

increases. Morphine blunts this response and thereby necessitates an even greater increase in PaCO_2 to stimulate ventilation. In our case, the patient was awakened at 4 AM for dressing changes. The pain and anxiety associated with this stimulation may have caused him to self-administer the maximum amount of morphine (42 button pushes and 15 mg of morphine). Once the dressing was completed, the patient went back to sleep, after having receiving a relatively large amount of morphine. This, coupled with his compromised airway, can easily account for his near-fatal respiratory distress.

Third, although this patient was in the SICU, he was not monitored adequately. A review of his chart found that during the critical 2 h before the patient's respiratory distress, respiration was not monitored or charted because of a nursing shortage and emergencies in other parts of the unit.

Morbid obesity frequently is associated with respiratory impairment. In patients who suffer from the Pickwickian or obesity hypoventilation syndrome, arterial blood gas analysis reveals hypoxemia and hypercarbia²; for this reason, it can be argued that these patients are not candidates for PCA. However, those suffering from obstructive sleep apnea syndrome frequently have normal blood gasses while awake and may not appear to be short of breath.³

It is these patients who may experience difficulties with PCA.

How then should we deal with postoperative pain in the obese patient? It is these patients whose respiratory status benefit most from optimal analgesia, particularly after abdominal and thoracic surgery. In retrospect it may have been safer for our patient to have received PCA only, instead of PCA plus a 2-mg/h basal infusion. In addition, the Bard PCA pump can be programmed with a 1-h limit on the amount of drug delivered; in our case this safety feature was not used. For obese patients, it may be prudent to use smaller boluses of drug or to employ longer lock-out times between injections. Finally, for very obese patients or for those who exhibit signs of sleep apnea syndrome, it may be wise to restrict PCA use to an intensively monitored setting or alternately to rely on nurse-administered injections of intramuscular analgesics.

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Hypotension during Transfusion of Autologous Blood

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Autologous blood transfusion is generally considered the safest and most preferable method of transfusion therapy, and its appropriate use is highly recommended.¹⁻⁴

Among its advantages is a decrease in the risk of transfusion-associated reactions, including those related to infection, alloimmunization, and incompatibility.^{1,2-4} However, this technique is not without hazards, which may include volume overload, bacterial contamination, and incompatibility due to clerical errors.^{2,5,6} We report a case of repeated hypotension in association with transfusion of autologous blood, a situation that we do not believe has been reported previously.

CASE REPORT

A 53-yr-old woman presented for cholecystectomy, open liver biopsy, and possible right hepatic lobectomy. A hepatic mass had been detected on an abdominal ultrasound obtained to evaluate epigastric pain and diarrhea. A computed tomograph confirmed the finding. Her medical history was remarkable for cholelithiasis, hypercholesterolemia, well-controlled hypertension, and palpitations believed to be caused by paroxysmal supraventricular tachycardia. She had previously undergone total abdominal hysterectomy and bilateral salpingo-oophorectomy for ovarian cancer and was not aware of any prior anesthetic complications. Her medications included verapamil, gemfibrozil, conjugated estrogens,

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and acetaminophen with codeine, and she reported allergies to tetracycline and radiographic contrast.

Her physical examination was unremarkable and included a regular cardiac rhythm, a heart rate of 80 beats per min, and blood pressure of 140/80 mmHg. She weighed 62 kg. Her hematocrit was 33.7%, and her serum calcium was 10.6 mg/dl. The patient's serum aspartate and alanine aminotransferases, alkaline phosphatase, bilirubin, creatinine, and prothrombin time all were within normal limits. Available in the blood bank were four autologous units of blood obtained within 6 weeks of surgery and six designated donor units of blood. All blood was preserved with the standard citrate, phosphate, dextrose, and adenine (CPDA-1) solution and stored at 4 °C. No units were frozen or otherwise processed. As far as we could determine, the patient was afebrile and well during the period of her donations.

Anesthesia was induced with fentanyl and thiopental. The patient's trachea was intubated after muscle relaxation had been achieved with vecuronium, and mechanical ventilation was initiated. Anesthesia was maintained with isoflurane and N₂O in O₂ supplemented with fentanyl; relaxation was continued with vecuronium. Radial artery and internal jugular catheters were inserted without difficulty.

The patient received 1 g cefazolin at the time of incision. Her cholecystectomy proceeded uneventfully. Because of the patient's history of contrast allergy, 50 mg diphenhydramine was administered before the use of 17 ml 50% diatrizoate meglumine (Renograffin[®]) for cholangiograms. Intraoperative ultrasonography revealed a 2.2-cm mass in the right lobe of the liver, and an excisional biopsy was undertaken.

