Persistent Supraventricular Tachycardia

Case Report with Review of Literature

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A summary of all reported cases of persistent supraventricular tachycardia, as contrasted with paroxysmal and repetitive tachycardia, is presented, along with an additional well-documented instance covering an 11-year period. The distinctive features of persistent tachycardia are included.

Only 13 cases of persistent supraventricular tachycardia have been reported in the available medical literature, and few of these have had prolonged follow-up study.1-9 Unlike paroxysmal tachycardia, persistent supraventricular tachycardia occurs rarely and is characteristically refractory to treatment. We wish to present an additional and well-documented case report of persistent auricular tachycardia of 11 years' duration in a 15 year old boy which has remained refractory to treatment, and to focus attention on the pertinent features of this type of obstinate arrhythmia. A summary of all the reported cases of established persistent supraventricular tachycardia is included (table 1).* Only those instances of continuous tachycardia of at least two months duration have been considered.

Case Report

A 14 year old white boy was admitted to Montefiore Hospital on March 24, 1952 (case no. 57407). Details of the mother’s pregnancy and delivery were in no way unusual. The child was not cyanotic or dyspneic at birth and no murmurs or arrhythmia were noted. Development was normal. At the age of 6 months a “somewhat harsh systolic murmur” over the entire heart was noted by the family physician, who made no mention then of an unusual cardiac rate. At 14 months, the boy was treated for bronchitis. There is no history of diphtheria, rheumatic fever, or scarlet fever. No other childhood illnesses are recorded.

The patient was admitted to another hospital on Nov. 2, 1942, with a history of sudden paralysis of the right side of his body; loss of speech and incontinence. Examination at that time revealed a well-nourished 4 year old boy with hemiplegia and aphasia. The heart rate was rapid. No murmurs were audible. The remainder of the examination was negative. Laboratory data were normal. No evidence of cardiac enlargement was demonstrated on roentgenographic examination. The initial electrocardiogram revealed a supraventricular tachycardia with 1:1 conduction at a rate of 270 per minute (fig. 1, 11-20-42). On the same day the rhythm reverted to a regular sinus tachycardia (fig. 1). This proved to be a transient change, for during the remainder of the hospitalization, an auricular tachycardia was present with a varying auricular rate up to 290 per minute, and a ventricular response ranging from 45 per minute to 290 per minute (fig. 1). Various maneuvers for breaking the arrhythmia, including carotid sinus and eyeball pressure, and therapeutic trials of quinidine and digitalis failed to alter the basic rhythm, though digitalis caused changes in the degree of block. It will be noted, however, that the single tracing obtained on Nov. 20, 1942 (fig. 1), revealed an apparent transient sinus tachycardia, which did not recur. The ventricular rate was reasonably well controlled, and no evidence of congestive heart failure appeared. Speech gradually returned and the patient recovered almost complete use of his right leg but only partial motion of his right arm.

Following discharge on March 3, 1943, the patient was referred to a convalescent home. On June 1, 1943, he developed signs of congestive heart failure with distended neck veins and an enlarged liver, requiring rapid digitalization. Between 1943 and 1950, periodic examinations at a cardiac clinic revealed persistent auricular tachycardia with a varying ventricular rate, confirmed by serial electrocardiograms.

The patient first came to our attention on June 9, 1951, and has been followed periodically since then. Examination at that time revealed a rapid heart rate. He was admitted to Montefiore Hospital for further study on March 24, 1952. The patient stated he was conscious of changes in his heart rate, particularly when it became very rapid, but was otherwise asymptomatic. At these times he would

* At the request of the Editor, the table summarizing these cases is being omitted, but will be furnished on request.

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Fig. 1. Electrocardiogram of 11-20-42 reveals a 1:1 tachycardia. Tracing just below of the same date demonstrates the probable sinus tachycardia. (See text.) Tracing of 1-14-43 reveals auricular tachycardia with a varying degree of block. Tracing taken on 2-9-43 shows a tachycardia with a 2:1 block.

Fig. 2. Electrocardiogram of April 1962 reveals the persistent auricular tachycardia, with a 2:1 block clearly demonstrated by esophageal lead taken at the auricular level.
Fig. 3. Electrocardiogram of 10-23-53 shows auricular tachycardia with runs of 1:1 conduction, and electrical alternans best seen in lead II.

decrease his activity until the rate slowed. Physical examination revealed a well-developed, well-nourished, moderately obese white boy with a spastic paralysis of the right arm, weakness of the right leg, and abnormal reflexes compatible with a previous cerebrovascular accident. No murmurs were audible, and the heart was not enlarged either on fluoroscopy or roentgenography. Laboratory examinations, including venous pressure and circulation times, were within normal limits. Radioactive iodine uptake and urinary 17-ketosteroids were normal. Electrocardiograms revealed auricular tachycardia with a 2:1 block confirmed by esophageal leads (fig. 2). Quinidine, Digoxin and Prostigmine in the usual therapeutic doses did not alter the basic rhythm. The auricular rate varied between 200 and 300 beats per minute. The ventricular rate was usually 110 to 120 beats per minute at rest and increased to 150 beats per minute on exercise, but variation occurred as shown in figures 1 and 2. On one occasion the ventricular rate was observed to change from 240 to 120 per minute within a matter of seconds.

