deliver the radiation, it is important to have monitors in a fixed position (*i.e.*, not on the moving bed) so the camera remains focused on the monitors and, therefore, visible to the anesthesiologist outside the room throughout the session.

In summary, providing a stable depth of sedation and anesthesia with a constant infusion of propofol (Diprivan) is an effective technique to accomplish a stereotactic radiotherapy session. The technique allows for easy transport among several sites in the course of the 6- to 10-h procedure and still allows prompt recovery.

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Third Degree Heart Block and Asystole Associated with Spinal Anesthesia

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CARDIAC arrest is a well-established complication of spinal anesthesia.[1–7] We present a case of spinal anesthesia-induced asystole in which onset and recovery could be recorded by means of Holter monitoring. Holter monitoring revealed that shortly after subarachnoid injection, a first degree heart block developed that, without any previous change in heart rate, progressed to a complete heart block. After successful resuscitation, a first degree heart block that persisted until 6 h after subarachnoid injection partly outlasted sensory and motor blockade.

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Case Report

A 68-yr-old patient was scheduled for elective total hip arthroplasty. Apart from arterial hypertension, treated with nifedipine, he had no history of cardiovascular disease. Physical examination and preoperative 12-lead electrocardiograph (ECG) revealed no abnormal condition. Premedication consisted of 7.5 mg oral midazolam administered 1 h before surgery. Intraoperative monitoring included pulse oximetry, noninvasive blood pressure measurement, and 2-lead ECG. On arrival in the operating room, blood pressure was 180/90 mmHg, and heart rate was 72 beats/min. A peripheral vein was cannulated, and 500 ml of Ringer's lactate solution was administered. The patient was turned to the left lateral decubitus position, and 4.0 ml of bupivacaine, 0.5%, in saline was injected into the subarachnoid space *via* a 22-gauge Quincke needle inserted at the L3–L4 interspace. Thereafter the patient was placed in the supine position. Approximately 5 min after the subarachnoid injection, the patient complained of nausea. Blood pressure at this time was 100/60 mmHg. Immediately thereafter a loss of consciousness followed by a tonic–clonic convul-

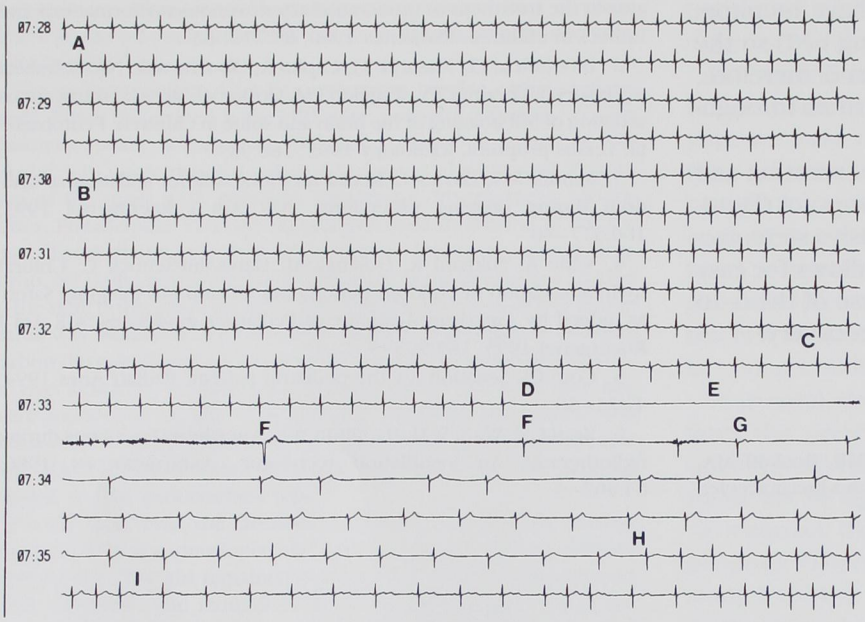

Fig. 1. Electrocardiograph printout obtained by continuous Holter monitoring illustrating the time course of electrocardiographic events. For the sake of clarity, only one of three leads is shown (CM3). Each line contains 30 s of electrocardiographic data. Numbers to the left denote the actual time in hours and minutes (printout starts at 7:28 A.M.). (*A*) Spinal anesthesia administered in the left lateral decubitus position. (*B*) Patient placed in supine position after administration of spinal anesthesia. (*C*) Onset of first degree heart block. (*D*) Onset of third degree heart block without ventricular electrical activity (P waves are not followed by an QRS complex). (*E*) Asystole (complete absence of electrical activity) after six P waves. (*F*) Thump-induced electrical activity. (*G*) Onset of atrioventricular junctional rhythm. (*H*) Atrioventricular junctional rhythm converts into sinus rhythm with first degree heart block. (*I*) Intermittent second degree heart block with a 2:1 conduction ratio.

