A Clinical Review of Autotransfusion and Its Role in Trauma

Lenworth M. Jacobs, MD, MPH, Jing Weng Hsieh, MD

Autotransfusion is defined as the collection and reinfusion of a patient's own blood. The first description of autotransfusion dates back to 1818, when Blundell used it in London to counter postpartum hemorrhage, with a mortality of 50%. Autotransfusion in the setting of chest trauma was first recorded by Elmendorf during battle in 1917 and in civilian life in 1931. The earliest reported American application of autotransfusion was attributed to Lockwood intraoperatively during a splenectomy in 1917.

Early literature of the 1920s and 1930s reported autotransfusion more commonly used for intra-abdominal hemorrhage, ie, ruptured ectopic pregnancies and traumatic vascular injuries. Its application in chest or pleural cavity injuries was limited to a few sporadic cases. The reluctance for such use stemmed from the fear of contamination of the shed blood.

With the advent of blood typing and cross matching, homologous blood from banks became the standard blood replacement in both the operating room and the emergency department, and interest in autotransfusion waned.

In recent decades, the combined effect of increasing demand for large quantities of readily available, compatible blood and the more stringent laws enacted to ensure the quality of donated blood has stimulated renewed interest and enthusiasm in autotransfusion. The experience with massive trauma in Vietnam and the many trauma victims of civilian motor vehicle accidents, shootings, and stabblings emphasized the need for a blood source other than banked blood.

Cardiopulmonary bypass surgery and experimental/clinical studies of the last ten years have established autotransfusion as a safe, economical, and practical means to supplement the overtaxed sources of homologous blood. The work of authors such as Symbas, Mattox, Anderson, Davidson, Klebanoff, Glover, Smith, and O'Riordan in the 1970s and 1980s demonstrated the value of routine implementation of autotransfusion in major emergency departments as well as in the operating room.

Autotransfusion Devices

Symbas' described the properties of the ideal autotransfusion device as follows: (1) rapid assembly, (2) relatively low cost, (3) ease of operation, (4) in-line filtration system, (5) minimized air-blood interface, (6) simplified anticoagulation, and (7) safety from air embolism and coagulopathies.

Several autotransfusion devices currently on the market fall into two broad categories: those with or without cell-washing ability. Each autotransfusion device has predominant use in either intraoperative open heart or elective general surgical (liver, vascular) procedures, in emergency departments for major acute trauma, or in blood banks for preparation of blood products.

One type of autotransfusion device used at Boston City Hospital was designed for trauma emergency department use and consists of disposable software and reusable hardware. The device is based on the principle of using wall suction (30 to 60 mm Hg) to draw blood from a traumatic hemothorax through a chest tube. The blood is filtered into a sterile, disposable, 1,900-mL-capacity liner within a rigid cannister; anticoagulation is administered through a volume control device (Figure). When the liner is full, it is disconnected from the chest tube. A microemboli filter (20 to 40 μm) and intravenous administration set is directly attached to the liner, and the autolo...
Gous blood is reinfused. The cost of setup is $48, thus, the cost per unit of autotransfused blood is $12 since a single liner accommodates 4 units of blood. The advantages of this system are its simplicity, ease of use and assembly, speed, and low cost. This device lacks the cell-washing capabilities of systems manufactured by other companies, which have their primary use in the operating room setting.

Hematologic Changes

Experimental and clinical studies of autotransfused blood from the peritoneal and pleural cavities have established definitive changes in platelet and fibrinogen levels. The number of platelets in salvaged blood have been shown to be considerably decreased, ranging from less than 71% baseline to 0% baseline. Not only is quantity affected, but so is quality; ie, platelet function appears to be abnormal. The aggregation properties of platelets are considerably depressed, resulting in increased bleeding times, sometimes longer than 24 hours. Davidson, Mattox, and Symbas' recommend that supplemental homologous donor platelets should be given. All authors agree that the number of platelets will return to baseline levels within 48 to 72 hours after autotransfusion.

Fibrinogen levels in salvaged blood are also decreased. As far back as Elmendorff in 1917 and Brown in 1931, it has been observed that thoracic cavity and peritoneal cavity blood does not clot. This has been attributed to the consequence of mechanical factors such as heart, lungs, and chest wall action, as well as biochemical reactions between the serosal lining of these cavities and blood. Brodie et al, in 1981, theorized that defibrination was a function or consequence of fibrinogenolysis. The true explanation of this phenomenon appears, however, to evade researchers at this time.

