Principles Governing 2:1 AV Block with Interference Dissociation

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Second-degree AV block is characterized by an intermittent interruption of conduction at the AV junction. The block of a supraventricular impulse affords the AV junction a period of rest and recovery so that the following impulse can be conducted once again. The blocked impulse also results in a long ventricular pause which may enable the escape of a subsidiary AV nodal or ventricular pacemaker. If the escape interval is equal to, or shorter than, the conduction interval (an interval terminated by a conducted beat), interference and dissociation between the supraventricular and escape rhythms will occur. When this is associated with a 2:1 AV block, the escape occurs frequently, resulting in long periods of dissociation. The arrhythmia may mimic other forms of conduction disturbances, and though first described by Mobitz in 1923, few cases have been reported. Examples have been published by Dressler (1929), Schott (1946), Dressler, Roesler, and Specter (1952), Magri (1953), Katz and Pick (1956), Marriott, Schubart, and Bradley (1958), and Szigayi, Taran, and Richman (1961). Dressler and Jonas (1962) have also produced an analogous arrhythmia in cases of 2:1 AV block by the implantation of an artificial ventricular pacemaker; the rhythm resulting from such a pacemaker, however, could more properly be viewed as an artificial ventricular parasytole. Despite these reports, relatively little is known of this arrhythmia which is often mistaken for other arrhythmias. Katz (1946) published several such examples as “almost complete AV block”; and Berman (1955) misinterpreted the condition as a “reverse Wenckebach phenomenon”—a “mechanism” or term that is unacceptable under any circumstances.

The purpose of this communication is to re-appraise the principles governing this arrhythmia, and to describe and analyse its two basic manifestations: (1) the association with an AV nodal escape rhythm; and (2) the association with a ventricular escape rhythm.

CASE REPORTS

Case 1. The electrocardiogram (Fig. 1, a continuous tracing of lead V1) was recorded from a 56-year-old African (negro) woman with hypertension who was on maintenance digitalis. The second strip shows AV dissociation between normal sinus rhythm and an idioventricular escape rhythm. The P waves bear no relation to the QRS complexes which have a right bundle-branch block pattern. The PP intervals of the entire tracing range from 69 to 77*, representing a sinus rate of 78 to 87 a minute. The RR intervals of the ventricular escape rhythm range from 140 to 146. QRS complexes with a normal configuration are present in strip 1 (3rd QRS complex), strip 3 (5th QRS complex), strip 4 (last QRS complex), and strip 5 (first two QRS complexes). These represent conducted sinus impulses. QRS complexes are also present whose shape is in between that of the QRS complex resulting from the conducted sinus impulse and the QRS complex resulting from the ventricular escape rhythm (strip 1, second QRS complex: strip 3, fourth QRS complex). These are fusion complexes or incomplete ventricular capture beats, resulting from the concomitant activation of the ventricles by the sinus and ectopic ventricular impulses. This manifestation establishes the ventricular origin of the escape rhythm. The other sections of the tracing again show complete AV dissociation. The RP intervals which are followed by conducted beats range from 88 to 107. The associated PR intervals range from 37 to 54. The shorter the RP interval, within this range, the longer the ensuing PR interval. This reflects the partial refractory period or recovery of the AV junctional conducting tissue, and may be expressed in the form of a recovery curve—obtained by plotting each RP interval against its ensuing PR interval. The RP:PR relation in this case is such that the resulting conduction cycles always equal 141 (± 2). This reflects a recovery curve of precisely 45 degrees. This is a rare manifestation, since most cases have a recovery curve less than 45 degrees.

