

LETTERS TO THE EDITOR

- *The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.*
- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 600 words.*
- *In general, no letter should contain more than six references (also typed with double spacing).*

Permanent pacing after cardiac transplantation

SIR,—Scott *et al* reported that only a few patients given a permanent pacemaker subsequently needed long-term rate support.¹ This accords with our experience and that of other groups.² We recently learned, however, that what appears to be transient sinus node dysfunction may not be.³ Only one out of six patients fitted with AAI(R) pacemakers that allowed for follow-up determination of sinus node recovery time also had a normal recovery phenomenon. One patient who had been in slow junctional escape rhythm until late in the postoperative period had permanent pacemaker placement before discharge. One year later he was in sinus rhythm at a rate of about 85 beats/min and a Holter recording showed that he was overriding the pacemaker most of the day. This accorded with the findings of Scott *et al* and Markewitz *et al*.^{1,2} The recovery phenomenon, however, was grossly abnormal with a postpacing pause of more than 4 s during which he had symptoms.³

While it is clear that an abnormal recovery time is not in itself an indication for pacemaker placement, as we stated in our original version of our paper,³ this may not be true in a patient with a cardiac transplant who has had symptoms. Though much is known about the incidence of sinus node dysfunction after cardiac transplantation, the actual incidence of symptoms remains unknown and may be underestimated because of the low threshold for postoperative pacemaker placement. In our series of 90 patients three recipients had to be given a pacemaker.⁴ Sinus node deficiency had been evident in these patients but its severity had been underestimated.⁴

We want to draw attention to the fact that late restoration of sinus rhythm in a patient with a heart transplant may indicate reversion to a latent type of sick sinus syndrome rather than normalisation of sinus node function. These patients may still be liable to severe symptoms.

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- 1 Scott CD, McComb JM, Dark JH, Bexton RS. Permanent pacing after cardiac transplantation. *Br Heart J* 1993;69:399-403.

- 2 Markewitz A, Schmoedel M, Noliert G, Überfuhr F, Weinhold C, Reichart B. Long-term results of pacemaker therapy after orthotopic heart transplantation. *J Cardiac Surg* 1993;8:411-6.
- 3 Heinz G, Kratochwill C, Buxbaum P, Kreiner G, Laufer G, Gössinger H, Laczovics A. Long-term intrinsic pacemaker function in patients paced for sinus node deficiency after cardiac transplantation. *PACE* 1992;15:2061-7.
- 4 Heinz G, Hirschl M, Buxbaum P, Laufer G, Gasic S, Laczovics A. Sinus node dysfunction after orthotopic cardiac transplantation: postoperative incidence and long-term implications. *PACE* 1992;15:731-7.

Are streptokinase antibodies clinically important?

SIR,—We read with interest the editorial by Dr M B Buchalter (1993;70:101-2) on the clinical significance of antistreptokinase antibodies and neutralisation titres. He suggests that it would seem sensible not to re-administer streptokinase until it is proven that these antibodies are of no clinical significance. The editorial concentrated on the persistence of antistreptokinase antibodies for at least four years after initial exposure to streptokinase.

A considerable number of patients re-infarct in the first few days after the initial infarction.¹ Lynch *et al* showed that antistreptokinase antibodies do not rise above pretreatment values until day four after the administration of streptokinase.² They did not measure neutralisation titres. We looked at the early values of antistreptokinase antibodies and neutralisation titres after streptokinase administration and found no rise in the neutralisation titres until day four after streptokinase.³ Therefore there may be an early window when streptokinase can be re-administered. These observations suggest that re-administration of streptokinase is appropriate during the three days after an initial dose of streptokinase or anistreplase. This has important financial implications in view of the price difference between streptokinase and non-antigenic thrombolytic agents.

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- 1 Rivers JT, White HD, Cross DB, Williams BF, Norris RM. Re-infarction after thrombolytic therapy for acute myocardial infarction followed by conservative management: incidence and effect of smoking. *J Am Coll Cardiol* 1990;16:340-8.
- 2 Lynch M, Littler WA, Pentecost BL, Stockley RA. Immunoglobulin response to intravenous streptokinase in acute myocardial infarction. *Br Heart J* 1991;66:139-42.
- 3 Lee HS, Cross SJ, Davidson R, Reid T, Jennings K. Raised levels of antistreptokinase antibody and neutralisation titres from four days to 54 months after administration of streptokinase or anistreplase. *Eur Heart J* 1993;14:84-9.

This letter was shown to the author, who replies as follows:

SIR,—I agree with Lee *et al* that as streptokinase antibodies do not seem to rise until day 4 after streptokinase administration, repeating the streptokinase before this time is likely to be safe and effective.

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Spontaneous ventricular defibrillation

SIR,—We read with pleasure the article of van Hemel and Kingma on self-terminating ventricular fibrillation.¹ They suggested that transient ventricular fibrillation is unusual and presumably occurs when there is a rapid and complete re-establishment of pre-existing normal electrophysiological properties in the myocardium. Moreover, they related the existence of transient ventricular fibrillation to the rapid electrophysiological improvement during the dynamic process of reperfusion.

We have worked on transient ventricular fibrillation for more than a decade and we want to question these two points. Firstly, transient ventricular fibrillation is not unusual. It has been reported in various mammals² and it is not so rare clinically.³ Secondly, during ventricular fibrillation there is no coronary circulation and reperfusion stops when ventricular fibrillation starts. For this reason the suggestion that an "increase in organisation of ventricular fibrillation into ventricular flutter" is a result of rapid electrophysiological improvement during the dynamic process of reperfusion (which does not exist during ventricular fibrillation) seems unlikely. Because reperfusion can not explain the synchronisation in ventricular fibrillation that takes place over time and leads to spontaneous defibrillation we propose an alternative explanation.

On the basis of our experimental studies⁴⁻⁶ we have hypothesised that transient ventricular fibrillation requires the maintenance of a high sympathetic and low parasympathetic level during ventricular fibrillation. This hypothesis was based on results obtained in studies⁴ showing that transient ventricular fibrillation is a normal feature in mammals that have predominantly sympathetic cardiac auto-regulation whereas sustained ventricular fibrillation occurs in those mammals that have predominantly parasympathetic auto-regulation.⁵ Moreover, it has been shown that sustained ventricular fibrillation can be transformed into transient ventricular fibrillation either by pretreatment with various compounds that increase the cardiac catecholamine concentration during ventricular fibrillation⁶ or by intracoronary administration of adrenaline during ventricular fibrillation.⁷

Our hypothesis suggests that a high cardiac catecholamine concentration improves intercellular coupling and intercellular synchronisation, leading different myocardial cells to fibrillate coordinately.⁷ This sympathetically induced synchronisation organises ventricular fibrillation into ventricular flutter and leads to subsequent spontaneous restoration of the sinus rhythm.

According to this assumption, the phenomenon described by van Hemel and Kingma can be related to the increases in sympathetic activity induced by ventricular fibrillation which increase the cardiac concentration of catecholamine over time. In a heart without structural heart disease, this increase in catecholamine concentration organises the configuration of the unsynchronised ventricular fibrillation into more organised "coarse" ventricular fibrillation, which in some cases terminates spontaneously.

The various problems involved in transient ventricular fibrillation were discussed