

# Atrial Flutter in Infants

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<b>OBJECTIVES</b>	We sought to characterize the clinical nature of atrial flutter (AFL) in a large cohort of infants.
<b>BACKGROUND</b>	There are no large studies describing the natural history of AFL in infants. Previous studies vary in the therapy used and expected prognosis.
<b>METHODS</b>	We reviewed the records of all children younger than 1 year of age who were diagnosed with AFL at our hospital during the past 25 years, excluding those with previous cardiac surgery.
<b>RESULTS</b>	We identified 50 infants with AFL. Most, 36 (72%), presented within the first 48 h of life. Congestive heart failure was evident in 10 infants, with 6 presenting at 1 day of age, and 4 presenting beyond 1 month of age. The remainder were asymptomatic. A large atrial septal defect was the only structural heart disease. Spontaneous conversion to sinus rhythm occurred in 13 (26%) infants. Sinus rhythm was restored in 20 of 23 (87%) attempts at direct current cardioversion and 7 of 22 (32%) attempts at transesophageal pacing; 7 required antiarrhythmic therapy. An additional arrhythmia, all supraventricular, appeared in 11 (22%) infants. The recurrence of AFL developed in 6 infants; 5 of 6 of these incidents occurred within 24 h of the first episode. All patients with recurrence had an additional arrhythmia.
<b>CONCLUSIONS</b>	Infants with AFL usually present within the first 2 days of life. No association was found with structural heart disease. Direct current cardioversion appears to be most effective at establishing sinus rhythm. Chronic AFL has the potential to cause cardiovascular compromise. Atrial flutter in the absence of other arrhythmias has a low risk of recurrence. Once in sinus rhythm, infants with AFL have an excellent prognosis and may not require chronic antiarrhythmic therapy. (J Am Coll Cardiol 2006;48:1040–6) © 2006 by the American College of Cardiology Foundation

Atrial flutter (AFL) is an uncommon arrhythmia in newborns and infants. The low incidence of AFL in this age group makes it difficult to study, and previous reports vary as to the most efficacious therapy and expected prognosis (1–9). These previous studies were limited by small, non-uniform patient populations. The purpose of this study was to characterize the natural history of AFL in a large cohort of infants, including presentation, therapy, and prognosis.

## METHODS

**Inclusion criteria.** We identified all infants younger than one year of age who presented to Texas Children's Hospital with AFL during the previous 25 years. All patients had AFL documented by a standard electrocardiogram (ECG) and confirmed by a pediatric cardiologist at Texas Children's Hospital (Fig. 1). Infants with previous cardiac surgery were excluded. When AFL was diagnosed by the use of fetal echocardiogram, it was not included unless the arrhythmia persisted after birth and was documented as described.

**Data collection.** Data were obtained by retrospective chart review of all infants 1 year of age or younger with a diagnosis of AFL. All medical records from our hospital were queried,

including Texas Children's Hospital inpatient records, the Cardiology Clinic computerized database, and the computerized log of ECGs performed. Clinical information collected included age at diagnosis, signs and symptoms at presentation, method of conversion to sinus rhythm, and medications. Prenatal and birth histories also were reviewed. The ECG features analyzed were rate of AFL, atrioventricular conduction ratio, recurrence, and the development of additional arrhythmias. Echocardiographic data were reviewed, including intracardiac anatomy, the presence of atrial dilation, and ventricular function. All ECGs, 24 h Holter monitors, echocardiogram results, and follow-up clinic visits were reviewed.

**Statistical analysis.** Descriptive statistics were used to characterize ECG and clinical features. Differences in continuous variables were compared using a two-tailed *t* test. The Fisher exact test was used in evaluating proportions between categorical variables. Statistical significance was defined as a *p* value of less than 0.05. Data was analyzed using SISA (Quantitative Skills, Hilversum, the Netherlands), on Internet Explorer version 6.0 (Microsoft Corporation, Redmond, Washington).

