sinus node or the blood supply to the sinus node. The interventional incision will not act as a substrate for atrial arrhythmias because there is no atrial tissue surrounding the incision around which a tachycardia circuit could propagate; the incision is anchored on either end by the nonconducting boundaries of the atriotomy and the right atrioventricular annulus. In fact, treatment of atrial arrhythmias by the Fontan revision surgeries with the right atrial maze procedures are designed create conduction block by similar but more extensive means.⁵

I agree that early postoperative sinus node dysfunction is associated with the development of late sinus node dysfunction and late atrial tachyarrhythmias. In our clinical trial, we reported a high percentage of early sinus node dysfunction in both the control and intervention groups, with no statistically significant difference between the two groups. Thus the sinus node dysfunction seen in this study was not attributable to the interventional atrial incision.

Our study reported only the short-term outcomes of our interventional atrial incision for the prevention of intra-atrial reentrant tachycardia in the lateral tunnel Fontan population. We have learned that, in contrast to previous clinical and animal studies, inducible perioperative atrial arrhythmias were not especially common clinically in these patients. Following up our cohort to assess long-term arrhythmia prevalence will provide a useful addition to our knowledge of the arrhythmia substrate in the Fontan population.

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Ventral cardiac denervation: Is it truly an effective prophylaxis against atrial fibrillation after coronary artery bypass grafting?

To the Editor:

We read with interest the article by Melo and colleagues¹ on ventral cardiac denervation (VCD) as a prophylaxis against atrial fibrillation (AF) after coronary revascularization. However, we wish to point out certain aspects of the methodology that may have affected the results. Post-CABG AF is paroxysmal in a large proportion of patients and does not always give rise to clinical symptoms. The fact that only 34%

and 33% of the patients in their VCD and control groups, respectively, had continuous telemetry monitoring for 4 days would have significantly reduced the AF detection rate, the primary end point of the study. Although statistically nonsignificant, the mixture of different myocardial preservation strategies used (crystalloid cardioplegia, blood cardioplegia, and off-pump) effectively reduced the advantage of the large number of patients in the study.

We conducted a study between January 2002 and April 2003 (Table 1) in which we compared the incidence of AF among 70 consecutive patients undergoing primary CABG with that among 70 consecutive patients who underwent VCD (with the same technique described by Melo and colleagues¹) in addition to primary CABG. All patients were operated by the same consultant surgeon with the same bypass method and myocardial preservation technique (antegrade-retrograde cold St Thomas' Hospital blood cardioplegia). All patients had continuous telemetry monitoring for 5 days after the operation. In con-

TABLE 1. Data and outcome after ventral cardiac denervation in patients undergoing CABG with or without VCD

Preoperative variables	VCD (n = 70)	No VCD (n = 70)	P value
Age (y, mean $\pm$ SD)	65 $\pm$ 10	65 $\pm$ 9	.7
Sex (M/F)	5:1	6:1	.6
Disease severity (%)			.8
Single vessel	1%	3%	
Double vessel	23%	22%	
Triple vessel	76%	75%	
Left ventricular function (%)			.5
>50%	76%	80%	
50%–30%	17%	16%	
< 30%	7%	4%	
Myocardial infarction (%)			.8
Never	69%	71%	
>30 d	28%	25%	
1–30 d	3%	4%	
Diabetes	16%	13%	.6
Hypertension (%)	65%	57%	.4
Chronic obstructive airway disease (%)	10%	11%	.8
Parsonnet score (mean $\pm$ SD)	7.1 $\pm$ 5.5	6.8 $\pm$ 6.5	.9
EuroSCORE (mean $\pm$ SD)	2.5 $\pm$ 1.7	2.3 $\pm$ 1.7	.6
β -Blockers (%)	69%	72%	.4
Crossclamp time (min, mean $\pm$ SD)	34.3 $\pm$ 7.5	33.4 $\pm$ 7.5	.6
Bypass time (min, mean $\pm$ SD)	57 $\pm$ 13.5	54 $\pm$ 12.2	.1
No. of grafts (mean $\pm$ SD)	2.8	2.9	.9
Postoperative inotropes (%)	13%	8%	.4
Reoperation (%)	1.4%	1.4%	>.999
Total blood loss (mL, mean $\pm$ SD)	603 $\pm$ 299	648 $\pm$ 412	.6
Length of stay (d, mean $\pm$ SD)	7 $\pm$ 0.8	7.5 $\pm$ 1.2	.8
AF (%)	29%	34%	.3

trast to the findings of Melo and colleagues,¹ and despite the younger age of our patients, we found no significant difference in the incidence of AF between the two well-matched groups in our study.

