Stayin' Alive: Updates in ACLS and Post-Cardiac Arrest Management

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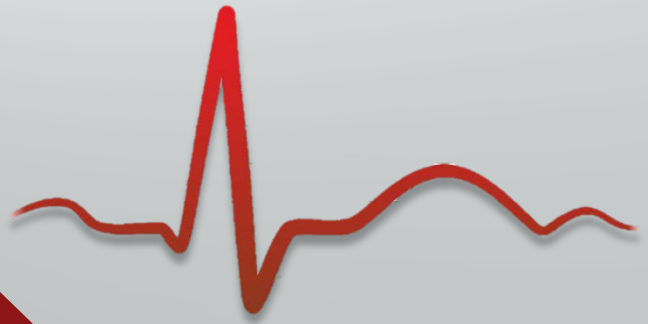


Disclosure

I have nothing to disclose concerning possible financial or personal relationships with commercial entities (or their competitors) that may be referenced in this presentation.

Objectives

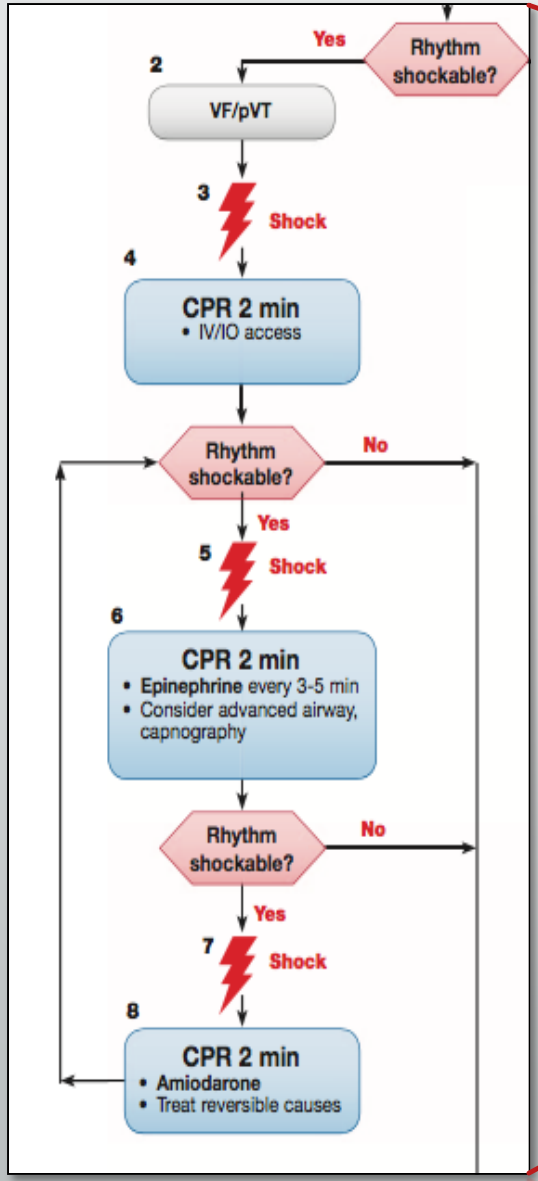
- Review fundamentals in management of adult cardiac arrest, focusing on changes in the 2015 ACLS guidelines
- Discuss evidence utilizing vasopressin during cardiac arrest leading to its removal from the ACLS algorithm
- Evaluate alteplase administration during cardiac arrest caused by suspected or confirmed pulmonary embolism
- Assess benefits and risks associated with sodium bicarbonate use during cardiac arrest given the recent drug shortage
- Discuss changes to targeted temperature management post-cardiac arrest and pharmacologic considerations during cooling

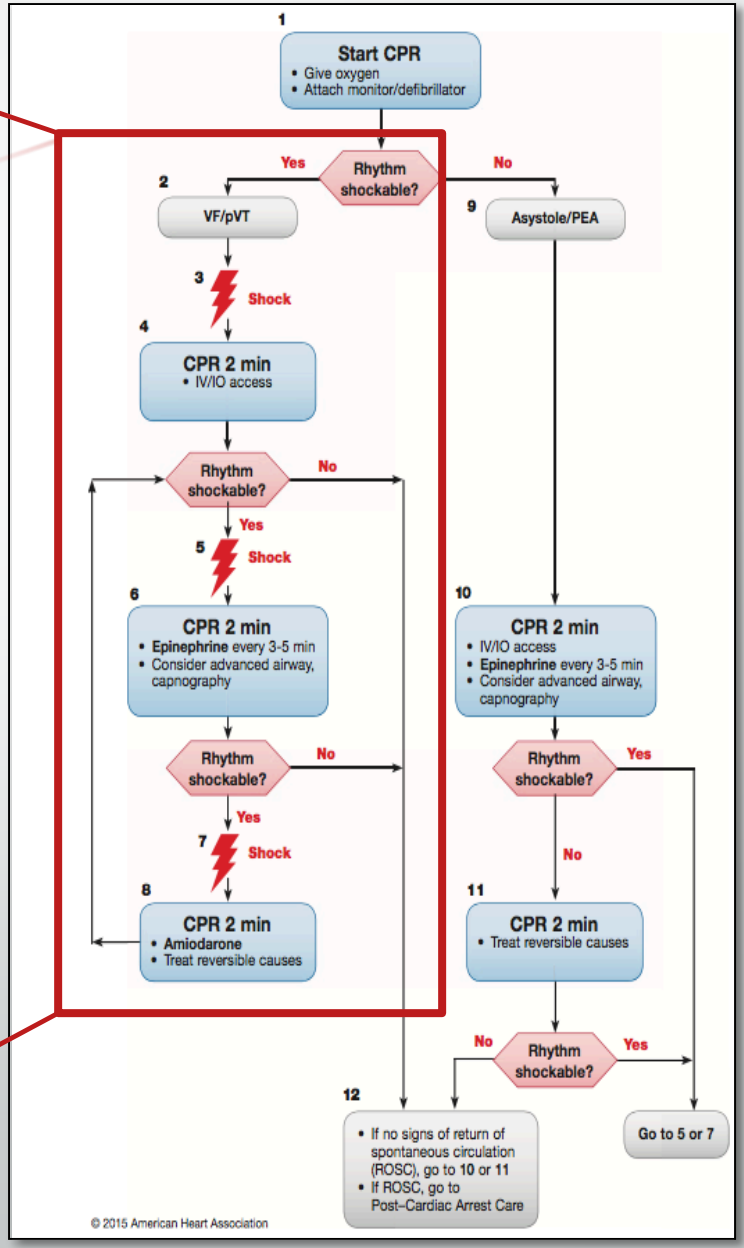
Cardiac Arrest

- *Sudden cessation of cardiac activity where the victim becomes unresponsive, with no normal breathing and no signs of circulation*
- 2016 statistics
  - Out-of-hospital cardiac arrest (OHCA)
    - Incidence: 350,000
    - Survival to discharge: 12%
  - In-hospital cardiac arrest (IHCA)
    - Incidence: 209,000
    - Survival to discharge: 24.8%

Link MS, et al. *Circulation.* 2015;132:S444-464.
http://www.cpr.heart.org/AHAECC/CPRAndECC/General/UCM_477263_Cardiac-Arrest-Statistics.jsp\

