Neuromonitoring and Emergency EEG

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48.1 Intraoperative EEG monitoring

IntraoOperative Neurophysiological Monitoring (IONM) is important for the evaluation of the CNS function during surgical procedures, where injury is possible. IONM can be helpful for the detection of an iatrogenic injury and for mapping of brain structures during the procedure, to avoid their damaging. EEG is one of the oldest and most commonly used modality for intraoperative neuromonitoring. Historically, it has been used at first for the identification of EEG patterns associated with anesthesia to determine the depth of anesthesia and adjusting the drug levels to achieve a predefined neural effect, such as burst suppression. Later, the evolution of intraoperative EEG use was also expanded to include monitoring for assessing cortical perfusion and oxygenation, as well as the identification of epileptiform activities, during a variety of vascular, cardiac and neurosurgical procedures [1, 2]. One of the most relevant application of intraoperative EEG monitoring is Carotid End Arterectomy (CEA). Neuromonitoring can also be useful during cardiovascular surgery and intracranial vessels embolization or during open-heart surgery and cardiopulmonary bypass, where there is risk for ischemic events that could produce both temporary and permanent EEG abnormalities. Among neurophysiologic techniques, EEG provides the fastest feedback with regard to fluctuations in the neurophysiologic dysfunction related to cerebral hypoperfusion. In physiological conditions, Cerebral Blood Flow (CBF) is about 70–50 mL/100 g/min. A reduction of flow to 35–25 mL/100 g/min is not generally able to provoke neuronal damage or to induce EEG modifications. However, a further CBF decrease (<25 mL/100 g/min) causes immediatly cerebral functional changes reflected by reversible EEG modifications and consisting firstly in the decrease in amplitude of the tracing, then in a progressive decrease of the rapid frequencies (alpha-beta) and, finally, in an overall slowdown of cerebral activity with appearance of delta waves. When CBF drops below 12 mL/100 g/min, EEG background activity progressively decreases until it is totally suppressed [3, 4].

Other than ischemic events, there is a wide spectrum of causes for EEG changes during surgery, such as hypotension, hypothermia, emodiluition, embolism and changes in anesthetic drug blood levels. With regard to hypothermia, this may be responsible of specific and evolutive EEG changes. If body temperature remains above 30 °C, hypothermia does not significantly impact electrophysiological findings. At temperatures below 29.6 °C, EEG tracings show a progressive diffuse slowings and periodic and rhythmic abnormalities and when temperature goes under 24.4 °C, a burst suppression or other malignant patterns (i.e., alpha/theta coma) appear. Finally, under 17.8 °C, a pattern of electrocerebral inactivity is reached [5]. During rewarming, the suppressed tracing returns to a burst suppression pattern and, progressively, to a continuous EEG.

48.1.1 EEG Monitoring During Carotid Endarterectomy

CEA is a surgical gold standard procedure for moderate-to-severe symptomatic and selected severe asymptomatic carotid stenosis patients. One of the most serious perioperative complications associated with CEA surgery is stroke, with a 30-day periprocedural stroke rate ranging between 2 and 6% [6]. Several factors contribute to this risk, but the most relevant remains the clamping of the carotid artery, which can induce cerebral ischemia, with consequent irreversible neurologic damage. A meta-analysis of existing literature demonstrated the diagnostic accuracy of EEG in predicting perioperative strokes. As a matter of fact, patients

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with strokes after carotid surgery have six times greater chance of experiencing an intraoperative change in EEG during CEA [6].

During CEA, the common carotid artery is temporarily clamped to allow the plaque removal from the internal carotid artery; in this phase, blood flow to the homolateral cerebral hemisphere is provided by collaterals, if the contralateral carotid is also not significantly stenotic and the circle of Willis communicating arteries are functional. If compensatory flow is not sufficient, the clamping performed without shunting (a temporary bypass connecting the sections below and above the blockage) may lead to cerebral ischemia. However, shunts are associated with iatrogenic problems and shunting itself may induce embolic events leading to perioperative stroke, so that they should only be used when necessary.

Based on this premise EEG monitoring is the most sensitive technique for giving indications to shunt positioning [7, 8]. The correlation between severity of EEG changes and degrees of reduction of the regional cerebral blood flow can help the surgeon decide whether there is absolute need for shunting, because mild EEG changes usually reverse with just an increase of systemic blood pressure, while more severe EEG changes require insertion of a temporary intraarterial shunt.

In order to properly evaluate EEG tracings during CEA, we must consider that general anesthesia provokes itself changes in the EEG activity. This depends mostly on the drugs used, doses and individual responses. Changes in EEG tracings can also happen due to a sudden hypotension. Severe electrocortical depression is observed when arterial blood pressure decreases of about 20–30 mmHg below the mean levels. Surgical stimulation of the vagus nerve or of the carotid glomus can also cause bradycardia or hypotension with consequent EEG modifications and then the cooperation between the surgical equipe, the anesthesiologists and the neurophysiologists is crucial for the proper management of patients undergoing CEA.

Before CEA, it is advisable that patients are submitted to standard EEG recording; this allows neurophysiologists to identify any pre-existing abnormalities, as the presence of both ipsilateral and/or contralateral focal EEG changes may increase the incidence of concomitant EEG abnormalities during clamping. Furthermore, predictors of EEG changes during CEA are represented by preoperative neurologic symptoms, ≥ 70% contralateral carotid and bilateral vertebral stenosis: these factors increase the possibility of clamping-induced ischemia, as detected by intraoperative EEG [8].

Chiappa et al. in 1979 classified the EEG changes within 20 sec of ICA clamping in five groups: (1) ipsilateral attenuation, (2) bilateral attenuation, (3) ipsilateral slowing without attenuation, (4) ipsilateral slowing with attenuation and (5) bilateral slowing and attenuation. These authors observed that the most common pattern (47%) was rapid unilateral or bilateral attenuation of background anesthetic-induced fast EEG activity [9].

The ACNS's intraoperative EEG guidelines (2000) defined three degrees of EEG changes caused by ischemia during clamping for CEA, in comparison with the preclamp post-induction baseline: (1) a decrease in background fast activity amplitude >50%, most apparent when using anesthetic agents that generate such fast activity, (2) an increase in delta-theta activity ≥50% (decrease in fast activity and increase in slow frequencies may be simultaneous) and (3) all raw EEG activity that progressively diminishes in amplitude and approaches electrocerebral inactivity [2, 10].

These various features of abnormalities, ipsilateral or bilateral, occur promptly after clamping (within 20–40 sec), while ischemic brain lesions need a few minutes to consolidate. Knowing these time intervals can really help surgeons regarding the decision to perform a shunt. For this reason, it may be useful to perform a clamping test for at least 1–2 min, to verify the times of the patient's tolerance after carotid clamping, evaluating the efficiency of collateral circulation.