sion was noted. The ECG and the absence of carotid artery pulse indicated asystole. Thump pacing was performed, atropine (0.5 mg) and ephedrine (20 mg) were administered intravenously, and the lungs were ventilated with 100% oxygen *via* a face mask. After 30 s the carotid artery pulse could be felt again. Within 2 min, blood pressure was restored (160/70 mmHg), and the patient regained consciousness. The sensory level at this time was at T5. Without further interventions, blood pressure (systolic pressure, 130–160 mmHg) and heart rate (80–95 beats/min) remained stable during the following 30 min, and the patient was awake and fully orientated. Therefore, we decided to proceed with surgery as planned. The maximum sensory level noted was T3. The further course was uneventful, and the patient was discharged home on postoperative day 12.

The patient had been enrolled in an ongoing clinical study on perioperative myocardial ischemia. He was monitored with a three-channel Holter ECG recorder (Marquette Electronics, series 8500, Marquette, Milwaukee, WI) using three modified bipolar leads (CM5, CM3, and aVF) from the afternoon before surgery until the fifth postoperative day. The Holter tapes were analyzed on a computerized analysis system (Marquette Laser SXP). The quality of the recordings was excellent, allowing reliable interpretation of the ECG data during the observation period.

Holter recordings revealed normal sinus rhythm (heart rate, 52–96 beats/min; PR interval, 0.14–0.18 ms) from the afternoon before surgery until the patient had been placed in the supine position after the subarachnoid injection (fig., point B). Approximately 4 min after the subarachnoid injection, a slow and gradual increase of the PR interval occurred, leading to the development of a first degree heart block (fig. 1, point C; fig. 2, top panel). Without any previous change in heart rate, a third degree heart block developed 5 min after the subarachnoid injection (fig. 1, point D; fig. 2, middle panel). After six P waves, none of which was followed by a QRS complex, no electrical activity was present (fig. 1, point E). Thirty seconds after the onset of asystole, an atrioventricular junctional rhythm developed (fig. 1, point G). One hundred ten seconds after the onset of asystole, the atrioventricular junctional rhythm converted to a sinus rhythm with first degree heart block (fig. 1, point H). During the following 5 min, a second degree heart block with a 2:1 conduction ratio was intermittently observed (fig. 1, point I; fig. 2, bottom panel). Thereafter first degree heart block was noted until approximately 6

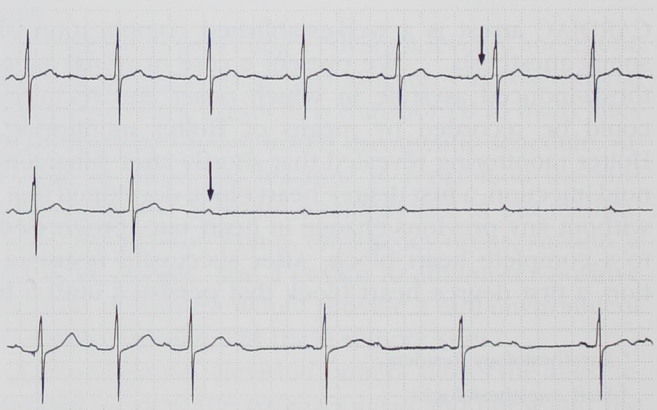

Fig. 2. Electrocardiograph printout (25 mm/s) obtained by continuous Holter monitoring. For the sake of clarity, only one out of three leads is shown (CM3). *Top panel,* Onset of first degree heart block. The arrow indicates the first beat with a PR interval ≥ 0.21 ms (corresponds to point C in fig. 1). *Middle panel,* First degree heart block progresses to third degree heart block (corresponds to point D in fig. 1). *Bottom panel,* First degree heart block with intermittent second degree heart block (corresponds to point I in fig. 1).

h after the subarachnoid injection (sensory level at this time < L_1) when the PR interval decreased below 0.2 ms. During the next 5 days, the patient remained in stable sinus rhythm (PR interval always below 0.2 ms). No ventricular ectopies more than Lown class II and no significant alterations of the ST segment occurred.

