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Medical Intelligence

Intraoperative Autotransfusion

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TRANSFUSION WITH HOMOLOGOUS BLOOD, when it is available, is associated with a significant incidence of complications. In addition, patients whose bloods for various reasons cannot be cross-matched may be denied elective surgery; others may succumb to massive blood loss when it is not possible to provide the volume of blood required. The bleeding patient is the most readily available source of compatible blood free of hazard of transmission of disease or isosensitization to formed elements. Autotransfusion is not a new practice; rather, it is becoming a sophisticated technique based on experience gained over the past 150 years.[36]

Historical Review

In 1818, James Blundell of Guy's Hospital, London, autotransfused ten women suffering from severe postpartum hemorrhage. Five of the patients lived.[2] William Highmore[17] is credited with bringing the procedure to the attention of the medical profession in a communication to *The Lancet* in 1874. Reporting independently in 1885 and 1886, Miller[25] and Duncan[11] cited cases of reinfusion of blood shed during lower-extremity amputations. In 1914, H. Johannes Thies,[35] of Liepzig, published the first case reports of autotransfusion in three women with ruptured ectopic pregnancies. Blood removed from the abdominal cavity was mixed with salt solution and administered into the substance of the thigh in the first case, while an arm vein and a vein in the omentum were used for infusion in the other two patients. In 1917, Elmendorf[14] reported the successful intravenous infusion of 300 ml of blood obtained from a young soldier's hemothorax.

Autotransfusion was introduced into the United States in 1917, when Lockwood[22] returned intravenously 750 ml of blood lost during splenectomy for Banti's disease. A similar case was reported by Burch[8] in 1922. The latter also recommended that contaminated blood not be thrown away, but be administered as a rectal drip.[7] Davis and Cushing,[10] in 1925, reported 22 cases of autologous blood replacement during or after major intracranial operations. Blood was suctioned from the wound, mixed with a few milliliters of sodium citrate solution, filtered through 40 or 50 thicknesses of sterile gauze, refiltered, and infused into a saphenous vein or a vein in the antecubital fossa. The technique allowed complete extirpation of meningiomas at one operation. Cushing's patients also received a continuous rectal saline drip. A review of autotransfusion in gynecology was published in 1923 by Farrar.[15]

In 1943, Griswold and Ortner[16] reported 100 cases of trauma to the abdomen or thorax in which autotransfusion was used. They associated one death with faulty technique. The patient sustained eight perforations of the small intestine, three of the descending colon, and two of the rectum. He died 62 hours after autotransfusion of 2,000 ml of blood. The infusion needle was found to be plugged with feces.

In a desperate attempt to save a young Samoan woman in profound shock from hemorrhage due to placenta previa, Leiato, in 1956, collected the vaginal blood in a sterile obstetrical basin. Anticoagulants were not available. The blood, therefore, was diluted with saline solution and infused intravenously. The patient, who previously had

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had no recordable blood pressure, was delivered of a normal viable infant and made an uneventful recovery. She received no homologous blood.[32]

More recently, Pathak and Stewart[25] reported the successful autotransfusion of more than 500 patients with ruptured ectopic pregnancies. Transfusion bottles containing ACD solution, standard plasma aspirating sets, a large bowl, and a conventional operating room suction machine were the only equipment needed to salvage and reinfuse intraperitoneal blood.

Autotransfusion of blood from acute hemothorax is rather extensively practiced and requires minimal equipment. Chest tube drainage may be collected in standard infusion bags containing CPD preservative or into chest bottles containing only saline solution.[33,34]

Modern Autotransfusion

Dyer[12,13] and Klebanoff[20,21] are credited with development of current techniques of autotransfusion. Working with Bentley Laboratories, Klebanoff developed an autotransfusion system (ATS) that has disposable components (fig. 1). Initial trials with the new apparatus were carried out in Viet Nam, where the minimal risk and life-saving value of the technique were demonstrated.

