Plasma Epinephrine and Norepinephrine during Anesthesia in a Patient with Orthostatic Hypotension

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The instability of cardiovascular reflexes in a patient with orthostatic hypotension may result in profound hypotension by anesthetics and severe hypertension by vasopressors during anesthesia. In addition, blunt responsiveness of the autonomic nervous system to noxious stimuli makes it difficult to evaluate the depth of anesthesia. This report descibes the anesthetic management and the changes in plasma epinephrine and norepinephrine obtained during anesthesia in a patient with orthostatic hypotension.

Report of a Case

A 18-year-old 50 kg man was scheduled to undergo total laryngectomy for repeated aspiration pneumonitis. He had been diagnosed as Kleinefelter's syndrome in another hospital. He was admitted to our hospital for detailed examination and treatment. Repeated pulmonary aspiration was brought about by paralysis of laryngopharyngeal muscles due to a brainstem tumor. Tracheostomy was performed to manage pulmonary aspiration. It was noted that his systolic blood pressure decreased in the sitting position from 110 mmHg to 60 mmHg without compensatory tachycardia. Therefore, he was diagnosed as orthostatic hypotension. Preanesthetic laboratory findings were normal except a mildly-increased Pa_{CO_2} (45 mmHg).

No preanesthetic medication was given. On arrival in the operating room, radial arterial and central venous catheters were inserted under local anesthesia for continuous pressure monitoring and blood sampling. Anesthesia was induced by stepwise increases in enflurane concentration from 0.5% to 3% in pure oxygen. Infusion of dopamine was started at a rate of 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$. The trachea was intubated through the tracheal stoma without muscle relaxant. As systolic blood pressure decreased to 70 mmHg after induction, lactated Ringer solution was rapidly infused and the rate of infusion of dopamine was increased to 7 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Blood pressure and heart rate were stabilized at adequate levels (fig. 1). During operation dopamine was administrated at a rate of 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and the concentration of enflurane was maintained at 2%, because the patient showed body movement without changes in blood pressure and heart rate at that below 1.5%. After the operation was completed, the infusion rate of dopamine was gradually decreased and the patient uneventfully emerged from anesthesia.

The venous blood was sampled 5 times: on arrival in the operating room, immediately after tracheal intubation, after start of the operation, about the middle of the operative course, and after tracheal extubation.

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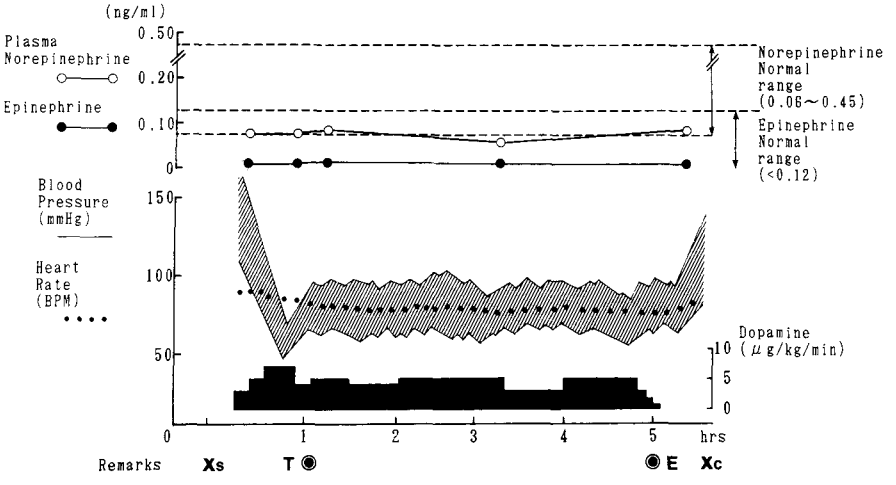

Fig. 1. The anesthetic course of the patient with orthostatic hypotension is shown in parallel with the changes in the plasma epinephrine and norepinephrine. The plasma levels of these hormones remained very low throughout the anesthetic course.

Xs: start of anesthesia, T: intubation through the tracheal stoma, E: extubation, Xc: completion of anesthesia.

Plasma epinephrine and norepinephrine were measured.

The levels of these hormones remained very low throughout his anesthetic course (fig. 1).

Discussion

It is well known that the patients with orthostatic hypotension show cardiovascular instability under general anesthesia.

Induction of anesthesia has been smoothly done with thiopental[1,3,4], fentanyl plus diazepam[2], but profound hypotension occured with ketamine[3]. Methoxyflurane[1], nitrous oxide plus halothane[2,3], ketamine[3], and droperidol[4] has been reported to cause severe hypotension during maintenance of anesthesia. In our case, development of hypotension was due probably to decreased sympathetic compensation for cardiovascular depression of enflurane.

On the other hand, hyperactivity to vasopressors such as norepinephrine[2], phenylephrine and ephedrin[3] has been observed in patients with orthostatic hypotension. Dopamine has been used without extreme hypertension[3], but there is a possibility of the occurrence of abnormal electrocardiographic changes with dopamine[3]. Because the responses to vasopressors in patients with orthostatic hypotension may be variable, these drugs must be carefully used.

Axelrod, et al.[5] emphasized the importance of fluid therapy for the patients with autonomic dysfunction. They could treat profund hypotension by sufficient fluid replacement and decreasing the depth of anesthesia without vasopressors. Our case did not receive sufficient fluid therapy before anesthesia. This might be a cause of profound hypotension during induction of anesthesia.

Plasma epinephrine and norepinephrine remained extremely low during this patient's anesthetic course, the levels of which were as low as preanesthetic ones. Such low levels of plasma epinephrine and norepinephrine suggest a blunt responsiveness of the sympathetic nervous system in the patients with orthostatic hypotension. This brings about the difficulty in evaluating the depth of anesthesia.

In summary, we described anesthesia for a patient with orthostatic hypotension. Profoud hypotension caused by enflurane anes-

thesia was well controlled by dopamine without abnormal responses. It was confirmed that plasma epinephrine and norepinephrine remained very low to noxious stimuli in the patients with orthostatic hypotension.

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