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ST Segment Depression during Labor and Delivery

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ECG changes suggestive of myocardial ischemia are common during cesarean delivery under regional anesthesia. To determine the time course, duration, and significance of these ECG changes, we monitored 111 parturients with continuous ambulatory ECG (Holter) during and after cesarean delivery. Twenty-two parturients undergoing vaginal delivery were similarly monitored. ST segment depression was present in 25% of patients undergoing cesarean delivery but was not found in those patients delivering vaginally. ST segment elevation was not detected in either group. The incidence of ST segment depression during cesarean delivery was similar with epidural (29%), spinal (17%), and general (18%) anesthesia, occurring most commonly in the 30 min following delivery ($P < 0.001$). Transthoracic echocardiographic imaging was performed in 23 patients undergoing cesarean section. Five of the 23 patients had seven episodes of intraoperative ST segment depression. Regional wall motion abnormalities were not present in any patient. A decrease in ejection fraction area greater than 15% from baseline or from previous interval ejection fraction area was present during four episodes of ST change. Three episodes of ST depression were not associated with significant decreases in ejection fraction area. Precordial Doppler monitoring for detection of venous air embolism in 25 patients revealed no association between the occurrence of venous air embolism and ST segment depression. We conclude that although significant myocardial impairment during cesarean delivery does not occur, episodes of ST depression may not all be merely an artifact of parturition. (Key words: Anesthesia, obstetric: cesarean delivery. Heart: ischemia. Monitoring: Doppler; echocardiography; electrocardiography.)

ISOLATED ST segment abnormalities are common during cesarean delivery.[1,2] Symptoms of chest pain and dyspnea associated with these electrocardiographic (ECG) changes have led to the speculation that myocardial ischemia occurs at this time. Prior study[1] of the ECG changes has been limited by intermittent sampling of the ECG at preselected time points during the course of cesarean delivery. Defining significant ECG changes as changes in heart rate, rhythm, and conduction also resulted in an extraordinarily high (47%) incidence of abnormalities in otherwise healthy women. Furthermore, ECG changes not always indicative of ischemia—the occurrence of premature ventricular contractions and ST segment changes lasting less than 60 s—were chosen to represent myocardial ischemia. The time course, duration, and significance of the ECG changes remained ill-defined.[1]

In the current study, with the aid of continuous ambulatory ECG (AECG) monitoring, we prospectively examined the prevalence and duration of ECG changes during labor and delivery. Transthoracic echocardiography also was used to determine if echocardiographic findings suggestive of myocardial ischemia or dysfunction were present during periods of ECG change. Furthermore, precordial Doppler monitoring was used to determine the relationship between the occurrence of venous air embolism (VAE) and ECG change.

Materials and Methods

Following approval from the Human Investigation Committee of the Yale University School of Medicine, 133 consecutive (based on equipment availability), healthy (ASA physical status 1 and 2) patients were monitored during labor and delivery using a calibrated amplitude-modulated AECG monitor (SpaceLabs AECG model 90205, Redmond, WA). In the 111 patients who underwent elective cesarean delivery, modified bipolar leads V3 and V5 were monitored from immediately before induction of anesthesia through the early postoperative period. Twenty-two laboring parturients were similarly monitored from varying stages of cervical dilation through stage three of labor. The AECG recordings were analyzed on a SpaceLabs FT2000 computerized analysis system. ST segment and heart rate trends were plotted. All significant changes observed in the trend plots were printed at 25 mm/s, and the ECG strips were independently reviewed by two investigators. Differences were resolved by consensus. Significant ECG changes were defined as either $\geq$0.1 mV horizontal or downsloping ST segment depression or $\geq$0.2 mV ST segment elevation in 1 lead measured at 60 ms after the J-point and persisting for at least 1 min. A 12-lead ECG was obtained on postoperative day 1 in the first 55 (when available) cesarean delivery patients.