It is also important to observe the reversal of EEG changes, either after inserting a temporary shunt and/or increasing the systemic blood pressure, because this resulted in a favorable neurologic outcome (Figs. 48.1 and 48.2).

Direct visual analysis of EEG monitoring during CEA offers an increased sensitivity and allows a prompt interaction between the neurophysiologist and the surgeon: the neurophysiologist suggests shunting, if any changes in EEG occurs. However, the methods of quantitative evaluation of EEG are also very useful, which can objectively assess the changes occurring during clamping, making the evaluation less operator-dependent. The most widely used quantitative EEG (qEEG) procedures employ Fast Fourier Transform (FFT) algorithms and the spectral array displays available are the Compressed Spectral Array (CSA), which displays EEG data as a pseudo-three-dimensional plot, and the Density Spectral Array (DSA) that depicts also the same values on a color-coded data. These trends, in addition to a visual inspection of the EEG, are particularly useful during monitoring, because they objectively highlight modifications due to frequency and power variations of EEG signals (Figs. 48.3 and 48.4).

When CBF decreases, alpha and beta relative power decrease, while delta and theta relative power tend to increase. Cursi et al. [11] have studied, for example, the usefulness of the desynchronization index (indicating the reduction of 8–15 Hz band power) for the detection of cerebral hypoperfusion during CEA. The values of desynchronization index exceeding 65% seem to correctly detect patients with cerebral hypoxic risk.

Another quantitative parameter, often used during EEG monitoring in CEA, is the Brain Symmetry Index (BSI). BSI quantifies the mean spectral asymmetry between the hemispheres, calculating the absolute value of the average

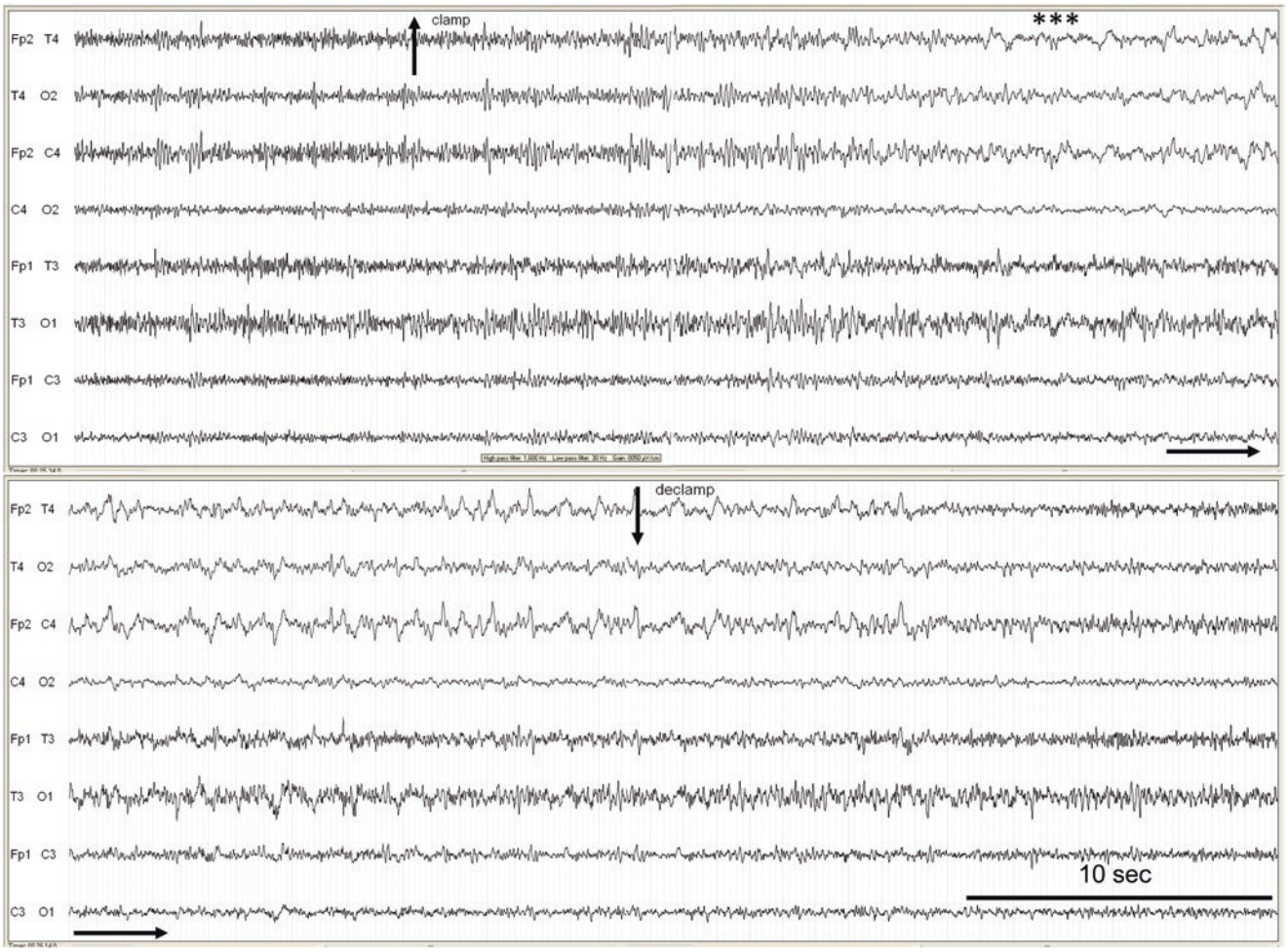

Fig. 48.1 EEG monitoring of 76-year-old patient during carotid endarterectomy. 20 s after clamping (***), EEG showed right delta slowings with tendency to diffusion which disappeared after declamping: this has represented an indication for shunt positioning

spectral density of the right and left hemisphere in the frequency range from 1 to 25 Hz. The BSI is normalized in the range between 0 (perfect symmetry for all channels) and 1 (complete asymmetry), with physiologic values of 0.05 to 0.08. The BSI may assist in the visual EEG analysis during CEA and provides a quantitative measure for EEG asymmetry due to cerebral hypoperfusion. Changes in the BSI of at least 0.06 during clamping test has an excellent correlation with visual EEG assessment and required shunting [3, 12].

