

Cell Salvage in Trauma

Fraser D. Rubens, MD, MSc, FRCSC,¹ Aneil Mujoomdar, MD, FRCSC,² and Homer C. Tien, MD, FRCSC^{3,4}

¹Division of Cardiac Surgery, University of Ottawa
Professor of Surgery, University of Ottawa Heart Institute
40 Ruskin Street

Ottawa, Ontario K1Y 4W7 Canada

frubens@ottawaheart.ca

²Division of Thoracic Surgery, University of Ottawa
Ottawa, Ontario, Canada

³Division of General Surgery and the Trauma Program,
Sunnybrook Health Sciences Centre, University of Toronto
Toronto, Ontario, Canada

⁴Canadian Field Hospital, Canadian Forces Health Services
Petawawa, Ontario, Canada

None of the authors have any conflict of interest with any product named in this article.

Learning Objectives: This clinical review of cell salvage will provide the traumatologist with specific aspects of understanding to facilitate the implementation of this technology into clinical practice. In particular, the specialist will be provided information to address the following goals. 1) To understand the basic techniques for the collection of blood from the wound for autotransfusion and the mechanisms for processing of this blood to remove potential contaminants. 2) To characterize the pathophysiology of salvaged blood prepared with and without centrifugal processing. 3) To understand the potential complications of the utilization of cell salvage, with particular reference to inflammation, infection, renal function, and coagulopathy. 4) To review the rationale for the use of cell salvage and to review the current clinical evidence supporting its implementation in trauma surgery. 5) To highlight the potential importance of this blood-conservation strategy in military conflict.

Abstract

There has been a resurgence of interest in cell salvage as a blood-conservation technology in many surgical specialties. Current limitations with regard to training and facility with the required equipment have thus far limited the expanded application of this strategy in the trauma setting. In this review, the pathophysiology of cell salvage and the consequences of centrifugal processing on the quality of the resultant transfusion product are reviewed. The majority of journal reports in the surgical literature supporting the clinical use of this strategy comprise observational studies; however, at least one well-designed, randomized, controlled trial has confirmed the potential of this application to decrease allogeneic blood transfusion in trauma recipients. The use of this approach is of particular interest in the military setting where access to fresh blood products may require collection of whole blood from soldiers, which may compromise their immediate health in a battle setting. Recommendations for technology innovation to advance the use of cell salvage in the trauma suite and in the battlefield are reviewed with the hope that well-designed trials can follow to aid in the implementation of this practice if proven effective.

Historical Overview

Long before the development of sophisticated operative techniques and anesthetic practice, physicians and surgeons were challenged with situations involving major hemorrhage secondary to warfare and urban violence. The concept of collecting this blood for retransfusion as a means of resuscitation was appealing and intuitive, thus supporting pioneering work in cell salvage as a blood-conservation strategy.¹ The development of modern blood banks subsequently removed much of the urgency that precipitated the evolution of this technology. Patients in extremis could now fairly rapidly receive type-specific and universal donor blood transfusions. However, this dependency on allogeneic products has recently been re-examined; peaking demands on blood banks, combined with fears of transfusion-related infections, have led to a re-emergence of the interest in cell salvage in a variety of disciplines including trauma. Recognizing that an integral part of the experience of specialists in trauma management includes the attainment of a sound understanding in transfusion practices and related technologies, the objective of this review will be to highlight the principles of cell salvage as it relates to this specialty.

Techniques of Cell Salvage

As compared with the early mechanics of cell salvage equipment (Fig. 1),² modern devices have advanced in terms of automation and sophistication, but in general they follow identical principles. Blood is aspirated into a closed suction device either by means of a double-lumen suction catheter in which heparin or citrate is entrained simultaneously, or through a drainage tube from a body cavity such as a chest tube. After this sterile collection, the blood passes through a filter designed to remove material greater than a specified diameter, (usually between 150 and 180 microns) before passing into a reservoir that has been primed with an anticoagulant.



Figure 1. Autotransfusion apparatus used successfully in World War II, consisting of a pool suction tip, a bottle with a two-hole stopper and citrate solution, tubing, a funnel and gauze filter, and an intravenous outfit. (Reproduced with permission from Griswold RA, Ortner AB. The use of autotransfusion in surgery of the serous cavities. *Surg Gynecol Obstet* 1943;77:167-77 [now *Journal of the American College of Surgeons*].)

At that point, the blood can be directly readministered (*unprocessed* blood) from the reservoir chamber into the patient either by gravity or through an infusion pump, usually after passage through a second filter (40 micron). This is an approach with a particular attraction in the trauma scenario in which immediate volume replacement may be necessary. However, if hemodynamics can temporarily be stabilized, an advance in cell salvage strategy can be applied in which the collected blood can be centrifugally washed to remove noncellular debris and potential contaminants prior to reinfusion (*processed* blood) using a device known as a “cell saver.”

A cell saver is typically composed of two subassemblies. The inner subassembly is stationary and contains the inlet and outlet port. The outer subassembly (which rotates) contains the primary processing chamber. Blood is separated based on differential densities of the components during centrifugation whereby the heavier elements form a boundary layer from the outside of the bowl inward. The lighter elements are displaced toward the core and then into a waste-collection container. Photo-optics are used to detect the maximum cell density and then to initiate the washing of the packed suspension with a selected volume of saline to further displace debris, plasma, free hemoglobin, and any anticoagulants via the outlet. Following washing, the remaining red cells are displaced into a collection bag and they may then be reinfused. The process is regulated by a microprocessor with internal air detectors and one-way valves.

