Wenckebach Periods of Alternate Beats
Clinical and Experimental Observations

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SUMMARY

Wenckebach periods of alternate beats (AW) can be described as a 2:1 atrioventricular (A-V) block in which the conducted P waves show progressive prolongation of the P-R interval of the Wenckebach type. However, while classical Wenckebach periods terminate with a single blocked P wave, AW necessarily ends with (or begins from) two consecutive blocked P waves. Five clinical cases and several experimental examples of AW are reported. Recovery curves of A-V conduction were constructed, and it was demonstrated that AW is related to a marked prolongation of both the absolute and relative refractory periods. All the cases were associated with intraventricular block. In addition, recording of His bundle potentials in one case, histological study of the conduction system in another, and the experimental observations, support the view that AW tends to occur below the A-V node, in one of the main ventricular conducting fascicles. Four of the five patients developed complete heart block and Adams-Stokes seizures.

Additional Indexing Words:
- Infranodal block
- Refractoriness
- Concealed conduction
- Hemiblocks
- Bundle branch block
- 2:1 atrioventricular block

WENCKEBACH PERIODS of alternate beats (hereupon called alternating Wenckebach or AW) can be described as a 2:1 A-V block, in which the conducted P waves show a progressive prolongation of the P-R interval, until one is blocked and the cycle starts again. Thus, one of every two P waves is blocked, and the other P wave is conducted, giving rise, independently, to Wenckebach periods. However, while classical Wenckebach periods terminate with a single blocked P wave, AW necessarily ends with (or begins from) two consecutive blocked P waves.

In a series of clinical and experimental observations of AW, recovery curves of A-V conduction were constructed, showing that AW is related to a marked prolongation of refractoriness within the intraventricular conduction system.

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Material and Methods

Clinical Observations

Five clinical cases of AW were analyzed (table 1). In all cases, long tracings were recorded. P-R intervals of conducted P waves, as well as blocked P waves, were matched against the preceding R-P intervals, and recovery curves of A-V conduction were constructed.1-4 The terms absolute (ARP) and relative refractory period (RRP) were used as classically described.4 In every case, the type of intraventricular conduction was determined from the conventional ECG, according to known criteria.5, 6

Only in case 4 did the AW occur spontaneously. In the other cases, the original ECG showed 2:1 A-V block, and the AW was provoked by increasing the sinoatrial rate with 1 mg of atropine sulphate intravenously or by inhalation of amyl nitrate.

A case of second degree A-V block, in which partial penetration of some of the blocked P waves simulated the presence of AW, was also studied.

Experimental Observations

Mongrel dogs weighing 12 to 15 kg were anesthetized with intravenous sodium pentobarbital (30 mg/kg body wt). The bundle branches or main divisions of the left bundle branch were cut or injured according to techniques previously described.5 One fascicle or two were totally interrupted, while the remaining fascicle (hereafter called the critically injured fascicle or CIF) was slightly injured, in order to affect conduction in it only partially. This was produced by gently scratching or pressing down the fascicle with a blunt needle.
introduced through the ventricular wall. After initial injury, total block occurred in the CIF, but conduction went back to normal in a few minutes. At that moment, it was relatively easy to provoke bradycardia-dependent or phase 4 block in the CIF (unpublished observations). However, when the injury was repeated several times, it was easier to obtain AW. Commonly, this occurred toward the end of the experiment or when injury was present for a longer time, and this is consistent with observations in our laboratory indicating that prolonged refractoriness in injured fascicles needs a certain time to develop.

Since the other fascicles were previously interrupted, complete A-V block always developed initially when the CIF was injured. After this, 2:1 A-V block was commonly observed, in addition to the intraventricular block related to the totally interrupted fascicles. Thus, the ECG showed 2:1 A-V block plus left bundle branch block (LBBB), or right bundle branch block (RBBB), alone or combined with left anterior (LAH) or posterior (LPH) hemiblock. This indicated that the 2:1 block was occurring at the level of the CIF, and was confirmed by His bundle recordings (see below). At that time, AW occurred spontaneously, or was more commonly provoked by rapid atrial pacing. Not uncommonly, classical Wenckebach periods also occurred in the CIF. The AW was commonly unstable and short lasting, due to the fact that the CIF tended always to return to a normal conduction. Another lesion was then necessary to reproduce the AW, following the usual sequence of total A-V block, second degree A-V block plus BBB, etc.

