Supraventricular Tachycardia with Left Aberrant Conduction due to Retrograde Invasion into the Left Bundle Branch

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SUMMARY
A description is given of a patient with an atrioventricular nodal tachycardia with left aberrant conduction changing into nonaberrant conduction after a ventricular premature beat. Functional left bundle-branch block by retrograde invasion into the left bundle branch is thought to be responsible for this phenomenon.

Additional Indexing Words:
Electrocardiogram  Nonaberrant conduction  Functional left bundle-branch block

During experimentally induced supraventricular tachycardia Moe and associates observed in the dog heart aberrant conduction in one of the bundle branches, caused by retrograde invasion into that bundle branch, causing functional bundle-branch block. As far as we know no clinical record has been reported. Recently we saw a patient with supraventricular tachycardia and left aberrant conduction, probably caused by this mechanism.

Report of Case
This 46-year-old man was seen outside our hospital by one of the members of our staff 30 min after the sudden onset of severe palpitations during a discussion of problems related to his business as wholesale dealer. The electrocardiogram to be discussed (figs. 1 and 2) was made at that time.

On admission the patient did not look ill. His blood pressure was 150/80 mm Hg. The pulse was regular, 220/min. Auscultation of the heart did not reveal any abnormalities apart from the rapid ventricular rate.

In the unipolar intra-atrial complexes (fig. 3), the regularly spaced QRS complexes (frequency, 220/min; QRS duration, 0.08 sec) were followed by P waves after 0.08 sec. The configuration of these P waves pointed to activation of the right atrium in a caudo-cranial direction (legend to fig. 3). A diagnosis of A-V nodal tachycardia with 1 to 1 retrograde atrial activation was made.

When the catheter was advanced into the right ventricle, a short burst of ventricular premature beats resulted, which terminated the tachycardia. The postarrhythmic ECG showed a regular sinus rhythm, rate 70/min, with a PQ interval of 0.16 sec. The QRS complexes were normal. There were no signs of preexcitation. The ST-T segment showed changes compatible with a post-tachycardia state. They had disappeared one day later.

Analysis of the ECG (Figs. 1 and 2)
Leads I to V1 show a regular tachycardia with a ventricular rate of 220/min. No P waves are seen. The QRS complex is 0.12 sec wide and has a complete left bundle-branch block configuration. Leads V3 to V6 show a regular tachycardia with a ventricular rate of 220/min. Again no definite P waves can be identified. The QRS duration now is 0.08 sec.

The interesting part of the ECG is lead V2 (fig. 2). On the left one sees a tachycardia (frequency, 220/min) with QRS complexes measuring 0.12 sec and showing a complete...
left bundle-branch block configuration. After a premature ventricular beat (arrow), the QRS width changes abruptly to 0.08 sec, the frequency of the tachycardia remaining exactly the same.

Discussion
Aberrant conduction is a regular finding in tachycardia of supraventricular origin. Sometimes at the start of the tachycardia a few aberrantly conducted complexes followed by nonaberrant conducted ones are present. In other cases aberrant conduction is present during the whole tachycardia. In the former situation the relation between the functional refractory periods of A-V node, His bundle, and bundle branches is such that the impulse arrives at the bundle branches when one of them is still refractory. The result is inability to pass that bundle branch leading to aberrant conduction.

Figure 1
ECG recorded 30 min after the onset of severe palpitations. Paper speed, 25 mm/sec. A regular tachycardia is seen with a ventricular rate of 220/min. Leads I to V₆ show complete left bundle-branch block complexes with a QRS width of 0.12 sec. No definite P waves are seen. Leads V₂ to V₆ show QRS complexes with a width of 0.08 sec. Again no P waves can be identified. The change from a QRS width of 0.12 sec to 0.08 sec takes place in lead V₂ after a ventricular premature beat (see fig. 2).
Figure 2

Lead V₆ at greater magnification. On the left QRS complexes with a width of 0.12 sec are seen. After a premature beat (marked by an arrow), the QRS width changes to 0.08 sec, the frequency of the tachycardia remaining the same throughout.

Figure 3

intracavitary lead right atrium
Supraventricular Tachycardia

Figure 4

Initiation, maintenance, and termination of left aberrant conduction during supraventricular tachycardia. (A) This shows how the first beat of a supraventricular tachycardia is unable to pass the left bundle branch, this branch being refractory. The impulse conducted through the right bundle branch travels through septal fibers to invade the left bundle branch in a retrograde direction. The frequency of the supraventricular tachycardia may be such that all following impulses coming from above reach the left bundle branch at the time when it has been made refractory by retrograde invasion from the preceding beat. Persistent left aberrant conduction results. (B) If the excitation pattern described in A is disturbed, for example, as shown here by a fortuitously timed ventricular premature beat, the left bundle branch is no longer refractory when the next supraventricular impulse arrives. Now at the same frequency of the supraventricular tachycardia nonaberrant conduction takes place.

The intra-atrial electrocardiogram is shown together with lead II. Paper speed, 50 mm/sec. The intra-atrial electrode was positioned here high in the right atrium. The P wave follows the QRS complex after 0.08 sec and is positive at this location; this suggests activation of the atrium in a caudo-cranial direction. This sequence is in accordance with the nodal origin of the tachycardia.
During the tachycardia, the functional refractory period of the bundle branch shortens relatively more than that of the A-V node and His bundle permitting, after a few beats, passage through that bundle branch again.\(^1\)

If aberrant complexes are present constantly during the tachycardia, it is presumed that the functional refractory period of one of the bundle branches does not shorten sufficiently during the tachycardia in relation to those in A-V node and His bundle. The impulse always arrives at the bundle branch when this is still refractory, resulting in persistence of aberrant conduction.