Carty, in 1972, demonstrated factors V, VIII, and X to be decreased. Darling et al reported on the intraoperative autotransfusion experience in major vascular surgery. They concluded that transfusions of less than 3,000 to 3,500 mL were safe, and that the transient disseminated intravascular coagulation (DIC) phenomena were not directly related to the autotransfused blood, but resulted from a dilutional coagulopathy and the systemic heparinization. Recently, Moore et al demonstrated experimentally in dogs the prolongation of bleeding time, whole blood clotting time, and thrombin time of DIC phenomena, and confirmed the previous platelet findings. Like platelet counts, the coagulation factors will also return to normal within 48 to 72 hours. The work of Bennett et al has shown transient and insignificant changes in prothrombin time and partial thromboplastin time.

Anticoagulation

At the Boston City Hospital emergency department, citrate phosphate dextrose (CPD) is used in all autotransfusion cases regardless of rate or amount of bleeding. The controversy regarding anticoagulant use in autotransfusion devices is solely a theoretical discussion. In practice, one should be prepared to use it. The controversy arises from the fact that defibrinated blood does not clot, and questions the need to use any anticoagulants. Davidson found that anticoagulants were necessary in cases involving massive great vessel bleeding, where brisk bleeding prevents defibrination and results in massive clotting of the autotransfusion device and tubings. In the majority of cases of chest trauma with "slower bleeding," Brown and Debbah, and Brodie et al, and Symbas agreed that anticoagulant use was not needed.

It would appear that autotransfusion is best utilized in cases of massive hemothorax encountered in the emergency department, and thus use of anticoagulant becomes essential. At the present time, CPD is recommended by most authors and manufacturers of autotransfusion devices. In the earlier period of intraoperative autotransfusion, heparin was used systematically and locally within the autotransfusion tubing and devices. Systemic heparinization carries an obvious drawback in the patient with multiple trauma who already has severe bleeding, externally and internally. Briefly mentioned in the literature is the use of...
acid citrate dextrose (ACD) as an anticoagulant.\textsuperscript{38} Citrate phosphate dextrose has the advantage over ACD of being less acidic and requiring less volume,\textsuperscript{11} and has none of the complications of heparin. Klebanoff\textsuperscript{20} and Reul et al\textsuperscript{11} found CPD to be well tolerated by both experimental and clinical models.

The suggested quantity is 25 to 30 mL of CPD in 500 mL of autotransfused blood, or a 1:7 ratio of CPD to blood (banked blood contains 70 mL of CPD per 500 mL). Myocardial failure induced by citrate toxicity and chelation of available ionized calcium has not been reported with the recommended amount of CPD. When less than 2,000 or 3,000 mL of autotransfused blood is transfused, the occurrence of clinically important DIC is rare.\textsuperscript{23,24} The recommendation is to supplement with fresh frozen plasma when autotransfusions are greater than 4,000 mL.\textsuperscript{25}

**Free Hemoglobin and Nephrotoxicity**

Another discussed, though less well known, aspect of autotransfusion is that of hemolysis of RBCs and the potential nephrotoxic effects of the resultant elevated free plasma hemoglobin. The mechanism of RBC damage appears to include both exposure to body cavities and the actual procedure of collecting and reinfusing the blood.\textsuperscript{23} Isolated free plasma hemoglobin levels elevated over an undefined critical level can result in renal insufficiency and ultimate failure.

Aaron et al\textsuperscript{22} speculated that blood hemoglobin levels above 100 to 135 mg/dL exceeded the binding capacities of heptoglobin, one of the major mechanisms by which free hemoglobin is cleared. When the renal threshold is reached, the precipitated hemoglobin can form the more toxic acids hematin and methemoglobin in the tubules, resulting in acute tubular necrosis. Recently, Rabiner\textsuperscript{26} has challenged this theory and questioned whether the free hemoglobin moiety or the stroma fraction is actually nephrotoxic. His work with stroma-free hemoglobin in normal and ischemic kidneys showed no substantial histological changes and no increases in serum or urine urea nitrogen and creatinine levels.

