J Anesth (1994) 8:352–355

Journal of Anesthesia

ST segment and T wave changes with the respiratory cycle during anesthesia for coronary artery bypass grafting

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Key words: Myocardial ischemia, ST segment, T wave, ECG, Mechanical ventilation, Coronary artery bypass grafting surgery

Introduction

Electrocardiographic changes with myocardial ischemia include ST segment and T wave alterations. In this report, we present a case in which the ST segment and T wave changed synchronously with respiration during coronary artery bypass grafting (CABG) surgery. There has been no report on changes of ST segment morphology along with respiration, the etiology of which may be similar to that of changes in T wave morphology [1,2]. However, this phenomenon may be noteworthy since it occurred during anesthetic management for CABG surgery and required special vigilance over ST segment and T wave changes.

Case Report

A 72-year-old man (height 162 cm, weight 59 kg) was scheduled for CABG surgery. Coronary angiography showed 50%, 75%, and 75% stenoses of the right, left anterior descending (LAD), and left circumflex (LCX) coronary arteries, respectively. Preoperative ECG revealed T wave inversions in leads II, III, aVF, and V_{3-6} in the absence of a Q wave. Preoperative laboratory examinations were all within normal limits.

Following his arrival at the operating room, ECG monitoring of standard limb and V_5 leads displayed on the oscilloscope equipped with the recorder (Hewlett-Packard 78254A and 78576A, Palo Alto, CA, USA). A flow-directed pulmonary artery catheter was placed in the pulmonary artery, and an intravenous administration of 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of nitroglycerin was started before the anesthetic induction. Fentanyl 1.0 mg, diazepam 10 mg, and vecuronium 8 mg were intravenously administered, and the trachea was intubated. Anesthesia was maintained by high-dose fentanyl 5.0 mg without inhaled anesthetics. Ventilation was mechanically controlled by a time-cycled ventilator (Servo 900C, Siemens-Elema, Sweden). The surgery proceeded uneventfully except for a temporary elevation of ST segment in lead II until cardiopulmonary bypass (CPB). Distal anastomoses of the saphenous vein grafts to the LCX and LAD were performed during 80 min of aortic cross clamping, followed by proximal anastomoses to the ascending aorta. The patient was separated from CPB while dopamine, nitroglycerin, and diltiazem were intravenously titrated at 5, 0.3, and 0.3 $\mu \cdot kg^{-1} \cdot min^{-1}$, respectively. Eighty-four minutes following discontinuation of CPB, changes in ST segments in leads II and V_5 (Figs. 1 and 2) were noted. These changes varied with the respiratory cycle. ST segments in leads II and V_5 were flattened during the inspiratory phase and depressed during the expiratory phase. When the patient was disconnected from the ventilator for seconds, the ST segments remained depressed (Fig. 3). Hemodynamic variables were then systemic artery pressure 131/70 mmHg, pulmonary artery pressure 24/11 mmHg, pulmonary *capillary* wedge pressure (PCWP) 4 mmHg, central venous pressure 2 mmHg, cardiac output 3.95 $l \cdot min^{-1}$, and heart rate 79 bpm. The PCWP tracing did not show any signs suggesting myocardial ischemia. The ST segment and T wave changes with the respiratory cycle continued for 30 min and gradually normalized while the mediastinum was closed. The ST segment depression persisted for a few minutes further while its morphology did not change

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Received for publication on July 30, 1993; accepted on December 16, 1993

