Autotransfusion in the surgical management of trauma and vascular disease

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The author focuses on the methodology and the results of the use of autotransfusion in the management of 15 massive hemorrhage cases that were caused by severe trauma or vascular disease.

The prompt availability of blood products is often crucial to survival in the care of patients with severe trauma or vascular disease. With an increasing frequency of trauma, emergency surgery, and complex elective procedures, the immediate need for banked blood cannot be met.1

Over the course of some 30 years, it has been determined that the known complications of stored homologous blood are serum hepatitis, acute reaction, and complex crossmatch.1,2,8 Interest in autotransfusion, the salvage of blood loss and immediate reinfusion, has been renewed because of the shortage of banked blood and its known complications.

Since autologous blood is more physiologic, autotransfusion has these advantages: (1) decreased transmission of disease, (2) decreased incidence of immunization, (3) availability of rare blood type, (4) elimination of technical errors in crossmatching, (5) less quantity of infused ACD (acid citrate dextrose), and (6) over-ruled religious objections.

In 1874, Highmore suggested that autotransfusion might be lifesaving after a patient's postpartum exsanguination. During splenectomy for Banti's syndrome, Lockwood used autotransfusion in America in 1914.

When Thies reprinted his experience in the treatment of ruptured ectopic pregnancy, interest was stimulated in autotransfusion, and the era of modern autotransfusion began. By 1920, Burch was able to accumulate 164 cases for review.4

A lack of practical retrieval and reinfusion hampered the early attempts at autotransfusion. The current technique of autotransfusion was developed by Dyer and Klebenoff in conjunction with the Bentley Laboratories. The initial trials were in Vietnam, where, the saving value of the technique was demonstrated.5,6

Actual cases and methodology

Intraoperative autotransfusion was used in 15 patients at Saint Paul Ramsey Hospital and Medical Center, Saint Paul, Minnesota, during the years 1975 and 1976. These patients, requiring emergency surgery, had massive hemorrhage resulting from severe trauma or vascular disease. The management of these cases and any complications will be reviewed here.

On arrival in the emergency room, 10 of these patients were transferred directly to a specifically designated operating room which is always kept ready for patients who have been selected by a triage in the emergency room. In the
The selection of patients, the following criteria were used: (1) the nature of the injury, (2) presence of a vascular disease, or (3) unstable vital signs.

The surgical, x-ray, and anesthesia staff were prepared to evaluate and treat the patient immediately upon his or her arrival in the operating room. The operating room table was cassetted so that x-rays could be taken without having to move the patient. The vital signs were checked, and an EKG monitor was used. Infusion lines were established with large bore catheters, and lactated Ringer's 5% dextrose solution was infused. By insertion of a Foley catheter into the bladder, an hourly urine output check was kept on all patients.

The decision to use the autotransfusor was strictly on the basis of apparent or anticipated blood loss. Though other systems are available, the Bentley autotransfusion system (Bentley ATS 100, Bentley Laboratories, Irvine, California) was used in all cases. The system was a modification of the cardiotomy reservoir used in open heart surgery. It included a plastic disposable pre-sterilized Bentley cardiotomy reservoir, disposable suction tubing, and a single rollerhead pump. After the pre-packaged unit was opened, the suction tip and tubing were retained by the scrub nurse.

The reservoir was placed in its holder, and the infusion tubing was inserted into the head of the roller pump. The system was primed with 250 ml of lactated Ringer's solution to which had been added aqueous heparin. The amount of the heparin used depended on the size of the patient.

Intermittently during excessive intraoperative bleeding, blood was aspirated from the abdominal or thoracic cavity. The patient's blood was pumped into a clear spherical 1500-ml capacity reservoir which was volume calibrated for easy reference. In the reservoir, the blood was filtered through a 125 micron-pore filter. Here, the bubbles or microemboli were removed, along with clots, particulate matter, and contaminants.

The maximum flow rate was 600 ml per minute.\textsuperscript{5,8} With prior training, the system can be assembled in 3-5 minutes. The greatest potential hazard was air emboli. For this reason, one person should have the sole responsibility of the autotransfusor.

The suction was kept below the blood level to prevent air fluid interface which causes hemolysis. The roller pump speed was kept at a minimum to lessen cellular damage. Pressure was exerted on the reservoir, forcing the blood through one or two intravenous lines, which included additional filtration. Special cannulas were not necessary. A constant level of 200 ml was maintained in the reservoir to avoid air embolus.