The majority of cell savers use a variation of the bell-shaped (conical) Latham bowl or a (cylindrical) Baylor bowl³ (Fig. 2). There are no human trials supporting a difference between the two in terms of significant clinical outcome. Alternatively, the Fresenius CATS (Fresenius Hemocare, Bad Homburg, Germany) system uses a washing chamber similar to that found in larger apheresis units. The separation chamber of the CATS represents a blood channel in the shape of a double spiral. The blood is pumped into the inner spiral while it is washed with saline. Rotation of the separation chamber drives the red cells to the outer spiral, while lighter substances such as fat may be removed from the inner spiral. One significant advantage of this device is that it may operate in a continuous fashion with small volumes, thus batch processing is unnecessary and the device may be applicable to small-volume losses such as in pediatric surgical cases. As there is no “settling” after centrifugation stops (such as with the two-bowl configurations), it is also likely that residual contamination with noncellular materials is minimized.^{4,5}

Regardless of the device used, centrifugal washing is extremely effective at hemoconcentrating the blood product. Unprocessed blood usually has a hematocrit of 20 volume %.^{6,7} With the bowl devices, hemoconcentration may be modest, yielding a hematocrit approximately twice the preprocessing level,⁷ and with the CATS, the final product may have a hematocrit as high as 70%^{8,9} due to an incorporated optical sensor that guarantees that the red cell product is pumped out only at this target hematocrit.

As anticipated, centrifugal washing also removes most soluble proteins and nonerythrocyte particulate matter, such as interleukin 6, tumor necrosis factor, thrombin antithrombin III complex (TAT), plasmin antiplasmin, and free hemoglobin.⁷ However, levels of some proteins such as TAT and free hemoglobin may not fall below systemic levels as the process of washing may cause further release from activated cells or the clearance may be hampered by different affinities of the measured variables to retained cells or incompletely washed-out proteins.⁷

The operation of the cell-saver device does require some additional training, and in most institutions its use is supervised either by a perfusionist, an anesthesiology technician, or a nurse with additional training in cell salvage. Most units in North America using this technology have an active cardiac surgery program.

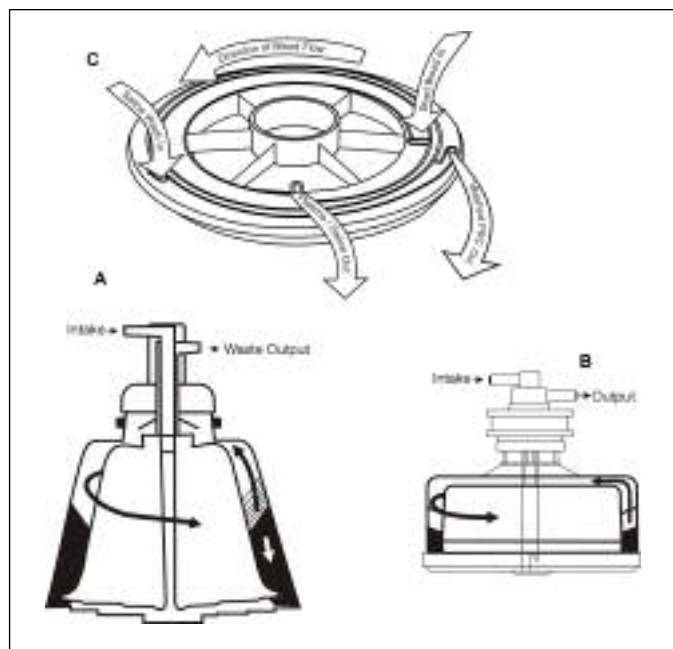


Figure 2. Schematics of centrifugal processing devices for salvaged blood. Conventional discontinuous (A) Latham (conical) and (B) Baylor (cylindrical) centrifuge bowls. (C) Fresenius CATS continuous system illustrating use of opposing double spiral to process collected blood. Cells from shed blood introduced centrally are forced peripherally against an opposing current of lighter saline wash. (Reproduced with permission from Rubens FD, Boodhwani M, Lavalee G, Mesana T. Perioperative red blood cell salvage. *Can J Anesth* 2003;50(6 Suppl):S31-S40.)

Pathophysiology of Salvaged Blood

Before universally advocating the use of cell salvage in cases of trauma and hemorrhage, it is essential to provide sufficient information on the function of this blood product to recruit clinician's confidence in this technology. In the acutely hypovolemic patient, it is intuitively understandable that collected shed blood may provide a rapid source of volume that can potentially minimize the hemodynamic effects of blood loss. However, does this product carry and deliver oxygen, and do the negative aspects of its administration outweigh the immediate benefit of large-volume replacement that it provides?

There is evidence to support that the morphology and function of red cells collected from a wound are normal and may in fact be better than allogeneic blood from a blood bank. Cells collected from shed unprocessed blood appear to maintain a normal membrane stability (as reflected by osmotic fragility) to a degree similar to that of peripherally sampled blood, but significantly less (and therefore better) than 5-week-old stored packed red cells.¹⁰ Collected shed blood also appears to have adequate stores of adenosine triphosphate and 2,3-diphosphoglycerate, with levels significantly higher than in banked blood ($P < .001$).¹⁰⁻¹²

Red blood cells that have been reinfused after cell salvage from a wound also appear to have acceptable in vivo survival. Schmidt et al¹³ isolated samples of red cells collected from the wound and from the peripheral systemic circulation, and these cells were simultaneously labeled with chromium and technetium. The authors found no difference between the 24-hour survivals of shed blood as compared with circulating peripheral blood. As well, there was no difference in the mean lifespan of shed blood (mean, 20.5 days;

range, 11.6–29) and circulating blood (mean, 22.7 days; range, 14.4–36.2; $P = .15$).

Therefore, in summary, evidence supports that salvaged red cells have normal functioning and survival as compared with blood simultaneously collected by venipuncture, and there is supportive evidence that this collected blood has a greater oxygen-carrying capacity than banked blood.