The Bentley autotransfusion unit (ATS)§ is a modification of the basic cardiotomy reservoir employed in open-heart surgery. The sterile disposable unit and tubing are opened at the operating table. The surgeon retains the suction tubing and passes the remainder off the table. A single roller-head pump is attached to the inflow tubing, which allows aspiration of blood from the patient to the reservoir and provides pressure at the air–fluid interface for reinfusion. The reservoir is primed with 200–300 ml of balanced salt solution, the system is purged of air, and the two outflow lines are connected to the patient. (Special cannulas are not necessary. Conventional 14-gauge intravenous catheters are suitable.) Blood is pumped into the clear, spherical 1,500-ml-capacity reservoir, where it is defoamed and filtered through a 125-micron-pore nylon filter. Generally, the surgeon determines the proper aspiration rate while the anesthesiologist controls reinfusion. Maximum flow rate is 600 ml/min.

Anticoagulation may be accomplished by various methods. The authors add 3 units of heparin per ml of priming fluid. No additional heparin is administered. Systemic heparinization, employing 2–3 mg/kg body weight, is used in several centers.[5,31] When systemic heparinization is used, a neutralizing dose of protamine sulfate may be given prior to termination of the operation. This method is suitable for elective vascular surgery. However, it is inappropriate for the patient with multiple trauma who may have sustained a closed extremity fracture or head trauma. A suction device that provides regional anticoagulation has recently been introduced by Aaron and colleagues.[1] The vacuum created as aspirated blood passes through a metering Venturi device in the suction handle draws heparin into a mixing chamber. Protamine may be added to the venous-return line. As an alternate method of anticoagulation, CPD or ACD preservative may be added when the pump is primed, and incrementally during autotransfusion.

If the rate of aspiration exceeds the maximum reinfusion rate, excess blood may be collected in standard blood-collecting units containing CPD or ACD preservative. These units may be administered when additional intravenous lines are established.

With minimal prior training, a physician, nurse, or surgical technician can assemble the system in five minutes. The greatest potential hazard of the unit is air embolism. For that reason, two safety features have been incorporated. An alarm alerts the pump operator when the blood level in the reservoir reaches approximately 200 ml. As an added precaution, a "dead man's switch" necessitates the technician's exerting positive control to operate the system rather than having to clamp the lines in an emergency. Nevertheless, it is imperative that the individual responsible for the pump have no other duty to perform during the surgical procedure.

Indications for autotransfusion, methods of anticoagulation, and effects of blood components on organ systems, as well as contraindications to use of the technique, remain controversial.

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Indications for Autotransfusion

Autotransfusion may be indicated in the following conditions: 1) blunt or penetrating trauma to the chest or abdomen; 2) ruptured ectopic pregnancy; 3) elective surgery associated with large-volume blood loss (*e.g.*, hepatic lobectomy, aortic aneurysm resection); 4) rare blood type or antibodies precluding crossmatching of blood.

In operative procedures involving slow bleeding (oozing), salvage is not practical due to excess trauma to cellular elements by the suction tip.

Acceptance of the technique by patients of the Jehovah's Witness faith varies with the individual sect.

Clinical Experience

Initally, use of the autotransfusion system (ATS) was limited to patients with ruptured ectopic pregnancies or those who sustained severe trauma. However, autotransfusion is now being used in other emergency procedures, as well as a variety of elective operations where blood losses in excess of 1,000 ml are anticipated. The authors' experience at the University of Texas Health Science Center at San Antonio Teaching Hospitals is summarized in table 1. Neither anesthetic technique nor monitoring procedures were influenced by the use of autotransfusion. Most patients had central venous pressure catheters and several had arterial lines.

Analysis of the effects of autologous blood in laboratory models and on patient morbidity and mortality is clouded by concomitant use of homologous blood. In the authors' experience, the clinical course of the patient correlates best with the total volume of blood transfused, rather than with quantity of autotransfused blood. There was no death among 29 patients in whose cases the total volumes of transfused blood were less than 3,500 ml. However, the mortality rate was 81 per cent in the group of 26 patients who required more than 7 liters. The largest single autotransfusion was 40,000 ml, administered to a 22-year-old rancher who had been kicked in the abdomen by his horse. He had sustained irreparable hepatic damage and died on the operating table. There was no clinical evidence of pulmonary insufficiency that could not be attributed to lung contusion, trauma, or aspiration.

