

Atrial Flutter: An Uncommon Pediatric Manifestation of Hyperthyroidism

ABSTRACT. *Objective.* Atrial flutter is an uncommon arrhythmia in the pediatric population except for the immediate newborn period or following atrial repair of congenital heart disease. In children the diagnosis of atrial flutter may be difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. Atrial flutter secondary to hyperthyroidism has been rarely reported in older adults, but there are no reports of children presenting with atrial flutter as the initial manifestation of hyperthyroidism.

Case Report. We report an interesting case of hyperthyroidism in a 3-year-old presenting with congestive heart failure and atrial flutter with 1:1 atrioventricular conduction. The responses to adenosine administration and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of hyperthyroidism.

Conclusion. When atrial flutter is encountered in a pediatric patient in whom there is 1:1 atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis. *Pediatrics* 1997;100(2). URL: <http://www.pediatrics.org/cgi/content/full/100/2/e11>; atrial flutter, hyperthyroidism, pediatric, adenosine.

Atrial flutter is an uncommon arrhythmia in the pediatric population. It is principally encountered in the newborn period or after atrial repair of congenital heart disease. Although hyperthyroidism is clearly known to be a cause of atrial flutter in older adults,^{1,2} there are no reports of children presenting with atrial flutter as the initial manifestation of thyrotoxicosis.

When children present with atrial flutter, the diagnosis is often difficult, attributable to rapid atrioventricular conduction and superimposition of flutter waves on QRS and T waves. The presence of hyperthyroidism would be expected to further affect these relationships. We report here an interesting case of thyrotoxicosis in a 3-year-old presenting with atrial flutter in whom the response to adenosine administration and to cardioversion were unusual and ultimately helpful in suggesting the diagnosis of thyrotoxicosis.

CASE REPORT

A 3-year-old black girl presented with an increased heart rate noted for 9 hours and decreased activity. She had a similar episode of "fast heart rate" 2 days previously. She had no history of fever, heat intolerance, neck pain, significant weight loss or hoarseness, but was extremely hyperactive. Her past medical history was only remarkable for scarlet fever treated with amoxicillin 2 months before admission. The family history was remarkable for a maternal aunt with an overactive thyroid and another cousin with insulin-dependent diabetes mellitus.

On physical examination, she was 17.3 kg and had a regular heart rate of 275 beats/minute, respiratory rate of 66 breaths/minute, blood pressure of 111/78 mm Hg, and a axillary temperature of 37.7°C. She had moderate retractions. There was a nontender, enlarged thyroid gland. The lung fields were clear. The

precordium was hyperdynamic with normal first and second heart sounds, and easily audible third and fourth heart sounds, but no murmurs. The liver was palpable 5 cm below the right costal margin, nontender, and extended slightly left of midline. The peripheral pulses were normal and equal without radial-femoral delay, and there was normal peripheral perfusion.

An electrocardiogram (ECG) showed a narrow QRS complex tachycardia with a QRS duration of 60 ms and a rate of 280 beats/minute (Fig 1). There were typical atrial flutter waves, best seen in leads II, V1, and V2, with 1:1 atrioventricular conduction. Laboratory studies included an arterial blood gas determination which showed a moderate metabolic acidosis, normal electrolytes with the exception of a serum bicarbonate of 15 mEq/L, and mild elevation in liver transaminases (AST, 179; ALT, 174). Chest radiograph demonstrated moderate cardiomegaly with a small right pleural effusion.

Adenosine at doses of 100, 200, 400, and 600 µg/kg was given by rapid peripheral intravenous infusion without a change in heart rate. The patient underwent successful direct current cardioversion using 10 Joules, to a sinus tachycardia with a rate of 180 beats/minute. (Fig 2). On echocardiogram, there was diminished ventricular function with a percent fractional shortening of 27%. There was left atrial enlargement (left atrial diameter of 29 mm), left ventricular dilatation (end diastolic dimension of 36 mm), and mild mitral regurgitation.

