Post-resuscitation Care

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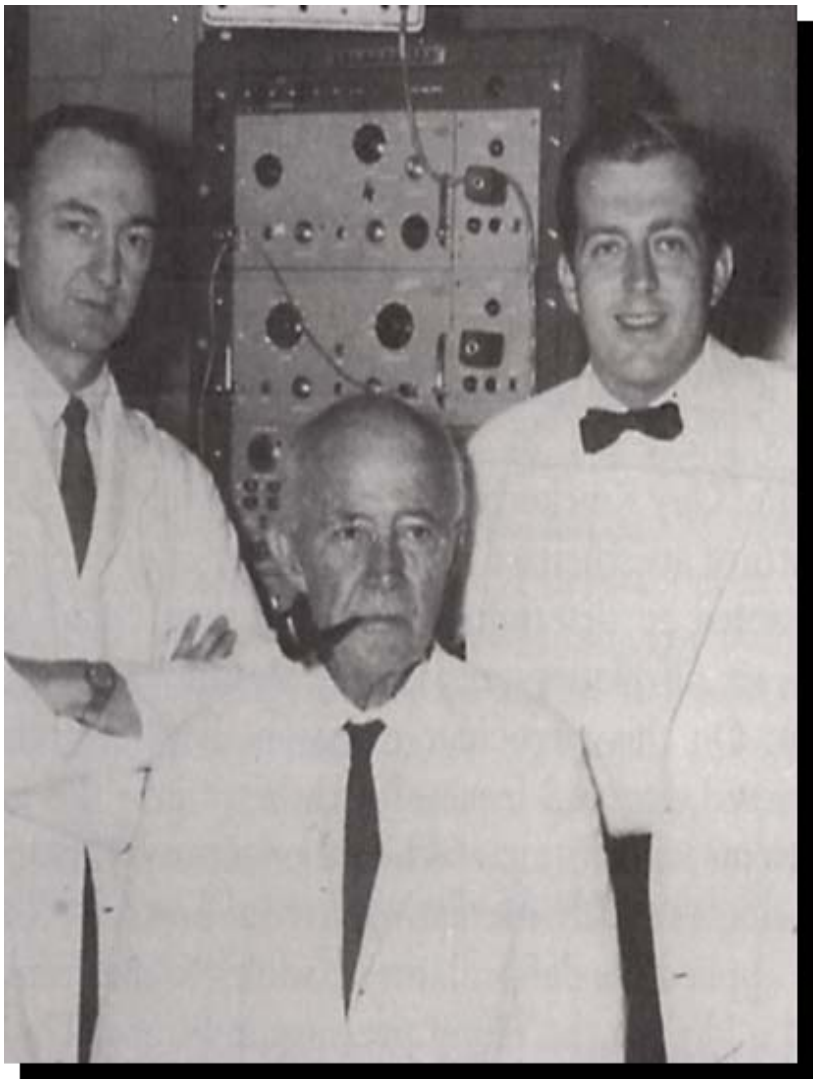
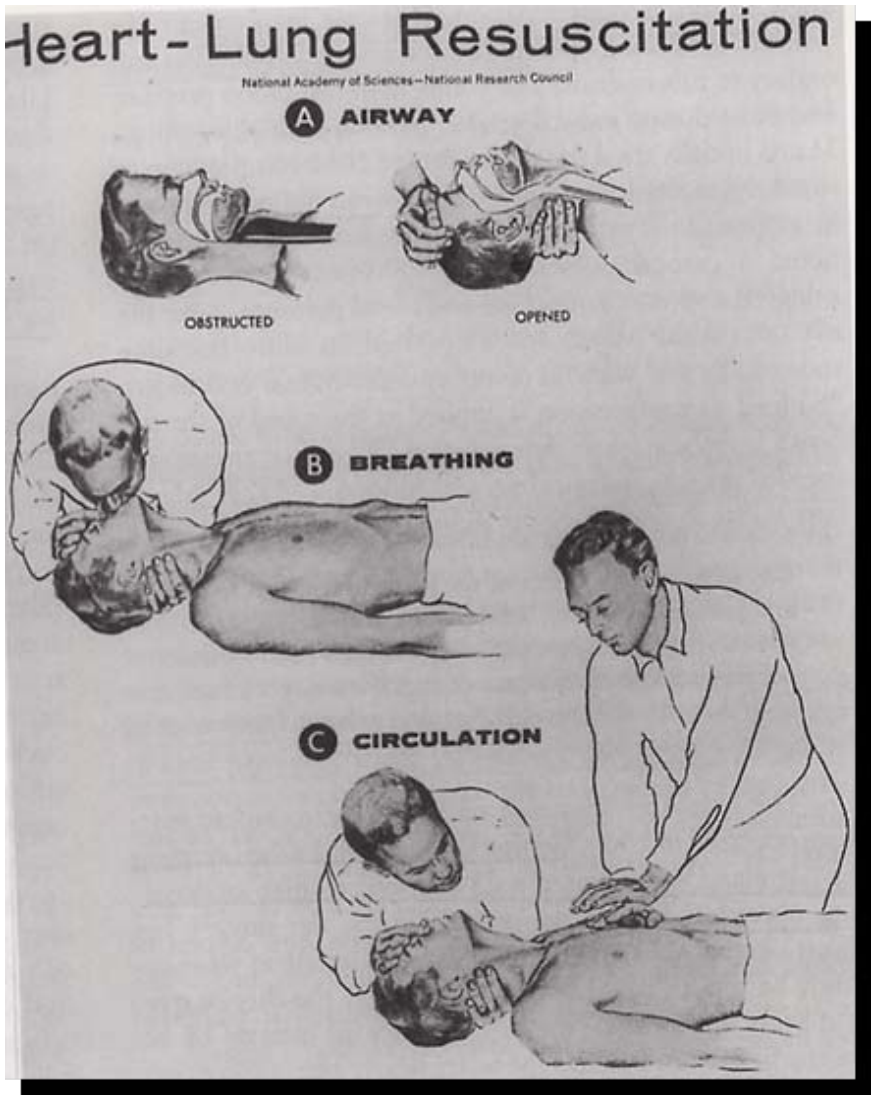




Dr.James R Jude, Dr. William Kouwenhoven, Dr.G Guy Knickerbocker at Johns Hopkins Hospital, 1961

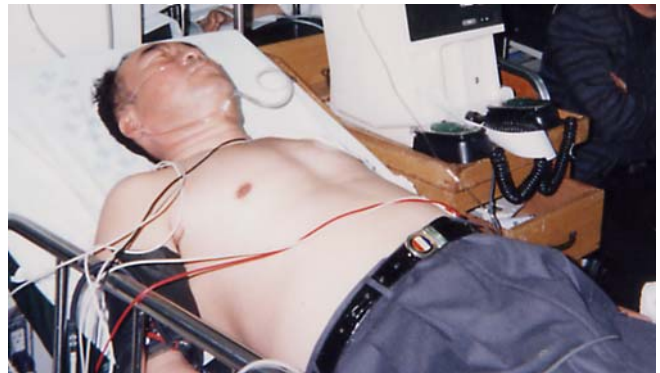
소방 119 구급대

대한응급의학회

대한순환기학회

대한심폐소생협회

BLS, ACLS Courses



Postresuscitation Coma

ROSC : 25-50% of Attempted CPR

80% of ROSC pts : initially Coma

Survival rate:18 % (6 M)

Good outcome:13 %(6 M)

By BRCT II

I/R Injury mechanism

Treatment

Outcome evaluation

Reperfusion injury

Fever

Hypotension

Hypoxia

Seizure

Glucose(hypo, Hyper)

Acid-base

Electrolytes

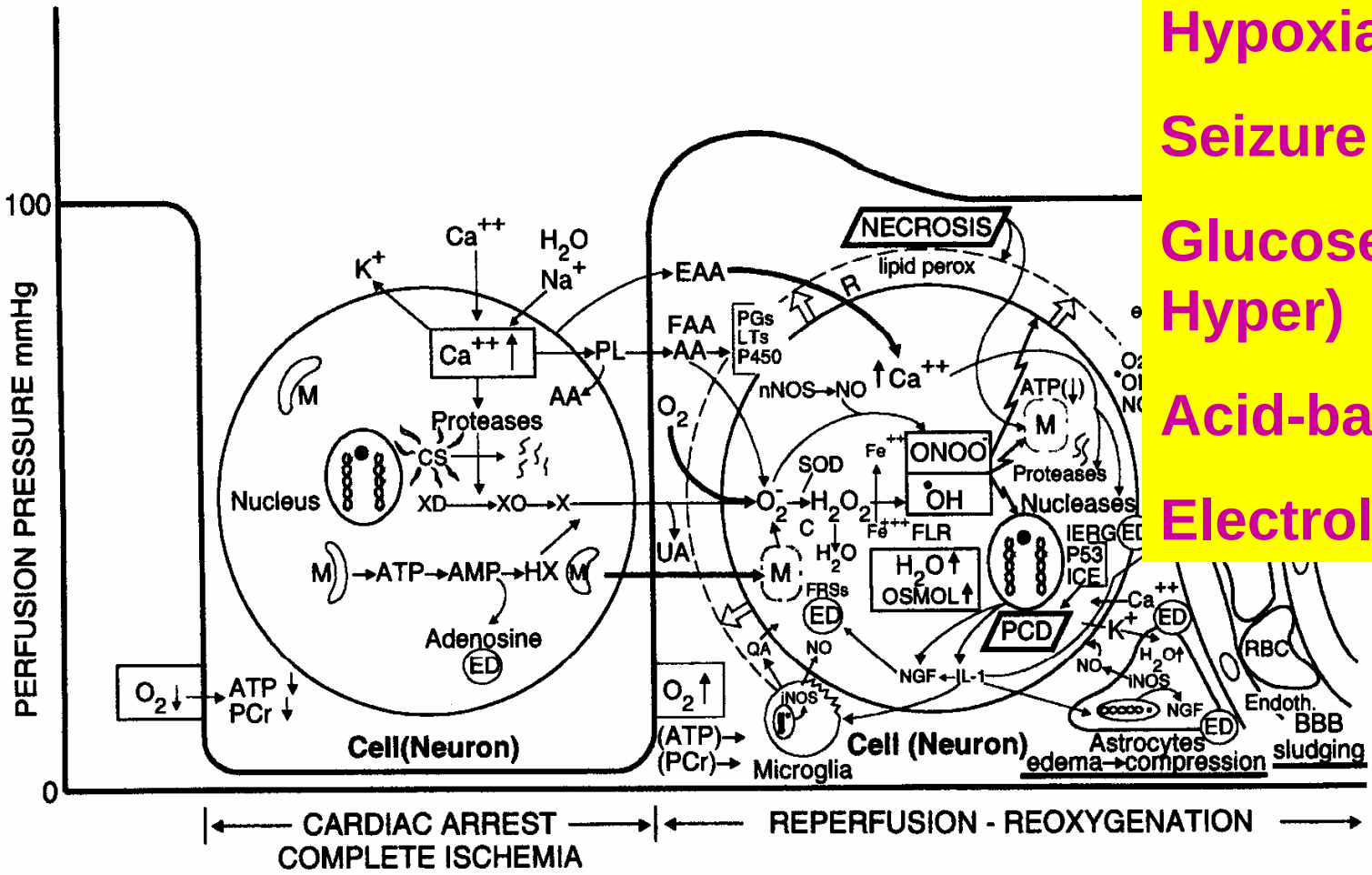


Figure 49.2. For legend see opposite page.

