

Clinical recognition and treatment of atrial ectopic tachycardia in newborns

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Summary

Questions: Atrial ectopic tachycardia (AET) may be difficult to diagnose in neonates and treatment can be complex especially in case of severe heart failure. This study addresses the clinical recognition and drug therapy of AET in neonates.

Methods: Retrospective chart and database review of neonates diagnosed with AET between 1994 and 2002.

Results: AET was diagnosed in 19 neonates at a median age of 18 days (range 0–64). A paroxysmal AET pattern was seen in 10 and a permanent in 9 patients. Tachycardia in the foetal or neonatal period indicated an arrhythmia in 8 patients while 11 showed non-specific symptoms. Severely depressed ventricular dysfunction was observed in 2 patients necessitating cardiovascular resuscitation in 1. The mean maximum paroxysmal AET rate was 213 bpm (range 178–227). For permanent AET, the median mean heart rate was 169 bpm (153–185) and the mean maximum heart rate was

212 bpm (range 196–274). Antiarrhythmic class Ic and III drugs alone or as combination therapy controlled AET in all 18 treated neonates and ventricular dysfunctions resolved. Proarrhythmic drug side effects were seen in 1 patient under propafenone therapy.

Conclusions: AET in neonates is frequently recognised as paroxysmal or permanent tachycardia. Symptoms are often non-specific even though neonates and infants may develop severe ventricular dysfunction. A high degree of awareness is mandatory for neonatologists, paediatricians and primary care physicians to recognise AET in neonates. Class Ic and III antiarrhythmic drugs are effective in the treatment of neonatal AET. Monitoring for proarrhythmic drug side effects is mandatory.

Key words: atrial ectopic tachycardia; newborn; antiarrhythmic therapy

Introduction

Supraventricular tachycardias (SVT) are observed in paediatric patients with an incidence of 0.1–0.4% [1]. Although atrioventricular reentrant tachycardias are the most frequent mechanism, atrial ectopic tachycardias (AET) account for 5–20% of SVT in the paediatric age group [2, 3]. In AET, an ectopic focus, which can be located somewhere in the atria or great veins, acts as a pacemaker with a rate faster than the sinus node [4, 5]. In contrast to atrioventricular reentrant tachycardias, AET can persist despite atrioventricular block (figure 1).

AET occur in paroxysmal and permanent forms. In the permanent form, Garson et al.

showed in a series of 11 children with AET, that 82% developed congestive heart failure [6]. Symptoms of AET may be initially absent, subtle or difficult to recognise especially in neonates. Thus, the diagnosis of AET could be delayed with subsequent development of heart failure. Once recognised, AET has a favourable prognosis in infants with a spontaneous resolution rate of over 90% within the first year of life [7].

Aim of this study was to evaluate clinical findings, which may facilitate timely recognition of AET in neonates and infants, and to assess the efficacy of antiarrhythmic drug therapy in this patient group.

Patients and methods

Patients

We retrospectively analysed clinical data, efficacy of drug therapy, long-term therapy and recurrences of arrhythmias in neonates with AET diagnosed between 1994 and 2002. Infants who demonstrated symptoms in the neonatal period were enrolled in the study. The study was approved by the local ethics committee and written patient consent for data collection obtained.

Diagnostics

AET was diagnosed electrocardiographically as described previously by Koike et al. (table 1) [8]. Standard ECG was made at presentation. Unless patients were haemodynamically unstable, 24-hour-ECG recordings

were obtained before drug therapy to facilitate analyses of efficacy and potential proarrhythmic effects of antiarrhythmic drug therapy. Minimum, maximum and mean heart rates were obtained from 24-hour-ECG. The maximum heart rate ever recorded in standard ECG or 24-hour-ECG was recorded for paroxysmal AET. A mean heart rate of >150 bpm on 24-hour-ECG was considered abnormal in the absence of sinus tachycardia inducing disease states.

Echocardiography was done before treatment to evaluate ventricular function and cardiac anatomy.

