manifestations of lupus erythematosus resistant to topical steroids and antimalarial drugs. Thalidomide has been reported to be of benefit in subacute cutaneous and chronic discoid lupus erythematosus as well as lupus erythematosus profundus.<sup>24</sup>

The mode of action of thalidomide is unclear, although its teratogenicity is well recognised. Care must be taken to monitor for the development of possible peripheral neuropathy. Despite these side effects thalidomide is useful in combating the often disfiguring cutaneous manifestations of lupus erythematosus.

NIGEL P BURROWS

Department of Dermatology, Addenbrooke's NHS Trust, Cambridge CB2 2QQ

- 1 Venables PJW. Diagnosis and treatment of systemic lupus erythematosus. BMJ 1993;307:663-6. (11 September.)
- 2 Naafs B, Bakkers EJM, Flinterman J, Faber WR. Thalidomide treatment of subacute cutaneous lupus erythematosus. Br J Dermatol 1982;107:83-6.
- Knop J, Bonsmann G, Happle R, Ludolph A, Matz DR, Mifsud EJ, et al. Thalidomide in the treatment of sixty cases of chronic discoid lupus erythematosus. Br J Dermatol 1983;108:461-6.
  Burrows NP, Walport MJ, Hammond AH, Davey N, Russell
- 4 Burrows NP, Walport MJ, Hammond AH, Davey N, Russell Jones R. Lupus erythematosus profundus with partial C4 deficiency to thalidomide. Br J Dermatol 1991;125:62-7.

# Medical management of rheumatoid arthritis

EDITOR,—In their reviewing of the medical management of rheumatoid arthritis D R Porter and R D Sturrock document the poor outcome achieved in the past.1 They identify the need to treat patients earlier but note the difficulty of doing this in the absence of any reliable predictive indicators. We question the statement that no reliable stable indicators are available as we recently published evidence that two genetic factors, combined with rheumatoid factor, produce clinically useful predictions.2 The findings of this pilot study have been confirmed in a consecutive series of 177 patients attending our early inflammatory arthritis clinic.3 This showed that clinically valuable predictions of radiological erosions at one year could be made. The presence of either rheumatoid factor or the conserved epitope had a sensitivity of erosive disease of 95%, permitting the identification of virtually all patients at risk of erosive disease. The presence of the conserved epitope plus rheumatoid factor had a specificity of 88%. This allows selection of high risk patients for more toxic intervention. A subgroup of patients with Dw4/Dw14 were identified with an even worse prognosis.

The importance of genetic typing is that it can be done at any stage of the disease process. This has become particularly relevant as patients are seen earlier in the disease in an attempt to prevent damage (current evidence indicates, that, though painful joints are improved at whatever stage of the disease patients are seen, function is only stabilised.4 Further pressure to treat patients early comes from evidence showing that patients with persistent active disease suffer harmful catabolic effects, such as osteoporosis—for example, patients with raised C reactive protein concentration for two years lose on average more than 10% of bone from their hips.5 Patients seen close to the onset of their disease are difficult to distinguish from each other on clinical grounds, hence the importance of these objective, stable measures.

Though there was once considerable debate about which patients should receive second line treatment, this has now changed. A recent survey from our national early arthritis group (which reflects national prescribing habits) indicates that 88% of patients diagnosed as having rheumatoid arthritis receive second line drugs in the first three years, and all but 5% of these receive them within the first 12 months. Debate now centres more on which second line drug to give rather than on whether to give one. The information obtained

from prognostic indicators allows this choice to be made on logical grounds. This approach also bypasses the argument, referred to by Porter and Sturrock, for inverting the therapeutic pyramid—that is, giving aggressive treatment to all patients. This is clearly inappropriate for the many patients who do well with less aggressive management.

PAUL EMERY ANDREW GOUGH MIKE SALMON JOE DEVLIN

Department of Rheumatology, Faculty of Medicine and Dentistry, University of Birmingham, Birmingham B15 2TT

- 1 Porter DR, Sturrock RD. Medical management of rheumatoid arthritis. BMJ 1993;307:425-8. (14 August.)
- 2 Emery P, Salmon M, Bradley H, Wordsworth P, Tunn E, Bacon PA, et al. Genetically determined factors as predictors of radiological change in patients with early symmetrical arthritis. BMJ 1992;305:1387-9.
- 3 Gough A, Faint J, Wordsworth P, Salmon M, Emery P. The HLA class II associations of rheumatoid arthritis confer severity not susceptibility [abstract]. Arthritis Rheum 1992; 35(9):47.
- 4 Young A, Cox N, Davis P, Dixie J, Emery P, James D, et al. Early rheumatoid arthritis (RA): clinical patterns and outcome during first 3 years in 207 patients. Br J Rheumatol 1993;32 (suppl 1):97.
- 5 Gough A, Lilley J, Devlin J, Eyre S, Emery P. Rapid bone loss in early rheumatoid arthritis (RA) due to disease activity. In: Christiansen C, Rüs B, eds. Osteoporosis. Proceedings 1993. Fourth international symposium on osteoporosis and consensus development conference. Rodovre, Denmark: Osteopress, 1993: 175-6.