- Most neurons tolerate up to ~15 min of normothermic ischemia *in vivo*

– Key Points –

- Identify and treat precipitating causes of the arrest (6H's and 5T's factors)
- Support care (Critical Care) – V/S, electrolyte, acid-base, seizure, glucose etc, at least 24hrs
- Therapeutic Hypothermia
- Outcome evaluation – SEP, N/Ex

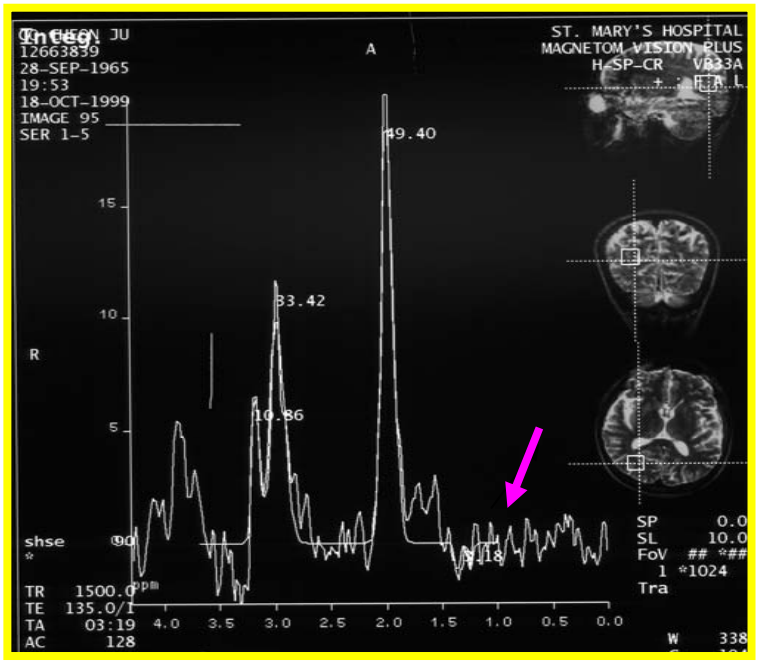
C A S E 1

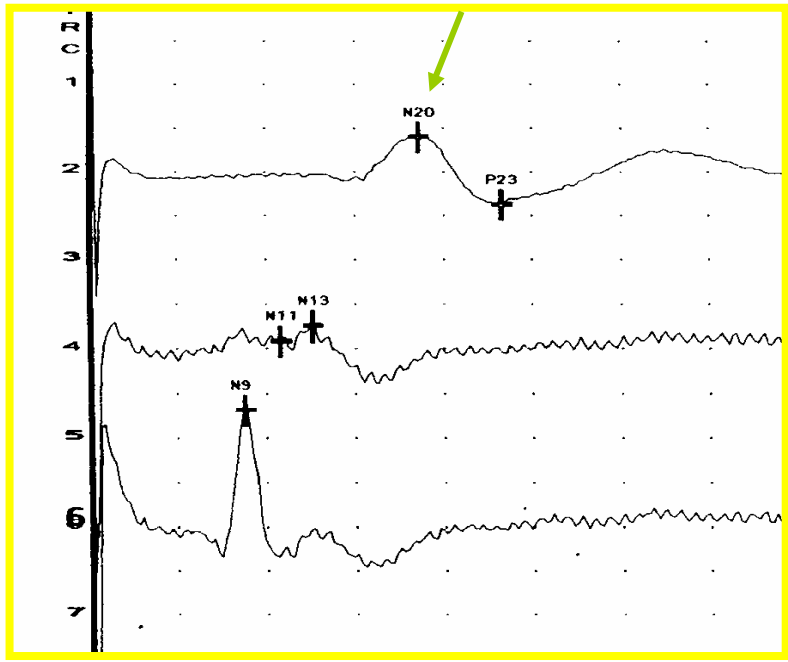
- M/34 고O주
- Near drowning, Arrest T:25min, Asystole,

  initial BT: 28°C

  CPR T:15min, bystander CPR(-), 119 no CPR
  Brain stem reflex(CR: 6hr)

  1D:GCS=3
  3D:GCS=5
  12D:GCS=15,
  Posthypoxic Myoclonus(Lance Adams)
  SEP,MRS,Echo=O.K





MRS : lactate(-) at 1.3ppm,

At 2D after ROSC

SSEP : N20 (+),

At 30hrs after ROSC

의식 회복후 상태

Lance-Adams syndrome(posthypoxic myoclonus)

Temperature Regulation

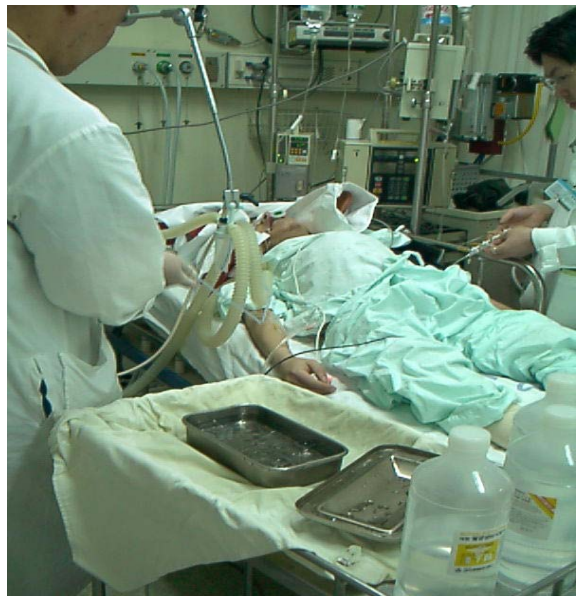
Hyperthermia

- 40 pts,PRS (Resuscitation 2001;49:273)
  - Peak axillary Temp. Above 39 °C within initial 72hrs

    all 20pts : dead(vs 3 of 20 pts, less than 39 °C)
- Either frequent use of antipyretics or “controlled normothermia” with cooling techniques
- Monitor the patient’s temperature after resuscitation and avoid hyperthermia

Induced Hypothermia

- Unconscious adult patients with ROSC after OHCA should be cooled to 32˚C to 34˚C for 12 to 24 hours when the initial rhythm was VF (Class IIa).
- With non-VF arrest out of hospital or for in-hospital arrest (Class IIb).



Bernard SA, NEJM 2002; 346:557-63
HACA Study Group, NEJM 2002; 346:549-56

Australian Study (Bernard SA)

Good outcome

Hypo- vs Normothermia

(21/43 vs 9/34,

49% vs 26%, **p=0.046**).

Mortality rate

Hypo- vs Normothermia

(22/43 vs 23/34,

51% vs 68%, **p=0.145**)

Primary causes of death

-Cardiac failure

-Brain death

-Severe neurologic injury

-Withdrawal of all active therapy

TABLE 5. OUTCOME OF PATIENTS AT DISCHARGE FROM THE HOSPITAL.

OUTCOME*	HYPOTHERMIA (N=43)	NORMOTHERMIA (N=34)
	number of patients	
Normal or minimal disability (able to care for self, discharged directly to home)	15	7
Moderate disability (discharged to a rehabilitation facility)	6	2
Severe disability, awake but completely dependent (discharged to a long-term nursing facility)	0	1
Severe disability, unconscious (discharged to a long-term nursing facility)	0	1
Death	22	23

*The difference between the rates of a good outcome (normal or with minimal or moderate disability) in the hypothermia and the normothermia groups (49 percent and 26 percent, respectively) was 23 percentage points (95 percent confidence interval, 13 to 43 percentage points; P=0.046). The unadjusted odds ratio for a good outcome in the hypothermia group as compared with the normothermia group was 2.65 (95 percent confidence interval, 1.02 to 6.88; P=0.046). The odds ratio for a good outcome in the hypothermia group as compared with the normothermia group, after adjustment by logistic regression for age and time from collapse to return of spontaneous circulation, was 5.25 (95 percent confidence interval, 1.47 to 18.76; P=0.011).

European Study(HACA Study Group)

TABLE 2. NEUROLOGIC OUTCOME AND MORTALITY AT SIX MONTHS.

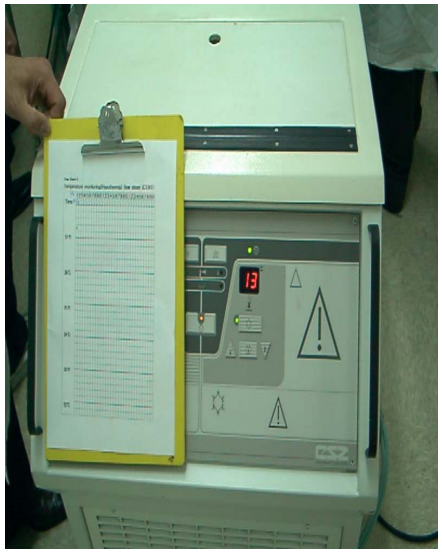
OUTCOME	NORMOTHERMIA	HYPOTHERMIA	RISK RATIO (95% CI)*	P VALUE†
	no./total no. (%)			
Favorable neurologic outcome‡	54/137 (39)	75/136 (55)	1.40 (1.08–1.81)	0.009
Death	76/138 (55)	56/137 (41)	0.74 (0.58–0.95)	0.02

*The risk ratio was calculated as the rate of a favorable neurologic outcome or the rate of death in the hypothermia group divided by the rate in the normothermia group. CI denotes confidence interval.

†Two-sided P values are based on Pearson's chi-square tests.

‡A favorable neurologic outcome was defined as a cerebral-performance category of 1 (good recovery) or 2 (moderate disability). One patient in the normothermia group and one in the hypothermia group were lost to neurologic follow-up.

Resuscitative hypothermia, ST.Mary's Hosp



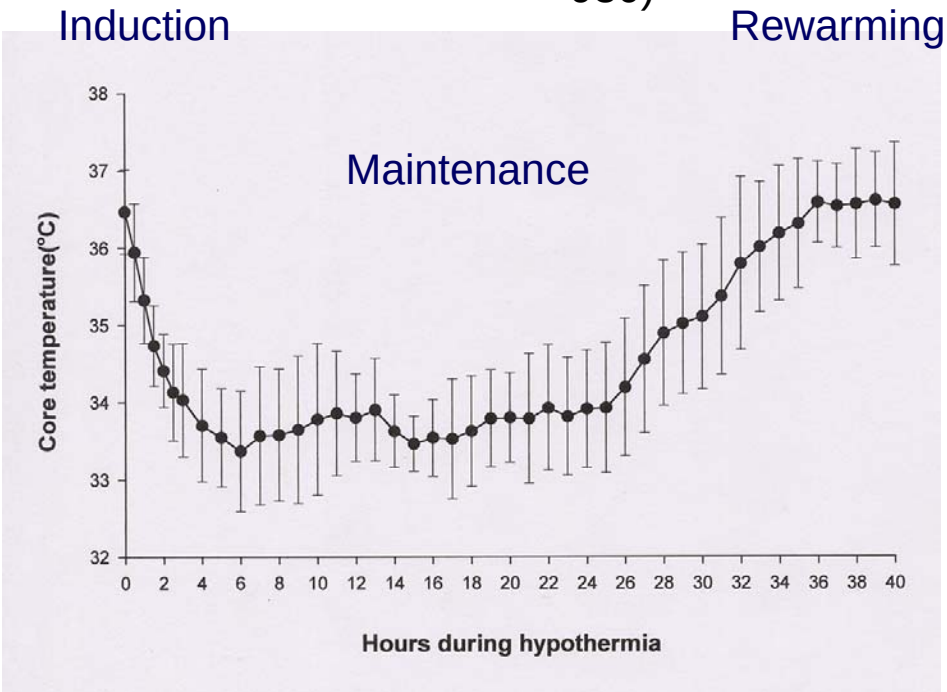

Results (1)

Patient (n=12)	Age (yr)	Arrest Time(min	ACLS Time(min	Initial Rhythm	Causes of cardiac arrest	Underlying Disease	**CPC**
1	60	10	15	PEA	R (Drug overdose)	Depression	1
2	19	25	10	Asystole	R (Drug overdose)	Depression	3
3	58	10	27	VF	C (AMI)		3
4	54	36	20	Asystole	R (Unknown)	HBP	1
5	37	24	4	PEA	R (Hanging)	Depression	5
6	51	10	10	Asystole	C (Dysrhythmia)		1
7	33	40	8	VF	C (AMI)	HBP,DM	5
8	57	2	8	Asystole	C (CHF)	HBP,DM	4
9	25	20	22	Asystole	C (dysrhythmia)		1
10	78	10	4	Asystole	R (Asphyxia)	HBP	4
11	49	30	10	Asystole	R (Asphyxia)	DM,CAD	4
12	55	25	5	Asystole	C (AMI)	CAD	5
SUM	486	200	127	VF (n=2)	C (cardiac,n=6)		Good (n=4)
AVR ± S.D	48 ±17	20 ± 12	12 ± 8	PEA (n=2) Asystole (n=8)	R (respiratory,n=6)		Poor (n=8)

2002, ACEP, oral presentation, Seatle, WA

Results (2)

Resuscitative Hypothermia	All Patients (n=12)	Outcome	
		Good (n=4)	Poor (n=8)
Initiation Temperature (℃)	36.5±0.5(35.8-37.5)	36.2±0.3(36-36.7)	36.6±0.6 (35.8-37.5)
Initiation Time (min)	129±113 (40-420)	188±159 (60-750)	99±78 (40-250)
Induction Time (min)	160±79 (80-330)	171±107 (100-685)	154±69 (80-300)
Rewarming Time (min)	605±190 (360-960)	603±284 (360-930)	606±106 (420-680)



