Diagnosis of Myocardial Injury by Real-time Recording of ST Segments of the Electrocardiogram in a Patient Receiving General Anesthesia for Electroconvulsive Therapy

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ELECTROCONVULSIVE therapy (ECT) is associated with short-lasting but important changes in autonomic nervous system activity.1–3 The autonomic response to ECT is characterized by initial parasympathetic stimulation immediately followed by subsequent sympathetic stimulation. Typically, transient bradycardia is first seen immediately after the seizure, after which sinus tachycardia and arterial hypertension are observed, frequently associated with cardiac arrhythmias.4–6 Though these changes usually are self-limited, a hyperdynamic state in most elderly patients represents an important risk factor.7 Hypotension after ECT warrants an immediate search for a cause. In this case report, we present arterial hypotension immediately after ECT with subsequent refractory myocardial failure and subendocardial infarction. We made the initial diagnosis of myocardial injury because a tabulated record of real-time ST-segment data was available at the bedside, allowing immediate decisions about appropriate pharmacologic support measures. To validate the observations in this patient, we also present data on ST segments of the electrocardiogram (ECG) and hemodynamic variables recorded during the first treatment in a comparison group of ten patients with the admission diagnosis of depression who presented for ECT.

References

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Patients and Methods

Comparison Group
In each of ten patients (age range 54–83 yr), we recorded ST segments of the ECG, arterial blood pressure (BP), and heart rate (HR) immediately before and up to 20 min after application of ECT. Patients were included in this group if, during the first ECT, systolic BP exceeded 180 mmHg, diastolic BP exceeded 90 mmHg, or HR exceeded 100 beats/min. We monitored arterial BP by means of a noninvasive BP monitor (Dinamap, Critikon, Tampa, FL) set at 1-min intervals. Pregelled self-adhesive silver-silver chloride electrodes were placed on the upper extremities bilaterally; lower extremity leads were placed in the midaxillary line on the iliac crests; and the precordial lead was placed beneath the left breast as close as possible to the V5 position. Leads II and V5 were displayed continuously on a monitor-oscilloscope with built-in ST-segment interpretation software (series 7000, Marquette Electronics, Milwaukee, WI). The signal was calibrated at 10 mm for each 1 mV, with a frequency response of 0.01–100 Hz at −3 dB. The HR and ST segments of the ECG were analyzed at 10-s intervals in leads I, II, and V5. All BPs and HRs recorded by the noninvasive BP monitor and the HR and ST-segment data recorded by the ECG were collected by an IBM XT computer, located at the bedside, by use of a customized computer program forming an integrated patient record, in which all individual BP, HR, and ST-segment data were tabulated in order of their occurrence. The tabulated record was displayed continuously on the computer monitor and simultaneously printed on paper. After at least one control measurement of BP and several measurements of HR and ST segments, anesthesia was induced with sodium methohexital and succinylcholine (both approximately 0.75 mg/kg), and the lungs were ventilated with oxygen via face mask. Taking the absence of the foot sole reflex as evidence of adequate muscular relaxation, a unilateral brief pulse stimulus was applied. Manual ventilation of the lungs was continued until spontaneous ventilation had resumed. Data were collected at 1, 3, 5, 10, and 20 min after application of the electroconvulsive stimulus (fig. 1).

Case Report
A 79-yr-old woman with a 6-month history of mental depression and with malnutrition was admitted to be considered for ECT. Concurrent medical diagnoses included insulin-dependent diabetes and glaucoma. In addition, severe coronary artery disease was present;

![Figure 1. Blood pressure, heart rate, and ST-segment values in a comparison group of 10 patients. Changes in blood pressure and heart rate were observed at 1 and 3 min; those in ST-segment variables were greatest in lead II at 1 min.](image-url)
three non-Q-wave myocardial infarctions had been documented in the preceding 12 yr. One month before the current admission, cardiac enlargement and pulmonary congestion were found by x-ray examination, but an acute myocardial infarction was ruled out on the basis of serial cardiac enzyme determinations. The ECG recorded 2 days before ECT showed sinus rhythm and nonspecific ST-segment and T-wave abnormalities in AVL and V6. Because of motor retardation, exercise tolerance could not be assessed readily, but the patient denied precordial discomfort and angina.

