Wenckebach phenomenon within the atria

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'Blocks of varying degrees can be produced between different portions of the auricular muscle in the dog. . . .'

Drury, A. N. and Regnier, M. (1927-29)

'You see only what you look for - you recognize only what you know. . . .'

Sossman, N. C., from Schamroth (1971)

A Wenckebach type of conduction disturbance occurred between the stimulated (high right atrial) site and the recording (low right atrial or coronary sinus) electrodes in two patients. The pacing rates ranged between 158 and 194 a minute. This phenomenon (also observed by Lewis, Feil, and Stroud, 1918-1920) was manifested by a gradual prolongation of the stimulus-to-atrial electrogram intervals until one stimulus failed to reach the recording electrodes. The localization of the site of delay and block could be made exclusively from the intracardiac recordings, since the surface leads showed what appeared to have been only an AV nodal Wenckebach. In Case 2 the intra- and interatrial as well as the atrioventricular conduction patterns were studied during intermittent paired high right atrial pacing. Though this patient had complete right bundle-branch block and left anterior hemiblock, the major area of delay was in the atria. The full recovery period of the latter occurred at a longer St1-St2 interval than that of the AV node or His Purkinje system.

A previous report from our department stressed the importance of His bundle electrograms in the analysis of unusual forms of AV nodal Wenckebach block (Castillo, Maytin, and Castellanos, 1971). It is well known that this conduction disturbance can occur at any anatomical region or junction where some form of delay is actually, or potentially, present. The most frequently involved sites are the atrio-Hisian, sinoatrial, His ventricular, and ectopic-ventricular junctions (Schamroth, 1967). Rarely, it can also originate in the atria as will be shown in the present communication which also emphasizes the diagnostic value of intracardiac recordings and atrial pacing.

Material and methods

The technique used in our department to obtain His bundle, and local, atrial, or ventricular electrograms has been presented previously (Castellanos, Castillo, and Myerburg, 1971; Castellanos and Castillo, 1972). After explaining the procedure and obtaining consent, a tripolar catheter electrode¹ was introduced percutaneously through a

femoral vein and, under fluoroscopic control, positioned across the septal leaflet of the tricuspid valve. When properly placed, this lead records low right atrium, His bundle, and right ventricular inflow tract electrograms. The latter is usually distorted, possibly by tricuspid valve motion. Additional catheters with similar characteristics were introduced through an antecubital vein to record and stimulate from the high right atrium, and in Case 2 from the coronary sinus. Filtered (40-400 Hz) bipolar electrograms were recorded (simultaneously with surface leads) after connexion to a distribution switch box which, in turn, was attached to an Electronics for Medicine recorder. Paper speed was 100 mm/sec. Continuous or intermittent paired atrial stimulation was performed with a pacemaker which delivered slightly underdamped pulses 2.5 msec in duration and twice diastolic threshold value. During sinus rhythm and high RA pacing, the interval between the beginning of the P wave (or the emission of the spike) and the local low RA electrogram was taken as a measurement of intra-atrial conduction time (normal values in our laboratory: 20-40 msec). The interval between the sharp deflection of the low RA electrogram and the His bundle deflection was used as an indication of AV nodal conduction time (normal values: 50 to 120 msec). Conduction time through the His bundle and

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other parts of the ventricular specialized conducting system was given by the HV interval (normal values: 35–55 msec). Coexisting delay in the bundle-branches and their divisions was identified according to the characteristic QRS changes. Specific information regarding 2 patients referred for electrophysiological studies will be presented in the description of each individual case.

Case reports

Case 1 Fig. 1 was obtained from a patient with calcific disease of the aortic valve and syncopal attacks. During sinus rhythm the morphology of the P wave as well as the PR interval were within normal limits. High RA pacing was performed at a rate of 194 a minute and the electrical activity of the atria studied through catheters placed in the low RA and in the coronary sinus. The latter probably explored part of the left atrium. Pacing from the coronary sinus (not shown) resulted in negative P waves in leads I, II, and III. The most important measurements made from Fig. 1 are presented in Table 1. St1, St2, St3, and St4 represent 4 consecutive impulses delivered to the high RA. The numbers after the

FIG. 1 Case 1. Wenckebach phenomenon within the atria. The conduction disturbance appeared between the pacing site (in the high right atrium) and the recording electrodes at the low right atrium (LRA) and left atrium (LA). BAE=bipolar atrial electrogram from the high right atrium. HBE=His bundle electrogram. CS=coronary sinus. St=stimulus artefact. Numbers refer to successive impulses (and their responses) delivered at a rate of 194 a minute. In this and all records, paper speed was 100 minutes/second.