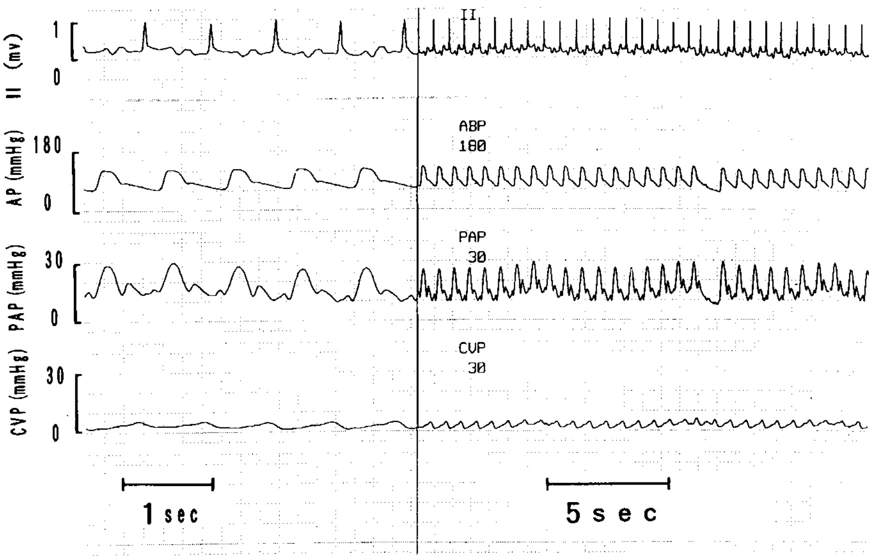

Fig. 1. ST segment and T wave changes in lead II with the respiratory cycle

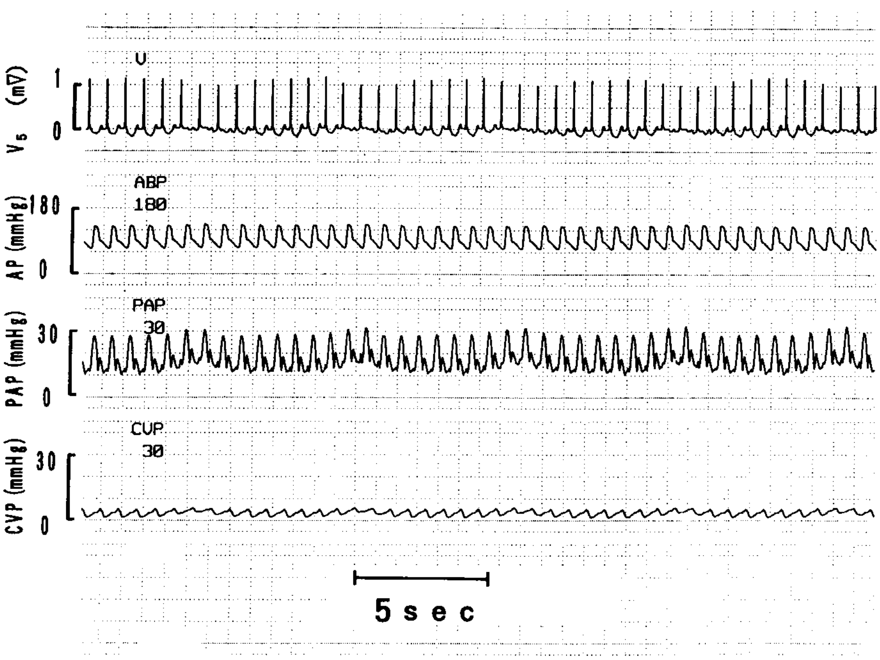

Fig. 2. ST segment and T wave changes in lead V_5 with the respiratory cycle

with the respiratory cycle. During the stay in the ICU for 2 days after surgery, ST segment changes associated with the respiratory cycle were not documented on the oscillographic display of leads II and V_5. A standard 12-lead ECG was recorded every morning for a week after surgery, and revealed inverted T waves in lead V_{4-5} which, however, did not change synchronously with the cycle of mechanical ventilation. The peak values of serum creatinine phosphokinase, aspartate aminotransferase, and alanine aminotransferase were 529 $IU \cdot l^{-1}$, 119 $IU \cdot l^{-1}$, and 73 $IU \cdot l^{-1}$, respectively, which were recorded on the 1st postoperative day. The patient postoperatively did not have any symptoms or laboratory data suggesting peri- or postoperative myocardial infarction, and was discharged from the hospital on the 44th postoperative day.

Discussion

The persistent depression of the ST segment during a temporary cessation of the mechanical ventilation (Fig. 3) suggests that ST segment and T wave changes along with the respiratory cycle were accompanied by some degree of myocardial ischemia. However, the cyclic change of ST segment and T wave morphology suggests an etiology other than ischemia. Battler et al. [3] stated that it should take 30 s or 4 min for complete or partial

