Tachycardia-induced cardiomyopathy caused by atrial flutter responding to DC cardioversion

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DISCUSSION

The concept that chronic tachycardias can lead to reversible left ventricular (LV) dysfunction is supported by animal models of chronic rapid pacing as well as human studies of improvement in LV function following tachycardia control (Shinbane et al, 1997). Experimental tachycardia cardiomyopathy was first described in 1962 (Whipple et al, 1962) and subsequently it has been demonstrated in numerous models that sustained rapid pacing can produce severe biventricular dysfunction.

Haemodymamic changes occur as early as 24 hours after pacing with continued deterioration in ventricular function for 3–5 weeks resulting in end-stage heart failure. Following termination of pacing there is significant improvement in LV ejection fraction (EF) with normalization after 1–2 weeks (Chow et al, 1990).

In humans, reversible cardiomyopathy with rate or rhythm control of incessant tachycardias have been described, usually in the context of atrial fibrillation, although similar findings have been seen with other atrial arrhythmias (Grogan et al, 1992). Several studies have documented a reversal of heart failure in such patients with atrial fibrillation after cardioversion to sinus rhythm (Kieny et al, 1992).

Our patient developed a dilated cardiomyopathy following surgery in conjunction with atrial flutter. On maintenance of sinus rhythm there was a significant improvement in ventricular dimensions and contractile function which returned to presurgical values (*Figure 1*). It should be pointed out that the initial measurement of LV function was made before surgery (demonstrating good contractile function with an EF of 49%) and that no absolute measurement of LV function was made in the immediate postoperative period before the patient developing atrial flutter. There is, however, no supportive evidence that the surgery itself caused his LV function to become significantly depressed.

CASE REPORT

A 58-year-old man presented in February 1996 to his local hospital with a true posterior myocardial infarction for which he received thrombolysis. He subsequently underwent exercise testing which was terminated because of chest pain and anterior ST depression of >2 mm after 6 minutes of a Naughton Balke protocol. He proceeded to coronary angiography which demonstrated severe triple vessel coronary artery disease. The left ventriculogram demonstrated posterior hypokinesis but overall left ventricular (LV) contractile function was well preserved with an ejection fraction (EF) calculated at 49%. He was in sinus rhythm and had chronic stable angina.

In September 1996 he underwent revascularization on prognostic grounds. Five days postoperatively he developed atrial flutter for which he was started on amiodarone 200 mg once daily and subsequently discharged. Two weeks later he was readmitted with progressive dyspnoea and peripheral oedema. He was found to be in clinical heart failure and atrial flutter with a ventricular rate of 150. He subsequently underwent a successful DC cardioversion reverting to sinus rhythm following a single 200J DC shock. He was discharged on amiodarone 200 mg once daily, frusemide 80 mg once daily, enalapril 10 mg once daily and aspirin 75 mg once daily.

In November 1996 he presented in atrial flutter with heart failure. An echocardiogram was performed showing a dilated hypokinetic left ventricle with an EF of approximately 20% (*Table 1*). He was anticoagulated and underwent a further DC cardioversion in February 1997 which successfully reverted him to sinus rhythm. Echocardiography was performed following reversion to sinus rhythm (*Table 1*) which revealed a dilated LV with global systolic dysfunction and functional mitral regurgitation (EF=30%). In April 1997 he reverted to atrial flutter and had significant effort dyspnoea with an exercise tolerance of 50 yards. He was again successfully cardioverted and commenced on sotalol 80 mg twice daily in an attempt to maintain sinus rhythm.

He subsequently remained in sinus rhythm with a marked symptomatic improvement (New York Heart Association Grade 1 dyspnoea and no clinical evidence of cardiac failure). Echocardiography in July 1997 demonstrated a significant improvement in LV dimensions and systolic contractile function (EF=50%) with values equivalent to pre-surgery.

TABLE 1.
Improvements seen on sequential transthoracic echocardiograms

	November 1996	February 1997	July 1997
Rhythm	Atrial flutter	Atrial flutter	Sinus rhythm
Left ventricular end-diastolic dimension (mm)	60	67	57
Left ventricular end-systolic dimension (mm)	54	57	42
Fractional shortening (%)	11	15	26
Ejection fraction (%)	20	30	50
Aorta (mm)	32	33	36
Left atrium (mm)	44	40	30
Right ventricle (mm)	33	34	25

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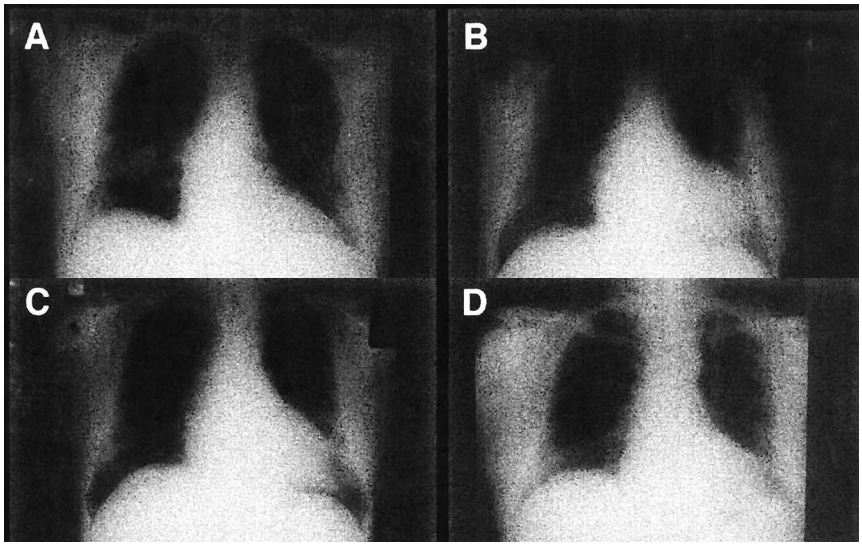

