Journal Reading

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Predictors of acute decompensation after admission in ED patients with sepsis

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Introduction (I)

- The patient who presents with or subsequently develops severe sepsis or septic shock
  - ✓ Rapid aggressive resuscitation
    → improving outcomes and decreasing mortality rates that range from 35% to 65%
  - ✓ ED care and disposition decisions have significant effects on outcome in patients with severe infection.
- In-hospital or 30-day mortality
  - ✓ not be the optimal measure for assessing infection severity in the ED itself

Introduction (II)

- Patients have worsening sepsis or develop septic shock on regular nursing floors have delays in processes of care as compared to those who develop septic shock in the intensive care unit (ICU).
  - ✓ identify factors predictive of decompensation on the inpatient floor within 48 hours after admission among those ED patients initially admitted to a regular nursing floor
  - ✓ transfer to an ICU within 48 hours of admission as the marker of acute decompensation

Methods (I)

- A case-control study of ED patients admitted to an urban tertiary care hospital
- Inclusion criteria
  - ✓ treatment in the ED and admission to a regular nursing (non-ICU) floor
  - ✓ proven or suspected infection in the ED
  - ✓ receipt of a hospital discharge diagnosis of sepsis (ICD-9-CM code of 038.49, 038.9, or 995.92)
- from January 1, 2003, to June 30, 2005

Method (II)

- stratified into 2 groups
  - ✓ ICU group：all patients who were transferred to an ICU within 48 hours of admission
  - ✓ non-ICU or floor group：all patients who remained on the regular nursing floor for at least 48 hours
- An infection was considered
  - ✓ documented ED diagnosis of an infectious condition
  - ✓ an ED diagnosis to “rule out” an infectious condition
  - ✓ blood cultures were ordered in the ED
- To assess the burden of comorbidities, the Charlson comorbidity index was calculated.

Charlson risk index

Condition	Assigned weights for diseases
Myocardial infarct	1
Heart failure	1
Peripheral vascular disease	1
Cerebrovascular disease	1
Dementia	1
Chronic pulmonary disease	1
Connective tissue disease	1
Ulcer disease	1
Mild liver disease	1
Diabetes	1
Hemiplegia	2
Moderate or severe renal disease	2
Diabetes with end organ damage	2
Any tumor	2
Leukemia	2
Lymphoma	2
Moderate or severe liver disease	3
Metastatic solid tumor	6
AIDS	6
Weighted comorbidity classes	
Low	0 points
Medium	1 to 2 points
High	3 to 4 points
Very high	≥5 points

Adapted from: Charlson, ME, Pompei, P, Ales, KL, et al, J Chron Dis 1987; 40:373.

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Method (III)

- Altered mental status
  - ✓ altered mental status, confusion, delirium, or dementia
- Immunosuppression
  - ✓ HIV, AIDS, cancer, multiple myeloma, chemotherapy, recent systemic steroid use (within 30 days), organ transplant, or use of any immunosuppressive medication
- fever as temperature of 38.0˚C or higher
- tachycardia as heart rate of 100 beats per minute or higher
- tachypnea as respiratory rate of 20 breaths per minute or higher
- hypotension as systolic blood pressure of less than 90 mm Hg
- low serum bicarbonate was defined as values less than 20 mmol/L

Method (IV)

- The primary study outcome variable was transfer to an ICU within 48 hours of admission.

Result (I)

Table 1 Characteristics of study patients

	Entire population (n = 78)	Transferred to ICU (n = 34)	Remained on regular floor (n = 44)
Proportions of dichotomous variables (n%)			
Male sex	52.5	64.7	43.2
Previously existing do-not-resuscitate order	18.2	16.1	23.1
Nursing home resident	27.6	11.7	38.6
Temperature, ≥38.0°C	32.0	17.6	43.2
Tachycardia (>100 beats/min)	64.1	64.7	63.6
Tachypnea (>20 breaths/min)	11.5	17.6	6.8
Hypotension (systolic, <90 mm Hg)	39.7	41.1	38.6
Low serum bicarbonate (<20 mmol/L)	32.9	55.9	14.3
Immunosuppression present [a]	26.9	35.3	20.4
Altered mental status in ED [b]	43.4	46.9	40.9
Vasopressors used in the ED	2.5	2.9	2.2
Antibiotics administered in the ED	79.5	55.9	97.7
Blood cultures ordered in the ED	48.7	37.5	56.8
Urine culture ordered in the ED	26.3	28.0	25.0

Result (II)

Table 2 Results of univariate testing for prediction of ICU transfer within 48 hours of admission. *P* values < .05 are bolded.

