stent placement in an superior vena cava obstruction with a decreased gradient documented by TEE (Fig. 2). In 2 patients with multilevel LVOT obstruction, TEE confirmed transthoracic echocardiographic and angiographic findings but was superior to both in defining the nature of the stenoses. A 13-year-old girl had a gradient of 50 mm Hg in the LVOT at catheterization; TEE alone detected the presence of a muscular tunnel. In a 7-year-old girl with recurrent LVOT obstruction with a gradient of 40 mm Hg at catheterization, TEE demonstrated a fibromuscular ridge, a discrete membrane, and recurrent growth of atrioventricular valve tissue after complete atrioventricular canal repair. On the basis of these findings, no interventions were performed at the time of catheterization in these two patients.

*Conclusions.* In this study, TEE performed in conjunction with cardiac catheterization provided crucial information regarding the atrial septum, postsurgical intraatrial obstructions of systemic and pulmonary venous connections, and the LVOT. This information may not be obtainable by transthoracic echocardiography or angiography. Our findings support the use of TEE to monitor the performance and success of these selected interventional catheterization procedures.

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## Chaotic atrial tachycardia in children

Mubadda A. Salim, MD,[a] Christopher L. Case, MD,[b] and Paul C. Gillette, MD[b] *Charleston, S. C.*

Chaotic atrial tachycardia (CAT) is characterized by the presence of three or more P-wave morphologic features on the surface electrocardiogram, absence of a dominant atrial pacemaker, and variable P-P, R-R, and P-R intervals with an atrial rate of >100 beats/min.[1] CAT is predominantly a disease of adults with respiratory illness. Fewer than 100 cases have been reported in children.[2-6] The correct diagnosis and treatment is important, because this arrhythmia can be difficult to control and can be fatal. We describe our experience with this arrhythmia, concentrating on the clinical presentation, acute and chronic treatment regimens, and outcome.

During the period between October 1986 and December 1993, five infants arrived at the Medical University of South Carolina with CAT. Over the same time period, 1500 new onset arrhythmias were diagnosed. This represents an incidence of 0.2%. We retrospectively reviewed the charts of these patients and evaluated their response to therapy. There were three boys and 2 girls aged 3.0 ± 3.7 months (Table I). Three of the five patients had symptoms at initial examination. Two patients had respiratory distress after several days of nonspecific symptoms, and one patient had tachycardia in utero along with polyhydramnion. At initial examination 3 infants showed signs of congestive heart failure and 2 had no abnormalities other than rapid irregular heart rate. Cardiomegaly was present on the chest radiographs of 3 patients. Diagnosis of CAT was made on the basis of 12-lead electrocardiogram; according to the diagnostic criteria (Fig. 1), the mean number of different P-wave morphologic features was 4 ± 2 (range 3 to 7). The atrial rate could only be estimated from the surface electrocardiogram. The PR interval ranged between 50 and 180 msec. The mean ventricular rate was 178 ± 25 beats/min. The QRS complex morphologic characteristics were normal in 4 patients; the fifth patient had an incomplete right bundle branch pattern. The mean QRS axis was 100 ± 37 degrees. Echocardiograms demonstrated 2 patients to have normal structure and function. Of the remaining 3 patients, 1 had a hypertrophic left ventricle and 2 had secundum atrial septal defects, with left superior vena cava to coronary sinus in 1. The left ventricular shortening fraction was 33% to 47%. Acute treatment of this arrhythmia was attempted in three patients at referring institutions. Adenosine in 1 patient and direct current cardioversion in two patients failed to terminate the arrhythmia. Acute therapy with procainamide in 3 patients, quinidine in 2 patients, and verapamil in 1 patient, were unsuccessful in terminating CAT.

All patients were hospitalized for chronic treatment. Digoxin was initially tried in all patients and was successful as the sole drug in 1 of 5 patients. This patient responded with conversion to sinus rhythm after 2 days of initiating therapy. Amiodarone, a second-line drug used in the 4 remaining patients, was successful in combination with digoxin in 1 patient. The third line of therapy was a combination of class 1C antiarrhythmic drugs with amiodarone and digoxin. This combination was used in 3 patients and was successful in 2. In patient 1, for whom the previous regimen failed, atenolol was added to the combination of digoxin, amiodarone, and encainide to control the

From the Divisions of Pediatric Cardiology, the [a]University of Tennessee and the [b]Medical University of South Carolina.

Reprint requests: Christopher L. Case, MD, Division of Pediatric Cardiology, Medical University of South Carolina, 171 Ashley Ave., Charleston, SC 29425-0680.

