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Intraoperative "Syncope": Evaluation with Tilt-table Testing

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The cause of acute, intraoperative hemodynamic deterioration in well-oxygenated patients is often unknown. The Bezold-Jarisch reflex has been suggested as a possible explanation in recent reports of patients who, without apparent warning, develop profound hypotension and bradycardia during spinal anesthesia.[1,2] The Bezold-Jarisch reflex originates from chemo- and mechanoreceptors in the left ventricle.[3] Stimulation of these receptors results in increased centrally mediated parasympathetic activity and decreased sympathetic activity with concomitant bradycardia, arterial vasodilation, and hypotension. We report a case of profound bradycardia and hypotension during general anesthesia in a patient in the sitting position. Follow-up tilt-table testing implicated the Bezold-Jarisch reflex as the underlying mechanism.

CASE REPORT

A 74-yr-old woman presented for shoulder reconstruction. Underlying medical problems included diet-controlled diabetes mellitus, hypertension, stable exertional angina, and hypercholesterolemia. She did not have a history of syncope. Medications included diltiazem, isosorbide dinitrate, furosemide, and nitroglycerin as needed. On physical examination she had a blood pressure of 140/60 mmHg, a regular pulse at 60 beats/min, and a weight of 86 kg. Her airway appeared normal, and cardiac examination revealed no murmurs or third or fourth heart sounds. A preoperative ECG was normal, with sinus rhythm at 59 beats/min, and was unchanged from previous tracings. Preoperative cardiac evaluation included a persantine-thallium scan that revealed mild inferolateral ischemia and an echocardiogram demonstrating normal ventricular function. Prior to induction of anesthesia, intravenous and radial arterial catheters were placed. No premedication was administered. Anesthesia was induced with thiopental 200 mg and fentanyl 400 μg in incremental doses. Tracheal intubation was facilitated with succinylcholine. During induction and intubation, hemodynamics were stable (blood pressure 150–160/70–80 mmHg and heart rate 50–55 beats/min). Anesthesia was maintained with nitrous oxide (50%), oxygen, isoflurane (≤ 0.5% inspired concentration).

The patient then was placed in the sitting position for surgery. After approximately 10 min of sitting upright, her blood pressure decreased to 40 to 50 mmHg (systolic) and heart rate decreased to 43 beats/min. She was placed in Trendelenburg's position; anesthetics were discontinued; and intravenous fluids and ephedrine (15 mg iv) were administered without appreciable change in heart rate or blood pressure. On the ECG (leads II and V), there was no evidence of ischemia. She received phenylephrine (total of 300 μg), and hypotension resolved over 15 min. Hemoglobin oxygen saturation was 100% throughout this episode. Atropine was not used immediately because of concerns about tachycardia in a patient with a history of angina and limited intraoperative monitoring to evaluate ischemia as a cause for her deterioration. The patient awoke promptly and was neurologically intact. Surgery was postponed. Subsequent ECGs remained unchanged, and myocardial infarction was ruled out.

Since the intraoperative hypotension and bradycardia were unexplained, tilt-table testing was used to evaluate the possibility of intraoperative neurally mediated vasovagal "syncope." While supine the patient's blood pressure and heart rate were normal. However, after 29 min of 60° head-up tilt, she suddenly developed dizziness, hypotension, and bradycardia (figs.1A and 1b). In view of this positive tilt-table test, with apparent replication of intraoperative hemodynamic events, she was diagnosed as having intraoperative vasovagal "syncope." She was treated with pindolol, 5 mg twice a day, a β-adrenergic blocker with intrinsic sympathomimetic activity. Following several days of therapy, repeat tilt-testing was negative (figs. 1C and 1D), and she was rescheduled for surgery.

Subsequently, anesthesia was induced with midazolam 4 mg intravenously, fentanyl 200 μg intravenously, and succinylcholine to facilitate tracheal intubation. Anesthesia was maintained with nitrous oxide, oxygen, and enflurane. Anesthesia induction, positioning, and surgery proceeded uneventfully.