In May 1952, following discharge from the hospital, the digitalis dosage was reduced and then discontinued. In June 1952, his ventricular rate rose to between 200 and 240 beats per minute, and the boy complained of palpitations and slight dyspnea on exertion. Redigitalization slowed his ventricular rate and he became asymptomatic. In June 1953, an electrocardiogram revealed the persistent auricular tachycardia with runs of 2:1 and occasionally 3:1 block. On Oct. 23, 1953, he again complained of palpitation and some dyspnea. An electrocardiogram revealed auricular tachycardia with runs of 1:1 conduction at a rate of 200 per minute, with electrical alternans (fig. 3). Without any treatment except sedation, and with no change in digitalis medication, the block increased to 2:1 and then 3:1, and the patient became again relatively asymptomatic. He is being maintained on digitalis.

Discussion

The diagnosis of auricular tachycardia was established in this child at the age of 4 following a cerebrovascular accident. Whether this rhythm has been present since birth cannot be determined. Periodic electrocardiograms taken over the past 11 years reveal the persistently rapid auricular rate with varying degrees of heart block (fig. 1). The possible single exception was the tracing dated Nov. 20, 1942 (fig. 1), which reveals a reversion to an apparent sinus tachycardia. However, this tracing may also be interpreted as showing a slow auricular tachycardia, inasmuch as the P wave and the P-R interval are the same as those present in the other electrocardiograms. This episode was of short duration and has never recurred on any of the numerous subsequent tracings. The only evidence of the possible presence of a sinus rhythm was provided on that occasion.

The sudden onset of the hemiplegia in the absence of any other illness, and the normal development since then suggests that the arrhythmia with probable embolization was responsible for the cerebrovascular accident. The absence of cardiac enlargement or murmurs appears to rule out the usual etiologic forms of heart disease. There is no history of myocarditis, nor evidence of a demonstrable
congenital defect. The auricular rate varies from time to time, but always remains between 200 and 300 per minute. The etiology of this is entirely unknown.

At the age of 4, signs of congestive heart failure developed, which responded well to intravenous digitalis. It is likely that a persistently high ventricular rate was related to this episode. The patient remained asymptomatic for many years thereafter, except for the awareness of changes in his heart rate.

In reviewing the literature for other established instances of persistent supraventricular tachycardia, of two months duration or more, only 13 other cases were found. We were careful to distinguish these cases from those with repetitive tachycardia. In this latter group evidence of normal sinus rhythm could be demonstrated repeatedly during the course of the tachycardia, whereas in the persistent type evidence of normal sinus rhythm could not be demonstrated for prolonged periods despite frequently repeated tracings.

Palpitation or awareness of the heartbeat was the most common symptom in the group, and was usually accompanied by slight shortness of breath, especially on exertion. However, five of the patients were asymptomatic, the arrhythmia having been discovered during examination for other reasons. The tachycardia first occurred before the age of 20 in 11 of the reported cases. There is no evidence that the arrhythmia was present at birth in any of the patients. One patient is said to have had tachycardia at 6 months of age, while another patient was 34 years of age when his tachycardia first began. The oldest patient of the group was first seen at the age of 59 because of a sacroiliac strain, but was otherwise asymptomatic although he was told of a tachycardia 43 years before.

Cardiac enlargement was noted in only three instances, and a murmur was described in only one case. No evidence of possible etiologic factors, such as myocarditis or thyroid disease, was found in any of these patients.

Congestive heart failure occurred during the course of the arrhythmia in three of the patients, including our own, and was readily controlled by the usual measures in each instance. In the case cited by Claiborne, which most closely resembles our own, congestive heart failure occurred at the age of 4 and the patient required digitalis thereafter. The other patient in whom congestive heart failure appeared was a 45 year old white man with a tachycardia which lasted 498 days.

The auricular rate in our patient has always remained between 200 and 300 per minute. In the eight other instances where the auricular rate was specifically recorded, it usually varied between 120 and 200 per minute. A varying degree of A-V block was not unusual, with a ventricular rate usually between 120 and 180 per minute.

This group of patients was characteristically refractory to treatment. Sedation, vagal stimulation, quinidine, Pronestyl, digitalis and other measures failed to alter the basic rhythm. Digitalis was the most effective drug used, for it slowed the ventricular rate in the majority of instances in which it was employed, though usually it had no effect on the auricular mechanism. In three cases digitalis temporarily abolished the tachycardia. In one of these patients, a 6 year old child, digitalis was ineffective in altering the rhythm when the child was 16 months of age but converted the tachycardia to regular sinus rhythm when the child was 6 years of age.

**Summary**

A 15 year old boy with a persistent and refractory supraventricular tachycardia, well-documented over an 11-year period, is presented. The complications of hemiplegia and heart failure have appeared despite the absence clinically detectable organic heart disease. A review of all other cases with persistent auricular tachycardia of at least two months duration is included. Attention is drawn to some of the distinctive features of persistent tachycardia, in contrast with paroxysmal and repetitive tachycardia.

**Sumario Español**

Se informa el caso de un niño de 15 años con una taquicardia supraventricular refractaria y persistente, bien documentada por un periodo de 11 años. Las complicaciones de hemiplegia
y descompensación cardíaca han aparecido no obstante la ausencia clínica de enfermedad orgánica cardíaca. Un repaso de todos los otros casos con taquicardia auricular persistente de por lo menos dos meses de duración se incluye. Se llama la atención ha algunas de las características distintivas de la taquicardia persistente, en contraste con la taquicardia paroxística y repetitiva.

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