DIFFERENCE has been observed in certain instances between the onset of QRS in intracavitary and surface limb leads. Simultaneous tracings demonstrate that when premature ventricular depolarization exists the early rapid growth in magnitude of potential recorded by intracavitary leads may be nearly or totally inapparent in surface leads. Following premature depolarization changes occur in intracavitary and surface leads that shed light on the electrical basis for surface electrocardiographic inscription of “fusion beats,” initial and terminal QRS abnormalities, and secondary ST-T wave changes.

Materials and Methods
Premature ventricular complexes were observed during the course of 50 consecutive right- and left-sided heart catheterizations performed separately or as combined procedures. The intracavitary electrocardiogram was recorded simultaneously with surface leads and usually with cavity pressure curves. A multichannel cathode-ray tube recorder* with a photographic recording system and a paper speed of 75 mm. per second was used to record tracings of simultaneous conventional electrocardiographic leads, unipolar intracavitary leads, and intracavitary pressures. Time lines in the records are separated by 0.02 second. Amplifier sensitivity was usually 30 mm. per millivolt. High-frequency amplifier cut-off in the illustrations conforms to that of direct-writing electrocardiographs. Cardiac pressures were recorded with strain gages.† Esophageal leads were recorded as unipolar leads with an esophageal electrode catheter.‡

Each electrocardiographic lead was recorded by separate sets of electrodes and separate but matched amplifiers. The surface lead generally selected for comparison with the intracavitary lead was the one most parallel to the QRS axis for the subject, since that was found to have a better signal-to-noise ratio than others. Two conventional surface leads were used to demonstrate that absence of changes in an isolated surface lead were not due to perpendicularity of an electrical current to the plane of that lead. Electrode catheters were used to record intracavitary electrocardiograms. Right intracavitary leads were recorded with Courand electrode catheters* connected to exploring electrodes of Wilson central terminals. Left intracavitary leads were obtained by means of a thin copper wire inserted through the polyethylene catheter utilized in percutaneous transthoracic catheterization of the left heart. The tip of the wire was at the distal end of the catheters and its proximal portion was connected to the exploring electrode of a Wilson central terminal. The intrinsic deflection of the intracavitary lead was used as a measure of time of occurrence of depolarization in the region of the electrode catheters.

Comment
This paper deals predominantly with right ventricular complexes because of the greater number of right cardiac catheterizations performed in our laboratory. Study of the more limited left heart catheterization material suggests that findings from the left will be qualitatively the same as from the right. During cardiac catheterization premature beats are frequently induced. All of the complexes in this study could be considered to be due to the presence of the catheter within the heart. We assume that premature complexes in the presence or absence of a cardiac catheter follow similar patterns and electrical principles. All the subjects in this study had acquired or congenital heart disease and were included in this study without regard to age, sex, or diagnosis. Tracings were selected to illustrate findings common to the group of patients catheterized.

Results

Figure 1
“Normal sinus rhythm” is present throughout the tracing. The third ventricular complex is

Simultaneous right ventricular intracavitary electrocardiogram (top), right ventricular intracavitary pressure curve (middle) and lead II electrocardiogram (bottom). Time scale in Figure 1 applies to all figures except Figure 4. The 30-mm. per millivolt scale in Figure 3 is applicable to all surface leads except in Figure 4. The intracavitary complexes were recorded at either 5 or 30 mm. per millivolt. The intracavitary leads in Figure 3, and the left ventricular leads in 5B and 8 were recorded at the former sensitivity to allow full visualization on the figures.

See legend for Figure 1.

Figure 1

Premature right ventricular depolarization manifested by the early intrinsic deflection of the right ventricular lead. Evidence of premature ventricular depolarization at its actual time of occurrence is lacking in lead II. Shortening of "P-R segment," increase of QRS amplitude, disappearance of S wave, and secondary ST-T-wave changes are seen after the premature right ventricular depolarization.

A notch is present near the bottom of the descending limb of the intracavitary QRS in the abnormal complex. This notch is present at the onset of the normal intracavitary complexes. The time intervals between these notches in all complexes are the same as the P-P intervals and the intervals between intracardiac pressure points.

Conclusions

It is likely that the notch and the subsequent portion of the intracavitary lead is due to the sequence of normal activation in the right ventricle. In the third beat premature ventricular depolarization is apparently aborted by normal processes. The area of myocardium responsible for the S wave may have been the source of the premature depolarization. Although the usual order of electrical events in the right ventricle is disturbed, there is no visible change in the pattern or rhythmicity of the right ventricular pressure curve.

Figure 2

In the second cardiac cycle premature depolarization is evident in the right ventricular lead, but simultaneous changes suggestive of early depolarization are not seen in the limb or atrial leads. Two hundredths of a second after the intrinsic deflection in the intracavitary lead the QRS complex of lead II shows an 0.04 second Q wave, decreased QRS amplitude, a slightly prolonged Q-T interval, S-T elevation, and secondary T-wave changes. The right atrial lead does not disclose significant simultaneous potential changes. In contrast to Figure 1, in which the rhythmicity of ventricular pressure pulses was unchanged, prematurity of right ventricular depolarization was accompanied by a 0.02-second earlier onset of rise of ventricular pressure.

