Atrioventricular Synchronization and Accrochage

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Complete heart block implies an absolute independence between atria and ventricles that does not in fact always exist. Segers showed that, after complete block was artificially produced in the frog’s heart, atria and ventricles would sometimes begin to beat exactly in phase, most commonly in a 2 to 1 ratio. He subsequently reported one clinical example of 2 to 1 A-V synchronization in a patient with complete heart block. Two further cases that may illustrate different varieties of synchronization are here presented.

Atrioventricular synchronization has received scant attention in the English literature. The purpose of this communication is to re-emphasize this phenomenon and to present electrocardiographic records that may represent clinical examples of it.

Definition of Terms

When separate and distinct cellular elements, having no anatomic continuity and possessing inherently different rhythms, are placed in contact with each other, they sometimes begin to discharge impulses simultaneously at a common rate; when this occurs, it is logically called synchronization. Such synchronization has long been known to occur in juxtaposed nervous tissues; it has also been shown to develop in fragments of heart muscle or in whole cardiac chambers placed in contact; and when complete block is present, it may develop in the living heart between atria and ventricles.

If the “hook-up” between cardiac elements is short-lived, lasting for only a few beats, after which the separate elements resume their former independent rhythms, Segers' has applied the term accrochage (a hooking together). Thus there is overlap between the two terms, accrochage being merely a brief bout of synchronization.

It may here be emphasized that atrioventricular synchronization does not mean merely the synchronous or almost synchronous contraction of atria and ventricles—else the definition would embrace A-V nodal rhythm. The essence of synchronization is that atria and ventricles respond to their own separate pacemakers which are, however, beating synchronously—two individuals retain their separate autonomy, yet walk for a while in step.

Review of Literature

1. Synchronization between Cardiac Fragments

In 1946 Segers' reported that if two fragments of frog heart were placed in contact, they would often begin to contract exactly in phase. He found that for this to occur the rates of the 2 fragments must not differ by more than 25 per cent: the incidence of synchronization was indeed proportional to the difference in rates—at a 25 per cent difference it became a rare phenomenon, whereas when rates were within 1 or 2 per cent of each other, prolonged synchronization developed in approximately half his experiments. The rate at which the two fragments “synchronized” was equal to, or more often slightly less than, that of the more rapidly beating fragment; thus synchronization was primarily the result of an acceleration of the more slowly beating fragment with minor if any slowing of the more rapid fragment.

Synchronization was also observed to occur in a 2 to 1 ratio, especially when an atrial fragment was placed in contact with a ventricular fragment. This 2 to 1 type of synchronization occurred in more than half the experiments when the inherent rate of the ventricular fragment was between 45 and 53 per cent of the rate of the atrial fragment. More rarely 3 to
1, 4 to 1, or even 3 to 2 ratios were observed. The 3 to 2 type of synchronization, in which every third systole of the more rapid rhythm coincides with every second systole of the slower, is clearly a more complex interrelationship than the others, as it involves systoles in both fragments that are "out of phase." Attention is drawn to it because the second record here presented (fig. 5) may represent a clinical example of a similarly complex interrelation between atria and ventricles.

In Segers’ experiments, synchronization, when it occurred, lasted variously from a few seconds (accrochage) to 10 minutes or more. That it was due to the synchronous discharge of the same two previously independent pacemakers was clear from the fact that the action currents from each fragment remained unchanged in form.

(2) Atrioventricular Synchronization in Experimental Heart Block

Many years ago Erlanger and Blackman in the dog’s heart and Demoor and Rijlant in the rabbit’s had shown that artificially created complete atrioventricular block was sometimes associated with periods of what we may now call synchronization between atria and ventricles. Their records were entirely mechanical, however, and therefore gave no indication of the electric mechanism of the synchronous discharge. From their data it was entirely possible that atria and ventricles were under the control of a single intermediate pacemaker rather than responding to two separate but synchronized pacemakers.

Segers was able to produce periods of synchronization or accrochage in the frog’s heart in which he had produced complete block by destroying the conducting tissues. The usual A-V ratio obtained was 2 to 1, but at times 1 to 1 or 3 to 1 synchronisation developed. This led him and his colleagues to explore the possibility that similar interactions between atria and ventricles might occur in patients with complete A-V block.

(3) Clinical Atrioventricular Synchronization

In 1929 van Buchem commented “atria and ventricles frequently do not function so independently in complete heart block as one might be inclined to think.” He described a patient with complete A-V block in whom the intrinsic atrial rate was slower than that of the ventricles; whenever the P wave, having “overtaken” and passed the QRS complex, reached a distance of 0.19 second after the onset of the QRS, then the rate of the atria accelerated to become identical with that of the ventricles. This observation was not documented with a published tracing. Subsequently, Fischer and Kisch also stated, but again without publishing illustrative tracings, that the rhythms of atrial and ventricular pacemakers could synchronize during complete A-V block.

In 1947 Segers described a patient with complete block whose atrial rate was constantly double that of the ventricles. That this was not simply a 2 to 1 A-V block was clear from the facts: (1) the P waves varied in their relationship to the QRS complexes—sometimes appearing before, sometimes after, and sometimes coinciding with them—but always remained closely “hooked” to them; (2) exercise increased the atrial rate without appreciably influencing that of the ventricles.

