NHS. For the present measurement of bone mass in the over 60s should be limited to those patients with a clinical indication and in whom decisions about treatment will be influenced by the result. Indications should include a request from the well informed patient herself and a need to confirm (or dispel) a radiographic impression of osteoporosis. In the future measurements of bone mineral density should be an integral part of the management of patients with osteoporosis. In this skeletal Utopia the role of densitometry in the over 60s will become more clear when we have powerful ways of influencing bone loss related to age. ROGER SMITH

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Asbestos diseases and compensation

Be wary of advising patients to sue

Two forms of recompense are available to those who develop industrial respiratory disease: industrial injuries benefit and damages in the courts. Industrial injuries benefit applies to people employed (not self employed) in an occupation in which the disease is prescribed by the Department of Social Security and depends on the diagnosis being agreed by a departmental medical board for respiratory diseases. Civil litigation is open to anyone who can persuade a lawyer that the employer was negligent in allowing the disease to occur.

Compensation for asbestos related disease is a particularly complex and emotive issue. Four diseases—pneumoconiosis (asbestosis), mesothelioma, bilateral diffuse pleural thickening, and lung cancer in the presence of either asbestosis or diffuse pleural thickening—are recognised as industrial diseases by the Department of Social Security in people occupationally exposed to asbestos. Every year some 500 people are awarded benefits for mesothelioma, 200 for asbestosis, 100 for diffuse fibrosis, and 60 for lung cancer in the presence of other asbestos related diseases. This last figure excludes many who were already receiving benefits for asbestosis or pleural fibrosis and subsequently developed lung cancer. Pleural plaques alone, being harmless and non-disabling, are not prescribed diseases.

Clearly, plenty of scope for disagreement exists about these diagnoses given the ease with which mesothelioma may be confused with pleural invasion by carcinoma, the well known interobserver variability in reading chest radiographs for irregular opacities and pleural changes, and the often poor correlation between measured dysfunction and professed respiratory disability. The Department of Social Security medical boards for respiratory diseases work to clearly laid down definitions, and in about a third of cases their verdict differs from that of the doctor who diagnosed the asbestos related disease. Such decisions may, however, be referred to a medical appeals tribunal. The doctors of the respiratory diseases board are glad to advise clinicians about claim procedures for individual patients, and a personal call or letter is often helpful. Widows or other dependants may make retrospective claims, and the boards will assess these on the evidence. If the claims are successful disability and constant attendance benefits may be payable for a period up to the date of death. Widows’ pensions are no longer payable.

Whether or not a claim for industrial injuries benefit is made or is successful, a person suffering from asbestos related disease may sue any previous employer for negligently allowing exposure to asbestos. If successful the litigant will be awarded damages assessed by the judge and based on the calculated loss of potential earnings together with a sum in recompense for pain and suffering. If the victim is dead the widow or other dependent relatives may pursue the action. Awards may be substantial but if the action is lost so may the costs. Litigants are normally supported by a trade union or by legal aid.

It is by no means certain that a person with an asbestos related disease will win. A court will rarely regard the mere presence of pleural plaques as disabling, although claims of anxiety or phobia about cancer as a consequence of the diagnosis may result in a moderate settlement. More commonly a claim is unsuccessful because the employer cannot be shown to have been negligent in the light of knowledge at the time. The fact that mesothelioma was not known to be a hazard of exposure to asbestos until 1960 may be a hindrance to proving negligence, although courts generally accept that this disease would have been prevented if the employer had adhered to regulations intended to prevent asbestosis. Plaintiffs who pursue a case unsuccessfully may end up worse off; anxious about their health and embittered at the perceived injustice.

What should the doctor do when advising a patient with asbestos related disease? Firstly, the association between exposure and disease must be plausible and the diagnosis reasonably secure; many patients have been made anxious by being told that they have asbestosis when their exposure has been trivial and their diagnosis is dubious. Secondly, the patient should be advised to apply for industrial injuries benefit if he or she has been employed in a prescribed occupation (one in which asbestos exposure was known to occur) and has one of the four scheduled diseases. The patient needs to fill in a form at the local social security office and will then be interviewed and referred to the board. The board
doctors will normally seek the clinical notes and radiographs as well as themselves assessing the patient clinically, radiologically, and functionally.