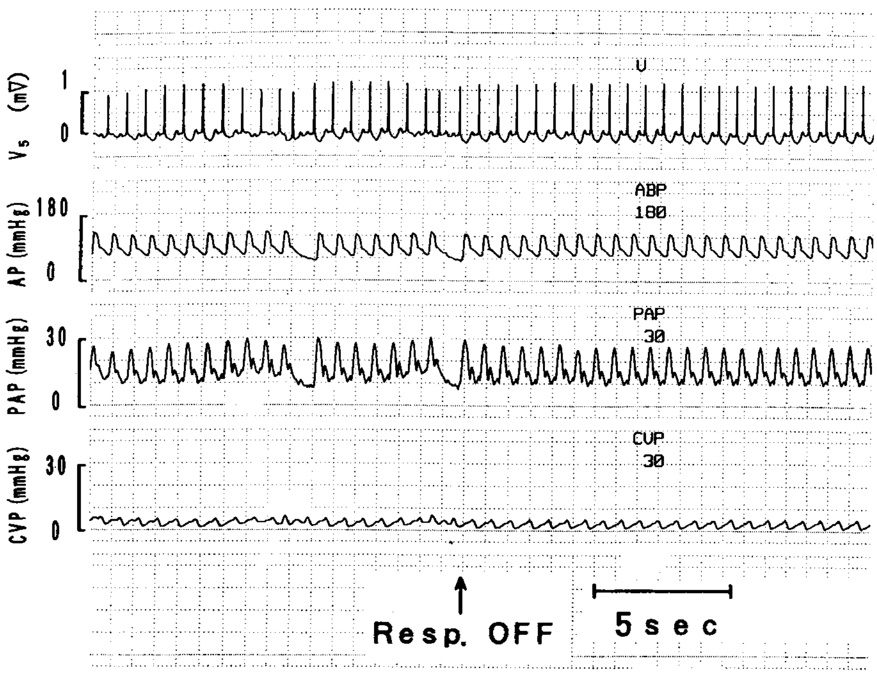

Fig. 3. Disappearance of ST segment and T waves changes in lead V_5, while the patient was weaned from the ventilator. *Resp*, respirator

obstruction of a coronary artery to produce ECG changes, respectively. Thus, even if there was a periodic occurrence of kinking or compression of coronary bypass grafts, it is highly unlikely that it would be manifested as synchronous ST segment or T wave changes.

There is a well-known respiratory variation of the heart position, namely clockwise and counterclockwise rotations in deep inspiration and expiration, respectively [1,4]. Oscillographic loop projection of vectrocardiogram shows the downward rotation of the QRS and T axes with increasing left axis deviation in the inspiratory phase [1]. Such effects might have been augmented in this case with a relatively large tidal volume of ventilation. However, such a simple anatomical change does not explain the findings in this case because the degree of variation in the amplitude of the R wave with respiration was far smaller than that of the T wave both in leads II and V_5.

Katz [5] suggested that the lungs behave as electrical insulators, whereas vessels containing blood act as conducting wires. There are several reports of interference with the conductance of cardiac electrical signals by the intracardiac blood, the pericardial effusion, and an amount of air in the lung and mediastinum associated with QRS, ST segment, and T wave changes [6–8]. In this case, there might be large varying of transfer impedance associated with periodic movements of the blood, the heart, and the lungs by the mechanical ventilation when the mediastinum was widely opened. In this context, it seems reasonable to expect that the range of ST segment and T waves became less when the mediastinum was closed.

There are also several reports of the T wave varying along with respiration which could be more likely explained by some neural effects [1,2]. Simonson et al. [1] observed the movements of QRS and T vectors in different directions associated with respiration, and suspected some vagal effect. Adams [2] demonstrated marked changes of T wave polarity in inspiration or with the Valsalva maneuver. However, there has been no report of ST segment changes with the respiratory cycle. In general, lung inflation stimulates parasympathetic neural activities where the bronchial and pulmonary C-fibers are involved [9–11]. Acetylcholine is subsequently released to increase the permeability of potassium through the cell membrane, allowing early repolarization of the Purkinje fibers [12]. This might have led to decreased negativity of T wave observed in the inspiratory phase. However, it remains to be investigated whether such neural effects can cause alterations of not only T wave but also ST segment morphology.

Lastly, there are artifacts related to an oscilloscopic monitor in the operating room [13]. In this case, however, the low frequency filters of the ECG was set at 4 cycles/s so that the tracing would be less susceptible to isoelectric line wandering by respiration, movement, or electrical artifacts [14,15]. Thus, ST segment and T waves were unlikely to be affected by respiratory movement, as shown by little wandering of the isoelectric line during respiration in Figs. 1 and 2.

Although the etiology remains to be investigated, this case showed that a sort of ischemic ECG pattern can be masked by the inspiratory phase of the respiratory cycle. The possibility of such a phenomenon should be always kept in mind during ECG monitoring in daily anesthetic practice, especially in anesthesia for cases undergoing CABG surgery [16–18] that require prompt diagnoses and treatments.

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