After successful cardioversion, the patient was started on maintenance digoxin, and a slowly decreasing sinus rate was observed. Laboratory studies at presentation confirmed the clinical suspicion of hyperthyroidism (normal values in parentheses): thyroid-stimulating hormone (high sensitivity), .002 mU/L (0.5 to 3.0); thyroxine (T₄), 12.1 (5.5 to 12); and free T₄ index, 19.5 (5.5 to 12). Repeat thyroid function tests done several days later indicated persistently severe thyrotoxicosis: free T₄, 3.1 ng/dL (0.6 to 2.0); free thyroxine index, 20.8; and triiodothyronine (T₃), 385 ng/dL (100 to 275). Antithyroglobulin antibody was not present but antithyroid microsomal antibody titer was elevated 1 to 6400 (normal, 0 to 100). Ultrasound imaging showed a homogeneously enlarged thyroid. Supersaturated potassium iodide therapy was begun. Her family declined antithyroid medication or radioactive iodine and she subsequently underwent surgical resection of her thyroid gland. All other viral and bacterial studies were negative. Pathologic evaluation was remarkable for a diffusely enlarged thyroid gland weighing 6 g with histologic evidence of chronic lymphocytic thyroiditis and diffuse lymphocytic follicles, consistent with Hashimoto's thyroiditis. Her postoperative course was complicated by transient hypocalcemia. Follow-up echocardiograms showed a left atrial diameter of 20 mm, improved myocardial function with a percent fractional shortening of 33%, and trace mitral regurgitation. Atrial flutter did not recur, and digoxin was successfully discontinued 3 months after institution.

DISCUSSION

This case is unique, in that thyrotoxicosis presented in a patient in the pediatric age group as atrial flutter. It is instructive, both for the uniqueness of the presentation, and for the fact that the clinical manifestations of atrial flutter were unusual and somewhat confusing initially, owing to the presence of hyperthyroidism. Typically, 1:1 atrioventricular conduction of atrial flutter is rare at any age, including children, and 2:1 conduction is more typical. Such 1:1 conduction may be associated with rapidly progressive congestive heart failure. Normally, the diagnosis of atrial flutter can be made by observing the response to intravenous adenosine, an approach which failed in this case despite a seemingly more-than-adequate administered dose. The unusual aspect of this presentation can be explained, when one consid-

Received for publication Oct 31, 1996; accepted Feb 18, 1997.

Reprint requests to (W.A.S.) Pediatric Cardiology, RBC 380, Rainbow Babies and Children's Hospital, 11100 Euclid Avenue, Cleveland, OH 44106-6011. PEDIATRICS (ISSN 0031 4005). Copyright © 1997 by the American Academy of Pediatrics.