A doctor who strongly believes that an employer has been negligent may find it difficult to resist the temptation to suggest that the patient sues. It is better to advise the patient to consult a trade union or a solicitor and offer to provide medical support for any claim. The patient will then get proper advice on negligence and the legal obligations of employers, matters with which doctors are usually unfamiliar. With pleural plaques it is mischievous to do other than tell the patient of the presence of harmless shadows in the radiograph and to explain that they carry no implications for health other than those arising from the known exposure to asbestos. Anxiety in such patients is iatrogenic and can be prevented by competent medical advice.

Finally, what should the doctor do when a patient suffering from asbestos related disease dies? In England and Wales deaths from industrial diseases should be reported to the coroner, who will carry out an inquest, which doctors 

Three types of erythromelalgia

Important to differentiate because treatment differs

The term erythromelalgia was first used in 1878 by Mitchell to describe a syndrome of red congestion and burning pain in the hands and feet. He distinguished it from the painful red limbs seen in some patients with gout or rheumatoid arthritis. Some confusion was introduced when Smith and Allen suggested changing the name to erythromelalgia in order to emphasise the symptoms of painful inflammation and warmth. They also showed that in their patients aspirin promptly relieved the burning pain for about three days. Recently erythromelalgia and erythermalgia have been used indiscriminately as synonyms—and to confuse matters further both have been described as primary or secondary, erythermalgia often associated with polycythaemia vera. There are, in fact, three syndromes of erythromelalgia that need to be distinguished for effective management.

The most common variant is erythromelalgia associated with thrombocythaemia. The clinical features of this syndrome are readily explicable by platelet mediated arteriolar inflammation and thrombosis—whether the thrombocythaemia is isolated or is associated with polycythaemia vera or myelofibrosis. The long lasting clinical relief given by aspirin is due to its irreversible inhibition of platelet cyclo-oxygenase activity.

The burning distress in the feet or hands of patients with thrombocythaemia is linked with local symptoms of redness, warmth, and swelling. Warmth aggravates the symptoms, but cold relieves them only slightly. More substantial relief is given by absolute and continued rest with the arm or leg raised. The redness is usually most obvious in the ball of the forefoot or one or more toes or both. In the arm the lesions are usually in the palm and finger tips. One hand is affected more often than both; when the lesions are bilateral they are asymmetrical. The picture may progress from a dull dusky mottled redness to dark purplish acrocyanosis and even peripheral gangrene. The histopathological findings are fibromuscular intimal proliferation and occlusive thrombosis of arterioles and digital arteries. Thrombocythaemic erythromelalgia is frequently not recognised because clinicians are unfamiliar with its typical appearances, but it may be common. In our series of 50 patients with thrombocythaemia 30 had erythromelalgia, but of 73 such patients from eight general hospitals in The Netherlands it was present in only 17. The relief of pain for several days after a single dose of aspirin is a reliable therapeutic test for this type of erythromelalgia.

By contrast, primary erythromelalgia is rare. Instead of the asymmetrical or unilateral localisation of thrombocythaemic erythromelalgia in the foot, toes, and fingers in adults, primary erythromelalgia begins in childhood or adolescence as bilateral symmetrical burning distress in the feet, ankles, and legs. There is a sparing of the toes and no progression to peripheral ischaemia or gangrene. The burning distress of primary erythromelalgia is easily elicited by exposure to warmth and by exercise—to such a degree that patients need to find some way of cooling their legs. The disorder occurred in more than one member in five families, suggesting some genetic basis. Six criteria are required for the diagnosis to be made: firstly, attacks of local red vasodilatation and congestion with increased local skin temperature and burning pain; secondly, the disorder is bilateral; thirdly, the attacks may easily be provoked and aggravated by exercise and heat; fourthly, cold, rest, and raising the affected limbs provides relief; fifthly, there must be no primary or associated disease; and, lastly, the condition is refractory to drug treatment. Only 13 case histories of primary erythromelalgia satisfying these criteria could be collected from published work.

The third and final variant is secondary erythromelalgia, described in association not only with gout, systemic lupus erythematosus, rheumatoid arthritis, cryoglobulinaemia, endarteritis obliterans, thromboangiitis obliterans, polyarteritis nodosa, and arteriosclerosis but also with diabetes mellitus, neurological conditions, vascular diseases, and secondary to vasoactive drugs. In none of these conditions has any platelet dysfunction been shown, and aspirin has no