

## Atrial tachyarrhythmias in infants

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This report describes clinical features and tactics of treatment of atrial tachyarrhythmias in infancy. Electrophysiologic study was performed in all 20 infants (2 weeks - 12 months old) in order to determine the mechanism of arrhythmia and to predict the clinical efficacy of management. Reciprocating mechanism was revealed in 12 infants. Atrial flutter was diagnosed for 10 infants among whom only 2 patients were older than two months. Six infants were found to have flutter conduction with a rate of 1:1. Four infants had congestive heart failure, 3 of them with a structurally normal heart. Half of the infants with atrial flutter needed long-term antiarrhythmic therapy. Electrotherapy for termination of atrial flutter was effective in all of them. Automatic atrial tachycardia in eight infants presented no major problems unless it became incessant and resistant to pharmacological treatment. The average tachycardia rate reached  $171 \pm 7$  beats/min. Atrial reciprocating tachycardia usually affects patients with diseased myocardium.

**Key words:** atrial tachyarrhythmia, atrial ectopic tachycardia, atrial flutter, infants.

Some of the atrial tachyarrhythmias in early infancy can be potentially dangerous even in a structurally normal heart because of short atrial cycle length and good conduction to the ventricles. Arrhythmias in infants are more likely to go unrecognized because infants are unable to communicate; therefore, their presence should be suspected in cases of bizarre behavior - excitement or apathy, loss of appetite, unexplained dyspnea, cyanosis or even collapse, if the treatment begins too late.

The aim of this study was to present the clinical signs according to electrophysiologic mechanism and to share tactics of treatment of atrial tachyarrhythmias in infancy.

### Material and Methods

There were 20 infants with atrial tachyarrhythmias among 40 infants who underwent transesophageal electrophysiologic study (EPS) in our institution during the last eight years. The intracardiac EPS was carried out additionally in five patients, among them in two (patient nos. 4 and 12) with atrial tachycardia. The age of the patients with atrial tachyarrhythmias ranged from 2 weeks to 1 year (13 of them were in their first 2 months).

Noninvasive technique of the electrophysiologic investigations of the heart was elaborated on the basis of the recording of transesophageal electrograms and differentially amplified surface electrocardiograms (DAEG) in sinus rhythm and during pacing-induced arrhythmias (Fig. 1). Various forms and regimens of heart pacing were used during the study.

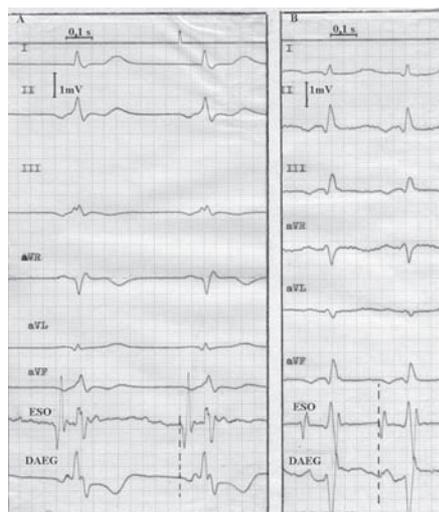


Fig. 1. ECG of a 12-month infant during automatic left atrial tachycardia (A) and 11-month infant during right atrial tachycardia (B).

The indications of transesophageal EPS were approved by our institution<sup>1</sup>. According to our protocol, all patients with tachyarrhythmia require transesophageal EPS, which provides the additional criteria for more precise evaluation of severity and prognosis of arrhythmia. The simplicity of performance permitted us to use this investigation even in suspicion of rhythm disorder.

By means of transesophageal EPS, we were able to estimate the function of the sinus node, anterograde atrioventricular (AV) conduction, refractory period of atria, and AV accessory connections; provoke and terminate tachycardia; identify its mechanism; and assess antiarrhythmic drug efficacy.

Automatic tachycardias cannot be initiated or terminated by programmed electrical stimulation, though rarely they can be induced by means of incremental or rapid transesophageal pacing. The acceleration of heart rate at the onset of tachycardia ("warming up" phenomenon) is common. Sometimes varying cycle lengths are monitored during this type of tachycardia. Overdrive pacing (artificial depolarization) results in a compensatory pause after which tachycardia continues (Fig. 2).

The reentrant tachycardia can be induced and terminated by programmed, incremental, rapid and other modes of pacing (Fig. 3).