Technical notes. In order to perform an intraoperative EEG, technicians need to be experienced and the full cooperation of staff is required. Neurophysiologists should be informed of all clinical and anesthesiological changes, to better interpret EEG patterns. Whereas this happens, bias can be overcome and EEG can be considered as one of the most efficient tools during this kind of surgery. EEG monitoring must be extended from the operative awake to the postoperative awake states. Cup or subdermal needle electrodes can be used: cup electrodes are fixed to the scalp by collodium or EC2, making the best contact and avoiding artifacts due to passive head movements during surgery; needle electrodes can be easily placed, but are also more unstable and have a higher impedance. A bipolar, anterior-posterior, 16 channel montage with placement of 21 electrodes is recommended. According to the intraoperative ACNS's guidelines [1, 10] a number of channels less than 8–12 is insufficient. The high-pass filter should be set at 0.53 Hz, the low-pass filter at 70 Hz, the sensitivity at 5 μV/mm. The aim of the montage is to identify general modifications of the pattern more than to analyze the wave shape of the single elements; for this reason, it is preferable to use a time sweep between 5 and 15 mm/s.

48.1.2 EEG Monitoring in Cardiothoracic Surgery

The indications for EEG monitoring during cardiothoracic surgery include the prompt identification of ischemic damages, both due to cardioembolic events and to hypoperfusion. Particularly important is the role of the EEG during some types of cardiothoracic surgery in which extracorporeal perfusion or hypothermic cardioplegia are used. Ascending

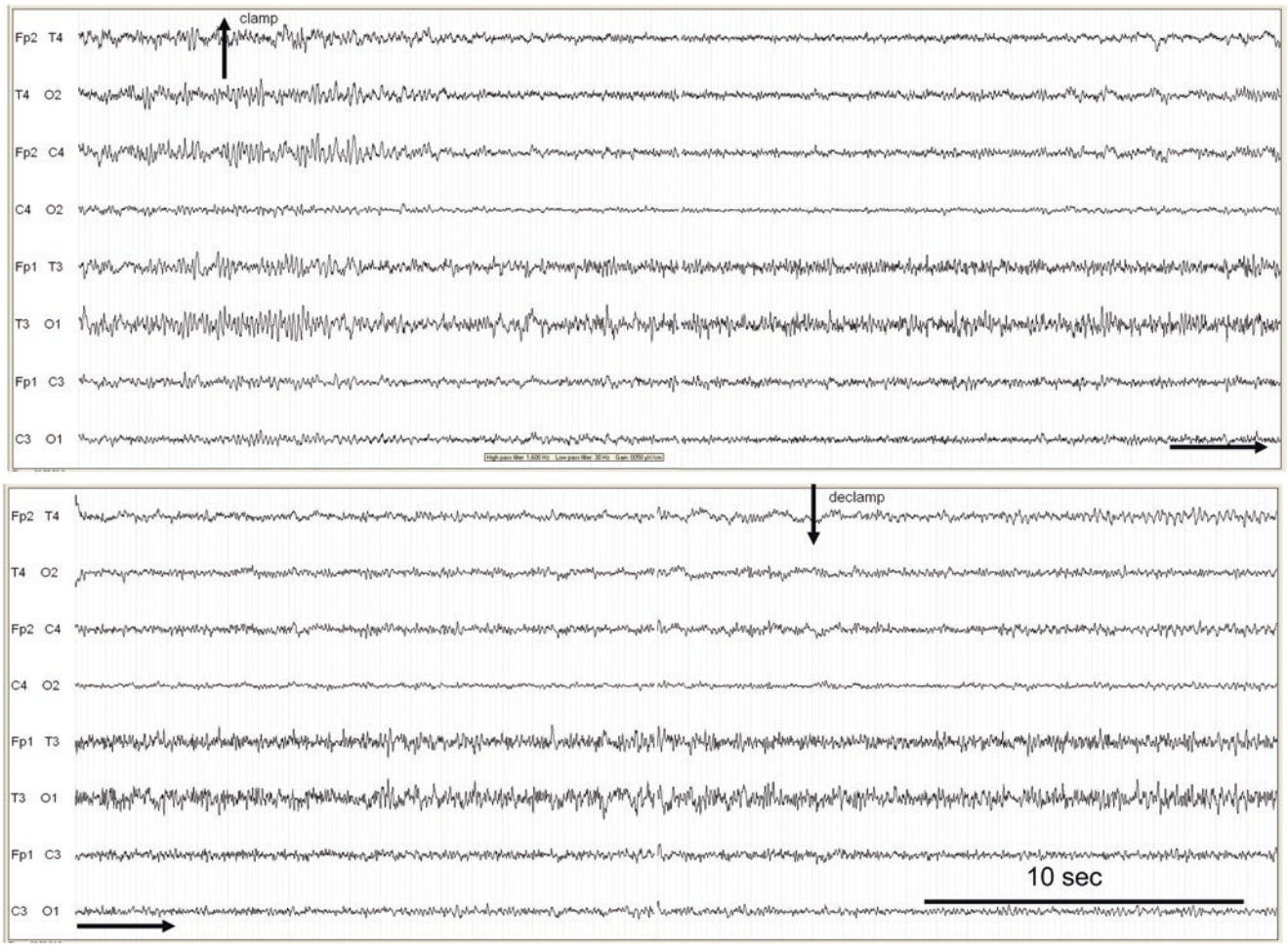

Fig. 48.2 Same patient of Fig. 48.1. After clamping, performed to remove the shunt, a right electrocortical suppression was observed: the suppression promptly disappeared after declamping

aortic and/or aortic arch surgery is a complex procedure that usually requires a Circulatory Arrest (CA) to achieve a surgical field that is free of cannulas and clamps. During this period, there is a high risk of ischemia of all organs, especially of the central nervous system. The safe duration of CA is controversial and will depend on the hypothermia achieved and the concomitant use of cerebral perfusion. The main pathophysiological aspects involved in cerebral ischemia-reperfusion are the consumption of Adenosine Tri Phosphate (ATP), the excitotoxicity of glutamate, the alterations of ionic homeostasis and the formation of oxygen free radicals. Measures that disrupt this cascade of events will theoretically have neuroprotective potential. However, a second pathway of neuronal death by apoptosis has been found, where mitochondria plays a major role and a third pathway due to necroptosis or programmed necrosis that does not depend on caspases. All of this would indicate that some measures that seem effective may have no long-term impact, because what they actually do is delay neuronal death [13]. Hypothermia acts by decreasing intracellular enzyme activity and Cerebral Metabolism Rate for O_2 ($CMRO_2$), thereby improving the balance between the supply and demand for oxygen. For each degree of temperature decrease, the $CMRO_2$ decreases an average of 6–7%, so at 25 °C the $CMRO_2$ decreases to 37% and at 15 °C to 15% of the basal. Cerebral blood flow is also reduced in a linear fashion as opposed to the decrease in the $CMRO_2$ that accelerates below 20 °C [14, 15].

