**LETTERS TO THE EDITOR**

- The British Heart Journal welcomes letters commenting on papers that it has published within the last 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**

**Sr,**—Scott et al reported that only a few patients given a permanent pacemaker subsequently needed long-term rate support. This accords with our experience and that of other groups.1,2 We recently learned, however, that what appears to be transient sinus node dysfunction may not be. Only one out of our 22 patients fitted with AAI (pace- 

makers that allowed for follow-up determi-

nation of sinus node recovery time also had a normal recovery phenomenon. One patient who had in slow junctional escape rhythm until late in the postopera-

tive 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 accords with the findings of Scott et al and Markewitz et al.3 The recovery phenomenon, however, was grossly abnormal with a postspacing pause of more than 4 s during which he had symptoms.4

While it is clear that an abnormal recov-

er time is not in itself an indication for pacemaker placement, as we stated in our original version of our paper, 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 postopera-

tive pacemaker placement. In our series of 90 patients three recipients had to be given a pacemaker.5 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 syn-

drome 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, Beaton RS. Permanent pacing after cardiac transplan-


2 Markewitz A, Schmoeckel M, Nollet G, 

Uferfuhr P, Weinhold C, Reicht M. Long-

term results of pacemaker therapy after 

orthotopic heart transplantation. J Cardiac 


4 Heinz G, Hirsch M, Busubam P, Laufer G, 

Gasic S, Laczovics A. Sinus node dysfunc-

tion after orthotopic cardiac transplantation: 


**Are streptokinase antibodies clinically important?**

**Sr,**—We read with interest the editorial by Dr M B Buchalter (1993;70:101–2) on the clinical significance of antistreptokinase antibodies and neutralisation tites. 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 anti-

bodies 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 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 tites. We looked at the early rise of antistreptokinase anti-

bodies and neutralisation tites after strept-

okinase administration and found no rise in the neutralisation tites 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 strep-

tokinase and non-antigenic thrombolytic agents.

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1 Rivers JT, White HD, Cross DB, Williams 

BF, Norris RM. Re-infarction after throm-

bolytic therapy for acute myocardial infarc-


2 Lynch L, Littler WA, Pentecost BL, Stockley 

RA. Immunoglobulin response to intra-

venous streptokinase in acute myocardial infarc-


3 Lee HS, Cross SJ, Davidson R, Reid T, 

Jennings K. Raised levels of antistreptoki-

nase antibodies and neutralisation titres from four days to 54 months after administration of streptokinase or anistreplase. Eur Heart J 1993;14:94–9.

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

**Sr,**—I agree with Lee et al that as strepto-

kinase antibodies do not seem to rise until 

day 4 after streptokinase administration, 

testing the streptokinase before this time 

is likely to be safe and effective.

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**Spontaneous ventricular defibrillation**

**Sr,**—We read with pleasure the article of van Hemel and Kingma on self-terming 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 in the myocardium. We recently recognised the related 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 reper-

fusion 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 reper-

fusion cannot explain the synchronisation between ventricular fibrillation that takes place over time and leads to spontaneous defibrillation we propose an alternative explanation.

On the basis of our experimental studies, we have hypothesised that transient ventricular fibrillation requires the maintenance of a high sympathetic and low parasymathetic level during ventricular fibrillation. This hypothesis was based on results obtained in studies 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.4 Moreover, it has been shown that sustained ventricular fibrillation can be transformed into transient ventricular fibril-

lation either by pretreatment with various compounds that increase the cardiac catecholamine concentration during ventricular fibrillation or by intracoronary administra-

tion of adrenaline during ventricular fibrillation.5

Our hypothesis suggests that a high card-

iac catecholamine concentration improves intercellular coupling and intercellular syn-

chronisation, leading different myocardial cells to fibrillate coordinate.6 This sympa-

thetically induced synchronisation organises ventricular fibrillation and induces transformation 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 for a time. In a heart without structural heart disease, this increase in catecholamine concentra-

tion organises the configuration of the unsynchronised ventricular fibrillation into more organised "coarse" ventricular fibrilation, which in some cases terminates spontaneously.

The various problems involved in tran-

sient ventricular fibrillation were discussed
at a workshop on antiarrhythmic drugs and self ventricular defibrillation held in Tel-Aviv on 6–7 May 1993. The lectures presented at this workshop are now in print in the *Journal of Basic and Clinical Physiology and Pharmacology.*

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This letter was shown to the authors, who reply as follows:

Sin,—We thank Resin, Blayer, and Manoach for their interesting suggestion that raised cardiac catecholamine concentrations may have caused termination of ventricular fibrillation in our patient. In addition to an increase in sympathetic activity, we propose that termination could also have been caused by depolarisation of nerve endings in the ischaemic myocardium upon repolarisation. This phenomenon is the result of the early efflux of potassium into the extracellular space. Experimental models of denervated hearts or of hearts with depleted catecholamine stores could be used to test these interesting hypotheses.

