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Usefulness of the Electrocardiogram in Diagnosing Mechanisms of Tachycardia

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Abstract. The electrocardiogram, despite its simplistic technological composition, remains a valuable tool in the diagnosis of pediatric arrythmias. In this article the characteristic features of different tachycardias are reviewed.

Key words: Electrocardiogram—Pediatrics—Arrhythmia—Tachycardia

It has been 100 years since Einthoven recorded the electrical activity of the heart using his string galvanometer. During the past century his method has rapidly evolved into the science of modern electrocardiography. Early pioneers also described arrhythmia mechanisms which have since been confirmed by intracardiac recordings. Reentry, for example, was described by Mayer in 1906 and 1908 [11, 12], by Mines in 1913 [13], and by Lewis in 1920 [7]. Modern-day invasive electrophysiology has furthered our understanding, confirming and clarifying the electrocardiographic (ECG) findings so that most diagnoses may now be made without invasive procedures.

Tachycardias may occur as a result of reentry, abnormal automaticity, and triggered activity. Reentry is the propagation of an impulse through tissue already activated by that same impulse [10]. Most tachyarrhythmias in the young result from a reentrant mechanism; for patients younger than 12 years an accessory connection (AC) is used, and atrioventricular (AV) nodal reentry (AVNRT) is more common in adolescence [3, 4]. Macro- and microatrial reentrant circuits may occur around surgical incisions, and most occur following extensive atrial surgery such as in the Fontan, Mustard, and Senning procedures [14, 17]. Reentrant arrhythmias are commonly initiated by an ectopic beat and often terminate with a P wave due to block in the AV node. Abnormal automaticity results from rapid phase 4 depolarization causing spontaneous impulses from tissues that may not normally possess this ability [10]. This may occur at any level (atrium, AV junction, and ventricle) and show rate variability with "warm-up" at initiation and "cooldown" prior to termination. They rarely terminate with AV block. Triggered activity occurs as a result of afterdepolarizations (membrane potentials that occur after the normal action potential), triggering tachycardias especially in those patients with abnormalities of repolarization such as the long QT syndrome [10].

An approach to the ECG interpretation of arrhythmias is summarized in Table 1. In order to diagnose the tachycardia mechanism, a 12-lead recording is preferable. Using rhythm strips, P waves may not be visible and the width of the QRS may be underestimated, with the initiation and termination being isoelectric in some leads.

A 12-lead ECG in sinus rhythm is extremely useful in patients with a history of or presenting with an unknown tachycardia [8]. The presence of a short PR interval and delta wave of ventricular preexcitation or varying PR intervals suggestive of dual AV nodal physiology indicates a potential reentrant circuit. Many infants and children with tachyarrhythmias have structurally normal hearts, but those with underlying congenital heart disease, especially following surgical intervention, frequently have conduction delays.

From the initial examination of the tachycardia ECG, the tachycardia can be broadly categorized as either-narrow or wide QRS complex. In infants and children, either may be primarily atrial or ventricular, but, as with adults, a wide QRS complex tachycardia should be considered ventricular until proven otherwise. Supraventricular tachycardia (SVT) with aberrancy has been reported to occur in up to 20% of episodes of SVT in children. Narrow complex ventricular tachycardia may occur in infants.

Narrow Complex Tachycardias

SVTs may be classified as atrial, those using only atrial tissue for initiation and maintenance with the AV node as a bystander, and as AV junctional, those requiring the AV junction as a necessary component (Table 2). The

Table 1. Approach to electrocardiographic diagnosis of arrhythmia

Identify P wave	
Present	
Upright in II, III, AVF—sinus rhythm, sinus node or sinoatrial reentry, high ectopic focus	
Negative in II, III, AVF—AV reentry, low ectopic focus	
Absent: sinus node disease, AVNRT, JET, VT	
P wave morphology	
Normal: sinus tachycardia, sinus node reentry, sinoatrial reentry	
Abnormal: ectopic atrial, atrial flutter, atrial fibrillation	
Relationship of P to QRS	
Preceding: sinus, atrial tachycardias, long RP reentrant tachycardias	
After: AVNRT, AVRT, VT	
Presence of AV or VA block	
Atrial rate > ventricular rate: sinus, atrial tachycardias	
Ventricular rate > atrial rate: JET, VT	

AV, atrioventricular; AVRT, AV reentry tachycardia using accessory connection; AVNRT, AV node reentry tachycardia; JET, junctional ectopic tachycardia; VT, ventricular tachycardia.