The maximal suppression of $CMRO_2$ occurs at Electro Ccerebral Inactivity (ECI), and cooling to deep hypothermia (≤18 °C) has evolved as the preferred technique for cerebral and systemic organ protection during deep hypothermic CA. However, the cooling time and temperature required to achieve ECI are highly variable among patients [16]. Therefore, EEG monitoring can identify when sufficient hypothermia is reached and permits the verification that it is maintained [17].

48.2 EEG Monitoring in ICU

Multimodal monitoring in ICU using a variety of techniques including clinical and laboratory evaluation, bedside neurophysiological monitoring with continuous or noncontinuous techniques and imaging, is fundamental for patients who

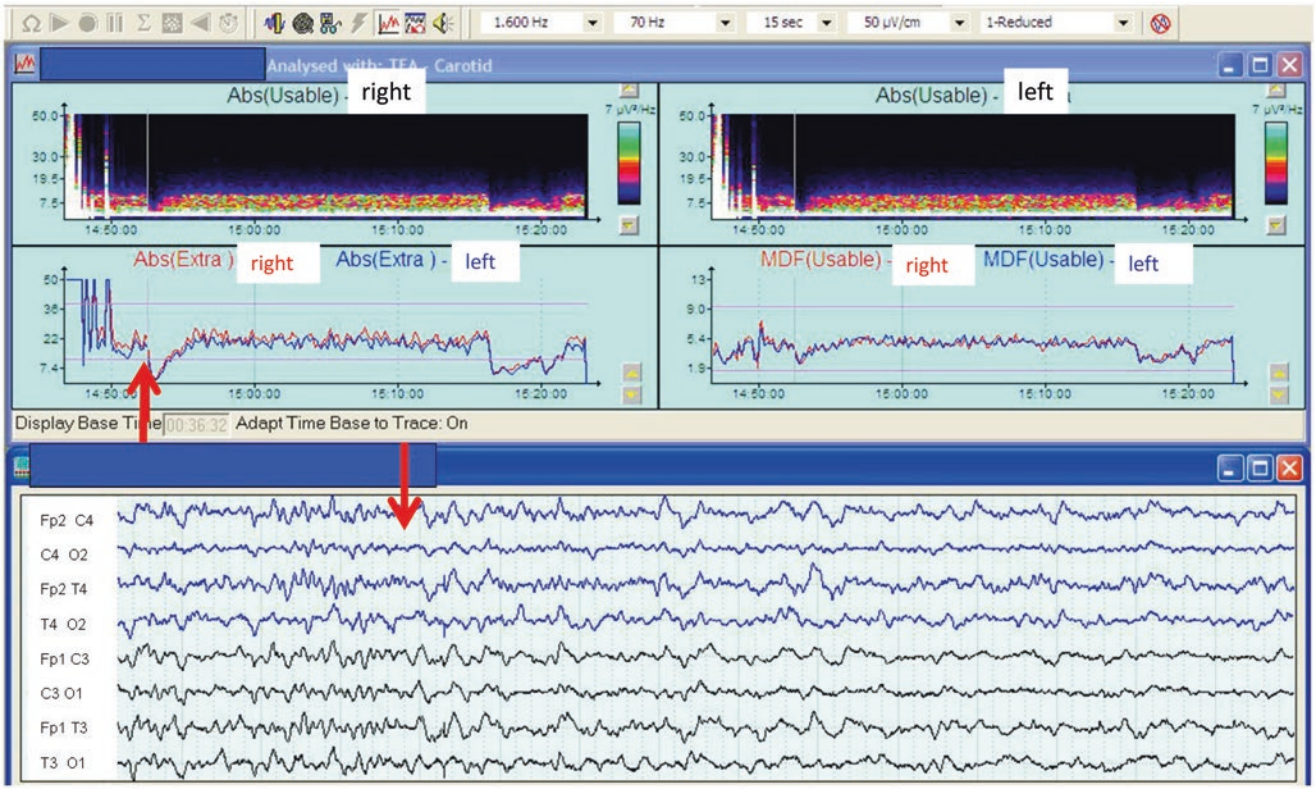

Fig. 48.3 EEG monitoring during left endarterectomy. Raw EEG after clamping (at arrow) showed a diffuse slowing. In the upper part of the figure, the density spectral array of absolute power and the Mean Dominant Frequency (MDF) is showed

Fig. 48.4 EEG monitoring during right endarterectomy. Clamping caused a marked, sudden suppression of the background activity. In the upper part of the figure, the trends of absolute power of the EEG frequencies and Mean Dominant Frequency (MDF) are shown. In the lower part, raw basal EEG is showed on the left and EEG during clamping on the right side

require neurocritical care. Clinical evaluation, essential precondition to any subsequent investigation, should include, in addition to the general neurological and clinical examination, the use of specific scales that make the contents intelligible through standardized scores: Glasgow Coma Scale (GCS), Full Outline of Un-Responsiveness (FOUR Score) and Glasgow Outcome Scale (GOS) for evaluation of coma state; Confusion Assessment Method for the ICU (CAM ICU) and Richmond Agitation Sedation Scale (RASS) for evaluation of delirium. Moreover, patient's clinical conditions should always be considered during the interpretation of all instrumental exams. Indication, timing and modalities of execution of all instrumental examinations should be decided by the neurophysiologist and the anesthesiologist together.

Among the neurophysiological tools, EEG is recommended in all patients with acute brain injury and unexplained and persistent consciousness disorders. It should be performed in emergency in patients with Convulsive Status Epilepticus (CSE) that do not return to functional baseline within 60 min after seizure medication and in patients with Refractory SE (RSE). EEG is required during therapeutic hypothermia and within 24 h of rewarming to exclude Non Convulsive Status Epilepticus (NCSE) in all comatose patients after cardiac arrest. EEG is suggested in comatose ICU patients, without an acute primary brain condition and with unexplained impairment of mental status or unexplained neurological deficits to exclude NCSE, particularly in those with severe sepsis or renal/hepatic failure. EEG can be useful to detect delayed cerebral ischemia in comatose patients affected by subarachnoid hemorrhage, in whom neurological examination is difficult or unreliable. Continuous EEG (cEEG) monitoring can also be an important diagnostic tool in comatose ICU patients without an acute primary brain condition and with unexplained impairment of mental status or unexplained neurological deficits, to exclude NCSE. On the other hand, EEG does not appear to be useful to detect cerebral ischemia and to target cerebral perfusion pressure in acute ischemic stroke patients [18]. The indications and utility of EEG monitoring in ICU are summarized in Table 48.1.

Standard EEG represents a fundamental instrument for the evaluation of cerebral function in ICU patients. Seriated EEGs provide extremely important data about neurological conditions, helping physicians to elaborate a correct diagnosis and also to decide which treatment is more suitable for every patient. It also provides a helpful tool for the prognosis. New digital equipments offer a noninvasive and easy to perform exams that do not interfere with patients' daily care.