On the day of ECT, we followed the protocol outlined above. After control measurements of arterial BP (104/61 mmHg) and HR (84 beats/min) were obtained, anesthesia was induced with methohexital (30 mg), muscular relaxation was produced with succinylcholine.

Fig. 2. Histograms of heart rates and blood pressures recorded in the patient presented in the case report. These values were obtained simultaneously with the ST segments in figure 3. Anesthesia was induced after one control measurement (at 09:26:09); the electroconvulsive stimulus was applied at 09:28:29.

Fig. 3. Histogram of ST segments in lead I, II and V5 of the electrocardiogram in the patient presented in the case report. ST-segment elevation in lead V5 was reversible, but that in lead II was irreversible.

(30 mg), the lungs were ventilated with oxygen, and ECT was administered by a unilateral brief pulse stimulus of 72 Ws, which was followed by a well-modified generalized seizure lasting for 22 s. Because of low systolic BPs immediately after ECT, ephedrine (15 mg) was administered intravenously, but 2 min later, BP was unmeasurable. Further details on BPs, HRs, and ST segments are presented in the histograms (figs. 2 and 3) and in the results below. On
the oscilloscope, we noted sinus rhythm with occasional ectopic ventricular beats; the computer record showed marked ST-segment elevation in lead II. Norepinephrine infusion was begun. An endotracheal tube was placed for controlled ventilation; initial arterial blood gas and pH measurement revealed arterial oxygen tension 582 mmHg, arterial carbon dioxide tension 20 mmHg, and pH 7.45. Simultaneously measured electrolyte values were Na 136 mm, K 3.8 mm, and glucose 242 mg/dl. After placement of invasive monitoring lines, arterial pressure reappeared and values measured simultaneously via the arterial cannula and by noninvasive technique differed by no more than 10 mmHg. Initially, cardiac output was 1 l/min with a pulmonary artery occluded pressure of 19 mmHg. The 12-lead ECG showed T-wave inversion in the lateral leads, consistent with persistent inferior wall ischemia. Nitroglycerin infusion was added.

Creatine phosphokinase and creatine kinase MB band levels 3 h after the event were 483 U/l and 28.0 U/l (5.8%), respectively; creatine kinase MB band levels peaked with 34.0 U/l 17 h after the ischemic event.

On the day after ECT, a 12-lead ECG showed T-wave inversion in lateral leads, the absence of Q waves, and cardiac enzyme elevation; the presumptive diagnosis was inferolateral subendocardial infarction. A protracted low-flow state then developed with cardiac output values ranging from 1.8 to 2.4 l/min at left ventricular filling pressures ranging from 16 to 19 mmHg. On the 4th day, the patient died after refractory hypotension and bradycardia. Permission for a postmortem examination was not granted.

Results

Comparison Group

As shown in figure 1, peak increases in systolic and diastolic pressures occurred at 1 min (from 157 ± 9.3 to 210 ± 21.5 and from 75 ± 3.5 to 111 ± 12.6 mmHg, respectively) and in HR (from 81 ± 5 to 124 ± 10 beats/min). These values returned to pre-ECT values 5 min after the stimulus. Decreases in BP were not observed. Peak changes in ST segment values also occurred at 1 min; those in lead II were greatest but with a relatively wide range of values. Values recorded at 3 min and beyond were similar to those obtained before ECT.

Patient Presented in Case Report

As shown in figure 2, immediately before induction of anesthesia, arterial BP was 104/60 mmHg with HR of 62 beats/min. Thirty seconds after induction of anesthesia, these values were 99/55 mmHg and 70 beats/min, respectively. However, 60 s later (i.e., immediately before application of the stimulus), BP had decreased to 58/35 mmHg. At this time, three successive HR readings by the ECG were 139, 155, and 144 beats/min, respectively, 10 s apart. Twenty seconds after application of the stimulus, arterial BP was 85/35 mmHg with a pulse rate of 68 beats/min; and 72 s after termination of the seizure, these values were 78/36 mmHg and 93 beats/min, respectively. Two minutes later, BP was unmeasurable. In the initial 3 min after ECT stimulation (i.e., between 09:29 and 09:32), HRs ranged from 74 to 124 beats/min.