The following cases illustrate two further possible forms of atrioventricular synchronization:

Case 1. A white male of 66 years was admitted to the hospital on December 27, 1954. He had enjoyed good health until 4 weeks before admission. At that time he developed a respiratory infection that was followed by progressive shortness of breath and chest pain. On admission he was cyanotic and dyspneic. The heart was regular at a rate of 130 and the blood pressure was 140/80. Rales were heard over both lung fields; the liver was not felt, and there was no edema. Blood urea was elevated and x-ray films of chest showed massive cardiac enlargement. A respiratory acidosis was reflected in carbon dioxide combining powers of 76 and 79 volumes per cent.

At first there was some response to measures directed at the congestive heart failure and pulmonary edema but on the seventeenth hospital day the patient suffered a cerebral accident with right hemiplegia. Two days later acute pulmonary edema developed but responded to treatment. Five days after this, on the twenty-fourth hospital day, another attack of pulmonary edema occurred. Once again the patient was rescued but after this he progressively deteriorated and died on Feb. 4, 1955.

Digitalis was administered as follows: during the
first three days in the hospital he received a total of 1.6 mg. of Cedilanid intramuscularly. During the next 14 days 0.2 mg. of Cedilanid was injected daily except on 2 days when a double dose was given. Oral gitalin was then administered for a further 16 days in a daily dose of 0.5 mg. except on 3 days when this dose was doubled. Thus, at the time of the tracing shown in figure 2, he had received in 33 days a total of 4.8 mg. of Cedilanid by intramuscular injection and 9.5 mg. of gitalin by mouth. Quinidine 0.2 Gm. every 6 hours was administered throughout most of the hospital course.

The salient features of serial electrocardiograms were as follows: 12/30/54: S-A tachycardia at 136 with nonspecific ST-T abnormalities; 1/20/55: Normal sinus rhythm with incomplete A-V block; 1/28/55: See figure 2 (discussed in detail below); 2/3/55: S-A tachycardia with complete A-V block; atrial rate 155, ventricular rate 120.

At autopsy the heart weighed 425 Gm. The left ventricle was dilated and its wall thickened. There was no gross evidence of coronary artery occlusion or of myocardial infarction. Microscopically, however, there were focal areas of fibrous tissue replacement in the wall of the left ventricle secondary to sclerotic changes in the arterioles. Sections taken from the septum in the region of the conducting tissues showed inflammatory cells, new vessel formation, and focal areas of fibrous tissue; these were considered to be evidence of a recent infarction, probably less than a month old.

The pattern observed in figures 1 and 2 would seem to admit of three conceivable interpretations. To dismiss the least acceptable first, since P waves in lead I were clearly visible in previous tracings and the QRST configuration was unchanged, a strip of lead I such as that shown in figure 1 would un-
doubtedly be interpreted as A-V nodal tachycardia of "mid-nodal" type. That this is probably not the mechanism becomes apparent when one sees P waves of sinus form emerging from time to time in what appear to be short runs of progressively increasing incomplete A-V block or of complete A-V block (fig. 2). It still remains a bare possibility that short bouts of sinus rhythm with A-V block alternate with runs of nodal tachycardia. This is rendered highly improbable, if not impossible, by the observed facts: (1) the ventricular rate, as soon as the P waves begin to coincide with the QRS complexes, becomes identical with the atrial rate as observed in the short stretches where P waves are visible, suggesting that the ventricles are now under atrial control. It is an almost inconceivable coincidence that, at the precise moment when the next sinus impulse is due, the A-V node should abruptly assume control at a rate identical with that of the S-A node; (2) that P waves of normal sinus form are seen partially submerged in the QRS complex, at the beginning (leads I, II and V1) and at the end (lead I) of the runs of supposed "nodal" rhythm. Moreover, it will be noted that in all the leads (except V1 where P is biphasic) the QRS complexes containing buried P waves are of greater amplitude than those situated between visible P waves. This suggests that the buried P waves add height (or depth in aVr) to the QRS complexes and, therefore, must be of the same polarity as the QRS. In every instance this polarity is consistent with normal sinus P waves rather than with waves of A-V nodal form.

Two more plausible possibilities suggest themselves, both of which invoke the mechanism of synchronization. One of these is that after short runs of independent rhythm, the atria and ventricles begin to synchronize. From the short runs of supposedly independent rhythm one sees that the ventricular rate is about 85 and the atrial rate 115. When synchronization occurs, as in Segers' experiments, the more slowly beating element accelerates to keep pace with the faster. If a diagram be made in Segers' mode, the A-V relationships supposed can be graphically shown as in figure 3.

![Figure 3](http://circ.ahajournals.org/)

**Fig. 3.** A-V relationships plotted (on the assumption of complete A-V block) from lead aVr, figure 2. The zero line represents the incidence of P waves and the black dots represent QRS complexes. Points plotted above the zero line thus represent successive "R-P intervals" (beginning with that of the first labeled P wave) before synchronization occurs; the fourth to the seventh beats represent synchronized P waves and QRS complexes; points plotted below the line represent succeeding "P-R intervals."