Systemic heparinization was not used in the trauma patients because of the multiple injuries, especially head trauma and distal fractures. While total body heparinization was used in the four vascular disease cases, aqueous heparin was added to the reservoir in smaller amounts. In slow oozing, salvage is not practical due to excess cellular damage by the suction tip.

**Results**

The autotransfusor was utilized in 15 patients. (Table 1.) The amount of homologous blood transfused ranged from 500 ml to 9500 ml, with an average of 3867 ml per patient. The intraoperative estimated blood loss varied from 1100 ml to 13,500 ml, with an average of 7507 ml. This blood loss represented the amount autotransfused plus blood loss in the field suction and in the sponges. The amount of autologous blood reinfused was estimated. The crystalloid solution administered ranged from 1100 ml to 5850 ml with an average of 3167 ml.

The mortality rate in this series of patients was 46.6%. This rate, including a late death at 38 days, was commensurate with the massive degree of injury. Five patients died in the operating room: two from cardiac arrest during resection of ruptured abdominal aortic aneurysms, one from disseminated intra-
vascular coagulopathy, one from massive injuries (brain death), and one from uncontrolled hemorrhage.

The sixth patient, who died in the recovery room, was in an auto accident which caused liver and hepatic vessel lacerations along with head and chest injuries. There was uncontrollable bleeding from all raw surfaces despite administration of fresh frozen plasma and cryoprecipitates. Her laboratory values were: fibrinogen 118 mg, prothrombin time 16.2 sec, partial thromboplastin time more than 180 sec, thrombin time 26.9 sec, and platelets 54,000. Her arterial blood gases on 100% oxygen were: pH 7.32, pO$_2$ 42, pCO$_2$ 40, total CO$_2$ 24.6, and CaHCO$_3$ 27.4. Shortly before she expired the patient’s urine output was zero, and she had a hypothermia of 32 degrees C. This patient received 9500 ml of homologous blood in addition to approximately 3000 ml of autologous blood.

The seventh patient, also an auto accident victim, had a traumatic rupture of the thoracic descending aorta. A graft was performed. After 38 postoperative days, he had a cardiac arrest and died.

The most persistent complication was bleeding or oozing. One patient, with an extensive

Table 1
Patients receiving intraoperative autotransfusion

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Type of injury</th>
<th>Organ</th>
<th>Estimated blood loss (ml)</th>
<th>Volume of homologous blood transfused (ml)</th>
<th>Complications</th>
<th>Results</th>
<th>Days in Hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>M</td>
<td>Gunshot wound</td>
<td>Small bowel, external iliac vein &amp; artery</td>
<td>15000</td>
<td>3500</td>
<td>High output renal failure</td>
<td>Alive</td>
<td>27 days</td>
</tr>
<tr>
<td>30</td>
<td>F</td>
<td>Blunt trauma</td>
<td>Liver, spleen</td>
<td>7000</td>
<td>5000</td>
<td>Hemangia coagulopathy</td>
<td>Alive</td>
<td>16 days</td>
</tr>
<tr>
<td>73</td>
<td>M</td>
<td>Vascular disease</td>
<td>Abdom. aortic aneurysm</td>
<td>10,000</td>
<td>1500</td>
<td>Impossible to place clamp to control size of rupture exsanguination</td>
<td>Died in Operating Room</td>
<td></td>
</tr>
<tr>
<td>67</td>
<td>M</td>
<td>Blunt trauma</td>
<td>Liver, spleen</td>
<td>11,500</td>
<td>3500</td>
<td>Coagulopathy</td>
<td>Died in Operating Room</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>Stab wound</td>
<td>Heart</td>
<td>3500</td>
<td>2000</td>
<td>Anemia</td>
<td>Alive</td>
<td>8 days</td>
</tr>
<tr>
<td>42</td>
<td>M</td>
<td>Blunt trauma</td>
<td>Liver</td>
<td>5750</td>
<td>4250</td>
<td>Oliguria, abscesses, anemia</td>
<td>Alive</td>
<td>26 days</td>
</tr>
<tr>
<td>70</td>
<td>F</td>
<td>Vascular disease</td>
<td>Distal aorto-iliac occlusion</td>
<td>3750</td>
<td>2750</td>
<td>Coagulopathy, pneumonia</td>
<td>Alive</td>
<td>26 days</td>
</tr>
<tr>
<td>70</td>
<td>M</td>
<td>Blunt trauma</td>
<td>Thoracic aorta</td>
<td>12,000</td>
<td>8500</td>
<td>Coagulopathy, hemoglobinuria, renal failure, acute tubular necrosis, respiratory sepsis</td>
<td>Late death</td>
<td>38 days</td>
</tr>
<tr>
<td>17</td>
<td>F</td>
<td>Blunt trauma</td>
<td>Liver, spleen, brain</td>
<td>6000</td>
<td>1500</td>
<td>Brain death</td>
<td>Died in Operating Room</td>
<td></td>
</tr>
<tr>
<td>83</td>
<td>M</td>
<td>Vascular disease</td>
<td>Abdom. aorta</td>
<td>7000</td>
<td>4000</td>
<td>Coagulopathy, bilateral pneumonia</td>
<td>Alive</td>
<td>28 days</td>
</tr>
<tr>
<td>27</td>
<td>M</td>
<td>Blunt trauma</td>
<td>R.t. subclavian vein</td>
<td>8000</td>
<td>4000</td>
<td>Coagulopathy</td>
<td>Alive</td>
<td>12 days</td>
</tr>
<tr>
<td>78</td>
<td>M</td>
<td>Vascular disease</td>
<td>Abdom. aorta (ruptured)</td>
<td>8000</td>
<td>4500</td>
<td>Cardiac standstill</td>
<td>Died in Operating Room</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>M</td>
<td>Blunt trauma</td>
<td>Liver, gallbladder</td>
<td>1100</td>
<td>500</td>
<td>None</td>
<td>Alive</td>
<td>12 days</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>Blunt trauma</td>
<td>Spleen, left lung</td>
<td>10,000</td>
<td>5000</td>
<td>Air leak, urine output, BP, pulmonary edema</td>
<td>Died in Operating Room</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>F</td>
<td>Blunt trauma</td>
<td>Liver, hepatic vessels, skull</td>
<td>13,800</td>
<td>9500</td>
<td>Coagulopathy, hemoglobinuria, hemolytic anemia, urine output, cardiac arrest</td>
<td>Died in Recovery Room</td>
<td></td>
</tr>
</tbody>
</table>