Adult Cardiac Arrest Algorithm





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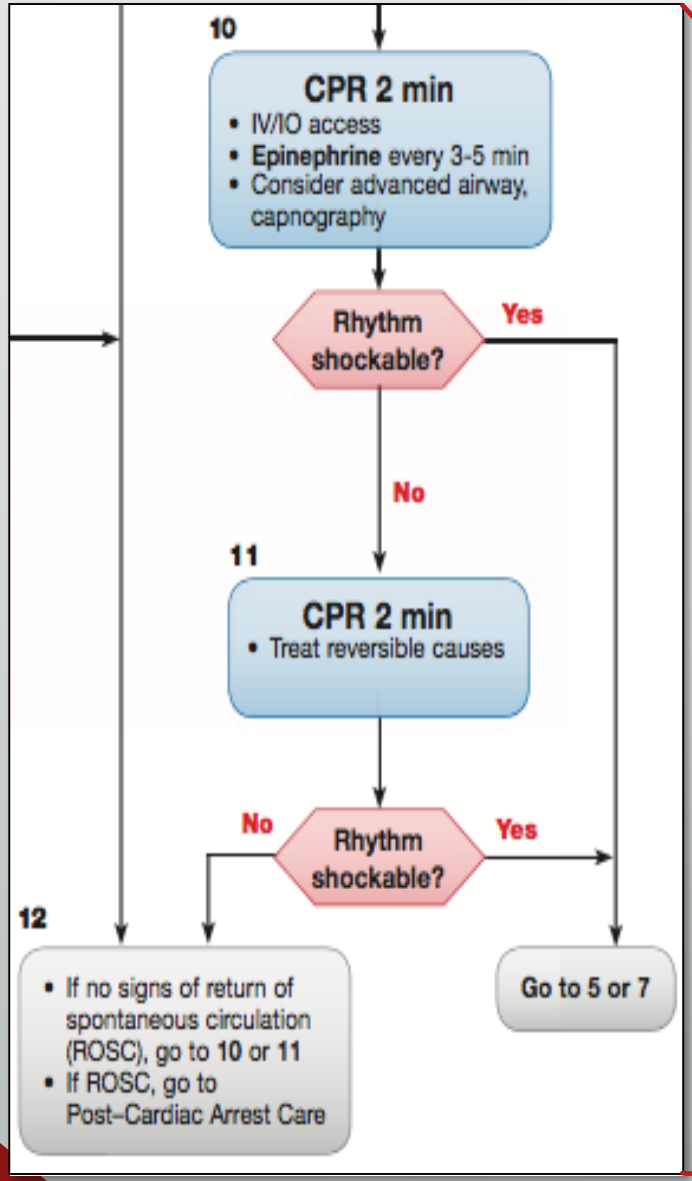
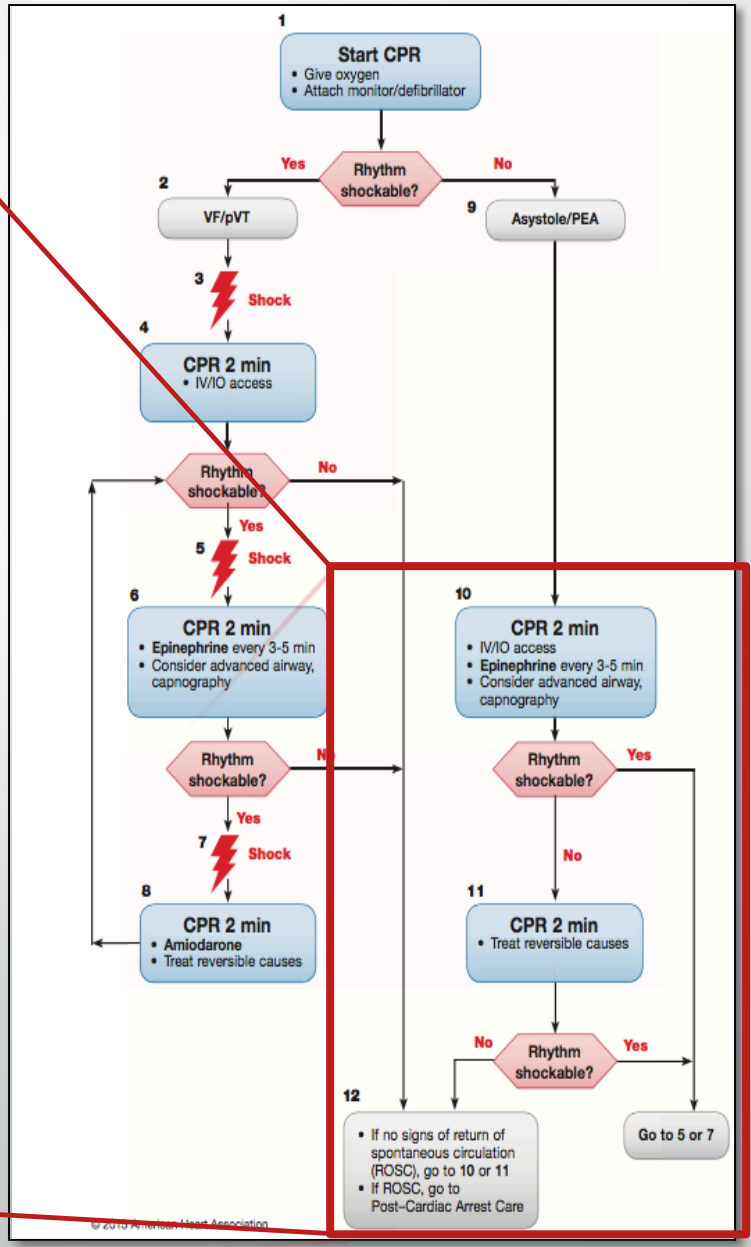
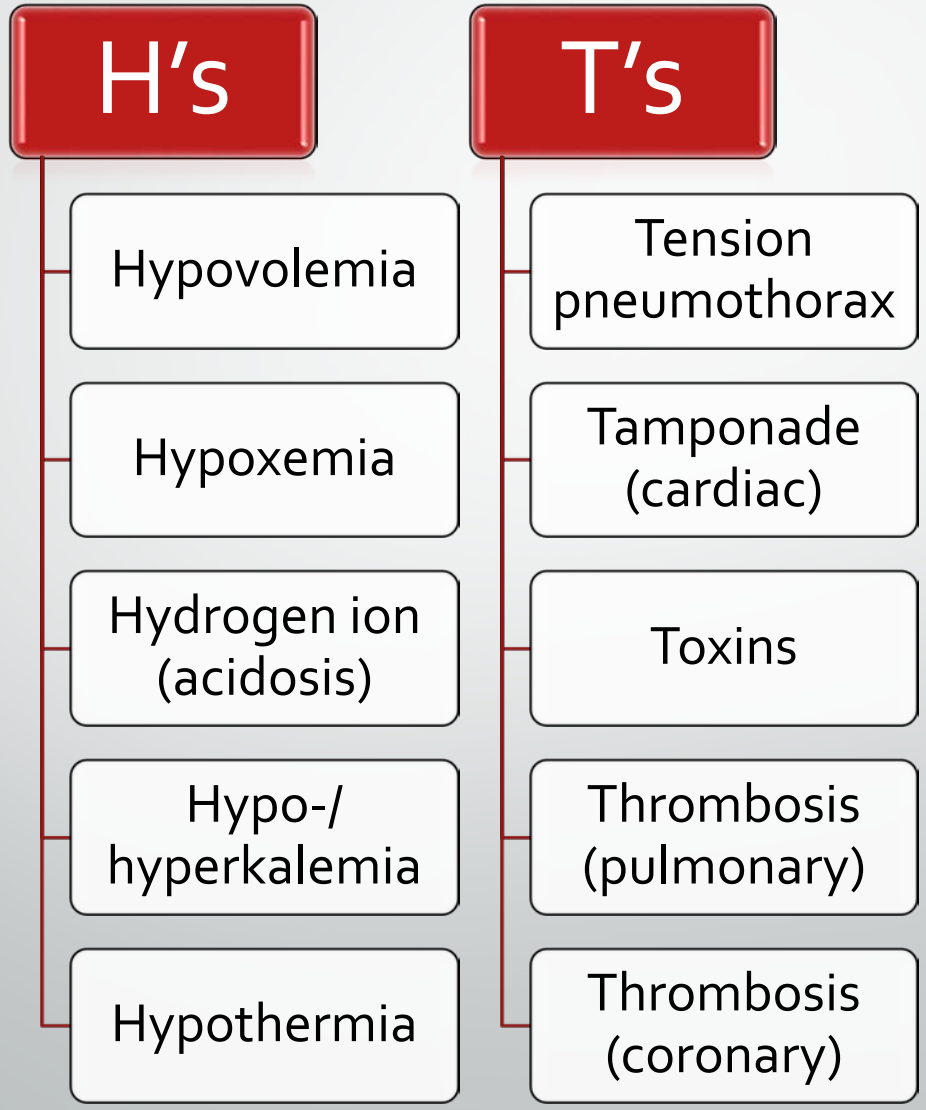
Adult Cardiac Arrest Algorithm





Link MS, et al. *Circulation.* 2015;132:S444-464.

