Esophageal Pacing: A Diagnostic and Therapeutic Tool

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SUMMARY The purpose of this study was to develop guidelines for reproducible esophageal pacing of the atria and to determine the incidence of successful initiation and termination of tachycardia using this technique in patients with a history of spontaneous supraventricular tachycardia (SVT).

Strength-duration curves were performed in 39 patients using a bipolar esophageal lead with a 2.9-cm inter-electrode distance. Unlike strength-duration curves normally obtained in cardiac tissue, which plateau at pulse durations more than 2.0 msec, the esophageal current threshold decreased progressively as pulse duration was increased to the limit of the stimulator (9.9 msec). At pulse durations of 8.0-9.9 msec, atrial capture was achieved in all patients. At progressively shorter pulse durations, capture was achieved in progressively fewer patients despite use of current up to 30 mA. Stable pacing was achieved in 26 of 39 patients with a pulse duration of 1.0 msec (mean threshold 21 mA), in 33 of 39 patients with a pulse duration of 2.0 msec (mean threshold 18 mA), and in 39 of 39 patients with a pulse duration of 9.9 msec (mean threshold 11 mA). The current requirements did not correlate with the amplitude of the unipolar or bipolar atrial electrogram recorded in the group as a whole, but the lowest thresholds in individual patients occurred at the site where the largest and most rapid atrial deflections were recorded.

In 38 patients with documented SVT, overdrive pacing from the esophagus was performed at cycle lengths of 240-400 msec using a pulse duration of 7.0-9.9 msec. Reciprocating tachycardia was induced in 35 of 38 patients and was terminated by overdrive pacing in 33 of 38 patients. Atrial fibrillation was induced incidentally in four patients; sinus rhythm returned spontaneously. Other effects included ventricular pacing in two, unmasking of latent preexcitation in three, induction of ventricular tachycardia by atrial pacing in two patients with a history of ventricular tachycardia, and phrenic pacing in one.

We conclude that atrial pacing can be achieved from the esophagus with minimal discomfort in the majority of patients; that lower pacing thresholds can be obtained with the use of wide pulse durations (7.0-9.9 msec) and a bipolar electrode with wide interelectrode distance (2.9 cm); that rapid atrial pacing from the esophagus can be used to induce and terminate SVT for diagnostic or therapeutic purposes; and that esophageal pacing provides a convenient way to assess repeatedly the efficacy of long-term drug therapy and to screen patients for preexcitation syndromes.

THE PROXIMITY of the esophagus to the atria is the basis of using esophageal electrodes to record electrical activity from the atria. Several investigators have suggested using this route to deliver electrical stimuli to the atria or ventricles for diagnostic and therapeutic purposes, but this application has been limited by lack of consistent capture and patient discomfort resulting from high current requirements.

We recently reported the value of recording ventriculoatrial (VA) intervals from the esophagus during supraventricular tachycardia (SVT) to help define the mechanism of SVT. An absolute VA interval shorter than 70 msec during SVT excluded participation of an accessory Kent bundle pathway. This diagnostic technique was limited by the need for tachycardia at the time of the esophageal recording. The possibility of using the same esophageal lead to induce tachycardia by atrial pacing prompted the present study, in which we attempted to develop guidelines for reproducible pacing of the atria and to assess our ability to induce and terminate SVT by esophageal pacing in a series of consecutive patients with documented SVT.

Materials and Methods

The study group consisted of 65 patients, 45 males and 20 females, mean age of 35 years (range 7-70 years). Forty-six patients had SVT associated with a concealed or manifest preexcitation syndrome, five had SVT due to reentry confined to the atrioventricular (AV) node, five had paroxysmal atrial fibrillation, three had postoperative restudy after surgical division of a Kent bundle, four had ventricular tachycardia, one patient had atrial tachycardia and one patient had sick sinus syndrome.

Patients were studied in the postabsorptive state. Antiarrhythmic therapy was discontinued for 48 hours. An i.v. catheter was always inserted before the procedure. In 50% of the patients, small i.v. doses of diazepam or meperidine were used for sedation.

A bipolar permanent transvenous pacing electrode (Medtronic 6992) with electrodes spaced at 29 mm
Threshold determinations were made as a function of interelectrode distances of 0-8 cm; the pulse duration was held constant at 9.9 msec.

