How to Approach an Arrhythmia

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Which Leads to Take

Diagnostic information for the analysis of cardiac rhythms can usually be gleaned from all the conventional electrocardiographic leads. The clearest and most useful information, however, is usually obtained from the following leads: (1) Leads which best reflect the atrial deflection. These are usually standard leads II, V₁, and S₃ (positive electrode over the fifth interspace to the right of the sternum; negative electrode over the manubrium sternum). (2) Leads which best reflect the configuration of the QRS complex. Leads which reflect the phasic aberrant ventricular conduction and ventricular ectopy, are lead V₁ and the monitor lead MCL₁—a modified CL₁ lead (positive pole over the fourth interspace to the right of the sternum; negative pole on the left shoulder). The added advantage of this lead is that it does not unduly hamper coronary care nursing.

The Analytic Approach to Cardiac Rhythms

The following analytic procedure is recommended for the elucidation of cardiac rhythms: (1) analysis of the atrial deflection; (2) analysis of the QRS complex; (3) analysis of the P:QRS relationship; (4) analysis of premature events; (5) analysis of pauses and late events; and (6) analysis of unexpected or “paradoxic” events.

Analysis of the Atrial Deflection

The atrial deflection is analyzed to determine whether the site of the pacemaker is within the atria, and whether the P' wave reflects retrograde atrial activation from an A-V nodal (junctional) or ventricular pacemaker, or whether it reflects retrograde atrial activation from a reciprocal sequence.

The Atrial Origin of the Pacemaker

A sinus P wave is pyramidal in shape with a smooth contour and a blunted apex. The mean manifest frontal-plane sinus P-wave axis is usually in the region of +40° to +60° with a range of 0 to +70.

It is normally diphasic in lead V₁. An ectopic atrial P wave is bizarre, tall, peaked and/or irregular in outline. A P' wave of left atrial origin may have a “dome and dart” appearance: a proximal rounded deflection—the “dome,” followed by a distal sharply pointed deflection—the “dart.” Left atrial origin is usually associated with a mean manifest frontal-plane P'-wave axis in the region of −80° to −120°.

Clearly, the distortion of the baseline by regular F (flutter) waves, or f (fibrillation) waves, must also connote supraventricular origin. The flutter-wave mean manifest frontal-plane axis is usually directed at −90°, resulting in negative deflections in standard leads II and III, and in lead aVF.

The P' Wave of Retrograde Atrial Activation

Retrograde Atrial Activation from an Impulse Arising in, or Conducted through, the A-V Node. Retrograde atrial activation may result from an A-V nodal pacemaker or from a ventricular impulse which is conducted through the A-V node. When this occurs, the P'-wave frontal-plane axis is usually in the region of −70° to −90°, resulting in negative P' waves in standard leads II and III, and lead aVF.

Retrograde Atrial Activation through an A-V Nodal Bypass. Retrograde atrial activation can also result from a reciprocal beat or atrial echo which is conducted through an ectopically located bypass (see below). The P' wave of reciprocal rhythm, particularly that resulting from a bypass connecting the left atrium and the left ventricle, i.e. the type A W-P-W syndrome, usually results in a P'-wave axis that is directed in the region of −90° to −120° on the frontal-plane reference system. This results in a P' wave that is negative in standard leads II and III, and in lead aVF, and negative or equiaphasic in standard lead I.

Analysis of the QRS Complex

It is necessary to determine whether the QRS complex of a cardiac rhythm is the expression of a conducted supraventricular impulse (arising in the S-A node, atria, or A-V node), or whether it represents ventricular ectopy. The differentiation does not usually involve any difficulty when the supraventricular impulse is associated with normal intraventricular conduction, and hence a normal,
relatively narrow, QRS complex. The differentiation, may, however, be very difficult when the supraventricular impulse is associated with phasic aberrant ventricular conduction—the momentary, temporary, or episodic abnormal intraventricular conduction of a supraventricular impulse—since the QRS complex will then be wide, bizarre, and mimic a QRS complex of ectopic ventricular origin. The following features will assist in the differentiation: (1) Relationship to preceding atrial activity (see below). (2) The QRS configuration. Differentiating diagnostic criteria can also be obtained from a careful analysis of the QRS complex, especially in the precordial leads, and particularly in leads V₁ and MCL₁. The following is based predominantly on the elegant studies of Marriott and associates.¹ ³

Features Favoring Phasic Aberrant Ventricular Conduction

A Right Bundle-Branch Block Pattern. Phasic aberrant ventricular conduction usually, though not invariably, reflects a right bundle-branch block pattern.