Melo and colleagues¹ failed to come up with a convincing reason for the claimed effectiveness of the technique. Anatomically, the route taken by autonomic nerves to the heart is highly variable²; a lesser but significant and variable proportion of autonomic nerves reaches the heart along the pulmonary veins and the back of the heart, so the actual denervation achieved in quantitative terms is not consistent or comparable between patients unless the innervation is mapped out before denervation. The inclusion of patients with diabetes (26% in the VCD group and 27% in the control group in their study and 16% and 13%, respectively, in our own study) and patients receiving β -blockers (75% in the VCD group and 81% in the control group in their study and 69% and 72%, respectively, in our study) gives rise to additional problems. Many patients with diabetes already have partial denervation from preexisting autonomic neuropathy, and it is debatable how much additional denervation is achieved surgically with the technique. β -Blockers themselves have antiarrhythmic effects and produce a state of near-total pharmacologic sympathetic denervation. Long-term β -blockade causes β -receptor hypersensitivity, necessitating reintroduction of β -blockers after the operation. The reintroduction of β -blockers in most cases in the postoperative period results in total cardiac sympathetic denervation and partial parasympathetic denervation, which raises questions about the whole hypothesis.

One can only meaningfully assess the effectiveness of the technique through a prospective, randomized multicenter trial excluding patients with diabetes and those receiving β -blockers. Histologic quantification of the amount and type of nerve fibrils in the excised fat pads is also necessary.

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Use of enoxaparin in cardiac surgery To the Editor:

We congratulate Dr Medalion and coworkers¹ for the results reported in their study recently published in the *Journal* that focused on the effects of enoxaparin in patients undergoing coronary operations. The optimal administration of low-molecular-weight heparin in cardiac surgery is a controversial issue.^{2,3} The use of enoxaparin in patients with prosthetic valves has also been recently described.^{4,5} Because we have routinely used enoxaparin both preoperatively and postoperatively in non-emergency cardiac patients at our institution during the last 5 years, we would like to outline some aspects of our experience.

When we started using enoxaparin in 1998, we had some concerns related to the potential bleeding hazards. Although we never measured anti-factor Xa activity, we did not experience any major bleeding complications that could primarily be related to the use of enoxaparin itself, and we gradually developed a simplified protocol

(Table 1), which is, however, significantly different from the one described. Doses of preoperative enoxaparin are much lower, and antiplatelet therapy is stopped on admission. On the other hand, a low dose of enoxaparin is given to all patients awaiting surgical intervention once daily (twice daily if the dose exceeds 4000 U/d). Also, patients who could potentially be stabilized with higher doses of enoxaparin and treated on an urgent basis, typically patients with coexistent left main disease and unstable angina, are instead treated as emergency cases and, as such, receive unfractionated heparin infusion until transfer to the operating room. Enoxaparin is also used after surgical intervention to minimize thromboembolic complications until a satisfactory international normalized ratio is attained in patients receiving oral anticoagulation and to prevent deep venous thrombosis.

In the study cited above,¹ although it is clear that patients classified as emergency cases receive heparin infusion, the authors do not specify when to administer subcutaneous enoxaparin versus intravenous heparin in urgent cases. The level of anticoagulation and the laboratory tests used (eg, partial thromboplastin time and activated clotting time) in patients receiving intravenous heparin are also not reported, and this might influence the measurements of anti-factor Xa activity at skin incision. Although results might be biased by a special accuracy in surgical hemostasis in patients enrolled in the study, bleeding rates were low in all subgroups despite high doses of enoxaparin (1 mg/kg twice daily),

TABLE 1. Use of enoxaparin before and after cardiac operations

Variable	Dose
Preoperative course	
Standard minimal dose	2000 U
Minimal dose in patients with atrial fibrillation or oral anticoagulation	4000 U
Maximal dose	2 × 4000 U
Conditions that modify preoperative doses	
Intravenous nitrates or left main stenosis >50%	2 × dose
Endocarditis or cardiac neoplasm	2 × dose
Body mass index >30	2 × dose
Body mass index <25	1/2 dose
Ticlopidine or clopidogrel withdrawal	1/2 dose
Hemoglobin <12 g/dL	1/2 dose
Postoperative course	
Standard dose	2000 U until mobilization
Oral anticoagulation or atrial fibrillation	4000 U until INR >2
Body mass index >30	4000 U until mobilization

One hundred units of enoxaparin = 1 mg.

INR, International normalized ratio.