CLINICAL PROGRESS

Interference Dissociation

By Ralph Miller, M.D., and Richard H. Sharrett, M.D.

INTERFERENCE dissociation is one of the most interesting arrhythmias. Although formerly considered a rarity, it is now recognized to be quite common; Katz and Pick, for example, found the surprisingly high incidence of 681 instances in a series of 50,000 consecutive patients. No doubt many cases escape detection simply because dissociation due to interference is usually transient, and by chance may not be recorded in a routine short tracing. However, all too frequently this arrhythmia is misdiagnosed, even by experienced electrocardiologists.

In the past, interference dissociation attracted little attention in clinical practice because it is essentially a benign disorder, insidious in onset, almost entirely asymptomatic, and of no great diagnostic or prognostic importance. In recent years, however, there has been a revival of interest in the arrhythmias in general, stimulated by advances in cardiovascular surgery and related fields. The complete electrocardiologist of today must be prepared to interpret correctly the most difficult arrhythmias, including all the complex variations of interference dissociation. For this reason we thought it would be timely to present a comprehensive as well as a detailed description of the electrocardiographic manifestations of interference dissociation.

Materials and Methods

Electrocardiograms obtained from 12 cases have been selected for analysis. These patients were observed either at the East Orange Veterans Administration Hospital, the Cardiac Clinic of the Newark Health Department, or in private practice. In some of the figures the cardiac mechanism is illustrated diagrammatically. The customary conventions are employed to indicate atrial and ventricular systole and atrioventricular conduction. The horizontal line through the atrioventricular (A-V) space indicates the level of the A-V nodal pacemaker, but no attempt is made to locate this position accurately. When P waves cannot be clearly seen because of superimposition upon QRS complexes, their position is set arbitrarily and related time intervals are roughly estimated. The unavoidable errors in timing that are thereby incurred are probably not significant. Time intervals are in hundredths of a second. P-R and R-P intervals are recorded when present.

The following discussion is based upon analysis of these electrocardiograms, experience with a large number of additional cases of interference dissociation and a review of the pertinent literature.

Nomenclature

In consulting the literature one is soon impressed by the obvious fact that there is no unanimity of usage of terms with reference to A-V nodal rhythm, A-V block, interference, A-V dissociation, interference dissociation and related phenomena. Standardization of the nomenclature for these arrhythmias is to be hoped for in the future. Meanwhile, the terminology adopted for our present purposes will be clarified when indicated.

A-V Nodal Rhythm

The term A-V nodal rhythm in general usage implies that the A-V node controls both the atria and ventricles. In this report, however, it will be used to indicate the presence of a consecutive series of A-V nodal impulses, whether these control the whole heart or only one set of chambers because of a conduction defect. No attempt is made to subdivide A-V nodal rhythm into upper, middle, and lower types. As was pointed out long ago, regardless of the location of the A-V nodal pacemaker, the electrocardiogram records merely the temporal relationship between the onset of atrial and ventricular depolarization. For example, the atria are activated before the ventricles when retrograde is shorter than antegrade conduction time, although both times may be normal, short, or prolonged. Therefore, the temporal relationship between P and R in A-V nodal rhythm is not analogous to the P-R...
TABLE 1. A-V Conduction Defects

<table>
<thead>
<tr>
<th>1.</th>
<th>Primary heart block (anatomic defects of A-V junction or abnormal refractory period)</th>
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<tbody>
<tr>
<td></td>
<td>A. First degree (delayed conduction)</td>
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<tr>
<td></td>
<td>1. antegrade</td>
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<td></td>
<td>2. retrograde</td>
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<td>3. bidirectional</td>
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<td>B. Second degree (incomplete block)</td>
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<td></td>
<td>1. antegrade</td>
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<td>2. retrograde</td>
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<td>3. bidirectional</td>
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<tr>
<td></td>
<td>C. Almost complete block</td>
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<td></td>
<td>1. antegrade or retrograde, with complete block in opposite direction</td>
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<td></td>
<td>2. bidirectional</td>
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<td></td>
<td>D. Complete (A-V dissociation)</td>
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<tr>
<td></td>
<td>1. undirectional (with any of the above degrees of block in opposite direction)</td>
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<td></td>
<td>2. bidirectional</td>
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<tr>
<td>2.</td>
<td>Secondary A-V block, or interference (normal refractory period)</td>
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<td></td>
<td>A. Ipsedirectional interference</td>
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<tr>
<td></td>
<td>1. delayed conduction</td>
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<td></td>
<td>2. dropped beats</td>
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<td>B. Contradirectional interference</td>
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<td>1. isolated</td>
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<td></td>
<td>a. direct</td>
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<tr>
<td></td>
<td>b. delayed</td>
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<tr>
<td></td>
<td>2. repetitive (interference dissociation, A-V dissociation due to interference)</td>
</tr>
<tr>
<td></td>
<td>a. undirectional with block in opposite direction, which may be:</td>
</tr>
<tr>
<td></td>
<td>1. primary, complete or incomplete</td>
</tr>
<tr>
<td></td>
<td>2. secondary, due to ipsedirectional interference</td>
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<tr>
<td></td>
<td>b. bidirectional</td>
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<td></td>
<td>3. interference dissociation with capture</td>
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<tr>
<td></td>
<td>a. atrial</td>
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<td></td>
<td>b. ventricular</td>
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<td></td>
<td>c. both atrial and ventricular</td>
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<td></td>
<td>4. combinations of primary or secondary heart block with interference dissociation</td>
</tr>
<tr>
<td></td>
<td>a. block above level of A-V nodal pace-maker</td>
</tr>
<tr>
<td></td>
<td>b. block below level of A-V nodal pace-maker</td>
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</tbody>
</table>

A-V Conduction Defects

A working classification of disturbances in conduction through the A-V junction is presented in Table 1.

A-V Block. The separation of A-V block into primary and secondary classes, as suggested by Zeisler, has been found most useful. Primary block is due to an anatomic defect or abnormal refractivity of the A-V junction. The block may be of any degree from simple delayed conduction to complete A-V dissociation. Rarely block may be almost complete, with transmission of an impulse only during the supernormal phase of conduction. It is important to realize that block may affect only antegrade or only retrograde conduction, or both, or it may involve conduction in both directions to different degrees.

Secondary A-V Block. Secondary block is due to interference, in the presence of a normal refractory period of the A-V junction. There are 2 types of interference, as follows:

1. Ipsedirectional interference: This occurs when the heart beat is so rapid that one impulse falls closely upon the heels of a preceding one traveling in the same direction and finds the A-V junction refractory, with consequent delay or failure of conduction. Examples are paroxysmal atrial tachycardia with first degree A-V block, or atrial flutter with 2:1 A-V block.

2. Contradirectional interference: In contrast, a second type of interference occurs when an impulse from the atrium finds the A-V junction refractory due to passage of a beat from a lower center back toward the atrium. The interference here is between 2 impulses moving in opposite directions and toward each other, that is contradirectionally. The interference may be direct, in which case the 2 waves of excitation meet head on and mutually obliterate each other. However, if one of the impulses precedes the other and is blocked, it may nevertheless set up a refractory period for subsequent passage of a contradirectional impulse. This phenomenon may be designated delayed contradirectional interference. An example is the prolonged P-R interval of the first beat following an interpolated ventricular extrasystole. In this paper the term interfe-
ence implies the presence of the contradirectional type, unless otherwise specified.

Contradirectional interference may be isolated or repetitive; repetitive contradirectional interference produces A-V dissociation.

A-V Dissociation. In A-V dissociation the atria and ventricles beat independently of each other in response to their own pacemakers. A-V dissociation may be due to primary complete heart block or to contradirectional repetitive interference.

Interference Dissociation. This term is used in the strict sense of A-V dissociation due to interference. No attempt is made to distinguish complete from incomplete interference dissociation because, as pointed out by others, with rare exceptions, a long tracing will prove that the dissociation is incomplete. Interference dissociation may be bidirectional or unidirectional with primary block in the opposite direction.

Capture. During interference dissociation capture is said to occur when favorable conditions of the refractory state permit an impulse from one center to pass through the A-V junction and control the entire heart for one or more beats. Ventricular capture, atrial capture or occasionally both may occur in any given case. The term capture is preferred to that of an interference beat because the latter usage may cause confusion with the concept of a blocked beat due to isolated interference.

Foci Between Which Interference Dissociation May Occur

Interference dissociation between a sinoatrial and an A-V nodal rhythm is the most common form. It may be regarded as the prototype of this arrhythmia, and its presence will be implied unless otherwise specified. However, interference dissociation can occur between 2 pacemakers in any part of the heart. It is even possible to have interference between 2 foci in a single heart chamber, without implicating the A-V junction. The theoretical foci between which interference dissociation may occur are assembled for reference in table 2. In all instances (in the absence of antegrade second degree A-V block) the lower focus forms impulses at a faster rate than the upper one. Obviously retrograde block must be present, protecting the slower upper focus. This block may be primary, of any degree, or secondary, due either to ipsedirectional or contradirectional interference. On the other hand, interference dissociation is possible in the presence of a faster upper than lower focus, if there is incomplete antegrade block, reducing the number of conductible beats. Paroxysmal tachycardia in an upper focus in the absence of A-V block may be dissociated due to interference from tachycardia in a lower center (fig. 17e). This combination of arrhythmias is termed simultaneous dissociated paroxysmal tachycardia. Rarely there may be interference between several foci active at the same time. Parasytole with simple interference may be considered a variant of interference dissociation.