Treat Reversible Causes



Link MS, et al. *Circulation.* 2015;132:S444-464.

High-Quality Evidence?

- Few randomized controlled human trials in cardiac arrest
  - Retrospective
  - Animal subjects
  - Case reports/series
  - Expert consensus
- Cornerstone of therapy: high-quality CPR and early defibrillation
  - ≥ 2 inches, 100-120 compressions/min
  - Allow complete chest recoil
  - Minimize interruptions

Link MS, et al. *Circulation.* 2015;132:S444-464.

Major Changes in the 2015 Update

- Vasopressin removed from the Adult Cardiac Arrest Algorithm
- Prognostication during CPR based on exhaled CO_2 measurements
- Bundle treatment of steroids, vasopressin, and epinephrine may provide some benefit in treating IHCA
- In cardiac arrest with non-shockable rhythm, the early provision of epinephrine is suggested

Link MS, et al. *Circulation.* 2015;132:S444-464.

Overview

- Vasopressin removal from ACLS algorithm
- Alteplase in cardiac arrest due to PE
- Sodium bicarbonate
- Targeted temperature management

Epinephrine vs. Vasopressin

- Epinephrine: alpha-adrenergic effects
  - Increase coronary perfusion pressure
  - Cerebral perfusion pressure
  - Dose: 1 mg IV/IO q 3-5 min
- Vasopressin: non-adrenergic peripheral vasoconstrictor
  - Coronary and renal vasoconstriction
  - Dose: 40 units IV/IO x 1
  - Cost: 20 unit vial – $135

Link MS, et al. *Circulation.* 2015;132:S444-464.

Why Vasopressin?

- Large release after cardiac arrest
  - Higher the endogenous concentration, greater chances of return of spontaneous circulation (ROSC)
- Long duration cardiac arrest
  - Severe hypoxia and acidosis
  - Vasopressin seems to be more effective than epinephrine in achieving ROSC
- Animal studies suggest vasopressin is associated with better vital organ perfusion and resuscitation rates than epinephrine for cardiac arrest

Lindner KJ, et al. *Lancet*. 1997;349:535-537.
Lindner KJ, et al. *Br Heart J*. 1996;75:145-150.
Lindner KJ, et al. *Ann Intern Med*. 1996;124:1061-1064.

Conflicting Data…

Trial	Patient Population	Intervention	Outcomes (Epi vs. Vaso)
Randomized comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation[1]	N = 40 VF OHCA refractory to defibrillation	Epinephrine 1 mg (n = 20) OR vasopressin 40 units (n = 20) as initial therapy	**Survival to admission: 35% vs. 70% (p = 0.06)** Survival at 24 hr: 20% vs. 60% (p = 0.02) Survival to discharge: 15% vs. 40% (p = 0.16)
Vasopressin versus epinephrine for in-hospital cardiac arrest: a randomized controlled trial[2]	N = 200 IHCA of any initial rhythm	Epinephrine 1 mg (n = 96) OR vasopressin 40 units (n = 104) as initial therapy	Survival to discharge: 14% vs. 12% (p = 0.67) Survival at 1 hr: 35% vs. 39% (p = 0.66)
A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation[3]	N = 1186 OHCA of any initial rhythm	Epinephrine 1 mg (n = 597) OR vasopressin 40 units (n = 589) If ROSC not achieved after 3 min, the same study drug was injected again	Survival to admission: 31.2% vs. 36.3% (p = 0.06) Survival to discharge: 9.9% vs. 9.9% (p = 0.99) <u>Asystole</u> Survival to admission: 20.3% vs. 29.0% (p = 0.02) Survival to discharge: 1.5% vs. 4.7% (p = 0.04)

VF = ventricular fibrillation

1. Lindner KJ, et al. *Lancet*. 1997;349:535-537.
2. Stiell IG, et al. *Lancet*. 2001;358:105-109.
3. Wenzel V, et al. *N Engl J Med*. 2004;350:105-113.

ORIGINAL ARTICLE

Vasopressin and Epinephrine vs. Epinephrine Alone in Cardiopulmonary Resuscitation

- Multicenter, randomized controlled trial of adults with out-of-hospital cardiac arrest
  - May 1, 2004 – April 30, 2006
- Epinephrine 1 mg + vasopressin 40 units vs. epinephrine 1 mg + placebo
  - Repeat if ROSC not restored within 3 min
  - Epinephrine thereafter
- Primary Endpoint: survival to hospital admission
- Secondary Endpoints: ROSC, survival to hospital discharge, good neurologic recovery, 1-year survival

Gueugniaud PY, et al. *N Engl J Med.* 2008;359:21-30.

Results

- Baseline characteristics similar between both groups
  - Exception: male sex (75.4% vs. 71.7%, p = 0.03)
- Witnessed cardiac arrest were more likely to survive to hospital admission (23.2% vs. 14.4%, p < 0.001)
- BLS ≤ 8 min and ACLS ≤ 12 min higher rate of survival to admission (38.3% vs. 20.5%, p = 0.001)

Table 2. Survival Data for the 2894 Patients in the Intention-to-Treat Population.*

End Point	Combination Treatment (N=1442)	Epinephrine Only (N=1452)	Relative Risk of Death (95% CI)	P Value
Survival to hospital admission — no. (%)	299 (20.7)	310 (21.3)	1.01 (0.97–1.05)	0.69
Survival to return of spontaneous circulation — no. (%)	413 (28.6)	428 (29.5)	1.01 (0.97–1.06)	0.62
Survival to hospital discharge — no./total no. (%)	24/1439 (1.7)	33/1448 (2.3)	1.01 (1.00–1.02)	0.24
1-Year survival — no./total no. (%)	18/1437 (1.3)	30/1447 (2.1)	1.01 (1.00–1.02)	0.09
Good neurologic recovery at hospital discharge — no./total no. (%)†	9/24 (37.5)	17/33 (51.5)	1.29 (0.81–2.06)	0.29

Gueugniaud PY, et al. *N Engl J Med.* 2008;359:21-30.

Results

- Post hoc subgroup analysis evaluating combination vs. epinephrine alone on rate of survival to discharge
  - Ventricular fibrillation: 6.8% vs. 10.3%, NS
  - Asystole: 1.3% vs. 1.0%, NS
  - **PEA: 0.0% vs. 5.8%, p = 0.02**
- Authors' Conclusions: Lack of superiority on outcomes suggests it may be futile to add vasopressin to epinephrine during ACLS for OHCA

Gueugniaud PY, et al. *N Engl J Med.* 2008;359:21-30.

