Prediction of poor outcome within the first 3 days of postanoxic coma

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Abstract—*Objective:* To determine the optimal timing of somatosensory evoked potential (SSEP) recordings and the additional value of clinical and biochemical variables for the prediction of poor outcome in patients who remain comatose after cardiopulmonary resuscitation (CPR). *Methods:* A prospective cohort study was conducted in 32 intensive care units including adult patients still unconscious 24 hours after CPR. Clinical, neurophysiologic, and biochemical variables were recorded 24, 48, and 72 hours after CPR and related to death or persisting unconsciousness after 1 month. *Results:* Of 407 included patients, 356 (87%) had a poor outcome. In 301 of 305 patients unconscious at 72 hours, at least one SSEP was recorded, and in 136 (45%), at least one recording showed bilateral absence of N20. All these patients had a poor outcome (95% CI of false positive rate 0 to 3%), irrespective of the timing of SSEP. In the same 305 patients, neuron-specific enolase (NSE) was determined at least once in 231, and all 138 (60%) with a value >33 μg/L at any time had a poor outcome (95% CI of false positive rate 0 to 3%). The test results of SSEP and NSE overlapped only partially. The performance of all clinical tests was inferior to SSEP and NSE testing, with lower prevalences of abnormal test results and wider 95% CI of false positive rates. *Conclusion:* Poor outcome in postanoxic coma can be reliably predicted with somatosensory evoked potentials and neuron-specific enolase as early as 24 hours after cardiopulmonary resuscitation in a substantial number of patients.

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The prediction of poor outcome in patients who remain unconscious after cardiopulmonary resuscitation (CPR) has recently been addressed in a number of systematic reviews. A positive predictive value of 100% has been demonstrated for the absence of early cortical responses (N20) of the somatosensory evoked potentials (SSEPs) in the first week after CPR,[1-3] and similar values were found for the absence of pupillary and corneal reflexes and of any motor response 3 days after the hypoxic–ischemic insult.[1,4] The usefulness of biochemical markers of brain damage in serum or CSF remained uncertain, because of the small number of patients in most studies and methodologic flaws in some.[5] With these reviews, a number of questions could not be answered. 1) What is the earliest time when SSEP results may be considered reliable? 2) Are the predictive values of separate variables additive? 3) Can biochemical markers of anoxic–ischemic brain damage contribute to the prediction of poor outcome? We designed our study (Prognosis in Postanoxic Coma) to address these questions.

Additional material related to this article can be found on the *Neurology* Web site. Go to www.neurology.org and scroll down the Table of Contents for the January 10 issue to find the title link for this article.

Methods. From January 2000 to May 2003, we performed a multicenter prospective cohort study to correlate early clinical, neurophysiologic, and biochemical findings with clinical outcome. To study prediction in regular clinical practice, we chose a pragmatic design, with various types of hospitals and without centralized assessment of the neurophysiologic tests. CSF studies were not feasible with this design. Patients admitted to the intensive care units of 32 Dutch hospitals (13 teaching hospitals and 19 nonteaching hospitals) were included. The study was approved by the ethical review boards of all participating hospitals.

Consecutive patients with CPR for primary or secondary circulatory arrest, persisting coma 24 hours after CPR, age 18 years or older, and informed consent from a legal representative were included. We defined coma as no eye opening to external stimuli, motor response to pain flexion or worse, and no speech. Exclusion criteria were a life expectancy of no more than several months caused by pre-existent disease, death by brain criteria after 24 hours, and concomitant head injury.

Timing of assessments. Baseline characteristics registered were gender, age, medical history, prearrest level of functioning, cause of the arrest, location of arrest, cardiac rhythm before CPR, time between arrest and initiation of CPR, and duration of CPR. All patients underwent standard assessments 24, 48, and 72 hours (±4 hours) after CPR, including neurologic examination, median nerve SSEP, and blood sampling at all time points and EEG at 72 hours. For practical reasons, SSEP recording was not

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always possible on weekends. If the 72-hour SSEP was due on a weekend day, the recording was postponed to Monday.

