

# Supraventricular Tachycardia in Infants: Epidemiology and Clinical Management

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**Abstract:** Supraventricular tachycardias (SVTs) are observed in 0,1-0,4% of the paediatric population and represent an important clinical problem with related significant health and social issues. Most tachycardias are paroxysmal, being associated with sudden onset and termination, and only a relatively small number of them is permanent, namely chronic. Paroxysmal tachycardias, in addition, can be either sustained (lasting > 30 seconds) or non-sustained whenever their duration is less. Most SVTs are due to re-entry, and only atrial tachycardia and junctional ectopic tachycardia are caused by enhanced automaticity. Atrial tachycardia, however, can also be due, although rarely, to re-entry or to triggered activity. A prompt recognition of these arrhythmias in children by pediatric cardiologist is essential for a correct clinical management of the patients. In this review, the epidemiologic data regarding the SVTs in pediatric age are reported along with the description of the pathophysiological mechanisms and the analysis of electrocardiographic findings to be considered for a correct clinical diagnosis and a rational therapeutic approach to these arrhythmias.

**Key Words:** Supraventricular tachycardia, infants, epidemiology, management.

## INTRODUCTION

The term “tachycardia” indicates a heart rate that is excessive, namely higher than a conventional limit. Tachycardia is easily recognized in adults on the basis of a heart rate > 100 per minute; in infants, on the contrary, definition of tachycardia is less and less simple, since the “normal” heart rate is variable according to age [1]. Table 1 shows the different ranges of normal heart rates at rest, in the sleeping or awaking state, and during effort, in different paediatric ages. It is evident that a rate of 180 per minute in the sleeping state may be still considered as “normal” in a neonate, whereas it must be classified as “tachycardia” in a 4-month-old infant.

**Table 1. Normal Heart Rate According to Age**

	Rest (Awake)	Rest (Sleep)	Effort (Cry,Fever,...)
0-3 months	100-190	80-180	up to 200
3 months-2 years	80-150	70-120	up to 200
2-10 years	75-110	60-90	up to 180
> 10 years	55-90	50-90	up to 180

Once the presence of tachycardia has been established, the question arises of whether the heart rate increase is due to exaggerated sinus node automaticity (sinus tachycardia) or to ectopic origin of the impulse. The answer is usually based on the electrocardiogram, but it may often rely upon clinical observation: tachycardia occurring during fever, for example, is almost invariably of sinus origin.

When defining a cardiac rhythm as “tachycardia” it should be remembered that although in most cases atria and ventricles share the same rate, not infrequently atrial and ventricular rates differ from each other, as it commonly occurs, for example, in atrial flutter or fibrillation. The term “tachycardia” must be applied independent of the rate increase occurrence only in one heart chamber (atrium or ventricle) or in both of them.

An important clinical issue concerning tachycardias is their clinical presentation: most tachycardias are paroxysmal, being associated with sudden onset and termination, and only a relatively small number of them is permanent, namely chronic. Paroxysmal

tachycardias, in addition, can be either sustained (lasting > 30 seconds) or non-sustained whenever their duration is less.

In the paediatric age, the great majority of ectopic tachycardias (i.e., those originating outside the sinus node) are supraventricular, whereas ventricular ones are far more rare. The present review will be focused on clinical management of supraventricular tachycardias (SVTs) in paediatric patients, including the ECG diagnosis, while treatment of SVTs will be discussed in another section of the issue.

## EPIDEMIOLOGY

Supraventricular tachycardias are observed in 0,1-0,4% of the paediatric population, [2] and the majority of them are paroxysmal. Table 2 shows a list of SVTs: although sinus tachycardia is mentioned, this will not be hereunder taken into account, being a physiological, rather than pathological, phenomenon.