The significance of leukocytes collected in cell salvage blood is as yet undetermined. Although excessive depletion from wound hemorrhage could potentially compromise the leukocyte count and thus immune function, this is unlikely. In fact, leukocytes are retained in shed blood and centrifugal processing does not necessarily result in their removal, regardless of the device used.^{7,14}

A consistent finding in all studies to date has been the documentation of very low platelet counts in salvaged blood, with counts lower than allogeneic blood.^{6,7,12} Centrifugal washing of shed blood further decreases this count (60 [49–66] before and 14 [11–17] after [$10^3/\text{mCL}$]). This drop in platelets is likely related to ongoing thrombosis at the wound site. This latter process also likely explains the active defibrinogenation of this blood due to fibrin formation in the wound,¹⁵ although the absolute level of fibrinogen in shed blood may be no different from that of banked blood.⁶ Axford et al⁶ documented that shed blood is also different from banked blood in terms of greater factor VIIIc activity, antithrombin III, protein C, plasminogen activity, and antiplasmin activity, although the clinical significance of these findings is not known.

Complications of Cell Salvage

Major complications that have been reported to be related to reinfusion of collected shed blood include air embolism, the induction of an inflammatory state, sepsis, hemolysis, and coagulopathy. Air embolism was one of the earliest recognized

complications, but it is less frequently seen as a problem because of improved automation systems and air detectors. Further, great care is taken in training programs to emphasize the importance of avoiding the use of pressure systems to infuse back shed blood.¹⁶

It is not unexpected that the reinfusion of unprocessed blood would be associated with changes associated with activation of the inflammatory system. Febrile reactions are seen in 55% of patients receiving unprocessed blood, whereas they are seen in only 20% of patients after receiving processed blood.¹⁷ Autotransfusion is also associated with activation of the complement system.¹⁸ Increased levels of complement factors C3a, C5a, and terminal complement complex have been demonstrated in aspirated processed blood from hip arthroplasty patients.¹⁹ The consequences of this activation have not been clarified, and in otherwise healthy patients they likely may be of no concern, but it has been suggested in the cardiac surgery population that it may be associated with decreased immunity that may lead to an increased infection rate.²⁰

Most clinicians have witnessed hemoglobinuria after readministration of shed blood, paralleled by an increase in plasma-free hemoglobin, particularly if the blood is not processed.^{6,7,15,18,21–24} There is no proof that readministering blood with this degree of hemolysis results in clinically relevant complications; however, the impact of this finding in patients with borderline renal function is untested.

Spread of sepsis, particularly after aspiration of products from contaminated wounds, is a significant concern. Cultures of unprocessed shed blood collected from chest tubes after elective cardiac surgery are positive in 21% to 48% of samples.^{25,26} The contamination rate appears to be increased after processing shed blood collected after elective surgical procedures.^{7,27–29} This contamination is unlikely the result of the introduction of new bacteria, but rather from the washing out of perioperative prophylactic antibiotics. On the other hand, this degree of

Table 1. Cell Salvage in Trauma

Study	Design	Patient Population	Results
Glover et al ⁴⁷ (1978)	Retrospective	183 for emergency surgery	100% CS, 14 patients with EC-CSB, 8/14 survived
Huth et al ⁶⁷ (1983)	Retrospective	33 for emergency surgery	25/33 received CSB, accounting for 11% of requirements. No difference in observed septicemia or coagulopathy
Jurkovich et al ⁴⁸ (1984)	Retrospective for trauma	85 for emergency surgery transfused	26% received CSB for 28% of total blood
Timberlake and McSwain ⁴⁹ (1988)	Retrospective	11 for emergency surgery for trauma	1/11 with EC-CSB. No deaths; 3/11 developed infectious wound complications
Horst et al ⁵⁰ (1992)	Retrospective	154 for emergency surgery for trauma	Averaged 8.0 units CSB; 31% developed coagulopathy ↑ risk coagulopathy if >15 units of CSB collected
Ozmen et al ⁶⁸ (1992)	Retrospective	70 for PATI: 50 treated with banked blood vs. 20 treated with CSB	No difference in infectious complications
Smith et al ⁶⁹ (1997)	Retrospective	126 for emergency surgery for abdominal trauma	Averaged 6.9 units CSB 75% of cases were cost-effective
Hughes et al ⁷⁰ (2001)	Retrospective	22 for emergency surgery for abdominal trauma	CSB accounted for 1/3 of transfusion requirements; mortality 23%
Bowley et al ⁵¹ (2006)	Prospective, randomized, controlled trial	44 for emergency surgery: 23 in control group and 21 in CSB group	Control group: 11.17 units blood CS group: 6.47 No difference in survival (35% vs. 33%)

PATI, penetrating abdominal trauma index; CSB, cell-salvaged blood; CS, cell salvage; EC-CSB, enteric-contaminated cell-salvaged blood.

contamination has not been proven clinically to contribute to systemic or wound infections in elective cases.

The trauma situation is of course different in that prophylactic antibiotics would not be present and the degrees of contamination may be much greater because of viscous injury. Boudreaux et al³⁰ showed in an in vitro model that centrifugal processing can reduce the levels of bacterial contamination after enteric contamination of blood. Further work demonstrated that survival after transfusion of contaminated blood in an animal model can be increased from 30% to 90% with the addition of antibiotics.³¹

Despite these positive advances with antibiotic coverage, it is still generally accepted that in cases of definite sepsis with wound contamination, cell salvage should not be used unless bleeding is life-threatening.³² In 1994, the British Committee for Standards in Hematology Blood Transfusion Task Force produced the Guidelines for Autologous Transfusion for Cell Salvage. These guidelines stated, "Cell salvage is appropriate where there is a clean wound" and "cell salvage techniques should not be used in the presence of bacterial contamination of the operative field."³³ Finally, regardless of the source, in compliance with current American Association of Blood Bank standards, cell salvage from any body cavity should be limited to the initial 6 hours of collection.