Updated 2015 Recommendation

- Vasopressin offers no advantage as a substitute for epinephrine in cardiac arrest (Class IIb, LOE B-R)

- Vasopressin in combination with epinephrine offers no advantage as a substitute for standard-dose epinephrine in cardiac arrest (Class IIb, LOE B-R)

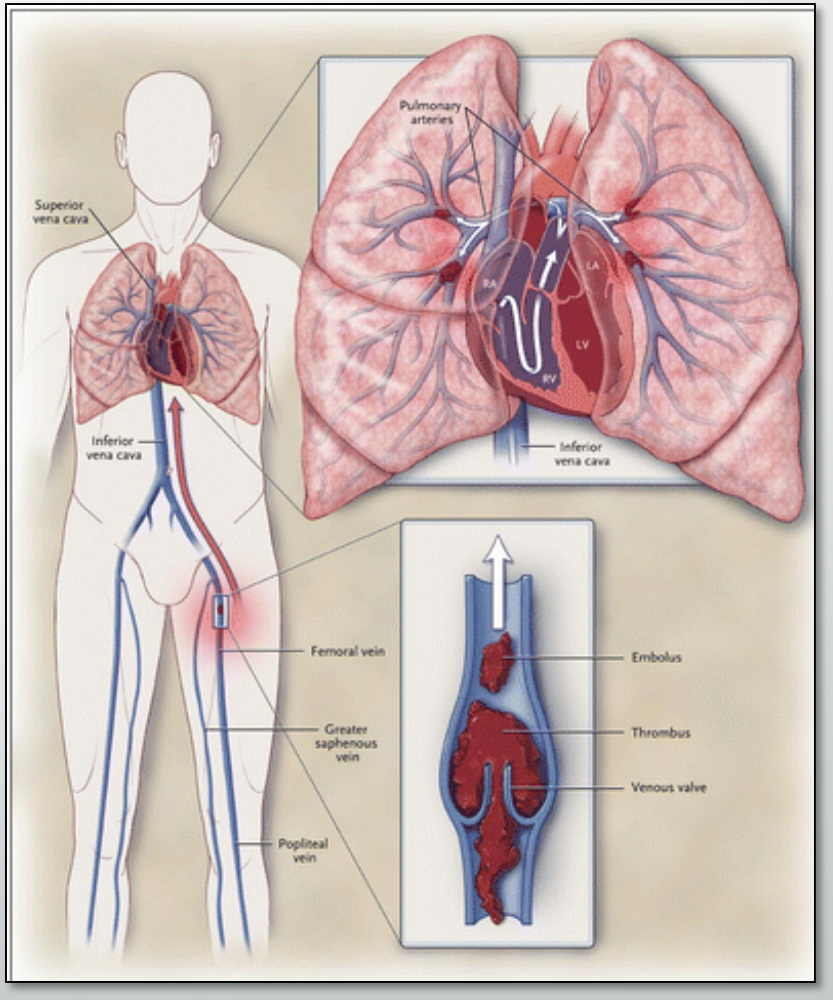
Link MS, et al. *Circulation.* 2015;132:S444-464.

Overview

- Vasopressin removal from the ACLS algorithm
- Alteplase in cardiac arrest due to PE
- Sodium bicarbonate
- Targeted temperature management

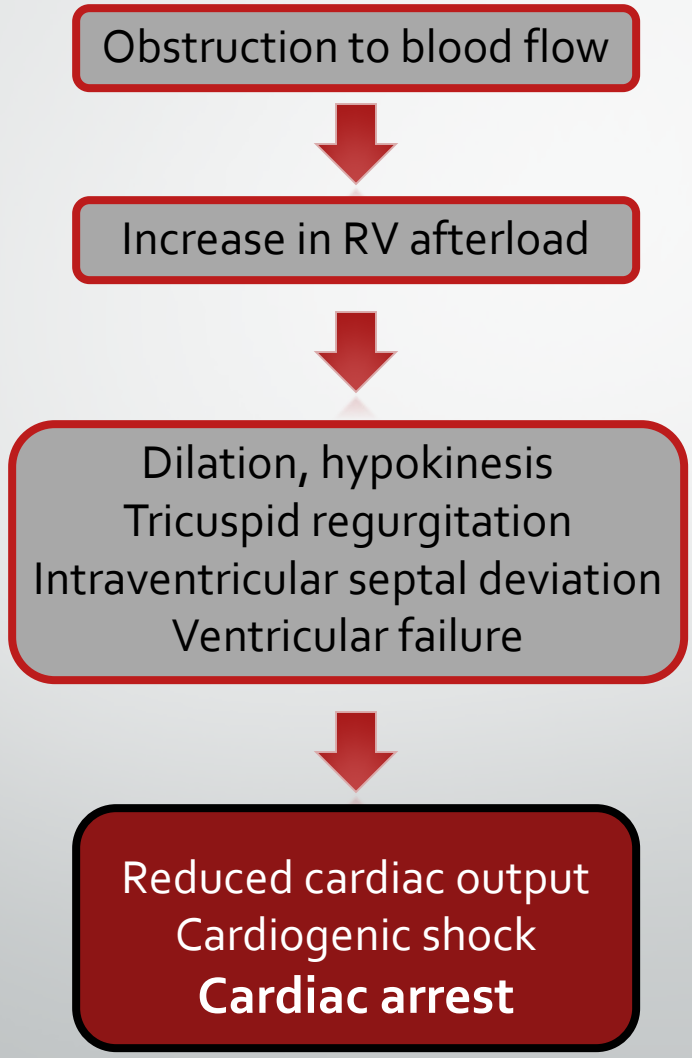
Acute Pulmonary Embolism

- Submassive vs. massive PE
  - Fulminant PE
- Less than 5% progress to cardiac arrest
  - Mortality: 65-90%
  - May occur within hours of symptom onset
  - Most common rhythm: PEA
- Variable, non-specific signs and symptoms
  - May lead to delayed diagnosis



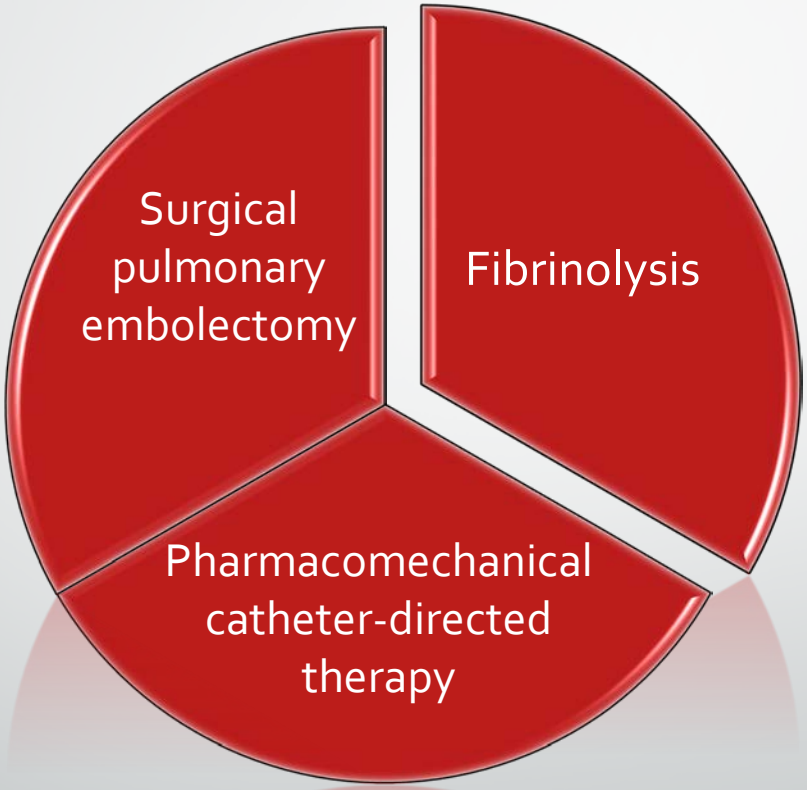
Bailen MR, et al. *Crit Care Med.* 2001;29:2211-2219.
Tapson VF. *N Engl J Med*. 2008;358:1037-1052.
Lavonas EJ, et al. *Circulation.* 2015;132:S501-518.