**Table 2. Supraventricular Tachycardias**

- Sinus tachycardia (ST)
- Atrial tachycardia (AT)
- Atrio-ventricular nodal re-entrant tachycardia (AVNRT)
- Atrio-ventricular re-entrant tachycardia (AVRT)
  - Orthodromic
  - Antidromic
- Atrio-ventricular re-entrant tachycardia due to a “slow” accessory pathway (PJRT)
- Junctional ectopic tachycardia (JET)
- Atrial flutter (AF)
- Atrial fibrillation

In paediatric age, SVT distribution is bimodal, with one peak in infancy and one at the age of 8-12 years [3]; the incidence of the different SVTs, however, is variable: the most common one is atrio-ventricular re-entrant tachycardia (AVRT), accounting for more or less 80% of all SVTs [4]. Atrial tachycardia (AT) is responsible for 5-20% of paediatric SVTs; this arrhythmia occurs either in paroxysmal or in permanent form, differing from AVRT, that is paroxysmal, apart from the so-called “permanent junctional reentrant tachycardia” (PJRT).

A-V nodal re-entrant tachycardia (AVNRT) is relatively rare in a paediatric population: it seldom occurs in neonates, and its inci-

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dence increases with aging, [4] being always lower with respect to AVRT (Fig. 1).

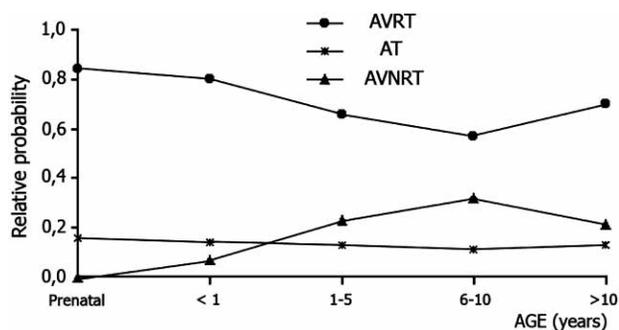


Fig. (1). Probability of supraventricular tachycardia type by age (modified from Kon Ko<sup>3</sup>).

Junctional ectopic tachycardia (JET) is an arrhythmia occurring almost exclusively in the paediatric age in its persistent or permanent form; it is unusual in adults, and not rarely occurs following cardiac surgery. Atrial flutter (AF) is extremely uncommon in the paediatric population [5-8], and atrial fibrillation is even more rare [1,9]. In the majority of cases, these arrhythmias are associated with the presence of structural heart disease.

Most episodes of SVT (about 40% of all SVTs in paediatric age) occur during the 1<sup>st</sup> month of life, being their prevalence less in the subsequent ages: 10% in the 1<sup>st</sup> year, 15% from the 1<sup>st</sup> to the 6<sup>th</sup> year. There is an inverse relationship between age of the first SVT attack and likelihood of recurrence: in 60% of patients presenting with SVT within the first 4-6 months, the attacks tend to disappear during the ensuing 6 months; almost all children with their first SVT episode after the 6<sup>th</sup> month, in contrast, have recurrences for at least 1 year, and 70% of them will have further episodes for more than 6 years [4,10-12]. On the other hand, the younger the patient with SVT, the higher the incidence of arrhythmia induced heart failure.

### MECHANISMS OF SUPRAVENTRICULAR TACHYCARDIA

Most SVTs (AVRT, AVNRT, PJRT, AF) are due to re-entry, and only AT and JET are caused by enhanced automaticity [13]. Atrial tachycardia, however, can also be due, although rarely, to re-entry or to triggered activity.

Re-entry is commonly initiated by a premature impulse, and a re-entrant SVT is often interrupted by a single premature impulse that succeeds in penetrating into the re-entry pathway, making this refractory, in such a way that the progression of the reciprocating impulse is hampered, and tachycardia ceases.

A-V nodal re-entrant tachycardia (AVNRT) is often caused by an atrial extrasystolic impulse conducted to the ventricles with a very long P-R interval; this is because the premature impulse is blocked anterogradely in the fast A-V nodal pathway, being conducted over the slow pathway only; the same impulse, then, may retrogradely traverse the fast pathway and, after reaching the atria, is again conducted to the ventricles over the slow pathway, and so on (Fig. 2A).

Atrio-ventricular re-entrant tachycardia (AVRT) due to an accessory A-V connection (the Kent bundle) is dependent upon a large circuit incorporating, apart from the accessory pathway itself, the A-V node, the His bundle, one ventricle and one atrium. In most cases, the initiating impulse (e.g., an atrial extrasystole) undergoes a block in the Kent bundle, being conducted to the ventricles over the A-V node and the His bundle only; the accessory pathway may, then, retrogradely conduct the impulse to the atria, initiating the circus movement responsible for tachycardia (Fig. 2B).

