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ST segment depression repeatedly induced by isoflurane inhalation

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Introduction

Since Reiz et al. [1] reported ST segment changes in electrocardiography (ECG), myocardial ischemic changes induced by isoflurane inhalation have been widely discussed as a coronary steal phenomenon. Results of experimental animal studies on coronary hemodynamics of isoflurane have been highly contradictory [2,3], providing no evidence of coronary steal or deleterious effects on coronary hemodynamics. In humans, Priebe suggested in his review article that only a subset of patients with "steal-prone" coronary anatomy is likely to be susceptible to the development of coronary steal; namely, patients with one or more total occlusions of a major coronary artery, and a concomitant hemodynamically significant stenosis of the collateral-supplying vessel [4].

This is a report of a patient with reproducible ST segment depression on the ECG who responded to inhalation of isoflurane but not enflurane, although she had no history of ischemic heart disease.

Case report

The patient, a 79-year-old woman, underwent vaginal hysterectomy for prolapse of the uterus. She had been taking oral nifedipine for hypertension for several years, but she had not experienced any ischemic heart attacks and was classified as NYHA class I. The ECG before surgery did not show any ischemic changes, but showed premature atrial contractions with a frequency of 6–10·min^{-1} (Fig. 1A). After premedication with 0.5 mg atropine and 15 mg pentazocine, anesthesia was induced with intravenous thiopental (200 mg) and succinylcholine (40 mg) followed by tracheal intubation. No ST changes on the ECG were observed during this induction period. Anesthesia was maintained with isoflurane and 66% N_2O under muscle paralysis by pancuronium bromide. Respiration was controlled.

About 9 min after the induction of anesthesia ST depression (0.12 mV) was noted (Fig. 1B). At this time 2% isoflurane was inhaled, and blood pressure (BP) and heart rate (HR) were 164/65 mmHg and 64 beats·min^{-1}, respectively. Although the concentration of isoflurane was reduced to 1.5%, ST then became further depressed (0.2 mV) together with decreases in BP to 134/71 mmHg and HR to 59 beats·min^{-1} (Fig. 1C). Since isoflurane was thought to have contributed to this ST change, 2% enflurane was substituted for 1.5% isoflurane. About 7 min later, ST depression disappeared (Fig. 1D) despite the fact that BP was at the same level and HR was higher than when ST depression was first noted under isoflurane anesthesia (Fig. 1B). Under careful observation of the ECG and systemic hemodynamics, 1.5% isoflurane was readministered to prove the contribution of isoflurane to the ST depression. About 5 min later, ST was again depressed by 0.15 mV with the elevation of BP to 180/80 mmHg and the maintenance of HR at 79 beats·min^{-1} (Fig. 1E). By substitution of enflurane for isoflurane the ST depression disappeared again, although BP and HR remained unchanged (Fig. 1F). After that, anesthesia was maintained with enflurane and the patient's recovery from anesthesia was uneventful. No cardiac complications were observed postoperatively.

Discussion

The present case clearly demonstrated that isoflurane can produce ST segment depression independently of systemic hemodynamic changes, since ST depression

