Parietal Focal Block: An Experimental and Electrocardiographic Study

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A method of producing focal ventricular block is described. The sequence of the electrocardiographic variations is ascribed to changes in the velocity and direction of the excitatory process in the ventricular wall. The epicardial electrocardiograms resemble those considered indicative of ventricular hypertrophy or of “incomplete” or of “complete” bundle branch block. When the focal block is pronounced a positive deflection appears in the cavity tracing. The ventricular blocks can be subdivided into “conduction blocks” and “fiber blocks,” the former produced by the delay of the stimulus in the specialized conduction system and the latter produced by the delay of the excitatory process in the ordinary heart muscle.

In the past, focal blocks have proved difficult to produce experimentally. Recently, however, Frau obtained satisfactory results by the infiltration of quinine salts into the ventricular walls. We have found that certain substances when injected into a coronary artery produce a parietal focal block. We believe that the results of these experiments have theoretic and practical importance. Preliminary observations have been already published.

Method

In 46 dogs 92 experiments were performed. Observations were made after splitting the sternum and opening the pericardial sac. Fresh saline solutions of 2.5 or 5 per cent cocaine chlorhydrate were used, and small amounts varying from 0.1 to 0.5 cc. were injected into the coronary arteries. In several experiments saturated solutions of morphine, 1 per cent solutions of strychnine and other substances were also injected. The results were similar to those produced by cocaine. In most instances the anterior descending coronary artery was used, but in several experiments injections were given into the smaller vessels seen upon the epicardial surface of the right or left ventricles. In two instances the injections were given before and after cutting the left branch of the bundle of His.

The right arm terminal of the electrocardiograph was connected to the right hind leg through a non-polarizable electrode, and the left arm terminal was connected to the exploring electrode through a similar nonpolarizable boot. A rather large, olive shaped, electrode of German silver was introduced in the ventricular cavities, the indifferent electrode was attached to the left hind leg. Direct leads were taken upon the epicardial surface of the ventricular muscle supplied by the artery in which the injection was given. Control electrocardiograms were always taken; during the experiments tracings were obtained from distant ventricular zones, and occasionally curves were recorded while the exploring electrode was moved slowly over the epicardial surface. Cavitary leads were obtained where the more illustrative electrocardiographic changes were observed; curves were also taken while the electrode was moved in the ventricular cavity. Direct-writing electrocardiographs were found to be useful in locating the most convenient points for obtaining permanent records.

Results

The saline solution of cocaine when injected in a coronary artery caused a “parietal focal block” (p.f.b.) in the ventricular territory irrigated by the vessel. The focal block developed rapidly while the injection was being given, and disappeared gradually in 15 to 30 minutes. The electrocardiograms taken while the exploring electrode was moved slowly over the epicardial surface demonstrated that the ventricular region where the parietal focal block was maximal was encircled by zones in which the degree of the block gradually decreased (fig. 1). The blocked ventricular zone was found...
Fig. 1. Left parietal focal block and left bundle branch block. Electrocardiogram 1 was taken upon the epicardial surface of the left ventricle before the experiment. Tracings 2 and 3 were recorded at the same point while parietal focal block was progressing. When focal block disappeared the left branch of the bundle of His was cut. Electrocardiogram 4 was taken at the same epicardial point when left bundle branch block was present. Tracings 5 and 6 were obtained when left bundle branch block and parietal focal block coexisted. The artery which was injected and the site of the exploring electrode are indicated in the diagram.

The electrocardiographic changes in parietal focal block. The lower tracing was taken while the exploring electrode was moved slowly over the epicardial surface following the direction indicated by the arrow. The electrocardiographic changes clearly demonstrated that the blocked region is encircled by ventricular muscle in which activation is normal and that the average degree of block is more pronounced in the central zone than in the periphery.

Fig. 2. Left parietal focal block. In this figure the control curves and those showing the sequence of the electrocardiographic manifestations in the epicardial and endocardial leads are seen. The curves were taken at short intervals during the experiments while the electrodes remained immobile.
to be surrounded by muscle in which the excitatory process was normal.

Disturbances of the heart rhythm and changes in the heart rate were rarely observed. In the ventricular wall where the block occurred the contractile activity locally decreased and this phenomenon had some relation to the degree of block.

