Ventricular Precontracting Area in the Wolff-Parkinson-White Syndrome Demonstration in Man

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By means of the analytic method of roentgenkymography of Cignolini, 11 typical cases of Wolff-Parkinson-White syndrome were studied. Comparison of the kymographic tracings with the synchronously registered electrocardiogram demonstrated precocious contraction of a limited ventricular area, situated in the left ventricle in the A type and in the right ventricle in the B type.

In the Wolff-Parkinson-White syndrome (W-P-W) the recognized causes of the pathognomonic electrocardiographic slow wave (the so-called delta wave) inserted between P and R are premature excitation of a ventricular area and an exceptionally slow transmyocardial conduction of the impulse, before it reaches the Purkinje network, and hence the whole of the myocardium. It has been experimentally verified that such conduction may occur in a peculiar way in a few limited ventricular areas localized in the arterial cone of the heart, and chiefly in the right or left ventricular area adjacent to the anterior segment of the interventricular septum, close to the base.

According to some authors, activation may originate in the septum; however, it diffuses first to one ventricle and then to the other. Following the conception of Rosenbaum et al., it would diffuse firstly to the left ventricle in the so-called A type (positive QRS in V1, V5, V2) and to the right one in the B type (negative QRS in V1, V5, V2).

Since investigative procedures employed to date (jugular phlebography, cardiac sphygmoscopy, roentgenkymography, electrokymography, etc.) have yielded uncertain and inconstant data on the mechanical effects of the ventricular pre-excitation phenomenon, a study employing Cignolini’s analytic method of roentgenkymography was undertaken. By this method it is possible to obtain detailed graphs that reflect the movement of almost...
every point of the cardiovascular profile and are always chronologically comparable among themselves together with synchronously registered electrocardiograms\textsuperscript{6,7} (figs. 1 and 2). Moreover, the single ventricular areas can be observed directly and not indirectly by means of the movements of the great vessels caused by them. This last method, though extensively utilized by many workers, is obviously incorrect, inasmuch as any given ventricular area may contract prematurely without causing a pressure gradient high enough to open the semilunar valves. This has been proved experimentally by Prinzmetal et al.;\textsuperscript{8} they stim-

![Diagram of the normal roentgenkymographic tracings compared with the electrocardiogram and phonocardiogram. Time intervals 0.02 second.](image)

**Fig. 2.** Diagram of the normal roentgenkymographic tracings compared with the electrocardiogram and phonocardiogram. Time intervals 0.02 second.

![Diagram of the W-P-W syndrome, A type. Note the position of the \(o\) point (beginning of the ventricular contraction) in the higher section (\(a\)) and in the lower section (\(b\)) of the left ventricle; \(o\) \(=\) beginning of the atrial contraction; \(z\) \(=\) protosystolic wave. Time intervals 0.02 second.](image)

**Fig. 3.** Case of W-P-W syndrome, A type. Note the position of the \(o\) point (beginning of the ventricular contraction) in the higher section (\(a\)) and in the lower section (\(b\)) of the left ventricle; \(o\) \(=\) beginning of the atrial contraction; \(z\) \(=\) protosystolic wave. Time intervals 0.02 second.

ulated the epicardial or endocardial surface of both ventricles electrically and produced the W-P-W pattern; a filmed record of the
contraction of the heart showed that in W-P-W systoles the mechanical activity of the heart was distinctive. Atrial contraction was normal, but it was not followed by the normal pause. On the contrary, ventricular activity followed immediately and 4 phases were rec-ognized: (1) premature contraction of the electrically activated myocardial area followed by a discontinuous diffusion of the contraction wave to the surrounding areas (no blood ejection from the ventricle takes place in this phase); (2) contraction of the remain-

Fig. 4. Another case of W-P-W syndrome, A type.

Fig. 5. Another case of W-P-W syndrome, A type.
The same case as that in figure 5, after the disappearance of the electrocardiographic abnormalities. The precontraction still persists.

ing ventricular myocardium, which occurred rapidly, as in normal systoles; (3) diastolic relaxation and protrusion of the precon-tracted area, while the remaining ventricular muscle was still contracted; (4) occurrence of diastole.