Site of Interference

Interference between 2 contradirectional impulses may occur at any level between their starting points. The most common mechanisms are illustrated in figure 1. Thus, interference between an S-A and A-V nodal impulses may take place in the A-V node, in the atrium (atrial

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### Table 2: Location of Foci Between Which Interference Dissociation Can Occur

<table>
<thead>
<tr>
<th>Foci</th>
<th>Lower portion of S-A node</th>
<th>Homogenetic or hetero-genetic rhythms</th>
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<tbody>
<tr>
<td>1. S-A Node</td>
<td>Atria</td>
<td></td>
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<tr>
<td></td>
<td>A-V node</td>
<td></td>
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<tr>
<td></td>
<td>His bundle</td>
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<tr>
<td></td>
<td>Ventricles</td>
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<tr>
<td>2. Atrium (Paroxysmal atrial</td>
<td>Two atrial foci</td>
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<tr>
<td>tachycardia, flutter or fibrillation with block)², ³</td>
<td>A-V node</td>
<td></td>
</tr>
<tr>
<td></td>
<td>His bundle</td>
<td></td>
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<tr>
<td></td>
<td>Ventricles</td>
<td></td>
</tr>
<tr>
<td>3. A-V node (A-V nodal rhythm,</td>
<td>Two A-V nodal foci⁴</td>
<td></td>
</tr>
<tr>
<td>tachycardia with block)</td>
<td>A-V node</td>
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<td></td>
<td>His bundle</td>
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<td></td>
<td>Ventricles</td>
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<td>4. Ventricles</td>
<td>Two ventricular foci</td>
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<tr>
<td>5. Complete A-V block with</td>
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<tr>
<td>dissociation between 2 foci</td>
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<td>6. Interference dissociation</td>
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<tr>
<td>between more than 2 foci</td>
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<tr>
<td>for example, S-A rhythm with</td>
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<tr>
<td>dissociation due to interference</td>
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<tr>
<td>with ventricular extrasystoles</td>
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<tr>
<td>and automatic beats⁵. ⁶</td>
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<tr>
<td>7. Ventricular parasystole with</td>
<td></td>
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<tr>
<td>simple interference⁷</td>
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</tbody>
</table>

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fusion beat) or at the S-A junction. Interference between an S-A and a ventricular impulse may occur in proximity to the idioventricular focus, in the ventricle (ventricular fusion beat), in the A-V node, in the atrium (atrial fusion beat) or at the S-A junction. Finally, interference between an A-V nodal and an idioventricular rhythm may occur in the A-V junction, in the ventricle (ventricular fusion beat) or in the immediate vicinity of the idioventricular pacemaker. Rarely the site of interference may shift (figs. 8 and 16).

**Zone of Potential Interference**

A clear concept of isolated interference is essential to the understanding of the more intricate problems of interference dissociation. The mechanism of isolated interference can best be visualized with the aid of simplified diagrams.

In figure 2a let there be a pause in the sinus rhythm after P2 so that an A-V nodal beat escapes. If retrograde is faster than forward conduction and an S-A beat does not appear before position P4, the A-V nodal pacemaker will control the atria as well as the ventricles. However, if a normal sinus impulse arises at any time between P3 and P4, it will interfere with the A-V nodal escaped beat. The time interval between P3 and P4 may conveniently be termed "the zone of potential interference." This zone is not to be confused with the phase of interference of Lewis and Master, or the phase of concealed conduction of Langendorf and Pick, which refers to that portion of the cardiac cycle between the absolute and relative refractory phase during which partial penetration of an impulse into the A-V junction takes place. In subsequent parts of figure 2 the pause preceding the A-V nodal or ventricular escaped beats is assumed to be constant, while conduction in either direction and the moment of appearance of oncoming S-A beats are the variables. In figure 2b forward and retrograde conduction times are equal and the zone of interference is slightly wider than in 2a. In figure 2c retrograde conduction is prolonged so that P3 comes after the ventricular systole, and the zone of interference is correspondingly increased. In figure 2d forward conduction time is prolonged, and this mechanism also lengthens the zone of interference. Note that the zone of interference in A-V nodal beats is equal to the sum of forward and retrograde conduction times between the atrium and the site of the A-V nodal pacemaker.

From the foregoing considerations certain important deductions may be derived, as follows: 1. It may be anticipated that interference, and dissociation due to interference, are more likely to occur when the A-V nodal rhythm is of that type in which retrograde conduction time is prolonged, since the zone of potential interference is thereby increased. 2.

**Fig. 1.** Theoretical sites of interference between: (A) S-A and A-V nodal impulses, (B) S-A and idioventricular impulses, and (C) A-V nodal and idioventricular impulses. The customary conventions are employed to indicate the origin of impulses in the S-A node, A-V junction and ventricle, atrial and ventricular systoles, and S-A and A-V conduction.

**Fig. 2.** Diagrammatic representation of the zone of potential interference. Solid dots and unbroken lines represent origin and conduction of active impulses. Open dots and broken lines represent theoretical position of interfering beats at the onset of the zone of potential interference.
Since A-V nodal rhythm of this type is much less common than that with preceding atrial activation, the explanation for the infrequency of interference dissociation (as well as reciprocal rhythm) in clinical practice is apparent. 3. Prolongation of antegrade conduction time also lengthens the zone of potential interference, and therefore first degree A-V block is a predisposing factor for interference dissociation. 4. In A-V nodal escaped beats most commonly the temporal relationships are such that there is interference with an oncoming S-A impulse. 5. However, if an escaped beat controls the entire heart, statistically the type with preceding atrial activation should be most common, and in our experience this is true with respect to escaped beats after atrial or A-V nodal extrasystoles.

We have not observed an A-V nodal escaped beat with preceding atrial activation after ventricular extrasystoles. However, this phenomenon is theoretically possible, particularly if the ventricular impulse is conducted retrograde to the atrium and discharges the S-A node. In the latter event, the postextrasyotolic pause and subsequent escape mechanism should be the same as following A-V nodal extrasystoles. Schott\(^8\) presented 2 cases of ventricular extrasystoles with retrograde conduction, followed by A-V nodal escaped beats with P preceding R, which he considered unique. Schott explained the preceding atrial excitation in the escaped beats on the basis of facilitation.

In figure 2e the zone of interference of an idioventricular beat is shown. The duration of this zone is equal to the sum of forward and retrograde conduction times between the atria and the ventricles. In figure 2f the zone of interference (P\(_{17}-P\(_{18}\)) of an idioventricular beat is compared to one (P\(_{14}-P\(_{15}\)) of an average A-V nodal beat starting at the same time. The difference in duration of the 2 zones of interference is equal to the time required for conduction from the A-V nodal focus to the ventricle and back.

The following deductions regarding idioventricular beats seem logical: 1. Since the zone of interference of idioventricular beats exceeds that of atrionodal beats, retrograde conduction is statistically less likely following the former.

2. The zone of interference is of relatively greater importance late in atrial diastole. This fact has been confirmed incidentally by Kistin and Landowne,\(^7\) and Bussan, Torin and Scherf,\(^8\) who found that retrograde conduction occurs most often after those ventricular extrasystoles that come early in diastole. 3. The zone of interference is of particular significance when the heart rate is rapid because it will then occupy a greater proportion of diastole. 4. Moreover, if the zone of interference should exceed the duration of the atrial cycle length, retrograde conduction would be impossible at all times. These factors help explain the infrequency of ventricular capture and of retrograde conduction to the atria in paroxysmal ventricular tachycardia.

The diagrams in figure 2 have been simplified for our present purposes. Actually the problem is complicated by such factors as the zone of delayed contradirectional interference, the location of the A-V nodal pacemaker, unusual prolongation of the refractory period, variation in conduction through the A-V junction at different levels, facilitation, reentry and, finally, abnormalities in sino-atrial conduction.

**Modes of Onset of Interference Dissociation**

There are several ways in which interference dissociation may originate. These will be divided into 3 categories, as follows:

1. *Escape Mechanism*: (Lower center slower than upper center)

   The A-V nodal focus may have a slower inherent rhythm than the S-A node, but it may fortuitously usurp control of the heart during a pause in the dominant rhythm. Such a pause may be due to deficient production of impulses (sinus arrest), blocked sinus impulses (sino-atrial block), and blocked impulses in the A-V node above the site of its pacemaker, or may follow extrasystoles and paroxysmal tachycardias of any type.\(^7\) If the duration of the atrial pause exceeds the nodal cycle length, an A-V nodal escaped beat is to be expected. The escaped beat may control the entire heart, but more often it interferes with the oncoming sinus beat (fig. 2). The escape may be isolated,
but occasionally favorable temporal relationships permit a consecutive series of escaped beats, resulting in dissociation. Since the sinus rhythm is faster than the escape rhythm it must quickly regain control of the heart beat, and, therefore, interference dissociation of this type is necessarily short lived (fig. 19a).

2. Homogenous Rhythms: (Lower center slightly faster than upper center)

Under certain conditions an A-V nodal focus may develop a slightly more rapid rhythm than the S-A node, and consequently control ventricular activation. If its rhythm is fast enough, the A-V nodal pacemaker will control the atria also. However, if the A-V nodal rhythm is only slightly faster than the S-A rhythm, interference dissociation may occur. The dissociation may be due to bidirectional interference or to forward interference and retrograde primary or secondary block. The onset of interference dissociation will be favored if the A-V nodal rhythm is of the type with preceding ventricular activation and consequently a wide zone of potential interference (fig. 2c). The A-V nodal rate may become faster than the S-A rate at any given moment under the following circumstances: 1. Depression of the S-A nodal rhythm, with the A-V nodal rate constant (figs. 4 and 5). 2. Slight acceleration of the A-V nodal rhythm with the S-A rate constant. 3. Combination of acceleration of the A-V and slowing of the S-A rhythms. 4 Depression of both pacemakers, with a greater effect on the S-A rhythm. 5. Acceleration of both rates but involving the A-V node to a greater degree.

In some cases it may be difficult to differentiate a homogenous rhythm from a heterogenous one, particularly when the rates of both foci are relatively rapid. This point is of more than academic importance because an active ectopic rhythm may require suppressive therapy, such as, quinidine, whereas no attempt should be made to abolish a potential escape rhythm.

3. Heterogenous Rhythms: (Lower focus much faster than upper one)

Active rhythms may originate in lower centers which are much more rapid than the S-A rhythm and ordinarily should take command of the entire heart. However, if retrograde block is present, antegrade dissociation results. Therefore, paroxysmal A-V nodal and ventricular tachycardia with retrograde, but not forward block are examples of interference dissociation. Of course, the retrograde block may be of any type: primary, complete or incomplete, or secondary. In the last case, if the conductible retrograde rhythm is slightly faster than the S-A rhythm, interference may occur in the upper portion of the A-V node and thus there will be a combination of ipsilateral and contralateral retrograde interference. At very fast rates in paroxysmal A-V nodal or ventricular tachycardia with delayed retrograde conduction, the zone of potential interference might exceed the R-R interval and ventricular capture would therefore be impossible.

