An atrionodal and nodo-Hisian gap phenomenon

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Two cases of the gap phenomenon are presented. The first was an example of a gap between the atrium and the AV node. In the second the gap was at the nodo-Hisian junction. At a critical H_1H_2 interval splitting of the premature His potential occurred. A decrease of the H_1H_2 interval led to the disappearance of the distal H_2 potential and to atrioventricular block. But at even shorter coupling intervals the H_1H_2 interval lengthened suddenly with resumption of the AV conduction. These observations provide further evidence that the gap phenomenon has a wider spectrum than was previously thought.

There is a curious phenomenon which will be observed rarely during programmed electrical stimulation studies of the right atrium when premature stimuli are introduced. By progressively moving the stimulus earlier in the atrial cycle a point is reached when the atrial response evoked is no longer conducted to the ventricles. Rarely a still more premature atrial extrasystole than that which was blocked is paradoxically followed by a ventricular response. These are the characteristic features of the so-called *gap phenomenon* (Moe, Mendez, and Han, 1965; Durrer, 1968; Goldreyer and Bigger, 1969; Wit *et al.*, 1970a; Gallagher *et al.*, 1973a, b; Agha *et al.*, 1973; Akhtar *et al.*, 1974a, b; Wu *et al.*, 1974). The explanation of it lies in the fact that the very early electrical impulse is delayed so long proximally to the original site of block that by the time it reaches that site the refractory period has been passed and conduction occurs (Wit *et al.*, 1970a; Gallagher *et al.*, 1973a, b). Depending on where in the proximal tissue the conduction delay takes place which allows atrioventricular conduction to be resumed, Gallagher *et al.* (1973a, b) distinguish arbitrarily between two types of gap. In the so-called type I the proximal region is the AV node, whereas in the type II it is the ventricular specialized conduction system.

Recently Wu *et al.* (1974) showed that this classification was an over simplification of the gap phenomenon and that it could also occur at other levels in the conducting system from the atria to the ventricles. The purpose of this paper is to present two cases which tend to reinforce the view of Wu *et al.* (1974).

Received 30 April 1975.

Patients and methods

After giving informed consent the two patients were studied in the non-sedated, post-absorptive state. Under fluoroscopic control two electrode catheters were introduced via the right femoral vein into the right heart. The first – a hexapolar catheter – was positioned against the high lateral wall of the right atrium. The distal two electrodes were used for stimulation and the next one or two for recording a uni- or bipolar high atrial electrogram. A second bipolar electrode catheter was positioned across the tricuspid valve to record the His bundle electrogram (HBE) at a filter setting of 100–270 cycles/s, thus utilizing a technique slightly modified from that described by Scherlag *et al.* (1969). Leads I, II, III, V₁, and the uni- or bipolar atrial electrogram (V_{RA} or B_{RA} respectively) were recorded simultaneously with the HBE either on an eight-channel photographic recorder (Hellige, Freiburg) or on an eight-channel direct-writing recorder (Siemens, Erlangen). All recordings were taken at a paper speed of 100 mm/s. The intervals between the atrial wave of the atrial electrogram (P) and the A wave of the HBE (A), the PA interval, and the A wave of the HBE and the His deflection (H), the AH interval, as well as the interval between the His deflection and the earliest time of ventricular activation on any lead (the HV interval) were measured.

The investigations were made by means of the extra-stimulus method (Wit *et al.*, 1970b). The right atrium was driven at about 5–10 beats/min faster than the sinus rhythm. The stimulation was by a specially designed pacemaker delivering premature or test stimuli (S₂) after every tenth basic driving beat (S₁). The premature beat interval (S₁S₂) was progressively shortened by 5–10 ms until the effective refractory period (ERP) of the right atrium was reached.