Table 2. Classification of narrow complex tachycardias

Atrial
Sinus tachycardia, sinus node reentry, sinoatrial reentry
Ectopic atrial tachycardia, atrial fibrillation
Atrial reentrant tachycardia, atrial flutter
AV junctional
AV node reentrant tachycardia
AV reentrant tachycardia using accessory connection
Junctional ectopic tachycardia

identification of the P wave and its relationship to the QRS is crucial in defining the mechanism. Atrially driven tachycardias will have identifiable atrial activity preceding each QRS. Sometimes the P wave is not obvious and one needs to search for it. Figure 1 shows a sinus tachycardia with first-degree AV block in a postoperative patient. Careful examination reveals a P wave distorting the peak of the previous T wave, excluding the presumptive diagnosis of junctional ectopic tachycardia (JET), a narrow complex tachycardia with no visible P wave common in the postoperative situation [5]. The P wave morphology and axis help differentiate primary atrial tachycardias from AV junctional tachycardias [4, 9, 18, 19]. Abnormal morphology indicates a focus other than the sinus node [8, 9, 16, 18]. Three or more morphologies define chaotic or multifocal atrial rhythm. Positive P waves in the inferior leads indicate high to low activation and therefore an atrial tachycardia. Conversely, negative P waves indicate low to high activation, indicating reentry from a lower focus. Features of atrial ectopic tachycardia are shown in Figure 2. The P wave morphology is different for the sinus and remains similar to that at onset. Rate variability with the warm-up characteristic of an automatic focus is present, the PR interval lengthens with the faster cycle length, and the tachycardia terminates with a QRS complex. The presence of AV block may assist in making the diagnosis: Primary atrial tachycardias are usually fast and varying degrees of AV block may result. Figure 3 is an ECG of a newborn with atrial flutter at 480 beats per minute (bpm) and 2:1 AV block with one episode of 4:1 AV block. The lower ventricular rate allows the typical sawtooth atrial complex with no isoelectric baseline to be easily seen. In tachycardias in which the P wave is exactly halfway between the QRS complexes, atrial tachycardia with 2:1 AV block should be suspected.

AV junctional tachycardias may have no visible P waves (typical AVNRT and JET) or P waves may follow each QRS (accessory connection reentry). The duration of the RP interval is dependent on the speed of the retrograde conduction. In most reentrant tachycardias the RP interval is shorter than the PR. With slow retrograde conduction the P wave advances toward the next QRS, resulting in a longer RP than PR interval [6, 20]. A retrograde P wave with a short RP is distinctly visible following each QRS in the AC reentrant tachycardia shown in Fig. 4. Figure 5 is an example of a long RP tachycardia with the negative P waves in leads II, III, AVF, and V4-6, closer to the following QRS complex. This tachycardia, commonly known as permanent junctional reciprocating tachycardia (PJRT), is usually incessant, with rate variability due to catecholamine sensitivity. Atypical (fast–slow) AVNRT also has a long RP interval and a superior P wave axis [20]. Typical AVNRT (slow–fast) may be difficult to distinguish from JET in young children because no P wave is visible due to the fast rate and simultaneous activation of atrium and ventricle (Fig. 6). Because JET usually occurs in the very young or postoperatively, the circumstances may make the diagnosis. Adenosine is useful in this situation, terminating the AVNRT by blocking AV node conduction while having no effect on JET [5]. In older children and adults with AVNRT, P waves may be identified as part of the QRS complex giving a pseudo-S wave in lead III and rsr′ in V1.

