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Transesophageal Atrial Pacing in Anesthetized Patients with Coronary Artery Disease

Hemodynamic Benefits Versus *Risk of Myocardial Ischemia*

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Background: **Transesophageal atrial pacing (TEAP) provides prompt and precise control of heart rate and improves hemodynamics in anesthetized patients with bradycardia and hypotension. The authors' purpose in this study was to examine the hemodynamic benefits of TEAP *versus* the risk of myocardial ischemia in patients about to undergo coronary artery bypass surgery.**

Methods: **Hemodynamics, ventricular filling pressures, mixed venous oxygen saturation, and end-diastolic, end-systolic, and fractional area change of the left ventricle, determined by transesophageal echocardiography (TEE), were measured after anesthesia induction with 30 μg/kg fentanyl and at incremental TEAP rates of 65, 70, 80, and 90 beats/min (bpm) in 40 adult patients. Monitoring for myocardial ischemia was accomplished with 12-lead electrocardiograms and biplane TEE assessment of left ventricular regional wall motion. Hemodynamics, electrocardiograms, and TEE measurements at each TEAP rate were compared with baseline awake measurements (except TEE) and with measurements obtained after anesthesia induction before TEAP.**

Results: **Sinus bradycardia occurred in 15 patients after anesthesia induction and was associated with a hypotensive response and a decrease in cardiac output in 10 patients. In these patients, TEAP restored diastolic blood pressure and cardiac output to baseline values at TEAP rates of 65 and 80 bpm, respectively. Stroke volume was similar to baseline measurements after anesthesia induction and at TEAP rates of 65, 70, and 80 bpm, but was significantly reduced from baseline at TEAP 90 bpm. Myocardial ischemia was detected in 7 and 5 patients at a TEAP rate of 80 and 90 bpm, respectively.**

Conclusions: **Control of heart rate with TEAP restores intraoperative hemodynamics to baseline in patients in whom bradycardia and a hypotensive response develop before coronary artery bypass surgery. When using TEAP for patients with severe coronary artery disease, these results support using the lowest TEAP rate titrated to achieve optimal hemodynamics, while closely monitoring for myocardial ischemia, especially at TEAP rates > 80 bpm. (Key words: Cardiac pacing: transesophageal. Coronary artery disease: myocardial ischemia. Echocardiography: transesophageal. Surgery: coronary artery bypass.)**

INDIVIDUALS with coronary artery disease have a limited ability to augment coronary blood flow commensurate with an increase of myocardial oxygen demands.[1,2] In the presence of restriction to coronary arterial blood flow, increased myocardial oxygen consumption secondary to increased heart rate can provoke myocardial ischemia and worsen ischemic injury.[1–7] Consequently, reduction in heart rate intraoperatively is usually viewed favorably as promoting myocardial oxygen metabolic balance in patients with coronary artery disease. In this group of patients, however, bradycardia often results in hypotension and reduced coronary perfusion pressure, which, potentially, also may precipitate myocardial ischemia.[6] In addition, bradycardia may lead to hemodynamic compromise in patients with dilated cardiomyopathy or more serious dysrhythmias in patients with sick sinus syndrome or prolonged QT intervals.[8–10]

The treatment of bradydysrhythmias during anesthesia may be problematic. Chronotropic drugs can either be ineffective or cause tachycardia and dysrhythmias.[11–14] Temporary cardiac pacing using epicardial, transvenous, or transcutaneous methods is not always clinically feasible on an emergent basis, and is also associated with other limitations.[15,16] It was reported that transesophageal atrial pacing (TEAP) provides prompt, effective, and precise control of heart rate in patients

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with sinus bradycardia and atrioventricular junctional rhythm during anesthesia.[12–14,17,18] Increased cardiac output and coronary perfusion pressure also was demonstrated to result from TEAP in patients undergoing coronary artery bypass graft (CABG) surgery.[13,14,17]

Hemodynamic improvements associated with TEAP in patients with coronary artery disease may, however, be complicated by the development of myocardial ischemia at high heart rates.[1–7] In studies of TEAP in CABG surgical patients, researchers found no evidence of myocardial ischemia.[13,14,17] Monitoring for ischemia in these studies was limited and, perhaps, episodes of myocardial ischemia would have been detected with more intensive monitoring.[19–21] Therefore, whether optimal hemodynamics using TEAP can be attained while avoiding myocardial ischemia is not clear. Our purpose in this study, therefore, was to prospectively (1) evaluate the effects of TEAP at several incremental heart rates on hemodynamics and other parameters of left ventricular (LV) function, and (2) assess for the development of myocardial ischemia during TEAP through use of 12-lead electrocardiographic (ECG) and transesophageal echocardiographic (TEE) monitoring in anesthetized patients before CABG surgery.