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**Letters**

**Cost effectiveness of prophylaxis in dental practice to prevent infective endocarditis**

Sin,—We compliment Gould and Buckingham on their thorough review of the cost effectiveness of antibiotic prophylaxis for dental extraction in patients at risk of endocarditis.1 Their article gives the opposite view to that presented by van der Meer et al1 from the Netherlands. We recently surveyed patients attending cardiology outpatient departments at Groby Road Hospital in Leicester1 and found that patients with high-risk and low-risk cardiac lesions for endocarditis rarely attended a dentist. More worrying, most did not recall relevant advice (70% of low-risk and 86% of high-risk patients, see our table 1). The estimate by Gould and Buckingham that 50% of patients at high-risk are not provided with prophylaxis is likely to be an underestimate. Unlike our questioned naires 90% of general practitioners and dentists said they would give prophylaxis to patients at high risk (fig 5). However, only 14% of general practitioners could identify at risk patients on their register and half of general practitioners and dentists thought that they did not receive adequate advice from their cardiac centre.

We wholeheartedly support Gould and Buckingham’s conclusion that the use of prophylaxis in dental practice could be expanded by improved communication between doctors and dentists. Patients also need to be aware of the need to keep healthy teeth and gums and for regular dental check-ups. We have designed a simplified endocarditis risk card for patients and sticker for patients’ records and medical notes which should facilitate communication between patient, doctor, and dentist. It is clear from previous surveys and the current survey that advice to patients should be simple, clear, repeated and, most importantly, given in writing. Because most patients at risk attended only cardiac clinics, the onus is on cardiologists to improve the current situation.

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**BRITISH CARDIAC SOCIETY NEWSLETTER**

Council met on 14 October and confirmed the programme for the annual meeting in Torquay next year. The comprehensive and exciting programme is anticipated with plenary sessions on the ‘Management of acute myocardial infarction: short and long term considerations’ and ‘Intravenous cardiac catheterisation: oesophageal ultrasound: Where is it going?’

Council considered the continuing turbulence in the internal market and supported the policy of pressing for a sensible contract system that is flexible and sensitive to the special interests and casemix of individual providers. Clinical guidelines, to be distinguished from protocols, by the Joint Audit Committee with the Royal College of Physicians (see below). Council was keen to support progress towards a comprehensive set of guidelines covering all major specialties. It expressed concern that they will be an important part of redefining the contractual process and lead to a much greater understanding of our specialty.

It was encouraging to hear of the formation of the Nigerian Healthcare Foundation. The president, Professor T A Lambo, and vice-president, Dr K K Akinyoye, of the foundation visited London recently and met with John Parker to discuss the help and cooperation that the British cardiologists could offer Nigerian cardiologists. Further discussions on practical forms of help are continuing.

**British Paediatric Cardiac Association**

Babulal Sethia writes:—This year (1993) has been a busy one for the association. The summer meeting in conjunction with the British Cardiac Society Annual Meeting at Wembley was extremely well attended and provided an opportunity for cardiac physicians and surgeons on the relative merits of transcatheter closure of the arterial duct, the role of thorascopic surgery and management of the left AV valve and atrio-ventricular septal defect. The Harrogate meeting on 26 and 27 November is due to cover heart failure and the use of stents in congenital heart disease. In 1994 our meeting in association with the British Cardiac Society in Torquay is planned to encompass the discussion of long-term outcome in patients with congenital heart disease together with a specific discussion on the issue of pregnancy in patients with congenital heart defects.”

“The BPCA had been taking an active role in response to the Calman report. Although our final response has not yet been agreed, we are trying to coordinate the views of all our members in order that a unified approach may be taken in discussions, including the Royal Colleges, the SAC, and other interested parties.”

**Joint Audit Committee**

David de Bono writes:—"The Joint Audit Committee of the British Cardiac Society and the Royal College of Physicians of London was set up to facilitate and encourage all types of audit activity in relation to cardiology. Recently the committee sponsored an investigation into ‘Times to hospital admission and thrombolysis in acute myocardial infarction’ (Birkhead JS, et al. *BMJ* 1994;305:844) and ‘The use of cardiac catheter complications’ (de Bono D. *Br Heart J* 1993;70:297–300). Both these studies are ongoing. The committee is also organising a series of workshops, in collaboration with the Research Unit of the Royal College of Physicians, with the aim of defining and publishing management guidelines and audit standards for common cardiac conditions. Draft guidelines on stable angina were published in July (de Bono D, Hopkins A, J R Coll Physicians Lond 1993;27:267–72) and the edited workshop papers are currently in press. A workshop on the management of myocardial infarction was held in September and further