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, Schmoeckel M, Noliert G, Überfuhr P, 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, Laczcovics 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, Laczcovics 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 reinfarct 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 readministered. 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 preexisting 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 mammals[2] 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 studies[4–6] 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 studies[4] showing that transient ventricular fibrillation is a normal feature 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 fibrillation[6] 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

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 press in the *Journal of Basic and Clinical Physiology and Pharmacology.*

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1 van Hemel NM, Kingma JH. A patient in whom self terminating ventricular fibrillation was a manifestation of myocardial reperfusion. *Br Heart J* 1993;**69**:568–71.
2 Wiggers CJ. The mechanism and nature of ventricular fibrillation. *Am Heart J* 1940;**20**: 399–412.
3 Clayton R, Higham D, Murray A, Campell R. Self terminating ventricular fibrillation [abstr]. *Am Coll Cardiol* 1992;**19**:265.
4 Manoach M, Netz H, Erez M, Weinstock M. Ventricular self defibrillation in mammals: age and drug dependence. *Age Ageing* 1980;**9**:112–6.
5 Manoach M, Varon D, Neuman M, Netz H. Spontaneous termination and initiation of ventricular fibrillation as a function of heart size, age, autonomic autoregulation and drugs: a comparative study on different species of different age. *Heart Vessels* 1987;**2** (suppl):56–68.
6 Manoach M, Erez M, Wozner D, Varon D. Ventricular defibrillating properties of catecholamine uptake inhibitors. *Life Sci* 1992; **51**:P159–64.
7 Manoach M, Erez M, Varon D. Properties required for self-ventricular defibrillation: influence of age and drugs. Editorial review. *Cardiol Elderly* 1993;1:337–44.

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

SIR,—We thank Reisin, 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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Cost effectiveness of prophylaxis in dental practice to prevent infective endocarditis

SIR,—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 al*[2] from the Netherlands. We recently surveyed patients attending cardiac outpatients department at Groby Road Hospital in Leicester[3] 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 indeed likely to be an underestimate. In reply to our questionnaires 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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1 Gould IM, Buckingham JK. Cost effectiveness of prophylaxis in dental practice to prevent infective endocarditis. *Br Heart J* 1993;**70**: 79–83.
2 van der Meer JTM, van Wijk W, Vandenbroucke JP, Valkenburg HA, Michel MF. Efficacy of antibiotic prophylaxis for prevention of native-valve endocarditis: nationwide case-control study in the Netherlands. *Lancet* 1992;**239**:135–9.
3 Forbat LN, Skehan JD. Failure provision of antibiotic prophylaxis for at-risk patients. *Eur Heart J* 1993;**14**:812–8.

BRITISH CARDIAC SOCIETY NEWSLETTER

Council met on 14 October and confirmed the programme for the annual meeting in Torquay next year. A comprehensive and exciting programme is anticipated with plenary sessions on the "Management of acute myocardial infarction: short and long term considerations" and "Intravascular and 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, are being developed 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 areas of cardiology because it considered that they will be an important part of refining the contractual process and lead to a much greater understanding of our specialty by the purchasers.

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 Akinroye, 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 promoted lively discussion between physicians and surgeons on the relative merits of transcatheter closure of the arterial duct, the role of thoracoscopic surgery and management of the left AV valve and atrioventricular septal defect. The Harrogate meeting on 26 and 27 November 1993 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 involving 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* 1992;**305**:445–8) and 'An audit 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**:267–73) and the edited workshop papers are currently in press. A workshop on the management of myocardial infarction was held in September and further