European Study

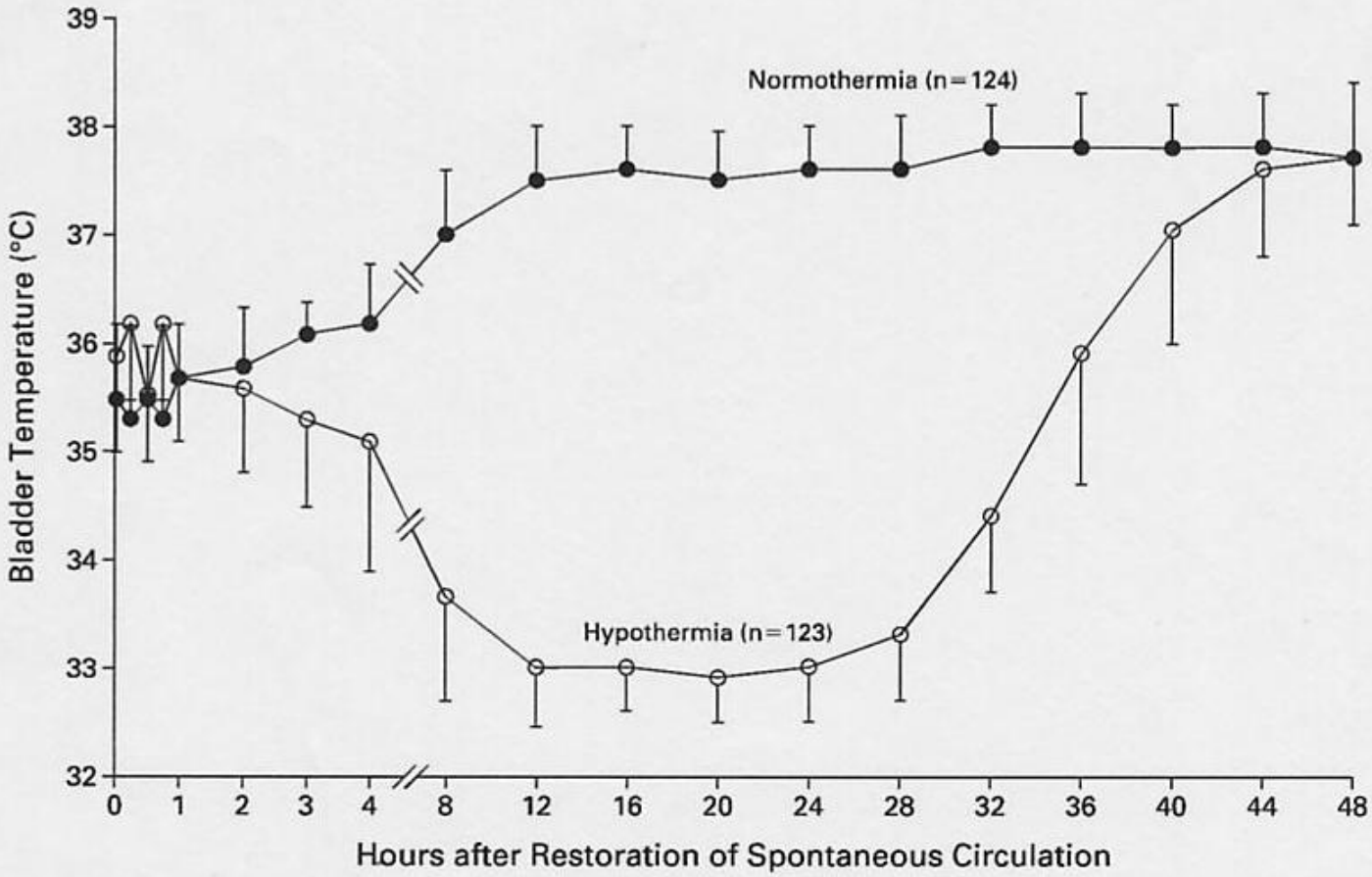
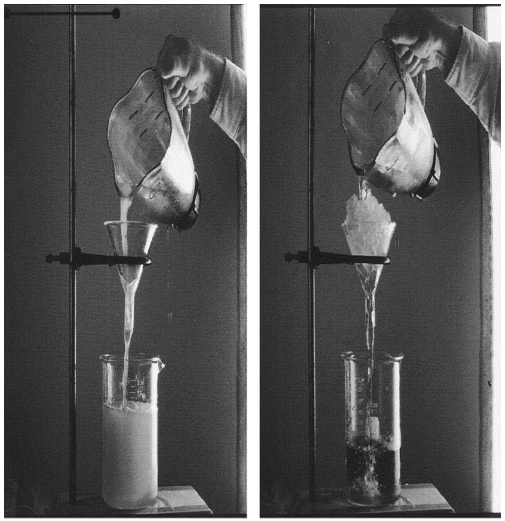


Figure 1. Bladder Temperature in the Normothermia and Hypothermia Groups.

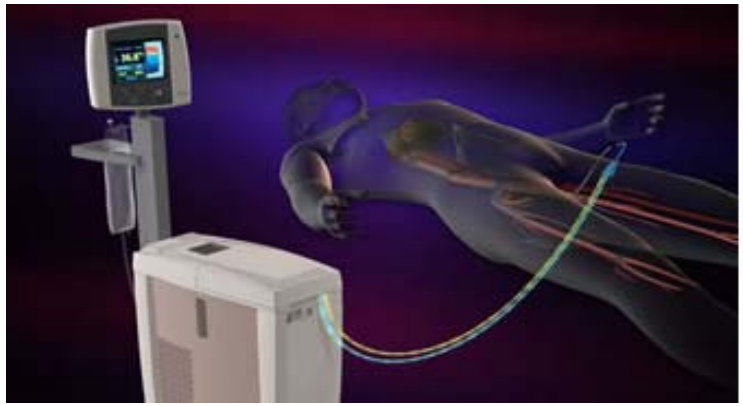
The T bars indicate the 75th percentile in the normothermia group and the 25th percentile in the hypothermia group. The target temperature in the hypothermia group was 32°C to 34°C, and the duration of cooling was 24 hours. Only patients with recorded temperatures were included in the analysis.

Internal cooling techniques



(cold saline techniques)

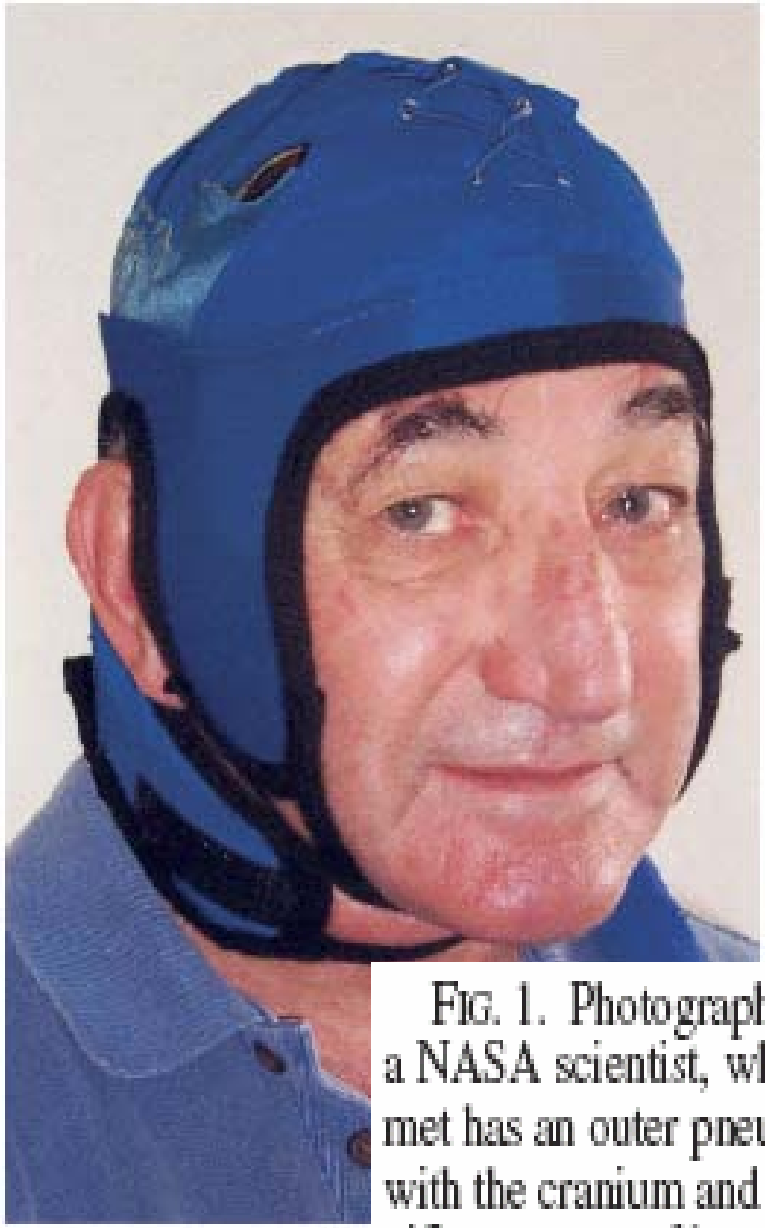
Ref.: Vanden hoek,TL,MD. CCM. volume 32(9) supplement Sep 2004;pps425



(Endovascular cooling catheter)

CoolGard 3000; Alsius

J Neurosurg 100:272, 2004



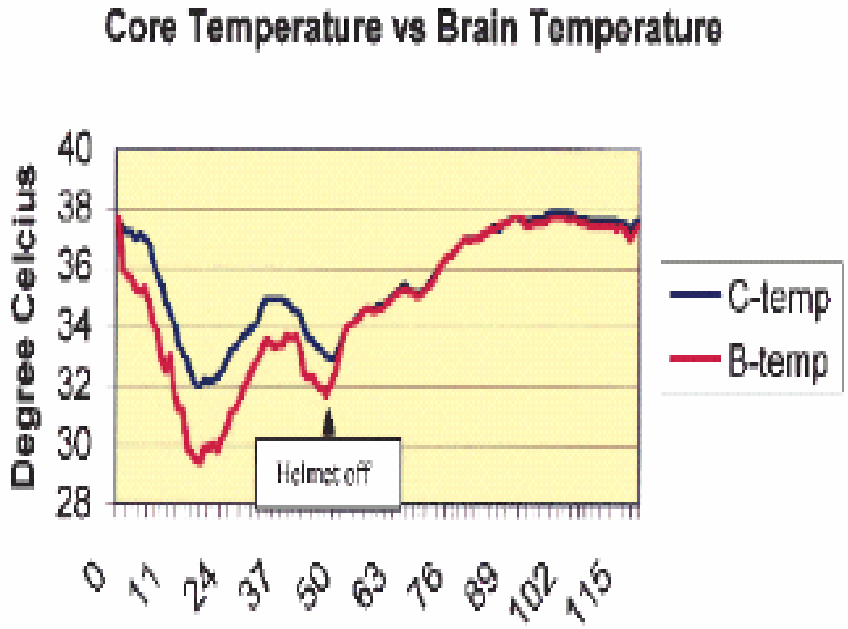


FIG. 1. Photograph showing the helmet worn by William Elkins, a NASA scientist, who invented this technology. The cooling helmet has an outer pneumatic liner pressurized to allow close contact with the cranium and neck. The device also is adjustable to fit a significant range of head sizes.

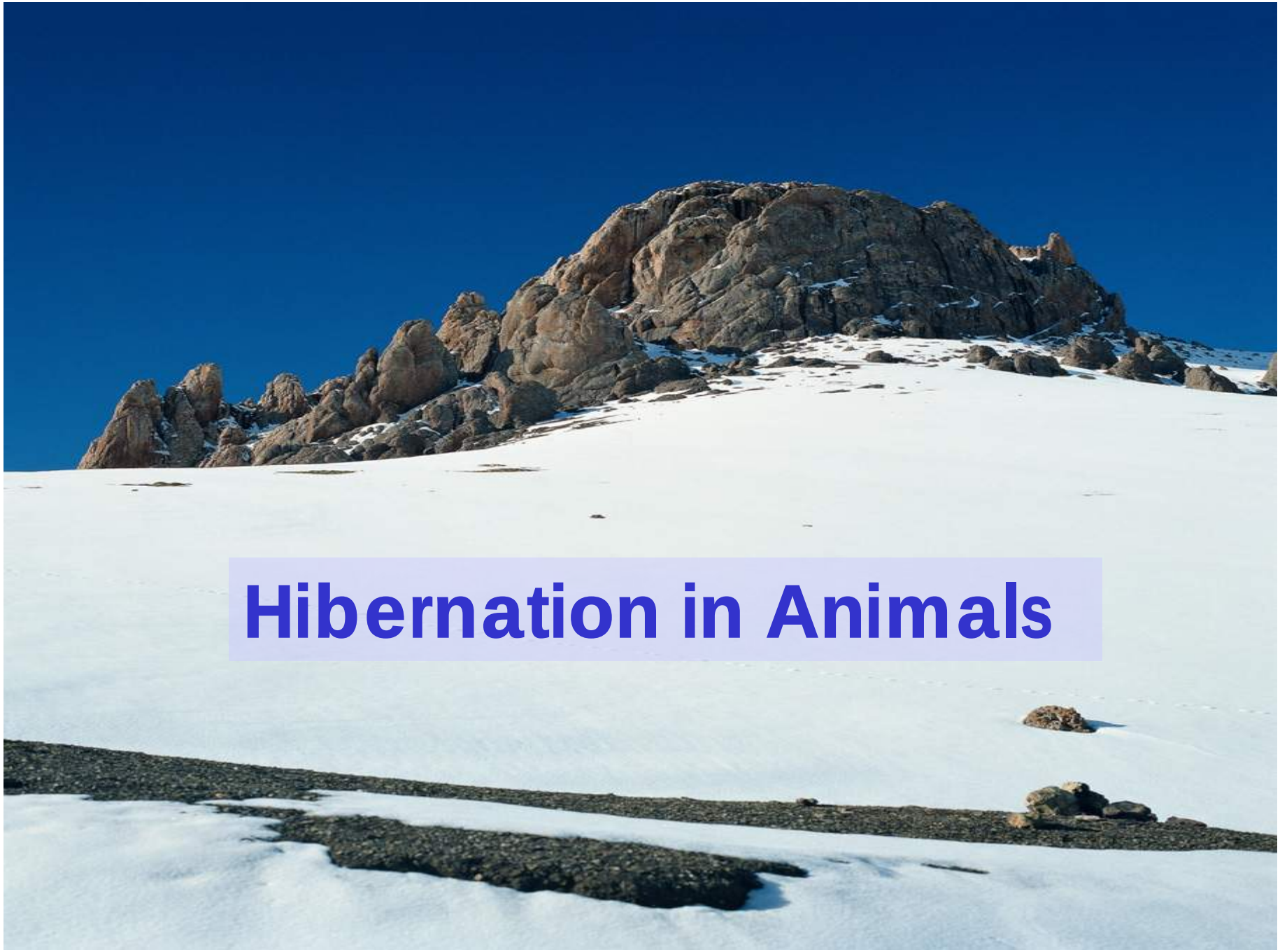
Regulated hypothermia

Forced hypothermia

- Physiological responses to maintain normothermia
  - Vigorous heat gain and conserving response (thermoregulatory system)
  - Blunt or delay achieving the hypothermic therapeutic target temp.
  - Create undue physiological and psychological stress (↑ shivering, increased catecholamine & cortisol)
  - Reduce the benefits of the hypothermic therapy.