The final concern with the use of cell salvage in the trauma suite relates to its potential contribution to a coagulopathy in a patient who is already systemically challenged. In animal models, massive reinfusion of unprocessed shed blood may lead to a state of disseminated intravascular coagulopathy³⁴ and this scenario has been mirrored in clinical cases.³⁵⁻³⁷ Much of the information with regard to induced coagulopathy with unprocessed blood comes from the cardiac surgery literature. Several clinical trials have demonstrated that the reinfusion of unprocessed blood in this group augments postoperative bleeding in amounts of 30% to 43% as compared with patients in whom the shed blood was discarded^{38,39} or the blood was processed.¹⁷ This effect has also been demonstrated in vascular surgery⁴⁰ and in surgery for spine trauma.⁴¹

There are several mechanisms by which reinfusion of shed blood, either processed or unprocessed, can contribute to coagulopathy. The increased bleeding in the cardiac surgery trials was associated with increased circulating levels of plasma fibrin-degradation products.³⁸ Shed blood also contains high levels of soluble markers of thrombin generation from the wound such as TAT15,²³ and prothrombin fragment 1,^{2,15} both known stimulators of endothelial tissue plasminogen-activator release.⁴² Whether this is a definitive mechanism of the increased bleeding that has been shown in these few clinical trials is not proven, however.^{6,38} On the other hand, processing of blood does prevent the expected rise in plasma D-dimer levels seen after reinfusion of the washed product.¹⁷

In summary, although reinfusion of unprocessed blood can provide immediate volume replacement and oxygen-carrying capacity, there is strong evidence that this product leads to overall increased bleeding. Processing of shed blood may remove the majority of the profibrinolytic components, but at the cost of the removal of large volumes of plasma proteins essential to the coagulation process, and this may contribute to a dilutional coagulopathy, a scenario recognized clinically by most surgeons and anesthesiologists.

Use of Cell Salvage in Trauma

A growing number of studies, including meta-analyses, have documented the efficacy of cell salvage in elective surgery⁴³ such that this technology has gained widespread acceptance in disciplines such as cardiac, vascular, orthopaedic, and neurosurgical. However, the use of cell salvage in trauma has not gained widespread

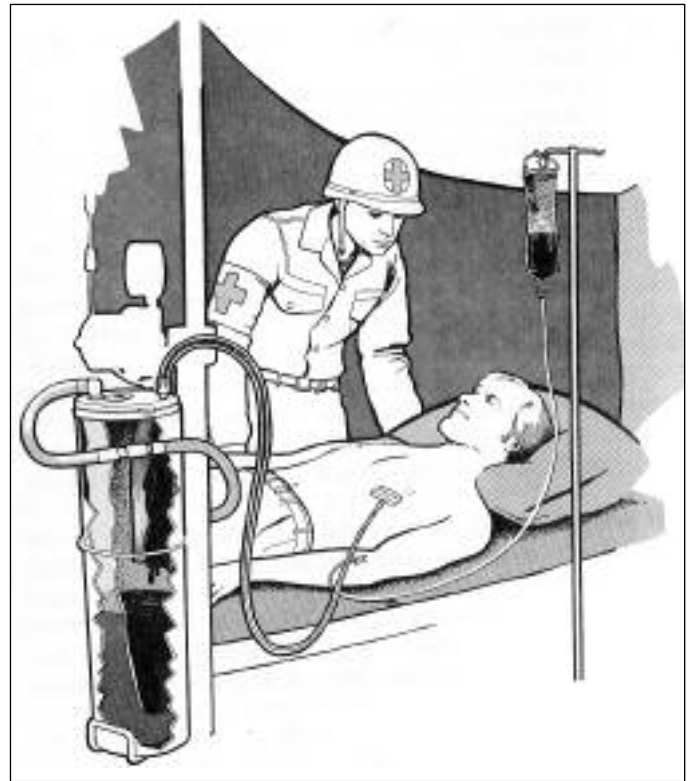


Figure 3. Autotransfusion in the battlefield. (Reproduced with permission from Rumisek JD. Autotransfusion of shed blood: an untapped battlefield resource. *Mil Med* 1982;147:193-6.)

recognition despite the obvious relevance of this problem to blood conservation, but there are now several reports supporting its application (Table 1). It has been estimated that trauma is responsible for as much as 10% to 15% of blood demands in urban centers, with up to 80% of trauma patients receiving blood transfusions at level I trauma centers.⁴⁴ Theoretically, cell salvage would be ideal in this setting, where the potential for massive blood loss exists and salvaged blood could be available quickly for resuscitation; however, the practicality of this application in busy trauma services must be taken into context.

Cell salvage in trauma can be applied in two broad circumstances. First, the technique can be used as an emergency source of blood volume replacement in the acutely hypovolemic patient who is actively bleeding into a body cavity. Most commonly, this involves patients who have sustained a massive hemothorax treated with tube thoracostomy with reinfusion of unprocessed shed blood.^{45,46} The authors believe this is a relatively safe and practical alternative that should be a recognized option by traumatologists. The only potential difficulty is that of transient coagulopathy, but this problem normalizes within 72 hours.⁴⁵

The second circumstance in which cell salvage may be useful involves trauma surgery wherein marked blood loss is anticipated once the operation commences. Surgery for spinal trauma represents a key example of this situation. Cavallieri et al⁴¹ retrospectively evaluated 238 cases of patients with this problem, in which blood transfusions were received by 118 patients; 53 received intraoperative blood salvage, and 65 did not (control group). The cell salvage group had a 47% reduction in homologous blood requirements (743 ± 1191 mL vs. 1403 ± 1453 mL; $P < .008$) and there was a 45% reduction in the number of patients who received homologous blood (45% vs. 82%; $P < .001$). Between these two

groups, no significant differences were observed in the evolution of hematocrit, platelet count, and coagulation markers, but there was a slight increase in postoperative blood loss was noted in the cell salvage group (465 ± 383 mL vs. 301 ± 292 mL; *P* < .01). The authors concluded that the efficiency of intraoperative blood salvage in emergency surgery for spine trauma is high and similar to that reported in elective spine surgery.⁴¹

