Death is the final common pathway of any severe illness. Therefore, the differential diagnosis of cardiac arrest is quite broad. Clinical context can often narrow this down, but sometimes history isn't available. Some of the more common causes of cardiac arrest are listed below, but this list is by no means exhaustive.

more common causes of cardiac arrest

- **Cardiac**
  - Myocardial infarction causing VT/VF.
  - Primary ventricular arrhythmia (e.g., VT arising from old myocardial scar, torsade de pointes).
  - Bradycardic arrest (e.g., due to heart block or medications).
  - Aortic dissection.
- Tamponade.
- Acute cor pulmonale due to massive pulmonary embolism.

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

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

**Others**
- Septic shock.
- Anaphylactic shock.
- Intracranial hemorrhage (e.g., 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. 37.8C? Cath versus no cath? PE or no PE?). A more important question is often: what happened to this patient?
A recent study highlights the yield of a contrasted pan-CT scan (head through pelvis) among cardiac arrest patients without an obvious etiology. This should be strongly considered, especially among patients being transported to the scanner for a head CT scan.

### Post-Arrest Investigations

#### Labs
- Fingerstick glucose.
- Basic labs (extended electrolytes, CBC, INR, PTT).
- Liver function tests.
- Lactate.
- Troponin.
- Blood cultures & procalcitonin if concern for sepsis.
- Pregnancy test PRN.
- ABG (after intubation & optimization of end tidal CO2)

#### EKG
- If initial EKG is abnormal, repeat it. Serial post-resuscitation EKGs can help sort out transient versus persistent ischemia.

#### POCUS
- Lungs: Exclude pneumothorax.
- Echocardiography (e.g., evaluate for tamponade, hypovolemia, RV failure).
- Abdomen: evaluate for peritoneal ascites/blood.
- DVT study if PE is suspected.

#### Imaging
- Chest X-ray.
- Noncontrast head CT.
- If the cause of arrest is not obvious, obtain CT head/chest/abdomen/pelvis to look for PE or occult foci of bleeding/sepsis.

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**targeted temperature management (TTM)**

### Background on Therapeutic Temperature Management

- The optimal temperature management for post-arrest patients has been controversial over the past 20 years. Hypothermia became widely recommended on the basis of two very small studies. (11856793, 11856794) Subsequently, larger and more robust studies disproved the benefit of hypothermia. (24237006, 31473324) This is further explored here.
- Best available evidence suggests that avoidance of fever is beneficial. An aggressive protocol for fever avoidance as employed in the TTM2 trial seems to be the optimal approach (TTM37.8).
- Hypothermia (33°C) should be restricted to the context of RCTs, for the following reasons:
  - Robust, modern RCTs do not show any benefit from 33°C (in terms of either mortality or neurological outcomes). (24237006, 31473324)
  - 33°C may increase hemodynamic instability (including bradycardia and cardiac arrhythmias causing hypotension). Bradycardia may be particularly dangerous among patients with underlying torsade de pointes. (17015798, 19845813)
  - 33°C may increase the utilization of paralytics, delay sedative metabolism, and prolong the duration of mechanical ventilation.
  - 33°C may delay awakening, thereby impairing the ability to provide accurate neuroprognostication.
  - Using a single TTM37.8 strategy for all patients (rather than juggling two protocols for both TTM33 & TTM37.8) 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).
- It remains unknown how to optimally interpret blood gas values among hypothermic patients (i.e., how to correct for hypothermia). (33765189)
who should receive TTM37.8 (versus no TTM)?

- It’s unclear precisely which patients may benefit from TTM37.8. Avoiding fever is likely beneficial for any patients with significant anoxic brain injury.
- Currently it is reasonable to provide TTM37.8 to any patient who is unresponsive following cardiac arrest (as defined by unresponsiveness to verbal commands). 

overview of how to perform TTM37.8

Getting started:
- (1) Start scheduled antipyretics (typically acetaminophen 1 gram q6hr, or lower doses in patients with malnutrition or cirrhosis).
- (2) Patients should receive an invasive, continuous temperature probe (e.g., bladder or esophageal temperature probe).
- Follow the temperature closely. If the temperature increases >37.8, then initiate adaptive external cooling (e.g., external cooling pads with circulating water titrated against the patient’s temperature).
- The target temperature should be set to 37.5°C.
- If shivering occurs, this should be managed in an organized and multimodal fashion (more on this below).

For patients who require adaptive external cooling, it’s unclear exactly how long to continue adaptive external cooling:
- Even after 40 hours, temperature could be detrimental to the injured brain. ERC/ESICM guidelines recommend avoiding a fever for at least 72 hours among patients who remain in coma. 
- In the TTM2 trial, between 40-72 hours the strategy of achieving normothermia was left up to clinician discretion. This leaves it unclear precisely when the temperature pads should be removed.
- 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 (more on this below).
for patients on adaptive external cooling, 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 >>37.5°C, this means that the device is warming the patient. This is often a poor prognostic sign.
- If the water bath temperature is <<37.5°C, the patient is “trying” to spike a fever. This has a few implications:
  - 1) This may be regarded as a “fever-equivalent.” For example, 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 removing the external cooling pads.

antipyretic therapies

- Scheduled antipyretics are extremely useful:
  - This may avoid the need for external cooling entirely.
  - If the patient does require external cooling, antipyretics will help reduce shivering. Continuation of antipyretics after physical cooling is discontinued may also help prevent rebound fever.
- Potential antipyretic agents:
  - Patients should receive acetaminophen (1 gram q6hr scheduled) unless contraindicated (e.g. by acute hepatic failure).
  - Steroid may be used as described below (although this remains controversial).
  - Additional agents which could be considered include NSAIDs, aspirin, and clonidine.(30393754, 27264198)

anti-shivering strategy

- 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).(27138855, 29278601, 24049601, 20653360, 21210305)
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).

