philic,‡ respiratory depression may be more common than with morphine. While other reports of epidural opioid overdose were due to avoidable medical personnel errors,[4,5],§ patient-generated overdoses are not. The unpredictability and potentially disastrous consequences of respiratory depression would suggest action to protect patients from overdosing themselves.

Patients are generally considered to be protected against the Valley Lab pump (IV7200) because it is a sophisticated, reliable, and accurate computerized device. It would be highly unlikely for a pump to spontaneously malfunction and cause an overdose of its infusate. It is not true, however, as demonstrated in this case, that the pumps are protected from the patients. The convenient but unsecured controls are easily adjusted by curious, self-destructive, drug-seeking, or confused patients.

To reduce this risk, a locking cover, as found on many patient-controlled analgesia pumps, might be desirable, effective, simple, and inexpensive. Until these pumps are made more secure, patients such as the one described above might not be suitable candidates for epidural infusions, and if pump controls are found to have been tampered with, perhaps infusions should be discontinued immediately.

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Superior Vena Cava Syndrome as a Complication of Change in Body Position during General Anesthesia

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Although superior vena cava syndrome (progressive obstruction or compression of the superior vena cava) manifests with distinctive and characteristic signs and symptoms, its causes are variable. The syndrome is frequently associated with mediastinal masses and chronic mediastinitis‡ but may also occur after insertion of central venous catheters.[1] We observed the development of superior vena cava syndrome after an inadvertent change in a patient's body position during surgery on the thoracic spine.

CASE REPORT

A 64-yr-old, 84-kg man who was essentially healthy except for a history of controlled hypertension had neurologic symptoms of low-back pain, bilateral pain of the lower extremities, and bladder incontinence. Physical examination indicated diffuse bilateral motor weakness of the lower extremities. Magnetic resonance imaging revealed severe spondylosis and stenosis at T11–12 and L4–5 and large osteophytes. His chest radiograph results were normal. The patient was scheduled for removal of a T11 spur and a lumbar laminectomy; a left lateral extracavitary approach was planned.

Before induction of anesthesia, an arterial and a central venous catheter were inserted *via* a radial artery and the right internal jugular vein, respectively. Chest x-ray confirmed the position of the tip of the central venous catheter in the lower part of the superior vena cava. Two 16-G peripheral intravenous (iv) catheters also were inserted. Anesthesia was induced with thiopental, vecuronium, and sufentanil and was maintained with isoflurane, N_2O, and a sufentanil iv infusion. The patient was rolled onto the operating room table and positioned semiprone with the right side down. A bean bag (Olympic Vac-Pac™, Olympic Medical Co., Seattle, WA) provided support. Other supports for pressure points consisted of two foam chest rolls; pillows placed under the left arm, left leg, and right knee; and foam padding placed under the iliac crest. The bean bag was made rigid by suction deflation,

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Key words: Veins: superior vena cava. Complications: superior vena cava obstruction.

‡ Hunter W: The history of an aneurysm of the aorta, with some remarks on aneurysms in general. Med Obser Inq (London) 1:323, 1757

and pressure points were rechecked by both the anesthesiologist and the neurosurgeon.

Before anesthesia, central venous pressure (CVP) was 12 mmHg and arterial blood pressure was 140/80 mmHg. Hemodynamic status remained stable for the first 4 h of surgery, with CVP in the 12–15 mmHg range. Approximately $4\frac{1}{2}$ h after the incision, CVP increased to 41 mmHg. Examination of the catheter site revealed no external kinks in the catheter, and blood was easily aspirated. We also noted that the beanbag was no longer rigid and that the chest rolls had shifted. The patient had thus shifted to a slightly more prone position. The patient was repositioned as much as possible, given the necessity of maintaining a sterile field. The bean bag was made rigid again, chest rolls were repositioned, and surgery resumed. Several minutes later, edema of the patient's head and neck was noted. Eventually, both upper extremities also became edematous. Central venous pressure continued to increase to 70 mmHg, and edema became increasingly severe.

