Post-cardiac arrest management

December 17, 2016 by Josh Farkas

Death is the final common pathway of any severe illness. Therefore, the differential diagnosis of cardiac arrest is quite broad. Clinical context can usually narrow this down considerably, but sometimes this isn’t available. More common causes are listed here, but this list is by no means exhaustive.

more common causes of cardiac arrest

- **Cardiac**
  - Myocardial infarction causing VT/VF
  - Other causes of VT/VF (e.g. VT arising from old myocardial scar, torsade de pointes)
- Bradycardic arrest (e.g. due to heart block or medications)
- Aortic dissection
- Tamponade

**Pulmonary**
- Upper airway obstruction (choking, angioedema)
- Respiratory arrest due to severe asthma or COPD
- Tension pneumothorax
- Pulmonary embolism

**Toxicologic/metabolic**
- Overdose (e.g. opioids)
- Hypoglycemia
- Hyperkalemia

**Others**
- Septic shock
- Intracranial hemorrhage (subarachnoid or intraparenchymal)
- Hemorrhage (e.g. intraperitoneal, gastrointestinal)
- Hypothermia

The cause of cardiac arrest should be vigorously investigated if it isn’t obvious. Unfortunately, we are often distracted by binary, close-ended questions (e.g. 33C vs. 36C? cath versus no cath?). A more important question is often: what happened to this patient? Below are some tests which are commonly useful here.

**post-cardiac arrest investigations**

- **Labs**
  - Fingerstick glucose
  - Basic labs (extended electrolytes, CBC, INR, PTT)
  - Lactate
  - Troponin
  - Blood cultures & procalcitonin if concern for sepsis
  - Urine toxicology may be considered

- **EKG**
- **Bedside echocardiography:**
  - Lung: Exclude pneumothorax
  - Echocardiography (e.g. evaluate for tamponade, hypovolemia, RV failure)
  - Abdomen: evaluate for peritoneal ascites/blood
  - DVT study if PE is suspected

- **Chest X-ray**
- **CT scans**
  - Noncontrast CT head (usually obtained)
  - Chest CT angiography if PE is a concern
  - CT abdomen/pelvis if sepsis suspected and source unknown.

**targeted temperature management (TTM)**

All patients undergoing TTM should target 36C

- This remains controversial, with guidelines accepting a range of temperature targets from 33-36C.
- Available evidence shows no benefit to hypothermia (33C) compared to normothermia (36C). In the absence of evidence, targeting 36C is prudent for several reasons:
  1. TTM36 is more hemodynamically stable than TTM33, which is relevant because these are often very unstable patients.
  2. TTM36 avoids electrolytic shifts associated with raising and lowering the temperature.
  3. Hypothermia at 33C suppresses immune function and associates with increased rates of pneumonia.1
- 4) TTM33 will induce bradycardia, which is dangerous in patients with underlying torsades de pointes.\(^2,3\)
- 5) Using a single 36C strategy for all patients (rather than juggling two protocols for both TTM33 & TTM36) avoids endless debates about this topic for every patient. This promotes uniform care while allowing us to focus on more important issues (e.g. investigating and treating the cause of arrest).
- 6) TTM36 has no contraindications, so that it can be applied to every patient in need of neuroprotection.
- More reasons to use TTM36 here [https://emcrit.org/pulmcrit/top-10-reasons-to-stop-cooling-to-33c/](https://emcrit.org/pulmcrit/top-10-reasons-to-stop-cooling-to-33c/).

**who should receive TTM36 (versus no TTM)?**

[Diagram of TTM36 decision tree]

- It’s unclear precisely which patients may benefit from TTM36. In theory, this treatment should be applied to any patients with significant anoxic brain injury.
- Patients with a shockable rhythm (VT/VF) probably benefit most, but TTM36 should be applied regardless of the initial rhythm.\(^4\)
- The degree of neurologic impairment which warrants TTM36 is unknown. The algorithm above is based on defining “unresponsiveness” as absence of response to verbal commands, consistent with Canadian guidelines.\(^5,6\) The post-arrest neurologic exam is a blunt tool to predict the risk of anoxic brain injury. When in doubt, it’s better to err on the side of protecting the brain with TTM36.

