A case study is presented of a 40-year-old female who underwent an elective abdominoplasty with the administration of general endotracheal anesthesia. The intra-operative course was complicated by an unsuspected, undiagnosed bleed from a branching artery at the bifurcation of the abdominal aorta, first retroperitoneally, then into the peritoneal cavity. The peritoneal hemorrhage was unrelated to the surgical procedure, because the abdominal cavity was not entered during the elective procedure.

Introduction
Hypotension is a symptom of all forms of shock: hypovolemic, cardiogenic and hyperdynamic. Rosenthal defines shock as "a syndrome of failure of the heart to pump blood into the aorta in sufficient quantity or under sufficient pressure to maintain the pressure-flow relationship for adequate tissue perfusion." Trauma is responsible for most cases of acute hemorrhagic shock; however, the spontaneous rupture of vascular aneurysms in the chest or abdomen can be another cause. A patient who presents with severe hemodynamic instability and shock requires some estimation of existing pathophysiology.1

Atherosclerosis and thrombophlebitis are the disease processes responsible for the majority of vascular pathology. Atherosclerosis primarily involves the arterial system and has a predilection for arteries with turbulent flow. Progression of the disease eventually results in arterial occlusion or aneurysmal dilatation secondary to loss of structural integrity of the arterial wall.2 The majority of arteriosclerotic aneurysms occur in older patients, those in the sixth or seventh decades of life, and are 10 times more frequent in men than women.3

The ability to obtain a thorough and complete history is of vital importance in establishing a diagnosis and selecting the proper treatment. An iatrogenic-induced unresponsiveness from anesthesia, negligible blood loss from the operative site and an essentially benign preoperative history were some of the many obstacles the author encountered in reaching a differential diagnosis of spontaneous peritoneal hemorrhage.

Case report
A 40-year-old, 59 kg, 167 cm Chilean female ASA I with abdominal panniculus and diastasis rectus presented for an elective abdominoplasty. Her medical history was essentially benign. Her surgical history included a hernia repair at age 10, an appendectomy at age 15 and removal of renal calculi at age 32. All prior surgery had been uneventful and performed while the patient was under general anesthesia. The patient was not on any medication and denied any known allergies to drugs. Preoperative laboratory data taken two days prior to surgery showed hemoglobin 13.6 g/dl, hematocrit 39.6%, platelets 276,000, sodium 139, potassium 3.7, chloride 108, CO₂ 25, glucose 79, blood urea nitrogen 11, pt 10.5/9.6 and ptt 31.6/27. Urinalysis revealed trace ketones. Vital signs were blood pres-
sure (BP) 105/68 mmHg and a pulse rate (PR) of 66 bpm.

On the day of surgery, the patient presented to the operating room, alert, unmedicated and voicing no complaints. A 16-gauge intravenous (IV) catheter was easily inserted into her left hand, and an infusion of lactated Ringer’s was started. Diazepam 5 mg IV was administered, with an appropriate response noted. On admission to the operating room, the patient was placed on the operating room table in the supine position, with bilateral armboards for support. Initial monitors included an ECG on lead II, automatic blood pressure cuff (Dinamap®), precordial stethoscope, oxygen analyzer, peripheral nerve stimulator, end-tidal CO₂ and a pulse oximeter attached with a finger probe. Immediate preinduction vital signs were BP 134/84, PR 86 and an SaO₂ of 99%. Oxygen 100% was administered for a period of 5 minutes, during which the patient received additional diazepam 5 mg IV and fentanyl 100 μg IV. The induction of general anesthesia was accomplished with sodium thiopental 250 mg.

Intubation was facilitated with atracurium 30 mg, and in an attempt to help blunt the cardiovascular response to intubation, 0.5% enflurane was administered via mask. Immediate preintubation vital signs were BP 106/54 and PR 80 bpm. A 7.0 mm endotracheal tube was inserted under direct visualization without difficulty. Negative abdominal sounds were auscultated with ventilation, while breath sounds were bilateral and equal over the lung fields. The immediate postlaryngoscopy and intubation BP was 138/90 mmHg and heart rate (HR) was 112, with the ECG monitor showing a normal sinus rhythm.

