Determinants of Atrioventricular Reentrant Paroxysmal Tachycardia in Patients with Wolff-Parkinson-White Syndrome

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SUMMARY Normal and anomalous pathway properties were evaluated in 50 patients with preexcitation to discover determinants of paroxysmal supraventricular tachycardia (PSVT). Twenty-eight patients had inducible PSVT and 22 had no inducible PSVT. Patients with inducible PSVT had: 1) ability for retrograde anomalous pathway conduction demonstrated with ventricular pacing at short paced cycle lengths (< 429 msec) and close coupling intervals (< 400 msec); and 2) antegrade AV nodal refractoriness less than anomalous pathway refractoriness during rapid and/or coupled atrial pacing with ability for exclusive normal pathway conduction for at least one beat at short atrial paced cycle lengths (< 375 msec) or close coupling intervals (< 320 msec).

Failure to induce PSVT was accounted for by one of the following: 1) absent or poor retrograde anomalous pathway conduction alone (six patients); 2) inadequate antegrade AV nodal properties alone (eight patients); 3) both inadequate antegrade AV nodal and retrograde anomalous pathway properties (seven patients); and 4) prolonged atrial refractoriness (one patient). There were no significant differences in anomalous pathway antegrade refractory periods between the patients with and without PSVT.

We conclude that the occurrence of PSVT in Wolff-Parkinson-White syndrome depends on the ability for retrograde anomalous pathway conduction and adequate antegrade AV nodal conduction. The inability to induce PSVT usually reflects inadequate retrograde anomalous and/or antegrade normal pathway properties. The ability to induce PSVT is independent of antegrade anomalous pathway properties.

PATIENTS WITH WOLFF-PARKINSON-WHITE syndrome may have recurrent paroxysmal supraventricular tachycardia (PSVT).1 If PSVT occurs in these patients, it is usually characterized by narrow QRS complexes, and reflects a circus movement with antegrade conduction via normal atrioventricular (AV) pathway and retrograde conduction via an anomalous extranodal pathway.2 Induction of this tachycardia can be achieved with atrial impulses which block in the anomalous pathway conducting antegrade to the normal pathway, or by ventricular impulses which block in the normal pathway and conduct retrogradely via the anomalous pathway.3,4 Why some patients with Wolff-Parkinson-White syndrome have PSVT and others do not is not clear.

In this study, we examined antegrade and retrograde conduction properties of normal and anomalous pathways to study the determinants of AV reentrant PSVT in patients with the Wolff-Parkinson-White syndrome. We compared preexcitation patients with and without inducible AV reentrant PSVT.

Methods

Patient Selection

All patients with typical preexcitation undergoing electrophysiological study in our laboratory between January 1974 and August 1977 were reviewed. The only requirement for inclusion in this study was a complete electrophysiological evaluation, including assessment of antegrade conduction with incremental atrial pacing and atrial extrastimulus technique and assessment of retrograde conduction with incremental pacing and ventricular extrastimulus technique.

Fifty patients met the above criteria and are the subjects of this report. Ages ranged from 13–64 years (38 ± 16; mean ± SD). Eighteen patients had left-sided, 22 had right-sided and 10 had septal preexcitation. In 11 of the patients preexcitation was intermittent. Thirty-two patients had no clinically demonstrable organic heart disease, six had arteriosclerotic heart disease, three hypertensive heart disease, five valvular heart disease, one congenital heart disease and three cardiomypathy.