Duration of Interference Dissociation

Interference dissociation tends to be a transient arrhythmia and rarely persists for any appreciable time without interruption. In some patients it may be encountered at every examination over a period of months or years, but it is always interrupted at frequent intervals. Persistent interference dissociation is possible only in the following situations: 1. In the presence of retrograde block and a high degree antegrade A-V block, if the critical R to P interval for forward conduction, plus the P-R interval for conducted beats exceeds the automatic ventricular period.2 In paroxysmal ventricular tachycardia with retrograde block if the refractory period of the bundle plus the P-R interval exceeds the ventricular cycle length. In rare cases of synchronization of the 2 rhythms.

Modes of Termination of Interference Dissociation

The termination of interference dissociation may take one of the following forms:

1. When dissociation is due to an accidental escape mechanism, as discussed previously it will of necessity be short-lived and the S-A pacemaker regains control of the heart after a few beats.

2. In homogenous rhythms changes may
occur in the relative rates of the upper and lower pacemakers so that the sinus impulses obtain a clear lead and terminate the dissociation. These changes are the reverse of events favoring the onset of interference dissociation (fig. 13).

3. Interference dissociation may be abolished by various maneuvers or drugs affecting the ectopic focus to a greater degree than the S-A node. Thus normal sinus rhythm may be restored by forced breathing, carotid sinus pressure (fig. 15), exercise, change of posture, or the administration of atropine. (Paradoxically, in some cases interference dissociation may be precipitated by these same factors.)

4. If the A-V node should suddenly develop a sufficiently fast rhythm, simple A-V nodal rhythm may replace interference dissociation (figs. 8 and 9).

5. Interference dissociation may be abolished by a ventricular capture (or a reciprocal beat) which discharges the ectopic pacemaker (fig. 9).

6. As a result of ventricular capture, or other types of transition,25,40 the sinus rhythm may be accelerated and take command of the heart (fig. 5).

7. An atrial capture may be followed by a run of A-V nodal rhythm when the retrograde beats discharge the S-A pacemaker, temporarily depressing its rhythmicity (fig. 9).

8. The transition from interference dissociation to normal sinus or A-V nodal rhythm may be abrupt or gradual. Indeed at times the one rhythm seems to slip imperceptibly into the other. This form of transition frequently is characterized by the presence of fusion beats (figs. 16 and 17a).43

9. At times the transition between the 2 rhythms may be accompanied by shifting of the pacemaker in the sinus node.2

10. Interference dissociation may be terminated by an atrial extrasystole which discharges the lower center on its way through the A-V junction (fig. 14). It is theoretically possible that a ventricular extrasystole with retrograde conduction could do the same thing.

**Comparative Rates of the Two Rhythms**

Classically in interference dissociation the lower rhythm is faster than the upper one. However, exceptions to the rule may be encountered, as follows:

1. Although the S-A rhythm is actually faster, the slower A-V nodal rhythm may escape after a pause, as previously described (fig. 19a).

2. Preceding the transition from interference dissociation to normal sinus rhythm there may be a short period during which the S-A nodal pacemaker warms up and is faster than the A-V nodal focus, but has not yet gained control of the heart beat (figs. 17 and 17a).

3. There may be varying degrees of S-A block so that the A-V rhythm is faster than the effective S-A rhythm.27

4. The sinus rhythm may be faster, but there may be A-V block above the site of the A-V nodal pacemaker.9 If the internodal interval is shorter than the time between conductible S-A beats, interference dissociation results (figs. 10 and 11).

5. The A-V nodal rhythm actually may be faster than the sinus rhythm, with resulting interference dissociation. However, if there is co-existent second degree block below the level of the A-V nodal pacemaker, the antegrade conductible A-V nodal beats will be less frequent than the atrial beats.

6. If synchronization occurs, the 2 rhythms may run along at the same rate for varying lengths of time.

It is obvious, therefore, that an A-V nodal rhythm faster than the sinus rhythm is not a necessary criterion for the diagnosis of interference dissociation.

**Aberrancy in A-V Nodal Rhythm**

In A-V nodal rhythm most often ventricular conduction is slightly aberrant. At times the aberrancy may be very marked, with resulting bizarre QRS complexes. In the case of A-V nodal extrasystoles or paroxysmal tachycardia the aberrancy of the ventricular responses is due to the normal refractoriness of the bundle branches, that is, ipsedirectional interference. This explanation obviously cannot apply to A-V nodal escape mechanisms. Prinzmetal and his group28,29 attributed the ventricular aberrancy in the Wolff-Parkinson-White syndrome as well as in A-V nodal escape beats to accelerated conduction through special pathways within the A-V junction tissue. However, Pick and
Katz\textsuperscript{20} have criticized this theory, and we concur with the opinion that it is untenable. Pick\textsuperscript{31} suggested that the aberrancy of ventricular conduction from A-V nodal escaped beats is due to utilization of paraspecific pathways of conduction\textsuperscript{22} by impulses originating in peripheral portions of the A-V node, in contrast to those originating more centrally. In order to confirm this theory it will be necessary to prove that the paraspecific fibers are actually functional.

Another possible explanation is that if the A-V nodal focus is located eccentrically its excitation wave may spread unevenly through the junction and reach one bundle branch in advance of the other. In this connection it has been shown that the A-V node is partitioned by longitudinal fibrous septa, which could account for the fact that longitudinal is more rapid than horizontal transmission\textsuperscript{6}.\textsuperscript{27} We find this hypothesis particularly appealing, because it is compatible with Scherf’s\textsuperscript{33} theory of functional longitudinal dissociation in reciprocal rhythm, and also with the explanation for the occurrence of both atrial and ventricular captures in the same case (q.v.). The same theory may be invoked to explain changes in contour of retrograde P waves (fig. 9).

In rare instances it is possible for the A-V nodal beats to give rise to more normal looking QRS complexes than the sino-atrial beats. This phenomenon occurs in the Wolff-Parkinson-White syndrome with interference dissociation (fig. 19). The A-V nodal beats in the Wolff-Parkinson-White syndrome may show some ventricular aberrancy due to fusion with a portion of a sino-atrial impulse passing over the anomalous pathway.\textsuperscript{26} In interference dissociation between S-A and an idioventricular rhythm in the presence of bundle-branch block it is possible for the idioventricular beats to appear supraventricular in contour if they originate in the interventricular septum.\textsuperscript{34}

In some cases it may be difficult, if not impossible, to differentiate A-V nodal escape with aberrant conduction from ventricular escape.

**Relationship of the P Wave to QRS Complexes**

In interference dissociation, due to the fact that the ventricular is faster than the atrial rate, the P wave appears to run up to the QRS complex, come abreast of it and finally pass it (figs. 8 and 9). This is the outstanding electrocardiographic manifestation of interference dissociation and is the main clue to its diagnosis. The progressive shortening of the P to R time has led to use of the term “reverse Wenckebach phenomenon.”\textsuperscript{35, 36} This term should be discarded because the P to R relationship in interference dissociation has nothing in common with second degree heart block. Moreover, the designation “reverse Wenckebach phenomenon” may cause confusion with the retrograde Wenckebach phenomenon in A-V nodal rhythm (fig. 9c).

When the P wave passes a QRS complex the following events may happen in subsequent beats. 1. Ventricular capture. 2. Atrial capture. 3. Synchronization or accrochage.\textsuperscript{22}

**Ventricular and Atrial Capture**

Ventricular capture occurs when, without significant change in rate of either the upper or lower rhythms, the P wave passes the QRS, finds the bundle nonrefractory and is conducted to the ventricle (fig. 9). Other types of transition between the 2 rhythms involved in interference dissociation are not to be considered true captures.

With ventricular capture there may be normal atrioventricular and intraventricular conduction. However, frequently A-V conduction is delayed or the capturing beat may be blocked. If the blocked impulse discharges the atrioventricular pacemaker, the effect of concealed conduction will be manifested by an irregularity in the ventricular rhythm.\textsuperscript{37} The bundle branches may be in a relative refractory state so that the QRS complexes of the ventricular capture will be aberrant (an example of ipse-directional interference). The ventricular capture in passing through the A-V node may discharge and depress its pacemaker, thus abolishing interference dissociation. If it does not discharge the A-V nodal pacemaker a
ventricular capture might be interpolated. Usually a ventricular capture has no effect on the S-A rhythm. However, in some cases the rate of the S-A pacemaker may be accelerated, and more rarely the S-A rate may slow down following a ventricular capture (fig. 7).

If conduction through the A-V junction is uniform the first ventricular cycle length following a capture may be the same, or nearly the same, as the usual nodal cycle length. If there is a localized delay in conduction of the ventricular capture below the site of the A-V nodal pacemaker (as compared to the usual A-V conduction) the next ventricular cycle will appear shortened. On the other hand, if the capture depresses the A-V nodal pacemaker the next internodal interval may be prolonged and the effect may extend over several cycles. In interference dissociation with an idioventricular rhythm, an incomplete capture would cause a ventricular fusion beat.

Analogous events come into play with regard to atrial capture. If a retrograde atrial beat discharges and depresses the S-A nodal pacemaker, a run of uncomplicated A-V nodal rhythm may result. If the atrial capture is only partial, an atrial fusion beat appears. In an atrial capture interference may occur at the S-A junction (fig. 1-14) so that the sinus rhythm will not be disturbed. In some cases an atrial capture may be accompanied by a reciprocal beat (fig. 9).

It is of interest that ventricular capture is much more frequent than atrial capture. Study of figures 2 and 3a will demonstrate that ventricular capture should be impossible unless there is retrograde block. Apparently in most cases this condition is met, and therefore atrial capture is encountered infrequently. In figure 3a the zone of interference of the blocked retrograde beat between P₁ and P₂ is evidently very short. Therefore P₄ falling at a distance beyond this zone, is conducted to the ventricle. P₃ comes during the refractory period of the bundle following the blocked retrograde beat and is not conducted (an example of delayed contradirectional interference).