DISCUSSION

Although intraoperative hypotension and bradycardia are not uncommon, there are several unusual features about this case. First, the onset of bradycardia and hypotension was quite abrupt, occurring prior to incision and after 10 min of stable hemodynamics while the patient was in the sitting position. Secondly, blood pressure and heart rate did not become normal when she was placed in Trendelenburg's position. Lastly, ephedrine had no effect on either bradycardia or hypotension. Our initial differential diagnosis included: acute myocardial ischemia/infarction, cerebrovascular event, drug reaction, and autonomic dysfunction. Drug reaction seemed unlikely; it soon became clear there was no clinical evidence for myocardial infarction or cerebrovascular event, and autonomic dysfunction secondary to diabetes mellitus was unlikely given the abruptness of hemodynamic deterioration. Before proceeding with surgery, we elected to

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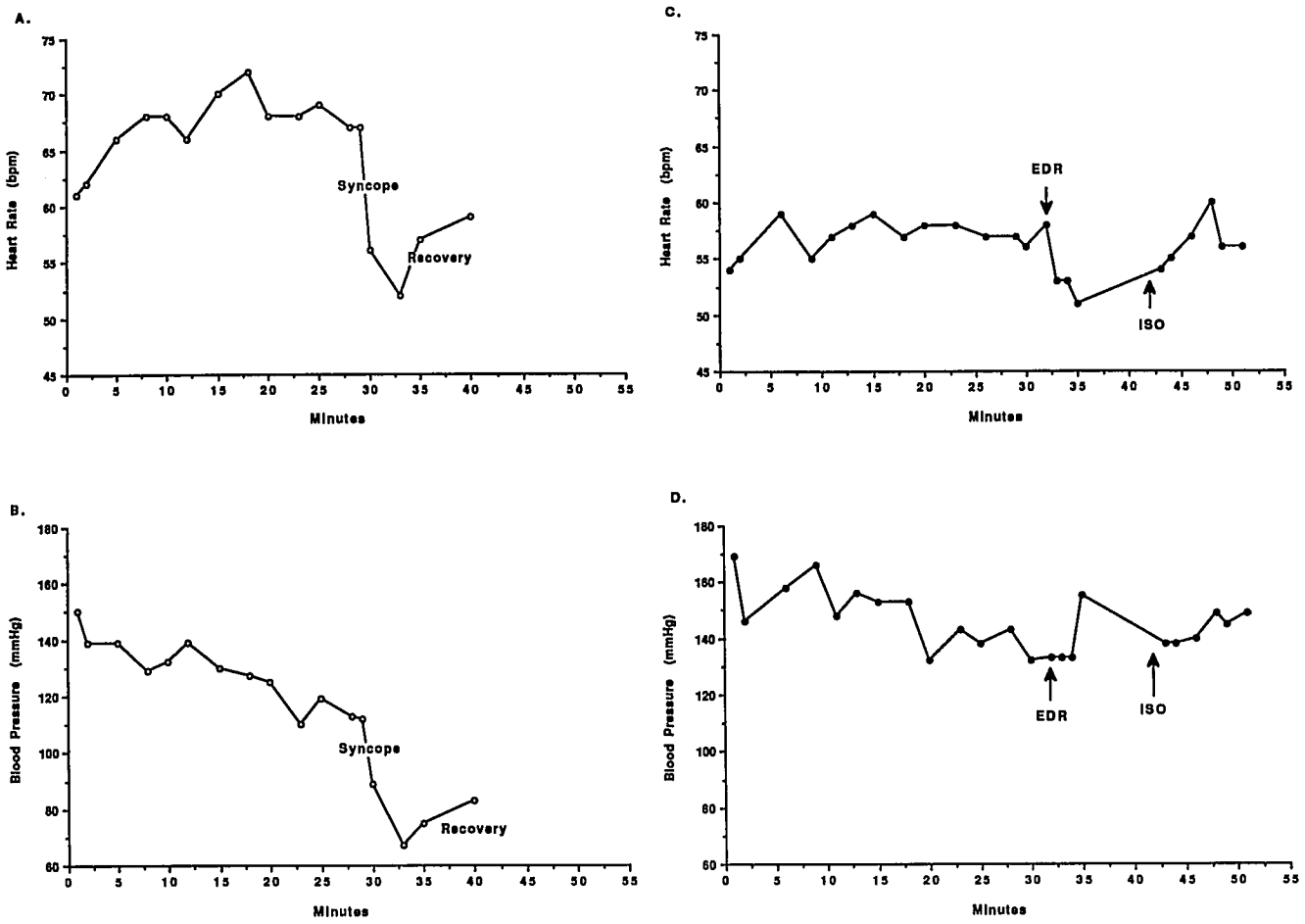