## RESULTS

**Age and presentation.** We identified 52 infants with AFL in the first year of life with no history of previous cardiac surgery. Two patients were excluded—1 infant with persistent fetal circulation and a large clot at the tip of an

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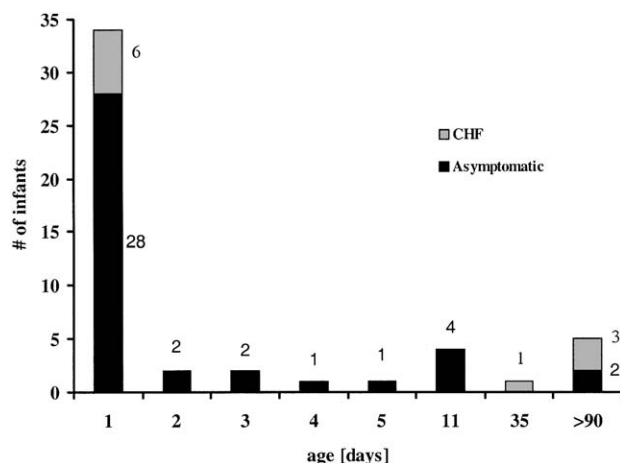
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#### Abbreviations and Acronyms

AFL	= atrial flutter
DC CV	= direct current cardioversion
DOL	= day of life
ECG	= electrocardiogram/electrocardiographic
SVT	= supraventricular tachycardia
TEP	= transesophageal pacing

umbilical venous catheter, and 1 infant who developed AFL in the postoperative period after lung resection with umbilical lines in place. Of the remaining 50 infants, there were 31 boys and 19 girls. The majority, 36 (72%), presented in AFL within the first 48 h of life; 29 at birth, 5 on day of life (DOL) 1, and 2 on DOL 2. A prenatal diagnosis of AFL had been made in 2 of these infants. An additional 8 infants presented before they were 1 month of age, and 6 presented beyond the first month of age, up to 210 days of life (Fig. 2).

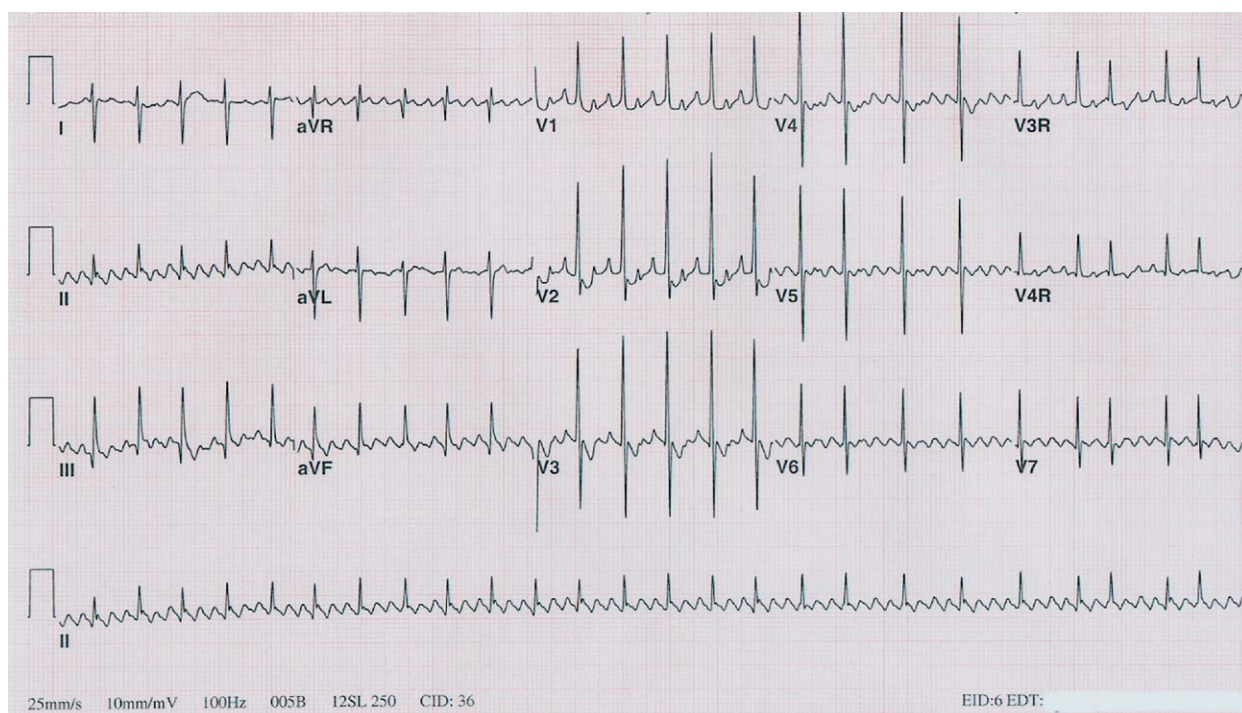
The majority of patients (40 of 50, 80%) presented with asymptomatic tachycardia noted on routine examination and monitoring. Ten infants (20%) presented with congestive heart failure and depressed ventricular function, as assessed by echocardiogram. Six infants presented in AFL at birth with cardiac compromise, including 5 infants with severe hydrops. Four infants presented with symptomatic congestive heart failure outside of the newborn period—1 at 5 weeks of life with a patent ductus arteriosus and large atrial septal defect and 3 with normal intracardiac anatomy who developed symptomatic heart failure when they were older than 3 months of life (Fig. 2). Infants who presented beyond 3 months of age were statistically more likely to