Antiarrhythmic therapy

Antiarrhythmic drug therapy was chosen according to the ventricular function (figure 2). In infants with good ventricular function (EF > 45%) propafenone (150–500 mg/m²/d) as first line, sotalolol (2–5 mg/kg/d) or flecainide (3–6 mg/kg/d) as second line and amiodarone (10 mg/kg/d for 10 days, thereafter 5–10 mg/kg/d) as third line drugs were given. Class Ic and class III drug combination therapies were used in case there was insufficient class Ic drug effect observed. Intravenous amiodarone with a 5 mg/kg over one hour loading dose followed by continuous infusion of 10–25 µg/kg/min was given in case of feeding difficulties or in infants with medium to severe cardiac dysfunction with a ventricular ejection fraction below 45%. Intravenous amiodarone was switched to oral amiodarone for a total loading phase of 10 mg/kg for ten days and thereafter reduced to 5–10 mg/kg/d. Amiodarone was used in infants with moderately to severely decreased ventricular function due to its low negative inotropic effect and high efficacy [9]. The treatment protocols were based on the observations by our group [9]. Follow-up was done every three months with standard and 24-hour-ECG. At the age of about one year, antiarrhythmic drug therapy was discontinued if no tachycardia recurrence was noted. Thyroid and liver function tests were done in patients receiving amiodarone at therapy start, hospital discharge and every six months while receiving amiodarone.

Statistics

Descriptive statistics were used as appropriate.

Figure 1

Example of a 24-hour ECG tracing showing AET partly with 2:1-block. Arrows with P indicate the P-waves of the ectopic focus.

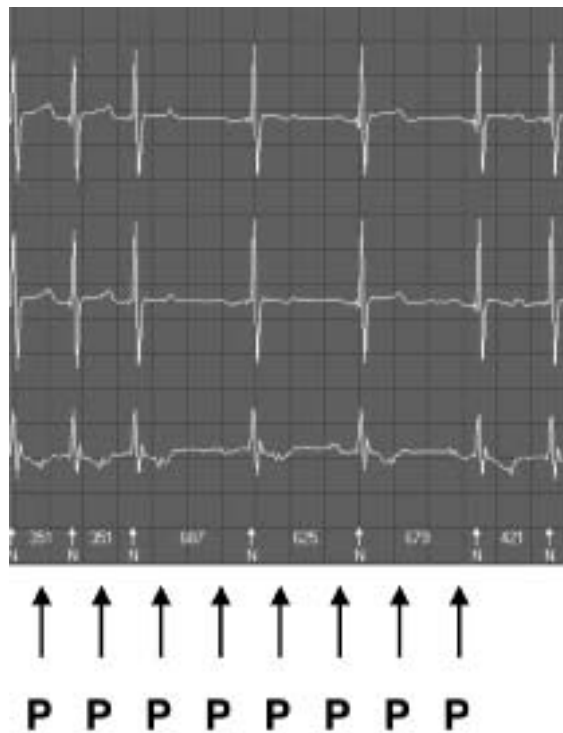


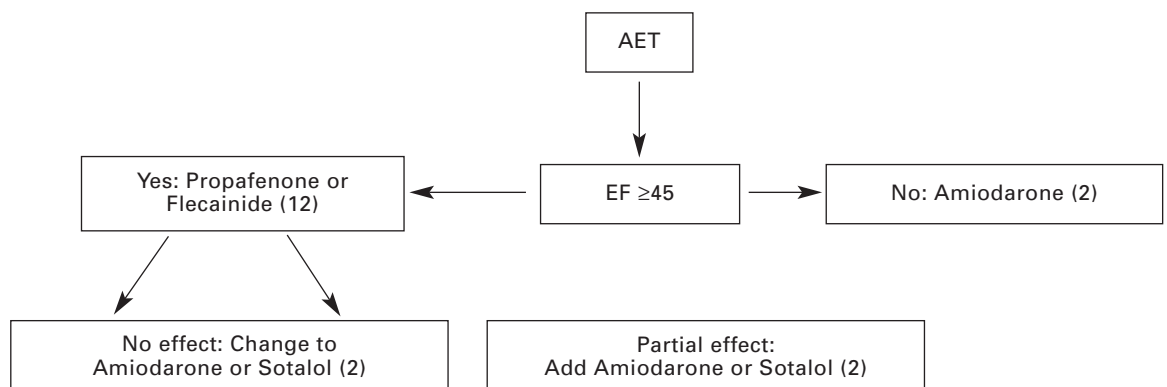
Table 1

Criteria for the diagnosis of AET as described by Koike et al. [8].

