EDITORIAL

MEDICINE AND THE MEDIA

Giving information to the public at large of medical matters in vivid form has an honourable lineage. The Old Testament chroniclers, and the disciples who reported the sayings and doings to Jesus would definitely have belonged to a Medical Journalists Association had such a body then been in existence. They described disease in simple terms — and the cure of it. They spread the word of the power of healing, knowing then, as we know now that sickness and health exert a powerful fascination alike for those who are sick and those who are well. They are communicators par excellence!

However in spite of this historical background doctors and journalists are unlikely ever fully to agree on the way medical matters should be presented to the public. This is fortunately a conflict of attitudes and style rather than of content. Both professionals agree that the public must be better informed of medical matters. Basically the medical journalists seek to inform his readers, his listeners or those who watch TV of what is new on the medical front and to explain how doctors go about the task of treating illness or disease. He (or she) attempts to do this in as short a space, or time, as possible and to do so as speedily as possible. Doctors on the other hand feel medicine is his private world. Some, perhaps many, genuinely believe that the task of passing on information about medical matters, or of interpreting what medicine is doing or trying to do is best done by doctors. They alone, they feel, can and should judge whether information ought to be imparted and if so how it should be presented.

Another contentious point between the doctor and the journalist is one of timing. A doctor often wonders, after having been approached by a journalist seeking help why it should matter whether he gives his answer to-day, tomorrow or next week. The media on the other hand spend enormous amounts of money and energy to be first with the news. Immediacy is their cornerstone since newspapers and magazines all have to sell their wares in the market place.

What then should a doctor sought by the media say and do when that distant voice on the phone solicits his help? One obvious answer would be "No comments!" but having decided against this negative response, it would appear the crucial decision one must make is "can I trust this man or woman on the phone." This mutual trust and confidence is paramount in any doctor-journalist relationship. Although journalists do not have the equivalent of the Hippocratic Oath, they are expected to preserve confidentiality of their source. Indeed journalists are being prosecuted and even jailed for refusing to reveal who told them what or how they came to know of certain information.

The next important point is one of professionalism. Many doctors fear and often rightly so that journalists may distort, sensationalize or trivialise what they say. A source of annoyance to many doctors, certainly as far as the printed word is concerned, is the flaring head-line with the eye-catching phrase that lures the reader to the smaller print below. An eminent visitor to Singapore recently Dr Patrick Steptoe is reported to have said that he has no objection to newspapers referring to ovum removal, in vitro fertilization and reimplantation but that he objects to the term "test-tube baby" tag attached to his work. An interesting point but would his pioneering work have received world-wide attention if head-line writers had used the term "in vitro fertilization" instead of "test-tube baby?" Nevertheless to attain a certain amount of proficiency medical journalists should do significant home-work and background research before tackling a topic. The practice of publishing word for word from a prepared script delivered at conferences is to be deplored.

Although it is unchallengeable that widespread publicity can create problems, the media can in the broadest sense educate the public to be aware of the need for new approaches to controversial issues. Has the time not come to back the "contracting-out" approach to the donation of kidneys and other spare-parts. Is there a peril in the computerizing of medical records with busybodies having access? What are the implications of making Singapore a "medical marketplace" — indeed should medicine ever be a saleable commodity. These are topics that require indepth study and research by our medical journalists.

Although there are potential areas of conflict, yet the opportunity for co-operation and harmony is never greater. Reasonably approached, most doctors are willing to help the Press unravel the complexities of modern medicine. However doctors still seek, and rightly so, an assurance that confidentiality will be respected. Although there is less fear of breaching one of the three A "sins" of medicine namely abortion, advertising and adultery, such assurances should always be given. However rogue antics like trying to gatecrash wards and private medical receptions, "ambushing" eminent visitors and making unsolicited visits to their hotel rooms late at night and harassing patients are to be condemned, not least by the media themselves if co-operation between journalists and doctors is to continue.

In the U.K. many efforts are being made to improve communication between medicine and media. The various Royal Colleges hold informal meetings at which senior doctors and media representatives meet to discuss matters of common interest. Workshops and seminars are held to iron out communication problems.

Doctors and journalists must come together for one reason if for no other: the need to prevent sickness by promoting health. This can in fact be effectively done as shown by the successful Press campaign against smoking. How much one may differ in the views concerning handling of medical information, one fact is inescapable: a nation's greatest wealth is its healthy citizens. Both medicine and media must rise to the occasion.

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Editor

SPECIAL ARTICLE

RHEUMATOID ARTHRITIS — AN OVERVIEW

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INTRODUCTION

Although one of the commonest of the inflammatory connective tissue diseases, rheumatoid arthritis (RA) remains one of the most enigmatic.

