

EDITORIAL COMMENT

Clinical Application of the Electrocardiogram*

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In this issue of the *Journal*, Charles Fisch (1) presents an excellent overview of the birth and development of the electrocardiograph machine. His article provides insightful commentary by an expert electrocardiographer who has spent a professional lifetime using that device to make

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fundamental observations about cardiac pathophysiology. The purpose of this editorial comment is to expand the findings covered in that review to demonstrate how clinical investigators have applied those advances in electrocardiography toward an understanding of the physiology of cardiac arrhythmias (2–4). This knowledge has enabled the *practicing* cardiologist, armed with no more than a pair of calipers and deductive reasoning, to use the 12-lead electrocardiogram (ECG) to diagnose arrhythmias in ways that rival the clinical electrophysiologist. In fact, the early electrocardiographers did just that, and the advent of invasive clinical electrophysiology has proven their insight correct in many instances, ranging from observations about concealed conduction, accessory pathway (Wolff-Parkinson-White) tachycardias, aberration, focal causes of atrial fibrillation, ventricular tachycardias, and others. Let us consider a few of the lessons that they learned.

Interpretation of supraventricular tachycardias. Based on their ECG presentation, paroxysmal supraventricular tachycardias (SVTs) can be classified into two major groups: short RP and long RP tachycardias, that is, the P wave during the SVT occurs either in the first or second half of the tachycardia cycle. Because the PR interval is inversely related to the RP interval, short RP tachycardias have a long PR interval, and long RP tachycardias have a short PR interval. Although exceptions to any classification are to be expected, this grouping includes the vast majority of SVTs and enables the clinician to deduce the SVT mechanism with relative ease and accuracy. From that information, appropriate therapeutic decisions follow.

SHORT RP SVTs. These SVTs are defined by having atrial activity 1) obscured by the QRS complex because of the simultaneous inscription of both, 2) occurring in the terminal portion of the QRS complex and often giving the appearance of an R' in lead V₁, or 3) present in the ST segment. Hence, the interval from the onset of the QRS complex to the onset of the P wave is short—"short RP SVT"—and in fact can be a negative value when the retrograde P wave is buried in the beginning portion of the QRS complex. Invasive electrophysiologic studies have shown that the most likely SVT for the first and second examples is atrioventricular nodal reentrant tachycardia (AVNRT), using the slow AV nodal pathway anterogradely and the fast AV nodal pathway retrogradely. An SVT traveling to the ventricle over the AV node and back to the atrium over an accessory pathway, called "atrioventricular reentrant tachycardia" (AVRT; Wolff-Parkinson-White syndrome), is the most likely cause of the third example, and less commonly the second. Thus, careful study of the ECG can explain these very common SVTs.

One can become even more sophisticated in diagnosing the SVT if an episode of functional bundle branch block (FBBB) also occurs. Prolongation of the SVT cycle length during FBBB is most consistent with an AVRT and the location of the accessory in the same ventricle that gave rise to the FBBB. Thus, prolongation of the SVT cycle length during a period of a functional *left* bundle branch block (LBBB) would be found during AVRT due to retrograde conduction over a *left*-sided accessory pathway; the same reasoning applies to functional right BBB (RBBB) and a *right*-sided accessory pathway. The cycle length of the SVT prolongs because, during the FBBB, the anterograde impulse must first activate the ventricle contralateral to the site of FBBB and then travel across the interventricular septum to reach the opposite ventricle, gain access to the accessory pathway, and activate the atrium retrogradely. Failure of the FBBB to prolong the cycle length of the AVRT occurs when the accessory pathway is located contralateral to the ventricle with the FBBB, in many AVRTs due to septal accessory pathways, and during non-WPW forms of SVT. The ECG algorithms based on the morphology of the delta wave of the WPW complex can also be used to determine the location of the accessory pathway.

LONG RP SVTs. These SVTs are characterized electrocardiographically by atrial activity located "just before" the next QRS complex, so that the P wave is located in the second half of the tachycardia cycle at a conductible PR interval of ≤ 300 ms or so. As such, this SVT creates a long interval from the preceding QRS complex to the next P wave. This type of ECG presentation is typical of an atrial tachycardia and two other SVTs. One is an unusual form of AVRT comprised of a slowly conducting accessory pathway that creates an incessant SVT, which pauses briefly for a few sinus beats and then resumes. This is called the "permanent

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form of junctional reciprocating tachycardia," or PJRT, and is important clinically because the incessant nature of the SVT can cause a tachycardia-induced cardiomyopathy. The impulse in PJRT travels to the ventricles over the normal conducting system and then back to the atria over the slowly conducting accessory pathway. The third type of long RP SVT is an unusual form of AVNRT, during which the impulse travels to the ventricle over the fast-conducting AV nodal pathway, and retrogradely over the slow-conducting pathway. In essence, it is the reverse route traveled by the usual type of AVNRT noted earlier in the text.

