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The usefulness of EEG, exogenous evoked potentials, and cognitive evoked potentials in the acute stage of post-anoxic and post-traumatic coma

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Abstract

Three-modality evoked potentials (TMEPs) have been used for several years in association with the EEG as a diagnostic and prognostic tool in acute anoxic or traumatic coma. Cognitive EPs have been recently introduced. EEG and cognitive EPs provide functional assessment of the cerebral cortex. TMEP parameters can be described by two indices : the index of global cortical function (IGCF) and the index of brainstem conduction (IBSC).

Although it remains a unique tool for epilepsy assessment, the value of EEG is largely limited by its high sensitivity to the electrical environmental noise, its dependence on sedative drugs, and its inability to test the brainstem. Major TMEP alterations (absence of cortical activities more than 24 hours after the onset of post-anoxic coma, major pontine involvement in head trauma) are associated in all cases with an ominous prognosis (death or vegetative state). However, even if mild TMEP changes are associated with a good prognosis in 65% (post-anoxic coma) to 90% (head trauma) of cases, some patients never recover despite exogenous TMEPs that are only mildly altered in the acute stage. Thus, cognitive EPs can usefully complement exogenous EPs as a prognostic tool in coma. Indeed, even if the absence of cognitive EPs in comatose patients does not have any prognostic value, their presence implies a very high (more than 90%) probability of consciousness recovery. The major technical challenge for the future will be the development of reliable tools for continuous EEG and TMEP monitoring.

Key words : EEG ; evoked potentials ; coma ; brain death ; prognosis ; anoxia ; head trauma**.**

Introduction

Neurophysiological methods (electroencephalogram : EEG, evoked potentials : EPs) provide functional assessment of the central nervous system (CNS). As such, their scope is similar to that of neurological examination (Glasgow Coma Scale : GCS, Glasgow-Liège Coma Scale : GLCS, focal nervous dysfunction) and complementary to that of structural methods (CT-Scan, magnetic resonance imaging : MRI). Indeed, CNS dysfunction can occur in the absence of anatomical lesions and CNS lesions can provoke functional dirsturbances that are outside the domains of clinical or electrophysiological examinations. Neurophysiological methods can be used as an adjunct to diagnose the origin of comatose states, as a tool to predict outcome, and for monitoring purposes. Some extensive reviews have been recently published on that subject (Chatrian *et al.*, 1996 ; Guérit, 1999a ; Guérit *et al.*, 1999a).

This paper will summarize our experience of EEG and EP recording at the acute stage of post-anoxic and post-traumatic coma. The domains and limits of neurophysiological methods will be reminded in a first step. Some relevant pathophysiological mechanisms that are involved in brain anoxia and head trauma will be presented in a second step. In a third step, we will present the main EEG and EP features of post-anoxic and post-traumatic coma and show how these can be explained by pathophysiology. The way by which these features can be used for diagnosis and prognosis and how to integrate these with clinical and structural examinations will be presented in a fourth step. In a fifth step, we will present some future directions for continuous neuromonitoring.

This paper will only summarize the rationale of our methods and the reader is referred to our previous papers for further methodological details (Guérit, 1992, 1999 ; Guérit *et al.*, 1993, 1999b).

Domains and limits of EEG and EPs (Table 1)

EEG

The EEG reflects cortical neuronal activity modulated by both physiological and pathological diencephalic and brainstem influences and possibly affected by metabolic and/or toxic factors. This explains that many abnormal EEG patterns are

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Table 1

Main neurophysiological tools available in the ICU (reprinted from Guérit, 1999a, with permission)

Neurophysiological tool	Components	Normal latency range (ms)	Corresponding central or peripheral nervous structures
EEG			Cerebral cortex, brainstem modulation
Cognitive EPs	MMN	< 200	Auditory cortex
	P300	> 300	Associative cortex, brainstem modulation
Visual EPs	peak I	< 60	Retina
Flash	peak III	< 100	Occipital cortex
	peak VII	150 - 250	Associative cortex, brainstem modulation
	RAD	> 250	Occipital cortex, brainstem modulation
Somatosensory EPs	Erb's point	< 12	Peripheral nerve
Median nerve	N13	12 - 16	Spinal cord (cervical)
	P14	13 - 18	Medulla
	P14-N20	< 7	Brainstem + subcortical transmission time
	N20	18 - 25	Parietal cortex (area 3b)
	N30	28 - 35	Frontal cortex
Auditory EPs	BAEP I	< 2	Auditory nerve
	BAEP II-V	< 6	Pons
	Middle-latency	<90	Auditory cortex
	Long-latency	> 100	Associative cortex, brainstem modulation