Electrophysiological Studies

Patients gave informed consent before electrophysiological study. All cardioactive drugs were discontinued at least 48 hours before the study. His bundle electrograms were recorded using a tripolar electrode catheter introduced percutaneously into the right femoral vein and placed at the tricuspid valve.5 A hexapolar catheter, introduced via the right antecubital vein, was advanced to the right ventricular apex. The two distal electrodes were used for ven-
tricular pacing. The proximal four electrodes were positioned against the right lateral atrial wall and were used for high and mid-right atrial recording and stimulation. A third quadripolar catheter, introduced into the left antecubital vein, was used for recording and stimulation from the coronary sinus. Electrocardiographic leads I, II, III and V₁, as well as atrial and His bundle electrograms, were simultaneously recorded on a multichannel oscilloscopic recorder (Electronics for Medicine DR-16, White Plains, New York) at paper speeds of 100 msec. Stimuli, provided by a programmable digital pulse generator (manufactured by M. Bloom, Philadelphia, Pa.), were 2 msec in duration and approximately twice diastolic threshold. Localization of anomalous pathways was performed as previously described, utilizing atrial pacing at multiple sites and by mapping during retrograde conduction.⁵ ⁶

Anomalous pathway antegrade properties were studies using responses to incremental atrial pacing and atrial extrastimulus technique. Incremental atrial pacing was performed at 10 beats/min increments until one or more of the following was achieved: 1) lack of atrial capture; 2) antegrade block in the anomalous pathway; 3) antegrade block in the normal pathway; or 4) achievement of a minimum paced cycle length of 240–333 msec (290 ± 27 msec; mean ± sd). Antegrade anomalous pathway refractory periods and echo zones were determined with atrial extrastimulus technique at two or more cycle lengths (one sinus or close to sinus), from an atrial site close to the anomalous pathway.⁵ ⁶ ⁸ ⁹ S₁, S₂, S₃, was utilized in some patients, but did not provide additional data to that obtained with incremental atrial pacing in regard to PSVT induction.

Anomalous pathway retrograde properties were studied using responses to incremental ventricular pacing and ventricular extrastimulus technique. The ventricles were paced at rates slightly above sinus and then at 10 beats/min increments until one of the following was achieved: 1) lack of ventricular capture; 2) ventriculo-atrial block; 3) retrograde block in the normal pathway (shown by induction of AV reentrant paroxysmal supraventricular tachycardia), or 4) achievement of a minimum paced cycle length of 250 to 429 msec; (321 ± 54 msec). Anomalous pathway refractory periods and echo zones were determined with ventricular extrastimulus technique during sinus rhythm or at a ventricular driven cycle length slightly shorter than sinus cycle length.⁴ Shorter ventricular paced cycle lengths were often utilized, but did not provide additional data to that obtained with incremental ventricular pacing in regard to PSVT induction.

Definitions

In this study, PSVT was defined as AV reentrant tachycardia using the normal AV pathway for antegrade conduction and the anomalous pathway for retrograde conduction.⁸ Other varieties of paroxysmal tachycardia (including antidromic) induced in these patients were not considered in this analysis. Retrograde anomalous pathway conduction during PSVT was proven by demonstration of one or more of the following criteria: 1) abnormal atrial retrograde activation sequence;⁷ ⁸ ¹⁰ 2) ability to capture the atria during PSVT with critically timed ventricular extrastimuli delivered when the His bundle was refractory;¹¹ 3) increase in ventriculoatrial (VA) conduction time during PSVT with development of bundle branch block ipsilateral to the anomalous pathway.¹² ¹³

The presence of retrograde anomalous pathway conduction during incremental ventricular pacing was suggested by demonstration of one or more of the following: 1) abnormal retrograde atrial activation sequence (23 patients);⁷ ⁸ ²) fixed VA conduction time during incremental ventricular pacing (37 patients);¹⁰ 3) PSVT induction with incremental ventricular pacing, not related to critical VA interval, suggesting induction via block in the normal pathway (15 patients);⁴ 4) retrograde activation sequence during incremental pacing identical to that observed during induced PSVT or AV reentrant atrial echoes (34 patients).

