

Treating Lazarus: Post-ROSC Care in 2015

Charles Bruen, MD

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No disclosures
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Persistent Precipitating Disease

- Identify reversible causes of arrest
- Tamponade, PE, PTX
- Distributive shock (septic, anaphylactic)
- Myocardial infarction
- Metabolic dearrangement

Post-Arrest Syndrome

- Systemic ischemic/reperfusion response
- Persistent physiologic derangements
- Sepsis-like inflammatory response and multiorgan dysfunction
- Separate from primary ischemic injury & dysfunction

Post-Arrest Syndrome

- Myocardial dysfunction / Stunning
 - Ischemia/Shocks
 - Systole/diastole
- Brain injury
- Coagulopathy
- Microvascular thrombosis
- Adrenal dysfunction

Hemodynamics

- MAPs >70mmHg had improved outcomes
- But...
 - If vasopressors used to achieve goal, then outcomes 2x worse
- And...
 - Use vasopressors to get to MAP, better than not getting to MAP goal at all

Hemodynamics

- More cardiac dysfunction seen at hypothermia temperatures
- Must assess reason for shock, and have that guide therapy
 - Vasodilatory sepsis
 - Hypovolemia
 - Cardiogenic

Oxygen

- Clearing ongoing hypoxia has negative impact post-ROSC
- Hyperoxia though is likely also detrimental
 - Increased metabolic use
 - Generation of ROS

Oxygen

During CPR

- NIRS measures rSO₂ (normally 60-80%)
 - Corresponds to cerebral SvO₂
 - During CPR, rSO₂ was high in survivors
 - <30% universal fatal
- High FiO₂ does not appear to expose brain tissue to hyperemic injury (little data)

Oxygen

Prehospital

- High FiO₂ was associated with histological evidence of brain injury
- Unclear if it is even possible to deliver titrated oxygen prehospital that avoids hyper/hypoxia
- Even if we could, would it affect outcomes

Oxygen

Inhospital

- Restrospective data supports avoiding hyperoxia
- Animal data very clear
- Increased PaO₂ may be marker of illness severity after ROSC

Oxygen

- Confound of measuring PaO₂ because of shock
 - Peripheral SpO₂
 - Peripheral PaO₂
- Slow introduction of oxygen back to system
- Aim for PaO₂ 100-150 mmHg
 - 5mmHg/deg below 37 on alpha-stat

Carbon Dioxide

- EtCO₂ valuable to predict ROSC
- Cerebral autoregulation dysfunction
- Hypercapnia (hypovent) increases CBF/
increases ICP
- Hypocapnia (hypervent) vasoconstricts
and decreases CBF and oxygen delivery

Carbon dioxide

- ICP Management
- Maintain Fowler position to increase venous drainage
- Avoid fever, agitation, seizures, high metabolic demand

Carbon dioxide

- Hypo is probably worse than hyper
- Goal 40-45 mmHg
- Alpha-stat or pH-stat

Ventilator Management

- Lungs may be damaged
 - Trauma (contusion/laceration), aspiration blood/vomit, PNA, pulmonary edema, PTX
- Post-arrest VILI
 - LTV, Prone positioning, lung rest with ELSO
- High VT compromises CBF
- 6 ml/kg or less

Glycemic Control

- Most post-arrest pts hyperglycemic
- Post-arrest
 - Want to avoid secondary injury
 - Hypermetabolic state
 - Hypoglycemia rapidly leads to harm

Glycemic Control

- Tight glucose control increases incidence of hypoglycemia
 - Even more incidence on TH
- Target 140-180 mg/dl

Seizures

- 36% of post-arrest puts have seizure activity
- 2-3% have status
- 0.5-1% have NCSE
- Reflects extent of injury, but also increased metabolic demand 2-3x

Seizures

- Brain Resuscitation Clinical Trial 1
 - No benefit for empiric prophylactic anticonvulsives
- Intermittent vs continuous EEG

Seizures

- If seizures/SE/NCSE develops, treat aggressively
- Good outcomes have been reported

Revascularization

- ACS certainly a major causes of SCD
- ACS a definitive reason for revascularization
- Up to 50% pts with OHCA had culprit lesion on angiography
 - True for STEMI
 - Also, NSTEMI -- 40% had lesion

