Intraventricular trifascicular block

Observations on conduction disturbance in bundle-branch system

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A case of intraventricular trifascicular block secondary to an inferior myocardial infarction is reported. The case demonstrates instances of 2:1 atrioventricular conduction due to alternating block in the right bundle-branch and block in the right bundle and both anterior and posterior divisions of the left bundle (trifascicular block). Instances of 1:1 AV conduction with grade I block and periods of type I (Mobitz) block are ascribed to a delayed and a Wenckebach type of conduction respectively in the posterior division of the left bundle.

Recent recognition of the trifascicular nature of the bundle-branch system has changed some of the concepts about AV conduction disturbances and the pathways of impulse propagation in the ventricular myocardium. In a recent review by Rosenbaum et al. (1969) no more than 20 cases of intraventricular trifascicular block were found while the authors recognized the various combinations that may arise secondary to the different grades of conduction disturbance in the three fascicles of the bundle-branch system. The following report deals with a further case of intraventricular trifascicular block and presents certain features of interest where different grades of AV conduction disturbance are explained on the basis of variable dysfunction of the three fascicles of the bundle-branch system.

Case report
A 54-year-old man was admitted to Kasr El-Aini Faculty of Medicine of Cairo University on March 1960. The patient, who was known to have diabetes and hypertension for some time, began to complain of anginal attacks one week before admission. The attacks were frequent, not always related to exertion, and lasted from a few up to 15 minutes. On examination, he was in no apparent distress. His pulse showed frequent irregularities and his blood pressure was 150/100 mm Hg. Cardiac examination revealed no abnormality. Continuous cardiological monitoring for half an hour showed frequent shifts between 1:1 and 2:1 AV response with occasional instances of the Wenckebach conduction. The patient received sublingual tablets of isoprenaline every 3 hours but experienced two separate attacks of fainting sensation not mounting to actual syncope within 12 hours and suddenly expired 24 hours later.

Description of cardiogram (Fig.) The record reveals two different QRS patterns and three types of AV conduction disturbance.

The first QRS pattern (type A) is always preceded by a normal PR interval of 0.19 sec and has a duration of 0.12 sec. The pattern reveals right bundle-branch block (RBBB) in the chest leads and a frontal plane axis of -40° with ST-T changes suggestive of inferolateral injury. The second QRS pattern (type B) is always preceded by a prolonged PR interval of at least 0.25 sec and has practically the same duration of 0.12 sec. The pattern reveals more prominent S waves in leads II and III shifting the frontal plane axis to -80°, and significant changes in the praecordial leads especially lead V6 which shows loss of the initial Q wave and a more prominent S wave. The ST-T changes of inferolateral injury pattern are less prominent. The three types of AV conduction disturbance are as follows.

1. Periods of 2:1 AV block occurring at an atrial rate of 65–71 beats per minute. The conducted P wave has a PR interval of 0.19 sec and is always followed by type (A) QRS pattern.
2. Periods of 1:1 AV conduction seen to take place at critical slowing of the atrial rate to 60–65 beats per minute. These periods always start at a P wave followed after a PR interval of 0.19 sec by type (A) QRS pattern, while the next P waves are conducted with constant PR intervals of 0.25 sec and are followed by type (B) QRS pattern.
3. Periods of Wenckebach conduction usually in the form of 3:2 or 4:3 periods. During instances of 3:2 conduction, the first P wave is followed
after a PR interval of 0.19 sec by type (A) QRS pattern, while the second P wave is followed after a PR interval of 0.25 to 0.4 sec by type (B) QRS pattern and the third P wave is blocked. During 4:3 Wenckebach periods, the second P wave is always followed by type (B) QRS pattern after a PR interval of at least 0.25 sec, while the third P wave reveals further prolongation of the PR interval before block of the fourth P wave (see lead V6).

**Interpretation of cardiogram** Type (A) QRS pattern represents right bundle-branch block. The presence of left axis deviation of −40° in the frontal plane is, however, unusual. This can be explained either by involvement of the diaphragmatic surface of the heart in myocardial infarction or by the presence of an 'incomplete' block in the anterior division of the left bundle or both. However, for the rest of the report the pattern will be considered to represent 'pure' RBBB. This will make the discussion easier and will not interfere much with the validity of the interpretations. Type (B) QRS pattern reveals more conspicuous left axis deviation in the frontal plane and represents RBBB + 'complete' block in the anterior division of the left bundle. The recognition of these two QRS patterns facilitates the interpretation of the AV conduction disturbance. Though concomitant disturbance at the AV node-His bundle cannot be excluded, yet the evidence points to exclusive disturbance at the bundle-branch system.