* All time intervals are expressed in hundredths of a second.
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**Case 2.** The electrocardiogram (Fig. 2, a continuous tracing of lead V1) was recorded from a 68-year-old African (Negro) man with calcific aortic valvular disease who was on maintenance digitalis therapy. The first strip shows AV dissociation between normal sinus rhythm and an idionodal escape rhythm; the P waves and QRS complexes are normal in shape and bear no relation to each other. The PP intervals range from 75 to 82, representing a sinus rate of 73 to 80 a minute. The idionodal escape rhythm is constant with an RR interval of 149, representing a rate of 40 beats a minute. This rhythm prevails throughout the tracing, with three exceptions. Three QRS complexes are inscribed prematurely, resulting in RR intervals of 120. This occurs in the second strip (4th QRS complex), 6th strip (3rd QRS complex), and 7th strip (4th QRS complex). Each of these prematurely inscribed QRS complexes is preceded by a P wave at a PR interval of 20. These QRS complexes represent conducted sinus impulses or capture beats—the momentary conduction to, and capture of, the ventricles by a supraventricular impulse. The AV nodal origin of the escape rhythm is established by the fact that the QRS complexes of the escape beats have the same shape and size as the QRS complexes of the conducted beats. Conduction of the sinus impulse to the ventricles is only possible when the P wave occurs during a critical period after a QRS complex, i.e. at a minimum RP interval of 98. All sinus impulses which occur with shorter RP intervals are blocked. There are no conducted sinus impulses with longer RP intervals, since the conducted sinus impulse penetrates into the AV nodal pacemaker and discharges it, thereby resetting the AV nodal cycle so that the following RP intervals occur within the refractory period of the conducted beat or the refractory period of the escape beat.

**DISCUSSION**

Interference-dissociation is a rhythm wherein two pacemakers concomitantly activate the heart; the...
conduction of their impulses is such that they meet and impede or interfere with each other's mutual progress. The term "interference" is thus used here in an electrical sense, and contrasts with the terminology used by Scherf (1926) who confines this term for a capture beat—the interference of one impulse with the rhythm of the other.

It should be emphasized that AV dissociation is never a primary diagnosis but merely connotes the presence of two pacemakers which concomitantly activate the heart. This may be brought about by a failure of one impulse to reach the domain of the other. This may, for example, be brought about by SA or AV block—AV dissociation by default; or it may be brought about by an enhancement of a subsidiary pacemaker, e.g. idionodal or idioventricular tachycardia. The mechanism in the two cases described above is one of default—a failure of the sinus impulse to arrive at the subsidiary pacemaker level, for it is the AV block which permits the escape of the subsidiary pacemaker.

The rhythm manifestation in simple interference-dissociation is dependent upon the rate-relation of the supraventricular and ventricular cycles, in other words, the relation between the sinus cycle and the AV nodal or ventricular cycle. When, however,
interference-dissociation complicates 2:1 AV block, the sinus cycle as such has little meaning or significance to the mechanism of interference, since the blocked impulse does not affect or interfere with the AV nodal or ventricular rhythm. It is every second sinus impulse that is pertinent to, and that may interfere with or usurp, the subsidiary rhythm. The effective or pertinent cycle is thus the interval represented by twice the sinus cycle or the conducted sinus cycle. The intersinus interval, which is effective at the level of a subsidiary pacemaker, has been termed the effective intersinus cycle (Scharroth and Dubb, 1965), or the effective atrial cycle (Marriott and Menendez, 1966). The effective intersinus interval may also be a conduction cycle, a cycle that is terminated by a conducted impulse but not necessarily begun with a conducted impulse.

The events immediately following the blocked impulse in 2:1 AV block are dependent upon the relation of the potential conduction cycle and the potential escape cycle. The following may arise.

1. The conduction cycle is shorter than the escape cycle. When this occurs, normal AV and intraventricular conduction of the sinus impulse will ensue.

2. The conduction cycle is longer than the escape cycle. When this occurs, the subsidiary pacemaker will gain control of the ventricles, and AV nodal or ventricular escape rhythm will ensue. This is usually accompanied by AV dissociation from the sinus impulse.

3. The conduction cycle is equal to the escape cycle. This will have the following effects. (a) In the case of a ventricular escape pacemaker, AV dissociation with ventricular fusion will ensue; (b) in the case of an AV nodal escape pacemaker, AV dissociation will ensue, but this simulates conducted sinus rhythm since the PR interval will be the same as the PR interval of a conducted sinus impulse (see below).

The aforementioned principles are evident in both the illustrated cases. The conduction cycle in Case I ranges from 138 (2 × 69) to 154 (2 × 78).

The escape cycle ranges from 140 to 146. These ranges overlap, and the opportunity consequently exists for either pacemaker to be dominant; this will depend upon which cycle is the shorter at any particular moment.