The anomalous pathway antegrade effective refractory period was defined as the longest atrial coupling interval (A₁A₂) at which block was achieved in the anomalous pathway.⁹ The anomalous pathway retrograde effective refractory period was defined as the longest ventricular coupling interval (V₁V₂) at which block occurred in the anomalous pathway.⁴

Results

Patients with Inducible PSVT

There were 28 patients in whom PSVT could be induced during electrophysiological studies. In these patients, PSVT could be induced by the following techniques: 1) sudden cessation of rapid atrial pacing (17 patients); 2) atrial extrastimulus technique (24 patients); 3) sudden cessation of rapid ventricular pacing (15 patients); and 4) ventricular extrastimulus technique (six patients). In all patients with inducible PSVT, the tachycardia could be initiated with incremental and/or coupled atrial stimulation. Induction of PSVT from the atria was always with impulses conducted via the normal pathway and blocked in the anomalous pathway. In those patients with induction with ventricular stimulation, PSVT always related to block in the normal pathway (a concealed event) and retrograde conduction via anomalous pathway.

Retrograde Anomalous Pathway Conduction Properties

All patients with inducible PSVT had intact anomalous pathway retrograde conduction (fig. 1). In 27 of the 28 patients, retrograde block in the anomalous pathway could not be achieved during incremental ventricular pacing. The shortest achieved ventricular paced cycle length in these 27 patients
Antegrade Normal Pathway Properties

In all patients with inducible PSVT there was an achievable paced atrial cycle length (17 patients) or coupling interval (24 patients) during atrial extrastimulus testing at which there was block in the anomalous pathway (see below) and antegrade conduction via the normal pathway (fig. 2). The shortest atrial paced cycle length with demonstrable exclusive normal pathway conduction ranged from 375–240 msec (302 ± 35 msec). The shortest coupling interval (atrial extrastimulus testing) with demonstrable exclusive normal pathway conduction ranged from 320–210 msec (258 ± 33 msec).

Since all 28 patients in this group had inducible PSVT, there was capability for sequential normal pathway conduction at cycle lengths at least equal to that of induced PSVT. Cycle lengths of induced PSVT ranged from 430–270 msec (329 ± 48 msec) (fig. 2). In most patients atrial paced cycle lengths shorter than PSVT cycle length were not tested.

Antegrade Anomalous Pathway Properties

Six of the 28 patients with inducible PSVT had intermittent preexcitation during sinus rhythm. In 15 of the remaining 22 patients, block in the anomalous pathway was achieved with atrial pacing at cycle lengths ranging from 240–462 msec (335 ± 66 msec). In seven patients, anomalous pathway conduction was intact at the shortest atrial paced cycle length tested, which ranged from 240–316 msec (287 ± 29 msec).

Anomalous pathway effective refractory periods could not be measured in the six patients who had intermittent preexcitation, but was considered approximately equal to sinus cycle length, which ranged from 450–1020 msec (745 ± 225 msec). Anomalous pathway antegrade effective refractory periods could also not be measured in three patients in whom anomalous pathway refractory periods were shorter than atrial functional refractory periods. In these patients, PSVT was induced after cessation of rapid atrial pacing. Anomalous pathway effective refractory periods in these three patients ranged from < 250 msec to < 340 msec (< 290 ± 46 msec). The anomalous pathway effective refractory period could be measured in the remaining 19 patients and ranged from 225–390 msec (296 ± 33 msec) (fig. 3, left).
FIGURE 2. Induction of paroxysmal supraventricular tachycardia (PSVT) with coupled atrial pacing in a patient with left-sided preexcitation. A distal coronary sinus electrogram is shown in Panel C. A1 represents the atrial electrogram of basic drive and A2 the premature atrial extrastimulus. A represents the atrial electrograms and H the His bundle electrograms. HRA = high right atrial electrogram; HBE = His bundle electrogram; CL = cycle length; DCS = distal coronary sinus electrogram.

Panel A. Atrial pacing from the coronary sinus at a CL of 600 msec. An atrial extrastimulus is introduced at an A1-A2 interval of 300 msec. Both A1 and A2 are conducted via the anomalous pathway.

Panel B. The A1-A2 interval is decreased to 290 msec and A2 is blocked in the anomalous pathway conducting via the normal pathway with induction of PSVT.

