EDITORIALS

RHEUMATOLOGY: A WAY AHEAD

Recent years have seen a number of attempts by rheumatologists to audit their activities [1-4]. In addition, a group of newly appointed rheumatologists offered opinions on how rheumatology should be organized and allied to other associated specialties [5,6]. Such efforts are to be applauded, since without them it is probable that we will simply drift on, providing the same historical pattern of care, vainly attempting to make a case for increased resources and protection from cuts. They do not, however, provide satisfactory solutions based upon well researched data, which could be applied anywhere other than in the immediate locality of the auditors. Even then, they probably represent too narrow a view of the problem.

The ARC Epidemiology Unit at Manchester has provided statistics to the medical profession and government on staffing levels and major deficiencies in relation to staffing in the United Kingdom [7,8]. From this, figures have been devised which indicate the number of rheumatologists required per unit of population and the sort of basic facilities required to support such consultant staff [7-9]. They have also indicated where major developments have occurred and the impact of these on medical services, such as that of joint replacement on orthopaedics. They have not, however, indicated the clinical spectrum which rheumatologists would be expected to manage.

Internal audits have often surprised the auditors [1]. There have been wide variations in referral rates and in the general case mix of patients between different units. There has also been variation in the proportion of review to new-patient appointments. It is not clear whether this last finding is related to different case mix within clinics, to the number of junior medical staff or to other provisions for the management of musculoskeletal diseases in the locality. Those who expressed dissatisfaction with the kind of cases referred to their clinics identify those patients with so-called minor rheumatological disorders as being burdensome. They also comment on the lack of training in the use of splints and other orthoses and the problems associated with long-term monitoring of patients on slow-acting antirheumatic drugs [5,6].

Orthopaedic surgeons also treat patients with locomotor diseases and disabilities. In addition, a large proportion of patients with these disorders are not referred to hospital by general practitioners [8], some of whom are also able to refer patients directly to physiotherapists, who themselves have considerable expertise in the assessment and treatment of locomotor disorders.

In the UK there are three times as many consultant orthopaedic surgeons as there are rheumatologists [10]. In Scotland, the ratio is even higher at 12.7:1, highlighting the major lack of rheumatologists in Scotland [11]. In addition, although the statistics available do not allow primary trauma cases to be separated out, orthopaedic surgeons see six times as many new-patient referrals as rheumatologists in England and Wales and twenty-two times more in Scotland. These figures also indicate that each consultant orthopaedic surgeon is responsible for twice as many new patients as each consultant rheumatologist. Many Regional Health Authorities are much less well off for rheumatologists than others and this is particularly marked in the Midlands, North of England, South West England, Wales and Scotland [8]. Orthopaedic provision, however, is more consistent throughout the UK [10,11].

In addition to trauma, orthopaedic surgeons also deal with a significant proportion of so-called minor musculoskeletal conditions and probably find them as much an unwanted burden as some rheumatologists. Certainly many orthopaedic surgeons would prefer to see only those patients where there is a serious possibility for the role of operative treatment or investigation.

What then of the general practitioner’s role? We do not know if those with high referral rates need to be moderated or those with low referral rates need to be encouraged. Nor do we know if high referral rates to rheumatology are matched by low referral rates to orthopaedics and physiotherapy and vice versa. Studies have been conducted of the benefits of educating general practitioners in periarticular injection techniques for the relief of selected musculoskeletal disorders. These have demonstrated at least a short-term benefit [12]. We do not, however, have data on longer term consequences of
this exercise. From discussion with general practitioners, it is my impression that lesions likely to benefit from local corticosteroid injection are seen comparatively infrequently by individual practitioners. Figures suggest that for an average list size of 2200, a general practitioner would see four patients with frozen shoulder per annum [7]. Not all may require injection treatment. In a recent study, an author with previous rheumatological training and working in general practice found only a small number of patients requiring corticosteroid injection. Unfortunately, no indication was given as to the period of time over which these patients were collected [13]. There is perhaps therefore a problem for a general practitioner in gaining expertise in this area. In addition, it is of interest to note that in a very small number of patients in this study, it was felt necessary to refer them for physiotherapy. In the same study, the number of patients requiring simple reassurance was high and it may be that more effort needs to be made in the direction of education and advice on how to assess patients with locomotor symptoms, in order to distinguish the significant from the non-significant and that requiring treatment from that which does not. However, this may be much more difficult to achieve than the teaching of a simple technique.