Revascularization

- Diagnostic Benefit
- Does intervention help?
 - Spaulding (Survival OR 5.2)
 - Dumas (Survival OR 2.1)
- Signals that benefits occur for QoL
 - Cognition, ADLs, social interaction

Revascularization

- Who to take?
- Often dont have symptoms or incomplete history
- Obvious non cardiac etiology
 - Exclude clear sepsis, trauma, metabolic disorder, respiratory failure, hemorrhage
- Definitely ruling-out ACS is difficult

Revascularization

- ECG not sens/spec immediately post-ROSC
- Features
 - Conventional STEMI criteria --STE, Sgarbossa, LBBB
 - Persistent ischemic features
 - Ventricular dysrhythmias
 - Cardiogenic shock
 - Substantial cTn
 - RWMA

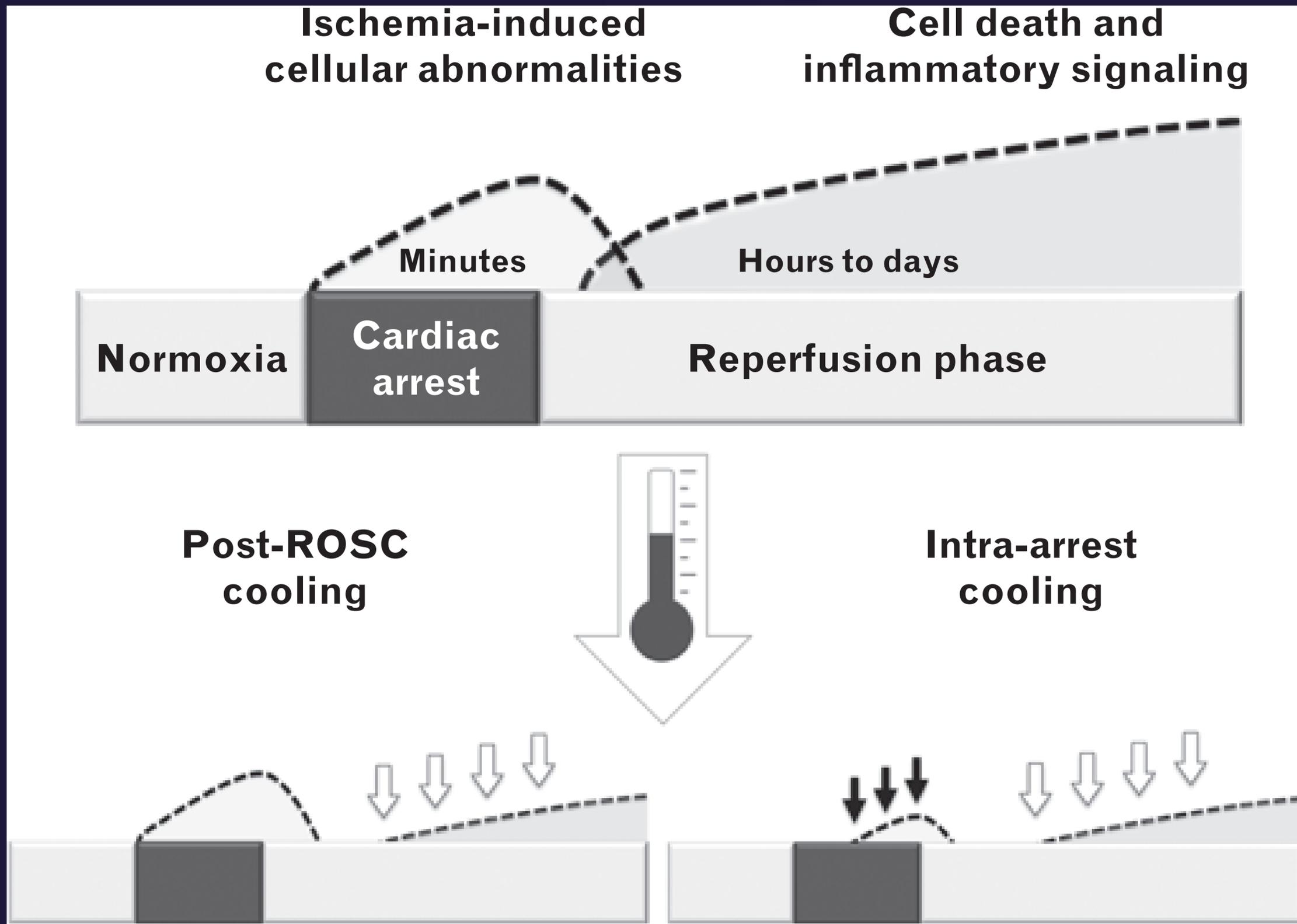
Temperature

- Two landmark trials
 - 2002. Same issue of NEJM
 - Bernard. HACA.
- Shockable rhythms
- Widespread adoption
 - Use expanded beyond original scope

Temperature

Timing

- Prehospital cooling. Seattle.
- 1364 pts. 2000 mL iced saline.
- 1 deg separation at admission
- Higher rearrests and pulmonary edema
- Possibly better for longer transports, but not proven



TTIM Trial

- 36deg
 - Median temp at arrival
 - Control devices wont stray into fever range
- Pragmatic trial — reflect current practice

TTM Results

- Neutral no benefit of either temperature in any outcome including adverse events
- Cannot say that cooling is unnecessary
- Does not add to literature of benefit of cooling

TTM Criticism

- Precision of temperature control
- Time to reach target temperature
 - 4 hrs for randomization
 - 4 hrs to get to temperature
- Less ill cohort

TTM Criticism