MMN : mismatch negativity.
RAD : rhythmic after-discharge.

highly unspecific as they can reflect either primary cortical dysfunction or abnormal influences on an otherwise healthy cerebral cortex. For example, diffuse EEG slowing or even an almost isolectric pattern can reflect direct, prognostically-relevant, cortical involvement in post-anoxic coma but can also be the consequence of associated metabolic or toxic influences and recover as soon as these influences have been removed. Another limitation of the EEG, which is not shared by EPs, is its major sensitivity to the electrical environmental noise, which can make it hardly interpretable, or even ambiguous, especially when high amplifications must be used as, for instance, in brain death (BD) confirmation. The main advantage of the EEG is that it provides on-line assessment of the cerebral cortex, contrary to the EPs, which summarize the functional status of the tested CNS structures over the time needed for averaging. This makes the EEG a unique tool for examining rapidly changing cortical states (interictal transients, seizures, triphasic waves, periodic patterns, sleep alternances, cortical reactivity to stimulations, short-term drug influences). Undoubtedly, the recent developments in EEG quantification will re-enhance its role in these circumstances (see last paragraph on continuous neuromonitoring).

EPs

Short-latency EPs (brainstem auditory EPs : BAEPs, short-latency somatosensory EPs : SEPs) evaluate the peripheral sensory apparatus (ear, peripheral nerve, spinal cord), the brainstem, the subcortical somatosensory pathways, and the primary parietal cortex (N20). Middle-latency EPs assess the temporal (middle-latency auditory EPs : MLAEPs), parietal and frontal (middle-latency SEPs), and occipital (visual EPs : VEPs) cortex. Both short- and middle-latency EPs provide direct assessment of the corresponding structures and are less influenced by the non-specific factors which can alter the EEG. Long-latency EPs depend on multiple, ill-defined, cortical generators and can be subdivided into exogenous EPs and cognitive EPs. These are actually under similar influences as the EEG.

Two main advantages of EPs, when compared to the EEG, are their relative insensitivity to the environmental noise (owing to the averaging process) and, before all, the fact that they provide straighthforward brainstem assessment. Two limitations of EPs must also be pointed out : first, they only provide assessment of the sensory pathways and can, therefore, remain unaltered in the presence of an anterior dysfunction limited to the motor pathways (this limitation can, at least theoretically, be bypassed by motor EPs to transcranial magnetic stimulation) ; and, second, they only provide reliable CNS assessment in the absence of major sensory pathologies, which is not guaranteed in comatose patients (cochlear involvement due to anoxia, peripheral nerve, spinal cord, VIIIth nerve or optic nerve lesions due to trauma).

EEG and EP semiology in coma

Overall, our neurophysiological assessment of comatose patients is based on three techniques : EEG, three-modality exogenous EPs : TMEPs

(VEPs, SEPs, and BAEPs), and cognitive EPs (P300).

Table 2 shows the main EEG alterations most commonly observed in the ICU.

TMEP recording gives rise to several parameters, which are synthetized by two indices : the index of global cortical function (IGCF) and the index of brainstem conduction (IBSC) (Guérit *et al.*, 1993). The IGCF is determined on the basis of VEPs and cortical SEPs, according to the rules described in Table 3. It is expressed in grades (1 to 4), which presents the advantage of coding the results into a language that is easily understood by intensivists. The IBSC is, first quantitatively, and then qualitatively, determined, according to the rules described in Table 4.

Cognitive EPs are obtained with a passive odd-ball auditory paradigm and are rated in qualitative terms of their presence or absence. More details about cognitive EP assessment in comatose patients can be found elsewhere (Fisher *et al.*, 1999, Guérit *et al.*, 1999b, Kane *et al.*, 2000).

Pathophysiology of post-anoxic and post-traumatic coma

The sensitivity of any neurophysiological technique depends on its ability to evaluate the CNS structures that are the most sensitive to the pathophysiological process at the origin of coma, which, in turn, depends on coma aetiology.