Panel C. AV reentrant PSVT utilizing normal pathway for antegrade and anomalous pathway for retrograde conduction. Note the abnormal sequence of retrograde atrial depolarization with coronary sinus atrial electrogram preceding both high right atrial and low right septal electrograms.
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Anomalous Pathway Antegrade Refractory Periods

Patients with PSVT (28 pts)  
Patients without PSVT (22 pts)

Patients Without Inducible PSVT

PSVT could not be induced in 22 patients. In all of these patients, the inability to induce PSVT appeared to reflect inadequate conduction in one or more components of the potential circus movement, and could be related to describable electrophysiological properties. Inadequate retrograde anomalous pathway conduction was noted in 13 patients, inadequate antegrade normal pathway conduction in 15 patients and inadequate atrial conduction in one patient (fig. 4).

Retrograde Anomalous Pathway Properties

In 13 of the 22 patients, PSVT was not inducible because of inadequate anomalous pathway retrograde conduction. These 13 included seven patients with inadequate antegrade AV nodal conduction.

In seven of the 13 patients, there was total inability for VA conduction at all paced ventricular cycle lengths (absence of both retrograde normal and anomalous pathway conduction). In six patients, intact ventriculo-atrial conduction via normal pathway was present during ventricular pacing. In these patients the presence of normal pathway retrograde conduction was suggested by demonstration of at least two or more of the following: 1) normal retrograde activation sequence; 2) increasing VA intervals and development of retrograde Wenckebach periodicity with incremental ventricular pacing (fig. 5A); 3) occurrence of ventricular echoes with total preexcitation (retrograde normal pathway and antegrade anomalous pathway conduction) during ventricular stimulation (fig. 5B); and 4) the presence of retrograde H potentials with constant or increasing H-A intervals during coupled ventricular stimulation.

In nine of the patients without inducible PSVT, the inability to induce PSVT did not relate to inadequate

Patients without PSVT (22 patients)

Inadequate antegrade AV nodal property
Inadequate atrial property
Inadequate retrograde anomalous pathway property
Inadequate antegrade AV nodal and retrograde anomalous pathway property

Figure 4. Electrophysiologic properties of the atria, normal and anomalous pathway in 22 patients without inducible paroxysmal supraventricular tachycardia (PSVT).
retrograde anomalous pathway properties. In eight of these nine patients, intact retrograde anomalous pathway conduction was present at the shortest tested ventricular paced cycle length, which ranged from 284–375 msec (327 ± 36 msec). In one of the nine patients, retrograde anomalous pathway block occurred at a ventricular paced cycle length of 400 msec. Anomalous pathway retrograde refractory periods could not be measured in eight of the nine patients because of limiting ventricular refractoriness. Retrograde anomalous pathway effective refractory period in these eight patients ranged from < 230 to < 340 msec (277 ± 39 msec). In one patient the anomalous pathway retrograde refractory period was 250 msec.

Antegrade Normal Pathway Properties

In 15 of the 22 patients, normal pathway conduction appeared inadequate for induction of PSVT. These 15 included seven patients with inadequate retrograde anomalous pathway conduction. In five of these 15 patients, AV block was present at both the longest atrial paced cycle length and at the longest A1A2 coupling interval at which anomalous pathway block was encountered (total absence of AV conduction) (fig. 6). The longest paced atrial cycle length with failure of anomalous pathway conduction and absent normal pathway conduction in these five patients ranged from 400–273 msec (327 ± 55 msec) (figs. 6A and B). The longest coupling interval in these
five patients with failure of anomalous pathway conduction and absence of normal pathway conduction ranged from 290–320 msec (303 ± 15 msec) (figs. 6C and D). In these five patients, AV nodal conduction was presumably less adequate than anomalous pathway conduction.

In four patients, measurable AV nodal properties appeared inadequate to sustain PSVT. In these four patients, the longest paced cycle length which produced block in the antegrade normal pathway ranged from 460–600 msec (515 ± 59 msec). The AV nodal effective refractory period in these four patients ranged from 380–530 msec (478 ± 70 msec).

