A-V Conduction in Auricular Flutter

By M. Besoain-Santander, M.D., A. Pick, M.D. and R. Langendorf, M.D.

Flutter of the auricles may be associated with either a regular or an irregular ventricular rate. The former occurs as a rule in the untreated patient, the latter as a result of digitalis therapy. An attempt was made to explain the irregular ventricular response in auricular flutter as a result of a conduction disturbance similar to that seen in sinus rhythm with second degree A-V block characterized by progressive lengthening of the A-V conduction time preceding the dropped beat (Wenckebach phenomenon). Such an approach was suggested by the peculiar arrangement of the ventricular beats in some cases of auricular flutter. Similarly, in order to account for certain features of the arrhythmia it was assumed that some of the non-conducted flutter impulses penetrate into the A-V junction and thus influence the conduction of the subsequent flutter impulses.

In auricular flutter, the tissues of the A-V junction are exposed to impulses occurring in rapid succession, the rate of which greatly exceeds that of normal sinus impulses. Our understanding of the protective function of the A-V junction which reduces the number of such rapid impulses conveyed to the ventricles to a rate more economical for the heart, is based on the classical studies of Lewis\(^1\) on the law of the heart. However, only a few attempts have been made\(^2-5\) to analyze the complex mechanism of the conduction disturbance which develops in the A-V junction during auricular flutter. It would appear that A-V conduction during auricular flutter follows some definite rules rather than being simply a matter of chance. A study of auricular flutter was, therefore, undertaken both in cases with regular A-V conduction and those with clinical ventricular arrhythmia due to apparently irregular A-V block.

The observations were made on 27 patients, 14 male and 13 female, between the ages of 21 and 90. The clinical diagnosis was hypertensive and/or arteriosclerotic heart disease in 22 cases and rheumatic heart disease in the remaining 5 cases. Fifty-nine records were obtained during auricular flutter and additional records were available for comparison in 17 cases before the onset or after the termination of the auricular flutter.

A-V Conduction in Untreated Cases of Auricular Flutter

In table 1 the type of A-V conduction in the first record with auricular flutter is listed. Six cases were already under medication, 21 were untreated at the time. Prior to treatment, the

<table>
<thead>
<tr>
<th>A-V conduction</th>
<th>Untreated Cases</th>
<th>Cases already under treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1:1 conduction</td>
<td>1 (4.8%)</td>
<td>1</td>
</tr>
<tr>
<td>2:1 conduction</td>
<td>10 (47.6%)</td>
<td></td>
</tr>
<tr>
<td>Irregular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>with basic 2:1 conduction</td>
<td>4 (19%)</td>
<td>2</td>
</tr>
<tr>
<td>with alternation of 2:1 and</td>
<td>1 (4.8%)</td>
<td></td>
</tr>
<tr>
<td>4:1 conduction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Totally irregular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>with ventricular rate over 100</td>
<td>2 (9.5%)</td>
<td>1</td>
</tr>
<tr>
<td>with ventricular rate below 80</td>
<td>3 (14.3%)</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>6</td>
</tr>
</tbody>
</table>

prevailing finding was a regular 2:1 A-V ratio (47.6 per cent). In 19 per cent, a basic 2:1 conduction was found with occasional changes to 1:1 or 4:1 A-V conduction. In most cases, both with regular and irregular conduction, the
ventricular rate was over 100 per minute. In 3 untreated cases it was below 80. These were patients in the older age group with evidence of arteriosclerotic heart disease. Two of them showed the pattern of right bundle branch system block and one, in addition, showed signs of a remote myocardial infarct. It appeared that in these cases an A-V conduction defect became apparent with the onset of the rapid auricular rate.6 Persistent 1:1 A-V conduction was seen in two cases. The first, reported elsewhere,7 appeared to be paroxysmal in origin and the underlying mechanism became obvious only under carotid sinus pressure, producing a transient 2:1 A-V conduction. In the other instance, 1:1 A-V conduction was seen for a short time after conversion of auricular fibrillation to flutter by quinidine. The single case of table 1 showing alternating 2:1 and 4:1 conduction will be discussed later (see fig. 4B).

