Cardiovascular Topics

Supraventricular tachycardia in children

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Summary

The mechanisms causing different supraventricular tachycardias can be identified with the aid of the 12-lead ECG using Tipple's approach. The main aims of this retrospective study were to use the 12-lead ECG to determine the underlying mechanisms of supraventricular arrhythmias and to evaluate the effectiveness of the treatment modalities used.

Forty-one patients were included in the study. The main findings were: nine of the 41 patients had atrial tachycardias while junctional tachycardia occurred in 32/41 of our patients. The underlying mechanisms causing the junctional tachycardias were: AVNRT (*n* = 21), AVRT (*n* = 10) and JET (*n* = 1). Of the 10 patients presenting with AVRT, eight were less than one year old. AVNRT occurred more often in the older age group (>1 year of age). Fifteen of the 41 patients had spontaneous cessation of their supraventricular tachycardia.

The drug most commonly used during the acute and long-term phases was digoxin. Amiodarone was used in six patients with an 80% success rate. In the early 80s verapamil was used in five patients with a 100% success rate. It is important to note that verapamil is no longer used in children due to its side effects. Lately, adenosine phosphate is the drug of choice in most supraventricular tachycardias.

The management of supraventricular tachycardias in paediatric practice is mainly based on clinical studies and individual experience. Care must therefore be taken to choose medication regimens that are likely to be effective with the minimum risk of potentiating abnormal haemodynamics or conduction.

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Supraventricular tachycardia, previously known as paroxysmal atrial tachycardia, is the most common significant arrhythmia in children.[1,2] The incidence of supraventricular tachycardia is estimated to be one in 250 to 1 000 children, which is far higher than the previously quoted (1967) incidence of one in every 25 000 children.[3] As methods of detection of arrhythmia improved, awareness of this condition increased. During the same time period, the number of cardiac surgery survivors have increased and so also the incidence of supraventricular tachycardia, which is one of the more common late postoperative complications.[1]

Information obtained by electrophysiological studies and ablation procedures furthered our understanding of the mechanisms causing supraventricular tachycardia, so that now, with the aid of the standard 12-lead electrocardiogram (ECG), most mechanisms causing arrhythmias can be diagnosed without invasive procedures.[4]

Three main mechanisms causing supraventricular tachycardias were identified: re-entry, abnormal automaticity and triggered activity.[2] It is generally agreed that accessory AV connections in certain types of supraventricular tachycardia are developmental anomalies.[5] Therefore, in children younger than 12 years of age, supraventricular tachycardias are usually caused by an accessory atrioventricular connection.[5] In the teenage years, atrioventricular nodal re-entrant tachycardia (AVNRT) is the commonest cause of supraventricular tachycardia.[2]

No data could be found of a South African study regarding supraventricular tachycardias in children.

Aim

The aims of this study were to analyse the demographic data of patients presenting with supraventricular tachycardias, to use the 12-lead ECG to determine the underlying mechanisms of the arrhythmia and to evaluate the effectiveness of the treatment modalities used.

Materials and methods

Supraventricular tachycardia is a rapid heart rate resulting from an abnormal mechanism, which originates proximal to the bifurcation of the bundle of His.[1] The rate of the tachycardias ranges from 220 to 320 beats per minute in infants, and from 150 to 250 beats per minute in older children.[2]

Based on the characteristics of the tachycardia, patients were divided into two groups: primary atrial tachycardias and atrioventricular (AV) junctional tachycardias. The 12-lead ECG recording was used to identify the underlying causative mechanism of the supraventricular tachycardia, making use of an algorithm, as suggested by MA Tipple.[6]

Electrocardiographic diagnosis of supraventricular arrhythmias (MA Tipple)[6]

(a) Identify P wave:

Present:	
Upright in II, III, AVF:	Sinus rhythm, sinus node, sinoatrial re-entry, high ectopic focus.
Negative in II, III, AVF:	Atrioventricular re-entry (AVRT), low ectopic focus.
Absent:	
Sinus node disease:	Atrioventricular nodal re-entry tachycardia (AVNRT), ectopic tachycardia (JET)

(*b) P wave morphology*:

Normal:	Sinus tachycardia, sinus node re-entry, sinoatrial re-entry.
Abnormal:	Ectopic atrial tachycardia, atrial flutter, atrial fibrillation.