During type (A) QRS pattern, the impulse reaches the ventricles through the two fascicles of the left bundle. On the other hand, during type (B) QRS pattern, the impulse is conducted through the posterior division of the left bundle alone. This pattern is always preceded by prolonged PR interval of at least 0.25 sec. This reveals that there is an conduction delay in the posterior division of the left bundle which becomes uncovered only by block of the conduction in the anterior division. The shorter PR interval of 0.19 sec preceding type (A) QRS pattern can now be explained by the relatively faster conduction through the anterior division.

During periods of 3:2 Wenckebach conduction, the first atrial impulse is conducted through both anterior and posterior divisions of the left bundle (though faster through the former division) and is blocked in the right bundle-branch alone (a monofascicular block). The second atrial impulse is conducted through the posterior division of the left bundle alone and is blocked in both the right bundle and the anterior division of the left bundle (a bifascicular block). The third atrial impulse shows complete AV block which is explained by failure of conduction in the three fascicles of the bundle-branch system (a trifascicular block).

During periods of 4:3 conduction the second and third atrial impulses reveal an increasing conduction delay through the posterior division of the left bundle before block of the fourth atrial beat. Lastly, instances of 2:1 block are explained by conduction through the two divisions of the left bundle alternating with a trifascicular block.

The observation that both 1:1 conduction in the posterior division of the left bundle and com-
plete block in the anterior division take place at a critical slowing of the atrial rate to 60 to 65 beats a minute is explained by the presence of a higher critical rate for conduction in the posterior division of the left bundle (60–65 beats per minute) and a slower critical rate for conduction in the anterior division. The latter probably lies somewhere between 35 beats a minute (the rate of the ventricular response during instances of 2:1 AV block) and 60 beats a minute (the slowest rate of ventricular response during 1:1 conduction in the posterior division).

Discussion

One or more of the fundamental patterns of AV conduction disturbance can be observed in the three fascicles of the bundle-branch system in the present case. The right bundle-branch shows a permanent third degree (complete) block while the posterior division of the left bundle shows both grade I block and type I (Mobitz) conduction. The anterior division of the left bundle may be revealing a type II (Mobitz) block. However, this postulate is difficult to substantiate since a fixed type of second degree AV block (2:1 periods in the present case) may be the result of a Wenckebach phenomenon and not a type II (Mobitz) block (Langendorf and Pick, 1968). On the other hand, the presence of normal PR interval preceding type (A) QRS pattern cannot exclude a significant conduction delay in the anterior division of the left bundle (though less severe than that of the posterior division). Apart, however, from this theoretical consideration this case reveals a higher degree of conduction disturbance in the right bundle, anterior division, and posterior division of the left bundle respectively, which agrees with their suggested vulnerability for conduction defects, which was based on both anatomical and physiological considerations (Rosenbaum, Elizari, and Lazzari, 1968).

The distinction between the functional nature of type I (Mobitz) block and the organic nature of type II block has been recently pointed out (Langendorf and Pick, 1968). Inferior infarction is usually associated with reversible ischaemic changes of the AV node-His bundle and as a rule leads to type I (Mobitz) block, while anteroseptal infarction associated with extensive lesion of the bifurcation or bundle-branches or both usually leads to type II block. Lepeschkin (1964) has theoretically related this observation to the greater difference in velocity between the slow conduction through the AV node and the rapid conduction through the bundle-branches. Thus a pathological prolongation of impulse conduction at the AV node will usually give rise to measurable change in the PR interval, while a constant delay simulating grade I block or an increasing delay simulating type I (Mobitz) block will not usually lead to measurable delay in the PR interval. In fact according to this consideration, Lepeschkin (1964) has suggested that analysis of AV conduction time may be helpful in locating the level of AV block in myocardial infarction. The demonstration of both grade I block and type I (Mobitz) conduction secondary to conduction disturbance in the posterior division of the left bundle in the presence of an inferior myocardial infarction will help to show that the above postulates are not absolute. The fact remains, however, that the distinction between the two types of AV block at the AV node-His bundle and the bundle-branch system is of major clinical repercussion in cases of acute myocardial infarction. The potential hazard of complete heart block and Adams-Stokes attacks in the latter situation usually entails a rapid interference through the use of an artificial pacemaker; a step that unfortunately was not undertaken in the present case.

References