Results

Case 1

The patient, a 67-year-old woman, gave a history of

FIG. 1 Atrionodal gap (Case 1). V_{RA} is a unipolar atrial lead. First and second beats in both panels were produced by the driving and testing stimuli respectively. TOP: At an S_1S_2 interval of 340 ms S_2 is conducted to the atria without supplementary latency. BOTTOM: At an S_1S_2 interval of 320 ms S_2 is conducted to the atria with increasing latency, so that S_1S_2 interval is shorter than A_1A_2 interval.

dizzy spells. A His bundle study was performed to assess the functional state of the AV conduction system. The atria were driven at a rate of 79/min (cycle length 760 ms). The A_1A_2 interval was identical to the S_1S_2 interval up to the premature beat interval of 330 ms (Fig. 1). At an S_1S_2 interval of 320 ms or less the premature stimulus was conducted within the atria with progressive delay so that the S_1S_2 interval was increasingly shorter than the corresponding A_1A_2 interval (Fig. 1). Thus an S_1S_2 interval of 280 ms was accompanied by an A_1A_2 interval of 340 ms and the premature atrial response was still conducted to the ventricles (Fig. 2). But a further 10 ms shortening of the S_1S_2 interval with a corresponding A_1A_2 interval of 330 ms led to block of the premature impulse within the AV node (Fig. 2). The impulse was not conducted to the bundle of His. The effective refractory period of the AV node was thus reached. The coupling intervals S_1S_2 of 260 ms and 250 ms were accompanied by A_1A_2 intervals of 330 ms or less

FIG. 2 Case 1, same notation. TOP: AV conduction still persists at S_1S_2 interval of 280 ms. BOTTOM: Shortening of S_1S_2 interval to 270 ms with corresponding A_1A_2 interval of 330 ms. The ERP of the AV node is reached and A_2 blocked above His bundle.

maintaining the AV block. However, at a premature beat interval of 240 ms or less AV conduction was resumed (Fig. 3). These coupling intervals had a corresponding A_1A_2 interval of 350 ms or more, thus exceeding the actual duration of the effective refractory period of the AV node. The prolongation of the A_1A_2 interval is explained by the delayed conduction in the atrial tissue at coupling intervals shorter than 320 ms. At these coupling intervals the P wave recorded in the unipolar atrial electrogram (V_{RA}) appeared broad and distorted with no definite intrinsic deflection. In addition to this prolongation of the intra-atrial conduction time the latency between the stimulus artefact and the subsequent atrial wave slightly increased. The increased latency seemed to play a minor role in the prolongation of the S_2A_2 interval as compared to the slowed impulse propagation within the atrium itself.

The study was repeated at faster heart rates of 96 and 115 beats/min. At A_1A_2 intervals of 320 and 340 ms, respectively, the effective refractory period of the AV node was reached. The premature beat was blocked above the recording site of the bundle of His. At S_1S_2 intervals shorter than 310 and 280 ms

FIG. 3 Case 1, same notation. TOP: Shortening of S_1S_2 interval to 240 ms is followed by lengthening of A_1A_2 interval to 350 ms. This value is longer than the ERP of the AV node and AV conduction is thus resumed. BOTTOM: At S_1S_2 interval of 210 ms the ERP of the atrium is reached.

respectively the conduction delay between S_2 and A_2 became gradually longer. Yet until the time of the effective refractory period of the right atrium, which was 190 ms for both rates, the prolongation of the A_1A_2 interval was not long enough to exceed the duration of the effective refractory period of the AV node. A gap phenomenon therefore could not ensue.

Case 2

The patient, a 42-year-old woman, had bouts of supraventricular tachycardia which were later shown to be attacks of atrial fibrillation. The atria were driven at a rate of 90 beats/min (cycle length 680 ms). Until the A_1A_2 interval was shortened to 350 ms, with a corresponding H_1H_2 interval of 370 ms, the premature H_2 potential was conducted to the ventricles with an H_2V_2 interval of 35 ms (Fig. 4). At an A_1A_2 interval of 340 ms the H_1H_2 interval also shortened by 10 ms (Fig. 4). H_2 was now split into two distinct components (H_2 and H_2') with an increase of the H_2V_2 interval to 45 ms. A further shortening of the coupling intervals was followed by an increase of the splitting of the His potential. At the A_1A_2 interval of 320 ms with a

FIG. 4 Case 2. Gap at the nodo-Hisian junction. B_{RA} is a bipolar atrial lead. TOP: A_1A_2 interval of 350 ms, with a corresponding H_1H_2 interval of 370 ms, is accompanied by a normal configuration of the H_2 potential. BOTTOM: Shortening of A_1A_2 interval to 340 ms leads to H_1H_2 interval of 360 ms with splitting of the H_2 potential into two distinct deflections.

FIG. 5 Case 2, same notation. TOP: At A_1A_2 interval of 320 ms corresponding H_1H_2 interval reaches 345 ms, whereas splitting of H_2 potential increases. BOTTOM: At A_1A_2 interval of 310 ms, with corresponding H_1H_2 interval of 340 ms, second deflection of H_2 is lost, so an AV block ensues.