(c) Relationship of P to QRS:

Proceeding:	Sinus tachycardia, atrial tachycardia, long PR re-entrant tachycardia.
After:	AVNRT, AVRT.

(d) Presence of AV or VA block:

Atrial rate > ventricular rate:	Sinus tachycardia, atrial tachycardia.
Ventricular rate > atrial rate:	JET.

All surface ECG tracings were taken at the usual paper speed (25 mm/sec) with usual standardisation (1 mV/10 mm). We infer the presence of a P-wave if the T-wave is quite pointed or if a notch is visible on the T-wave.

Definitions

Primary atrial tachycardia is that in which the primary electrophysiological disturbance is restricted to atrial tissue and it includes: sinus tachycardia, sinus node re-entry, ectopic atrial tachycardia, atrial fibrillation, atrial re-entrant tachycardia and atrial flutter.[6] Atrially driven tachycardias have identifiable atrial activity preceding each QRS complex.

Atrioventricular tachycardias require the atrioventricular (AV) junction as a necessary component and include AV re-entrant tachycardia using an accessory connection, atrioventricular node re-entrant tachycardia (AVNRT) and junctional ectopic tachycardia (JET).[6] These tachycardias usually have an abnormal P-wave morphology and axis, which indicate a focus other than the sinus node.

Reciprocating tachycardias involve accessory pathways:

- Orthodromic reciprocating tachycardia is the most common form where activation is from atria to ventricles through the normal AV node, returning via the accessory pathway to the atria.[5] A narrow QRS complex results, during which P-waves are inscribed after the QRS complex (RP > PR).
- Antidromic reciprocating tachycardia shows a prolonged QRS duration with conduction anterograde over an accessory connection and retrograde conduction via either the AV node or an additional accessory connection.[7]
- In Wolff-Parkinson-White, an accessory pathway (e.g. Bundle of Kent) links the atria and ventricles and bypasses the AV node. The pathway might manifest itself by a delta wave in sinus rhythm or might be concealed when it operates only during the tachycardia.[4]

Atrioventricular nodal re-entry tachycardia (AVNRT) is due to re-entry within the AV node and is caused by a circuit operating within and in the proximity of the AV node. Although AV node anatomy and function are incompletely understood, patients with dual AV nodal physiology may be considered to have functional slow and fast AV nodal pathways. The usual tachycardia of AVNRT is based on an anterograde propagation of the impulse through the slow pathway and by faster and retrograde activation through the fast pathway.[5]

Junctional ectopic tachycardia (JET) usually occurs in the very young, or postoperatively and is characterised by an abnormal P-wave axis, normal QRS axis and significant variability in overall rate.

Design

This is a retrospective, descriptive study of 41 children admitted from 1982 to 1998 with supraventricular tachycardias. The following data were extracted from the patient folders:

- Age at onset of the first episode of supraventricular tachycardia
- Gender
- Predisposing factors such as: fever, sepsis, drugs, thyroid abnormalities, myocarditis
- Associated factors such as: congenital heart disease, postoperative to cardiac surgery and Wolff-Parkinson-White (WPW) syndrome
- Signs and symptoms present with initial episode
- Presence of congestive heart failure with the first episode

- Type and response of initial treatment for the first episode (success is defined as a cessation of supraventricular tachycardia for at least one hour)
- Long-term treatment and response to such treatment
- Presence of supraventricular tachycardia on follow-up (at least one episode of ECG-documented supraventricular tachycardia within one year of the last clinic visit).

Inclusion criteria

Children younger than 13 years of age had to have had a 12-lead surface ECG, or 24-hour ambulatory Holter study, taken during the supraventricular tachycardia and in sinus rhythm.