**Figure 2.** Age at presentation for infants with atrial flutter, distinguishing infants who were asymptomatic or with symptoms of congestive heart failure (CHF) secondary to atrial flutter.

develop congestive heart failure ( $p = 0.035$ ). Most patients (33 of 50, 66%) had a single episode of AFL, were asymptomatic, and easily converted to sinus rhythm. Seventeen patients (34%) had AFL complicated by congestive heart failure, recurrence, an additional arrhythmia, or AFL that was refractory to conversion to sinus rhythm. These patients are listed in Table 1 as “Infants With Complicated Atrial Flutter.”

Infants with congestive heart failure were of 2 groups. Of those presenting symptomatic on DOL 1, 5 of 6 were noted to be in tachycardia in utero at 36 to 40 weeks of gestation, prompting delivery by induced vaginal delivery or Cesarean. Five of these newborns had either AFL that was refractory



**Figure 1.** Twelve-lead electrocardiogram showing sawtooth pattern of atrial flutter with 2:1 atrioventricular conduction.

**Table 1.** Characteristics of Infants With Complicated Atrial Flutter

Patient	Age at Diagnosis (Days)	CHF	Additional Arrhythmias	Response to Conversion Attempts	Recurrence	Maintenance Medications
1	1	Yes	SVT	TEP SR	—	Digoxin, propranolol
2	1	Yes	AFIB	TEP SR	Yes	Digoxin
3	1	Yes	SVT	Failed TEP Flecainide SR Amiodarone SR	Yes	Digoxin, amiodarone
4	1	Yes	SVT, WPW	Failed CV ×6 Quinidine SR	—	Quinidine, digoxin
5	1	Yes	—	CV SR	—	Digoxin
6	1	Yes	—	Failed TEP, CV Procainamide SR	—	No follow-up
7	35	Yes PDA, ASD	—	CV SR	—	Digoxin
8	84	Yes	—	Failed TEP CV SR	—	Digoxin
9	90	Yes	—	CV SR	—	Quinidine, digoxin
10	125	Yes	—	Failed CV ×3, Propranolol SR	—	Propranolol
11	1	—	AET	Failed TEP, CV Amiodarone SR	Yes	Amiodarone, propranolol, digoxin
12	1	—	SVT	Failed TEP and CV Procainamide + CV SR	Yes	Flecainide, digoxin
13	1	—	AET	Spontaneous SR	—	Sotalol, digoxin
14	11	—	SVT	Flecainide SR	Yes	Flecainide, digoxin
15	1	—	SVT	TEP SR	—	Propranolol, digoxin
16	1	—	AET	Failed TEP Spontaneous SR	Yes	Flecainide, digoxin
17	16	—	SVT	TEP SR	—	No follow-up

AET = atrial ectopic tachycardia; AFIB = atrial fibrillation; ASD = atrial septal defect; CHF = congestive heart failure; CV = direct current cardioversion; PDA = patent ductus arteriosus; TEP = transesophageal pacing; SR = sinus rhythm; SVT = supraventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.

