Hemodynamic improvement in hemorrhagic shock by aortic balloon occlusion and hypertonic saline solutions

Vascular Surgery, Department of Surgery, Escola Paulista de Medicina, São Paulo, SP, Brazil

The initial treatment of uncontrolled hemorrhagic shock from an abdominal source is controversial. The hemodynamic effects of transfemoral diaphragmatic aortic occlusion with a balloon followed by a single bolus of hypertonic saline solutions have been evaluated in 28 dogs. The animals were submitted to pressure-driven hemorrhage for 90 min, according to mean arterial pressure in the abdominal aorta and randomized into four groups, according to the treatment employed at 34 min after hemorrhage. Group 1 dogs (controls) received isotonic NaCl (0.9%, 308 mOsm/l, 4 ml/kg) without aortic occlusion; group 2 underwent aortic occlusion and received isotonic NaCl (0.9%, 308 mOsm/l, 4 ml/kg); group 3 were occluded and received hypertonic NaCl (7.5%, 2400 mOsm/l, 4 ml/kg); group 4 were occluded and received hypertonic sodium acetate (10.5%, 2400 mOsm/l, 4 ml/kg). There were no significant differences between groups at basal measures and also after 30 min of continuous bleeding, when animals presented with severe shock, and significant decreases in mean arterial pressure, cardiac index, systolic index and cardiac filling pressures; the systemic vascular resistance index was increased. Control animals remained in severe shock throughout the experiment and three died. The recovery of mean arterial pressure in aortic-occ.1uded dogs given isotonic NaCl was associated with a marked increase in systemic vascular resistance index, without improvements in cardiac index, systolic index and cardiac filling pressures. In occluded dogs given hypertonic NaCl and NaAc the mean arterial pressure recovery lasted longer, with lower increases in systemic vascular resistance index, while the cardiac index, systolic index and cardiac filling pressures showed a marked albeit transient increase. Injection of hypertonic saline following aortic occlusion produced significantly better hemodynamic profiles and should be seriously considered for the first treatment in severe uncontrolled hemorrhagic shock from an abdominal vascular source.

Keywords: hemorrhagic shock, aortic occlusion, hypertonic solutions, shock therapy, vascular trauma, aortic aneurysm

Severe hemorrhagic shock from massive blood loss is the most frequent cause of the high mortality associated with the two most frequent abdominal vascular 'catastrophes' — abdominal vascular trauma, affecting mainly young and otherwise healthy patients, and ruptured abdominal aortic aneurysms, affecting older patients with frequent coronary and cerebrovascular diseases. The main prognostic factors in patients who reach the hospital alive are the initial systolic blood pressure and the duration of shock. Anesthesia and abdominal decompression, secondary to laparotomy, may precipitate cardiac arrest in patients in severe shock. Proximal aortic control in such patients is difficult with active bleeding, and the anatomic derangement caused by hematoma predisposes the patient to inadvertent venous and intestinal lesions that
worsen the prognosis\textsuperscript{11–18}. For patients with severe hemodynamic instability, several authors recommend thoracotomy and descending aortic cross-clamping before laparotomy to reduce blood loss and preserve flow to the cerebral and coronary vessels\textsuperscript{13,16,19–30}. Aortic compression or cross-clamping at the diaphragmatic hiatus to avoid the additional morbidity of a thoracotomy has been preferred by others\textsuperscript{18,19,31,32}.

The use of preoperative intra-aortic balloon occlusion was first conceived and applied during the Korean War\textsuperscript{33,34} and has subsequently gained popularity. It is frequently used as a fast, easy and effective way to gain control of the proximal aorta, which allows immediate reduction in blood loss, recovery of arterial pressure, better hemodynamic stability during anesthesia and fewer problems with abdominal decompression during laparotomy\textsuperscript{13,16,35–37}.

The control of hemorrhage must also be the primary therapeutic intervention in abdominal vascular trauma or rupture of an aortic aneurysm. The use of the MAST suit, initially thought to control bleeding as well as improve hemodynamic patterns, is no longer recommended because it does not provide effective bleeding control nor improve outcome in clinical studies\textsuperscript{38,39}.

Thus, aortic balloon occlusion has been extensively recommended for preoperative control of intra-abdominal bleeding, especially in patients close to a moribund state as a result of penetrating trauma to the torso\textsuperscript{37}.