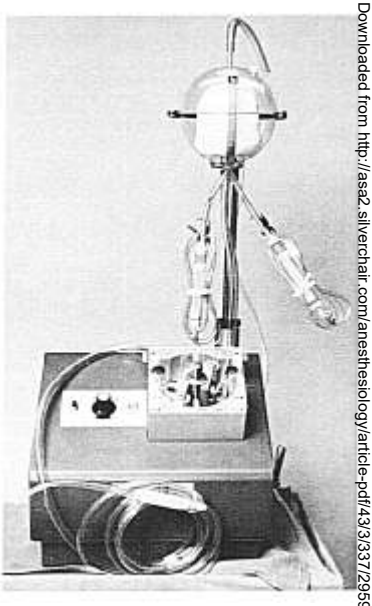

FIG. 1. Bentley autotransfusion system.

The following case reports are representative of the authors' experience:

Report of Two Cases

Patient 1. A 58-year-old white woman was admitted with a diagnosis of Felty's syndrome (hypersplenism, pancytopenia, and rheumatoid arthritis) of ten years' duration. In view of her progressive downhill course, splenectomy appeared to be indicated. Vital signs on admission were blood pressure 106/70 torr, pulse rate 98/min, and respiratory rate 22/min. There was evidence of marked weight loss and arthritic deformity of the extremities. The patient had a tracheostomy which had been performed for tracheal stenosis two years previously. Hematologic examination indicated marked anemia (hemoglobin 7.3 g/100 ml, hematocrit 22 per cent) and leukopenia (leukocyte count $0.9 \times 10^3/mm^3$). Examination of a peripheral blood smear revealed the platelets to be decreased in number and the erythrocytes to be grossly abnormal. The bone marrow was

TABLE 1. Summary of Clinical Experience with Autotransfusion

Operative Procedure		Volume of Autotransfusion (ml)		
Type	Number	Range	Average	Total
Ruptured ectopic pregnancy	13	500– 8,500	1,985	25,800
Penetrating trauma	22	600–14,000	4,045	89,000
Blunt trauma	13	500–40,000	6,754	87,800
Elective procedures:	13			
Laminectomy	1			
Portacaval shunt	1			
Hepatectomy	2	200– 8,500	2,462	32,000
Abdominoperineal resection	1			
Splenectomy	1			
Nephrectomy	1			
Miscellaneous emergency procedures				
Ventricular perforation secondary to cardiac catheterization	1			
Ruptured hepatic abscess	1			
Renal transplant–anastomotic breakdown	1			
Laparotomy–laceration secondary to liver biopsy	1	500–18,000	5,060	50,600
Ruptured aortic aneurysm	3			
Ruptured hepatoma	1			
Laparotomy post-vaginal hysterectomy	1			
Ruptured ovarian cyst	1			
TOTAL	71	200–40,000	4,017*	285,200

* Mean value.

hypercellular with a compensatory increase in granulocytic products. Prothrombin time was 13.1/11.9 sec, fibrinogen 65 mg/100 ml,[†] partial thromboplastin time 48/33 sec. Attempts at routine crossmatching of blood were fruitless. Subsequently, three isoantibodies (Kell, E_1, Fy) were identified, and a fourth unidentified antibody was suspected. It was ultimately possible to obtain two units of compatible whole blood after screening 150 units. In view of the size of the spleen, technical difficulty during the operative procedure was anticipated. The autotransfusion unit was therefore prepared for use on a stand-by basis. Estimated blood loss during operation was 2,000 ml. The patient received 950 ml of blood by autotransfusion together with the available 1,000 ml of bank blood and 2,000 ml of balanced salt solution. Immediately postoperatively the hemoglobin was 6.8 g/100 ml, hematocrit 22 per cent, free plasma hemoglobin 100 mg/100 ml, leukocyte count 0.46 $\times 10^3$/mm^3; urinalysis indicated a trace of free hemoglobin. The patient made a relatively uneventful recovery, with progressive improvement of the hemogram. Two months after splenectomy the patient died in acute respiratory failure.