Wide Complex Tachycardias

The differential diagnosis of a wide complex tachycardia is (1) ventricular tachycardia (VT), (2) SVT with aberrancy or preexisting bundle branch block, (3) antidromic SVT (antegrade down pathway, retrograde up AV node), and (4) atrial arrhythmia conducted via an accessory pathway (Table 3) [15, 21]. Ventricular tachycardia may result from reentry, abnormal automaticity, or triggered activity. Associated pathological findings (scarring postcardiac surgery, cardiomyopathies, and infantile Purkinje cell hamartomas) are commonly present but VT may occur in patients with structurally normal hearts. For pa-

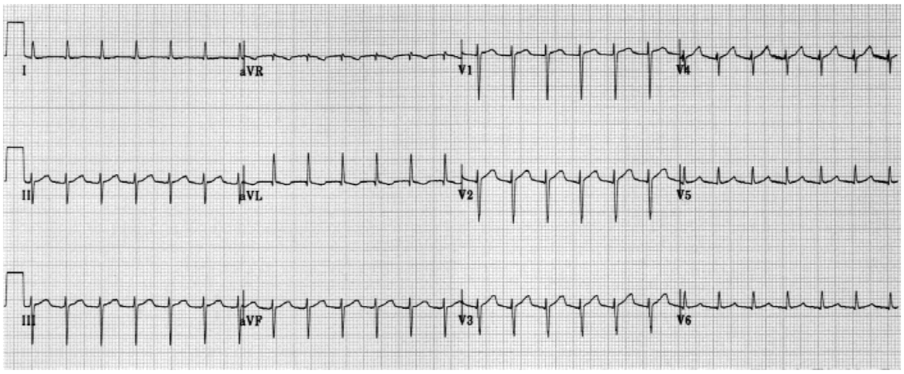

Fig. 1. ECG of a 4-year-old child a few hours following the Fontan operation for tricuspid atresia. Note the deformation on the apex of the T wave indicating the presence of a P wave. Sinus tachycardia 150 beats per minute; first-degree AV block.

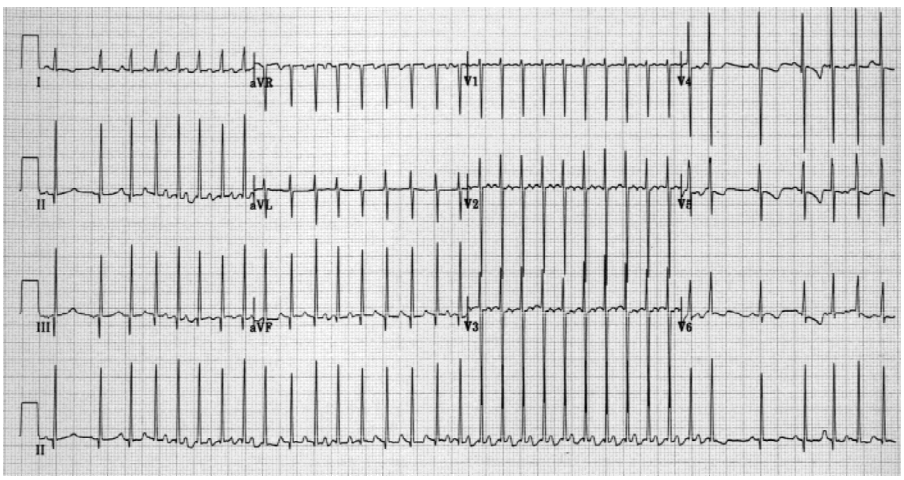

Fig. 2. A 4-day-old infant with critical aortic stenosis. This ECG shows an atrial ectopic focus initiating an atrial ectopic tachycardia (beat 3). The tachycardia shows rate variability and warm-up characteristics of an automatic focus, similar P waves throughout the tachycardia, and PR prolongation. It terminates with cessation of the ectopic focus.

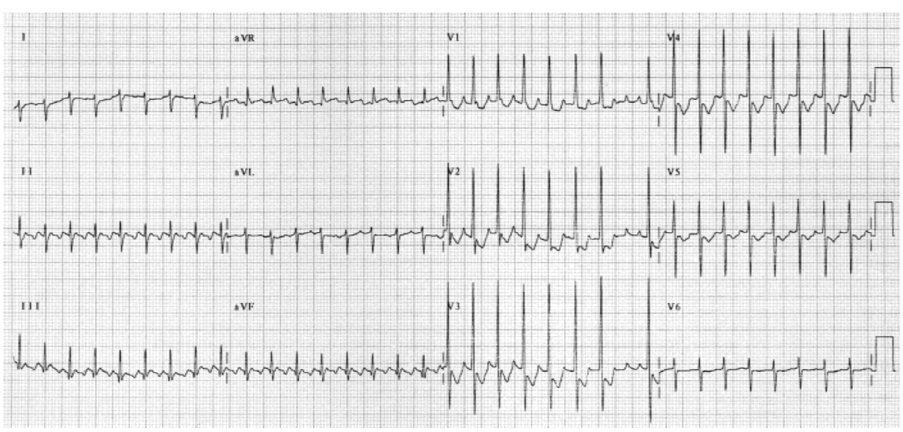

Fig. 3. Atrial flutter with 2:1 to 4:1 AV block in a 1-hour-old infant. Flutter rate is 480 beats per minute.

