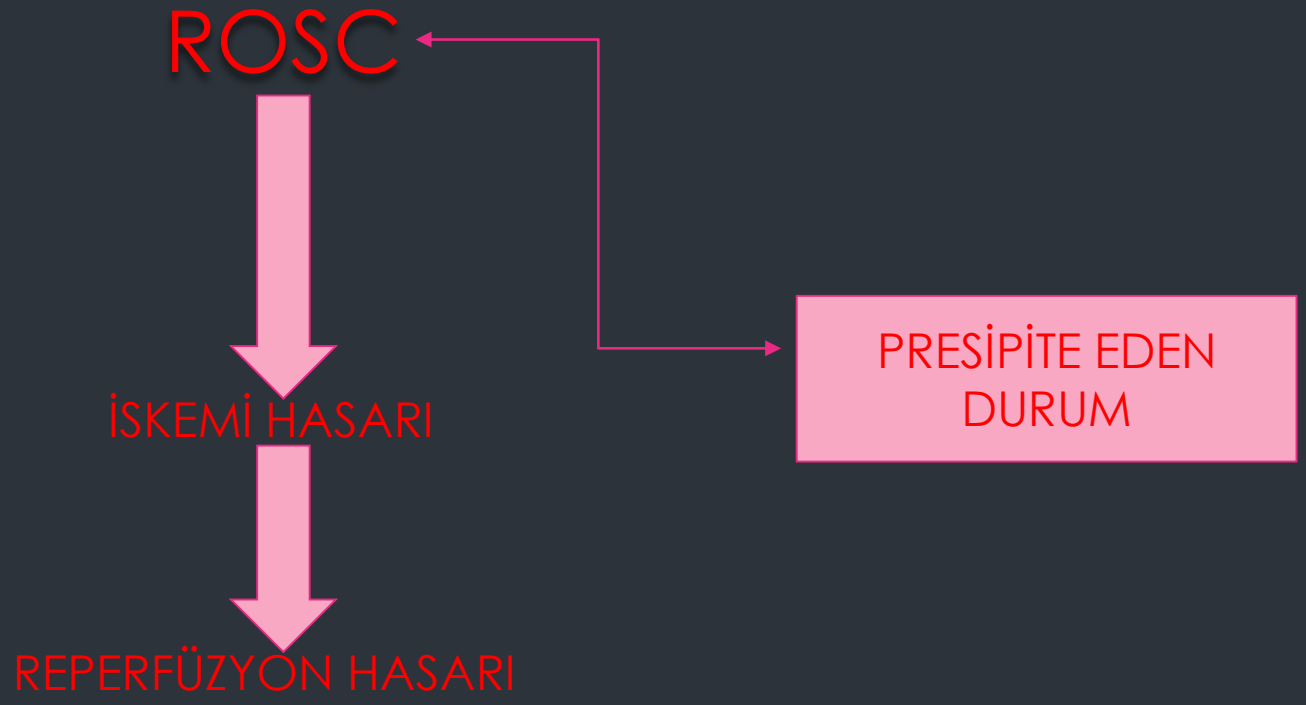
Post ressusitatif bakım

Doç. Dr. İlhan KORKMAZ

CUMHURİYET ÜNİVERSİTESİ

Post-Kardiyak Arrest Sendromu



PKA SONRASI MORTALİTE

- KARDİYAK ARREST:1-3 GÜN
- BEYİN ÖLÜMÜ: GEÇ DÖNEM ÖLÜMLER

Pka NÖROLOJİK HASAR

- HASTANE DIŞI ARRESTLERİN YBÜ 2/3'ÜNDE ÖLÜM NEDENİ
- HASTANE İÇİ %25 ÖLÜM NEDENİ
- KOMA
- KONVÜLZİYON
- MYOKLONUS
- NÖROKOGNİTİF BOZUKLUKLAR
- BEYİN ÖLÜMÜ

- **NÖROLOJİK HASARI ARTTIRAN FAKTÖRLER**
- MİKRODOLAŞIM BOZUKLUĞU
- BOZULMUŞ OTOREGÜLASYON
- HİPOTANSİYON
- HİPERKARBİ
- HİPOKSEMİ
- HİPEROKSEMİ
- ATEŞ
- HİPO-HİPERGLİSEMİ
- KONVÜLZİYONLAR

PKA SONRASI HEDEFLER

- Havayolu ve solunum
- Dolaşım
- Nörolojik sağ kalımda hedefler

Havayolu ve solunum desteği

%94

Oximetry-Guided Reoxygenation Improves Neurological Outcome After Experimental Cardiac Arrest

Irina S. Balan, PhD; Gary Fiskum, PhD; Julie Hazelton, MS; Cynthia Cotto-Cumba, MD; Robert E. Rosenthal, MD

Stroke. 2006;37:3008-3013

Conclusions—A clinically applicable protocol designed to reduce postresuscitative hyperoxia after CA results in significant neuroprotection. Clinical trials of controlled normoxia after CA/restoration of spontaneous circulation should strongly be considered.

Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality. Kilgannon JH, Jones AE, Shapiro NI, et al. JAMA 2010;303:2165–71.

Conclusion: Among patients admitted to the ICU following resuscitation from cardiac arrest, arterial hyperoxia was independently associated with increased in-hospital mortality compared with either hypoxia or normoxia.

ABSTRACT

Aims: To investigate the feasibility of delivering titrated oxygen therapy to adults with return of spontaneous circulation (ROSC) following out-of-hospital cardiac arrest (OHCA) caused by ventricular fibrillation (VF) or ventricular tachycardia (VT).

