

covery room who is constantly stimulated by the nursing staff and remains awake will show less respiratory depression than the patient who is not stimulated and falls asleep.

A final consideration in these patients would be an adverse drug interaction resulting in either increased respiratory depression or in prolongation of the action of alfentanil. Neither of these patients received any anesthetic medications other than alfentanil, nitrous oxide, relaxant, and thiopental. In this group of drugs, only alfentanil, thiopental, and the muscle relaxants have significant respiratory depressant properties. Since neither patient had received more than 1 mg/kg of thiopental during the last hour of the case or more than 5 mg/kg for the entire case, respiratory depression from thiopental is unlikely. Also, neither patient showed any evidence of residual neuromuscular blockade. Drug interactions between alfentanil and the antihypertensive medications, labetalol and nitroglycerine, have not been previously reported. Whether these drugs played a role in these cases is unknown, but such an interaction is highly unlikely.

Clinically, both of these patients exhibited delayed respiratory depression similar to that observed in some patients after fentanyl administration. The rigidity observed in the second patient has also been observed in patients with postoperative respiratory depression following fentanyl.¹⁰ The clinician should be aware that delayed respiratory depression may occur when alfentanil infusions are used for long cases. The fact that alfentanil is a short-acting drug should not lure the clinician into omitting

close respiratory monitoring and appropriate patient stimulation during the recovery period.

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Atrial Esophageal Pacing in Patients Undergoing Coronary Artery Bypass Grafting: Effect of Previous Cardiac Operations and Body Surface Area

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Atrial esophageal pacing is a fast, easy, relatively benign method of cardiac pacing. In patients with bradycardia,

pacing increases cardiac output and raises coronary perfusion pressure,¹ and, therefore, may be useful in patients at risk of developing bradycardia and ischemia intraoperatively. This is especially true in patients undergoing coronary artery bypass grafting secondary to their underlying disease and the drugs they commonly receive, namely beta adrenergic blockers and calcium channel entry blockers. Induction of anesthesia, especially with high-dose opioids and post-tracheal intubation, is frequently attended by bradycardia.

The factors limiting use of esophageal pacing include pacing current threshold levels, inability to consistently pace the ventricle, inadvertent diaphragmatic pacing, and the potential for esophageal injury. The mechanical as-

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pects of the esophageal catheter design and delivery of pacing current have been investigated previously. Pacing current threshold is affected by pulse duration, esophageal catheter size, interelectrode spacing, and pulse shape.²⁻³ There is evidence to demonstrate that the esophagus is not damaged by the electrical current during esophageal cardiac pacing.⁴

With the exception of determining the optimal depth of insertion of pacing electrodes into the esophagus, the effect of patient variables on esophageal pacing current threshold requirements is not clear.⁵ This study was thus primarily designed to determine the effects of a previous cardiac operation on pacing current threshold, our hypothesis being that scar tissue might alter the threshold. Secondly, the effects of body size and previous myocardial infarction were observed. Also, hemodynamic changes before and after the induction of pacing in patients with bradycardia were documented.

MATERIALS AND METHODS

With informed consent and institutional approval, 11 patients scheduled for coronary artery bypass grafting (CABG) for the first time and ten patients undergoing the operation for the second time were enrolled. Patients with esophageal disease, who gave a history of pericarditis or cardiomyopathy, or who were not in sinus rhythm were excluded. Patient weight, body surface area, and previous history of myocardial infarction were recorded. Monitoring of all patients included systemic and pulmonary arterial blood pressures in addition to the surface electrocardiogram. Cardiac output was determined by thermodilution. All patients were premedicated with morphine or methadone im and oral diazepam. Anesthesia was induced with iv fentanyl and vecuronium, and then their tracheas were intubated. A Portex Cardiosophagoscope® with an interelectrode distance of 10 cm was inserted orally to a depth determined by the greatest P wave deflection on the bipolar atrial electrogram. Pacing current thresholds were determined for all patients at 2, 4, 6, 8, and 10 msec pulse duration using a pulse generator designed and built at our institution for this purpose. For those patients with post-induction bradycardia (heart rate less than or equal to 60 bpm) atrial pacing was instituted at a pulse duration of 10 msec, a current great enough to maintain constant pacing, and a rate 1½ times baseline. Profiles of several hemodynamic variables were obtained prior to and after the commencement of pacing.

Hotelling's T^2 test was applied to test the null hypothesis that the two populations (first time *versus* redo CABG

TABLE 1. Pacing Current Thresholds

Pulse Duration (msec)	Mean Current Threshold (mamps)		<i>t</i> Statistic	<i>P</i> Value
	First Time	Redo		
2	27.6 ± 7.9	28.9 ± 4.0	.46	.65
4	24.0 ± 7.2	25.6 ± 4.1	.61	.55
6	20.9 ± 6.2	21.9 ± 4.4	.45	.66
8	19.5 ± 6.0	20.2 ± 4.1	.28	.78
10	17.8 ± 6.0	18.9 ± 3.9	.49	.63

Hotelling's $T^2 = 7.3$, $F_{5,15} = 1.2$, and $P = 0.37$. All values are mean ± SD.

patients) which were sampled did not differ in their mean capture current at any of the five pulse durations used. This test was also applied to compare mean capture currents between patients who had and had not suffered a previous myocardial infarction. Correlation of body size to current threshold levels, both by weight and body surface area, was analyzed with a linear least squares of the means method. Comparisons of hemodynamic profiles, baseline and after commencement of pacing, was performed with two-tailed *t* tests.