Variables	OR (95% CI)	*P*
Age	0.99 (0.96-1.01)	.354
Male sex	2.40 (0.96-6.07)	.061
Previously existing do-not-resuscitate order	0.64 (0.13-3.20)	.587
Nursing home resident	0.21 (0.06-0.71)	**.012**
Charlson comorbidity score	1.15 (0.91-1.47)	.242
Immunosuppression	2.12 (0.77-5.86)	.147
Altered mental status in ED	1.27 (0.51-3.19)	.605
Temperature—initial (°F)	0.56 (0.39-0.78)	**.001**
Heart rate—initial	1.00 (0.98-1.02)	.719
Respiratory rate—initial	1.01 (0.95-1.07)	.833
Systolic blood pressure—initial (mm Hg)	0.99 (0.97-1.00)	.191
Temperature—highest in ED (°F)	0.68 (0.53-0.88)	**.003**
Heart rate—highest in ED	1.00 (0.98-1.02)	.936
Respiratory rate—highest in ED	1.03 (0.96-1.11)	.318
Systolic blood pressure—lowest in ED (mm Hg)	1.00 (0.98-1.01)	.704
Oxygen saturation—lowest in ED	1.12 (0.96-1.29)	.141
White blood cell count (K/μL)	0.98 (0.92-1.050	.624
Hemoglobin level (g/dL)	1.15 (0.95-1.39)	.140
Platelets (K/μL)	1.00 (1.00-1.00)	.303
Band forms (%)	1.02 (0.94-1.12)	.601
Sodium (mmol/L)	1.04 (0.96-1.13)	.362
Potassium (mmol/L)	1.08 (0.66-1.78)	.761
Blood urea nitrogen (mg/dL)	1.00 (0.99-1.02)	.848
Creatinine (mg/dL)	0.91 (0.78-1.05)	.187
Glucose (mg/dL)	1.00 (0.99-1.01)	.900
Serum bicarbonate (mmol/L)	0.88 (0.81-0.96)	**.004**
Absence of fever (temperature, <38.0°C)	3.65 (1.24-10.71)	**.018**
Tachycardia (>100 beats/min)	1.05 (0.41-2.66)	.922
Tachypnea (>20 breaths/min)	2.92 (0.68-12.70)	.151
Hypotension (systolic, <90 mm Hg)	1.11 (0.44-2.77)	.820
Low serum bicarbonate (<20 mmol/L)	7.60 (2.54-22.8)	**<.001**

Result (III)

- In the multivariate analysis
  - ✓Only absence of fever and decreased serum bicarbonate were significant contributors to the model (for prediction of ICU transfer).
    - low serum bicarbonate (＜20 mmol/L) odds ratio (OR) = 7.40 (95% CI, 2.35-23.30)
    - absence of fever OR = 3.66 (95% CI, 1.11-12.60)

Result (IV)

Table 3 Patient outcomes stratified by group as a percentage or mean with 95% CIs

Outcome	Entire population (n = 78)	ICU group (n = 34)	Non-ICU group (n = 44)	*P*
In-hospital mortality	20.5 (12.2-31.2)	32 .3 (17.4-50.5)	11.4 (3.8-24.6)	.045
Intubation within 48 h	24.4 (15.3-35.4)	61.3 (42.2-78.2)	0 (0-8.0)	<.001
Mean hospital length of stay (d)	14.1 (11.4-16.7)	16.8 (11.9-21.6)	12.0 (9.2-14.8)	.07