## Diagnosis in histopathology

#### Is often unclear

EDITOR,—In the absence of appropriate information it is wrong to make specific comments about recent events in Birmingham, where a pathologist has been criticised for misdiagnoses.¹ Most reports in the press, however, have shown a worrying lack of insight into the nosological status of histopathological diagnoses.

Managers (and even many clinicians) may have been misled into believing that lesions submitted to histopathology laboratories are easily recognisable as benign or malignant. It also seems to have been accepted that review of a case by a solitary independent expert can provide the correct answer. Nothing is further from the truth. In practice, a moderate number of submitted lesions prove extremely difficult to interpret and the diagnosis is uncertain. Furthermore, when such lesions are reviewed by panels of experts half of the experts may classify them as benign and half as malignant.

Virtually all of diagnostic histopathology is riddled with such problems, and bone lesions are probably the most complex and difficult to diagnose. Perhaps for these lesions more than any other, the final diagnosis must rest on careful interdisciplinary correlation, integrating information from all clinical specialties. Even then, the accurate diagnosis may not become evident until after several years of follow up. The review of one expert's diagnosis by another is not an acceptable option realistically. As a minimum, several experts must be used, and even then it must be appreciated that the diagnosis offered by the group is a consensus opinion rather than guaranteed to be right.

In all clinical specialties consultants who persistently perform badly—diagnosis and practice—compared with the consensus of their colleagues must be identified. To achieve this aim, British gynaecological cytopathologists remain unique in subjecting themselves to yearly proficiency testing. It seems essential that, in addition to medical audit, similar quality assurance is incorporated into all branches of medicine. It is perhaps ironic that in many areas such programmes are already in place for histopathology. As a profession, we must

also decide how to act once people who persistently perform badly have been identified by such systems.

DAVID SLATER

Rotherham District Hospital, Rotherham S60 2UD

1 Kingman S. Birmingham pathologist criticised for misdiagnoses. BMJ 1993;307:581. (4 September.)

### A multidisciplinary affair

EDITOR,—It is well known that a team, comprising not only a pathologist but also clinicians and radiologists, is required to diagnose bone tumours. The history of the condition, radiological appearance, and site of the biopsy specimen all influence the diagnosis and have to be taken into account. Pathological examination alone can result in a false diagnosis. The inquiry at the South Birmingham Tumour Service<sup>1</sup> should therefore look not only at the tissue samples but also at the procedures for diagnosis, the radiographs (with a bone radiologist), and the clinical notes. Sharon Kingman reports that a 9 year old boy with osteomyelitis was treated for a bone tumour<sup>1</sup>; this strongly suggests a lack of teamwork and input from a radiologist.

Before putting the entire blame on Dr Starkie the inquiry should also look at the diagnostic team at the South Birmingham Tumour Service. Did it exist?

G M STEINER

Sheffield Children's Hospital, Sheffield S10 2TH

1 Kingman S. Birmingham pathologist criticised for misdiagnoses. BMJ 1993;307:581. (4 September.)

# Use of diltiazem in sport

EDITOR,—Further to reports of use of anabolic steroids by people participating in sport, we wish to highlight a potential problem that we have encountered in our locality.

A bodybuilder suffering severe abdominal cramps after training attended the accident and emergency department. Questioning elicited the fact that he regularly used anabolic steroids but that recently, to augment his training, he had been taking the calcium channel blocker diltiazem in doses far exceeding the maximum dose recommended in the *British National Formulary* (480 mg a day).<sup>2</sup> His symptoms were eased rapidly by an injection of hyoscine butylbromide, and as no other side effects were noted he was discharged.

Apparently, the use of diltiazem is well established locally among bodybuilders and rugby footballers. The drug's side effects, such as gastrointestinal disturbance, are well documented,<sup>2</sup> but potential cardiac rhythmic disturbances could be fatal

There is scientific evidence that diltiazem increases maximum oxygen consumption after training,' but we are intrigued and worried about how the drug is obtained. Our patient declined to disclose his source of supply.

HUW RICHARDS MANDY GROCUTT MICHAEL McCABE

Accident and Emergency Department, Morriston Hospital, Morriston, Swansea SA6 6NT

- 1 Kennedy M. Anabolic steroids in sport. BMJ 1993;307:567-8. (28 August.)
- 2 BMA and Royal Pharmaceutical Society of Great British national formulary, number 25. London: BMA, RPSGB, 1993: 04.5
- 3 Stewart KJ, Effron MB, Valenti SA, Keleman MH. Effects of Diltiazem or propanolol during exercise training of hypertensive men. Men and Science in Sport and Exercise 1990;22:171-7.

BMJ VOLUME 307 9 OCTOBER 1993