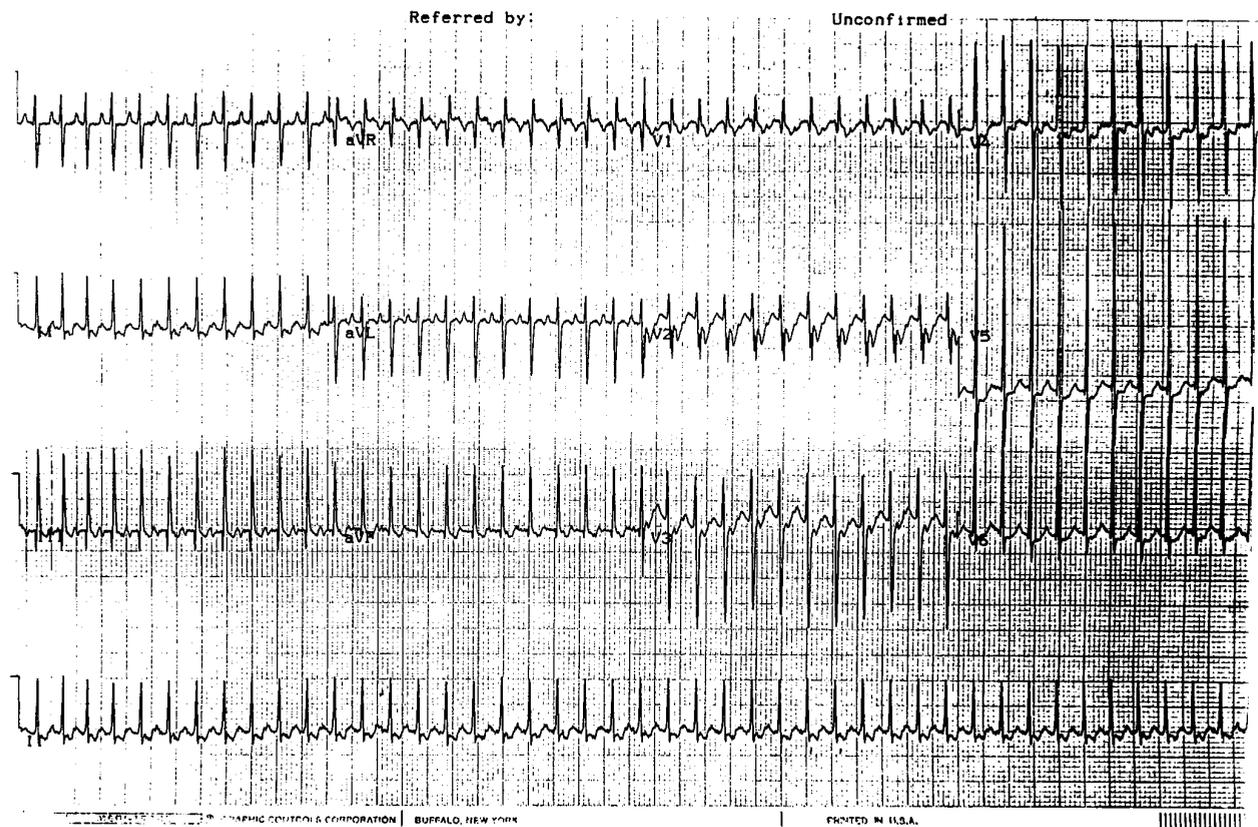


Fig 1. Twelve-lead ECG at presentation demonstrating a narrow QRS tachycardia with 1:1 atrioventricular conduction. Negative flutter waves are best seen in lead V2, but are also well seen in V1, V3, and the inferior limb leads (II, III, and aVF).

ers the effect of hyperthyroidism on the heart and conducting tissue.

Atrial flutter as a manifestation of hyperthyroidism, to the best of our knowledge, has not been previously reported in a child. Previous studies evaluating children with hyperthyroidism have reported that sinus tachycardia, increased ECG voltages, left atrial hypertrophy, left ventricular hypertrophy, or combined ventricular hypertrophy are the most commonly seen electrocardiographic effects.³ Very rarely, atrial fibrillation, ventricular fibrillation, or complete atrioventricular block have also been documented.⁴ In the largest published study evaluating 380 children and young adults with atrial flutter, none were attributable to hyperthyroidism.⁵ From a different perspective, review of several series of thyrotoxicosis in childhood encompassing 279 patients failed to identify any with atrial flutter.⁶⁻¹⁰

Serum thyroid hormone has direct and indirect effects on the heart. T₃ and T₄ both stimulate the Ca-ATPase of the myocardial sarcolemma resulting in decreased extracellular calcium, accelerated diastolic relaxation, and enhanced sinoatrial automaticity. T₄ influences automaticity of the sinoatrial node and atrial tissue by increasing the rate of phase four depolarization resulting in a decreased action potential duration and increased frequency of sinoatrial node discharge. The latter effects when coupled with the enhanced catecholamine response also seen in thyrotoxicosis, leads to shortening of the atrial effective refractory period as well as decreasing the atrial

stimulation threshold in late diastole.^{11,12} Shortening of atrial refractoriness may act to promote atrial flutter, by allowing areas of block to recover quickly enough to allow reentry.¹³

Thyroid hormone has also been shown by Goel et al⁴ in 1972 to increase atrioventricular conduction in hyperthyroid dogs. In the latter study hyperthyroid denervated dog hearts were shown to have a decreased atrioventricular node functional refractory period and conduction time allowing for faster ventricular rates independent of the sympathetic nervous system. This may help to explain why patients with thyrotoxicosis and supraventricular tachycardia tend to have faster ventricular rates despite normal to subnormal levels of serum catecholamines.¹⁴⁻¹⁷ Others have shown that the increased catecholamine sensitivity may be due to either increased adrenergic receptor density or G-protein synthesis and not attributable to increased levels of serum catecholamines.¹⁸

In normal doses, the endogenous nucleoside adenosine inhibits atrioventricular conduction and, when given to patients with atrial flutter, the brief interruption of atrioventricular conduction reveals the flutter waves. However, it failed to have any such effect in this patient despite very high doses. The lack of a response to adenosine was unusual, but explainable when one considers the known effects of thyroid hormone. Adenosine acts by competing for binding at the adenosine 1 receptor which seems to mediate the inhibition of atrioventricular node conduction. In this case however, it may be that thyroid hormone also competitively binds, induces increased inactiva-