Discussion & Conclusion (I)

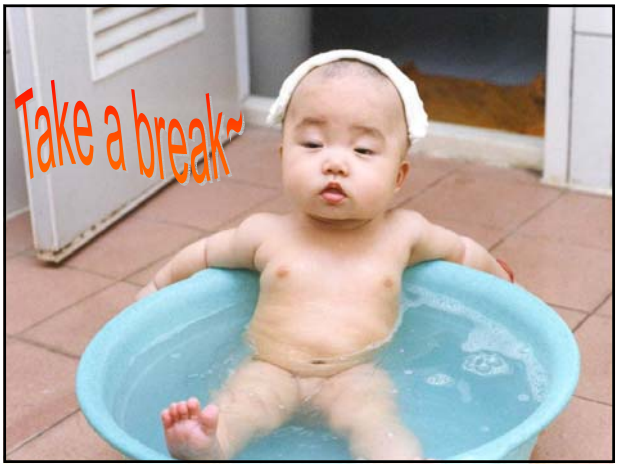
- In this study of variables available to the ED physician, only serum bicarbonate and absence of fever were independently predictive of patient decompensation.
  - ✓the low bicarbonate likely represents occult hypoperfusion
  - ✓lactate? (study patients enrolled in a period before widespread use of lactate)
- patients who develop sepsis on regular nursing floors get worse or delayed care

→ a trend toward increased mortality in the floor patients

→ rapid, early, aggressive identification and treatment of severe sepsis/septic shock, correct ED admission decisions are critical

Discussion & Conclusion (II)

- Emergency and admitting physicians should consider the possibility of worsening infection severity in patients with low serum bicarbonate.
- In addition, the absence of fever should not be used as a sign that a patient does not have severe infection.



Predictors of poor neurologic outcome in patients after cardiac arrest treated with hypothermia: A retrospective study

Resuscitation (2011), doi:10.1016/j.resuscitation.2011.02.020

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Introduction (I)

- Clinical signs, electrophysiological and/or biochemical tests with a false positive rate of 0% and a narrow confidence interval are currently used early after cardiac arrest to identify a subset of patients with a poor prognosis.
  - ✓clinical practice parameters as described by the Quality Standards Subcommittee of the American Academy of Neurology (AAN)

Introduction (II)

- Mild therapeutic hypothermia during the post-cardiac arrest period
  - ✓ a marked effect on the cerebral changes
    - ➢ metabolism, cerebral blood flow, inflammatory response and neuro-excitatory pathways
  - ✓ Sedatives (midazolam) additionally influence the time to regaining consciousness
    - ➢ hypothermia influences the metabolism
      1. an increase in plasma concentrations
      2. possibly depressed cytochrome P 450(CYP)3A4 and CYP3A5 activity
    - ➢ impaired hepatic and renal function
    - → midazolam and its active metabolites can accumulate resulting in an unpredictably prolonged sedative effect
  - ✓ parameters may not be applicable to patients after hypothermia

Materials and methods (I)

- Patients
  - ✓ retrospective cohort study
  - ✓ consecutive adult patients with return of spontaneous circulation (ROSC) after cardiac arrest and treated with mild hypothermia
  - ✓ admitted to the ICU of the Radboud University Nijmegen Medical Centre
  - ✓ Medical Centre between January 2007 and November 2009

Materials and methods (II)

- Standard care
  - ✓ all comatose patients with a Glasgow Coma Scale (GCS) ≤ 8 after cardiac arrest were admitted to the ICU
    - ➢ monitored and treated according to international clinical standards and the institutional protocol
  - ✓ cooled to 32–34 ℃ for 24h by infusion of cold fluids and surface cooling, followed by passive rewarming
  - ✓ sedated with midazolam and/or propofol
    - ✓ Ramsay score of 6
    - ✓ analgesia using sufentanyl of morphine during hypothermia
  - ✓ Continuous sedation was stopped at normothermia.
  - ✓ clinical signs of shivering
    - ➢ treated with extra sedation, analgesia or rocuronium
  - ✓ vasoactive or inotropic support
    - ➢ maintain a mean arterial blood pressure > 80mmHg