Methods and Materials

All procedures of this study met the approval of our Institutional Review Board and received individual informed consent. Forty-five patients scheduled for elective CABG surgery were enrolled. Coronary angiography was performed before surgery, and the degree of coronary arterial stenosis was quantified according to standard institutional procedures. Patients were excluded from this study for the following: emergency procedures, combined valvular surgery, myocardial infarction < 3 months before surgery, left ventricular ejection fraction < 35%, left main coronary artery stenosis > 70%, intraaortic balloon pump, preexisting cardiac pacemakers, atrioventricular conduction abnormalities, atrial fibrillation, left bundle branch conduction block, left ventricular hypertrophy, preoperative digoxin use, and esophageal varices, stricture, or tumor.

Preoperative cardiac medications were continued until the time of surgery, and the patients were premedicated with morphine sulfate, lorazepam, and scopolamine. Patient monitoring included radial arterial and oximetric pulmonary arterial catheters (Opticath, Abbott Laboratories, North Chicago, IL) and a 12-lead ECG (PageWriter XLi, Hewlett Packard). Anesthesia was induced with 30 μg/kg fentanyl, and vecuronium was given for muscle relaxation. After tracheal intubation, a TEAP electrode catheter (TAPCATH, Arzco Medical Systems, Vernon Hills, IL) was inserted to 50 cm from the infraalveolar ridge to pacing electrodes. A biplane TEE probe (Hewlett Packard Sonos OR, Andover, MA) was inserted. After TEE probe placement, the TEAP electrode was withdrawn to the level of maximal atrial depolarization and connected to a pulse generator (Model 7A, Arzco Medical Systems, Vernon Hills, IL).

Protocol

Before anesthesia induction, a 12-lead ECG and baseline hemodynamic measurements were obtained. Hemodynamic measurements included systolic, mean and diastolic systemic and pulmonary arterial pressures, central venous pressure, pulmonary capillary wedge pressure, cardiac output, stroke volume, systemic and pulmonary vascular resistance, and mixed venous oxygen saturation. Cardiac output was determined by thermodilution through use of the average of three room temperature, saline injectate measurements. At any study point, cardiac output measurements that varied by >10% of the previous measurement were disregarded, and the measurement was repeated. Hemodynamic measurements and 12-lead ECG were repeated after tracheal intubation. At this time, baseline TEE images of the LV also were obtained. Cardiac pacing was initiated with a pulse width of 10 msec and varying amplitude (10–20 mA), as required to ensure atrial capture. Atrial pacing was performed in an incremental manner in each patient at 65, 70, 80, and 90 beats/min (bpm), with each rate maintained for 3–5 min. If the patient's rate was >65 bpm, pacing was started at 70 bpm. Patients with heart rates > 70 bpm were excluded from analysis. The pacing study protocol was performed in the absence of surgical or other patient stimulation. Hemodynamic measurements, 12-lead ECG, and TEE recordings were repeated at the end of each specified pacing heart rate. The pacing protocol was terminated if myocardial ischemia was detected clinically, as judged by the clinicians carrying for the patient, using either the operating room ECG monitor or 12-lead ECG for diagnosis.

