Contour of Electrically Induced Premature Ventricular Beats Before and After Experimental Myocardial Infarction in Dog

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Ventricular premature beats represent the commonest arrhythmias in man. Such beats are often present, both in heart disease and in the normal heart, although they occur more frequently in persons with diseased hearts. Because the anatomical site of origin and the direction of spread of premature ventricular systoles cannot be determined on clinical grounds, it is generally agreed that such complexes have no electrocardiographic characteristic findings that point to the presence or absence of underlying cardiac disease. Nevertheless, isolated case reports have suggested that premature ventricular systoles may have final ventricular deflections characteristic of acute myocardial infarction. Recent clinical analyses also suggested that bizarre forms of depolarization and of repolarization of premature ventricular systoles are almost exclusively found in persons who have underlying myocardial disease.

This study attempts to obtain experimental evidence for this concept by determining if premature ventricular complexes are characteristically deformed by acute experimental cardiac injury.

Methods

Thirteen dogs, weighing 9 to 19 Kg., were used. The dogs were anesthetized with intravenous pentobarbital (30 to 35 mg./Kg.). Respiration was supported after tracheostomy with a respirator using room air. The chest was opened by a midline sternum-splitting incision. The pericardium was incised and its edges sewn loosely to the lateral thoracic walls. Every effort was made to change the position of the heart as little as possible. Barbed platinum-tipped stimulating electrodes were inserted into the anterolateral right ventricular wall and into the midlateral left ventricular wall. The exact position of these electrodes varied from dog to dog. In three dogs, the electrodes broke or fell out. Electrodes were reinserted within 5 mm. of the previous site of puncture. In dogs 8, 9, and 10, additional electrodes were placed in an area on the left ventricular wall where an infarct was anticipated. These regions became infarcted in dogs 8 and 9. Slight transient T-wave changes were seen as the electrodes were placed in position. Otherwise, no changes were observed.

Myocardial infarction was induced by Harris's method of double ligation. In dogs 1 through 5, only the anterior descending branch of the left coronary artery was ligated. In the other dogs, the circumflex branch of the left coronary artery was ligated usually together with a small branch of the anterior descending coronary artery.

The six limb leads were monitored on the oscilloscope but only two leads, usually aV_L and aV_R, were recorded. In dogs 12 and 13, additional epicardial leads were recorded, one from the anterior right ventricular wall and one from the left ventricular wall 2 to 3 cm. caudal to the stimulating electrode. These epicardial leads were taken with a small saline-soaked pledget of cotton as the contact medium. The position of these leads before and after infarction were within 5 mm. of each other.

A Grass stimulator served as the source of the electrical impulses. Frequency, duration, and voltage of the stimulus were controlled. The minimal stimulus necessary to produce a ventricular complex was used. This stimulus varied with each dog and from ventricle to ventricle. The voltage varied from 6 to 130 mv. The duration of the stimulus was 0.01 second, but in two instances was 0.02 second. The frequency was one stimulus a second.
in all dogs except one. In this one, the frequency was increased to 1.5 a second so as to be out of phase with the sinus beats. In most instances, the stimulus was the same before and after infarction. In four, the voltage was greater and in one, less after infarction.

**Results**

In dogs 1, 5, 6, and 7 electrocardiographic evidence of myocardial infarction failed to occur although gross evidence of infarction was clearly visible. There was also no change in the electrically induced postinfarction premature complexes. However, if the heart was displaced so that the infarcted region was at least partially in contact with some portion of the thoracic wall, electrocardiographic evidence of infarction appeared and the electrically induced complex assumed the characteristic deformities described below.