The electrocardiographic changes in the epicardial leads. In right or left parietal focal block the terminal S waves when present in the control tracings disappeared rapidly when the block progressed (figs. 3 and 4). In left focal block the Q waves, when recorded in the control curve, persisted or became more prominent when the block progressed. Occasionally block progressed even more the RS duration was prolonged and a notch heralding the late positive deflection was seen in the ascending limb of the S wave. In all cases in pronounced right parietal focal block the ventricular complexes were of the rsR' type (figs. 3 and 4). In the unipolar direct epicardial leads in left and right parietal focal block tall and broad positive deflections were always recorded. As the block progressed the size of these deflections increased and their summits were inscribed gradually later (figs. 1, 2, 3, and 4).

The electrocardiographic changes in the cavitary leads. If the exploring electrode was placed far from the ventricular wall where the parietal focal block occurred the form of the electrocardiogram remained essentially the same. When the electrode was placed near to or in contact with the ventricular wall where the focal block occurred, conspicuous variations were observed. The cavitary potential became less negative while the block was incipient, and during developed block a late positive upstroke was recorded (figs. 2, 3 and 4). The size of the late positive deflection increased as the block increased. In right parietal focal block the endocardial electrocardiogram was of the rsR' or rSR' type (figs. 3 and 4). In left parietal focal block the cavitary tracings were of the Qr or QR type (fig. 2). When the exploring electrode in the ventricular cavity was moved away from the wall exhibiting focal block the size of the late upstroke diminished and finally

Fig. 3. Right parietal focal block. Electrocardiograms recorded while the parietal focal block progressed. When the block begins the size of the R and S waves diminishes, the RS relation changes but no definite variation in the RS duration is observed. A notch heralds the late R wave. When the block is pronounced the ventricular complex is of the rsR' type. Cavitary leads taken before the experiment and when the block was pronounced can be seen at the lower right.
disappeared. In all experiments a late positive deflection in the cavitary tracing was recorded only when parietal focal block was already evident (figs. 2 and 4).

The electrocardiographic changes in left parietal focal block complicated by left bundle branch block. When these experiments were performed, soon after the injection notable electrocardiographic variations were observed (fig. 1). The form of the ventricular complexes was strikingly modified and the QRS interval was prolonged. The sequence of the electrocardiographic manifestations was similar to those recorded in uncomplicated cases of parietal focal block.

**Discussion**

Conduction blocks and fiber blocks. In the advanced stages of parietal focal block the tracings are no longer similar to those seen in experimental bundle branch block. This observation suggests that when parietal focal block is pronounced the course of the excitatory proc-

![Image](http://circ.ahajournals.org/content/57/1/111.F.full)

**Fig. 4. Right parietal focal block.** Epicardial and endocardial leads taken at intervals until the normal curve reappeared. In epicardial as well as endocardial leads small initial R waves are present. When parietal focal block is severe the negative downstroke following the initial positive deflection becomes prominent. Terminal S waves recorded when ventricular excitation is normal disappear soon after the block begins. Variations in the size of the late positive deflections are obvious. In cavitary leads positive late deflections are present only while parietal focal block is pronounced; when the block disappears the negativity of the cavitary potential gradually increases. Tracings resemble those which in man are considered representative of right ventricular hypertrophy, “incomplete” and “complete” right bundle branch block.

ess is delayed in the muscle fiber itself. When one of the branches of the bundle of His is cut, no immediate changes can be expected in the intrinsic condition of the muscle fibers located in distant parts of the septum or in the ventricular walls. Experimentally produced bundle branch block is a good example of a “conduction block.” As may be seen in figure 1, when
a left parietal focal block is produced after the section of the left branch of the bundle of His, notable variations in the shape and duration of the ventricular complexes are observed; these changes can be ascribed only to the slow and abnormal course of the excitatory process in the ordinary ventricular muscle. The foregoing observations demonstrated the existence of "fiber blocks." Since in the experiments herein discussed the blocking substance was injected into a coronary artery, it must be expected that in the ventricular territory irrigated by the vessel all of the muscle elements, even those located near the endocardium, were damaged; consequently the parietal focal block produced by this method probably represents a mixture of "conduction" and "fiber" block. The subdivision of ventricular blocks into "conduction" and "fiber" block is consistent with the existing physiologic data. The Purkinje network delivers impulses more or less promptly to the contractile elements, but once the stimulus reaches the muscle fibers the spread and further course of the excitatory process must depend upon the actual condition of the ordinary cardiac fibers. In a future paper the electrocardiographic manifestations accompanying severe "fiber" block will be discussed.