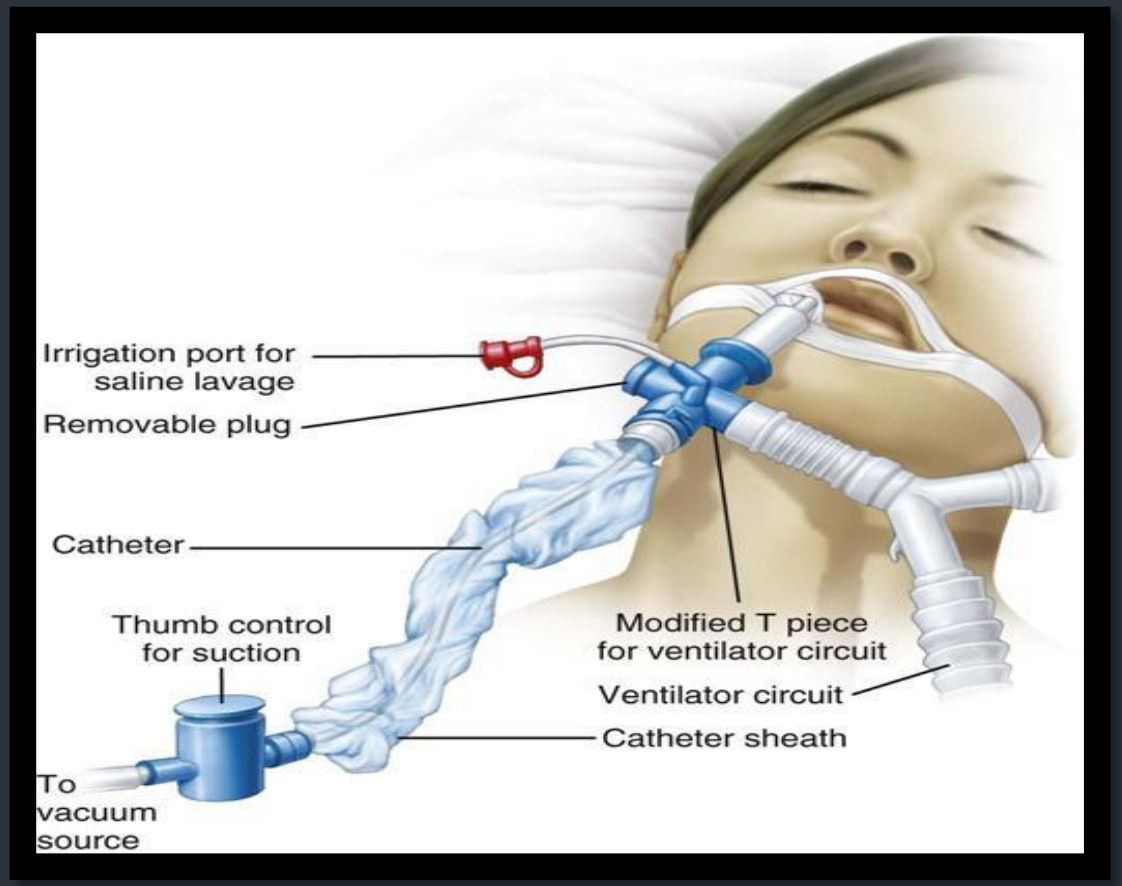
Methods: We used a multicentre, randomised, single blind, parallel groups design to compare titrated and standard oxygen therapy in adults resuscitated from VF/VT OHCA. The intervention commenced in the community following ROSC and was maintained in the emergency department and the Intensive Care Unit. The primary end point was the median oxygen saturation by pulse oximetry (SpO_2) in the pre-hospital period.

Results: 159 OHCA patients were screened and 18 were randomised. 17 participants were analysed: nine in the standard care group and eight in the titrated oxygen group. In the pre-hospital period, SpO_2 measurements were lower in the titrated oxygen therapy group than the standard care group (difference in medians 11.3%; 95% CI 1.0–20.5%). Low measured oxygen saturation (SpO_2 < 88%) occurred in 7/8 of patients in the titrated oxygen group and 3/9 of patients in the standard care group ($P = 0.05$). Following hospital admission, good separation of oxygen exposure between the groups was achieved without a significant increase in hypoxia events. The trial was terminated because accumulated data led the Data Safety Monitoring Board and Management Committee to conclude that safe delivery of titrated oxygen therapy in the pre-hospital period was not feasible.

Conclusions: Titration of oxygen in the pre-hospital period following OHCA was not feasible; it may be feasible to titrate oxygen safely after arrival in hospital.

Given the evidence of harm after myocardial infarction and the possibility of increased neurological injury after cardiac arrest, **as soon as arterial blood oxygen saturation** can be monitored reliably (by blood gas analysis and/or pulse oximetry), **titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%**. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration