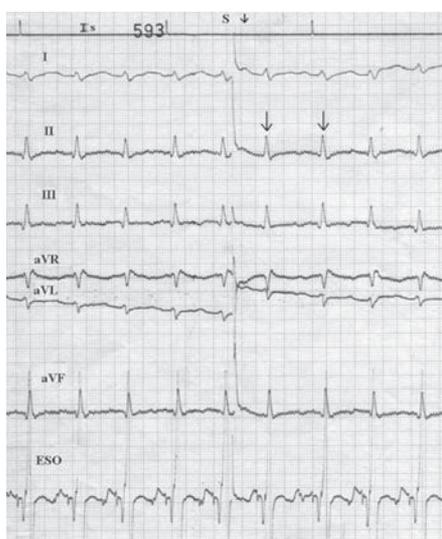


Fig. 2. Programmed stimulation during the focal automatic atrial tachycardia. "Reset" phenomenon – the presence of extrasystole (the first finger) followed by a pause (between the fingers) exceeding the length of P-P.

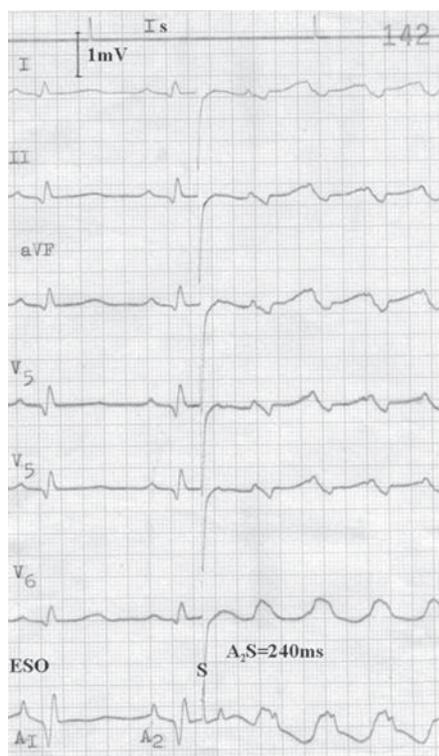


Fig. 3. Induction of focal microreentrant atrial tachycardia using programmed transesophageal pacing.

Intracardiac EPS in infants was performed with regard to specifying localization of the automatic focus.

In our retrospective study, we reviewed the clinical reports of 20 infants with atrial tachyarrhythmias.

## Results

The clinical and echocardiographic findings of our patients are summarized in Table I.

According to the classification of atrial tachycardias<sup>22</sup>, focal atrial tachycardia was established in 10 infants. Microreentry mechanism was identified in two patients among them (nos. 1 and 11). Though the tachycardia rate in both infants was less than 240 beats/min, the episodes were severe enough to induce heart failure. Premature atrial coupled contractions were registered in a one-month-old patient (39 weeks of gestation, weight at birth - 2745 g, born with signs of fetal hydrops) immediately after the birth. His mother has been treated for syphilis during pregnancy. The episode of atrial tachycardia was terminated by transesophageal atrial

**Table I.** Clinical and Echocardiographic Features of Patients with Atrial Tachyarrhythmia

Patient	Age (months)	Tachyarrhythmia	Echocardiographic findings
1	1	ART	↓ LVF
2	6	AF/WPW	EFE, ↓ LVF
3	3 weeks	AAT	Normal
4	12	AAT	Normal
5	2	AF	↓ LVF
6	2	AF	Normal
7	6	AAT	Normal
8	8	AF	OSD, ↓ LVF
9	1	AF	Normal
10	2 weeks	AF/WPW	Normal
11	10	ART	↓ LVF
12	11	AAT	Normal
13	2	AF	↓ LVF
14	1	AF	Normal
15	1	AF/WPW	Normal
16	2	AAT	↓ LVF
17	12	AAT	Normal
18	1	AF/WPW	↓ LVF
19	1	MAT	Normal
20	2	MAT	OSD

ART: Atrial micro reentrant tachycardia. ↓ LVF: Decreased left ventricular function. AF: Atrial flutter. OSD: Ostium secundum defect. EFE: Endocardial fibroelastosis. WPW: Wolff-Parkinson-White pattern. AAT: Automatic atrial tachycardia. MAT: Multifocal atrial tachycardia.