The Ramsay sedation scale

Clinical score	Patient characteristics
1	Awake; Agitated or restless or both
2	Awake; Cooperative, oriented, and tranquil
3	Awake but responds to commands only
4	Asleep; brisk response to light glabellar tap or loud auditory stimulus
5	Asleep; sluggish response to light glabellar tap or loud auditory stimulus
6	Asleep; no response to glabellar tap or loud auditory stimulus

Redrawn from Ramsay, MA, Savage, TM, Simpson, BR, Goodwin, R, Br Med J 1974; 2:656.

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Materials and methods (III)

- Assessment of neurological prognosis (I)
  - ✓ bilateral median nerve somatosensory evoked potential (SSEP) study
    - ➢ if a patient remained comatose (defined as a motor score of 4 or less) after rewarming and cessation of sedation
  - ✓ If the cortical N20 response was bilaterally absent (with peripheral responses present) supportive care was withdrawn.
  - ✓ Where unilateral or bilateral cortical N20 responses to the SSEP were present, a motor score of M1-2 or absent pupillary reactions to light or absent corneal reflexes at day 3
    - ➢ not result in withdrawal of active treatment
  - ✓ motor score of M1-2 in combination with absent pupillary reactions and absent corneal reflexes
    - ➢ active treatment was withdrawn

Materials and methods (IV)

- Assessment of neurological prognosis (II)
  - ✓ Myoclonia was treated with sedation using midazolom and/or anti-epileptic drugs (valproic acid or levetiracetam).
    - ➢ EMG artefacts with (cortical myoclonus) or without (subcortical myoclonus) EEG spike correlates
      1. cortical myoclonia was present
      - → sedative and anti-epileptic drugs was initiated and medication given for a minimum period of 24 h
  - ✓ An electroencephalogram (EEG) was performed in case of suspected seizures or persistent coma.
  - ✓ EEG consisting of
    - ➢ generalized suppression, burst suppression or generalized periodic discharges
    - → poor prognosis

Materials and methods (V)

- Assessment of neurological prognosis (III)
  - ✓ Glasgow Outcome Scale (GOS) was determined at least 3 months after hospital discharge from the patients' correspondence or after consultation of the family physician
    - ➢ (1) death; (2) persistent vegetative state; (3) severe disability; (4)moderate disability; and (5) good recovery
    - ➢ A favorable neurological recovery was defined as a GOS score of 4 or 5

Materials and methods (VI)

- Patient data
  - ✓ analyzed from the day of admission to the ICU until day 7, discharge from the ICU or death
  - ✓ Comorbidity
    - ➢ smoking, a history of hypertension, diabetes, acute kidney injury (an eGFR < 60ml/min), liver dysfunction (serum bilirubin > 20 μ mol/l), ischemic heart disease (NYHA class III and IV, myocardial infarction or coronary arterial bypass grafting), heart failure (NYHA class III and IV), cardiac arrhythmias, cerebral pathology or malignancy
  - ✓ Complications
    - ➢ new cardiac arrest requiring chest compressions, hemorrhage requiring transfusion, arrhythmias, acute kidney injury (RIFLE criteria 2–3) and/or liver dysfunction (serum bilirubin > 20 μ mol/l)

Materials and methods (VII)

- Data analysis
  - ✓ positive predictive value (PPV) = the probability that the patient had an unfavourable outcome when a positive test result was observed

Results (I)

Demographic and clinical characteristics.