As shown in figure 3, ST segments in lead I showed a moderate change from −0.3 mm before ECT to −0.9 mm after ECT. In contrast, major changes of ST segments were recorded in lead II (from −1.2 mm before ECT to +2.8 mm after ECT), and this elevation was irreversible, consistent with persistent infarct related ischemia. The ST segment elevation recorded in lead V5, in excess of 1.1 mm, reverted after about 3 min.

Discussion

Fatal myocardial failure with a subendocardial infarction developed after anesthesia and ECT in this diabetic patient with known severe coronary artery disease. Though the patient was at high risk for cardiovascular complications, ECT was initiated because of unresponsiveness of the depressive illness to pharmacologic treatment. We emphasize that the factor triggering the ischemic event remains undetermined and that we cannot establish a causal relationship between ECT and the observed myocardial injury, because hypotension and tachycardia were recorded immediately before the stimulus. In fact, the severe cardiac complication might have been attributed to ECT if the noninvasive BP had been set to measure variables every 3 min (as is customary in operating and recovery rooms) and if a computer record with accurate time-stamping of all physiologic data and clinical annotations had not been available.

The patient did not complain of chest pain after ECT, but the initial diagnosis of myocardial infarction was made when protracted hypotension and marked irreversible ST segment elevation in lead II were first seen. These changes are consistent with infarct related ischemia. In contrast, the ST-segment changes in lead V5 were greater than 1.1 mm and lasted about 3 min but were reversible. Cardiac enzymes were elevated later, and therefore, the diagnosis of myocardial infarction was made. However, because ECG tracings after the ischemic event showed an absence of Q waves and because no classic evolution of a transmural infarction was observed, the infarction was termed subendocardial. Ischemic events cannot be attributed to the electroconvulsive stimulus itself.

The present acute ST elevation immediately after ECT
is comparable to that described for ECT-related myocardial stunning with transient regional and global dysfunction but without an increase in cardiac isoenzymes.9

Though ST segment abnormalities have been validated with simultaneously recorded myocardial perfusion defects in subjects with exercise,10,11 the detection of myocardial ischemia in high-risk patients by ST analysis alone is limited. However, support for the validity of the initial diagnosis of myocardial ischemia on the basis of ST segment values as shown in figure 3 comes from two observations. First, the acute appearance of persistent ST segment elevation in lead II was followed by a documented increase in cardiac enzyme concentrations. Second, the pattern of ST segment changes with persistent elevation in lead II in the patient presented here is in contrast to that observed in the comparison group of patients with the transient, variable, and modest ST depression in lead II. We excluded hypothermia, hypokalemia, and hypoglycemia12 as possible sources of ST segment changes. Furthermore, because cardiac arrhythmias and conduction defects13 interfere with appropriate ST-segment analysis, ST segment values were not reported between 09:29:38 and 09:33:09 (fig. 3).

The computer collected all data from the noninvasive BP monitor and the ECG; HRs derived from these separate sources less than 10 s apart and recorded at the time of the ischemic event were correlated closely. Therefore, we consider the HRs reported by the ECG (fig. 2) valid. In figures 1 and 2, HRs are shown that were supplied by the ECG because of its higher resolution. Because we do not have an ECG tracing obtained before, during, and immediately after the ischemic event, the type of tachycardia associated with the high HR readings shown in the lower panel of figure 2 remains unknown.

Though predictors of an adverse outcome in patients treated with ECT has not been established, in a prospective study,8 ECG tracings and cardiac enzyme determinations were negative for myocardial infarction or ischemia in 29 patients 6 h and up to 3 days after ECT. According to another study,14 ST depression occurred in 11% of patients predominantly during the convulsive phase and resolved within 2 min. Fatal complications of ECT are rare. Though the precise relationship of ECT and a cause of death may be difficult to establish, in a literature survey,4 10 of 31 fatalities met the criteria for a causal relationship between ECT and death.