Technicians should take note of any variation of awareness or consciousness of the patient during the whole duration of the recording, as well as of any factor that could influence tracing interpretation. If there is any obstacle to electrodes placement that can generate artifacts, it must be annotated so that the physician can take it into account when reporting the results. It is also very important to collect a detailed medical history and medical therapies. Previous exams, especially neuroradiological, may be useful to better individuate the main clinical aspects.

Table 48.1 Indications and utility of cEEG monitoring in Intensive Care Unit (ICU)

Detection of Non Convulsive Status Epilepticus (NCSE)
Diagnosis, follow-up, and therapy monitoring of seizures and status epilepticus (CSE, NCSE)
Characterization of paroxystic clinical manifestations – Repetitive motor manifestations. – Sudden spasms, nystagmus, ocular deviation, and chewings. – Autonomic paroxystic manifestations (brady-tachycardia, flushing, etc.).
Evaluation of consciousness during particular clinical conditions – Locked-in syndrome. – Serious neuromuscular diseases. – Frontal syndromes. – Akinetic mutism.
Monitoring of the effects of pharmacological therapies – Level of sedation. – Treatment of intracranial hypertension.
Prevention and monitoring of vasospasm – After subaracnoid hemorrhage. – During and after vascular neurosurgical procedures.
Evaluation of altered mental states
Prognosis of coma (>postanoxic and post-traumatic)
Diagnosis of brain death

The most useful information that can be obtained from ICU EEG monitoring are:

- Reactivity of the tracing to external stimuli.
- Identification of focal or diffuse epileptiform abnormalities and evaluation of AntiEpileptic Drugs (AEDs) efficacy.
- Detection of peculiar EEG patterns of coma useful for a prognostic evaluation.

Reactivity to external stimuli is fundamental in the evaluation of comatose patients.

EEG reactivity to external stimuli depends upon the intactness of anatomic pathways different from the lemniscus-thalamus-parietal axis known to carry somatosensory evoked responses [19] and consisting of interconnected stations. This complexand non completely clarified pathway is composed of projections from reticular nuclei of brainstem to diffuse thalamic projection system that relays to the cortex.

In Chap. 46, we have already explained various types of reactivity and testing modalities in comatose patients, through visual (eye opening under light), auditory (clapping, loud name calling) and nociceptive stimuli; while it is assumed that the first stimulus will be more informative, pain is generally viewed as the most effective. Furthermore, pain stimuli are routinely included in the physical examination of coma patients; even if they do not show a behavioral response to external stimuli, a cortical reaction to noxious

stimuli can be reflected by a change in EEG background activity [20]. In addition, tactile stimuli should be tested bilaterally on both extremities and the face, in order to avoid any area that may be affected by other kinds of lesions (e.g., spinal lesions).

Focal EEG abnormalities are often suggestive of specific pathologies; for example, Lateralized Periodic Discharges (LPDs) may be indicative of specific conditions such as herpetic encephalitis or intracerebral hemorrhage. Therefore, widespread slow activity is more frequently associated with bilateral, toxic, or dysmetabolic cortical damage, while bilateral and synchronous EEG patterns, often intermittent and monomorphic, are suggestive of deep subcortical lesions. Specific EEG patterns during coma are described in detail in Chap. 46.

The duration of EEG recordings depends on clinical request and it should be at least 30 min.

Other polygraphic data (EKG, EMG, oxygen saturation, PNG) are helpful to obtain more accurate interpretation of the EEG. When available, back-averaging analysis technique may be useful to correlate motor features to cortical epileptiform activity in comatose patients (subtle status epilepticus). This offline analysis method allows an association to be made between cortical epileptiform discharges and correspondent EMG patterns in patients with motor symptoms.

Video-EEG recording results are very useful, especially in patients with epileptic seizures (both convulsive or non-convulsive) or in patients with other motor symptoms, to avoid inadequate pharmacological therapies.

48.2.1 Continuous EEG (cEEG)

As ICU patients often present changes of their electroclinical condition (due to modifications of therapy, sleep-wake rhythm, seizures, etc.), seriated EEG recording results may be often inadequate.

Technological advances have improved the chances to easily collect, analyze and transmit the great deal of information deriving from a long-lasting recording.

Technical improvement converted cEEG from a research tool to a valid diagnostic instrument in the management of ICU patients, integrating the informations with other ICU monitoring systems (blood pressure, intracranial pressure, SaO_2, temperature) (Figs. 48.5 and 48.6).

Among all neuromonitoring tools, EEG has some advantages. It has a good spatial resolution (it can record at the same time the electrical activity of different cortical regions) and an excellent temporal resolution; it can be used to investigate both structural and functional disorders (seizures, post anoxic damages, toxic or dysmetabolic encephalopathies) (Table 48.2).

However, neuromonitoring lasting for days may have management problems. One of the most relevant con-

Fig. 48.5 Example of multimodal neuromonitoring in ICU. On the left: *HR* Heart Tate, *ABP* Arterial Blood Pressure, *ICP* Intra Cranial Pressure, *CPP* Cerebral Perfusion Pressure, *PbtO2* Pressure - brain tissue oxygen, *PRx* Pressure Reactivity index, *DSA* Density Spectral Array

Fig. 48.6 An example of control room in an Intensive Care Unit ICU

Table 48.2 Characteristics of various instrumental techniques for the evaluation of cerebral functions in comatose patients

	Practicality	Spatial resolution	Temporal resolution	Specificity	Interpretation
cEEG	++++	+++	++++	++++	+
Evoked potentials	+++	++	++	++	+
Transcranial Doppler	+++	++	++	++	+
Neuroimaging (CT-MR)	+	++++	+	+++	++
IBP monitoring	++++	+	+++	++	+++

cEEG continuous EEG, *CT* computed tomography, *MR* magnetic resonance, *IBP* intracranial blood pressure

sists in the need to maintaining correct electrodes placement and to assure a valid connection between the patient and the recording equipment. In ICUs, many factors can trigger artifacts: ventilators can cause either mechanical or electric artifacts; caring procedures can produce EEG patterns similar to physiological or pathological conditions. Even an electrode disconnection can produce signals not immediately recognizable as artifacts.

Hirsch et al. [21], in their sample of ICU patients undergoing cEEG monitoring, demonstrated a common periodic pattern as a reaction to various stimuli (Stimulus-Induced Rhythmic, Periodic, or Ictal Discharges, SIRPIDs). SIRPIDs are a relatively common phenomenon in the critically ill patients and their prevalence is reported in 10–34% of EEG neuromonitorings [22]. In order to differentiate SIRPIDs from critical spontaneous activity, a video-EEG recording can be helpful; technical personnel always need to register any stimulus that patients receive. Given the complexity of cEEG recording, a highly trained team is required; it should include neurologists, technicians, nurses and neuro-anesthesiologists.

