Therapeutic Hypothermia

- Theory
    - Many cells do not die from the actual hypoxia and hypoperfusion. Instead, this insult triggers harmful cascades that lead to cell death.

- Benefits of Therapeutic Hypothermia
    - Interrupts direct apoptosis cascade
    - Inhibits free radical production associated with re-perfusion
    - Blunts the inflammatory cascade (ice on injury mechanism)
    - Stabilizes cell membranes
    - Decreases metabolic activity

- Work-up
    - Need baseline neurological function. Use train-of-four testing if needed to r/o affects of neuromuscular blockade.
    - Asymmetrical neurological findings are not expected.
    - Cool unless the patient has very purposeful movements or follow basic commands
    - Diffuse bilateral infiltrates are typically due to aspiration or CHF. This is usually secondary to the arrest.
    - Stat ECHO with read is required.
    - Very high lactate (>15) think about abdominal pathology or compartment syndrome.
    - CAD without AMI is often the etiology. Cardiogenic shock should be treated with cath and AIBP. Increasing cardiac markers (> 5) should consider cath.

- Important aspects of cooling
    - Vent
        - DO NOT HYPERVENTILATE! Hyperventilation causes vasoconstriction. pCO2 40-45. Do not use vent to compensate for metabolic acidosis unless life-threatening. No data to support using temp corrected ABGs or not.
        - Cooling decreasing MV requirements. Thus, ABGs should be q 4-6hr to avoid hyperventilation.
        - DO NOT hyper-oxgenate! In some studies it almost doubles worse outcomes but studies are conflicting.
    - BP
        - MAP at least 65. Many centers believe MAP should target 80-100 to maximize brain perfusion.
        - CVP 8-12. UOP > 0.5ml/kg per hour

        - Can use ScvO2 target > 70.
        - 2 L fluids then levophed or epic
        - IABP and cooling not contraindicated. Some centers use cooling in patients with temporary cardiogenic shock.
        - Use dobutamine or milrinone in patient not hitting physiological endpoints
        - Only use amio if patient has continued issues with rhythms. Do not use prophylactically.
        - Hypothermia diuresis is likely.
    - Cooling
        - Higher cooling temperatures decrease bleeding risks. Coffee-grounds shouldn't prevent cooling.
        - Sedation and paralysis of "non-responsive" patients will often assist in cooling by treating shivers too fine to visualize.
        - Management of temperature post-24hr is probably just as important as the initial cooling process. Using hypothermia catheter to accomplish this is okay.

- Contraversies
    - Prehospital cooling
        - Cold IV fluids pre-hospital do not help and may hurt.
            - http://jama.jamanetwork.com/article.aspx?articleid=1778673
            - Cold fluids in truck yielded same mortality and neurological outcome.
            - Higher rates of rearrest and increased pulmonary edema.
        - However, registry databases suggests shows that "hypothermia delay" decrease chances of positive neurological recovery.
    - Difference between 32 vs 34 vs 36 C unclear
        - Studies have shown that lower temperatures decrease cerebral edema and seizures.
        - However, temp goals 33 vs 36 have not been shown to improve outcomes.
            - http://www.nejm.org/doi/full/10.1056/NEJMoa1310519
    - Blood pressure targets
        - http://www.resuscitationjournal.com/article/S0300-9572(14)00890-9/abstract?cc=y=
            - Artificially increasing MAP didn't improve outcomes.
    - Neuron-specific enolase (send out)
        - Measures degree of brain damage and may predict mortality.
    - Sedation
        - Higher levels of sedative medications during hypothermia may independently be neuroprotective.
    - 72hr post-arrest evaluations
        - Although still recommended for prognosis, the 72hr evaluation is probably less accurate after hypothermia.