VENTRICULAR PREMATURE BEATS IN THE DIAGNOSIS OF MYOCARDIAL INFARCTION

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The importance of ventricular premature beats (VPB) in the clinical diagnosis of myocardial infarctions has been pointed out by several authors. Dressler (1943) reported a case in which the electrocardiographic signs of infarction were present in such beats (deep and slurred Q waves in lead III) and absent in the sinus beats. Simonson et al. (1945), Bellet (1953), Scherf and Schott (1953), Katz et al. (1958), Silverman and Salomon (1959), and Anttonen et al. (1959) recognized that myocardial infarctions can be diagnosed from VPB and, at times, even earlier from these cycles than from the sinus beats.

The similarity of ventricular activation in VPB and in right bundle-branch block (RBBB) and left bundle-branch block (LBBB) has long been accepted. Consequently, VPB with unipolar patterns of LBBB are right VPB, and VPB with patterns of RBBB are left VPB. The same considerations apply also for supraventricular premature beats (SVPB) with aberrant conduction, since the aberration is due to some degree of either RBBB or LBBB (Bisteni et al., 1960). Thus, right VPB and SVPB with aberration similar to that in LBBB are analysed in the same manner as sinus beats with LBBB. In fact, in these three situations the process of ventricular activation follows a similar sequence: the right ventricle is activated before the left. This type of reasoning applies also for left VPB, RBBB, and SVPB with RBBB: in these three instances the left ventricle is activated before the right.

A better knowledge of the ventricular activation process in normal conditions and in bundle-branch block has served for a new approach to the diagnosis and localization of myocardial infarctions. Sodi-Pallares et al. (1957, 1960) have shown that tracings with electrical signs of infarction are better understood when analysed in the light of recent studies concerning the ventricular activation process (Sodi-Pallares et al., 1955; Medrano et al., 1956, 1957, and 1958). It has been demonstrated also that septal infarctions may be more easily recognized in the presence of bundle-branch blocks (Sodi-Pallares, 1956), in contrast with the view generally held. On the basis of these considerations the significance of experimental and clinical VPB in the diagnosis of myocardial infarction is studied in this paper.

Material and Method

Experimental. Right and left VPB were produced in 12 dogs, before and after ligation of the anterior descending branch of the left coronary artery, according to the technique of Harris (1950). The ectopic beats were produced by tapping directly on the appropriate epicardial surface, or by means of a Grass Stimulator Model S4-CR with application of a stimulus of 4 volts for one millisecond at many points over either ventricle.
In all experiments the usual twelve electrocardiographic leads were recorded and unipolar epicardial leads at various points were also registered in three animals. Recordings were obtained with a Sanborn Poly-Viso machine model 150 or with a Grass six-channel polygraph model 932, using paper speeds of 50 and 60 mm./sec., respectively. The observations were carried out up to 48 hours after coronary artery ligation. At the end of the experiments, the dogs were sacrificed and the myocardial lesions were studied and carefully localized.

Clinical. The electrocardiograms of patients from the files of the Instituto Nacional de Cardiología de México with the diagnosis of myocardial infarction were reviewed. The electrocardiograms of one hundred cases showing premature beats (ventricular and supraventricular) were studied in detail. Correlation with the findings at necropsy, when this was available, was made.

Results

Experimental. The unipolar patterns of VPB, produced before ligation of the anterior descending coronary artery, were similar to the patterns observed in experimental bundle-branch block. Right VPB resembled the morphologies of LBBB, and left VPB those of RBBB. Another important generalization from our studies in the normal dog’s heart was that VPB, either right or left, having a QRS complex with a positive area never presented an initial negativity (Q wave) in leads that reflected the potential variations of the epicardial surfaces of either ventricle. In other words, the VPB never showed a QR type of unipolar pattern (initial negativity followed by a positivity), irrespective of their sites of origin in the ventricles. Only after myocardial infarctions were produced, did right or left VPB present a QR pattern (QR, QRs, or Qrs complexes) in leads that reflected the potential variations of the ventricles.

Two conditions are necessary for the VPB to have a diagnostic significance of myocardial infarction. First, the unipolar form must be of the QR and not of the QS type, since the latter can be found at epicardial and precordial points close to, or corresponding to, the site of origin of the VPB in the ventricle. Secondly, the QR pattern of VPB must be recorded in leads that

![Fig. 1.—(A) Leads V4, V5, and V6 recorded simultaneously as a control; (B) and (C) The same tracings obtained one hour after occlusion of the anterior descending coronary artery. The third beat is a premature right ventricular beat. Note the Q waves in the extrasystolic complexes while there is no initial negativity in the sinus complexes.](http://heart.bmj.com/br-heart-j-first-published-as-10.1136/hrt.23.5.521-on-1-september-1961)
Fig. 2.—Premature right ventricular beats produced mechanically and occurring before the P wave. High degree of aberration.

reflect the potential variations of the ventricles. Therefore, QR patterns recorded in leads aVR and aVL have no value, since these leads in the dog are oriented to the right and to the left atrium. In fact, QR complexes are usual patterns in the right atrium for left VPB (and RBBB) and in the left atrium for right VPB (and LBBB), in the absence of myocardial infarction.

