*Acta Paediatrica Hungarica, 25 (4), pp. 385—394 (1984)*

# The electrophysiological peculiarities of childhood tachyarrhythmias

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Electrophysiological analysis of 20 patients in child or adolescent age affected by paroxysmal tachycardia has been performed. In 16 cases the tachycardia was supraventricular and in 4 cases it was ventricular. Evaluation of the electrophysiological parameters revealed certain characteristics of childhood tachyarrhythmias, *viz. (1)* adequate sinus node function, absence of sinus dysfunction, *(2)* absence of brachycardia, *(3)* acceleration of anterograde and retrograde atrioventricular impulse conduction systems.

Traditional surface electrocardiography already permitted recognition of cardiac dysrhythmias and their types. In the last two decades development of electrophysiology has furnished innumerable data useful in the pathomechanism of various dysrhythmias [3, 7]. The bulk of these data has been obtained in adults, thus they cannot be adapted to the disturbances observed in children and newborns. Thereby we were prompted to examine the tachyarrhythmias of children in order to reveal eventual characteristics.

## Patients and Methods

During the period 1975—1983, 20 children or adolescents, were admitted to our department for electrophysiological evaluation. Their age was between 4 and 18 years. All suffered from severe, sometimes life-threatening attacks of tachyarrhythmia hardly responsive to drug therapy. There were 12 girls and 8 boys. All adolescents had the complaint for many years, patients whose arrhythmia began after the fourteenth birthday were excluded from this study.

A twelve-channel ECG, bidirectional chest X-ray, echocardiography and laboratory tests including determination of serum sodium and potassium were performed in all patients. Electrophysiological evaluation was carried out in alert patients or under ketamine anaesthesia. Antiarrhythmic drug treatment was suspended for the 24 hours preceding the procedure. Through the right and left femoral veins 2—4 multipolar electrode catheters were introduced into the heart: into the upper part of the right atrium (close to the sinus node in the angle between the vena cava superior and the right atrium), into the lower part of the right atrium, to the bundle of His and into the right ventricle. In case of an open foramen ovale a catheter was introduced into the left atrium and the left ventricle as well. If this was not possible, registration of the left atrium was carried out by an electrode introduced into the coronary sinus. The intracavital EG and His EG was registered and programmed electric stimulation was carried out by a Medtronic 5325 device. Table I offers a list of electrophysiological parameters examined in this study.

We studied the mode of precipitation and termination of the tachycardia by atrial and ventricular electric stimulation. The stimulation was carried out by applying single ($S_1$ technique), multiple ($S_{1-2}$ technique) or continuous stimuli.

In 10 patients the intrinsic heart rate was determined by autonomous blockade.

TABLE I

Electrophysiological parameters measured in the study

— cycle length of the basal rhythm and the tachycardia (CL)
— total atrioventricular conduction time (P-Q interval)
— duration of impulse conduction within the Aschoff-Tawara node (A-H interval)
— duration of impulse conduction within the His-Purkinje system (H-V interval)
— breadth of QRS complex
— duration of the electrical systole of the heart (Q-T interval)
— relationship of the ventricular effective refractory period to the duration of electrical systole of the heart (VERP/Q-T quotient)
— functional condition of the atrioventricular conduction system during atrial electrical stimulation performed with increasing frequency
— sinus node recovery time (SNRT), corrected for sinus node frequency (CSNRT)
— sinoatrial conduction time (SACT)
— atrial effective refractory period (AERP), atrial functional refractory period (AFRP) by early stimulus technique
— atrioventricular node refractory time (AVNRT)
— ventricular effective refractory period (VERP)
— retrograde conduction (occurring during ventricular stimulation)
— effect of carotis sinus compression (CSC) on the above parameters