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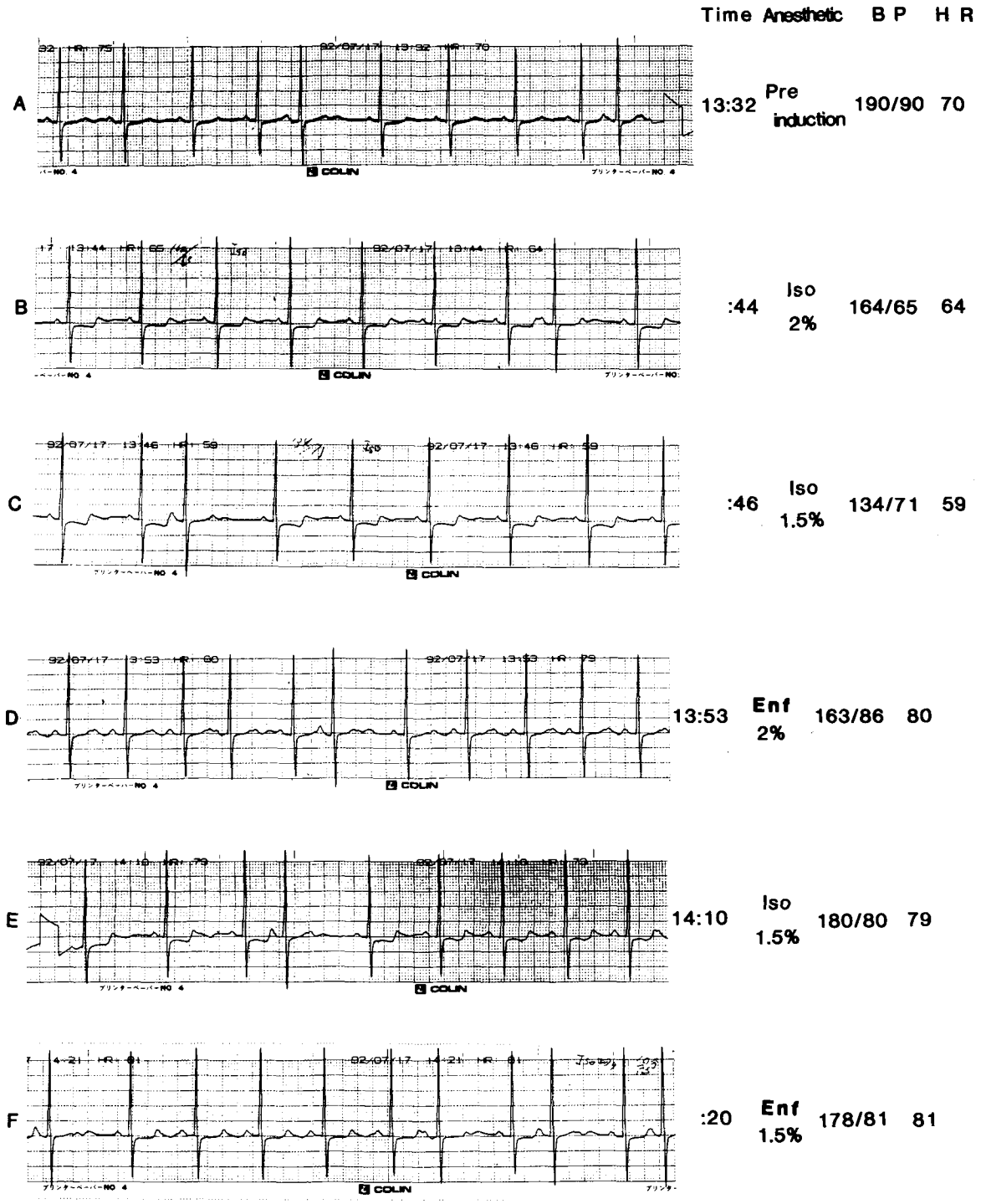

Fig. 1A–F. **A,C** ECG changes in response to inhalation of isoflurane. ECG records were from lead II. **B,C,E** ST depression appeared by inhalation of isoflurane. **D,F** ST depression disappeared when enflurane was substituted for isoflurane. *Iso*, isoflurane; *Enf*, enflurane. *BP*, blood pressure in mmHg; *HR*, heart rate in beats·min^{-1}; *Time*, when this record was taken.

occurred only when isoflurane was inhaled, but not when enflurane was inhaled, despite the existence of various systemic hemodynamic conditions. Because ST depression reflects myocardial ischemic change, the coronary steal phenomenon resulting from the strong coronary vasodilative action of isoflurane is suggested to be present in this case. This case differs from previously reported cases in that this patient did not have an obvious history of ischemic heart disease. All patients with ST changes who responded to isoflurane administration in Reiz's report had histories of angina or myocardial infarctions [1]. Reiz's patients, therefore, might belong to the "steal-prone" group described by Priebe [4]. However, the presence of preexisting abnormality of coronary circulation cannot be ruled out completely in this case. Silent myocardial ischemia has been reported to exist in a considerable number of preoperative patients [5], and a continuous ECG record using ambulatory ECG monitor and an examination of coronary circulation using angiography were not performed on this patient. Another significant finding in this case is that the ST depression was reproducible by inhaling isoflurane. There have been few clinical reports demonstrating repeatedly induced ST depression responding to isoflurane inhalation.

In summary, ST depression was repeatedly induced by inhalation of isoflurane in a patient who had no history of obvious coronary heart disease.

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