Echocardiography

The biplane TEE probe was positioned to obtain images of the LV at the mid-papillary muscle level in two planes: the transverse–short axis plane, in which the

septal, anterior, lateral, inferior–posterior walls can be visualized, and the longitudinal long-axis plane, in which inferior, anterior, and apical walls are imaged. Echocardiographic images were recorded on VHS tape and later arranged in a split screen, side-by-side, cine-loop format for comparison of baseline images with those obtained at each pacing rate with the order of the images scrambled. The recorded echocardiograms were reviewed off-line by an experienced echocardiographer blinded to hemodynamic data and TEAP rates (ECG not recorded on TEE images). Regional wall motion was assessed through use of a semi-quantitative score (1 = normal wall motion, 2 = hypokinesis, 3 = akinesis, 4 = dyskinesis) of the images at baseline and at each paced heart rate, using a 12-segment modification of the system recommended by the American Society of Echocardiography.[22] Left ventricular end-diastolic (EDA) and end-systolic (ESA) areas also were measured off-line by planimetry at the endocardial/blood border interface from images obtained in the short-axis view at the mid-papillary muscle level. Care was taken to ensure that the imaging plane was the same for all measurement intervals. The average of three EDA and ESA measurements were determined at baseline, after anesthesia induction, and at each paced heart rate. Fractional area change (FAC = (EDA − ESA)/EDA) also was calculated. Based on FAC determinations obtained immediately after anesthesia induction, left ventricular function was categorized as normal if FAC ≥ 45%, or moderately reduced for FAC < 45%

Myocardial Ischemia Definitions

Twelve-lead ECGs obtained at each point in the protocol were reviewed independently by two physicians. The ECG definition of myocardial ischemia was ≥1 mV shift in the ST segment from the isoelectric point determined 60 ms after the J point. Inclusion of a shift in the ST segment as compatible with a myocardial ischemic episode required the acceptance of both reviewers. The echocardiographic definition of myocardial ischemia was an increase in LV regional wall motion score of greater than 1 grade compared with baseline. The intra-individual concordance of our laboratory using this TEE method for the determination of new regional wall motion changes was determined to be 92%, and the inter-individual concordance was 93%.[23] The development of myocardial ischemia at each TEAP rate was considered for episodes meeting the ECG and/or echocardiographic definitions determined off-line (*i.e.*, not based on responses determined during pacing protocol).

Data Analysis

Hemodynamic and TEE-derived measures of LV dimension were compared with baseline measurements obtained before anesthesia induction (hemodynamic measurements only) and after anesthesia but before TEAP. To assess hemodynamic improvements with TEAP, sinus bradycardia was defined as heart rate < 60 bpm, and bradycardia-associated hypotension was defined as mean arterial pressure ≤ 70 mmHg or decreases in mean arterial pressure ≥ 25% compared with values obtained before anesthesia induction. Continuous data, determined to be distributed normally by Bartlett's test, were analyzed with one-way analysis of variance with Bonferroni correction for multiple comparisons. Discrete data were analyzed with Fisher's exact test. Significance was considered for values of $P < 0.05$.

Results

Five patients were eliminated from analysis because of heart rate > 75 bpm after anesthesia induction (n = 4) or because of technical difficulties with TEAP (n = 1). Demographic information for the remaining 40 patients is shown in table 1, with patients grouped by the presence or absence of bradycardia associated with a hypotensive response after anesthesia induction. Fifteen patients had heart rates < 60 bpm after anesthesia induction, and 3 of these patients had heart rates < 50 bpm. Of the 15 patients with bradycardia, 10 met the criteria for having hypotension. No episodes of atrioventricular junctional rhythm were observed. There were no differences in age between patients with and without hypotension during anesthesia induction, nor were there differences between these two groups with respect to medication use before surgery or with the other characteristics listed. Likewise, there were no differences in the distribution of coronary artery stenosis between patient groups, with the exception of no patients in the hypotensive group having left main coronary arterial stenosis from 50–70%. There also were no differences between groups in the number of patients with normal or moderately reduced LV function or in baseline wall motion score. Characteristics for those patients with evidence of myocardial ischemia developing during TEAP, regardless of hemodynamic response to induction of anesthesia, are listed in table

Table 1. Demographic Information for Patients with and without Hypotension and for Patients from Both Groups Developing Myocardial Ischemia with TEAP