Clinical assessment. Within the first 24 hours post CPR, sedatives and muscle relaxants were stopped. When this was not possible, propofol was used as the standard sedative, and this medication was stopped shortly before clinical assessment and EEG recording. Morphinomimetics and benzodiazepines were avoided as much as possible. Clinical features registered included cardiovascular stability (spontaneously stable, stable with medication, unstable), Glasgow Coma Scale, pupillary and corneal reflexes of each eye (present or absent), eye movements (spontaneously roving, on cervico-ocular testing, absent), and seizure or myoclonus (absent, sporadic, status). We also recorded the Acute Physiology and Chronic Health Evaluation-II (APACHE-II) score over the first 24 hours. Outcome was registered after 1 month using the Glasgow Outcome Scale. Poor outcome was defined as either death or persisting unconsciousness after 1 month. In patients who died, the probable cause of death was registered. In surviving patients, 1-year outcome was assessed by telephone contact with the patient, a family member, or the general practitioner of the patient.

SSEPs. We recorded SSEPs with standard procedures.[6,7] Local clinical neurophysiologists in each center assessed the recordings. The results for the N20 were documented for each side separately as absent (only in the presence of a cervical potential) or present or the recording was judged to be technically insufficient. For our primary analysis, we defined "SSEP absent" as N20 absent on both sides and "SSEP not absent" as all remaining combinations of left and right recordings.

EEG. Local clinical neurophysiologists assessed the EEG recordings. They coded their findings using the classification of Hockaday et al.[8] In addition, they registered the presence or absence of a burst-suppression pattern and of epileptiform activity (absent, sporadic, frequent, status).

Determination of S-100B and neuron-specific enolase. Blood sampling and measurements were done using standard procedures.[9] For our primary analysis, we defined abnormal values as >0.7 μg/L for S-100B and >33 μg/L for neuron-specific enolase (NSE), based on our meta-analysis in which these values resulted in the lowest false positive rates.[5]

Blinding. Treating physicians were blinded for the results of the first and second day SSEP and for all blood tests. Absence of SSEP at 72 hours was considered a sufficiently reliable predictor of poor outcome to allow its use for treatment decisions,[1-3] and the result of 72-hour SSEP testing was therefore made available to the treating physicians.

Treatment and treatment restrictions. The protocol prescribed no specific treatments. Standard care in Dutch intensive care units generally consists of standard supportive care, valproate or phenytoin for epilepsy, and valproate or clonazepam for myoclonus. During the last phase of the study, induced hypothermia (32 to 34 °C) was gradually introduced as a treatment of anoxic-ischemic brain damage.[10,11] These patients were included. From a methodologic point of view, it was desirable that all patients receive maximal treatment during a certain period, but this could not be realized on ethical and practical grounds. Treating physicians could decide to stop or forego further treatment of patients with established predictors of poor outcome. With regard to neurologic prognosis, they were offered the following guidelines: 1) Chances for survival or recovery of consciousness are virtually nil when the N20 of the SSEP or the pupillary reflexes or motor responses are absent or when the EEG is isoelectric at 72 hours or later; 2) chances for survival without severe disability are virtually nil with a burst-suppression EEG at 72 hours or later or with motor response flexion or worse at 7 days or later.[1,12] Timing and motivation of any change in treatment level were documented. Treatment levels were no treatment restrictions; treatment restricted to standard supportive care, such as can be given on regular wards (no-resuscitation order, weaning from the ventilator and transfer to the regular ward, no readmission to the intensive care unit; all other treatment given when necessary); and no treatment other than palliation.[13]

Analyses. We designed the study to test two hypotheses: 1) The bilateral absence of N20 of the SSEP 24 and 48 hours after CPR predicts poor outcome as accurate as the absence of such responses 72 hours after CPR; 2) in a number of patients in whom N20 of the SSEP is present in the first 72 hours after CPR, poor prognosis can be accurately predicted with clinical, EEG, or biochemical variables. Hypothesis 1 was tested with descriptive statistics on the basis of 2 × 2 tables with "bilateral absence of N20" and "poor outcome" as present or absent. We calculated false positive rates (patients with abnormal test result and favorable outcome/all patients with abnormal test result) and positive likelihood ratios with their 95% CIs. To calculate likelihood ratios when one of the cells of the 2 × 2 table contained no observations, we added a value of 0.5 to each cell. For the second hypothesis, we performed univariate analysis with similar descriptive statistics, using all remaining variables (table 1). We used predefined cut-off levels for the primary analysis of NSE and S100b levels (see above). The performance of these tests was further evaluated with receiver operating characteristic curves. Sample size calculation was based on 5% as the upper limit of the CI of a false positive rate for a prognostic variable, which means that we would need 80 patients to identify such a variable. With a conservative estimation of the prevalence of useful predictors of 20%, 400 patients were required to identify such variables reliably.