Pathophysiology of brain anoxia

Two factors determine the sensitivity of a given brain structure to global brain hypoperfusion : its basal metabolic rate and its situation with respect to the major vessels. The higher basal metabolic rate of the cerebral hemispheres, when compared to the brainstem, and of the grey matter, when compared to the white matter, explains the elective sensitivity of the cerebral cortex and hippocampus, and the relative brainstem resistance. The second factor explains, in addition, the elective sensitivity of the medio-frontal and parietal cortices. Consequently, the EEG, cognitive EPs, and the IGCF are the most sensitive parameters, while the IBSC is less likely to be affected and, as a consequence, is actually irrelevant for prognosis.

Pathophysiology of traumatic coma

The pathophysiology of head trauma is more complex as three main factors determine both the current medical status and the patient outcome : (1) the presence, location, and degree of reversibility of primary focal brain lesions, which can involve either the cerebral hemispheres or the brainstem (especially the midbrain) ; (2) the extent of the dif-

Table 2

EEG features in the ICU (1) (reprinted from Guérit *et al.*, 1999a, with permission)

1. Background activity

1.1. Normal alpha (occipital, reactive to eye opening)
1.2. «Alpha coma» (frontal, areactive)
1.3. Drug-induced activities in the alpha range (fronto-central)
1.4. Theta
1.5. «Theta coma»
1.6. Beta (symmetric/asymmetric)
1.7. Delta (diffuse)
1.8. Delta (focal)
1.9. Spindles (symmetric/asymmetric)

2. Symmetry, reactivity, variability

2.1. Asymmetry (not posterior)
2.2. Posterior suppression
2.3. Reactivity
  2.3.4. Voltage reduction
  2.3.5. K-complexes
  2.3.6. prolonged bursts of delta waves
2.4. Variability

3. Additional patterns (non pathological)

3.1. K-complexes

4. Additional patterns (pathological)

4.1. Intermittent Rythmic Delta Activity (IRDA) (frontal or occipital)
  4.1.1. related to stimulation
  4.1.2. unrelated to stimulation
4.2. Triphasic waves
4.3. Episodic Low-Amplitude Events (ELAE)
4.4. Alternating pattern (related to Cheynes-Stokes respiration)
4.5. Epileptiform activity
  4.5.1. Generalized
  4.5.2. Periodic Lateralized Epileptiform Discharges (PLEDs)
  4.5.3. Focal spikes
4.6. Burst suppression
4.7. Periodic spiking
4.8. Low voltage Pattern
4.9. Electrocerebral Silence

(1) (adapted from Rae-Grant *et al.*, 1991 and Niedermeyer and Lopes da Silva, 1982).

Table 3

Determination of the index of global cortical function (IGCF) [reprinted from Guérit, 1999a with permission]

	VEPs	SEPs
Grade 0	Normal	Normal
Grade 1	Increased peak III latency Peak VII present	Normal N20, P24, and P27 N30 present
Grade 2	Increased peak III latency Peak VII absent	Normal N20 and P24 N30 absent
Grade 3	Increased peak III latency No subsequent activities	Normal N20 No subsequent activities
Grade 4	No reproducible VEPs ERG present	No cortical activities P14 present

Table 4

Qualitative brainstem assessment [reprinted from Guérit, 1999a, with permission]

Level of brainstem lesion	SEPs	Middle-latency AEPs	BAEPs
Midbrain	Normal P14 N20 delayed or absent	Abnormal	Normal
Pons	Normal P14 N20 delayed or absent	Abnormal	Abnormal
Medulla	Absent P14 N20 delayed or absent	Normal	Normal

fuse axonal injuries in the cerebral hemispheres ; and (3) the immediate and long-term consequences of brain edema and subsequent increases in intracranial pressure (brain herniation). That is, both the IGCF and IBSC can be altered – and prognostically relevant – in head trauma.

EEG and EP features of post-anoxic and post-traumatic coma

Post-anoxic coma

Overal, anoxic comas are characterized by a dissociation between abnormal EEG and IGCF, and preserved IBSC (in the absence of other factors interfering with the IBSC : alcoholism, diabetes, kidney insufficiency, drugs). This could be forecasted on the basis of pathophysiology. Increasing degrees of brain anoxia first give rise to aspecific EEG slowing (with or without reactivity) followed by the appearance of what is considered as the "malignant EEG patterns" (low-amplitude delta, alpha coma, burst suppression). Theoretically, electrocerebral silence (ECS) may occur. All degrees of IGCF involvement can be observed in exogeneous EPs, from Grade 1 to 4, the prognostic value of these will be dealt with in the next section. Although BAEPs are usually normal even in the presence of major IGCF alterations, transient alterations due to post-anoxic cochlear involvement can occur at the very acute stage of coma (Sohmer *et al.*, 1986).