The Different Types of A-V Conduction in the Entire Group of Cases

As shown in table 2, our cases can be divided into (a) cases with regular ventricular action due to a constant A-V ratio and fixed relation of QRS complexes to a preceding F wave and (b) cases with irregular ventricular action, due to varying A-V conduction, characterized by a changing relation of QRS to the preceding F wave. A fixed F-R distance was assumed if the QRS complex appeared consistently at identical points on the flutter waves. In cases of complete A-V block a regular ventricular rate occurs with changing F-R distances. Transitions between groups (a) and (b) were seen, as in fig. 1, in which a rhythmic ventricular action with a constant F-R distance was occasionally interrupted by an irregular ventricular response.

(a) Fixed F-R Relation. A constant F-R interval would be expected in these cases; however, as is the rule in auricular flutter, the determination of exact values of the A-V conduction time (F-R interval) is difficult, for two reasons: first, the point on a flutter wave at which the impulse begins to travel towards the ventricles cannot be established2, 6; therefore, measurements of A-V conduction represent only approximate and relative values. For the sake of simplicity, this point in all of our records was assumed to coincide with the beginning of the ascending part of an F wave. As long as our criteria of the “beginning” of an F wave remain the same our conclusions based on relative values of A-V conduction remain valid. The other difficulty consists in the determination of which of the flutter waves, preceding a QRS, is conducted. Usually F waves are seen to coincide partially with a QRS complex and it seems improbable5, 6 that such an F wave is transmitted, since the distance (0.07-0.14 sec.) from its “beginning” to the onset of QRS appears too short. The alternative, prolonged A-V conduction of one of the preceding F waves, seems more likely.

A 2:1 A-V ratio was found to be the most common type of A-V conduction (35.6 per cent) in all analyzed tracings. For reasons given above, the time of A-V conduction was assumed to be 0.26 to 0.46 second. Similarly, in our cases of regular 4:1 conduction, the conduction time varied between 0.33 and 0.43 second assuming that A-V conduction is from the last completed
F wave preceding a QRS. If one assumes that conduction is from the second last completed F wave preceding QRS, the respective values in our cases would be 0.45 to 0.78 second.

Such marked delay of A-V conduction as compared with P-R intervals during sinus rhythm is found regularly in auricular flutter even if the ventricular rate is within the range seen in sinus tachycardia or normal sinus rhythm. To our knowledge, Wenckebach and Winterberg were the first\(^4\) to explain the long duration of the F-R interval by assuming some penetration into the A-V junction of the preceding auricular impulse. The non-conducted F impulses, by shortening the time for re-

\[\text{(b) Variable F-R Relation.}\]

Except in the presence of complete A-V block, these cases are associated with a more or less marked arrhythmia of the ventricles. This irregularity may be complete, similar to that seen in auricular fibrillation, or there may be an allorohythmic repetition of certain groups, or a fundamentally rhythmic action of the ventricles may be disturbed only occasionally by some irregularity. One such case is shown in fig. 1. A regular 2:1 A-V ratio, manifested by a regular sequence of QRS complexes with fixed F-R distances is disturbed in two instances. For reasons discussed above, the A-V conduction time of the regular beats is as-

\[\text{Fig. 1. The conventions in the diagram below this and the following records are those used customarily. A-V represents the spread of a flutter impulse through the A-V junction between the auricles (A) and the ventricles (V). Oblique lines at different angles indicate varying speed of impulse conduction through the A-V junction; the short lines at right angles to the oblique lines, blockage of the impulse. Varying lengths of oblique lines, representing blocked impulses indicate the varying distance to which the impulse penetrates into the A-V junction.}

\[\text{Lead III: Auricular flutter with regular 2:1 A-V conduction, disturbed by a single instance (eleventh QRS) of 1:1 conduction. Discussed in text.}\]

covery in that portion of the junctional tissues into which they penetrate, are responsible for the prolongation of the A-V conduction time of the subsequent flutter impulse. Further details of such concealed conduction\(^5\) in the A-V junction, as indicated in all of our diagrams, will be discussed later in connection with irregular conduction of auricular flutter waves.

