency syndrome.

The benign malarias

Drug prophylaxis against malaria is mainly directed towards the malignant falciparum type. This type rarely causes illness more than four weeks after exposure or, four weeks after discontinuing prophylaxis, where there is partial drug resistance. The benign *Plasmodium ovalii* and *vivax* have a dormant or hypnozoite phase which allows parasites to persist in the liver. Illness then can occur months or sometimes years after leaving the endemic area. It is important to consider this diagnosis when delayed fever occurs and also to advise travellers that these benign forms will only be prevented for the duration of drug prophylaxis. It is possible to try and eradicate residual liver hypnozoites with a course of primaquine when ordinary prophylaxis has been discontinued. However, the risk of side effects usually makes this inappropriate. *Plasmodium vivax* infection is a particular problem in those returning from the Indian sub-continent and *P ovalii* in those returning from Africa, where it may be on the increase, especially in West African countries.

Testing for viral hepatitis

In addition to testing for both hepatitis A and B virus markers, many virus laboratories now have available tests for both C and D (delta) infection.

Serological tests for hepatitis A infection include detecting immunoglobulin M (IgM) which is the marker of acute infection and immunoglobulin G (IgG) which is helpful in confirming past infection. The IgG test is useful in determining whether a positive IgG test can obviate the need for giving immunoglobulin to those such as travellers to countries where food and water hygiene is poor. Many travellers over the age of 50 years are immune following infection which may have been asymptomatic.

Useful tests for hepatitis B include surface antigen (HbSag) and cor IgM. HbSag is present in both acute and chronic infections, whereas cor IgM, consistently present in acute cases, is only occasionally positive in carriers. The presence of E antigen denotes high infectivity. E antigen carriers are thought to be those in whom complications such as cirrhosis and hepatoma are most likely to occur.

Antibodies against hepatitis C, which like hepatitis B is spread sexually or through blood or blood products, become detectable around six weeks after infection. With current tests therefore it is not possible to make the diagnosis during an acute hepatitis C illness. However, if serological evidence of this infection is sought during convalescence it can explain a substantial proportion of sporadic non-A non-B hepatitis. Hepatitis C infection can take place concurrently with hepatitis B.

Hepatitis D infection (delta) has been discussed in a previous update. It cannot occur without co-existent hepatitis B infection with which it may be concurrent. Infection later on can explain some apparent hepatitis B 'relapses'.

Epidemic non-A non-B hepatitis spread by the faecal–oral route that causes both sporadic cases and outbreaks especially in Africa and Asia is due at least in part to hepatitis E for which serological testing may be available in the near future.

Contibuted by Dr E Walker, Communicable Diseases (Scotland) Unit, Ruchill Hospital, Glasgow G20 9NB (041 946 7120), from whom further information about the current topics can be obtained.