Exclusion criteria

Incomplete data or absence of a 12-lead surface ECG or 24-hour Holter study.

Results

There were 41 patients who fulfilled the inclusion criteria: 22 males and 19 females (ratio M:F = 1,18:1) with a mean age of 3 months (range: in utero to 13 years) at presentation. Twenty of the patients, 13 male and 11 female, were less than 1 year of age. The age of onset and the gender of the patients are displayed in Fig 1.

Predisposing and associated factors were found in 49% of patients and are summarised in Table I. The most common complaint in older patients (>1 year of age) was of palpitations. Fifteen of the 16 patients presenting with congestive heart failure were less than one year of age.

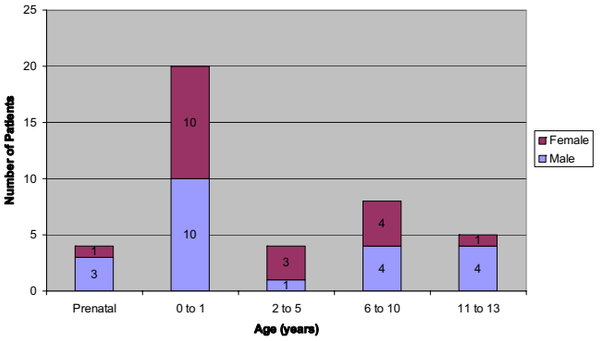

Fig. 1. Age at onset of supraventricular tachycardia and gender

Junctional tachycardia occurred in 32/41 of our patients, while atrial tachycardia was present in 9/41. The underlying mechanisms causing the junctional tachycardias were: AVNRT (n = 21), AVRT (n = 10) and JET (n = 1). Of the 10 patients presenting with AVRT, eight patients were less than one year of age. AVNRT occurred more often in the older age group (>1 year of age).

Fifteen of the 41 patients presenting with supraventricular tachycardia had spontaneous cessation of their supraventricular tachycardia (atrial tachycardia two patients, AVNRT nine and AVRT four). The response to treatment of the remaining 26 patients is summarised in Table II.

Digoxin was the drug most commonly used and was successful in terminating the supraventricular tachycardia in 81% of patients. Synchronised DC electrical cardioversion was the next most common method used, with a success rate of 43%. Verapamil (five patients) as well as amiodarone

TABLE I. PREDISPOSING AND OTHER FACTORS ASSOCIATED WITH SUPRAVENTRICULAR TACHYCARDIA

	Associated factors			*Predisposing factors*			
Age (months)	*Pre-op CHD*	*Postop CHD*	*WPW*	*Myocarditis*	*Fever*	*Sepsis*	*Drugs*
Foetal	VSD, PDA						Mother, lofepramine
0.25	Ebstein						
0.5	VSD, PDA						
0.5			*			*	
0.5			*	*			
0.5			*				
0.7			*		*		
1					*		
1			*				
1				*			
1.5	PDA						
2			*	*			
2			*				
3	Floppy mitral valve					*E. coli*	
3					*		
5.5				*			
8					*		
16		VSD					
84			*				
144			*				
Total	**5**	**1**	**9**	**4**	**4**	**2**	**1**

Abbreviations: CHD = congenital heart disease; WPW = Wolff-Parkinson-White syndrome; VSD = ventricular septal defect; PDA = patent ductus arteriosus; Pre-op = pre-operative; Postop = postoperative.