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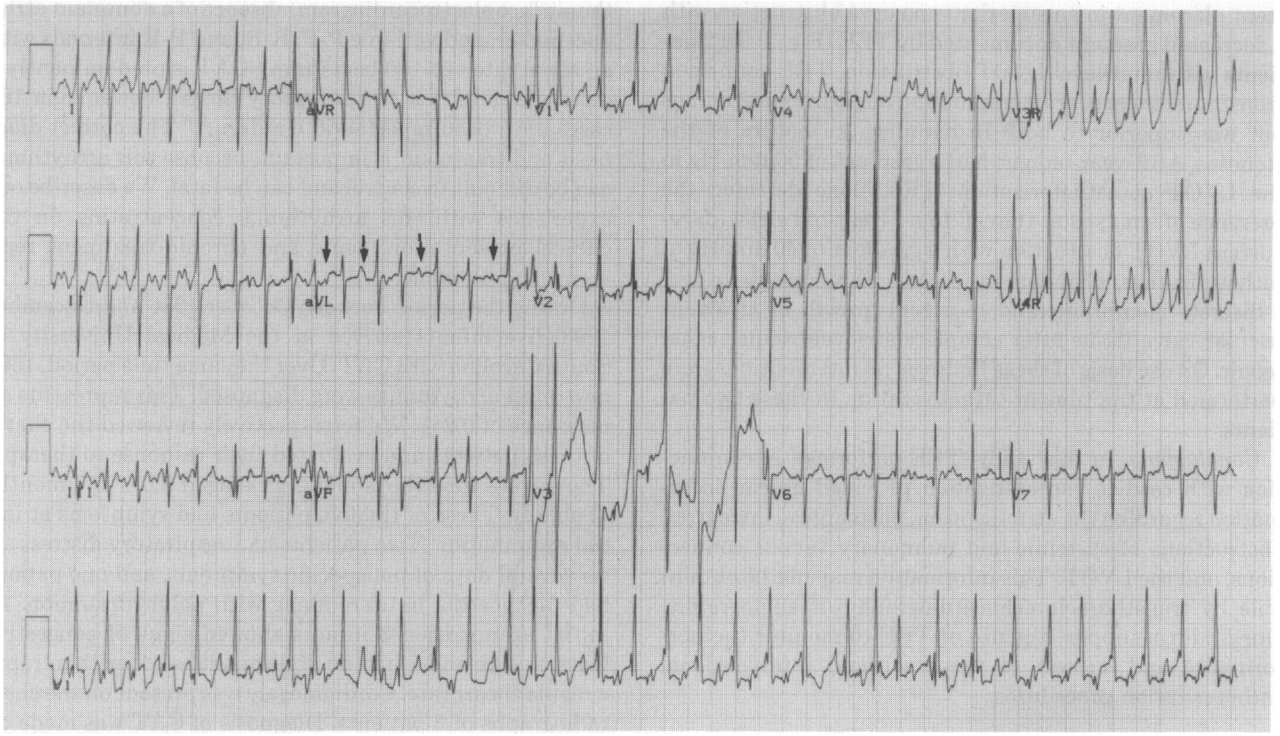


**Fig. 1.** Chaotic atrial tachycardia in patient 3. Average ventricular rate is 218 beats/min. Four different P-wave morphologic characteristics are marked by *arrows*.

ventricular response. This child also had surgical closure of an atrial septal defect at 3 months of age after a catheterization documented pulmonary/systemic flow ratio of 3.2. This patient's CAT proved to be the most resistant to therapy and was not under control at his death at the age of 16 months from respiratory and cardiac decompensation caused by complications of CAT therapy. This patient was the first of this series; we had no further deaths from this arrhythmia.

Electrophysiologic studies were performed on patients 1 and 2. The electrophysiologic study of patient 1 was performed when the patient was at 3 months of age and receiving amiodarone and digoxin; the study showed only short runs of rapid atrial tachycardia and no ventricular arrhythmias induced. A second study 3 months later to evaluate a wide complex tachycardia that developed while the patient was receiving amiodarone, flecainide, and digoxin showed atrial flutter with aberrant conduction. Burst atrial pacing converted him to sinus rhythm. Sinus node function was within the normal limits, with no inducible arrhythmias. Patient 2 was receiving amiodarone, encainide, and digoxin at the time of the study (age 9 months), which revealed the presence of predominant sinus rhythm with spontaneous short runs of atrial tachycardia; no arrhythmia was induced. A follow-up study with the same therapeutic regimen was performed 3 months later while the patient was catheterized for evaluation of his atrial septal defect. This study revealed sinus rhythm and no inducible arrhythmias. There was no significant difference between the patients with and without structural heart abnormalities in the number of drugs used during their hospitalization, the time from initiation of therapy until they were converted to sinus rhythm, or the length of follow-up.