Initial QRS Vector Similar to that of the Normally Conducted Supraventricular Impulse. Since right bundle-branch block has minimal or no influence on the initial QRS deflection, phasic aberrant ventricular conduction will tend to have an initial QRS complex similar to that of the normally conducted impulse.

A Triphasic-rSR' Complex in Lead V₁ or Lead MCL₁. This contrasts with the monophasic or diphasic form in the lead V₁ usually associated with ventricular ectopy.

qR, qRs, or qRS Complex in Lead V₆. Note that a tiny q wave in lead V₆ is rarely seen in ventricular ectopy.

Features Favoring Ventricular Ectopy

Monophasic or Diphasic Configurations in Lead V₁ or Lead MCL₁. It is particularly suggestive of ventricular ectopy when an RR' deflection manifests in lead V₁ and the initial R wave is taller than the ensuing R' deflection.

A QS Complex in Lead V₆.

An rS Complex in Lead V₆. This manifestation can also occur when right bundle-branch block is associated with left anterior hemiblock.

Concordant QRS Complexes in the Precordial Leads. Dominantly positive QRS complexes in all the precordial leads (this may also be due to the type A W-P-W syndrome), or dominantly negative QRS complexes in all the precordial leads.

Deepest QRS Complex in Lead V₁ or V₂. In left bundle-branch block the deepest QRS complexes are usually found in leads V₁ and V₂, whereas in ventricular ectopy the deepest QRS complex usually occurs in lead V₁ or V₂.

A Bizarre Frontal-Plane QRS Axis. For example, an axis of −120°. Note that the presence of A-V dissociation does not help in the differentiation since a dissociated A-V nodal rhythm could also be conducted with phasic aberrant ventricular conduction.

Analysis of P-QRS Relationship

It is important to note whether the P wave and the QRS complexes are related, i.e. whether each QRS complex is preceded or followed by a causally related P wave at a constant P-R or R-P interval. The diagnosis is usually simple in the case of normal or relatively slow rates, when the long intervening pauses make it easy to determine whether the P wave precedes the related QRS complex or whether it follows the related QRS complex, an expression of retrograde V-A conduction. When the rhythm is fast, however, as in paroxysmal tachycardia, identification of the causal relationship may be extremely difficult unless the beginning of the rhythm is seen.

With anterograde conduction of supraventricular impulses it is necessary to determine whether all conducted impulses, i.e. all impulses related to a QRS complex, have a fixed P-R relationship, or whether, for example, there is a progressive prolongation of the conduction time, as in the Wenckebach phenomenon, in which case there will also be a progressive decrease in the R-R intervals. When the P waves and QRS complexes are not constantly related, some form of A-V dissociation is usually present. This is particularly so when the P-R intervals appear to diminish progressively.

A-V dissociation is a general and nonspecific term which refers to any rhythm where the atria and the ventricles are activated concomitantly but independently, one pacemaker activating the atria and the other the ventricles. This may occur for a single or momentary episode, for example a ventricular extrasystole which is dissociated from a sinus impulse, both impulses interfering with each other's mutual progress within the A-V node; or the dissociation may be permanent as in established complete A-V block. A-V dissociation is thus never a primary diagnosis and usually has little meaning unless further qualified.

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"Interference"

The term "interference" has been interpreted in two different senses which have opposite meanings and which are, in fact, mutually exclusive.

The Electrophysiologic Interpretation. "Interference" is used in the electrophysiologic sense to connote the mutual impedance of the impulses from two pacemakers which prevent each other's onward conduction as a result of the refractoriness effected by each pacemaker. This may occur anywhere in the heart, for example in the ventricles, each impulse activating part of the ventricles and thereby resulting in a ventricular fusion complex, in the A-V node resulting in dissociated P and QRS complexes, or in the atria resulting in an atrial fusion complex, each impulse activating part of the atria.

The "Capture" Interpretation. The term "interference" has also been used to describe a capture beat. In this sense the conducted sinus or atrial impulse "interferes" with the rhythm of the ventricles, and the capture beat is consequently termed an "interference beat."

There seems little doubt, however, that the use of the term interference in the electrophysiologic sense is logically, semantically, and physiologically the correct one, and the reader is referred to the other reviews on the subject.

Analysis of Premature Events

The tracing is analyzed to determine whether there is a sudden premature event. Such a premature event may be due to an extrasystole, a parasystolic beat, a capture beat, or a reciprocal beat.