Induction Termination of Tachycardia

In 38 consecutive patients with a history of SVT, we tried to induce and terminate their arrhythmias. The majority of these patients subsequently underwent a detailed electrophysiology study. We tried to induce SVT by rapid pacing (cycle length 240-400 msec) using a wide pulse duration (7.0-9.9 msec) and enough current to maintain atrial capture (mean 15 mA). After induction of SVT and recording of the VA intervals, we tried to terminate SVT using the same pacing criteria.

Results

Placement of Esophageal Electrode

Satisfactory atrial electrograms were recorded with the esophageal electrode in all 65 patients. Maximal bipolar potentials ranged from 0.3-1.8 mV (mean 0.9 ± 0.34 mV) (± SD). The maximal unipolar potential ranged from 0.5-3.6 mV (mean 1.3 ± 0.6 mV). The maximal unipolar potential was recorded 27-45 cm from the nares (mean 38.5 ± 5 cm).

Strength-Duration Curves

Strength-duration curves were performed in 39 patients (fig. 2). In this patient, no capture occurred at 0.2-msec pulse duration; capture at 0.4-msec pulse duration required 29 mA, but with increasing pulse duration, current requirements decreased. Unlike strength-duration curves normally obtained in cardiac tissue, which plateau at pulse durations more than 2 msec, the current requirements continually decreased as pulse duration was increased up to the limit of the stimulator (9.9 msec).

The group results of strength-duration determinations in all 39 patients are shown in figure 3. In all pa-
patients, the current requirements decreased as pulse duration was increased. Stable atrial pacing was achieved in 26 of 39 patients using a pulse duration of 1.0 msec (mean threshold 21 ± 5 mA); in 33 of 39 patients using a pulse duration of 2.0 msec (mean threshold 18 ± 5 mA); and in 39 of 39 patients using a pulse duration of 9.9 msec (mean threshold 11 ± 5 mA). In our experience, moderate discomfort accompanies delivery of current in excess of 18 mA to the esophagus. Conventional external stimulating devices are typically limited to pulse durations of 2.0 msec. Pacing at a pulse duration of 2.0 msec would therefore result in a lower success rate, higher current requirements, and more symptoms in patients in whom atrial capture occurred.

The minimal pulse duration at which successful capture occurred ranged from 0.1–8.0 msec (average 1.2 ± 1.6 msec). The current thresholds at this minimal pulse duration ranged from 15–30 mA (mean 27 ± 3 mA).

One might expect that the larger the amplitude of the bipolar or unipolar electrogram recorded, the closer the proximity of the esophageal lead to the atria; thus, patients with atrial electrograms of greater amplitude might be expected to manifest lower pacing thresholds than patients with atrial electrograms of smaller amplitude. However, analysis of group results for the current threshold determined at a pulse duration of 9.9 msec showed a poor correlation with the amplitude of either the bipolar atrial electrogram (r = 0.22) or the unipolar atrial electrogram (r = −0.04).

Ventricular pacing via the esophagus (fig. 4) was observed in only two patients during withdrawal of the lead from the stomach to the esophagus. In both patients, a large ventricular electrogram without evidence of an atrial electrogram was recorded at the time ventricular pacing was noted. The anatomic position of the ventricles relative to the esophagus in these two patients probably facilitated ventricular pacing. Ventricular pacing was not noted when the lead was positioned with a predominant atrial deflection before institution of pacing.

Threshold as a Function of Interelectrode Distance

In seven patients, threshold determinations were made as a function of interelectrode distances of 0–8 cm, with the pulse duration held constant at 9.9 msec. In this subgroup (table 1), the optimal spacing appeared to be 3 cm, compared with 2.9 cm for performing strength-duration curves.

Induction and Termination of Tachycardia

Thirty-eight patients were entered into this part of the study, 33 with reentry utilizing VA accessory pathways and five with reentry confined to the AV node. Supraventricular tachycardia was induced one or more times in 35 of 38 patients (figs. 5 and 6). Tachycardia could not be induced in three patients who had manifest preexcitation and short antegrade effective refractory periods of their accessory pathways. SVT could not be induced with catheter

**Figure 3.** Esophageal strength-duration determinations in 39 patients. The current threshold is plotted as a function of pulse duration. The number of patients successfully paced using current up to 30 mA is indicated by N. See text for discussion.