Acute Pulmonary Embolism

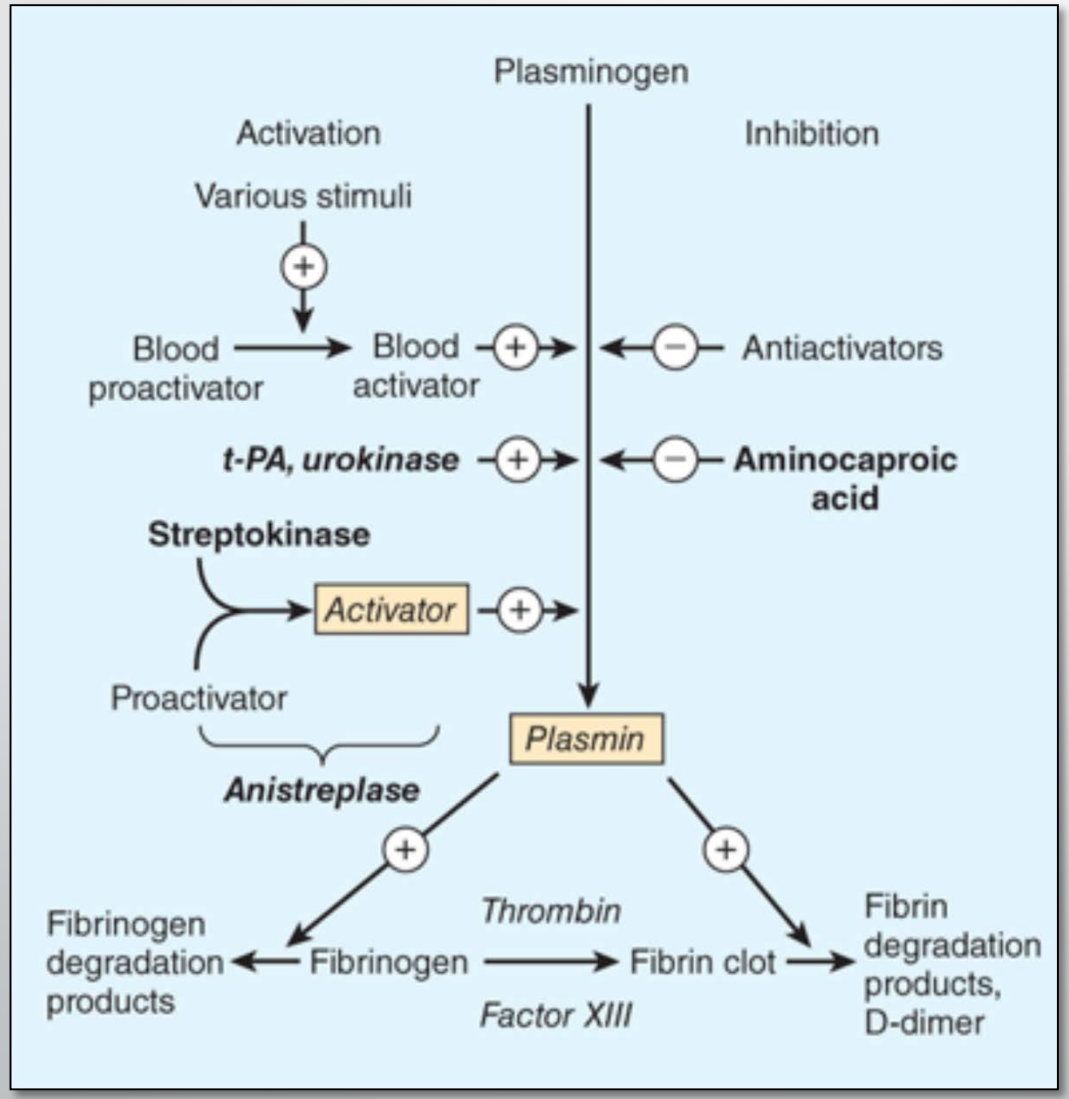


Bailen MR, et al. *Crit Care Med.* 2001;29:2211-2219.

Treatment Options



Fibrinolysis



Katzung BG, Trevor AJ. *Basic & Clinical Pharmacology, 13e*; 2015

Alteplase

- Mechanism: convert plasminogen to plasmin, leading to degradation of the fibrin matrix within a thrombus
  - During CPR: improved microcirculatory reperfusion and hemodynamic stability
- Duration of action: ~80% cleared in 10 min; fibrinolytic activity persists for ~1 hour post-infusion
- Only selective thrombolytic agent FDA approved for PE
- Cost
  - Alteplase 50 mg vial: $3988
  - Alteplase 100 mg vial: $7976

Tapson VF. *N Engl J Med*. 2008;358:1037-1052..

Thrombolytics in ACLS

Trial	Patient Population	Intervention	Outcomes
Efficacy and safety of thrombolytic therapy after initially unsuccessful cardiopulmonary resuscitation: a prospective clinical trial[1]	N = 40 study drugs N = 50 historical control OHCA where ROSC not achieved in 15 min	rt-PA 50 mg + heparin 5000 units vs. control If ROSC not achieved in following 30 min, repeat	**ROSC: 68% vs. 44% (p = 0.026)** Admitted: 58% vs. 30% (p = 0.009) No difference in bleeding complications, 24 hr survival, or hospital discharge
Tissue plasminogen activator in cardiac arrest with pulseless electrical activity[2]	N = 117 t-PA N = 116 placebo OHCA (undifferentiated) with initial rhythm of PEA	t-PA 100 mg over 15 min vs. placebo	**Survival to discharge: 0.9% vs. 0.0% (p = 0.99)** No difference in ROSC, survival to admission, hemorrhage Avg 32.1 min from CPR to t-PA Only 1 case with confirmed PE
Thrombolysis during resuscitation for out-of-hospital cardiac arrest[3] (TROICA Trial)	N = 525 in each group OHCA	Tenecteplase vs. placebo	**30-day survival:** **14.7% vs. 17.0% (p = 0.36)** No difference in ROSC, hospital admission or discharge More intracranial hemorrhage in tenecteplase (2.7% vs. 0.4%, p = 0.006)

1. Bottiger BW, et al. *Lancet*. 2001;357:1583-1585.
2. Abu-Laban RB, et al. *N Engl J Med*. 2002;356:1522-1528.
3. Bottiger BW, et al. *N Engl J Med*. 2008;359:2651-2662.

Thrombolytics in ACLS

Objective	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Patient Demographics	Female 45 years old	Male 28 years old	Male 34 years old	Male 73 years old	Male 76 years old	Female 56 years old
Presenting rhythm	PEA	PEA	Asystole	PEA	Asystole	PEA
CPR Initiation	Immediate	After 5 min	After ≥ 10 min	Immediate	Immediate	Immediate
rt-PA delay	60 min	30 min	30 min	15 min	Immediate	15 min
rt-PA Regimen	50 mg bolus + 50 mg bolus after 30 min	50 mg bolus + 50 mg bolus after 30 min	50 mg bolus	50 mg bolus + 50 mg bolus after 30 min	50 mg bolus + 50 mg bolus after 30 min	50 mg bolus + 50 mg bolus after 30 min
Total CPR time	70 min	45 min	90 min	30 min	5 min	40 min
Post-CPR complications	Hemorrhage at injection sites	None	None	None	Hemorrhage at injection sites, UGIB	None
Outcomes	Alive at 1 yr	Death	Death	Alive at 1 yr	Stroke at 1 yr	Alive at 6 mon

UGIB = Upper gastrointestinal bleed

ROSC and survival to discharge: 4/6 (66.7%)
Mortality cases had no possibility of survival (carcinoma of the pancreas, CPR initiation > 10 min)
No fatal hemorrhages reported

Ruiz-Bailen M, et al. *Resuscitation.* 2001;51:97-101.

Thrombolytics in ACLS

- Do not delay thrombolytic therapy for confirmation when high clinical suspicion
  - Minimize time from collapse to administration of therapy
- Dosing options: 50 mg vs. 100 mg
- Continue resuscitation for at least 15-20 min if ROSC is not achieved

<u>AHA ACLS Guideline Recommendations</u>

In patients with cardiac arrest and without known PE, routine fibrinolytic treatment given during CPR shows no benefit and is not recommended (Class III, LOE A)

In patients with confirmed PE as the precipitant of cardiac arrest, thrombolysis, surgical embolectomy, and mechanical embolectomy are reasonable emergency treatment options (Class IIa, LOE C-LD)

Thrombolysis may be considered when cardiac arrest is suspected to be caused by PE (Class IIb, LOE C-LD)

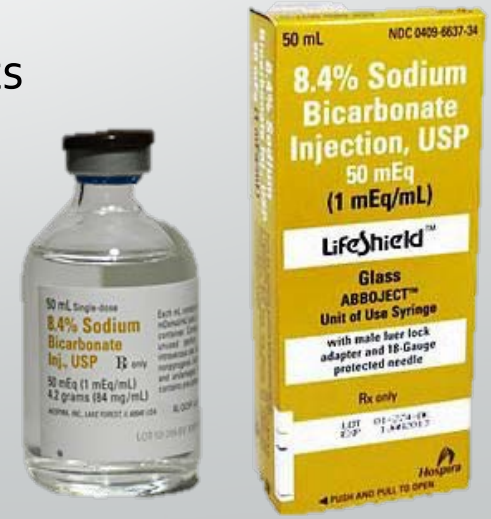
Lavonas EJ, et al. *Circulation.* 2015;132:S501-518.