to multiple attempts at conversion or developed an additional arrhythmia requiring treatment (Patient #1, #2, #3, #4, and #6). The sixth newborn was converted to sinus rhythm by a single attempt at direct current cardioversion (DC CV) and had no further recurrences. The remaining four symptomatic infants presented with symptoms outside of the newborn period after development of congestive heart failure. Of these 4, 2 had AFL that was difficult to convert to sinus rhythm (Patient #8 and #10), and 1 infant had a large atrial septal defect and patent ductus arteriosus (Patient #7). She subsequently had resolution of congestive heart failure symptoms after restoring sinus rhythm.

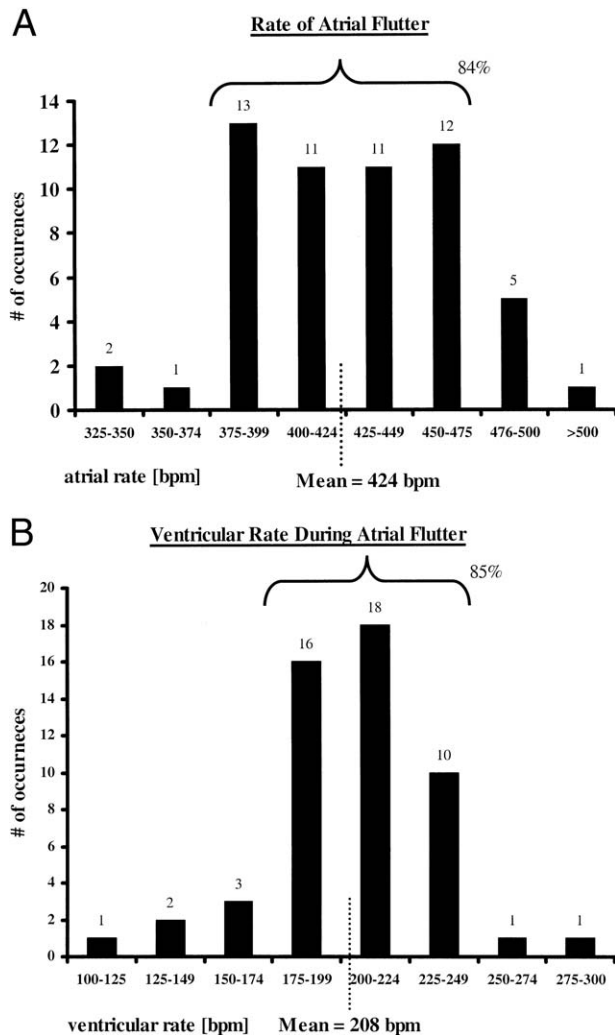
**ECG features.** The diagnosis of AFL was made by a surface ECG in all patients. The mean rate of AFL was  $424 \pm 46$  beats/min (range 340 to 580 beats/min). Mean ventricular response rate was  $208 \pm 28$  beats/min (range 125 to 280 beats/min) (Fig. 3). Atrioventricular conduction assessed by ECG was 2:1 in 75% and was variable in the remaining episodes. No statistical differences were found in ECG features between those who responded well to treatment, developed additional arrhythmias, became symptomatic, or had a recurrence of AFL.

**Echocardiographic features.** All infants had a structurally normal heart by echocardiogram except for 1 patient, who had an atrial septal defect and patent ductus arteriosus. She presented at 5 weeks of age with congestive heart failure, was converted to sinus rhythm by DC CV, treated for heart

failure, and was then lost to follow-up. She represented at 5 years of age with cardiomegaly and subsequently underwent surgical ligation of her patent ductus arteriosus and closure of her atrial septal defect. Cardiomegaly improved, and she has had no recurrence of arrhythmia. Ventricular function was depressed in 10 infants (20%), as shown by decreased shortening fraction on echocardiogram, and was normal in the remaining 40 infants. After restoration of sinus rhythm, all instances of ventricular dysfunction resolved within a matter of weeks. No patients in the study group had evidence of atrial thrombus formation.