When the conduction cycle is shorter than the potential escape cycle, sinus rhythm with 2:1 AV block supervenes. This is evident at the end of strip 4 and the beginning of strip 5 in Fig. 1, where the conduction cycle ranges from 140 to 144; the escape cycle must therefore be longer than this. When the conduction cycle is equal to the escape cycle, or when it approximates the escape cycle within a narrow range (defined below), fusion complexes will occur. This is evident in strip 3 of Fig. 1 where the third escape cycle measures 140, and the conduction cycle measures 146. Fusion will still occur even if the escape cycle is shorter than the conduction cycle, provided that the difference does not exceed twice the duration of the QRS complex, or, more accurately, that the difference does not exceed the sum of anterograde and retrograde conduction times. This principle is illustrated in diagram II of Fig. 3—an idealized diagram constructed for equal anterograde and retrograde conduction times. The principle would, however, remain essentially the same if these times were unequal. The effective intersinus interval—or the conduction interval—is illustrated as S1–S3. The resulting RR interval is illustrated as R1–R2. Sinus impulse—S1—discharges the ectopic focus at E1. Sinus impulse—S2—is blocked. E2 is the point derived by measuring the duration of the QRS complex backwards from point X—the beginning of the QRS complex. E2–X thus represents retrograde conduction through the ventricles; X–R2 represents anterograde conduction through the ventricles. E3–R2 is equal to twice the QRS duration. If the ectopic cycle is equal to, or less than, the interval E1–E3, e.g. an interval illustrated as E1–E2, the ectopic impulse will anticipate the arrival of the sinus impulse and will consequently gain control of the ventricles. If the ectopic cycle terminates

![Diagrams illustrating the principles governing the development of AV dissociation in cases of 2:1 AV block. Diagram I—with an AV nodal pacemaker. Diagram II—with a ventricular pacemaker. See text.](http://heart.bmj.com/br-heart-j-first-published-as-10-1136-hrt-31-6-780-on-1-november-1969-downloaded-from-http://heart.bmj.com/)
during the interval E3-R2, e.g. at E4, ventricular fusion will occur, for it is during this narrow range that both impulses invade the ventricles simultaneously. If the ectopic cycle is longer than the interval E1-R2, it will be anticipated by the sinus impulse which will thus have complete control of the ventricles. It is thus evident that the ectopic cycle must be appreciably shorter than the sinus cycle to enable the ectopic pacemaker to gain complete control of the ventricles (compare the situation with an AV nodal pacemaker—see below).

The arrhythmic manifestation in Case 1 is further illustrated in diagram B of Fig. 4. The escape cycle is depicted as the interval E1-E2. With relatively short RP intervals, e.g. intervals E1-S1, or E1-S2, the sinus impulse is blocked since it encounters a refractory AV node. With a relatively longer RP interval, e.g. E1-S3, the sinus impulse (S3) encounters a relatively responsive AV node and is therefore conducted to the ventricles. The AV node is partially refractory since the PR interval is prolonged. The conduction cycle E1-S3 is shorter than the escape cycle E1-E2, and S3 therefore anticipates E2, effecting a complete capture of the ventricles. When the RP interval is still longer, e.g. E1-S4, the conduction cycle equals the escape cycle E1-E2. Impulses S4 and E2 thus invade the ventricles simultaneously, thereby recording a fusion complex. With still longer RP intervals, e.g. E1-S5, the conduction cycle becomes longer than the escape cycle. E2 therefore anticipates S5 at the ventricular level and AV dissociation occurs. It will be noted that there is only a small interval or "window" in the ectopic cycle during which conduction of the sinus impulse can occur. This "window" is represented by the interval S3-S4 (between the arrows). It is evident that if the potential conduction cycle (at ventricular level), viz. RP + PR is always longer than the escape cycle, dissociation will be permanent; and this is precisely the same manifestation presented by complete AV block. The principle is illustrated in diagram A of Fig. 4. E1-E2 is always shorter than E1-S3 + the potential PR interval of S3.

The differentiation of a conducted beat from an escape beat is relatively simple in the case of a subsidiary ventricular pacemaker (Case 1); the difference in QRS configuration makes such distinction easy. This difference is not present in the case of an AV nodal escape pacemaker. In this case, both the conducted sinus and AV nodal impulses give rise to identical QRS complexes, for ventricular fusion does not occur. This statement may with justification be challenged by the purist, for ventricular fusion complexes may occur between impulses of sinus and AV nodal origin if these impulses are conducted through different AV junctional pathways. However, this phenomenon is so rare that it does not detract significantly from the merit of the stated principle. This is evident in Case 2 where the only means of identifying the conducted sinus impulse is by virtue of its relative prematurity. The ectopic cycle is constant at 149. The conduction cycle (RR + PR) = 120. Whenever a sinus impulse occurs with an RP of 100 it encounters a responsive AV node and is conducted to the ventricles. The effective sinus cycle ranges from 148 to 164, and thus the AV nodal rhythm—whose cycle does not exceed 149—is usually dominant. The conducted sinus impulse discharges the AV nodal pacemaker during its passage through the AV node. The events following the blocked impulse will again depend upon the relation of the effective inter-sinus interval and the escape cycle. The following events are theoretically possible and are illustrated in diagram I of Fig. 3. The effective inter-sinus interval—the conduction cycle—is represented by the interval S1-S3. Sinus impulse—S1—during its passage through the AV node, discharges the AV nodal pacemaker (at E1). Sinus impulse—S2—is blocked. The subsequent events will depend upon the relation of the potential conduction cycle and the potential escape cycle. The following may arise.