During the surgical preparation period, a Foley catheter was inserted, along with a nasal temperature probe, and a condenser humidifier was placed into the breathing circuit. The operating table was placed in the Fowler’s position, with the back elevated 30 degrees. Prior to placement of the surgical incision, the patient received an additional 600 μg of fentanyl, and the N₂O concentration was increased to 66%. The mechanical ventilator settings indicated a tidal volume of 850 cc at a rate of 6 breaths per minute, with peak inspiratory pressures noted to be 20 cm/H₂O. During the 20-minute interval from intubation to incision, the vital signs had stabilized at BP 90-100/46-56 and PR 60-66. Approximately 10 minutes after the surgical incision was made, the patient received cefazolin II gm IV push, at the request of the surgeon.

Within 5 minutes of receiving the antibiotic, hypotension was first noted. The BP was 68/48; the PR 72. Lung fields were auscultated and found to be clear and equal bilaterally, with an SaO₂ reading of 99% and an ETCO₂ of 28 mmHg. The rate of IV fluid infusion was increased; the enflurane was discontinued; and ephedrine 10 mg IV was given. The only noted change in vital signs was an increase in HR to 100 bpm. A second dose of ephedrine 10 mg IV push was administered within 10 minutes after the first dose, and the N₂O/O₂ concentration was changed to 50%. With still no change in BP and a neosynephrine infusion was being prepared, a third dose of ephedrine 10 mg IV was given. The patient’s PR increased to 150, and BP increased to 98/52. At this point, the patient had received 2 L of IV fluids, had a urine output of 100 cc, and the surgeon had removed the excess abdominal tissue. The surgical field blood loss had been minimal.

Maintaining the operating room table in its original position in order to facilitate closure of the skin, the bed was brought into a Trendelenberg position. Attempts to insert a radial arterial line were unsuccessful bilaterally, although initially a radial pulse was palpable. A large bore IV catheter was inserted into the left external jugular vein, and additional lactated Ringer’s was infused. While the neosynephrine infusion was started, neosynephrine 100 μg IV push was given twice. When no significant response was noted, the N₂O was discontinued. Scopolamine 0.2 mg IV was administered in order to minimize any recall.

Additional surgical support was provided to obtain an arterial cutdown and to expedite closure of the surgical incision. However, the radial arteries were so constricted that not only was bilateral identification of the radial arteries difficult, but once a 20-gauge catheter was inserted, no blood could be aspirated. From the surgical field, attempts to obtain or even locate the femoral artery were unsuccessful. At this point, the BP, via Dinamap®, was averaging 30-40 torr systolic, but a carotid pulse was always present. A “wide open” dopamine infusion, at a concentration of 8 μg/cc, was also started. In order to eliminate any possible adrenocortical hypofunction, hydrocortisone 100 mg IV push was given twice.

Throughout the entire course of events, the patient did not move, the peripheral SaO₂ remained at 98% and the cardiac monitor showed a sinus tachycardia. The abdominal skin edges were approximated and closed without difficulty. The abdomen was not noted to be distended.

After closure of the wound, approximately 130 minutes after incision, a 12-lead ECG, followed by a chest x-ray, was obtained to rule out any cardiac etiology. A right subclavian line was inserted, and blood for multiple laboratory tests was obtained.
Simultaneously, upon receiving the hemoglobin and hematocrit results of 3.9 g/dl and 9.4%, respectively, the patient’s abdomen became distended. The patient was responsive to deep pain and responding to simple commands with the nod of her head. The BP was fluctuating between 60-110 systolic, with PR between 130-150 bpm.

Immediately, type-specific blood was infused under pressure through the three IV lines, and the patient was prepared for an exploratory laparotomy. The only additional medication administered was scopolamine 0.2 mg IV. With placement of the laparotomy incision, the patient flexed her legs and turned her head. Her BP was 102/44. As needed, additional muscle relaxation was given. Upon entering the abdomen, approximately 3,000 cc of blood was evacuated. A retroperitoneal hematoma was noted, and the retroperitoneum was opened. The aorta was cross clamped. The site of hemorrhage, an avulsion of a branching artery at the bifurcation of the abdominal aorta, was identified. The rent was oversewn, and total cross clamp time was 21 minutes. As the intravascular volume was replaced, the anesthetic requirement increased. The neosynephrine infusion was discontinued, and the dopamine infusion was continued at low dose for its dopaminergic effect.

