

The Role of Transesophageal Atrial Stimulation in the Evaluation of Supraventricular Tachycardias

J. RES

Hospital DE HEEL, Department of Cardiology, Zaandam, The Netherlands

R. VAN WOERSEM

Boven IJ Hospital, Amsterdam, The Netherlands

Summary

In order to treat patients that exhibit supraventricular tachycardias, the tachycardia mechanism and the conduction characteristics of a possible accessory pathway must be established. For this purpose, transesophageal atrial stimulation was performed on 69 consecutive and symptomatic patients, with or without pre-excitation, in order to induce regular supraventricular tachycardia and atrial fibrillation. The results of transesophageal atrial stimulation were also compared with noninvasive tests including standard ECG's during symptoms, exercise tests, and Holter monitoring. Seventy supraventricular tachycardias were induced in 49 patients; arrhythmias could not be induced in 9 patients. Reentrant tachycardias were characterized by using several criteria such as the esophageal ventriculo-atrial interval and AV dissociation. All supraventricular tachycardias could be characterized. The shortest R-R interval conducted with pre-excitation could be determined in 23 patients, and a short effective refractory period (≤ 270 ms) was found in 16 patients. In 33 out of 58 (57 %) patients, ECG's with spontaneous supraventricular tachycardia were recorded with a single type of arrhythmia: in 30 patients on standard ECG. In 11 patients, supraventricular tachycardia was recorded during Holter monitoring, but it provided additional data in only 3 patients. In only 1 patient, a spontaneous arrhythmia was recorded, but no supraventricular tachycardia could be induced with transesophageal atrial stimulation. The regular supraventricular tachycardia could not be characterized reliably on the ECG in 14 out of 24 supraventricular tachycardia due to an indiscernible P wave (11 cases) or incorrect diagnosis (3 cases). In conclusion, transesophageal atrial stimulation can help discover and correctly diagnose arrhythmias. In addition, the conduction characteristics of the accessory pathway can be determined. The results of transesophageal atrial stimulation are favorable compared to the results of noninvasive testing. Therefore, transesophageal atrial stimulation as a simple, direct, and useful tool can be applied in patients with pre-excitation and/or in patients exhibiting a supraventricular tachycardia with an unknown mechanism, and in whom radiofrequency ablation is not directly indicated.

Key Words

Supraventricular tachycardias, transesophageal atrial stimulation, reentrant tachycardias, accessory pathways

Introduction

Electrophysiologic studies on patients with supraventricular tachycardias (SVT) are necessary for two major reasons. First, the mechanism of the SVT has to be understood, in order to optimize the treatment plan. For example, patients with infrequent AV nodal (junctional) reentrant tachycardias should be treated either directly with mechanisms to terminate the SVT or with intermittent antiarrhythmic drug therapy. Furthermore,

the antiarrhythmic drug treatment should be closely monitored since digitalis and verapamil can be dangerous in a patient who has pre-excitation or paroxysmal atrial fibrillation with pre-excitation [1-5]. Patients who have accessory pathways (AP) resulting in an SVT and who are resistant to pharmacotherapy may be proper candidates for radiofrequency catheter ablation of the AP [6,7]. Patients with AV nodal reentrant

tachycardias can be treated successfully with class Ic drugs [8,9], calcium blocking agents [10,11], or sotalol [12]. Second, in the presence of an AP, the conduction characteristics of the AP must be determined in order to prevent life threatening arrhythmias from occurring. In addition, the antegrade-effective refractory period of the AP and the shortest RR-interval of the pre-excited beats have to be calculated.

Different approaches have been proposed for those patients with documented regular SVT's in the presence or absence of pre-excitation (Wolff-Parkinson-White syndrome). On the one hand, noninvasive tests can be performed using a standard 12-channel ECG and a series of exercise tests to assess the presence of pre-excitation and to provoke arrhythmias [13,14]. Ambulatory 24-hour Holter monitoring can be performed to document the SVT and their correlation with complaints [15,16]. The duration of the effective refractory period can be assessed by administering exercise tests and using the Holter monitor [17,18]. However, these methods are not reliable, because they underestimate the risk of rapid conduction of AP and the possibility of sudden death in some patients [19-21]. Examination of the ECG during the regular SVT is very useful in diagnosing the mechanism of SVT, if the P wave is discernible [22-24]. However, recognition of the P wave on the surface ECG may be difficult to find. Atrial flutter and particularly atrial fibrillation can be diagnosed almost immediately.

Invasive electrophysiologic studies have been proposed for the symptomatic patient or the patient with pre-excitation. An invasive, multi-catheter electrophysiologic study with programmed electrical stimulation of the heart is very effective for the induction and the characterization of SVT [25]. In addition to disclosing the mechanism of SVT, the localization of the AP can be determined and subsequent special electrophysiological interventions can be exercised [7,25].

An alternative, quick, and noninvasive approach is transesophageal atrial stimulation (TRAS) [26,27]. Various characteristics are used to characterize the different types of SVT. The recording of the esophageal ventriculoatrial interval (VA_{eso}) during a reentrant tachycardia and possible AV dissociation are essential for the characterization of the tachycardias, such as AV nodal (junctional) reentrant tachycardias, reentrant tachycardias using an AP, and atrial tachycardias [26,28].

Rationale

In this study, we evaluated the clinical utility of TRAS in those patients who exhibited regular supraventricular tachycardias or who indicated pre-excitation on the standard ECG. The TRAS evaluation had to establish the mechanism of the SVT and the conduction characteristics of the AP and / or normal AV connection.

Materials and Methods

Patients

The study group consisted of 69 patients: 24 women and 45 men, mean age 39.2 ± 14.9 years (range 16 to 72), who were being evaluated because of documented SVT, or for symptoms in the presence of overt pre-excitation. Symptoms included syncope, dizziness, palpitations (either regular or irregular), or a combination of these. The patients were also questioned regarding their use of antiarrhythmic medication, as well as the rate and duration of their palpitations.