Two cases with an apparently constant 3:1 ratio of auricular to ventricular activation showed a slight alternation of F-R distances varying between 0.35 and 0.41 second (or 0.57–0.65 sec. respectively), and were, therefore, listed in table 2 under group b. Conditions seemed to be present comparable with those discussed below in connection with figure 3A and B.
regular 2:1 conduction, as indicated in the diagram of fig. 1. The different F-R intervals bear an inverse relationship to the respective R-F distances, reflecting the incomplete recovery in the A-V junction, which is also responsible for the prolongation of the A-V conduction time during the part of the record with regular ventricular rate. In figure 2A a marked ventricular irregularity is seen. Most of the R-R intervals are slightly longer than two F-F intervals. The shorter R-R intervals a functional and constant A-V block high in the node reduced by half the number of auricular impulses crossing the A-V junction and another lower region of delayed conduction produced the progressive type of A-V block with (single) dropped beats.

In all records listed in table 2 as irregular, with a basic 2:1 or 4:1 A-V conduction, it was possible to establish an explanation similar to that shown for figure 2A. Another example is shown in figure 2B, where the long

tend to show progressive shortening preceding a long interval and the longer R-R intervals measure less than two short ones. Thus, the ventricular arrhythmia resembles that produced by a second degree A-V block of the Wenckebach type. Furthermore, as seen in typical Wenckebach periods, the R-R interval following the ventricular pause is found to be longer than that preceding the pause. To explain the whole mechanism of A-V conduction, it is assumed that all flutter impulses penetrate some distance into the upper part of the A-V conduction system and that only every second one is transmitted to the lower portion. Thus, R-R cycles are more frequent. Again it can be seen that these long R-R intervals are both shorter than the sum of two short R-R or four F-F intervals, while the short R-R cycles are longer than two F-F intervals. The assumed conditions of A-V conduction and the twofold block is shown diagrammatically below the tracing.

In some cases, the lower area of block gives rise to regularly recurring dropped beats, so that ventricular allorhythmia is produced (fig. 3A–D). Figure 3A represents a case of ventricular "bigeminy" during auricular flutter. Short intervals (0.74–0.75 sec.) alternate with
longer ones (0.80-0.81 sec.). The short R-R distances are longer than two F-F intervals, the longer R-R are shorter than four F-F intervals. Here, a 3:2 block of the Wenckebach type in the lower part of the A-V junction is assumed to be paired with a regular 2:1 response in the upper part. Penetration of apparently blocked flutter impulses into the latter region is indicated in the diagram to explain the prolonged A-V conduction time after relatively long ventricular intervals. However, since the sum of two consecutive R-R intervals

Fig. 3. Conventions as in figure 1. “Ventricular bigeminy” during auricular flutter. Note varying extent of penetration of non-conducted auricular impulses. Discussed in text. Compare with figures 6 and 7. (A) Lead II: 2:1 alternating with 4:1 A-V conduction. (B) Lead II: regular 3:1 A-V conduction with alternation of the F-R intervals of the conducted beats. (C) Lead II: 4:1 alternating with 6:1 A-V conduction. (D) Lead II: 2:1 alternating with 3:1 A-V conduction.
equals six F-F intervals, an alternative explanation, namely regular 3:1 block has to be considered and is shown below in an analogous case (fig. 3B). On this last assumption, a more complicated mechanism involving different degrees of penetration of blocked auricular impulses has to be postulated to account for alternation of the length of the F-R intervals. Such varying penetration could be expected to occur because of the varying cycle length preceding each attempt at conduction. Whereas this uneven (3:1) conduction ratio could not be excluded in two cases listed as such in table 2, it appeared less likely in others such as the case shown in figure 3A. In this patient the flutter attack started with a regular 2:1 conduction and, under treatment with digitalis, more and more instances of 4:1 conduction appeared until the latter ratio was finally established. The tracing of figure 3A was obtained during an intermediate period.