Of the 76 parturients undergoing cesarean delivery under epidural anesthesia, 23 were also monitored with transthoracic echocardiography using the Hewlett-Packard Sonos 500 Ultrasound System (Andover, MA) and a 3.5-MHz phased array transducer. Baseline parasternal long axis and midpapillary short axis views were obtained

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immediately before induction of anesthesia. Parasternal short-axis views were then recorded continuously on one-half–inch videotape from induction until surgical skin closure. The echocardiographic images were analyzed by an investigator as well as by a reviewer blinded to the clinical course of the patient. Wall motion analysis consisted first of a division of the short-axis view into four segments (anterior, lateral, posterior, and septal). Each segment was analyzed visually for abnormalities in wall thickening and systolic endocardial excursion and assigned a numerical grade: 0 = normal wall motion; 1 = hypokinesia; 2 = akinesia; and 3 = dyskinesia. Video images were then digitized and analyzed on a Franklin Quantitative Analysis System using an edge detection algorithm.

Ejection fraction areas (EFA; end-diastolic area − end-systolic area]/end-diastolic area) were calculated for each patient at baseline and at six intervals: 1) induction to skin incision; 2) skin incision to uterine incision; 3) uterine incision to delivery; 4) delivery to uterine closure; 5) uterine closure to skin closure; and 6) skin closure to transport to the recovery room. When multiple measurements were made for any given interval, an average of all EFA values for that interval was calculated. A decrease in EFA of greater than 15% from baseline was considered significant.

To determine the relationship between the ECG changes and VAE during cesarean delivery, 25 of the remaining 88 cesarean section patients were monitored with a precordial Doppler (Medasonics Versatone D8, Mountainview, CA). A 2.4-MHz transducer was placed parasternally over the right fourth intercostal space before skin incision. Appropriate placement of the precordial Doppler transducer was confirmed with intravenous agitated saline injections at the start and the end of surgery. An air trap (Ivex-RF, 0.8-μm filter; Abbott Medical Products, North Chicago, IL) was placed proximal to the intravenous angiocatheter to decrease the incidence of iatrogenically introduced air. The audible Doppler signal was monitored during the cesarean section by an investigator and was recorded onto standard audio cassette tape for analysis by a reviewer blinded to the operative events and unimpeded by operating room noise. A positive Doppler change was defined as the occurrence of a change in the rhythmic sound of the normal precordial Doppler signal resulting in a signal similar to that produced during injection of agitated saline. A significant decrease in hemoglobin oxygen saturation (Sp_{O_2}) (Ohmeda 3700 Biox pulse oximeter) was defined as a decrease to less than 95% or to 3 percentage points less than the patient's baseline while breathing room air.

Anesthetic technique for a given patient was determined by the anesthesia care team. Patients having regional anesthesia for cesarean delivery received at least 1 l of dextrose-free crystalloid solution before induction of anesthesia. Lumbar epidural anesthesia was performed using a 17-G Weiss needle and a 19-G Teflon catheter. Excellent anesthetic and analgesic levels (sensory level T3–T5) for cesarean delivery were achieved using incremental doses of 2% lidocaine with 1:200,000 epinephrine and 0.1 mEq/ml sodium bicarbonate. Spinal anesthesia was performed with a 25- or 26-G spinal needle using hyperbaric 0.75% bupivacaine. Intrathecal or epidural fentanyl was added at the discretion of the anesthesia care team. Supplemental oxygen was provided by face mask until delivery.

Parturients receiving general anesthesia breathed 100% oxygen by face mask and underwent a rapid-sequence induction with cricoid pressure using thiopental and/or ketamine and succinylcholine. Before delivery of the fetus, anesthesia was maintained with 50% nitrous oxide and enflurane or isoflurane. Intravenous diazepam and fentanyl were administered after delivery. Left uterine displacement was maintained throughout the operative procedure in all parturients. Blood pressure was monitored in all patients using the SpaceLabs 90305 patient monitor. Hypotension was defined as a systolic blood pressure less than 100 mmHg. Hypertension was defined as a systolic blood pressure greater than 120% of baseline.