antiarrhythmic 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).
- For most patients, observation without antiarrhythmics is usually recommended.
- If the patient is hypertensive, beta-blockers may be considered as an antiarrhythmic option.

management of post-arrest SIRS & steroid administration

- Cardiac arrest of any cause may cause cytokine release and a sepsis-like clinical syndrome. Features may include vasopressor dependent shock and transient reduction in systolic heart function (similar to septic cardiomyopathy; this often improves over time with supportive care). Management 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. More on the use of antibiotics below.
- Steroid administration is controversial. For example, routine administration of steroid post-cardiac arrest is recommended by the ESIICM/SCCM guidelines, but not the ERC/ESICM guidelines. Benefits of steroid may include:
  - Improved hemodynamic stability, prevention of post-arrest multi-organ failure.
  - Antipyretic effect helps prevent shivering and rebound fever.
- Methylprednisolone (e.g., 60-125 mg) may be given intra-arrest. Post-arrest, a stress dose steroid may be considered (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. (30308548)

blood pressure target?

- Anoxic brain injury may cause elevated intracranial pressure as well as impaired autoregulation, leading to malperfusion at lower blood pressures. For this reason, targeting an elevated MAP (e.g. >75 mm) may be reasonable.

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.

pulmonary optimization

normocapnia (pCO2 35-45 mm)

- Goals
  - Most current guidelines recommend targeting normocapnia (35-45 mm Hg or 4.5-6 kPa).
  - 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 (35-45 mm Hg or 4.5-6 kPa). 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 94-98% or PaO2 of roughly 75-100 mmHg. (33765189)
- 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 counterbalanced with a phenylephrine or norepinephrine infusion.
  - Dexmedetomidine causes bradycardia, which may be counterbalanced 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). (25304078)
- 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. (Neuromuscular paralysis, for example 10 mg vecuronium, will be needed to obtain an accurate EEG without muscle artifact).
    - 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.
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

- One very small, single-center study found benefit from a three-day prophylactic course of ampicillin-sulbactam. (15754197)
- ANTHARTIC trial (31693806)
  - 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.

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.

algorithm

- Labs
  - Fingerstick glucose.
  - Basic labs (extended electrolytes, CBC, INR, PTT).
  - Liver function tests.
  - Lactate.
  - Troponin.
  - Blood cultures & procalcitonin if concern for sepsis.
  - Pregnancy test PRN.
  - ABG (after intubation & optimization of end tidal CO2)

- EKG
  - If initial EKG is abnormal, repeat it. Serial post-resuscitation EKGs can help sort out transient versus persistent ischemia.

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

- Imaging
  - Chest X-ray.
  - Noncontrast head CT.
  - If the cause of arrest is not obvious, obtain CT

- The Internal Bask of Critical Care.
Post-Arrest Management

Evaluation & Treatment of any inciting event (see box 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 often >75 mm.
- May consider stress steroid (e.g., 50 mg hydrocortisone IV q6hr).

Pulmonary
- Target normoxia (oxygen saturation 94-98%, PaO2 75-100 mm).
- Target normocapnia (adjust ventilator to achieve end tidal CO2 ~30-35 mm, then check ABG/VBG. Target pCO2 35-45 mm).
- Consider 2-day antibiotic course to prevent pneumonia if lung infiltrates on CXR (usually ampicillin/sulbactam or ceftriaxone).

Renal
- K >3.5 mEq, 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 neurological examination).
- 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 an organized, multimodal fashion (see table).

Other
- DVT prophylaxis.
- GI prophylaxis (pantoprazole 40 mg PO/Iv).
- Enteral nutrition if no procedure planned.

TTM37.8 - Multimodal control of temperature & shivering

Post-arrest patient unable to follow verbal commands

Basic temperature package
- Insert continuous temperature monitor (bladder or esophageal).
- Scheduled acetaminophen (usually 1 gram q6hr; if alcoholic, cirrhotic, or <50kg then 650 mg q8).

if T<37.8
Continue close monitoring for fever (T>37.8).

if T>37.8
Advanced temperature package & Tier-1 anti-shiver
- External adaptive cooling device (e.g., Arctic Sun). Set this to maintain a target temperature of 37.5.
- Buspirone 30 mg per tube q8hr-q12hr scheduled.
- Magnesium replenition to target a normal level (e.g. >2 mg/dL or >0.8 mM).

Tier-2 anti-shivering therapies
- Warming hands & feet (tricks body into feeling warm).
- Dexmedetomidine and/or propofol infusions.
- Ketamine infusion 0.2-0.3 mg/kg/hour (low, analgesic dose).
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 (PulmCrit)
- Pragmatic comparison 33C vs. 36C (PulmCrit)
- Targeted temperature trial changes everything (podcast, EMCrit)
- Prophylactic antibiotics after cardiac arrest? (PulmCrit)
- What happened to VSE (Vasopressin/Steroid/Epinephrine)? (PulmCrit)
- Post-cardiac arrest care in 2013 with Stephen Bernard: Parts I and II (podcast, EMCrit)
- Post-ROSC checklist by ALiEM.
- Post-cardiac arrest care (Chris Nickson, LITFL)
- RINSE trial of pre-hospital cooling (Ken Milne, SGEM)

**references**