Discussion

Our case suggests that a new first degree heart block during spinal anesthesia may be a warning sign of impending complete heart block or asystole. Moreover, a spinal anesthesia-induced first degree heart block may persist for a prolonged period of time after the level of spinal anesthesia has receded below the thoracic dermatomes associated with sympathetic innervation of the heart. However, the present case illustrates that the value of first degree heart block as a warning sign of impending complete heart block may be limited by the rapidity with which first degree heart block may progress to complete heart block or asystole and the difficulty in detecting first degree heart block on a bedside ECG screen. The diagnosis of first degree and third degree heart block was only made retrospectively by analyzing the Holter recordings.

Although the occurrence of severe bradycardia and asystole during spinal anesthesia has been repeatedly reported,[1-7] the underlying mechanism remain a matter of debate. Proposed theories include (1) reflex-induced bradycardia resulting from a decreased venous return; and (2) an unopposed vagal tone by spinal anesthesia-induced thoracic sympathectomy. Current knowledge favors the former hypothesis: lumbar epidural anesthesia, confined to the lower spinal segments, induced an enhancement of cardiac vagal tone resulting from venous pooling.[8] Likewise, clinical, biochemical, and echocardiographic findings in healthy volunteers, suffering from a decrease in blood pressure and heart rate during lumbar epidural anesthesia, conform to central blood volume depletion, leading to an increase in vagal tone.[9] Anzai and Nishikawa showed that head-up body tilt induced similar increases in heart rate during either low (analgesic level T10) or high level (analgesic level T4) of spinal anesthesia.[10] This indicates that a T4 sensory level of spinal anesthesia does not produce total sympathetic blockade and, therefore, challenges the concept of spinal anesthesia-induced thoracic sympathectomy.

Although the mechanism of lumbar neuraxial anesthesia-induced bradycardia or asystole is presently unknown, the final pathway is most likely an absolute or relative increase in parasympathetic activity. It is noteworthy that nausea, a parasympathomimetic event, was the first symptom in our patient. An increase in parasympathetic activity slows conduction through the nodal region of the AV node. Higher degree heart blocks have been described to occur during spinal anesthesia in the presence or absence of preexisting conduction abnormalities.[11-15] Thus, in keeping with these previous reports, our case emphasizes that spinal anesthesia may seriously affect atrioventricular conduction. Moreover, our case illustrates that a first degree and even a third degree heart block may be missed on line. Thus heart blocks may occur more frequently during spinal anesthesia than presently thought, and impairment of atrioventricular conduction, induced by an increase in parasympathetic activity, might be an important cause of asystole during spinal anesthesia. In the present case, asystole occurred in a patient treated with the calcium antagonist nifedipine. Unlike verapamil, nifedipine does not affect atrioventricular conduction. It is therefore unlikely that nifedipine promoted or aggravated the development of heart block;

In a closed-claims study, Caplan *et al.* described 14 cases of poor outcome after a cardiac arrest during spinal anesthesia.[1] However, as in our case, favorable outcomes were described in several reports of severe bradycardia or asystole during spinal anesthesia.[2-6,11-15] Auroy *et al.* reported that 6 of 26 cardiac arrests related to 40,640 cases of spinal anesthesia were fatal.[7] Risk of death was significantly associated with higher age, higher ASA physical status class, later occurrence of cardiac arrest, and total hip arthroplasty.[7]

Our case reemphasizes that in a witnessed cardiac arrest, prompt treatment is crucial. This implies strict adherence to established safety standards, *i.e.*, the presence of qualified anesthesia personnel throughout the procedure and the continuous monitoring of the electrocardiogram. The initial treatment of asystole consists in precordial chest thumping[4] and the administration of vagolytic and sympathomimetic drugs.

The tonic-clonic convulsion of our patient appears to be an unusual observation. During a prospective survey, no seizures were described in 40,640 cases of spinal anesthesia.[7] The same survey demonstrated that all seizures related to more than 100,000 cases of regional anesthesia were preceded by classical symptoms of systemic local anesthetic toxicity. Given a stable T5-T3 level and the small dose (20 mg) of bupivacaine adminis-

tered, we can exclude the possibility of systemic toxicity. The most likely explanation for the tonic-clonic convulsion is cerebral hypoxemia, resulting from cerebral hypoperfusion.

It is noteworthy that previous reports indicate that acute bradycardia or asystole may occur at any time during neuraxial anesthesia.[3,16] In our case, first degree heart block persisted longer than the sensory blockade of the upper thoracic dermatomes. Liguori and Sharrock reported a case of severe bradycardia 210 min after epidural injection, when sensory and motor modalities had considerably receded.[16] Both their observation and our case suggest that neuraxial anesthesia-induced bradycardia or alterations in atrioventricular conduction may, at least partly, outlast sensory or motor blockade. If this hypothesis were to be confirmed, this would have important implications for postoperative monitoring and timing of discharge of patients to the ward.

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