Statistical analysis was performed using chi square (anesthetic technique, incidence of hypotension, incidence of ST depression during vaginal delivery), Fisher's exact test (VAE), chi square goodness of fit test (incidence of ST depression after cesarean delivery), repeated-measures ANOVA (EFA, end-diastolic area), and the t test for independent samples (patient characteristics) with $P < 0.05$ considered significant.

Results

Demographics and ECG characteristics for the patients monitored with continuous AECG during cesarean and vaginal delivery are provided in tables 1 and 2. ST segment elevation did not occur in any patient. Significant ST segment depression was present in 25% (28 of 111) of all patients undergoing cesarean delivery. The incidence of ST segment depression was similar during spinal (17%, 3 of 18), general (18%, 3 of 17), or epidural anesthesia (29%, 22 of 76) (P = not significant). ST segment depression was most common in the 30 min after delivery ($P < 0.001$) (fig. 1) and was immediately preceded by hypotension in only 4 of 36 episodes of ST change (fig. 2). In the 55 patients (including 17 of 28 AECG-positive) who had postoperative ECGs, no significant abnormalities were seen.

None (0 of 22) of the parturients monitored during vaginal delivery had significant ST segment depression ($P = 0.004$). Eleven patients received epidural anesthesia; in 9 (9 of 11) of these patients, AECG monitoring was begun after induction of epidural anesthesia.

Five of the 23 patients (22%) monitored with trans-

TABLE 1. Patient Characteristics for AECG Patients

	Cesarean Section						Labor		MVP	
	Epidural		Spinal		General					
ST Segment Depression	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent
N	22	54	3	15	3	14	0	2	2	9
Age (yr)	31.6 (4.3)	30.4 (5.1)	28.7 (4.2)	31.6 (6.1)	27.0 (13.9)	29.1 (5.2)	NAP	27.7 (5.8)	37.5 (7.8)	32.4 (2.8)
Baseline heart rate (beats/min)	85 (11.5)	87 (13.7)	79 (16.8)	81 (12.4)	77 (8.3)	92* (8.6)	NAP	82 (12.4)	70 (14.4)	83 (12.4)
Baseline systolic blood pressure (mmHg)	116 (11.6)	121 (14.5)	121 (11.4)	119 (11.7)	115 (5.0)	120 (11.2)	NAP	112 (11.9)	109 (1.4)	112 (6.8)
Baseline diastolic blood pressure (mmHg)	77 (7.9)	73 (9.9)	78 (14.4)	74 (8.0)	76 (6.9)	72 (9.1)	NAP	69 (9.2)	71 (12.7)	75 (9.5)
Maximum heart rate (beats/min) achieved in OR	142 (18.4)	133 (20.4)	142 (16.0)	125 (13.9)	125 (4.2)	139 (17.8)	NAP	148 (21.0)	124 (2.3)	131 (7.2)
Preoperative hematocrit (%)	36 (3.1)	36 (4.0)	34 (5.1)	37 (3.9)	33 (2.0)	36 (4.3)	—	—	—	—
Postoperative hematocrit (%)	33 (2.6)	33 (4.7)	28 (7.0)	33 (3.9)	29 (2.0)	32 (4.8)	—	—	—	—
Hypotension (%)	55	44	100	80	0	7	—	—	—	—
Ephedrine use (mg)	20.8 (9.5)	16.5 (8.9)	25.0 (8.7)	18.8 (11.3)	NAP	10.0 (0)	—	—	—	—
Chest pain (number)	4	1	0	0	NAP	NAP	—	—	—	—

Standard deviations appear in parentheses.
NAP = not applicable; MVP = mitral valve prolapse.

* $P < 0.05$.