Nonetheless, conditions that would precipitate hemolysis should be avoided, and perhaps more important in preventing renal failure is the avoidance of shock and acidosis. Sym¬
bas\textsuperscript{27} and Reul et al\textsuperscript{11} reported free plasma hemoglobin levels of greater than 350 mg/dL in autotransfused blood. Klebanoff\textsuperscript{20} reported no known complications of autotransfusion to kidneys in his work. O'Riordan\textsuperscript{20} reported transient increases of serum creatinine levels. At this time, renal insufficiency would appear to be a relative contraindication to autotransfusion.\textsuperscript{20}

To minimize hemolysis, two aspects of the autotransfusion procedure have been modified. Suction pressures should be limited to 10 to 15 mm Hg in the emergency department closed-chest tube drainage system, and between 40 and 60 mm Hg intraoperatively.\textsuperscript{23,24} To further minimize hemolysis, suctioning should be from pooled blood; the suction tip should be kept below the surface of the blood, thereby reducing the degree of air-blood interface.\textsuperscript{23,24}

Reul et al\textsuperscript{22} suggested the use of mannitol to prevent renal insufficiency and failure, and O'Riordan\textsuperscript{20} used furosemide to maintain adequate diuresis. Recommendations in the literature are not clear as to diuretic use.

**Sepsis**

Another controversial area deals with sepsis, or what to do when suspicion is high regarding contamination of the shed blood. Griswold and Ortner,\textsuperscript{28} in the early 1940s, recognized this potential problem, and stated that the infusion of contaminated blood was better than no blood. Klebanoff\textsuperscript{20} reported that experimental models tolerated infusion of contaminated blood very well. Glover et al\textsuperscript{22} showed that transient bacteremias occurred, but blood cultures were negative 24 hours after the autotransfusion. Their work, done in the early 1970s, confirmed that earlier sentiment. Dog studies demonstrated that shock was the overwhelming and determining factor in morbidity and mortality due to transfusion of contaminated blood. When circulatory loss was greater than 40%, substantial increases in morbidity and mortality occurred in the face of transfusion of such blood.\textsuperscript{22} Blood contaminated with liver, pancreas, and bile has been infused clinically by Glover et al,\textsuperscript{22} Griswold and Ortner,\textsuperscript{28} and ourselves, and experimentally by Klebanoff,\textsuperscript{20} without any sequelae. Blood contaminated with urine or salvaged during prostate resections has been infused with the rare occurrence of transient renal failure.\textsuperscript{22,29,30}

Recommendations to prevent septic complications include discarding blood that is more than 4 hours old, especially when obtained from an open wound; discarding fecal contaminated blood; and recognizing that autotransfusion has absolutely no storage capacity (Table).\textsuperscript{31} Although experimentally and clinically contaminated blood has been used, the consensus is that contaminated shed blood should be discarded in all but the most desperate situations. In situations in which homologous blood is readily available, the inherent risk of transfusing contaminated blood is too great; indeed, in urban settings with modern blood banks and an available Red Cross, this is an absolute contraindication. In the small rural emergency department with little or no bank blood availability, autotransfusion’s advantages outweigh its disadvantages.\textsuperscript{31}

Broad-spectrum antibiotics are indicated if contamination of autotransfusion blood is suspected; this is generally agreed on by all authors. Glover et al\textsuperscript{22} reported in 1978 that antibiotics could increase survival from 30% to 90% in shock experimental models transfused with contaminated blood. The efficacy of cephalosporins + combination triple therapy (aminoglycosides, clindamycin, peni¬cillin) has not been adequately tested to date.

**Microemboli**

The complication of microaggregates and microembolism, and their potential sequelae of renal and pulmonary disfunction, is well established.\textsuperscript{31-33} Platelet-fibrin-leukocyte microaggregates, and fat microaggregates when embolized, can cause respiratory distress syndrome and renal failure. The work of Bennett et al\textsuperscript{34} on dog lung models after massive autotransfusion and banked-blood transfusions showed a decrease in both groups of arterovenous oxygen gradient, but to a lesser degree with
autotransfused blood; also, unlike banked blood, autotransfused models did not show increases in pulmonary vascular resistance or endobronchial pressures. In these experimental models, fewer animals exhibited microscopic evidence of pulmonary changes or damage in the autotransfusion group. Bennett et al.\(^1\) concluded that autotransfused blood was less deleterious, with less microembolic complications, than banked blood.

