Letters to the Editor must not exceed 400 words in length and may be subject to editing or abridgment. Letters must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Only some letters will be published. Authors of those selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication.

Useful Understanding of Postoperative Atrial Fibrillation

To the Editor:

Hogue et al¹ concluded: “In the hour before AF [atrial fibrillation] after CABG surgery, higher heart rate and lower heart rate complexity compared with values in control patients were independent predictors of AF.” Their careful study and stochastic analyses showed that this was statistically significant, but their observations were not clinically significant for the following reasons.

After CABG operations, many patients have AF for a few minutes or hours without a fast ventricular rate or emboli. Even new AF that lasts for a day or more usually disappears spontaneously without bad results. In a series of 100 consecutive patients who had cardiac operations, all patients who entered the hospital in regular sinus rhythm left the hospital in regular sinus rhythm in spite of intervening AF.² Predicting harmless as well as harmful AF as a single set does not influence treatment or help the patients in other ways.

Concerning patients who acquire AF after acute myocardial infarction (MI), the American College of Cardiology/American Heart Association stated, “Although AF after acute MI is usually transient, heparin therapy should be given to patients not already receiving it.” Hogue et al³ reported 1 patient who had AF and then appeared to have a fatal cerebral embolism on postoperative day 2. There was time to give this patient heparin, and the patient then appeared to have a fatal cerebral embolism on postoperative day 2. There was time to give this patient heparin, and the patient probably received it. Predicting that the patient would have AF did not help have AF.

Predicting which patients will have persistent hazardous AF is a different matter. Such a prediction would justify vigorous treatments in these few patients to prevent AF. Such treatment would not be reasonable for all patients at risk for this arrhythmia after CABG operations.

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Misdiagnosis of New Bifascicular Block

To the Editor:

I enjoyed the contribution of Kaji et al⁴ in the “Images in Cardiovascular Medicine” section of the journal. I have found an error, and because I believe that this part of Circulation is very popular and is read by a large number of physicians, it would be an opportunity to call attention to a frequently committed misdiagnosis. I am referring to the characterization of the ECG made by the authors as showing “a new bifascicular block.” Apparently the authors made this diagnosis on the basis of the widened QRS complex in lead V₃ (which they interpreted as a right bundle-branch block) and the leftward shift of the frontal QRS axis well depicted in leads aVL and aVF (which they attributed to left anterior hemiblock).

However, the ECG features noted in their case and the precipitating clinical circumstances are typical of the “giant R waves syndrome” noted in the hyperacute phase of transmural ischemic injury.²⁻⁶ ECG patterns identical to the one depicted in the case under discussion are seen in the setting of the very early phase of transmural myocardial infarction and coronary vaso-spasms (Prinzmetal’s angina) and immediately after acute coronary occlusion in the animal laboratory.²⁻⁶

The diagnostic particulars of the ECG include an increase in the R amplitude of waves and the decrease or disappearance of S waves (vide leads aVL, V₂, and V₃ of the authors case), ST-segment elevation merging with the R waves (V₃), and widening of the QRS complex (αVL, 3, αVF, V₃), often indistinguishable from ventricular tachycardia, in which the P waves are obscured and the rate is fast.

The mechanism of this ECG syndrome has nothing to do with a compromise of conduction at 1 or more sites of the conduction system and is believed to constitute a “focal” block or delay in conduction in an area of severe transmural ischemic injury. In such a territory, late slow conduction produces delayed enhanced (due to lack of opposition) depolarization vectors, pointing toward the ECG leads reflecting the particular region involved (vide, positive potentials in aVL, V₂, and V₃ and negative in 3 and aVF in this case).¹⁻⁵

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Human Basic Fibroblast Growth Factor Induces Angiogenesis in Hen Eggs and Rat Hearts

To the Editor:

In the February 24, 1998, issue of Circulation, Schumacher et al⁷ report on their work concerning the production of human basic fibroblast growth factor (FGF-1), 2 series of animal experiments using FGF-1, and the clinical use of the growth factor in patients undergoing CABG surgery. They have to be congratulated for their persistent effort of genetically producing, experimentally testing, and clinically applying an angiogenic

1250
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1251

growth factor in patients with coronary artery disease (CHD) for the first time. The authors themselves say at one point in the discussion that the possibility of inducing angiogenesis in the human heart “has been widely discussed for many years but never before attempted.”

There is complete agreement with Schumacher and coworkers that they were the first to attempt human myocardial neangiogenesis. At the same time, the clinical data presented of the 20 patients treated with FGF-1 and of the 20 individuals subjected to inactivated FGF-1 are not at all convincing that the authors “established for the first time the efficacy of FGF-1 for the treatment of CHD, and were able to demonstrate that it can induce neangiogenesis...” The only thing the authors are able to demonstrate persuasively is that the area surrounding the bypassed left anterior descending coronary artery in their Figure 6A (digital subtraction angiography) is grayer than that in Figure 6B. To claim on the basis of this example that “the formation of capillaries could also be demonstrated in humans” is severely exaggerated, because the reader does not know anything about the timing of acquisition of those images with respect to the contrast injection. A difference of 250 ms (frequency of 4 images per second) between the image acquisition for Figure 6A and B and not the different treatment modalities could be the reason for the variable gray shades. The not completely intelligible sentence in the Results section that the angiograms of both treated and control groups “show comparable distances between the beginning of the injection and visualization of the medium” further raises the question of exactly where the contrast dye was injected and how much of it was injected in the 2 study groups. To state (in the Abstract) on the basis of Figure 7 that “[a] capillary network sprouting from the proximal part of the coronary artery could be shown to have bypassed the stenoses and rejoined the distal parts of the vessel” in response to FGF-1 treatment is equally speculative, because probably not even the authors know whether collaterals, if present, weren’t already there before bypass surgery and FGF-1 therapy.