Demographic and clinical characteristics $N = 103$	Favourable $N = 36$ (35.0%)	Unfavourable $N = 67$	p Value
Male versus female, n (%)	24/36 (66.7) versus 12/36 (33.3%)	52/67 (77.6%) versus 15/67 (22.4%)	0.228
Age (yrs)	62.5 (53.8–70.3)	64 (54.3–76.0)	0.078
Body mass index (kg/m²)	24.9 (22.9–27.0)	25.7 (24.1–27.8)	0.351
APACHE II score	26.0 (20.5–30.0)	30.0 (26.8–33.3)	0.001
SAPS	61.0 (49.0–72.5)	72.0 (60.3–81.0)	0.001
Comorbidity before admission n (%)	24/36 (66.7%)	51/67 (76.1%)	0.304
Hypertension	13/36 (36.1%)	16/67 (23.9%)	0.204
Diabetes	2/36 (5.6%)	4/67 (6.0%)	0.932
Renal failure	1/36 (2.8%)	5/67 (7.5%)	0.333
Liver failure	0/36 (0%)	1/67 (1.5%)	0.461
Ischemic heart disease	10/36 (27.8%)	22/67 (32.8%)	0.597
Heart failure	5/36 (13.9%)	12/67 (17.9%)	0.600
Arrhythmia	6/36 (16.7%)	14/67 (20.9%)	0.605
Neurological disease	4/36 (11.1%)	13/67 (19.4%)	0.280
Malignancy	2/36 (5.6%)	3/67 (4.5%)	0.784
Origin of the arrest n (%)			
Primary cardiac	32/36 (88.9%)	45/67 (67.2%)	0.016
Primary hypoxic	1/36 (2.8%)	5/67 (7.5%)	0.333
Acute intracerebral lesion	0/36 (0%)	1 (1.5%)	0.461
Other/unknown	3/36 (8.3%)	16 (23.9%)	0.052
Initial cardiac rhythm n (%)			
VF/VT (most common)	34/36 (94.4%)	38/67 (56.7%)	<0.001
Asystole/pseudo	1/36 (2.8%)	20/67 (29.9%)	0.001
PEA	1/36 (2.8%)	8/67 (11.9%)	0.116
Unknown	0/36 (0%)	1/67 (1.5%)	0.461
Time collapse and ROSC (min)	15 (10–20)	25 (20–45)	0.001
Emergency cardiac treatment n (%)			
Thrombolysis	0/36 (0%)	0/67 (0%)	NA
Emergency CABG	0/36 (0%)	1/67 (1.5%)	0.170
IABP	11/36 (27.8%)	12/67 (17.9%)	0.142
PTA (most common)	16/36 (44.4%)	20/67 (29.9%)	0.139
Complications during ICU stay n (%)	23/36 (63.9%)	53/67 (79.1%)	0.033
Cardiac arrest	5/36 (13.9%)	16/67 (23.8%)	0.203
Hemorrhage	4/36 (11.1%)	2/67 (3.0%)	0.102
Arrhythmia	18/36 (50.0%)	33/67 (49.3%)	0.941
Renal failure	1/36 (2.8%)	16/67 (23.9%)	0.020
Liver failure	3/36 (8.3%)	6/67 (9.0%)	0.879
Length of stay in ICU (days)	5.5 (3.0–7.0)	3.0 (2.0–7.0)	0.011

Result (II)

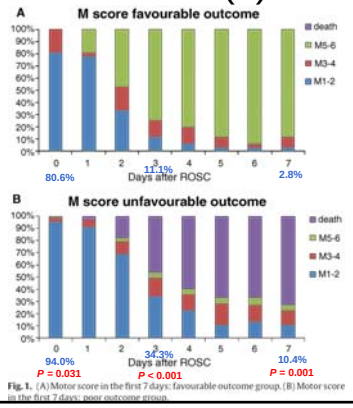


Fig. 1. (A) Motor score in the first 7 days: favourable outcome group. (B) Motor score in the first 7 days: poor outcome group.

Results (III)

Table 2
Combination of an M1 or M2 or absent pupillary reactions to light or absent corneal reflexes on day 3.

	Favourable $N = 36$	Unfavourable $N = 67$	p Value	PPV (95% CI)
Day 3 M1-2 or no PR or no CR	4/36 (11.1%)	54/67 (80.6%)	<0.001*	0.93 (0.82–0.98)
Day 3 M1-2 and no PR and no CR	0/36 (0%)	10/67 (14.9%)	0.013*	1.00 (0.66–1.00)

* Fisher's exact test.