Many rheumatologists will have received a significant part of their training in academic centres. Some will have spent time in clinical research, whilst others will have, in addition or instead, spent time in laboratory research. Bearing in mind the significant variations in the type of cases seen in different types of clinics, it is quite possible that training in departments dealing primarily with inflammatory joint disease and connective tissue diseases, with a smattering of osteoarthritis, does not necessarily prepare the incumbent senior registrar for subsequent exposure to the wide variety of commoner, more mundane and minor but to the patient painful and disabling, musculoskeletal diseases, for which his future general practitioner colleagues will expect advice and treatment.

It seems, therefore, that to approach the perceived problem of inappropriate referral to rheumatology clinics and failure of general practitioners to initiate simple and effective treatments, only from the standpoint of rheumatologists, is not going to provide solutions. Patients wish to have an expeditious diagnosis, explanation, treatment and, where necessary, investigation of their medical disorders. They wish the practitioner concerned to have the necessary expertise and to have that expertise demonstrated to them. If it is lacking or perceived to be lacking, then referral for a further opinion will be required [2]. Currently little is known of the referral patterns to individual specialties throughout the UK or the factors which may influence these. Individuals are unlikely to be able to study this effectively as colleagues may be overconcerned about sectional interests and successful comparisons between Health Authorities would be unlikely, due to the lack of standardization of methodology. An organization such as the British League Against Rheumatism (BLAR) with its affiliated societies covering all the medical interests should be well placed to initiate such a study, using the well established epidemiological facilities available in the UK. Such a study would also have the advantages of a standard approach, thereby allowing consideration of the type of changes which might be most appropriate in individual localities, by comparing their characteristics with studied areas.

Until we have a better understanding of the factors influencing the pattern of treatment and referral by general practitioners, it will be difficult to provide little other than opinions on solutions. Perhaps, however, rheumatologists should recognize that they are only one specialty dealing with the musculoskeletal disorders and should attempt to take a wider view. Recognition of the significant role and expertise of other professionals in the management of these disorders is an initial step. Closer links and cooperation with other professionals in determining the best use of all the resources available should prove worthwhile. For instance, could we cooperate further in undergraduate education to ensure covering the essentials and avoiding overlap? Should we have a degree of combined training for orthopaedic surgeons and rheumatologists? Could we collaborate in devising investigation and treatment protocols for common disorders, and if so, should these be used in collaborative clinics between rheumatologists and orthopaedic surgeons? How much do medical practitioners need to know of the details of physical therapy and how should they assess the appropriateness of their referrals for such therapy? Computerized systems have been demonstrated to be of benefit in reducing some
of the problems of monitoring of slow-acting drugs [14,15], but to date they have not been widely used.

Whatever solutions are devised, they are unlikely to be durable if they are based upon data generated from or the views of only one of the specialty groups managing musculoskeletal disease.

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REFERENCES

RELAPSING POLYCHONDRITIS
RELAPSING polychondritis (RP) is an episodic, progressive, inflammatory disease of cartilaginous structures which may be accompanied by inflammatory lesions of the special sense organs, the joints and the cardiovascular system. Clinical studies have enabled the establishment of diagnostic criteria on an empirical basis [1]. These include the following clinical features: (1) recurrent auricular chondritis; (2) a non-erosive, inflammatory, polyarthritis; (3) nasal chondritis; (4) inflammation of ocular structures including the uveal tract, cornea, conjunctiva, sclera and episclera (reviewed in [2]); (5) chondritis of the respiratory tract involving laryngeal and tracheal cartilages; (6) cochlear and/or vestibular damage manifest by neurosensory hearing loss, tinnitus or vertigo. The combination of either three or more such features, or one clinical feature together with a positive biopsy, or chondritis in two or more separate anatomical sites with a therapeutic response to corticosteroids are regarded as diagnostic [1, 3]. In general the presenting symptoms are related to one of the six major sites of inflammation, the commonest being auricular chondritis (26%) or arthritis (23%), with symptoms related to nasal chondritis (13%), ocular disease (14%) and audiovestibular disturbance (6%) occurring less frequently [1]. However, some 2% of cases present with miscellaneous symptoms and one unusual mode of presentation is a pyrexia of unknown origin as reported in the current edition of this journal [4]. Interestingly, fever is not unusual in RP, being present in 44% of patients at some stage of the disease and in 22% at presentation [2]. However, it rarely occurs in the absence of other characteristic clinical features but clearly poses a problem when it does, as there are no diagnostic laboratory tests.