**Figure 4.** Ventricular pacing during esophageal stimulation. In this example, the esophageal lead was passed to the level of the stomach. Pacing was performed using a pulse duration of 9.9 msec and a current of 15 mA. The esophageal lead was progressively withdrawn to the level of the atrium. Ventricular pacing occurred initially; upon withdrawal of the esophageal lead to the level of the atrium, atrial pacing alone was noted.
tachycardia. Ventricular stimulation was required to induce tachycardia. SVT was terminated one or more times in 33 of 38 patients. In one patient SVT could not be terminated and required drug therapy. In the remaining four patients, SVT was converted to atrial fibrillation (fig. 7), which spontaneously reverted to sinus rhythm. The same response to overdrive pacing was observed in these patients during subsequent electrophysiologic study using intracardiac pacing.

We did not try in this study to induce atrial fibrillation. Such a study is under way and preliminary results suggest that atrial fibrillation can be induced by this technique to characterize the ventricular response during atrial fibrillation in patients with pre-excitation syndromes.

Other Effects Observed During Esophageal Pacing

Esophageal pacing resulted in unmasking of pre-excitation due to a left lateral accessory AV pathway in three patients (fig. 8). Four patients with a history of ventricular tachycardia were studied and rapid atrial pacing resulted in induction of ventricular tachycardia in two. In one of these patients, the ventricular tachycardia was nonsustained; in the other, tachycardia was terminated by overdrive atrial pacing. In two of the four patients who had sustained ventricular tachycardia, overdrive esophageal atrial pacing during tachycardia demonstrated fusion and capture beats. In these cases, atrial pacing was combined with the administration of atropine (0.5 and 1.0 mg), which enhanced AV nodal conduction and permitted ventricular overdrive. Phrenic pacing was observed with high pacing thresholds in only one of 65 patients. Finally, one patient with incessant SVT demonstrated 2:1 AV block associated with a slower ventricular response during rapid esophageal pacing,

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<th>Bipolar separation (cm)</th>
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The pulse duration was constant at 9.9 msec.

FIGURE 5. Induction and termination of supraventricular tachycardia in a patient with documented reentry in the atrioventricular (AV) node. (left) Sinus tachycardia is initially present because of the prior administration of 1.0 mg of atropine to facilitate induction of reentry in the AV node. Overdrive atrial pacing by means of the esophageal electrode at a cycle length of 260 msec successfully initiates a supraventricular tachycardia. (right) Delivery of stimuli at the same cycle length results in a return to sinus rhythm (NSR) after a single junctional escape beat. RT = reciprocating tachycardia.

FIGURE 6. Esophageal (ESO) recordings obtained during study of a patient with supraventricular tachycardia due to reentry in the atroventricular (AV) node (same patient as in figure 5). Bipolar recordings during sinus rhythm (NSR) and after the induction of supraventricular tachycardia using overdrive esophageal pacing are shown. During supraventricular tachycardia, the atrial electrogram recorded from the esophagus occurs nearly simultaneously with the QRS complex. RT = reciprocating tachycardia; I = standard ECG lead I; A = atrial electrogram.
and was stabilized for several hours until a transvenous catheter could be implanted before open heart surgery.

Patient Tolerance of Esophageal Pacing

All patients experienced discomfort, most frequently described as a mild burning sensation or chest pain and likened to that of indigestion. Most patients tolerated this discomfort easily. Occasional moderate discomfort was noted, almost invariably in patients with thresholds in excess of 18 mA. Intravenous diazepam or meperidine was administered to approximately half of the patients. No patient had discomfort severe enough to force discontinuation of the study. Approximately 15% of the patients underwent two or more studies for assessment of drug therapy or for termination of tachycardia.

