CARDIAC AND PULMONARY REPLACEMENT

ATRIAL FLUTTER: A NEWLY RECOGNIZED COMPLICATION OF PEDIATRIC LUNG TRANSPLANTATION

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Background and methods: Atrial flutter after pediatric lung transplantation has not previously been reported. We reviewed the records of 78 children who underwent lung transplantation at our institution to characterize the incidence and clinical course of postoperative atrial flutter. The diagnosis was based on either a surface or transesophageal electrocardiogram that demonstrated a fixed cycle length atrial tachycardia that did not require ventricular participation. **Results:** Atrial flutter occurred in seven of 62 (11.3%) patients who underwent bilateral sequential lung transplantation, zero of 10 patients after single lung transplantation, and zero of six patients after heart-lung transplantation. Ages of the patients with atrial flutter ranged from 2.5 months to 14 years. Electrocardiographic findings among patients varied with respect to p-wave morphology and atrioventricular conduction. No patient had a prior atrial arrhythmia or coexisting structural cardiac disease. None had atrial flutter in relation to a rejection episode. Two patients had atrial flutter transiently during the first postoperative day, but it resolved spontaneously. Five patients had recurrent atrial flutter that began 13 ± 7 days after the operation. The mean cycle length of atrial flutter was 196 ± 65 msec. The arrhythmia was unresponsive to digoxin in four patients to whom it was administered. It was controlled with procainamide in four patients and with flecainide in one. At 1 and 6 months after lung transplantation, procainamide was discontinued in two patients without recurrence. One patient died of bronchiolitis obliterans 6 months after the operation while still receiving flecainide. Two patients continue to receive procainamide 4 and 7 months after transplantation. **Conclusions:** (1) Atrial flutter commonly occurs after bilateral lung transplantation in children. (2) Electrocardiographic manifestations are variable. (3) Type 1 antiarrhythmic agents provide satisfactory control. (J Thorac Cardiovasc Surg 1996;112:984-91)

Lung transplantation is increasingly being considered as a viable treatment alternative for children who have a variety of end-stage restrictive and obstructive pulmonary diseases or end-stage pulmonary vascular diseases. Intermediate-term results

are encouraging.¹⁻³ Complications of the procedure include rejection, those consequent to immunosuppression, and those resulting from the operative procedure, including phrenic nerve palsy, bronchial stenosis, and vascular anastomotic strictures. Ar-

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rhythmias have not been described after lung transplantation, in either children or adults.

We observed atrial flutter (AFL), or intraatrial reentrant tachycardia, in numerous children after lung transplantation at our institution. The purpose of this study was to characterize the prevalence, clinical course, and response to therapy of AFL after pediatric lung transplantation.

Methods

Population and clinical variables. All patients undergoing lung transplantation or heart-lung transplantation between January 1990 and October 1995 at St. Louis Children's Hospital were identified through a retrospective review of hospital records. No patients were excluded from the analysis. Records were reviewed to identify pertinent demographic data including age and sex. Clinical data recorded included preoperative diagnosis, type of transplant (single lung, bilateral sequential lung, or heart-lung), type of donor (cadaveric or living-related donor), operative technique, onset of AFL, cycle length, therapy received, and outcome.

Operative technique. A standard technique of pulmonary transplantation was used with end-to-end pulmonary arterial and bronchial anastomoses.⁴ The pulmonary venous anastomoses were performed by placing a vascular clamp on the left atrium to include the pulmonary vein orifices on the appropriate side. The stumps of these veins were excised and the atrial tissue between them divided, providing a wide orifice of atrial tissue for the anastomosis to the donor left atrial cuff. Heart-lung transplantation was performed with end-to-end tracheal, aortic, and right atrial anastomoses.⁵

Diagnosis of AFL. The diagnosis of AFL was predominantly determined from the surface electrocardiogram during periods of variable atrioventricular nodal conduction, where consecutive nonconducted p waves of constant morphology, polarity, and cycle length were distinguishable. Transesophageal electrocardiography was used in selected instances to clarify the relationship between atrial and ventricular activity. Criteria to distinguish AFL from ectopic atrial tachycardia were pace termination, termination by direct-current cardioversion, sudden onset without "warm-up" or acceleration of rate, and a cycle length less than 200 msec.