Post-traumatic coma

Traumatic comas are associated with more complex neurophysiological patterns, which can be explained by the, also more complex, pathophysiology of head trauma. Four qualitative patterns were described (Guérit *et al.*, 1993) :

• Pattern 1 is similar to that seen in anoxia with EEG and IGCF alterations, which never exceed Grade 2, and IBSC preservation. This pattern corresponds to brain edema without any brainstem involvement.

• Pattern 2 corresponds to midbrain dysfunction. It reflects the elective midbrain sensitivity to brisk head deceleration. The EEG and IGCF are variably altered. BAEPs are usually normal or merely exhibit modifications of the V/I amplitude ratio. SEPs are characterized by a normal P14 contrasting with unilateral or bilateral abnormalities of N20, which can be delayed or absent. Midbrain dysfunction can also be demonstrated on the basis of a dissociation between normal BAEPs and abnormal MLAEPs (Fischer *et al.*, 1994). As we will see later on, the major interest of Pattern 2 is that it can remain compatible with good outcome, despite major clinical alterations (GCS equal or lower than 4).

• Pattern 3 is characterized by the association of deeply altered IGCF (Grade 3 or 4) and signs of pontine involvement, as demonstrated by BAEP destructuration. It corresponds to transtentorial herniation.

• Pattern 4 is that of BD. It is characterized by the absence of activities of intracranial origin contrasting with the preservation of extracranial activities (electroretinogram, sensory nerve action potential, cervical components) (Guérit, 1992). The interest of EPs to confirm BD will be discussed later on.

Clinical use of EEG and EPs in coma

As we have seen in the introduction, EEG and EPs can be used for diagnosis, prognosis, and as a continuous neuromonitoring tool. Although their domain is similar to that of the clinical examination, they present two major advantages : their insensitivity to muscle blockers, which makes these tests particularly valuable in curarized patients who cannot be clinically evaluated, and the fact that they provide more precise quantitative data that can be used more easily and more completely than the clinical evaluation for the assessment of the patient's evolution.

Diagnosis and prognosis will be dealt with in this section and the future perspectives of continuous neuromonitoring will be considered in the last paragraph.

Diagnosis

Owing to their lack of aetiologic specificity, the EEG and EPs are seldomly used for diagnosis, except in some circumstances : the differentiation between toxic-metabolic factors and structural lesions in comas of unknown aetiology, the diagnosis of brainstem lesions, the diagnosis of de-efferented states and of psychogenic unresponsiveness, and BD confirmation.

Differentiation between toxic-metabolic factors and structural lesions

The EEG features which support the metabolic/toxic hypothesis are the presence of triphasic waves, suggesting a metabolic encephalopathy, and the presence of beta rhythms, which favors the toxic hypothesis (although beta rhythms can also be observed in upper brainstem lesions interrupting the cholinergic influences on the thalamus). For the EPs, mere latency prolongations in the absence of morphological alterations favour the hypothesis of dysfunction of structurally intact sensory pathways and are usually prognostically irrelevant.

Diagnosis of brainstem lesions

Especially when the hazard of patient transportation contra-indicates CT-Scan or MRI, the EPs remain a unique tool to demonstrate brainstem dysfunction interfering with BAEPs and/or SEPs. In some instances, BAEPs can demonstrate unexpected brainstem lesions in patients who were initially considered as post-anoxic, in which case heart failure or respiratory arrest are likely to be the consequence of primary brainstem damage rather than the primary cause of coma.

The locked-in syndrome and psychogenic unresponsiveness

In the absence of other interfering factors, both cognitive and exogeneous EPs are normal in psychogenic unresponsiveness. The EEG can be normal or of low-voltage, in which case its reactivity should be cautiously tested.

In the locked-in syndrome, the EEG and VEPs are usually normal. Cognitive EPs are theoretically normal but the auditory P300 can be absent if the brainstem lesion interrupts the auditory pathways, in which case it can be useful to record visual P300s. Similarily, the BAEPs and SEPs are variably involved as a function of the extent of pontine damage. Motor EPs can confirm the involvement of the cortico-spinal tracts (Facco *et al.*, 1993).