Some patient characteristics and the type of prior arrhythmias are given in Table 1. The surface ECG showed overt pre-excitation in 37 (53 %) patients. Syncope was the most significant symptom in 9 (13 %) patients. Major symptoms were absent in 4 patients whose characteristics of pre-excitation had to be explored. An abnormal ECG was found in 17 of the 69 (25 %) patients. The most frequent abnormality was mitral valve prolapse (8 patients). Sixty-four percent of the patients were on antiarrhythmic drug therapy prior to the TRAS evaluation.

Noninvasive Evaluation

When an SVT was recorded during an earlier admission to a hospital or an emergency room, the ECG was reviewed and the type of SVT was classified. All tachycardias were classified by an investigator who did not know the results of the TRAS. In the case of a discernible P wave, the mechanism was determined; otherwise, the tachycardia mechanism was classified as unknown. Usually, patients with a long history of symptoms were asked when and where an ECG had been recorded during a symptomatic tachycardia. Recordings of documented tachycardias were collected in 33 out of 58 (57 %) patients. The standard noninvasive evaluation included a symptom-limited exercise test in all patients and one or more Holter monitors. An ECG was recorded for every patient. Holter monitors

were repeated in 61 patients who had no arrhythmias during the first recording. Whenever appropriate, a more extensive diagnostic evaluation was performed to determine the underlying structural heart disease.

Transesophageal Study

Antiarrhythmic medication was discontinued for at least 5 half-times before testing, and beta-blockers for at least one week. The possibility of discomfort during insertion of the catheter and the esophageal stimulation was explained to each patient. Informed consent was obtained for all patients.

The patients were studied in the post-absorptive state and did not receive sedation or analgesic drugs. While the patient was sitting, the esophageal electrode was inserted and stimulation was conducted. Some patients preferred to be in a semi-supine position on a hospital bed during the test. If the hexapolar balloon or the quadripolar flexible lead were used, the nose and throat were lightly anesthetized with 1 % lidocaine spray or 2 % lidocaine jelly. Local anesthesia was not used with the pill electrode. A description of the electrodes can be found in literature [26-31]. The pill electrode, the hexapolar balloon electrode, and the quadripolar flexible lead were applied in 11, 26, and 48 patients, respectively. They were successfully applied in 5, 15, and 38 patients, respectively. Positioning of the electrode was guided by the atrial electrogram. The electrode with the largest and steepest unipolar atrial deflection was used as the cathode and the electrode with the second best atrial deflection was used as the anode. In general, surface electrograms with leads I, II, III, V1, and V6, and at least one transesophageal atrial electrogram were recorded simultaneously (100 mm/sec) on the 6-channel ink jet recorder (Siemens Elema Mingograf 62) or on a multichannel digital electrophysiology recording unit (Bard Lab System 24). The esophageal atrial electrogram was digitally filtered to diminish the stimulus artifacts [32]. Pacing was performed with a dedicated TRAS device (model 2380, High Output Stimulator, Medtronic, USA), which provided stimulation with a minimum cycle length (CL) of 100 ms, at a pulse width of 20 ms, and with a maximum amplitude of 40 mA. This device can be used as a booster in connection with a standard programmable electrophysiological stimulator (UHS 20, Biotronik, Germany). To ensure consistent atrial capture, stimulation was started with a current that was 20 % above threshold. The output was increased if capture was lost and decreased if serious

	Mean \pm sd	Range	n
Age	39.2 \pm 14.9	16 - 72 years	69
Female	39.2 \pm 15.0	16 - 69 years	24 (35 %)
Male	37.9 \pm 14.7	16 - 72 years	45 (65 %)*
* p = NS (female vs male)			
Symptoms	Mean \pm sd	Range	
Duration	8.1 \pm 8.8	0.5 - 32 years	
Attack	87 \pm 112	1 - 600 min	
Frequency of attacks	23 \pm 39	1 - 150 x/year	
Type of symptoms	Patients	Percent	
Syncopes	9	13	
Dizziness	17	25	
Palpitations	56	81	
Absence of symptoms	4	6	
Combinations of symptoms	17	25	
Echocardiographic findings (n = 69)	Patients	Percent	
Normal echocardiogram	52	74	
Mitral valve prolapse	8	12	
Aortic valve pathology	3	4	
Mitral valve pathology	2	3	
Tricuspid insufficiency	2	3	
Concentric LVH	1	2	
Hypertrophic cardiomyopathy	1	2	
Patients on prior anti-arrhythmic therapy (AA-drug)	1	\geq 2 drugs	
Patients (n = 44)	21	23	
Number of medications (n = 75)	n	% per patient	
Verapamil	23	33	
Flecainide	13	19	
Sotalol	9	13	
Amiodarone	7	10	
Beta-blockade	6	9	
Digoxin	5	7	
Propafenon	4	6	
Disopyramide	4	6	
Kinidine	3	4	
Ajmaline	1	1	

Table 1. Characteristics of patients with regular supraventricular tachycardias, or Wolff-Parkinson-White Syndrome, evaluated with TRAS.

chest discomfort occurred. This usually resulted in a current that was 30 % above the stimulation threshold. Standard stimulation protocols were employed [33]. Incremental pacing was performed to the CL at which either second degree AV block (Wenckebach)

occurred, or 1:1 antegrade conduction over the accessory pathway ceased, or a minimum CL of 250 ms (240 bpm) was reached. Due to impeded sensing of the atrium, extra stimuli were applied during sinus rhythm in a small number of patients. In all patients, the antegrade atrioventricular refractory period was measured at a basic CL of 600 ms (100 bpm) using the extra stimuli. If a tachycardia could not be induced, a second extra stimulus was given. This was followed by short bursts (3 - 10 sec) of rapid atrial pacing at CL starting at 300 ms, until a minimum of 100 ms (200 till 600 bpm) was achieved in order to induce atrial fibrillation. In the early phase of the study, the aggressive induction of atrial fibrillation was not used.

Medication was not given during or after the stimulation procedure except in one patient who received intravenous flecainide because of persistent atrial fibrillation after induction. All regular tachycardias were terminated by TRAS using two to eight stimuli at 90 % to 75 % of the CL of the tachycardia.