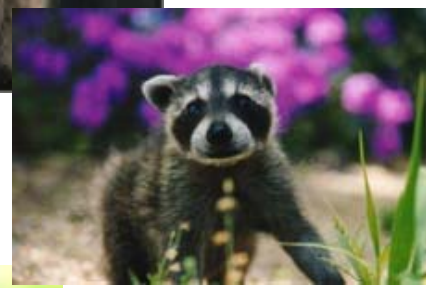
Regulated hypothermia

- Reduced body temp via a reduction in the set-point of thermoregulation
  - The theoretical advantage of mild hypothermia more quickly
  - Less stress (↓ shivering, increased catecholamine & cortisol)


Hibernation in Animals

- **Bats**
  - **normal heart beat :**
    400 /min
  - **during hibernation :**
    11-25 /min
  - **saves 99.3 % of their energy.**

- **Bears**
  - **survive six months without eating, drinking, urinating or defecating**
  - **The largest and most dramatic hibernators**
  - **during hibernation their body temperature does not drop despite slowing their breathing and heart rates.**
  - **so bears put on large fat reserves**

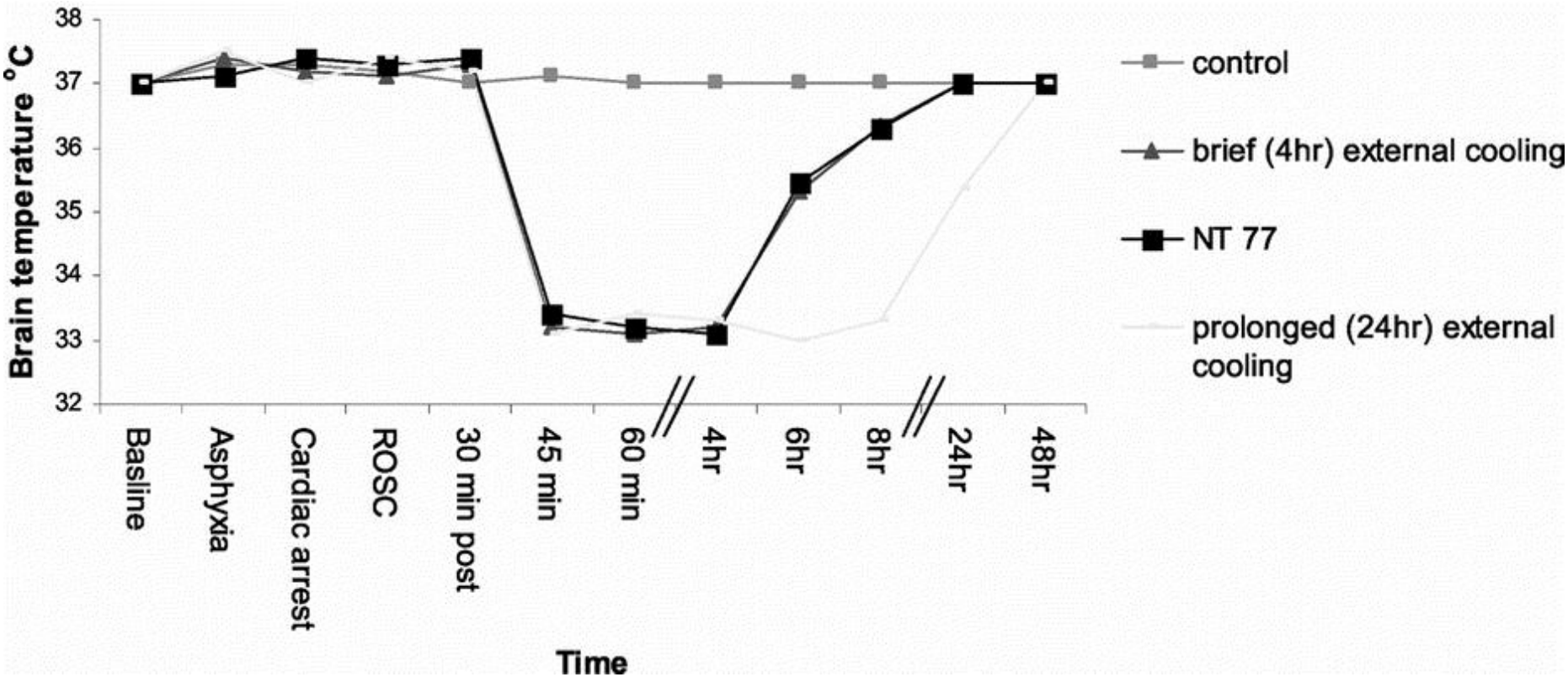


- Hibernation induction trigger, an 88 kd peptide :
  - in the serum of hibernating groud squirrels
  - increase survival time in a multiorgan preparation model with dogs (J thorac Cadiovascular Surg 1991; 102:224)
- D-Ala2, D-Leu5-enkephalin (DADLE) : extends hypothermic preservation time of the lung (J thorac Cadiovascular Surg 1996; 111:259)
- Neurotensin 77 : induce hibernation for several hrs in rats (CCM 2004;32:806)
- H_2S : reversible hibernation in mice by the way of inhibition oxidative phosphorylation (Science 2005;308:518)

Neurotensin

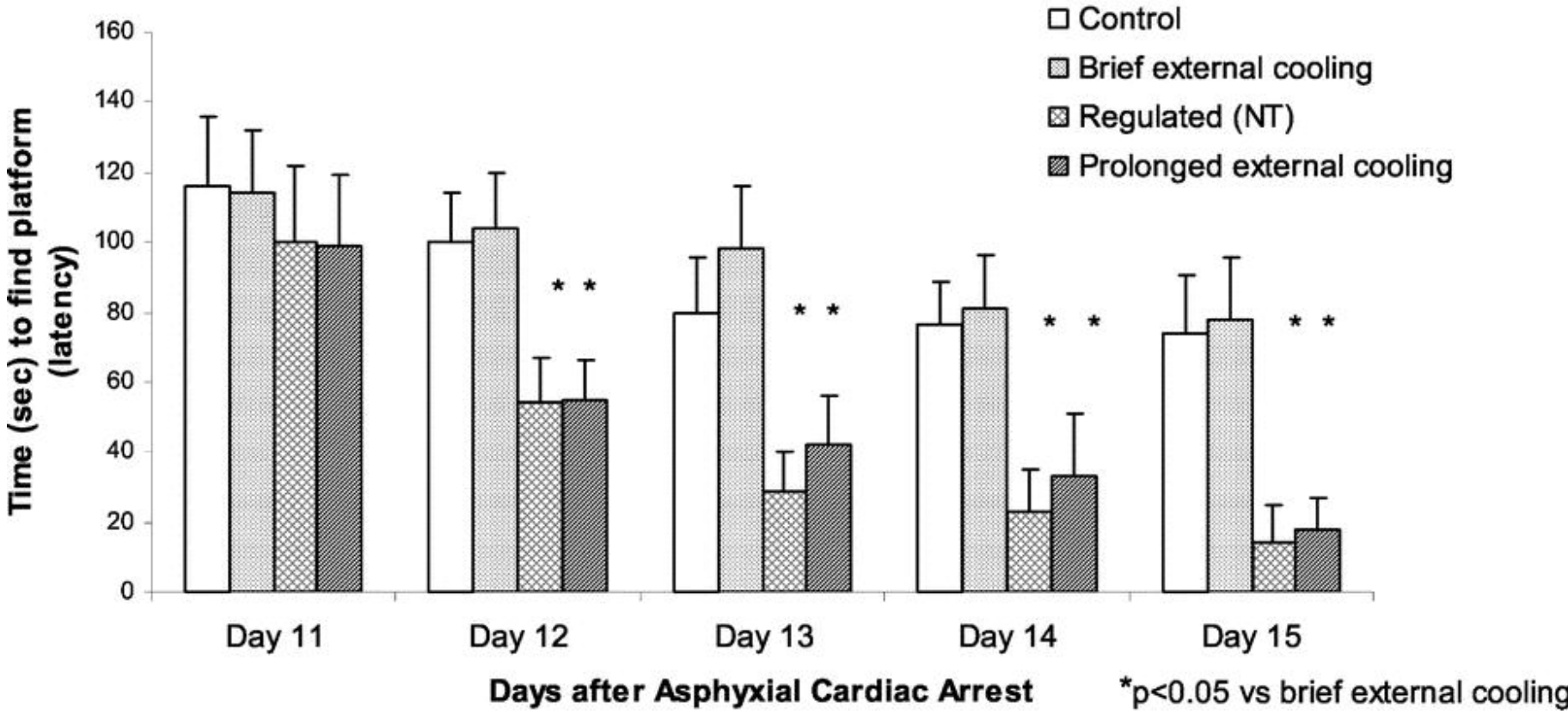
- An endogeneous tridecapeptide
- Elevation during hibernation (Comp biochem physiol C 1983)
- Induces hypothermia by activation of neurotensin receptors in the brain
- Normally degraded rapidly by circulating peptidases found in the blood

Figure 1. Brain temperature was measured telemetrically and recorded during surgical preparation and for 24 hrs after reperfusion from asphyxial cardiac arrest



Neurotensin-induced hypothermia improves neurologic outcome after hypoxic-ischemia
Critical Care Medicine. 32(3):806-810, March 2004

Figure 3. Performance (latency time) in the Morris maze 11–15 days after reperfusion from asphyxial cardiac arrest in rats



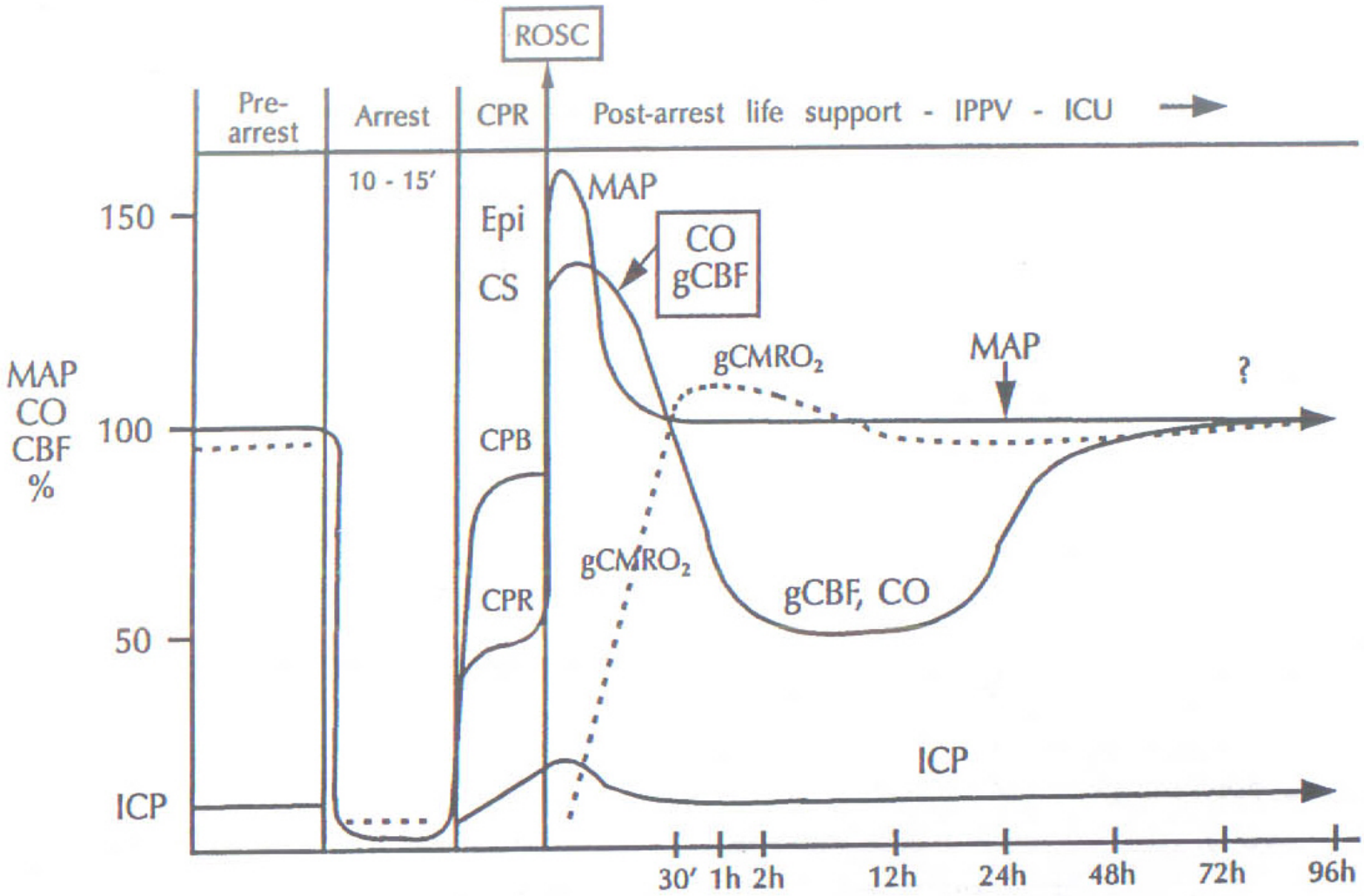
Neurotensin-induced hypothermia improves neurologic outcome after hypoxic-ischemia
Critical Care Medicine. 32(3):806-810, March 2004

Support - Cardiovascular System

- Transient myocardial stunning& dysfunction
  - lasts 48-72hrs, high level of cathecholamine
  - low cardiac output, followed vasodilation
    Tx.: Fluid, vasoactive, inotropics, inodilator
    (JACC 2002;40:2110)
- Hemodynamic instability: Echo & cardiac evaluation within the first 24 hrs after arrest
- Early corticosteroid supplementation (adrenal insufficiency)?
- Prophylactic of antiarrhythmics: insufficiency of evidence
- Continue infusion of antiarrhythmics associated with ROSC (Class Indeterminate)
- β-blocker with ischemic heart disease if there are no Clx.