The nomenclature for reentrant arrhythmias within the atrium has been debated, especially with respect to congenital heart disease. Some restrict the term *atrial flutter* to those patients with classic inverted "saw-toothed" p waves in the inferior leads, preferring the term *intra-atrial reentrant tachycardia* for all other atrial arrhythmias involving macroentry. For our purposes, any presumed macroreentrant atrial arrhythmia was classified as AFL.

Drug monitoring. Patients begun on antiarrhythmic therapy were monitored with serial electrocardiograms, 24-hour Holter monitors, echocardiograms, drug levels, and complete blood counts.

Table I. Patient characteristics

	AFL (n = 7)	No AFL (n = 71)	p Value
Age	7.4 ± 5.9 yr (2.5 mo to 14 yr)	9.9 ± 6.4 yr (1 day to 25 yr)	0.40
Sex			0.99
Male	3 (43%)	28 (39%)	
Female	4 (57%)	43 (61%)	
Procedure			0.18
BSLT	7	55	
SLT	0	10	
HLT	0	6	
Donor type			0.25
Cadaveric	6	69	
Living-related	1	2	
6 mo survival	83%	79%	0.99

BSLT, Bilateral sequential lung transplantation; SLT, single lung transplantation; HLT, heart-lung transplantation.

Statistical analysis. All values for each group are expressed as mean ± standard deviation. Statistical significance of paired data was determined by Student's paired *t* test. Categorical data were compared by means of χ^2 analyses (SYSTAT 5.0, SYSTAT Inc., Evanston, Ill.). A *p* value less than 0.05 was considered statistically significant.

Results

Prevalence and presentation of AFL. Between 1990 and 1995, lung transplantation or heart-lung transplantation was performed in 78 patients, with a mean age of 9.7 ± 6.3 years. Among 62 patients who underwent bilateral sequential lung transplantation, postoperative AFL developed in seven (11.3%). No episodes of AFL were documented in children after single lung transplantation (*n* = 10) or heart-lung transplantation (*n* = 6). En-bloc double lung transplantation was not performed. Other clinical variables, including age, sex, and donor type, and 6-month survival did not significantly differ between patients who had AFL and those who did not (Table I).

No patient who had postoperative AFL had co-existing structural cardiac disease that may have contributed to the arrhythmia. All were in normal sinus rhythm before the operation. No occurrence of postoperative AFL was associated with an episode of rejection.

AFL was generally well tolerated. No patient had hemodynamic collapse. However, symptoms developed in three patients, including palpitations in patient 5 and dyspnea in patients 2 and 4. Routine intensive care unit monitoring identified AFL in the other children. In two patients, AFL occurred transiently during the first postoperative day and re-

Fig. 1. AFL with inverted p waves. A 12-lead electrocardiogram demonstrates the initiation of AFL in patient 1, marked by the arrow in the continuous lead II rhythm strip. Consecutive, nonconducted p waves are inverted in lead aVF, characteristic of spontaneous human type I AFL. The cycle length of the arrhythmia is 160 msec. AFL, Atrial flutter; AV, atrioventricular; f, flutter wave; SR, sinus rhythm.

Table II. Clinical characteristics of patients with postoperative AFL

Patient	Age	Sex	Diagnosis	Onset of AFL (postop day)	Cycle length (msec)	Acute drug therapy
1	2.5 mo	M	Primary pulmonary hypertension	24	160	D, P
2	11 yr	F	Primary pulmonary hypertension	12	160	D, P
3	10 yr	M	Cystic fibrosis	8	150	D, F
4	14 yr	F	Cystic fibrosis	8	300	D, P
5*	13 yr	F	Cystic fibrosis	11	280	P
6	12.5 mo	M	Fibrosing alveolitis	1	160	None
7	2.5 yr	F	Bronchiolitis obliterans	1	160	None

D, Digoxin; F, flecainide; P, procainamide.