When a tachycardia was induced, the CL of the tachycardia was recorded and the VA_{eso} interval was measured. The VA_{eso} interval was defined as the interval from the onset of ventricular activation to the steep atrial deflection on the esophageal atrial electrogram [26]. When a bundle branch block was temporarily present during the tachycardia, the CL and the VA_{eso} were measured, before and after the transition of a wide QRS tachycardia into a narrow QRS tachycardia. The tachycardias were characterized as AV nodal (junctional) reentrant tachycardia (RT), orthodromic AV reentrant tachycardia using an accessory pathway (orthodromic RT), antidromic RT, atrial tachycardia, atrial flutter or atrial fibrillation.

Criteria for Diagnosis

The following criteria were used for AV nodal RT:

- the tachycardia was induced by a single atrial extra stimulus resulting in a prolonged AV interval;
- demonstration of a discontinuous AV conduction curve (AV jump), i.e., a critical prolongation of the AV interval (≥ 50 ms) with small shortening (10 ms) of the coupling interval of the atrial extra stimulus; and
- VA_{eso} less than 70 ms [26].

The criteria for orthodromic RT were as follows:

- Wolff-Parkinson-White syndrome was present on the surface ECG or could be provoked with atrial extra stimuli;

- orthodromic RT was induced when the atrial extra stimulus was antegradely blocked in the accessory pathway (AP) and conducted in the AV node, with or without bundle branch block;
- transition of a wide QRS tachycardia from a narrow QRS occurred during the tachycardia;
- VA_{eso} was greater than 70 ms; and
- a concealed AP was diagnosed when all criteria were fulfilled (except for the presence of pre-excitation on the surface ECG).

Atrial tachycardia was distinguished from atrial flutter by the absence of P waves on the surface ECG and a CL less than 250 ms. (rate < 240 bpm). Absence of correlation of the AA interval with the AV interval during induction and termination was also required. Both tachycardias should also have shown AV dissociation during the spontaneous tachycardia after vagal maneuvers.

When atrial fibrillation was induced, the shortest RR interval with pre-excitation (SRR - PE) was noted. When the induction of atrial fibrillation failed or was too short (< 30 sec) for the evaluation of the conduction over the AV node and the AP, the SRR-PE was taken during very rapid atrial pacing (CL ≤ 150 ms = ≥ 400 bpm).

Statistical Methods

The student's t test was used for comparing tachycardia features, as well as for conduction and refractoriness characteristics. The Fischer exact test was used for statistical evaluation of categorical data.

Results

TRAS - Failure Rate and Side Effects

The TRAS procedure failed in a total of 11 (15 %) patients. In 3 patients, the introduction of 1 or more of the 3 electrodes failed, and in 6 patients, the stimulation caused pain before constant atrial capture could be achieved. In 2 additional patients, hyperventilation and hyperactive peristalsis precluded the completion of the stimulation protocol. The side effects and the occurrence of pain are listed in Table 2. A flexible lead was used in the majority of patients because of the low failure rate with respect to its introduction (6 %) and the relatively low failure rate of atrial stimulation (15 %). In 1 patient, the stimulation caused severe pain that could barely be tolerated. In 7 patients, pain was moderate and tolerable. In 29 patients, pain was complete-

ly absent. Occasionally, temporary loss of capture was noticed which necessitated repeating the stimulation sequence. In these patients, there was no need to increase the stimulation amplitude. Temporary loss of capture was observed in 8 patients without pain, 10 with mild pain, and 5 with moderate pain.

In 3 patients, atrial fibrillation was inadvertently induced due to oversensing of a false signal in the entrance circuitry of the high output stimulator; this functioned as a booster in connection with the programmed stimulator. In all 3 patients, the atrial fibrillation stopped spontaneously after a few minutes. The burst stimulation intended to induce atrial fibrillation at the end of the stimulation protocol could then be omitted in these patients. The mean stimulation threshold in all patients was 10.4 ± 3.6 mA (range 7 - 25), and the mean ratio between the stimulation threshold and the maximum output was 1.29 ± 0.26 (range 1.0 - 2.8).

Electrophysiologic Measurements

The electrophysiologic data are given in Table 3. The effective refractory period of the AV node (ERPAV) was determined in 57 (98 %) patients who were available for TRAS. The ERPAV was 264 ± 54 ms (range 160 - 410). In 37 patients, pre-excitation was clearly visible on the surface ECG, and in 1 patient with a left lateral accessory pathway the pre-excitation was concealed during sinus rhythm. However, in only 28 out of the suitable 38 (74 %) patients with pre-excitation, the effective refractory period of the AP (ERPAP) was measured. The ERPAP was 300 ± 105 ms (range 210 - 750). A short ERPAP, defined by ≤ 250 ms, was found in 8 patients with a very short ERPAP of 210 ms in 1 patient.

Rapid atrial stimulation was employed in 46 patients, and in 25 (54 %) patients (54 %), atrial fibrillation was successfully induced (see Figure 1). The shortest RR interval with pre-excitation (SRR-PE) during atrial fibrillation or during rapid atrial stimulation was 296 ± 55 ms (range 190 - 430) in 23 patients. In 5 patients the SRR-PE was available, both during atrial fibrillation and during rapid atrial pacing. In 3 of these patients the SRR-PE at rapid atrial pacing was 20 ms shorter than the SRR-PE during atrial fibrillation. In 4 patients a very short SRR-PE (≤ 250 ms) was found. There were no signs of conduction over a second accessory pathway. A reentrant tachycardia (orthodromic RT, AV nodal RT or antidromic RT) was induced in 31 patients.

	Incomplete procedures (n = 11)	Complete procedures (n = 58)	All procedures (n = 69)
Vomiting at Introduction	3	-	3
Painful stimulation	6	-	6
Hyperventilation	1	-	1
Hyperactive peristalsis	1	-	1
Runaway of stimulator	-	3	3
Stimulation n. phrenicus	-	1	1
Atrial arrhythmias	-	3	3
All	11	7	18
Painful stimulation			
Severe pain	6	1	7
Moderate pain	-	7	7
Mild pain	-	21	21
No pain	-	29	29
Intermittent loss of capture	-	23	23
Electrodes used	17	68	85
Number per patient	1.5	1.2	1.2
One electrode	7	49	56
Two electrodes	2	8	10
Three electrodes	2	1	3
	Mean \pm sd	Range	
Stimulation threshold	10.4 \pm 3.6 mA	7.0 - 25.0	
Maximum output	12.8 \pm 4.1 mA	10 - 25.0	
Ratio of maximum output vs. threshold	1.29 \pm 0.26	1.0 - 2.8	

Table 2. List of side effects and characteristics of TRAS in 69 patients.

