1976). Furthermore, women make claims on sickness policies significantly more than men and many insurance companies reflect this with higher premiums for women clients.

The above table from an article submitted to the Journal of the Institute of Actuaries *gives further factual evidence —Ed.*

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HAY FEVER

Sir,

In the prize-winning essay on hay fever (May *Journal,* p.265) it is said that cell-mediated immunity is mediated by IgE. My understanding is that IgE is an antibody involved in anaphylaxis which is classed as humoral immunity, while cell-mediated immunity involving reactions such as delayed hyposensitivity and graft rejection involves the T-lymphocytes, and not IgE.

As the essay is entitled "The management of hay fever in general practice" it is a pity that it does not really discuss the evidence for and against skin testing and desensitization. It is also surprising considering that when quite a lot of space is devoted to immunology no mention is made of the mechanism of desensitization, which I believe involves IgE 'blocking antibodies'.

Finally, the three full pages of references, 175 in all, must be a record. Unfortunately many seem to come straight from the pages of *Index Medicus* without being discussed in the essay, whilst many contentious points are made without any quoted references.

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JAMES MACKENZIE LECTURE

Sir,

Professor J. McCormick has challenged (April *Journal,* p.248) my statement in the 1978 Mackenzie Lecture (January *Journal,* p.7) that in Mackenzie's day coronary thrombosis "was not there". He supports his belief that Mackenzie "encountered many infarctions" by referring to case histories described in Mackenzie's two books *Diseases of the Heart* (1913) and *Angina Pectoris* (1923) suggesting that "in many instances cases are supported by careful postmortem evidence".

Study of these books reveals no support at all for Professor McCormick's viewpoint:

1. Of the 160 cases detailed in *Angina Pectoris,* only 22 were subjected to postmortem examination which Mackenzie described as "somewhat imperfect". Of the 22 postmortem cases, at the very most 10 demonstrated myocardial degeneration due only to ischaemia associated with coronary atheroma. The remainder had suffered from rheumatic endocarditis, syphilitic aortitis, or from some other disease.
2. From the histories of the 138 cases not subjected to postmortem (some were still living at the time of writing) it is impossible in many instances to make any diagnosis at all. Some were probably functional, others appeared to be suffering from rheumatic or syphillitic cardiac disease, or from diseases of the lung, or from cancer.
3. The importance of valvular disease or syphilitic aortitis as a cause of angina in those days is further demonstrated by Mackenzie's statement *(Angina Pectoris,* p.89) that 90 cases of angina—presumably out of a total of 284 whose deaths he had recorded—were associated with aortic incompetence and that "angina pectoris in early life always raises the suspicion of syphilitic aortitis" (p. 78).
4. I could find no record at all of death occurring within 48 hours of onset of angina in a previously healthy patient.
5. Nor could I find any record of typical non-fatal attacks occurring in a previously symptom-free patient.

For an understanding of the history of coronary disease it is essential to remember the two main manifestations of anginal attacks (Michaels, 1966).

The first manifestation, due to coronary ischaemia progressing over a number of years, often going on to death in old age when the myocardium may be found to be degenerate and fibrosed, was first described by Heberden in the eighteenth century (Heberden, 1802). It was not very common until the present century (Osler, 1910). Mackenzie himself suffered from this form of angina.

The second manifestation—sudden infarction coming out of the blue in a previously symptom-free patient, ending in death within a few days or in recovery—is now so common that it is often described as an epidemic. No description of this second manifestation appeared in medical literature before the present century. It was not recorded in Edinburgh before 1928 (Gilchrist, 1971) and it does not appear to have been known in Glasgow in the mid-1920s (Henderson, 1964). We still do not understand the pathology of coronary thrombosis *(BMJ,* 1979) nor the relationships between thrombus formation and arterial disease, but I believe there is no doubt at all of the fact that widespread infarction striking men in their 40s, 50s, or 60s in large numbers is a new disease of the twentieth century.

I am sorry that Professor McCormick found part of my lecture "impossible". I was merely trying to say that if we are to understand the cause, and so prevent the degenerations which beset us, we must study nature's laws and seek to obey them. If some readers find this theme impossible I doubt if I can help them, but I implore them at least to read some of my references, particularly *The Saccharine Disease* (Cleave, 1974) from whose work so much of my material and inspiration originated.

Before shrugging it all off as "impossible" readers should be reminded that Cleave has this year been awarded the Harben Gold Medal for his services to public health, an honour which he thus shares with names such as Pasteur, Lister, Koch, Ross, and Fleming.

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