His bundle recordings were taken according to a previously reported technique, and left bundle branch recordings were obtained with a special, still unpublished procedure developed by one of us (MVE), and which consisted essentially in "hooking" the fascicle with an electrode introduced through the right ventricular wall. This allowed a stable recording of the His and LBB potentials throughout all the maneuvering of long experiments. Not all the attempts were successful, and AW was provoked in approximately one of every three to four experiments. Five examples of AW were thus obtained.

Table 1

<table>
<thead>
<tr>
<th>Age</th>
<th>Diagnosis</th>
<th>Conduction disturbances</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4 years VSD closed spontaneously</td>
<td>2:1 A-V block</td>
<td>Died</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Complete A-V block</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>RBBB + intermittent LAH</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>44 years Chronic chagasic myocarditis</td>
<td>First degree A-V block + LBBB</td>
<td>Adams-Stokes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2:1 A-V block + RBBB + LAH</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>77 years Lenegre's or Lev's disease</td>
<td>High degree A-V block + RBBB + LAH</td>
<td>Adams-Stokes</td>
</tr>
<tr>
<td>4</td>
<td>3 days Complex congenital heart disease Levoposition</td>
<td>LAH</td>
<td>Died</td>
</tr>
<tr>
<td>5</td>
<td>78 years Lenegre's or Lev's disease</td>
<td>2:1 A-V block + RBBB</td>
<td>Adams-Stokes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Complete A-V block</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: VSD = ventricular septal defect; A-V = atrioventricular; RBBB = right bundle branch block; LAH = left anterior hemiblock; LBBB = left bundle branch block.

Figure 1

Case 1. Typical 4:3 AW. In the diagram, P-P, P-R and R-R intervals are indicated in hundredths of seconds. Each AW period is terminated by two blocked P waves.
WENCKEBACH PERIODS OF ALTERNATE BEATS

Results

Clinical Observations

A 4-year-old girl was found to have RBBB, intermittent LAH, and 2:1 A-V block (case 1). Later, she developed complete heart block with Adams-Stokes seizures and died at the age of 6. At autopsy, there was evidence of a high ventricular septal defect which had closed spontaneously, by apposition of the septal leaflet of the tricuspid valve. Related to this, there was extensive fibrosis in the region of the membranous septum, involving the branching portion of the His bundle and the origin of both bundle branches. The A-V node was free of lesions and the rest of the heart was normal. At the time when the ECG showed pure RBBB and 2:1 A-V block, typical AW was induced by amyl nitrate inhalation (fig. 1). Two 4:3 AW periods may be seen. In each, one of every two P waves is blocked, while the intercalated P waves are conducted with a progressive increase of the P-R interval, until two consecutive P waves are blocked. In the two cycles, the maximum increment in the P-R duration occurs not at the beginning but at the end of the period. Thus, the R-R intervals progressively increase, instead of decreasing as in classical Wenekebach periods. From the electrocardiographic and anatomic data, the AW was located at the level of the main LBB.

Figure 2 shows another example of AW, from a 44-year-old man with chronic Chagasic myocarditis (case 2). At the age of 41, an ECG showed first degree A-V block and LBBB. At the time of the study, the ECG showed RBBB with LAH and 2:1 A-V block. AW occurred after an i.v. injection of

atropine sulphate. Four AW periods are shown, each terminated by two consecutive blocked P waves. The RBBB with LAH, and the previous occurrence of LBBB, suggested that the AW took place in the main LBB, or its posterior division.