TABLE II

| | Age (yr) sex | Diagnosis | TC frequency beat/min | AERP ms | VERP ms | Wenckoback-point anterograde | Wenckoback-point retrograde | Q-T |
|---|---|---|---|---|---|---|---|---|
| 1 | 12 ♀ | AF | 352—176 | 170 | 190 | 178 | — | normal |
| 2 | 11 ♀ | AF + fibr. | 400—150 | 150 | 160 | 174 | — | normal |
| 3 | 13 ♂ | ATA | 220 | 160 | 180 | 200 | 210 | normal |
| 4 | 17 ♀ | ATA | 200 | 150 | 170 | 180 | — | normal |
| 5 | 13 ♀ | ATA | 200—300 | 140 | 200 | 220 | — | normal |
| 6 | 17 ♂ | ATA | 200 | 140 | 180 | 210 | 260 | normal |
| 7 | 14 ♀ | ATA | 180 | 160 | 180 | 200 | 250 | normal |
| 8 | 8 ♂ | ATA | 186 | 180 | 200 | 206 | — | normal |
| 9 | 15 ♀ | ATA | 168 | 250 | 250 | 188 | 230 | normal |
| 10 | 5 ♀ | ATA | 160 | 160 | 170 | 168 | 220 | normal |
| 11 | 9 ♂ | ATA | 120 | 210 | 220 | 132 | — | normal |
| 12 | 12 ♂ | ATA | — | 150 | 160 | 110 | — | normal |
| 13 | 10 ♀ | ATA | 220—110 | — | — | 150 | 280 | normal |
| 14 | 16 ♂ | JT | 120—140 | 120 | 180 | 180 | 190 | normal |
| 15 | 13 ♂ | JT | 160—178 | 220 | 210 | 180 | — | normal |
| 16 | 4 ♀ | JT | 176 | 170 | 200 | 174 | 220 | normal |
| 17 | 5 ♀ | VT | 140 | 140 | 170 | — | — | 315 ms × 0.54 |
| 18 | 18 ♀ | VT | 160 | 160 | 170 | — | — | 340 ms × 0.5 |
| 19 | 13 ♀ | VT | — | 210 | 200 | 200 | — | normal |
| 20 | 10 ♂ | VT | 150—180 | 220 | 220 | 180 | 200 | normal |

AF = atrial flutter
ATA = atrial tachyarrhythmia
JT = junctional tachycardia
VT = ventricular tachycardia
x = VERP/Q-T
TC = tachycardia

## Results

The results obtained in the 20 patients are shown in Table II. The tachyarrhythmia proved to be supraventricular in 16 cases (SVTA), in four patients it was of ventricular origin (VT). Tables III and IV show the classification of paroxysmal tachycardia into types. In two patients the supraventricular tachyarrhythmia manifested as paroxysmal atrial flutter (AF). In one case the atrial frequency was 352/min, in the other 300/min. In the first case (Case 1) AF alternated with normal sinus rhythm, in the other case (Case 2) AF went over to atrial fibrillation during electric stimulation. Here the basic rhythm was furnished by a "wandering pacemaker". The CSNRT value exhibited a pathological prolongation (800 ms). In Case 1, AF could be abolished by $S_{1-2}$ programmed stimulation while in Case 2 only DC shock proved to be effective. During the attack of both patients a 2 : 1 atrioventricular block was present.

Atrial paroxysmal tachycardia (ATA) was observed in 11 patients. In seven patients a reentry mechanism could be demonstrated (Figure 1), in four patients (Cases 4, 5, 12

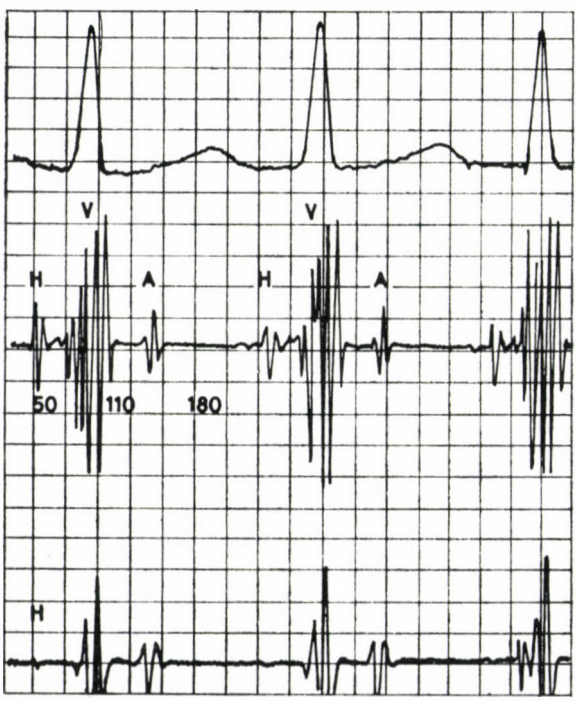


**Fig. 1. Tachycardia of reentry type. Paper speed: 100 mm/sec. The impulse proceeds through an accessory pathway from the ventricle to the atrium (110 ms), there it provokes an atrial echo (A wave), and slowing down in the A-V node (A-H: 180 ms) it returns by the bundle of His to the ventricle (H-V: 50 ms)**