Of exceptional interest are those cases in which both atrial and ventricular captures occur. The simplest explanation for this phenomenon is that the retrograde block is intermittent. When retrograde block is absent an atrial capture would be expected; when present, ventricular capture would occur.

However, an alternate explanation may be offered. We have been impressed recently by observation of several cases in which there were both atrial and ventricular captures, accompanied by reciprocal rhythm (fig. 9). Similar cases have appeared in the literature. This suggests a relationship between bidirectional capture and reciprocal rhythm. If ventricular capture occurs in the absence of retrograde A-V block, the S-A impulse must somehow by-pass the A-V nodal impulse as shown in figure 3b. This could occur in the presence of functional longitudinal dissociation in the A-V node. An A-V nodal beat may start out, be transmitted rapidly to the ventricle, and simultaneously spread toward the atrium at a retarded rate in a localized longitudinal pathway. Before the retrograde impulse can reach the atrium a sinus nodal impulse may have already activated the atrium and proceeded down the unexcited section of the A-V node, and finding the lower part of the A-V junction nonrefractory, be transmitted to the ventricle. Meanwhile the retrograde A-V nodal beat will be blocked at the atrial boundary owing to refractivity of the atria following the impulse P₅. In other words, simultaneous bidirectional conduction through the A-V junction could well account for the foregoing phenomenon. In the hypothetical instance in figure 3b if the beat P₅ had not occurred, there might have been a reciprocal beat as well as an atrial capture.

![Figure 3](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.81.3.811)

**Fig. 3.** Possible mechanisms explaining occurrence of atrial and ventricular captures in the same case. A. Retrograde block. B. Simultaneous bidirectional conduction through A-V node.
HEART BLOCK AND INTERFERENCE

Interference dissociation is frequently associated with other forms of heart block.

Antegrade Block. First degree A-V block produces a prolonged zone of potential interference and consequently increases the chances of interference (fig. 2b). Persistent dissociation due to interference may result when the ventricular cycle length is shorter than the combined lengths of the refractory period of the bundle and the P-R interval.20

The presence of second degree A-V block above the level of the A-V nodal pacemaker makes interference dissociation possible in cases in which the S-A rhythm is faster than the lower one. This is the underlying mechanism of paroxysmal atrial tachycardia, flutter, and fibrillation with interference dissociation. Whenever the automatic ventricular cycle is longer than 1 sinus period but shorter than 2 atrial cycles, dissociation with interference in the presence of 2:1 A-V block should be considered.19 There is a critical period for the R to P time which determines when a S-A impulse will be conducted. Usually the R to P time exceeds the normal sinus period but this is not always true (fig. 10). If the critical R to P time plus the time required for forward conduction exceeds the ventricular period, persistent dissociation may occur and closely resemble A-V dissociation due to complete primary block.19

Rarely in interference dissociation incomplete A-V block occurs below the site of the A-V nodal pacemaker. When a capturing beat is blocked below the A-V nodal pacemaker, the condition has been termed double interference or dissociation with ladder type of interference.41

In the presence of complete A-V block interference dissociation is possible between 2 foci above, or 2 foci below the site of block.

Retrograde Block. Since in interference dissociation the A-V nodal pacemaker is usually the faster one, it would control the entire heart unless there were retrograde block. It is to be emphasized that this block may be of several types.

1. Probably in most cases retrograde conduction is slower than forward conduction. This factor as well as first degree retrograde block lengthens the zone of potential interference (fig. 2c), and thereby predisposes to interference dissociation, particularly at rapid rates.

2. Complete primary retrograde block may be present. In this event no impulses from the A-V node would enter the atrium under any circumstances.

3. When the ventricular rate is excessively rapid, as in paroxysmal A-V nodal or ventricular tachycardia, incomplete primary or secondary block may occur. The rate of the retrograde conductible beats may be slightly faster than the rate of the S-A beats and therefore retrograde interference dissociation will occur.

DIFFERENTIAL DIAGNOSIS

There are a number of arrhythmias that must be differentiated from interference dissociation. The most important are the following:

1. First degree heart block with a long P-R interval, with the P wave lying close to the preceding QRS. Tracings of this type closely resemble interference dissociation with synchronization. However, in a long tracing when the heart rate changes either spontaneously or in response to the sinus reactions the P-R interval will remain constant.

2. Second degree A-V block with a P-R interval longer than the P-P interval.2 This is seen as a manifestation of 2:1 block, or in the Wenckebach phenomenon. In the former instance the relationship will become clear when the rate of the sinus rhythm changes. The Wenckebach phenomenon is differentiated because of the marked irregularity in the ventricular rhythm.

3. Almost complete block with V-A or A-V response. Differentiation of this condition from interference dissociation has been briefly discussed in the report of case 7. Winternitz and Langendorf41 suggested that complete, or almost complete, antegrade block and normal retrograde conduction may lead to interference dissociation in reverse. However, in such cases it is necessary to rule out the presence of al-
most complete retrograde block, with conduction only during the supernormal phase.

4. Complete A-V block. As has been pointed out the rate of the atrial as compared to the ventricular rhythm is not an absolute criterion for differentiation, since the ventricular rate may be faster than the conductible atrial rate in interference dissociation, and rarely in complete block the atrial rate may be slower than the ventricular rate. The main point of differentiation is that in complete block in long tracings there will never be any evidence of conduction across the A-V junction.

5. Atrial fibrillation with complete block presents the same picture as atrial fibrillation with interference dissociation. However, when the dissociation is of short duration, it is probably due to interference.

6. Interference dissociation between an S-A and A-V nodal rhythm with aberrant ventricular conduction may sometimes be confused with interference dissociation between S-A and idioventricular rhythms. The latter in turn would have to be differentiated from interference between an S-A and A-V nodal rhythm in the presence of bundle-branch block. Long tracings recording the onset and termination of interference dissociation, or the presence of isolated extrasystoles or escaped beats should resolve this problem. Ventricular captures or fusion beats are diagnostic of an idioventricular rhythm.

7. Ventricular parasystole requires special consideration in the differential diagnosis of interference dissociation. In parasystole the rhythm of the lower center is slower than the upper one. This is probably the most important distinction. The ventricular center is protected from the higher center by an entrance block, that is, there is unidirectional forward block into the parasystolic center and retrograde interference. This is the reverse of ordinary interference, in which there is forward interference and retrograde block. The differentiation from the mechanism shown in figure 1-B2 where a ventricular focus is not discharged by a sinus beat is that in the latter instance the focus is protected by interference and not by a permanent block, and potentially the ventricular focus could be discharged at other times in the cardiac cycle; in parasystole the ventricular focus is protected at all times. In interference dissociation the 2 foci may be protected from one another by block or interference at a distance; for example, the sinus nodal pacemaker may be protected from a ventricular impulse during ventricular tachycardia by retrograde block in the A-V junction. In parasystole the protecting block is in the immediate vicinity of the ventricular pacemaker. In rare instances ventricular parasystole may complicate interference dissociation between 2 other foci.

Presentation of Cases

Case 1

E. B. This 63-year-old white man had recently developed the anginal syndrome. By coincidence he suffered a sudden attack of coronary thrombosis while a routine electrocardiogram was being made. The accompanying lead I (fig. 4) was obtained within a few minutes of the onset of pain, before any medication had been given.

The atria are under the control of the sinus nodal pacemaker. Phasic sinus arrhythmia is present, with an atrial cycle length varying from 0.84 to 1.04 seconds. On the other hand, the internodal interval is fixed at 1.01 seconds. The P waves numbered 4, 5, 6, 9, and 10 are conducted to the ventricle, terminating cycles 0.86 to 1.01 seconds in duration. Interference dissociation occurs in the remaining portions of this tracing. The ventricular complexes in response to sinus nodal impulses differ slightly in contour from those of A-V nodal origin. Respiratory variations in the form of QRS also occur throughout the tracing. At the transition from normal sinus rhythm to interference dissociation, between R4 and R5, the cycle length is equal to the usual internodal interval for this case. This suggests that the A-V nodal focus is located low down in the junctional tissues, since the A-V nodal impulse must have begun to develop immediately preceding the onset of QRS.

Comment. This case illustrates one of the simplest forms of interference dissociation, with sinus arrhythmia and a fixed internodal interval. As the S-A rate waxes and wanes, there is a shift between normal sinus rhythm and interference dissociation. The ventricular complexes in response to A-V nodal stimuli are slightly aberrant.
Case 2

W. R. This patient was a 58-year-old Negro man with hypertensive heart disease and digitalis intoxication. A short strip of lead II is shown in figure 5. The first beat is of A-V nodal origin with retrograde slower than antegrade conduction time. The R to P time is 0.14 second. P₃–R₃ represents isolated interference between S-A and A-V nodal impulses. The third and fourth beats are of normal sinus origin, with a P-R interval of 0.16 second and an R-R interval of 0.76 second. The sinus rhythm is depressed after P₂ allowing the A-V node to escape and a bidirectional interference dissociation results. After P₃, the sinus rhythm accelerates and regains command of the heart at P₅. At the transition from interference dissociation to normal sinus rhythm the atrial cycle length shortens and P₅ also is conducted to the ventricle. Following P₅, S-A rhythmicity is depressed and interference occurs.

The inherent rhythm of the A-V node is regular, with a cycle length of 1.12 seconds. The intervals, R₃–R₄ and R₄–R₅, introducing the first nodal beats following sinus beats, are slightly shorter, measuring 1.10 seconds. This slight discrepancy in timing indicates that the A-V nodal impulses must have started to form shortly before the onset of QRS₄ and QRS₅. The ventricular complexes of A-V nodal origin are slightly aberrant. Between P₄ and P₅, the A-V nodal rhythm is actually faster than the S-A rhythm, yet the A-V nodal pacemaker does not gain control of the atria, despite the absence of retrograde block. However, owing to the fact that retrograde is longer than forward conduction, the zone of potential interference is increased, and dissociation occurs. Of course, if the S-A node did not quicken its pace and take command of the heart at P₅, the A-V nodal impulses eventually would have effected an atrial capture.