*Organ from living related donor.

solved spontaneously without any therapeutic interventions. The remaining five children had recurrent paroxysmal AFL, initial episodes occurring 8 to 24 days after the operation (mean 13 ± 7 days). The mean atrial cycle length of the tachyarrhythmia was 196 ± 65 msec (Table II).

Reentry was clearly established as the mechanism of the tachycardia in most patients. This was demonstrated by termination of the arrhythmia with overdrive pacing in patient 2 and by direct-current cardioversion in patient 4. In patients 1 and 3, the onset of the tachycardia without any evidence of

“warm-up” or acceleration of rate was detected. Patients 6 and 7 were presumed to have a reentrant mechanism because of their tachycardia cycle lengths of 160 msec, shorter than has been described clinically for ectopic atrial tachycardias.

Electrocardiographic manifestations of AFL. The electrocardiographic manifestations of AFL were varied. In patient 1, inverted saw-toothed p waves, characteristic of human type I AFL,⁶ were present in the inferior leads (Fig. 1). At the onset of the tachycardia, consecutive, nonconducted p waves with a cycle length of 160 msec were evident.

Fig. 2. AFL with upright p waves. A 12-lead electrocardiogram demonstrates AFL with a cycle length of 160 msec and variable atrioventricular conduction in patient 2. Nonconducted p waves are upright in leads II, III, and aVF. *f*, Flutter wave.

Subsequently, the heart conducted 2:1 through the atrioventricular node. After stabilization of atrioventricular conduction, the R-R interval became fixed at a cycle length of 320 msec; the nonconducted p waves were concealed in the QRS complexes. Had the initiation not been captured, it would have been difficult to correctly establish the diagnosis of AFL with 2:1 atrioventricular block from the remainder of the electrocardiogram. In patient 2 (Fig. 2), variable atrioventricular conduction also permitted the diagnosis of AFL. The atrial cycle length of 160 msec was identical to that of patient 1; however, the p waves were of opposite polarity.

In addition to p wave differences, variability in atrioventricular conduction resulted in ostensibly different tracings that may have been the result of a single underlying rhythm disturbance. Patient 4 at times had an irregular atrial tachycardia and on other occasions had a fixed cycle length atrial tachycardia (Fig. 3). Although an initial electrocardiogram resembled atrial fibrillation, a transesophageal electrogram performed at that time demonstrated a regularly irregular atrial rhythm. The atrial irregularity, combined with variable atrioventricular con-

duction, produced irregularly irregular R-R intervals on the surface electrocardiogram, typical of atrial fibrillation. A subsequent Holter monitor evaluation revealed AFL, as periods of slowed atrioventricular conduction unmasked the flutter waves with a cycle length of 300 msec. During the same monitoring period, a wide QRS complex tachycardia of identical cycle length, probably representing AFL with aberrant ventricular conduction, was also present in the same patient.

Acute management of AFL. Digoxin was administered as primary therapy in four of five patients but was unsuccessful in every instance. AFL was successfully controlled with procainamide in four patients and with flecainide in one. AFL was terminated acutely by esophageal atrial overdrive pacing in one patient (Fig. 4) and by direct-current cardioversion in another (patient 4) before the start of chronic pharmacologic therapy.

Follow-up. Follow-up ranged from 2 to 12 months (mean 7.1 ± 3.8 months). No proarrhythmic, negative inotropic or other side effects related to antiarrhythmic therapy were noted. Neither of the two children who had transient AFL on the first postoperative day (patients 6 and 7) had a recur-

