Refining thinking on type A behaviour and coronary heart disease

Clinical observation of patients led cardiologists in the late 1950s to describe a "coronary prone behaviour pattern" (the type A behaviour pattern). The features of type A behaviour include an aggressive competitiveness together with an intense, sustained drive for achievement, a pressing sense of time urgency (struggling against time deadlines), and hostility, which is often well rationalised. Those who show these characteristics are designated type A; those who do not are designated type B. Although reference is often made to a type A personality, the type A behaviour pattern is not a strictly personality type. Rather it is a constellation of behaviours manifested under particular circumstances (often stressful) by susceptible individuals.

Type A behaviour can be measured reliably using Rosenman's structured interview, which has recently developed into a videotaped clinical interview. Attempts have been made to show specific biochemical or physiological correlates of type A behaviour that might account for its relation to coronary heart disease, but the findings have been inconsistent. Many apparently relevant studies may, however, be faulted. For example, critics highlight the failure of some studies to correlate type A behaviour with coronary artery disease assessed angiographically, but such differences may be caused by selection bias. Further, the association between the type A behaviour pattern and coronary heart disease may be independent of a correlation with angiographic abnormalities.

Three large prospective American studies of people free of coronary heart disease have examined whether the type A behaviour pattern will predict coronary heart disease. In the Western Collaborative Group and Framingham studies the incidence of coronary heart disease was significantly greater in type A than type B subjects even after controlling for other risk factors such as cigarette smoking, blood pressure, and serum cholesterol concentration. Shekelle et al failed to confirm these results, but some of their other findings are difficult to explain. For example, their study differed from the other two in specifically selecting those at high risk of developing coronary heart disease. Yet their subjects had a lower mean annual incidence of myocardial infarction or sudden cardiac death than comparable subjects in the other two studies.

Is the type A behaviour pattern applicable only to middle class American men? Clearly no, because type A behaviour is associated with a significantly increased prevalence, incidence, or both, of coronary heart disease in both sexes and in various cultures. Depending on the method of assessment, the type A behaviour pattern is independent of other common emotional or personality variables and of social class. In the first large British prospective study of the type A behaviour pattern Johnston and his colleagues (p 86) confirm an association of type A behaviour with the prevalence of coronary heart disease, but not with the subsequent incidence of myocardial infarction.

Type A behaviour is not a unitary concept. The structured interview rates observed behaviour as well as the responses to specific questions, and self rating questionnaires, although easier to use, clearly do not allow observational ratings; their scores therefore show only modest correlations with those obtained using the structured interview. Furthermore, the various self rating instruments also correlate poorly with each other, suggesting that each measures a different component of the type A behaviour pattern. Which components are most important in contributing to coronary heart disease is still not clear, which complicates comparisons between studies using different methods of assessment.

Type A patients who have had a myocardial infarct show characteristic components of the type A behaviour pattern that differ from those of patients with angina, raising the possibility that different aspects of type A behaviour may be associated with different manifestations of coronary heart disease. This could contribute to the finding by some researchers (including Johnston et al) of a positive association between type A behaviour and either the prevalence or incidence of coronary heart disease but not both, since samples have different proportions of particular types of coronary heart disease.

The nature of type A behaviour requires that particular care should be taken to avoid systematic bias in patient selection. In a recent study of patients who had survived a myocardial infarct 318 of 866 declined to participate in the type A behaviour pattern assessment, which might explain the failure to find an association of the behaviour with coronary artery disease. Similarly, patients referred for coronary artery angiography or admitted to a coronary care unit cannot be considered representative of those with coronary heart disease. In patients admitted to a coronary care unit Ahnve et al found the highest type A scores among those who turned out not to have coronary heart disease.
Renal micropolyarteritis

The kidneys are commonly affected in polyarteritis, and several recent reports suggest that they are most often affected when the inflammation damages mainly the smaller arteries and the vessels distal to them. This renal disease is now often called micropolyarteritis, though the systemic manifestations are still described as angiitis or vasculitis; the term allergic, hypersensitivity, malignant, or necrotising is sometimes added to draw attention to other important aspects of the condition.

Because the vascular lesions are not confined to the kidney patients are often referred initially to general physicians, dermatologists, or rheumatologists. Gastroenterologists may see patients with disease of the gut, liver, or spleen, and the eyes are often affected. Wegener’s granulomatosis, which is a closely associated condition, may present to the ear, nose, and throat or chest department. Infection will often be suspected because of a flu-like prodromal illness or because of the fever and malaise that usually accompany renal micropolyarteritis.

The kidneys may be affected early or late in the disease, and any doctor managing a case of proved or suspected angiitis must regularly check the plasma creatinine concentration and examine the urine for red cells and casts. A nephrologist should be called immediately renal damage is suspected as it may progress rapidly to advanced renal failure, which not only reduces the chance of retrieving renal function but also increases the hazards of immunosuppressive treatment.

In a patient with renal micropolyarteritis the doctor is more likely to be faced with the diagnosis by biopsy of the kidney rather than of more accessible tissues, and renal biopsy has been shown to be safe. The histological appearances have been well described, but the typical foci of vascular and glomerular necrosis may be absent and the diagnosis may still remain extrarenal evidence. Seldom is there evidence of immune complex deposition, although tests for circulating complexes are often positive. In a patient with a lung haemorrhage or purpuric rash immunohistological investigation may, be helpful since it will show the absence of the linear immunoglobulin pattern of Goodpasture’s syndrome and the IgA deposits typical of Henoch-Schönlein syndrome.

Though spontaneous remission has been reported, immunosuppressive treatment should not be delayed if the patient has haematuria, uraemia, or a raised erythrocyte sedimentation rate. Many treatment regimens have been tried, but the yardstick against which they must be measured is the prednisolone-cyclophosphamide combination described by Fauci’s group. They give excellent advice about adjusting doses to fit the severity of the disease, both initially and during follow-up. Their results in Wegener’s granuloma and vasculitis of the kidney are far better than those in earlier reports.

The recent studies from London and Sheffield confirm that the prognosis in renal micropolyarteritis is more favourable than was believed and better than in classic polyarteritis nodosa. Most patients now survive the first few months with useful renal function, and they then have an excellent chance of avoiding long term haemodialysis. Even patients requiring dialysis sometimes improve enough for it to be stopped.

The most recent development is that workers in Holland and Bass and Wade in their study of patients referred for coronary artery angiography found higher type A scores among those with normal coronary arteries. Although type A people may not admit to cardiovascular symptoms under some circumstances, one who presents in casualty might insist on being thoroughly investigated. Similarly, type A people seem to experience more stressful life events than type B people, particularly related to work, although it might be that type A people report more work associated events than their type B colleagues.

Clearly the type A behaviour pattern is complex, as is its association with coronary heart disease, and we need to recognise shortcomings in the studies done so far. Even if well designed studies show a significant association between the type A behaviour pattern and coronary heart disease they will not prove causation. The best evidence would come from intervention studies, and Friedman and colleagues have reported that reducing type A behaviour by counselling after myocardial infarction leads to a dramatic and sustained drop in subsequent cardiac morbidity. This kind of result surely justifies further investigation of the type A behaviour pattern rather than its dismissal for its inconsistent track record.

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