Table 1 *Variables and values used in analyses*

Clinical characteristics
• APACHE-II score during first 24 h (>25 vs ≤25)*
• Circulation (unstable vs spontaneously stable)
• Epilepsy or myoclonus (present vs absent)
• Pupillary reflexes (bilaterally absent vs. present)
• Corneal reflexes (bilaterally absent vs present)
• Eye movements (absent vs spontaneous or on cervico-ocular testing)
• Motor response (absent vs present spontaneously or with painful stimuli)
Neurophysiologic variables
• SSEP N20 (bilaterally absent vs not bilaterally absent)
• EEG at 72 h (Hockaday grade 5 vs others)*
• EEG at 72 h (burst suppression vs no burst suppression)*
• EEG at 72 h (epileptiform activity vs. no epileptiform activity)*
Biochemical variables
• Serum NSE (>33 vs ≤33 μg/L)
• Serum S-100B (>0.7 vs ≤0.7 μg/L)

All variables except (*) tested at 24, 48, and 72 (±4) h after cardiopulmonary resuscitation.

SSEP = somatosensory evoked potential; APACHE-II = Acute Physiology and Chronic Health Evaluation; NSE neuron-specific enolase.

Results. We included 407 patients in the study (see table E-1 on the *Neurology* Web site at www.neurology.org). Six centers included more than 20 patients, 8 centers between 20 and 10, and 18 centers fewer than 10. Poor outcome occurred in 356 (87%) of the patients, 349 of whom had died. Of the 51 patients who were conscious after 1 month, 34 were severely disabled, 10 moderately disabled, and 7 had made a good recovery. Of the 34 patients who were severely disabled after 1 month, 11 had died after 1 year, 13 remained severely disabled, and 10 had become independent (moderate disability or good recovery).

Characteristics of neurophysiologic, biochemical, and clinical tests are presented in tables 2 to 4. The absence of SSEP, EEG Hockaday grade 5, and serum levels of NSE of >33 μg/L were superior to all other variables with regard to the percentage of abnormal test results (high), false

Table 2 *Prediction of poor outcome with neurophysiologic variables*

Variable/time after CPR, h	Patients tested, n (patients without treatment restrictions)	Abnormal test result, % (95% CI)	False positive rate,* % (95% CI)	Positive likelihood ratio (95% CI)
No SSEP (N20) bilaterally				
24	254 (193)	38 (32–44)	0 (0–4)	29 (2–454)
48	246 (164)	36 (30–42)	0 (0–4)	27 (2–427)
72	281 (170)	41 (35–46)	0 (0–3)	25 (2–394)
24–72†	301 (175)	45‡ (40–51)	0 (0–3)	25 (2–383)
EEG no activity ≥20 μV				
72	282 (165)	28 (23–33)	0 (0–5)	17 (1–272)
EEG burst-suppression pattern				
72	276 (163)	8 (5–12)	0 (0–15)	5 (0–81)
EEG status epilepticus				
72	282 (166)	9 (6–13)	7 (1–24)	1 (0–5)

* Patients with abnormal test result and favorable outcome/all patients with abnormal test result (1 − positive predictive value).
† Refers to 305 patients who were still comatose after 72 h and in whom SSEP testing had been performed at least once.
‡ At least one abnormal test result.

CPR = cardiopulmonary resuscitation; SSEP = somatosensory evoked potential.

positive rates (low), and likelihood ratios (high). No false positive predictions were made with any of these variables, with the upper limit of the 95% CI of their false positive rates below 5%.