TABLE 2. AECG Characteristics of Monitored Patients

	Cesarean Section			
	Epidural	Spinal	General	Labor
N	76	18	17	22
ST segment depression (% of patients)	22 (29)	3 (17)	3 (18)	0 (0)
Mean duration of monitoring (min) (range)	719.6 (140–2567)	389.4 (158–1473)	580.9 (131–1510)	270.3 (55–1134)
Mean duration of ST depression (min) (range)	14.0 (1–57)	8.5 (2.5–18.5)	2.0 (1–4)	—
Mean heart rate at the onset of ST depression (beats/min) (range)	116 (93–149)	127 (104–148)	119 (115–125)	—
No. of patients with multiple episodes of ST depression	6	2	0	—
Episodes detected in lead V3 only	4	3	1	—
Episodes detected in lead V5 only	11	2	1	—
Episodes detected in both leads V3 and V5	13	0	1	—

thoracic echocardiography had 7 episodes of intraoperative ST segment depression. Regional wall motion abnormalities were not found in any patient (grade = 0). There were also no differences, during any interval of cesarean delivery, in end-diastolic areas and EFA between the patients with and without ST depression (P = not significant).

A decrease in EFA greater than 15% from baseline was present during three episodes (one with anginal chest pain) of ST segment depression. One episode of ST change was associated with a greater than 15% decrease in EFA from that of the previous interval but not from baseline. Three episodes of ST segment depression had no associated significant decrease in EFA. No episode of EFA decrease was associated with hypertension. Intraobserver variability in the calculation of EFA was 5.6%, and interobserver variability was 6.8%.

Changes compatible with VAE were detected in 6 of the 25 (24%) patients monitored with a precordial Doppler (fig. 3). No patient (0 of 25) had both VAE and significant ECG changes. ECG change was present in 2 patients without VAE (P = not significant). One of 6 patients with VAE complained of chest pain. No (0 of 25) patient complained of dyspnea. VAE was not followed by hypotension in any patient. Significant decreases in Sp_{O_2} were seen in 2 patients with VAE and in 8 patients without VAE. The decrease in Sp_{O_2} followed the administration of fentanyl or diazepam in six of these 10 patients.

Eight percent of monitored patients (11 of 133) provided a history of mitral valve prolapse (MVP) in the predelivery evaluation. Significant ECG changes were present in 18% (2 of 11) and occurred before induction in one patient and after delivery in the second patient. Patient characteristics for the MVP group are given in table 1.

Finally, transthoracic echocardiographic imaging revealed probable amniotic fluid embolism in one patient. However, ST segment depression was not present in that patient.

Discussion

ECG changes are common during cesarean delivery. Because of the similarities between angina pectoris and the symptoms reported by patients undergoing cesarean delivery under regional anesthesia, it has been suggested that these ECG changes may represent myocardial ischemia.[1] In an attempt to clarify the time course, duration, and significance of these ECG changes, we used transthoracic echocardiography and continuously monitored bipolar leads V3 and V5 with an AECG monitor. Leads I, aVL, and V5 had been previously described as the most sensitive for detecting ECG changes,[1] yet we chose bipolar leads V3 and V5 because they demonstrated a similar sensitivity to a 12-lead system for detecting ischemia during exercise in patients with coronary artery disease.[3]

A variety of theories have been developed to explain the potential for myocardial ischemia during cesarean de-

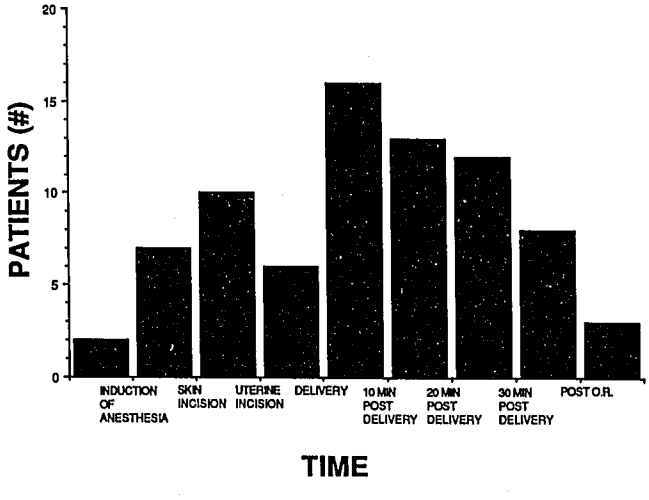

FIG. 1. Incidence of ST segment depression during cesarean delivery. ST-segment depression was most common in the 30 min after delivery.