**anatomy of TTM36**

[Diagram of TTM36 timeline and temperature control]

This schematic is based on the TTM-1 trial by Nielsen et al, which is currently the best validated protocol to TTM36.\(^7\) However, nobody really knows the optimal way to do this.

**pay attention to the water bath temperature**

- While the patient is on an external adaptive cooling device, the temperature of the water bath provides insight into the patient’s thermoregulation.
- If the water bath temperature is >>36C, this means that the device is warming the patient. This is often a poor prognostic sign.
If the water bath temperature is <<36°C, the patient is "trying" to spike a fever. This has a few implications:

1) This may be regarded as a "fever-equivalent." Particularly if the patient has chest infiltrates on X-ray, consider treatment for aspiration pneumonia.
2) If the external cooling device is removed, the patient is likely to subsequently spike a fever. This is a contraindication to discontinuation of external cooling pads.

**Ideal duration of external adaptive cooling?**

- Nobody knows. Beyond 36 hours the TTM trial left this to the clinician's discretion, so no definite guidelines exist.
- External adaptive cooling will improve the precision of temperature control. If the cooling pads aren't causing a problem, leaving them on longer will more effectively prevent fever.
- If the water bath temperature is very low, this is a contraindication to removing cooling pads (as discussed above).

**Anti-pyretic therapies**

- A robust antipyretic regimen is essential:
  - While on external cooling, this will help prevent shivering.
  - After external cooling is discontinued, this is the primary mode of fever prevention.
- Standard anti-pyretic regimen:
  - 1) Patients should receive acetaminophen (1 gram q6hr *scheduled*) unless contraindicated (e.g. by acute hepatic failure).
  - 2) Steroid should be used as described below.
- PRN antipyretics: It's uncommon for a patient to break through scheduled acetaminophen and steroid. Should this occur, additional antipyretics which may be considered include NSAIDs, aspirin, and clonidine.⁸⁻⁹

**Anti-shivering package**

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Treatments</th>
</tr>
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</table>
| **Step I** | - Acetaminophen 1,000 mg PO q6hr scheduled (NOT PRN)  
- Steroid (e.g. 50 mg IV q6 hydrocortisone or 50 mg prednisone daily)  
- Buspirone 30 mg per tube q8hr-q12hr scheduled  
- Magnesium replenishment to target normal levels (e.g. >2 mg/dL or >0.8 mM) |
| **Step II** | - Warming hands & feet (tricks body into feeling warm)  
- Dexmedetomidine infusion (or, if this cannot be tolerated, propofol infusion)  
- Ketamine infusion 0.2-0.3 mg/kg/hour  
- Magnesium to target a mildly supranormal level (3.4 mg/dL or 1.2-1.6 mM)  
- Ondansetron 4 mg IV q8hr |
| **Step III** | - Fentanyl boluses PRN (softer than meperidine)  
- Paralysis is the treatment of last resort |

An organized, multimodal approach to shivering is essential. Routine implementation of Step-I treatments will prevent the need for more aggressive interventions. Longer-acting medications (e.g. fentanyl infusion) and paralysis can nearly always be avoided.

• A structured and aggressive approach to shivering is essential to avoid unnecessary use of opioids and paralysis (which will delay extubation and generally promote iatrogenic harm).¹⁰⁻¹⁴

**Cardiovascular interventions**

**Coronary disease management**

- Myocardial ischemia should be strongly considered in any adult with cardiac arrest of unclear cause (especially VT/VF).
- Patients with EKG findings consistent with occlusive MI require emergent catheterization. However, some patients with non-occlusive MI may also benefit from catheterization (e.g. patients with shock or recurrent arrhythmia). If ischemia is suspected to be the cause of arrest, cardiology should be consulted. Additional therapies for MI may also be indicated (e.g. aspirin, heparin infusion).