FIG. 6 Case 2, same notation. TOP: At A_1A_2 interval of 290 ms H_1H_2 interval increases to value of 410 ms, which restores normal AV conduction. BOTTOM: At S_1S_2 interval of 200 ms the ERP of the right atrium is reached.

corresponding H_1H_2 interval of 345 ms the duration between the two components of H_2 reached 30 ms (Fig. 5). At an H_1H_2 interval of 340 ms, produced by a shortening of the A_1A_2 interval to 310 and 300 ms, only the first minor deflection of the H_2 potential appeared (Fig. 5). This minor deflection could be constantly recorded after the A_2 potential.

A block between the two components of the H_2 potential was thus responsible for the failure of conduction of the atrial impulse to the ventricles. At a still shorter A_1A_2 interval of 290 ms the A_2H_2 interval increased from 110 to 190 ms, so that the corresponding H_1H_2 interval was now 410 ms (Fig. 6 (top) and Fig. 7). This was much longer than 345 ms, at which the block between H_2 and H'_2 had appeared, and longer also than 360 ms—the interval at which the fractionation of the H_2 potential into two deflections occurred. Thus the corresponding H_2 potential was of normal configuration and the ensuing H_2V_2 interval of normal duration. The shortening of the A_1A_2 interval to 280 and 270 ms maintained the AV conduction because of an H_1H_2 interval of 410 and 420 ms, respectively. Reducing the coupling intervals S_1S_2 to 260 ms or less led to A_1A_2 intervals of 270 or 280 ms with corresponding H_1H_2 intervals of 410 and 420 ms respectively. This occurred because of an increasing delay between S_2 and A_2 . The effective refractory period of the right atrium was reached at a S_1S_2 interval of 200 ms (Fig. 6).

FIG. 7 Gap at nodo-Hisian junction. A_1A_2 interval (on abscissa) is plotted against H_1H_2 interval (X) and the V_1V_2 interval (O) on ordinate. At H_1H_2 interval of 360 ms splitting of the H_2 potential occurs which became greater at shorter intervals. At H_1H_2 interval of 340 ms only the first H_2 deflection appears. Between an A_1A_2 interval of 300 and 290 ms the H_1H_2 interval suddenly increases from 340 to 410 ms with disappearance of H_2 splitting and of AV block.

When the atria were driven at a cycle length of 500 ms (rate 120/min) and the study repeated we were unable to reproduce the gap phenomenon. The functional refractory period (FRP) of the AV node – the shortest interval between two successive His bundle responses, both propagated from the atrium – was now at 360 ms but no splitting of H_2 or block was observed.

Discussion

The gap phenomenon can occur when there are two regions in the chain of the impulse transmission through the heart in which the following conditions are encountered (Wit *et al.*, 1970a; Gallagher *et al.*, 1973a, b; Wu *et al.*, 1974). (1) The functional refractory period of the proximal region is shorter than the effective refractory period of the distal one. This condition makes the occurrence of the block possible. (2) At shorter coupling intervals at which a block occurs a conduction delay ensues in the proximal region. Thus the impulse will be retained in this region so long that the distal one regains its excitability and restores the conduction to the ventricles. This explains the disappearance of the block at shorter coupling intervals.

A distinction has been made between type I and type II gap phenomena based on the localization of the proximal region of conduction delay (Galla-

gher *et al.*, 1975a, b; Agha *et al.*, 1973). In the type I gap it is the AV node, whereas in the type II gap the conduction delay is in the ventricular specialized conduction system. In both types of gap the distal region of block is in the ventricular specialized conduction system.

As Wu *et al.* (1974) showed, there are other possible sites of conduction delay in addition to the AV node (as in type I) and the ventricular specialized conduction system (as in type II). They are in the atrium and in the bundle of His itself. Wu *et al.* described a case in which the gap occurred between the His bundle as the proximal region and the ventricular specialized conduction system or a bundle-branch as the distal region. They therefore proposed that the previous classification should be replaced by one based on the proximal and distal regions involved in the gap phenomenon. Our first case is an example of a gap between the right atrium as the proximal region and the AV node as the distal region. Similar cases have been described not only by Wu *et al.* (1974) but also by Narula (1973), who named it 'pseudo-supernormal AV conduction.'