Abdominal trauma represents a controversial application for cell salvage. Unlike cases of massive hemothorax, in traumatic injury to the abdomen with hemoperitoneum, the diagnosis may be delayed and there is greater concern of bacterial contamination of peritoneal contents because of concomitant bowel injury. Most of the reports in the trauma literature supporting this application have involved case series. Glover et al.⁴⁷ in 1978, described their experience with cell salvage in 183 emergency abdominal operations. It was noted that 14 patients received enteric-contaminated salvaged blood, with 8 survivors, of whom 4 had positive blood cultures postoperatively. Jurkovich et al.⁴⁸ reported 22 trauma patients in whom cell salvage was used at laparotomy; 82% of the patients received more than two units of cell-salvaged blood. As a whole, cell-salvaged blood provided 18% of the transfusion requirements of their entire cohort of trauma patients (85 patients). Six patients received enteric-contaminated cell-salvaged blood, three had positive reservoir blood cultures, but none had positive postoperative blood cultures. One of the six did succumb to sepsis-related multiorgan failure 3 weeks after injury. Of the 22 patients who received cell-salvaged blood, 7 developed coagulopathy (32%) as compared with 14% of those who received only homologous blood in their abdominal trauma cohort. Mortality rates were 27% in those who received cell-salvaged blood versus 25% in those who did not.⁴⁸ In 1988, Timberlake and McSwain⁴⁹ reported on 11 patients who had suffered thoracoabdominal trauma. All underwent intraoperative cell salvage of enteric-contaminated shed blood and all patients survived; however, three developed infectious wound complications.

Lastly, Horst et al.⁵⁰ studied 154 trauma patients in whom cell-salvaged blood was used aggressively in 88% of patients, providing 33% of the transfusion products in this cohort. Thirty-one percent developed moderate-to-severe coagulopathy. Risk factors for coagulopathy included the transfusion of more than 15 units of cell-salvaged blood, transfusion of more than 50 units of total blood, and transfusion of more than 10 units of enteric-contaminated cell-salvaged blood. They also concluded that cell salvage was a cost-effective technology if more than two units of cell-salvaged blood was transfused.

The only prospective, randomized, controlled trial that has addressed the use of cell salvage in abdominal trauma was completed by Bowley et al.⁵¹ in 2006. Forty-four patients were randomized into two groups, to receive either processed cell-salvaged blood along with allogenic blood (21 patients) or only

allogenic blood (control, 23 patients) if required. The mean number of units of allogenic blood transfused in the control group was 11.2 ± 6.1 units versus 6.5 ± 5.1 units in the cell salvage group (*P* = .008). The mean volume of salvaged blood transfused in the cell salvage group was 1.49 ± 0.62 liters. Enteric contamination was similar in the two groups. Survival in the control group was 35% versus 33% in the cell salvage group (*P* = NS).

The liberal use of laparoscopy may further change the potential for aggressive cell salvage as many specialized trauma units and military hospitals employ this technology in the initial triage. The feasibility of this approach has been demonstrated by Zantut et al.,⁵² who reported on 21 patients who underwent laparoscopic examination and subsequent aspiration of intraperitoneal blood after abdominal trauma. This procedure allowed them to diagnose if the blood was contaminated and, once cleared, allowed for immediate reinfusion, which was specifically of benefit in a patient who was a Jehovah's Witness. The technique should be relatively easily accomplished by the simple attachment of the laparoscopic suction catheter to a cell-saver machine in a manner similar to a chest tube.

Cell Salvage on the Battlefield

Military trauma care may also benefit from cell salvage techniques (Fig. 3). One application may be in the prehospital scenario with special operations forces (SOF). SOF operators work in far-forward locations, and can be hours or days away from a supporting hospital. As a result, SOF medics have advanced medical skills in order to provide life-saving interventions and ongoing support to critically injured casualties until they reach definitive care. SOF medics can perform several advanced procedures including intubation, escharotomies for severe burns, and intraosseous cannulation for fluid therapy,⁵³⁻⁵⁵ and almost all SOF medics are trained to perform needle and tube thoracostomies.⁵⁵ SOF medics, however, do not have the ability to transfuse patients; this capability deficiency is related to the logistical difficulties of maintaining a refrigerated blood supply in the prehospital setting.

There is tremendous interest in developing solutions to this problem. Military trauma researchers have devoted extensive resources to developing "artificial blood" that does not require refrigeration or special storage.⁵⁶⁻⁵⁸ As this is still in the development stage, acquiring techniques to perform cell salvage in the field, specifically from a hemothorax, is of great interest. This technique may provide enough volume and oxygen-carrying replacement therapy to sustain bleeding soldiers until they reach definitive care, where an ensuing coagulopathy or sepsis can be treated.

Another application for cell salvage may be in military field hospitals. These units can sometimes be faced with mass casualty situations in which the tremendous demand for allogeneic blood overwhelms existing supplies. The current solution to this difficult problem is to implement the "walking blood bank" strategy. Military

Table 2. Balancing Cell Salvage in Trauma

Advantages	Disadvantages
Reduced use of banked blood	Requires trained personnel
No risk of ABO incompatibility	Time required for collection, spinning, and washing before available for retransfusion
No increased risk of infectious complications	Expense of disposables and equipment
No transfusion reactions	Risk coagulopathy with large volumes of cell salvage (>15 units)
Likely cost-effective if >2 units transfused	

members are prescreened prior to deployment for transfusion-related infectious diseases. While overseas, if there is an acute demand for red blood cells, volunteers are immediately requested for blood donation. Blood is drawn and then reinfused into patients, after a repeat partial screen for (human immunodeficiency virus, hepatitis B and C) infectious disease in the donated blood is completed.