**Anti-arrhythmic therapy?**
Antiarrhythmic medications (e.g. amiodarone) may be indicated in the following situations:

1. Recurrent ventricular tachycardia or abundant ectopy.
2. Patient with VT/VF arrest who is pending catheterization (as a bridge to revascularization).
3. For most patients, observation without antiarrhythmics is usually recommended.
4. If the patient is hypertensive, beta-blockers may be considered as an anti-arrhythmic option.

steroid

Routine administration of steroid post-cardiac arrest is recommended by the ESICM/SCCM guidelines. Evidence regarding steroid is discussed here. Methylprednisolone (e.g. 60-125 mg) may be given intra-arrest. Post-arrest, a stress dose steroid may be started (e.g. 50 mg hydrocortisone IV q6hr, or simply prednisone 40-50 mg daily).

- One retrospective study suggested that high doses of steroid (defined as >50 mg/day prednisone) is undesirable.
- Benefits of steroid may include:
  - Improved hemodynamic stability, prevention of post-arrest multi-organ failure.
  - Anti-pyretic effect helps prevent shivering and rebound fever.
- Duration of steroid therapy is unclear. After the patient has stabilized hemodynamically, my preference is to continue some steroid until ~5 days post-arrest as an antipyretic (e.g. 40 mg prednisone daily).

management of post-arrest SIRS

- Cardiac arrest of any cause may cause cytokine release and a sepsis-like clinical syndrome. Features may include:
  - Vasopressor-dependent shock
  - Transient reduction in LV ejection fraction (similar to septic cardiomyopathy; this often improves over time with supportive care).
- The treatment of this is similar to the treatment of septic shock (e.g., judicious fluid resuscitation, vasopressor support tailored to hemodynamics and bedside echocardiography).
  - If there is any possibility of infection (e.g. chest infiltrates), empiric antibiotic is reasonable while awaiting culture results.

blood pressure target?

- Anoxic brain injury may impair the brain’s ability to regulate its perfusion, causing malperfusion at lower blood pressures. For this reason, targeting an elevated MAP (e.g. >75 mm) is commonly recommended.

anticipate post-CPR mechanical complications

- CPR itself is a mechanical trauma, with numerous potential complications:
  - Pneumothorax, hemothorax
  - Splenic or liver laceration, causing hemoperitoneum
- If shock develops within a few days of CPR, perform a bedside ultrasound exam to exclude hemoperitoneum or pneumothorax.

normocapnia (pCO2 35-45 mm)

- Goals
  - Most current guidelines recommend targeting normocapnia.
  - Hypocapnia is probably the most dangerous, as this will cause cerebral vasoconstriction and reduced brain perfusion.
  - Hypercapnia causes cerebral vasodilation. This might be good (increased perfusion) or it might be harmful (increased brain edema, elevated intracranial pressure). Studies are underway currently investigating this. For now, hypercapnia should be avoided.
- Strategy
  - Immediately after intubation, adjust the minute ventilation to achieve an end-tidal CO2 of 30-35 mm. Since pCO2 is always above the end-tidal CO2, this will generally put the pCO2 into a safe range.
  - Only after the end tidal CO2 is optimized, obtain an ABG/VBG to verify that the pCO2 is within the target range. Adjust the ventilator as needed, and continue to carefully follow the end tidal CO2.
**normoxia**

- Both hypoxemia and hyperoxia seem to be harmful.
- A reasonable target may be an oxygen saturation of 92-96% or PaO2 of roughly 80-150 mm.
- The most common mistake here is leaving the ventilation set to 100% FiO2 for hours. The FiO2 will always be 100% immediately after intubation, but this should be down-titrated as rapidly as possible.
  - Remember: you don't need a blood gas to titrate the FiO2. FiO2 can be decreased immediately post-intubation, with titration based on pulse oximetry.

**neurologic issues**

**sedation**

- Either dexmedetomidine or propofol may be used for sedation. Both have the following properties:
  - 1) Don't interfere with neurologic examination (e.g. propofol may be held for exams).
  - 2) Decrease shivering.
  - 3) Will not accumulate or delay extubation.
- Some patients will have difficulty tolerating these agents due to hypotension. This can generally be managed by co-administration of a vasopressor or inotrope to balance out hemodynamic effects of the propofol or dexmedetomidine.
  - Propofol causes vasodilation, which may be counter-balanced with a phenylephrine or norepinephrine infusion.
  - Dexmedetomidine causes bradycardia, which may be counter-balanced with a low-dose epinephrine or dobutamine infusion.
- The best strategy here might be a combination of dexmedetomidine and pain-dose ketamine infusions (KetaDex):
  - Ketamine and dexmedetomidine have synergistic analgesic and anti-shivering effects.
  - Both ketamine and dexmedetomidine tend to prevent delirium and facilitate extubation.
- **Longer acting drugs should be avoided (even fentanyl),** as this may impair neuroprognostication.