Figure 1. Serial chest radiographs demonstrating reversible cardiomegaly developing with atrial flutter (AF) with normalization following restoration of sinus rhythm (SR). a. February 1996 (SR). b. February 1997 (AF). c. April 1997 (AF). d. August 1997 (SR).

First there was no evidence of perioperative infarction which may have contributed to reduced LV function following surgery (there were no diagnostic electrocardiographic changes of infarction during this period and no cardiac enzyme rise). Another mechanism by which depressed LV function may occur following cardiac surgery which is generally fully reversible would be the phenomenon of myocardial stunning. Stunning has been shown to occur following coronary artery bypass grafting but studies have shown that the LV dysfunction caused by stunning in the context of cardiac surgery recovers completely in 24–48 hours (Gray et al, 1979; Reduto et al, 1981).

The prolonged LV dysfunction in our patient cannot be explained by either myocardial stunning or infarction. It seems very likely that his LV dysfunction was due to his incessant arrhythmia. This is supported by the fact that his EF and ventricular dimensions normalized after termination of the arrhythmia. These findings are consistent with a reversible tachycardia cardiomyopathy which responded to cardioversion and restoration of sinus rhythm. HM

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Grogan M, Smith HC, Gersh BJ, Wood DL (1992) Left ventricular dysfunction due to atrial fibrillation in patients believed to have idiopathic dilated cardiomyopathy. *Am J Cardiol* **69:** 1570–3

Kieny JR, Sacrez A, Facello A et al (1992) Increase in radionuclide left ventricular ejection fraction after cardioversion of chronic atrial fibrillation in idiopathic dilated cardiomyopathy. *Eur Heart J* **13:** 1290–5

Reduto LA, Lowrie GM, Reid JW (1981) Sequential post operative assessment of left ventricular performance with gated cardiac pool imaging following aortocoronary bypass grafting. *Am Heart J* **101:** 59–66

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Whipple GH, Sheffield LT, Woodman EG, Theopilis C, Friedman S (1962) Reversible congestve heart failure due to chronic rapid stimulation of the normal heart. *Proc N Engl Cardiovas Soc* **20:** 39–40

In the Public's View...

The Patients' Association and informed consent: a conflict of interest?

Sorry, I preferred Clare Rayner when she was an agony aunt. She spoke a good deal of sense. She had the advantage of looking like someone's favourite aunt, someone you really could go to with a problem and get a friendly cuddle as well as advice. Now, as a spokesperson for the Patient's Association, she is a leading player in the current wave of criticism of doctors.

In the current climate, it wasn't long before medical research was likely to come under fire, and it was always likely to involve babies. When the media coo over medical miracles saving tiny babies, do they think the wonder treatments emerged suddenly one morning, falling out of a cereal packet? Almost all effective treatments that are not obviously effective will have undergone trials in which selfless patients have been the subjects. Informed consent for medical research is a difficult enough subject for simple studies in adults; it is even more difficult when the subjects are babies. Now some parents in the Midlands are complaining that their babies were entered into studies of a new type of ventilator, but were told only of the disadvantages of the old type, and were unaware that it was research at all.

Enter Clare Rayner, in conversation with the chairperson of the BMA ethics committee on BBC Radio 4's *Today* programme. First she assumed that the parents complaint was valid. We do not know yet; there is to be an enquiry. By 1989, when the research began, informed consent was taken seriously. If informed consent was not obtained that is not just unsatisfactory; it is indefensible. But we do not know. It did not stop Clare Rayner speaking as if research doctors did not usually bother.

Her suggestion for avoiding abuse of research subjects was a patient advocate: someone who could be with the patient and doctor while the research was being discussed, to help them ask questions and understand what was being asked of them. She also asked for patients to be given more time. This is all very well, but time is something that researchers in neonatal medicine don't have much of.

Clare Rayner did not expand on where we were expected to find all these trained and knowledgeable patient advocates, needed at all hours and for all sorts of different research projects. Then it occurred to me that these people did used to exist. They were called nurses. But now too many nurses want or have to be more like doctors and managers, which inevitably alters their relationship with patients.

And Clare, patients sometimes do not remember very well. Seeing a fit, healthy man preoperatively for an operation on his prostate, I just mentioned that he was, of course, in the laser study, wasn't he? The patient denied all knowledge despite, 3 days earlier, having been given an information sheet and having signed a research consent form. HM

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