Bilinci kapalılarda Ventilasyonun Kontrolü

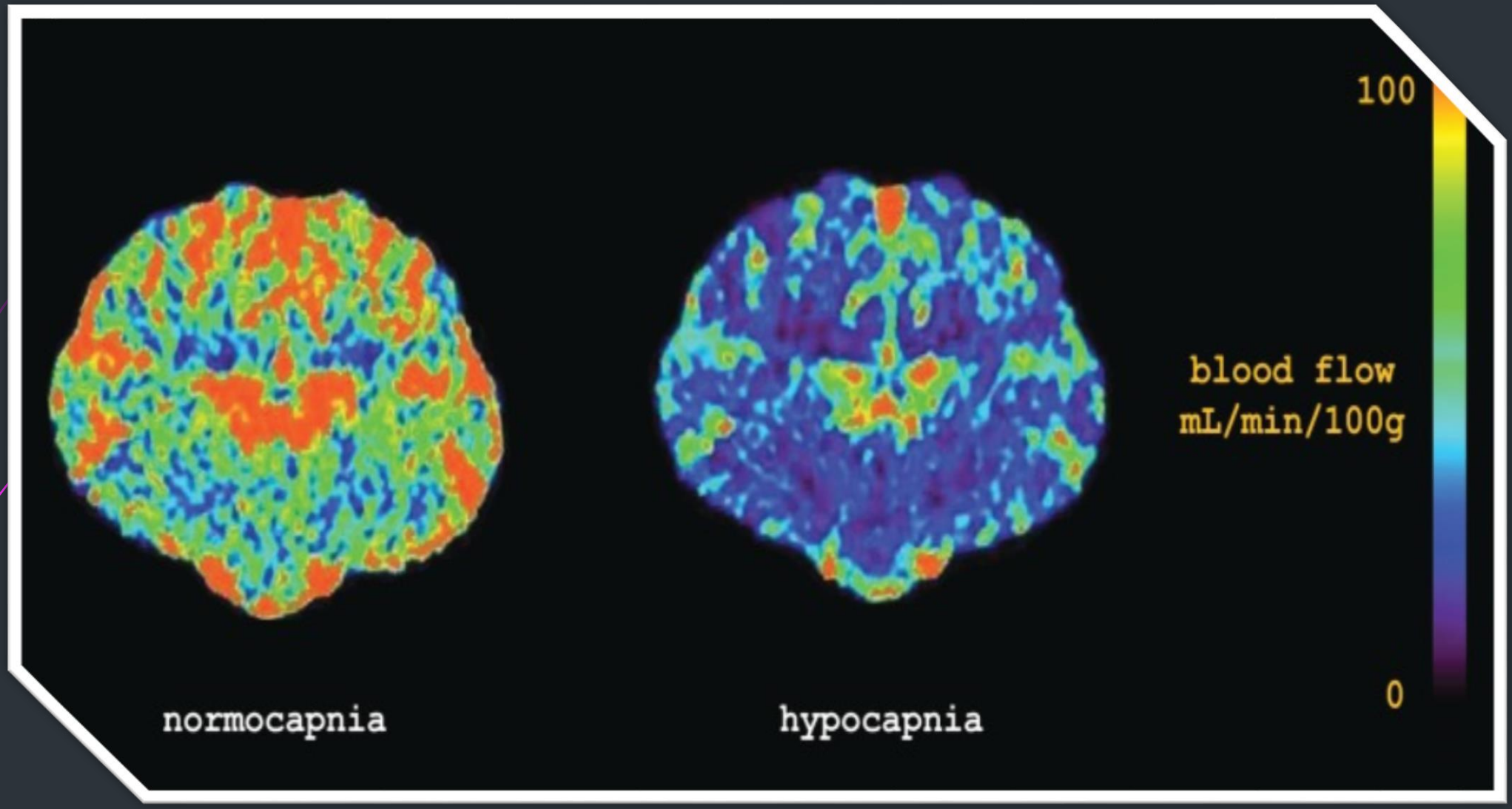


CPR SONRASI KARBONDİOKSİT

We studied 16,542 consecutive patients admitted to 125 ANZ ICUs after CA between 2000 and 2011. Using the APD-$PaCO_2$ (obtained within 24 h of ICU admission), 3010 (18.2%) were classified into the hypo- ($PaCO_2$ < 35 mmHg), 6705 (40.5%) into the normo- (35–45 mmHg) and 6827 (41.3%) into the hypercapnia (>45 mmHg) group. The hypocapnia group, compared with the normocapnia group, had a trend toward higher in-hospital mortality (OR 1.12 [95% CI 1.00–1.24, $p = 0.04$]), lower rate of discharge home (OR 0.81 [0.70–0.94, $p < 0.01$]) and higher likelihood of fulfilling composite adverse outcome of death and no discharge home (OR 1.23 [1.10–1.37, $p < 0.001$]). In contrast, the hypercapnia group had similar in-hospital mortality (OR 1.06 [0.97–1.15, $p = 0.19$]) but higher rate of discharge home among survivors (OR 1.16 [1.03–1.32, $p = 0.01$]) and similar likelihood of fulfilling the composite outcome (OR 0.97 [0.89–1.06, $p = 0.52$]). Cox-proportional hazards modelling supported these findings.

Conclusions: Hypo- and hypercapnia are common after ICU admission post-CA. Compared with normocapnia, hypocapnia was independently associated with worse clinical outcomes and hypercapnia a greater likelihood of discharge home among survivors.

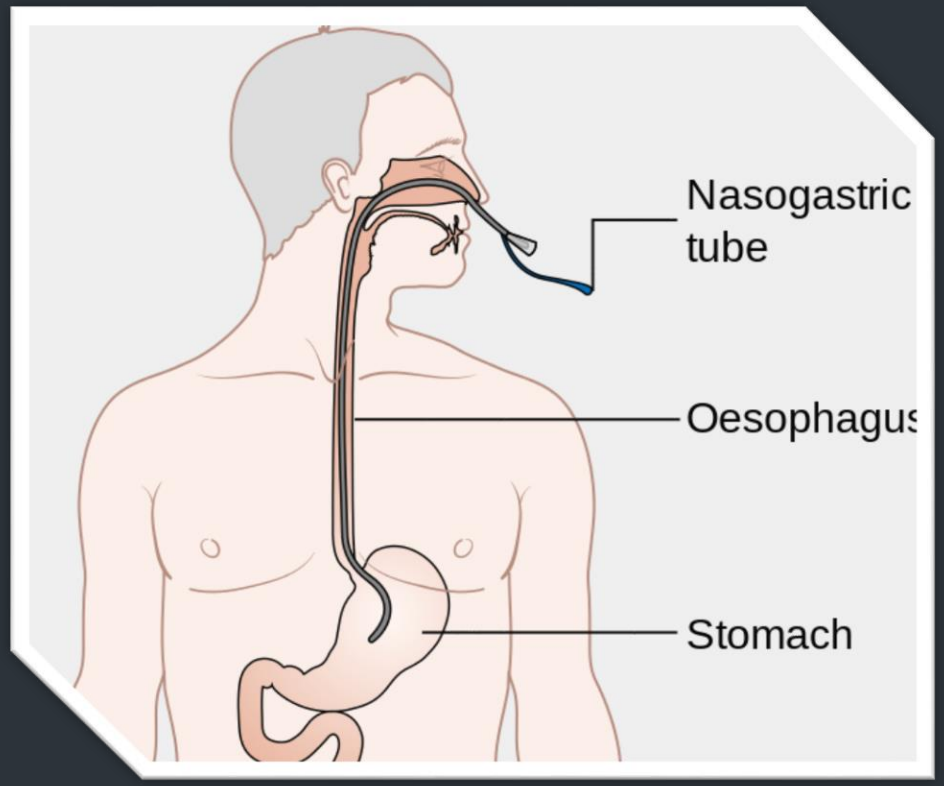
Conclusions: In this multicenter study, hypercapnia was associated with good 12-month outcome in patients resuscitated from out-of-hospital cardiac arrest. We were unable to verify any harm from hyperoxia exposure. Further trials should focus on whether moderate hypercapnia during postcardiac arrest care improves outcome. (Crit Care Med 2014; 42:1463–1470)


100
blood flow
mL/min/100g
0
normocapnia
hypocapnia

Ventilasyon şekli

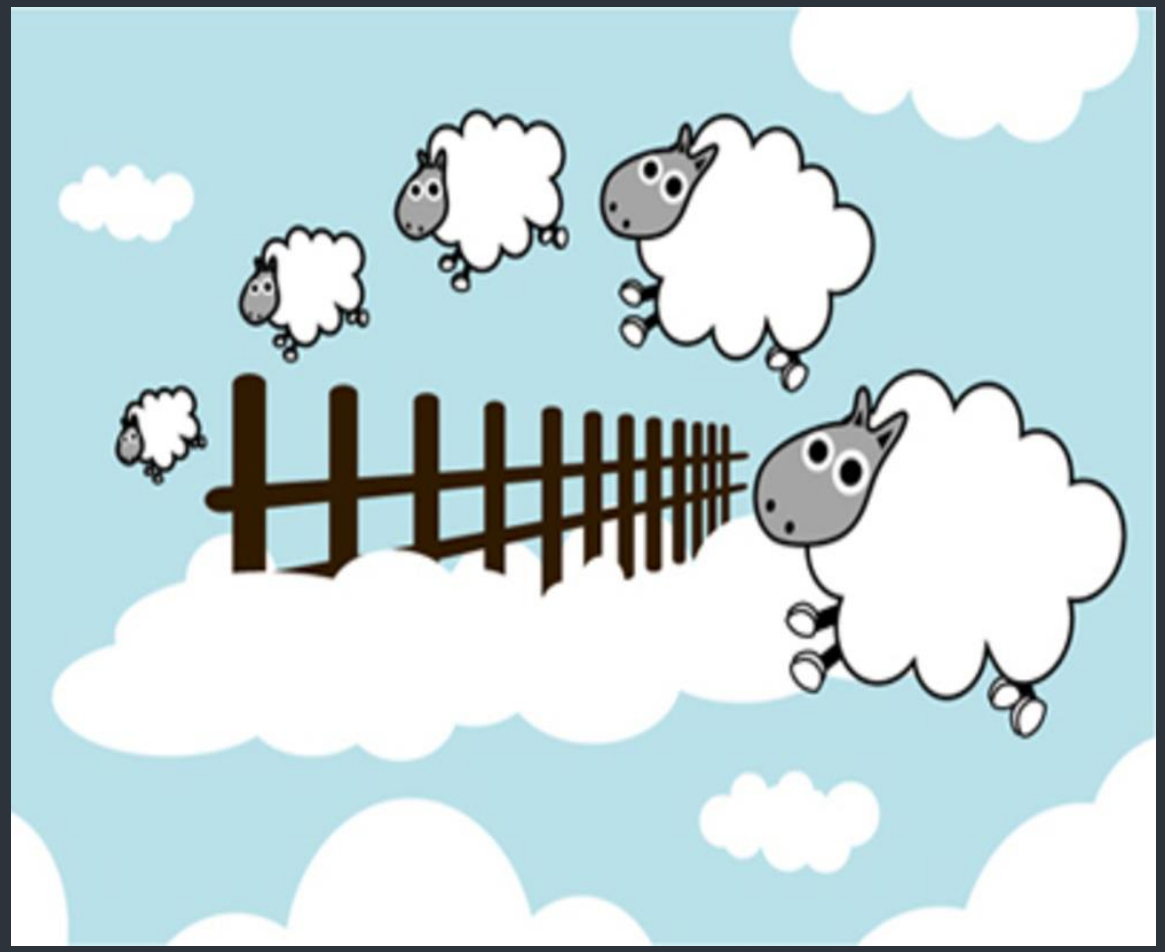
- Tidal volume 6–8 ml/ kg
- PEEP: 4–8 cm H_2O