In 256 patients, at least two SSEP recordings hade been done. In nine of these, N20 was present in the first and absent in a later recording. In five other patients, N20 was absent at 24 hours, but the response recurred in a later recording. All these 14 patients had a poor outcome. In 167 patients, serum NSE had been determined at all three time intervals, and all 88 (53%) patients with at least one abnormal NSE value had a poor outcome. The results of SSEP recording and NSE determination only partially overlapped (see table E-2 on the *Neurology* Web site). All 192 patients in whom one of the tests was abnormal had a poor outcome (95% CI of the false positive rate 0 to 2%). Most of the abnormal test results were obtained 24 hours after CPR (74% of abnormal results for SSEP, 65% for NSE, and 78% for one of these tests). Overlap between SSEP and EEG results was more extensive, but still incomplete: Of all patients without absent SSEP, only 13% had a low voltage EEG (Hockaday grade 5; all voltages <20 μV) or a burst-suppression pattern. Of the 305 patients who were still unconscious after 72 hours, no abnormal test result for SSEP, NSE, or EEG was found in 105, 81 (77%) of whom had a poor outcome.

Receiver operating characteristic curves for the two biochemical tests validated the cut-off value for NSE we selected from our structured review,[5] but not that for S100b, which proved more time dependent than that of NSE (fig-

Table 3 *Prediction of poor outcome with biochemical variables*

Variable/time after CPR, h	Patients tested, n (patients without treatment restrictions)	Abnormal test result, % (95% CI)	False positive rate,* % (95% CI)	Positive likelihood ratio (95% CI)
NSE > 33 μg/L				
24	272 (206)	42 (36–48)	0 (0–3)	36 (2–563)
48	241 (157)	52 (46–59)	0 (0–3)	45 (3–715)
72	209 (108)	46 (40–53)	0 (0–4)	39 (3–610)
24–72†	231 (110)	60‡ (53–66)	0 (0–3)	23 (2–357)
S100b > 0.7 μg/L				
24	273 (207)	45 (40–51)	3 (1–8)	5 (2–12)
48	238 (155)	44 (38–50)	2 (0–7)	9 (2–36)
72	207 (108)	35 (29–42)	0 (0–5)	30 (2–466)
24–72†	230 (110)	53‡ (46–59)	2 (1–7)	3 (1–9)

* Patients with abnormal test result and favorable outcome/all patients with abnormal test result (1 − positive predictive value).
† Refers to 305 patients who were still comatose after 72 h and in whom NSE or S100b testing had been performed at least once.
‡ At least one abnormal test result.

CPR = cardiopulmonary resuscitation; NSE = neuron-specific enolase.

Table 4 *Prediction of poor outcome with clinical variables*

Variable/time after CPR, h	Patients tested, n (patients without treatment restrictions)	Abnormal test result, % (95% CI)	False positive rate,* % (95% CI)	Positive likelihood ratio (95% CI)
APACHE-II score >25				
First 24	366 (274)	51 (46–56)	8 (4–12)	2 (1–3)
Circulation unstable				
24	394 (299)	57 (52–62)	14 (10–19)	1 (1–1)
48	353 (238)	47 (41–52)	10 (6–15)	1 (1–2)
72	298 (175)	39 (34–45)	8 (4–14)	1 (1–2)
Epilepsy or myoclonus (no status)				
24	374 (301)	33 (29–38)	6 (3–12)	2 (1–4)
48	341 (237)	19 (14–23)	6 (2–16)	2 (1–5)
72	292 (177)	14 (10–18)	2 (0–13)	4 (1–29)
Myoclonus status				
24	396 (301)	4 (2–6)	0 (0–21)	5 (0–81)
48	350 (237)	1 (0–3)	0 (0–52)	1 (0–25)
72	300 (177)	2 (1–4)	0 (0–52)	1 (0–20)
Status epilepticus				
24	395 (301)	2 (1–4)	0 (0–41)	2 (0–39)
48	351 (237)	1 (0–3)	0 (0–60)	1 (0–21)
72	300 (177)	1 (0–3)	0 (0–71)	1 (0–14)
No pupillary or corneal reflexes				
24	386 (301)	12 (9–16)	4 (0–14)	4 (1–15)
48	338 (236)	12 (8–15)	2 (0–13)	5 (1–36)
72	289 (177)	13 (9–17)	0 (0–9)	8 (1–121)
No motor response				
24	395 (300)	59 (54–64)	9 (6–14)	1 (1–2)
48	351 (236)	44 (38–50)	6 (3–10)	1 (1–2)
72	300 (177)	35 (29–42)	5 (2–9)	2 (1–4)

* Patients with abnormal test result and favorable outcome/all patients with abnormal test result (1 − positive predictive value).