- Midazolam and propofol sedatives are neuroprotective and doses wernt recorded
- High rate of rewarming
- Large portion of bystander CPR (70%)
- Sedation and shivering not protocolized

TTM Criticism

- Short BLS time <1min
- Higher deaths in 33group before prognositication (33 v 36)
- Poor cardac function and multiorgan failure
- Too generalizable
- Miss subgroups that may benefit

Prognostication

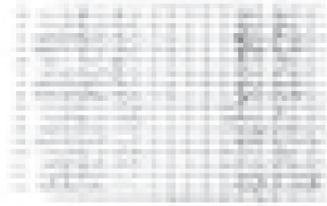
- AAN 2006
- No brain stem reflexes at anytime
- Day 1 — Myoclonus, Status
- Day 3 — Absent SSEP, NSE >33,
Absent motor reflexes

Prognostication

- Was before hypothermia era
- Case reports have challenged these criteria
 - First motor, then NSE, SSEP
 - Even Myoclonic Status Epilepticus



EEG



SSEPs

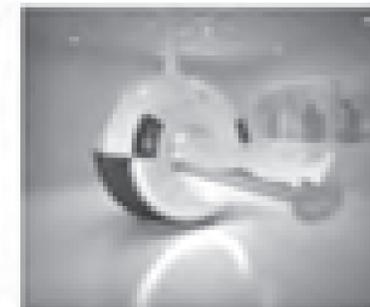
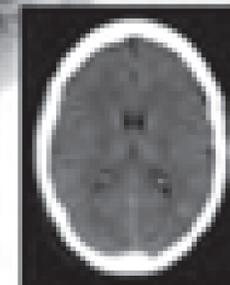


Biomarkers

Neurological examination



Imaging



Clinical Exam

- Pupils false prediction in 1/3 cases
- More reliable at 72 hours (but not perfect)
- TTMT waited until 118 hr
- Pupilometer

Clinical Exam

- Corneal reflexes less reliable than pupils (FPR 5%)
- Motor exam sens/ not spec (FPR (10-40%))
- Both affected by sedatives/paralytics

Clinical Exam

- Myoclonus
- Sign of CNS injury
- With/without epileptiform activity
- Generalized myoclonus (face, trunk, limbs) >30min is MSE
- Can be confused with Lance-Adams Syndrome
- 9% with myoclonus s/p TH had good outcomes

Electrophysiology

- Short-latency Somatosensory evoked potentials (SSEP)
 - Less confounding by sedatives
- In non-TH, accurate predictor of outcome at 24 hours.
- In TH after rewarming, predictor but not as good
- Self-fulfilling prophecy