The most important alternative cause of arterial hypotension immediately after ECT is severe hypovolemia, frequently seen in patients with major depression because of prolonged anorexia with insufficient fluid intake. Because the computer record showed ST segment elevation, the initial treatment of hypotension in this patient included pharmacologic cardiovascular support with norepinephrine, rather than a challenge with intravenous fluids.

Finally, we used an ECG monitor, which is limited to analysis of ST segments in only three leads. However, the presented technique of real-time display of ST-segment values, BP, and HRs, both on the computer screen and on paper, contrasts favorably with Holter monitoring. Traditionally, Holter data are not immediately available, as they are reviewed and reported retrospectively. A modified Holter device equipped with an alarm function that signals acute perturbations of ST segments was described recently.15 However, this device is limited in that the actual ST segment values cannot be seen at the time the alarm sounds.

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References

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Intraarterial Vasodilator Administration to Restore Pulse Oximeter Function

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PULSE oximetry measurement of peripheral arterial hemoglobin oxygen saturation is considered standard care during administration of anesthesia. A recent review discussed the scientific and clinical issues surrounding this technology.1 Several circumstances may make pulse oximetry readings inaccurate or unobtainable, including conditions related to the patient (carboxy- and methemoglobinemia, peripheral vasocclusion, hypotension), use of dyes (methylene blue), and interference from operating room equipment (electrocautery).1 One common reason for poor or absent pulse oximeter function is peripheral vasocclusion. When this situation occurs, common strategies to maintain pulse oximeter function include placing the pulse oximeter probe on sites other than the fingertip (ears, nose, toes), and wrapping the hand to conserve heat. Enhancing blood flow to a finger with a digital block using local anesthesia was recently reported to restore pulse oximeter function.2 Here we report intraarterial administration of vasodilators (nitroglycerin and hydralazine) through an ipsilateral indwelling radial artery catheter as a simple alternative to reverse local vasoconstriction and restore pulse oximeter function.

Case Report

A 54-yr-old woman, American Society of Anesthesiologists physical status 3, was admitted with vascular insufficiency and infection of the distal right leg. History included hypertension, non-insulin-dependent diabetes mellitus, hemodialysis-dependent renal failure, left hemisphere cerebrovascular accident 18 yr ago, and 30 pack/yr cigarette use. Medications were insulin, vancomycin, cefazidime, and metronidazole. Physical examination showed blood pressure 128/72 mmHg in the right arm, heart rate 88 beats/min, and weight 56 kg. There was a left carotid bruit. Lungs were clear to auscultation and percussion, cardiac sounds revealed an S4 and systolic ejection murmur, and no edema was present. A native vessel arteriovenous fistula was present in the left forearm. No pulse was palpable in the right leg, and the right second toe was dark and painful. Hematocrit was 21.5%. After arteriography was performed, a right leg femoral-anterior tibial bypass procedure was planned.

In the operating room, a 20-G right radial arterial catheter and a lumbar epidural catheter were placed. Bupivacaine (0.5%) was given through the lumbar epidural catheter to maintain a T8 level of anesthesia. Axillary temperature was 35.1°C. Both hands were wrapped in plastic bags, and the room temperature was maintained at 24°C. Twenty minutes after incision, the pulse oximeter stopped functioning, being unable to detect a digital pulse. Both hands were cool to touch. No pulse oximetry signal could be generated from any of the fingers, from either ear, or the nose. Blood pressure was stable throughout this period, and axillary temperature was 35.2°C.

The pulse oximeter dysfunction was thought to be due to peripheral vasoconstriction. To provide local vasodilation, 10 µg intraarterial nitroglycerin in a 2-ml volume was injected over 1–2 min through the radial artery catheter with the pulse oximeter probe placed on the right second fingertip. Pulse oximeter function was restored within 1 min of completing the injection and maintained for 12 min; repeat doses of 10 µg nitroglycerin were given and maintained pulse