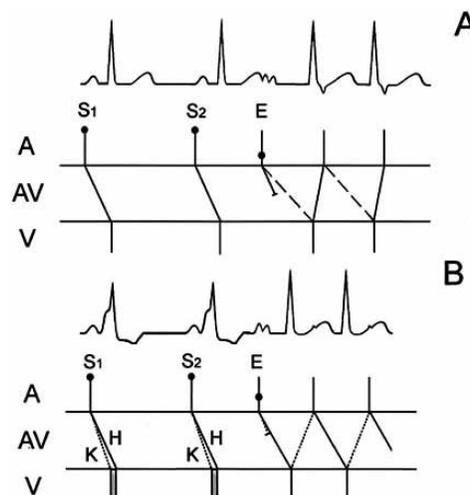


Fig. (2). Initiation of re-entrant supraventricular tachycardia by an atrial extrasystole. Panel A: A-V nodal re-entrant tachycardia. S1, S2: sinus impulses. E: atrial extrasystolic impulse. Continuous lines in the A-V section represent conduction over the fast A-V nodal pathway; dashed lines correspond to conduction over the slow A-V nodal pathway. Panel B: Atrio-ventricular re-entrant tachycardia. S1, S2: sinus impulses. E: atrial extrasystolic impulse. Continuous lines in the A-V section represent conduction over the His bundle (H); dotted lines correspond to conduction over the Kent bundle (K).

The great majority of AVRTs are orthodromic (namely, the impulse is conducted to the ventricles over the His bundle and returns to the atria through the Kent bundle); this results in narrow QRS complexes in the absence of pre-existing bundle branch block or aberrant conduction. Far more rarely, AVRT is antidromic, since conduction to the ventricles occurs over the accessory pathway and retrograde conduction to the atria over the A-V node. In such a case, QRS complexes are wide, as an expression of ventricular activation dependent exclusively on the Kent bundle. Antidromic AVRT can also occur in patients with more than one accessory pathway: under these circumstances, accessory pathways provide both the anterograde limb and the retrograde limb of the circuit.

A-V re-entry is also responsible for the so-called PJRT. The term "Permanent Junctional Reentrant Tachycardia" (PJRT) is misleading, and should be avoided, although very popular. This kind of SVT, thus, is not strictly "junctional", being an A-V reciprocating tachycardia whose circuit incorporates a slowly conducting accessory pathway [14,15]. This kind of tachycardia is often not initiated by a premature impulse, but by a sinus impulse, provided that the sinus cycle length is shorter than the anterograde refractory period of the accessory pathway [16]. Fig. (3) shows that in the first 2 beats the sinus impulse reaches the ventricles through the A-V node and the His bundle only, since conduction over the accessory pathway is

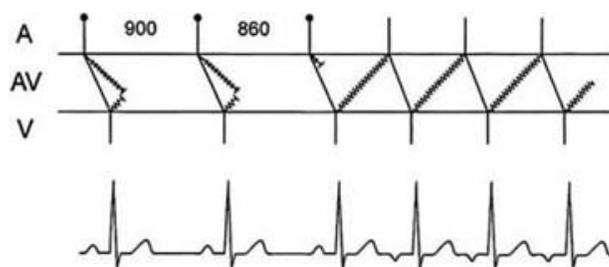


Fig. (3). Initiation of PJRT (A-V re-entrant tachycardia due to a slowly conducting accessory pathway) by a sinus impulse. Continuous lines in the A-V section represent conduction over the A-V node and the His bundle; zigzag lines correspond to conduction over the "slow" accessory pathway.

extremely slow: following ventricular depolarization, the impulse penetrates in retrograde direction the accessory pathway, colliding with the anterograde impulse. A slight shortening of the sinus cycle length from 900 to 860 msec (3<sup>rd</sup> beat) results in anterograde block of the sinus impulse in the accessory pathway; after ventricular activation, therefore, retrograde conduction from the ventricles to the atria over the accessory pathway is now possible, giving rise to the re-entrant tachycardia.

The re-entry mechanism is also responsible for atrial flutter (AF). In most cases, the re-entry circuit is confined to the right atrium, and the wave front circulates around the ostia of the caval veins, the interatrial septum and the lateral wall of the atrium. Some "barriers", as the crista terminalis, play a key role in permitting uninterrupted circulation of the impulse.