FIG. 1. The changes in heart rate and blood pressure during two head-up tilt table tests. During the initial test (*A*, *B*), the patient was placed in a 60° head-up tilt position, and after 29 min she developed syncope. She was immediately returned to the horizontal position. Blood pressure and heart rate were recorded over the subsequent 10 min (recovery). During her second test, while receiving pindolol therapy (*C*, *D*), the patient was placed in a 60° head-up tilt position, and after 30 min she remained asymptomatic. With this protocol, if patients do not develop syncope after 30 min of head-up tilt, two different pharmacologic provocateurs are used to induce vasovagal syncope. She received edrophonium (EDR) 10 mg intravenously while in a 60° head-up tilt position without developing syncope. She was then given isoproterenol (ISO) titrated to a heart rate 20% > baseline (1–4 μg/min intravenously) while in a 60° head-up tilt position. Syncope was not induced.

evaluate the patient for a potential predisposition to vasovagal syncope with tilt-table testing.

Tilt-table testing has recently become a valuable procedure in the evaluation of unexplained syncope. The test relies on a series of responses to venous pooling when the patient is shifted from a horizontal plane to a 60° head-up position.[3–8] Venous pooling (500–1,000 ml blood) causes increased sympathetic tone and vigorous ventricular contractility. In susceptible patients, this triggers the Bezold-Jarisch reflex. When the sympathetic response is inappropriate, increased sympathetic activity causes ventricular hypercontractility and the firing of C-afferent fibers. The C-afferent fibers trigger a two-pronged medullary-mediated reflex: one is a sympatholytic effect on the arterial tree, with vasodepression; the second is increased vagotonia and bradycardia.[3,8–10] Thus, increased levels of circulating catecholamines, including administration of ephedrine, may paradoxically augment the Bezold-Jarisch reflex.[3–5,9,10]

We routinely use head-up tilt testing alone and with pharmacologic provocateurs, such as edrophonium and isoproterenol, to trigger neurally mediated vasovagal syncope in susceptible patients.[4,11] With head-up tilt alone, our patient responded in a typical vasovagal fashion with an abrupt onset of hypotension and bradycardia at 29 min, which is characteristic of patients with neurally mediated syncope.[6] β Blockers may blunt the sympathetic stimulus leading to the Bezold-Jarisch reflex, and they are often effective therapy in these patients.[3,12,13]

In our patient, hypotension and bradycardia were reproduced with head-up tilt but completely prevented with a β blocker. Pindolol was chosen since it results in less

bradycardia when compared with other β blockers. Hypotension and bradycardia did not recur after induction of general anesthesia and head-up positioning while the patient received pindolol. Although differences in anesthetic technique cannot be discounted, in the absence of any anesthetic agents the patient developed typical vasovagal syncope during tilt-table testing. However, she did not experience syncope during tilt-table testing while receiving pindolol. We therefore believe that pindolol played a significant role in the successful hemodynamic outcome of her operation. While there were some differences in the anesthetic technique between the first and second inductions that may have accounted for different hemodynamic outcomes, it is more likely that this patient experienced the Bezold-Jarisch reflex during her first anesthetic induction. She probably did not respond to ephedrine because additional catecholamines paradoxically potentiated this reflex. When the Bezold-Jarisch reflex was blunted with a β blocker, the patient remained hemodynamically stable intraoperatively despite head-up tilt.

Although diabetes mellitus may have caused this patient's intraoperative deterioration, it is unlikely.[14] She had only mild diabetes requiring no drug therapy. More importantly, β blockers are not known to be effective therapy in patients with diabetic dysautonomia.

We believe the Bezold-Jarisch reflex may be an important cause of unexplained intraoperative morbidity. The sudden onset of hypotension and bradycardia in anesthetized, well-oxygenated patients is alarming and poorly understood. The Bezold-Jarisch reflex has been postulated to be the mechanism in patients experiencing this syndrome during spinal anesthesia.[1,2] In these patients the decrease in ventricular end-diastolic volume and secondary compensatory ventricular hypercontraction around an almost empty chamber is believed to trigger the Bezold-Jarisch reflex.[2] In our case involving general anesthesia in the sitting position, the combination of venous pooling and predisposition to the Bezold-Jarisch reflex likely contributed to this patient's sudden hemodynamic deterioration, or intraoperative "syncope." In this patient, tilt-table testing was helpful in identifying the Bezold-Jarisch reflex as the most likely cause of her initial hemodynamic collapse. Moreover, repeat tilt-table testing on pindolol therapy played an important role in her subsequent management. The Bezold-Jarisch reflex may be largely unappreciated in anesthetized patients, and tilt-table–guided therapy may be useful in patients with a history of unexplained, abrupt intraoperative hypotension and bradycardia.

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