Intravenous infusion has been the mainstay of pre-hospital and emergency room management of these patients, but infusion of conventional crystalloid solutions appears to be of little benefit in the pre-hospital treatment of critically injured trauma victims when large-volume resuscitation is usually impossible\textsuperscript{40–42}. Smaller-volume infusion of hypertonic saline solutions has been the subject of both laboratory and clinical studies, demonstrating a rapid expansion of plasma volume by an osmotic fluid shift from the intracellular to the intravascular space. This infusion, providing a rapid and sustained stabilization of cardiac output and arterial pressure, increases microcirculation and decreases splanchnic resistance, reduces initial fluid requirements, temporarily reduces cerebral edema in head trauma victims, and improves survival as a consequence of the rapid reversal of shock during the initial phase of injury\textsuperscript{43–61}. Several authors believe that raising blood pressure with vigorous fluid infusion, or with hypertonic saline preoperatively, may result in greater blood loss in uncontrolled hemorrhage, which would worsen prognosis\textsuperscript{41,42,62–67}.

The use of the hypertonic solution with the attractive immediate effects obtained by small-volume infusion after the aortic control by balloon occlusion in severe hemorrhagic shock has not been described previously\textsuperscript{68,69}.

The aim of the present study was to determine the hemodynamic effects of the aortic balloon occlusion followed by hypertonic saline solutions in the treatment of severe uncontrolled hemorrhagic shock.

Materials and methods

Animals

This project was approved by the Scientific Committee of the Escola Paulista de Medicina and the experiments were performed in line with the NIH guidelines for the use of experimental animals. Experiments were performed on 28 mongrel dogs fed standard dog chow and given water \textit{ad libitum} for 1 week in the divisional kennel. Food was removed 12 h and water 6 h before induction of anesthesia with pentobarbital sodium, 25 mg/kg. Small doses of pentobarbital were given throughout the experiment to provide superficial anesthesia and to allow animals to breathe room air spontaneously through a cuffed tracheal cannula.

Hemodynamic procedures

Polyethylene cannulas (8 Fr) were inserted into the right femoral vein to allow infusion of solutions in the inferior vena cava; the right carotid artery was dissected and cannulated to allow continuous monitoring of mean arterial pressure (MAP) at the proximal aorta and the right femoral artery to monitor mean arterial pressure of the abdominal aorta and to control the rate of bleeding. A three-way thermodilution 7 Fr catheter was introduced under radioscopic monitoring through the right external jugular vein to the right pulmonary arterial branch to monitor mean pulmonary arterial pressure and pulmonary wedge pressure; cardiac output was computed by thermodilution after intravenous injections of 5 ml of saline at 4°C by means of a Gould SP1435 cardiac output computer (Gould Inc., Cleveland, Ohio, USA). Pressures were determined and registered by a Gould–Statham p23Db transducer (Gould Inc.) and hematocrits by microcentrifugation. The left femoral artery was dissected and prepared for cannulation with an 8 Fr Fogarty occlusion catheter after 34 min of blood loss, when, under radioscopic monitoring, the descending aorta at the level of the tenth thoracic vertebrae (T10) was occluded (Figure 1).

Uncontrolled hemorrhagic shock was produced by the pressure-driven hemorrhage method\textsuperscript{61,70}, namely, an initial bleeding rate (25 ml/min) was set and reset minute-to-minute in proportion to the prevailing MAP of the abdominal aorta. This initial bleeding rate was based on previous studies\textsuperscript{61,70} that showed severe shock to be induced within a time interval (30 min) compatible with average pre-hospital delay in medical/paramedical care, and was kept fixed until MAP of the abdominal aorta (MAPAA) fell to or below 100 mmHg. Thereafter, the bleeding rate (BR) at any given minute (BR\textsubscript{n}) was equal to \( BR = 25 \times MAPAA_{n-1}/100 \), where MAPAA\textsubscript{(n-1)} was the MAP of the
Hemodynamic improvement in hemorrhagic shock: L. F. Poli de Figueiredo et al.

Figure 1 Experimental preparation. 1. endotracheal tube; 2. aortic balloon occlusion at T10 level; 3. mean arterial pressure measurement in abdominal aorta; 4. infusion of solutions into inferior vena cava; 5. triple-lumen thermodilution catheter at pulmonary arterial branch; 6. cardiac output computer; 7. proximal arterial pressure monitor

abdominal aorta in the previous minute. BRn was adjusted to MAP\(_{(n-1)}\) on a minute-by-minute basis, according to the following schedule: the total volume of blood scheduled for removal during the \((n-1)\)th min was withdrawn from the cannulated right carotid artery over the first 30 s of the minute; the average MAP prevailing during the last 30 s of the minute was used to determine bleeding rate\(^{64,70}\).