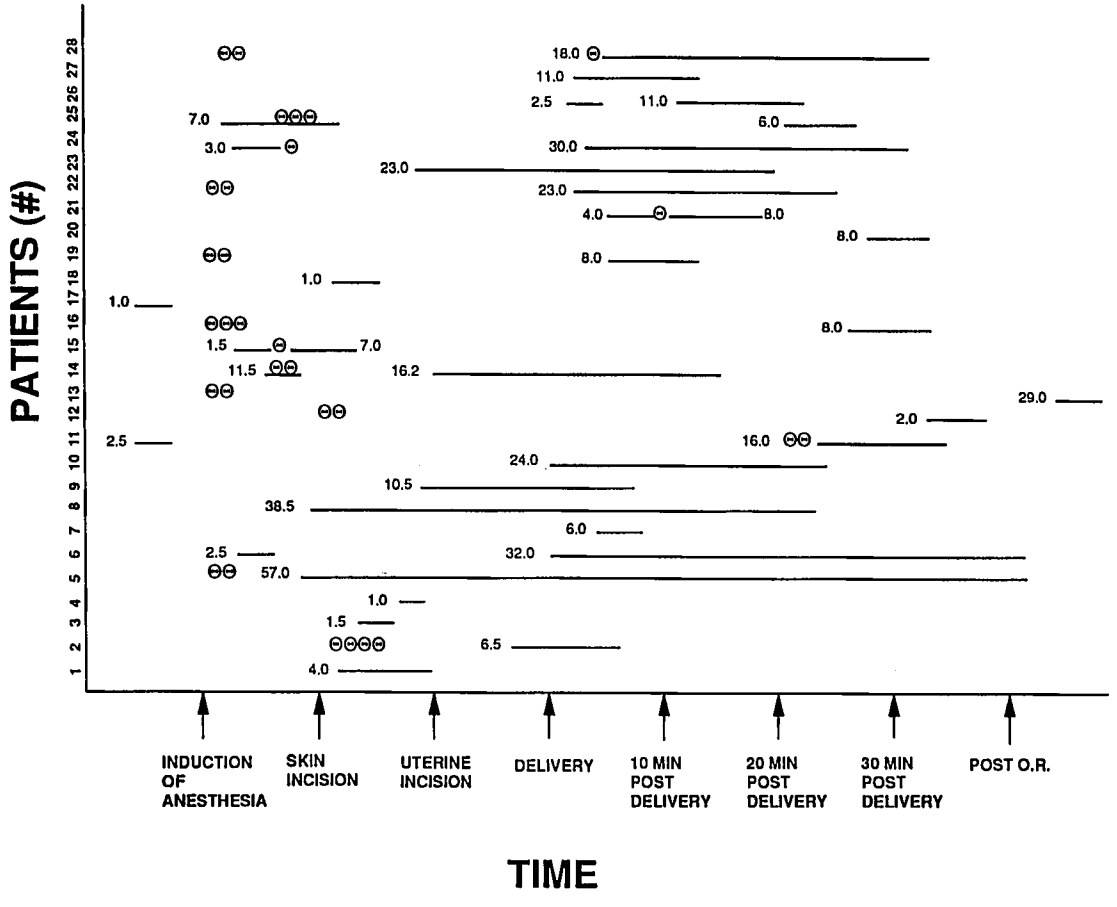

FIG. 2. Timing and duration of ST segment depression during cesarean delivery. The numbers are the duration of ECG change in minutes. Θ = the occurrence of hypotension. ST segment depression was immediately preceded by hypotension in only four episodes.

livery. Acute hypervolemia from overhydration and uterine contraction–related autotransfusion in combination with a local anesthetic–induced sympathetic block has been speculated to produce increases in left ventricular end-diastolic volume and therefore increases in myocardial oxygen demand.[1] In our study, end-diastolic area—an estimate of ventricular volume—was similar in all patients during all intervals of cesarean delivery. ST segment depression also occurred under general anesthesia, during which sympathectomy does not occur and prehydration of the patient is limited to smaller volumes. This suggests that increases in left ventricular end-diastolic volume are not responsible for ST segment changes during cesarean delivery.