An example of induction is given in Figure 2. In 10 (32 %) cases, the tachycardia stopped spontaneously,

	Mean ± sd	Range	n	Percent	Suitable for evaluation
ERP-AV node	264 ± 54	160 - 410	57	98	58
ERP-AP	300 ± 105	210 - 750	28	74	38
SRR-PE (AFib or pacing)	296 ± 55	190 - 430	23	61	38
CL of AV RT	344 ± 40	290 - 440	29	94	31
VA _{eso} of RT	113 ± 54	10 - 240	29	100	29
CL of atrial RT	262 ± 89	195 - 500	9	64	14

AFib = atrial fibrillation
 Pacing = rapid atrial stimulation
 AP = accessory pathway
 AV node = atrioventricular node
 CL = cycle length (in msec)
 ERP = effective refractory period
 RT = reentrant tachycardia
 SRR-PE = shortest RR-interval conducted with pre-excited beats
 VA_{eso} = ventriculo-atrial interval on esophageal recording

Table 3. Determination of effective refractory period of atrioventricular (AV) nodal conduction and accessory pathway (AP), and cycle length (CL) of induced reentrant tachycardia.

and in the other 21 (68 %) cases, the tachycardia was actively terminated by overdrive pacing. Transition of bundle branch block into narrow QRS tachycardia was observed in 10 patients; 4 times with left bundle branch block, 4 times with right bundle branch block, and 2 times with both forms of block during a series of induced tachycardias. In 2 patients,

the VA_{eso} showed a distinct difference between the wide and the narrow QRS tachycardias, which were 50 ms and 20 ms, respectively. In the first case, the AP was localized at the left lateral site and concerned a left bundle branch block. In the second case, the accessory pathway was localized at the posterolateral site and a left bundle branch block was present during the wide QRS tachycardia.

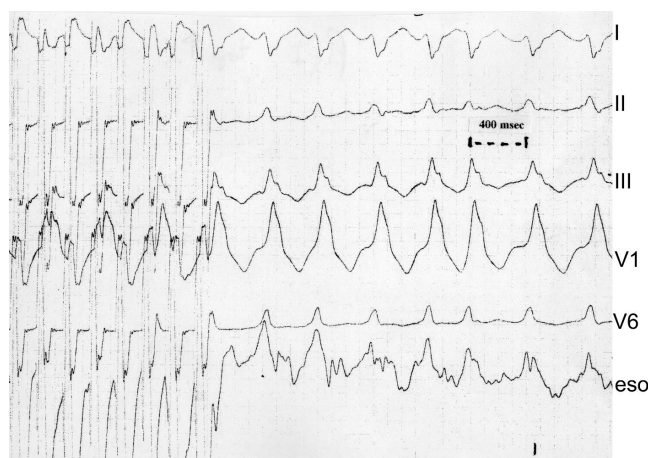


Figure 1. Induction of atrial fibrillation after burst stimulation. An irregular rhythm is shown on the lower channel representing the esophageal electrode, which has been used for recording of the atrial activity only. The surface ECG is represented by the leads I, II, III, V1 and V6. The AV conduction is purely over the accessory pathway resulting in wide QRS complexes, and the shortest RR interval between pre-excited beats is 280 ms. (paper speed is 50 mm/sec).

Types of Tachycardias Diagnosed

The different types of induced tachycardias are listed in Table 4. Characterization of the tachycardia was applied in all induced tachycardias using the VA_{eso}-intervals as the most important criteria. In Figure 2, the induction of an orthodromic reentrant tachycardia is shown in a patient with concealed accessory pathway (absence of pre-excitation during atrial pacing). Special attention has been paid to the AV relationship before attempting overdrive stimulation. In 1 particular patient, an SVT with a very short VA_{eso}-interval indicated an AV nodal reentrant tachycardia; the 1:1 AV relationship was interrupted after rapid overdrive stimulation suggested an atrial tachycardia.

Some characteristics of the SVT, such as the mode of induction, are given in Table 5. In 2 patients, more supraventricular tachycardias (apart from atrial fibrillation) were induced. In 1 patient, both an AV nodal RT and an orthodromic RT were induced, and both tachycardias were distinguished from one another by the

Type of SVT	TRAS		Surface ECG			
	Induced	Correct	Recorded diagnosis	Correct diagnosis	Incorrect diagnosis	Unknown diagnosis
Orthodromic RT	19	19	14	7	1	6
AV nodal RT	11	11	8	2	1	5
Antidromic RT	1	1	0	0	0	0
Atrial tachycardia	7	7	2	1	1	0
Atrial flutter	7	7	2	2	0	0
Atrial fibrillation	25	25	11	11	0	0
Induced tachyarrhythmias	70	70	37	23	3	11

Table 4. Type of SVT characterized by TRAS versus spontaneous SVT.

VA_{eso}-interval. In another patient, several types of tachycardias were induced: AV nodal RT, orthodromic RT, antidromic RT, and atrial tachycardias. All these tachycardias were distinguished from one another by applying the above-mentioned criteria. During the antidromic tachycardia, the wide QRS showed an activation pattern similar to the pre-excitation pattern.

Atrial fibrillation was induced in 25 patients, which proved to be useful for observing possible concealed pre-excitation in patients with unknown pre-excitation. One patient showed pre-excitation which was not observed during sinus rhythm. In the patient with clearly visible pre-excitation on the surface ECG, the SRR-PE was measured.