TABLE II. EMERGENCY TREATMENT OF SUPRAVENTRICULAR TACHYCARDIA AND RESPONSE

		Age (months)	*Heart failure*	*Vagal manoeuvres*	*DC cardioversion*	*Adenosine*	*Digoxin*	*Verapamil*	*Amiodarone*
Primary atrial tachycardia		0	No				Yes		
		0	No		No		No		Yes
		0.25	Yes		No				Yes
		0.5	Yes			Yes			
		1.5	Yes				Yes		
		2	Yes				Yes		
		3	Yes				Yes	Yes	
Junctional tachycardia	AVNRT	0	Yes		Yes	Yes			Yes
		0.3	No				Yes		
		0.5	Yes		No		No		Yes
		1	Yes		Yes		No		No
		1	Yes				Yes		
		1	Yes					Yes	
		1	No				Yes	Yes	
		3	Yes	No			Yes		
		8	No	No			Yes		
		72	No			Yes			
		84	No				Yes		
		96	No					Yes	
	AVRT	0	No				Yes		
		0.5	Yes		Yes	No			
		0.7	Yes				Yes		
		2	Yes		No			Yes	
		2	Yes	No			Yes		
		84	No	Yes					
	JET	16	Yes						Yes

(six patients) had a success rate of 100% and 80% respectively, with no complications or fatalities. Vagal stimulation (carotid sinus massage) was successful in only one seven-year-old patient. In the three infants where vagal stimulation was tried it was ineffective.

After the initial cardioversion, 80% of patients were put on long-term treatment. Digoxin was used in 77% of these patients. Twelve of the 33 patients on long-term therapy had breakthrough episodes within the first year after initiation of therapy for supraventricular tachycardia. Two patients received radio-frequency ablation. After adjustment of the dosage of the drugs, none of the remaining 10 patients had any breakthroughs.

Thyroid function tests were done every six months in patients on amiodarone. One developed hypothyroidism and one a retinopathy. Both these abnormalities normalised after discontinuation of the drugs.

All patients younger than one year of age were kept on maintenance therapy for two years after the last documented episode of supraventricular tachycardia before discontinuation of therapy.

Discussion

Supraventricular tachycardia is a common abnormality of heart rhythm in the paediatric patient, often presenting before the age of four months.[1,8] Thirty per cent of infants lose supraventricular tachycardia inducibility by one year of age and therefore clinical supraventricular tachycardia recurrences are uncommon. Some children have no recurrences of supraventricular tachycardia after the initial presentation.[9]

The term supraventricular tachycardia includes a wide variety of causative mechanisms, which were brought to the fore by electrophysiologic studies. These mechanisms are re-entry tachycardias, increased automaticity and increased triggered activity. Some of these mechanisms are confined to the atria while others involve both atria and ventricles. Classifying the tachycardia by its mechanism and origin, as well as determining associated risk factors for treatment (such as congenital heart disease, myocarditis, cardiomyopathy) allow the clinician to choose the best treatment modality for the patient. It is therefore no longer adequate to identify a narrow-complex tachycardia as a paroxysmal supraventricular tachycardia.[7,14]

Studies have shown that the clinical features, predisposing and associated factors, mechanisms of supraventricular tachycardia, subsequent responses to treatment and the eventual outcome of patients presenting with supraventricular tachycardia vary greatly according to the patient's age. The data in our study correlate with these findings.

Fifty-four per cent of children in our study had their first episode of supraventricular tachycardia before four months of age. These findings correlate with Garson *et al*.[1] and Anderson *et al*.,[10] who reported incidences of 38% and 59% respectively. There were 13 male and 11 female infants under the age of one year. The male preponderance of supraventricular tachycardia when the onset is in infancy has been verified in several reports, but is not found in any age groups after infancy.[11]

Predisposing or associated factors were present in 49% of the patients and included myocarditis, fever, infection and drug exposure (24%), WPW syndrome (22%), and congenital heart disease (15%). Ninety-four per cent of