CPR = cardiopulmonary resuscitation; APACHE-II = Acute Physiology and Chronic Health Evaluation.

ure). With the mandatory high specificity, sensitivity of NSE testing is clearly superior to that of S100b testing.

Treatment had been restricted in 23% of patients at 24 hours and in 28% at 48 hours. In all but two patients, these restrictions were of category B. Despite blinding of the treating physicians for the 24- and 48-hour SSEP and all NSE values, treatment was more often restricted in patients with absent SSEP and abnormal NSE values (see table E-3 on the *Neurology* Web site).

Only 10 patients were treated with induced hypothermia, 8 of whom had a poor outcome (not significantly different from untreated patients). These 10 patients included 4 in whom at least one SSEP had been absent and 3 in whom serum NSE had been above the cut-off value at least once; all these patients had a poor outcome.

Discussion. We have demonstrated that the bilateral absence of the N20 of the SSEP in patients with postanoxic coma of at least 24 hours' duration is invariably associated with poor outcome. In 45% of patients, SSEP were absent within the first 3 days after CPR, in about three-fourths of these already after 24 hours. Absent SSEP were found more often than any other neurophysiologic or clinical predictor with 100% predictive value. Serum NSE above 33 μg/L proved to be equally accurate, with a prevalence of 60%. An important additional finding, confirming earlier reports,[14,15] was the incomplete overlap of the results of SSEP and NSE. The prevalence of at least one abnormal test result derived from patients in whom both tests were performed was 66%. Finally, in patients with no absent SSEP and NSE ≤33 μg/L, a small number with poor outcome could be identified with EEG (burst suppression or no voltage >20 μV). Of all 356 patients with poor outcome, this outcome could be reliably predicted with these three variables in the first 3 days after CPR in 252 (71%).

We tested a large number of variables (see table

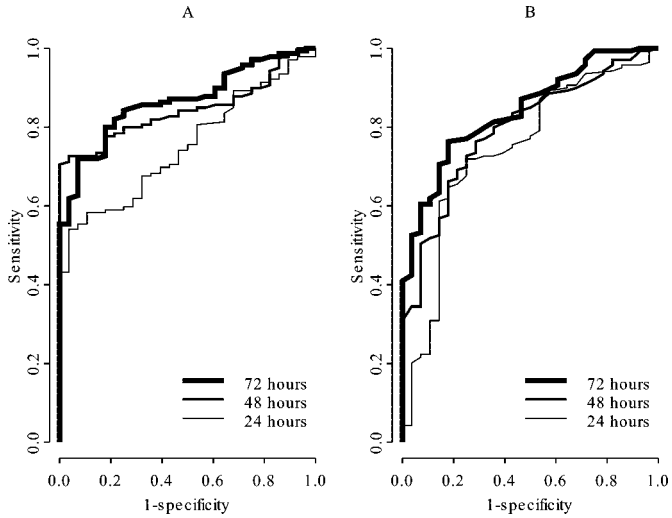

Figure. Receiver operating characteristic curves for the prediction of poor outcome by serum levels of neuron-specific enolase (NSE) (A) and S100b (B) at 24, 48, and 72 hours after cardiopulmonary resuscitation. At (1 − specificity) = 0, maximum cut-off levels for NSE are 31.7 μg/L at 24 hours, 23.8 at 48 hours, and 32.3 at 72 hours; for S100b, 6.9 μg/L at 24 hours, 1.1 at 48 hours, and 0.7 at 72 hours.