Diagnosis of SVT on the Surface ECG

Table 4 gives insight into the efficiency of the conventional surface ECG recording in documenting a tachycardia. Recording a spontaneous tachycardia during an episode of symptoms was a relatively efficacious procedure. It provided informative recordings in 30 out of 58 (52 %) patients. Prolonged Holter monitoring and exercise tests produced tachycardia ECG-recordings in only 3 additional patients, bringing the total to 33 (57 %). The recordings confirmed 37 arrhythmias, 24 regular tachycardias, and 13 irregular tachycardias (atrial fibrillation and atrial flutter with irregular AV conduction). In 4 patients, both types of arrhythmias were found. In only 1 patient, a regular SVT was diagnosed on the ECG during a symptomatic period, while with TRAS no SVT were induced.

An SVT was correctly diagnosed in 23 out of 37

(62 %) recorded tachycardias. However, only 37 spontaneous tachycardias out of a possible 70 SVT's could be recorded. The diagnosis of the SVT was incorrect in 3 cases. The diagnosis could not be made in 11 patients because the P wave was masked by the QRS complex or by ST-segment abnormalities (see Figure 3). The

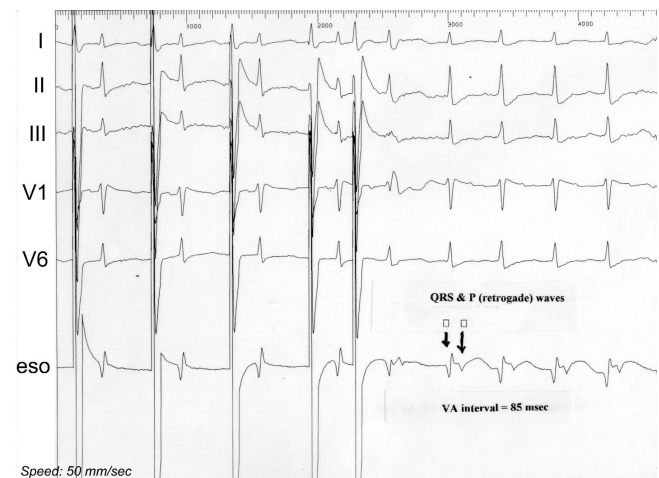


Figure 2. The ECG shows the surface ECG with the leads I, II, III, V1, V6 and the esophageal atrial recording. After 4 beats of the basic drive (s) at a CL of 600 ms an extra stimulus (s1) was given, which was conducted through the AV node, and subsequently a reentrant tachycardia was induced. The characterization of the reentrant tachycardia was based on a concealed pathway. This was due to the absence of pre-excitation during the basic drive and after extrasystoles, the regularity of the tachycardia, the constant 1:1 A-V relation, and a VA_{eso} ≥ 70 ms. (The ECG was stored on a digital recorder and printed at a paper speed of 50 mm/sec).

	n	Percent
Type of AV RT	31 / 58	53
Orthodromic reentrant tachycardia	19 / 58	33
AV nodal reentrant tachycardia	11 / 58	19
Antidromic reentrant tachycardia	1 / 58	2
Mode of induction of AV RT		
Induction by 1 or 2 extrasystole	31 / 31	100 #
Dual AV nodal pathways (AV jump)	11 / 13	80 **
Induction of AV nodal RT	9 / 11	81
No induction of AV nodal RT	2 / 11	19
Mode of termination		
Spontaneous termination of RT	10 / 31	32
Overdrive stimulation of RT with 2-8 ES	21 / 31	68
Tachycardia related characteristics of RT		
Transition wide into narrow QRS RT	10 / 31	32
Left BBB	4 / 10	40
Right BBB	4 / 10	40
Left & Right BBB	2 / 10	20
Alternans of QRS during AV RT	1 / 31	3
1:1 AV relation		31 / 31 100
All atrial reentrant tachycardias	14 / 58	24
Presence of AV dissociation	14 / 14	100
CL < 250 msec (= atrial tachycardia)	7 / 58	12
CL > 250 msec (= atrial flutter)	7 / 58	12
Rapid atrial stimulation	40 / 58	69
Induction of atrial fibrillation	25 / 40	63

in the case of WPW-syndrome the tachycardia was induced when the pre-excitation disappeared at a shorter extrastimulus interval.

** the "jump in AV-interval" was probably masked by the pre-excitation in 2 patients, but an AV nodal RT with a short VA_{min} could also be induced.

BBB = bundle branch block

ES = extrasystole

Table 5. Type of TRAS induced SVT's and electrophysiologic characteristics of the induced tachycardias.

ECG's with atrial fibrillation and flutter were all correctly diagnosed, in contrast to the regular (reentrant) tachycardias. In 10 out of 39 (26 %) regular tachycardias, a correct diagnosis was made on the surface ECG (Table 4).

Discussion

TRAS is successful in the induction, termination, and classification of different types of arrhythmias in selected groups of patients. The reentrant tachycardia can be induced and terminated by atrial stimulation, and the P wave can be recorded during the tachycardia. Furthermore, the main electrophysiologic parameters of the Wolff-Parkinson-White syndrome can be mea-

sured during atrial stimulation, both during the reentrant tachycardia and during atrial fibrillation.

In patients with documented reentrant tachycardias, a 100 % induction rate has been achieved with TRAS [34-36]. However, in groups of patients with a positive history of palpitations, the success rate was found to vary between 23 and 100 % [37-40]. Most of these studies were based on small groups of patients (n ≤ 25). In addition, these studies showed differences in stimulation protocols, age of the patients, and study objectives. Some studies were performed in combination with exercise or the intravenous administration of isoproterenol [38]; other studies used only straight pacing with decremental intervals to induce the reentrant tachycardias [28,37,39]. In general, reports indicate