The use of additional in-line micropore filters with the standard 170-µm filter in blood administration sets is controversial. Reul et al.\(^2\) suggested 40-µm micropores as a compromise between optimal blood infusion rate and the trapping of microaggregates and platelets. Davidson\(^3\) recommended 20-µm micropore filters, and cited no cases of pulmonary insufficiency with this size. The work of Litwin et al.\(^4\) in various types of blood transfusion filters demonstrated their usefulness in removing numerous microaggregates at even 170-µm pore size. The work of Moore et al.\(^5\) with methylprednisolone sodium succinate in massive autotransfused experimental model lungs suggested that corticosteroids may have considerable protective ability by preventing the platelet-releasing reaction. They found no hemodynamic changes in corticosteroid-treated models, and morphological changes in only 50% of these models v 90% of control models.

### Air Embolism

Finally, the complication of air embolism is an uncommon and fatal consequence of autotransfusion. Discussion of this topic is more germane to the last decade. All reported cases were associated with the automated roller pump units, best exemplified by the Bentley system, in which the reservoirs were inadvertently allowed to run dry, resulting in air embolism and fatality.\(^6\) Since the removal from the market of the Bentley system in 1973, this complication has been practically unrecorded. With the present emergency department autotransfusion setup, the frequency of air embolism approaches that for transfusion of a banked unit of blood.\(^7\)

### Advantages

Although autotransfusion’s complications are numerous, its advantages over banked blood are considerable. In the acutely injured patient in need of immediate blood, autotransfusion is readily available and, obviously, compatible. The 45-minute period necessary for a normal typing and cross matching procedure is eliminated. The collection and reinfusion device is simple and easily adaptable to any emergency department and requires only a few minutes to set up. Autotransfusion is safe; no hemolytic, febrile, or allergic reactions have been reported. Immunological sensitization from homotransfused leukocytes and platelets is avoided. Autotransfusion eliminates the risk of transmutable diseases of blood, eg, hepatitis, malaria, syphilis, and, more recently, acquired immune deficiency syndrome. Autotransfusion-salvaged blood is already warm, a distinctly desirable characteristic in the rapidly fluid-resuscitated patient in whom hypothermia can be a serious problem. The fresh RBCs of autotransfusion demonstrate survival times equivalent to those of venous blood cells,\(^8\) better oxygen transport capacities, and greater concentration of diphosphoglycerate levels than 4-day-old banked blood.\(^9\) The hematocrit value ranges between 25% and 35%, and although the platelets are decreased, they remain viable. Labile clotting factors are present in greater concentrations than in banked blood.\(^10\) The relatively normal metabolic balance of autotransfusion prevents hyperkalemia, hypocalcemia, and acidosis, often associated with transfusion of banked blood.\(^11\)\(^,\)\(^12\) There is also less risk of overtransfusion and inadvertent circulatory overload with autotransfusion.\(^13\)

Economically, autotransfusion has been shown to result in substantial cost savings. The cost of personnel and equipment for cross matching is eliminated. The normal cost of a unit of whole blood from the blood bank is $25 to $75. Davidson\(^3\) calculated the cost of the first 3 units of autotransfused blood to be $12 a unit. Probably more important, autotransfusion conserves a limited human resource; blood that is normally wasted is saved and reinused, allowing the equivalent units of banked blood to be available for other use.

Finally, some religious sects will occasionally accept autotransfusion as the only method of blood transfusion compatible with their practices.

### Conclusions

Autotransfusion, in the past 160 years of its history, has been shown to be safe, economical, and to possess many desirable advantages over homologous blood. The increase in national demand for banked blood, and the tighter controls imposed on the donor pool, have promoted the routine use of autotransfusion in major emergency departments and operating rooms and established it as a practical and essential supplement.
to the homologous source. The complications of autotransfusion are not benign, but with closer attention to equipment and the stated recommendations, their occurrence can be minimized.

References


American Cancer Society Inc, Research Development Program Grants

The American Cancer Society would like to call attention to its Research Development Program Grants as a mechanism for the rapid funding of timely clinical research projects of great promise.

The Research Development Program Grants were designed to provide speedy funding for new research ideas, for cancer control, unusual projects, or unanticipated needs for equipment and travel. Additionally, they may be used to provide short-term support with limited funding for clinical research of exceptional promise, and which, therefore, should be initiated with a minimum of delay.

Application forms and further information may be obtained from the Research Department, American Cancer Society Inc, 777 Third Ave, New York, NY 10017.

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