Among the many involved questions in RA is "why so localised?" What directs the sequence of events leading to an inflammation so intensely centred on the synovium. Although the more widespread effects of immune-complex mediated damage, such as vasculitis and nephritis are an occasionally prominent feature, the severity of the synovitis is greater than that seen in any other connective tissue disease.

EPIDEMIOLOGY

RA may well be a disease of modern times. Little evidence of this easily recognisable disease exists in texts, paintings or skeletons before the last century. Intriguingly, Buchanan has suggested that RA may also be a disease in decline — with other connective tissue diseases such as systemic lupus erythematosus and vasculitis gradually emerging worldwide with increasing prevalence. Be that as it may, RA both medically, socially and economically is a major disease problem in most countries. Although prevalence does appear to vary — cold, wet or humid climates producing higher figures, interpretation is difficult. For example, the prevalence in England and Jamaica may well be similar.

In the UK the disease affects up to 2% of the population, with females outnumbering males by 3 : 1. The disease reaches a peak frequency in the 5th or 6th decades.

PATHOLOGY

The predominant inflammatory response within the synovium is lymphocytic. Studies using monoclonal antisera in attempts to define predominant T-cell subset types have given widely differing results. One possible reason for these reported differences may be the stage of inflammation at which the immunofluorescent studies were made.

At the present time, it appears that in the early RA infiltrate, OKT 4 helper T cells predominate, while later, OKT 8 (suppresor T cells) predominate.

Although the initiating factors in the sequence leading to disturbances in T & B cell function are unknown, the sequelae have been well studied, and are, in essence, a summary of the inflammatory process. Thus antibodies particularly anti IgG — are formed, immune complexes are abundant, and complement activation initiated. The synovial fluid exudate is inflammatory and complement levels are low (the serum complement usually remaining normal). Inflammatory mediators are released, including kinins, collagenases and other enzymes, and the end stage of inflammatory cartilage and joint erosion reached if the process continues. A proportion of the local inflammatory reaction may be self-perpetuating — for example, Type 2 collagen can, under certain circumstances, itself cause synovitis.

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Collagen — anti-collagen complexes may contribute to the mass of intra-articular immune complexes though by far the major constituent is rheumatoid factor (RF) — up to 98% of identifiable complexes consisting of IgG-anti IgM.

SYSTEMIC PATHOLOGY

Rheumatoid nodules, with their characteristic histology, are distributed on pressure points, along tendons, and, very occasionally, systemically in the lungs, sclera, heart. There appears to be a considerable ethnic variation in the tendency to rheumatoid nodule development, their being rare in certain Chinese and in some black populations. There have been suggestions that the tendency to nodule formation is HLA-linked, but this has yet to be substantiated.

Systemic vasculitis, occasionally acute and resembling polyarteritis nodosa, is a rare complication. The reasons for this switch in disease pattern in RA from synovitis to vasculitis are unclear. In studies undertaken in our unit, reticulo-endothelial clearance of IgG-coated erythrocytes (a probe of Fc receptor cell function) was grossly impaired in the majority of patients with vasculitis, compared with the relatively normal clearance in uncomplicated RA. Although this suggests that impaired RES clearance of complexes may be one factor in the altered circulation and distribution of complexes seen in RA vasculitis, the aetiology of vasculitis remains far from clear.

SJOGREN'S SYNDROME

Up to one third of RA patients suffer from the additional features of dry eyes and dry mouth characteristic of Sjogren's Syndrome. The histology is of lymphocyte infiltration of the exocrine glands, particularly the salivary and lacrimal glands, occasionally obliterating excrocrine glandular tissue and function.

In addition to the local problems of kerato-conjunctivitis sicca and xerostomia, these patients may suffer from interstitial nephritis (one wonders whether some cases of analgesic nephropathy in RA are complicated by or with Sjogren's syndrome), a tendency to drug allergies (both gold and penicillamine allergy are approximately twice as common in this subset of RA patients), and more overt evidence of B cell proliferation, with hyperglobulinaemia, anti-nuclear (particularly anti-Ro) antibodies, organ — specific antibodies and high mean levels of circulating complexes. A rare complication of primary Sjogrens (but, disputably, not of sjogren's associated with RA) is the development of lymphoma.

RHEUMATOID FACTOR

In the USA and UK, approximately 80% of patients have demonstrable circulating IgM antiglobulins, conventionally tested by slide latex or haemagglutination techniques. While the time-honoured division of RA into "seropositive" and "seronegative" is over-simplistic, and subject to over-interpretation, it has historically been of immeasurable value in the early attempts at delineation of the "HLA B27" from the HLA-B8" group of diseases. For the individual patient, the presence or absence of RF carries only general prognostic importance.