The Extrasystole

An extrasystole is an impulse which arises in an ectopic atrial, A-V nodal, or ventricular focus and is premature in relation to the prevailing rhythm. A further important characteristic is that it usually has a fixed or constant coupling time to the preceding sinus beat, i.e. the coupling interval is constant for all extrasystoles arising from the same focus. This indicates that the extrasystole is in some way related to or precipitated by the preceding sinus beat (compare Parasystole below).

The Distribution of Ventricular Extrasystoles: The Rule of Bigeminy. The precipitation of a ventricular extrasystole is at times favored by a long preceding cycle—R-R interval. The compensatory pause of the ventricular extrasystole constitutes another long cycle which, in turn, tends to precipitate a further ventricular extrasystole. Thus bigeminal rhythm tends to be perpetuated and the phenomenon has consequently been termed the rule of bigeminy.

Concealed Ventricular Extrasystoles. When ventricular extrasystoles occur in a basic bigeminal pattern and then tend to intermit for longer intervals, the sinus beats between the ventricular extrasystoles are often found to occur in odd numbers only. This has been attributed to a continuous bigeminal rhythm with some of the ectopic impulses being complicated by exit block. The distribution is referred to as concealed extrasystolic ventricular bigeminy. An analogous phenomenon may complicate ventricular extrasystoles which occur after every two sinus beats. When this rhythm intermits for longer periods, the intervening sinus beats tend to occur in numbers which conform to the formula of (a multiple of 3 + 2. This is termed concealed extrasystolic ventricular trigeminy.

Parasystole

Parasystole is the expression of an independent ectopic rhythm whose pacemaker is somehow protected in its immediate vicinity from the impulses of the dominant (usually faster) sinus pacemaker, and is thus able to continue its pacemaking discharge at its own inherent slow rate, undisturbed by the impulses of the faster sinus pacemaker. The ectopic impulses activate the surrounding myocardium whenever the myocardium is responsive, i.e. when it is not refractory consequent to prior activation by the sinus impulses. The parasystolic pacemaker is usually situated in the ventricles, or less commonly in the A-V node or in the atria. The independent rhythm of ventricular parasystole results in ectopic ventricular beats, abnormal QRS complexes, which are not related to preceding sinus beats; i.e. there is marked variation of the coupling intervals. Because of the regular, independent rhythm (some of whose discharges are manifest), the longer interectopic intervals will be in simple multiples of the shortest interectopic interval or the ectopic cycle length. Furthermore, occasional fortuitous invasion of the ventricle by both the supraventricular and ectopic impulses will result in a ventricular fusion complex, i.e. a QRS complex whose configuration resembles in part that of the "pure" conducted ectopic ventricular beat.

Atrial parasystole is a rhythm which is manifested as two independent and unrelated atrial deflections,
usually a normal sinus P wave and the P’ wave of an ectopic atrial pacemaker. The basic properties and characteristics of atrial parasystole are the same as those of ventricular parasystole. There are premature P’ waves which have varying coupling and their interectopic intervals are in simple multiples of a common denominator. There are occasional atrial fusion beats. Since both the pacemakers are situated in the same physiologic chamber, the bialtrial chamber, the ectopic impulses tend to discharge the sinus pacemaker whose cycle recommences from this moment of discharge. As a result, there is a tendency for the sinus P wave to be linked or coupled to the ectopic P’ wave. This is known as reversed coupling. The ectopic atrial impulse may be conducted to the ventricles if and when it finds the A-V node recovered consequent to possible prior activation by the sinus impulse. This distinguishes atrial parasystole from complete intra-atrial block—an expression of atrial dissociation. Complete intraatrial block also appears as two sets of independent and unrelated atrial P waves, but the P waves of one set—the ectopic atrial impulses—are never followed by QRS complexes, since the ectopic atrial impulses cannot exit from the area of tissue which they activate, i.e. they are blocked from the surrounding atrial tissue.

The Capture Beat

In cases of interference-dissociation, the impulses from a supraventricular pacemaker and a faster subsidiary (ventricular or A-V nodal) pacemaker interfere with each other within the A-V node. The sinus impulses cannot be conducted to the ventricles as a result of lower A-V nodal refractoriness consequent to partial retrograde penetration of the ventricular impulses; and the ventricular or A-V nodal impulses cannot be conducted to the atria as a result of the upper A-V nodal refractoriness consequent to partial penetration by the sinus impulses. At times, however, the sinus impulses may, with critical timing, arrive at the A-V node when it has recovered from its partial retrograde activation by the ventricular impulse. When this occurs, the sinus impulse can be conducted through the A-V node to activate, and thus momentarily “capture,” the ventricles. This isolated or momentary activation of the ventricles by the sinus impulse constitutes a capture beat.