pacing, and digoxin (0.05 mg/kg load) was prescribed afterwards in order to prevent further episodes. Another 10-month-old boy was suffering from viral myocarditis, and his atrial tachycardia with complete right bundle branch block (RBBB) occurred on the fourth day of the illness (Fig. 4). The sinus rhythm



**Fig. 4.** ECG of a 10-month infant. (A) Atrial reentrant tachycardia (R-R=260 msec) with complete right bundle branch block. (B) Conversion of the atrial tachycardia to sinus rhythm by rapid atrial pacing.

was restored by synchronized cardioversion. Digoxin was prescribed to prevent further episodes of tachycardia. Numerous paroxysms of tachycardia repeatedly occurred later during respiratory infections in both infants and they needed combination of antiarrhythmic drugs (propafenone and amiodarone) to prevent further palpitations. The antiarrhythmics were discontinued at the age of 1.5 years in the first infant, and at the age of 3 years in the second. Duration of the follow-up was two and five years, respectively.

Automatic mechanism of focal atrial tachycardia was revealed in eight infants (2 with multifocal atrial tachycardia). There was no evidence of primary structural heart disease, except in one two-month-old girl (no. 2) with atrial septal defect and multifocal atrial tachycardia (Fig. 5). Fetal hypoxia was found in three patients. The mother of one of them had been treated for syphilis during pregnancy. In one six-month-old girl, tachycardia developed after abdominal surgery for intussusception. She underwent radiofrequency ablation only at the age of 5 years. Tachycardia rate varied between 150 and 280 beats/min. Only one two-month-old boy (no.16) was symptomatic, with tachycardia identified prenatally and persisting



Fig. 5. ECG of two-month girl with multifocal atrial tachycardia.

throughout a two-month period with a rate of 260-280 beats/min. Six infants needed a combination of antiarrhythmic drugs (digoxin, propafenone, sotalol, amiodarone). A single antiarrhythmic (digoxin) was prescribed for two infants with multifocal atrial tachycardia. Tachycardia ceased in both infants after 10 days of digoxin therapy.

Atrial flutter was diagnosed in 10 infants, among whom only two infants were older than two months. Both of them presented with structural heart disease: atrial septal defect and endocardial fibroelastosis. The girl (patient no. 2) admitted with Wolff-Parkinson-White (WPW) syndrome. In this case, the first severe (pallor, dyspnea, cyanosis after) paroxysm occurred and terminated spontaneously at the age of two months. The ECG during this attack was not registered in the local hospital. The second episode of tachyarrhythmia (atrial flutter followed by ventricular fibrillation, which was terminated by cardioversion) was induced during EPS. Because of the short anterograde refractory period (190 ms) of the accessory pathway, amiodarone was prescribed to prevent further tachyarrhythmia attacks. WPW syndrome was revealed in another three infants (patient nos. 10, 15, 18) with atrial flutter and structurally normal heart. Two of them (nos. 10 and 15) had two types of paroxysms (orthodromic and atrial flutter). Amiodarone therapy was effective in all of them. Among 10 infants with atrial flutter, six infants were detected to have atrial flutter with a ventricular conduction rate of 1:1 (P-P varied between 286 and 400 beats/min) (Fig. 6). Four patients had atrial premature contractions before the initiation of atrial tachyarrhythmia.

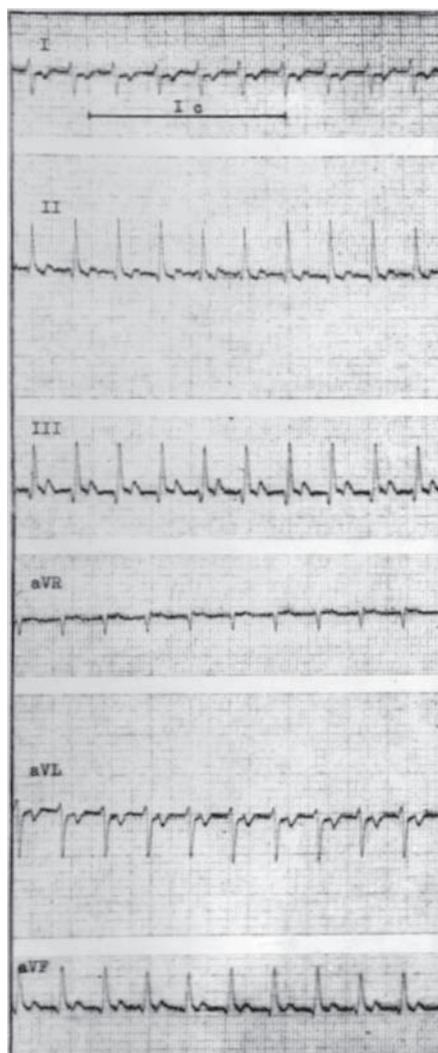


Fig. 6. ECG of a one-month infant [Atrial flutter of 1:1 (P-P=210 ms)].