Conclusions

Premature onset of the ventricular pressure may or may not accompany premature ventricular depolarization. Premature right ventricular depolarization can produce isolated complexes in which QRS contours and secondary ST-T-wave changes in the limb leads are similar to patterns associated with myocardial infarction.

Figure 3

Normal sinus rhythm is present throughout the tracing. The third beat shows premature right
ventricular depolarization of about 0.03 second in both ventricular leads taken with separate electrodes 1 mm. apart on the same catheter. Lead II demonstrates a short P-R interval and a small positive upstroke before the usual R wave. The right atrial lead shows a minor change in the downstroke of its QRS but no clear evidence of right ventricular prematurity. In the right ventricular pressure curve, the third beat is approximately 0.02 second shorter than the others. Only the descending limb of the right ventricular pressure curve shows minimal alteration in its contour.

**Conclusions**

Early depolarization of a portion of myocardium may not materially affect the basic pattern of other intracavitary deflections, and surface electrocardiographic changes may be minimal. In this tracing the QRS deflection in the right atrial lead showed no significant change due to the right ventricular depolarization, suggesting that the abnormality was generated by a small myocardial area. This may be the reason why only minimal changes in duration and contour of pressure waves accompany premature ventricular depolarization. In this tracing terminal forces of depolarization seen in the normal beats have apparently shifted to the initial portion of the abnormal cycle without disturbing the depolarization of the myocardial areas responsible for major QRS and ventricular pressure curves.

**Figure 4**

The first sequence is the normal one for this subject with atrial fibrillation. Electrode catheter RV₁ was at the tricuspid valve level and RV₂ was at the pulmonic valve level. The second and third sequences showed markedly asynchronous depolarization within the same ventricle. Depolarization occurred prematurely 0.04 second earlier at the tricuspid valve level than at pulmonic valve. Lead II showed a small Q wave, a prolonged QRS of lower than normal voltage, and ST-T-wave changes. Pressure pulse contours are normally different at each valve level (1st complex). The duration of ventricular systole decreased after the asynchronous depolarization (second and third complexes).

**Conclusions**

Within the same ventricle there are areas of myocardium more susceptible than others to premature depolarization; conversely premature depolarization represents a segmental or localized change in pattern of depolarization. The shortened
ventricular systole may be due to premature depolarization but could also be a manifestation of changes in R-R interval.

Figures 5A and 5B

In the third cycle of 5A right ventricular depolarization is 0.09 second premature and right ventricular pressure is 0.03 second early. Lead I shows no changes directly simultaneous with the early inscription of the intrinsic deflection. Immediately following premature ventricular depolarization lead I shows a decreased P-R interval, a Q wave of 0.03 second duration, decreased QRS amplitude, and Q-T prolongation.

Figure 5B from the same subject shows the same situation in the first two cardiac cycles. A simultaneous left ventricular lead has also been recorded. Approximately a 0.1-second difference exists between the onset of the intrinsic deflection in the right and left ventricular complexes during the time that the surface lead records the Q wave.

In the normal third complex there is no longer a marked "phase" difference between the intrinsic deflection of right and left ventricles. The period for the intrinsic deflections of the left ventricle is constant for all cycles as is the P-P interval. The period for the intrinsic deflection of the right ventricle is not constant because of premature right ventricular depolarizations.

Conclusions

Since atrial and left ventricular complexes are regular throughout the tracing, the abnormal surface complexes may be assumed to be due to right ventricular premature depolarization. A series of beats, as well as isolated ones (fig. 3) with changes like those associated with myocardial infarction can occur with premature right ventricular depolarization. The marked electrical
asynchronism between left and right ventricles was the apparent cause of the surface lead changes.

Figures 6A and 6B

The periods between the intrinsic deflection of all right atrial P waves and right atrial QRS complexes are identical and constant in these two continuous tracings. The P waves in lead II do not change in contour or timing. Since the intrinsic deflection of the atrial P wave is constant, the intrinsic deflection of the right ventricular lead can be referred to it for timing purposes. The intrinsic deflection of the right ventricle does not maintain a constant period. In the first cycle a small Q wave and a small S wave are present in lead II along with an isoelectric T wave. In subsequent cycles the Q and S waves are no longer present, the “P-R segment” is of varying duration, the QRS duration increases slightly, the QRS amplitude becomes greater, and secondary ST-T-wave changes occur.

Conclusions

The degree of shortening of “P-R segment,” QRS abnormalities, and secondary ST-T-wave changes in the limb lead reflect the degree of prematurity of ventricular depolarization. As the interval between atrial and ventricular depolarization shortens (measured here from intrinsic deflection of the atrial P wave to that of the right ventricular QRS) these changes become more pronounced. In an individual subject similar surface electrocardiographic complexes tend to have the same atrial ventricular depolarization time. (Examples: Sequences 3 in figure 6A and 3 in 6B, sequences 1 and 2 in 6B). The decreasing P-R interval with increasing changes in QRS suggests the “concertina effect” in the Wolff-Parkinson-White syndrome.