Neuss and Schlepper (1973) described a patient with WPW syndrome type A. At a given coupling interval during atrial stimulation the effective refractory period of the accessory pathway was reached and the conduction to the ventricles followed only through the AV node-His system. At shorter coupling intervals the intra-atrial conduction time was so prolonged that the impulse reached the accessory pathway out of the refractory period and the WPW pattern reappeared. This last case represents, in fact, a particular form of gap with the right atrium as the proximal region and the accessory pathway as the distal region.

Our second case is similar to Case 3 of Wu *et al.* (1974), in which there was a gap between the AV node and the His bundle. In contrast to our case the site of the block in their case was distal to the recording site of the second component of H_2 . Our case is unique in that the block occurred between the two deflections of the split H potential. The validity of a split H potential has been proved by means of simultaneous recordings of trans-membrane potential and a bipolar extracellular electrogram (Varghese *et al.*, 1973). The study showed a good correlation between the two forms of recording.

Most authors agree that splitting of the H potential represents a first degree intra-Hisian block (Narula *et al.*, 1971; Peuch *et al.*, 1972; El-Sherif *et al.*, 1974; Fleischmann and Pop, 1975). A second degree intra-Hisian block appears as an intermittent loss of the second component sometimes preceded by a prolongation of the interval between the two

components (Narula *et al.*, 1971; Peuch *et al.*, 1972). If the first component is too small to be recorded the block can be wrongly interpreted as occurring in the AV node and not in the His bundle. This was reported by El-Sherif *et al.* (1974). We think that if the recording catheter had been advanced a few millimetres into the right ventricle the first component of H_2 would have been lost and a nodal site of block would be postulated. Therefore a distinction between a nodal and a Hisian block will sometimes be difficult to make.

The resumption of AV conduction was accomplished in our second case by a sudden increase in the AH interval. Though the sudden increase of the AV nodal time may be interpreted as a manifestation of longitudinal AV nodal dissociation (Denes *et al.*, 1973; Denes, Wu, and Rosen, 1974) echo beats or bouts of supraventricular re-entrant tachycardia did not occur in our patient.

The effect of the driving rate on the occurrence of the gap phenomenon has been emphasized by Wit *et al.* (1970a). They showed that a gap between the AV node as the proximal region and the ventricular specialized conduction system as the distal region occurred more often at lower rates. This is explained by the influence of the driving rate on the refractory periods of the tissues involved. At lower rates the effective refractory period of the ventricular specialized conduction system is increased while the functional refractory period of the AV node will be shortened or sometimes lengthened. If there is a shortening of the functional refractory period of the AV node associated with a lengthening of the effective refractory period of the ventricular specialized conduction system a gap phenomenon occurs more easily with the lower rate. The same is true for the gap at the nodo-Hisian junction, and explains why in our second case the gap phenomenon was observed at a lower but not at a higher rate.

In our first patient also the gap occurred only at lower rates. Wu *et al.* (1974) predicted that a gap between the atria and the AV node would occur more easily at higher rates. Shorter cycle lengths tend to shorten the effective refractory period of the atrium and lengthen the effective refractory period of the AV node (Wit *et al.*, 1970a) and AV block will occur more easily. Yet, despite the opposing effects on the effective refractory periods of the atrium and the AV node, the increase in conduction delay at shorter coupling intervals between the test stimulus and the atrial deflection in the His bundle electrogram will not always be long enough to allow the passage of the impulse over the AV node to return.