In 1965 Moe and associates\(^1\) however, suggested an alternate explanation for the presence of aberrantly conducted complexes during the supraventricular tachycardia, based upon beautifully executed experiments in the dog heart. They demonstrated that during regular driving of the dog heart either in the atrium or at the His bundle, a premature stimulus, given after an appropriate delay at the atrium or His bundle, reaches the bundle branches when one of them is still refractory. The impulse is then blocked in that bundle branch and travels through the other one, spreads over the septum and re-enters the blocked bundle branch from below to activate part of that bundle branch in a retrograde way. The next stimulus, given at a correct interval after the first premature one, travels down the His bundle to meet refractory tissue in the bundle branch that has been activated in a retrograde way by the former stimulus. Again aberrant conduction results. If the correct stimulus frequency is chosen, the sequence can be continued indefinitely, all QRS complexes showing the same aberrant conduction. Initiation and continuation of this type of aberrant conduction depends upon: (1) a correct delay between the last complex of the regular driven rhythm and the first premature stimulus, in order to have this stimulus arrive at the bundle branches, when one of them is still refractory; and (2) an appropriate interval between the following stimuli, which have to reach the bundle branch exactly at the time, when it is made refractory by retrograde invasion from the foregoing impulse.

It is understandable that in order to maintain aberrant conduction the stimulus interval named under point 2 lies between narrow limits. When this sequence of impulses is interrupted by a stimulus given too early, this pattern is broken immediately, resulting in disappearance of aberrant conduction.

Moe and associates\(^1\) pointed to the greater vulnerability in the dog heart of transmission failure in the right bundle branch. They showed however that repetitive aberrant conduction, due to retrograde invasion in the bundle branch, is also possible for the left bundle branch.

Our electrocardiogram shows that during the tachycardia at the same ventricular rate, both left aberrant and nonaberrant conduction take place. The ventricular rate in itself, is apparently not the only determinant of whether or not aberrant conduction results. The change from aberrant to nonaberrant conduction happens after a premature ventricular beat.

Following Moe's lines, we would like to suggest that in our patient, at the beginning of the tachycardia, the prematurely occurring first beat found the left bundle branch refractory and was conducted over the right bundle branch, invaded the septal muscle fibers, and activated the left bundle branch in a retrograde direction. We do not know at what level of the left bundle branch this took place. The second beat of the tachycardia arrived at the left bundle, after this was made refractory by the retrograde invasion into that bundle by the first beat. Again this second beat was conducted in a left aberrant way. The same goes for the third and consecutive beats of the tachycardia. As pointed out above, this special sequence of activation of the respective parts of the conduction system is essential for maintenance of the aberrant conduction. When this was interrupted by the ventricular premature beat, the left aberrant conduction disappeared immediately, revealing a supra-
ventricular tachycardia with "normal" QRS width. Figure 4 illustrates initiation, maintenance, and termination of the left aberrant conduction.

Reference


Atrial Fibrillation

The awakening was a sudden one. In the first week of October, 1909, a man came to my out-patient department at the City of London Hospital; he showed paroxysms of a peculiar form of tachycardia. It is not necessary to enter into a detailed description of the curves obtained from him at this and his subsequent attendances at University College Hospital. It will be sufficient if it is stated that I was confronted with a case in which I believed that a rhythm coming from the "node" was clearly demonstrable. It was a new method, namely the electro-cardiographic, which gave the facts to argue from. It was this patient who first convinced me that the heart irregularity of which I speak to-day could not be due to a "nodal rhythm", for the signs which he presented were totally different from those exhibited by the other patients. His attacks were of regular tachycardia, and it brought home the contrast, and emphasized the irregularity of the other condition as its leading quality. From the day on which I first saw this patient my interest was centred in the cause of the irregularity, so commonly seen in heart failure, the condition then attributed by others to "nodal rhythm." . . .

We may now go back to a paper written by Cushny and Edmunds. In 1906 these authors described the arterial curves obtained in dogs when the auricle was thrown into fibrillation, and they compared them with the curves of a patient in whom paroxysms of irregular tachycardia had occurred from time to time. They tentatively suggested that the irregularity might have had a similar origin in patient and dog. I believe that it was a remembrance of this paper, read some time before and forgotten, which finally led me to examine the irregularity of auricular fibrillation more carefully.

Comparing the electric curves in my possession, it immediately became obvious that discovery was not far away, and a further and larger series of jugular and electric curves were taken from a dog. My conviction on the subject dates from the experiment performed on this animal on November 11th, 1909. Having sufficient data I visited Dr. Mackenzie, for it was imperative that if the new view were to gather force I should win his assent. In taking venous curves from the dog I used the ordinary clinical polygraph, obtaining venous and radial curves, so that they had the appearance of clinical records. There were normal curves showing a waves, and curves from the fibrillating heart in which the venous pulse was of the ventricular form, and from which all sign of auricular contraction had vanished. I placed them before him without comment, and he stated his belief, as I had hoped, that they were from a patient with "nodal rhythm." From this moment my task was easy, for the electric curves which I had obtained were equally convincing.

It was in this manner that the auricular fibrillation theory won Dr. Mackenzie's support—support so generously given and so valuable, for it was withdrawn from the rival hypothesis, "nodal rhythm." But several points remain to be cleared. . . .

It was then that I published my preliminary communication. Almost immediately afterwards a paper by Rothberger and Winterburg came into my hands for the first time, and in it these writers also emphasized the similarity of the electric curves in the experimental and clinical conditions. The observations of Rothberger and Winterburg actually preceded my own, and were published in July, 1909; they have since been confirmed by a number of other workers.—Thomas Lewis: A Lecture on Evidences of Auricular Fibrillation, Treated Historically. Brit Med J 1:58, 1912.
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