NG



Sedasyon

- Sedasyon yüksek düzeyde önerilmektedir.

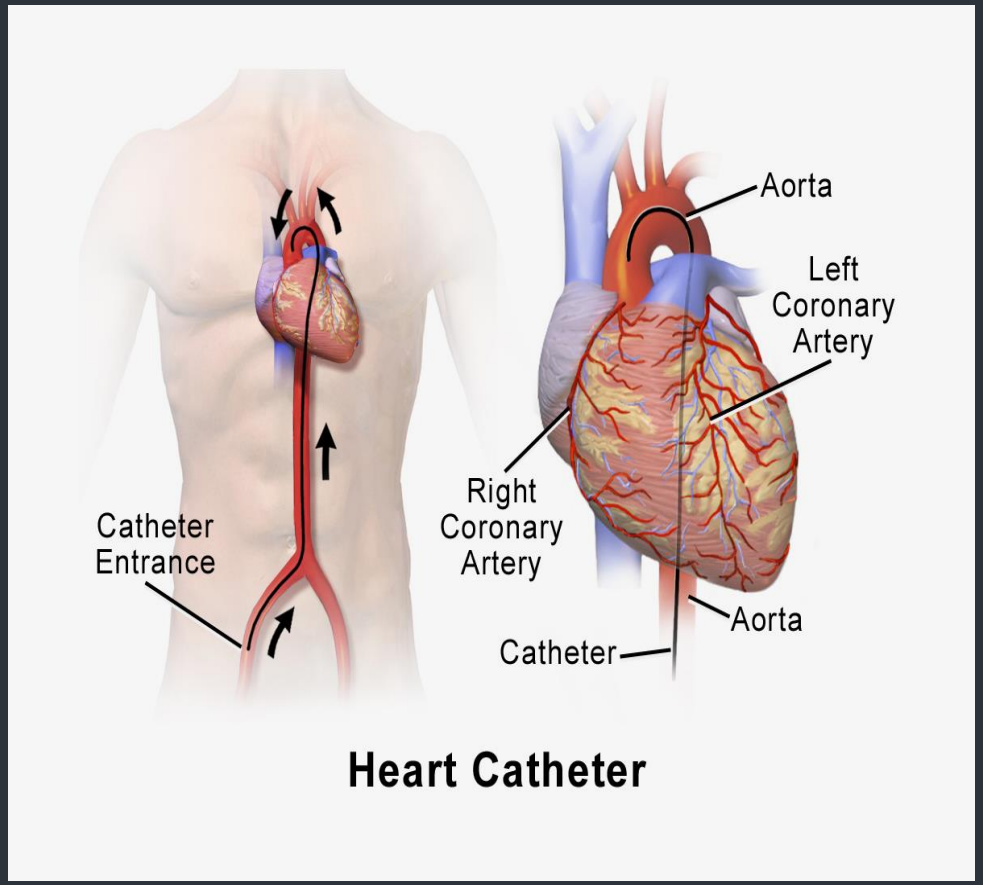


Nöromuskuler ajanların kullanımı

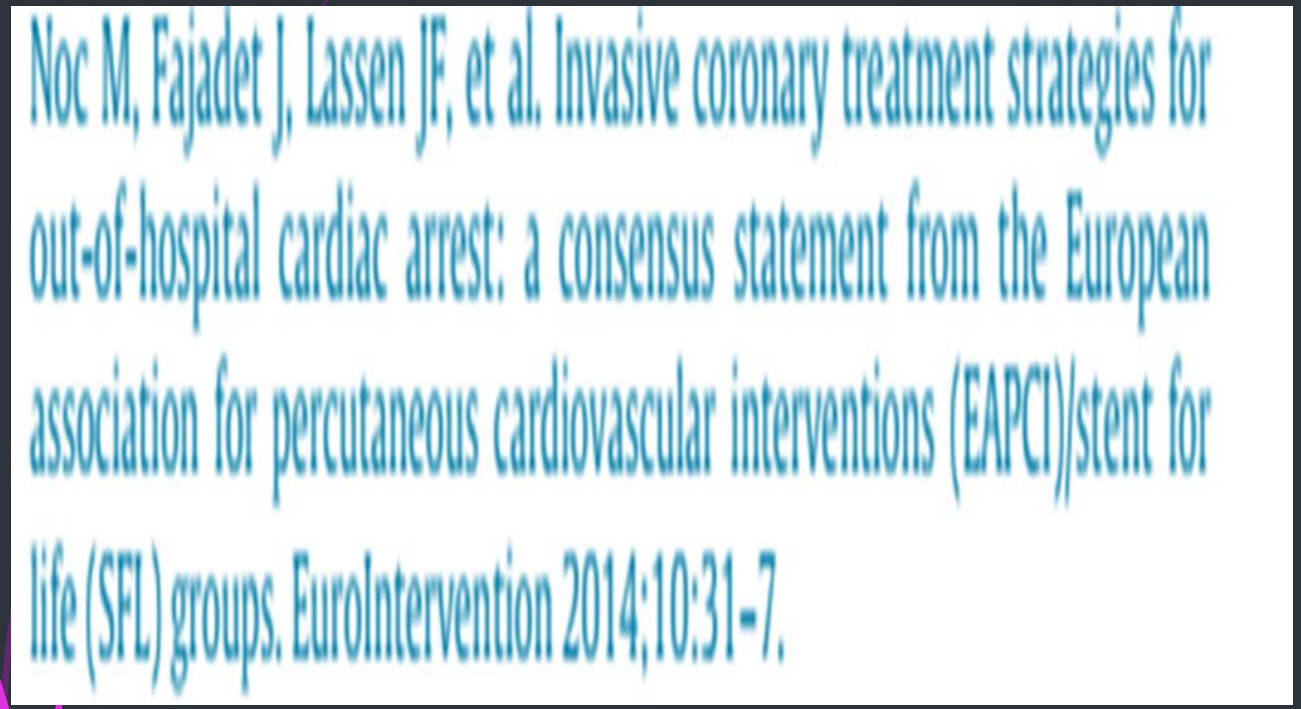
24 SAAT NÖROMUSKULER AJANLARLA VENTİLASYON DESTEĞİ VERİLEN HASTALARDA SAĞKALIM ORANI(%78/%41) VE LAKTAT KLİRENSİNDE ANLAMLI OLARAK HIZLI DÜZELME GÖRÜLMÜŞTÜR