Distinct P-waves with a normal or minimal prolonged PR-Interval
Identical P-wave morphology of first and subsequent tachycardia beats
Warm-up phenomenon at initiation of tachycardia
Abnormalities in atrial rate, P-wave-axis or morphology features
Observation of second-degree atrioventricular block without interruption of the atrial rhythm
Wide variability in tachycardia rate
Inability to terminate the tachycardia by programmed stimulation or cardioversion

Figure 2

Stepwise treatment strategy for AET according to ejection fraction (EF). In brackets are the numbers of patients on that therapy.



Results

Diagnostics

A total of 19 infants with neonatal AET were diagnosed. All patients had a structurally normal heart. Clinical data and antiarrhythmic drug therapy are summarised in table 2 and figure 2. Possible heart failure or AET related symptoms were detected in 11 neonates while an isolated increased heart rate in the foetal or neonatal period indicated an arrhythmia in 8 patients.

Of 7 neonates with foetal tachycardia 1 mother received therapy with digoxin and flecainide which lowered foetal heart rate, but finally cesarean section was done at 36th week of gestation. In 2 other cases of foetal tachycardia, caesarean section was done because of maternal indications. Birth weight of all neonates with foetal tachycardia were within normal limits and none showed hydrops foetalis.

Cardiopulmonary resuscitation was required in 1 infant with cardiovascular collapse.

Maximum heart rate recorded for paroxysmal AET was median 213 bpm (range 178–227). For permanent AET, the mean heart rate was median 169 bpm (153–185) and the maximum rate 212 bpm (range 196–274).

Antiarrhythmic therapy

Antiarrhythmic drug therapy was given to 18 infants (table 2 and figure 2). No therapy was re-

quired in an infant with only rare and well-tolerated paroxysmal episodes of AET, which were not more relevant than normal daily sinus tachycardias. All patients converted to sinus rhythm on antiarrhythmic drug therapy although rate control cannot be excluded in some neonates with AET P wave morphology similar to the sinus P wave. Ventricular function normalised before hospital discharge in all infants with previously decreased function.

AET was successfully treated with flecainide in one child after propafenone and sotalol failed.

Propafenone therapy with 260 mg/m²/d had to be stopped because of QRS widening and atrioventricular block as proarrhythmic side effects in one infant two weeks after starting therapy. In all other patients no relevant drug side effects were noted. Thyroid and liver function tests showed normal values in all patients receiving amiodarone.

Antiarrhythmic drugs were successfully stopped in 14 patients after one year of therapy while 4 patients were still on medication after dosage reductions or transient cessation of antiarrhythmic medications demonstrated recurrences of AET. In one patient radiofrequency catheter ablation was performed successfully at three years of age.

Table 2

Data and treatment.

Case	Sex	Age at diagnosis (days)	Symptoms	LV-Function	Heart rate in initial ECG	Effective drug treatment	Combination therapy with
10 patients with paroxysmal AET:							
1	M	64	Tachycardia in paediatric office	Normal	173	Propafenone	
2	F	17	Respiratory distress syndrome	Normal	160	Propafenone	
3	M	24	Tachycardia in paediatric office and heart murmur	Normal	148	Propafenone	
4	M	28	Foetal tachycardia and tachycardia in paediatric office	Normal	219	Propafenone	
5	M	54	Heart failure, cardiovascular collapse	↓↓↓	220	Amiodarone iv	
6	M	1	Foetal tachycardia and heart failure	↓↓	227	Amiodarone iv	
7	M	4	Gastroschisis, heart failure, tachycardia	↓	160	Amiodarone iv	
8	M	24	Pale skin	Normal	214	Propafenone	
9	M	3	Foetal and postnatal tachycardia	Normal	200	Propafenone	Sotalol
10	F	21	Cyanosis while drinking, Tachycardia in paediatric office	Normal	200	Propafenone	
9 patients with permanent AET:							
1	F	10	Foetal tachycardia, pale skin	Normal	206	Propafenone	
2	F	13	Tachycardia in paediatric office	Normal	183	Propafenone	
3	M	3	Tachycardia in paediatric office, drinking deterioration	Normal	170	Flecainide	Amiodarone
4	M	14	Foetal tachycardia	↓	150	Sotalol	
5	M	28	Tachycardia in paediatric office, excessive sweating	normal	177	Propafenone	
6	M	21	Tachycardia in pediatric office, edema	↓	188	Propafenone	
7	M	4	Foetal tachycardia	normal	200	Propafenone	
8	M	18	Foetal tachycardia and tachycardia in paediatric office	normal	212	none	
9	F	35	Tachycardia in paediatric office, cyanosis while drinking	normal	175	Propafenone	

