first round of sessions, we should have a better idea of the case mix and the difficulties the local general practitioners face. The final audit would help in further developing the training of local trainees (in general practice) and in the continuing professional development of general practitioners.

Another added advantage is the improvement in communication between primary and secondary care and a better understanding of each other's problems. On the other hand, better awareness of musculoskeletal problems may lead to a paradoxical increase in the number of referrals. However, it should lead to a better patient care.

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Authors' reply

I agree with Dr Ali Jawad that teaching of general practitioners and general practitioner registrars is best done within primary care itself, reflecting the mix of musculoskeletal problems that present within the community.

Various groups around the country are putting in place teaching similar to that described and, in particular, the Primary Care Rheumatology Society is setting up a series of meetings led by society members looking at common problems presenting to GPs.

Our first round of training meetings is based on shoulder problems and the initial meetings have been greeted with enthusiasm by participating GPs. We hope to expand this programme to include other regional problems, osteoporosis, and inflammatory arthritis. I think that the main benefit of teaching in this way is that it produces positive practical outcomes for examination techniques, treatment options, and the demonstration and practice of practical procedures, such as steroid injections.

Education for GPs, which is perceived to be generated from within primary care, with consultant input as appropriate, seems to be well received by GPs.

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LETTERS TO THE EDITOR

Magnitude of the genetic component in juvenile idiopathic arthritis

Genetic factors undoubtedly play a part at least in some forms of juvenile idiopathic arthritis (JIA). Yet it is commonly believed that the risk to a sibling of a patient with JIA is not particularly strong as shown by the rarity of reported multicase families.[1]

Multicase JIA families have been traced systematically at the Rheumatism Foundation Hospital in Heinola, Finland, over a period of 15 years. A total of 41 families with 88 affected siblings (34 boys, 54 girls) were found fulfilling the Durban criteria for JIA.[2] In 60 (68%) of these 88 patients the disease was pauciarticular and, in most instances, it ran a mild course. The mean age at JIA diagnosis was 4.6 years.

Over the same period, eight sets of MZ twins were found; two twin pairs were concordant for JIA.

The incidence of JIA in Finland is 14/100 000 in the paediatric population.[3] This corresponds to about 150 cases a year. Because the mean age at diagnosis was 7 years, the total number of cases of JIA in the paediatric population is about 1200, and the prevalence of the disease is about 1 per 1000, in a population of 1.2 million under 16 years of age in the country. The incidence and prevalence figures are similar to those reported from other countries for which data are available.[4] For instance, it has been estimated that in the United States with a population 50 times larger than that of Finland there are about 71 000 cases of JIA.[1]

Over the 15 year period of study 2300 patients were seen at the paediatric department of the Rheumatism Foundation Hospital. Of these, about 90% had JIA. It can thus be estimated that the population of JIA cases from which all the recorded multicase families were derived amounted to about 2000. This corresponds to 60% of JIA cases in the country.

There are about four MZ births per 1000 in the population. Thus our eight MZ pairs indicated that most, if not all, such pairs had been traced in the study group. Considering the population prevalence of JIA (1 per 1000) the concordance rate of 25% implies a relative risk of about 250 for a MZ twin. In adult rheumatoid arthritis the risk was found to be nine.[5]

The average number of children in Finnish families is 1.8; 45% of families have only one child, 38% have two children, 13% have three children, and 4% four or more children. In adult rheumatoid arthritis the relative risk for a sibling is about three. In view of the population prevalence of JIA, this finding of 41 multicase families in the basic population of 2000 indicates that the relative risk in JIA is much higher.

These findings cannot perhaps be extended to other populations, though there is no reason to believe that this would not be the case. In any event, genome wide screening of Finnish patients offers a promise that new susceptibility loci outside the HLA region may be found.

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A case of rheumatoid meningitis positive for perinuclear antineutrophil cytoplasmic antibody

Rheumatoid meningitis (RM) is a pachymeningitis rarely occurring in rheumatoid arthritis (RA). The pathophysiology of RM is not clear, though a diffuse vasculitis in the dura mater has been shown in some patients.[1] Here we present a patient with RM who had a raised serum level of perinuclear antineutrophil cytoplasmic antibody (P-ANCA), which is known to be associated with vasculitic diseases.

A 72 year old woman was admitted to our hospital because of headache. Twenty two years before she had been diagnosed as having RA and treated with non-steroidal anti-inflammatory drugs, without complete remission. Seven months before admission, polyarthralgia deteriorated and a numbness of the hands and feet and a low grade fever occurred, and she lost 10 kg in 2 months. She was given prednisolone 15 mg/day and her symptoms disappeared in a few weeks; the dose of prednisolone was tapered to 5 mg/day over three months. Five weeks before admission a left temporal headache occurred and gradually worsened; then she was admitted to the hospital. On physical examination, there were paraesthesia in the hands and feet, but no meningeal signs, papilloedema, visual field defects, cranial nerve palsies, ataxia or pathological reflexes were present. In laboratory studies, urine analysis and blood chemistry were normal. Table 1 shows the serological findings. Analysis of cerebrospinal fluid was normal and multiple cultures were negative for micro-organisms. Magnetic resonance imaging (MRI) of the brain disclosed a diffusely thickened dura mater along the left cerebral hemisphere and the cerebellar tentorium was enhanced after gadolinium pentetic acid (Gd-DTPA) administration (figs 1A and B). Biopsy of the dura mater was refused by the patient. After various causes of pachymeningitis[2–4] such as sarcoidosis, Wegener's granulomatosis, malignant lymphoma, and a number of infectious diseases were excluded, a diagnosis of RM was made. The dose of prednisolone was increased to 50 mg/day, and the headache disappeared in two weeks. Raised levels of rheumatoid factor (2390 fell to 153 IU/ml) and P-ANCA (65 fell to <10 EU) gradually normalised in eight weeks. Brain MRI performed three months later showed no dural thickening (figs 1C and D).