In six of the patients with noninducible PSVT and inadequate normal pathway conduction, the normal pathway manifested inability for sequential antegrade conduction. In these six patients, normal pathway conduction was adequate for only a single atrial echo. The second atrial echo failed to conduct to the His bundle (fig. 7).

In the remaining seven patients without inducible PSVT, AV nodal properties appeared adequate for PSVT induction (lack of PSVT induction reflecting inadequate conduction in the retrograde anomalous pathway in six and atrium in one patient). In all seven patients, there was an achievable paced atrial cycle length (five patients) or coupling interval during atrial extrastimulus testing (six patients) at which there was block in the anomalous pathway and antegrade conduction via the normal pathway. The shortest paced cycle length with exclusive antegrade normal pathway conduction ranged from 284–400 msec (341 ± 43 msec). The shortest coupling interval (atrial extrastimulus testing) with exclusive normal pathway conduction ranged from 250–450 msec (318 ± 70 msec).

Atrial Properties

In one of the 22 patients, the atrial refractory period was 450 msec (all other patients having < 350 msec).
This patient had intact retrograde anomalous pathway conduction and a normal pathway refractory period less than anomalous pathway refractory period. Prolonged atrial refractoriness above appeared to account for the absence of PSVT in this patient.

Antegrade Anomalous Pathway Properties

Five of the 22 patients without inducible PSVT had intermittent preexcitation during sinus rhythm. In 12 of the remaining 17 patients, antegrade block in the anomalous pathway occurred at an atrial paced cycle length of 273–426 msec (364 ± 72 msec). In five patients, anomalous pathway conduction was intact at the shortest atrial paced cycle length tested, which ranged from 261–333 msec (296 ± 26 msec).

Anomalous pathway antegrade refractory period could not be measured in the five patients who had intermittent preexcitation, but was considered approximately equal to sinus cycle length which ranged from 700–1000 msec (879 ± 126 msec). Anomalous pathway antegrade refractory period could not be measured in two patients, in whom anomalous pathway refractory periods were shorter than atrial functional refractory periods. In these patients, the anomalous pathway effective refractory periods were, respectively, less than 225 and 305 msec. The anomalous pathway effective refractory period could be measured in the remaining 15 patients, and ranged from 270–530 msec (323 ± 69 msec) (fig. 3, right).

Comparison of Antegrade Anomalous Pathway Properties in Patients With and Without PSVT

There were no significant differences in anomalous pathway properties between patients with and without inducible PSVT (fig. 3). Six of the 28 patients (21%) with inducible PSVT had intermittent preexcitation, while five of 22 (23%) patients without PSVT had intermittent preexcitation (NS). The mean atrial paced cycle length producing antegrade block in the anomalous pathway was 335 ± 17 msec (mean ± SEM) in patients with inducible PSVT and 364 ± 21 msec in patients without inducible PSVT (NS). The mean anomalous pathway antegrade effective refractory period was 296 ± 8 msec in patients with inducible PSVT and 323 ± 18 msec in patients without inducible PSVT (NS) (fig. 3).

Relationship of Tachycardia Induction to Clinically Observed PSVT

Of the 28 patients with PSVT inducible in the catheterization laboratory nineteen (68%) had spontaneous electrocardiographically documented PSVT. Eight patients (28%) had only paroxysmal palpitations and one (4%) was asymptomatic.

Of the 22 patients without inducible PSVT in the catheterization laboratory, none had electrocardiographically documented PSVT. Eleven patients (50%) had paroxysmal palpitations and 11 (50%) were asymptomatic. Of the 13 patients with inadequate retrograde anomalous pathway conduction, nine (69%) were asymptomatic, while of the eight patients with only inadequate antegrade normal pathway conduction, six (75%) had paroxysmal palpitations.