Against the argument for complete block with idioventricular rhythm is the fact that the ventricular rhythm, during the periods where P waves are apparent, is not regular; there is a progressive shortening of the R-R interval. Furthermore, if we accept complete A-V block, two obvious differences from the usual behavior of Segers' synchronizing tissues present themselves and perhaps deserve comment: (1) the intrinsic rhythms of the two pacemakers are rather widely separated, the ventricular rate (85) being only 74 per cent of the atrial (115); (2) during the periods of accrochage, synchronization is achieved exclusively through acceleration of the ventricles with no retarding of the atria.

The remaining and most attractive possibility is that the record shows only incomplete A-V block with P-R intervals progressively lengthening until synchronization occurs. Beginning with the first labeled P wave in lead aVr (fig. 2) we may measure successive P-R intervals as 0.26, 0.42, and 0.50 second. The next P wave is then lost in the QRS complex and the P-R interval is, therefore, equal to the R-R interval (0.54 sec.). For the next few beats the P-R interval remains equal to the
R-R interval, so that the P wave “belonging” to the next QRS complex remains concealed in the preceding QRS. If this is the correct interpretation, it represents synchronization of the atrial impulse with the ventricular impulse of the preceding cardiac cycle and the A-V relationships may be represented as in figure 4. If this explanation is acceptable, it is necessary to assume that the P-R interval occasionally exceeds the R-R interval. Thus the QRS immediately following the first labeled P wave in lead I (fig. 2) must represent the ventricular response to the P wave buried in the preceding QRS complex.

Case 2. A 49-year-old white man had his first known attack of rheumatic fever at the age of 38. At that time his physician told him that he must have had rheumatic heart disease for many years. For the past 10 years he has had mild exertional dyspnea. Three years ago the electrocardiogram showed right bundle branch block. One week before the tracing here presented (fig. 5) he “nearly fainted” at work—he felt weak, his heart pounded, and he became noticeably pale. During the next few days he had rather frequent spells of similar type; he lost consciousness in none of them. Since the time of this tracing, spells have become infrequent and a subsequent electrocardiogram has shown constant complete A-V block.

The tracing in figure 5 is included solely because it may represent the clinical counterpart of the unusual form of 3 to 2 synchronization observed by Segers in his animal experiments. In the tracing sinus rhythm at a rate of about 85 with incomplete A-V block alternates with complete block and idioventricular rhythm at a rate of about 58. The periods of sinus rhythm are associated with right bundle branch block, the pattern of which is identical with that constantly observed in the record a year previously. In the 12 lead electrocardiogram from which figure 5 is taken, the mechanisms rapidly alternated—the longest run of sinus rhythm observed being 16 beats and the longest of idioventricular rhythm, 15 beats. Each run of idioventricular rhythm began with an idioventricular QRS complex which coincided exactly with the expected time of the next P wave.

A “ventriculo-atrial” arrhythmia, of the type to which attention was first drawn by Erlanger and Blackman in 1910 and which has been the subject of many observations and discussions since, was also to be seen in the tracing during the bouts of complete block with idioventricular rhythm. This is clearly discernible in the strip reproduced in figure 5—the P-P intervals containing ventricular complexes are shorter than the non-QRS-containing P-P intervals.

**Mechanism of Synchronization.**

When independent cardiac fragments or chambers are placed in contact, it is obviously possible that the influences mutually exerted may be either mechanical or electric or both. Segers showed that the phenomenon could be mediated by electric influences alone; for he found that synchronization developed in heart fragments that were mechanically isolated and were connected only by a “bridge”
of electrolytes (Ringer's solution). But he also demonstrated that mechanical stimuli alone could induce synchronization; for he showed that, if two isolated ventricles were fixed on the 2 branches of an inverted Y-shaped cannula filled with a noneconducting medium (liquid paraffin), so that the contraction of either ventricle would displace paraffin into the other, thus raising the pressure within it, synchronization not infrequently developed.

Whether mechanical or electric stimuli play the decisive role in establishing atrioventricular synchronization in the presence of complete block remains to be determined; at present one can only speculate. What seems clear is that, in the presence of complete A-V block, all degrees of independence between atria and ventricles can exist. At one end of the scale is the classic concept of complete independence with each moiety beating regularly and being entirely uninfluenced by the other. At the other end of the scale comes prolonged atrioventricular synchronization. In between are shorter and less stable periods of synchronization (acrochage); and minor and often transient disturbances of one another’s rhythm, such as the ventriculo-atrial arrhythmia mentioned above the mechanism of which has remained a subject of hot dispute for decades.

Summary

1. The subject of synchronization (and acrochage) between independent cardiac elements is reviewed.
2. An example of A-V block, with short periods of presumable synchronization between atria and ventricles in a patient with recent myocardial infarction involving the septum, is presented. Possible interpretations of the electrocardiogram are discussed in detail.
3. A second patient with rheumatic heart disease, who has recently developed complete A-V block, presents a 3 to 2 A-V ratio that may represent a complex form of synchronization.
4. The mechanism of synchronization is briefly discussed.

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References

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