Coronary ligature in dogs 8 through 13 produced definite electrocardiographic evidence of myocardial infarction. These findings were the classical ones of ST-T segment deviations from the base line and T-wave changes. In addition, less prominent but definite changes in the QRS complex were observed. In two, the initial and terminal portions were deformed. In the others, definite but more subtle changes in the terminal deflections of the QRS were present. Commonly, this consisted of obliteration of a small terminal R wave following a large S-wave deflection and comparable changes in the other limb lead. The changes in the electrically induced postinfarction premature ventricular complexes mimicked these findings very closely except that they were usually more marked. The more premature the premature beats, the more exaggerated the abnormalities compared to those of the sinus propagated ventricular complexes. Moreover, at times the final deflection of the electrically induced premature systoles had contours suggesting an older lesion than that of the sinus propagated complexes, in the sense that terminal negativity was confined to the former. Not only were the ST-T segments more deviated from the base line, but notching of the QRS at times appeared at the time of inscription of the terminal segments of the postinfarct QRS complex. Only rarely were the changes in the electrically induced complexes less marked than those in normally conducted ones. All of these findings were present regardless of whether the right or left ventricle was electrically stimulated (fig. 1).

The findings in dogs 9, 10, and 12 are of particular interest because premature ventricular systoles after clinical myocardial infarction are thought to arise at the border
of the ischemic zone of infarction. In these dogs, left ventricular stimulating electrodes were placed by chance at the left anterior superior border of the infarct. In dogs 9 and 12, premature complexes induced by left ventricular stimulating electrodes had abnormalities greater in amplitude, whereas those from right ventricular stimulating electrodes had abnormalities smaller in amplitude but both were very similar in contour to those of the sinus propagated ventricular complexes. However, this relationship in magnitude was reversed in dog 10.

Spontaneous premature ventricular systoles also were recorded after myocardial infarction. These complexes were also characteristic of infarction and similar in contour to the electrically induced ones.

In no instance did the electrically induced premature ventricular beats fail to show the diagnostic pattern of infarction seen in the sinus propagated beats.

**Discussion**

These findings lend support to the clinical concept that premature ventricular complexes may be significantly and characteristically deformed by underlying cardiac disease. In all instances, experimental myocardial infarction produced characteristic changes in the final deflection of the electrically induced ventricular premature beats if such characteristic findings were present in beats of normal origin. Subtle changes in depolarization also developed.

That changes in the premature ventricular complexes are actually produced by the infarcted region is strongly supported by the findings in dogs 1, 5, 6, and 7. In these dogs, electrocardiographic evidence of the grossly visible myocardial infarction was not produced until the infarcted region was brought in contact with the thoracic wall. Sayen, Katcher, and Peirce have shown that in maximally insulated hearts, moderate-sized anterior and posterior ischemic areas can be produced without any electrocardiographic changes remote from the ischemic zone. Furthermore, evidence of the origin of these changes is suggested by our experiments which indicate that ligation of an additional coronary artery in a previously infarcted heart with electrocardiographic signs of injury produced comparable changes in the injury currents of the sinus-propagated and electrically induced ventricular complexes (fig. 2).

The findings suggest that the contour of ventricular premature beats may be of value in the diagnosis of acute myocardial infarction. Our findings, however, fail to explain occasional instances in the human with acute infarction in which ventricular extrasystoles do not display the characteristic deformity present in beats of sinus origin.

**Summary**

Ventricular premature beats were produced by stimulating electrodes inserted into the anterolateral right ventricular and into the
midlateral left ventricular wall of 13 open-chest anesthetized dogs. In 9 of the 13, acute myocardial infarction deformed the final deflection of the experimentally induced ventricular extrasystoles so that they mimicked the diagnostic pattern present in the beats of sinus origin. In 4 of the 13, no changes occurred in either the extrasystoles or in the beats of sinus origin until the infarcted region was brought into contact with the thoracic wall, at which time diagnostic patterns of infarction appeared in both types of ventricular complexes. Reinfarction produced by ligating an additional coronary branch produced comparable changes in the final deflection of the premature and sinus-propagated beat. These findings suggest that the inscription of the terminal deflections of premature ventricular beats is dominated by events within the infarcted region. They support the clinical concept that the configuration of ventricular extrasystoles may be of value in the recognition of acute myocardial infarction. The findings do not explain, however, the occasional absence of diagnostic findings in ventricular premature beats in electrocardiograms otherwise diagnostic of human myocardial infarction.

References
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