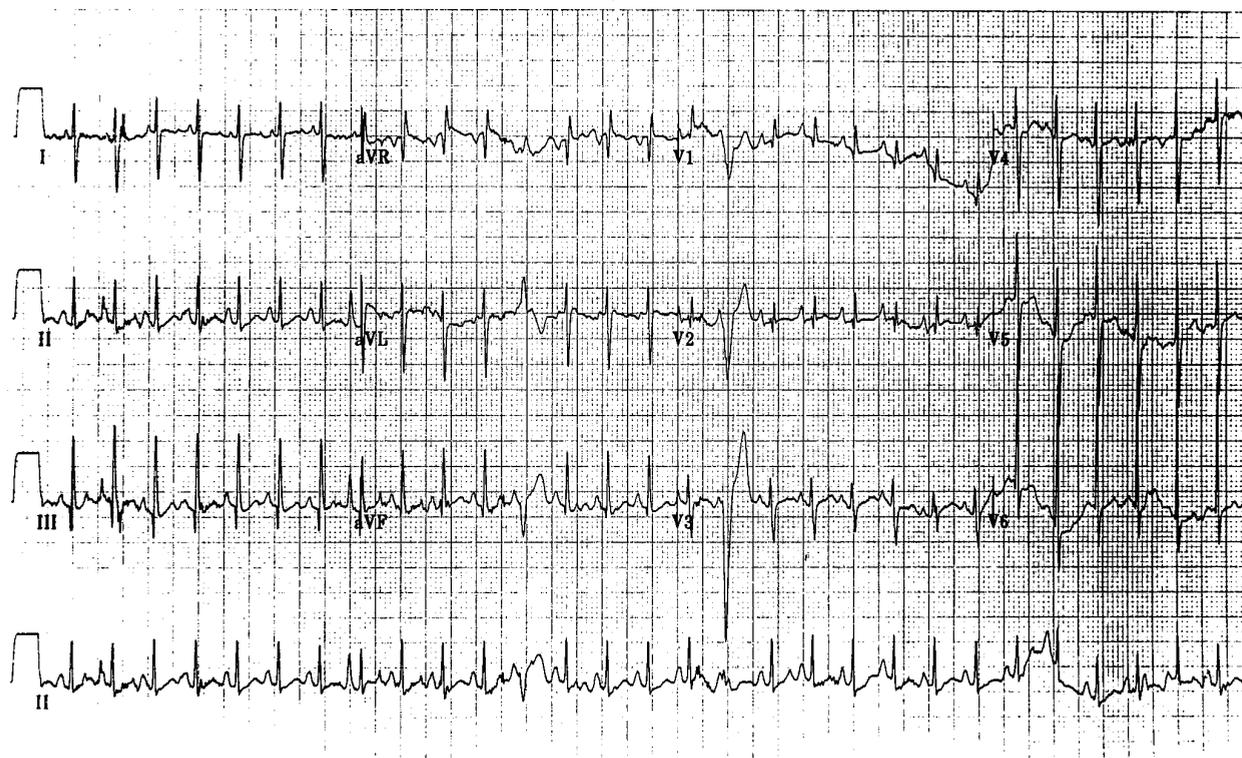


Fig 2. Twelve-lead ECG 1 hour after DC cardioversion demonstrates sinus tachycardia with right atrial enlargement. Note that the previously seen deep flutter waves in V2 are clearly not present on this sinus rhythm ECG, and therefore were not part of the T wave during atrial flutter in Fig 1.

tion, or negatively influences the interaction between adenosine and its receptor.

Finally, the significant sinus tachycardia seen well after cardioversion was also unusual for patients with isolated atrial flutter, but entirely consistent with a diagnosis of hyperthyroidism, and this provided a clue that the arrhythmia was not the primary problem. This was confirmed after the laboratory studies demonstrated a thyrotoxic profile. Although one might ascribe such tachycardia to the possible presence of tachycardia-induced cardiomyopathy, in this patient, the shortening fraction was in fact in the normal range immediately after cardioversion, and so a severe cardiomyopathy was unlikely.¹⁹ The question of whether this patient had an inherent predilection to develop atrial flutter which was brought out by hyperthyroidism, or whether the patient developed atrial flutter secondary to electrophysiologic changes precipitated by hyperthyroidism has not been resolved. If atrial flutter were to recur, an electrophysiologic study may be warranted.

In summary, when atrial flutter is encountered in a pediatric patient in whom there is 1:1 atrioventricular conduction, a lack of a response to adenosine, and persistent sinus tachycardia after cardioversion, the clinician should be alert to the possibility of thyrotoxicosis.

WILLIAM A. SUAREZ, MD
 GEORGE F. VAN HARE, MD
 ISAIAH D. WEXLER, MD
 Divisions of Cardiology and Endocrinology
 Department of Pediatrics

Case Western Reserve University School of
 Medicine
 Cleveland, OH 44106

JAMES E. ARNOLD, MD
 Division of Pediatric Otolaryngology
 Department of Otolaryngology-Head and
 Neck Surgery
 Case Western Reserve University School of
 Medicine
 Cleveland, OH 44106

