tunities for surgical consultants have therefore changed little in the past quinquenium.

Similarly, with regard to the senior registrar grade in general surgery, again there has been only small improvement. The average age of the senior registrar group has decreased considerably, and parity has almost been established between the number of senior registrars completing training and the number of consultant vacancies available each year. This is certainly a step in the right direction, but there has been no appreciable increase in the number of senior registrars completing training each year or in the number of new consultant posts available. On the other hand no attempt whatsoever has been made to deal with the registrar problem, to which we drew attention three years ago.[3] In 1965 there were 626 general surgical registrars competing for 153 senior registrar posts and in 1970 there were 644 surgical registrars competing for 138 senior registrar posts. Thus probably at present there are five middle-grade registrars in post for every senior surgical registrar—clearly a situation which cannot be allowed to continue. Further progress cannot be made unless the number of registrar training posts is substantially reduced and related to the 46 or so vacancies occurring each year in the senior registrar establishment. These proposals are inextricably linked with those of coping with the work load and the service commitment to patients, especially in the district hospitals. The present proposals for an appreciable increase in the number of surgical consultants will, in part, help to rectify this, but it is also vital that a realistic career structure should be devised if the present anxieties of the hospital junior staff are to be allayed.

What has been achieved for the senior registrar grade now needs to be attempted for the registrar grade, and the Joint Committee for Higher Surgical Training and other interested bodies should consider this urgently. The gentle breeze we noted in 1968 continues to blow in the field of postgraduate surgical education. But it is not yet a wind of change, and furthermore not all the nods it has produced are those of acquiescence.

[1] Joint Committee for Higher Surgical Training, *Second Report*. London, Royal College of Surgeons of England, 1971.
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[3] *British Medical Journal*, 1968, **4**, 401.
[4] *Lancet*, 1966, **2**, 1456.
[5] *Lancet*, 1970, **1**, 944.
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Non-rheumatic Myopericarditis

Acute rheumatic carditis may be in danger of passing unrecognized as the classical form of rheumatic fever becomes rarer. In elderly patients, especially, bacterial endocarditis may likewise remain undiagnosed until irreversible cardiac damage ensues. The report of the Rheumatic Fever Working Party[1] clearly showed that the long-term effects of rheumatic carditis are closely related to the degree of cardiac damage at the time of initial diagnosis and treatment. That early recognition affects the long-term prognosis in other forms of myocarditis is probably also true. Though diphtheritic and Fiedler's[2] (idiopathic or isolated) myocarditis were recognized in the past, "myocarditis" on its own was long considered a questionable diagnosis reached as a last resort.

It is only rather recently that the large number of potential causes of myocarditis have been appreciated. They include toxic substances—such as emetine and alcohol—bacteria, spirochaetes, rickettsiae, fungi, parasites, and viruses, and autoimmune reactions. However, in most of these conditions the generalized systemic reaction or disease dominates the clinical picture and the cardiac symptoms are of minor importance.

Such causes of myocarditis were initially identified from necropsy studies.[3] Overt clinical disease is usually rare in the acute stage, but advances in laboratory techniques have allowed viruses in particular to be more frequently recognized. Laboratory diagnosis is based on culture from faeces and sputum and from changing antibody titres to a specific virus. Poliomyelitis virus has been cultured from heart muscle. Though many viral illnesses have been associated with the occasional case of myocarditis (including psittacosis, measles, mumps, variola, vaccinia, and infectious mononucleosis), the most commonly involved are the Coxsackie viruses. These are divided into groups A and B (with a number of subgroups) according to the pattern of disease they induce in suckling mice.

Coxsackie B can induce pericarditis, myocarditis, and meningitis in man. As well as causing Bornholm disease (or epidemic pleurodynia) it is responsible for some cases of "acute benign pericarditis." A report from Colindale in 1967[4] on 1, 160 Coxsackie B5 infections noted that 31% had meningeal symptoms, 23% myalgia or Bornholm disease, 15% respiratory disease, and 5% cardiac involvement (with two deaths). W. G. Smith[5] reported 10 adult cases of Coxsackie B myocarditis, with seven complete recoveries. Most previous reports of this condition from newborn babies and infants had shown a far gloomier prognosis.[6] T. W. Mattingley[7] also noted this difference between the high infant and low adult mortality from this disease.

In many epidemics, largely affecting adults, electrocardiographic changes have indicated myopericarditis.[8] Between 5 and 12% of patients have been affected.[9] G. S. Sainani[10] suggested that some of the patients might end up with chronic cardiac lesions, but the frequency with which acute attacks progress to chronic heart disease remains unknown. J. F. Goodwin[11] reported a follow-up of 74 patients with "primary congestive cardiomyopathy," of whom 55% had died. In some of these cases alcohol, and in others infection, may have caused the heart condition. E. N. Silber[12] reported cases associated with respiratory infections, some of influenzal origin, and recorded their subsequent course as acutely fatal, acute with recovery, subacute with recovery, or chronic with late death.