At this point, the thoracic phase of surgery was complete. The lumbar laminectomy was postponed so that the patient could be repositioned for optimal venous drainage of the head and neck. The wound was closed, and the patient was placed in a 45°, head-up, semi-Fowler's position with the head in the midline. With the patient in this position, CVP returned to 20 mmHg. The patient was allowed to regain consciousness to the point at which he could obey commands and at which he appeared neurologically intact. He was then resedated and admitted to the surgical intensive care unit; the trachea remained intubated. At this time, the head and neck were extremely edematous and tense on palpation, and the tongue was swollen and protruding from the mouth. Postoperative anterior–posterior chest x-ray revealed no evidence of widening of the mediastinum, and the position of the catheter was unchanged.

Over the next 24–36 h, edema involving the head, neck, and upper extremities resolved completely. Central venous pressures remained normal, and the trachea was extubated 36 h after surgery. No neurologic sequelae were apparent.

Discussion

In 1757, William Hunter provided the first description of superior vena cava syndrome, which occurred in a patient with syphilitic aortic aneurysm.‡ Since then, the syndrome has been attributed to several disease processes of the mediastinum. Thoracic neoplasms account for as many as 97% of all cases.[1] Because the superior vena cava crosses the right main bronchus, lung cancer (especially of the right lung) is a common cause. Less common sources include chronic mediastinitis due to histoplasmosis and aortic aneurysm. The current widespread use of central venous and pulmonary artery catheters has added yet another source of superior vena cava syndrome—clot formation around the central catheter.[2] Clot formation has also caused the syndrome after placement of transvenous pacemakers.[3]

The superior vena cava is a thin-walled, low-pressure system that is vulnerable to compression. It is surrounded by lymph nodes and, lying adjacent to the aorta, traverses the right side of the mediastinum. A considerable portion of the superior vena cava is enclosed in the pericardial reflection and is therefore relatively fixed.[4] The main collateral channel for the superior vena cava is the azygous vein, which is able to dilate and compensate for venous drainage during the early stages of insidious superior vena cava compression.

The diagnosis of superior vena cava syndrome is usually based on clinical signs. The conscious patient may complain of hoarseness, shortness of breath, coughing, and visual disturbances. Advanced cases sometimes include alterations in consciousness. Physical findings are proportional to the rapidity of development of the syndrome and include dilation of collateral veins and edema of the upper chest, arms, palpebra, and conjunctiva. Glossal and periorbital edema are not uncommon. Venous stasis may cause cyanosis of the skin.[4] Also, central venous hypertension sometimes results in inappropriate secretion of antidiuretic hormone.[5] As might be expected, the most common radiographic abnormality is widening of the superior aspect of the mediastinum, especially on the right side.[6]

Our patient displayed many of the classic signs of superior vena cava syndrome, and the presence of an indwelling central venous catheter allowed us to document the significant increase in CVP. The cause of the increased pressures is not readily apparent. The possibility of laceration of the superior vena cava by the guidewire and subsequent formation of a mediastinal hematoma are made less likely by the postoperative chest x-ray showing no evidence of widening of the mediastinum. The temporal relationship of the increase in CVP to the change in position and the patient's hemodynamic stability for $4\frac{1}{2}$ h after placement of the catheter also make mediastinal hematoma less likely. An enlarged mediastinal structure impinging significantly on the superior vena cava after the change in body position might have been the cause of the increased CVP. Again, an enlarged mediastinal structure was not noted on either the preoperative or postoperative chest x-ray but remains a possibility. Another possibility would be an intravascular anomaly of the superior vena cava that was made worse by the change in position. The likelihood of such an occurrence would be highest in the distal superior vena cava, which is relatively fixed by the pericardial reflection.

Although this complication has the potential for serious consequences, we cannot recommend, on the basis of one case report, routine monitoring of CVP in all patients in the lateral decubitus position. We do believe, however, that this case reinforces the importance of patient positioning and describes a possible major complication of malpositioning.

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Anesthetic Considerations for Severe Ovarian Hyperstimulation Syndrome

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Severe ovarian hyperstimulation syndrome (OHSS) due to exogenous gonadotropin administration is a life-threatening condition associated with ascites, pleural effusions, oliguria, electrolyte abnormalities, hemoconcentration, hypercoagulability, and hypotension.[1]

We present the first recorded case of anesthetic management for the patient with severe ovarian hyperstimulation syndrome.