In figure 3C the basic mechanism is similar, but the degree of delay in A-V conduction is greater. The long R-R intervals (1.23–1.24 sec.) are shorter than six F-F intervals and the short R-R (0.97–0.98 sec.) longer than four F-F intervals. The total sum of a short plus a succeeding long interval equals ten F-F intervals. If we again assume a double region of block, every second auricular impulse would be blocked at the upper level and, of the remaining ones, alternately every second, then every third, would reach the ventricles. A detailed analysis of the tracing seems to confirm this assumption. It can be seen that following the first conducted impulse with a conduction time (F-R interval) of 0.42 second full conduction is not re-established with an R-F distance of 0.44 second (R-F distance of the fourth F wave) and only the sixth F wave (R-F distance 0.88 sec.) is transmitted to the ventricles with an F-R interval of 0.32 second. Due to the marked shortening of the latter, the following fourth F wave now occurs 0.54 second after a QRS, this interval permitting a ventricular response at an F-R interval of 0.43 second. Although these figures seem to be in close accordance with the fundamental laws of recovery and conduction, it should be realized that they represent only relative and average values—changes of the refractory period as a result of concealed A-V conduction are not taken into consideration. As will be discussed later, the true state of recovery does not appear to be identical at all levels of the A-V junction since the penetration of non-conducted flutter impulses is expected to be more or less inversely proportional to the preceding cycle length (fig. 6). It would appear that such a mechanism plays its part in the production of alternating 4:1 and 6:1 conduction in this case.

A somewhat different mechanism seemed to produce the allorhythmia present in tracing 3D. There too, an alternation of long and short R-R intervals is seen and the sum of two such consecutive R-R intervals equals five F-F intervals. This is the only case of our series where no other explanation than alternation of 2:1 and 3:1 A-V conduction could be established. The prolonged A-V conduction time of 0.30 second after an R-R interval of 0.60 second may be the consequence of concealed conduction of preceding auricular impulses. The failure of every second F wave following the QRS with the longer conduction time to be transmitted to the ventricles could be explained, as was figure 3C, either by a shortened recovery time or by a different degree of A-V penetration by the blocked flutter impulses. The latter is assumed in the diagram to this figure.

Alternation of the A-V conduction time, giving rise to ventricular bigeminy as presented in figure 3A–D, was seen in 9 of our cases. This allorhythmia was persistent throughout two of the records and disturbed by some other irregularity in the others.

In figure 4 there is illustrated a case listed in table 2 as almost complete A-V block. The auricular flutter rate is 260, the ventricular rate in Lead III is 52 with slight variations of R-R distances not exceeding 0.03 second. The persistent variations of F-R distances with a regular rate of both auricles and ventricles suggest the presence of complete A-V block and an idioventricular rhythm in these leads. However, in Lead V5, a single ventricular beat appearing prematurely (ventricular capture) interrupts the regular ventricular action.

Finally, in 11.8 per cent of the records (table
2) we were unable to find a definite and consistent relationship of auricular and ventricular activation. If in such cases the ventricular rate is slow and irregular and the F-R intervals vary throughout the record, one could consider the possibility of complete A-V block above the nodal pacemaker and a second degree A-V block with the Wenckebach phenomenon below it. Analogous cases with multiple regions of block in the A-V junction in the presence of auricular fibrillation have been reported from this department.9 The failure of a satisfactory analysis in some cases is, however, not surprising in view of the complexity of the factors, which determine conduction of flutter impulses viz. concealed conduction with different degree of penetration and the ensuing variations of the duration of refractory period and recovery time at different levels of the A-V junction.

A-V Conduction and Ventricular Premature Beats

In 6 of our records premature ventricular complexes of apparently ectopic origin were seen and their interference with A-V conduction of flutter impulses could be studied. In figure 5A a basic 2:1 A-V conduction with an F-R interval of 0.32 second is present. A ventricular premature beat interferes with the conduction of an F wave in the lower part of the A-V junction, but apparently not with the concealed conduction of the succeeding F wave, since the F-R interval following the compensatory pause remains prolonged (0.30 sec.).

In figures 5B and 5C, tracings of another case are shown taken on different days. The patient was treated with digitalis and a regular F-R ratio of 4:1 was established. In both tracings a premature beat occurring relatively late in diastole did not produce a "compensatory pause." Two possible explanations are indicated in the diagrams. In figure 5B the premature beat is considered to be of ectopic ventricular origin and interfering with A-V conduction. The prolongation of the subsequent F-R interval may be ascribed either to retrograde conduction of the premature beat or to the shorter R-F distance of the conducted flutter wave. This prolonged F-R distance, following the premature complex is not seen in figure 5C. An alternative explanation, viz. transient 3:2 block in the lower part of
the A-V junction with aberrant intraventricular conduction of the second flutter impulse, seems more adequate and is so indicated in the diagram.