Figure 3 shows a His bundle recording from another case of AW (case 3). This was a 77-year-old man, with high grade A-V block and RBBB with LAH. The progressive prolongation of the P-R intervals was related to a prolongation of the H-V intervals, while the A-H intervals remained constant. The blocked P waves were all followed by an H, indicating the infranodal origin of the conduction defect.

False AW

Partial penetration of impulses or concealed conduction may cause prolongation of a succeeding P-R interval and eventually simulate AW, as shown in figure 4. This ECG was recorded in an 82-year-old man, whose basic tracing showed RBBB with intermittent LAH and LPH and variable P-R intervals. At first glance, the ECG looks like AW. However, long R-P intervals are followed by P-R intervals longer than those following shorter R-P intervals, indicating a lack of R-P/P-R relationship, which never occurs in true AW. In addition, the apparent AW does not end with two blocked P waves. Careful analysis showed that the varying P-R intervals were related to different degrees of penetration of the atrial impulses.

Experimental Observations

Figure 5 illustrates an experimental example of AW. The RBB and anterior division of the LBB

![Figure 2](image-url)

Case 2. The two strips are continuous. The upper strip shows a long AW period, followed in the lower strip by three 3:2 AW periods. Intervals, in hundredths of seconds.
were cut and the posterior division of the LBB was slightly injured. The ECG showed RBBB with LAH and 2:1 A-V block. When the atria were driven at a rate of 127 beats per min, 3:2 AW occurred. In each period, every other P wave was blocked below the H, whereas the intercalated P waves were conducted with an increase in the P-R interval at the expense of the H-V interval, with a normal and

Figure 3
Case 3. Leads V2, II and His bundle electrogram (HBE) simultaneously recorded. An AW period is illustrated. Every other P wave is blocked below the H (arrows), while the intercalated P waves are conducted with a progressive prolongation of the P-R interval, at the expense of the H-V interval. The A-H interval remains constant. The AW is terminated by two consecutive P waves blocked below the H, followed by a ventricular escape (E). The H-V intervals are indicated in milliseconds.

Figure 4
False AW periods. The two strips (lead V1) are discontinuous. The first QRS complex in both strips corresponds to a ventricular escape. All the other QRS complexes are of sinoatrial origin. The varying P-R intervals are related to different degrees of partial penetration of the "blocked" P waves, as illustrated in the diagram. Despite the fact that only one of every two P waves is conducted to the ventricles, there is not complete periodicity, and the false AW is not terminated by two consecutive blocked P waves, as in fig. 1 to 3. Intervals are indicated in hundredths of seconds.
Constant A-H interval. Terminating each AW period, two consecutive P waves were blocked below the H. The AW occurred at the level of the moderately injured posterior division of the LBB. Similar results were obtained in three other experiments.

**AW Caused by a Combination of Classical Wenckebach in the His Bundle and 2:1 Block in the Right Bundle Branch**

The features of the experiment shown in figure 6 were obtained only once, but may be important regarding the mechanism of AW. The LBB was cut and the RBB was slightly injured. The ECG showed then LBBB and 2:1 A-V block, which was located in the slightly injured RBB. However, at the very beginning of the experiment, the His bundle was also injured when it was hooked for electrographic recording. Later in the experiment, when the atria were driven at a rate of 142 beats per min, the tracings in figure 6 were obtained. The P waves were followed by a LB potential. However, only one of every two LB is followed by a QRS. All throughout, the LB-V interval was constant. At the same time, the A-LB space increased progressively in a Wenckebach fashion, until one P wave was not followed by a LB. At that moment, two consecutive P waves were blocked, one below the LB and the other above the LB, terminating the AW period. Thus, there is a Wenckebach block between atrial and LB activation, which is assumed to occur in the slightly injured His bundle (it would be unlikely to have nodal Wenckebach at this rate of stimulation), plus 2:1 block in the slightly injured RBB. This experiment shows that AW can result from two independent lesions: a higher lesion causing classical Wenckebach block, and lower lesion causing 2:1 block.

