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Letter to the editor

Transient cardiac depression during suprapubic prostatectomy under subarachoid block

OSAMU SHIMODA and RYOSUKE NAKAYAMA

Department of Anesthesiology, Kumamoto University School of Medicine, 1-1-1 Honjo, Kumamoto 860, Japan

To the editor:

We report an episode of transient cardiac depression that occurred during suprapubic prostatectomy under subarachnoid block.

A 78-year-old man (weight, 78 kg, height, 168 cm) was scheduled for suprapubic prostatectomy. His medical history included Minamata disease, hypertension, and rheumatoid arthritis without complications, and he had cerebellar disorder, constriction of the visual field, and hypalgesia of the limbs. Subarachnoid block was performed, using a 23-gauge spinal needle with 1.5 ml of 0.3% dibucaine (Percamin-S; specific gravity 1.037/15°C; Teikoku Kagaku) with 50 μg of epinephrine, with the patient in the left recumbent position. After the analgesic level between T8-S5 was confirmed by the pin prick method, the surgery was begun. Immediately after the surgeon's index finger was inserted along the roof of the urethra between the lobes of the prostate, heart rate and blood pressure decreased suddenly, from 106 bpm and 135/85 mmHg to 41 bpm (Fig. 1) and 81/39 mmHg, respectively. The ST segments of the electrocardiograph were depressed from −0.7 to −2.6 mm. The patient exhibited dyspnea, although oxygen saturation on the pulse oximeter remained at 99%. Blood loss was minimal and the analgesic level was confirmed below T7. Atropine (0.5 mg) and ephedrine (8 mg) were administered intravenously. Three min later, heart rate and blood pressure had increased, to 86 bpm and 115/68 mmHg, respectively. Thereafter, the patient's discomfort was relieved. The remaining surgical procedures were carried out uneventfully.

Several cases of adverse responses caused by vagal reflex have been reported during transurethral resection under subarachnoid block.[1,2] In these patients, cardiovascular collapse was thought to have been provoked by incomplete subarachnoid block, overdistension of the bladder and/or rapid micturition. The adverse responses in our patient showed some similarity to those reported. A similar mechanism may have acted as the trigger in the events in our patient, because the bladder and prostate are closely adjacent. Although the sensory blockade was assessed as below T7, it is possible that the pin prick method may have been inappropriate for assessing analgesic level in the present patient, since he exhibited a peripheral nerve disorder.

Autonomic nervous activity in patients with Minamata disease is not well documented. Heart rate varia-

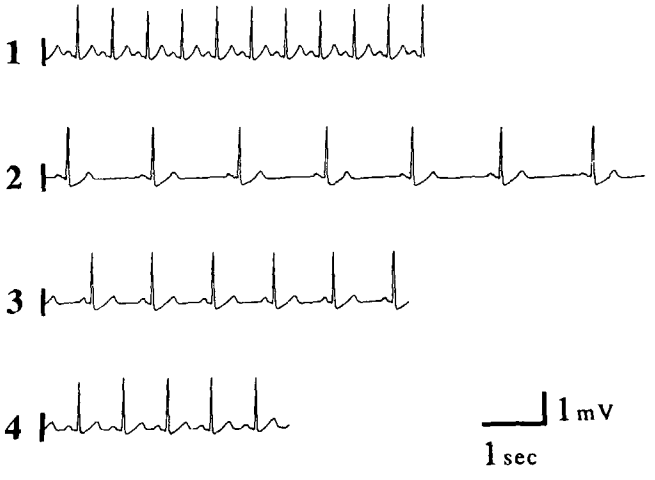

Fig. 1. Changes in lead II electrocardiograph. Before bradycardia (*1*; HR 101), bradycardia due to vagal reflex (*2*; HR 41), and findings 1 min (*3*; HR 62) and 3 min (*4*; HR 86) after intravenous atropine and ephedrine

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tions in our patient, evaluated 10 days postoperatively, showed mild suppression of both the high and the low frequency spectra. Although absence of a response to atropine is often reported in bradycardia due to autonomic neuropathy,[3,4] the present patient had a well-preserved reactivity to atropine; this suggests that the dysautonomia in this patient was not a critical factor in the cause of the brady cardia. The cardiac depression could have been prevented by the prophylactic administration of atropine.

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