Dolaşım

- Koroner dolaşım
- STEMI-LBBB 80% akut koroner lezyon tespit edilmiş
- Kanıt düzeyi düşük gözlemsel çalışmalarla desteklenmiştir.



Non - STEMI



- Yaş
- CPR süresi
- Hemodinamik instabilite
- Geliş kalp ritmi
- Hastaneye başvuru esnasında nörolojik durumu
- Kardiyak etiyoloji olasılığı

PTCA AHA 2015

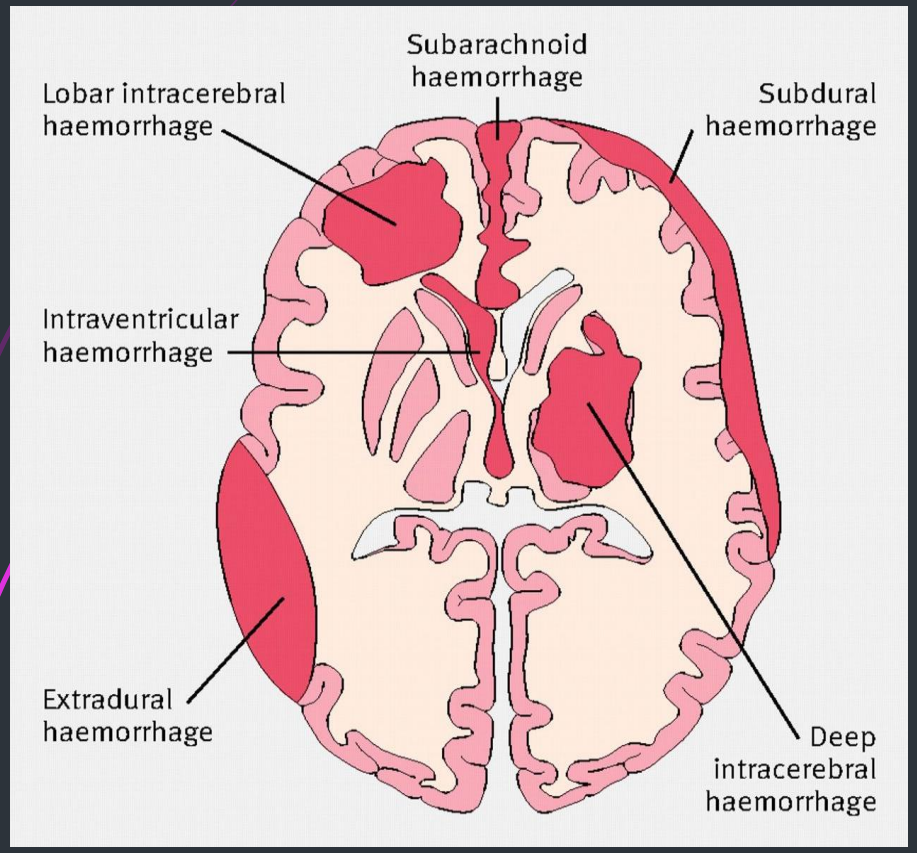
A 12-lead ECG should be obtained as soon as possible after ROSC to determine whether acute ST elevation is present. (Class I, LOE B)

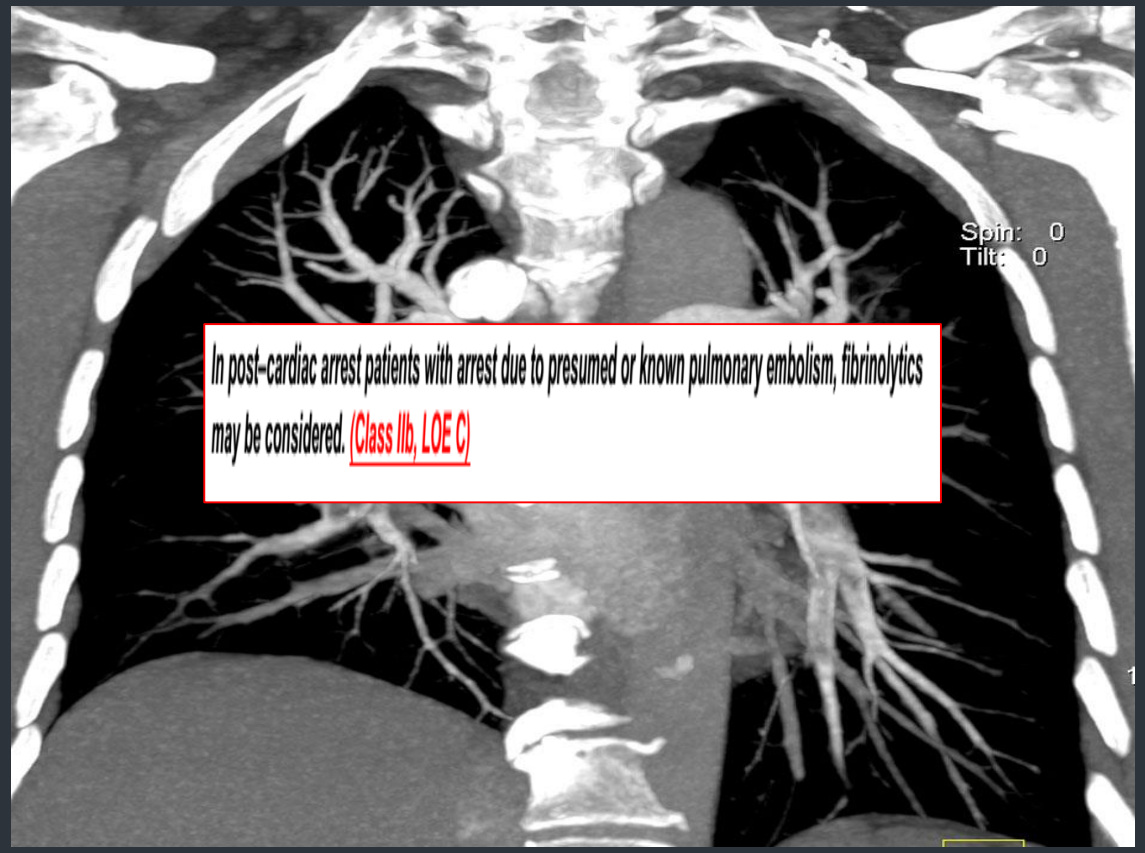
Coronary angiography should be performed emergently (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac etiology of arrest and ST elevation on ECG. (Class I, LOE B-NR)