tients postcardiac surgery, the importance of having a reference 12-lead ECG in sinus rhythm is demonstrated in Fig. 6. A 10-year-old boy presented with this wide complex tachycardia (rate 244 bpm). Review of his sinus ECG revealed right bundle branch block with the morphology virtually identical to that of the tachycardia. Closer examination showed a 1:1 VA conduction with retrograde P waves following each QRS (best seen in lead V2), suggesting an AV reentrant mechanism rather than VT.

Several algorithms for wide complex tachycardias have been reported to differentiate VT from SVT with aberrancy (Tables 4 and 5). Characteristics of VT are classically (1) wide complex QRS with left axis devia-

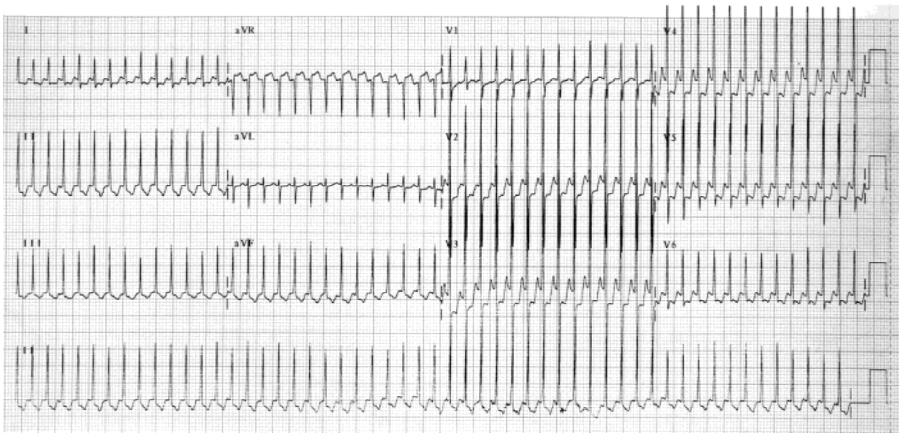

Fig. 4. Narrow complex tachycardia in an 11-month-old infant. Distinct retrograde P waves may be seen deforming the ST segment. An accessory pathway was present allowing reentrant tachycardia.

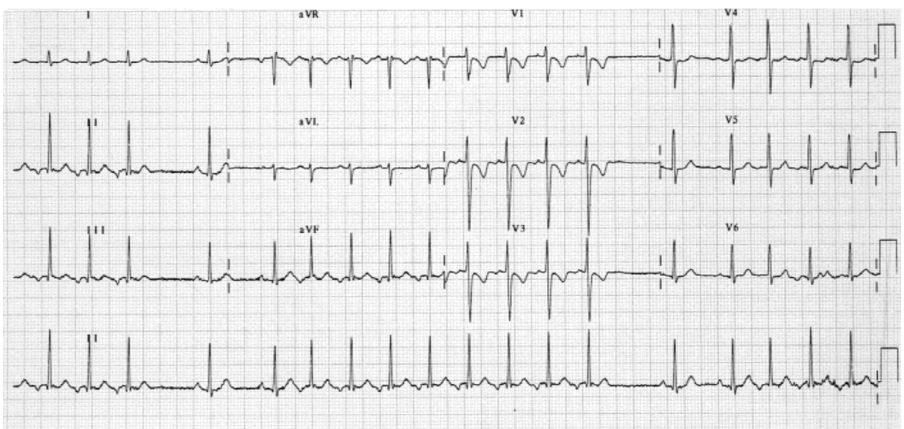

Fig. 5. Intermittent narrow complex tachycardia, rate 120 beats per minute with negative P waves in II, III, and aVF. Beats 4, 5, 14, and 15 are sinus. RP interval is longer than PR. Nine-year-old with PJRT.

