Tachyarrythmias Following Coronary Artery Bypass Grafting

To the Editor:

Ducceschi et al *(Winter 1999)* [1] discuss the clinical predictors of in-hospital ventricular tachyarrythmias following coronary artery bypass grafting. It would have been interesting to know if any of the patients had ventricular arrhythmias pre-operatively. Although the differences were not significant, I note that nearly twice as many patients had ejection fraction of less than 30% in the VT group. Perhaps even more interesting was that less than half of the patients with VT were receiving beta-blockers when compared to those in the group which did not develop significant arrhythmia.

I think the authors should have defined VT (was it more than 30 seconds duration, above a certain rate, or requiring cardioversion?). The number of patients with arrhythmias was small and I presume this is why VT and VF were combined. I would like to have known how many patients developed VT which deteriorated to VF and how many had primary VF.

It is interesting that the VT patients were younger as I would have expected the older age group to be more susceptible.

Having identified the independent correlates for VT by multi-variate analysis, it is interesting to speculate as to whether these patients should be treated prior to the onset of VT/VF at least with beta-blockers or possibly with amiodarone.

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Anomalous Left Coronary Artery From the Pulmonary Artery

The study on Trends in Anomalous Left Coronary Artery from Pulmonary Artery by Wolleneck G. published in the recent issue of *Heart Views* (1) is appreciated. The modalities and current trends in management and the importance of aggressive surgical strategy, critical to the outcome, are highlighted. Additional and interesting aspects of this condition were published in 1997 by Schwartz et al (2). In their surgical review of 42 patients over 18 years, a number of factors were analyzed to predict mortality. Twenty-seven of their patients had preoperative mitral regurgitation (16 mild, 8 moderate, 3 severe). Fourteen patients underwent direct re-implantation of the left coronary artery to the aorta, Twenty-six patients underwent an intrapulmonary baffling technique while two patients underwent extrapulmonary baffling technique. Their significant finding was: ONLY severe mitral regurgitation was associated with increased mortality; NOT age, body surface area, preoperative shortening fraction or LV end diastolic dimension. The severity of LV dysfunction did not predict mortality or recovery of LV function. This is in contrast with other studies where severity of LV dysfunction correlated with the mortality (3,4). In addition, the authors noted the frequent occurrence of postoperative supravalvar pulmonary stenosis as a complication with intrapulmonary baffle (76%) and with reimplantation (22%), though not so with extrapulmonary baffle. As reported by Wolleneck G, the authors of this study also conclude that the appropriate operative management of patients with moderate to severe MR is "not clear".

Another study from the same center in 1999 showed significant benefit with the additional use of LVAD in children with severe LV dysfunction (5). Over a one-year period thirty-one patients underwent dual coronary repair of ALCAPA. Seven of these children who could not be weaned from the cardiopulmonary bypass were placed on mechanical LVAD support . Five of these patients survived with significant improvement in function and 2 required late MV repair. LVAD support was required for a median of 43.5 hours. Significant complications from the use of LVAD were reported during the procedure. There were 2 deaths out of the seven patients. Use of LVAD as an adjunctive modality may significantly improve the survival rates in a small group of patients with difficulty in weaning from the bypass.

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Heart Rate Variability following Myocardial Infarction

I was very interested to read the paper by Di Micco and colleagues (Summer 2000) on the loss of heart rate variability following myocardial infarction and its correlation with arrhythmic events and mortality (1). Diabetic autonomic neuropathy is a well-documented cause of loss of heart rate variability (2-4) it is often asymptomatic, and evidence for its existence has to be sought by measuring the influence of breathing and posture on the heart rate (5). Unexplained sudden death has been reported in patients with diabetic autonomic neuropathy and it has been suggested that arrhythmias may have been responsible. The occurrence of myocardial infarction in diabetic patients with autonomic neuropathy brings together two potent causes of loss of heart rate variability and thus increasing the risk of malignant ventricular arrhythmias and death.

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