Emergency coronary angiography is reasonable for select (eg, electrically or hemodynamically unstable) adult patients who are comatose after OHCA of suspected cardiac origin but without ST elevation on ECG. (Class IIa, LOE B-NR)

Coronary angiography is reasonable in post-cardiac arrest patients for whom coronary angiography is indicated regardless of whether the patient is comatose or awake. (Class IIa, LOE C-LD)

Tomografik görüntüleme

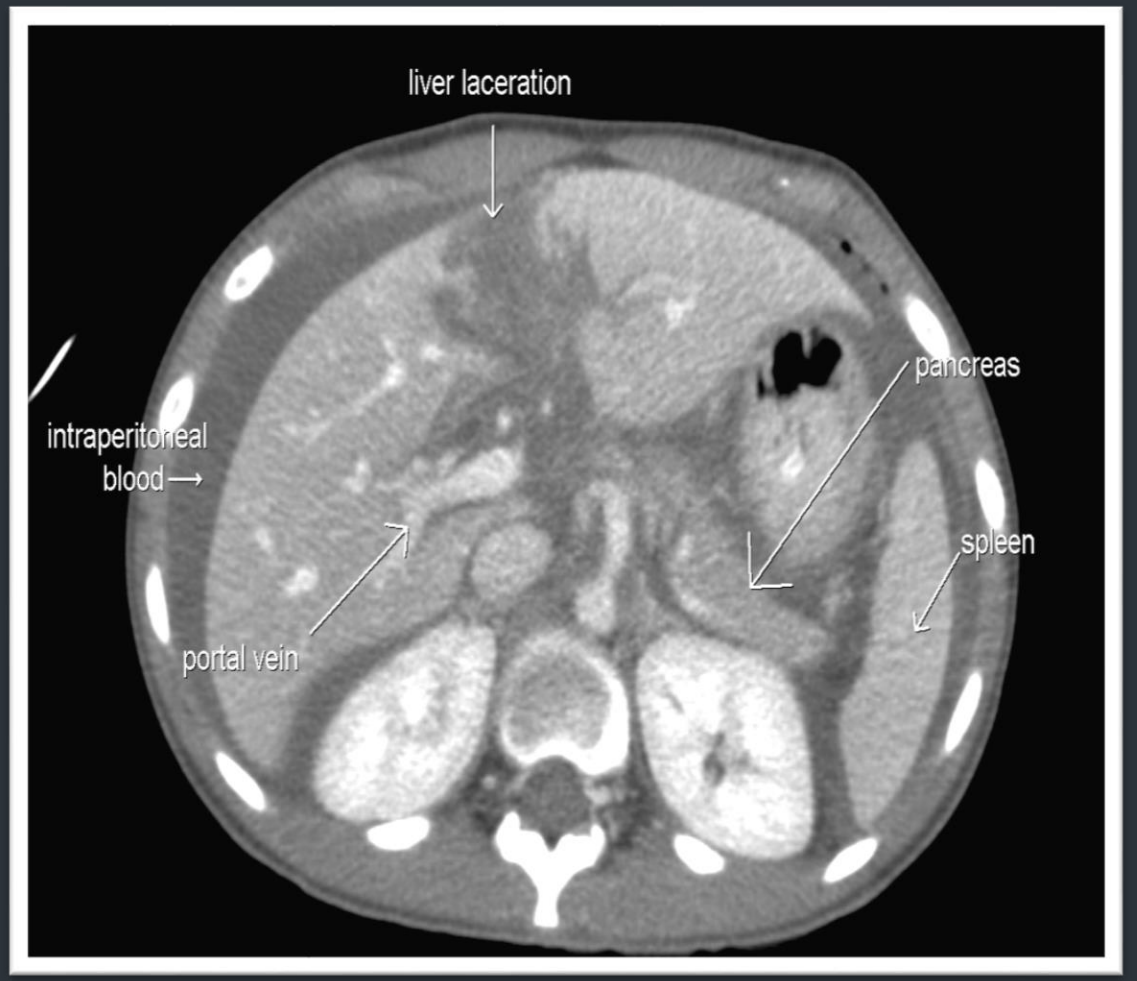




Kardiyak nedenler ön planda düşünülmüyorsa

Abdominal görüntüleme

- Laktat >15 mmol/L
- Travma öyküsü
- Peritonit bulgusu



Hemodinamik stabilizasyon

Hedef kan basıncı ne olmalı ??

Neurologic Critical Care

Arterial Blood Pressure and Neurologic Outcome After Resuscitation From Cardiac Arrest*

J. Hope Kilgannon, MD[1]; Brian W. Roberts, MD[1]; Alan E. Jones, MD[2]; Neil Mittal, MD[1]; Evan Cohen, MD[1]; Jessica Mitchell, MD[1,3]; Michael E. Chansky, MD[1]; Stephen Trzeciak, MD, MPH[1,3]

70 mm Hg. This threshold (mean arterial pressure > 70 mm Hg) had the strongest association with good neurologic outcome (odds ratio, 4.11; 95% CI, 1.34–12.66; $p = 0.014$). A sustained intrinsic hypertensive surge was relatively uncommon and was not associated with neurologic outcome.