In Fig. 1 is shown a right VPB (unipolar pattern of LBBB), produced before (A) and one hour after (B and C) coronary artery ligation. In the sinus beats no signs of infarction are seen, while the VPB is diagnostic of infarction because of its QR form (C). The initial positivity of the VPB in (B) is due to the ascending limb of the P wave. This VPB could hardly be considered a “fusion beat” in view of the conspicuous aberration and the duration of the ventricular complex (Bisten et al., 1960).

The control tracing of another dog, in which right VPB were provoked before (Fig. 2) and at the end (Fig. 3) of the P waves is shown in the corresponding figure. Only in the latter were “fusion beats” produced. Note that the VPB do not show initial Q waves. Tracings were taken 48 hours after coronary ligation in this animal (Fig. 4). The ectopic beats are of spontaneous origin and are discharged at different times in the cardiac cycle, some before and some after the P waves, thus producing varying degrees of “fusion beats” and consequently different degrees of aberration. The sinus ventricular complexes do not present signs of myocardial necrosis but only signs of injury and ischaemia. On the other hand, the VPB now begin with significant Q waves
The ectopic beats were provoked late in diastole, at the end of the P wave ("fusion beat").

which are deeper and wider as the ectopic stimulus discharges closer to the preceding T wave, and therefore shows more aberration. The ectopic beats with much aberration resemble incomplete LBBB with Q waves. Patterns similar to these have been described by Sodi-Pallares et al. (1960) in human septal infarctions with complete or incomplete LBBB. Necropsy study of this dog revealed an infarction of the lower half of the interventricular septum.

In another dog, right (Fig. 5) and left (Fig. 6) VPB were produced. Column A in each figure shows the control precordial leads. Again no QR patterns are recorded. In column B of both figures are the electrocardiograms obtained one and a half hours after coronary artery ligation. Column C shows the tracings obtained six hours (Fig. 5) and seven hours (Fig. 6) after ligation. Comparison of the sinus beats before and after ligation reveals the appearance of a significant degree of injury. The VPB also present striking changes. The right VPB (Fig. 5, columns B and C) show qRs in V5 and qR in V6, and the left VPB (Fig. 6, column C) show QR complexes from V1 to V3.

A similar behaviour of the VPB is also seen in the direct epicardial leads (Fig. 7) in another experiment. Lead II is a simultaneous recording of three epicardial points: point 1, at the free wall of the right ventricle; point 2, on the left ventricle near the anterior edge of the interventricular septum; and point 3, on the lateral wall of the left ventricle. The right VPB before coronary artery ligation (column A) are of the RS type at points 1 and 2, and of the R type at point 3. One and a half hours after the ligation (column B), a small q wave appears in the sinus beats at points 2 and 3 with a marked positive displacement of the RS-T segment. A right VPB (second beat, column B) shows a deep and wide Q wave only at point 2, which was subsequently found to be at the centre of an infarcted area, while points 1 and 3 were at the periphery.

Clinical. The clinical studies of the value of the VPB in the diagnosis of myocardial infarction are in agreement with the experimental findings. Fig. 8 is the electrocardiogram of a
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FIG. 4.—Same animal as in Fig. 3. Tracing taken 48 hours after the production of coronary occlusion. Various degrees of "fusion beats" are seen. The signs of necrosis are present in the premature beats. Note that the Q waves are deeper and wider as the ectopic stimulus discharges closer to the preceding T wave.

patient in whom the sinus beats show a marked degree of RBBB with Q waves from V1 to V5, corresponding to an infarction of the middle and inferior thirds of the interventricular septum. The second beat in all leads is a left VPB with a significant initial negativity and a shape similar to RBBB. Both the sinus beats and the VPB have a similar pattern, since in both there is a similar asynchronism in ventricular activation, the left ventricle being activated before the right.

In Fig. 9 a myocardial infarction is recognized from the sinus beats (QS in V2 and Qrs in V3), as well as from the VPB (QR in V1 and Qrs in V2). In both of these patients (Fig. 8 and 9) the infarction can be diagnosed from both the sinus complexes and the VPB.