![Diagram](image)

**Fig. 5.** Conventions as in figure 1. In addition, broken lines indicate aberrant conduction within the ventricles. Ventricular premature beats during auricular flutter. Discussed in text. (A) Lead III and (B) Lead V4: Bizarre ventricular complex of ectopic origin. (C) Lead III: Bizarre ventricular complex due to aberrant intraventricular conduction of a transmitted flutter impulse.

**Comment**

Our analysis of tracings of auricular flutter has been based on the fundamental laws valid for A-V conduction during sinus rhythm. However, some differences are to be expected in view of the rapid rate of auricular impulses and the longer refractory period of the A-V junction compared with that of the auricular muscle. Thus, a latent conduction defect may become apparent with auricular impulses occurring in rapid succession. Again, in cases of auricular flutter and complete A-V block, this block may disappear with the onset of sinus rhythm. The more effective action of digitalis on A-V conduction in auricular flutter and fibrillation may be explained in a similar way.

The usual A-V conduction found at the onset of auricular flutter is 2:1. This condition is a manifestation of interference with transmission due to the fact that the refractory period of the A-V junction is normally longer than the F-F interval. 1:1 conduction occurs only exceptionally. It may be the consequence...
of a relatively slow flutter rate of the auricles due to quinidine or of an abbreviation of the recovery time of A-V conduction such as seen in children or after exercise or emotion.

In our material it was found that cases with an initial 2:1 ventricular response in auricular flutter had, during sinus rhythm, a P-R interval of from 0.12 to 0.18 second. On the other hand, cases with first and second degree A-V block during sinus rhythm developed 4:1 block or Wenckebach periods with the onset of auricular flutter. It would, therefore, appear that a ratio of A-V conduction greater than 2:1 in auricular flutter can be considered as the first sign of a disturbance of A-V conduction, corresponding to P-R prolongation during sinus rhythm. However, sometimes the A-V conduction defect may be so slight as to become manifest only in auricular flutter. It has been demonstrated experimentally\(^2\) that in the dog under faradically maintained auricular fibrillation the ventricular rate is an index of A-V conductivity and a similar concept should be applicable to the human heart. Thus a ventricular rate over 100 per min. during auricular flutter should be considered normal. Most commonly, A-V conduction delay is caused by digitalis; it may, however, be due to some other cause, as in three of our cases with an initial ventricular rate of less than 80 per min.

The normal A-V conduction time (F-R interval) in auricular flutter with 2:1 conduction was found to be between 0.26 and 0.46 second. This marked and consistent prolongation has an adequate explanation in concealed conduction of the non-conducted alternate auricular impulses.

Abnormal A-V conduction in auricular flutter first appears as a transition of 2:1 to 4:1 ratio and only rarely in the form of 3:1 conduction. In view of this experience, Katz\(^2\) and Ashman and Hull\(^3\) suggested the existence of two levels of block in the A-V junction. The analysis of our cases with a ventricular arrhythmia confirms this assumption. It would appear that the upper region of A-V block represents a normal functional phenomenon associated with the rapid auricular rate, whereas the real block occurs in the lower region of the A-V conduction system. Consequently the latter, if constant, appears as a multiple of the functional A-V ratio of 2:1. Alternating 2:1 and 4:1 conduction can explain more adequately some cases (see fig. 3A) in which a 3:1 ratio has to be considered. A progressive Wenckebach type of A-V block during auricular flutter has been postulated by others\(^4, 6, 14, 15\) for this mechanism. The peculiar grouping of the ventricular complexes in some of our cases with irregular ventricular beating, as demonstrated in figure 2, also displays the typical structure of Wenckebach periods.