To be able to assess the efficacy of the potentially very important new treatment modality of growth factors for ischemic heart disease, precise functional measurements of collateral flow have to be used.2 This is not only important because numerous growth factors can potentially be judged regarding their effect on collateral capillary vessels (angiogenesis) or larger, functionally more important conductance collaterals (arteriogenesis),3 it is also relevant because the effect of a collateral-promoting, angiogenic substance has to be characterized in light of its atherogenic response to stress.

As a minor point, it has to be mentioned that the study by Yanagisawa-Miwa was performed in dogs and not in rabbits.

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**Worried to Death?**

*To the Editor:*

We were interested to read the report of the 70-year-old woman who developed ST-segment changes, elevated cardiac enzymes, and ventricular akinesis on hearing of her husband’s death.1 Bereavement or other significant life stressors have been associated with life-threatening arrhythmias and increased mortality. Although clinical laboratories by studies have demonstrated that acute psychological stress produces ischemia in patients with coronary artery disease,2 rarely has an acute ischemic response to real-life psychological stress been clinically documented in an otherwise healthy individual.

Prospective studies, however, have demonstrated that otherwise healthy individuals with chronically elevated anxiety are at greater risk of sudden cardiac death (SCD).3 Anxiety increases the risk of SCD, with even low-to-moderate levels of anxiety being sufficient to produce some elevation in risk.4 Recently, we reported a dose-dependent relationship between anxiety level and impaired vagal reflex control of heart rate in young healthy volunteers,4 suggesting that low vagal cardiac control may mediate the relationship between anxiety and SCD. Reduced vagal cardiac control, as measured by low heart rate variability, clearly predicts cardiac risk. In addition, low levels of vagal control of heart rate have been found in survivors of SCD with no evidence of coronary disease.5

Acute increases in stress may evoke acute myocardial ischemia, life-threatening arrhythmias, and death in some individuals. Understanding the mechanisms by which stress injures these events may allow us to identify individuals who are susceptible to stress-induced cardiac responses and may ultimately help us develop effective preventive strategies. Although the significance of the sympathetic nervous system in the production of stress-induced arrhythmias is well known, recently vagal cardiac control has also been shown to be important in the prediction of fatal arrhythmias. The data from our laboratory and others suggest that individuals with low vagal control may represent a group with increased susceptibility to stress.

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**Response**

We agree that a demonstration of a relationship between psychological stress (in particular, grief) and a transient compromise of coronary perfusion is rare, which is why we were pleased to present our case to the physician public. The mechanism by
which a stressor such as intense bereavement leads to a severe reduction of coronary blood flow is not clear and may be multifactorial. Clearly, autonomic imbalance, as pointed out by Watkins and Blumenthal, is of paramount importance. A sympathetic discharge, evident in this case during our patient’s acute emotional response, can cause marked changes in coronary vascular tone and rheology by effects on such diverse factors as coagulability, platelet adhesion, and endothelial function.1,2 Patients with low vagal tone may be particularly susceptible to these phenomena, because we know they are particularly susceptible to the electrical instability that is the substrate for sudden cardiac death.3 Whether our patient was so disposed is unknowable. We did not discern any signals of dysautonomia in her case, such as ventricular arrhythmia, or marked alterations in heart rate, and her blood pressure remained steadily normal after her acute trauma. We did not perform any formal measurements of autonomic tone, nor did we assess her vascular response to autonomic manipulation.

We sincerely appreciate the comments made by Drs Watkins and Blumenthal. They underscore the need for a deeper understanding of the interaction between grief reactions and major cardiac events, a phenomenon that needs to be expected and recognized by all clinicians.4,5

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Transcatheter Alcohol Septal Ablation for Hypertrophic Obstructive Cardiomyopathy

To the Editor:

I read with interest the report by Lakkis et al1 on transcatheter alcohol septal ablation for patients with hypertrophic obstructive cardiomyopathy (HOCM). However, 11 (33%) of their 33 patients developed complete heart block and required permanent pacemaker implantation. This high rate, which has not changed significantly from the rate of 40% previously reported by the same authors in 1997 on a smaller series of patients,2 represents an important complication of this novel procedure.

Furthermore, it should be noted that pacing in HOCM itself carried a high complication rate. In 1 series of 83 patients, infection occurred in 10%, electrode displacement in 12%, and death in 1% due to right ventricular perforation.3

Therefore, it is time for a controlled trial to be performed before this experimental procedure becomes an accepted clinical treatment. Or at least, a prospective registry should be set up to assess the frequency of these complications.4 Braunwald5 proposed the National Heart, Lung, and Blood Institute as such an organization.

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Response

We thank Dr Cheng for his letter. We, too, were concerned about the high incidence of complete heart block early in our series. We have reviewed the last 50 consecutive patients who presented without a pacemaker, and only 3 (6%) required permanent pacing after the procedure. This incidence of complete heart block compares favorably with that of myomectomy surgery. We believe that the lower incidence of complete heart block is due to modifications of our technique, including the use of myocardial contrast echocardiography.

We completely agree that the time has come for a prospective registry to assess the frequency of complications and the efficacy of the procedure on a national basis. We have approached the National Heart, Blood, and Lung Institute for sponsorship.

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