LV-Function: normal (EF >55%); ↓ (EF 45–55%); ↓↓ (EF 30–45%); ↓↓↓ (EF <30%)

EF = Ejection fraction, iv = intravenous, LV = left ventricular

Discussion

This study focuses on clinical recognition of AET and demonstrates that foetal tachycardias or tachycardias detected at paediatric routine visits are often the first clinical sign of AET. Noteworthy is that more than a third of all patients showed foetal tachycardia. This rate is higher than in the group described by Bauersfeld et al., where 3 out of 19 infants had foetal arrhythmias while the other patients showed signs of congestive heart failure [7]. Tachycardia detection was rather accidental in 3 patients during a routine paediatric visit. Symptoms in 11 neonates were fairly non-specific and demonstrated that a high level of awareness is mandatory to recognise subtle symptoms or an isolated increased heart rate as sign of an underlying AET. However, 2 patients presented with moderate to severe ventricular dysfunction and showed that AET, especially when diagnosed with delay might be a life threatening arrhythmia.

One major problem is the differentiation between AET and sinus tachycardia, which can be caused by various disease states such as infection, anaemia, pain, hypovolaemia, hyperthyreosis, structural heart disease or cardiomyopathy. Garson and Gillette showed that standard and 24-hour-ECG are valuable diagnostic tools to diagnose AET [11, 15]. ECG norm values were published by Davignon et al. 25 years ago [16]. Lately, a task force of the European Society of Cardiology published guidelines for the interpretation of neonatal ECG taking into account the Davignon values [17]. Norm values for standard ECG heart rates from birth to day 7 were defined as 90–166 bpm (mean 129) and from day 7 to 30 as 107–182 bpm (mean 149). For 24-hour-ECG we also had consistent with others, defined a mean heart rate of 130 bpm plus 2 standard deviations which means 150 bpm or higher to be abnormal [18].

As described in other studies class Ic (propafenone, flecainide) and class III (sotalol, amiodarone) antiarrhythmic drugs proved to be effective in all treated patients with an acceptable safety profile [7, 10, 12–14]. However, a class Ic and class III antiarrhythmic drug combination therapy was necessary in two infants. One infant showed two weeks after starting therapy a proarrhythmic propafenone effect with QRS widening and atrioventricular block. The delayed proarrhythmic effect was most likely caused by a slow

metabolism in this particular patient. However, this illustrates that careful ECG monitoring during starting therapy and follow-up is mandatory. Furthermore antiarrhythmic drug therapy risks and benefits have to be well balanced.

As a result of successful AET control, decreased ventricular function normalised in all children within weeks. This relatively fast recovery may point to the great remodelling potential of the neonatal and infant myocardium.

Although AET has reportedly a chance of spontaneous resolution of 75% to 95% in the first year of life, resolution was only seen in 73% of our patients [7, 19]. To avoid long-term antiarrhythmic drug therapy, radiofrequency catheter ablation was successfully performed in one child who showed no spontaneous resolution for three years. In case of antiarrhythmic drug failure, catheter ablation would also be a last therapeutic resort for infants with AET.

Limitations of this study are its retrospective design and, similar to other paediatric arrhythmia studies, the relatively small patient number although the study comprises the largest number of neonatal patients with AET.

In conclusion, AET in neonates is frequently recognised as paroxysmal or permanent tachycardia that may already have occurred in the foetus. Although neonates and infants may develop severe ventricular dysfunction or even cardiovascular collapse due to AET, symptoms may be non-specific. Therefore, a high degree of awareness is mandatory for neonatologists, paediatricians and primary care physicians to recognise AET in this age group.