As a matter of fact, if some patterns could be easily recognized by relatively "nonexpert" personnel, many others may only be detected by experienced neurophysiologists that can give the right interpretation of the trace and can distinguish and exclude eventual artifacts.

Literature shows how cEEG monitoring is extremely helpful in identifying subclinical epileptiform discharges (single nonconvulsive seizures or nonconvulsive status epilepticus) [21, 23]. cEEG monitoring is thus useful in the diagnosis of nonconvulsive epileptic seizures or purely electrical discharges and in the monitoring of status epilepticus when consciousness disorders persist after initial treatment [24]. Symptoms of nonconvulsive seizures can be quite misleading, often wrongly reported as postictal states, psy-

chiatric disorders, strokes, or metabolic encephalopathy. This might explain why nonconvulsive seizures are often underestimated (27–34% of cases) in the ICU [24–27].

A prospective study, conducted by Towne et al., showed that NCSE could be diagnosed via cEEG in 8% of unexplained comas in children or adult patients with no prior history of convulsive seizures [28].

cEEG has demonstrated to be very useful in early diagnosis of ischemic events in patients with any situation leading to a significant decrease in cerebral blood flow. In the case of subarachnoid hemorrhage complicated with vasospasm, ischemic complications are witnessed by a decrease in the alpha/delta ratio during cEEG. This enables an early diagnosis of ischemia at a stage where it is still reversible, helping physicians to quickly adapt therapeutic measures [29]. Other indications are monitoring of all cerebral hemorrhages, severe traumatic brain injuries and encephalitis; in all of these clinical indications, the European Recommendations recently published by the European Society for Intensive Care Medicine (ESICM) reported that continuous EEG was highly beneficial for patients [30].

The diagnosis of nonconvulsive seizures, which can evolve in NCSE, is essential in the ICU, especially in unexplained disorders of consciousness, since their presence has been correlated to a poorer prognosis and a delay in diagnosis appears to increase patient mortality [31]. The persistence of electrical SE has been associated with a higher mortality rate vs. generalized clinical SE, requiring an increase in AE treatment. The possible progression of clinical SE toward subtle SE also validates using EEG monitoring in the care management of generalized SE when disorders of consciousness persist after the initial treatment [27, 32].

Another relevant indication for cEEG is the differential diagnosis between postanoxic encephalopathy and SE, after cardiac arrest. As a matter of fact, after such event, EEG can reveal different types of abnormalities (burst suppression pattern, spike, spike and wave, polyspikes and wave, diffuse polyspikes or triphasic wave activities, often periodic or quasiperiodic). The simultaneous presence of myoclonus and the epileptiform EEG abnormalities should not always be considered, nor treated, as SE, because of the probable subcortical origin of the myoclonus (Fig. 48.7).

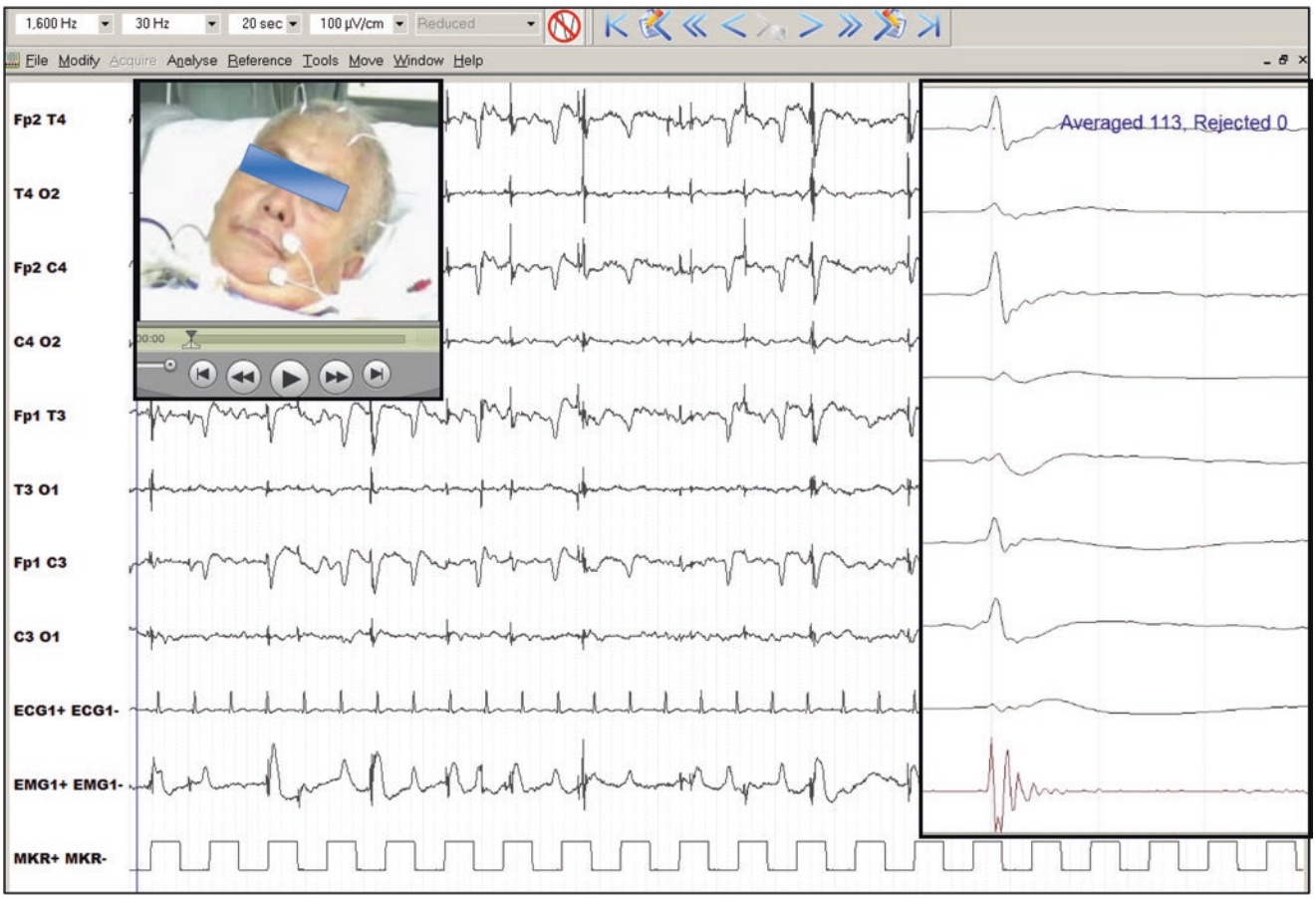

Fig. 48.7 A 71-year-old patient affected by postanoxic encephalopathy due to cardiac arrest. After 15 days diffuse facial myoclonic movements appeared, refractory to antiepileptic treatments. Simultaneous EEG and EMG monitoring didn't show any correlation between cortical and muscular activity (recorded by orbicularis oris muscle), as confirmed by back-averaging analysis