The presence of circulating IgG rheumatoid factor, more difficult to quantitate, has been linked with vasculitis, but figures vary widely. Recently, in a study of IgA rheumatoid factors, were noted high titres of circulating IgA RF in patients with primary Sjogren's syndrome — a finding of interest in view of the predominantly mucosal nature of Sjogren's.

THE AETIOLOGY OF RA

1. GENETICS

Studies by Stastny and subsequently by others have shown that classical seropositive RA patients possess the HLA antigen HLA D4 with twice the normal background frequency.

Obviously, as studies from other countries appear, different, local, HLA association are being reported. Nonetheless, the HLA studies underscore earlier epidemiological and serological studies suggesting a genetic influence.

SEX RATIO

Polyarteritis Nodosa apart, the connective tissue diseases all have a female predominance. Until recently, this difference (which may rise to 30 : 1 in young SLE patients) has been little studied. Studies during the past 10 years have shown that sex hormones have a profound effect not only on most measurable aspect of the immune system, but on the expression of clinical disease pattern.

In SLE, for example, abnormal metabolism of oestrogen has been demonstrated in certain patients. In the NZB mouse, the animal model for SLE, the majority of females perish from their lupus nephritis within their first year. By suitable experimental manipulation of sex hormones, up to 90%/year survival in the same group has been achieved. Clearly, in practical terms, the means at our clinical disposal for therapeutic alterations of sex hormone levels are very limited, but such studies do emphasise the multifactorial nature of many of the these diseases, and point to new lines of therapeutic research.

INFECTIVE AGENTS

Streptococci, mycoplasmas, rubella, EB virus and other agents all have their advocates. Studies have been so intense that it is probably fair to surmise that none of the known existing infectious agents — in isolation — is the cause of RA. A more attractive hypothesis is similar to that held in reactive arthritis — that in genetically predisposed individuals, a variety of organisms or even chemicals may trigger a variety of inflammatory responses, the pattern of which we call RA.

SOIL AND SEED

When the pleasure ship "Little Rock" sailed out of Trieste with 1200 men on board, one of the clearest examples of the interaction of infective and genetic factors in the production of arthritis was about to be acted out.

At sea, one half of the men developed Shigella dysentry. A dozen or so went on to classical Reiter's syndrome, or post-dysentric 'reactive' arthritis.

When Calin and others traced these men many years later, in addition to being B27, all the men who had developed post-dysentric Reiters had gone on to more chronic inflammatory disease — Spondylitis, iritis or urethritis.

Another example of soil and seed is discussed by Professor Panayi in this symposium — the suggestion that gold abreactions in RA might be more common in HLA D3 individuals.

Last year, my group at the Hammersmith Hospital — in association with Professor Batchelor reported a third example — hydralazine lupus, in whom there was a D4 association. Indeed so strong was the association that a female who was D4 as well as being a slow acetylator would almost predictably develop arthritis on continuing hydralazine consumption.

It is tempting to suggest that a similar state exists in RA — that is — the continuing exposure to an antigen or antigens results in disease in preselected individuals.

Food and arthritis

Classical allergy studies have concentrated on the aquisition of antigen via the blood, respiratory tract or skin, but until recently, evidence for gut absorption of antigens was scanty. The studies of Soothill, Brostoff and others have demonstrated that protein antigens may enter the circulation via the gut, and may induce an antibody response.

RA patients, including the most perspicatious and conservative, frequently give clear histories of exacerbation of symptoms following the ingestion of certain foods or food products. We are currently studying the pathways of macromolecule absorbtion in patients with arthritis — "gut associated" as well as those not clearly associated with gastrointestinal symptoms or disease.

We recently described a patient whose severe, aggressive erosive disease subsided on excluding cheese products from the diet. When she was re-challenged with cheese, not only did the disease flare dramatically, but acute phase reactants and circulating complexes, which had disappeared from the circulation, returned in high titer.

It is this author's prejudice that the absorption of potential antigens — proteins, food products, bacterial cell wall antigens etc, from the gut, may well prove to be an important cause of continuing disease in certain patients.

TREATMENT

The days of patronising arthritis-homes and the 16-aspirin-a-day and plaster-of-Paris-splints are mercifully over, or should be over. Though one may cavill at the profits made by the major pharmaceutical houses, they have, in no small way, given immeasurable practical help to treatment by their research and development programs. "Non steroidals", though weak, are safe, and infinitely better tolerated than aspirin.

The earlier use of second line drugs — Gold and penicillamine and even, in some cases, antimalarials, must be chalked up as a therapeutic 'advance' albeit a minor one. The advances in orthopedic surgery are clearer still.

In any chronic disease, the state of mind of the patient is paramount. It would be comforting (though perhaps complacent) to think that the development of rheumatology, with physicians at least interested and positive about the rheumatic diseases, has itself been a factor in the improved management of rheumatoid arthritis.