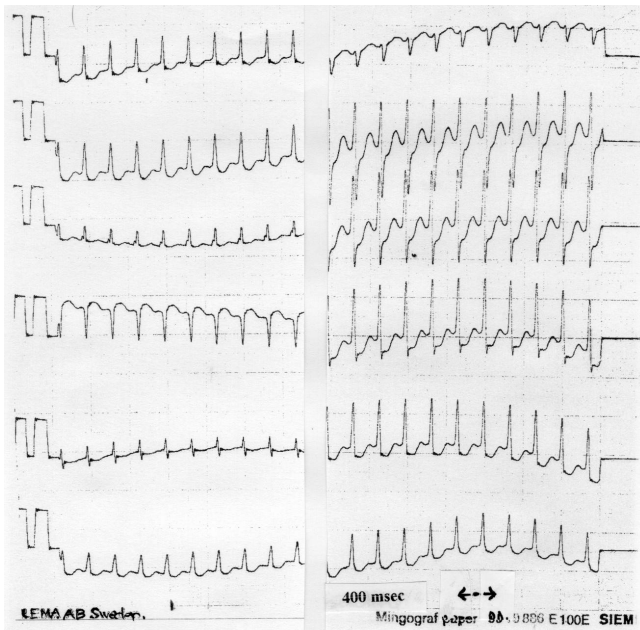


Figure 3. A 12-channel surface ECG recording of a regular SVT and a P wave is not clearly discernible, possibly due to ST segment abnormalities. This figure characterizes a regular reentrant tachycardia with a narrow QRS-complex, which was based on a Wolff-Parkinson-White syndrome with a left posterolateral localization of an accessory pathway. The cycle length of the tachycardia was 270 ms (rate of 220 bpm, paper speed of 25 mm/sec).

high success rates whenever a three-phase protocol was used that resembled the one in our study:

- decremental pacing until Wenckebach conduction occurred in the AV node; loss of 1:1 conduction over the AP or a minimum interval of 250 ms was reached;
- extrasystoles during spontaneous rhythm and after several runs at a basic drive with at least 1 CL of 600 ms; and
- burst stimulation starting at large intervals, up to very short intervals of 150 or even 100 ms [34-36].

A few studies have also reported the success rate of the induction of atrial fibrillation in patients with Wolff-Parkinson-White syndrome. In 3 studies, a success rate of 91 to 100 % was obtained, but these studies were small (≤ 11 patients). In larger studies, the success rate varied from 24 % to 96 % [37,38,41-43]. In our series, the success rate of atrial fibrillation was 63 % (25 out of 40 attempts). This rate is rather low compared to some of the previous studies mentioned. In part, this was due to the use of a less aggressive pacing protocol

in the early phase of the study. In a later phase, special attention was given to the induction of atrial fibrillation in patients with antegrade conduction over the AP, in order to estimate the conduction characteristics of this accessory pathway and to elicit conduction over a possible second pathway.

Invasive Evaluation

The patient with an antidromic reentrant tachycardia also showed other types of arrhythmias including an AV nodal reentrant tachycardia and an orthodromic RT. It is our hypothesis that one of the limbs in the AV node conducts the impulse from the ventricle to the atrium and the antegrade AV conduction uses the eccentric AP. The differential diagnosis of an atrial tachycardia (which is conducted over the accessory pathway with a 1:1 relationship) has to be taken into account. Such complicated cases show the limits of what can be achieved by noninvasive methods such as TRAS. Multicatheter ECG recordings monitoring the effect of carefully chosen premature beats may still be called for to elucidate the real activation pattern in such patients. In this particular case, the diagnosis of antidromic reentrant tachycardia was confirmed by the use of invasive methods.

Invasive electrophysiology studies are still needed for the evaluation of patients with complex supraventricular tachycardias, and when using radiofrequency catheter ablation for intervention. The major disadvantages of the intracardiac stimulation are the need for fluoroscopy, the risk in accessing the vasculature, and the expense related to personnel, catheters and equipment. The risk of an invasive electrophysiologic study is limited. However, at least 5 study-related deaths have been reported and reviews from other laboratories have noted serious complications in about 0.6 % or 0.7 % of cases [25,44,45]. These included deep venous thrombosis, pulmonary embolism, infection, pneumothorax, and bleeding or hematoma at the puncture site.

In this study, the rate of side effects with TRAS was not very low, but resulted in mild inconveniences of a very temporary nature. These types of minor side effects are not even reported in the literature when dealing with complications of invasive electrophysiologic studies.

Noninvasive Evaluation

Evaluation of patients using traditional noninvasive

tests is also another study approach. Noninvasive evaluation of the effective refractory period can be conducted using Holter recordings and exercise tests. A sudden disappearance of the pre-excitation during an increase in heart rate is correlated with a long refractory period of the accessory pathway [18,46,47]. This has also been seen in the Holter monitor recordings. Intermittent pre-excitation suggests a benign prognosis and is correlated with a long effective refractory period of the accessory pathway [18]. However, assessment of risk in an individual patient cannot be based on electrocardiographic data that is derived indirectly. Determination of the effective refractory period, and especially the shortest pre-excited RR interval, is of major importance for the risk assessment of an individual patient, even when a patient is asymptomatic [19,48]. Induction of atrial fibrillation is of crucial importance. The definite risk of a patient with a short pre-excited RR-interval has to be determined. Despite the very low rate of sudden death in a prospective study, the shortest pre-excited RR interval (SRR-PE) of less than 250 ms is a nonspecific marker of sudden death [49]. Sudden death as the initial manifestation of Wolff-Parkinson-White syndrome is extremely rare, yet it cannot be overlooked [50,51].

Conclusion

One of the advantages of TRAS is the ability to perform an investigation in an ambulatory care setting. This greatly reduces the costs of the investigation. This study shows that an invasive electrophysiologic study is not needed to determine the mechanism of SVT and to assess the risk of an AP. For TRAS, only an "adapted" stimulator and special electrodes are needed. A defibrillator should always be available. In the laboratory, 1 or 2 people can perform the study in less than one and a half hours.

One of the theoretical drawbacks of TRAS is that ventricular stimulation cannot usually be accomplished through the esophagus. This implies that an evaluation of ventricular arrhythmias requires an invasive study. However, as this study has shown, an invasive study is not needed for the evaluation of supraventricular tachycardias. The relationship between the esophagus and the atrium is much closer than between the ventricle and the esophagus [52,53]. When the TRAS method is applied for ventricular stimulation, a very high stimulus intensity would be needed to capture the

ventricles, which in turn causes pain. However, inadvertent ventricular stimulation has to be avoided, particularly at high pacing rates. Such accidental, direct ventricular capture has occasionally been described [28]. Indirect stimulation of the ventricle is possible through AV conduction. This can occur even in the presence of a ventricular tachycardia. In rare cases, a ventricular tachycardia was terminated by overdrive stimulation of the atrium, and 1:1 AV conduction was found to result in capture of the ventricle and subsequent termination of the tachycardia [54].