Atrial dilation was a common finding, observed in 18 of 50 infants (36%). No relationship was found among atrial dilation and flutter rate, atrioventricular conduction ratio, the presence of an additional arrhythmia, or flutter recurrence. Of 18 patients with atrial dilation, only 2 had significant atrioventricular valve regurgitation, which was moderate in both cases. Of note, atrial dilation was present in all patients with ventricular dysfunction and was related to a later age at presentation. The mean age at presentation for patients without atrial dilation was 6 days, whereas for patients with atrial dilation, the mean age was 31 days, although result did not reach statistical significance, with a *p* value of 0.1. This suggests the atrial dilation may have resulted from the combination of 2 factors seen in patients with a longer duration of tachycardia—the chronic loss of





**Figure 3.** (A) Rate of atrial flutter. (B) Ventricular rate during atrial flutter. bpm = beats/min.

atrioventricular synchrony, and the development of ventricular dysfunction after prolonged tachycardia.

**Conversion to sinus rhythm.** All patients successfully converted to sinus rhythm either spontaneously or by intervention with DC CV, transesophageal pacing (TEP), and/or antiarrhythmic drugs. Outcomes of each method of conversion are shown in Figure 4. Twenty infants were ultimately converted by DC CV (20 of 23 attempts; 87%), and 7 infants were converted to sinus rhythm by TEP (7 of 22 attempts; 32%). Two infants developed atrial fibrillation during attempts at TEP, which reverted spontaneously to sinus rhythm. There were no other complications due to TEP or cardioversion. Spontaneous conversion to sinus rhythm occurred in 13 infants. All infants who converted spontaneously did so within a few hours of the diagnosis.

Pharmacologic therapy was instituted in 15 infants. Twelve received digoxin loading as first-line therapy. Sinus rhythm occurred in 4 infants within hours of beginning the digoxin load; the remaining eight required additional intervention. Seven infants received antiarrhythmic treatment

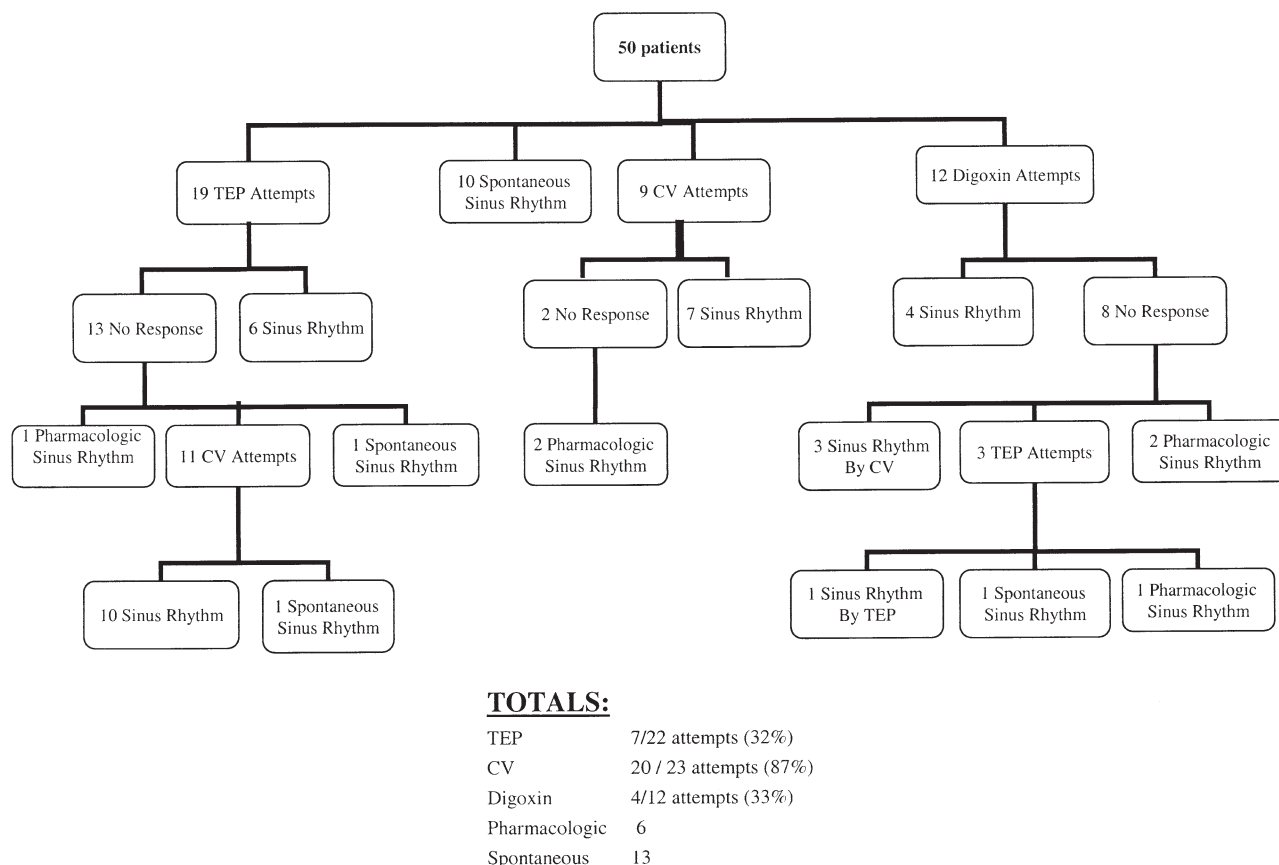
secondary to refractory AFL that persisted despite multiple attempts at conversion (Table 1) (Patient #3, #4, #6, #10, #11, #12, and #14). Patient #3 had successful control of the AFL but developed ventricular tachycardia while on flecainide; the flecainide was then changed to amiodarone. Patient #10 converted to sinus rhythm after 2 weeks on propranolol. Patient #12 was converted to sinus rhythm by DC CV after loading with procainamide and is included in the successful DC CV group. Of patients requiring antiarrhythmic therapy, 1 responded to amiodarone, 1 to flecainide, 1 to amiodarone and flecainide combined, 2 to procainamide, and 1 each to quinidine and propranolol.