1), and the 100% predictive value of some of these could well have arisen by chance. This is borne out by the fact that some patients (2/57) have been reported with NSE values >33 μg/L who survived.[5,15] On the other hand, the low false positive rates for SSEP and serum NSE confirm our previous findings[1,5] and are supported by subsequently reported data.[2,3,14-16] Our results must therefore be regarded sufficiently robust to warrant application in clinical practice.

The patient characteristics at inclusion do not differ from previously described cohorts with regard to age and sex distribution, initial rhythm disturbance, proportion of out-of-hospital arrests, and comorbidity. The proportion of patients with poor outcome (87%) is at the upper end of the wide range reported for both retrospectively and prospectively studied cohorts, with mortality rates varying from 28 to 92%.[1] The main difference between these cohorts and the one we studied was the time of inclusion: We included only patients still unconscious 24 hours after CPR, whereas all other studies included patients with much shorter periods of unconsciousness, varying from the time of return of spontaneous circulation to a few hours thereafter. Because most patients who recover consciousness do so within the first day, inclusion of patients at a later stage explains the high proportion of patients with poor outcome in our cohort.

We have defined death or persisting unconsciousness after 1 month as "poor outcome." This was based on our previous argumentation that the chance of recovery of consciousness in patients who are still unconscious 1 month after CPR is virtually nil, especially when the SSEPs are absent.[1] Patients who were conscious but severely disabled after 1 month were not included in the "poor outcome" category, because further improvement in these patients may occur. In our cohort, one-third of such patients lived independently 1 year after the cardiac arrest. One could argue that prediction of poor outcome should be aimed at the clinical condition after a longer period, say 1 year, and should then include "severe disability," a condition many patients would like to avoid. We could not find useful differences between the small numbers of patients who were severely disabled or living independently after 1 year. Obviously, a major effort would be required to find reliable rules for such a prediction.

Regular clinical practice, with its more or less tacit knowledge about predictors of poor outcome, is incompatible with a strict experimental study design. The tendency to restrict treatment selectively in patients with characteristics recognized or presumed to be predictive of poor outcome may then result in the finding that such characteristics are indeed good predictors of poor outcome (the fallacy of a "self-fulfilling prophecy"). We could not (and did not wish to) prevent physicians to restrict treatment. In our protocol, we provided guidelines for such decisions, with as most important characteristic the postponement of such decisions to at least 72 hours after CPR. Despite this, some restriction had been implemented in 23% of patients after 24 hours and in 28% after 48 hours and significantly more often in patients with absent SSEP or high values of serum NSE at these time points, test results of which the treating physicians were not aware. This could be explained by the higher prevalence in these patients of clinical variables generally recognized to be associated with poor outcome (see table E-3 on the *Neurology* Web site). An alternative or complementary explanation of insufficient blinding would only apply to the SSEP results and therefore seems unlikely. It is apparent that selective treatment restrictions are difficult to prevent. The main conclusions of our study, however, are not invalidated by this finding, because two-thirds of the patients with absent SSEP at 24 and more than half at 48 hours were treated without restrictions. Despite this maximal treatment, all these patients had a poor outcome.

During the last phase of our study, induced hypothermia (32 to 34 °C) became gradually accepted as a treatment of anoxic–ischemic brain damage.[10,11] Although we have included some patients who were treated with hypothermia, our results are essentially derived from patients in whom no such treatment has been given. The question then is whether our results could be applied to patients treated with hypothermia. The need for predicting outcome and changing treatment options accordingly only arises, of course, after the treatment, that is, when the patient is normothermic again. Because the effects of low body temperature on SSEP are immediately reversed with rewarming, SSEP can then be used safely. Furthermore, cooling to 30 °C influences the

latencies of the cortical responses, but not the responses themselves.[17,18] All this has recently been substantiated in a study in which all patients with postanoxic coma who were treated with hypothermia and had absent SSEP while hypothermic had a poor outcome.[19] In another study, serum NSE levels at 24, 36, and 48 hours associated with poor outcome were significantly higher in patients treated with induced hypothermia compared with those of untreated patients.[16] However, in no patient in this series, irrespective of treatment and outcome, values greater than 33 μg/L were found, so that no false prediction of poor outcome would have been made when this cut-off had been applied. We conclude that SSEP recording and serum NSE determination can be used in patients who are treated with hypothermia.