Prognosis

Influence of non-cerebral factors

Before using the EEG and EPs as tools for prognosis, it is mandatory to verify that their alterations are not the consequence of interfering, primary non-neurological, factors. The main factors that are liable to interfere are metabolic disturbances and sedative drugs, body temperature, and peripheral sensory pathologies. Note that the latter can interfere, not only with sensory EPs, but also with EEG reactivity.

To deal with non-neurological factors is sometimes a very difficult task. As a rule of thumb, the prognosis associated with a given degree of EEG or EP alteration is all the more favorable as another non-neurological factor is liable to cause similar alterations. This holds especially true for any EEG alteration (except the burst-suppression pattern) and mild to moderate IGCF changes (Grade 1 to 3), while non-neurological factors almost never account for major IGCF alterations (Grade 4) or BAEP changes suggesting structural pontine lesions.

Post-anoxic coma

The value of the EEG to predict outcome in post-anoxic coma is limited by the widespread use of sedative drugs. The prognostically relevant EEG patterns are a mildly altered EEG with good reactivity, which usually implies a good prognosis, and the malignant EEG changes, which are usually (but not systematically) associated with an ominous prognosis (Berkhoff *et al.*, 2000).

The prognostic value of exogenous EPs depends on the degree of IGCF changes. In our series, Grade 1, 2, 3, and 4 IGCF observed between the 1st and the 3d day after the acute episode were associated with 65%, 40%, 15% and 0% of good outcomes (characterized by the ability to regain independent life, irrespective of whether cognitive functions fully recover or not), respectively (Guérit *et al.*, 1993).

Two points are worth being emphasized :

1. The fact that all patients with Grade 4 IFGC (that is, absence of cortical components including VEPs and SEP N20) eventually died or remained vegetative is in keeping with the rest of the literature. In particular, a recent meta-analysis of Zandbergen *et al.* (1998) confirmed that SEP recording is the most useful method for predicting poor outcome in anoxic-ischaemic patients. Practically, the observation of Grade 4 IGCF at least 24 hours after the acute episode questions the ethical acceptability of pursuing resuscitation in these hopeless cases.
2. The fact that even the mildest IGCF alterations remain associated with a poor outcome in 35% of cases, which underlines the relatively low value of exogenous EPs for predicting good outcome in anoxic-ischemic coma. We have recently conducted a cognitive EP study in the acute stage of anoxic coma (Guérit *et al.*, 1999b). In keeping with other recent studies (Fischer *et al.*, 1999 ; Kane *et al.*, 2000), we have shown that, while the absence of cognitive EPs does not allow to draw any conclusion in terms of poor outcome, their presence predicts consciousness recovery (with possible cognitive sequellae) in more than 90% of cases. Practically, we recommend first to record exogenous EPs to identify the worst cases and, in the absence of major IGCF alterations, to record cognitive EP in order

to identify the cases with a maximal probability of consciousness recovery.

Head trauma

The EEG seems even more unrewarding for predicting outcome in head trauma than in brain anoxia except for the persistence of EEG reactivity, which is correlated with a good outcome (Rae-Grant *et al.*, 1991 ; Gutling *et al.*, 1995). The prognostic value of exogenous EPs actually depends on which EP pattern is observed :

1. Pattern 1 predicts a good outcome in 80% (IGCF Grade 2) to 90% (IGCF Grade 1) of the cases. That is, for a given IGCF grade, the prognosis of traumatic patients looks better than that of anoxic patients, which is likely to be explained by the fact that the functional alterations observed in head trauma could reflect reversible brain edema, while similar alterations in brain anoxia would reflect irreversible cytotoxic neuronal damage.
2. Although worse than that of Pattern 1, the prognosis of patients with Pattern 2 (midbrain dysfunction) seems to depend on two factors : the reversibility of the midbrain dysfunction and the extent of associated diffuse axonal lesions. This justifies to perform MRI, whenever possible. Noteworthy, Pattern 2 can be associated with a good outcome despite a very poor clinical examination (GCS 4) and recovery can sometimes occur after a very long period during which the patient is considered as vegetative (up to 6 months in one patient of our series). Hantson (personal communication) recently observed that evidence of midbrain dysfunction could be demonstrated in most traumatic cases presenting at admission with GCS<8 and normal CT scan and that more than 80% of these patients eventually recovered.
3. Pattern 3 (transtentorial herniation) has been associated with death in all patients of our series and Pattern 4 is that of BD, which, by definition, is equivalent to individual death.