1. The escape cycle (illustrated as E1-E4) is longer than the conduction cycle (illustrated as
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When this occurs, the sinus impulse anticipates the ectopic impulse, and a conducted sinus beat will ensue.

(2) The potential conduction cycle is longer than the ectopic cycle (illustrated as E1-E2). When this occurs the ectopic impulse anticipates the sinus impulse and usurps control of the ventricles (dotted line). Complete dissociation between the sinus and idioventricular rhythm ensues, and this simulates complete AV block precisely (Dressler et al., 1952). Indeed, the only definite clue in the whole tracing that this is not an example of complete AV block, is the presence of the three capture beats. This could easily be missed with the customary practice of recording only short strips of the 12 conventional leads. This manifestation further underlines the principle stressed by Marriott et al. (1958) that an absence of forward conduction is not necessarily evidence of complete AV block. It also explains how such unsatisfactory terminology as “almost complete AV block” (Katz, 1946) can be used for this arrhythmia.

(3) The conduction cycle is exactly equal to the ectopic cycle (illustrated by the interval E1-E3). When this occurs, it is impossible to tell whether the QRS complex results from a conducted sinus or an AV nodal impulse, for the associated PR interval will be the same for both the conducted sinus beat and the dissociated AV nodal beat. It will be noted that it is relatively easy for an AV nodal pacemaker to gain control of the ventricles; the ectopic cycle need only be fractionally shorter than the sinus cycle for the AV nodal pacemaker to assume control. The slightest abbreviation of the PR interval would indicate that the QRS complex is of AV nodal origin. This is in contrast to the situation with a ventricular pacemaker where there must be a relatively greater abbreviation of the ectopic cycle for the ventricular pacemaker to assume complete control of the ventricles (see above). Furthermore, irrespective of where the sinus impulse occurs during the AV nodal cycle, if the conduction cycle equals the escape cycle, temporary fortuitous synchronization will occur. This simulates conducted sinus rhythm and is evident in strips 3, 4, and 5 of Fig. 2 where the rhythm appears to be conducted sinus rhythm with 2:1 AV block and a prolonged PR interval. Analysis of the complete tracing, however, clearly indicates that AV dissociation is in fact present during this period.

Thus, it is evident that long recordings are necessary to facilitate the accurate analysis of this arrhythmia, especially so in the case of a subsidiary AV nodal pacemaker. When the ectopic rhythm is manifest (as described in (2) above) or when the distinction cannot be made (as described in (3) above), the underlying mechanism may be revealed by increasing the sinus rate—as with slight exercise. This will make the sinus rhythm dominant and unmask the true basic underlying mechanism of 2:1 AV block.

The aforementioned manifestations will only occur when ventricular or AV nodal escape complicates 2:1 AV block. When such escape complicates 3:2 AV block, the resulting arrhythmia will be an escape-capture bigeminy (Bradley and Marriott, 1958; Schamroth and Dubb, 1965).

The recognition of 2:1 AV block with interference dissociation has two important physiological and clinical implications. The accurate elucidation of the arrhythmia will reveal that conduction is still possible. The frequency and constancy of the escape indicates the adequacy of the “rescuing” subsidiary pacemaker.

SUMMARY

The principles governing 2:1 AV block with interference dissociation are analysed and described with respect to its two basic manifestations: (1) the association with an AV nodal escape rhythm, and (2) the association with a ventricular escape rhythm. The recognition of this arrhythmia has important physiological and clinical implications. The differentiation from complete AV block indicates that conduction is still possible. The frequency and constancy of the escape indicates the adequacy of the “rescuing” subsidiary pacemaker.

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REFERENCES


