Acute Myocardial Infarction Revealed by Interpolated Premature Ventricular Contractions*

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A 68-YEAR-OLD MAN WAS HOSPITALIZED following the onset of crushing precordial chest pain.

Admission electrocardiogram (Fig. 1)

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**Figure 1:** Admission electrocardiogram shows small Q waves in leads II, III, and AVF (residual of previous inferior myocardial infarction), and ST segment depression compatible with digitalis effect or subendocardial ischemia.

**Figure 2:** Lead V3 continuous, demonstrates interpolated premature ventricular contractions (arrows) displaying the classic changes of an acute anterior wall myocardial infarction. The ST-T segment elevation represents a reversal of the usual secondary abnormalities seen with ventricular ectopic beats.
The following day an arrhythmia occurred. A repeat electrocardiogram (Fig. 2) revealed frequent unifocal interpolated ventricular extrasystoles. Although the pause following these prematurities was less than compensatory, ventricular ectopia was indicated by: (1) anomalous diphasic QRS complexes, without preceding P waves; (2) constant interval between the anomalous and sinus beats (fixed coupling); and (3) initial vector of the anomalous complexes dissimilar from that of flanking conducted sinus impulses. The configuration of these premature contractions suggested origin from an infarcted area.

Serial electrocardiograms were compatible with an acute anterior myocardial infarction (Fig. 3). A more extensive lesion than was evident in the sinus beats was implied by the early occurrence of interpolated premature ventricular contractions exhibiting the classic infarction pattern in several precordial leads. This corroborated the wide area of necrosis suggested by maximum SGOT level of 195 units.

Experimentally, ligation of the anterior descending branch of the left coronary artery engenders excitation of the infarcted area with production of premature ventricular contractions having an infarct morphology. Clinically, the electrocardiographic configuration of such ectopic ventricular mechanisms may suggest acute or remote myocardial infarction when it is not evident in the conducted supraventricular leads or is masked by a conduction defect. As in the present case, morphologic analysis of premature ventricular contractions may also be of supplemental value in determining the location of infarction and extent of necrosis.

The infarction premature ventricular contraction occasionally occurs in apparently normal subjects. Accordingly, this electrophysiologic clue must be correlated with other clinical findings in adjudging its diagnostic significance.

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