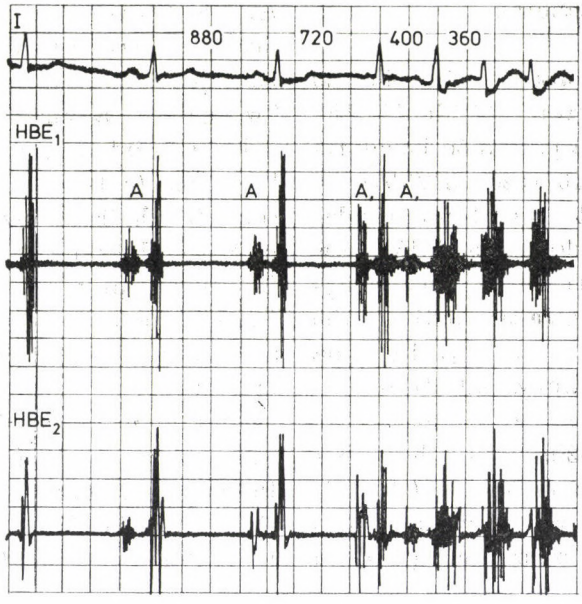


FIG. 2. Ectopic type atrial tachycardia (paper speed: 25 mm/sec). The basal cycle length is 800 ms. This is replaced by an accelerated atrial activity (CL: 880-720-400-360 ms) (A). Higher frequencies induce right Tawara branch block

360 ms) (A). Higher frequencies induce right Tawara branch block

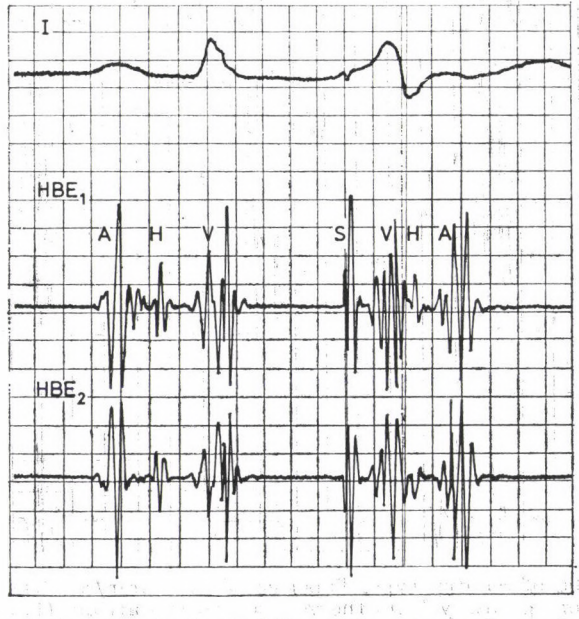


FIG. 3. Early ventricular stimulation is followed by retrograde conduction. The impulse returns to the atrium through the bundle of His. Paper speed: 100 mm/sec. S: stimulus

and 13) the tachycardia was sustained by an ectopic focus (Figure 2). The mean heart rate was 170/min in this group. In six patients (Cases 1—6) the atrial effective refractory period was shorter than the ventricular one ($\geq$ 20 ms). The Wenckebach point measured by serial atrial electric stimulation (atrioventricular block) appeared at a heart rate of 178/min on the average. In six of the patients affected by ATA (Cases 3, 6, 7, 9, 10 and 13), retrograde conduction was observed (Figure 3). The functional block of the retrograde conduction system elicited by ventricular stimulation occurred at an average frequency rate of 240/min. In Case 13 even a stimulation of 280/min failed to elicit a retrograde block. At the same time, during the ectopic tachycardia originating from the lower part of the right atrium a 2 : 1 anterograde A—V block was encountered.