Support - *Ventilatory Parameters*

- Hyperventilation → hypocapnea (PCO_2 ↓)
  → cbr. vasoconstriction → CBF ↓
  → cerebral ischemia and ischemic injury
- Hyperventilation → 기도압 ↑ auto PEEP
  → CVP & ICP ↑ → CBF ↓
  → cerebral ischemic injury ↑
- Routine hyperventilation is detrimental (Class III)


ROSC
Pre-arrest
Arrest
CPR
Post-arrest life support - IPPV - ICU
10 - 15'
MAP
CO
gCBF
Epi
CS
CPB
CPR
gCMRO2
MAP
?
gCMRO2
gCBF, CO
ICP
MAP
CO
CBF
%
150
100
50
ICP
30'
1h
2h
12h
24h
48h
72h
96h

Support - CNS

- Optimize cerebral perfusion pressure
- Treat hyperthermia + therapeutic hypothermia
- Seizures control by anticonvulsant therapy initiated (Class IIa)
- Routine seizure prophylaxis (Class Indeterminate)

Thrombolytic agents during CPR

- 90 OHCA, Heparin & rt-PA (Lancet 2001;357:1583)
  - A prospective pilot intervention trial in pts undergoing CPR
  - ROSC (68% vs 48%)
  - Alive at 24 hrs after CA (35% vs 25%)
  - Hospital discharge (15% vs 8%)
  - No bleeding complication

- 108 pts, rt-PA (Resuscitation 2001;50:71)
  - A retrospective case control study

- Large European multicenter trial (TROICA trial)-large scale, randomized, controlled clinical trial, over 1000 pts, over 40 centers

Intravascular fibrin formation & microthrombosis; distributed throughout the entire microcirculation after CA

Outcome Prediction of Postresuscitation Coma

Happy Faces ^ ^ *

CPC 1, 2

Unhappy Faces ^ ^ *

CPC 3

CPC 4

CPC 5

Aggressive early management ?

Withdrawal of Critical Care Support ?

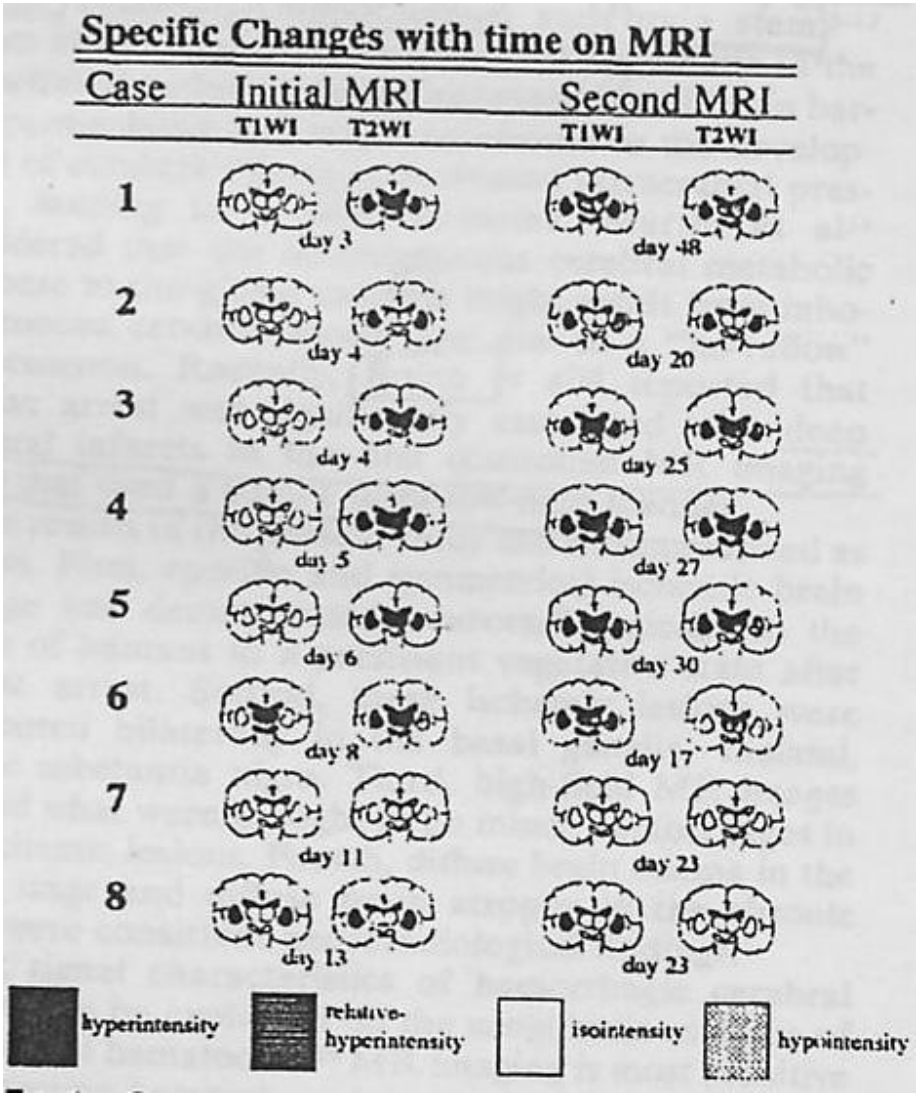
가장 효과적인
예후 예측 인자는???

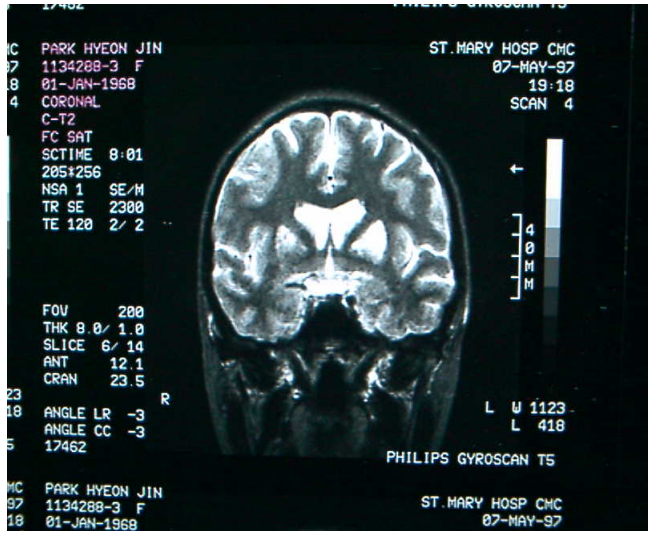
St.Mary's Hosp Case

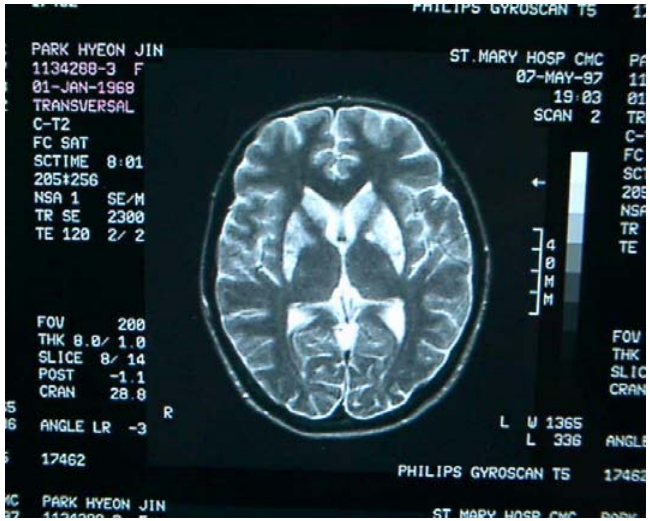
96.05

Stroke 1994:25;2091-2095

박 0 0 MRI findings







St.Mary's Hosp Case : 박 0 0

1997.05.01

1. Neurologic examination
2. Electrophysiologic Studies (SEP,EEG)
3. Imaging (CT, MRI, MRS, Diffusion Imaging)
4. Biochemical markers (NSE, S-100, CK-BB)
5. Inflammatory markers (IL-6, IL-8)
6. Combination of predictive markers

1. Neurologic Examination

Is This Patient Dead, Vegetative, or Severely Neurologically Impaired ?

Assessing Outcome for Comatose Survivors of Cardiac Arrest

1914pts (11 studies)

Booth, et. Al. JAMA,, 2004

Table 5. Pooled Clinical Signs in the Prognosis of Post–Cardiac Arrest Coma

Source	LR of Poor Neurological Outcome (95% Confidence Interval)	
	Positive	Negative
	At 24 Hours	
Absent Withdrawal to Pain		
Summary LR	**4.7 (2.2-9.8)**	**0.2 (0.1-0.6)**
Edgren et al[34]	3.9 (1.1-14)	0.4 (0.2-0.8)
Levy et al[39]	6.8 (2.3-19.8)	0.2 (0.2-0.3)
Sasser[42]	5.1 (3.6-7.3)	0.2 (0.1-0.2)
Snyder et al[43]	6.5 (1.0-42.0)	0.3 (0.1-0.7)
Absent Pupil Response		
Summary LR	**10.2 (1.8-48.6)**	**0.8 (0.4-1.4)**
Chen et al[34]	0.9 (0.0-19.1)	1.0 (0.8-1.2)
Edgren et al[34]	5.6 (0.3-95.0)	0.8 (0.6-1.1)
Levy et al[39]	10.7 (0.7-170.0)	0.8 (0.7-0.9)
Sasser[42]	39.2 (5.6-276.6)	0.6 (0.6-0.7)
Absent Motor Response		
Summary LR	**4.9 (1.6-13.0)**	**0.6 (0.3-1.3)**
Chen et al[34]	3.7 (0.2-59.1)	0.9 (0.6-1.1)
Levy et al[39]	5.5 (1.4-21.0)	0.6 (0.5-0.8)
Sasser[42]	7.6 (4.6-12.6)	0.4 (0.3-0.4)
Snyder et al[43]	3.5 (0.5-24.3)	0.7 (0.5-1.1)
Absent Corneal Reflex		
Summary LR	**12.9 (2.0-68.7)**	**0.6 (0.2-1.9)**
Edgren et al[34]	1.8 (0.2-15.4)	0.9 (0.7-1.2)
Levy et al[39]	14.8 (0.9-233.0)	0.7 (0.7-0.8)
Sasser[42]	90.9 (5.7-1442.9)	0.4 (0.4-0.5)

Booth, et. Al. JAMA, 2004

Table 5. Pooled Clinical Signs in the Prognosis of Post–Cardiac Arrest Coma

Source	LR of Poor Neurological Outcome (95% Confidence Interval)	
	Positive	Negative
	At 72 Hours	
Absent Pupil Response		
Summary LR	**3.4 (0.5-23.6)**	**0.9 (0.4-2.1)**
Chen et al[34]	0.9 (0.0-19.1)	1.0 (0.8-1.2)
Edgren et al[37]	5.3 (0.3-84.0)	0.8 (0.7-1.0)
Levy et al[39]	5.8 (0.4-94.0)	0.9 (0.8-1.0)
Absent Motor Response		
Summary LR	**9.2 (2.1-49.4)**	**0.7 (0.3-1.3)**
Chen et al[34]	2.0 (0.1-34.8)	0.9 (0.7-1.2)
Edgren et al[37]	12.6 (0.8-193.0)	0.6 (0.5-0.7)
Levy et al[39]	16.5 (1.1-261.0)	0.7 (0.6-0.8)
Snyder et al[43]	3.0 (0.2-38.8)	0.6 (0.3-1.1)
Seizure or Myoclonus†		
Summary LR	**1.4 (0.5-3.9)**	**0.8 (0.3-2.1)**
Krumholz et al[38]	1.7 (0.8-3.4)	0.7 (0.5-1.0)
Levy et al[39]	1.1 (0.5-2.3)	1.0 (0.8-1.2)
Snyder et al[44]	1.7 (0.7-4.2)	0.8 (0.6-1.1)

Abbreviation: LR, likelihood ratio.
*Times reflect number of hours since cardiac arrest.
†These figures refer to the presence of seizures or myoclonus at any time after cardiac arrest.