The capture beat may be recognized by the following features: (1) It is always related to a preceding P wave. (2) It is always premature. (3) The QRS complex may be normal, resembling that of a normally conducted sinus impulse, or it may be conducted with phasic aberrant ventricular conduction, thus usually having a right bundle-branch block or triphasic configuration. Furthermore, the capturing impulse may effect a partial capture only, activating only a part of the ventricles; i.e. the capturing impulse invades the ventricles concomitantly with the ectopic ventricular impulse thereby constituting a ventricular fusion complex. (4) At times, the capturing impulse may penetrate the A-V node without reaching the ventricles thus constituting a concealed A-V nodal capture. The penetrating impulse discharges the A-V nodal pacemaker and thus resets the A-V nodal cycle, resulting in an unexpected pause. This is a form of concealed conduction, conduction that is not manifested electrocardiographically but whose presence is inferred by an ensuing disturbance of conduction and/or impulse formation.

Reciprocal Beats: Reciprocal Rhythm

In reciprocal rhythm an impulse arises in the atria, the A-V node (A-V junction) or ventricles, and during its conduction through the A-V node enters another hitherto unused pathway and returns to reactivate the same chamber (atria or ventricles) once again. The relatively rapid return of the impulse results in a premature event. In all cases of reciprocal rhythm the returning impulse may repeatedly reenter the original conduction pathway, thereby continuing the reciprocal rhythm as a reciprocating tachycardia, a continuous circus movement, i.e. an anterograde conduction through one pathway and retrograde conduction through the other.

Reciprocal Rhythm of Atrial Origin. In reciprocal rhythm of atrial origin a sinus or, less commonly, an ectopic atrial impulse, after activating the atria, enters the A-V node and returns through another A-V nodal pathway to reactivate the atria a second time. The retrograde activation results in a “retrograde” or inverted P’ wave—an atrial echo. This is reflected as a P-QRS-P’ “sandwich.” A prolonged P-R interval is usually necessary to permit the recovery of the upper A-V nodal tissues and/or the atria so that they can respond once again to the returning impulse. The P’ wave may be mistaken for a blocked atrial or A-V nodal extrasystole. Differentiation is usually made on the following basis: (1) A reciprocal P’ wave is usually associated with a long preceding P-R interval. Such selective linkage would be very fortuitous in the case of an atrial or A-V nodal

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extrasystole. (2) The occurrence in the same tracing of A-V nodal or ventricular beats with “retrograde” P’ waves identical to the postulated echo P’ wave reflects the latter’s retrograde character.

**Reciprocal Rhythm of A-V Nodal Origin.** In this rhythm an A-V nodal impulse is conducted anterogradely to the ventricles and retrogradely to the atria. The retrogradely conducted impulse also enters another A-V nodal pathway which enables it to return to and activate the ventricles once again. This results in the early inscription of another normal QRS complex, or a complex that is complicated by aberration (because of the relative prematurity). A prolonged retrograde conduction time is usually necessary so as to provide the lower A-V nodal tissues ample recovery time in order to enable them to respond to the returning impulse. Thus, the reciprocal beat is often associated with the last beat of a retrograde Wenckebach sequence—the beat with the longest retrograde conduction time.

**Reciprocal Rhythm of Ventricular Origin.** In this rhythm, a ventricular impulse, usually a ventricular extrasystole, is conducted retrogradely through the A-V node, and returns through another A-V nodal pathway to activate the ventricles a second time. The reciprocal beat is then reflected by a premature normal QRS complex. The reciprocal beat may, because of its prematurity, also be complicated by aberration.

**Analysis of Pauses and Late Events**

When regular rhythm is interrupted by a long or relatively long pause, the following mechanisms may be responsible:

1. The pause may be due to a “dropped” beat resulting from second-degree A-V block. When this occurs, a supraventricular P wave which is on time is not followed by a QRS complex.

2. The pause may be due to a “dropped” beat resulting from second-degree S-A block. When this occurs, a complete cycle or P-QRS complex is omitted, and the long pause is approximately twice that of the normal sinus cycle.