Fig. 3. Variable electrocardiographic manifestations of AFL in the same patient. **A**, A 12-lead electrocardiogram in patient 4 demonstrates a rhythm with irregular R-R intervals, resembling atrial fibrillation. **B**, An esophageal electrocardiogram performed at the same time demonstrates a regularly irregular atrial rhythm. The atrial complexes occur at alternating cycle lengths of 260 and 510 msec. These depolarizations may represent sinus beats followed by atrial echo beats or they may be manifestations of a protected reentrant circuit with a cycle length of 260 msec, from which there is 3:2 exit block. **C**, A Holter recording in which slowed atrioventricular conduction unmasks the flutter waves. The cycle length of the tachycardia is 300 msec. **D**, A wide QRS complex tachycardia on the same Holter recording with an identical cycle length of 300 msec, probably representing AFL with aberrant ventricular conduction. *A*, Atrial depolarization; *ESO*, esophageal atrial lead; *III*, surface electrocardiogram lead III; *ESO/III*, hybrid lead of *ESO* and *III*; *f*, flutter wave; *V*, ventricular depolarization.

rence. In patients 5 and 1, procainamide was discontinued at 1 and 6 months after the operation, respectively, without recurrence. Six months after lung transplantation, patient 3 died of bronchiolitis obliterans while still receiving flecainide; before his death, this patient was free of AFL. Patients 2 and 4 continued to receive procainamide, 7 and 4 months after the operation, respectively. Three months after the operation, discontinuation of procainamide in patient 4 resulted in recurrence of AFL within 24 hours.

Discussion

Prevalence and diagnosis of AFL. AFL has not previously been reported after lung transplantation in children or adults. The experience with lung transplantation in children is limited. The relatively high percentage of patients with postoperative AFL that we observed is in part a function of the large volume of our pediatric lung transplant experience. However, AFL may also be underdiagnosed in this

setting as a result of inconsistent and variable electrocardiographic findings.

Establishing the diagnosis of AFL after lung transplantation necessitates a high index of suspicion. The atrial cycle lengths encompass a wide range. The axis and morphology of flutter waves may differ among children. Variability in atrioventricular conduction can result in irregular R-R intervals. Wide complex tachycardias may also represent AFL with aberrant ventricular conduction. Fixed cycle length tachycardias in children after lung transplantation should not be labeled as nonspecific paroxysmal supraventricular tachycardia until AFL has been excluded. A 12-lead electrocardiogram is more useful than a rhythm strip, because diagnostic nonconducted p waves may not be apparent in all leads. In perplexing diagnostic cases, inducing atrioventricular nodal conduction block with either vagal maneuvers or adenosine may be helpful. A transesophageal atrial electrocardiogram may also assist in achieving the correct diagnosis. In addition, a

Fig. 4. Esophageal pacing terminates AFL. Surface electrocardiogram leads II and aVL demonstrate a narrow complex tachycardia of fixed cycle length in patient 2. The diagnosis of AFL in this child was previously established (Fig. 2). The *arrow* marks the onset of the stimulus artifact from transesophageal pacing at a cycle length of 180 msec. After cessation of atrial burst pacing, the patient is in sinus rhythm. *AFL*, Atrial flutter; *SR*, sinus rhythm.

temporary atrial epicardial electrode implanted at the time of the operation may aid in the diagnosis of postoperative rhythm disorders and may permit prompt termination of AFL with atrial overdrive pacing techniques.

Irregular rhythms, as determined by R-R intervals, must be carefully scrutinized. Although such arrhythmias may represent atrial fibrillation or ectopic atrial tachycardias with varying exit block, they may also represent variants of AFL. In patient 4 (see Fig. 3), transesophageal electrocardiography demonstrated a regularly irregular atrial rhythm with alternating atrial cycle lengths of 260 and 510 msec. This repetitive atrial pattern may have represented sinus rhythm followed by atrial echo beats. However, it may have instead represented a protected reentrant circuit with a cycle length of 260 msec, from which there was 3:2 interatrial exit block. The ensuing Holter monitor tracing in this patient clearly manifested AFL with a cycle length of 300 msec. We speculate that as the tachycardia slowed from a cycle length of 260 to 300 msec, 1:1 interatrial conduction was permitted. This phenomenon, in which abnormalities in interatrial conduction during reentry can produce a surface electrocardiogram with variable R-R intervals and without typical flutter waves, has been previously described⁷ and was termed "impure flutter" by Lewis.⁸

Management of AFL. Various modalities are available for the treatment of patients with AFL. Atrial overdrive pacing and direct-current cardioversion were used in isolated instances in our series and are preferred if rapid restoration of sinus rhythm is desired. Digoxin, considered by some the drug of choice for AFL in children,⁹ was invariably unsuccessful in our patients. Conversely, type 1

antiarrhythmic agents were consistently beneficial and well tolerated.