Overview

- Vasopressin removal from ACLS algorithm
- Alteplase in cardiac arrest due to PE
- Sodium bicarbonate
- Targeted temperature management

Sodium Bicarbonate

- Tissue acidosis occurs during severe ischemia seen in cardiac arrest
  - Rapid accumulation of CO_2
  - Slow accumulation of lactic acid
- Until 1986, sodium bicarbonate (buffer therapy) was an integral component of ACLS
  - Lack of benefit and possibly deleterious effects
  - Compressions and hyperventilation sufficient
- 50 mEq/ 50 mL = 1 ampule



Dybvik T, et al. *Resuscitation*. 1995:89-95.

Theoretical Disadvantages

- Reduce systemic vascular resistance
- Extracellular alkalosis → shift in oxyhemoglobin saturation curve → inhibit oxygen release
- Hypernatremia
- Excess CO_2 causing intracellular acidosis
- Exacerbate central venous acidosis and inactivate catecholamines

Dybvik T, et al. *Resuscitation*. 1995:89-95.

Sodium bicarbonate improves outcome in prolonged prehospital cardiac arrest☆

Rade B. Vukmir MD, JD, FCCP, FACEP*, Laurence Katz MD
Sodium Bicarbonate Study Group[1]

- Prospective, randomized, double-blind study in OHCA refractory to defibrillation
- Early administration of empirical (1 mEq/kg) sodium bicarbonate (n = 420) vs. placebo (n = 372)
  - Brief (< 5 min), moderate (5-15 min), and prolonged (>15 min) down time
- Primary outcome: survival to admission 13.9% vs. 13.8%, p = 0.199
  - Prolonged (> 15 min) down time: 32.8% vs. 15.4%, p = 0.007
- Conclusions: early administration of sodium bicarbonate has no effect on overall outcome in OHCA

Vukmir RB, et al. *American Journal of Emergency Medicine.* 2006;24:156-161.

Sodium Bicarbonate: When to use it?

- Pre-existing metabolic acidosis
- Hyperkalemia
- Overdose causing QRS ≥ 120 msec

AHA ACLS Guideline Recommendations

Routine use of sodium bicarbonate is not recommended for patients with cardiac arrest (Class III, LOE B)

Link MS, et al. *Circulation.* 2015;132:S444-464.

Overview

- Vasopressin removal from ACLS algorithm
- Alteplase in cardiac arrest due to PE
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Post-Cardiac Arrest Syndrome

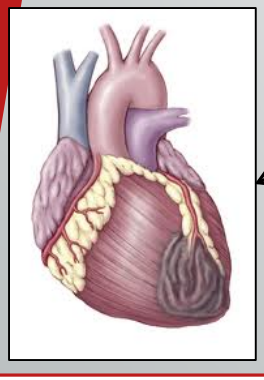
- Poor prognosis despite achieving ROSC
  - 12% of out-of-hospital arrests survive to discharge
  - 24.8% of in-hospital arrests survive to discharge
- ~80% of patients remain comatose for more than an hour post-resuscitation
  - Secondary brain injury
  - Myocardial dysfunction
  - Systemic ischemia
  - Consequences of the disorder that caused the arrest
  - Ongoing inflammation and injury

Post-Cardiac Arrest Syndrome

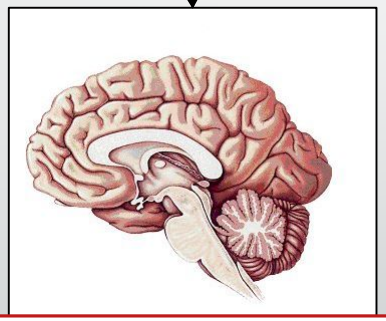
Cardiac Arrest → No Blood Flow/O_2 Deprivation → Ischemic Injury

Coagulopathy/ Toxic Metabolites

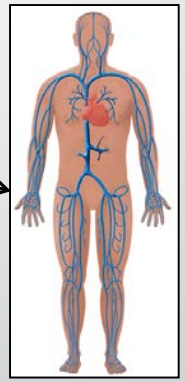
ROSC



- Myocardial dysfunction
- "Stunning"



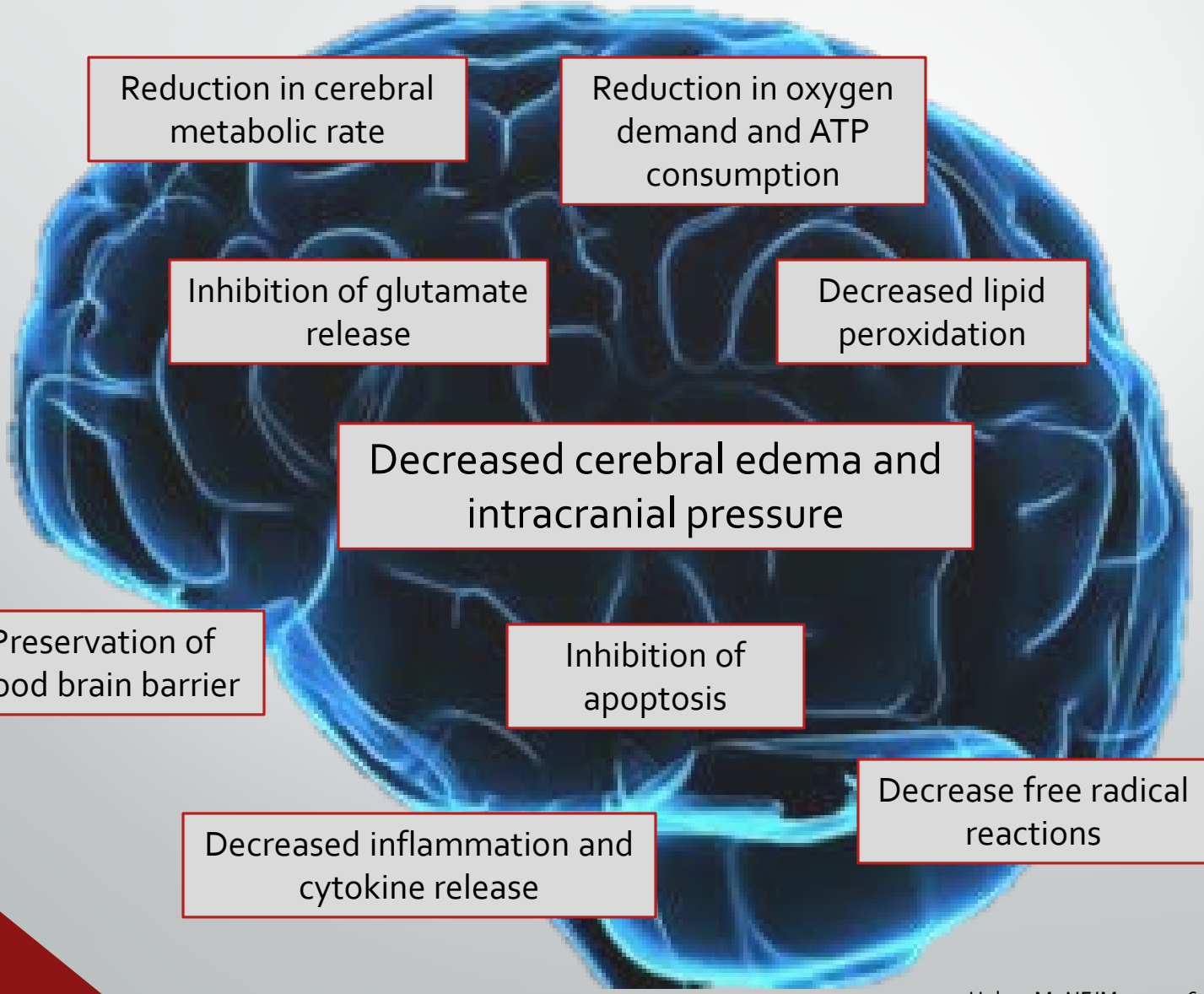
- Hyperemic macro-circulation
- Excitotoxicity
- Disrupted Ca^{2+} homeostasis
- Free radical formation
- Activation of apoptosis



- Ischemic metabolites
- Poor O_2 delivery
- ↑Immune & coagulation cascade

Neumar, et al. *Circulation*. 2008; 118:2452.
Stub, et al. *Circulation*. 2011; 123:1428.