3. The pause may be due to a blocked or nonconducted atrial extrasystole. When this occurs, a very premature and bizarre P’ wave is not followed by a QRS complex. It may, however, be difficult to see the premature P’ wave which may be hidden within or partially obscured by the preceding T wave. Thus, when a relatively long pause interrupts regular rhythm the preceding T wave should be carefully scrutinized for the possibility of a superimposed P’ wave of a very early atrial extrasystole.

4. The pause may be due to a blocked or nonconducted A-V nodal extrasystole. The diagnosis is very difficult since, in contrast to a blocked atrial extrasystole, no P’ wave is evident. Diagnosis is based on the effect of interpolation, namely a prolongation of the ensuing P-R interval—a form of concealed conduction.14

5. The pause may be due to a concealed A-V nodal capture.

6. The long pause may be an expression of marked sinus arrhythmia. It then occurs during a gradual waxing and waning of R-R intervals.

**The Late Event: Escape Beats**

A long pause is not infrequently terminated by an escape beat—an ectopic discharge—which may be atrial, A-V nodal, or ventricular in origin. This occurs because of nonarrival (default) of the sinus impulse, and which may, for example, be due to sinus bradycardia, S-A, or A-V block. The escape beat is always late and has the features of an ectopic atrial deflection, an A-V nodal deflection, or a ventricular deflection. The escape may occur for a single beat and then be usurped once again by the sinus pacemaker which has recovered from its default, or, when the default of the supraventricular pacemaker persists for a longer period, the escape may continue in the form of an escape rhythm: idionodal or idioventricular rhythm. The escape rhythm is always a slow rhythm.

**Analysis of “Unexpected” or “Paradoxic” Events**

The “Supernormal” Phase of Conduction

The supernormal phase of conduction is a paradoxical or unexpected event which is occasionally seen during periods of high-grade A-V block, “complete” A-V block, or indeed any form of complete A-V dissociation with a relatively slow subsidiary pacemaker. The phenomenon is manifested as with momentary A-V conduction during “complete” or high-grade A-V block. A relatively early beat is conducted, constituting a capture beat, whereas relatively late impulses, which should encounter even more responsive tissue, are blocked.

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Such a paradoxical capture by an early supraventricular impulse associated with the block of later supraventricular impulses is termed the supernormal phase of conduction.\textsuperscript{15} The term supernormal phase is in a sense a misnomer, because conduction is not better than normal but is usually better than the prevailing depressed state of conduction. Recently, Moe, Childers, and Merideth (1968)\textsuperscript{16} have shown that many cases of so-called supernormal conduction can be explained on the logical basis of intricate forms of concealed conduction and the complex interplay of subsidiary and supraventricular pacemaker.

The Wedensky Phenomenon

A paradoxic event may be manifested in the form of a relatively long (0.75 sec) period of facilitated or enhanced conduction which occurs early during the escape or subsidiary cycle in high-grade A-V block. Conduction occurs during the period immediately after the escape beat, whereas the supraventricular impulses which occur later are blocked. The period of enhancement is relatively long when compared with the momentary enhancement which manifests as the supernormal phase of conduction.

The Wedensky phenomena have been invoked to explain this manifestation.\textsuperscript{17} Wedensky, in 1886,\textsuperscript{18} described a relatively long enhancing effect which followed the application of a maximal shock or stimulus to a neuromuscular preparation. A subthreshold stimulus, too small to evoke a response, could evoke a response if preceded by a maximal shock. Wedensky attributed this phenomenon to relatively prolonged lowered threshold of excitability following the maximal shock. This is termed the Wedensky effect. Wedensky (1903)\textsuperscript{19} further demonstrated, in a neural preparation, that an impulse arriving at a blocked zone enhanced the excitability of the nerve beyond the block. The condition of the nerve distal to the block is changed even though the enhancing impulse is not conducted through a blocked zone to depolarize the distal part of the nerve. This enhancement is probably brought about by a lowering of threshold, an electrotonic effect, so that a subthreshold stimulus becomes temporarily threshold.

These principles may be applied to the paradox of relatively long periods of early enhancement during high-grade A-V block. The escape impulse arrives at the area of A-V block, and by Wedensky facilitation (probably electrotonus) lowers the threshold of the area beyond the block. This enables an ensuing supraventricular impulse to be conducted anterogradely. Maintenance of A-V conduction, i.e. conduction of the second and ensuing sinus impulse, is due to the Wedensky effect. Each conducted sinus impulse is likened to the experimental maximal induction shock, which temporarily lowers the threshold of the depressed area within the A-V node. A physiologic phenomenon which must also be considered as a possible explanation for this manifestation is fluctuating vagal effect from the escape beats.

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