Discussion

The use of an esophageal lead to stimulate the heart was first reported by Zoll in 1952 as a technique for pacing the ventricles. Later investigators reported use of unipolar or bipolar esophageal leads to stimulate the atria or ventricles.2-14 In general these applications were undertaken with pulse durations of 2.0 msec or less, which resulted in high current requirements, patient discomfort, pacing of thoracoabdominal muscles and inconsistent capture. Ordinarily, strength-duration curves performed in cardiac tissue in close contact with the stimulating electrode show a plateau with little change in threshold achieved by increasing the pulse duration in excess of 2.0 msec.16 In this study we demonstrated a progressive decline in current requirement up to a pulse duration of 9.9 msec. This relationship has not been reported, and suggests a capacitance effect due to the distance and nature of the tissues between the esophagus and atrium. The combined effect of using a long pulse duration and a wide interelectrode distance permitted reliable atrial capture at comparatively low thresholds in our series.

Esophageal pacing provides a relatively noninvasive method for pacing the atria without fluoroscopy, sterile precautions or cardiac catheterization, and thus compares favorably with temporary transvenous atrial pacing. Consistent atrial capture, especially at rapid pacing rates, may be difficult by the transvenous route, which requires placing the pacing catheter in the right atrial appendage or the coronary sinus.

Esophageal pacing can also be performed ex-

FIGURE 7. Inadvertent induction of atrial fibrillation during attempts to overdrive supraventricular tachycardia in a patient with documented Wolff-Parkinson-White syndrome. Supraventricular tachycardia with a normal QRS complex is initially present with a cycle length of 330 msec. Four stimuli are delivered from the esophageal electrode at a cycle length of 240 msec, resulting in atrial fibrillation with antegrade conduction occurring over an accessory pathway in the left lateral atrioventricular groove. The atrial fibrillation spontaneously reverts to sinus rhythm. The artifact between the second and third stimuli was caused by movement of the patient. Atrial fibrillation was also elicited by overdrive atrial pacing during subsequent invasive study of this patient.

FIGURE 8. Unmasking of latent preexcitation in a patient with documented Wolff-Parkinson-White syndrome. Sinus rhythm is initially present in this patient, who had a history of supraventricular tachycardia and a normal resting ECG. Initiation of atrial pacing from the esophagus at a cycle length of 400 msec results in unmasking of preexcitation, which was subsequently shown to be due to an accessory pathway in the left lateral atrioventricular groove.
peditiously in ambulatory, bedside or emergency situations and can be repeated chronically. Because atrial pacing is performed from outside the heart, esophageal pacing is particularly well suited to the study of patients with known or suspected preexcitation. We previously showed that accessory pathways located on the tricuspid annulus or in the septal area can be partially or completely obtunded temporarily owing to catheter trauma. More recently, a left free-wall accessory pathway was traumatized in our laboratory during manipulation of a coronary sinus catheter and has not regained function after 1 year. PACing in patients with preexcitation can be accomplished from the esophagus without disrupting the accessory pathway.

Certain precautions seem reasonable. PACing of the ventricle occurred in two of 65 patients, and only when the esophageal lead was advanced far enough to record a large ventricular electrogram. Nevertheless, pacing should always be instituted at a slow rate to ensure that the ventricle is not inadvertently paced at rapid rates. Atrial fibrillation may be inadvertently induced by rapid atrial pacing and may result in a rapid ventricular response in patients with preexcitation syndromes. For the above reasons, an intravenous catheter should be in place before esophageal pacing, and cardioversion equipment should be readily available. All recording and stimulating equipment should be electrically isolated.

In this study, we used esophageal pacing to reliably induce and terminate reentrant SVT. However, esophageal pacing may be extended to all reported uses of temporary atrial pacing. Possible indications include determination of sinus node recovery times, stress of AV conduction and screening for latent antegrade preexcitation, temporary management of bradyarrhythmias due to sinus node dysfunction, induction and termination of supraventricular tachycardias for diagnostic or therapeutic purposes,19 induction of atrial fibrillation in patients with preexcitation for prognostic considerations, conversion of flutter to sinus rhythm or atrial fibrillation with a slower ventricular response, demonstration of capture and fusion in patients with ventricular tachycardia, and repeated assessment of the long-term efficacy of drug therapy for supraventricular arrhythmias.

Addendum

Since submission of this manuscript, we have learned of another study: Stery H, Prager H, Koller H: Transesophageal rapid stimulation of the left atrium in atrial tachycardias. Z Kardiol 67: 136, 1978

Acknowledgment

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