**The Recovery of A-V Impulse Conduction**

Figure 7 shows the recovery curves obtained from the five clinical cases and two of the canine experiments. In every case, there was a definite inverse R-P/P-R relationship. In each curve, three conduction ranges were identified: an early range during which P waves failed to be conducted to the ventricles; an intermediate range, during which the P-R intervals decreased more or less gradually; and a late range of normal or stable A-V conduction. With all possible limitations, these three ranges bear a relationship to the ARP, RRP and full
Experimental example of AW, due to classical Wenckebach periods in the His bundle, combined with 2:1 block in the RBB. Leads III, VI and left bundle electrogram (LBE) simultaneously recorded. The LBB was cut below the recording place, and the RBB and His bundle were slightly injured. The atra were driven at a rate of 142 beats/min. S = stimulus artifact; A = atrial deflection; LB = left bundle deflection; V = ventricular deflection. There is a progressive prolongation of the A-LBE intervals, until one A is not followed by a LBE, indicating a classical Wenckebach periodicity between atrial and LBB activation, which occurs within the slightly injured His bundle. At the same time, there is a 2:1 block between LBB and ventricular activation (note that only every other LBE is followed by a V), which occurs in the slightly injured RBB. The AW is terminated by two blocked P waves, one below the LBE, and one above the LBE. In the diagram, intervals are indicated in milliseconds.

Completion of recovery, respectively. Thus, AW seems to be related to a marked prolongation of refractoriness, at the expense of both the ARP and RRP.

Recovery curves of A-V conduction, from five clinical cases and two experimental examples of AW, based on the classical R-P/P-R relationship. P-R intervals (abscissa) and P-R intervals (ordinate), are indicated in hundredths of seconds. Note the marked prolongation of conduction times, reflected in the P-R interval, which can occur at the level of the ventricular conducting fascicles.
**Discussion**

**The Number of Lesions Causing AW**

AW may result from a single lesion causing an extreme prolongation of both the ARP and RRP, or may be the consequence of two independent lesions, one of which causes classical Wenckebach (which does not require such a marked prolongation of refractoriness), while the other lesion, downstream of the first, causes 2:1 block. In the latter case, the two lesions must operate in series and never in parallel, and the AW may occur more readily if the Wenckebach block is located upstream of the 2:1 block. However, AW may also occur if the 2:1 block is superior to the classical Wenckebach block; in such a case, prolongation of refractoriness in the lower lesion must be so great that AW may occur within the lower lesion itself, if the rate of stimulation is appropriate.

To determine whether AW results from a double lesion, a recording electrode must be positioned in between the two lesions. This is possible with a His bundle recording, if the upper lesion occurs in the A-V node and the lower lesion in a ventricular fascicle; or with an electrogram of the proximal part of the bundle branches, if the upper lesion is in the His bundle and the lower lesion in one of the bundle branches, as in fig. 6. However, if a double lesion occurs further down in a ventricular fascicle, it may be extremely difficult to differentiate this from the effect of a single lesion. Accordingly, in all the other clinical as well as experimental observations, it was impossible to determine whether the AW was due to a single or double lesion. Fortunately, a beautiful demonstration of AW apparently caused by a single lesion was provided by a case reported by Schuilenburg and Durrer.\(^9\)

The patient (case 2 in this report) was a 27-year-old man whose basic ECG showed RBBB and 2:1 A-V block. A His bundle recording demonstrated that the 2:1 block occurred in the His bundle, which showed a split deflection (H and H') with a constant A-H interval, and a 2:1 H-H' block. When the atrial rate was increased to 110 beats/min (by pacing), a typical AW occurred and, while every other atrial impulse was blocked between H and H', the intercalated impulses showed a typical Wenckebach progression also between H and H', until two consecutive H deflections failed to be followed by H'. The authors described this as an "H-H' Wenckebach period superimposed upon a 2:1 H-H' block." Among several possible interpretations, they favor explanation that the critically injured region was located within the His bundle, and the whole explanation was based on "the supposition that the zone of tissue with conduction impairment has a very long refractory period," which was actually confirmed by the application of atrial tests stimuli. This case lends further support to the interpretation presented in this paper regarding the existence of AW caused by a single lesion.