Booth, et. Al. JAMA, 2004

2005. AHA.
Recommendation

- Predictors of poor neurologic outcome
  (1) absent corneal reflex at 24 hrs
  (2) absent pupillary response at 24 hrs
  (3) absent withdrawal to pain at 24 hrs
  (4) no motor response at 24 hrs
  (5) no motor response at 72 hrs

Advantage and Limitation of Neurologic Exam

Advantage

- Universal availability
- Ease of performance
- First line of information

Limitation

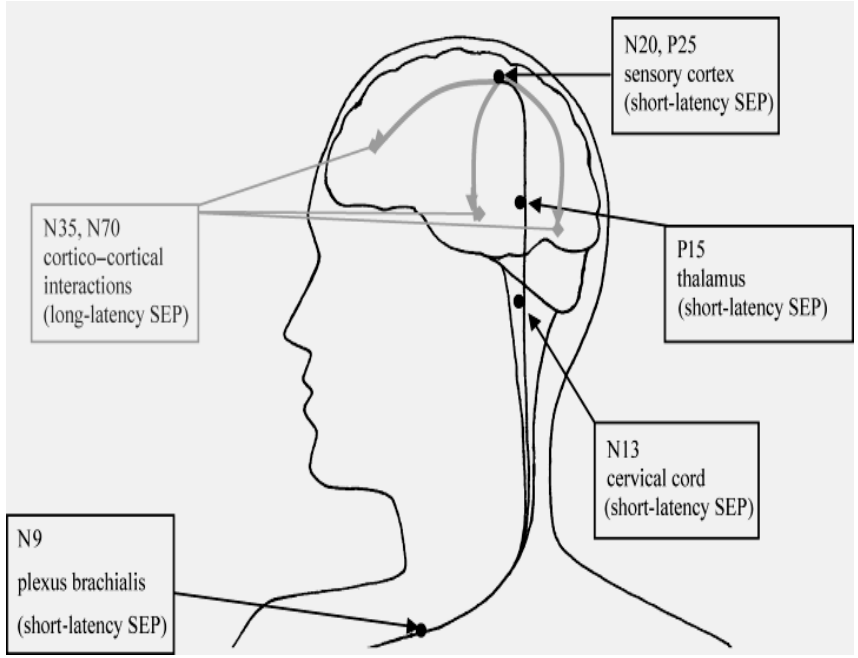
- Many resuscitated pts : still under analgesia and sedation
- : thus not fully assessable by neurologic exam
- Not objective
- Relative wide C.I

2. Electrophysiologic Studies (SEP)

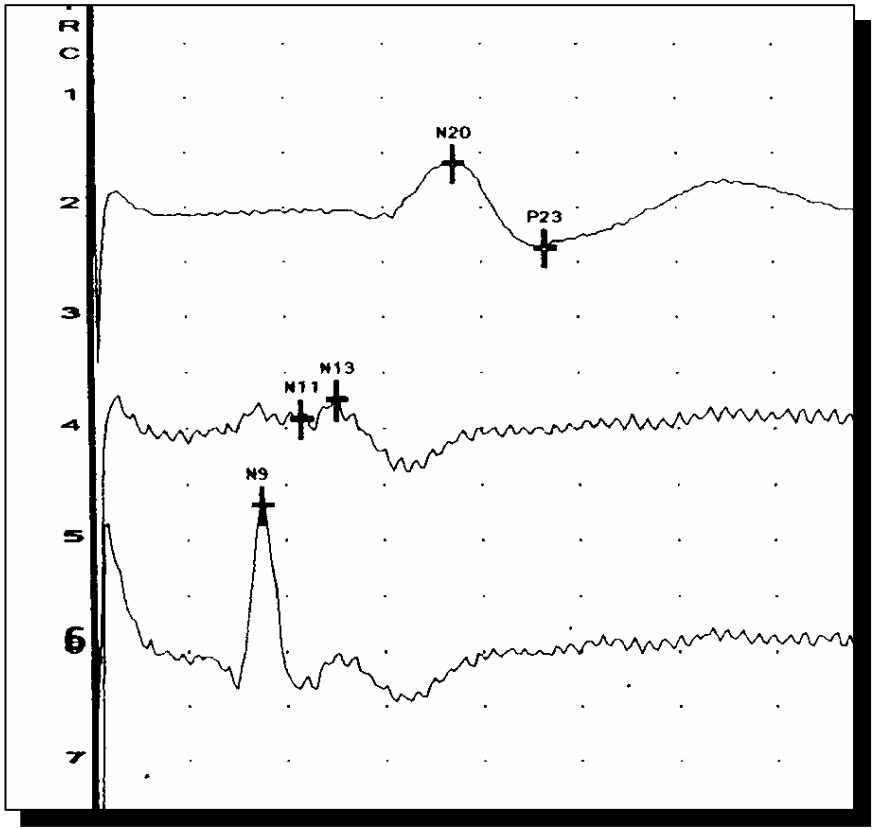
Somatosensory Evoked Potential (SEP)

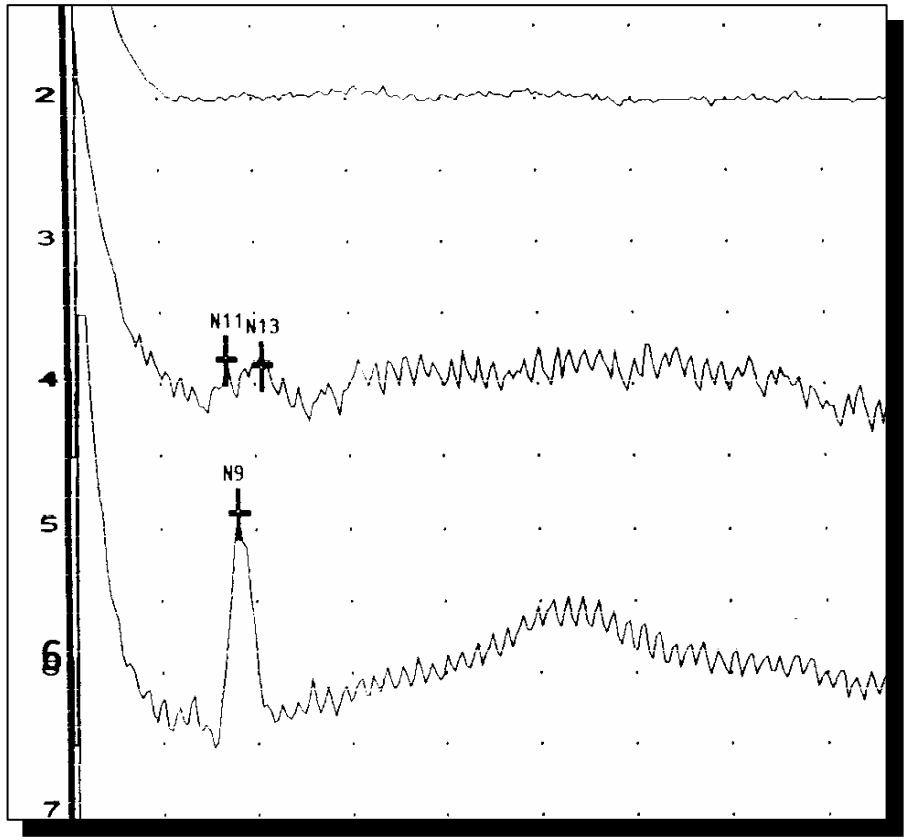
Electrode

1) Erb point(N9 peak)
2) 7th cervical level(C7 : N13 peak)
3) Sensory Cortical level
   (C3', C5': N20 peak)



Somatosensory Evoked Potential





Review of the use somatosensory evoked potentials in the prediction of outcome after severe brain injury

Bradley G. et al., Crit Care Med Vol.29,No.1

1804 pts, reviewed 44 studies

Bradley G, Crit Care Med, 2001

	PLR (95% C.I)	PPV	Sensitive
Normal SEP (favorable outcome)	4.04(3.10-5.28)	71.2% (394/553)	59% (394/668)
Bilat. Abs SEP (unfavorable outcome)	11.41(7.93-16.42)	98.5% (765/777)	46.2% (765/1657)

PLR : Positive liklihood ratio, PPV : Positive predictive value , SEP : Somatosensery evoked potential
95% C.I (Confidence Interval)

Bradley G, Crit Care Med, 2001

Table 4. Reappearance of bilaterally absent somatosensory evoked potentials in patients who had favorable outcomes

Study	Number of Patients	Type of Injury	Comments
Cusumano (54)	1	Traumatic	Edema, elevated ICP, basal bifrontal lesions
DeLecluse (71)	5	Traumatic	—
DeMeirleir (16)	2	Other	Reye's syndrome, postdecompressive craniectomy
Facco (20)	1	Traumatic	Elevated ICP
Guerit (67)	1	Traumatic	Brainstem bleed
Krieger (31)	1	Traumatic	Postdecompressive craniectomy
Lindsay (33)	1	Traumatic	—
Pohlmann-Eden (39)	1	Traumatic	Brainstem bleed
Rumpl (42)	1	Traumatic	Brainstem bleed
Synek (69)	3	Traumatic	Hyperthermia
Taylor (44)	1	Other	Lightning strike
Zegers de Beyl (72)	4	Traumatic	—

Other, Not traumatic, hypoxic-ischemic encephalopathy, or infection; ICP, intracranial pressure.

Bradley G, Crit Care Med, 2001

Table 5. Requirements for interpretation of bilaterally absent somatosensory evoked potentials as a predictor of poor outcome

Specific diagnosis
No focal lesion preventing impulse from reaching cortex (11)
No subdural or extradural collections to impede the recording of the cortical response (11)
No decompressive craniotomy in previous 48 hrs (11, 16, 31)
Coma not caused by lightning injury (44)
Coma not caused by reversible conditions (e.g., drug overdose)
Age appropriate methodology (cut-off at ~4 mos of age) (77)
Recording made and interpreted by experienced personnel (1–4)
Recording made >24 hrs after injury
Recording duplicated after 24 hrs

Bradley G, Crit Care Med, 2001

The time dependency of SSEPs in comatose CA survivors. 25 pts

- Within 24hr after ROSC there was a significant improvement in SEP
Therefore we recommend allowing a period of at least 24hr after ROSC

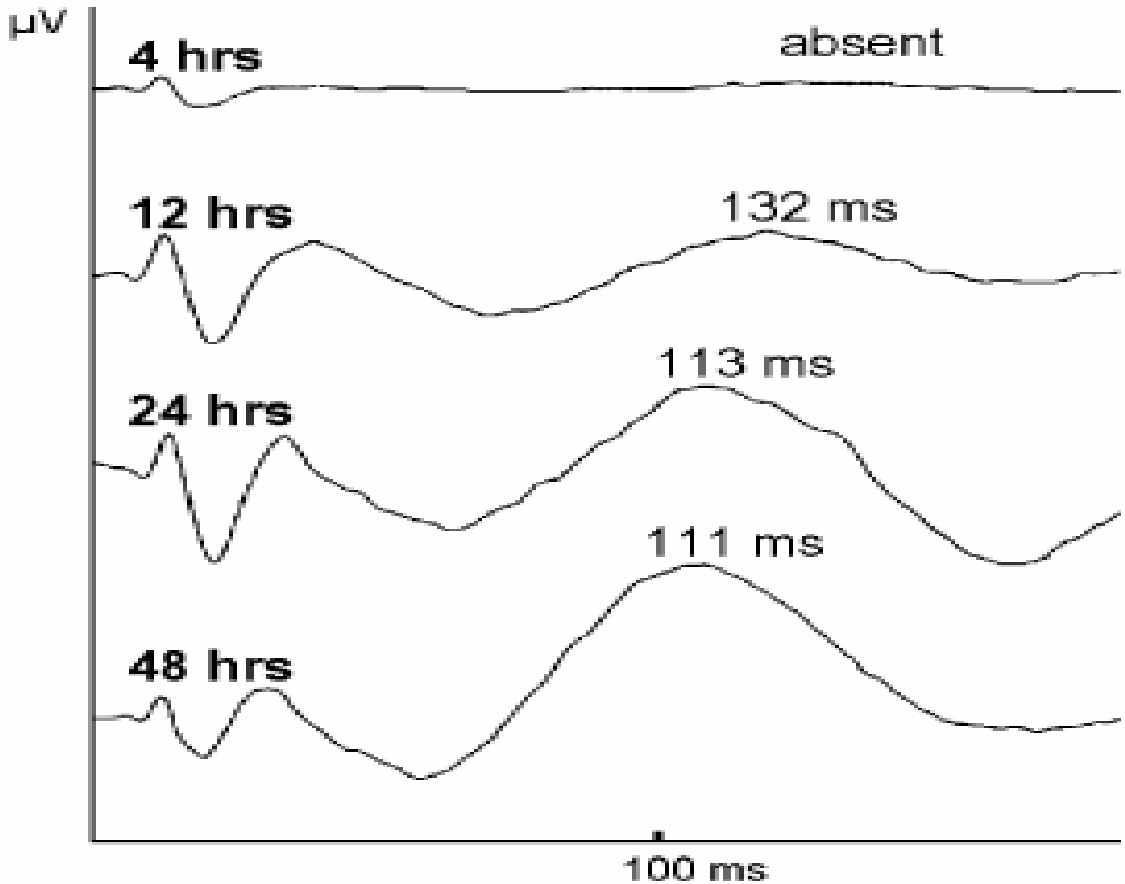


Fig. 1 Alterations in long-latency SEP within 48 h after ROSC demonstrate in one comatose cardiac arrest survivor

Gendo A, Vienna, Austria, Intensive Care Med, 2001

2005. AHA.
Recommendation

- Prediction of poor neurologic outcome

  Bilateral median nerve SEP (N20)
  at least 72 hrs with hypoxic-anoxic coma

Proton Magnetic Resonance Spectroscopy(MRS)

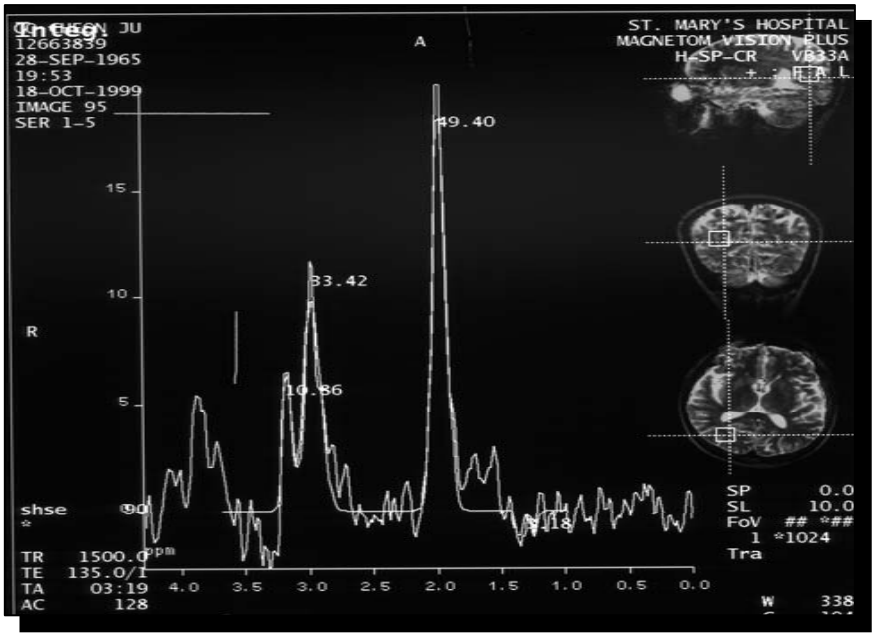


Figure.1 Case 5,

Spectra from PO region at 48 hours after ROSC showed

no evidence of any lactate signal.