The mean follow-up duration was 23 ± 23 months (range 1 to 59). Four of the 5 patients are alive and in sinus rhythm. In the patient treated only with digoxin, sinus rhythm has been preserved for 2 years with this medication only. Amiodarone therapy in the remaining 4 patients was discontinued in 1 patient after 3 years of control and in 1 patient because of possible pulmonary side effects (although he was not in sinus rhythm). Two patients are currently receiving chronic maintenance therapy of amiodarone. Of the 3 patients in whom class 1C antiarrhythmic drugs were used, 1 patient continues to receive flecainide, 1 was receiving encainide at the time of his death, and 1 had encainide safely discontinued 1 year after therapy was started.

CAT in infants appears to have a favorable prognosis when it responds to therapy.[1] It has been associated with congenital heart disease with or without prior surgical intervention,[2-4] with severe systemic infection,[2] and with myocarditis.[4] However, it is most often found in children with normal heart structure. The cause of CAT is unclear but may be related to the size of the atria[2] or the presence of a suture line on the atria in patients after surgery.[3] The control of the systemic infection, when present, resulted in

**Table I.** Clinical data of patients

| *Patient no.* | *Age at onset of arrhythmia* | *Sex* | *Symptoms* | *Duration of CAT* | *Heart structure* | *Medical problems* | *Duration of CAT after therapy* | *Follow-up duration (mo)* | *Rhythm on follow-up/ Outcome* | *Medications* |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 38 days | M | Respiratory distress | 15 mo | Secundum ASD; Qp:Qs = 3.2:1; large right + left atria; left superior vena cava to coronary sinus. | Prematurity; meconium aspiration; left broncho-malacia; failure to thrive; neurodevelop-mental delay | 15 mo | 15 | CAT/Died of respiratory and cardiac complications | Digoxin, encainide, atenolol |
| 2 | 6 mo | M | None | 70 day | Secundum ASD; large left + right atria; Qp:Qs = 1.7:1 | None | 22 days | 59 | Sinus/Alive | Digoxin, furosemide |
| 3 | In utero | F | Polyhydram-nion, tachycardia, right pleural effusion | 62 day | Concentric LV hypertrophy, endocardial thickening; normal anatomy | None | 3 days | 7 | Sinus/Alive | Digoxin, amiodarone, flecainide, furosemide |
| 4 | 8 mo | M | Respiratory distress | 2 day | Normal anatomy | None | 2 days | 31 | Sinus/Alive | Digoxin |
| 5 | 3 days | F | None | 14 day | Normal anatomy | None | 3 days | 1 | Sinus/Alive PAC | Digoxin, amiodarone |

*ASD*, Atrial septal defect; *LV*, left ventricle, *PAC*, premature atrial contractions; *Qp*, pulmonary blood flow; *Qs*, systemic blood flow.

conversion to sinus rhythm in some patients.[2] Both flecainide[5] and amiodarone[6] have been reported used to successfully treat CAT. In our experience, digoxin alone was successful as the sole agent in only one patient. Adding amiodarone therapy proved effective in 1 patient, and the combination of amiodarone and digoxin with a class 1C agent was effective in 2 patients. The only patient who did not respond to therapy had other complicating conditions. He was premature with meconium aspiration and had left-sided bronchomalacia. The surgery to close his atrial septal defect removed the hemodynamic burden of the left-to-right shunt but did not make the management of his arrhythmia any easier. The scar on the right atrial wall may have contributed to the continued persistence of the CAT. The goal of our therapy protocol was to control the ventricular response and to restore sinus rhythm. This report includes patients from as early as 1986, a time when current methods of controlling the ventricular response (i.e., ablation of the atrioventricular node and the His bundle) were not available. On the basis of this experience, we initially treat patients with CAT with digoxin. Amiodarone and a class 1C agent are added sequentially to attain sinus rhythm. The withdrawal of these drugs is performed carefully and after sinus rhythm has been restored with no episodes of breakthrough arrhythmias. In conclusion, CAT is an uncommon arrhythmia in children. In the majority of cases, combination therapy of a class 1 and class 3 antiarrhythmic agent is necessary for rhythm control.

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