Patient 2. A 28-year-old Mexican-American man sustained a gunshot wound to the right arm. The bullet traversed the right pleural space and exited in the left anterior axillary line. Upon arrival in the emergency room, the patient had a blood pressure of 60/0 torr, pulse rate 120/min, and respiratory rate 42/min. Breath sounds were decreased on the right and normal on the left. A thoracostomy tube was placed on the right, with immediate return of 1,500 ml of blood, which was saved. The initial hemoglobin was 12.0 g/100 ml, hematocrit 39.4 per cent, leukocyte count 15×10^3/mm^3. The patient received 2,500 ml of type-specific bank blood and was taken to the operating room for exploratory thoracotomy. A severe laceration of the right lung was evident and the right internal mammary artery was found to be transected. Estimated blood loss during the operative procedure was 5,200 ml. Twelve hundred milliliters of blood were aspirated into the wall suc-

[†] Hypofibrinogenemia is not a component of Felty's syndrome.

tion prior to preparation of the autotransfuser. The remaining shed blood, 4,000 ml, was returned to the patient. At the end of operation, oozing from all raw surfaces indicated a definite coagulopathy, but coagulation studies were not performed at this time. Blood replacement was obviously inadequate, and bank blood was not immediately available. Therefore, the 1,500 ml brought up from the Emergency Room were aspirated from the Thoraseal reservoir and returned to the patient. An additional 1,500 ml of chest-drainage blood collected in the Thoraseal unit in the recovery room was also autotransfused. The patient received 3,900 ml of balanced salt solution and eight units of fresh frozen plasma. Postoperatively the hemoglobin was 6.2 g/100 ml, hematocrit 17.7 per cent, prothrombin time 47 per cent, partial thromboplastin time 66/31 sec, and platelet count 72,000/mm³. Plasma free hemoglobin was 100 mg/100 ml. The patient received an additional three units of whole bank blood in the first three postoperative days. The hemoglobin rose to 14 g/100 ml and results of coagulation studies returned to normal. The patient subsequently made an uneventful recovery and was discharged on the eleventh postoperative day.

Effect of Autotransfusion on Blood Elements

Knowledge of hematologic integrity after autotransfusion is vital, but far from complete. Serial postoperative study of the hemogram is complicated by the acute-phase reactivity of leukocytes, platelets, and fibrinogen; hemodilution by crystalloid infusions; metabolic derangements induced by underlying disease, hypotension, and trauma; and changes associated with administration of homologous blood.

The composition, structure, and integrity of the formed elements of autotransfused blood vary with the conditions under which the blood is collected. Chemical and morphologic changes are induced during aspiration, compression by roller pumps, turbulent flow and foaming, air–fluid interfacing, and passage through filters. However, mechanical aspects of autotransfusion account for only a portion of cellular alteration. Normal defense mechanisms of serous linings of body cavities react to erythrocytes as foreign bodies by lysing them. The shed blood also loses coagulation factors.

While some rely heavily on comparative hematocrits, they have not been valuable in the authors' patients, since most received homologous blood as well as crystalloid infusions. Brener[6] found erythrocyte mass to be reduced by approximately 50 per cent in five patients who received an average of 4.5 liters of autotransfused blood during aortic resections. In a comparative postinfusion study of bank blood and autotransfused blood, Aaron *et al.*[1] found the hematocrit of the former to be 46.5 ± 10.5 per cent, while that of the latter was 17 ± 3.3 per cent. Erythrocyte counts were 5.0 ± 1.1 cells/cu mm and 2.0 ± .4 cells /cu mm, respectively.

Evidence suggests that erythrocytes that survive autotransfusion retain a normal life span and are capable of normal oxygen transport. In experimental and clinical studies, Symbas *et al.*[33] showed the half-survival time of autotransfused erythrocytes to be normal. Bennet *et al.*,[3] using ^{51}Cr tagging of erythrocytes, found the mean half-survival of autotransfused cells to be 17.5 days (SE = .7), compared with 17.0 days (SE = .9) for control erythrocytes.

Carty and Barr[9] studied the composition of intraperitoneal blood from 12 patients with ruptured ectopic pregnancies. The volumes of blood ranged from 1 to 3 liters, and had been in the peritoneal cavity for as long as 6 hours. Mean hemoglobin was 9.4 g/100 ml, packed cell volume 30 per cent, and mean corpuscular hemoglobin concentration 31 per cent. The mean corpuscular hemoglobin concentration values indicate that the hemoglobin in the blood from the peritoneal cavity was almost entirely intracellular and therefore potentially capable of normal oxygen transport.