The QRS changes in the epicardial leads. Under normal conditions in direct unipolar epicardial leads the S waves are not due to the electrical activity of the underlying explored zone, but to the electrical effects of some distant ventricular regions which are activated later. In right or left parietal focal block the terminal S waves diminish in size or disappear rapidly when the block begins. In the blocked region excitation is still progressing when the activation is finished in the rest of the ventricular muscle; consequently, even when the focal block is incipient, there are no distant electrical forces that may produce negative potentials by the time the final part of the electrocardiogram is recorded.

The Q waves are due to the electrical activity of the ventricular regions that are activated earlier than the explored zone. Since the spread of the excitation is delayed in the blocked region, in left parietal focal block the Q waves may persist, become more prominent, or are present only while the block is severe. In right parietal focal block the septum is not involved, therefore no changes can be expected in the initial positive deflection; similar observations have been made in human right ventricular blocks. The S waves following the initial R deflections in right parietal focal block and the Q waves in left parietal focal block are produced by a similar process.

In the electrocardiograms obtained in the blocked region we may expect active balancing electrical forces only when activation is progressing in the rest of the ventricular muscle. Hence, while the initial part of the ventricular complex is influenced by distant electrical forces, the final part of the tracing represents the unbalanced potential variations of the explored zone. Under normal conditions a positive deflection is inscribed in a unipolar direct epicardial lead if the voltage developed across the wall beneath the exploring electrode increases more rapidly than the negativity of the adjacent ventricular cavity. Once the ventricular muscle normally activated has passed into the resting electrical state, negative cavity potentials no longer exist. Hence, in direct unipolar leads taken upon the epicardial surface of an island of ventricular muscle that is activated later, striking changes may be expected by the time the negativity of the adjacent ventricular cavity decreases or disappears. The size of the positive deflections in the epicardial leads in canine right or left parietal focal block may be partially due to the disappearance of the negative potential in the ventricular cavities. However, the presence of a positive deflection in the opposite endocardial leads clearly indicates that in determining the form and size of these deflections the abnormal course of the excitatory process in the ventricular wall also plays an important role.

When the right parietal focal block begins (fig. 3), there is a delay in the activation of the ventricular wall beneath the electrode; hence the size of the R and S waves diminishes because the voltage developed across the explored ventricular wall is balancing the negative potentials of the ventricular cavity. The variations in the size and relation of the R and S waves that are important in the diagnosis of
right ventricular hypertrophy can, in this case, be ascribed to a minor degree of block. Striking changes may be observed in the form of the electrocardiograms while no definite prolongation of RS duration is noted. When right parietal focal block is more pronounced the explored ventricular wall becomes active even later, consequently the potential variations of the ventricular wall beneath the electrode are unbalanced and the late positive deflection rises above the base line. The foregoing analysis may explain the rSR' type of ventricular complexes not only in canine parietal focal block but also in cases of human right ventricular block.3

The QRS changes in the cavity electrocardiogram. The analysis of the electrocardiographic changes observed in man when ventricular blocks are progressing, suggests that in the involved ventricular walls the vector that represents the average direction of the excitation must be parallel or nearly parallel to the epicardial or endocardial surfaces.5

Under normal conditions, in human beings or in dogs, the outward spread of the excitation in the ventricular walls produces negative potentials in leads taken inside the ventricular cavities.3 When the parietal focal block is incipient the negative potentials diminish. In parietal focal block the activation of the rest of the heart muscle remains unchanged, consequently, variations in the magnitude of the cavity potentials solely can be ascribed to changes in the electrical activity of the ventricular wall where the block is progressing. In minor degrees of block the ventricular wall contributes less effectively or not at all to the production of either positive or negative potentials in the ventricular cavities. When the parietal focal block is severe a late positive deflection is recorded in the cavity lead, and the size of this deflection increases as the block increases. A similar positive deflection is simultaneously inscribed in the opposite epicardial lead. Positive potentials in two leads taken upon the opposite surfaces of the same ventricular wall suggest that the average direction of the electrical forces developed in the ventricular wall is parallel, or nearly so, to the epicardial or endocardial surfaces.2