He recovered consciousness at 12 days after ROSC and

has shown mild memory impairment.

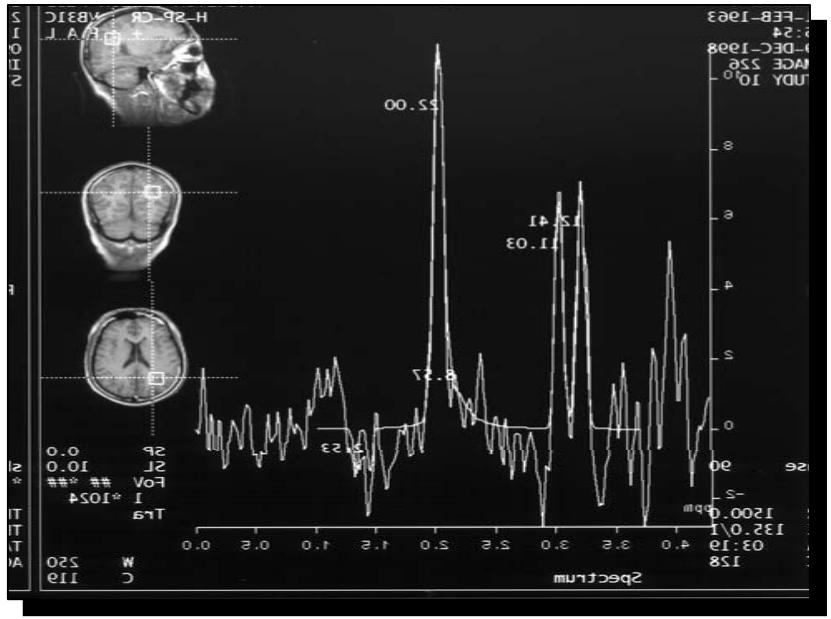


Figure.2 Case 9,

Spectra from PO region at 72 hours after ROSC showed

elevated lactate peak at 1.3 ppm.

He presented with persistent vegetative state.

27 pts, Lac(+) Sensitivity : 78.9%, Specificity : 100%

LR(+) : 13.9, LR(-) : 0.2

St. Mary Hosp., Ann Emerg Med 2001:38(4); S39

Summery of MRS

- Advantage
  - Greater advantage than clinical evaluation
  - Not influenced by sedating drugs or other medical treatment
  - Objective parameter
- Limitation
  - Expensive
  - Availability
  - Need intensive care monitoring during test

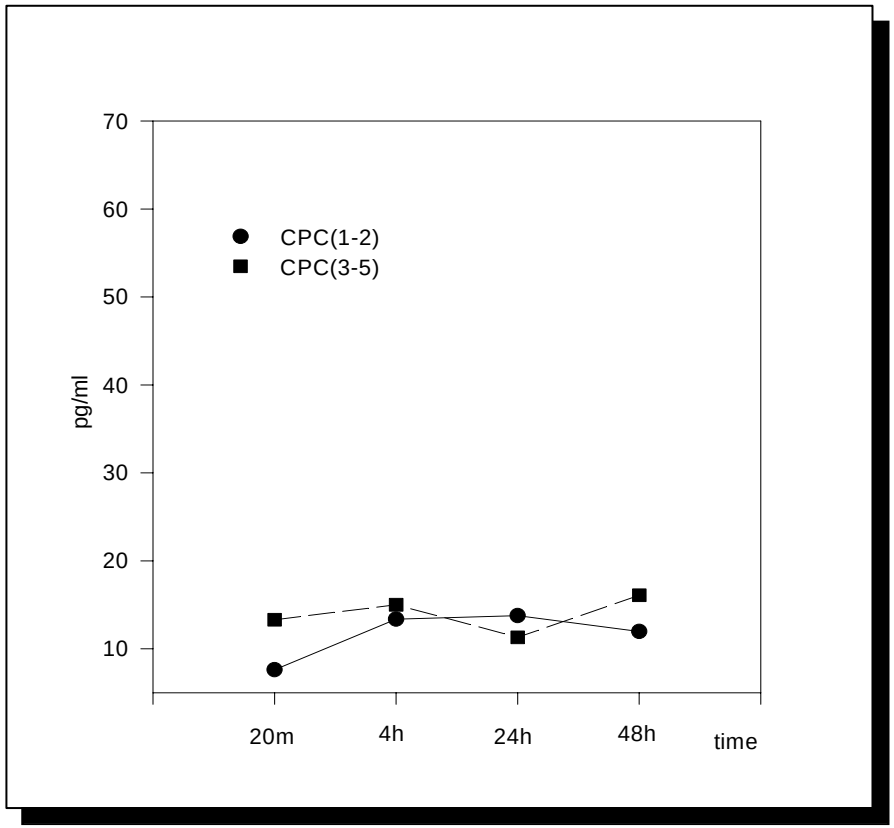


Figure1. The comparison of the time course between the course between the CPC(1-2) and CPC(3-5)in CSF IL-1 concentration

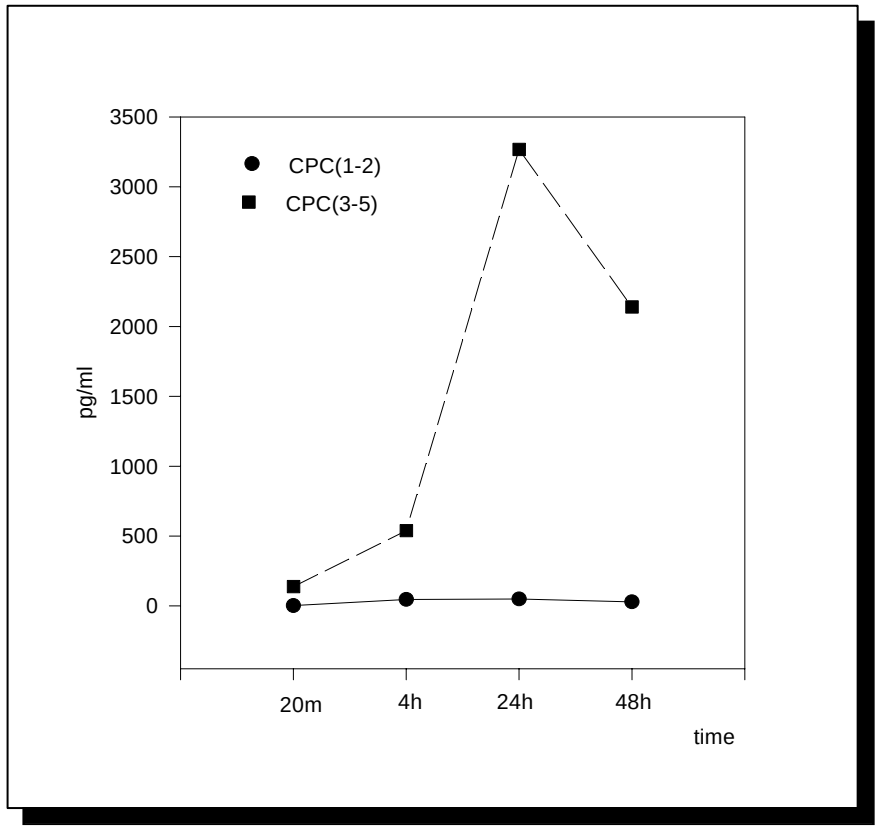
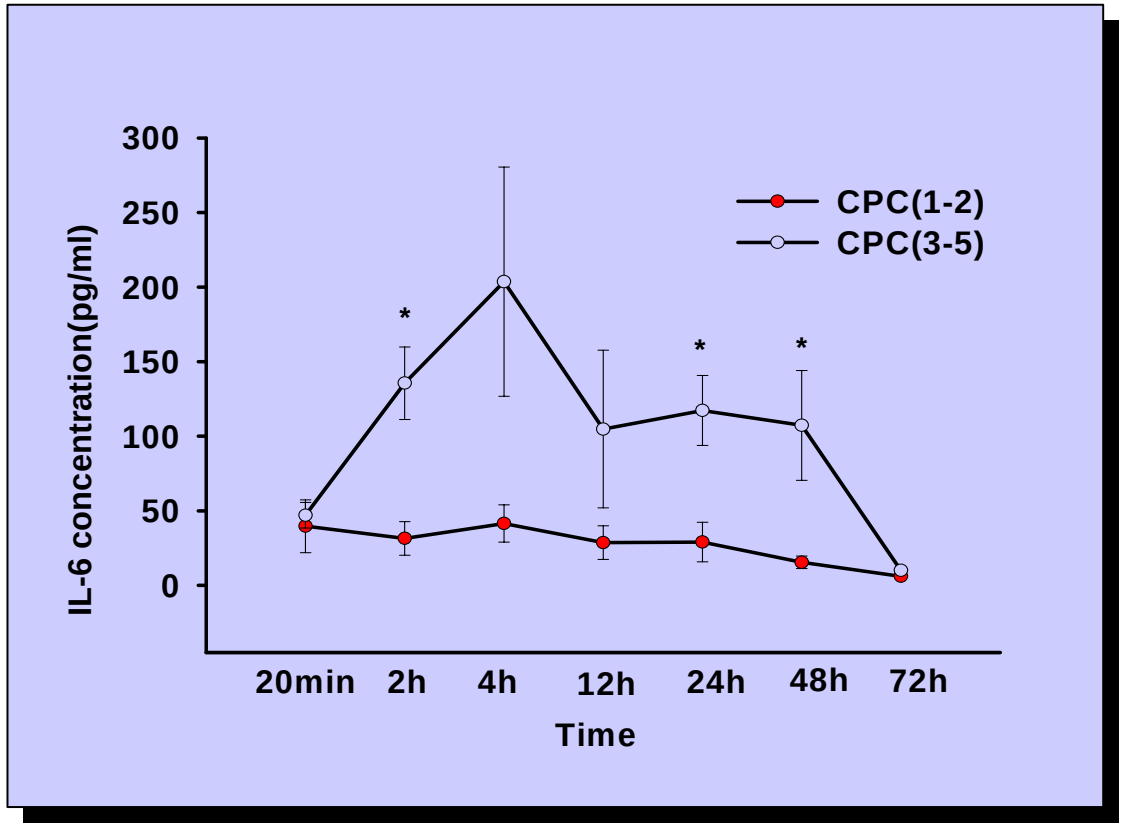


Figure2. The comparison of the time course between the course between the CPC(1-2) and CPC(3-5)in CSF IL-6 concentration



Patterns of evolution of serum IL-6 after successful CPR.