The occurrence of uneven conduction ratios during auricular flutter seems unquestionable and is shown in figures 3B and D. This fact does not necessarily exclude the presence of the assumed double region of block, if the effect of the length of the preceding cycle on the conduction of the subsequent impulse is taken into consideration. In their studies on A-V conduction in the mammalian heart, Lewis and Master\(^4\) have clearly demonstrated that the response to a stimulus may depend on the duration of the preceding cycle. Prolongation of this cycle is followed by a longer period of actual and apparent unresponsiveness, and the point at which recovery begins is shifted farther into diastole. However, the conducted or partially conducted beat, terminating this cycle, exerts an influence upon conduction not only of the first impulse that follows, but also of the second one. The diagram in figure 6 demonstrates how the conduction of impulse D is influenced by the degree of penetration of the non-conducted impulse B. The conduction of impulse D is determined (1) by the rest period (r) in the A-V junction between D and the preceding conducted impulse C and (2) by the length of the cycle preceding C (unshaded area). This cycle equals approximately the distance B-C in the upper portion of the A-V junction and the distance A-C in its lower portion. With deeper penetration of B as in figure 6B, the shorter cycle length (B-C) in the greater part of the A-V junction tends to enhance the conduction of impulse D by shortening the refractory period after impulse C. However, the opposite effect of impulse B on the conduction of impulse C shortens the
time of recovery between impulses C and D and thus inhibits the conduction of D. If the latter effect outbalances the former, impulse D fails to be conducted as an indirect result of deeper penetration of the non-conducted impulse B. And furthermore, of two impulses which are both apparently blocked due to a short rest period in the A-V junction, the one occurring after a shorter cycle penetrates deeper into the conduction system. Thus, different conditions of recovery are produced at different levels of the junctional tissues, the total effect of which becomes manifest as success, failure or prolonged duration of a subsequent conduction. It would appear that this conduction system. Lewis and Master have pointed out that deviations from the expected recovery curve occur when conduction is in a critical state as with the last conducted impulse preceding a dropped beat. Undoubtedly, variations in penetration of non-conducted impulses occur under similar circumstances and are responsible for variations in conduction of subsequent beats. This is clearly illustrated in figure 7, which represents a case with Wenckebach periods and concealed A-V conduction during sinus rhythm. Without appreciable change in the length of the preceding cycle, a non-conducted impulse (5th P wave) in figure 7A penetrates the A-V junction partially, as evidenced by the P-R prolongation of the subsequent beat, or penetrates more deeply (7B) and prevents conduction of the following beat, or (7C) becomes a conducted impulse. Finally, another factor which may also come into play in cases of auricular flutter with depressed A-V conduction is the supernormal phase of recovery which may likewise account for some unexplained variations of A-V conduction.

With a higher degree of conduction delay, an A-V ratio of 6:1 may be produced as in our figure 3C. A 5:1 ratio was not found among our cases, but may occur under conditions

- This case was previously published by H. K. Hellerstein as an instance of atrial infarction, proved at autopsy (Am. Heart J. 36: 422, 1948).

![Diagram](http://circ.ahajournals.org/)

**FIG. 6.** Schematic diagram to illustrate the influence of the extent of penetration of a non-conducted auricular impulse upon transmission of the next two successive impulses. Discussed in text. Conventions as in figure 1; in addition, A, B, C and D are successive auricular impulses. The broken line (r) indicates the duration of the rest period in the A-V junction. The shaded areas indicate the extent of shortening of a cycle by concealed A-V conduction.

complex mechanism is effective in cases of auricular flutter where no definite A-V ratio can be established (as in 11.8 per cent of our observations listed in table 2).

Actually, our assumption of a constant degree of penetration of the non-conducted impulses into the higher level of the A-V junction where the halving of the rate occurs is a simplification. We know that at this level too, varying penetration occurs which has been assumed to explain the alternation of short and long R-R intervals both in auricular flutter with 2:1 A-V conduction and in sinus rhythm with 2:1 A-V block.

Variations in cycle length are not the only factor controlling the degree of penetration of non-conducted auricular impulses into the conduction system.
discussed above. The next step would be 8:1 A-V conduction; however, the low ventricular rate produced with this ratio permits the appearance of nodal escapes which obscure the true A-V ratio. If finally the nodal beats become dominating, the pattern of complete A-V block characterized by regular ventricular rate and varying F-R intervals may appear in the tracing. In one instance of our series (figure occurring under digitalis medication, it may be of clinical importance to distinguish between extrasystolic ventricular bigeminy and aberration of intraventricular conduction of transmitted auricular impulses.