All infants with symptomatic heart failure underwent prompt intervention to restore sinus rhythm, 5 with initial attempts by DC CV and 5 with TEP. Three of these patients were successfully DC cardioverted, and 2 were successfully overdrive paced. There were 3 failed initial attempts at TEP, 2 of whom were successfully cardioverted. Three patients ultimately failed multiple attempts to restore sinus rhythm and required antiarrhythmic therapy.

**Recurrence and long-term prognosis.** Six infants (12%) experienced a recurrence of AFL (Table 1). One infant had a single recurrent episode (Patient #16), 2 had two recurrences (Patient #2 and #11), and three experienced multiple recurrent episodes of AFL that required antiarrhythmic therapy to control (Patient #3, #12, and #14). The AFL recurred within 24 h of termination in 5 of 6 instances, and 1 infant had a later recurrence of AFL 5 days after his initial episode. There were no recurrences of AFL outside of this 5-day period, including over long-term follow-up (mean duration of 27 months for the 41 patients in whom follow-up data was available).

Eleven infants (22%) had a second arrhythmia in addition to AFL. These included supraventricular tachycardia (SVT) (7 patients), atrial ectopic tachycardia (3 patients), and atrial fibrillation (1 patient) (Table 1). One patient had evidence of pre-excitation, which was not evident during tachycardia. In 9 of 11 instances, the additional arrhythmia developed within 48 h of presentation with AFL (range 1 to 8 days). In infants with an additional arrhythmia, the recurrence rate of AFL was 63% (6 of 11). All infants with a recurrence of AFL had an additional arrhythmia. Of the 39 infants without an additional arrhythmia, none had a recurrence. This result represented a statistically increased risk of recurrence over infants without an additional arrhythmia ( $p < 0.001$ ). Patients with AFL that was refractory to conversion to sinus rhythm also were more likely to demonstrate an additional arrhythmia ( $p = 0.005$ ). An additional arrhythmia occurred in 4 of the 6 patients with refractory AFL who required antiarrhythmic medication (Table 1).