	No Hypotension (n = 30)	Hypotension (n = 10)	Myocardial Ischemia (n = 12)
Age*	64.2 ± 7.6	67.5 ± 11.8	62.2 ± 8.8
Hypertension	12 (40%)	9 (90%)	5 (42%)
Diabetes mellitus	5 (17%)	3 (30%)	2 (17%)
History of MI	11 (48%)	5 (50%)	8 (67%)
Tobacco use	10 (33%)	0	3 (25%)
Medication			
β-blockers	17 (57%)	8 (80%)	9 (75%)
Ca^{++} channel blockers	11 (37%)	5 (50%)	6 (50%)
Nitroglycerin	18 (60%)	10 (100%)	9 (75%)
ACE inhibitors	8 (27%)	4 (40%)	4 (33%)
Aspirin	19 (63%)	8 (80%)	8 (67%)
Heparin	10 (33%)	4 (40%)	5 (42%)
Coronary arterial stenosis			
LAD >70%	27 (90%)	10 (100%)	11 (92%)
Circumflex >70%	19 (63%)	4 (40%)	7 (58%)
RCA >70%	24 (80%)	8 (80%)	10 (83%)
Left main 50–70%	6 (20%)	0	2 (17%)
LV function			
Normal	6 (20%)	1 (10%)	2 (17%)
Moderately reduced	24 (80%)	9 (90%)	10 (83%)
Baseline wall motion score*	14.1 ± 3.4	13.8 ± 3.4	13.9 ± 2.5

MI = myocardial infarction; β-blockers = β-adrenergic blocking drugs; Ca^{++} channel blockers = calcium channel blocking drugs; LAD = left anterior descending coronary artery; RCA = right coronary artery; LV = left ventricular.

* Mean ± SD.

1. There were no significant differences between patients with and without myocardial ischemia for any of the patient characteristics listed, including baseline LV function and wall motion score.

Hemodynamic and echocardiographic results are shown in table 2. Heart rate was higher (72 ± 7 bpm) before anesthesia induction in patients in whom bradycardia and a hypotensive response later developed compared with patients who remained normotensive after anesthesia induction (60 ± 10 bpm; $P = 0.004$). Baseline awake heart rate and heart rate after anesthesia induction were no different in patients with or without myocardial ischemia with TEAP. There were no other differences observed in the baseline hemodynamic measurements between patients with and without hypotension or between patients with and without myocardial ischemia. After anesthesia induction and tracheal intubation, systolic, diastolic, and mean systemic and systolic pulmonary arterial pressures and cardiac output were all significantly lower than baseline awake measurements in patients in the hypotensive group. Compared with baseline awake measurements, there were no significant differences in any of the hemodynamic measurements after anesthesia induction in patients without hypotension.

Institution of TEAP resulted in increases in the hemodynamic parameters compared with measurements obtained immediately after anesthesia induction, although most of the measurements remained significantly less than baseline measurements. Systemic diastolic blood pressure and cardiac output, however, were restored to values similar to those measured before anesthesia induction at TEAP rates of 65 bpm and 80 bpm, respectively. Compared with awake values, stroke volume was no different after anesthesia induction or with TEAP rates of 65, 70, and 80 bpm, but was significantly less than awake baseline at a TEAP rate of 90 bpm. Compared with baseline awake values, there were no significant changes observed in pulmonary diastolic arterial pressure, central venous pressure, pulmonary capillary wedge pressure, mixed venous oxygen saturation, or vascular resistances after anesthesia induction or with TEAP in the hypotensive group. Decreases in ESA and EDA with each successive paced heart rate in the hypotensive group were not significantly different from measurements after anesthesia induction. There also was no difference in FAC at each TEAP rate.

Results of 12-lead ECG and TEE monitoring for myocardial ischemia at each TEAP rate are shown in table 3. No episodes of myocardial ischemia were observed after anesthesia induction. In 12 (30%) patients, evidence of myocardial ischemia during TEAP developed. Myocardial ischemia was not observed at TEAP rates of 65 and 70 bpm. Seven patients had myocardial ischemia at a TEAP rate of 80 bpm, with 5 of these patients having the ischemic episode diagnosed on the basis of TEE criteria and 2 patients based on both ECG and TEE criteria. Five patients had new evidence of myocardial ischemia at a TEAP rate of 90 bpm, whereas myocardial ischemia detected at a TEAP rate of 80 bpm persisted at a TEAP rate of 90 bpm in 5 additional patients. In 2 patients, the study protocol was aborted at a TEAP rate of 80 bpm, because of the clinical diagnosis of myocardial ischemia (confirmed by off-line analysis). Myocardial ischemia was detected off-line in 5 other patients at a TEAP rate of 80 bpm, but in these patients, the ischemic episodes were not detected clinically and, therefore, the pacing protocol continued to a TEAP rate of 90 bpm. In patients who developed myocardial