The mechanism of atrial fibrillation is more complex, and involves both automaticity and re-entry: the most recent point of view assumes that multiple foci, most of which are located within the pulmonary veins, fire irregularly, and initiate re-entry in multiple micro-circuits.

Automatic tachycardias originate from ectopic pacemakers that are able to fire with an unexpectedly high rate of discharge. Not rarely, automatic SVTs, arising from the atrium or the His bundle, are congenital.

### CLINICAL PRESENTATION

The infant or child with SVT may be asymptomatic, but not rarely there are clinical manifestations more or less suggestive of the rhythm disorder; at the other end of the spectrum, syncope or even acute hemodynamic collapse may occur, although very rarely. Children may complain of palpitation if they suffer from paroxysmal SVT; permanent SVT, in contrast, is seldom associated with palpitation but may provoke symptoms of heart failure [17,18]. In particular, AVRT (the most common paroxysmal SVT) is symptomatic unless its rate is relatively low; in contrast, re-entrant tachycardia due to a slow accessory pathway (PJRT), that is often permanent, is almost never associated with palpitation, but not rarely results in heart failure [19]. Atrial tachycardia is often symptomatic, particularly in its incessant form, which is resistant to conventional antiarrhythmic medication, [20] whereas junctional ectopic tachycardia (JET) is asymptomatic for palpitation in most cases. Atrial fibrillation in children is rare, and can be, at times, associated with preexcitation; in this condition, a very high ventricular rate may be attained, whenever the anterograde refractory period of the accessory pathway is short, resulting in significant risk for ventricular fibrillation and cardiac arrest.

Clinical investigation should start from the family history, that may reveal cases of arrhythmias or sudden juvenile death; in symptomatic children the history is very important to distinguish paroxysmal SVT from sinus tachycardia (Table 3). The neonate, on the other hand, is unable to express specific symptoms, and SVT episodes may occur unrecognized as long as heart failure becomes

manifest. In addition, the baby may show refusal to feed, failure to thrive, breathlessness. At times, a baby in sinus rhythm with severe acute heart failure or cardiogenic shock of unexplained origin suffers from repetitive long episodes of SVT with very high heart rate [21]; whenever during clinical examination tachycardia is absent, the diagnosis may only arise from prolonged monitoring.

Physical examination during tachycardia provides several keys, including 1) heart rate; 2) regularity or irregularity of tachycardia (an irregular rhythm suggests either atrial fibrillation or AT/AF with variable A-V conduction ratio); 3) duration of tachycardia episodes, in case of non sustained tachycardia; 4) signs of A-V dissociation (in particular, variable loudness of the 1<sup>st</sup> sound) leading to a diagnosis of ventricular tachycardia or junctional ectopic tachycardia; 5) signs of heart failure or cardiogenic shock; and 6) murmurs pinpointing to the presence of congenital heart disease, although detection and analysis of murmurs is difficult at high heart rates.

### ELECTROCARDIOGRAM

Analysis of the 12-lead ECG both during sinus rhythm and in tachycardia is the most important key to the diagnosis of SVT, and permits distinction among the various SVTs in the large majority of cases. Items to be taken into account include: 1) heart rate, 2) sustained versus non-sustained tachycardia, 3) width and duration of QRS complexes; 4) P wave analysis, answering the following questions: *a.* are P waves evident or not? *b.* what is P wave configuration and axis? *c.* is there any constant relationship between P waves and QRS complexes? *d.* what is the ratio between P waves and QRS complexes? 5) analysis of tachycardia initiation and termination; 6) response to vagal manoeuvres, 7) response to drugs.

The ECG during sinus rhythm may reveal preexcitation (short P-R interval, delta wave, wide QRS complex, ST-T wave changes) pointing out a diagnosis of AVRT (WPW syndrome).

