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"Fluidless" resuscitation with permissive hypotension via impedance threshold device therapy compared with normal saline resuscitation in a porcine model of severe hemorrhage.

Metzger A, Rees J, Segal N, McKnite S, Matsuura T, Convertino VA, Gerhardt RT, Lurie KG.

BACKGROUND: One approach to improve outcomes after trauma and hemorrhage is to follow the principles of permissive hypotension by avoiding intravascular overpressure and thereby preventing dislodgement of platelet plugs early in the clotting process. We hypothesized that augmentation of negative intrathoracic pressure (nITP) by treatment with an impedance threshold device would improve hemodynamics without compromising permissive hypotension or causing hemodilution, whereas aggressive fluid resuscitation with normal saline (NS) would result in hemodilution and SBPs that are too high for permissive hypotension and capable of clot dislodgement.

METHODS: Thirty-four spontaneously breathing anesthetized female pigs (30.6 ± 0.5 kg) were subjected to a fixed 55% hemorrhage over 30 minutes; block randomized to nITP, no treatment, or intravenous bolus of 1-L NS; and evaluated over 30 minutes. Results are reported as mean ± SEM.

RESULTS: Average systolic blood pressures (SBPs) (mm Hg) 30 minutes after the study interventions were as follows: nITP, 82.1 ± 2.9; no treatment, 69.4 ± 4.0; NS 89.3 ± 5.2. Maximum SBPs during the initial 15 minutes of treatment were as follows: nITP, 88.0 ± 4.3; no treatment, 70.8 ± 4.3; and NS, 131 ± 7.6. After 30 minutes, mean pulse pressure (mm Hg) was significantly higher in the nITP group (nITP, 32.3 ± 2.2) versus the no-treatment group (21.5 ± 1.5 controls) ($p < 0.05$), and the mean hematocrit was 25.2 ± 0.8 in the nITP group versus 19 ± 0.6 in the NS group ($p < 0.001$).

CONCLUSION: In this porcine model of hemorrhagic shock, nITP therapy significantly improved SBP and pulse pressure for 30 minutes without overcompensation compared with controls with no treatment. By contrast, aggressive fluid resuscitation with NS but not nITP resulted in a significant rise in SBP to more than 100 mm Hg within minutes of initiating therapy that could cause a further reduction in hematocrit and clot dislodgment.