Discussion

Electrophysiological and pathological studies suggest that most patients with Wolff-Parkinson-White syndrome have an extranodal anomalous pathway (Kent bundle). In these patients, the most common variety of paroxysmal tachycardia is AV reentrant, utilizing the Kent bundle for retrograde conduction and normal AV pathway for antegrade conduction. In this circus movement tachycardia, the atria and ventricles are final common pathways.
ability of patients with preexcitation to sustain this paroxysmal tachycardia is in the electrophysiological properties of the components of the circus movement. On theoretical grounds, one would hypothesize that sustained AV reentrant tachycardia could occur in those patients with preexcitation who had capability for repetitive antegrade normal pathway conduction (the most critical component being the AV node) and capability for repetitive retrograde anomalous pathway conduction.

Wellens and coworkers reported electrophysiological studies in 107 patients with Wolff-Parkinson-White syndrome.\(^4\) In 57 of these patients, circus movement PSVT was inducible using atrial premature stimulation (77%) or ventricular premature stimulation (49%). Wellens also noted that 98 of his patients had intact, and nine had absent, ventriculo-atrial conduction. He did not report results of incremental atrial and ventricular pacing. He also did not compare electrophysiological properties in those patients with and without inducible PSVT.

Sung et al. studied 29 patients with left-sided pre-excitation using programmed ventricular stimulation.\(^4\) He related PSVT induction to the pattern of retrograde conduction. Specifically, PSVT could not be induced in the absence of both retrograde normal and anomalous pathway conduction, or in the presence of exclusive normal or simultaneous normal and anomalous pathway conduction. PSVT could be induced when there was retrograde block in the normal pathway with conduction via the anomalous pathway. Sung examined only induction of PSVT from the ventricles, and did not report results of antegrade normal and anomalous pathway properties, or results of atrial induction of PSVT.

In the present study we compared patients with and without PSVT inducible by any technique. We were able to compare antegrade and retrograde electrophysiological properties of normal and anomalous pathway in those patients with and without inducible PSVT.

All of the patients with inducible PSVT had intact retrograde anomalous pathway conduction. In these patients, retrograde anomalous pathway conduction appeared adequate for maintenance of PSVT, shown by the ability to retrogradely conduct at relatively short paced ventricular lengths (≤ 429 msec) and short coupling intervals (≤ 400 msec).

All patients with inducible PSVT also had ability for repetitive antegrade normal pathway conduction. This facet of conduction was more difficult to demonstrate, since intact anomalous pathway conduction interfered with measurement of AV nodal properties. However, the adequacy of antegrade normal pathway conduction was demonstrated in all patients by demonstration of exclusive normal pathway conduction at either short atrial paced cycle lengths (≤ 375 msec) and/or at short coupling intervals during extrastimulus testing (≤ 320 msec). The presence of PSVT in these patients, was another demonstration of the ability for repetitive normal pathway conduc-

tion at short cycle lengths. Although paced cycle lengths shorter than that of PSVT cycle length were usually not tested in these patients, we presume that AV nodal conduction often would have been intact at atrial paced cycle lengths somewhat shorter than the cycle length of the induced PSVT.

Having accomplished description of the electrophysiological properties in patients with inducible PSVT, the patients without inducible PSVT could be scrutinized. Again, one could theoretically predict that inability to induce sustained PSVT would be related to inadequate conduction in one or more portions of the circus movement. In patients without inducible PSVT, inadequate retrograde anomalous pathway conduction was frequent. This was demonstrated by total absence of ventriculo-atrial conduction with ventricular pacing (retrograde block in both normal and anomalous pathways), or failure of retrograde anomalous pathway conduction at relatively long paced ventricular cycle lengths.

