The role of deliberate hypotension

Richard P Dutton

Treatment of haemorrhagic shock consists of finding the source of bleeding and correcting it, and restoring normal intravascular fluid volume (American College of Surgeons Committee on Trauma, 1997). There is general consensus that fluid administration is ultimately required for successful resuscitation. In the long run, restoration of fluid volume is required to support systemic perfusion and restore aerobic metabolism. Failure to do so is associated with poor clinical outcomes (Abramson et al, 1993). For this reason, the advanced trauma life support curriculum recommends administration of up to 2 litres of intravenous fluids early in resuscitation, followed by continued blood and fluid therapy to support a normal systolic blood pressure.

Early aggressive intravenous therapy of the patient who is still actively haemorrhaging may be counterproductive, however. Increase in preload leads to increased cardiac output and blood pressure, with reversal of compensatory vasoconstriction in injured vessels. Non-blood intravenous solutions dilute oxygen-carrying capacity, and platelet and coagulation factor concentration. Rapid fluid administration may contribute to systemic hypothermia and electrolyte abnormalities, and common isotonic crystalloid solutions are potent immune system activators (Rhee et al, 1998). The net result is a substantial increase in haemorrhage volume, with the potential for creation of a downward spiral of bleeding, fluid administration and further bleeding. The possibility of a benefit from deliberate under-administration of fluids during early resuscitation – before definitive control of haemorrhage – has been explored by a number of authors over the past two decades, in both animal models and clinical practice.

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IN SUPPORT OF DELIBERATE HYPOTENSIVE RESUSCITATION

Laboratory data

Beginning with Shaftan et al (1965), many have sought to characterize the fine balance between exacerbation of haemorrhage and support of perfusion in early haemorrhagic shock. They examined a non-lethal model of femoral artery bleeding in dogs, concluding that blood loss was greatest when the blood pressure was supported by either fluid therapy or vasopressors, and that spontaneous haemostasis occurred fastest with either no resuscitation or active administration of vasodilators. Stern et al (1993) was one of several to use an aortic injury model in swine, demonstrating that survival was best when fluids were administered but titrated to a lower than normal blood pressure.

Capone et al (1995) constructed an elegant model of tail laceration haemorrhage in rats, incorporating a period of 'prehospital' resuscitation before definitive surgical repair of the injury. He also demonstrated increased mortality with either no resuscitation or resuscitation to a normal blood pressure, finding that survival was greatest when fluids were given but the blood pressure was kept lower than normal.

Shoemaker et al (1996) presented an excellent summary of the extensive animal literature supporting deliberate hypotensive resuscitation. Further studies using similar models of uncontrolled haemorrhage but more complex monitoring technology have demonstrated that oxygen delivery (perfusion) may actually be highest at a lower than normal blood pressure, presumably because of decreased haemodilution (Riddez et al, 1998).

Human data

Clinical application of this approach began in the early 1990s. Bickell et al (1994) showed improved outcome following deliberate restriction of early fluid therapy to victims of penetrating torso trauma. Demetriades et al (1996) noted improved survival in a similar population of penetrating trauma patients delivered to the hospital by private conveyance, as compared to ambulance service. In a retrospective review of patients resuscitated using a rapid infusion system, Hambly and Dutton (1996) noted that mortality was greater than expected, and that case-matched controls received less fluid and had improved survival.

Dutton et al (2002) conducted the most recently published clinical trial of deliberate hypotensive resuscitation. Blunt and penetrating

trauma patients (n=110) were randomized to fluid therapy at a mean arterial pressure (MAP) of 60 mmHg *vs* 80 mmHg until the end of active bleeding. Mortality was identical in the two groups, despite a higher injury severity in the low pressure group, suggesting, at the very least, the safety of deliberate hypotensive resuscitation. This finding has encouraged the further application of this technique to patient populations at high risk for ongoing haemorrhage that is difficult to surgically control, such as those with liver or pelvic bleeding.

CONCLUSIONS

There is little doubt that increasing the amount of fluid administered to the actively haemorrhaging patient increases the rate of bleeding, with potentially deleterious consequences. Less well understood is what lower threshold of perfusion is safely tolerated in these patients. Improved monitoring, particularly for subtle indicators of shock, will allow for more precise titration of fluid therapy in the future, allowing the clinician to safely navigate between increased haemorrhage on the one hand and hypoperfusion on the other. In combination with steady improvement in the ability to diagnose and control the source of bleeding, controlled hypotensive resuscitation offers real hope for improving outcomes from traumatic haemorrhage. HM

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KEY POINTS

- In the patient with unconrolled haemorrhage, any increase in blood pressure will lead to increased bleeding, clot wash-out and haemodilution.
- Deliberate hypotension is a viable strategy if it does not increase ischaemic injury and results in more rapid haemostasis.
- Better real-time monitoring of tissue oxygen delivery will allow for more precision in resuscitation in the future.