Comment. The interesting features in this case are as follows: 1. Interference dissociation occurs during periods of depression of the S-A nodal pacemaker. 2. The interference dissociation is bidirectional. 3. Retrograde conduction time from the A-V node to the atrium is longer than forward conduction time to the ventricle. 4. The resulting time differential, with widening of the zone of potential interference, favors establishment of interference dissociation rather than atrionodal rhythm. 5. The transition from interference dissociation to normal sinus rhythm is accompanied by enhancement of the S-A nodal rhythmicity. (Blumgart and Gargill ascribed this phenomenon to improvement in coronary artery blood flow to the S-A node following 2 ventricular systoles in rapid succession. Cutts suggested that coronary blood flow to the sinus node may be maximal when the atria and ventricle beat in their normal sequence.)

Case 3

I. I. This patient was a 62-year-old Negro woman with hypertensive and arteriosclerotic heart dis-
ease, and digitalis intoxication. Selected strips of lead II are shown in figure 6. In strip a, for the first 3 cycles interference dissociation is present between S-A and A-V nodal pacemakers. The atrial cycle length increases abruptly from 1.16 seconds between P₂ and P₃ to 1.68 seconds between P₃ and P₄. At the same time the ventricular cycle length decreases from 1.26 between R₁ and R₄, to 1.23 between R₃ and R₄, and 1.20 seconds between R₂ and R₄. The combination of the lengthening of the P-P interval and the shortening of the R-R interval causes P₄ to fall outside of the refractory period of R₄ and, therefore, to be conducted to the ventricle. There is no evidence of atrial activity accompanying R₄, although sufficient time is available for retrograde conduction. In long tracings in this case a retrograde atrial beat was never recorded and, therefore, evidently complete retrograde block is present. The interval R₄–R₅ measures 0.69 second and the ventricular beat R₅ appears normal in contour. Following R₅, 2 pairs of ventricular systoles occur. They consist of an A-V nodal beat followed by a normal sinus beat (pseudoreciprocal rhythm). Since the S-A impulse encounters the relative refractory phase of the bundle, both atrioventricular and intraventricular conduction are impaired. The P-R interval between P₃ and R₄ is 0.22 second and the interval R₃–R₄ measures 0.52 second. The interval R₄–R₅ is 0.54 second and the P-R interval between P₅ and R₅ is 0.24 second. The interval R₄–R₅ is slightly longer than R₃–R₄ and there is a lesser degree of aberrancy of R₅ as compared to R₃. Interference dissociation recurs in the last 2 cycles of this strip.

In strip b, the first beat is of A-V nodal origin with retrograde block. P₂ falls outside of the refractory phase of R₁, and is conducted normally after a P-R interval of 0.16 and an R-R interval of 0.72 second. P₃ is an atrial extrasystole, followed by a normal ventricular response after a normal P-R interval and an R-R interval of 0.48 second. The same sequence, A-V nodal beat, normal sinus beat and atrial extrasystole, occurs between R₄ and R₅. The interval between the normal sinus beat P₇ and the atrial extrasystole P₉ is 0.54 second and intraventricular conduction is normal. R₉ is an A-V nodal escaped beat and R₁₀, a normal sinus beat with an aberrant ventricular response after a prolonged P-R interval of 0.24 and an R-R interval of 0.55 second. The cycle preceding an atrial extrasystole is shorter than most of the intervals between escaped beats and normal sinus beats, yet intraventricular conduction is normal after atrial extrasystoles and aberrant in the latter instance. The explanation for this may be that the atrial extrasystoles occur after a comparatively short cycle (R₉–R₈ and R₁₀–R₁ in strip b), while the sinus beats following escaped beats terminate a short cycle after a long one (R₄–R₅ and R₁–R₂ in strip a, and R₉–R₁₀ in strip b). Since the refractory period of the junctional tissues varies directly with the length of the preceding cycle, the cause for this phenomenon is apparent. It is of interest that the P-P interval shortens following an atrial extrasystole, and the beats P₉ and P₁₀ are conducted. These short intervals may be due to improved blood supply to the S-A node following 3 beats in close succession, that is, R₁₁, R₁₂, R₁₃ and R₁₄, R₁₅, R₁₆ in strip b.⁴⁴

**Fig. 6. Case 3. Selected strips of lead II.**
Comment. The salient features demonstrated in this tracing are as follows: 1. Interference dissociation in the presence of marked sino-auricular depression. 2. Pseudoreciprocal rhythm. 3. Aberrant intraventricular conduction in relation to the length of the preceding cycles. 4. Effect of atrial extrasystoles on the subsequent sinus rhythm. 5. Termination of interference dissociation by a combination of acceleration of the ventricular rhythm and depression of the sinus rhythm. (This is an apparent paradox, since the same factors in the presence of a normal sinus rhythm may produce interference dissociation.)

Case 4

A. W. Figure 7, a selected strip of lead II, was obtained from a 60-year-old white man with arteriosclerotic heart disease. No medication that might have influenced the rhythm had been given.

Interference dissociation between an S-A and A-V nodal rhythm is present. The rates of both the S-A and A-V nodal foci vary independently. In the first 4 cycles the P-P interval lengthens progressively while the A-V nodal rate is fairly constant. As a result, P₄ comes far enough beyond R₄ to fall in the relative refractory phase of the bundle, and it is conducted to the ventricle after a P-R interval of 0.28 and an R-R interval of 0.46 second. The ventricular capture following P₄ occurs after a P-R interval of 0.28 second and an R-R interval of 0.45 second. Following the capture by P₄ the atrial cycle length increases to 0.76 second. There is a less marked increase in the P-P interval of 0.69 second after the capture of P₄. The latter temporal relationship is such that it allows a second sinus beat to be conducted (P₅₋₆₋₇₋₈). The lengthening of the interatrial cycle length embracing the ventricular capture is unusual. An increase in vagal tone caused by the early captured ventricular beat may be sufficient to overcome any effect of increased blood flow and hence cause slowing of the S-A rate. An alternate and perhaps preferable explanation is that the intervals P₄₋₅ and P₅₋₆ would have increased, even if there were no intervening ventricular beat, due to the presence of sinus arrhythmia, which of itself set up conditions favorable for the capture. The impulse from the S-A node which gives rise to the ventricular capture passes through the A-V nodal focus and discharges it. The foreshortening of the internodal intervals between R₄₋₅ and R₅₋₆ is due to the fact that there is a delay in the passage of the capturing impulse below the site of the A-V nodal focus.

Comment. This tracing demonstrates interference dissociation between S-A and A-V nodal rhythms in which the 2 vary in rate independently and according to no set pattern. Ventricular capture occurs on 2 occasions, with foreshortening of the succeeding internodal cycle length, due to a conduction delay below the level of the A-V nodal pacemaker. Following ventricular capture the atrial cycle length increases, but this is probably a fortuitous event due to the presence of sinus arrhythmia.

Case 5

D. A. This 60-year-old white man had had a thyroidectomy because of a toxic nodular goiter. He was receiving digitalis, and postoperatively was given quinidine for persistent atrial fibrillation. The tracing in figure 8 was obtained after termination of the atrial fibrillation. Selected strips, cut from a long lead II, are presented for analysis.

In strip a the atrial mechanism cannot be definitely determined. The ventricular rhythm is perfectly regular. The ventricular complexes are of the RS type and are followed by small positive deflections that at first glance might be thought to be R prime waves or upright P waves.

In strip b right carotid sinus pressure was applied between the arrow markers. The ventricular rhythm slows gradually and the eighth ventricular beat interferes with a preceding normal S-A beat.
The next 3 beats are of normal sinus origin. At this point it might be suggested that the positive deflection after the S wave in strip a is an upright P wave with interference dissociation and synchronization between the S-A and A-V nodal rhythms. However, strips c and d clearly delineate the rhythm.

Following the first beat in strip c interference dissociation occurs, with the P wave running up to the QRS complex, coming abreast of it at R4 and passing it at R5. At R11 the A-V node takes control of the entire heart. The P wave coinciding with R10 is probably an atrial fusion beat. (Compare fig. 1A, mechanisms 1, 2, 3, and 5.)

The A-V nodal rhythm continues throughout strip d. Towards the end of the tracing the relationship of P to QRS is similar to that in strip a. The retrograde P wave in this case is of the minus-plus type with a prominent late upright deflection. It is the final portion of the retrograde P wave that appears after the QRS complexes in strip a and resembles an upright P wave. Also, superimposition of the negative phase of the P wave upon the S wave causes the latter to appear deeper.

Comment. This case demonstrates an A-V nodal rhythm that could be converted at will to interference dissociation and then normal sinus rhythm by carotid sinus stimulation. Stimulation of the carotid sinus caused slowing of both the S-A and A-V nodal rhythms but affected the latter to a greater degree. As recovery from carotid stimulation took place the rates of both rhythms increased but the A-V nodal rhythm became relatively more rapid than the sinus rhythm, so that first interference dissociation and then simple A-V nodal rhythm resulted.

Case 6

M. P. This 60-year-old white man was suffering from arteriosclerotic and pulmonary heart disease. Selected strips of a long lead II obtained on July 1, 1954, are shown in figure 9. The patient was not receiving digitalis or quinidine at that time.

In strip a the first P wave is conducted to the ventricle after a P-R interval of 0.14 second. Following this interference dissociation occurs between S-A and A-V nodal rhythms. The atrial cycle length for the most part is 0.53 to 0.54 second in duration. Possibly due to changes in vagal tone, the atrial cycle length increases to 0.58 second between P7-P8 and P11-P12. The A-V nodal cycle length measures 0.47 to 0.49 second. Following the normal beat at the beginning of this strip the P wave runs up to the QRS complex, comes abreast of it at R4, and begins to pass it at R5. P5 occurs long enough after R5 to fall within the relative refractory phase of the bundle and it is conducted to the ventricle after a P-R interval of 0.24 second. This ventricular capture shortens the R-R interval to 0.44 second. Two conducted sinus beats, P3 and P4, follow the ventricular capture. This phenomenon may be due to the fact that the S-A impulses P1, P3, and P4 in passing through the A-V node discharged and temporarily depressed its pacemaker. However, it soon picks up enough speed to interfere again with the sinus rhythm at P5. Interference dissociation continues to the end of the strip. The interval R9-R10 is shortened due to the fact that the P-R interval preceding R4 is prolonged while the next P-R interval is normal.