In conclusion, TRAS is a feasible technique in patients who have a history of regular tachycardia, with or without pre-excitation. It provides valuable information with respect to the mechanism of the tachycardia, and it allows for the shortest pre-excited RR-interval to be determined. Minor side effects do occur, but complications are absent. TRAS is a cost-effective tool in the evaluation of patients with Wolff-Parkinson-White syndrome.

References

- [1] Wellens HJJ, Durrer D. Effect of digitalis on atrio-ventricular conduction and circus movement tachycardias in patients with Wolff-Parkinson-White syndrome. *Circulation*. 1973; 47: 1229-1233.
- [2] Dreifus LS, Haiat R, Watanabe Y, et al. Ventricular fibrillation: A possible mechanism of sudden death in patients with Wolff-Parkinson-White syndrome. *Circulation*. 1971; 43: 520-527.
- [3] Garratt C, Antoniou A, Ward D, et al. Misuse of verapamil in pre-excited atrial fibrillation. *Lancet*. 1989; 1(8634): 367-369.
- [4] McGovern B, Garan H, Ruskin JN. Precipitation of cardiac arrest by verapamil in patients with Wolff-Parkinson-White syndrome. *Ann Intern Med*. 1986; 104: 791-794.
- [5] Gulamhusein S, Ko P, Klein GJ. Ventricular fibrillation following verapamil in the Wolff-Parkinson-White syndrome. *Am Heart J*. 1983; 106: 145-147.
- [6] Jackman WM, Wang XZ, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. *N Engl J Med*. 1991; 324: 1605-1611.
- [7] Calkins H, Sousa J, el-Atassi R, et al. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. *N Engl J Med*. 1991; 324: 1612-1618.
- [8] Neuss H, Schlepper M. Long-term efficacy and safety of flecainide for supraventricular tachycardia. *Am J Cardiol*. 1988; 62: 56D-61D.
- [9] Cockrell JL, Scheinman MM, Titus C, et al. Safety and efficacy of oral flecainide therapy in patients with atrioventricular re-entrant tachycardia. *Ann Intern Med*. 1991; 114: 189-194.

- [10] Yeh SJ, Fu M, Lin FC, et al. Serial electrophysiologic studies of the effects of oral diltiazem on paroxysmal supraventricular tachycardia. *Chest*. 1985; 87: 639-643.
- [11] Klein GJ, Gulamhusein S, Prystowsky EN, et al. Comparison of the electrophysiological effects of intravenous and oral verapamil in patients with paroxysmal supraventricular tachycardia. *Am J Cardiol*. 1982; 49: 117-124.
- [12] Kunze KP, Schlüter M, Kuck KH. Sotalol in patients with Wolff-Parkinson-White syndrome. *Circulation*. 1987; 75: 1050-1057.
- [13] Lévy S, Broustet JP, Clémenty J, et al. Syndrome de Wolff-Parkinson-White. Corrélations entre l'exploration électrophysiologique et l'effet de l'épreuve d'effort sur l'aspect électrocardiographique de préexcitation. *Arch Mal Coeur*. 1979; 72: 634-640.
- [14] Bricker JT, Porter CJ, Garson A Jr., et al. Exercise testing in children with Wolff-Parkinson-White syndrome. *Am J Cardiol*. 1985; 55: 1001-1004.
- [15] Darmanata JI. Ambulatory Oesophageal electrocardiographic recording. Thesis, Amsterdam, 1985.
- [16] Lipski J, Cohen L, Espinoza J, et al. Value of Holter monitoring in assessing cardiac arrhythmias in symptomatic patients. *Am J Cardiol*. 1976; 37: 102-107.
- [17] Sharma AD, Yee R, Guiraudon G, et al. Sensitivity and specificity of invasive and noninvasive testing for risk of sudden death in Wolff-Parkinson-White syndrome. *J Am Coll Cardiol*. 1987; 10: 373-381.
- [18] Klein GJ, Gulamhusein SS. Intermittent preexcitation in the Wolff-Parkinson-White syndrome. *Am J Cardiol*. 1983; 52: 292-296.
- [19] Waldo AL, Akhtar M, Benditt DG, et al. Appropriate electrophysiologic study and treatment of patients with the Wolff-Parkinson-White syndrome. Report of the North American Society of Pacing and Electrophysiology Policy Conference held in New Orleans, Louisiana, May 14, 1986. *PACE*. 1988; 11: 536-543.
- [20] Critelli G., Gallagher JJ, Perticone F, et al. Evaluation of non-invasive tests for identifying patients with preexcitation at risk of rapid ventricular response. *Am Heart J*. 1984; 108: 905-908.
- [21] Daubert C, Ollitrault J, Descaves C, et al. Failure of the exercise test to predict the anterograde refractory period of the accessory pathway in Wolff Parkinson White syndrome. *PACE*. 1988; 11: 1130-1138.
- [22] Klein GJ, Sharma AD, Yee R, et al. Classification of supraventricular tachycardias. *Am J Cardiol*. 1987; 60: D 27-31.
- [23] Bär FE, Brugada P, Dassen WRM, et al. Differential diagnosis of tachycardia with narrow QRS complex (shorter than 0.12 second). *Am J Cardiol*. 1984; 54: 555-560.
- [24] Kay GN, Pressley JC, Packer DL, et al. Value of the 12-lead electrocardiogram in discriminating atrioventricular nodal reciprocating tachycardia from circus movement atrioventricular tachycardia utilizing a retrograde accessory pathway. *Am J Cardiol*. 1987; 59: 296-300.
- [25] Hammill SC, Sugrue DD, Gersh BJ, et al. Clinical intracardiac electrophysiologic testing: technique, diagnostic indications, and therapeutic uses. *Mayo Clin Proc*. 1986; 61: 478-503.
- [26] Gallagher JJ, Smith WM, Kasell J, et al. Use of the esophageal lead in the diagnosis of mechanisms of reciprocating supraventricular tachycardia. *PACE*. 1980; 3: 440-451.
- [27] Benson Jr. DW, Dunnigan A, Benditt DG. Follow-up evaluation of infant paroxysmal atrial tachycardia: transesophageal study. *Circulation*. 1987; 75: 542-549.
- [28] Gallagher JJ, Smith WM, Kerr CR, et al. Esophageal pacing: A diagnostic and therapeutic tool. *Circulation*. 1982; 65: 336-341.
- [29] Res JCJ, Woersem RJ van, Dekker E, et al. Transesophageal atrial pacing- stimulation and discomfort thresholds: the role of electrode configuration and pulse width. *PACE*. 1991; 14: 1359-1365.
- [30] Lunati M, Vignati G, Gasparini M, et al. Transesophageal pacing in childhood. *New trends in Arrhythmias*. 1987; 3: 183-189.
- [31] Arzbaeher R. A pill electrode for the study of cardiac arrhythmia. *Med Instrum*. 1978; 12: 277-281.
- [32] Benson Jr. DW, Jadvar H, Strasburger JF. Utility of a stimulus artifact suppressor for transesophageal pacing. *Am J Cardiol*. 1990; 65: 393-394.
- [33] Gallagher JJ, Pritchett ELC, Sealy WC, et al. The preexcitation syndromes. *Prog Cardiovasc Dis*. 1978; 20: 285-327.
- [34] Benson Jr. DW, Dunnigan A, Benditt DG. Atrial pacing from the esophagus in the diagnosis and management of tachycardia and palpitations. *J Pediatr*. 1983; 102: 40-46.
- [35] Benson Jr. DW, Dunnigan A, Benditt DG, et al. Transesophageal study of infant supraventricular tachycardia: electrophysiologic characteristics. *Am J Cardiol*. 1983; 52: 1002-1006.
- [36] Fujiseki Y, Okuno M, Fujino H, et al. Transesophageal atrial pacing in paroxysmal supraventricular tachycardia in infants and children. *Acta Paediatr Jpn. Overseas Ed*. 1987; 29: 605-613.
- [37] Cebron JP, Le Marec H, Victor J, et al. Wolff-Parkinson-White syndrome. Value of transesophageal atrial stimulation coupled with exercise test for the study of antero-grade conduction in the accessory pathway. *Arch Mal Coeur*. 1989; 82: 159-166.
- [38] Delise P, D'Este D, Bonso A, et al. Usefulness of transesophageal electrophysiologic study during stress test in the evaluation of supraventricular tachycardia occurring during effort. *G Ital Cardiol*. 1989; 19: 1094-1104.
- [39] Harte MT, Teo KK, Horgan JH. The diagnosis and management of supraventricular tachycardia by transesophageal cardiac stimulation and recording. *Chest*. 1988; 93: 339-344.
- [40] Patruno N, Critelli G, Pulignano G, et al. Asymptomatic preexcitation. Identification of potential risk using transesophageal pacing. *Cardiologia*. 1989; 34: 777 - 781.
- [41] Disertori M, Vergara G, Inama G, et al. Protocol for the use of transesophageal cardiac stimulation in the diagnosis and treatment of arrhythmias. *G Ital Cardiol*. 1984; 14: 958-959.
- [42] Kieny JR, Kraenner C, Facello A, et al. Value of transesophageal atrial pacing to evaluate the accessory pathway and the effect of treatment in Wolff-Parkinson-White syndrome. *Arch Mal Coeur Vaiss*. 1989; 82: 1585-1593.