TABLE 1 Case 1: intervals (msec) measured during atrial pacing at a rate of 194/min

Atrioventricular					
Beat	Beat 2	Beat 3			
St1-LRA1 = 50	St2-LRA2 = 60	St3-LRA3 = 80			
LRA1-H1 = 85	LRA2-H2 = 105	LRA3-H3 = 105			
H1-V1 = 55	H2-V2 = 55	H3-V3 = 55			
St1-V1 = 190	St2-V2 = 220	St3-V3 = 240			
Intra- and interatrial					
St1-LRA1 = 50	St2-LRA2 = 60	St3-LRA3 = 80			
St1-LA1 = 100	St2-LA2 = 110	St3-LA3 = 130			

corresponding low RA, LA, and His bundle responses are used in reference to the stimulus which induced them. The first three spikes produced effective P waves which were conducted to the ventricles with increasing St-V intervals (190, 220, and 240 msec) and gradual shortening of the ventricular cycles (from 345 to 335). Finally, St4 failed to produce a P wave. In general, the surface electrocardiogram shows some features of an AV nodal Wenckebach phenomenon.

It should be noted that, whereas the increase in the St-V intervals from beat 1 to beat 2 occurred at the expense of both St-low RA and low RA-His bundle intervals, that between beats 2 and 3 resulted exclusively from a prolongation of the St-low RA interval. Moreover, the bottom part of Table 1 shows that these changes were associated with a gradual increase in the St-LA interval. The time elapsing between the inscription of the low RA and LA electrograms was the same in the three beats.

Though the findings in the case suggest a Wenckebach type of conduction disturbance occurring between the paced high RA site and the recording electrodes, no definite conclusions could be drawn regarding the area(s) where this phenomenon occurred. Nevertheless this must have been somewhere 'within' the atria, since both stimulation and recording were performed from different parts of these chambers.

Case 2 A 66-year-old patient with complete right bundle-branch block, left anterior hemiblock, and frequent extrasystoles was referred for intracardiac studies. During sinus rhythm (Fig. 2, left) the PR interval was within normal limits. AV nodal conduction time (low RA-His bundle interval) was also normal, but the HV interval was prolonged. In the presence of complete right bundle-branch block and left anterior hemiblock this indicates an associated conduction disturbance in the low His bundle, or in the left branch, or in its posterior division. High RA pacing at a rate of 100 a minute (Fig. 2, right) increased the St-V interval to 245 msec. Since the AV nodal and His-ventricular conduction times did not change, this delay was localized to the atria. In fact, the St-low RA interval measured 115 msec.

The effects of continuous high RA stimulation at a rate of 158 a minute can be seen in Fig. 3.

FIG. 2 Case 2. Intervals measured during sinus rhythm (left) and high right atrial pacing at a rate of 100/minute (right). Note that the second beat shows a conspicuous prolongation of the St-LRA interval (115 msec).

The most important measurements made from this Figure are presented in Table 2. There was a gradual prolongation of the St-V intervals from 245 msec (in beat 1) to 310 msec (in beat 6) which was due, exclusively, to a progressive increase of the St-low RA intervals from 105 to 170 msec. The low RA-His bundle intervals did not change during pacing. Though the HV interval increased from 60 msec (in sinus beats and after St1) to 70 msec after St2, it henceforward maintained the same value throughout St6. Finally, St7 was not followed by a response at the low RA.

In summary, the surface electrocardiogram showed what seems to be an AV nodal Wencke-

TABLE 2 Case 2: intervals (msec) measured during atrial pacing at a rate of 158 min

	Sinus	St1	St2	St3	St4	St5	St6	St7	St8
PR	160	—	—	—	—	—	—	—	—
PA	30	—	—	—	—	—	—	—	—
St-V	—	245	275	275	275	300	310	—	250
St-low RA	—	105	135	135	135	160	170	—	115
Low RA-H	70	70	70	70	70	70	70	—	70
HV	60	60	70	70	70	70	70	—	60

bach, in which failure of the impulse to reach the ventricles was not due to AV block, but to the absence of the expected P wave. Intracardiac recordings were essential to determine the exact nature of the conduction disturbance. The pacing-exposed intra-atrial block (seen with rates as low as 100/min: Fig. 2) was attributed to severe atrial disease. The latter obviously coexisted with complete right bundle-branch block and left anterior hemiblock.

In this patient, additional studies were performed with the specific purpose of evaluating the importance of atrial events in the analysis of total AV conduction. Intra-atrial, AV, nodal, and distal to His bundle conduction patterns were studied by means of driving stimuli (St1) delivered to the high RA at a rate of 100/min while the cycle was scanned (by St2) after every 8 driving impulse at 10 to 20 msec intervals. Fig. 4, which was constructed by plotting the St1-St2 intervals (abscissa) against the LRA1-LRA2, H1-H2, and V1-V2 intervals (ordinates), was used because it includes the contribution of the atria to the total delay in AV conduction (Castellanos *et al.*, in press). This graph also shows localization of the sites where conduction delay can occur (atria, AV node, and His-Purkinje system), delineating the mechanisms of these differences. For instance, Fig. 4 shows that the full recovery of atria occurred at a St1-St2 delay of 560 msec since the curves depicting the LRA1-LRA2 intervals deviated from the oblique line of identity (theoretical line of no delay) at an interval of 550 msec.

FIG. 3 Case 2. Wenckebach phenomenon within the atria. The conduction disturbance appeared between the pacing site at the high right atrium (HRA) and the low right atrial (LRA) electrogram recorded by the HBE lead.