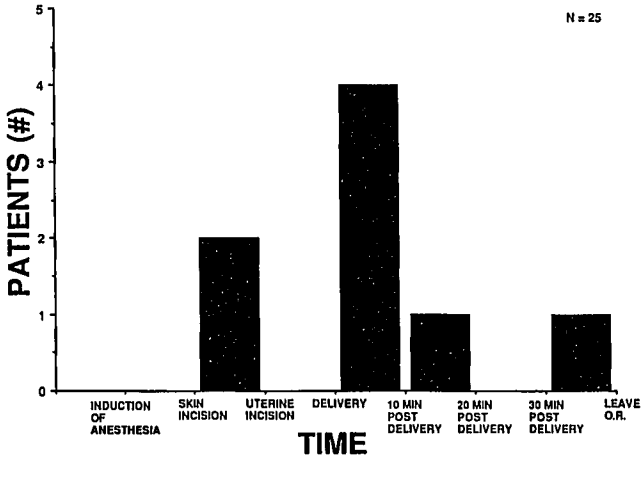

FIG. 3. Incidence of venous air embolism (VAE) during cesarean delivery. Two patients had two episodes of VAE.

The use of a vasopressor such as ephedrine to treat hypotension has also been hypothesized to increase myocardial oxygen demand.[1] This study demonstrates that while hypotension and the subsequent use of ephedrine was most frequent in the period after induction of anesthesia, there was no relationship between the occurrence of hypotension and ST segment change (fig. 2). ST segment depression was immediately preceded by the administration of ephedrine in only 4 of 36 episodes. Furthermore, the incidence of hypotension was similar in those with and without ST change.

VAE have long been held to be a cause of ECG change during cesarean delivery.[4] The low incidence of VAE in the current study may have been related to our use of an

air trap proximal to the patient's intravenous angiocatheter and our efforts to limit intraoperative interference of analysis by recording the Doppler sounds for delayed as well as repeated analysis. Although the incidence of VAE (24%) in our study was less than previously reported,[5] there was no relationship between the occurrence of VAE and ST segment depression (P = not significant). In fact, no patient had both VAE and ECG changes. Furthermore, decreases in Sp_{O_2} seen during cesarean delivery may have been related to the administration of sedatives. Although we cannot discount the possibility that VAE do produce symptoms of dyspnea and chest pain, it is unlikely that they are responsible for the ECG changes.[6] VAE remains a potentially devastating complication during cesarean delivery.[7] The frequent occurrence of VAE without significant hemodynamic complications, however, suggests that the majority of detectable episodes have limited clinical significance.

MVP has also been reported to produce ECG changes.[8] Patients with MVP who exhibit exercise intolerance and have normal coronary arteries may have ST segment depression when exercised.[9] The physiology of clinically silent MVP, prolapse demonstrated only by echocardiograpy, is such that symptoms are present only in a volume-underloaded ventricle. A marked decrease in the degree of MVP during pregnancy possibly related to the increase in blood volume has also been described.[10] In our study, ST segment depression was present in only 18% (2 of 11) of the patients with a history of MVP.

Other potential causes of ECG changes are hyperventilation; translational movement of the heart and hormonal changes after delivery; body position; and electrolyte disturbances. However, the absence of significant ECG changes during labor and vaginal delivery suggests that these are not significant etiologic factors. Coronary vasospasm is also unlikely in our study because ST elevation was not found.

In the parturient, the myocardial effects of amniotic fluid embolism are yet to be defined.[11–13] In our study, pulmonary embolism of amniotic fluid debris was detected by transthoracic echocardiography in one patient. ECG change, however, was not present in that patient.

It is unlikely that tachycardia alone is responsible for the ECG changes seen during cesarean delivery.[2] ST segment changes have been produced by sudden strenuous exercise in otherwise healthy subjects.[14,15] ST depression was not present, however, when the same heart rate was achieved by increments of exercise. Similarly, in the present study, a heart rate higher than or equal to that present at the onset of ST segment depression was recorded without concomitant ST depression in 78% of the AECG-positive patients. It is possible that the increases in cardiac output after fetal delivery, a period when ST depression was most common, represents sudden strenuous exercise to the parturient.