- [43] Vergara G, Furlanello F, Disertori M, et al. Induction of supraventricular tachyarrhythmia at rest and during exercise with transoesophageal atrial pacing in the electrophysiological evaluation of asymptomatic athletes with Wolff-Parkinson-White syndrome. *Eur Heart J*. 1988; 9: 1119-1125.
- [44] Krikler DM, Davies MJ, Rowland E, et al. Sudden death in hypertrophic cardiomyopathy: associated with accessory atrioventricular pathways. *Br Heart J*. 1980; 43: 245-251.
- [45] Horowitz LN. Safety of electrophysiologic studies. *Circulation*. 1986; 73(Suppl II): 28-31.
- [46] Strasberg B, Ashley WW, Wyndham CR, et al. Treadmill exercise testing in the Wolff-Parkinson-White syndrome. *Am J Cardiol*. 1980; 45: 742-748.
- [47] Lévy S, Broustet JP, Clémenty J, et al. Value of noninvasive techniques in the Wolff-Parkinson-White syndrome with particular reference to exercise testing. In: *Cardiac Arrhythmias*. Lévy S, Scheinmann MM (eds.). Futura Publishing Company, Mount Kisco, NY, 1984: 301-312.
- [48] Klein GJ, Prystowsky EN, Yee R, et al. Asymptomatic Wolff-Parkinson-White. Should we intervene? *Circulation*. 1989; 80: 1902-1905.
- [49] Milstein S, Sharma AD, Klein GJ. Electrophysiologic profile of asymptomatic Wolff-Parkinson-White pattern. *Am J Cardiol*. 1986; 57: 1097-1100.
- [50] Guize L, Soria R, Chaouat JC, et al. Prevalence and course of Wolff-Parkinson-White syndrome in a population of 138,048 subjects. *Ann Med Interne (Paris)*. 1985; 136: 474-478.
- [51] Klein GJ, Bashore TM, Sellers TD, et al. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med*. 1979; 301: 1080-1085.
- [52] Crawford TM, Dick 2nd M, Bank E, et al. Transesophageal atrial pacing: importance of the atrial-eso-phageal relationship. *Med Instrum*. 1986; 20: 40-44.
- [53] Binkley PF, Bush CA, Kolibash AJ, et al. The anatomic relationship of the esophageal lead to the left atrium. *PACE*. 1982; 5: 853-859.
- [54] Katz A, Knilans TK, Prystowsky EN. Bedside termination of sustained ventricular tachycardia by transesophageal atrial pacing. *PACE*. 1992; 15: 849-853.