Editorial comment
Spontaneous early clinical remission of myocarditis without immunosuppressive treatment

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When cases of "cardiomyopathy" make an unexpected recovery from critical heart failure it is likely that they were suffering from myocarditis. Even the lay press has given publicity to young previously fit young people suddenly smitten by devastating illness, saved from imminent demise by introduction of a pump assist device and progressing to apparent full recovery. The drama is in the removal of the device because the heart had recovered. It had not after all been needed as a "bridge to transplantation" but only as a "bridge to recovery".

The potential for recovery in life threatening myocarditis has long been known to cardiologists[1] and never ceases to surprise because there have been no clinical criteria to predict which patients will do well. Some recover seemingly normal ventricular function but biopsies during the acute phase suggest that some cardiovascular reserve must have been lost because myocytolysis is one of the diagnostic criteria[2]. Another unanswered question is how many will progress to dilated cardiomyopathy. Women with *peripartum* "cardiomyopathy" show changes of acute myocarditis if biopsied early after onset[3]. Fulminating cases have been transplanted but as considerable recovery occurs in many but not all survivors, transplantation in the acute phase is not appropriate.

In a recent study 147 cases of myocarditis seen over a 13 year period were followed up for an average of 5.6 years. Fifteen cases were classed as fulminant because of their severity with rapid onset and fever. Of these 93% were alive without transplant 11 years after biopsy compared with 45% of 132 cases with only "acute" myocarditis[4]. Both in this study and an earlier one left ventricular dilatation was not as pronounced in patients with fulminant myocarditis who were more likely to recover than clinically milder cases with bigger ventricles[1].

The prevalence and natural history of acute myocarditis remain unknown because most cases are not recognised. Although sudden death may occur, symptoms may be nonspecific or absent. The causes of myocarditis are numerous and this too will influence the natural history. Reversible toxic myocarditis occurs in diphtheria and sometimes also in infective endocarditis. Myocarditis in sarcoidosis and the collagen vascular diseases usually result from an autoimmune vasculitis. Some medicines such as emetine and industrial solvents such as the halogenated hydrocarbons can also be toxic to the myocardium.

Most cases of myocarditis with onset in otherwise healthy people probably have an infectious origin, although the pathogenesis is not yet fully understood and an aetiological agent is rarely found. Persistent viral infection of the myocardium was first demonstrated a decade ago. In western countries entero-viruses, especially coxsackie B 1-6 serotypes, are the most frequent. Diagnosis is easiest during epidemics but difficult in isolated cases. These are not seen by cardiologists unless they suffer chest pain or develop arrhythmia, heart failure or collapse, the majority being dealt with in the primary care system.

Chest pain may mimic myocardial infarction. Chest pain may also be due to associated acute pericarditis but the two do not always go together and the clinical em-

phasis is usually on one or the other. Whereas arrhythmias or conduction disturbances may be life threatening with only mild focal injury, more widespread inflammation is necessary before cardiac dysfunction is sufficient to cause symptoms.

Although evidence of myocyte necrosis, myocytolysis is one of the requirements for biopsy diagnosis and clearly is not reversible, the rapid improvement in cardiac function described in the paper by Bossone et al.[5] must reside in a rapidly reversible paresis of viable myocytes by cytokines such as tumour necrosis factor alpha and nitric oxide synthase but what turns the expression of these cytokines on or switches them off again before they have caused irreparable injury is unknown.

The importance of autoimmune mechanisms was investigated in a randomised controlled trial of the use of immunosuppressive agents in biopsy proven cases of acute myocarditis[6]. Benefit was not shown but the trial was weakened by failure to include immunohistochemical techniques in the biopsy criteria as well as by frequent protocol violations.

The 6 patients described by Bossone et al.[5], who enjoyed rapid spontaneous recovery, showed no significant dilatation either of the left ventricle or the left atrium in 5 of the 6 cases, although severe depression of contractile function had put the patients into NYHA class III or IV heart failure. The 9 patients who made slower progress with less complete recovery showed greater chamber dilatation as determined by transthoracic echo. The biopsy diagnosis was based on the Dallas criteria and thus did not include immunohistochemical evidence[2].

Myocardial depression was transient in the 6 out of 15 patients who improved spontaneously and the ejection fraction at discharge was already back to normal, the patients clinically well. With such rapid improvement it is remarkable that biopsy was carried out at all. The recognition of their acute myocarditis was clearly dependent upon having been admitted to a university hospital. Their longer term well being will be of great interest. If they were like McCarthy and Lieberman's fulminant cases they should do well but Bossone's cases may yet fall into the common category of patients who present with a dilated cardiomyopathy but have no memory of a transient acute illness.

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