Can the increased myocardial oxygen demand produced by parturition limit the vasodilator reserve of the parturient? A reduction in coronary vasodilator reserve can lead to angina in patients with normal coronary arteries and has been termed "Syndrome X." That myocardial ischemia occurs in patients with Syndrome X is suggested by the decrease in left ventricular peak filling velocity present during ST segment depression. Notably, wall motion abnormalities were not present during periods of ischemia.[16]

The diagnostic and prognostic value of exercise-induced ST segment changes in women have been extensively studied. In symptom-free women and those with atypical chest pain, exercise-induced ST segment depression has proven to be a poor predictor of future coronary events.[17] False positive ST segment changes also have been reported with AECG recordings. However, in one study of the AECG in a normal population, five of eight normal patients with ST segment change demonstrated a functional impairment on radionuclide ventriculography.[18] Thallium imaging, a technique with greater diagnostic and prognostic sensitivity for coronary artery disease, was normal in all five of these patients. In our population of relatively healthy women, it is clear that coronary artery disease is not the issue at hand. Rather, a functional impairment produced by pregnancy may be present. The decreases in EFA seen in this study may be suggestive of myocardial dysfunction. EFA has been shown to correlate well with ejection fraction obtained by scintigraphy ($r = 0.82$).[19] A larger study population may have revealed more definitive patterns of EFA change.

The absence of regional wall motion abnormalities in our study does not, however, preclude the occurrence of subendocardial ischemia. A comparison of two-dimensional echocardiographic recognition of myocardial injury with *post mortem* studies has revealed that normal wall motion is occasionally associated with subendocardial injury.[20] While the midpapillary short-axis view of the left ventricle represents areas of the myocardium supplied by the three major epicardial arteries, wall motion abnormalities produced by the involvement of peripheral arterioles may not be seen at this short-axis view. In a patient without coronary artery disease and with a normal left ventricle, imaging of multiple echocardiographic planes (apical, mitral valve, midpapillary) may have been more likely to detect subendocardial ischemia. Just as ischemic myocardium can mechanically impair normal adjacent tissue by a "tethering" effect, it is also possible that hyperkinetic myocardium, as seen in this study and by Palmer *et al.*,[21] can mask hypokinesia in an adjacent segment.[16]

Other limitations to the use of transthoracic echocardiography in our study includes the potential for a change in imaging planes produced by transducer movement. Haendchen *et al.*,[22] in a study of 50 dogs and 32 humans,

demonstrated no statistical difference when the EFA measured at the midpapillary muscle level was compared to either the high or low papillary muscle levels. The midpapillary short-axis view was chosen in the current study because slight changes in transducer location are readily reflected as changes in papillary muscle morphology. Patients were excluded from our echocardiographic study if wide variations in the imaging plane were present. Finally, in an effort to minimize the potential error related to transducer movement, greater than 15% decrease in EFA was defined as significant.

End-diastolic area, as measured in our study, also may not represent true ventricular volume because it is only a two-dimensional measure of volume. The correlation between transesophageally measured end-diastolic area and left ventricular volume measurement obtained by gated pool scintigraphy following coronary artery bypass graft surgery has been reported to be only fair ($r = 0.74$).[19] Careful studies of two-dimensional echocardiographic volume accuracy performed in an isolated heart preparation have demonstrated that the accuracy of two-dimensional measurements was significantly reduced when fewer than four cross-sectional views of the heart were used to assess left ventricular volume.[23] Nevertheless, routine hemodynamic parameters were insensitive measures of left ventricular volume when compared with visual estimates obtained by transesophageal echocardiography.[24]

In conclusion, ST segment depression is common during cesarean delivery and does not vary according to anesthetic technique but was notably absent in parturients delivering vaginally. Although echocardiographic evidence in this study suggests that there is no myocardial ischemia with functional impairment during cesarean delivery, it does appear that ST changes are not all merely an artifact of parturition.

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