Infarction might be thought to be localized to the posterior and inferior aspects of the heart if only the sinus beats were considered in Fig. 10 (deep Q waves in leads II and III). However, the VPB seen in leads V1, V4, V5, and V6 begin with slurred Q waves and suggest that the area of necrosis extends also to the anterolateral aspect of the heart. Necropsy revealed that the entire extent of the infarction was as suggested by the complete study of the electrocardiogram. This case
demonstrates how the VPB may often permit a more precise localization of an infarction than do the sinus beats with no asynchronism of ventricular activation.

Incomplete LBBB is shown in Fig. 11. The QS complexes of sinus origin in leads III, V1, V2, and V3 can be ascribed to the block and are not suggestive of myocardial infarction. However, the morphology of the VPB in V3 (qR) strongly suggests infarction of the septum, which was subsequently proven at necropsy. In Fig. 12 the sinus beats with normal conduction reveal only an antero-lateral ischemia. The VPB in lead III (QR) and in leads V4, V5 (qRs) are diagnostic of a septal infarction. At necropsy a healed infarction of the lower half of the septum with extension to the apex and free wall of the left ventricle was found.

The sinus beats of the electrocardiogram presented in Fig. 13 are suggestive of extensive anterior ischemia. However, myocardial infarction can be diagnosed from the VPB in V3. The T wave of this beat suggests primary ischemic changes. We believe that injury and ischemia can be diagnosed from characteristic alterations of the RS–T segment and T wave of VPB. These investigations will be the subject of future studies.

DISCUSSION

Our experimental and clinical studies support the concept that myocardial infarctions can be diagnosed from VPB, even though the normal sinus beats do not allow this diagnosis. The unipolar patterns of VPB that have been found to have diagnostic value are of the QR type (QR, QRs or Qrs complexes). The VPB, irrespective of their site of origin showed a QR pattern only after the experimental production of myocardial infarction. It should be noted, as mentioned earlier, that the QR pattern of the VPB must be recorded in the leads which reflect the potential variations of the epicardial surface of the appropriate ventricle.

We have already pointed out several times in this study that a similarity exists between the sequence of activation of VPB and BBB which determines in turn the similarity in shape of the
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Fig. 7.—Lead II recorded simultaneously with epicardial tracings at the free right ventricular wall (1) and at the free left ventricular wall (2 and 3). (A) Control, (B) after coronary occlusion. Note the deep Q wave in the extrasystole at point 2 in (B), only after the production of myocardial infarction.

Fig. 8.—Electrocardiogram of a patient with right bundle-branch block and signs of an infarction of the middle and lower thirds of the septum. The infarction is recognized in the extrasystolic complexes as well as in the sinus beats.

complexes derived in both situations. It is reasonable, then, to interpret the QR pattern of a right VPB as we have previously interpreted the QR pattern in LBBB (Sodi-Pallares, 1956) and also the QR of a left VPB as that of RBBB. It should be emphasized that the QR morphology in a right VPB (LBBB) is more commonly seen in the left precordial leads (Fig. 5) and the QR morphology in a left VPB (RBBB) in the right precordial leads (Fig. 6). In other words, the QR is more commonly found in the potentials derived from the ventricle that is the last to be activated, since the epicardial potential variations from the ventricle that is activated earlier very often show patterns of the QS or rS type, (with or without infarction), but not QR complexes. (Note rS morphologies in V1, V2, V3, col. C, Fig. 5 and QS morphologies in V4, V5, V6, col. C, Fig. 6.) Finally it is obvious that these observations apply equally to the supraventricular VPB with aberrant conduction.

The diagnostic value of ectopic beats may be assessed in the following ways.

1. The infarction may be diagnosed from the sinus beats as well as from the ectopic beats (Fig. 5, 6, 8, and 9).
2. The infarction may be diagnosed from the sinus beats as well as from the ectopic beats, but the latter are of more value in defining localization and extent (Fig. 4 and 10).
3. The infarction may be diagnosed from the ectopic beats and not from the sinus beats (Fig. 1, 11, and 12).

The important question to be answered is why an infarction is not revealed by the basic sinus rhythm with normal ventricular activation but is revealed by the presence of VPB or SVPB with aberrant ventricular response. This question and its answer constitute an excellent example of what we have come to call “deductive electrocardiography”, that is, the mental process of understanding the patterns reflected in any electrocardiogram as a function of the sequence of ventricular activation. Let us consider a concrete example relating to this question.

As an example of forces that are easily recognized during the normal activation process one may choose those that correspond to the activation of the middle third of the left septal mass. This activation gives rise to an initial positivity in V1 and V2; and an initial negativity in V5 and V6.
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Fig. 11.—Doubtful signs of posterior myocardial infarction in the sinus beats. The form of the extrasystole in V3 strongly suggests an infarction of the septum, which was proved at necropsy.