Pathak and Stewart[28] autotransfused 530 patients with aspirated blood from the peritoneal cavity during laparotomy for ruptured ectopic pregnancies. The homoglobins of peritoneal blood ranged from 5 to 13 g/100 ml, with a mean of 8.1 g/100 ml while venous blood hemoglobins ranged from 6.1 to 13.2 g/100 ml, with a mean of 10.2 g/100 ml.

Plasma free hemoglobin was present in most of the authors' patients in whom determinations were made, the highest value being 250 mg/100 ml. Although mild to moderate hemoglobinuria occurred in most patients, there was no case with either clinical or laboratory evidence of postoperative renal failure.

Brener *et al.*[5] reported postoperative plasma

free hemoglobin levels of 50 to 600 mg/100 ml, with a mean of 175 mg/100 ml, in 20 patients autotransfused an average of 1.5 liters each. One-fourth of the patients had gross hemoglobinuria. Levels of free hemoglobin ranging from 496 to 1,696 mg/100 ml were present in blood sampled immediately distal to the roller pump. Plasma hemoglobin levels were normal in all patients by the second day. Aaron *et al.*[1] found plasma free hemoglobin levels to be 1,000 ± 625 mg/100 ml in aspirated blood. Free plasma hemoglobin levels of 350 per cent of control have been reported in blood aspirated from hemothoraces in dogs after 45 minutes of contact with the pleura. Plasma hemoglobin levels of blood left in the pleural cavity for 6 hours increased to 1,175 per cent of control.[23]

The amount of hemolyzed blood that can be infused safely is not known. There are at least three plasma-binding systems for free hemoglobin. The appearance of hemoglobinuria represents a saturation of the plasma-binding systems as well as an exceeding of the absorptive capacity of the proximal renal tubes. The absence of clinical hemoglobinuria implies only that maximal tubular absorption has yet to be reached, not that hemoglobin has not been taken up by proximal tubular cells. Only a small proportion of available free hemoglobin appears in urine; most of unbound hemoglobin is taken up in renal and hepatic parenchyma.[1] However, the hemoglobin molecule may not be nephrotoxic. In fact, stroma-free hemoglobin solution has been suggested as the "ideal" blood substitute.[29] Renal damage may, on the other hand, be related to intravascular coagulation secondary to the coagulant activity of the stromal element of hemolyzed blood.[1]

Coagulopathies occur in patients who are autotransfused. Clotting disorders are also prevalent in patients who receive large-volume homologous transfusions. Miller[26] has defined massive transfusion as the acute administration of more than one and a half times the patient's estimated blood volume. In a study of coagulation defects associated with massive blood transfusion in battle casualities, he and his colleagues found that patients manifested bleeding tendencies when 30 units of blood had been administered. They considered the primary etiologic factor to be dilutional thrombocytopenia.[27]

Clinical evidence of coagulopathy was apparent in 21 per cent of the authors' patients. Laboratory abnormalities consisted of low platelet count, prolonged prothrombin time and partial thromboplastin time, and, in one case, factor VIII deficiency. Of those who died, four were known to have had serious coagulation defects preoperatively, and six had sustained fatal wounds with uncontrollable hemorrhage. Most patients received fresh frozen plasma, and many received platelet infusions; therefore, precise analysis of the effects of autotransfusion on clotting is impossible. Platelet counts, prothrombin time, and partial thromboplastin time were significantly affected in many patients without clinical evidence of coagulopathy. All of these values returned to normal in the postoperative period.

Coagulopathy may be related to several factors. Platelets are trapped by filters in the system. Not all blood is salvaged; thus, some clotting factors are lost. Rapid utilization of platelets and clotting factors to levels below those necessary for hemostasis may occur in the syndrome of disseminated intravascular coagulation.[18] Reinfusion of hemolyzed erythrocytes may produce hypercoagulability followed by intravascular coagulation. Splenectomy has been shown to accentuate the latter phenomenon, and many trauma patients undergo splenectomy.[30]

In experimental studies, Bennett *et al.*[3] found that platelet counts were decreased, but not low enough to cause concern clinically. Rakower and Worth[31] reported that postoperative platelet transfusions have always been required in patients autotransfused with more than 6 liters. Aaron *et al.*[1] reported the platelet count of aspirated blood to be 348 ± 141 × 10^3 cu mm.