At the beginning of strip b interference dissociation is present. The third atrial impulse is conducted and a ventricular capture results. As in strip a, 2 normal sinus beats follow the capture. Interference recurs between P5 and R7 and continues to P9, which is an atrial capture. Since the A-V nodal rhythm is faster than the S-A rhythm at this point, a run of A-V nodal beats ensues (P9, P9, and P10). Conduction of the retrograde beat P10 is prolonged to 0.22 second. This retrograde beat re-enters a pathway in the A-V node long enough after R10 to be conducted to the ventricle with a manifest forward conduction time of 0.25 second. The re-entrant beat apparently discharges and depresses the A-V nodal pacemaker, causing a pause in the atrial rhythm of 0.58 second, which is terminated by a normal sinus beat, P13. Interference dissociation then recurs. It is possible that a blocked re-entrant beat occurred after P9, setting the stage for the reciprocal beat at P13. The same factor of concealed conduction of a previous beat causing areas of refractivity in the bundle might account for the aberrancy of P13 as compared to other retrograde P waves in this tracing. It is of interest that in this short tracing are seen normal sinus rhythm, interference dissociation, ventricular capture, atrial capture, A-V nodal rhythm, and reciprocal rhythm.

Similar cases have been reported by others. Strip c begins with a run of A-V nodal rhythm with a progressively increasing R to P time, which suddenly lengthens to 0.24 second after R4 (the
retrograde Wenckebach phenomenon). This results in an increase in the atrial interval, $P_5-P_6$, without a change in the corresponding R-R interval. Therefore, the lengthening of the atrial cycle is due simply to an increase in retrograde conduction time and not to a change in rate. $P_4$ falls far enough after $R_4$ to accommodate a re-entrant mechanism and $R_5$ is a reciprocal beat. Again note the aberrancy of $P_4$. $P_5$ may be an atrial fusion beat. The fact that the retrograde beat $P_5$ precedes $R_5$ may reflect a prolongation of antegrade relative to retrograde conduction time due to the proximity of $R_6$ to $R_4$. Following $P_5$ dissociation recurs and persists until $R_6$, when the A-V nodal pacemaker assumes command of the whole heart. Reciprocation occurs following $P_6$. The reciprocal beat discharges the A-V nodal pacemaker and a normal sinus mechanism ensues at $P_7$. The last beat in this tracing is either an A-V nodal beat or an interference beat. In the first reciprocal beat the R to P time is 0.24 second and the P to R time is 0.22 second, while in the second reciprocal beat R to P measures 0.29 and P to R, 0.17. In the reciprocal beat in strip b, R to P measures 0.22 and P to R, 0.25 second. This conforms with the well-known phenomenon that in reciprocal rhythm there is an inverse relationship between R to P and P to R times.\(^{15}\)

**Comment.** The following interesting features are illustrated by this case: 1. The close relationship of normal sinus rhythm, interference dissociation, atrial capture, ventricular capture, and reciprocal rhythm. 2. Demonstration of all these variants in a single short tracing. 3. The depressing effect on either the S-A or A-V nodal rhythms by premature discharge of one focus by an impulse from the other. 4. The possibility of concealed conduction of a blocked re-entrant beat setting the stage for reciprocal rhythm in the following beat, and explaining variations in the contour of the atrial complexes. 5. The apparent shortening of the ventricular cycle length following a ventricular capture with a prolonged P-R interval.

**Case 7**

G. G. The clinical diagnosis of this 56-year-old white man was arteriosclerotic heart disease, en-
Fig. 10. Case 7. Selected strips of tracings (lead II) obtained on 3 different occasions.

The enlarged heart, second degree A-V block, nodal escaped beats, interference dissociation, and the an- 
ginal syndrome. In childhood he had had diphtheria, 
which may have been an etiologic factor for the heart 
block. Selected strips of lead II obtained on 
different occasions are presented in figure 10.

In strip a, normal sinus rhythm with 2:1 A-V 
block is present. The atrial cycle length varies 
from 0.72 to 0.78 second, due to ventriculophasic sinus 
arrhythmia. The P-R interval of conducted impulse 
measures 0.22 to 0.23 second. The ventricular 
cycle length is between 1.48 and 1.52 seconds.

In strip b every third atrial impulse only is con- 
ducted to the ventricle, after an interval of 0.28 
second. Each conducted beat is followed by a 
blocked P wave, and the latter in turn by inter- 
ference between an S-A impulse and an A-V nodal 
escaped beat. The atrial rate is slower than in strip 
a; the cycle length measures 0.84 to 0.90 second. 
The escaped beats occur after a pause of 1.44 to 
1.46 seconds, thus causing a bigeminal rhythm. 
The form of the escaped beats is slightly aberrant, as 
indicated by absence of the S wave which identifies the 
sinus beats. The fact that the dominant S-A 
 rhythm is slower in strip b than in strip a allows 
time for escape of the lower focus.

In strip c interference dissociation is present. The 
S-A rhythm is slower than on the 2 previous occa- 
sions, with a cycle length of 0.86 to 0.92 second. 
The internodal interval (1.72 seconds) also is longer 
than in strip b. Ventricular captures occur at R2 and 
R3, after a P-R interval of 0.36 second. It may be 
assumed that basically 2:1 A-V block is present, 
since this conduction defect was constant during 
the previous year. The ventricular rate is faster 
than that of the conductible atrial beats, and since 
there is retrograde block interference dissociation 
results. The QRS complexes of A-V nodal origin 
have the same contour as the escaped beats in strip b.

In strip b isolated escape occurs, while in strip c 
interference dissociation is encountered, although in 
both instances the ventricular is faster than the 
conductible atrial rate. However the ventricular 
rate is relatively more rapid in strip b, (average P-P 
interval 0.85 second, internodal interval 1.45 sec- 
onds) than in strip c (P-P interval 0.89 second, R-R 
interval 1.72 seconds). As a result, in strip b the 
escape beats fall early enough before the next P 
wave to allow the latter to be conducted to the ven- 
tricle, whereas in strip c the P wave following the 
nodal beats most frequently arrives during the re-
fractory period of the bundle. This allows repetitive 
interference before a ventricular capture finally 
occur.

The rhythm in strip c might easily be mistaken 
for almost complete heart block with occasional
A-V response. To prove the existence of the latter arrhythmia it is necessary to demonstrate a critical interval during which A-V conduction occurs, so that any P wave falling before or after this interval will be blocked. Upon analysis of the long tracing from which strip e was obtained these criteria were not fulfilled. Conduction did not take place unless the R to P time was greater than 0.70 second. However, an upper limit of R to P time after which block would again result could not be demonstrated. Another point against the possibility of almost complete block in this case is that over a long period of observation complete block did not develop.

Comment. This case demonstrates interference dissociation in the presence of second degree A-V block. It fulfills the criteria set down for this arrhythmia by Dressler, Roesier, and Specter. The genesis of the arrhythmia from 2:1 A-V block to second degree A-V block with nodal escape and finally to interference dissociation, and the relation of these rhythms to the atrial and ventricular rates are demonstrated.

Case 8

E. W. This 68-year-old white man was being treated for congestive heart failure due to hypertensive and arteriosclerotic heart disease. He also had progressive muscular dystrophy, with possible cardiac involvement. Following administration of a mercurial diuretic he had an unusually marked response that precipitated digitalis intoxication, evidenced by paroxysmal atrial tachycardia with block. Figure 11 illustrates the arrhythmia. Paroxysmal atrial tachycardia is present with shifting of the ectopic pacemaker. There are obvious differences in contour of the P waves preceding the fourth, thirteenth, and fifteenth ventricular beats. The P-P interval averages 0.34 second. The minor variations in atrial rhythm may be related to shifting of the ectopic pacemaker or to ventriculophasic arrhythmia secondary to the dropped beats. Second degree A-V block is present with the Wenckebach phenomenon. For the most part each third atrial beat is blocked. In the pause following QRS there is a pair of ventricular escaped beats. QRS is either a ventricular escaped beat or fusion beat. A similar sequence occurs after QRS. The tenth, eleventh, and twelfth QRS complexes are ventricular in origin. QRS is probably a ventricular fusion beat. The interval between idioventricular beats measures 0.60 to 0.66 second. The R-R interval of the conducted beats varies from 0.46 to 0.62 second.

Comment. In this case the actual atrial rhythm is faster than the idioventricular rhythm. However, due to the presence of second degree antegrade A-V block, the idioventricular rhythm is faster at times than the conductible atrial rhythm. Thus, interference dissociation occurs between a paroxysmal atrial tachycardia and an idioventricular rhythm.

Case 9

P. K. This 67-year-old white man was admitted to the hospital on June 17, 1954, following an attack of acute coronary thrombosis. An electrocardiogram just prior to admission showed complete heart block. Later that day second degree A-V block was present, with nodal escaped beats and interference dissociation. Interference dissociation was recorded at all subsequent examinations until July 12, 1954. Selected strips (lead II) of several electrocardiograms taken during this period are presented for analysis.

In figure 12 the first 3 beats are of normal sinus origin with P-P intervals of 0.68 to 0.72 second. The first P-R interval is prolonged to 0.24 second, the next 2 intervals to 0.26 second. P1 is blocked and R1 is in response to an A-V nodal escape, which touches off a run of interference dissociation. Although during the rest of this strip the S-A pacemaker is slightly faster than the atrionodal one, it cannot quite gain control of the ventricles and interference dissociation persists. R4 might be in response to P4, but the corresponding P-R interval is probably too short for this patient. Interest centers about the pause in the ventricular rhythm between R3 and R4, which is 1.16 seconds in duration. If P4 were conducted through the site of the A-V nodal pacemaker, and discharged it, there should have been a longer pause before R4 because the in-
INTERFERENCE DISSOCIATION

In figure 13, strip a, interference dissociation is present between the first 4 S-A and A-V nodal beats. The rates of the 2 pacemakers are quite similar. Following the fourth atrial and ventricular impulses both pacemakers slow down but the A-V node is depressed to a greater degree, allowing P5 to be conducted to the ventricle after a prolonged P-R interval of 0.32 second. P6 falls within the absolute refractory period of the bundle after R4.
and is blocked above the level of the A-V nodal pacemaker. There is delay in formation of the next nodal beat, with the interval measuring 1.33 seconds between R₅ and R₄. P₄ encounters interference, P₅ is conducted and P₆ is blocked. Interference recurs between R₄ and P₆.