**Summary and Conclusions**

1. Fifty-nine electrocardiograms of auricular flutter obtained on 27 patients were analyzed

![Diagram](https://example.com/diagram.png)

**Fig. 7.** The effect of the extent of penetration of a non-conducted auricular impulse (fifth P wave) upon the conduction of a subsequent impulse (6th P wave) in a case of sinus rhythm with 2nd degree A-V block. Note that the varying penetration of the fifth auricular impulse does not depend upon the length of the preceding cycle. Conventions as in figure 1. (A) Lead CF, (B) Lead II, (C) Lead I.

4) incompleteness of the A-V block was manifested by occasional ventricular capture similar to that seen in sinus rhythm.

In figure 5 it was shown that premature beats with aberrant intraventricular conduction appearing during auricular flutter are not always of ectopic origin, but may be produced with A-V conduction if the refractory period of the ventricular muscle is prolonged due to a preceding long ventricular cycle. Thus a ventricular bigeminy can be imitated with variations of the ratio of A-V conduction. If this in order to determine the ratio of A-V conduction and study the factors controlling A-V conduction in auricular flutter.

2. In the untreated patient, an A-V ratio of 2:1 with a constant F-R interval and a regular ventricular rate was most common and a ventricular rate of over 100 was considered evidence of normal A-V conductivity. The 2:1 A-V ratio was explained as a phenomenon of physiological interference of the rapid auricular impulses and the relatively long refractory period of the A-V junction.
ratio with constant F-R interval or a transition from 2:1 to 4:1 ratio with varying F-R interval was seen most often after digitalization.

3. One-half of the records of auricular flutter with an irregular ventricular rate revealed an arrangement of the ventricular complexes in definite groups identical in structure with the Wenckebach periods observed in cases of sinus rhythm with second degree A-V block. Such records were interpreted as indicating a double region of block, with interference and halving of the auricular rate at the higher level of the A-V junction and block with the Wenckebach phenomenon at the lower, affecting only those impulses which passed the upper region of block.

4. The evidence for the double region of block in cases of auricular flutter with irregular ventricular conduction supports a like explanation for the predominance of even ratios of A-V conduction in auricular flutter.

5. The consistent finding of long A-V conduction times (F-R intervals) in auricular flutter (0.26-0.46 sec.) is ascribed to concealed conduction of non-conducted auricular impulses preceding the conducted ones.

6. Analysis of the records suggests varying extent of penetration of non-conducted auricular impulses as a result of variations of the refractory period of the junctional tissues; such varying concealed conduction is responsible for variations in conduction of the subsequent beats.

7. Penetration into the A-V junction of a non-conducted auricular impulse impairs the conduction of the next impulse and, by so doing, indirectly inhibits also the conduction of the second subsequent impulse; the latter effect is opposite to its simultaneous enhancing effect on the refractory period of this beat, brought about by shortening of the preceding cycle due to the concealed conduction.

8. The complex interplay of the various factors controlling A-V conduction and the unpredictable deviations occurring when conduction is in a critical condition due to the rapid stimulation, are responsible for the failure to unravel the mechanism of A-V conduction in a number of cases of auricular flutter with irregular ventricular response.

9. Premature beats of bizarre contour, appearing during auricular flutter, may be due to activation of an ectopic focus or to aberrant intraventricular conduction of transmitted flutter impulses. The latter interpretation should be considered, if the aberrant beat terminates a short cycle which follows a long ventricular pause.

ACKNOWLEDGMENT

The authors wish to express their gratitude to Dr. Louis N. Katz for his help and criticism.

REFERENCES


2 ——: The Mechanism and Graphic Registration of the Heart Beat, ed. 3. London, Shaw and Sons, 1925.