Table 2. Hemodynamic and Echo
Patients with Bradycardia and H

	Awak
Heart rate (beats/min)	72 ±
Systolic BP (mmHg)	156 ±
Diastolic BP (mmHg)	70 ±
Mean arterial pressure (mmHg)	102 ±
Systolic PAP (mmHg)	43 ±
Diastolic PAP (mmHg)	14 ±
CVP (mmHg)	13 ±
PCWP (mmHg)	15 ±
SV (ml/beat)	65 ±
CO (l/min)	5 ±
PVR (dyne·s·cm^{-5})	230 ±
SVR (dyne·s·cm^{-5})	1,442 ±
Sv_{O_2} (%)	80 ±
EDA (cm^2)	-
ESA (cm^2)	-
FAC	-

TEAP = transesophageal atrial pacing;

* $P < 0.05$ *versus* Awake.

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Table 3. Number of Patients
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Both ECG and TEE Criteria at

TEAP Rate (beats/min)	n	EC
65	0	
70	0	
80	7	
90	10	

ECG = electrocardiography; TEE = t
= transesophageal atrial pacing.

Table 2. Hemodynamic and Echocardiographic Results at Baseline, after Anesthesia Induction, and at Each TEAP Rate for Patients with Bradycardia and Hypotension

	Awake	Anesthesia	65 beats/min	70 beats/min	80 beats/min	90 beats/min
Heart rate (beats/min)	72 ± 7	59 ± 6*	—	—	—	
Systolic BP (mmHg)	156 ± 17	107 ± 14*	110 ± 12*	113 ± 14*	119 ± 14*	121 ± 17*
Diastolic BP (mmHg)	70 ± 10	51 ± 3*	57 ± 6	58 ± 8	60 ± 9	62 ± 11
Mean arterial pressure (mmHg)	102 ± 11	69 ± 5*	76 ± 8*	77 ± 11*	81 ± 11*	83 ± 14*
Systolic PAP (mmHg)	43 ± 8	30 ± 5*	30.7 ± 5*	28 ± 6*	28 ± 5*	30 ± 5*
Diastolic PAP (mmHg)	14 ± 3	16 ± 3	16 ± 3	15 ± 5	16 ± 4	17 ± 4
CVP (mmHg)	13 ± 5	11 ± 4	12 ± 3	10 ± 3	11.4	10 ± 4
PCWP (mmHg)	15 ± 6	11 ± 4	12 ± 4	11 ± 5	11 ± 4	13 ± 4
SV (ml/beat)	65 ± 17	59 ± 11	57 ± 9	54 ± 9	52 ± 9	45 ± 7*
CO (l/min)	5 ± 1.1	3 ± 0.5*	4 ± 0.5*	4 ± 0.6*	4 ± 0.7	4 ± 0.5
PVR (dyne · s · cm^{-5})	230 ± 70	219 ± 34	213 ± 60	192 ± 64	210 ± 74	192 ± 55
SVR (dyne · s · cm^{-5})	1,442 ± 341	1,373 ± 228	1,449 ± 281	1,418 ± 296	1,357 ± 244	1,521 ± 150
Sv_{O_2} (%)	80 ± 5	81 ± 3	81 ± 4	81 ± 5	83 ± 4	85 ± 3
EDA (cm^2)	—	17 ± 4	16 ± 5	15 ± 5	14 ± 4	14 ± 5
ESA (cm^2)	—	8 ± 2	8 ± 3	7 ± 3	7 ± 2	7 ± 3
FAC	—	0.54 ± 0.07	0.51 ± 0.08	0.54 ± 0.08	0.52 ± 0.08	0.54 ± 0.08

TEAP = transesophageal atrial pacing; MAP = mean arterial pressure; PAP = pulmonary artery pressure; BP = blood pressure.

