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LETTERS TO THE EDITOR

Prognosis in Cardiac Tumors in Infants and Children

The result of the management of the seven cases of cardiac tumors reported by Bini et al. (1) was quite discouraging as only one child survived 3 months. In a recent review of cardiac tumors reported in the English language between 1972 and 1977 (2), I found 47 reported pediatric cases with a survival rate of more than 6 months in 55% of the cases. Forty-four of these tumors were benign with a survival rate of 57%. The survival rate increased significantly if the diagnosis of cardiac tumor was made preoperatively. The reason the survival rate was so low in the series of Bini et al. is probably that their cases were unusually difficult, perhaps because they report from a sophisticated referral center where only the worst cases may be referred. In the seven cases, six tumors were benign, and only one of the patients survived 3 months. Four of the tumors were rhabdomyomas, which appear to have been unusually severe, because all four were associated with tuberous sclerosis. Of the 23 reported rhabdomyoma cases between 1972 and 1977, tuberous sclerosis was present in only approximately 50%. In a less selective case series than that presented from Birmingham, Alabama, the prognosis for infants and children with cardiac tumors appears to be better than that reflected in the article by Bini et al.

In the same issue of JACC, Martin and Boak (3) claim that the diagnosis of cardiac tumors is usually made postmortem. In our review of 263 cases of cardiac tumor reported in 1972 to 1977, 205 (78%) had a preoperative diagnosis that greatly improved the prognosis.

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Ventricular Response to Atrial Fibrillation

With his editorial, Meijler (1) has reopened a controversy that has lain dormant for over 10 years. His studies of the ventricular rhythm in atrial fibrillation using autocorrellation as the crucial measurement have not demonstrated any "patterning" or nonrandom sequences of beat to beat intervals. Therefore, he asserts that the ventricular rhythm in response to atrial fibrillation is "random," and that certain drugs such as digitalis and quinidine do not affect this property of the ventricular rhythm in atrial fibrillation.

However, studies by several other techniques that are independent of each other demonstrate that the ventricular response to atrial fibrillation in human beings is not random, and instead is patterned in various ways (2–5). The simplest of these consists of casting histograms of ventricular beat to beat intervals that occur during inspiration, expiration and apnea in the presence of quiet, normal respiration and with Cheyne-Stokes respiration. The histograms of ventricular beat to beat intervals during apnea are markedly different from those during expiration, and slightly different from those inscribed during inspiration. Because respiration is a rhythmic process, it is clear that the ventricular response to atrial fibrillation changes in a rhythmic fashion and that the autocorrelogram is simply not sufficiently sensitive to record this rhythmicity.

More important is the fact that many more "patterned" sequences of ventricular beat to beat intervals occur in the majority of patients with atrial fibrillation than would be expected by chance. Our group studied only three types of many possible sequence patterns. These were sequences of equal intervals (intervals that varied no more than 32.5 ms from the first interval of the sequence), sequences of intervals that diminished in duration as expected in Mobitz type I second degree block and sequences of intervals that were multiples of a single common denominator, as expected in varying second degree block (3:2, 3:1, 4:2, etc.). These sequences were located (by computer) in recordings including 7,000 to 15,000 beats (per recording) and classified by the number of beats each sequence contained and by the interval length of their membership. We then calculated from the overall beat to beat interval histogram how many sequences of each type, membership size and interval length would be expected by chance alone in the recording at hand, and compared the number actually found with the number expected. Only calculations based on more than 1,000 intervals with probability (p) values less than 0.001 were called "sequences." Using these rigorous definitions, sequences of patterned intervals were found in most of our patients (6).

In some instances this was so because in the time that it takes to record 7,500 to 15,000 heartbeats, patients with atrial fibrillation may develop paroxysms of atrial flutter, ventricular tachycardia, junctional tachycardia or escape rhythm. However, these nonfibrillatory mechanisms were not the cause of most of the sequences we found. To ascertain this fact, all the sequences were located on the magnetic tape and the scalar electrocardiogram and tachogram reconstituted and then visually inspected. It is clear from these studies and also those by others that the ventricular response in atrial fibrillation is not random. It has also been found that quinidine, digitalis glycosides and other drugs significantly affect the nonrandom character of the ventricular response in atrial fibrillation in human beings.

I agree with Meijler that the mechanism whereby the rhythm in atrial fibrillation is established is still unknown. A detailed discussion of the various possibilities has been presented elsewhere (7). However, there can be no doubt that the integrative properties

of the atrioventricular node are not explained by its state of refractoriness or by concealed conduction alone.

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Reply

I agree that there is a problem—a word I prefer to "controversy"— in respect to different interpretation of the outcome of the analysis of the ventricular response to atrial fibrillation. This is manifest in what Urbach wrote in 1973 (his Ref. 6):

"The pattern of the ventricular response to uncomplicated atrial fibrillation depends primarily on the average pulse rate. Most patients with chronic atrial fibrillation and a ventricular response of 70 to 90 beats per minute have a very irregular rhythm. Their shortest ventricular beat to beat intervals measure 0.4 to 0.7 seconds, their longest 1.3 to 2.3 seconds. There are very few successive intervals that are equal in duration, and there are even fewer that are otherwise patterned." Study of the histogram produces data on variations in RR interval duration, not on RR interval sequence.

Much remains to be learned about the ventricular response in atrial fibrillation. My observations to which Urbach has responded are a modest attempt to clarify the problem.

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ST Segment Changes in Lead aVR

Billadello et al. (1) do not mention ST changes in leads other than the inferior and precordial ones. It would be interesting to know whether there was ST segment elevation or depression in lead aVR. This lead integrates the electrical activity of the different parts of the left ventricle in a more balanced way. An ST segment elevation in lead aVR, in the presence of an acute inferior wall myocardial infarction (with ST elevation), should be highly specific for anterior wall ischemia.

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1. Billadello JJ, Smith JL, Ludbrook PA, et al. Implications of "reciprocal" ST segment depression associated with acute myocardial infarction identified by positron tomography. J Am Coll Cardiol 1983;2:616–24.

Reply

We have reviewed the qualifying electrocardiograms from our series of 20 patients and have noted ST segment elevation or depression 0.1 mV or more in lead aVR in 4 patients. Three of nine patients with inferior myocardial infarction with "reciprocal" ST segment depression in the anterior precordial leads demonstrated ST segment depression in lead aVR, and one of these nine patients showed ST segment elevation in lead aVR. The patient with ST elevation in lead aVR and one of three patients with ST depression in lead aVR demonstrated decreased apparent accumulation of ^{11}C-palmitate in the anterior left ventricular wall documented tomographically, possibly reflecting anterior left ventricular ischemia. Thus, our data indicate that the presence of ST segment deviation in lead aVR in the presence of acute inferior wall myocardial infarction is neither a sensitive nor a specific indicator of anterior wall ischemia in our population.

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QRS Scoring Systems for Estimating Ventricular Function

Young et al. (1) considered the correlation of information obtained from the electrocardiogram with that obtained from radionuclide angiography in patients with ischemic heart disease. Previous studies from our institution (2) and those of others (3–5) have evaluated the performance of the QRS scoring system developed by Selvester and coworkers and simplified and validated by our group. In patients after an acute myocardial infarction, the correlations with left ventricular ejection fraction ranged from -0.61 to -0.88. A correlation of 0.72 was also reported when comparing the QRS score with infarct size estimation by creatine kinase (CK)-