References

- Agha, A. S., Castellanos, A., Wells, D., Ross, M. D., Befeler, B., and Myerburg, R. J. (1973). Type I, Type II, and Type III gaps in bundle-branch conduction. *Circulation*, **47**, 325.
- Akhtar, M., Damato, A. N., Batsford, W. P., Caracta, A. R., Vargas, G., and Lau, S. H. (1974a). Unmasking and conversion of gap phenomenon in the human heart. *Circulation*, **49**, 624.
- Akhtar, M., Damato, A. N., Caracta, A. R., Batsford, W. P., Josephson, M. A., and Lau, S. H. (1974b). Electrophysiologic effects of atropine on atrioventricular conduction studied by His bundle electrogram. *American Journal of Cardiology*, **33**, 333.
- Bharati, S., Lev, M., Wu, D., Denes, P., and Rosen, K. M. (1974). Pathophysiologic correlations in two cases of split His-bundle potentials. *Circulation*, **49**, 615.
- Denes, P., Wu, D., Dhingra, R. C., Chuquimia, R., and Rosen, K. M. (1973). Demonstration of dual A-V nodal pathways in patients with paroxysmal supraventricular tachycardia. *Circulation*, **48**, 549.
- Denes, P., Wu, D., and Rosen, K. M. (1974). Demonstration of dual A-V pathways in a patient with Lown-Ganong-Levine syndrome. *Chest*, **65**, 343.
- Durrer, D. (1968). Electrical aspects of human cardiac activity. A clinical-physiological approach to excitation and stimulation. *Cardiovascular Research*, **2**, 1.
- El-Sherif, N., Scherlag, B. J., Lazzara, R., Hope, R., Williams, D. O., and Samet, P. (1974). The pathophysiology of tachycardia-dependent paroxysmal atrioventricular block after acute myocardial ischemia: experimental and clinical observations. *Circulation*, **50**, 515.
- Fleischmann, D., Mathey, D., Bleifeld, W., Irnich, W., and Effert, S. (1973). His-Bündel-Elektrographie bei Patienten mit intraventrikulären Leitungsstörungen. *Klinische Wochenschrift*, **51**, 1066.
- Fleischmann, D., and Pop, T. (1976). Nachweis von Reizleitungsstörungen im Bündel mittels intrakardialer Ableitung. *Zeitschrift für Kardiologie*. In preparation.
- Gallagher, J. J., Damato, A. N., Caracta, A. R., Varghese, P. J., Josephson, M. E., and Lau, S. H. (1973a). Gap in A-V conduction in man: types I and II. *American Heart Journal*, **85**, 78.
- Gallagher, J. J., Damato, A. N., Varghese, P. J., Caracta, A. R., Josephson, M. E., and Lau, S. H. (1973b). Gap in A-V conduction in man. Type I and Type II. *Clinical Research*, **20**, 373.
- Goldreyer, B. N., and Bigger, J. T. (1969). Spontaneous and induced reentrant tachycardia. *Annals of Internal Medicine*, **70**, 87.
- Moe, G. K., Mendez, C., and Han, J. (1965). Aberrant A-V impulse propagation in the dog heart. A study of functional bundle branch block. *Circulation Research*, **16**, 261.
- Narula, O. S. (1973). Conduction disorders in the AV transmission system. In *Cardiac Arrhythmias*, p. 259. Ed. by L. S. Dreifus and W. Likoff. Grune and Stratton, New York and London.
- Narula, O. S., Scherlag, B. J., Samet, P., and Javier, R. P. (1971). Atrioventricular block: localization and classification by His bundle recordings. *American Journal of Medicine*, **50**, 146.
- Neuss, H., and Schlepper, M. (1973). Befunde zur super-normalen atrio-ventrikulären Erregungsleitung. *Zeitschrift für Kardiologie*, **62**, 267.
- Peuch, P., Grolleau-Raoux, R., Latour, H., Cabasson, J., Robin, J. M., Baissus, C., and Gilbert, M. (1972). Diagnostic des blocs tronculaires hisiens par l'enregistrement endocavitaire et la stimulation du faisceau de His. *Archives des Maladies du Cœur et des Vaisseaux*, **65**, 315.
- Scherlag, B. J., Lau, S. H., Helfant, R. H., Berkowitz, W. D., Stein, E., and Damato, A. N. (1969). Catheter technique for recording His bundle activity in man. *Circulation*, **39**, 13.
- Varghese, P. J., Elizari, M. V., Lau, S. H., and Damato, A. N. (1973). His bundle electrograms of dog. Correlation with intracellular recordings. *Circulation*, **48**, 753.
- Wit, A. L., Damato, A. N., Weiss, M. B., and Steiner, C. (1970a). Phenomenon of the gap in atrioventricular conduction in the human heart. *Circulation Research*, **27**, 679.
- Wit, A. L., Weiss, M. B., Berkowitz, W. D., Rosen, K. M., Steiner, C., and Damato, A. N. (1970b). Patterns of atrioventricular conduction in the human heart. *Circulation Research*, **27**, 345.
- Wu, D., Denes, P., Dhingra, R., and Rosen, K. M. (1974). Nature of the gap phenomenon in man. *Circulation Research*, **34**, 682.

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