* $P < 0.05$ *versus* Awake.

ischemia at a TEAP rate of 90 bpm, the diagnosis of the ischemic episode was based only on ECG criteria in 2 patients, on TEE criteria in 5 patients, and on both TEE and ECG criteria in 3 patients. Myocardial ischemia during TEAP occurred in 7 patients without hypotension after anesthesia induction and in 5 patients from the hypotensive group (P = n.s.). There were no difference in any of the hemodynamic parameters, including cardiac filling pressures during myocardial ischemia compared with measurements from patients without ischemia at the same TEAP rate. Evidence of myocardial ischemia resolved after several minutes in those patients in whom the ischemic episodes were detected clinically.

Table 3. Number of Patients Experiencing Myocardial Ischemia Based on ECG Criteria Only, TEE Criteria Only, or Both ECG and TEE Criteria at Each TEAP Rate

TEAP Rate (beats/min)	n	ECG Only	TEE Only	TEE and ECG
65	0	0	0	0
70	0	0	0	0
80	7	0	5	2
90	10	2	5	3

ECG = electrocardiography; TEE = transesophageal echocardiography; TEAP = transesophageal atrial pacing.

Discussion

A major concern with the use of TEAP in patients with coronary artery disease is that of precipitating myocardial ischemia with increased heart rates.[1–7] In studies of patients undergoing CABG surgery, researchers reported no evidence of myocardial ischemia with TEAP at the heart rates used.[13,14,17] In these reports, however, monitoring for myocardial ischemia was limited to 7-lead ECG monitoring that included lead V_5. The detection of myocardial ischemia perioperatively can be increased with the use of more ECG leads and by monitoring of regional myocardial function with TEE.[19–21] In contrast to previous studies, when using these more sensitive monitoring methods, myocardial ischemia was observed in the current study in 30% of patients during TEAP, occurring at heart rates of 80 and 90 bpm in 7 and 5 patients, respectively. To focus on the effects of TEAP on myocardial oxygen balance, we attempted to limit the number of potential variables that could contribute to the development of myocardial ischemia. Therefore, TEAP was performed in the absence of surgical stimulation and at a time when systolic arterial blood pressure, left ventricular dimension, and pulmonary capillary wedge pressure were all similar to those measured before TEAP, suggesting that the only variable increasing myocardial oxygen consumption

was faster heart rate. The presence of myocardial ischemia in 5 patients using both 12-lead ECG and TEE criteria also suggests that, although the number of myocardial ischemic episodes may decrease with stricter diagnostic criteria (*i.e.,* change in regional wall motion score > 1), the observations that myocardial ischemia may occur with TEAP would appear to persist.

The development of myocardial ischemia is complex, and depends on a dynamic balance of multiple variables affecting the myocardial oxygen supply/demand ratio. In this study, no episodes of myocardial ischemia were observed at TEAP rates of 65 and 70 bpm. We cannot exclude the possibility that the observation period in this study after each increase in heart rate (3–5 min) was inadequate and that myocardial ischemia could have occurred at TEAP rates < 80 bpm had we maintained TEAP at these rates longer. Laboratory and clinical investigations indicate, however, that in the presence of restriction to coronary artery blood flow, myocardial ischemia will become evident in 2–3 min when oxygen consumption is suddenly increased.[1,2,7,24–26] In subjects with coronary artery disease, increases in coronary blood flow in response to pacing-induced increases in heart rate and myocardial oxygen consumption also were shown to reach a steady level within 6 s, suggesting that the new level of myocardial oxygen/demand is achieved quickly.[1] Regardless, most spontaneous myocardial ischemic episodes before CABG surgery occur without acute increases in heart rate and appear more dependent on myocardial oxygen supply than on increased demand.[21,27,28] Therefore, because of these dynamic myocardial oxygen supply/demand characteristics, maintenance of TEAP at any rate, even <80 bpm, in this population of patients does not preclude the development of myocardial ischemia.