TABLE III. GUIDELINES FOR THE TREATMENT OF SUPRAVENTRICULAR TACHYCARDIAS

Type of supraventricular tachycardia	*Termination*		*Maintenance treatment*		
	Drugs/other options	*Dose*	*Drug*	*Dose (oral/day)*	*Doses/day*
Sinus tachycardia	Treat underlying cause				
Atrial tachycardia: ectopic and multi-focal tachycardia	Amiodarone Digoxin	5 mg/kg ivi loading 30 mcg/kg	Amiodarone Digoxin	2–5 mg/kg 10 mcg/kg	1 2
Sinoatrial re-entrant	See AVNRT/AVRT				
Atrial fibrillation and flutter	1. Haemodynamically unstable: DC cardioversion (1)	0.25–2 J/kg	*d,l*-sotalol (5)	2–8 mg/kg	2
	2. Haemodynamically stable: a) No CHF: (2) Amiodarone	5 mg/kg ivi loading	Amiodarone WPW: Propanolol (7)	2–5 mg/kg 1–3 mg/kg	1 3–4
	b) No CHF: Procainamide	infants 5–7mg/kg children 7–15 mg/kg slow ivi			
	Ibutilide	0.01 mg/kg ivi			
AVNRT and AVRT	1. Haemodynamically unstable: DC cardioversion (1) Oesophageal overdrive pacing	0.25–2 J/kg	Digoxin Propanolol	10 mcg/kg 1–3 mg/kg	2 3–4
	2. Haemodynamically stable: Vagal manoeuvres (3) Adenosine (4) Procainamide	50–100 mcg/kg ivi bolus infants 5–7mg/kg slow ivi children 7–15 mg/kg slow ivi	*d,l*-sotalol WPW: Propanolol (7)	2–8 mg/kg 1–3 mg/kg	2 3–4
Junctional ectopic tachycardia (JET)	Amiodarone (6)	5 mg/kg loading	Amiodarone	2–5 mg/kg	1

Notes:
(1) DC cardioversion: direct-current (DC) cardioversion is synchronised to QRS
(2) Congestive heart failure (CHF)
(3) Vagal manoeuvres:
Infants – apply ice to the face, gagging with tongue blade
Children – above methods plus unilateral carotid massage and Valsalva manoeuvre doing a handstand
(4) Adenosine is administered by rapid bolus at the usual starting dose of 100 mcg/kg/bolus with increases of 50 to 100 mcg/kg per bolus (maximum dose: 350 mcg/kg) if unsuccessful.
(5) Class 111a drugs such as *d,l*-sotalol and amiodarone are effective in 70% and 80% of cases in prevention of atrial flutter. The side-effects of amiodarone make it a l*ess* attractive option than *d,l*-sotalol.
(6) Postoperative JET is a life-threatening arrhythmia. In addition to hypothermia and atrial pacing, antiarrhythmia drugs are essential to overcome the first few critical days after surgery. Amiodarone is effective in both congenital and postoperative JET.
(7) Although propanolol and other beta-blockers are the drugs of choice in children with WPW; radio-frequency catheter ablation of the accessory pathway is the only curative treatment and therefore the preferred option.

patients with congestive heart failure occurred in patients under the age of one year.

Intravenous digoxin was the most frequently used drug and had a success rate of 81%. The mothers of three babies with foetal tachycardia received digoxin during pregnancy. It was successful in two of the three cases. Newer studies also suggest the use of sotalol for antenatal supraventricular tachycardias because it passes freely through the placental barrier.[12]

Cardioversion has been reported as being 98% successful in the acute treatment of supraventricular tachycardia.[13] Synchronised direct electrical current cardioversion had a success rate of 43% in our study. This might be due to an insufficient energy dose delivered. The maximum dose that was given to the four patients that did not respond was 2 watt-sec/kg, which is far less than the recommended maximum dose of 10 watt-sec/kg.[6]

Verapamil, which was used during the early 1980s, was

very effective in terminating supraventricular tachycardias in our study. It is known to cause severe haemodynamic deterioration in infants and in patients with heart failure and is therefore contraindicated in this group.[14] Due to its side effects, it is at present not being used.

Digoxin was the drug most commonly used for long-term therapy. The majority of recurrences were within the first year of age: 11 of the 13 patients that relapsed were infants younger than one year.

Recommendations

In contrast with adult cardiology, there are no controlled studies on the treatment of children with supraventricular tachycardias. Therefore, in paediatric practice, the management of supraventricular tachycardias is mainly based on clinical studies and individual experience.[14] Care must be taken to choose medication regimens that are likely to be effective with the minimum risk of potentiating abnormal haemodynamics or conduction (Table III).

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