Fig. 12.—Premature ventricular beats in leads V3, V4, and V5 suggesting the diagnosis of a necrotic area. A healed infarction of the lower half of the septum was encountered at necropsy. The sinus beats do not suggest infarction.
If an infarction involves the middle third of the interventricular septum, these forces will not be produced and the infarction may then be diagnosed by the absence of the forces that are easily identified in the normal activation process (Sodi-Pallares, 1960). Since the forces generated by the lower third of the left septal mass are not easily identified during normal activation, because of the electrical predominance of the forces of the free left ventricular wall, we cannot diagnose infarctions in this area in the presence of normal conduction. However, with a right VPB or a SVVPB with some degree of LBBB or sinus rhythm with LBBB the activation of the lower portion of the septum becomes relatively dominant and separated as a result of the asynchronous ventricular activation and its vector is then easily recognized in the upstroke of the wide and aberrant QRS complex of the left precordial leads.

As we have described (Sodi-Pallares, 1956), this vector is oriented toward the left, downward and slightly backward since it is recognized as a positivity in V5 and V6 (Fig. 14A). When an infarction occurs in this region and particularly in the anterior portion of the lower left septal mass, this vector is changed and now points more backward and probably upward giving rise to a Q wave in V5 and V6 (Fig. 14B).

It is worth stressing again that this type of infarction can be recognized only in the presence of asynchronous ventricular activation, since it is obscured in the case of normal ventricular activation, as discussed earlier. Furthermore, even in the presence of ventricular asynchrony, such an infarction can be diagnosed only if the unipolar patterns are interpreted in the light of the knowledge of the ventricular activation process.

Fig. 4 shows an example in which the duration, depth, and aberrancy of the Q wave increases with the degree of ventricular asynchrony. In V5 of Fig. 4 the first two beats are sinus beats with normal ventricular activation and no abnormal Q waves are seen. In the third beat which is a "fusion-beat" (Bisteni et al., 1960), there is a slight degree of aberration and a Q wave is inscribed. The fourth beat, also a "fusion-beat", occurs earlier in relation to the P wave which means greater dissociation and the Q wave is now wider and deeper. Finally, when the ventricular aberration is greatest, as in the fifth beat (VPB without fusion), the Q wave is seen to be widest and deepest of all. This clearly demonstrates the role of the asynchronous ventricular activation in revealing the infarction.
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Fig. 14.—Scheme showing the vector of activation of the left septal mass in the presence of left bundle-branch block: (A) without infarction; (B) with infarction in the anterior portion of the lower septal mass.

The considerations expressed are in agreement with the localization of the infarction in the post-mortem studies. In all the animals the infarctions involved the lower half of the anterior portions of the interventricular septum with or without extension to the upper anterior portions and to the lower posterior regions of the septum. There was extension also to the free left ventricular wall in all of the dogs.

In the three clinical cases with necropsy data available, the lower portions of the septum were involved, with extension to the free left ventricular wall in one case. If the myocardial infarction is localized in the free left ventricular wall without involvement of the interventricular septum, the premature beats will not be of aid in the recognition of the necrotic tissue. For example, a right VPB will produce an RS complex in the left ventricular cavity which will then be transmitted through the electrical window produced by the infarction of the lateral wall and thus will be reflected in V5 and V6 solely as an RS complex and no Q wave will be seen. With the same type of infarction a left ventricular premature beat will determine a QS complex and not just an initial Q wave so that neither will this tracing be characteristic of an infarction. Similar methods of analysis can be applied to the complexes of bundle-branch block.

SUMMARY

Experimental and clinical studies on the significance of ventricular premature beats in the diagnosis of myocardial infarction are reported.

The unipolar patterns of VPB, that are diagnostic of myocardial infarction are of the QR type (Qr, Qrs, and Qrs complexes).

The post-mortem studies revealed that the interventricular septum was always involved.

The conclusion is reached that any ectopic beat with aberrant conduction, irrespective of its site
of origin (right or left ventricle, ventricular or supraventricular), is diagnostic of myocardial infarction when the ectopic beat presents a QR type of morphology in leads reflecting the potential variations of the ventricles.

A comparison of the diagnostic value of ectopic beats and sinus beats with normal conduction, can be summarized according to the three main possibilities, thus:

1. the infarction may be diagnosed from the sinus beats as well as from the ectopic beats;
2. the infarction may be diagnosed from the sinus beats and from the ectopic beats, but the latter are of greater value in defining its localization and extent; or
3. the infarction may be diagnosed only by the patterns of the ectopic beats and not by those of the sinus beats.

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REFERENCES