Conclusions: We found that time-weighted average mean arterial pressure was associated with good neurologic outcome at a threshold of mean arterial pressure greater than 70 mm Hg. (*Crit Care Med* 2014; 42:2083–2091)

AHA 2015

Avoiding and immediately correcting hypotension (systolic blood pressure less than 90 mm Hg, MAP less than 65 mm Hg) during postresuscitation care may be reasonable. (Class IIb, LOE C-LD)

Dolaşım parametereleri

2015 AHA

- İdrar çıkışı
- Santral venöz O2

ERC 2015

- MAP??
- İdrar çıkışı;1ml/kh/saat
- K+ 4-4.5mmol
- Adrenal yetmezlik desteği??
- ICD

Beyin dokusunun korunması

- Hemodinamik olarak
- Sedasyon
- Konvülziyonlar ve tedavisi

Cerebral perfüzyon

Beyin ödemi → Bozulmuş beyin perfüzyonu

Arrest öncesi OAB

Beyin Perfüzyonu İçin Sedasyon

Yararları

- Hedef hipotermi sıcaklığına daha erken ulaşılır
- Oksijen tüketimini azaltır

- TTH
- Ajan??
- Benzodiazepin grubu
- Opioidler
  - Propofol
  - Alfentanil
  - Remifentanil

It is reasonable to consider the titrated use of sedation and analgesia in critically ill patients who require mechanical ventilation or shivering suppression during induced hypothermia after cardiac arrest.
(Class IIb, LOE C)

Konvülziyonların kontrolü

- İlk nöbette nedenleri ekarte edin
- Myoklonik kasılmalar
- Nöromuskuler blokör ajan kullanımı
- EEG takibinin olmaması

- Sodyum valproat
- Levetiracetam
- Fenitoin
- Benzodiazepinler
- Propofol
- Barbiturat
- İlaç karşılaştırması yok

Glukoz düzeyinin kontrolü

ERC 2015

- Sıkı kan şekeri kontrolü(72-108mg/dl)
- Esnek kan şekeri kontrolü(<180mg/dl)

Hipotermi

- Global cerebral hipoksi-iskemi sonrası nöroprotektiftir
- Her 1◦C cerebral oksijen tüketimini %6 azaltır
- Eksitatör amino asit salınımını azaltır
- Serbest radikallerin salınımı azalır
- 32-36

Resuscitation 85 (2014) 1480–1487

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

The inflammatory response after out-of-hospital cardiac arrest is not modified by targeted temperature management at 33 °C or 36 °C☆

John Bro-Jeppesen[a,*], Jesper Kjaergaard[a], Michael Wanscher[b], Niklas Nielsen[c], Hans Friberg[d], Mette Bjerre[e], Christian Hassager[a]

Level of TTM did not modify level of the inflammatory markers IL-1β, IL-6, TNF-α, IL-4, IL-10, CRP and PCT, (p = NS for each inflammatory marker).

Conclusions: Level of inflammatory response was associated with severity of PCAS with IL-6 being consistently and more strongly associated with severity of PCAS than the inflammatory markers CRP and PCT. The systemic inflammatory response after CA was not modified by TTM at 33 °C or 36 °C.

Hedeflenmiş sıcaklık ile hipotermi tedavisi(TTM) kimlere uygulanmalı

- Hastane dışı şoklanabilir kardiyak arrest sonrası bilinci kapalı hastalarda (güçlü öneri-kanıt düzeyi düşük)
- Hastane dışı şoklanamayan kardiyak arrest sonrası bilinci kapalı hastalarda(zayıf öneri- çok düşük kanıt düzeyi)
- Hastane içi kardiyak arrest sonrası bilinci kapalı kalanlarda(zayıf öneri-çok düşük kanıt düzeyi)
- Tedaviye başlananlarda 24 saat hedef sıcaklıkta tutulmalıdır(zayıf öneri-çok düşük kanıt düzeyi)

Hipotermide fizyolojik değişiklikler ve yan etkiler

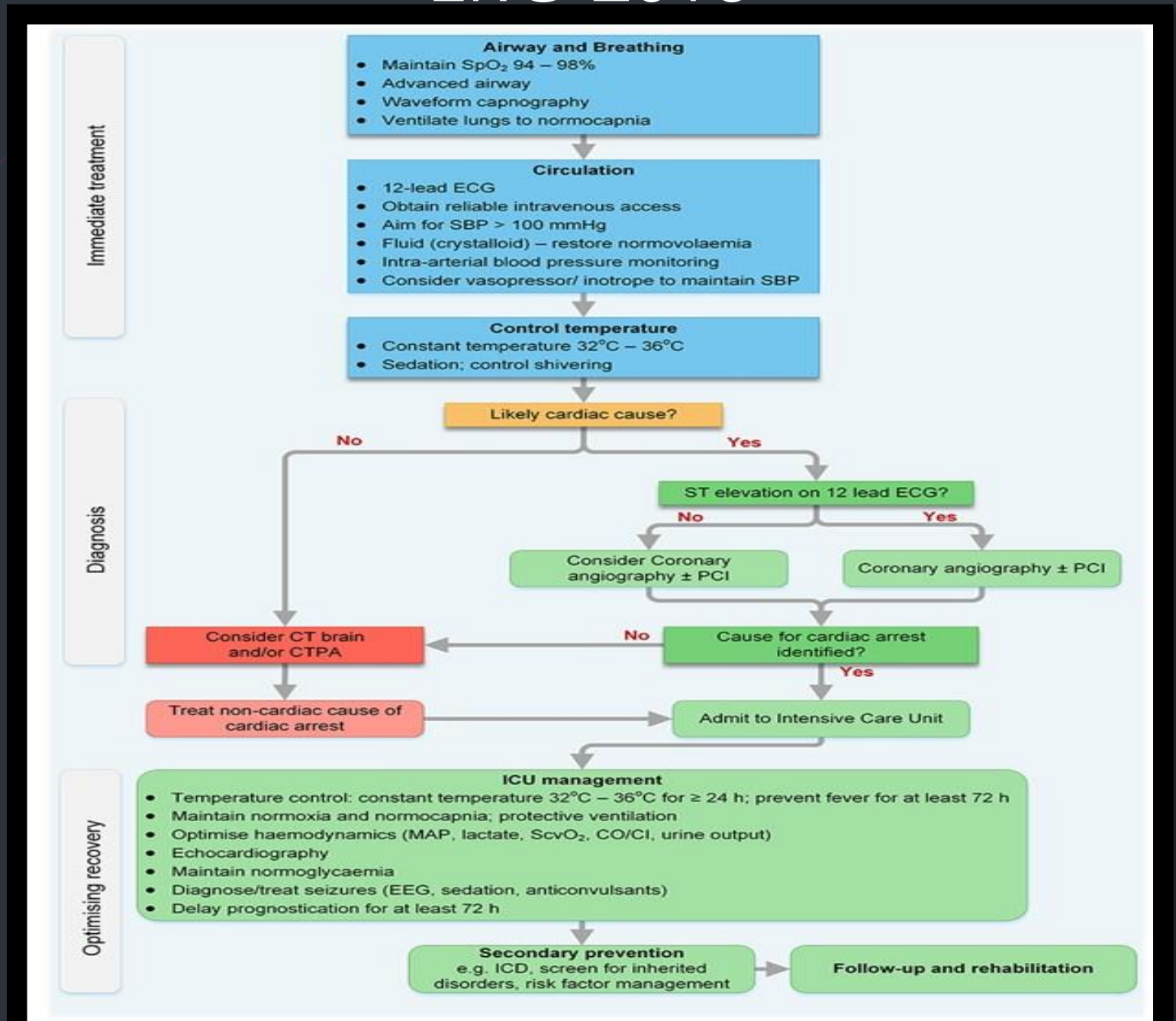
- Titreme ısı artışına neden olabilir
- Bradikardi
- Diüretik etki ve elektrolit bozukluğu
  - Hipofosfatemi
  - Hipokalemi
  - Hipokalsemi
  - Hipomagnezemi
- Hiperglisemi
- Koagulasyon bozukluğu
- İmmün sistemi baskılar ve enfeksiyonu arttırabilir
- Sedatif ve nöromuskuler ajanların eliminasyon hızı %30 azalır