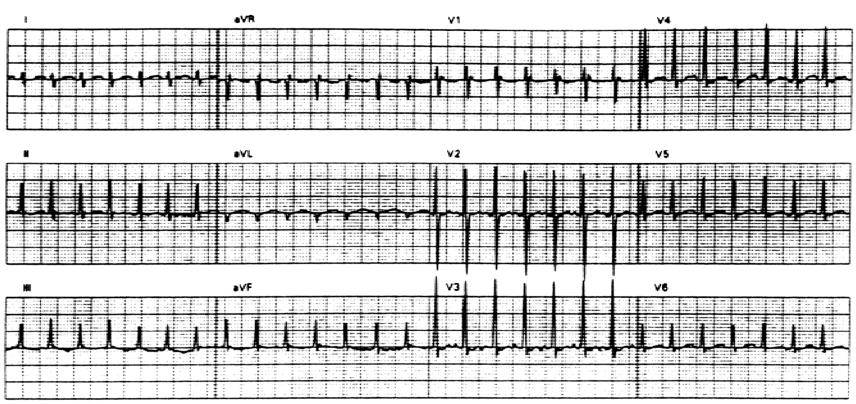

Fig. 6. AV node reentry tachycardia in a 12-year-old. The retrograde P wave is identifiable as a pseudo-S wave in lead III and rsr′ in V1.

Table 3. Differential diagnosis of wide complex tachycardia

Ventricular tachycardia
Supraventricular tachycardia (SVT) with intraventricular aberrant conduction
Antidromic SVT (antegrade accessory connection, retrograde up AV node)
Atrial arrhythmia conducted via accessory connection to ventricle

tion, (2) VA dissociation, and (3) fusion beats. Brugada et al. [2] and Antunes [1] developed an approach using four additional criteria: (1) the absence of RS pattern, (2) RS interval (onset of R to nadir of S) > 100 msec, (3) AV dissociation, (4) morphology of QRS in V_1 and V_6 [right bundle branch block (RBBB) or left bundle branch block]. Using these criteria in a stepwise approach in 384 patients with VT and 170 with SVT with aberrancy, the

Table 4. Wide complex tachycardia: criteria favoring VT[a]

Classical criteria	
	QRS duration >15 msec longer than 98th percentile for age
	Left axis deviation
	VA dissociation
	Fusion beats
Antunes–Brugada criteria—stepwise approach	
	Absence of RS complex in precordial leads
	R to S interval > 100 msec in one precordial lead
	More QRS complexes than P waves
	Morphology criteria for VT present in V1 and V6

VA, ventricular–atrial; VT, ventricular tachycardia.
[a] From Antunes et al. [1] and Brugada et al. [2].

Table 5. Morphological criteria favoring VT[a]

RBBB morphology	
	Triphasic pattern of QRS
	Left axis deviation
	R:S ratio <1 in V6
LBBB morphology	
	qR or QS in V6
	R wave in V1 or V2 >30 msec
	Any Q wave in V6
	R to nadir S duration >60 msec in V1, V2
	Notching on downstroke of S wave V1, V2

LBBB, left bundle branch block; RBBB, right bundle branch block; VT, ventricular tachycardia.
[a] From Antunes et al. [1] and Brugada et al. [2].