In three patients (Cases 14, 15 and 16) the SVTA was of junctional origin. The paroxysm set in by a His potential, the impulse was conducted by the His-Purkinje system to the ventricles and only then to the atria in a retrograde manner. The anterograde conduction from the atrium was slower in all cases than the retrograde one. In Case 15 an intra-His block was also found. Ventricular stimulation resulted in retrograde conduction only in two patients (Cases 14 and 16).

Table IV lists the patients affected by ventricular tachyarrhythmia (VT). In a 13-year old girl the electrophysiological evaluation was indicated by repetitive ventricular extrasystole (Case 19). They were multifocal, the

Table III

Supraventricular tachyarrhythmias (n: 16)

| | |
|---|---|
| Paroxysmal atrial flutter | 2 |
| Atrial tachycardia | 11 |
| Junctional tachyarrhythmia | 3 |

Table IV

Ventricular tachyarrhythmias (n: 4)

| | |
|---|---|
| Ventricular irritability | 1 |
| Torsades de pointes | 3 |
| ventricular fibrillation | 1 |
| long Q—T | 2 |

Table V

Accessory pathway (n: 7)

| | |
|---|---|
| Shortened P-Q (L-G-L syndrome) | 3 |
| W-P-W syndrome | 3 |
| Latent | 1 |

number of premature beats did not exceed four. In this patient mitral prolapse and an accessory conduction pathway were detected in addition to a shortened P—Q interval (A—V interval, 80 ms). In three patients (Cases 17, 18 and 20) VT manifested occasionally in form of torsades de pointes. In a girl of five years, with a VT, atrial irritability and double impulse conduction were verified. In two patients the Q—T interval corrected for frequency was prolonged and the VERP/Q—T quotient pathologically shortened ($<0.75$). In Case 20 the VT of a rate of 150 to 180/min could be elicited only by left ventricular stimulation. The VT exhibited transition to ventricular fibrillation but this spontaneously ceased after 2 seconds. The basal frequency rate was a 46—60/min sinus bradycardia. A double response achieved by early atrial stimuli suggested an accessory pathway. The refractory period of one path was 220 ms, that of the other 190 ms. In this patient the Q—T distance, its rate corrected time and the VERP/Q—T quotient were normal ($>0.75$).

In seven patients out of the twenty the presence of an accessory pathway could be demonstrated (Table V). The Lown—Ganong—Levine syndrome was present in two cases of supraventricular and one case of ventricular tachyarrhythmia (Cases 9, 14 and 19). An extranodal accessory pathway of Kent type was verified in Cases 6, 7 (SVTA) and 20 (VT). In a boy of five years (Case 10) the presence of a latent accessory pathway was shown by two different ventricular responses elicited by programmed atrial stimulation.

In 13 patients the tachycardia could be provoked and extinguished by $S_1$ and $S_{1-2}$ techniques alike (Figure 4). The paroxysm could be initiated in eight cases from the right atrium, in one case from the left ventricle and in three cases from the right atrium and the right ventricle. In one patient (Case 2) the atrial flutter or fibrillation could only be abolished by DC shock. In three patients the tachycardia could only be provoked and terminated by frequent stimulation (burst) as can be seen in Figure 5.

## Discussion

In this study the electrophysiological findings of 20 patients of child or adolescent age, affected by paroxysmal supraventricular or ventricular tachycardia have been analysed. The group was homogeneous in respect of absence of a heart defect or electrolyte abnormality. The cardinal and exclusive symptom was tachyarrhythmia appearing in attacks. In a case affected by ventricular irritabily there was a mitral prolapse verified by echocardiography, this was not accompanied by mitral insufficiency. Atrial flutter occurred in a patient seven years after surgery for a secundum type atrial septum defect.