The patient's hematocrit was 29% as this phase began. During the resection, the patient lost approximately 300 ml of blood in 10 min. Although the bleeding had been well controlled, because of the brief time period over which the loss had occurred and the expectation of continued venous bleeding, one unit of autologous packed red blood cells was transfused through a fluid warmer over approximately 5 min. Initially, with transfusion, the patient's blood pressure increased from 95/60 to 110/65 mmHg. However, 1 min after the blood transfusion was begun, her blood pressure decreased precipitously to 50/30 mmHg; her heart rate was unchanged at 70 beats per min. The hypotension was confirmed by sphygmomanometer. At this point, there was no significant bleeding nor was there compression of the inferior vena cava. The patient was not flushed, and there was no hematuria. The end-tidal CO₂ tension (P_{ET}CO₂) was unchanged, and the electrocardiogram demonstrated sinus rhythm without ST-segment, QT-interval, or T-wave changes (in modified lead V5). Her temperature remained 35.4° C. With the infusion of 300 ml crystalloid and 5 mg ephedrine and a decrease in the inspired concentration of isoflurane, the patient's blood pressure increased to 120/65 mmHg within 1 min of its initial decline and remained stable. The blood transfusion was completed without further problem.

One half hour later, the estimated total blood loss was 400 ml, and the patient's hematocrit was 28%. Because further venous oozing was expected postoperatively, transfusion of a second unit of autologous blood was begun as closure of the incision commenced. This unit was whole blood. At approximately the same time, 1.25 mg droperidol was given as an antiemetic, and 24 ml 0.25% bupivacaine was infiltrated subcutaneously at the incision. After transfusion of about one half (250 ml) of the unit of blood over 10 min, the patient's blood pressure decreased over approximately 3 min from 100/55 to 75/45 mmHg despite a decrease in the isoflurane concentration. Her heart rate increased from 60 to 75 beats per min. There was no flushing, hematuria, abnormality of the electrocardiogram, or change in the end-tidal concentration of CO₂. The patient's temperature was 35.2° C. The transfusion was stopped, and the patient's blood pressure was stabilized at 100/60 mmHg within 1 min after administration of 1 l crystalloid and 10 mg ephedrine.

The remainder of the procedure and the patient's postoperative course were uneventful, except for a mild urinary tract infection. Her

hematocrit and serum calcium concentration immediately after surgery were 32.9% and 7.5 mg/dl, respectively. The postoperative nadir of the hematocrit was 27.6% 11 h after surgery. There were no further transfusions. The final pathology of the tumor was focal nodular hyperplasia.

Inspection of blood bank records and the labels on the transfused units revealed no clerical errors. The patient's ABO and rhesus factor (Rh) type and that of the residual in the bags of transfused blood were rechecked and were identical. Tests for free hemoglobin in the patient's blood and urine and a direct Coomb's test on her blood performed within 3.5 h of the second transfusion were negative. Bacterial cultures of the blood remaining in the bags also were negative. A review of the records of the monthly quality control tests on the blood warmers in the operating room indicated that all warmers were functioning normally during the time of the patient's operation.

DISCUSSION

Physicians often do not apply the same criteria for transfusion of autologous blood as for transfusion of homologous blood and are more likely to use the former if it is available.^{2,6-8} In fact, some physicians transfuse all available units of autologous blood in an effort to return all donated blood to the patient.^{6,7} However, while the use of autologous blood avoids many of the potential problems associated with homologous blood, it is not without hazard. In addition to reviewing the mechanisms by which blood transfusion can cause hypotension (table 1), the following discussion highlights some of the risks of autologous blood.

Hypotension associated with blood transfusion can be mediated by the immune system. The most dreaded immune-mediated reaction is the hemolytic transfusion reaction from ABO incompatibility, and hypotension is one of the main clinical manifestations of such a reaction.⁹ In the anesthetized patient, hypotension, hemorrhagic diathesis, and hemoglobinuria are the only clues to an incompatibility reaction.^{9,10} Even units of supposed autologous blood are susceptible to this catastrophe, because 61% of fatal transfusion reactions are the result of clerical errors, such as the switching of labels.¹¹ In our case, confirmation of correct typing and crossmatching as well as negative tests for direct Coomb's antibody and free hemoglobin in the recipient's blood and urine excluded a hemolytic reaction.

