Vectorcardiographic Observations of Postextrasystolic T Wave Changes Associated with Interpolated Ventricular Beats*

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Vectorcardiographic observations in a patient exhibiting postextrasystolic T wave changes associated with interpolated extrasystoles were undertaken. The T wave changes observed were found to be “primary” and not secondary to aberrant ventricular depolarization. It is suggested that hemodynamic factors may play a role in the genesis of these T wave changes.

A recent study of the postextrasystolic T wave changes, associated with interpolated premature ventricular beats, revealed that this phenomenon is not rare and, unlike the T wave changes which occasionally occur in the first sinus beat following the more common premature ventricular beats with compensatory pauses, are not usually related to the presence of significant heart disease.† Despite the establishment of the clinical significance of this phenomenon, the cause has remained obscure. One theory was that of Scherf2 who proposed that the postextrasystolic T wave changes seen after interpolated extrasystoles are secondary to aberrancy in ventricular depolarization. Although several electrocardiograms of the patients described in a previous study demonstrated slight aberrancy in the QRS complex of the postextrasystolic beat, many of them did not, and in those that did, the T wave changes were in excess of what might be expected from secondary T wave changes of QRS aberrancy.† An opportunity to further evaluate this aspect of the problem arose when a patient was observed to be having occasional interpolated extrasystoles with postextrasystolic T wave changes during a routine clinic visit.

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MATERIAL AND METHODS

The vectorcardiograms were recorded with the Electronics for Medicine Simultrace recorder, model DR-8.* The lead system employed was that of Frank.3 Electrocardiograms were recorded by standard means employing a paper speed of 25 mm per second.

The patient was a 61-year-old man with a known past history of myocardial infarction. He received a routine complete cardiology evaluation. He had been asymptomatic for several years and was feeling well at the time of the examination.

RESULTS

Figure 1 reveals a representative complex from each lead of a standard 12 lead electrocardiogram, in addition to inspiratory leads III-2 and AVF-2. The latter two leads have been shown to be of value in the elicitation and evaluation of arrhythmias.4 Figure 2, line A, reveals the vectorcardiographic findings of the regular sinus beats occurring in this patient. Line B reveals vectorcardiograms of first sinus beats following interpolated premature ventricular systoles.

As can be seen in Figure 1, leads III-2 and AVF-2 reveal interpolated premature ventricular systoles followed by regular sinus beats with T wave changes not seen in the other beats. Analysis of the vectorcardiograms in Figure 2 reveals no difference in the “QRS” loops of the pre-extrasystolic beats (line A) when compared with the postextrasystolic “QRS” loops (line B). However, significant differences in the “T” loops are easily recognized.

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FIGURE 1. Representative complexes from each lead of a standard 12 lead electrocardiogram along with inspiratory leads III-2 and AVF-2. Note the interpolated extrasystoles in leads III-2 and AVF-2 with the postextrasystolic T wave changes.

FIGURE 2. Line A reveals the vectorcardiographic findings of the regular sinus beats occurring in the patient reported. Line B reveals vectorcardiograms of first sinus beats following interpolated premature ventricular systoles. Note that no difference is seen in the "QRS" loops of the pre-extrasystolic beats (Line A) when compared with the postextrasystolic "QRS" loops (Line B). However significant differences in the "T" loops are easily recognized.
DISCUSSION

The above findings would seem to be strong evidence in favor of the concept that the T wave changes seen in the postextrasystolic beats associated with interpolated premature ventricular beats are "primary" and not secondary to altered depolarization of the ventricles. Thus, the present study does not corroborate the theory that these T wave changes are secondary to aberrancy in ventricular depolarization; at least in the patient studied.

The cause of this altered repolarization remains unknown. Hemodynamic factors, however, are known to influence repolarization and may be important in the situation under question. This possibility seems plausible when one considers that the T wave is recorded during mechanical systole of the ventricles, as opposed to the QRS which occurs just prior to mechanical systole. Thus, the electrophysiologic events associated with the T wave may well be influenced by the mechanical forces and an increased gradient of pressure across the ventricular wall. In fact this is the most widely accepted explanation for the fact that the normal electrocardiogram records positive deflections for both QRS and T waves as opposed to the normal isolated muscle cell which lacks a gradient and records a repolarization wave or "T wave" opposite in direction to the depolarization wave or "QRS". In the normal heart, depolarization progresses from endocardium to epicardium; whereas the repolarization forces start at the epicardium and end at the endocardium. Therefore, any hemodynamic factor which alters the pressure gradient might be expected to alter the T wave. The postextrasystolic T wave changes often seen associated with ventricular extrasystoles and compensatory pauses (non-interpolated) have usually been attributed to prolonged ventricular filling during the compensatory pause with the mechanical effect of overdistention of the ventricles. These T wave changes are often associated with the presence of significant heart disease.2,6,8 The postextrasystolic T wave changes seen with interpolated premature ventricular beats, on the other hand, have not been shown to be statistically related to heart disease.1 However, this does not exclude the possibility of these T wave changes occurring in patients with established heart disease, such as the case studied in this report.

With both types of extrasystoles existing on the same electrocardiographic strip, it has been observed that T wave changes can exist following interpolated extrasystoles while not occurring after extrasystoles with compensatory pauses. It has been suggested, therefore, that different mechanisms are responsible for the genesis of these T wave changes.1 It is of some interest that the shorter diastolic interval following the interpolated type of extrasystole is associated with diminished diastolic filling and presumably a lesser pressure gradient from endocardium to epicardium. One might speculate that the diminished pressure gradient associated with the sinus beat following an interpolated premature extrasystole might simply "allow" the T wave to return partially to its hypothetical "normal position," i.e. opposite in direction to the QRS.

REFERENCES


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