Give oxygen, get a blood pressure... but don't overdo it

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Medical practice is based on a bedrock of conventional doctrine yet can be readily influenced by papers from high-impact factor journals and adroit marketing by opinion leaders and/or industry. These fads and fashions variably stand the test of time – some become ensconced as established dogma, some are jettisoned for ever, while others are discarded then belatedly rediscovered. Aspects of cardiorespiratory management of the trauma patient fall into all the above categories. Alas, the evidence base that makes these 'facts' incontrovertible is largely lacking. This article will attempt to steer a course through fact and supposition and will conclude with recommendations that, to the author at least, are founded in commonsense.

HISTORICAL PRACTICE

The management of trauma during the London Blitz in 1940 and in the front line in Italy in 1944–5 was studied by Grant and Reeve (1951). In the Blitz they noted that blood transfusion was

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deemed a last resort, and only then given slowly and in small amounts. Early transfusion was considered dangerous because of the likelihood of renewing bleeding, while rapid or large transfusions were thought likely to overburden the circulation and cause pulmonary oedema. It was thought useless to attempt restoration of a moribund patient. However, by the end of the war, blood transfusion had become an established therapy and the authors commented that adequate replacement of blood loss was an important factor in mitigating illness in the late post-operation period. Today, the same discussions continue.

Protagonists claim, on the one hand, that a low blood pressure will lessen the risk of renewed bleeding and/or reduce the degree of blood loss, with less consumption coagulopathy and, potentially, a lower risk of multiorgan failure. The other school of thought argues that a critically low oxygen delivery and a reduced driving blood pressure will compromise organ perfusion, and thus precipitate multiorgan failure.

A recent Cochrane systematic review of the evidence was sadly underwhelming because of the paucity of randomized, controlled trial (RCT) data (Kwan et al, 2004). The authors commented that they:

> **'… found no evidence from RCTs for or against early or larger volume of IV [intravenous] fluid administration in uncontrolled haemorrhage'.**

They noted the continuing uncertainty about the best fluid administration strategy in bleeding trauma patients and emphasized the need for further studies. Of the three RCTs on the topic comparing early *vs* delayed fluid resuscitation, only the study by Bickell et al (1994) yielded a positive result. However, it should be stressed that their patient population received penetrating torso trauma, were delivered to a high-quality trauma centre where 'time-to-knife' was short, and systolic blood pressures on arrival at hospital were not particularly low (averaging 113 mmHg). The resuscitation fluid given was Ringer's acetate, a solution not used in Europe and over which some doubt should be cast in view of the decrease in acetate metabolism by hypoperfused muscle. Extrapolation cannot in all cases be made to other patient groups, including those suffering blunt trauma or head injury. The largest trial to date comparing larger *vs* smaller volume resuscitation, performed by Dutton et al (2002) in 110 patients suffering either blunt or penetrating trauma, found no difference in mortality when aiming for either a blood pressure of 70 or 100 mmHg systolic.

HOW IMPORTANT IS BLOOD PRESSURE?

Perhaps we should not be even debating ideal pressure as this measure is well recognized to be a poor indicator of circulating blood volume. Even Grant and Reeve in their Second World War study commented on how restoring blood pressure to 100 mmHg or over was no guarantee of adequacy of resuscitation (Grant and Reeve, 1951). A useful analogy can be drawn to perioperative circulatory optimization in patients undergoing high-risk elective or emergency surgery. Kern and Shoemaker (2002) undertook a systematic review and found significant benefit (averaging a 23% reduction in mortality) was obtained through achieving a 'supranormal' circulation using fluid loading and/or inotropes before the onset of organ failure to achieve specified values of cardiac output and oxygen delivery and consumption.