Targeted Temperature Management



Holzer M. *NEJM* 2010; 363(13): 1256-1264.

Landmark Trials Assessing Therapeutic Hypothermia

Trial	Patient Population	Intervention	Outcomes
Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest[1]	N = 138 (hypothermia) N = 137 (normothermia) RCT of comatose patients with ROSC after OHCA due to ventricular fibrillation	Therapeutic hypothermia (32-34°C) over 24 hr vs. normothermia	**Favorable neurologic (CPC 1 or 2) outcome at 6 months: 55% vs. 39%, p = 0.009** Mortality at 6 months: 41% vs. 55%, p = 0.02 Complications within 7 days: no difference
Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia[2]	N = 43 (hypothermia) N = 34 (normothermia) RCT of comatose patients with ROSC after OHCA due to ventricular fibrillation	Therapeutic hypothermia 33°C within 2 hr of ROSC for 12 hr vs. normothermia	**Survival to hospital discharge with sufficient neurologic function: 49% vs. 26%, p = 0.046** OR 5.25 (1.47-18.76; p = 0.011) No difference in frequency of adverse events

2010 AHA ACLS Guideline Recommendations

Comatose adult patients with ROSC after out-of-hospital VF cardiac arrest should be cooled to 32-34°C for 12-24 hours

Considerations for comatose adult patients with ROSC after in-hospital cardiac arrest of any initial rhythm or after out-of-hospital cardiac arrest with an initial rhythm of PEA or asystole

1. Holzer M, et al. *N Engl J Med.* 2002;346:549-556.
2. Bernard SA, et al. *N Engl J Med.* 2002;346:557-563.
Peberdy MA, et al. *Circulation.* 2010;122:S768-786.

ORIGINAL ARTICLE

Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest

- Multicenter, randomized controlled trial of adults who remain unconscious (GCS ≤ 8) after ROSC from out-of-hospital cardiac arrest of presumed cardiac cause
  - November 2010 – January 2013
- Targeted temperature management 33°C vs. 36°C for 24 hr
- Primary Endpoint: all-cause mortality
- Secondary Endpoints: composite of poor neurologic function or death at 6 months

Nielsen N, et al. *N Engl J Med.* 2013;369:2197-2206.

TTM Trial: Results

Adverse Events

- ≥ 1 serious adverse event: 93% vs. 90%, p = 0.09
- Hypokalemia: 19% vs. 13%, p = 0.02
- Shivering: 30% vs. 34%, p = 0.20

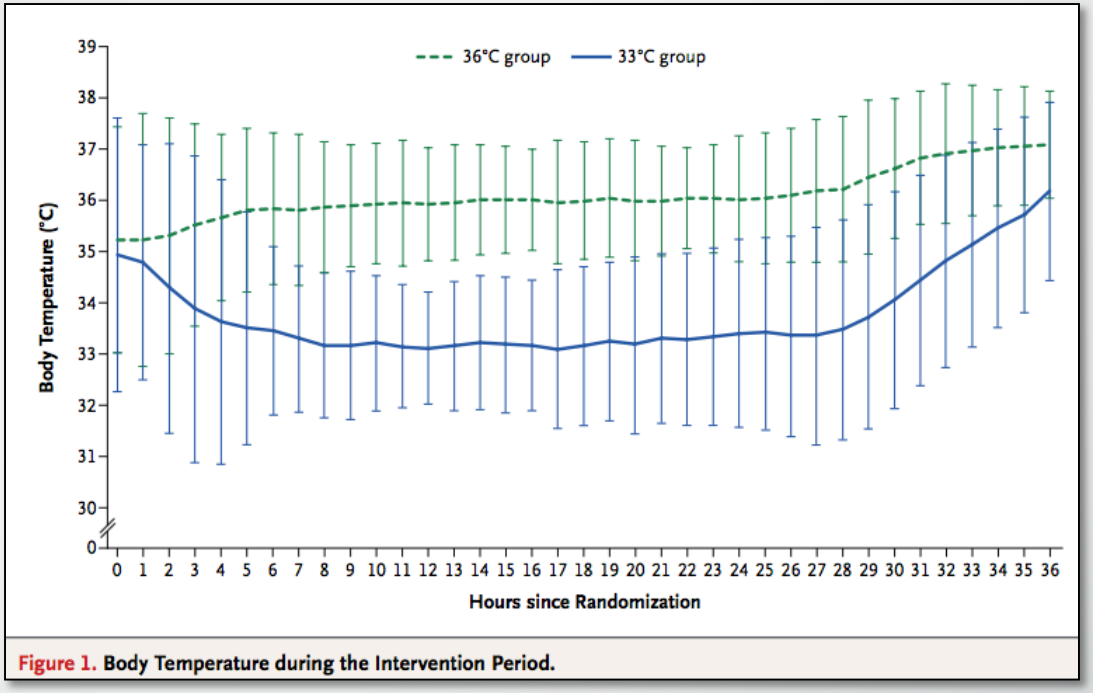


Figure 1. Body Temperature during the Intervention Period.

Table 2. Outcomes.

Outcome	33°C Group	36°C Group	Hazard Ratio or Risk Ratio (95% CI)*	P Value
	no./total no. (%)			
Primary outcome: deaths at end of trial	235/473 (50)	225/466 (48)	1.06 (0.89–1.28)	0.51
Secondary outcomes				
Neurologic function at follow-up†				
CPC of 3–5	251/469 (54)	242/464 (52)	1.02 (0.88–1.16)	0.78
Modified Rankin scale score of 4–6	245/469 (52)	239/464 (52)	1.01 (0.89–1.14)	0.87
Deaths at 180 days	226/473 (48)	220/466 (47)	1.01 (0.87–1.15)	0.92

Nielsen N, et al. *N Engl J Med.* 2013;369:2197-2206.