A-V Conduction in Auricular Flutter
M. BESOAIN-SANTANDER, A. PICK and R. LANGENDORF

Circulation. 1950;2:604-616
doi: 10.1161/01.CIR.2.4.604

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1950 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/2/4/604

An erratum has been published regarding this article. Please see the attached page for:
/content/3/6/x.2.full.pdf

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/
CONTRIBUTORS TO THIS ISSUE

Continued from page viii

FREDERICK OLMSTED, A.B.
Member, Assistant Staff, Research Division, Cleveland Clinic Foundation, Cleveland, Ohio

IRVINE H. PAGE, M.D.
Director of Research, Cleveland Clinic, Cleveland, Ohio

R. PICK, M.D.
Research Associate, Cardiovascular Department, Medical Research Institute, Michael Reese Hospital, Chicago, Ill.

LEON PORDY, M.D.
Research Assistant in Cardiology; Clinical Assistant, Cardiac Clinic, The Mount Sinai Hospital, New York, N. Y.

C. THORPE RAY, M.D.
Department of Medicine, Tulane University School of Medicine and Charity Hospital of Louisiana, New Orleans, La.

S. RODBARD, PH.D.
Assistant Director, Cardiovascular Department, Medical Research Institute, Michael Reese Hospital, Chicago, Ill.

MELVIN H. RODMAN, M.D.
Senior Resident in Medicine, Veterans Administration Hospital, West Roxbury, Mass.

CHARLES R. SHUMAN, M.D., M.S. (MED.)
Instructor in Medicine, Temple University School of Medicine and Hospital, Philadelphia, Pa.

MARVIN H. SLEISENGER, M.D.
Formerly Resident in Medicine, Beth Israel Hospital, Boston, Mass.; at present, Research Fellow, Department of Gastroenterology, University of Pennsylvania Hospital, Philadelphia, Pa.

EUGENE R. SNYDER, M.D.
Attending Otolaryngologist, The Mount Sinai Hospital, New York, N. Y.

WALTER SOMERVILLE, M.D.
Formerly Assistant in Medicine, Peter Bent Brigham Hospital and Harvard Medical School, Boston, Mass.; at present, Staff, Middlesex Hospital, London, England

JEREMIAH STAMLER, M.D.
Herbert G. Mayer Memorial Fellow; Research Associate, Cardiovascular Department, Medical Research Institute, Michael Reese Hospital, Chicago, Ill.

JOHN J. THORPE, M.D.
Instructor in Clinical Medicine, New York University Post-Graduate Medical School, New York, N. Y.

THEODORE B. VAN ITALLIE, M.D.
Resident in Medicine, St. Luke's Hospital, New York, N. Y.; now Research Fellow in Nutrition, School of Public Health, Harvard University, Boston, Mass.

WELDON J. WALKER, LT. COL., U. S. A.
Medical Corps, U. S. Army; formerly Assistant Chief, Medical Service, and Cardiologist, Gorgas Hospital, Ancon, Canal Zone; at present, Junior Associate in Medicine, Peter Bent Brigham Hospital, Boston, Mass.

JAMES A. WIER, LT. COL., U. S. A.
Medical Corps, U. S. Army; Senior Resident in Medicine, Gorgas Hospital, Ancon, Canal Zone

TRAVIS WINSOR, M.D.
Director, Nash Cardiovascular Foundation, and Staff Member, The Hospital of the Good Samaritan; Assistant Clinical Professor of Medicine, University of Southern California School of Medicine, Los Angeles, Calif.

JACOB ZATUCHNI, M.D., M.S. (MED.)
Instructor in Medicine, Temple University School of Medicine and Hospital, Philadelphia, Pa.

SAMUEL J. ZOECKLER, M.D.
Department of Internal Medicine, Veterans Administration Hospital, Des Moines, Iowa

CORRECTION: In the article entitled "The Metabolism of Lactate and Pyruvate in Children with Congenital Heart Disease" by Richard J. Havel and Elton Watkins, Jr. (Circulation 2: 536, 1950) the following footnote should have appeared: "This work was done under the auspices of a Life Insurance Medical Research Student Fellowship Grant to Dr. Havel."

In the article entitled "A-V Conduction in Auricular Flutter" by M. Besoain-Santander, A. Pick, and R. Langendorf (Circulation 2: 604, 1950), it was not clearly stated that Dr. M. Besoain-Santander was a Fellow of the Division of International Health of the U. S. Public Health Service.