Our series is too small to permit firm recommendations regarding the appropriate length of treatment. However, it appears that some children lose their vulnerability to AFL within a few months after lung transplantation whereas others require pharmacologic therapy for longer periods. On the basis of our preliminary experience, we have adopted a policy of empiric discontinuation of antiarrhythmic medication 6 months after lung transplantation in children who have not had a recurrence and who are otherwise clinically well.

Etiology of AFL. The reasons for the occurrence of AFL as a complication of pediatric pulmonary transplantation are speculative. Few specific functional conditions exist that would increase the likelihood of AFL after lung transplantation. In cardiac transplantation, an equivocal association has been established between sustained AFL and organ rejection.¹⁰⁻¹³ No association between rejection and AFL was observed in the present series. Even if other factors were important, such as alterations in general sympathetic drive or cardiac conduction after general anesthesia or cardiopulmonary bypass, other conditions must exist, because AFL is exceedingly uncommon in children and young adults after other procedures that use such measures.¹⁴

AFL is a common complication after a variety of operations for congenital heart disease in which complex atrial suture lines and incisions are used. Examples include the Mustard operation for transposition of the great arteries¹⁵ and the Fontan procedure for single ventricle pathology.¹⁶ Conversely, AFL in children without gross structural heart disease or in children after cardiac operations

not involving the atria is remarkably uncommon. Animal model simulations of the Mustard procedure and modified Fontan repair have emphasized the importance of anatomic barriers in the reentrant circuits,^{17, 18} in concordance with numerous previous investigations in which AFL has been demonstrated to occur by a mechanism of reentry involving circus movement around a central anatomic obstacle.¹⁹⁻²¹ We therefore hypothesize that AFL after pediatric lung transplantation is related to a surgical alteration in normal atrial anatomy. The pattern of left atrial incisions and suture lines placed during the performance of the pulmonary venous anastomoses might create the fundamental substrate (electrical discontinuity) for the inducibility of AFL. This theory is supported by our observation that no instances of AFL occurred in children after single lung or heart-lung transplantation. A heart-lung transplantation involves no left atrial anastomoses. In single lung transplantation, only one left atrial anastomosis is performed. The resultant area of conduction block may be too small to support a stable reentrant tachycardia in this circumstance. Perhaps the larger region of conduction block created in the left atrium with bilateral lung transplantation contributes to the greater prevalence of AFL in this group.

Limitations of the study. Rigid electrophysiologic criteria to distinguish automaticity from reentry were not demonstrated for three patients. However, in patients 6 and 7, the tachycardia cycle lengths were considerably shorter than has been previously reported for ectopic atrial tachycardia. Only in patient 5 could abnormalities of automaticity as the underlying mechanism of the observed arrhythmia not be definitively excluded. However, ectopic atrial tachycardias are uncommon after operations for congenital heart disease when compared with AFL.²²

Because of the prompt response of AFL to type I antiarrhythmic drugs, intracardiac mapping was not performed. Therefore no data are available regarding the critical sites of reentry within the atria. AFL in our patients was identified because of intensive care unit monitoring or cardiac telemetry or because specific symptoms directed further investigations. Routine atrial electrograms were not monitored and routine postoperative Holter monitoring was not used. Thus, the prevalence of postoperative AFL may have been underestimated in this series.

Conclusions

AFL commonly occurs after bilateral lung transplantation in children. The electrocardiographic manifestations are variable. Type 1 antiarrhythmic agents provide satisfactory control. Further studies are warranted to elucidate the mechanism of AFL in this setting.

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