The following properties of childhood tachyarrhythmias were found:

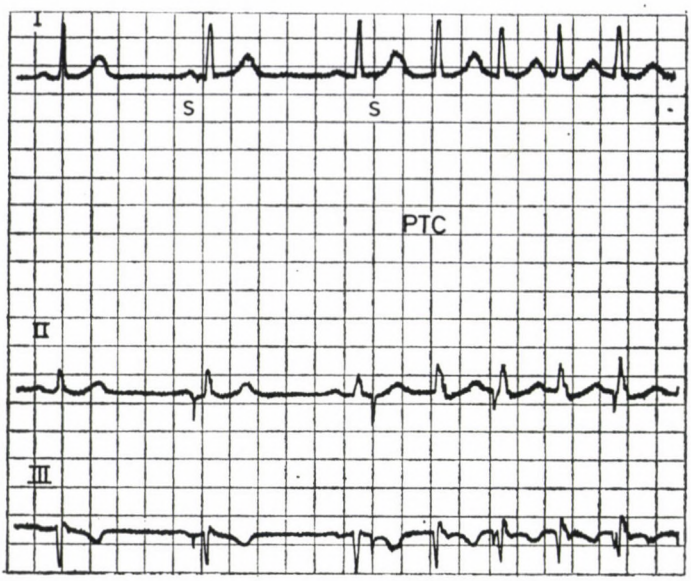


FIG. 4. SVTA elicited by single atrial stimuli (S). Paper speed: 25 mm/s

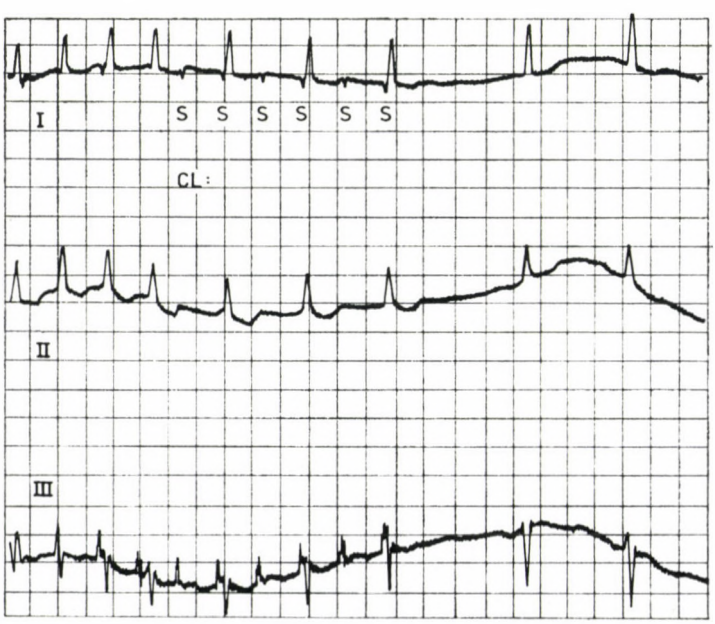


FIG. 5. Interruption of SVTA by frequent atrial stimulus series (S). Paper speed: 25 mm/s

1. Sinus node function is good, there was no sinus dysfunction.

2. Absence of bradycardia.

3. Acceleration of the anterograde and retrograde atrioventricular conduction system(s).

The spontaneous and intrinsic sinus activity measured during atropine ± beta blockade has again proved the well-known fact that sinus node frequency is higher in childhood than in adults. A pathological prolongation of the CSNRT was seen in one patient only, in a girl previously operated upon for atrial septum defect [10]. In this case the disturbance of sinusnode function could be attributed to iatrogenic sick sinus syndrome [9]. In all other patients SNRT, CSNRT and SACT values were normal. Thus, in childhood normal sinus node function may be anticipated in patients affected by tachyarrhythmia as long as they do not have a heart defect or have not been subjected to heart surgery. The appropriate sinus steering is the explanation for the fact that severe bradycardia is rare. It was encountered in our material in a single case. Both disturbed sinus control and bradycardia increase the risk of tachyarrhythmia. In adults, sinus node dysfunction is an arrhythmogenic factor principally in the bradycardia-tachycardia syndrome, in paroxysmal atrial flutter and fibrillation [4]. Atrial fibrillation is very rare in children. Atrial flutter can be recognized in surface ECG. The best proof, however, can be obtained by intracavital leads. The V wave reflecting ventricular activity is preceded by an H wave, with a normal or prolonged H—V time. As a consequence of the extremely frequent atrial rate a 2 : 1 atrioventricular block occurred in both of our cases.

