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.1 Only one out of six patients fitted with AAI(R) pacemakers that allowed for follow-up determination of sinus node function recovered a normal rhythm after pacing. We interpret our results as showing that, at least in some patients, sinus node dysfunction may be a transient phenomenon. We suggest that sinus node function may be assessed during a period of pacing by Holter monitoring. We also recommend that such an assessment be made at least four weeks after pacing.

H S LEE  S J CROSS  K JENNINGS
Department of Cardiology, Aberdeen Royal Infirmary, Aberdeen AB2 2ZB


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, re-equilibrating the streptokinase before this time is likely to be safe and effective.

M BUCHALTER
Department of Cardiology, University Hospital of Wales, Heath Park, Cardiff CF4 4XX

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 reinfarct in the first few days after the initial infarction.1 Buchalter 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 discharge rates 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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M BUCHALTER
Department of Cardiology, University Hospital of Wales, Heath Park, Cardiff CF4 4XX

Spontaneous ventricular defibrillation

SIR,—We read with pleasure the article of van Hemel and Kingma on self-terminating ventricular fibrillation.1 They suggest that transient ventricular fibrillation is unusual and presumably occurs when there is a rapid and complete re-establishment of pre-existing normal electrophysiological properties either by the myocardial fibres or 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 of ventricular fibrillation which takes place over time and leads to spontaneous defibrillation we propose an alternative explanation.

On the basis of our experimental studies4 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 studies showing that normal ventricular fibrillation is a rare event in mammals that have predominantly sympathetic cardiac autoregulation whereas sustained ventricular fibrillation occurs in those mammals that have predominantly parasympathetic autoregulation.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.8 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 catecholamines.9 In a heart with normal 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

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


Permanente pacing after cardiac transplantation

Gottfried Heinz

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