Besides replacing oxygen-carrying capacity, transfusing whole blood has the advantage of replacing clotting factors, platelets, and fibrinogen; some clinicians argue that whole blood is the ideal resuscitative fluid.^{59,60} However, this walking blood bank strategy has limitations. There are adverse effects associated with donating one unit of whole blood.⁶¹⁻⁶³ These effects (sore arm, bruising, and fatigue) may have few consequences in the course of a normal day's activity in Canada or the United States. However, on the battlefield, a sore arm or increased fatigue may have lethal consequences to the walking blood bank donor. Using processed autologous blood in the field hospital setting may offset some shortage in allogeneic blood, and reduce the need for whole blood from healthy donors.

Cost-effectiveness of Cell Salvage in Trauma

Systematic reviews of cell salvage performed in elective settings have shown it to be a cost-effective technology depending on the volume collected from the wound. Torella et al⁶⁴ concluded that more than two units of salvaged blood must be collected to derive economic benefit from this approach. This volume is less than that determined by Keeling et al,⁶⁵ who estimated a target of four units. Smith et al⁶⁶ retrospectively reviewed their experience with cell salvage in trauma during a 3-year period. Based on estimated blood losses and quantity of blood transfused, costs of auto transfusion were compared with estimated blood bank costs. They concluded that intraoperative cell salvage was cost-effective in 75% of their cases. It should be noted that cell-salvaged blood accounted for 45% of total blood transfused. From the available data, it would seem reasonable that the more frequently cell-salvaged blood was used, the greater likelihood of it being cost-effective.

Conclusion

In order to integrate cell salvage technology in high-volume trauma centers or in battlefield hospitals, several issues must be addressed. 1) Technical innovations must be introduced to facilitate and automate its use, particularly if this strategy is to be used in the far-forward battlefield and field hospital situations. 2) Strategies to minimize the coagulopathy observed with autotransfusion must be developed. 3) New paradigms for abdominal trauma management that involve immediate diagnostic peritoneal lavage or laparoscopy, in order to rule out bowel injuries and to introduce catheters to collect shed blood, need to be developed.

Although the evidence is limited for most aspects of cell salvage in abdominal trauma, we believe that we can confidently recommend that units dealing with a high volume of penetrating thoracic trauma should have immediate access to systems that allow for reinfusion of unprocessed shed blood. Although not ideal, it may provide a window of opportunity with supported oxygen delivery and hemodynamic stability until allogeneic products are available. In one of the author's (F.D.R.) clinical practice in cardiac surgery, a chest tube system (Atrium Medical Corp., Hudson, NH) is used that provides a sterile output at the base of the reservoir. If the collected volume is <1,000 mL in 6 hours, the contents are discarded as the yield from processing is not cost-effective relative to the cost of the disposables of the cell saver. Volumes collected greater than this amount in the hemodynamically stable patient would be processed

with the cell saver and readministered. In the rare situation of a sudden unexpected hemorrhage through the chest tubes, the blood can be immediately diverted into the patient without processing, while awaiting a surgeon for reopening and hemorrhage control.

With the ever-growing demand placed on blood banks, the limited pool of willing and able donors, and the risk of transfusion-related infections, cell salvage should be re-evaluated by traumatologists (Table 2). Trauma by its very nature results in clinical scenarios characterized by extensive circulatory derangement and compromised oxygen delivery, and in the absence of a ready supply of allogeneic blood, cell salvage can provide rapid resuscitation. Further, its judicious use may result in a decrease in the overall burden of allogeneic blood transfused in each patient. We believe the risk/benefit ratio of this simple procedure demands that it be re-examined with a critical eye with the hope of gathering support to test this strategy in well-designed trials.

References

- Duncan J. On reinfusion of blood in primary and other amputations. *Br Med J* 1886;1:192-7.
- Griswold RA, Ortner AB. The use of autotransfusion in surgery of the serous cavities. *Surg Gynecol Obstet* 1943;77:167-77.
- Rubens FD. An update on perioperative blood salvage in cardiac surgery. *Transfusion Altern Transfusion Med* 2005;7(1):20-8.
- Booke M, Fobker M, Fingerhut D, et al. Fat elimination during intraoperative autotransfusion: an in vitro investigation. *Anesth Analg* 1997;85(5):959-62.
- Shulman G. Quality of processed blood for autotransfusion. *J Extra Corpor Technol* 2000;32(1):11-9.
- Axford TC, Dearani JA, Ragno G, et al. Safety and therapeutic effectiveness of reinfused shed blood after open heart surgery. *Ann Thorac Surg* 1994;57:615-22.
- Reents W, Babin-Ebell J, Misoph MR, Schwarzkopf A, Elert O. Influence of different autotransfusion devices on the quality of salvaged blood. *Ann Thorac Surg* 1999; 68:58-62.
- Orr MD. Autotransfusion: intraoperative scavenging. *Anesthesiol Clin* 1982;24:97-117.
- Cordell AR, Lavender SW. An appraisal of blood salvage techniques in vascular and cardiac operations. *Ann Thorac Surg* 1981;31:421-5.
- Schmidt H, Kongsgaard UE, Geiran O, Brosstad F. Autotransfusion after open heart surgery: quality of shed mediastinal blood compared to banked blood. *Acta Anaesthesiol Scand* 1995;39:1062-5.
- Heaton WA, Keegan T, Holme S, Momoda G. Evaluation of 99mtechnetium/51chromium post-transfusion recovery of red cells stored in saline, adenine, glucose, mannitol for 42 days. *Vox Sang* 1989;57(1):37-42.
- McShane AJ, Power C, Jackson F, et al. Autotransfusion: quality of blood prepared with a red cell processing device. *Br J Anaesth* 1987;59:1035-9.
- Schmidt H, Lund JO, Nielsen SL. Autotransfused shed mediastinal blood has normal erythrocyte survival. *Ann Thorac Surg* 1996;62:105-8.
- Perttala J, Leino L, Poyhonen M, Salo M. Leukocyte content in blood processed by autotransfusion devices during open heart surgery. *Acta Anaesthesiol Scand* 1995;39:445-8.
- Flom-Halvorsen HI, Øvrum E, Tangen G, et al. Autotransfusion in coronary artery bypass grafting: disparity in laboratory tests and clinical performance. *J Thorac Cardiovasc Surg* 2000;118:610-7.
- Linden JV, Kaplan HS, Murphy MT. Fatal air embolism due to perioperative blood recovery. *Anesth Analg* 2002;84:422-6.
- Vertrees RA, Conti VR, Lick SD, et al. Adverse effects of postoperative infusion of shed mediastinal blood. *Ann Thorac Surg* 1996;62(3):717-23.
- Kongsgaard UE, Tollofsrud S, Brosstad F, Øvrum E, Bjørnskaug L. Autotransfusion after open heart surgery: characteristics of shed mediastinal blood and its influence on the plasma proteases in circulating blood. *Acta Anaesthesiol Scand* 1991;35(1):71-6.
- Arnestad JP, Hyllner M, Bengtson JP, et al. Removal of activated complement from shed blood: comparison of high- and low-dilutional haemofiltration. *Acta Anaesthesiol Scand* 1998;42(7):811-5.
- Body SC, Birmingham J, Parks R, et al. Safety and efficacy of shed mediastinal blood transfusion after cardiac surgery: a multicenter observational study. Multicenter Study of Perioperative Ischemia Research Group. *J Cardiothorac Vasc Anesth* 1999;13(4):410-6.