Paroxysmal atrial tachyarrhythmia is the commonest form of tachyarrhythmia in childhood. According to Keith and Nadas [4] one in 25 000 children suffers from atrial tachyarrhythmia and 5% of the affected children has a demonstrable heart defect. The paroxysms appear as early as during the first six months of life. Conspicuously, the recurrence rate of the attacks is higher if the first paroxysm occurs after the first year of life. Cowan observed intrauterine onset of atrial tachyarrhythmia accompanied by oedema, sometimes by hydrocephalus manifest at birth [4, 6]. In our own cases too, a reentry mechanism was present more frequently than an ectopic focus. In tachycardia caused by an ectopic focus no retrograde conduction, no A wave with critical coupling or no slow A—V conduction was shown. The shortened AERP related to the VERP may be evaluated as a sign of electrical inhomogeneity of the heart.

Junctional tachycardia is a rare phenomenon in children, except during postoperative periods. Retrograde conduction could be verified in two patients. In one this was accompanied by L-G-L syndrome. The atrioventricular conduction in both cases was slower than the retrograde one. A block in the His-bundle, in the His—Purkinje system was demonstrated in one case.

In adults continuous atrial stimulation of increasing rate usually induces a functional block of the atrioventricular node, usually at a rate of 150/min; in our own patients, however, higher rates were necessary in all but two patients. Also, the refractory period of the A—V node measured by programmed single impulses ($S_1$ technique) was found to be shorter. A 2 : 1 atriventricular block occurred in 3 patients only, at an atrial rate of 220, 300 and 352/min, respectively. Our findings demonstrate that in both anterograde and retrograde conduction the atrioventricular conductance is better in children than in adults. These electrophysiological findings explain the fact that pressure on the carotis sinus was ineffective in our patients. A pathological effect of the carotis sinus reflex can only be achieved if its points of attack, the sinus node, the sinoatrial conduction and the atroventricular node are damaged.

While SVTA does not cause severe haemodynamic failure in children, VT may lead to serious consequences [5]. In three out of four cases in our material the attacks of tachycardia were accompanied by vertigo or fainting. Case 18, a girl of 18 years, who had been treated for prolonged Q—T syndrome and VT with beta-blocking drugs and a pacemaker, died suddenly one year after the study. In respect to this severe, sometimes fatal complication, the supraventricular or ventricular origin of the VT seems to play no part. Still because of adequate therapy, the mechanism of VT should be explored with all efforts.

In three of our patients an accessory pathway could be found, in one patient a Kent-type extranodal pathway, in two patients a Mahaim bundle was verified. It may be stated that in the overwhelming majority of childhood VT cases a reentry mechanism is at work, using extranodal and intranodal pathways and the Tawara branches. Intraventricular microreentry occurs mostly in adults affected by local ischaemic heart diseases like myocardial infarction. It may be assumed that in the majority of ventricular tachyarrhythmias due to a reentry mechanism the impulse starts from or passes by the atrium, therefore it may be regarded as supraventricular.

Another important factor in the pathogenesis of VT, a prolonged Q—T interval, plays a role also in children. The association of long Q—T time and VT is well-known [1, 2, 8]. In a previous study we found that not only the prolongation of the electric systole of the heart but also the relative shortening of the VERP markedly increases ventricular vulnerability. The VERP/Q—T quotient is abnormal if its value falls below 0.75. In two cases with prolonged Q—T interval the value of the above mentioned quotient was also markedly decreased. In other words, a long Q—T syndrome must be suspected in every child affected by ventricular tachyarrhythmia. In cases with VT characterised by broad QRS complexes and regular morphology a

reentry mechanism with or without atrial participation is probable while in cases with attacks of torsades de pointes type, interference of several ectopic foci may be anticipated.

The site or origin and the sustaining mechanism of the attacks of tachycardia are similar to those observed in adults. Still, the automatism and the reactions of the young heart to parasympathetic and sympathetic stimuli are quite different. This explains the electrophysiological properties. The automatic rate of the sinus node and the ectopic sites is higher, their dysfunction is rarely encountered in children. In paroxysmal tachycardia of children and adolescents sinoatrial and atrioventricular conduction disturbances are rare, blocks occur at higher frequencies in anterograde and retrograde directions alike. As a consequence, bradycardia is a less frequent risk factor for tachycardia in childhood.

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*Received December 23, 1983*

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