Complete hemodynamic profiles were determined at baseline conditions, just before hemorrhage (0 min), and at 30, 40, 60 and 90 min of pressure-driven hemorrhage. The animals were submitted to the same experimental protocol and assigned randomly into four experimental groups \((n = 7\) per group\) according to the intervention employed at minute 34: group 1 (controls) received isotonic NaCl \((0.9\%, 308 \text{ mOsm/l, } 4 \text{ ml/kg})\) and no aortic occlusion; group 2 underwent aortic occlusion and received isotonic NaCl \((0.9\%, 308 \text{ mOsm/l, } 4 \text{ ml/kg})\); group 3 underwent aortic occlusion and received hypertonic NaCl \((7.5\%, 2400 \text{ mOsm/l, } 4 \text{ ml/kg})\); and group 4 underwent aortic occlusion and received hypertonic sodium acetate (NaAc) \((10.5\%, 2400 \text{ mOsm/l, } 4 \text{ ml/kg})\).

All solutions were given as a bolus injected during a 2-min interval into the inferior vena cava immediately after the aortic balloon occlusion at T10, as described earlier.

Statistical analysis

The weights of the animals were compared by one-way analysis of variance. The hematocrit and hemodynamic data were compared by multivariate profile analysis appropriate for each comparison as described by Wilks, Roy and Lawley–Hartley\(^{71,72}\). The significant differences were assigned for a \(P\) value of < 0.01.

Results

The body weight of the experimental animals, hematocrit and all hemodynamic data were similar between groups at baseline conditions. Pressure-driven hemorrhage produced a similar severe shock state in all animals during the first 34 min when a mean 42% of the estimated initial blood volume was removed. All groups showed significant decreases in MAP, cardiac index, systolic index and in mean pulmonary arterial and wedge pressures; systemic vascular resistance rose, but pulmonary vascular resistance showed no significant alterations (Figures 2–10).

Control animals remained in this severe condition of hemodynamic derangement after the small infusion of isotonic saline, and presented a further increase in pulmonary vascular resistance (Figure 9), in spite of very low cardiac filling pressures (Figures 6 and 7); three animals from this group died close to the end of the experiment, when almost 70% of the estimated initial blood volume had been extracted (Figure 2).

![Figure 2](https://example.com/figure2.png) Cumulative blood loss (ml/kg) during pressure-driven hemorrhage and after treatment. No significant differences were observed between groups throughout the experiment. ○, controls; □, occlusion + isotonic NaCl; △, occlusion + hypertonic NaCl; A, occlusion + hypertonic NaAc
Figure 3 Mean arterial pressure (mmHg) during pressure-driven hemorrhage and after treatment. ●: controls; ○: occlusion + isotonic NaCl; ◇: occlusion + hypertonic NaCl; ▲: occlusion + hypertonic NaAc. (*P<0.01; †P<0.01)

Figure 6 Mean pulmonary arterial pressure (mmHg) during pressure-driven hemorrhage and after treatment. ●: controls; ○: occlusion + isotonic NaCl; ◇: occlusion + hypertonic NaCl; ▲: occlusion + hypertonic NaAc. (*P<0.01)

Figure 4 Cardiac index (l.min⁻¹.m⁻²) during pressure-driven hemorrhage and after treatment. ●: controls; ○: occlusion + isotonic NaCl; ◇: occlusion + hypertonic NaCl; ▲: occlusion + hypertonic NaAc. (*P<0.01; †P<0.01)

Figure 7 Mean pulmonary wedge pressure (mmHg) during pressure-driven hemorrhage and after treatment. ●: controls; ○: occlusion + isotonic NaCl; ◇: occlusion + hypertonic NaCl; ▲: occlusion + hypertonic NaAc. (*P<0.01; †P<0.01)

Figure 5 Systolic index (ml.beat⁻¹.m⁻²) during pressure-driven hemorrhage and after treatment. ●: controls; ○: occlusion + isotonic NaCl; ◇: occlusion + hypertonic NaCl; ▲: occlusion + hypertonic NaAc. (*P<0.01; †P<0.01)

Figure 8 Systemic vascular resistance index (dyna.sec.cm⁻⁴.m⁻²) during pressure-driven hemorrhage and after treatment. ●: controls; ○: occlusion + isotonic NaCl; ◇: occlusion + hypertonic NaCl; ▲: occlusion + hypertonic NaAc. (*P<0.01; †P<0.01; ‡P<0.01)
Hemodynamic improvement in hemorrhagic shock: L. F. Poli de Figueiredo et al.

All groups submitted to aortic occlusion presented a significant reduction in MAP of the abdominal aorta, inferior to that in controls (Figure 10). Consequently, the rate of bleeding was reduced in obedience to the pressure-driven hemorrhage model. However, the cumulative blood loss did not show significant differences between all groups during the entire experiment (Figure 2). Aortic occlusion followed by isotonic saline provided a complete recovery of the initial MAP that was maintained until 60 min of blood loss, similar to that in occluded dogs given either hypertonic NaCl or NaAc (Figure 3), but this recovery was due only to a marked increase in systemic vascular resistance (Figure 8), with no other benefits in cardiac output, systolic volume or cardiac filling pressures (Figures 4–7). On completion of the experiment, dogs given isotonic saline showed significantly lower mean arterial pressure than those occluded and given hypertonic NaCl or NaAc (Figure 3) in spite of an extremely elevated systemic vascular resistance index (Figure 8). Pulmonary vascular resistance also showed a marked increase (Figure 9).