The clinical presentation of such patients has often been delayed and so recognized only at an advanced phase of cardiac dysfunction, usually when the patient has frank pulmonary oedema or congestive failure. When recognition was so late the prognosis was poor. J. P. Segal and colleagues[13] reviewed 159 cases of "idiopathic" myocarditis and noted certain earlier signs—conduction disturbance and dysrhythmias, particularly ectopic beats and atrial fibrillation. On radiography the size of heart was usually but not always shown to be increased. Auscultation might disclose a pericardial rub, an atrial triple rhythm, or summation gallop. Most authors agree that E.C.G. changes are nonspecific, with changes in S-T and T waves predominating. Most forms of myocarditis, like the cardiomyopathies, are apt to give rise to mural thromboses, leading to the risks of systemic or pulmonary emboli, and these may even be the presenting complaint.

The milder forms of acute myocarditis are often unrecognized clinically. Their prevalence is unknown, but they must be rare in relation to the frequency of viral disease. Features to alert the clinician to the diagnosis include tachycardia out of proportion to the patient's temperature and in the absence of anaemia or thyrotoxicosis; both of these must be remembered in the differential diagnosis. Suggestive symptoms include a recent respiratory infection followed by undue fatigue, pleural or pericardial pain, dyspnoea, and palpitation. On examination, excessive tachycardia at rest and on mild effort, or ectopic beats on effort, may be found. A triple rhythm is a more frequent auscultatory finding than murmurs. The E.C.G. may show changes in the T and R-T waves, and radiographs may show lung field changes and cardiomegaly. Confusion can be caused by the chance finding of abnormal signs or E.C.G. changes in patients already suffering from one of the chronic cardiomyopathies; however, in these patients the more severe E.C.G. changes are usually diagnostic. Occasionally "silent" coronary disease is suspected, and coronary angiography may be required to exclude this possibility.

Therapeutically, rest has been shown to be the best measure[14] in a variety of forms of myocarditis.

As the disease becomes better recognized it is increasingly clear that myocarditis in adults no longer has so gloomy a prognosis as was formerly believed. K. Bergström and colleagues[15] have studied 15 such adults one to four years after the acute illness (excluding cases of rheumatic carditis). In the acute illness of these patients presenting symptoms and signs included tachycardia, precordial pain, pericardial rub, and triple rhythm. Serum aspartate aminotransferase (SGOT) and the erythrocyte sedimentation rate were increased in some cases, and electrocardiographic changes were found in most. Follow-up studies showed that 14 of the 15 patients returned to work after 1½-8 months. E.C.G. changes were still found in five patients; 13 were clinically well one to four years later. The authors advised cautious physical training as part of the convalescence for their patients. As with rheumatic carditis, it would appear that the long-term prognosis may be related to the degree of myocardial damage at the time of the initial diagnosis, when bed rest is likewise the most effective treatment.

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Fats and Multiple Sclerosis

Approaches to the effective treatment of multiple sclerosis start from scattered points and follow diverse trails.[1] In recent years the application of refined biochemical methods to the analysis of body tissues has disclosed or elucidated abnormal chemical functions in many complex neurological disorders, and it is a constant hope that on the basis of such evidence a satisfactory interpretation of the cause of the disease may open the way to cure.

R. L. Swank[2 3] has drawn attention to the possibility that the high incidence of multiple sclerosis in some parts of the world can be related to dietary habits and that in those areas diets generally tend to be rich in saturated fatty acids. He has also suggested that modification of the diet for patients with multiple sclerosis in such a way as to reduce their intake of total fat and include a greater proportion of unsaturated fatty acids will improve the patient's functional condition and reduce the relapse rate. Furthermore biochemical studies of the brain lipids have shown that in cases of multiple sclerosis the relative proportions of saturated and unsaturated fatty acids are different from their proportions in healthy people.[4] R. H. S. Thompson[5] also noted a lowering of the serum linoleate in patients with this disease. Another thought-provoking fact was the discovery[6] that rats bred on a diet deficient in unsaturated acids showed an increased susceptibility to the development of experimental allergic encephalitis, which is regarded by some authorities as a disorder similar to multiple sclerosis.

Now J. Belin and colleagues[7] have carried this line of chemical investigation a step further. They have studied the levels of oleate and linoleate in the serum lipids of patients suffering from multiple sclerosis and of controls, and they have shown that the percentage of linoleate in the patients with multiple sclerosis was lower than in the controls while that of the oleate was higher. The two groups of patients were then fed with linoleate supplement in the form of an emulsion based on sunflower seed oil. In both groups the percentage of the linoleate levels rose strikingly, but it seemed to fall rather more rapidly in the patients with multiple sclerosis after the period of feeding was over. They have also provided other interesting details about the modification of the levels of oleate and linoleate in various separated fractions of lipids in the serum. Belin and his co-workers wisely refrain from making dogmatic assertions on the basis of their findings and accept the fact that the low linoleate levels in multiple sclerosis are difficult to explain. Since the absorptive mechanism is unimpaired, they have suggested tentatively that the rate of loss or of utilization of linoleate may be abnormally high in patients with multiple sclerosis. Current work by K. J. Zilkha and colleagues[7] on supplemental feeding with linoleate thus deserves to be continued.

The precise meaning of these biochemical studies in relation to the aetiology of multiple sclerosis remains obscure. It seems unlikely that they point to any direct causal link, but perhaps the unsaturated fatty acids modify in some protective way a process of acute demyelination which is initially triggered by an entirely different mechanism. Advocates of the allergic and slow virus theories will therefore not be upset by the linoleate experiments. But certainly there seems to be little harm in adding either sunflower seed or corn oil to the normal diets of patients suffering from multiple sclerosis. We can await the long-term outcome with interest.

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