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JOURNAL READING

Hypothermia in multisystem trauma

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Introduction

- Exsanguinating hemorrhage
- Warm ischemic time:
  - → 5 mins for brain
  - → 20 mins for heart
- Ischemia & reperfusion injury

➢ Hypothermia might play a role

Pattern of trauma

- Leading death cause < 44y/o
- CNS injury and exsanguination
- Aggressive IV resuscitation is ineffective or even harmful before hemorrhage control
- No control of hemorrhage and reestablished perfusion→ death
- Hypotension in TBI→ still harmful

Pattern of trauma

- hemorrhagic shock and resuscitation induce ischemia-reperfusion injury (free radicals, activation of inflammation…)

➢ Modifying resuscitation fluids
➢ Pharmacologic agent
➢ Therapeutic hypothermia

Limitations

- can only be induced after the insult
- OHCA don't have ↓ ↓ blood volume
- Initiated in hemodynamic collapse and must be maintain well
- Dirty, contaminated wounds + hypothermia induced immunosupression

Spontaneous vs Induced hypothermia -- Cause

- Develops in trauma
- Ischemia leading to depletion of cellular energy→ can't maintain temperature
- environmental exposure
- Cold infusion
- Heat loss from open cavity
- Induced in a controlled fashion for therapeutic purposes
- Normal cellular energy reserves

Spontaneous vs Induced hypothermia -- Significance

- Failure of homeostatic mechanisms
- Worsen by Shivering
- Poor prognostic sign→ "lethal triad" (hypothermia, coagulopathy, acidosis)
- Lower metabolic rate
- Shivering prevented by sedatives and paralytics
- May benefit critically injured trauma patients

In CNS injury

- Spinal cord→ few studies, induced intraoperatively, epidurally or subcutaneously
- TBI→ 33 ℃ for 24hr (Marion et al, 1997), <46 y/o (Clifton et al, 2002), better neurological outcomes

In hypotension

- 30% blood volume loss (54% motality)
- by surface cooling→ prolonged survival
- Hypothermia + limited resuscitation→ improved survival (Kim et al, 1997)
- ↓ metabolic of heart with nl. function (meyer et al, 1988)
- ≧ 33℃→ ↓ arrhythmia and coagulopathy
- Hypothermia:
  - During shock and early resuscitation
  - Rewarming during late resuscitation

In pulseless

- Depend on types
- Blunt, severe, multisystem→ not feasible
- Penetrating→ repairable hemorrhage→ ED thoractomy, CPB and hypothermia (< 15℃)
  - → for body: ↓ 10℃ = ↓ 50% energy usage
  - → for brain: ↓ 10℃ = ↓ 60% energy usage
- Buying time to repair injuries and resuscitate

In pulseless

- Dog models
- Hypothermia with CPB, normothermic hemorrhagic shock, hypothermic circulatory arrest→ good

  - Tisherman et al, 1990 &1991; Capone et al, 1996
- Rapidly cooling through aortic catheter (10℃), buy time before CPB→ good in arrest for 15, 20, 30, and even 90 mins

  - Woods et al, 1999; Behringer et al, 2000&2003

Refined preclinical studies

- Multiple vascular and nonvascular injuries resulting in uncontrolled hemorrhage and shock in swine
- Open chest CPB with acellular organ preservation fluids and heat exchanger
- Rapid hypothermia (10℃) with 2℃/min
- Repair injuries
- Rewarming (0.5℃/min) & Blood transfusion

Refined preclinical studies

- ＞75% longterm survival rate and normal cognitive functions
- Maximum time for hypothermic arrest with good outcome is 60 mins
- Extending ＞120 mins resulted in significantly worse outcomes
- Sigmoid laceration→ repair & no infection
- Spleen injury→ partial splenectomy & controlled bleeding

Conclusions

- Key cellular mechanism studies
  - Pharmacologic agent + hypothermia
- Spontaneous hypothermia→ active rewarm, damage control, resuscitation
- Therapeutic hypothermia→ Buy time for hemorrhage control, rewarm, resuscitation
  - Aortic flushing or open chest CPB
  - Rapid & profound hypothermia might be beneficial for severe injured patient

Thanks for attention~!!