The use of hypertonic NaCl (7.5%) after aortic occlusion provided a more sustained blood pressure recovery throughout the experiment (Figure 3), associated with a much smaller increase in systemic vascular resistance (Figure 8). There were also significant transient increases in cardiac output, systolic volume and cardiac filling pressure, without significant alterations in pulmonary vascular resistance (Figures 4–7 and 9).

The use of hypertonic sodium acetate provided the most significant hemodynamic improvement immediately after infusion, and by far the greatest initial cardiac output (Figure 4), systolic volume (Figure 5), and cardiac filling pressures (Figures 6 and 7). The MAP recovery was similar to that in occluded dogs given isotonic or hypertonic NaCl (Figure 3), but the systemic vascular resistance index was lower than initial baseline determinations (Figure 8). After 60 min, the hemodynamics after hypertonic sodium acetate were similar to those of hypertonic saline-treated dogs, except for a lower systemic vascular resistance index and at 90 min showed lower MAP than hypertonic saline-treated dogs, but higher than in those given isotonic saline (Figure 3).

The hematocrit did not vary in controls and isotonic saline-treated dogs throughout the experiment. Injection of both hypertonic solutions caused a significant immediate decrease that remained stable (Figure 11).

At 90 min of blood loss, cardiac output, systolic volume and cardiac filling pressures were similar between groups (Figures 4–7), but no deaths occurred before the end of the experiment in all animals submitted to aortic occlusion. Dogs given isotonic saline showed significantly lower MAP (Figure 3) and higher systemic and pulmonary vascular resistance than those given hypertonic NaCl or NaAc (Figures 8 and 9).
Discussion

Pressure-driven hemorrhage is a new experimental model of hemorrhage that simulates blood loss through a large arterial wound in a worst possible scenario, because arterial bleeding is governed by transmural pressure, with a number of naturally intervening factors that reduce blood loss (excluded in this model), such as vasoconstriction, coagulation and tamponade. The bleeding rate behaved as a simple linear function of prevailing blood pressure at the abdominal aorta and demonstrated a typical picture of severe uncontrolled hemorrhagic shock. The model is easily reproduced, the present findings being very similar to those of Rocha e Silva et al., with severe hypotension, very low filling pressures and cardiac output, associated with increasing systemic and pulmonary resistance.

The clinical picture of a patient with a large abdominal vascular lesion is similar to this magnitude of shock, and for older patients with ruptured abdominal aneurysms, the presence of co-existing occlusive lesions further reduces tolerance to low-flow states. Emergency aortic control is therefore mandatory. Several techniques are described, but aortic balloon occlusion is a fast and relatively easy way to gain control of the situation before surgery. The hemodynamic benefits of aortic occlusion during severe hemorrhagic shock were demonstrated by Dunn et al., when blood pressure recovered by increased systemic vascular resistance and improved cardiac output and systolic volume after blood transfusion. A multicenter trial that used transfemoral aortic balloon occlusion in hypertensive victims of penetrating abdominal trauma, showed benefits only when blood pressure increased with this maneuver. The present experiments showed that occlusion provides blood pressure recovery by a marked increase in vascular resistance, benefiting coronary and cerebral flows, but no other hemodynamic improvement was noted. Dunn and colleagues re-infused the shed blood, observing better hemodynamic profiles. However, with severe abdominal trauma, blood is not immediately available and large amounts of fluid infusions take time. Hypertonic solutions have been used safely and have benefited large numbers of patients. The impressive results obtained with these small volumes of hypertonic saline solution after occlusion strongly support its clinical use. It was found in the present study that blood pressure recovers not exclusively by an increase in vascular resistance, which occurs later, but as a result of the immediate increase in cardiac filling pressures and cardiac output. This fact is documented extensively in a large number of studies, and was highly significant in the present experiment.

Controversy in the literature about raising the blood pressure with fluids and solutions before hemorrhage control further supports the authors' findings that the significant benefits of hypertonic solutions can greatly and rapidly aid in the control of hemorrhage from an abdominal vascular source — the most important maneuver in hemorrhagic shock patients.

It is concluded that the use of small-volume hypertonic saline solutions after aortic occlusion should be strongly considered for initial treatment in patients with abdominal vascular trauma or ruptured aortic aneurysms with a rapid onset of hemorrhagic shock.

References

Hemodynamic improvement in hemorrhagic shock: L. F. Poli de Figueiredo et al.


Paper accepted 14 July 1994