A similar approach instituted after the onset of multiorgan failure resulted in no difference, emphasizing the need for active, early resuscitation. Even prompt postoperative resuscitation directed towards achieving specific targets – mixed venous oxygen saturation and blood lactate by Polonen et al (2000) and stroke volume by McKendry et al (2004) – significantly reduced hospital stay after cardiac surgery. The latter study is notable for using minimally invasive cardiac output monitoring (oesophageal Doppler), a technology previously used to optimize stroke volume in several intraoperative studies, again with outcome benefit (Sinclair et al, 1997; Gan et al, 2002). This technology, among others, is now available to measure cardiac output rapidly, reliably and relatively non-invasively. A surrogate for the adequacy of cardiac output, namely central venous oxygen saturation, was successfully used in a septic population presenting to an emergency department in Detroit by Rivers and colleagues (2001). They targeted a central venous saturation above 70%, and mirrored the results obtained in trauma or major surgery, lowering hospital mortality from 46.5% to 30.5%.

THE ROLE OF OXYGEN

The oxygen delivery equation comprises haemoglobin, cardiac output and arterial oxygen saturation. To leave an acutely traumatized patient hypoxaemic will also contribute to the development of a tissue oxygen debt and an increased likelihood of developing organ dysfunction. Few clinical studies are available. Resuscitation with air was compared to 100% oxygen in neonates (Vento et al, 2001). Air resuscitation appeared to be safe, associated with faster clinical recovery and less biochemical evidence of oxidative stress; however, no differences were shown on long-term follow up (Saugstad et al, 2003). The immaturity of neonatal lungs and the unaccustomed exposure to high-level oxygen are plausible explanations for oxygen toxicity in this cohort. The increased risk of a worsened ischaemia-reperfusion injury by hyperoxia has not been demonstrated in adult patients. A small study in patients with severe head injury used cerebral microdialysis to measure lactate and glucose levels in patients treated with oxygen levels up to 100% over a 6-hour period (Menzel et al, 1999). Compared to a control cohort treated conventionally, the mean dialysate lactate levels decreased by 40% yet glucose levels were somewhat variable. The significance of this finding has yet to be determined.

Animal haemorrhage studies suggest both the potential to harm from hyperoxic ventilation, including coronary vasoconstriction and decreased coronary blood flow (Kemming et al, 2004), but also benefit with an increased survival rate found in a partially resuscitated pig haemorrhage model (Meier et al, 2004). Another interesting laboratory study of hyperoxia demonstrated decreased microcirculatory vessel diameter and flow (Tsai et al, 2003), thus a high arterial oxygen tension may potentially compromise tissue oxygenation. The benefits of hyperoxia, at least in terms of oxygen delivery, are limited unless the patient is moved to a hyperbaric environment to increase the amount of oxygen dissolved in plasma. Therfore, a more rational approach to oxygenation would be to titrate supplemental oxygen therapy to achieve arterial oxygen saturations of 98–99%, thereby avoiding the damaging extremes of both hyperoxia and hypoxia.

CONCLUSIONS

Our resuscitation goals should shift away from an outdated reliance on blood pressure and 'blind' administration of high-flow oxygen toward a more directed, hi-tech monitored approach involving stroke volume optimization with maintenance of normoxia and an adequate haematocrit. The blood pressure target should be viewed as a secondary, albeit still important, objective that should be individualized. A chronic hypertensive is likely to require higher perfusion pressures than a previously fit young adult yet the optimal value should be sought by reference to markers of adequacy such as urine output, blood pressure or lack of electrocardiographic features of ischaemia. In this manner we are more likely to improve prognosis. HM

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KEY POINTS

- Targeted cardiorespiratory goals should be sought using more sophisticated, yet easily applied, monitoring devices.
- Stroke volume should be optimized using a Frank–Starling-type approach, i.e. no further increase in stroke volume following a fluid challenge.
- The adequacy of tissue oxygen delivery should be confirmed by keeping central venous oxygen saturation values above 70%, in addition to other clinical markers such as urine output and metabolic acidosis.
- Blood pressure should be maintained at a level that provides an adequate perfusion pressure for the individual patient. In general, this can be at levels considerably below normal values but should not be excessively low.
- Arterial oxygen saturation should be maintained at 98–99%. While it is reasonable to use high-flow, high-concentration oxygen as a component of immediate 'ABC' resuscitation, this should rapidly be titrated downwards to prevent prolonged hyperoxia.