Hemodynamic improvements with TEAP in CABG patients with bradycardia and systemic hypotension was reported.[13,14,17] Backofen *et al.*[13] showed increases in mean and diastolic systemic arterial pressures from 66 ± 11 to 78 ± 11 mmHg and 47 ± 7 to 59 ± 8 mmHg, respectively, with TEAP rates of 68 and 78 bpm in patients who receive large doses of fentanyl for CABG surgery. Our results confirm these findings and those from other investigations in which researchers observed significant increases in systolic, mean, and diastolic systemic arterial pressures with TEAP in patients undergoing coronary artery surgery.[14,17] Similar to these other studies, increases in cardiac output, albeit modest, also were observed with TEAP. We also showed that coronary artery perfusion pressure can be restored to a level similar to that measured in the awake state with a TEAP rate as low as 65 bpm. This study also extends prior reports by documenting the TEE effects of incremental TEAP.

Experimentally, chronotropic-associated improvements in cardiac output reach a maximum above which further increases in heart rate will no longer increase cardiac output.[3] In these canine experiments, the plateau of heart rate-related increases in cardiac output occur between 90 and 180 bpm.[3] In patients without myocardial ischemia during diagnostic stress testing, atrial pacing at heart rates between 107 ± 12 bpm and 132 ± 15 bpm also were shown to result in no change in cardiac output, but to progressively decrease stroke volume.[29] Improvements in cardiac output with TEAP in this study were related to the increase in heart rate because stroke volume and actually decreased at a TEAP rate of 90 bpm (table 2). Taken together, these results suggest not only that the risk of myocardial ischemia increases with incremental increases in TEAP rates, but that the hemodynamic benefits of higher heart rates may be limited in this population of patients.

As previously noted, limitations exist with the use of both ECG and TEE to assess for myocardial ischemia, including that of intermittent and noncontinuous sampling.[21,30,31] The use of biplane TEE in the current study enabled us to obtain transverse and longitudinal images of the LV, thereby increasing the myocardial segments evaluated.[32] However, it is possible that myocardial ischemia may have occurred in myocardial segments not assessed by two-dimensional imaging. Changing loading conditions of the heart secondary to faster heart rates and tethering effects from areas of prior ischemic damage of the myocardium also could have unmasked areas of regional LV contractile dysfunction that could be misinterpreted as resulting from myocardial ischemia.[30,31] Despite these limitations, 12-lead ECG monitoring was shown to have a sensitivity rate of approximately 67% in the detection of myocardial ischemia during diagnostic TEAP stress testing.[26] Under similar conditions, TEE was shown to have sensitivity and specificity rates of 90–93% and 87–100%, respectively, in the detection of myocardial ischemia, and to be highly concordant with scintigraphic findings of myocardial ischemia.[24] In addition to the above considerations, other limitations of this study include the short pacing interval used and the failure to assess for myocardial injury with postoperative CK-MB enzymes, troponin I, or serial ECGs. Our goal, in the current study, was to evaluate for the risk of developing myocardial

ischemia with TEAP. Whereas serial ECGs and myocardial enzyme information may have been interesting in light of our findings, evaluating whether the use of TEAP in patients with coronary artery disease can contribute to myocardial injury would require a larger number of patients to achieve statistical power.

Based on our data, increasing the heart rate with TEAP restores hemodynamics in anesthetized patients in whom bradycardia and hypotension develop before CABG surgery. In patients with severe coronary artery disease, these results suggest that TEAP rate should be titrated to the lowest rate at which optimal hemodynamic improvements are achieved, while closely monitoring for myocardial ischemia, especially at rates >80 bpm.

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Mivacurium Ne Muscles of the in Humans

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Background: **Laryngeal muscle cheal intubation. Time to peak and intensity of blockade less a with the adductor pollicis. The a to determine the neuromuscular laryngeal adductor muscles and**

Methods: **In 22 adults, anesth tained with propofol and alfent of the adductor pollicis as re sponse was evaluated by meas the cuff of a tracheal tube positi after train-of-four stimulation. 0.14 mg·kg^{-1}) was given intrav**

Results: **With 0.07 mg·kg^{-1} m ± 40 s (mean ± SD) at the larynx pollicis, respectively (P < 0.00 18% and 95 ± 8%, respectively recovery was 11.1 ± 2.9 min ar (P < 0.001). With 0.14 mg·kg^{-1} was more rapid at the local c adductor pollicis (201 ± 59 s, 90 ± 7% and 99 ± 1% (P < 0.0 was 16.4 ± 4.9 min and 28.4 ± 7**

Conclusions: **With mivacurium at the laryngeal muscles but b**

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