The mainstay of outcome prediction has always been the clinical neurologic examination, and the algorithms of Levy et al.[12] based on eye opening, motor response, and brainstem reflexes have been widely used for nontreatment decisions. Direct comparison of clinical and laboratory tests has been rare, and structured reviews could only summarize evidence for separate categories of tests.[1-5] With our study, we could make such a comparison, and it is evident that the predictive value of some laboratory tests (SSEP and serum NSE) is superior to that of clinical tests in terms of low false positive rates and high prevalence of abnormal test results. Based on all available evidence,[1-5,14-16] confirmed and extended by the current results, we therefore propose the following strategy: Patients who are still unconscious at least 24 hours after an anoxic–ischemic insult undergo SSEP recordings and determination of serum NSE. When N20 is bilaterally absent or serum NSE is >33 μg/L, further treatment will be withheld. When equivocal SSEP recordings are obtained and serum NSE is ≤33 μg/L, repeat SSEP testing is indicated. Repeat testing in patients in whom both tests were initially normal may identify additional patients with poor prognosis within the first 72 hours. In patients with normal SSEP and NSE results after 72 hours, additional findings that are sufficient to forego further treatment are absence of corneal or pupillary reflexes after 72 hours, or an EEG with burst suppression, or minimal or absent cortical activity. With this proposed testing strategy, most patients who will not recover consciousness can be reliably identified within the first 3 days after CPR. Finally, we emphasize that no tests are available that can reliably predict the recovery of consciousness or the quality of life in surviving patients.

Appendix

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Neuro*Images*

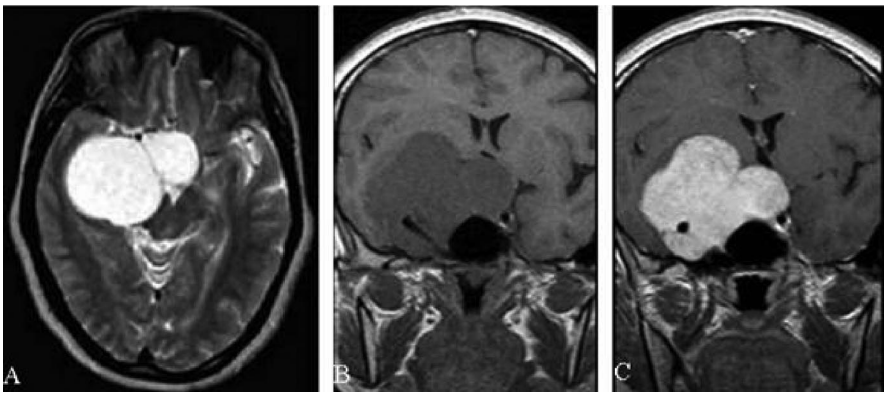

Figure. (A) Axial T2-weighted MR image reveals a well-circumscribed, large, lobulated, markedly hyperintense lesion in the right cavernous sinus with mass effect on the midbrain. (B) Coronal T1-weighted MR image shows a well-circumscribed, large, lobulated, hypointense lesion in the right parasellar region with encasement of the right internal carotid artery and sellar extension. (C) Coronal T1-weighted postgadolinium MR image shows an intense and homogenous enhancement of the lobulated right cavernous sinus lesion.

Cavernoma of cavernous sinus

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A 40-year-old woman presented with right-sided ophthalmoplegia, ptosis, and retro-orbital pain. MRI showed a large, lobulated, hyperintense lesion in the right cavernous sinus on T2-weighted image (figure, A). The lesion was hypointense on T1-weighted image (figure, B), with intense enhancement (figure, C). The imaging differentials are cavernoma, meningioma, and schwannoma. Cavernous sinus cavernoma is a rare vascular malformation, which represents 3% of all benign cavernous sinus tumors.[1] Marked hyperintensity on T2-weighted images with intense and homogenous enhancement are characteristic. Red cell–labeled blood pool scintigram is more specific for diagnosis.[2] Preoperative diagnosis is crucial because of intraoperative profuse hemorrhage.

The authors report no conflicts of interest.

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