RESULTS

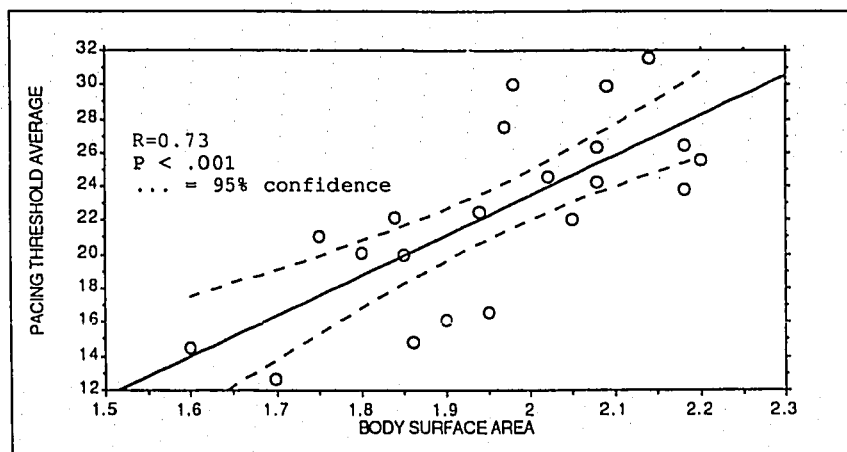
The average age of all patients was 54 yr with a range of 39–72 yr. Two patients had single vessel disease, three had two vessel disease, 12 had three vessel disease, and four had left main coronary artery disease. There was no significant difference between first-time CABG patients and redo CABG patients in extent of disease or cardiac function (mean ejection fraction 0.47 ± 0.10 (SD) *versus* 0.45 ± 0.12).

There was not a significant difference in age, extent of disease, or cardiac function of the patients who had post-intubation bradycardia from those who did not. Six of these patients were taking both a beta adrenergic blocker and either verapamil or diltiazem, three patients were taking beta adrenergic blockers alone, one a calcium entry blocker alone, and one was taking neither drug.

Pacing current thresholds decreased for all patients with increasing pulse duration in a manner previously described.⁶ There was no significant difference between the pacing current thresholds for the patients undergoing coronary artery bypass grafting for the first time compared to those undergoing the operation subsequently (table 1). There was no significant difference in pacing current thresholds between patients who had had myocardial infarctions from those who had not. Location of the infarct had no effect on the ability to pace. There was a positive correlation between pacing current threshold levels and body size, the best correlate being body surface area (fig. 1).

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FIG. 1. Relationship of pacing threshold average (mamps) to body surface area (meters²).



In the 11 patients who became bradycardic post-tracheal intubation, esophageal pacing significantly ($P < 0.05$) increased cardiac index, systemic blood pressure, heart rate, and cardiac index with the commencement of pacing. Systemic blood pressure increased, and pulmonary artery mean and diastolic blood pressure increased a clinically insignificant amount. Pulmonary systolic pressure did not change. Systemic vascular resistance fell slightly (table 2).

No dysrhythmias or signs of myocardial ischemia, as determined by continuous recording of leads II and V, were noted during the time the heart was being paced. Ventricular pacing was not noted in any patient.

DISCUSSION

Little is known about patient variables in determining esophageal pacing thresholds. Until now, most work has concentrated on the equipment and methods to decrease current threshold levels. One might expect that pacing current thresholds would be higher in patients who had

undergone a previous cardiac operation because of the development of adhesions and scar tissue around the heart. However, we could find no evidence that this was the case, and it was not more difficult to pace patients who had previously undergone coronary bypass surgery. Similarly, it has been suggested that patients with old myocardial infarctions may be more difficult to atrially pace from the esophageal approach,⁷ but we found no significant difference in pacing current thresholds between patients with or without old myocardial infarctions. Furthermore, location of the infarct seemed to have no effect on pacing current thresholds. This widens the applicability of this mode of pacing in a group of patients likely to develop intraoperative bradycardia.

We found a positive correlation between body surface area and pacing current threshold. One might empirically expect this to be the case, since the pacing current must generally traverse more extracardiac tissue in a larger patient than in a smaller one. The high pacing current threshold levels in this study are a result of using a pacing catheter with a large interelectrode distance. This was used because it is a commercially available, inexpensive, and disposable catheter marketed for recording atrial electrograms.