INTERMEDIATE RP TACHYCARDIA. Several infrequently occurring SVTs can give rise to tachycardias that have PR and RP intervals of about the same duration so that the P wave is found midway in the tachycardia cycle. They include AVNRT when the impulse travels over two slowly conducting pathways, unusual forms of AVRTs, and some atrial tachycardias.

SPONTANEOUS ONSET OR TERMINATION OF THE SVT. A sustained SVT initiated by a premature atrial complex (PAC) causing block in an accessory pathway is most likely an AVRT, whereas a sustained SVT started by a PAC that significantly prolongs the PR interval is probably AVNRT.

Documenting the termination of the SVT can also provide a clue as to its mechanism. An SVT always stopping with a P wave rather than a QRS complex as the electrical event last inscribed in the ECG is unlikely to be an atrial tachycardia, because the atrial focus would always have to block en route to the ventricle at the same time it stopped discharging, an unlikely set of coincidences to happen repeatedly. Far more likely is either AVNRT or AVRT, during which atrial activity blocks before reaching the ventricle, thus interrupting the reentrant loop and terminating the tachycardia. Similarly, an SVT that persists uninterruptedly despite blocked P waves is almost certainly an atrial tachycardia, rarely AVNRT, and *never* the usual forms of AVRT.

Atrial flutter and atrial fibrillation. **ATRIAL FLUTTER.** Although atrial flutter can originate in either atrium, frequently due to reentry around an area of fibrosis, the most common form of atrial flutter is attributable to a large reentrant loop confined to the right atrium that travels in a counterclockwise direction, caudocranially in the interatrial septum and craniocaudally in the right atrial free wall. This common form of atrial flutter is characterized electrocardiographically by sawtooth flutter waves that are negative in leads II, III, and aVF and show continual electrical activity (lack of an isoelectric interval between flutter waves). In this type of atrial flutter, an area of slow conduction is present in the posterolateral to posteromedial inferior right atrium, between the tricuspid valve annulus and the inferior vena cava orifice. This critical isthmus of slowed conduction is crucial to the maintenance of the reentrant conduction and represents the site of successful ablation that eliminates atrial flutter.

ATRIAL FIBRILLATION. The exciting new development in our thinking about atrial fibrillation is the recognition that

discharge from a single rapidly firing focus can precipitate and perpetuate this tachycardia (5). The controversy over whether multiple wavelets of reentry cause atrial fibrillation or a single rapidly firing focus causes atrial fibrillation has existed for 50 years. Laboratory and clinical data have supported both concepts, and very probably both forms exist clinically. However, the fact that a single focus can definitely cause some forms of atrial fibrillation facilitates catheter ablation to eliminate the arrhythmia and, thus, offers a cure for some patients. Patients with this type of atrial fibrillation are frequently young with structurally normal hearts, have paroxysmal episodes, and the ECG often shows bursts of an atrial tachycardia or frequent premature atrial complexes, sometimes initiating the atrial fibrillation. Fascinatingly, the atrial focus is most commonly located in the pulmonary veins, more often in the upper than in the lower veins, and can be ablated by transeptal catheter techniques. The P-wave morphology of the PAC or atrial tachycardia can be used to help locate the responsible pulmonary vein.

Interpretation of wide complex tachycardias. Distinguishing aberrant ventricular conduction of supraventricular origin from a ventricular tachycardia (VT) is critical for making appropriate patient management decisions. As with the analysis of SVTs, the 12-lead ECG can provide clues—based on the presence of specific QRS cycle sequences, QRS morphology, and the relationship of P waves to QRS complexes—that enable the clinician in most instances to diagnose SVT versus VT.

Fusion and capture complexes, and the presence of AV dissociation, provide the strongest ECG evidence of VT; a tachycardia consistently starting with a premature P wave, having a very short (100 ms) RP interval, a QRS configuration known to be supraventricular, P-wave and QRS complex linked to suggest that ventricular activation depends on atrial discharge, and slowing or termination of the tachycardia by vagal maneuvers, are all consistent with an SVT.