In patients without inducible PSVT, AV nodal conduction was also frequently inadequate for sustaining circus movement tachycardia. This was less readily demonstrated, since the existence of intact anomalous pathway conduction made quantitation of antegrade AV nodal properties difficult. However, in a number of patients AV nodal properties appeared inadequate for PSVT induction as shown by one of the following: 1) AV nodal refractoriness greater than anomalous pathway refractoriness at all tested coupling intervals with extrastimulus technique and at all atrial paced cycle lengths. In these patients, there was no demonstrable antegrade AV nodal conduction when anomalous pathway block was achieved. In this group, AV nodal conduction, although not quantifiable, was certainly less adequate than anomalous pathway conduction. In this group of patients, retrograde anomalous pathway conduction was often excellent, so the failure to induce PSVT with ventricular stimulation was another measure of inadequate AV nodal conduction; 2) development of AV nodal Wenckebach periodicity in patients with long anomalous pathway refractory periods, at relatively long atrial paced cycle lengths. Antegrade AV nodal properties in this group of patients were clearly less adequate than in those patients with inducible PSVT (see results); 3) the inability for antegrade conduction of more than one single echo. This phenomenon was demonstrated with atrial extrastimulus testing (or incremental atrial pacing). In these patients, exclusive antegrade normal pathway conduction could be achieved, with retrograde conduction back to the atria for a single atrial echo. The single atrial echo was unable to conduct antegrady to the His bundle, suggesting inability for sequential antegrade AV nodal conduction at short coupling intervals. In addition, patients in this group demonstrated a similar phenomenon with ventricular extrastimuli. In these patients, ventricular extrastimuli could conduct retrogradely via anomalous pathway to the atria, with return to the ventricles via normal pathway, and sub-
sequent return to the atria via the anomalous pathway. The latter atrial impulse was then blocked proximal to the His bundle, suggesting inability for antegrade conduction of two sequential beats via the AV node.

In patients with and without inducible PSVT, atrial properties were almost universally suitable for sustaining of induced PSVT. Prolonged atrial refractoriness prevented induction of PSVT in only one patient. The ventricles did not appear to be a limiting factor for PSVT induction in any of the patients.

The question might arise as to whether our noninducible PSVT group might have included patients capable of having PSVT on the day of electrophysiological study. If this were the case, then the failure to induce PSVT could be related to technical limitations such as inadequate pacing sites or modes of stimulation, rather than an inherent limitation somewhere in the potential circus movement. This seems unlikely for several reasons: 1) All patients were studied with multiple modes of stimulation, including incremental pacing, atrial extrastimulus technique, ventricular incremental pacing and ventricular extra-stimulus technique. Data concerning shorter driven cycle lengths with extrastimulus testing or S1S2S3 stimulation did not offer additional instances of PSVT induction. Our use of multiple attempts at rapid incremental pacing obviated the necessity for S1S2S3 stimulation. Rapid incremental pacing allows peel back of atrial refractoriness (allowing closer coupling intervals to be applied to the anomalous pathway), potentiates anomalous pathway block due to closely coupled stimulation, and potentiates the achievement of critical AV nodal delays for induction of the echo phenomenon. Rapid ventricular incremental pacing similarly obviates the need for ventricular extra-stimulus technique at short cycle lengths by potentiating retrograde block in normal pathway. 2) In almost all patients without inducible PSVT, there were sites of depressed conduction, which explained inability to induce sustained PSVT.

In only two patients (the two patients in whom AV nodal refractoriness was longer than anomalous pathway refractoriness) does the possibility of technical failure to induce PSVT seem possible. These two patients had excellent antegrade and retrograde anomalous pathway conduction. If one postulated that the failure to induce PSVT reflected inability to initiate antegrade normal pathway conduction because of excellent anomalous pathway properties, then these patients should have had PSVT easily inducible from below. For example, ventricular extrastimuli in these patients should conceal in the node (at close coupling intervals), conduct retrogradely via anomalous pathway, and thus induce PSVT, if antegrade AV nodal properties were suitable. As discussed above, AV nodal properties as judged from antegrade stimulation seemed inadequate. One extremely unlikely combination of properties could explain our inability to induce PSVT in these patients. If one postulated that both antegrade anomalous and normal pathway properties were excellent (with the first better than the second), and that both retrograde anomalous and normal pathway properties were excellent (with the second better than the first), then it is possible that no delivered stimuli from any site could induce PSVT, although there was adequate antegrade AV nodal and adequate retrograde anomalous pathway properties for sustaining PSVT.

