The most likely mechanism for creation of the TEF was erosion of the patient's nasogastric tube through the anterior esophageal wall into the trachea. The finding that the defect was larger on the esophageal side than on the tracheal side makes bronchoscopy an unlikely cause. None of the other common causes of TEF in adults (radiation therapy, trauma, corrosive burn, malignancy, congenital defect, or tuberculosis) apply to this patient.

There are several causes of air leak after tracheal intubation, most of which are easily verifiable. The most common are inadequate depth of ETT insertion (with the ETT cuff straddling the vocal cords), inadequate cuff inflation, and inadvertent placement of an esophageal stethoscope or nasogastric tube into the trachea.

Exclusion of these causes, in the presence of an increased leak associated with removal of the esophageal stethoscope, led to a presumptive diagnosis of TEF. This diagnosis was confirmed postoperatively. During directed questioning postoperatively, the patient admitted to a history of coughing when drinking fluids (especially carbonated beverages).

Despite several weeks of keeping the patient as *non per os* and providing nutrition *via* a feeding tube, the fistula failed to close. Subsequently, the patient underwent esophagoscopy and injection of the fistula with a fibrin sealant. Her coughing immediately ceased, and she remains asymptomatic.

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The Changes in Bispectral Index during a Hypovolemic Cardiac Arrest

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ANESTHESIOLOGISTS for many years have sought to monitor brain function during surgery to assist in proper administration of sedative–hypnotic agents and to detect early signs of inadequate cerebral perfusion. A processed electroencephalographic device (Aspect Medical Systems, Natick, MA) newly approved by the Food and Drug Administration produces a unitless scale from 0 to 100 that correlates with a patient's hypnotic state.[1] At the top of the scale (100), a patient is awake and responsive. As hypnotics are administered, the scale (Bispectral Index, BIS) decreases in a dose-related fashion. Generally, free recall is lost at 70 and consciousness is lost at 60.[2] As the Index approaches 0, burst suppression becomes more prominent as electroencephalographic activity is lost.

We describe a patient in whom this monitor was used for titration of anesthetic agents for a tricuspid valve replacement, during which she had transient hypovolemic cardiac arrest. The BIS and hemodynamics were stored real-time into a laptop computer. This case report shows the potential value of BIS monitoring, not only for intraoperative drug titration, but also as a sign of return of cerebral function after an intraoperative threat to neurologic integrity. It was possible to track how the patient was responding to resuscitation.

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Key words: Bispectral index; cerebral ischemia; hypotension.

Case Report

The patient was a 22-yr-old woman scheduled for tricuspid valve replacement. She had undergone closure of a ventricular septal defect

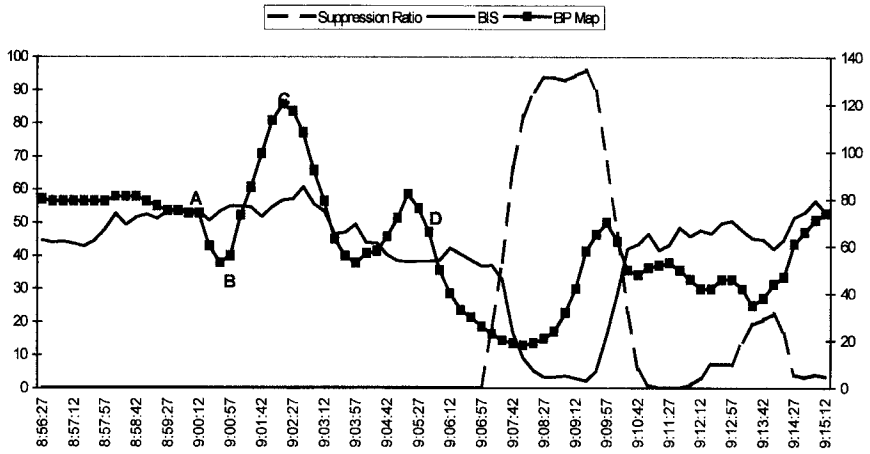

Fig. 1. Changes in the Bispectral Index with severe hypotension.

in the distant past, and now presented with pulmonary artery hypertension and progressive right ventricular failure because of tricuspid valve insufficiency.

She received 10 mg morphine sulfate and 5 mg midazolam (in divided doses) for sedation during (peripheral) intravenous (8.5 French), arterial and internal jugular (9 French) line placement. Induction then consisted of 25 μg sufentanil, 125 mg thiopental, and 6 mg pancuronium with 0.25 $\mu g \cdot kg^{-1} \cdot h^{-1}$ sufentanil and 0.5% isoflurane for maintenance. The BIS decreased to 55 with this induction sequence.

During sternotomy, using an oscillating saw, the right atrium was lacerated. There was sudden loss of blood volume within 2 min. (fig. 1) Despite having seemingly adequate intravenous access, we were unable to maintain cardiac filling, and the patient's systolic blood pressure decreased to 20 mmHg (fig. 1). Extra surgical personnel were called in to assist with immediate femoral cannulation and start cardiopulmonary bypass. Simultaneously, another surgical team repaired the damage to the atrium. During the whole sequence, an automatic record was kept of heart rate, mean arterial pressure, and BIS value on a laptop computer.

