Inability to Easily Predict Individual Deaths in a Hemorrhage and Resuscitation Model

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Introduction
In the US, the leading cause of death in people under 44 is trauma. Many of these deaths occur from hemorrhage. Currently, patients can be placed within mortality risk groups based on severity of trauma but individual mortality cannot be accurately predicted.

Methods (continued)

Results (continued)

Predicated 70% Clinical Mortality Model

By investigating trends in individuals within a high risk group, using those same variables that are used for patient population separation (high risk of death versus low risk of death), we hoped to definitively identify eventual non-survivors from survivors early, during hemorrhage (well before cardiovascular collapse occurs).

Methods

Results

Discussion

References

Support

Surgery Education and Trauma at Iowa Methodist Medical Center, Veterans Administration Central Iowa Health Care System, Drake University, Fraternal Order of the Eagles, Iowa Space Grant Consortium.

Packed Red Blood Cells

8 anesthetized (thiopental infusion, cephalic vein), ventilated (end-tidal PCO2, 30-35 mmHg for arterial PCO2, 35-40 mmHg at baseline), instrumented, purpose bred high dose subjected to pressure titrated severe hemorrhage, low-pressure stabilization, and resuscitation protocol.

Pressure driven protocol - standardizes hemorrhages between trials to achieve low blood flows associated with a high risk of immediate cardiovascular collapse and systemic metabolic debts associated with irreversible shock.

Hemorrhage

Pressure driven protocol - standardizes hemorrhages between trials to achieve low blood flows associated with a high risk of immediate cardiovascular collapse and systemic metabolic debts associated with irreversible shock.

N1 had the highest stroke volume index, while N2 had a stroke volume index in the middle of the six survivors. End hemorrhage Svo2 percentages were indistinguishable from each other.

N1 consistently had the highest Svo2 percentage at around 80%, while N2 was intermixed with the four lowest survivors. N1 and N2 shed 38 ml/kg and 40 ml/kg of blood respectively, which coincided with two survivors, but less than the other four survivors that ranged 45-56 ml/kg of blood shed by the end of hemorrhage.

N1 had the third highest shock index at the end of hemorrhage, while N2's shock index was intermixed with 4 survivors. N1 and N2 pulse pressures were both mixed in with the 4 survivors.

We consistently observe an 20% mortality following our hemorrhage protocol regardless of resuscitation interventions. This confirms the suitability of the protocol for seeking early trend differences between eventual survivors and non-survivors.

Others have reported on many of the same variables we investigated, but in the clinical, short post-trauma setting. For example, Holcomb et al. found a blood pressure threshold value useful for indicating the need for a life saving intervention. But only 88% of trauma patients with below threshold values required a life saving intervention while 26% of non-indicated patients also needed a life saving intervention.

Looking at a large number of flow, pressure, and metabolic related variables in our known high mortality risk group, we determined that definitive trend differences during hemorrhage for indicating eventual individual non-survivors were not present.