Hypotension is also one of the main presenting signs of another immune-mediated reaction—anaphylaxis to

TABLE 1. Causes of Hypotension Associated with Blood Transfusion

Hemolytic transfusion reaction
Anaphylaxis caused by anti-IgA antibodies
Prekallikrein activator in plasma protein fraction
Bacterial contamination*
Hypocalcemia (citrate intoxication)*
Hypersensitivity to plasticizers, sterilizers, and stabilizers in bags and tubing*
Venous air embolism*

* Causes that can be associated with autologous blood transfusion.

plasma proteins.^{12,13} Several cases of anaphylactic reactions from interaction of recipient anti-immunoglobulin A (IgA) antibodies in IgA-deficient patients against donor IgA have been reported.¹³ Anti-IgA of limited specificity has also been implicated as a cause of hypersensitivity reactions in patients with normal serum concentrations of IgA.¹⁴ There have been other cases in which the cause of an apparent hypersensitivity reaction could not be determined.¹⁵ None of these mechanisms explains the hypotension in our case, because postoperative laboratory tests and reexamination of all clerical data confirmed that the blood was indeed autologous.

Hypotension, vasodilatation, nausea, and sweating have been noted after the rapid administration of plasma protein fractions. Prekallikrein-activator, a degradation product of factor XII (Hageman factor), is presumed to produce this effect by triggering the release of bradykinin in recipients.¹⁶ Immunoglobulin preparations and factor VII concentrates may contain prekallikrein activator, but packed red cells and whole blood do not undergo extensive processing involving contact with foreign surfaces, and thus do not contain prekallikrein activator.¹⁷

Bacteremia is a rare complication of blood transfusion that should be considered in any severe, unexplained transfusion reaction. Transfusion of infected blood can cause immediate shock, presumably from bacterial endotoxins or an interaction between natural antibodies and bacterial antigens.^{18,19} Bacteria can be introduced into a unit of blood during collection,¹⁹ or they can enter the plastic bags through pinholes.²⁰ The most common contaminants of stored blood are *Pseudomonas* species as well as coliforms and achromabacters.²¹ In addition, *Yersinia enterocolitica* in donor blood has been reported to cause fever and hypotension in recipients.²² Cultures at 35° C from the remaining blood in the bags used for transfusion in our case were negative. However, 25% of bacterial contaminants are missed by culture at this temperature and are detected by culture only at 20–25° C.²³ Perhaps we failed to detect such contamination in our case, but it is unlikely that both units were infected.

Rapid administration of blood can lead to hypotension due to the transient hypocalcemia caused by the citrate used as an anticoagulant in banked blood.¹⁰ However, Denlinger *et al.* showed that in patients free from hepatic or renal disease and undergoing radical cancer surgery, whole blood given as fast as 150 ml · 70 kg⁻¹ · min⁻¹ did not decrease the mean arterial pressure by more than 10 mmHg.²⁴ They showed that the transient decrease in ionized calcium lasts less than 10 min and hypothesized that redistribution of citrate in extracellular fluid, possibly in

conjunction with hepatic and renal clearance, explains the time course. Our patient had normal liver and kidney function and a normal serum calcium preoperatively, and the transfusions were given at a rate lower than Denlinger's maximum rate. However, she was undergoing hepatic surgery, and we cannot exclude the possibility that this affected citrate clearance and contributed to the hypotension. Unfortunately, at the time of the procedure, our hospital did not have the capability to measure ionized calcium concentrations. However, it seems unlikely that her liver function would have been so globally affected during the procedure. Moreover, we did not note QT-interval prolongation.

Verapamil also is capable of causing hypotension and bradycardia.²⁵ However, the patient had been taking the drug for an extended period without ill effect. Although the verapamil may have contributed to the hypotension, perhaps by blunting reflex tachycardia, it seems unlikely that it was the primary agent. Another possibility is that the verapamil exaggerated any transient hypocalcemia resulting from rapid transfusion, but we are not aware of any evidence of this phenomenon in the literature.

The plasticizer di(2-ethylhexyl)phthalate (DEHP), which is mixed with the polyvinyl chloride used to make blood storage bags, can leach into stored blood. A metabolite of DEHP has been reported to cause hypotension and bradycardia in rats.²⁶ However, the blood concentrations necessary to cause hypotension should occur only during massive transfusion of blood that has been stored more than 35 days.²⁶ We transfused only one and one half units.

Given the patient's history of allergic reactions, a hypersensitivity reaction to unidentified allergens in the blood bags remains a possibility. Sterilizers and stabilizers used in the plastic bags that hold blood include ethylene oxide and organic compounds containing lead, barium, cadmium, and tin.²⁷ Acute hypersensitivity reactions to the ethylene oxide used to sterilize plastic bags and tubing have been reported in hemodialysis patients, plateletpheresis donors, and a recipient of factor VIII.^{28–30} In the patient who received the factor VIII transfusions, severe hypotension was reported. Finally, Morse *et al.* have suggested that the products of complement activation are present in blood products and theoretically may cause anaphylactoid reactions when infused.³¹

Of course, the hypotension may have been caused by something other than the blood transfusions. The surgical blood loss had ceased by the time the first transfusion was begun, and the blood pressure had begun initially to increase with volume expansion. The ongoing blood loss was minimal during the second transfusion, which corresponded with surgical closure.