AHA 2015

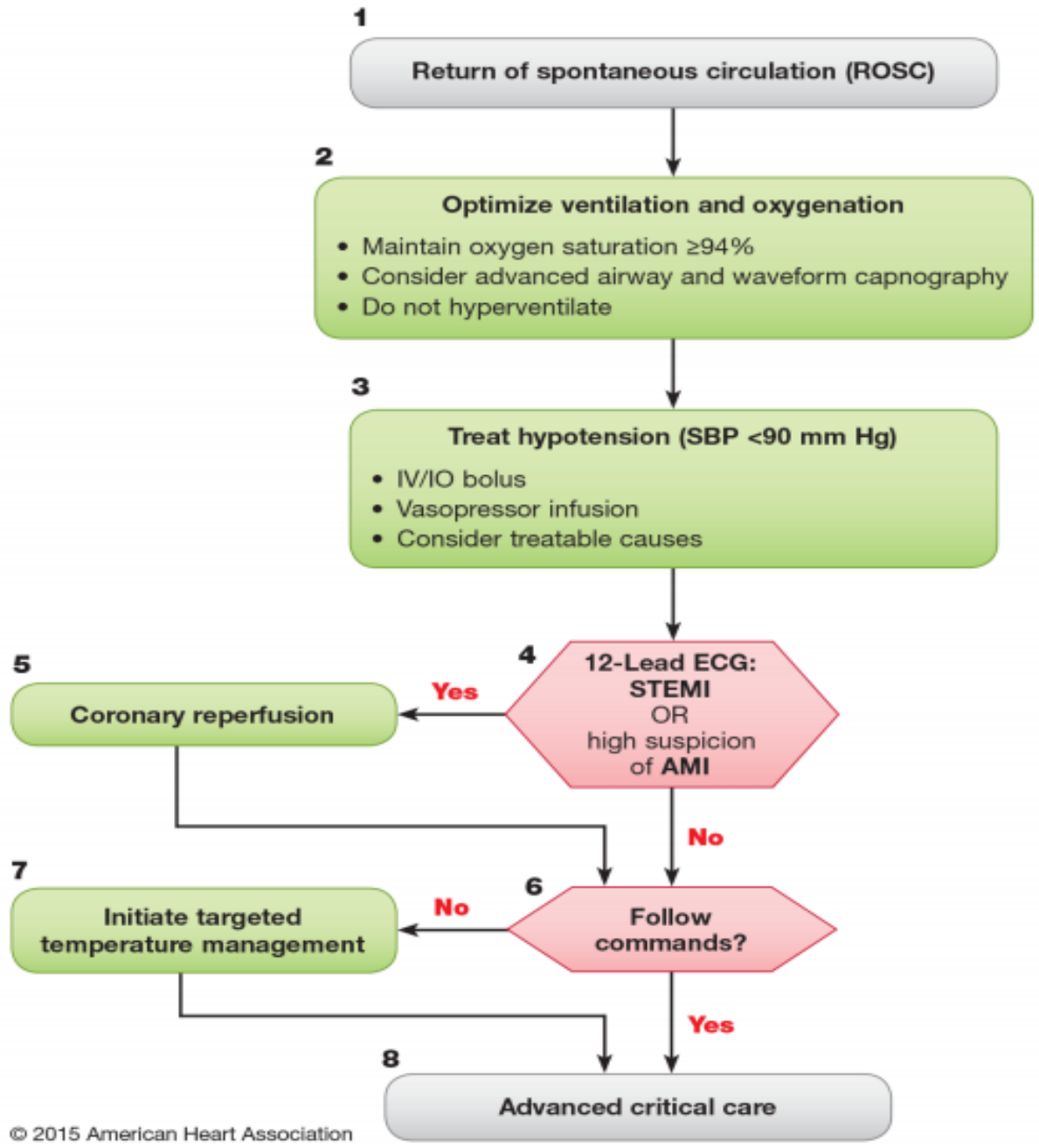
We recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest have TTM (Class I, LOE B-R for VF/pVT OHCA; for non-VF/pVT (ie, "nonshockable") and in-hospital cardiac arrest). (Class I, LOE C-EO)

We recommend selecting and maintaining a constant temperature between 32°C and 36°C during TTM. (Class I, LOE B-R)

ERC 2015


Immediate treatment
Airway and Breathing
Maintain SpO_2 94 – 98%
Advanced airway
Waveform capnography
Ventilate lungs to normocapnia
Circulation
12-lead ECG
Obtain reliable intravenous access
Aim for SBP > 100 mmHg
Fluid (crystalloid) – restore normovolaemia
Intra-arterial blood pressure monitoring
Consider vasopressor/ inotrope to maintain SBP
Control temperature
Constant temperature 32°C – 36°C
Sedation; control shivering
Diagnosis
Likely cardiac cause?
No
Yes
ST elevation on 12 lead ECG?
No
Yes
Consider Coronary angiography ± PCI
Coronary angiography ± PCI
Consider CT brain and/or CTPA
No
Cause for cardiac arrest identified?
Yes
Treat non-cardiac cause of cardiac arrest
Admit to Intensive Care Unit
Optimising recovery
ICU management
Temperature control: constant temperature 32°C – 36°C for ≥ 24 h; prevent fever for at least 72 h
Maintain normoxia and normocapnia; protective ventilation
Optimise haemodynamics (MAP, lactate, $ScvO_2$, CO/CI, urine output)
Echocardiography
Maintain normoglycaemia
Diagnose/treat seizures (EEG, sedation, anticonvulsants)
Delay prognostication for at least 72 h
Secondary prevention
e.g. ICD, screen for inherited disorders, risk factor management
Follow-up and rehabilitation

Adult Immediate Post–Cardiac Arrest Care Algorithm—2015 Update



Doses/Details

Ventilation/oxygenation:
Avoid excessive ventilation. Start at 10 breaths/min and titrate to target P_{ETCO_2} of 35-40 mm Hg.
When feasible, titrate F_{IO_2} to minimum necessary to achieve Spo_2 ≥94%.

IV bolus:
Approximately 1-2 L normal saline or lactated Ringer's

Epinephrine IV infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Dopamine IV infusion:
5-10 mcg/kg per minute

Norepinephrine IV infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmona
- **T**hrombosis, corona