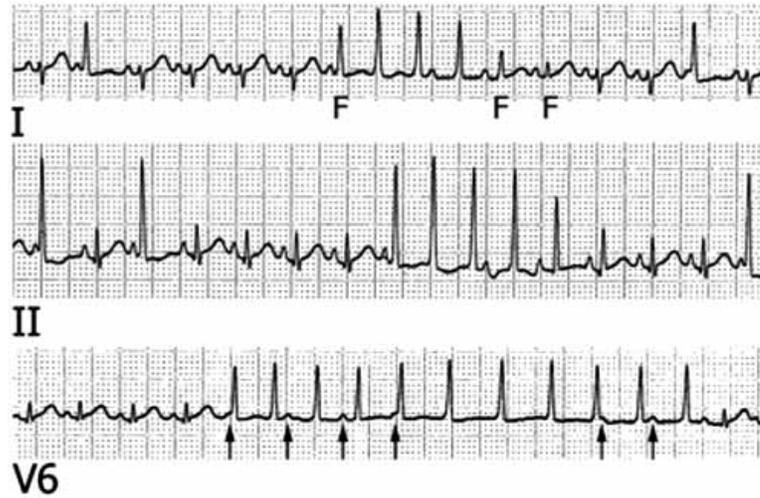
In the presence of wide QRS complexes tachycardia, a detailed analysis is necessary: first of all, it should be pointed out that in neonates or small babies the QRS duration is not very prolonged even during ventricular tachycardia (VT); distinction between STV and VT is based, like in adults, on several items including A-V dissociation, capture and fusion beats (Fig. 4), P/QRS ratio <1, precordial concordance, QRS complex configuration analysis in the chest leads to assess the presence or absence of signs suggestive of ectopy. Wide QRS complexes tachycardia can be also a preexcited tachycardia, either an antidromic form of AVRT or an AT/AF with conduction of the atrial impulse to the ventricles over an accessory pathway. An irregular wide QRS complex tachycardia is in most cases a preexcited atrial fibrillation.

### P Waves Search and Analysis in Narrow QRS Tachycardia

When the heart rate is high, P waves are usually barely discernible, since they are superimposed upon, or hidden within, the QRS complex or the T wave. It is, thus, necessary a detailed analy-

**Table 3. Distinction of Supraventricular Paroxysmal Tachycardia from Sinus Tachycardia on the Basis of History**

Question	Paroxysmal Tachycardia Likely	Paroxysmal Tachycardia Unlikely
How many episodes?	Several	One
Frequency of episodes?	Monthly	Daily
Duration?	5-30 minutes	Seconds, hours
Circumstances?	Any situation	Effort, night
How the child looks?	Pale,	Red
Site of palpitation?	Chest, neck	Chest
How does palpitation disappear?	Swallowing, vomiting, yawning	Rest



**Fig. (4).** Non-sustained ventricular tachycardia in a newborn (non-simultaneous strips of leads I, II and V6). Fusion beats are labelled F. Arrows point out sinus P waves dissociated from QRS complexes.

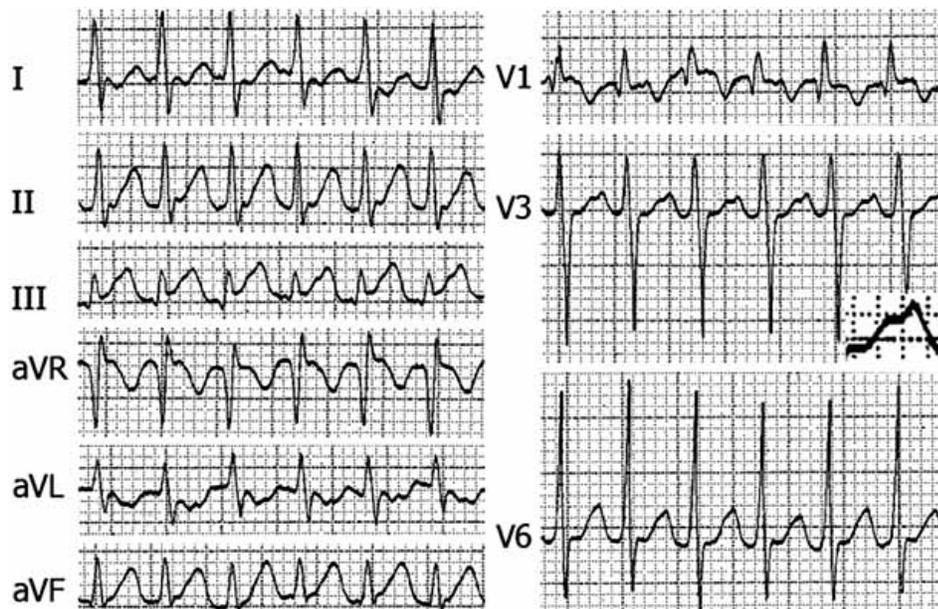
sis in order to discover, within the ST segment or the T wave, small notches or “humps” that reveal the presence of a P wave. A set of signs (defined as “leading signs”) have been proposed to recognize a P wave superimposed upon the T wave (Fig. 5) [22,23].