As can be seen in figure 1, (A) at 9:00 AM, just after incision, vital signs and BIS were stable. However, with sternotomy, large amounts of blood were lost, causing hypotension (approximately 9:01 AM; [B]), which was treated with a rapid infusion of phenylephrine (C), resulting in a rise of the mean arterial blood pressure to 85 mmHg. After the patient started to exsanguinate, blood pressure could not be maintained despite adding as much heated volume as possible (D). Approximately 4 min after the start of the sternotomy, she underwent hypovolemic arrest, with mean arterial pressure decreasing to 20 mmHg. It took approximately 9 min for the patient's blood pressure to be stabilized as cardiopulmonary bypass was initiated. During this time, manual cardiopulmonary resuscitation was performed as best as possible.

Of note are the concurrent tracings of BIS and blood pressure. As the blood pressure decreased with cardiac laceration, the decrease in BIS lagged behind the decrease in blood pressure by approximately 2 min. This may be caused, in part, by the 30- to 60-s delay inherent to the smoothing algorithm used to calculate BIS, and autoregulation and the lag between decreases in blood pressure, cerebral blood flow, and neuronal dysfunction. As blood pressure was corrected by volume replenishment (*via* femoral arterial cannulation), an additional 2 min was necessary for neuronal function, as indicated by the BIS, to return to the premorbid level. During the time in which neuronal function, as indicated by the BIS, was low, the burst suppression ratio was high. As cerebral blood flow was reinstituted, the BIS returned and the percentage of burst suppression decreased. During the case, we believed that the return of the BIS was a reassuring sign of successful resuscitation.

The remainder of the case continued without difficulty. The patient was able to be weaned from cardiopulmonary bypass without problem or vasoactive agents. Because of our concern about possible ischemic brain injury because of the episode of hypotension, the patient was awakened promptly after skin closure. She awoke without incident and was able to move herself off the operating room table onto the stretcher. She was extubated within 4 h of intensive care unit arrival.

Discussion

Using the the BIS in cardiac surgery and in the ambulatory setting has been shown to optimize titration of anesthetic and hypnotic agents[3] to achieve faster recovery times.[4]

While using the BIS in our usual fashion, we suddenly were confronted with a crisis. The sudden loss and return of blood pressure evoked a parallel but delayed pattern in the BIS, with transient electroencephalographic silence (100% burst suppression). The reversible nature of the BIS (electroencephalography) suggested that the patient was not going to experience permanent ischemic brain injury. In fact, that turned out to be the case.

It is clearly acknowledged by the makers of the product that the BIS is not intended as an ischemic monitor nor as a predictive device. Further studies need to be performed to evaluate such applications. However, this case shows how the availability of a simple-to-use monitor of brain function can provide useful new information regarding patient response to unexpected events.

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Extreme Intraoperative Blood Loss and Hemodilution in a Jehovah's Witness: New Aspects in Postoperative Management

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IN patients who refuse any blood components, global oxygen transport, cerebral oxygen delivery, and blood coagulation may be compromised during massive blood loss. This case report describes potential therapeutic options to improve blood coagulation and to monitor the adequacy of cerebral oxygen delivery by using the patient's own consciousness.

Case Report

We report the case of an 41-yr-old female Jehovah's Witness. Her body weight was 47 kg and her height was 160 cm. She was admitted for elective hysterectomy as a result of a uterine and intraligamentary myoma and failed intubation that occurred at the referring hospital. Her history was remarkable for a severe primary chronic polyarthritis. A preoperative echocardiography indicated a slight aortic insufficiency (regurgitation $< 30\%$) caused by degenerative alterations of the aortic valve. Ejection fraction was normal, and the patient was not limited in her physical activity. At the preoperative visit she provided a signed statement documenting her wish to refuse any blood components. Her hematocrit was 37.4% (hemoglobin, 12.6 g/dl), platelet count was 273,000/μl, and prothrombin time was < 10.6 s (normal, 10.6–12.9 s). Other laboratory findings were in the normal range. Blood pressure was 115/75 mmHg, and heart rate was 100 beats/min.

An awake fiberoptic intubation was planned. After successful tracheal intubation, anesthesia was induced with fentanyl, etomidate, and midazolam, and muscle paralysis was achieved with atracurium. Anesthesia was maintained with continuous propofol and remifentanil infusions. Blood pressure was measured noninvasively.

A large myoma was found during laparotomy (diameter, 12 cm; 865 g) that extended into the broad ligament and displaced the right ureter. Massive bleeding occurred from numerous blood vessels from the pelvic floor, supplying the intraligamentous myoma. To maintain normovolemia and blood pressure, a total of 5,000 ml of Ringer's lactate, 1,000 ml of 6% hydroxyethyl starch with a mean molecular weight of 200 kd (Fresenius, Bad Homburg, Germany), and 2,000 ml of 4% modified fluid gelatin (Braun Medical, Melsungen, Germany) was infused. In addition, norepinephrine (up to 170 ng $\cdot$ kg^{-1} $\cdot$ min^{-1}) and dopamine (4.3 μg $\cdot$ kg^{-1} $\cdot$ min^{-1}) were infused, and supplementary bolus doses of epinephrine and norepinephrine were administered. Hematocrit was 10% 2 h after incision and 8% at the end of the surgery. A severe coagulopathy developed, as evidenced by a prothrombin time of 39.5 s, a nonmeasurable activated partial prothrombin time, and a fibrinogen < 0.5 g/l. Platelet count was 113,000/μl. Two million units of aprotinin (Bayer, Leverkusen, Germany) were administered, followed by 500,000 U/h (total of 3 million units). In addition, metabolic acidosis was apparent with a *p*H of 7.28 and a base excess of -8.3 mM/l.

Finally, surgical hemostasis was achieved. A central venous access

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Key words: Erythropoietin; ICU management; prolonged anemia; surgery.