In strip b, for the first 3 cycles interference dissociation is present. However, following P₅ the atrial rhythm suddenly speeds up while the A-V nodal rhythm slows down, and P₆ is conducted after a P-R interval of 0.22 second. P₃ and P₅ are blocked and followed by nodal escaped beats with interference. The inherent nodal rhythm must be slower in the second half of this strip to allow P₇ and P₈ to be conducted without interference.

In the 2 preceding strips and in figure 12, the block was presumed to be above the level of the A-V nodal focus with a pause before initiation of an A-V nodal beat. In strip c, a different mechanism for the pause following blocked P waves is encountered. Interference dissociation is present throughout this tracing. Between R₃ and R₄ the ventricular rhythm accelerates and the cycle length decreases from 0.80 to 0.72 second. There is a pause of 1.40 seconds between R₃ and R₄ during which P₅ is blocked. This sequence differs from those in strips a and b, and figure 12, in which the P waves preceding the blocked beats are conducted. In strip 2c the blocked beat P₅ is not preceded by a conducted one. Furthermore, P₅ occurs long enough after R₄ to have been conducted to the ventricle. The key to solution of this problem is furnished by analysis of the spacing of the ventricular beats. The ventricular rate speeds up before the pause after R₄, the shortest interval precedes the pause and the first cycle following the pause is greater (0.80 second) than that preceding it (0.72 second), and the pause itself is shorter than the sum of any 2 consecutive short cycles. This arrangement is typical of the Wenckebach phenomenon and suggests the presence of an area of block below the level of the A-V nodal pacemaker. The nodal beat between R₄ and R₅ is concealed, with antegrade block, while interfering in a retrograde direction with the oncoming sinus beat, P₅. Similarly, concealed interference could account for the blocked beat P₆.

Figure 14 shows the effect of an atrial extrasystole during interference dissociation. The fifth P wave represents an extrasystole that is conducted to the ventricle. En route it discharges and depresses the A-V nodal pacemaker, with subsequent restoration of normal sinus rhythm. The P-R interval increases slightly in subsequent beats and the eleventh P wave is blocked above the level of the A-V nodal pacemaker. Interference dissociation then recurs.

Figure 15 demonstrates one effect of carotid sinus pressure in interference dissociation. Left carotid sinus pressure causes the S-A rhythm to slow down to a greater degree than the A-V nodal rhythm. This causes the P waves to fall further beyond QRS and allows a ventricular capture by the fifth atrial beat. Normal sinus rhythm is established at a slow rate but the A-V nodal pacemaker continues in a depressed state, so that the S-A rhythm persists to the end of this tracing.

Comment. The following are some of the interesting features in this case: 1. Interference dissociation in the presence of an atrial rhythm faster than the A-V nodal rhythm. 2. Onset of atrial nodal rhythm with a pre-automatic pause. 3. Slowing down of both S-A and A-V nodal rates, but the latter to a greater degree, allowing the S-A node to take control of the heart. 4. S-A nodal rhythm speeding up while A-V nodal rhythm slows down, breaking up interference dissociation. 5. Presence of block below the level of the A-V nodal pacemaker with the Wenckebach phenomenon, and concealed interference. 6. Termination of interference dissociation by a conducted atrial
extrasystole which discharges and depresses the A-V nodal pacemaker. 7. Effect of carotid pressure on interference dissociation, causing restoration of normal sinus rhythm by slowing the S-A more than the A-V nodal rate.

**Case 10**

A. V., a 62-year-old white man, was suffering from arteriosclerotic heart disease with congestive failure and multiple disturbances of rhythm. At the time the tracing in figure 16 was obtained he was receiving digitals. Ventricular tachycardia is present at the beginning of strip a and for the first few beats the atrial mechanism is not visible. Immediately following R1 a retrograde P wave can be detected. After 3 retrograde atrial beats a P wave drops out. Following R4, retrograde conduction is resumed until termination of the ventricular tachycardia after R17. Then there are 2 ventricular escaped beats of multifocal origin. The first normal sinus beat occurs at R4 and introduces a sinus tachycardia. Rn is a ventricular extrasystole. The P-P interval during the sinus tachycardia measures 0.48 second, as compared to an R-R interval of 0.46 second during the ventricular tachycardia.

In strip b the tracing starts with a sinus tachycardia. This is followed by dissociation due to interference with a ventricular tachycardia that is only slightly faster than the sinus rhythm. At first the interference occurs in the ventricle with production of ventricular fusion beats (R1 through R4). At R4 the site of interference shifts to the A-V junction, and after R6 retrograde conduction to the atria occurs and the ventricular focus controls the entire heart. (Compare with fig. 1B, mechanisms 1, 3, 4 and 7.)

**Comment.** In this case a sinus tachycardia competes for control of the heart with a slightly faster ventricular tachycardia. Because of slight difference in rates, the transition from one rhythm to the other is gradual and marked by the presence of ventricular fusion beats. The dissociation is due to bidirectional interference, since there is normal retrograde as well as antegrade conduction. Malinow and Langendorf present a strikingly similar case in their authoritative paper on the mechanism of fusion beats.

**Case 11**

T. M. This 60-year-old white man was suffering from arteriosclerotic heart disease complicated by multiple arrhythmias. Long leads were obtained on frequent occasions, and selected portions of lead II are presented for analysis.

In figure 17, strip a, normal sinus rhythm is present at an approximate rate of 75 per minute. The first 2 QRS complexes are of supraventricular origin. The P-R interval measures 0.24 second. The fifth to the tenth ventricular systoles constitute a run of idioventricular rhythm with interference dissociation. The idioventricular cycle length is 0.76–0.84 second. Thus, for part of the time during dissociation the ventricular rhythm is faster than the atrial rhythm, but preceding the transition to normal sinus rhythm it is actually slower for a short time. As might be anticipated when the 2 independent rhythms have about the same rate, fusion beats appear at the onset and termination of interference dissociation (R9, R10, and R11). The onset of interference dissociation coincides with a slight increase in rate of the idioventricular pacemaker and a slight decrease in the S-A rate, and the reverse occurs in the transition from idioventricular to normal sinus rhythm.

In strip b, the first 2 beats are of normal sinus origin. The third ventricular complex is an extrasystole from the same focus (designated focus 1) as the idioventricular rhythm in strip a. The fifth ventricular complex is an extrasystole from a second idioventricular focus, designated focus 2. It is followed by another extrasystole from focus 1 and then by 2 automatic beats of similar origin. Thus, interference dissociation is present from the fifth to the eighth ventricular beats.

In strip c the second beat is a ventricular extrasystole from focus 2. A pair of similar extrasystoles occur at R10 and R11, followed by a run of automatic
beats from the same focus. The idioventricular rhythm originating in focus 2 is interrupted by an interpolated ventricular extrasystole from focus 1 (QRS). The eleventh beat is a ventricular capture. QRS is of normal sinus origin. The last 2 beats are a ventricular extrasystole and an automatic beat from focus 2.

In strip d paroxysmal A-V nodal tachycardia is present and is terminated by carotid sinus pressure, signaled by the solid black line. Preceding the end of the tachycardia there are 2 ventricular extrasystoles that do not disturb the A-V nodal rhythm. In strip e, A-V nodal tachycardia is present, with a cycle length of 0.42 second. The first, third, and fifth ventricular beats are extrasystoles and the last one introduces a short run of ventricular tachycardia. A second run of paroxysmal ventricular tachycardia is present from the seventeenth to the twenty-first ventricular systoles. In this tracing, during ventricular tachycardia retrograde conduction from the ventricle to the atrium does not occur owing to interference below the level of the A-V nodal pacemaker. On other occasions in this case, during sustained ventricular tachycardia, retrograde conduction to the atrium did occur.

Comment. This case shows the following interesting features: 1. Interference dissociation between a normal sinus rhythm and multifocal idioventricular rhythms. 2. Idioventricular rhythm preceded and followed by fusion in the ventricle between supraventricular and idioventricular beats. 3. Idioventricular rhythm triggered off by ventricular extrasystoles. 4. Idioventricular rhythm with interference dissociation interrupted by interpolated ventricular extrasystoles. 5. Simultaneous dissociated atrionodal and ventricular tachycardia.

Case 12

T. S. This 64-year-old white man was suffering from multiple myeloma. Frequent electrocardiograms showed the presence of the Wolff-Parkinson-White syndrome. Short strips of the standard limb leads and precordial leads V1 and V4 are shown in figure 18 for purposes of orientation. The P-R interval measures 0.12 second and the QRS interval also is 0.12 second. A typical Wolff-Parkinson-White pattern is present, with positive delta waves in leads V1 and V4, and a small negative delta wave in lead II.

On July 7, 1955, unusual disturbances in rhythm

Fig. 17. Case 11. Selected strips of lead II obtained on different occasions.
developed, and 2 selected strips of lead II are presented for analysis in figure 19. In strip a, the dominant atrial rhythm is of normal sinus origin interrupted by atrial extrasystoles after each normal beat. P waves numbered 4, 5, and 7 are not clearly visible due to superimposition upon QRS complexes. Their position has been approximated after comparison with similar intervals in this tracing. For example, the time between P4 and P5 is the same as between P1 and P4; and the interval P5-P6 is analogous to P4-P5. The coupling of the extrasystoles to the sinus beats is fixed at 0.52 second, with the exception of the first one, in which the pre-extrasystolic cycle measures 0.46 and the returning cycle 0.72 second. The returning cycle throughout the rest of the tracing is usually 0.68 second. The sum of the intervals preceding and following extrasystoles is 1.20 seconds for the most part, corresponding to the interval between 2 normal sino-atrial cycles in strip b. The QRS complexes in response to the atrial extrasystoles differ slightly in contour from those of S-A origin. This aberrancy is due to differences in the extent of pre-excitation of the ventricle, depending upon the distance of the ectopic focus from the anomalous atrioventricular connection. The QRS complexes that follow normal P waves at positions 9, 11, and 13 differ from one another, from the usual Wolff-Parkinson-White complexes for this case, and also from those QRS complexes of A-V nodal origin. They probably represent fusion between a nodal escaped beat and an impulse from the atra traversing the anomalous A-V pathway. The post-extrasystolic interval, 0.68 second, approaches that of the internodal cycle between R4 and R7 (0.66 second). The P-R interval of the S-A beats and the atrial extrasystoles is between 0.10 and 0.12 second.