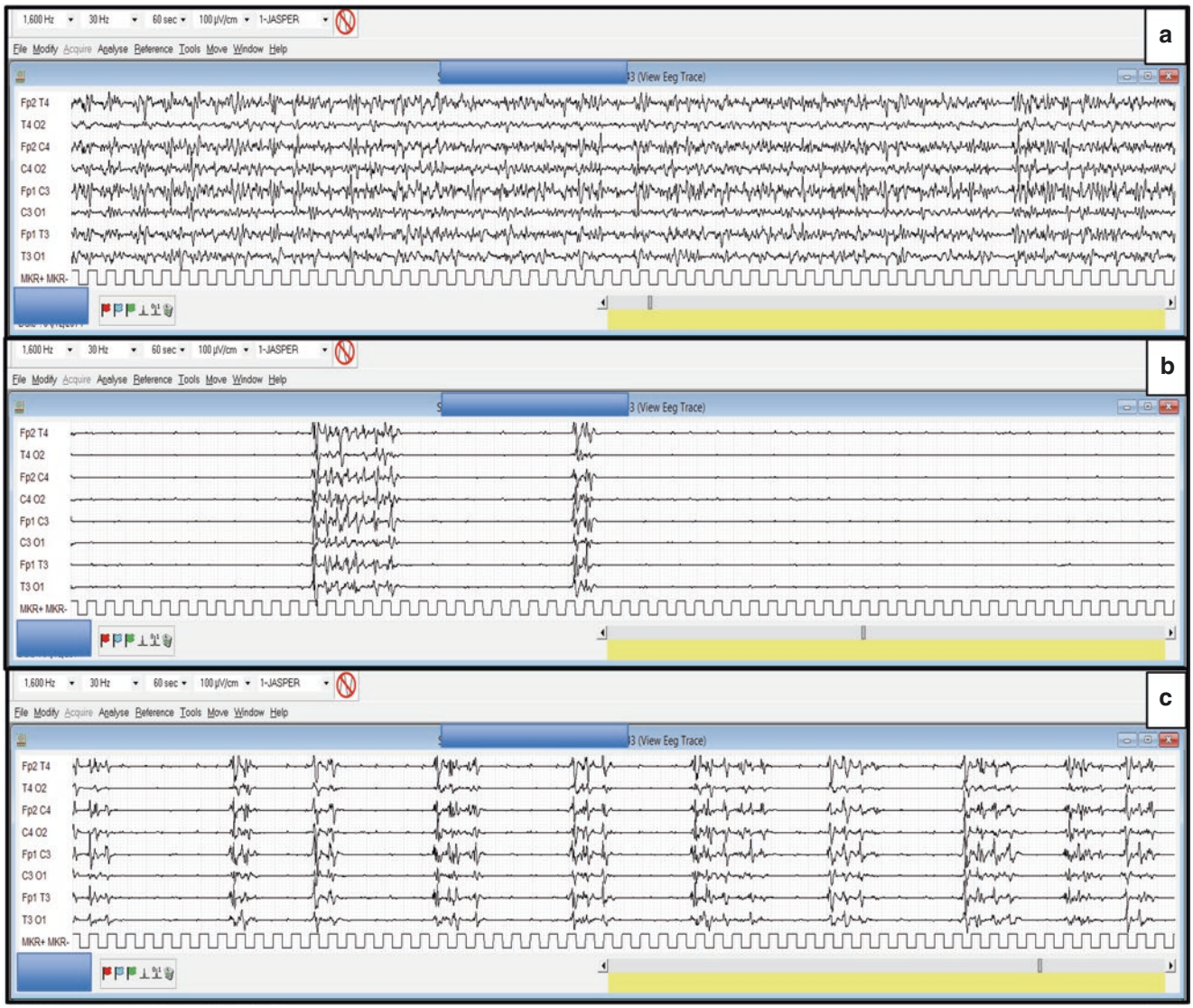

Fig. 48.8 Continuous EEG (cEEG) monitoring in a 76-year-old patient with traumatic brain injury. (**a**) Continuous diffuse epileptiform activity. (**b**) After a bolus of propofol (1 mg/kg) followed by an infusion of 20 mg/kg/h for 35 min, the cEEG showed a pattern of burst suppression with suppression periods >70% of the trace. (**c**) Few minutes after the interruption of propofol infusion, the periods of suppression became progressively shorter

EEG interpretation is always difficult in this context and long-term recording could help prompt detection of these abnormalities and redefine their significance and prognostic value [27, 33, 34].

Finally, cEEG can be used to determine the effects of different kinds of sedation and their optimal level to protect the brain, after severe traumatic brain injury (Fig. 48.8). However, the relevance of cEEG is under discussion in this situation, as most ICUs have the possibility of monitoring the depth of sedation (limiting the oversedation) by the Bi Spectral Index (BIS) [35]. It is based on an algorithm producing a dimensional scale going from 0 (suppressed EEG) to 100 (wake). This scale derives from a combination between direct signal recording from two frontal electrodes (Fp1 and Fp2 electrodes) and data representative of each level of sedation (from a database of EEG recordings). BIS cannot replace cEEG, but it can give an immediate quantitative result restrictively on the depth of anesthesia. For a complete description about the effects of anesthetic drugs on CNS and BIS, consult Chap. 45.