FIG. 4 Case 2. Graphs relating the St_1 - St_2 intervals (abscissa) to the LRA_1 - LRA_2 , H_1 - H_2 , and V_1 - V_2 intervals (in the ordinates). In this type of graph, any deviation to the left of the oblique line (theoretical line of no delay) indicates that conduction is occurring through incompletely recovered tissues. In Fig. 4 to 6, the terms St_1 and St_2 will be used in reference to driving and testing stimuli, respectively. Driving rate was 100/minute.

From the latter to 490 msec the atrial curve descended to the left (above) this line. It coincided with the H_1 - H_2 and V_1 - V_2 , indicating, that in this part of the cycle, the additional delay in total AV conduction time was limited to the atria (Fig. 5, left). At St_1 - St_2 intervals ranging between 490 and 420 msec the LRA_1 - LRA_2 and

H_1 - H_2 curves were superimposed to the left of the oblique line, but the V_1 - V_2 values were longer (Fig. 5, right). This suggests that at these intervals the increase in AV time occurred in both atria and His-Purkinje system. At St_1 - St_2 intervals ranging between 410 and 330 msec the delays occurred in the three areas, as indicated by the separation of the corresponding curves, all of which were located to the left of the oblique line of identity.

The long St_2 - LRA_2 intervals in Fig. 6 suggest that the delay in total AV conduction was more significant at the atrial level. In other words, the St_2 - LRA_2 intervals were longer than the LRA_2 - H_2 or H_2 - V_2 intervals. The gradual prolongation of the St_2 - LRA_2 interval was proportional to the decrease of the St_1 - St_2 delays, proving that the LRA_2 deflections were indeed related to the corresponding testing impulses.

To summarize, the graph in Fig. 5 showed that though AV conduction could be impaired at all three levels, the major area of delay was located within the atria. This delay was significant enough to have given the AV node and ventricular specialized conduction system enough time to have recovered somewhat by the time the atrial impulse reached the AV node. Hence, it could not be determined whether, in the absence of atrial delay, the H_1 - H_2 and V_1 - V_2 intervals would have measured what they did at the shorter St_1 - St_2 intervals.

Discussion

Few articles have mentioned that a Wenckebach phenomenon could occur within the human atria. Conduction disturbances in these chambers are usually either partial (first degree) or complete (Schamroth, 1971). In 1967, Schamroth presented the tracing

FIG. 5 Case 2. At a St_1 - St_2 interval of 490 msec (left) the conduction disturbance occurred only at the atrial level (LRA_1 - LRA_2 , H_1 - H_2 , and V_1 - V_2 intervals (505 msec) longer than St_1 - St_2 interval (490 msec)). On the right the delay occurred both at the atrial level (LRA_1 - LRA_2 and H_1 - H_2 intervals (460 msec) longer than St_1 - St_2 intervals (450 msec)) and also at the H-V level (V_1 - V_2 interval (480 msec) longer than LRA_1 - LRA_2 and H_1 - H_2 intervals (460 msec)).

FIG. 6 Case 2. The progressive increase in the St_2 - LRA_2 intervals occurring at decreasing St_1 - St_2 intervals indicates that the LRA deflections were indeed related to the corresponding testing stimuli.

from a patient with atrial tachycardia and 7:6 block between the ectopic focus and the surrounding atrial myocardium. The PP intervals showed a gradual decrease until one P wave was blocked.

Scherf and Cohen (1964) commented on a case reported by Decherd, Ruskin, and Brindley in 1946. The electrocardiogram showed sinoatrial block and alternation between two peaks of a wide P wave. Both groups of authors postulated the following sequence of

events: the first sinus impulse failed to activate the atria, the second was delayed in one atrium only, and the third was delayed in both atria. Hence, the arrhythmia was considered to be a combination of sinoatrial and interatrial block.

Lewis *et al.* (1918-1920) performed several experiments in which they stimulated and recorded from the dog's superior vena cava, close to the right atria. In dog KQ (their Fig. 8) the pacing rate was 369 a minute. The stimulus-to-response intervals showed a gradual prolongation (from 37 msec to 140 msec) before the development of 2:1 block.

Different degrees of intra-atrial block, including Wenckebach phenomenon, were observed by Drury and Regnier (1927-29), Drury and Andrus (1924), and Lewis and Drury (1923) when strips of atrial muscle were cooled, subjected to pressure, or perfused with Locke's solution on the acid side of neutrality.

A Wenckebach phenomenon within the atria has also been observed by other authors who have used intracardiac and specialized conduction system recordings. Narula *et al.* (1971) noted that some patients with an abnormal control high RA-low RA (or what they call PA) intervals exhibited a progressive prolongation of this interval during atrial pacing. They postulated, as we did in the present study, that the sites of pacing and recording were constant throughout the recording period. Such a stability cannot be achieved in all cases, this being a valid objection which can be raised against the interpretation of our findings.

Yet, if a Wenckebach type of block can occur within the atria in experimental studies and in certain clinical conditions, there is no reason why it cannot also be present during atrial stimulation. The incidence of this conduction disturbance might be higher if the interpreter becomes aware of its existence. This is particularly liable to occur whenever atrial stimulation and recording are performed from various sites.

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