**Roentgenkymographic Studies**

In a series of typical cases, detailed roentgenkymographic tracings were performed. Apart from variations in general morphology corresponding to the different physical types of the patients, a peculiar abnormality was detected, which we believe to be almost pathognomonic of the syndrome (figs. 3-10).

This consisted of a very clearly premature onset of the ventricular contraction (so-called point \(c\)) in the higher section of the left ventricle in the A type and of the right ventricle in the B type. The significance of this finding was indicated by the fact that this \(c\) point was earlier, by .04 to over .08 second, than the corresponding one in the lower sections of the same ventricle (fig. 11). In con-
trast, the c point of the lower ventricular sections occurred about .03 second after the onset of QRS, as occurs in normal beats.

In other words, it is clear that the lower sections of the ventricles are not late in starting to contract. Contrariwise, the higher sections begin contracting much earlier, coincident with the abnormal delta wave on the electrocardiogram. In placing the c point, it should be kept in mind that the onset of systole, which is represented by the apex of the angle composed by the diastolic and systolic boundaries of the ventricular tracings, is closely followed by the protosystolic wave s.

This last, which is scarcely recognizable in the proximity of the cardiac apex, becomes more evident toward the base, where it often makes up the outer point of the whole tracing. It may also be noted that s wave, which is not the expression of a local contraction, but an effect of the whole ventricular systole, is rarely asynchronous in the different sections of the heart. Furthermore, the great vessels widen in a normal chronologic sequence after the ventricular contraction. The atrial waves also are normal in morphology and chronology. Every other roentgenkymographic component is quite normal.

In some cases a kymogram was recorded after the electrocardiographic abnormalities disappeared following intravenous procaine amide. In these tracings the precontraction was also clearly recorded (figs. 6, 8 and 10).
However, it was not always possible to demonstrate the ventricular precontraction area in all cases: this was possible in 8 of our 11 cases. This failure occurred because the area was exceedingly small or was situated in points scarcely controllable by the kymographic analysis, especially in the B type of W-P-W syndrome. Moreover, as already stated, the degree of precontraction may change (in our cases from .04 to .08 second), sometimes even in the same subject.

**DISCUSSION**

It may be concluded that the kymographic tracings clearly confirm the existence of a "precontraction area," situated in the arterial cone of the left ventricle in the W-P-W syndrome A type, and of the right ventricle in the B type. It seems, accordingly, that the arrival of a premature excitation at a limited area of the ventricular myocardium is definitely demonstrated also in man. The delta wave is the expression of the slow progression of the impulse from the pre-excited area to the adjacent ventricular myocardium. Despite the delay produced by this phenomenon, the excitation reaches the whole ventricular myocardium earlier than the impulse traveling along the Tawarian pathways (fig. 12). When the electrocardiogram does not show the typical abnormalities of the syndrome, it may be assumed that pre-excitation and precontraction still exist in a given ventricular area, but the excitation reaches the remaining ventricular myocardium by way of physiologic pathways and the impulse arising in the abnormal area is blocked.

In summary, it is evident that in W-P-W syndrome there exist 2 pathways of ventricular excitation. The anomalous pathway, of anatomic or simply functional nature, conducts the impulse faster than the normal one, so that the excitation reaches the ventricles earlier, and particularly an area located near the base of the right or left ventricle. While this area contracts the excitation passes to the remaining ventricular myocardium with a certain delay (delta wave), but always faster, however, than the normal impulse, traveling along the normal path-
VENTRICULAR PRECONTRACTING AREA

Studies carried out in the Wolff-Parkinson-White syndrome by means of the analytic roentgenokinography have shown for the first time in man a precocious contraction of a limited ventricular area, which is situated in proximity of the base, in the left ventricle in the A type and in the right ventricle in the B type. White syndrome, which, therefore, finds the ventricles already in a refractory state.

When, either spontaneously or by means of pharmacologic agents, the impulse arising from the pre-excited area is blocked, the ventricles are excited in the normal way; but the ventricular precontraction still persists.

Figs. 12. Diagram of the 2 atrioventricular conduction pathways in the WPW syndrome (presympathetic area).

REFERENCES


Ventricular Precontracting Area in the Wolff-Parkinson-White Syndrome: Demonstration in Man
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