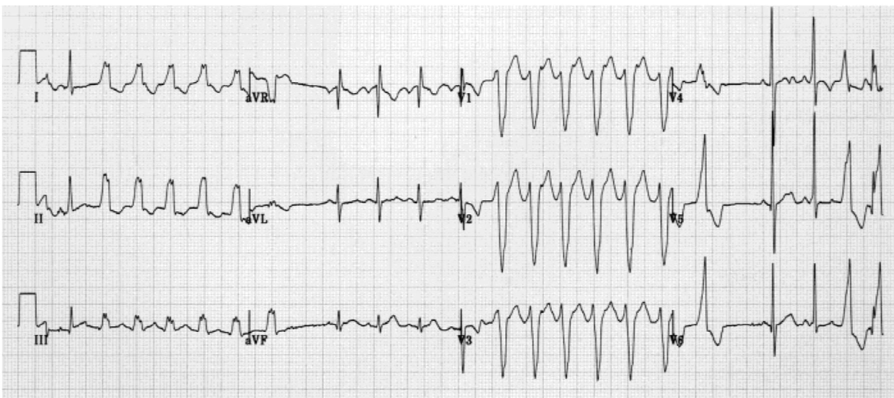

Fig. 7. (**A**) Wide complex QRS tachycardia 244 beats per minute. RS pattern $V_{2–6}$. RBBB morphology with 1:1 VA conduction (retrograde P waves visible in ST segment following each QRS). (**B**) Same patient in sinus rhythm showing RBBB morphology identical to that of the tachycardia. Ten-year-old child postventricular septal defect repair.

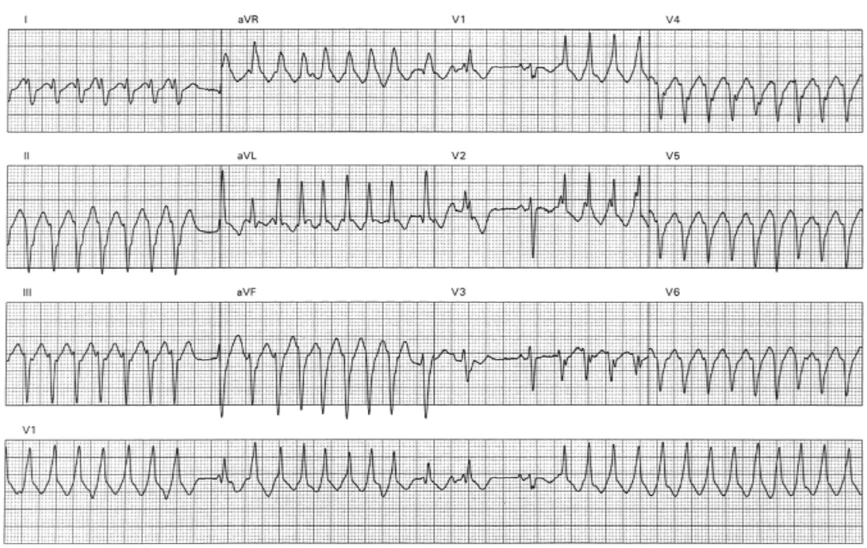

Fig. 8. A 1-year-old child with intermittent, accelerated ventricular rhythm. Rate is only slightly above sinus, RS pattern is present but RS interval is >100 msec, and VA dissociation is present.

correct diagnosis was made with a sensitivity of 0.987 and specificity of 0.96.

Accelerated ventricular rhythm, a slow form of VT that occurs in infants and older children with normal hearts, typically shows a variable rate less than 20% above sinus (Fig. 7). Exercise-induced VT (Fig. 8) resulting in syncope in a 13-year-old has a superior right axis with RBBB, suggesting a lower left ventricular septal focus of probable abnormal automaticity or reentry. Using the previous criteria for the identification of VT in Figs. 6–8, the correct diagnosis would be made in each tracing.

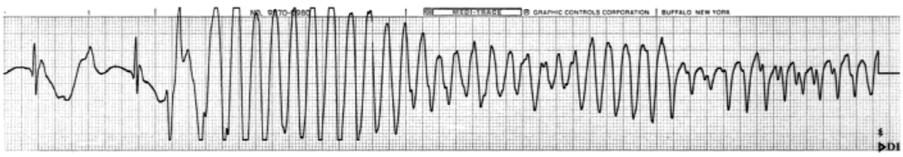

Fig. 9. Ventricular tachycardia in a 13-year-old with a history of exercise-induced syncope. RS pattern is absent, and VA dissociation and fusion beats are present. The tachycardia was ablated in the mid-lower septal region of the left ventricle.

After depolarizations, occurring in patients with abnormal repolarization as with the long QT syndrome and cardiomyopathies, may trigger ventricular tachycardias (Fig. 9). Abnormal T waves, T wave alternans, and long QT intervals in the resting ECG identify the patients at risk.

Summary

An accurate arrhythmia diagnosis may be made from most 12-lead ECGs if they are examined in detail and with a sleuth mentality, permitting the patient to receive appropriate emergency treatment, targeted medical therapy, and elective invasive procedures.

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