Of patients who developed SVT, 1 had an isolated episode of atrial fibrillation, 6 had frequent nonsustained episodes of SVT in the first few days of life that required continued drug therapy, and 1 had persistent SVT throughout the first month of life, which ultimately was controlled by amiodarone. None have had recurrence of SVT in late



**Figure 4.** Conversion of atrial flutter to sinus rhythm in 50 infants. CV = direct current cardioversion; TEP = transesophageal pacing.

follow-up. The 3 patients with atrial ectopic tachycardia (AET) had incessant tachycardia throughout the newborn period. Patient 1 had persistent AET throughout the first week of life until being administered flecainide; this patient experienced no further recurrences over the course of 5 years. Patient 13 was treated with sotalol and converted to sinus rhythm at 6 weeks of age. He did well until having a recurrence of AET at the age of 7 years. Patient #11 was able to achieve rate control only with amiodarone, propranolol, and digoxin, but was stable in AET until converting to sinus rhythm at six months of age, after which he had no further recurrence through follow-up to 16 months of age.

Of the 50 infants, 41 (82%) underwent follow-up at Texas Children's Hospital Cardiology Clinic. Maintenance medications were given to 36 of the 41 infants observed in follow-up; 5 received no medications. Antiarrhythmic drugs other than digoxin (flecainide, propranolol, sotalol, quinidine, and amiodarone) were used as chronic therapy in 11 infants. Of these 11 infants, 9 had an additional arrhythmia that required continued therapy, 1 was treated with propranolol after failing conversion attempts at DC CV, and 1 was treated with quinidine once in sinus rhythm after presenting in congestive heart failure. Once sinus rhythm was established, no patient had a recurrence of AFL during long-term follow up outside of the early recurrences described previously. Of the 5 infants who did not receive

chronic therapy, none had a recurrence. All cases of decreased ventricular function resolved within weeks of restoring sinus rhythm, as documented by repeat echocardiogram.

## DISCUSSION

This study is the largest to date of infants younger than one year of age with AFL. In general, AFL in infants is a well-tolerated arrhythmia, despite the often great atrial and ventricular rates. The rate of AFL in our population of infants was more rapid than that typically seen in adults with AFL, up to 580 beats/min, with rapid ventricular response rates, up to 280 beats/min. The diagnosis can be made from the surface ECG, which most frequently demonstrates 2:1 or variable atrioventricular conduction. Similar to previous studies, we found no association between infant AFL and structural congenital heart disease (4,5,9). By design, our study excluded infants with a history of previous cardiac surgery to study the nature of AFL in native myocardium, unaltered by scar formation. Despite the low incidence of structural heart disease, obtaining an echocardiogram can be useful to evaluate ventricular function because a significant number of infants did have ventricular dysfunction.

The potential exists, however, for cardiac compromise and the development of congestive heart failure. The devel-

opment of symptoms did not correlate with rapid atrial or ventricular rates or conduction ratio but appeared to be related to the duration of the tachycardia, which is consistent with the findings of Naheed et al. (10) and Martin and Hernandez (7). Of the 10 infants who presented with symptoms of congestive heart failure, all had a history compatible with prolonged tachycardia. Infants who were symptomatic were of 2 groups. Of those 6 presenting symptomatic on DOL 1, 5 were noted to be in tachycardia in utero at 36 to 40 weeks of gestation, prompting delivery by induced vaginal delivery or Cesarean. Five of the 6 continued with persistent tachycardia after birth; 3 had multiple recurrences of AFL, 4 had an additional arrhythmia, and 1 had AFL refractory to multiple attempts at cardioversion until the addition of procainamide. The remaining four presented outside of the newborn period after the development of symptoms of congestive heart failure. These 2 groups share a common potential mechanism for the development of ventricular dysfunction in that both likely had prolonged periods of tachycardia before detection, either before birth or after discharge from the nursery until the development of symptoms.

We found DC CV and TEP to be safe methods to attempt restoration of sinus rhythm. There was a low incidence of atrial fibrillation during attempts at TEP, and no complications from DC CV were observed. Direct current cardioversion appeared more successful than TEP at establishing sinus rhythm. However, our conversion rate for TEP was lower than previously reported (5,11,12). In this retrospective study, without data available regarding pacing protocols, we are unable to compare the effectiveness of these 2 modalities. A prospective study would be better suited to compare the efficacy of DC CV versus TEP for conversion of AFL.