21. Thurer RL, Lytle BW, Cosgrove DM, Loop FD. Autotransfusion following cardiac operations: a randomized, prospective study. *Ann Thorac Surg* 1979;27(6):500-7.
22. Adan A, Brutel de la Rivière A, Haas F, van Zalk A, de Nooij E. Autotransfusion of drained mediastinal blood after cardiac surgery: a reappraisal. *Thorac Cardiovasc Surg* 1988;36:10-4.
23. Schulze HJ, Wendel HP, Khalighi K, Heller W, Seboldt H. The quality of autotransfused chest-drainage blood after cardiac surgery: a study of coagulation factors. *Thorac Cardiovasc Surg* 1996;44:183-7.
24. Ward HB, Smith RR, Landis KP, et al. Prospective, randomized trial of autotransfusion after routine cardiac operations. *Ann Thorac Surg* 1993;56(1):137-41.
25. Davies MJ, Cronin KC, Moran P, Mears L, Booth RJ. Autologous blood transfusion for major vascular surgery using the Sorenson Receptal Device. *Anaesth Intensive Care* 1987;15(3):282-8.
26. Wollinsky KH, Oethinger M, Buchele M, et al. Autotransfusion--bacterial contamination during hip arthroplasty and efficacy of cefuroxime prophylaxis. A randomized controlled study of 40 patients. *Acta Orthop Scand* 1997;68(3):225-30.
27. Bennett JG. Autotransfusion of drained mediastinal blood. *Thorac Cardiovasc Surg* 1982;30:28-30.
28. Schwieger IM, Gallagher CJ, Finlayson DC, Daly WL, Maher KL. Incidence of Cell-Saver contamination during cardiopulmonary bypass. *Ann Thorac Surg* 1989;48(1):51-3.
29. Bland LA, Villarino ME, Arduino MJ, et al. Bacteriologic and endotoxin analysis of salvaged blood used in autologous transfusions during cardiac operations. *J Thorac Cardiovasc Surg* 1992;103(3):582-8.
30. Boudreaux JP, Bornside GH, Cohn J Jr. Emergency autotransfusion: partial cleansing of bacteria-laden blood by cell washing. *J Trauma* 1983;23(1):31-5.
31. Smith RN, Yaw PB, Glover JL. Autotransfusion of contaminated intraperitoneal blood: an experimental study. *J Trauma* 1978;18(5):341-4.
32. Dzik WH, Sherburne B. Intraoperative blood salvage: medical controversies. *Transfus Med Rev* 1990;4(3):208-35.
33. Napier JA, Bruce M, Chapman J, et al. Guidelines for autologous transfusion. II. Perioperative haemodilution and cell salvage. British Committee for Standards in Haematology Blood Transfusion Task Force. Autologous Transfusion Working Party. *Br J Anaesth* 1997;78(6):768-71.
34. Stillman RM, Wrezlewicz WW, Stanczewski B, et al. The haematological hazards of autotransfusion. *Br J Surg* 1976;63(8):651-4.
35. McKie JS, Herzenberg JE. Coagulopathy complicating intraoperative blood salvage in a patient who had idiopathic scoliosis. A case report. *J Bone Joint Surg Am* 1997;79(9):1391-4.
36. Milne AA, Drummond GB, Paterson DA, Murphy WG, Ruckley CV. Disseminated intravascular coagulation after aortic aneurysm repair, intraoperative salvage autotransfusion, and aprotinin. *Lancet* 1994;344(8920):470-1.
37. Murray DJ, Gress K, Weinstein SL. Coagulopathy after reinfusion of autologous scavenged red blood cells. *Anesth Analg* 1992;75(1):125-9.
38. de Haan J, Schonberger J, Haan J, van Oeveren W, Eijgelaar A. Tissue-type plasminogen activator and fibrin monomers synergistically cause platelet dysfunction during retransfusion of shed blood after cardiopulmonary bypass. *J Thorac Cardiovasc Surg* 1993;106:1017-23.
39. Martin J, Robitaille D, Perrault LP, et al. Reinfusion of mediastinal blood after heart surgery. *J Thorac Cardiovasc Surg* 2000;120(3):499-504.
40. Long GW, Glover JL, Bendick PJ, et al. Cell washing versus immediate reinfusion of intraoperatively shed blood during abdominal aortic aneurysm repair. *Am J Surg* 1993;166(2):97-102.
41. Cavallieri S, Riou B, Roche S, et al. Intraoperative autologous transfusion in emergency surgery for spine trauma. *J Trauma* 1994;36(5):639-43.
42. Kitaguchi H, Hijikata A, Hirata M. Effect of thrombin on plasminogen activator release from isolated perfused dog leg. *Thromb Res* 1979;16:407-15.
43. Hahn C, Tam SKC, Vlahakes GJ, Hilgenberg AD, Akins CW, Buckley MJ. Repeat aortic root replacement. *Ann Thorac Surg* 1998;66:88-91.
44. Como JJ, Dutton RP, Scalea TM, Edelman BB, Hess JR. Blood transfusion rates in the care of acute trauma. *Transfusion* 2004;44(6):809-13.