Although they provided interesting results in some patients, the specific interest of cognitive EPs seems less evident in head trauma than in brain anoxia. This is likely to be explained by the higher probability of focal brain damage interfering with P300 genesis.

Importance of the time elapsed from the acute episode

The prognostic value of both the EEG and the EPs depends on the time elapsed from the acute episode, esepecially in post-anoxic coma. Indeed, important alterations should be more cautiously interpreted within the first 24 hours after the acute episode, while the better prognostic value of mild alterations vanishes in patients who are still unresponsive more than 7 to 10 days after the acute episode. This finding can be explained by the fact that both reversible neuronal sideration and irreversible neuronal lesions account for the neurophysiological abnormalities that are observed at the acute stage of coma, while only irreversible structural neuronal lesions can explain equivalent EEG or EP changes that are observed later on.

EEG and EP confirmation of brain death

BD corresponds to the irreversible destruction of the whole encephalon, including the brainstem. The EEG is isoelectric. All EPs intracranially generated are lost while activities of extracranial origin are preserved. Practically, VEPs are restricted to the electroretinogram, BAEPs are either null or restricted to peak I, and SEPs are restricted to peripheral nerve and cervical activities (Guérit, 1992).

Fortunately, and contrary to what occurred in many other countries, the Belgian law doesn't impose any precise test to confirm BD. The diagnosis of BD is basically clinical (coma of known origin, areactivity, loss of brainstem reflexes, apnea). Confirmatory tests are mandatory whenever one has to rule out the possible role of misleading factors (CNS depressants, deep hypothermia, encephalitis, polyradiculopathy with cranial nerve involvement). They are also useful in that they can provide redundancy and, therefore, increased diagnostic safety.

Confirmatory tests can be subdivided into neurophysiological tests (EEG, EPs) and tests that directly assess intracranial circulation (four-vessel arteriography, radio-isotopic studies, brain-imaging techniques, and transcranial Doppler). We have recently reviewed the respective interests of the currently available methods for BD confirmation (Guérit, 1999b). Three criteria should determine the choice of the test used to confirm BD : it should yield vivid and unambiguous results, it should be conducted at the patient's bedside, and it should rule out the influence of misleading conditions (Task Force on Death and Dying of the Institute of Society, Ethics, and the Life Sciences, 1972). We estimate that these criteria are not fulfilled by EEG, owing to its ambiguity, especially due to the need to use high amplifications in a noisy environment, and its inability to rule out misleading conditions, as it can be isolelectric in patients in whom a clinical picture similar to that of BD is actually consecutive to sedative drugs. By contrast, these are perfectly fulfilled by EPs (Guérit, 1992) and transcranial Doppler (Ducrocq *et al.*, 1998). In particular, it has been demonstrated that brainstem EPs are preserved in the presence of sedative drug intoxications mimicking the BD pattern (de Tourtchaninoff *et al.*, 1999 ; Hantson *et al.*, 2000). Therefore, we

consider that, if handled by experts, these currently constitute the best BD bedside confirmatory tools. In particular, their widespread use could decrease the number of indications for arteriography and non-bedside radioisotopic studies, thereby limiting the risks associated with patient transportation.

New perspectives : continuous neuromonitoring

The necessity to shift from sporadic EEG and EP recordings to continuous neuromonitoring is becoming increasingly manifest in the OR and in the ICU and several applications of continuous neuromonitoring have already been published : analysis of EEG power variability for the early detection of vasospasm after subrachnoid hemorrhage (Vespa el al., 1997), automatic subclinical seizure detection in patients with metabolic disturbances (Jordan, 1995), continuous monitoring of the EEG spectra and BAEPs and SEPs in head trauma (Pfurtscheller), continuous EEG and EP monitoring for early detection of brain and spinal cord ischemia (Guérit, 1998).

Nevertheless, the development of continuous neuromonitoring raises several new specific problems. One of these problems is that the interpretation of neurophysiological data is usually outside the scope of the surgeons, intensivists or anesthesiologists, who precisely need this information for proper clinical management. Hence, the neurophysiologist cannot be present throughout the whole period of monitoring. Several difficulties are still to be overcome to free the neurophysiologist from most technical problems and to render the neurophysiological information readily accessible to non-neurophysiologists. This supposes a multidisciplinary approach, including engineers, neurophysiologists and intensivists, and a specific training of the nursing team.

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