Table 3
Clinical myoclonus in patients with a good outcome and in patients with a poor outcome. (40/103 (38.8%))

Clinical myoclonus	Favourable $N = 36$ (4/36 (11.1%))	Unfavourable $N = 67$ (36/67 (53.7%))	p Value (< 0.001)	PPV (95% CI) (0.90 (0.75-0.97))
Spontaneous	2/36 (5.6%)	24/67 (35.8%)	0.001	0.92 (0.73–0.99)
Stimulus sensitive	1/36 (2.8%)	5/67 (7.5%)	0.027	0.83 (0.36–0.99)
Both	1/36 (2.8%)	7/67 (10.4%)	0.166	0.89 (0.47–0.99)

- Of the 40 patients with clinical myoclonus, 18 underwent at least one EEG.
  - ✓ In 4 of these 18 patients (22.2%) cortical myoclonus was observed, in the others the jerks were subcortical in origin.
  - ✓ All 4 patients with cortical myoclonus had a poor outcome.

Result (IV)

- SSEP of both median nerves
  - ✓total 46 patients (8 patients inconclusive result)
  - ✓Uni- or bilateral cortical responses were present in 44.7% of the patients with a poor outcome and 87.5% of patients with a favorable outcome.
  - ✓no cortical N20 response in 18/38 (47.4%) of the patients with a poor outcome (PPV 1.0 (0.78–1.00))
  - ✓None of the patients with a bilateral absent cortical N20 response had a GCS motor score better than 4.

Results (V)

Table 4
Recorded encephalograms in 27 patients.

N = 27	Favourable(N = 4)	Unfavourable(N = 23)	p Value	PPV (95% CI)
Excessive theta	1/4 (25.0%)	3/23 (13.0%)	0.534	0.75 (0.22–0.99)
Predominant delta	0/4 (0.0%)	2/23 (8.7%)	0.540	1.00 (0.20–1.00)
Triphasic waves	1/4 (25.0%)	3/23 (13.0%)	0.534	0.75 (0.22–0.99)
Suppression	1/4 (25.0%)	9/23 (39.1%)	0.589	0.90 (0.54–0.99)
Burst suppression	0/4 (0.0%)	2/23 (8.7%)	0.540	1.00 (0.20–1.00)
PLEDs	3/4 (75%)	14/23 (60.9%)	0.589	0.82 (0.56–0.95)
Unreactive	0/4 (0.0%)	15/23 (65.2%)	0.015	1.00 (0.75–1.00)

1/4 (25%) | 11/23 (47.8%) | 0.396 | 0.92 (0.59–0.99)

PLEDs = Periodic lateralized epileptiform discharges

- anti-epileptic treatment for at least 24 h was performed in 16 patients (most commonly with benzodiazepines combined with valproic acid)
  - ✓ failed to improve consciousness or EEG patterns in 14/15 (93.3%) patients with a poor outcome and in none of the patients with a good outcome (p = 0.125) (clinical improvement：2)
  - ✓ PPV = 1.00 (0.73-1.00)

Discussion & Conclusion (I)

- The prognosis of post-anoxic encephalopathy in patients treated with hypothermia is difficult to predict in an early phase.
- After treatment with mild hypothermia, the motor score of the GCS gradually improved during the first 7 days after ROSC.
- At day 3, the current clinical practice parameters such as the motor score, pupillary reflex or corneal reflex were unreliable in predicting a poor outcome.
- The predictive value of EEG patterns in predicting a poor outcome was low, except for reactivity of the EEG to noxious stimuli.

Discussion & Conclusion (II)

- Treatment with mild therapeutic hypothermia and its concomitant administration of sedatives decreases the predictive value of the currently accepted clinical parameters.
- The neurological examination is of limited value in accurately predicting outcome.
- A combination of all 3 clinical parameters (pupillary, corneal reflexes and motor response) occurred in only a minority of patients with a poor outcome, resulting in a low sensitivity.