Upon completion of surgery, 326 minutes from the initial incision, the patient was transferred to the intensive care unit, nonalert, intubated and on mechanical ventilation. Vital signs on admission to the intensive care unit (ICU), were BP 92/60 torr and HR 108 bpm. Intra-operative fluids totaled 18,750 cc of crystalloid; lactated Ringer's 14,250 cc, normal saline 4,000 cc and D5W 500 cc, plus 6 units of packed red blood cells and 2 units of fresh frozen plasma. The total urine output for the case was 450 cc. The initial complete blood count, on admission to ICU, was white blood cell 14,200, hemoglobin 10.9 g/dl, hematocrit 32.9% and platelets 295K.

Within 30 minutes after admission to ICU, the patient was alert and able to provide some late but valuable information. By asking her yes and no questions and utilizing the patient’s ability to point to her right lumbar region, it was learned that she had been experiencing intermittent episodes of right lumbar discomfort for approximately 3 weeks. Later it was learned that the patient and her spouse had attributed the discomfort to muscle strain, since the initial onset occurred while chopping wood. Six hours after surgery, the patient met extubation criteria and was extubated without difficulty. Her remaining hospital course was complicated by discovery of a right pneumothorax, requiring a chest tube on postoperative day 4. Otherwise, the patient’s progress was satisfactory, and she was discharged home on her 15th postoperative day.

Discussion

There are many causes for hypotension during general anesthesia, ranging from excessive blood loss—usually associated directly with the operation performed—to some obscure pathology, as is found in certain endocrine disorders. Initially, one attempts to rule out the most common and/or obvious causes. With a PR in the 60s and a systolic BP of approximately 90 torr immediately prior to placement of the surgical incision, the possibility of an overdose of anesthesia for this particular patient was eliminated. Also, hypovolemia from fluid or blood loss was ruled out, because the estimated blood loss was negligible when the hypotension began. The patient’s fluid deficit had been replaced with IV fluids.

The first significant decrease in BP was noted within minutes of the patient receiving IV antibiotics. Although the incidence of anaphylactic reactions during anesthesia in the United States is unknown, the occurrence in Australia is between 1:5,000 and 1:25,000 anesthetics, with a mortality rate of 3.4%. Cardiovascularly, hypotension followed by sinus tachycardia, without dysrythmias, was the first clinical sign of instability. The hypotension could have been attributed to hyperdynamic shock. When hyperdynamic shock is secondary to anaphylaxis, there may be a relative hypovolemia present (caused by vasodilation), along with an absolute hypovolemia (due to increased capillary permeability) resulting in a loss of circulating volume. Since the patient exhibited no cutaneous or respiratory signs of anaphylaxis, the possibility of an allergic reaction seemed unlikely.

With a negative cardiac history and low cardiac risk factors, cardiogenic shock also seemed unlikely, but possible. Although the electrocardiographic changes seen with ischemia are usually detected after ventricular pathology has occurred, there were no ischemic changes or ectopy noted on the 3-lead ECG. Initially, the benefit of an intra-operative 12-lead ECG was not feasible without seriously compromising sterile technique and risking major wound contamination. It was at the completion of surgery, when access for invasive cardiac monitoring was being secured and a 12-lead ECG was being taken, that the diagnosis of an intra-abdominal hemorrhage was made. The distended abdomen, along with the patient’s multiple laboratory values, provided the definitive answer. The abdomen, just
minutes before, was flaccid enough to allow for approximation of skin edges after an abdominoplasty.

In retrospect, it was probably during intubation that the previously contained retroperitoneal bleed ruptured into the peritoneal cavity. Once rupture into the peritoneal cavity occurs, mortality approaches 100%2.2 Efforts to obtain a diagnosis of an intra-abdominal bleed obviously were hampered by general anesthesia, patient positioning, extraperitoneal surgery and a negative history. Also overlooked in this patient was one of the most frequent causes of circulatory inadequacy in a young, healthy patient: hypovolemic shock secondary to hemorrhage.

REFERENCES


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