Figure 7

The second sequence shows premature right ventricular depolarization following premature atrial activity. A shift toward right axis deviation is present. Lead I has a Q wave of 0.03 second duration, decreased QRS amplitude, and minor T-wave changes. Lead III has greater amplitude of QRS and T. Right ventricular QRS and T amplitude greatly increased after premature activation of right ventricle. Right ventricular pressure was also early and had during the abnormal sequence a decrease in systolic pressure and alteration of its configuration. The phonocardiogram preceding the instance of prematurity demonstrated splitting of the second sound.

Conclusions

Prematurity of ventricular activation causes shifts of electrical axis as seen in surface leads. The large ventricular amplitude found during premature activation is not proportionally displayed on the surface although it is suggested by changes in the electrical axis of the forces of the premature depolarization. The large electrical potential of premature depolarization is not associated with an increment of ventricular pressure but rather with a decrease and a change in the pressure curve.

Figure 8

The first and fourth complexes are normal for the subject. The third complex is an interpolated premature ventricular contraction. The tracing shows simultaneous right and left ventricular leads (left ventricle was recorded at one-sixth the sensitivity of the other leads) left atrial, and lead II. The P waves in the left atrial curve show “normal sinus rhythm” throughout. The second complex demonstrates approximately 0.04 second early depolarization of left ventricle. The amplitude of the left ventricular QRS increased about twice and QRS area about four times during the instance of prematurity, but the right ventricular complex shows no change, and early left ventricular depolarization was evident in right ventricle by ST-T-wave changes only. The left atrial QRS showed a Q wave approximately three times deeper than found during the normal beats. Left atrium and lead II have a shorter than normal P-R interval. The QRS is slightly increased in amplitude in lead II and the ST-T-wave area is more depressed than in normal beats.

Conclusions

Left ventricular premature depolarization produces surface and intracavitary changes similar to changes seen during premature right ventricular depolarization.

Discussion

It is suggested by these studies that the initial electrical forces of premature ventricular complexes begin before they are apparent on conventional surface leads. In instances
in which a portion of myocardium depolarizes early its potential does not have its usual phase relationships to other potentials. Premature depolarization of an area of musculature will produce surface electrocardiographic changes when the electrical discharge is sufficiently out of phase from other voltages large enough to obscure it and when its voltage is of sufficient magnitude not to be lost in transmission to the surface. Minor degrees of premature depolarization or discharges of low amplitude may not be readily apparent at conventional sensitivity of 10 mm. per millivolt.

Electrocardiographic tracings recorded with high amplification suggest that there is normally an overlap of atrial and ventricular potentials in the “P-R segment” which is not apparent in tracings recorded at conventional amplification. Figure 6 demonstrates the relationship of surface “P-R segment” and QRS changes to the time difference between generation of atrial potentials and right ventricular depolarization. Changes in the initial part of QRS complexes may be due to superimposition of atrial potentials and initial forces of ventricular depolarization as “fusion beats.” This mechanism may produce surface Q waves. Myocardial infarction has been attributed to these changes in isolated beats by other authors. This seems unlikely in the present cases in view of lack of clinical, historical, or postmortem evidence of infarction and the frequency with which these changes occur during catheterization. It is possible, however, that an infarction could produce electrical asynchrony as shown in figure 5, or the electrical and mechanical asynchrony associated with premature depolarization as shown in figures 2, 3, 4, and 7, and thus cause the initial QRS abnormalities in surface leads usually associated with infarction.

During premature ventricular depolarization the depolarizing potential is shifted from its customary locus producing changes in the terminal portions of the QRS and ST-T wave. The secondary ST-T-wave changes, demonstrated in figure 6, have a constant relationship to the time interval between premature ventricular and atrial depolarization. The mechanisms of production of surface lead abnormalities in positive two-step exercise or Master’s tests may be due to the time difference between atrial and ventricular depolarization. It is conceivable that premature ventricular depolarization of an area of myocardium may occur during exercise tests.

**Summary**

Surface “fusion beats,” beats with initial and terminal QRS abnormalities, and accompanying secondary ST-T-wave changes appear following intracavitary evidence of premature depolarization. In instances of premature activation of a portion of myocardium, the intracavitary deflection of premature ventricular depolarization may not be apparent in surface leads and, if present, may be reflected in surface leads only by subtle changes in wave configuration that include (1) shortening of the P-R interval, (2) prolongation or amplitude changes or both in the QRS, (3) changes in contour of initial and terminal portion of the QRS, sometimes resembling those of infarction, and (4) secondary ST-T-wave changes. Evidence of electrical asynchronism in a single ventricle and between ventricles is presented.

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References


Man Has Three Separate States of Existence

Some Divines count Adam thirty years old at his Creation, because they suppose him created in the perfect age and stature of man. And surely we are all out of the computation of our age, and every man is some months elder than he bethinks him; for we live, move, have a being, and are subject to the actions of the elements, and the malice of diseases, in that other World, the truest Microcosm, the Womb of our Mother.—Sir Thomas Browne. Religio Medici. Edited by W. A. Greenhill, M.D. London, Macmillan and Co., Ltd., 1950, p. 63.
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