Electrophysiology

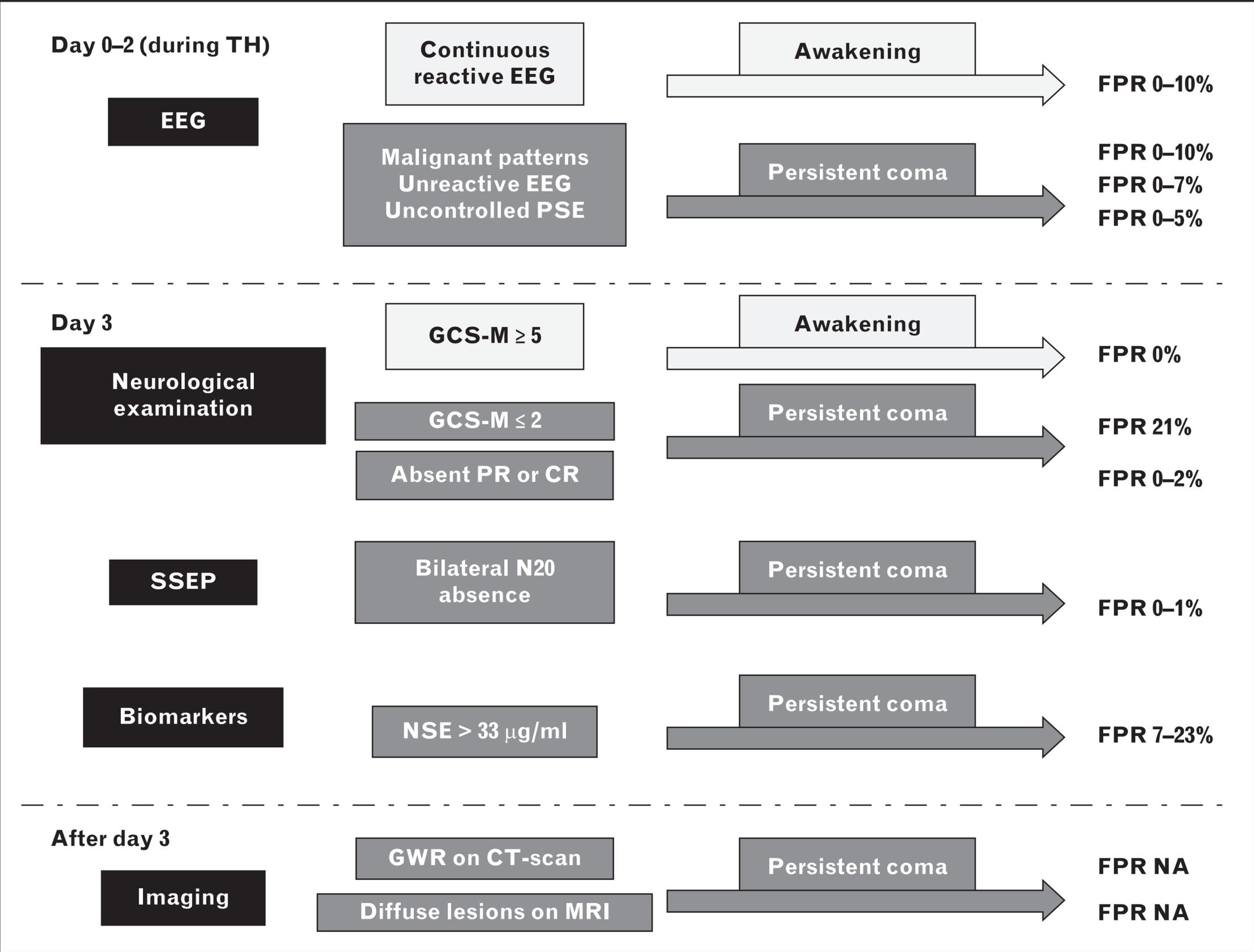
- EEG
- Malignant patterns — burst suppression, low voltage
- Inconsistent definition and interpretation

Biomarkers

- Neuron-specific enolase (Neurons)
- S-100 protein (Schwann cells)
- Markers of cell death, but not function

Neuroimaging

- Only gives structural not functional information
- HCT
 - Diffuse brain edema, Ratio Grey/White matter
- MRI
 - DWI — neuronal cytotoxic edema
 - Especially in basal ganglia and posterior cortical



What To Do

- Nothing immediate
- Almost nothing early
- Wait, then wait, and then wait some more
- Multimodal