Four of 10 infants had congestive heart failure (3 of them with structurally normal heart). In eight patients, atrial flutter was terminated by means of electrotherapy (transesophageal pacing or cardioversion). Intravenous digoxin was effective in one case, and spontaneous termination occurred in one infant. Long-term antiarrhythmic therapy (amiodarone or digoxin) was prescribed for five infants (patient nos. 2, 10, 13, 15, 18).

### Discussion

Tachyarrhythmias confined to the atria are not usually given much emphasis for their relative frequency of occurrence or for morbidity and mortality, particularly in cases of a structurally normal heart. However, some of them with high

rate can result in left ventricular dysfunction in infants because of difficulties in recognizing onset of paroxysm. Augmented atrial excitability and short refractory period due to prevailing cholinergic innervation determine the high rate of atrial tachycardia and inadequate myocardial inotropy - heart failure. Such a tachyarrhythmia is atrial flutter<sup>2-4</sup>. The rate of flutter is around 300 beats/min in most cases<sup>5</sup>. In our patients, the atrial flutter varied between 286 and 400 beats/min. Atrial flutter in infants differs from other arrhythmias mostly in its clinical features<sup>5</sup>. It may occur with no apparent heart disease as early as during the first month of their life<sup>6,7</sup>. A characteristic feature is atrial flutter with ventricular conduction rate of 1:1 even in the absence of accessory pathways (in 6 out of 10 patients with atrial flutter in our study) because of physiologically high atrioventricular conduction velocity at this age. A very rapid rate and physiologically weak inotropy of the cardiac muscle can lead to severe hemodynamic disturbances during atrial flutter of 1:1. Therefore, atrial flutter in infancy is a serious rhythm disorder even in the absence of heart disease and needs the fastest and surest method of conversion<sup>2,8-10</sup>. In most cases, it is easily converted to sinus rhythm by electrical conversion (direct current cardioversion or atrial transesophageal pacing). According to many authors, there is a good long-term prognosis for atrial flutter in the infants<sup>4-7,10</sup>. Patients with a structurally normal heart and without accessory connections do not need long-term antiarrhythmic treatment.

Atrial reciprocating tachycardia (ART) in infants occurs rarely and usually affects patients with myocarditis or cardiomyopathy and causes heart failure<sup>11</sup>. Recurrence of ART is common, and patients are in need of oral antiarrhythmic medications to prevent further episodes of tachycardia<sup>8,12</sup>. Amiodarone used alone or in combination with another antiarrhythmic drug is useful.

Atrial automatic tachycardia (AAT) is a common cause of chronic tachycardia in children, and it is resistant to pharmacologic therapy<sup>13,14</sup>. According to our data, however, AAT presents no major problems for infants unless it becomes incessant. The average tachycardia rate in our study reached  $171 \pm 7$  beats/min and was close to physiologic rate. Heart failure developed only in one two-month-old patient with incessant

tachycardia of 200 beats/min persisting from the prenatal period. Some authors present opposite data<sup>15-17</sup>, but it is unclear if the diagnosis of their patients was confirmed by EPS. In many cases, control of AAT can be achieved by using a combination of class IC and III antiarrhythmic agents<sup>14,18,19</sup>. However, the most effective treatment is radiofrequency catheter ablation, which is currently applied to infants as well<sup>13,15,20,21</sup>.

In conclusion, palpitations or symptoms suggesting tachyarrhythmia in infants need an evaluation of the mechanism of arrhythmia because the course, prognosis and treatment of separate tachyarrhythmias are different. In the absence of structural heart disease, atrial tachyarrhythmias are common arrhythmias in infancy. Automatic tachycardia poses no major problems, whereas atrial flutter in early infancy constitutes a severe rhythm disturbance. It has to be recognized and terminated as soon as possible because it often leads to heart failure.

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