*Includes volume autotransfused plus volume lost on sponges and induction
liver lobe resection, had a prothrombin time of more than 60 sec, partial thromboplastin time of more than 180 sec, and a thrombin time of more than 120 sec. His platelet count was 45,000. He died during the operative period of persistent severe coagulopathy.

Several factors may cause platelet damage: (1) trapped platelets by the filters in the system, (2) lost clotting factors—not all blood salvaged, (3) possible rapid utilization of platelets and clotting factors to lower levels than necessary for hemostasis, or (4) hypercoagulability—causation thought to be reinfusion of hemolyzed erythrocytes. As a result, hypercoagulability may cause intravascular coagulation, and this condition is accentuated by a splenectomy. Many trauma patients undergo splenectomies.5-9

Transient hemoglobinuria, observed in three patients, was corrected with forced diuresis. Renal failure was noted in two patients. One patient had a high output failure because of decreased renal perfusion caused by prolonged hypotension. Fourteen days postoperatively, a second patient had a low output renal failure and acute tubular necrosis. Yet, in another patient, oliguria was corrected with fluids and plasma.

Several patients had anemia. Cellular destruction was thought to be caused by extravascular tissue contact. The lysis of erythrocytes is a normal foreign body defense mechanism of serious linings of body cavities. Evidence suggests that erythrocytes which survive autotransfusion have a normal life span.8

There were two cases of pneumonia, one of tracheitis, one of pancreatitis, and one of respiratory sepsis. Infection did not develop in the patient who had a small bowel resection; for two weeks postoperatively, he had been treated with Cleocin.6

Conclusion

The records of 15 autotransfused patients who required emergency surgery for trauma or vascular disease have been reviewed. All of these patients received homologous blood. Thrombocytopenia, hypofibrinogenemia, and hemolysis are related to patients who had massive blood loss. This coagulopathy is similar to that which occurs with homologous blood. There is not much to be gained by the use of homologous blood, and autologous blood is more physiologic. The need for large volumes of blood can be met immediately without waiting for banked blood. The patients tolerate plasma free hemoglobin, provided acidosis and inadequate kidney perfusion are prevented.7

Autotransfusion is crucial to survival. It will be more practical when the anticoagulant for intraoperative autotransfusion is absolutely determined. Reservoir heparinization is not practical for large volume anticoagulation.9,10 When the amount of anticoagulant is insufficient, the tendency toward disseminated intravascular coagulopathy increases. The coagulopathy directly related to autotransfusion was impossible to determine because the patients also received homologous blood. The anticoagulation difficulties deserve further laboratory and clinical investigation.

REFERENCES

AUTHOR

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