Class Ic and III antiarrhythmic drugs are efficient in the treatment of neonatal AET although careful monitoring for proarrhythmic drug side effects is necessary.

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References

- 1 Garson A, Gillette PC, Mc Namara D. Supraventricular Tachycardia in children: clinical features, response to treatment and longterm follow-up in 217 patients. *J Pediatrics*. 1981;98:875–82.
- 2 Weindling SN, Saul JP, Walsh EP. Efficacy and risks of medical therapy for supraventricular tachycardia in neonates and infants. *Am Heart J*. 1996;131:66–72.
- 3 Walsh EP, Saul JP, Hulse JE, Rhodes LA, Hordof AJ, Mayer JE, Lock JE. Transcatheter ablation of ectopic atrial tachycardia in young patients using radiofrequency current. *Circulation*. 1992;86:1138–46.
- 4 Goldreyer BN, Gallagher JJ, Damato AN. The electrophysiologic demonstration of automatic ectopic atrial tachycardia in man. *Am Heart J*. 1973;85:205–15.
- 5 Gillette PC, Garson A Jr. Electrophysiologic and pharmacologic characteristics of automatic ectopic atrial tachycardia. *Circulation*. 1977;56:571–5.
- 6 Garson A Jr, Gillette PC. Electrophysiologic studies of supraventricular tachycardia in children. I. Clinical-electrophysiologic correlations. *Am Heart J*. 1981;102:233–50.
- 7 Bauersfeld U, Gow RM, Hamilton RM, Izukawa T. Treatment of atrial ectopic tachycardia in infants <6 months old. *Am Heart J*. 1995;129:1145–8.
- 8 Koike K, Hesslein PS, Finlay CD, Williams WG, Izukawa T, Freedom RM. Atrial automatic tachycardia in children. *Am J Cardiol*. 1988;61:1127–30.
- 9 Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. *Eur J Pediatr*. 2003;162:880–4.
- 10 Bauersfeld U, Pfammatter J-P, Jaeggi E. Treatment of supraventricular tachycardias in the new millenium- drugs or radiofrequency catheter ablation? *Eur J Pediatr*. 2001;160:1–9.
- 11 Ludomirsky A, Garson A Jr. Supraventricular tachycardia. In: Gillette PC, Garson A jr. Eds. *Pediatric arrhythmias: electrophysiology and pacing*. Philadelphia: W.B. Saunders: 1990:380–426.
- 12 Von Bernuth G, Engelhardt W, Kramer HH, Singer H, Schneider P, Ulmer H, et al. Atrial automatic tachycardia in infancy and childhood. *Eur Heart J*. 1992;13:1410–5.
- 13 Etheridge S, Craig J, Compton S. Amiodarone is safe and highly effective therapy for supraventricular tachycardia in infants. *Am Heart J*. 2001;141:105–10.
- 14 Janousek J, Paul T. Safety of oral propafenone in the treatment of arrhythmias in infants and children (European Retrospective multicenter study). *Am J Cardiol*. 1998;81:1121–4.
- 15 Garson A Jr, Gillette PC. Electrophysiologic studies of supraventricular tachycardia in children. II. Prediction of specific mechanism by noninvasive features. *Am Heart J*. 1981;102:383–8.
- 16 Davignon A, Rautaharju P, Barselle E, Soumis F, Megelas M. Normal ECG standards for infants and children. *Pediatr Cardiol*. 1979/1980;1:123–34.
- 17 Schwartz PJ, Garson A Jr, Paul T, Stramba-Badiale M, Vetter VL, Wren C. European Society of Cardiology. Guidelines for the interpretation of the neonatal electrocardiogram. A task force of the European Society of Cardiology. *Eur Heart J*. 2002;23:1329–44.
- 18 Mehta KM, Super DM, Connuck D, Salvator A, Singer L, Fradley LG, et al. Heart rate variability in healthy newborn infants. *Am J Cardiol*. 2002;89:50–3.
- 19 Nahhed ZJ, Strasburger JE, Benson DW Jr, Deal BJ. Natural history and management strategies of automatic atrial tachycardia in children. *Am J Cardiol*. 1995;75:405–7.

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