CASE REPORT

A 39-yr-old woman who had previously been in good general health and who was diagnosed with primary infertility underwent ovarian stimulation, *in vitro* fertilization, and embryo transfer. After follicle-stimulating hormone and luteinizing hormone administration, serum estradiol concentrations reached 7,280 pg/ml, a very high level compared to the 1,500–2,000 pg/ml level that is frequently sought. Ten thousand international units of human chorionic gonadotropin (HCG) was given and 40 oocytes were retrieved, of which 28 were fertilized and six were transferred to the patient. Development of nausea, vomiting, and abdominal pain on the day of embryo transfer (and 3 days after HCG administration) prompted admission to the hospital (table 1). Initial treatment consisted of bed rest and 50 g albumin intravenously (iv) every 12 h. On hospital day 2, the patient developed abdominal distension and polycythemia. Fluid intake was restricted to intravenous fluids administered at 50 ml/h, and 25 mg indomethacin by mouth was begun. The next day she complained of dyspnea, worsening of her abdominal pain, and vomiting. Physical examination demonstrated increased abdominal girth, ıbsent breath sounds over the lower half of both posterior lung fields, and an increase in hematocrit to 68%. Hematocrit decreased to 43%, and furosemide was added to the regimen.

Over the next 8 days, abdominal distension and dyspnea worsened; her weight increased by 9 kg; and pitting edema developed over the sacrum and the lower extremities. Beta HCG levels increased, indicating pregnancy. Renal failure developed as BUN and creatinine increased to 65 mg/dl and 2.0 mg/dl, respectively. Dopamine was started at a dose of 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$ in an attempt to increase renal blood flow. Despite this, urine output decreased to less than 10 ml/h, and creatinine clearance decreased to 10 ml/min. Chest x-rays had not been obtained, but arterial oxygen tension during breathing of supplemental oxygen was 119 mmHg.

To prevent irreversible renal damage, termination of pregnancy by suction dilatation and curettage was deemed necessary. On hospital day 12, she was brought to the operating room. By this point, her normal weight of 54.5 kg had increased to 63.6 kg. Blood pressure was 110/70 mmHg, pulse was 80 beats per min, and respiratory rate was 20 breaths per min. Serum sodium was 128 mEq/l, potassium was 4.1 mEq/l, BUN was 65 mg/dl, creatinine was 2.0 mg/dl, hemoglobin was 11.5 g/dl, and hematocrit was 32%. On arrival in the operating room, an iv catheter was in place and working well. Monitoring with an automated blood pressure cuff, ECG, apical stethoscope, pulse oximeter (Sp_{O_2}), and mass spectrometer was established. While the patient breathed 100% oxygen *via* mask, anesthesia was induced with 1 mg midazolam and 100 mg ketamine, and maintained with 1 mg midazolam and 50 mg ketamine. She remained hemodynamically stable with an Sp_{O_2} of 99–100%. Spontaneous ventilation was continued throughout the procedure, and blood loss was estimated at 200 ml. After the 10-min operation, she was taken to the recovery room fully conscious but disoriented. After an uncomplicated 2-h recovery room stay, she was fully alert and oriented. On return to her hospital room, spontaneous diuresis of large volumes of urine ensued. Over the next few days, serum potassium decreased to a nadir of 2.9 mEq/l, and she was treated with oral potassium supplements. Otherwise, the electrolyte abnormalities, ascites, and pleural effusions resolved uneventfully. All parameters returned to normal by the sixth postoperative week.

DISCUSSION

Ovarian hyperstimulation syndrome following ovulation induction with HCG is clinically evident in 11% of cases and identifiable by ultrasound in 44% of cases.[2] Between 0.4 and 4% of treatment cycles will result in development of severe OHSS.[3,4] The pathophysiology of OHSS represents an entire spectrum of abnormalities. The most common form is called mild OHSS. It consists solely of elevated estrogen and progesterone concentrations but lacks significant clinical signs and symptoms. This form is self-limiting and usually resolves with the onset of menses. Moderate OHSS is recognized clinically by abdominal distension, nausea, vomiting, diarrhea, ovarian

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