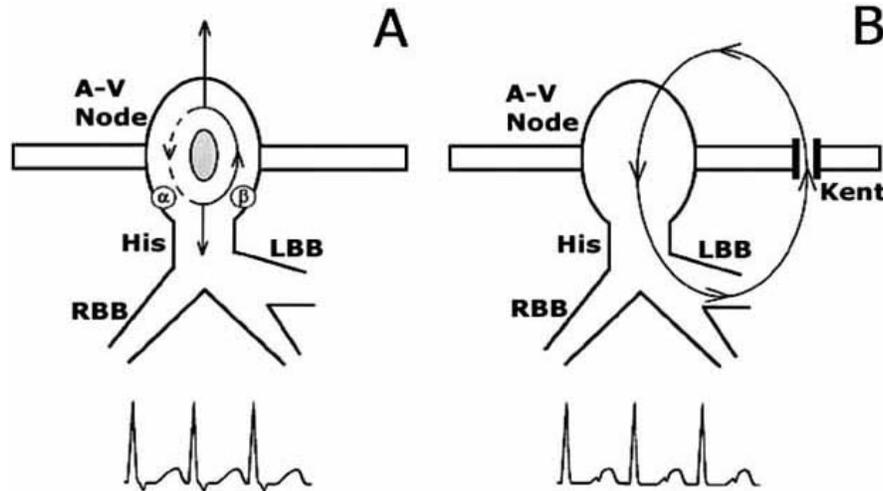
**Fig. (5).** “Leading signs” to recognize the presence of a P wave in narrow QRS complexes supraventricular tachycardia (modified from Oreto<sup>23</sup>).

Whenever, in a narrow QRS complexes SVT, a P wave is present within the T wave of all beats, the most likely diagnosis is AVRT (Fig. 6); this is because in SVT due to an accessory pathway atrial activation is never simultaneous with ventricular depolarization, but follows this with an interval  $\geq 70$  msec (Fig. 7B); accordingly, the P wave occurs during ventricular repolarization, and deforms the T wave. In contrast, AVNRT is characterized by more or less simultaneous atrial and ventricular depolarization (Fig. 7A), in such a way that P wave is totally hidden within the QRS complex or, less commonly, emerges from this as a pseudo-s wave (or pseudo-q wave) in the inferior leads and pseudo-r’ wave in lead V1. It should be pointed out, however, that not only AVRT, but also AT can be associated with P waves outside the QRS complex, within the T wave.

Whenever P waves are visible, it is useful to determine the direction of P vector on the frontal plane ( $\hat{A}P$ ). In the most common re-entrant SVTs (AVRT, AVNRT), this vector is directed superiorly, so that, apart from a few exceptions, the presence of positive P waves in the inferior leads (a feature that reveals a P wave axis



**Fig. (6).** Supraventricular tachycardia due to an accessory pathway (AVRT) in an infant. A biphasic T wave (“camel sign”) is evident in lead V3, particularly in the enlargement of T wave.



**Fig. (7).** Diagrams representing the mechanisms of A-V nodal re-entrant tachycardia (A) and A-V re-entrant tachycardia due to an accessory pathway (B).

pointing inferiorly) rules out both AVNRT and AVRT. In some cases, the P wave axis provides information not only on tachycardia mechanism, but also on specific characters of the arrhythmia; for example, an SVT with P wave axis directed superiorly and to the right is almost invariably an AVRT due to a left accessory pathway, and far less commonly an AT arising from the left atrium.