Quality as well as quantity of platelets must be considered. Are the remaining platelets similar to those in stored blood, or are they functional? Brener[6] found that platelets in aspirated blood do not aggregate. These platelets did not take up or release ^{14}C-serotonin in response to ADP or epinephrine—a more sensitive examination for platelet viability. However, following reinfusion of

as much as 4.5 liters of aspirated blood, circulating platelets functioned normally. Whether this was due to *in-vivo* metabolic restoration of the damaged platelets, sequestration of altered platelets by the spleen, or dilution with recipient normal platelets is unknown.

There is little doubt that platelet aggregates form following contact between shed blood and connective tissue. The screen filtration pressue (SFP) of blood passed at a constant rate through a 20-micron mesh screen has been shown to correlate with the presence of microaggregates.[4,22] Aaron *et al.*[1] found SFP's of both allotransfused and bank blood to be elevated. In canine studies, Bennett *et al.*[3] found that SFP decreased from an immeasurably high value to a measurable but elevated figure after filtration through the Bentley autotransfusion reservoir filter. Dacron-wool filtration was effective in bringing SFP of blood back to normal levels.

The deposition of platelet aggregates in the lung in the form of microemboli has been implicated in the syndrome of posttraumatic pulmonary insufficiency following transfusion with bank blood.[23] McNamara *et al.*[24] reported a relationship between the number of units of blood transfused and post-injury hypoxemia. The mechanism of pulmonary damage may be related to release of bronchoconstrictive and vasoactive substances from platelets and/or mechanical vascular obstruction. Bennett *et al.*[4] found that the majority of animals whose lungs were perfused with stored blood had microscopic evidence of interstitial pulmonary edema, vascular hemorrhage, intra-alveolar fluid, and alveolar congestion. Significantly fewer animals showed these changes when lungs were perfused with autotransfused blood. It was the authors' conclusion that autotransfused blood was significantly less injurious to the lung than was filtered blood stored for three weeks. Wright *et al.*[37] report autotransfusion of as much as four times the calculated blood volume in subhuman primates without significant development of microaggregates or pulmonary insufficiency. Rakower and Worth[31] found no example of the wet-lung syndrome, pulmonary edema, or persistent hypoxia when arterial blood gases and chest x-rays were examined several times daily in the early post-autotransfusion period.

Evidence that microaggregates formed in autotransfusion blood are composed primarily of adhesive platelets that deaggregate rapidly *in vivo*, whereas stored blood aggregates are composed of a variety of cellular particles that do not rapidly deaggregate, is accumulating.[4]

Reports relating intravascular coagulation to autotransfusion are sparse and conflicting. Brener *et al.*[5] state that fibrin and fibrinogen degradation products were present before autotransfusion in his patients and were not increased by the procedure. He and his coworkers found no evidence of disturbances in coagulation in 20 patients autotransfused during abdominal aortic resections.[6] Rakower and Worth,[31] on the other hand, in studying both canine models and patients, found that massive autotransfusion was always associated with defibrination. In intraoperative autotransfusion experiments with baboons, Kingsley *et al.*[19] found a decrease in fibrinogen, high levels of fibrin split products (FSP) and a hypocoagulable state, suggesting DIC, with consumption of clotting factors and fibrinolysis. The addition of cell-washing plasmapheresis eliminated FSP, but a hypocoagulable state also resulted, presumably from loss of clotting factors during plasmapheresis. In evaluating the effects of autotransfusion on blood elements of autotransfused dogs, Bennett *et al.*[3] found fibrinogen levels were increased rather than decreased. In experimental and clinical studies of autotransfusion from hemothorax, Symbas[33] found markedly decreased fibrinogen levels. However, the levels were elevated in many dogs and in several patients after the second postinfusion day, presumably a response to hemorrhage, trauma, or both.

Contraindications to Autotransfusion

Relative contraindications to the use of autotransfusion include: 1) Gastrointestinal tract contamination of salvaged blood. In experimental studies, Klebanoff *et al.*[21] introduced 10 ml of solid fecal material during autotransfusion. Blood cultures of all dogs were positive during the immediate post-

transfusion period, and negative at the end of 24 hours. Half of the animals received 250 mg chloramphenicol; half received no antibiotic. All animals survived.