The lack of difference in antegrade anomalous pathway properties in those patients with and without inducible PSVT surprised us. There was a wide range of antegrade anomalous pathway properties in both groups, ranging from excellent antegrade conduction to intermittent preexcitation during sinus rhythm. There were no statistically significant differences in measurable antegrade anomalous pathway properties in the two groups of patients with noninducible and inducible PSVT. Thus, antegrade anomalous pathway properties did not appear to determine whether PSVT could be induced in a given patient on a given day.

We have demonstrated that the clinical occurrence of spontaneous PSVT is related to the ability to induce PSVT in the catheterization laboratory. Specifically, the majority of patients with inducible PSVT in the catheterization laboratory had paroxysmal palpitations (96%) with documented PSVT in 68%. In those patients without inducible PSVT 50% were asymptomatic and none had documented PSVT, although 50% had paroxysmal palpitation. It appears that the frequency of PSVT might be related to both frequency of inducing premature atrial and ventricular contractions and the comparative refractoriness of normal and anomalous pathways. However, if a patient did not have adequate retrograde anomalous pathway conduction and adequate antegrade normal pathway conduction, PSVT could not occur. We believe that effective prophylactic drug therapy should affect the properties of either antegrade normal pathway, or retrograde anomalous pathway, making them similar to those seen in the group of patients without inducible PSVT.

Even patients who do not have capability for PSVT, either because of poor antegrade AV nodal or poor retrograde anomalous pathway conduction, are still subject to the hazards of rapid ventricular responses during atrial fibrillation when the antegrade refractory period of the anomalous pathway is short.

References
5. Scherlag BJ, Lau SN, Helfant RH, Stein E, Berkowitz WD,
Effects of Self-Induced Starvation on Cardiac Size and Function in Anorexia Nervosa

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SUMMARY Cardiac size, function and rhythm were examined in 11 patients with anorexia nervosa. Mean left ventricular, left atrial and aortic dimensions on echocardiogram were below normal adult values at baseline. In addition to decreased cardiac dimensions — ventricular ectopy, relative hypotension, bradycardia and blunted heart rate — response to exercise were noted. Left ventricular systolic function, however, was unimpaired as indicated by normal echocardiographic fractional shortening, and by normal exercise augmentation of ejection fraction determined by radionuclide cineangiography. Eight of the patients responded to treatment with a mean weight gain of 32%. In these eight, cardiac dimensions increased toward normal: left ventricular dimension increased by 13%; left atrial dimension by 20%; aortic dimension by 15% and estimated left ventricular mass by 20%. We conclude that abnormalities of heart size and rhythm occur in patients with anorexia nervosa. However, cardiac dimensions, including left ventricular mass, may increase following nutritional rehabilitation, accompanied by an increase in heart rate and blood pressure.

DEATH IN ANOREXIA NERVOSA may be sudden, and the mortality rate is the highest of any psychiatric disease.1 Self-enforced starvation in this condition results in severe reduction of body mass, comparable to that of famine victims.2 Despite intensive investigation of its psychiatric3 and endocrine4,5 manifestations, little has been written about the cardiovascular system in this disease, which offers a clinical model for the study of protein-calorie deprivation.

The recent development of noninvasive diagnostic techniques such as echocardiography, radionuclide cineangiography and Holter ambulatory electrocardiographic monitoring, permit more accurate estimation of cardiac size, and cardiac mechanical and electrical function, than previously possible. We have used these techniques to study patients with anorexia nervosa before and after weight gain. The data from these patients may be pertinent to the potential reversibility of the effects of starvation on cardiac size and function in the victims of famine and other causes of nutritional deprivation.

Methods

Patient Population

Eleven patients admitted to the Clinical Center of the National Institutes of Health with the diagnosis of anorexia nervosa as defined by the criteria of Feighner et al.6 were studied. Informed consent was obtained from each patient before the study. Each was admitted for evaluation and treatment of anorexia nervosa; none had cardiovascular symptoms or previously identified cardiovascular abnormalities. All had normal serum electrolytes. All were white females varying in
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