Data are expressed as mean ± SEM, * p < 0.05

St.Mary's Hosp. , Ann Emerg Med 2000:36(4); S91

Combination of Predictive factors

Sensitivity, specificity, positive and negative likelihood ratio of numerous tests and results in predicting poor outcome after coma

Indicator of poor prognosis	Outcome		Sensitivity (%)	Specificity (%)	LR+	LR−	$p^{\dagger}$
	Poor*	Good*					
SEP, bilateral absent N20 (n=35)	13/24	0/11	54.2	100	12.9	0.5	0.002
SEP, uni−/bilateral absent N20 (n=35)	15/24	1/11	62.5	90.9	6.9	0.4	0.003
MRS, lactate(+) (n=27)	15/19	0/8	78.9	100	13.9	0.2	0.000
SEP, bilateral absent N20 when MRS also examed (n=26)	11/18	0/8	61.1	100	10.9	0.4	0.007
MRS, lactate(+), SEP, bilateral absent N20, or both (n=26)	17/18	0/8	94.4	100	16.6	0.1	0.000

St.Mary's Hosp. Unpubulished data,

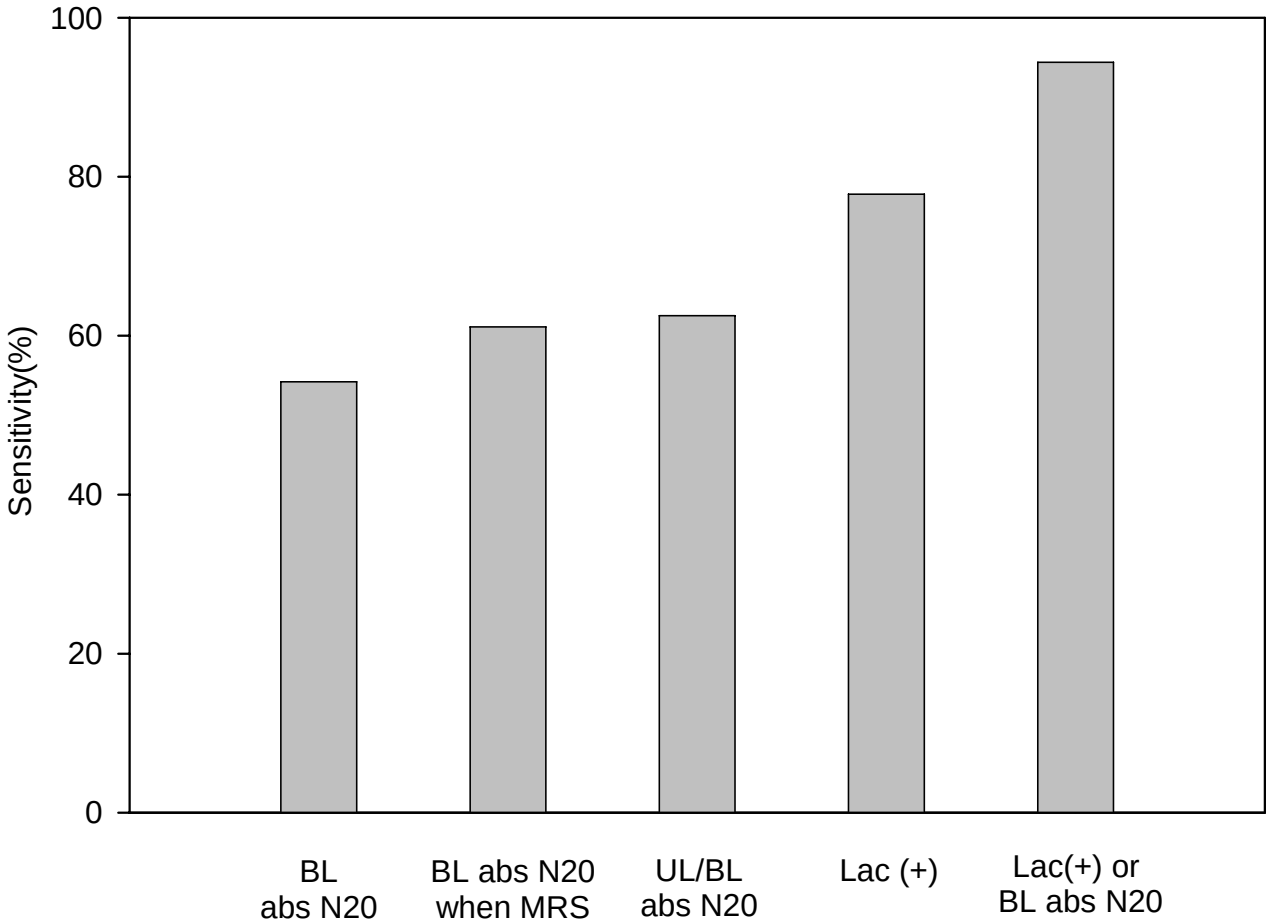


Figure 1**.** Sensitivity of various criteria for predicting poor outcome.
Specificity of all criteria except 'UL/BL abs N20' is 100% in the population tested.
UL = unilateral; BL = bilateral; Lac = lactate; abs = absent.

St.Mary's Hosp., Unpubulished data,

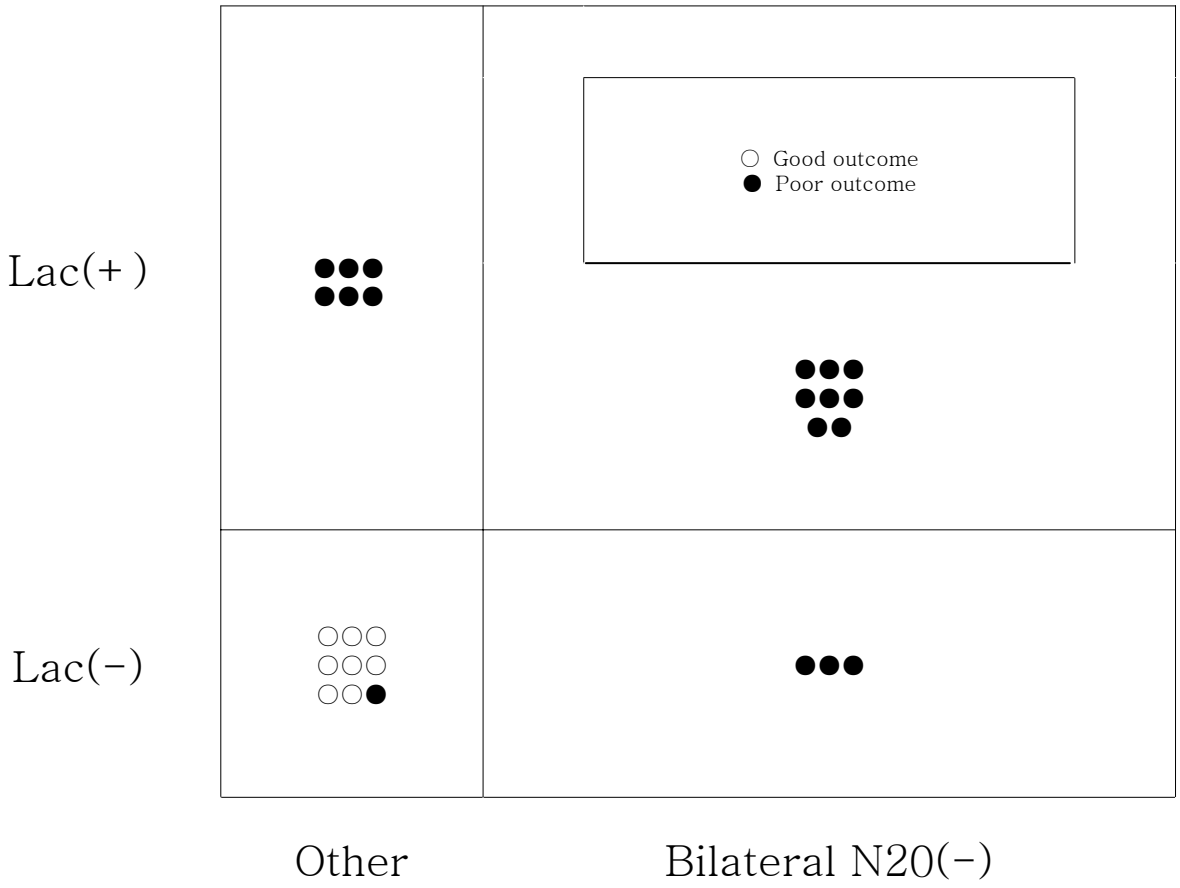
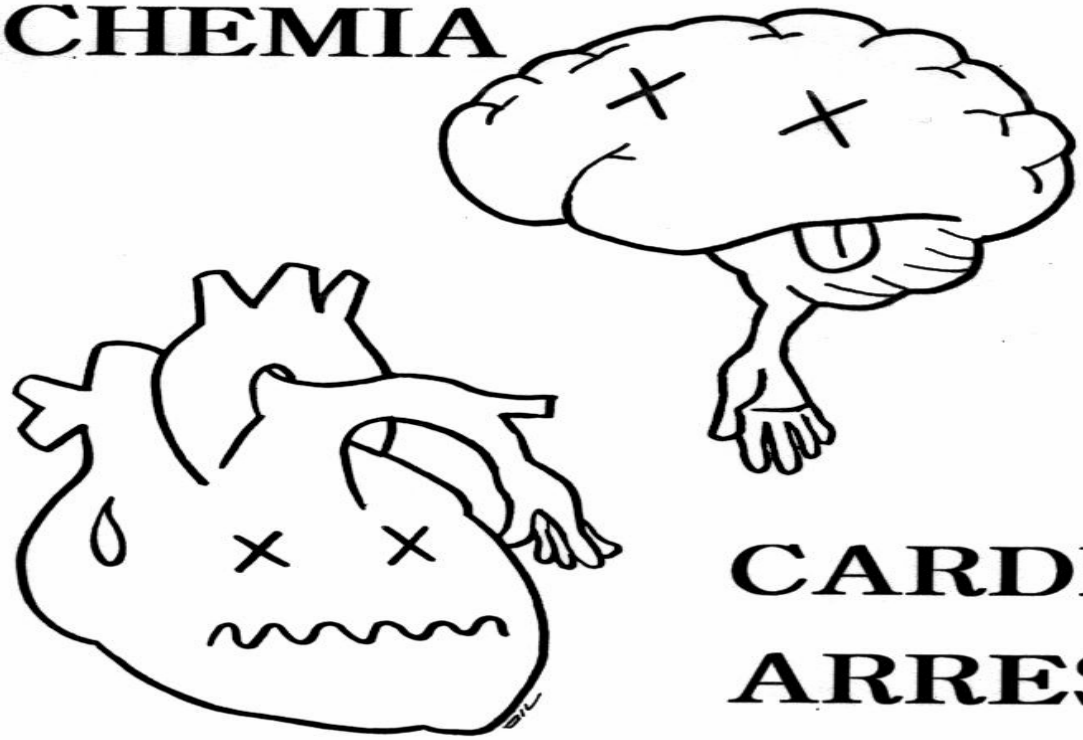


Figure 2. MRS lactate, SEP N20, and outcome in 26 coma patients with cardiac arrest. BL = bilateral; Lac = lactate; abs = absent.

St.Mary's Hosp., Unpubulished data,

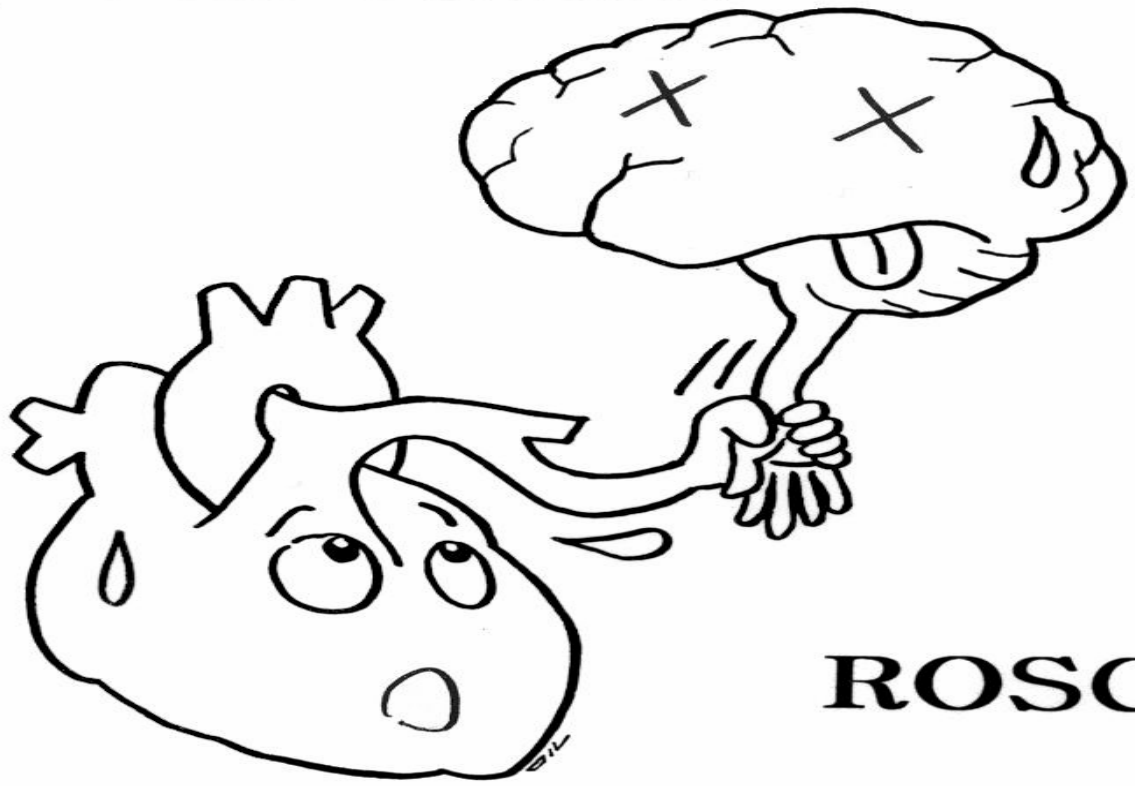
GLOBAL ISCHEMIA



CARDIAC ARREST

BLS ACLS

REPERFUSION



ROSC

PLS

SUCCESS!

CPCR


Thanks for your attention !!
Photo by Falkor
www.sooni.net