The pause following the first atrial extrasystole P2 is longer than the others in this tracing; and it is terminated by a nodal escaped beat that introduces a run of interference dissociation. The internodal cycle length is 0.66 second. This interval is longer than the average R-R interval during the remainder of this tracing, although it is shorter than most of the post-extrasystolic intervals, which measure 0.68 second. The fortuitous timing, however, allows a short run of dissociation. The QRS complexes in response to the A-V nodal focus measure 0.08 second and are almost entirely upright, with a small terminal S wave. The S-T segments are depressed and the T waves inverted. The atrial extrasystole P3 occurs early enough in the cycle to capture the ventricle, discharge the A-V nodal focus and thus terminate the dissociation. It is of interest that in this tracing one atrial extrasystole induces interference dissociation, while another abolishes it. The nodal rhythm is interrupted by a premature ventricular contraction at R4.

In strip b, P1 is probably an extrasystole, followed by a pause of 0.72 second, which allows the A-V

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**Fig. 18.** Case 12. Standard limb leads and precordial leads V1 and V4.

**Fig. 19.** Case 12. Selected strips of lead II.
node to escape. Following Pz the atrial rhythm is regular, with a cycle length of 0.60 second except for the interval Pz–P+. The ventricular cycle length in response to the A-V nodal pacemaker varies from 0.56 to 0.59 second and is shorter than the P-P interval. P waves cannot be clearly seen at positions 3, 11, 12, 13, and 14. In the thirteenth, fourteenth, and fifteenth ventricular complexes the S wave disappears and the S-T segment approaches the isoelectric line, probably due to superimposition of P waves upon the late portion of ventricular depolarization. QRS5 is probably a fusion beat, similar to QRS6 and QRS15 in strip a.

R4 and R7 are ventricular extrasystoles. R7 is followed by a retrograde atrial impulse that discharges the S-A node and causes the succeeding P-P interval to lengthen to 0.78 second. R4 is apparently from the same focus as R7, but it is not followed by retrograde conduction and the atrial rhythm is not disturbed. Apparently an atrial extrasystole is superimposed on R4, thus giving rise to interference in the A-V junction between an atrial and ventricular extrasystole. The intermodal interval increases spontaneously after R15 and allows resumption of normal sinus rhythm with Wolff-Parkinson-White type responses.

Comment. This case demonstrates the following interesting features: 1. The Wolff-Parkinson-White syndrome with return to normal ventricular excitation in the presence of A-V nodal rhythm. 2. Varying types of ventricular fusion in the presence of the Wolff-Parkinson-White syndrome, following S-A nodal beats, atrial extrasystoles, and A-V nodal escaped beats. 3. Interference dissociation initiated and terminated by atrial extrasystoles. 4. Termination of interference dissociation by spontaneous depression of an A-V nodal pacemaker. 5. Interference between atrial and ventricular extrasystoles. 6. Ventricular extrasystoles with retrograde conduction to the atrium in the presence of A-V nodal rhythm with interference dissociation, an unusual type of atrial capture.

A similar case has been presented by Mali- now and Langendorff.26

SUMMARY

Contradirectional interference results when 2 stimuli arising in different foci in any part of the heart spread in opposite directions toward each other. Interference dissociation is defined as that type of dissociation which is due to repetitive contradirectional interference.

The electrocardiograms of 12 cases have been selected for analysis to illustrate many of the important features of interference dissociation.

The various centers between which dissociation may theoretically occur are listed for reference. This series includes examples of dissociation due to interference between the following pairs of rhythms: Normal sinus rhythm and A-V nodal rhythm, normal sinus rhythm and multifocal idioventricular rhythms, sinus tachycardia and paroxysmal ventricular tachycardia, paroxysmal atrial tachycardia with block and idioventricular rhythm, simultaneous dissociated paroxysmal A-V nodal and ventricular tachycardias, and normal sinus rhythm and A-V nodal rhythm in the presence of the Wolff-Parkinson-White syndrome.

Interference may occur at any level between the points of origin of the 2 rhythms, and the most common sites of interference are illustrated diagrammatically.

Interference at the upper end of the A-V junction is of particular importance, and a clear understanding of isolated interference in this region is of help in resolving some of the more difficult problems of dissociation. For this reason the concept of the zone of potential interference is introduced. This is the time interval in the cardiac cycle during which interference between an A-V nodal, or ventricular, and oncoming S-A beat may be anticipated. Certain important deductions are derived from theoretical considerations of the zone of potential interference.

The various mechanisms leading to the onset and termination of interference dissociation are described, and the rates of the dissociated rhythms are compared.

Aberrancy of the ventricular response to A-V nodal impulses is discussed. This phenomenon is ascribed to uneven spread of the excitation wave from an eccentrically located focus, due to the fact that in the A-V node longitudinal is faster than horizontal transmission.

Ventricular and atrial captures are considered in detail. Of particular interest is the occasional occurrence of both types of capture
in the same case. This paradox is explained on the basis of either intermittent retrograde block or simultaneous bidirectional conduction through the A-V node in the presence of functional longitudinal dissociation. The relationship of other forms of heart block to interference dissociation is pointed out. Heart block predisposes to, and frequently complicates, interference dissociation. A differential diagnosis between interference dissociation and other disturbances of rhythm is presented. Interference dissociation is one of the most complex arrhythmias and familiarity with all its possible variations is essential to the correct interpretation of any given tracing.

SUMMARIO IN INTERLINGUA

Interferentia contradirectional resulta quando 2 stimulos a origine in differente focus in aliquo parte del idioventricular se propaga in direction contrari le un verso le altre. Dissociation per interferentia es define in le typo de dissociation que es causate per repetitive interference contradirectional.

Le electrocardiogrammas de 12 casos esseva seligite e analysate como illustration de multes del importante aspectos de dissociation per interferentia.

Le varie centros inter le quales dissociation es theoricamente possibile es listate pro objectivos de referentia. Iste serie include exemplis de dissociation causate per interferentia inter le sequento pares de rhythmos: Normal rhythm sinusal e rhythm nodal atrio-ventricular; normal rhythm sinusal e multifocal rhythmos idioventricular, tachycardia sinusal e tachycardia ventricular paroxysmal, tachycardia atrial paroxysmal con bloco e rhythm idioventricular, tachycardias nodal atrio-ventricular e ventricular dissociate simultane, e normal rhythm sinusal e rhythm nodal atrio-ventricular in le presentia del syndrome Wolff-Parkinson-White.

Interferentia pote occurrer a non importa qual nivello inter le punctos de origine del 2 rhythmos. Le plus commun sitos de interferentia es illustrate diagrammaticamente.

Interferentia al extremitate superior del junction atrio-ventricular es de importantia particular. Un comprension precise de interference isolate in iste region es de adjuta in resolver certes del plus difficile problemas de dissociation. Pro iste ration le concepto del zona de interferentia potential es introducire. Ilo es le intervallo de tempore in le cyclo cardiac durante le qual interferentia pote expectare inter un pulso nodal atrio-ventricular o ventricular e un pulso sino-atratial in preparation. Certe deductiones importante es derivate ab considerationes theorico del zona de interferentia potential.

Le varie mechanismos que effectua le declaration e le termination del dissociation per interferentia es describite, e le rapiditate del dissociate rhythmos es comparate.

Aberrantia del responsa ventricular a impulsi de nodal atrio-ventricular es discutite. Iste phenomeno es ascribe in a propagation in-erqual del unda excitatatori ab un foco de location excentric, in consequentia del facto que in le nodo atrio-ventricular le transmission longitudinal es plus rapide que le transmission horizontal.

Capturas ventricular e atrial es considerate in detalone. Le occurrentia occasional de ambe typos de captura in le mesme case es de interesse particular. Iste paradoxo se explica super le base de intermittente bloco retrograde o de simultanea conduction bidirectional a transverso le nodo atrio-ventricular in le presentia de un functional dissociation longitudinal. Es signalate le relation inter altere formas de bloco cardiac e dissociation per interferentia. Bloco cardiac es un facuto que predispose a dissociation per interferentia e que frequentemente complica lo.

Es presentate un diagnose differential de dissociation per interferentia e altere disturbationes de rhythmso.

Dissociation per interferentia es un del plus complexe arrhythmias. Familiaritate con omne su variationes possibile es essential in le interpretation correcte de omne registration particular.

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REFERENCES


The material in this report came from patients dying in the hospital or at home under the care of physicians associated with the clinic or from autopsies done at the request of the coroner's office. Studies included cutting cross sections of epicardial vessels at regular intervals of 3 to 5 mm. and making transverse slices of the ventricles with microscopic examination of muscle from portions of the ventricles supplied by all branches of the coronary arteries. Significant coronary lesions were found in these patients, the highest incidence being in the age range from 50 to 59 years, where 75 per cent of the individuals had lesions. A correlation is made with the clinical circumstances of coronary artery disease. The author points out that in those patients with coronary artery disease and acute myocardial infarctions clinically, the mechanism of death is difficult to establish. Of 133 patients studied, 57 or 43 per cent died with evidence of myocardial failure. Thirty-one or 23 per cent of the patients died with repeated attacks of chest pain and without evidence of myocardial failure. This was diagnosed as coronary failure and there was no pathologic evidence of new infarction. Twenty or 15 per cent of the patients died of rupture of the heart. The rupture of the heart resulted first from a dissection in the endocardium with bleeding into the myocardium, and a final dissection of the hematoma through the epicardium into the pericardial space. In the 133 patients, only 8 who died had associated thromboembolic complications. In 250 patients with coronary artery disease in whom acute infarction had occurred in the past, 64 or 26 per cent died suddenly without evidence of repeated myocardial infarction. The author stresses that patients with coronary artery disease, including patients with healed myocardial infarction may die on the presumed basis of acute myocardial ischemia without infarction.

HARVEY
Interference Dissociation
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