**Study limitations.** One of the limitations faced in this study relates to the wide range of time periods during which study patients were treated, during which the management strategies were not uniform. Across patients in this study, the initial attempts at conversion were more commonly TEP; however, an approximately equal number of patients ultimately underwent attempts at DC CV and TEP. A number of infants spontaneously converted to sinus rhythm before initial attempts at conversion, as well as a small number who failed repeated attempts at electrical conversion and required antiarrhythmic medications. Overall, infants with symptomatic heart failure underwent prompt intervention to restore sinus rhythm; none of these infants converted to sinus rhythm spontaneously. This result likely represents a more urgent triage of symptomatic infants to undergo immediate intervention, as well as potentially a more persistent state of tachycardia resulting in the development of symptoms, that was less likely to convert spontaneously.

Spontaneous conversion to sinus rhythm occurred in 13 (26%) infants. Although exact data on the time of conversion to sinus rhythm were not available, all reverted to sinus rhythm within 24 h of diagnosis. We also saw conversion of

4 of the 12 infants who received digoxin therapy initially, and all converted within hours of receiving the first dose. Although previously the use of digoxin has been supported as initial therapy for pharmacologic conversion (2), its efficacy has been brought into question based on the widely variable time between the initiation of digoxin therapy and conversion to sinus rhythm (5,7). It may be argued that the 4 patients who converted after receiving digoxin therapy in our study may have actually done so spontaneously. We agree with those authors who, based on efficacy, do not recommend digoxin as a first-line therapy for conversion to sinus rhythm (3,5,7). Electrical conversion by DC CV is the most effective method to establish sinus rhythm, although in the asymptomatic infant, a waiting period of 6 to 12 h may be considered, given the possibility of spontaneous conversion. In symptomatic infants, we recommend prompt use of DC CV.

The presence of an additional arrhythmia was the only statistically significant factor identified for recurrent and refractory AFL. Additional supraventricular arrhythmias appear to be fairly frequent (22%). Infants with an additional arrhythmia had a significantly increased risk of recurrence of AFL, and of having AFL that was refractory to attempts at conversion. When either a recurrence of AFL or an additional arrhythmia occurred, it was most likely to be evident within 48 h of termination of the first episode and was unlikely beyond 72 h. Previous reports have shown an association between AFL and the presence of accessory pathways (8). In our patient population, only one infant had evidence of pre-excitation, which was not manifested during AFL. Given the frequency of re-entrant SVT in our patient population (7 of 50, 14%) and that the most likely mechanism in this age group is an accessory pathway, one might even speculate an association of AFL with concealed pathways.

Chronic maintenance therapy was given to most of the infants. Most received digoxin, whereas those with an additional arrhythmia or refractory AFL received other antiarrhythmic medications. The fact that the majority received therapy makes it difficult to assess the need for chronic drug therapy in these infants. However, in our more recent experience with five infants who did not receive any maintenance therapy, none had a recurrence, suggesting that in infants with uncomplicated, asymptomatic AFL who convert spontaneously or respond easily to electrical conversion to sinus rhythm, maintenance therapy may not be necessary.

Atrial flutter remains a rare tachycardia in the newborn and young infant and usually presents in the newborn period with asymptomatic tachycardia. Congestive heart failure may develop and appears to be most related to the duration of the tachycardia. Isolated AFL in a newborn does not suggest an underlying structural heart defect, even if signs or symptoms of congestive heart failure are present. Infants with AFL respond well to DC CV but may convert to sinus rhythm spontaneously. On occasion, AFL may be refractory to attempts at electrical cardioversion or have recurrences

requiring the use of antiarrhythmic medications. Additional supraventricular arrhythmias, if present, greatly increase the risk for the recurrence of AFL. In the absence of additional arrhythmias, infants with AFL have an excellent prognosis once in sinus rhythm with a low risk of recurrence, and chronic antiarrhythmic therapy is unlikely to be necessary.

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