45. O'Riordan WD. Autotransfusion in the emergency department of a community hospital. *JACEP* 1977;6(6):233-7.
46. Sinclair A, Jacobs LM, Jr. Emergency department autotransfusion for trauma victims. *Med Instrum* 1982;16(6):283-6.
47. Glover JL, Smith R, Yaw PB, et al. Autotransfusion of blood contaminated by intestinal contents. *JACEP* 1978;7(4):142-4.
48. Jurkovich GJ, Moore EE, Medina G. Autotransfusion in trauma. A pragmatic analysis. *Am J Surg* 1984;148(6):782-5.
49. Timberlake GA, McSwain NE, Jr. Autotransfusion of blood contaminated by enteric contents: a potentially life-saving measure in the massively hemorrhaging trauma patient? *J Trauma* 1988;28(6):855-7.
50. Horst HM, Dlugos S, Fath JJ, et al. Coagulopathy and intraoperative blood salvage (IBS). *J Trauma* 1992;32(5):646-52.
51. Bowley DM, Barker P, Boffard KD. Intraoperative blood salvage in penetrating abdominal trauma: a randomised, controlled trial. *World J Surg* 2006;30(6):1074-80.
52. Zantut LF, Machado MA, Volpe P, Poggetti RS, Birolini D. Autotransfusion with laparoscopically salvaged blood in trauma: report on 21 cases. *Surg Laparosc Endosc* 1996;6(1):46-8.
53. Brown TL, Skinner AM. Burn injury care for Special Forces and far-forward deployed troops. *Mil Med* 2005;170(11):919-20.
54. Calkins MD, Fitzgerald G, Bentley TB, Burris D. Intraosseous infusion devices: a comparison for potential use in special operations. *J Trauma* 2000;48(6):1068-74.
55. Butler FK, Jr., Hagmann J, Butler EG. Tactical combat casualty care in special operations. *Mil Med* 1996;161(Suppl):3-16.
56. Gurney J, Philbin N, Rice J, et al. A hemoglobin based oxygen carrier, bovine polymerized hemoglobin (HBOC-201) versus Hetastarch (HEX) in an uncontrolled liver injury hemorrhagic shock swine model with delayed evacuation. *J Trauma* 2004;57(4):726-38.
57. Philbin N, Rice J, Gurney J, et al. A hemoglobin-based oxygen carrier, bovine polymerized hemoglobin (HBOC-201) versus hetastarch (HEX) in a moderate severity hemorrhagic shock swine model with delayed evacuation. *Resuscitation* 2005;66(3):367-78.
58. Johnson T, Arnaud F, Dong F, et al. Bovine polymerized hemoglobin (hemoglobin-based oxygen carrier-201) resuscitation in three swine models of hemorrhagic shock with militarily relevant delayed evacuation--effects on histopathology and organ function. *Crit Care Med* 2006;34(5):1464-74.
59. Kauvar DS, Holcomb JB, Norris GC, Hess JR. Fresh whole blood transfusion: a controversial military practice. *J Trauma* 2006;61(1):181-4.
60. Repine TB, Perkins JG, Kauvar DS, Blackburne L. The use of fresh whole blood in massive transfusion. *J Trauma* 2006;60(6 Suppl):S59-69.
61. Newman BH, Newman DT, Ahmad R, Roth AJ. The effect of whole-blood donor adverse events on blood donor return rates. *Transfusion* 2006;46(8):1374-9.
62. Newman BH, Pichette S, Pichette D, Dzaka E. Adverse effects in blood donors after whole-blood donation: a study of 1000 blood donors interviewed 3 weeks after whole-blood donation. *Transfusion* 2003;43(5):598-603.
63. Newman BH, Roth AJ. Estimating the probability of a blood donation adverse event based on 1000 interviewed whole-blood donors. *Transfusion* 2005;45(11):1715-21.
64. Torella F, Haynes SL, Kirwan CC, Bhatt AN, McCollum CN. Acute normovolemic hemodilution and intraoperative cell salvage in aortic surgery. *J Vasc Surg* 2002;36(1):31-4.
65. Keeling MM, Gray LA, Jr., Brink MA, Hillerich VK, Bland KI. Intraoperative autotransfusion. Experience in 725 consecutive cases. *Ann Surg* 1983;197(5):536-41.
66. Smith LA, Barker DE, Burns RP. Autotransfusion utilization in abdominal trauma. *Am Surg* 1997;63(1):47-9.
67. Huth JF, Maier RV, Pavlin EG, Carrico CJ. Utilization of blood recycling in nonelective surgery. *Arch Surg* 1983;118(5):626-30.
68. Ozmen V, McSwain NR Jr, Nichols RL, Smith J, Flint LM. Autotransfusion of potentially culture-positive blood (CPB) in abdominal trauma: preliminary data from a prospective study. *J Trauma* 1992;32(1):36-9.
69. Smith LA, Barker DE, Burns RP. Autotransfusion utilization in abdominal trauma. *Am Surg* 1997;63(1):47-9.
70. Hughes LG, Thomas DW, Wareham K, et al. Intraoperative blood salvage in abdominal trauma: a review of 5 years' experience. *Anaesthesia* 2001;56(3):217-20.