Discussion & Conclusion (III)

- Electrophysiological tests, combined with clinical parameters may facilitate prognostication in patients after cardiac arrest.
  - ✓bilateral absence of cortical N20 responses of median nerve SSEP 24 h after admission in patients treated without hypothermia
  - → invariably correlates with a poor neurologic outcome
  - ✓hypothermia does not change the accuracy of the SSEP for the prediction of permanent coma?
  - → the need for prospective studies to evaluate the predictive value of bilateral absent N20 responses after hypothermia

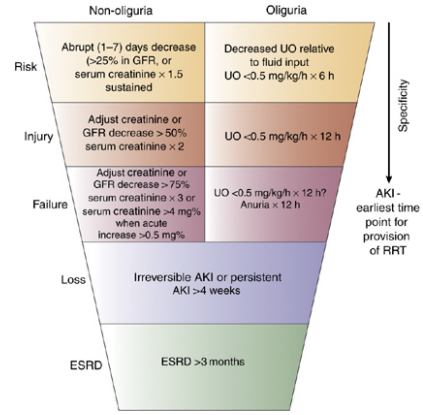
Discussion & Conclusion (IV)

- Known adverse EEG patterns such as burst-suppression, suppression or periodic epileptiform discharges failed to accurately predict outcome in our population.
  - ✓burst-suppression or isoelectric pattern
  - → associated with a poor outcome, but its specificity was not 100% and confidence intervals relatively wide
  - ✓After induction of hypothermia all patients initially demonstrate a slow burst-suppression EEG pattern with long periods of suppression, which gradually subsides.
  - ✓The occurrence of these patterns after rewarming is associated with a poor outcome, but fails the specificity to accurately predict the prognosis.

Discussion & Conclusion (V)

- ✓ An unreactive EEG background to noxious stimuli was strongly associated with a poor prognosis in our population.
- ✓ Since EEG reactivity had a false positive rate for mortality of 7%, it cannot be used as a single parameter for prognostication.

- No single clinical or electrophysiological parameter has sufficient accuracy to determine prognosis and decision making in patients after cardiac arrest, treated with hypothermia.
- Early prognostication in patients with post-anoxic encephalopathy will probably require a multimodal approach, combining a number of clinical and electrophysiological tests.



Kidney International 71, 971-976 (May (2) 2007)

表一：急性腎損傷之診斷標準[6]

	RIFLE Criteria (2002)		AKIN Criteira (2005)		
Stage	GFR Criteria	Urine Output Critieria	Serum Creatinine Criteria	Urine Output Criteria	Stage
Risk	↑ Scr X 1.5 or ↓ GFR > 25%	UO < 0.5 ml/kg/hr X 6 hrs	↑ Scr ≧ 0.3 mg/dl, or ↑ to ≧ 1.5- to 2-fold from baseline	UO < 0.5 ml/kg/hr ≧ 6 hrs	1
Injury	↑ Scr X 2 or ↓ GFR > 50%	UO < 0.5 ml/kg/hr X 12 hrs	↑ Scr > 2- to 3-fold from baseline	UO < 0.5 ml/kg/hr ≧ 12 hrs	2
Failure	↑ Scr X 3 or ↓ GFR > 75% or Scr ≧ 4 mg/dl (acute ↑ ≧ 0.5 mg/dl)	UO < 0.3 ml/kg/hr X 24 hrs or anuria X 12 hs	↑ Scr >3-fold from baseline, with an acute increase of at least 0.5 mg/dl or individual who received RRT	UO < 0.3 ml/kg/hr ≧ 24 hrs	3
Loss	Persistent ARF = complete loss of renal function > 4 wks		Diagnostic criteria: within 48 hrs - absolute increase in serum creatinine ≧ 0.3 mg/dl, or a percentage increase in serum creatinine ≧ 50%, or a reduction in urine output (< 0.5 ml/kg/hr > 6 hours)		
ESRD	End stage renal disease (complete loss of renal function > 3 months)				

Scr: serum creatinine; GFR: glomerular filtration rate; UO: urine output.

內科學誌 2009；20：320-334