spectral measures of heart rate variability and turbulence slope after APCs) revealed any significant change before the onset of spontaneous atrial fibrillation episode.

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REPLY

We are very pleased to get an intellectual input from Wichterle and Malik related to the mechanisms of heart rate (HR) turbulence after atrial premature beats (APBs). The major observation of our study (1) was that "paradoxical" increase of R-R intervals after APBs, resulting in a positive turbulence onset (TO), precedes the spontaneous onset of atrial fibrillation (AF) episodes. Our interpretation was that enhanced vagal responses to APBs might be the major factor behind this phenomenon.

Wichterle and Malik propose another potential mechanism, discussed also in our report, that resetting of sinus node activity after APBs might be the possible mechanism behind the paradoxical TO. The resetting phenomenon might then reflect the change in the origin or prematurity of APBs in the vicinity of AF episodes. For example, the APBs originating from the pulmonary veins might result in a different resetting of the sinus node.

As commented on by Wichterle and Malik, the TO after APBs has actually no relationship with other markers of autonomic tone measured from 24-h electrocardiograph recordings. This observation is consistent with our previous study (2). However, it should be noted that tonic autonomic regulation may be completely different from reflex regulation in response to acute hemodynamic fluctuation. Therefore, the lack of this correlation does not exclude the potential contribution of vagal reflexes in response to APBs, and it is evident that we do not seem to have enough data at the moment to precisely define the mechanisms of HR behavior after APBs. New study designs are needed to clarify this issue—for example, studies where APBs are delivered from various sites of atria, including pulmonary veins, with and without autonomic blockade.

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Genetic Testing in Clinics for Congenital Heart Disease in Adults

We read with interest the report by Beauchesne et al. (1) regarding 22q11.2 microdeletion in adults with selected conotruncal abnormalities. We have pursued a policy of screening for 22q11.2 microdeletion in such patients who are seen in a dedicated clinic for congenital heart disease in adults. This, however, is not a tertiary center, but a large district United Kingdom general hospital. As such, the numbers are smaller and have been performed as the patients come through clinic. To date, 8 of 16 patients with pulmonary atresia ventricular septal defect have been tested and 3 patients have 22q11.2 microdeletion (37.5%). One of these is of Chinese ethnic origin but probably has dysmorphic features. He had a sister who died with truncus arteriosus. The second patient has clear dysmorphic features with mental retardation. The third does not have dysmorphic features but is mentally retarded. Thirty-two of 55 patients with tetralogy of Fallot have been screened. None have 22q11.2 microdeletion, but one with very dysmorphic features has deletion of the long arm of C11 and is thought to have Jacobsen syndrome, which has been associated with endocardial cushion defects and coarctation of the aorta (2); we have not found this or 22q11.2 microdeletion in 4 of 33 patients with endocardial cushion defects or 20 of 88 coarctation patients tested so far. We agree with the investigators that screening of such high-risk patients is mandatory—especially given the implications for 50% transmission. As the genetic basis for congenital heart disease becomes clearer, more assiduous attention is likely to be needed, especially for those involved in maternal medicine.

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REPLY

We appreciate the interest of Dr. Freeman and her colleagues in our recent publication on 22q11.2 microdeletion in adults (1). The data they present are in keeping with the literature that indicates certain conotruncal anomalies, such as pulmonary atresia/VSD, are frequently associated with 22q11.2 microdeletion. Although the patients who were positive for 22q11.2 microdeletion in their center had "classic" features of the syndrome (two-thirds had dysmorphic features and two-thirds significant developmental delay), in our prospective cohort a significant proportion did not have these findings. We would also like to remind the readership that at the present time our position on screening adults is that it should be individualized as opposed to mandatory. We believe screening should be *considered* in patients with "high-risk" cardiac lesions, or if there is the presence of specific clinical features that are associated with 22q11.2 microdeletion as listed in our report (1). The pros and cons of screening should be discussed with each patient so that a decision, based on informed consent, can be made (2).

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CORRECTION

Costa RA, Mintz GS, Carlier SG, Lansky AJ, Moussa I, Fujii K, Takebayashi H, Yasuda T, Costa JR Jr., Tsuchiya Y, Jensen LO, Cristea E, Mehran R, Dangas GD, Iyer S, Collins M, Kreps EM, Colombo A, Stone GW, Leon MB, Moses JW. Bifurcation Coronary Lesions Treated With the "Crush" Technique: An Intravascular Ultrasound Analysis. J Am Coll Cardiol 2005;46:599–605.

In the opening paragraph of the article, the second sentence should read: "Regardless of the technique, restenosis rates after bare metal stenting were high (40% to 60%), especially at the ostium of the side branch (SB) (1–3) where lesions frequently present with negative remodeling before PCI and suboptimal angiographic results after PCI (1,4)."

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