It has been confirmed that traction on abdominal mesentery can cause a decrease in blood pressure, probably

† Swierczynska Z: Natural antibacterial antibodies as a cause of shock following blood transfusion. *Schweiz Z Path Bakt* 20:211–218, 1957

through a decrease in systemic vascular resistance caused by prostacyclin release from the bowel under traction.³² This may explain only the first instance of hypotension in our case, because the second occurred during closure of the wound.

Air embolism from the surgical field or from air introduced inadvertently through the transfusion tubing can lead to hypotension.^{33,34} Less than 100 ml is required by the latter route to cause cardiovascular collapse.³⁴ No change in PET_{CO_2} was noted to support venous air embolism in our case.

Intravascular injection of contrast media also can result in hypotension; the risk of reaction during intravenous cholangiography is about 8%.³⁵ Even if inadvertent intravascular injection of contrast occurred in our case, this would not explain the second episode of hypotension.

Depending on the dose administered, intravascular injection of bupivacaine can cause an increase or decrease in blood pressure,³⁶ but this would not be pertinent to the first instance of hypotension. Furthermore, bupivacaine is well known to cause ventricular dysrhythmias,³⁶ but none was noted in our case.

Droperidol can occasionally cause severe hypotension because of its effects on the central nervous system and its peripheral α -adrenergic blockade.³⁷ It is unlikely that droperidol caused hypotension in our case because it was not given in association with both episodes of decreased blood pressure, and the patient received two subsequent doses of droperidol in the recovery room without adverse effect.

In summary, we have presented a case in which two episodes of hypotension were temporally associated with transfusion of autologous blood. Although we have not been able to determine the exact cause of the hypotension, an allergic or anaphylactoid reaction seems most plausible, given that all other likely mechanisms have been excluded. Although rare, allergic and anaphylactoid phenomena have been reported, as explained above.²⁸⁻³¹ Immunoglobulin and complement concentrations from the patient's serum might have given further indirect evidence of such reactions, but they were not obtained. However, more importantly, the case illustrates that although it may avoid many of the risks of homologous transfusion, autologous blood may not be without danger. Patients are still susceptible to bacteremia from infected units, hypersensitivity reactions, air embolism, citrate intoxication, and volume overload. Perhaps most sobering is that clerical errors can still occur with autologous blood, and that these account for over 60% of the fatal transfusion reactions reported.

Guidelines have been published recently for perioperative blood transfusion, but they do not address the question of whether autologous and homologous blood should be treated differently.³⁸ We believe that less strin-

gent indications for transfusion of autologous blood are appropriate because it is safer than homologous blood. However, one should avoid using autologous blood for transfusion merely because it is available but rather, just as with homologous blood, choose it only when benefit to the patient is likely. It should be used only when significant blood loss has occurred, when further bleeding is anticipated, or when signs or symptoms of anemia are present.

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Transient Paraplegia during Posterior Cervical Osteotomy

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A procedure for correction of severe cervical kyphosis performed under local anesthesia provided an opportunity for unique observations regarding the progression of evoked response changes and physical signs and symptoms during the evolution and regression of paraplegia.

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CASE REPORT

The patient was a 62-yr-old man with ankylosing spondylitis and a severe cervical kyphosis. He was scheduled for posterior cervical osteotomy and realignment of the neck. Six months prior to the time of surgery, he had sustained a fracture at C7 in association with a fall. That injury resulted in an aggravation of the kyphosis and in numbness and decreased sensation in a right C6 distribution. He was otherwise neurologically intact. The cervical kyphosis was estimated to be 90° (chin-brow angle) and was such that his chin rested continually on his sternum. Mouth opening was limited and his ambulation was impaired because he was unable to look forward. His only regular medication was baclofen. He had last undergone a general anesthetic 6 yr prior to the current procedure. That anesthetic was reported by the patient to have been uneventful, and he could not recall the details of airway management. The anesthetic and surgical plan was to perform the procedure under local anesthesia and to be prepared to induce a brief period of general anesthesia if the realignment maneuver (which entails elective fracturing of the heavily calcified anterior longitudinal ligament) was not tolerated in the awake state. Somatosensory evoked response (SSER) monitoring was to be performed in order to permit