There is a well documented association of RP with rheumatic and autoimmune disease in
some 25–35% of cases. Such conditions include adult or juvenile rheumatoid arthritis, systemic lupus erythematosus, Sjögren’s syndrome, systemic sclerosis, autoimmune thyroid disease, ulcerative colitis, glomerulonephritis, pernicious anaemia and systemic vasculitis [1, 2]. A further addition to this list is the case report of Smith et al. [5] in this issue describing the coexistence of RP and Weber–Christian disease. In their patient, recurrent abdominal pain due to mesenteric panniculitis was the main symptom. The authors emphasize that visceral involvement with abdominal pain is not a feature of classical RP and alternative causes must be sought. Exclusion of an associated vasculitis is of particular importance as this obviates the need for cytotoxic agents and improves the prognosis. Usually, episodes of auricular and nasal chondritis subside spontaneously within 5–10 days [1] and may be managed with non-steroidal anti-inflammatory drugs alone. Nevertheless, treatment for RP involves the use of large doses of prednisolone in 75% of patients. This serves to decrease the frequency and severity of attacks and resort to cytotoxic agents is not required. However, the impression remains that corticosteroids do not stop the progression of disease in aggressive cases. Indeed the disease carries a 20–30% mortality rate (75% 5-year survival) with saddle nose deformity, arthritis, laryngotracheal strictures, systemic vasculitis, microhaematuria and anaemia in patients under the age of 51 years being identified as poor prognostic factors [1, 2, 6]. In approximately 50% of cases death is related to either involvement of the respiratory tract (respiratory failure due to airway collapse or pneumonia due to airway obstruction), vasculitic or cardiovascular complications, or infection consequent on immunosuppressive therapy.

The pathogenesis of this perplexing condition remains unclear. The characteristic pathological changes seen with light microscopy are the loss of basophilic staining of cartilage matrix, perichondral inflammation and cartilage destruction and replacement by fibrous tissue. The perichondral inflammatory infiltrate comprises lymphocytes and plasma cells together with fibrocytic and endothelial cell proliferation. The adjacent chondrocytes become vacuolated, pyknotic changes are observed in the nuclei and eventually loss of cell outline and phagocytosis by macrophages occurs [1, 7]. Initial studies focused on the presence of anticartilage antibodies (ACA) in the serum of affected individuals. Hughes et al. [8] found ACA in two out of three cases and demonstrated the loss of all glycosaminoglycans in affected cartilage and vessel walls suggesting the loss of the cell type responsible for the maintenance and repair of connective tissue. Subsequently other authors [9, 10] have demonstrated the presence of ACA in up to 66% of RP patients [10] and a proportion of these antibodies are directed towards type II collagen [9]. These antibodies are present at the onset of disease and the titres appear to correlate with disease activity.

In contrast, other studies have failed to demonstrate ACA in the sera of RP patients [11, 12]. Some authors have found evidence for a cell-mediated immune response directed towards cartilage components [11, 12]. Thus Rajapakse et al. [12] were able to demonstrate increased lymphocyte transformation and macrophage migration inhibition in response to human laryngeal cartilage proteoglycan in two subjects with RP. Furthermore, the immunohistological study of Svenson et al. [13] revealed a predominance of HLA-DR positive macrophages and a significant number of CD4 positive, helper-inducer T-lymphocytes in the cellular infiltrate, suggesting a cell-mediated immune mechanism. In addition animal models of RP in which rats immunized with type II collagen develop arthritis and auricular chondritis also point to the development of autoimmunity to cartilage components as being of aetiological importance [14, 15].

These clinical and laboratory observations suggest that immunological mechanisms are important in the initiation and perpetuation of RP. Central to this hypothesis is the development of an immune response to immunogenic epitopes present in cartilage components such as proteoglycan and type II collagen [16]. The resulting perichondral cellular infiltrate is presumably the site of cytokine production (e.g. interleukin-1, interferon-γ, tumour necrosis factor), with consequent neutrophil, macrophage and chondrocyte activation leading to the enzymatic degradation of cartilage matrix [17, 18]. Similar chronic inflammatory lesions in the eye and cardiovascular system would arise through the presence of shared antigenic epitopes present in these tissues [16].

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REFERENCES