When referring to the "number of lesions" causing AW, it should be made clear that we imply the lesions strictly responsible for the physiologic changes determining the occurrence of the AW, hence, those occurring in the critically injured fascicle or fascicles. In most observations, clinical as well as experimental, interruptive lesions were also present in other conducting fascicles.

**The Location of the Lesions Causing AW**

Whether dealing with a single or double lesion, AW occurred most commonly at the intraventricular level. In all clinical cases, AW was associated with intraventricular block, RBBB in four cases and LAH in three. This suggested that the AW occurred most commonly at the level of the main LBB, or one of its divisions, and was verified histologically in case one, through a His bundle recording in case three, and of course, directly in the canine experiments. Thus, there is no question that AW is most commonly infranodal in location. However, this is not meant to imply that AW cannot occur in the A-V node, and some reported cases of atypical Wenckebach periods in atrial flutter and atrial tachycardia\(^10\)–\(^13\) probably represent examples of nodal AW.

**The Clinical Significance of AW**

The infranodal location of AW is of great academic and clinical importance. It is well known that classical Wenckebach periods occur predominantly in the A-V node, whereas Mobitz II block is invariably infranodal.\(^14\)–\(^16\) However, this rule has exceptions, and it has been proven, clinically as well as experimentally, that Wenckebach periods may occur in the His bundle,\(^16\) bundle branches,\(^17\)–\(^18\) or main divisions of the LBB.\(^19\)–\(^20\) Thus, it is not surprising that AW should occur below the A-V node. Although the reasons are unknown, the important clinical point is that AW carries a bad prognosis, insofar as A-V conduction is concerned. Thus, of the five clinical cases, four developed Adams-Stokes seizures and complete heart block. This lends further support to the contention\(^21\) that it is the anatomic location of the A-V conduction
problem that more basically determines the prognosis, and that A-V block related to intraventricular conduction system involvement is much more serious than A-V block related to nodal involvement. AW tends to occur in patients in whom one or two fascicles are totally interrupted, and in whom the remaining fascicle is the only one that can still transmit the impulse, albeit precariously. Under such conditions, implantation of an artificial pacemaker seems mandatory.

The Mechanism of AW

In the presence of a great prolongation of refactoriness, the occurrence of AW is related to the sinoatrial rate. For example, in the idealized diagram of fig. 8, in which an ARP lasts 0.90 sec and a RRP lasts 0.30 sec, with a normal rate between 70 and 90 beats/min (P-P intervals of 0.85 to 0.65 sec), a P wave will fall on the ARP and be blocked, while the next P wave will fall after recovery is completed and be normally conducted. The result will be 2:1 A-V block. However, if the rate is accelerated beyond 100, when a P wave will fall on the ARP and be blocked, the next P wave will fall on the RRP and will be conducted with a prolonged P-R interval. A third P wave will fall again on the ARP, but closer to the preceding QRS, and will again be blocked, whereas the fourth P wave will fall on the RRP also closer to the preceding QRS, and will be conducted with a P-R interval longer than that of the second P wave. The AW is thus set into motion, until one of the even P waves will finally hit the ARP and will also be blocked. At that moment, both odd and even P waves will be consecutively blocked, the following P wave will fall beyond the total recovery time, and the cycle will be repeated. However, it should be noted that, for the sake of simplicity and in order to fulfill a didactic purpose, both the effects of rate and prolonged conduction upon refactoriness, have been omitted from the diagram.

It is understandable that, in all cases, AW was observed when the sinoatrial rate was relatively rapid, either spontaneously or during tachycardia-inducing maneuvers. In the final analysis, AW is a particular form of 2:1 A-V block in which the AW only occurs at appropriately rapid rates. Of course, if the rate is extremely low, even 1:1 A-V conduction may occur; and if the rate is too rapid, perhaps one of every three P waves would give rise independently to Wenckebach periods (unpublished observations), or bizarre forms of second degree A-V block may develop.

References

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