**noncontrast head CT**

- Cardiac arrest may occasionally reflect a neurologic catastrophe (e.g. subarachnoid hemorrhage).\(^{17}\)
- Noncontrast head CT should be considered for patients with arrest of unclear cause. It can also occasionally provide some prognostic information (any visible edema on CT is a fairly poor sign).
- This is generally not mission-critical, so it shouldn't delay other treatments.

**EEG monitoring**

- Video EEG monitoring plays two important roles: neuroprognostication and detection of seizure.
- Unless the patient is following commands, video EEG monitoring should be initiated (if available).

**early neuroprognostication**

- Early neuroprognostication is generally impossible, with two exceptions:
  - (1) Brain death: If cranial nerves and respiratory drive are absent, this should prompt evaluation for brain death. If brain death criteria are met then any further therapy is futile.
    - Note that brain death diagnosis requires normothermia and exclusion of lingering effects from intoxication.
  - (2) Early-onset myoclonic status epilepticus:
    - Clinical criteria:
      - Occurs soon after cardiac arrest (typically within 24 hours).
      - Lasts >30 minutes
      - Spontaneous, repetitive, unequivocal, ongoing myoclonic jerking involving face, limbs, and trunk.
      - Patient is unresponsive.
      - EEG shows burst-suppression pattern.\(^{18}\)
    - Significance: true early-onset myoclonic status epilepticus is generally inconsistent with a good neurologic outcome. These patients will often be observed for some days to confirm lack of neurologic improvement, but the likelihood of a poor outcome should be shared with the family up-front.
aspiration pneumonia & antibiotic prophylaxis

why aspiration pneumonia is potentially problematic

- Many patients aspirate during cardiac arrest.
- Patients are intubated and mechanically ventilated, usually with poor mental status — conditions which do not promote secretion clearance.
- Early diagnosis of pneumonia is impossible for many reasons (inability to measure a fever due to therapeutic temperature monitoring, inability to report symptoms due to intubated/sedated status, masking of mild hypoxemia because patients are on mechanical ventilation already).

evidentiary support for prophylactic antibiotics

- ANTHARTIC trial
  - Multi-center RCT involving 194 patients randomized to placebo versus 2 days therapy with amoxicillin-clavulanate.
  - The primary endpoint (ventilator-associated pneumonia) was successfully reduced by this intervention (from 34% to 19%, p=0.03).
  - However, the study was underpowered to prove that this translated into improvements in mortality or ventilator-free days.
  - Generalizability may be limited, as the trial was restricted to patients with out of hospital shockable arrest.
  - Further discussion of the trial here (https://emcrit.org/pulmcrit/anthartic/).

bottom line?

- Use of prophylactic antibiotics following cardiac arrest remains controversial. Overall, it appears safe and is supported by available evidence.
- If prophylactic antibiotics are used:
  - (a) Reasonably narrow agents should be used (e.g. ideally ampicillin/sulbactam, or possibly ceftriaxone monotherapy).
  - (b) The course should be limited to 48 hours.
- If prophylactic antibiotics aren't used, then there should be a low threshold to initiate antibiotics if the patient shows any signs of pneumonia.

post-cardiac arrest investigations

- Labs
  - Fingerstick glucose
  - Basic labs (extended electrolytes, CBC, INR, PTT)
  - Lactate
  - Troponin
  - Blood cultures & procalcitonin if concern for sepsis
  - Urine toxicology may be considered
- EKG
- Bedside echocardiography:
  - Lung: Exclude pneumothorax
  - Echocardiography (e.g. evaluate for tamponade, hypovolemia, RV failure)
  - Abdomen: evaluate for peritoneal ascites/blood
  - DVT study if PE is suspected
- Chest X-ray
- CT scans
  - Noncontrasted CT head (usually obtained)
  - Chest CT angiography if PE is a concern
  - CT abdomen/pelvis if sepsis suspected and source unknown.