Analysis of specific QRS contours during the tachycardia can be helpful in diagnosing VT, and include a QRS with left axis deviation and a duration exceeding 140 ms, with a normal duration during sinus rhythm. During VT with a RBBB appearance, the QRS complex in lead V_1 is often monophasic or biphasic with an initial deflection different from that of the sinus-initiated complex, and it has a small R and large S wave or a QS pattern in lead V_6 . During an LBBB pattern, the QRS axis can be rightward and have a broad, prolonged (exceeding 40 ms) R wave in lead V_1 and a small Q-large R wave or QS pattern in lead V_6 . Aberrant supraventricular complexes often have an RSR' in lead V_1 and are initiated by a long-short cycle sequence. A wide complex tachycardia with irregular intervals, and rates exceeding 200/min, should raise the question of atrial fibrillation and conduction over an accessory pathway. Other ECG features can be helpful, and various algorithms have been applied. It is important to stress that exceptions to all of the above criteria exist, and one must rely on sound

clinical judgment and consider the ECG only one of several helpful ancillary tests.

LESS COMMON FORMS OF VTS. The 12-lead ECG during sinus rhythm or VT often contains distinctive features that provide insight into the nature of the underlying heart disease, the location of the VT (important for catheter ablation), or the mechanism responsible for the VT. Each of these factors can be helpful in making therapeutic decisions and in advising the patient about his or her prognosis. Although in-depth description of these characteristics can be found in more comprehensive reports (3,4), several aspects will be reviewed here.

The presence of an RSR' in lead V₁ associated with upward coving of the ST segment in leads V₁ through V₃ and a normal QT interval may indicate that the patient has the newly described Brugada syndrome, an inherited genetic disorder affecting the repolarization function of the sodium channel (6). Interestingly, a slightly different abnormality of the same gene, which in the Brugada syndrome does not prolong repolarization, is also responsible for one of the long QT syndromes (LQT3). The Brugada syndrome is characterized clinically by ventricular tachyarrhythmias and sudden death, and it may be responsible for the so-called "night terrors" of many Asian cultures (3). The symptomatic patient requires treatment with an implantable cardioverter defibrillator (ICD).

Finding a prolonged QT interval during sinus rhythm signifies an inherited or acquired repolarization disorder called the "long QT syndrome" (LQTS) (7). The inherited form of LQTS has as many as six different responsible genotypes with several phenotypes, and can predispose to a specific kind of VT, called "torsade de pointes." This VT has a characteristic polymorphic ECG appearance of the QRS, which appears to be "twisting about on its points." A fairly specific electrophysiologic mechanism, "early afterdepolarizations," caused by a malfunctioning sodium or potassium channel, appears to be responsible for torsade de pointes, which can be treated by drugs or an ICD. An unknown number of youngsters present with seizures due to hypotension from the torsade de pointes, and they are misdiagnosed with epilepsy. Some patients predisposed to drug-induced, acquired LQTS may actually have an inherited form that makes them vulnerable to the QT-prolonging, proarrhythmic effects of many drugs.

Several types of cardiomyopathies can cause ventricular tachyarrhythmias and may demonstrate uniquely appearing ECGs during sinus rhythm. For example, arrhythmogenic right ventricular cardiomyopathy (8) can be characterized during sinus rhythm by an epsilon wave (a notch on the terminal portion of the QRS in lead V₁ due to delayed right ventricular activation) and T-wave negativity in the anterior precordial leads. This disorder, due to fatty replacement of the right and, less commonly, left ventricular myocardium, can result in lethal ventricular arrhythmias. Patients with hypertrophic cardiomyopathy (9) can exhibit large voltages consistent with ventricular hypertrophy and deep Q waves often confused with myocardial infarction, whereas patients

who have muscular dystrophy (10) can have tall R waves in the anterior precordial leads reminiscent of a true posterior myocardial infarction. Patients with a dilated cardiomyopathy can have VT due to bundle branch reentry that often has an LBBB contour and can be treated with radiofrequency catheter ablation.

Finally, two types of VTs that occur in patients with apparently structurally normal hearts include those originating in the right ventricular outflow tract that have a LBBB-inferior axis morphology, and those coming from the left ventricular septum that have a RBBB and left axis deviation contour. Both are relatively easily eliminated with radiofrequency catheter ablation.

Conclusions. It is clear from this brief accounting that the ECG has become an essential tool in diagnosing cardiac arrhythmias. Naturally, the ECG has many other important clinical applications not reviewed in this article. The ECG interpretation of arrhythmias has been greatly aided by the work of electrophysiologists who, over the last 30 years, have forged an impressive foundation of knowledge using invasive techniques. Today, a skilled electrocardiographer wielding a pair of calipers can feel quite comfortable in understanding a broad array of arrhythmias based on a careful study of a 12-lead ECG.

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