Several of the authors' patients sustained wounds involving the stomach, liver, kidney, or small intestine. The majority had blood cultures immediately postoperatively. Results were consistently negative.

2) Concomitant distant fractures or suspected head trauma (when systemic heparinization is employed).

3) Presence of cancer cells. One of the authors' patients underwent hepatic lobectomy for metastatic Wilms' tumor. Two thousand milliliters of blood were autotransfused; no homologous blood was administered. Although the child made an uneventful recovery, he subsequently developed multiple pulmonary metastases. What role autotransfusion played is unknown.

Summary

Criteria for use of autotransfusion are still being delineated. After reviewing the experience of others and employing autotransfusion in 71 patients, to whom 285,200 ml (570 units) of blood were administered, the authors suggest the following guidelines

Autotransfusion should be considered for any patient who sustains blood loss in excess of 1,000 ml in the absence of gastrointestinal contamination.

The ATS unit should be prepared in advance when cross-matching of blood is precluded by abnormal antibodies, major loss is anticipated during elective surgery, or patients of the Jehovah's Witness faith agree to autotransfusion for major surgery.

Satisfactory anticoagulation may be achieved by the addition of heparin to the pump, or systemic or regional heparinization. ACD or CPD may be substituted for heparin. Systemic anticoagulation should be used with caution in patients sustaining multiple trauma, particularly if closed extremity fractures are present or head injury is suspected.

Incorporation of additional in-line 20–40-μ-pore filters as a precaution against microemboli-induced pulmonary damage is desirable, if not mandatory.

In order to minimize extravascular blood–tissue interaction and thereby reduce the likelihood of induced clotting abnormalities, aspiration of blood from the surgical site should be confined to the period of rapid blood loss. In addition, surgeons should be encouraged to avoid excess mechanical trauma to the shed blood by keeping the suction tip beneath the blood–air interface.

Patients who are autotransfused may require administration of fresh frozen plasma or platelets, just as do patients receiving massive homologous transfusions.

Although there has been no report linking renal failure to autotransfusion, prophylactic enforced diuresis with mannitol or furosemide should be considered in patients sustaining prolonged periods of hypovolemic shock prior to or during autotransfusion.

If air embolism is to be prevented, it is imperative that the individual responsible for the pump have no duty to perform other than operating the autotransfusion unit.

When the alternative is exsanguination, autotransfusion should be considered despite the possibility of contamination, tumor-cell emboli, or closed-space hemorrhage.

Twentieth century anesthesiologists and surgeons might well profit from the recommendations of an earlier colleague. John Duncan,[11] of Edinburgh, writing in the *British Medical Journal* in 1886, cautioned that practitioners should not "neglect stringent precautions needful to avoid the contingencies of septicity and embolism, lest it [re-infusion of blood] should fall into discredit from too wide an application." He continued, "I advocate it as perfectly sane and capable of saving many lives in the major operations of surgery. I make it a routine practice in all the larger amputations because there is no risk and every ounce of blood is serviceable . . . to my own mind the principle of re-infusing the patient is now definitely established."

ADDENDUM

Since submission of the manuscript, the authors have autotransfused an additional 47 patients. They total volume of blood autotransfused in 118 patients was 455,600 ml (911 units).