The significant increases in cardiac index and systemic blood pressure with pacing in patients with bradycardia confirm data previously reported by Backofen *et al.*⁸ Our data add to the growing body of evidence that esophageal pacing in the setting of intraoperative bradycardia is effective in precisely controlling an increase in the heart rate and therefore increasing cardiac output and arterial blood pressure. Perhaps more importantly, coronary perfusion pressure, as estimated by systemic diastolic pressure, is increased with pacing. We used a relatively high cutoff heart rate (60 bpm) for instituting pacing to determine hemodynamic effects. Backofen's group had used a cutoff of 55 bpm with pacing increases to either 68 or 78 bpm, but the results from the two studies are very similar. In both studies there was a decrease in stroke volume of

TABLE 2. Hemodynamic Changes in Response to Esophageal Pacing

	Before Pacing	After Pacing	Difference
Heart Rate (bpm)	50 ± 7	82 ± 8	32 ± 10*
Cardiac Index (l/min/m ²)	2.0 ± 0.4	2.8 ± 0.6	0.8 ± 0.5*
Systemic Blood Pressure (mmHg)			
Systolic	111 ± 16	123 ± 22	12 ± 9*
Mean	73 ± 11	86 ± 13	13 ± 8*
Diastolic	57 ± 8	66 ± 9	9 ± 6*
Pulmonary Blood Pressure (mmHg)			
Systolic	25 ± 8	27 ± 8*	2 ± 3
Mean	19 ± 6	22 ± 6	3 ± 3*
Diastolic	14 ± 5	17 ± 6	3 ± 3*
Systemic Vascular Resistance (dynes·sec/cm ⁵)	1258 ± 318	1051 ± 269	-207 ± 242*

* Indicates significance at the $P < 0.05$ level. All values are mean ± SD.

about 25% with pacing, but there was no indication of myocardial ischemia. The proper indications to institute pacing and the desired endpoints are subject to debate, but we have shown that, when desired, atrial esophageal pacing can be used in precisely controlling the heart rate in the setting of bradycardia and possible hypotension, which cannot be done by pharmacologic means.

Our study dealt only with atrial pacing, and all of our patients were in sinus rhythm. It should be emphasized that esophageal cardiac pacing cannot be used in patients with second or third degree heart block because of the inability to adequately and consistently pace the ventricle *via* the esophageal route.

In conclusion, we have shown body size does, but previous cardiac operative status does not, influence esophageal pacing current threshold. Increases in systemic blood pressure and cardiac output resulting from pacing were confirmed, and, therefore, we have defined better the effectiveness of the esophageal route of cardiac pacing.

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Thiopental for Phantom Limb Pain during Spinal Anesthesia

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Recurrences of phantom limb pain, occurring during a subsequent spinal anesthesia, have been described in several patients with previous lower limb amputations.¹⁻⁶ However, no therapy has been uniformly effective. We describe three cases where the pain was completely abolished by intravenous administration of sub-anesthetic doses of thiopental.

CASE REPORTS

Case 1. A 68-yr-old, 43-kg woman with advanced tabetic arthropathy was scheduled for the right mid-thigh amputation. She had a history

of severe pain by neural syphilis and had been taking analgesics herself for the past 30 yr. Two years previously she was admitted to our hospital, where morphine 30-40 mg daily, and other analgesics and tranquilizers had been administered. After a left mid-thigh amputation, performed 6 months previously, she had experienced severe phantom limb pain, but had been pain-free for 2 weeks prior to the second operation. Oral flunitrazepam 0.5 mg was given 90 min preoperatively. In the right lateral position, the spinal anesthesia was introduced with a 23-gauge needle at the L2-3 interspace using 14 mg tetracaine and 80 µg morphine in 2.8 ml of 10% glucose. Subsequently, the patient was put into the supine position. Five minutes later, she started to complain of phantom limb pain which was much more severe than that she had before. The sensory level to pin prick was at T6 bilaterally. Intravenous administration of 60 mg (1.4 mg/kg) thiopental immediately, though not completely, relieved the pain. Because of her restlessness, three doses of 5 mg diazepam were administered iv 5, 10, and 15 min after thiopental injection. Although the patient was still alert and somewhat restless, the pain completely disappeared and did not occur again. The operation was performed as arranged. The sensory level to pin prick was at T10 bilaterally at the end of the surgery.

Case 2. A 77-yr-old, 37-kg man with arteriosclerosis obliterans of lower extremities was scheduled for the right below-knee amputation. Two months previously, he underwent an amputation through the middle of the right foot, but experienced no pain thereafter. Oral flunitrazepam 0.5 mg was given 90 min preoperatively. In the right lateral position, the spinal anesthesia was introduced with a 25-gauge needle at the L3-4 interspace using 8 mg tetracaine and 2 mg phenylephrine in 1.6 ml of 10% glucose. He was kept in the same position for 15 min. The sensory level to pin prick was at L1, only in the right half of his body. Then he was turned into the supine position. Ten

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