TTM Trial: Conclusions

- No significant differences between 33°C and 36°C in overall mortality or composite of poor neurologic function or death at 180 days
- No difference in harm
- Hypothermia influences all organ systems, any potential benefits should be balanced with possible side effects

<u>2015 AHA ACLS Guideline Recommendations</u>

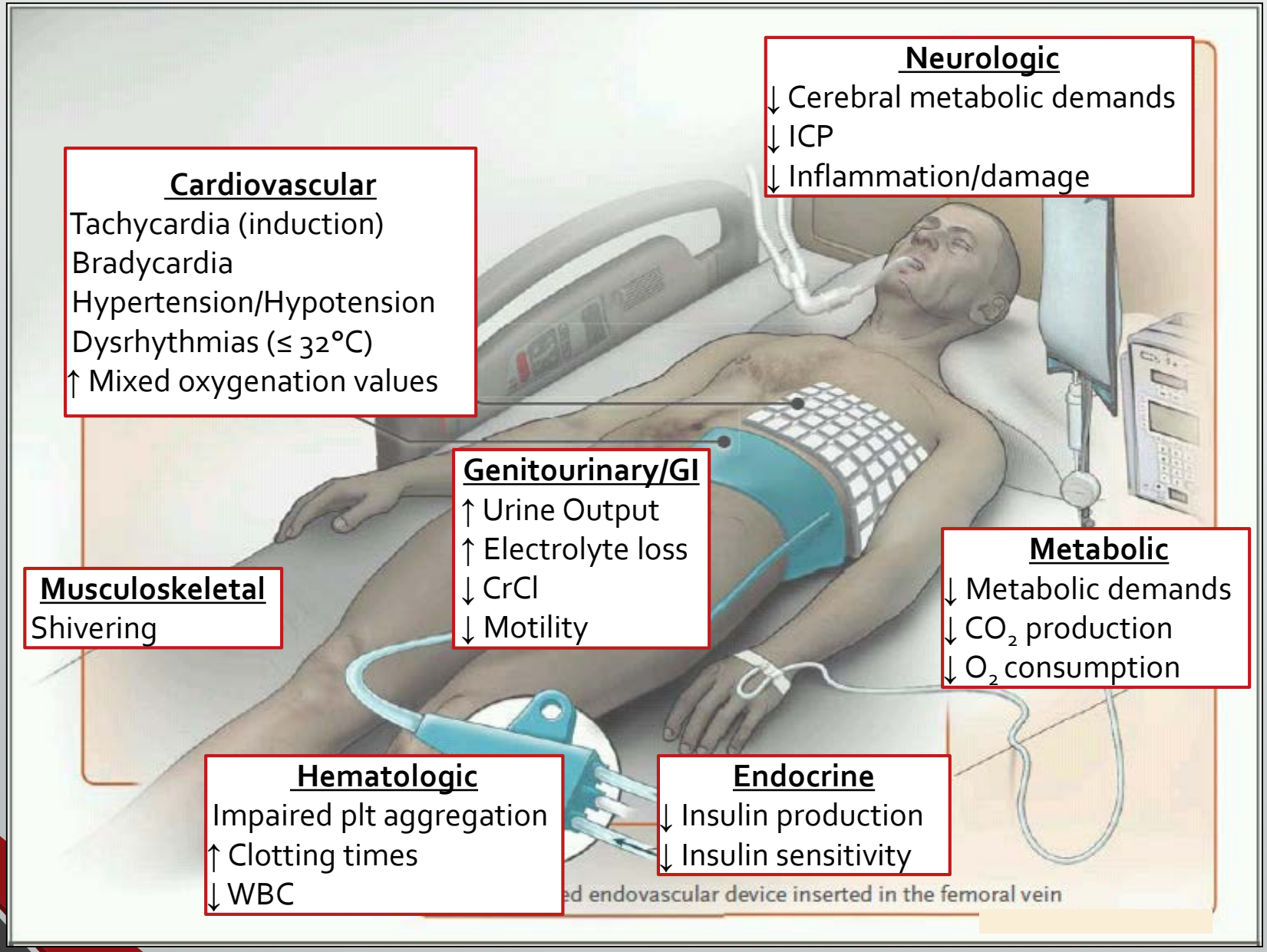
Comatose (i.e., lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest should have TTM (Class I)

Select and maintain a constant temperature between 32°C and 36°C during TTM (Class I)

Recommend **against** the routine pre-hospital cooling of patients after ROSC with rapid infusion of cold intravenous fluids (Class III: no benefit)

Nielsen N, et al. *N Engl J Med.* 2013;369:2197-2206.
Link MS, et al. *Circulation.* 2015;132:S444-464

Physiologic Effects of Cooling



Holzer, et al. *N Engl J Med.* 2010;363:1256.

Altered Pharmacokinetics and Pharmacodynamics

- Temperature-dependent enzyme-mediated reactions
  - Decrease in rate of drug metabolism by the liver
  - Reduction in drug clearance
- Temperature affect response to drugs
  - Blunted effects of drugs
  - Changes in volume of distribution
- Careful monitoring to assess for toxicity and efficacy

Polderman KH. *Crit Care Med.* 2009;37:S186-202.
Tortorici MA, et al. *Crit Care Med*. 2007;35:2196-2204

Pharmacologic Considerations during TTM

- Sedation
  - Midazolam: metabolized by CYP3A4 and 3A5
    - 5-fold increase in plasma concentrations and 100-fold decrease in systemic clearance in < 35°C
  - Propofol: metabolized by CYP2B6
    - Increased concentrations by 30% during hypothermia (34°C vs. 37°C)
- Analgesia
  - Fentanyl: metabolized by CYP3A4
    - Plasma concentrations 2-fold higher and clearance decreased 3.7-fold during hypothermia
  - Morphine: metabolized by hepatic glucuronidation
    - Reduced affinity at the μ-receptor as temperature decreased

Polderman KH. *Crit Care Med.* 2009;37:S186-202.
Tortorici MA, et al. *Crit Care Med*. 2007;35:2196-2204

Pharmacologic Considerations during TTM

- Electrolyte management
  - Monitor K^+, Mg^{2+}, PO_4^- closely
    - Renal tubule dysfunction
    - Electrolyte shifts intra-/extracellular
- Glycemic management
  - Decreased insulin production and receptor sensitivity
  - Increased glycemic variability is associated with increased mortality
  - Hyperglycemia during induction and maintenance
  - Hypoglycemia during rewarming

Bernard S. *Neurol Clin* 2006; 24: 61-71
Mirzoyev, et al. *Resuscitation*. 2010;81:1632

Summary

- The 2015 AHA ACLS Guidelines contain several important updates including the removal of vasopressin from the cardiac arrest algorithm
- Thrombolytic administration should be considered when there is confirmed or high suspicion of pulmonary embolism causing cardiac arrest
- Evidence of sodium bicarbonate in ACLS does not show benefit and should not be routinely used
- Comatose patients successfully resuscitated should undergo targeted temperature management (32-36°C), with careful monitoring of medication therapy due to altered pharmacologic properties during cooling

Assessment Question#1

Why was vasopressin removed from the arrest algorithm in the 2015 ACLS Guidelines?

a) Epinephrine was shown to be superior to vasopressin in achieving ROSC and survival to hospital discharge in two large randomized controlled trials

b) Vasopressin is associated with worse vital organ perfusion in animal models during cardiac arrest

c) Vasopressin as a substitute to or in combination with epinephrine has shown no additional benefit and is considered futile to administer during cardiac arrest

d) The exponential increase in cost of vasopressin caused the AHA to remove it from the algorithm

Assessment Question #2

AM is a 55 year-old female admitted to the Surgical ICU with left tibia/fibula fractures and multiple rib fractures after a high-velocity motor vehicle collision. Seven days into her admission, she was diagnosed with a DVT and began therapeutic anticoagulation. The next day, her condition deteriorated and she became unresponsive and found to be in a PEA arrest. CPR began immediately, and a member of the cardiac arrest team asks, "Should we give t-PA?"

What is an appropriate response?

a) Alteplase should be given immediately and administered over 2 hours while continuing CPR

b) Alteplase should be administered immediately as a bolus and chest compressions should continue for at least 15-20 minutes or until ROSC achieved

c) Alteplase should be administered as a bolus after 15-20 minutes of unsuccessful CPR

d) Alteplase should not be administered given the high risk of life threatening hemorrhage

Assessment Question #3

Which of the following is an appropriate pharmacologic consideration for a patient receiving targeted temperature management of 34°C?

a) Fentanyl and midazolam infusion rates should be increased given the rapid clearance of both drugs during hypothermia

b) Frequent glucose monitoring and subsequent insulin administration is necessary given the risk for hyperglycemia during hypothermia

c) TTM can cause increased affinity of drugs such as morphine to the receptors, causing increased potency

d) Drugs metabolized via CYP450 enzymes are the safest to use during TTM as these are temperature independent enzymatic reactions

Stayin' Alive: Updates in ACLS and Post-Cardiac Arrest Management

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