REFERENCES

1. Talley JD, Wathen MS, Hurst JW. Hyperthyroid-induced atrial flutter-fibrillation with profound sinoatrial nodal pauses due to small amounts of digoxin, verapamil, and propranolol. *Clin Cardiol.* 1989; 12:45-47
2. Creigler LL, Allen CB, Mark H. Relation of atrial flutter to Graves' disease. *Mt Sinai J Med.* 1986;53:548-549
3. Pilapil V, Watson DG. Electrocardiogram in hyperthyroid children. *Am J Dis Child.* 1970;119:245-248
4. Goel BG, Hanson CS, Han J. A-V conduction in hyper- and hypothyroid dogs. *Am Heart J.* 1972;83:504-511
5. Garson A, Bink-Boelkens M, Hesslein PS, et al. Atrial flutter in the young: A collaborative study of 380 Cases. *J Am Coll Cardiol.* 1985;6: 871-878
6. Nordyke RA, Gilbert Jr. FI, Harada ASM. Graves disease. *Arch Intern Med.* 1988;148:626-631
7. Barnes HV, Blizzard RM. Antithyroid drug therapy for toxic diffuse goiter (Graves disease): thirty years experience in children and adolescents. *Pediatrics.* 1977;91:313-320
8. Kogut MD, Kaplan SA, Collipp PJ, Tiamsic T, Boyle D. Treatment of hyperthyroidism in children: analysis of 45 patients. *N Engl J Med.* 1965;272:217-221
9. Vaidya VA, Bongiovani AM, Parks JS, et al. Twenty-two years experience in the medical management of juvenile thyrotoxicosis. *Pediatrics.* 1974;54:565-570
10. Saxena KM, Crawford JD, Talbot NB. Childhood thyrotoxicosis: a long-

- term perspective. *Br Med J*. 1964;7:1153–1158
11. Talley JD, Wathen MS, Hurst JW. Hyperthyroid-induced atrial fibrillation with profound sinoatrial nodal pauses due to small doses of digoxin, verapamil, and propranolol. *Clin Cardiol*. 1989;12:45–47
 12. Deutsch P, Kronzon I, Weiss EC. Unusually rapid atrial rate in a patient with thyrotoxicosis and atrial flutter. *Chest*. 1975;67:350–351
 13. Waldo AL. What's new in atrial flutter. In: Fish C, Surawicz B, eds. *Advances in Cardiac Electrophysiology and Arrhythmias*. New York, NY: Elsevier Science Publishing Co, Inc; 1991:176–185
 14. Landsberg L. Catecholamines and hyperthyroidism. *Clin Endocrinol Metab*. 1977;6:697–713
 15. Coluombe P, Dussault JH, Walker P. Plasma catecholamines in hyperthyroidism and hypothyroidism. *Metabolism*. 1976;25:973–978
 16. Bayliss RI, Edwards OM. Urinary excretion of free catecholamines in Graves' disease. *Endocrinology*. 1971;49:167–173
 17. Beaven MA, Costa E, Brodie BB. The turnover of norepinephrine in thyrotoxic and nonthyrotoxic mice. *Life Sci*. 1963;4:241–246
 18. Levey GG. Catecholamine-thyroid hormone interactions and the cardiovascular manifestations of hyperthyroidism. *Am J Med*. 1990;88:642–646
 19. Fenelon G, Wijns W, Andries E, Brugada P. Tachycardiomyopathy. Mechanisms and clinical implications. *PACE*. 1996;19:95106

Atrial Flutter: An Uncommon Pediatric Manifestation of Hyperthyroidism

William A. Suarez, George F. Van Hare, Isaiah D. Wexler and James E. Arnold

Pediatrics 1997;100:e11

DOI: 10.1542/peds.100.2.e11

Updated Information & Services

including high resolution figures, can be found at:
<http://pediatrics.aappublications.org/content/100/2/e11>

References

This article cites 18 articles, 1 of which you can access for free at:
<http://pediatrics.aappublications.org/content/100/2/e11#BIBL>

Subspecialty Collections

This article, along with others on similar topics, appears in the following collection(s):

Surgery

http://www.aappublications.org/cgi/collection/surgery_sub

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:

<http://www.aappublications.org/site/misc/Permissions.xhtml>

Reprints

Information about ordering reprints can be found online:

<http://www.aappublications.org/site/misc/reprints.xhtml>

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Atrial Flutter: An Uncommon Pediatric Manifestation of Hyperthyroidism

William A. Suarez, George F. Van Hare, Isaiah D. Wexler and James E. Arnold

Pediatrics 1997;100:e11

DOI: 10.1542/peds.100.2.e11

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://pediatrics.aappublications.org/content/100/2/e11>

Pediatrics is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. Pediatrics is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 1997 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 1073-0397.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