References

1. Aaron RK, Beazley RM, Riggle GC: Hematologic integrity after intra-operative allotransfusion comparison with bank blood. Arch Surg 108:831–837, 1974
2. Bankoff GA: Milestones in Medicine. London, Sir Isaac Pitman and Sons, Ltd., 1961
3. Bennett SH, Geelhold GW, Gralnick HR, et al: Effects of autotransfusion on blood elements. Am J Surg 125:273–279, 1973
4. Bennett SH, Geelhold GW, Terrill RE, et al: Pulmonary effects of autotransfused blood. Am J Surg 125:696–702, 1973
5. Brener BJ, Raines JK, Darling R: Intraoperative autotransfusion in abdominal aortic resections. Arch Surg 107:78–84, 1973
6. Brener BJ: Autotransfusion—safe at any speed? Arch Surg 108:761, 1974
7. Burch LE: Autotransfusion. Surg Gynecol Obstet 36:811–814, 1923
8. Burch LE: Autotransfusion. Trans Southern Surg Assoc 35:25–35, 1922
9. Carty MJ, Barr RD: The hemoglobin content of autotransfused blood in acute ectopic pregnancy. J Obstet Gynecol Br Commonw 79:1137–1138, 1972
10. Davis LE, Cushing H: Experiences with blood replacement during or after major intracranial operations. Surg Gynecol Obstet 40:310–322, 1925
11. Duncan J: On re-infusion of blood in primary and other amputations. Br Med J 1:192, 1886
12. Dyer RH: Intra-operative autotransfusion, a preliminary report and a new method. A J Surg 112:874–878, 1966
13. Dyer RH, Alexander JT, Brighton CT: Atraumatic aspiration of whole blood for intraoperative autotransfusion. Am J Surg 123:510–514, 1972
14. Elmendorf: Veber wiederinfusion nach punktion eines frishen haemathorax. Munchen Med Wochenschr 64:36–37, 1917
15. Farrar LK: Autotransfusion—blood transfusion in gynecology. Surg Gynecol Obstet 36:454–461, 1923
16. Griswold RA, Ortner AB: The use of autotransfusion in surgery of the serous cavities. Surg Gynecol Obstet 77:167–177, 1943
17. Highmore W: Overlooked source of bloodsupply for transfusion in post-partum haemorrhage. Lancet 1:89, 1874
18. Karpatkin M: Diagnosis and management of disseminated intravascular coagulation. Pediatr Clin North Am 18:23–38, 1971
19. Kingsley JR, Valeri CR, Peters H, et al: Citrate anti-coagulation and on-line cell washing in intra-operative autotransfusion in the baboon. Surg Forum 24:258–260, 1973
20. Klebanoff G: Early clinical experience with a disposable unit for the intra-operative salvage and reinfusion of blood loss (intraoperative autotransfusion) Am J Surg 126: 718–722, 1970
21. Klebanoff G, Phillips J, Evans W: Use of a disposable autotransfusion unit under varying conditions of contamination. Am J Surg 120: 351–354, 1970
22. Lockwood CD: Surgical treatment of Banti's disease. Surg Gynecol Obstet 25:188–191, 1917
23. McNamara JJ, Burran EL, Larson E, et al: Effect of debris in stored blood on pulmonary microvasculature. Ann Thorac Surg 14:133–139, 1972
24. McNamara JJ, Molet MD, Stremple JF: Screen filtration pressure in combat casualties. Ann Surg 172:334–341, 1970
25. Miller AG: Case of amputation at the hip joint in which reinjection of blood was performed and recovery took place. Edinburgh Med J 31:721–722, 1885
26. Miller RD: Complications of massive blood transfusions. ANESTHESIOLOGY 39:82–93, 1973
27. Miller RD, Robbins TO, Tong MJ, et al: Coagulation defects associated with massive blood transfusions. Ann Surg 174:794–801, 1971
28. Pathak UN, Stewart DB: Autotransfusion in ruptured ectopic pregnancy. Lancet 1:961–964, 1970
29. Peskin GW, O'Brien K, Rabines SF: Stroma-free hemoglobin solution: The "ideal" blood transfusions. Ann Surg 174:794–801, 1971
30. Rabiner SF, Friedman LH: The role of intravascular hemolysis and the reticuloendothelial system in the production of a hypercoagulable state. Br J Haematol 14:105–118, 1968
31. Rakower SR, Worth MH: Autotransfusion: Perspective and critical problems. J Trauma 13:573–574, 1973
32. Schilling JA: Discussion of a paper by Langston HT, Miles G, Dalessandro W: Further experience with autologous blood transfusions. Ann Surg 158:333–337, 1967
33. Symbas PN: Autotransfusion from hemothorax: Experimental and clinical studies. J Trauma 12:689–695, 1972
34. Symbas PN, Levin JM, Ferrier FL, et al: A study of autotransfusion from hemothorax. Southern Med J 62:671–674, 1969
35. Theis HJ: Zur behandlung der extrauteringravidi tat. Zentralbl Gynaekol 38:1191, 1914
36. Wilson JD, Taswell HF: Autotransfusion: Historical review and preliminary report on a new method. Mayo Clin Proc 43:26–35, 1968
37. Wright CB, Geelhold GW, Mason KG: Autotransfusion in the subhuman primate. Amer J Surg 128:49–53, 1974