In narrow QRS tachycardia with A-V dissociation, P waves independent of ventricular complexes, and occurring with regular P-P intervals, are relatively easy to be discovered (Fig. 8); such a pattern immediately suggests a diagnosis of junctional tachycardia (JET), the only SVT in which A-V dissociation is possible [24-27]. The same diagnosis should be entertained in the presence of SVT with constant R-R intervals, irregular P-P intervals, superiorly directed P wave axis, and P/QRS ratio <1: these features demonstrate that tachycardia arises from the His bundle, but some impulses undergo a retrograde block in the A-V node and fail to reach the atria [24-27].



**Fig. (8).** Junctional ectopic tachycardia in a newborn. Arrows indicate sinus P waves dissociated from QRS complexes.

An extremely uncommon form of P/QRS ratio occurring during SVT is the 1:2 ratio. This is characterized by 2 ventricular responses for each atrial impulse, and in the majority of cases represents a manifestation of normal sinus rhythm with each sinus impulse conducted separately and in sequence to the ventricles by the fast and the slow A-V nodal pathways, so that 2 QRS complexes arise from any P wave [28].

In the presence of P/QRS ratio >1 (more P waves than QRS complexes), AT or AF must be diagnosed: AVRT cannot be associated with this condition, but requires without any exception 1:1 A-V ratio. In AVNRT, however, a 2:1 P/QRS ratio may occur, although rarely [29]. At times, rapid discharge from multiple atrial foci is responsible for chaotic (multifocal) AT; [30-32] this rare arrhythmia is characterized by P waves of 3 or more different configurations, with variable P-P, P-R and R-R intervals.

Atrial fibrillation does not represent, usually, a diagnostic problem, being associated with irregularly irregular R-R intervals and absence of clearly discernible P waves, replaced by “f” waves. When the ventricular response is very fast, however, the R-R interval variability can be so slight that the arrhythmia resembles any other form associated with constant ventricular intervals.

#### Analysis of Tachycardia Initiation and Termination

This is a useful tool in the differential diagnosis of SVTs. In most cases, AVNRT is initiated by an atrial extrasystole conducted with a very long P-R interval (Fig. 2A), whereas AVRT may be initiated by either an atrial or a ventricular extrasystole. An SVT interrupted by a single ventricular extrasystole is almost always an AVRT because: 1) interruption by a single premature stimulus strongly suggests a re-entry mechanism, and 2) a ventricular premature impulse is unlikely to enter retrogradely the A-V node in time to depolarize a section of the circuit, making this unsuitable for the further progression of the reciprocating wavefront. This is because a ventricular premature impulse originates relatively far from the A-V node, being separated from this by a bundle branch and the His bundle; in contrast, an ectopic ventricular impulse can arise from a site very close to the re-entry circuit of AVRT, particularly if the accessory pathway is ipsilateral to the ventricle responsible for the extrasystole.

A-V re-entrant tachycardia due to a slowly conducting accessory pathway is often initiated by a sinus impulse rather than by an extrasystole, a situation that cannot be found in any other tachycardia.

Junctional ectopic tachycardia often starts with a premature impulse nearly simultaneous with, and dissociated from, a sinus P wave; this pattern rules out any other SVT.

#### Response to Vagal Manoeuvres and to Drugs

In neonates and in babies, vagal stimulation is commonly performed by eliciting the “diving reflex”: this is obtained by cooling the face, either with immersion in cold water or with a bag (e.g., a surgical glove) filled of iced water [33,34]. Parasympathetic stimulation has two different scopes: interruption of a re-entrant SVT

and/or identification of the SVT mechanism. Re-entrant tachycardias can be interrupted by the intense vagal stimulation, resulting in increased A-V nodal refractoriness and possible block of the reciprocating impulse; this may happen for AVRT, AVNRT, PJRT. On the other hand, in AT or AF impairment of A-V nodal conduction induced by vagal stimulation often results in non-conducted atrial impulses, thereby clarifying the mechanism underlying SVT [35-36]. In JET with 1:1 retrograde conduction to the atria, vagal tone increase can result in retrograde block of some impulses arising from the His bundle pacemaker, making immediately clear the diagnosis.

The same effect exerted by vagal stimulation can be obtained by drugs that provoke a sudden and marked depression of A-V nodal conduction (Adenosine or ATP); in such a way it is possible either interruption of re-entrant SVTs or unmasking of P or F waves in AT or AF, respectively [35,36].

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