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Editorial for:

Carrick MM, Morrison CA, Tapia NM, et al. Intraoperative hypotensive resuscitation for patients undergoing laparotomy or thoracotomy for trauma: Early termination of a randomized prospective clinical trial. J Trauma Acute Care Surg 2016; 80:

886-896.

What are we to make of the recent paper by Carrick et al. which found no benefit to survival from deliberate intraoperative hypotension in victims of penetrating trauma? Should this change our management of these patients?

No.

Why not? On the face of it, this seems like a convincing study by a reputable group. It was conducted with exemption from informed consent. The primary and secondary endpoints were established a priori. The enrolled patients were well-balanced for risk factors. And the outcomes were remarkably similar in their lack of difference for both mortality and major postoperative morbidities. So why shouldn't we believe the results?

First, the authors appear to be studying a moot problem. It took them 6 years to enroll the 180 patients in this trial (which was stopped early – they intended to include 270). Given the rapid pace of change in healthcare, this makes it probable that patients at the end of the trial were treated differently than patients at the beginning. And the small numbers themselves make it statistically unlikely that a small difference will be detected.

Second, the way the study and control groups were established reflects a change in how hypotension during acute resuscitation is currently viewed. The MAP target of 65 in the 'control' group would have been considered low in the deliberate hypotension trials of 15-20 years ago. So really this was a comparison of 'low' to 'really-low.' Again, this makes it unlikely that outcomes would be different.

Third, the effects of anesthesia on blood pressure were completely ignored. There is a single cryptic statement to the effect that the anesthesiologists could not deliberately lower pressure to a MAP of 55 in the study group. No comparison of MAC equivalents between groups was offered, so it is impossible to know if the low group was: 1) in hemorrhagic shock with minimal anesthetic and limited fluid replacement, or 2) adequately anesthetized while receiving less



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fluid. Ditto for the control group. Because anesthesia was uncontrolled, and thus the reversal of compensatory vasoconstriction is unknown, it is hard to interpret the outcomes. Most human trials of acute resuscitation share this issue – without a true understanding of the patient's physiology it is difficult to isolate the effects of any one intervention.

But we can still learn from this trial. The very good news is that the mortality observed is less than half of that in the original paper by this group in 1994. Something we are doing is making a difference: prehospital transport; damage control technique; new surgical instruments; topical hemostatics; earlier transfusion; or less crystalloid. Both the study and control group received substantially better care than two decades ago – something we can all take credit for!