algorithm

https://emcrit.org/ibcc/post-arrest/#aspiration_pneumonia_&_antibiotic_prophylaxis
post-cardiac arrest checklist

- Evaluation & Treatment of any inciting event (see list above)
- Cardiac
  - Review EKG & bedside echo.
  - Consult cardiology if arrest may be due to MI or primary arrhythmia (no consult needed if obvious non-cardiac cause).
  - Consider aspirin, P2Y12-inhibitor, and heparin if highly suspicious for MI.
  - MAP goal > 75 mm
  - Steroid per SCCM/ESICM guidelines (e.g., 50 mg hydrocortisone IV q6hr).
- Pulmonary
  - Target normoxia (oxygen saturation 92-96% -or- pO2 60-150 mm).
  - Target normocarbia (adjust ventilator to achieve end tidal CO2 ~20-35 mm, then check ABG/VBG. Target pCO2 35-45 mm).
  - Consider 2-day antibiotic course to prevent pneumonia (ideally ampicillin/subactam; alternative = ceftriaxone)
- Renal
  - K >4 mEq/L, Mg >2 mg/dL
- Neurologic
  - TTM @36C & video EEG, unless patient is awake and neurologically intact.
  - Sedate with propofol or dexmedetomidine (avoid longer-acting drugs that obscure neuro exam).
  - Add ketamine 0.1-0.3 mg/kg/hr for pain or shivering. Avoid fentanyl.
  - Acetaminophen 1,000 mg PO q6hr scheduled
  - Buspirone 30mg PO q8hr
  - Treat shivering in organized, multimodal fashion (table below).
- Other
  - DVT prophylaxis
  - GI prophylaxis (pantoprazole 40 mg PO/IV)
  - Enteral nutrition if no procedure planned.

multimodal management of shivering during TTM

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- Steroid (e.g., 50 mg IV q6 hydrocortisone or 50 mg prednisone daily)  
- Buspirone 30 mg per tube q8hr-q12hr scheduled  
- Magnesium repletion to target normal level (e.g. >2 mg/dL or >0.8 mEq/L) |
| Step II  | - Warming hands & feet (tricks body into feeling warm)  
- Dexmedetomidine infusion (or, if this cannot be tolerated, propofol infusion)  
- Ketamine infusion 0.2-0.3 mg/kg/hour  
- Magnesium to target a mildly supranormal level (3-4 mg/dL or 1.2-1.6 mEq/L)  
- Ondansetron 4 mg IV q8hr |
| Step III | - Fentanyl bolus PRN (safer than meperidine)  
- Paralysis is the treatment of last resort. |

An organized, multimodal approach to shivering is essential. Routine implementation of Step I treatments will prevent the need for more aggressive interventions. Longer-acting medications (e.g. fentanyl infusion) and paralysis can rarely always be avoided.

The Podcast Episode

https://emcrit.org/ibcc/post-arrest/#aspiration_pneumonia__antibiotic_prophylaxis
Excessive focus on temperature manipulation, while ignoring other essential components of management (e.g., investigation and treatment of the cause of the arrest).

Packing patients in ice prior to transfer to a referral center may cause dangerous, uncontrolled hypothermia.

Be cautious about early extubation of a patient who is still undergoing TTM – this can make it difficult to achieve control of shivering (use of sedatives is limited in a non-intubated patient).

Don’t prognosticate future cardiac function based on post-arrest ejection fraction, which often improves over time.

Avoid fentanyl infusions or benzodiazepines if possible. These may delay awakening, interfere with neuroprognostication, and prolong ventilation time.

Going further:

- Top 10 reasons to stop cooling to 33C
- Pragmatic comparison 33°C vs. 36°C
- Targeted temperature trial changes everything
- Post-cardiac arrest care in 2013 with Stephen Bernard: Parts I and II
- Post-ROSC checklist by ALiEM.
- Post-cardiac arrest care
- RINSE trial of pre-hospital cooling (Ken Milne, SGEM)


18. Neuromuscular paralysis (e.g. 10 mg vecuronium) will be needed to obtain an accurate EEG, without muscle artifact.