Probably the spread of the excitatory process in the ventricular wall where a severe block occurs can be compared to the course of the excitation in the normal auricular muscle.2 The experiments of Pruitt, Essex and Burchell in isolated strips of heart muscle support this point.6 It was evident in all experiments that the positive upstroke in the cavity lead appeared only when the block was severe. The presence of a positive potential in the ventricular cavities can be considered a reliable index of the degree of block. It is important to mention that Sodi-Pallares, Estandia, Soberon and Rodriguez, using cavity leads, found a positive potential only when the human left ventricular block was definitely established.7 The electrocardiograms become normal when the velocity of the excitation wave increases and when the excitatory process spreads radially from the endocardium towards the epicardium.2

The changes in the T waves. In normal conditions the direction and contour of the T waves depend at least partially upon the relative duration of systole in the endocardial and in the epicardial layers.8 In parietal focal block the size and contour of the positive deflections representing depolarization can be correlated to the size and contour of the negative waves representing recovery. Considering the abnormal and slow course of the excitatory process in parietal focal block, it may be suspected that the muscular units that are activated first are first in recovering, and the muscle fibers that are activated later are also later in passing to resting electrical state.

Conclusions

The form and duration of the epicardial and endocardial electrocardiograms in the different types of ventricular block depend mainly upon the velocity and direction of the excitatory process in the ventricular walls. The tracings obtained when right or left parietal focal block is incipient are similar to those that can be considered representative of right or left ventricular hypertrophy. The electrocardiograms recorded when parietal focal block is pronounced resemble those of “incomplete” or “complete” bundle branch block. In direct or semidirect unipolar leads, when the ventricular
excitation is normal, the voltage developed across the ventricular wall and other characteristics may indicate the thickness of the explored heart muscle. It is also evident that under normal conditions the differences existing between the precordial leads exploring the right and left ventricles can be ascribed mainly to the different thicknesses of the underlying ventricular walls. Under pathologic conditions it is not known if the tracings considered indicative of right or left ventricular hypertrophy actually represent "conduction block," "fiber block" or both. However, notorious variations in the form of the electrocardiograms may be expected only if a ventricular block exists.

It follows that probably the designation bundle branch block is often but not always correctly applied. The experiments demonstrated that the tracings obtained in focal blocks can be mistaken for those observed in bundle branch blocks. It is probable that in human pathologic states "conduction block" can often be complicated by "fiber block." It may be presumed that "fiber block" has more definite clinical significance than "conduction block." Pure "conduction block" is compatible with a normal ventricular muscle, while "fiber block" indicates disturbances of the contractile muscle elements.

**Summary**

In ventricular blocks the form and the duration of the electrocardiograms can be ascribed mainly to changes in the velocity and direction of the excitatory process in the ventricular walls. Any delay in the passage of the impulse through the branches of the bundle of His, its subdivisions or the Purkinje network is defined as "conduction block." The delay of the excitatory process in the ordinary heart muscle is defined as "fiber block." "Mixed block" of these two categories also exists.

In cases of canine experimental focal block the form and other characteristics of the electrocardiograms resemble those tracings that in humans may be considered representative of right or left ventricular hypertrophy, or of "incomplete" or "complete" right or left bundle branch blocks.

In parietal focal block positive and broad deflections are recorded in leads taken on the epicardial and endocardial surfaces. In leads taken at opposite points separated only by the thickness of the ventricular wall, positive and broad deflections clearly indicate the average direction and velocity of the excitatory process in the ventricular wall where the block occurs.

The analysis of the QRS-T changes in experimental focal block helps explain the form of the epicardial and endocardial electrocardiograms in the different types of human ventricular blocks.

**SUMARIO ESPAÑOL**

Un método de producir bloque focal ventricular se describe. La secuencia de variaciones electrocardiográficas es ascrita a los cambios en la velocidad y dirección del proceso excitatorio en la pared ventricular. Los electrocardiogramas epicardiales se asemejan a aquellos considerados indicativos de hipertrofia ventricular o de bloque completo o incompleto del paquete de His. Cuando el bloque focal es pronunciado una defleción positiva aparece en el trazado cavitario. Los bloques ventriculares pueden ser subdivididos en bloques de conducción y en bloques de fibra, el primero producido por el retardamiento del impulso en el circuito de conducción especializado y el segundo producido por el retardamiento del proceso excitatorio en el músculo del miocardio.

**REFERENCES**

1. **Frau, G.** La pathogenie des troubles de la conduction ventriculaire. Cardiologia **19**: 2, 1951.


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