However, inspective analysis of non-processed tracings still represents the main method for revision and evaluation of EEG. Nevertheless, traditional inspective analysis requires a consistent amount of time and it is not very sensitive toward slow-onset abnormalities, such as the progressive decreasing of high waves or relative changes in a specific frequency band. In addition, as previously stated, this method requires a team of experts to be always available. For these reasons,

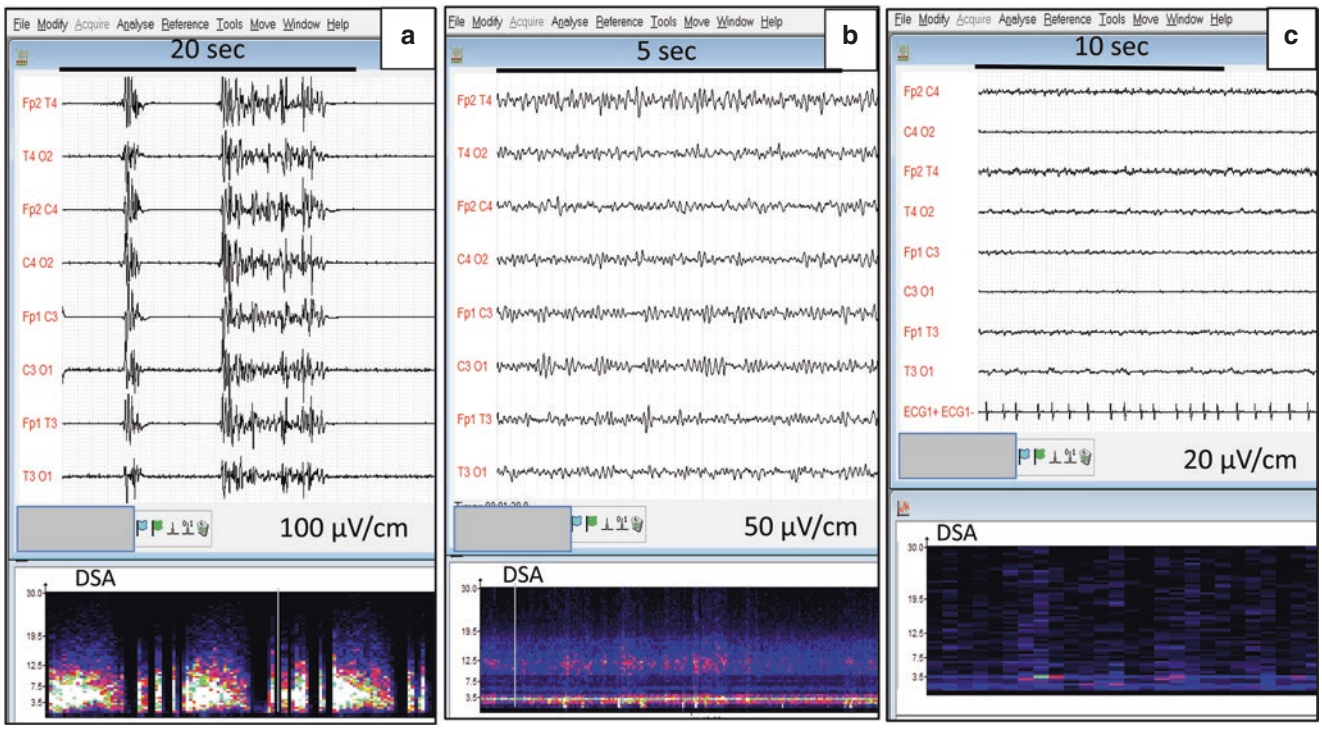

Fig. 48.9 Continuous EEG monitoring in a 56-year-old patient after cardiac arrest: raw EEG and Density Spectral Array (DSA). (**a**) day 1: burst-suppression pattern. (**b**) day 2: areactive alpha-coma of low amplitude. (**c**) day 3: attenuation/suppression of electrocerebral activity. LFF = 1 Hz, HFF = 30 Hz

alternative visualization methods were developed, allowing a simpler display through a quantification of EEG signals. They can be analyzed as number or visualized as graphics. Spectral analysis of cEEG can also be represented in the form of Compressed Spectral Array (CSA), Density Spectral Array (DSA), and amplitude-integrated EEG (aEEG) (Fig. 48.9).

Anyway original, raw tracings must always be available to verify the meaning of some abnormalities or trends. This is because some apparently significant patterns might be artifactual, especially in complex environments such as ICUs. Several programs were developed to automatically identify critical patterns but, up to now, they trigger many false positives.

48.3 Emergency EEG (eEEG)

EEG is a reliable and useful diagnostic tool in a number of clearly defined emergency situations. However, emergency EEG (eEEG) ordering policies at one hospital may not be applicable to others because of differences in staffing, payment difficulties, or local issues. These factors usually limit performing an EEG in emergency departments (EDs) [36].

The term "emergency" for a test means that the results will affect the patient management and outcome; consequently, an emergency test should be performed regardless the time and day. In an ideal situation, the EEG should be available anytime of the day 24/7, and it should only be requested by neurologists. However, the definition of eEEG has not yet been standardized. Varelas et al. defined eEEG an EEG requested from the attending physician—not necessarily a neurologist—for emergency indications on non-elective bases, performed within 1 h from the request [37]. Benbadis in 2008 named "stat" EEG (from the Latin word "statim," meaning immediately) an examination that should be available anytime any day (24 h/7 days), ordered only by neurologists or neurosurgeons, performed stat and interpreted stat (2–4 h from the request) [38]. The risk of this scenario is the abuse of the technique, and it could be ordered inappropriately [36]; an average response time of 3 h from request to initial reading is acceptable for eEEG [38, 39].

In a clinical policy statement, the American College of Emergency Physicians (ACEP) recommended that EEG should be considered in patients suspected of being in a non-convulsive status epilepticus or in subtle convulsive status epilepticus and concluded that no clear recommendation for ordering eEEG in other clinical situations may be made on the basis of available data [40].

However, based on the clinical experience of many emergency department in the world, the main situations in which eEEG is required are:

- Suspected SENC.
- Diagnosis of brain death.
- Altered mental states.
- Focal neurological deficits especially if there is discordance between clinical and neuroradiological data.
- Coma of uncertain origin.
- Suspected encephalitis, especially of infectious nature.
- Differential diagnosis with Psychogenic Non-Epileptic Seizures (PNES).

Apart from the other indications that have been abundantly discussed above and in the specific chapters, altered mental states deserve a particular mention.

Up to 10% of patients in the emergency department present with seizures or altered mental status (AMS) [40, 41]. The most common causes of AMS in the emergency departments are neurologic, accounting for 28% of discharge diagnoses in one study of 317 patients presenting with AMS [41]. AMS is a significant challenge to the emergency physician, because it is often the clinical feature of a wide spectra of pathologies, and its etiology may be difficult to individuate [42]. Moreover, clinical evaluation can be difficult, especially if a detailed history cannot be obtained and the collaboration of the patient is not complete. Not surprisingly, rates of emergency departments resources use, hospital admission and death for this diagnosis are disproportionately high relative to the percentage of thepopulation this group represents [43]. In these cases, EEG is a very useful diagnostic tool, because it is the only method that could provide an objective picture of the clinical situation. Nevertheless, it is still underused or often even not available in emergency [44]. Proposed reasons for this underuse include lack of availability of a technologist, difficulty and time required for signal acquisition [45], lack of timely expert signal interpretation and lack of awareness of utility of EEG in this setting [39, 41]. Two different examples of the utility of eEEG are showed in Figs. 48.10 and 48.11.

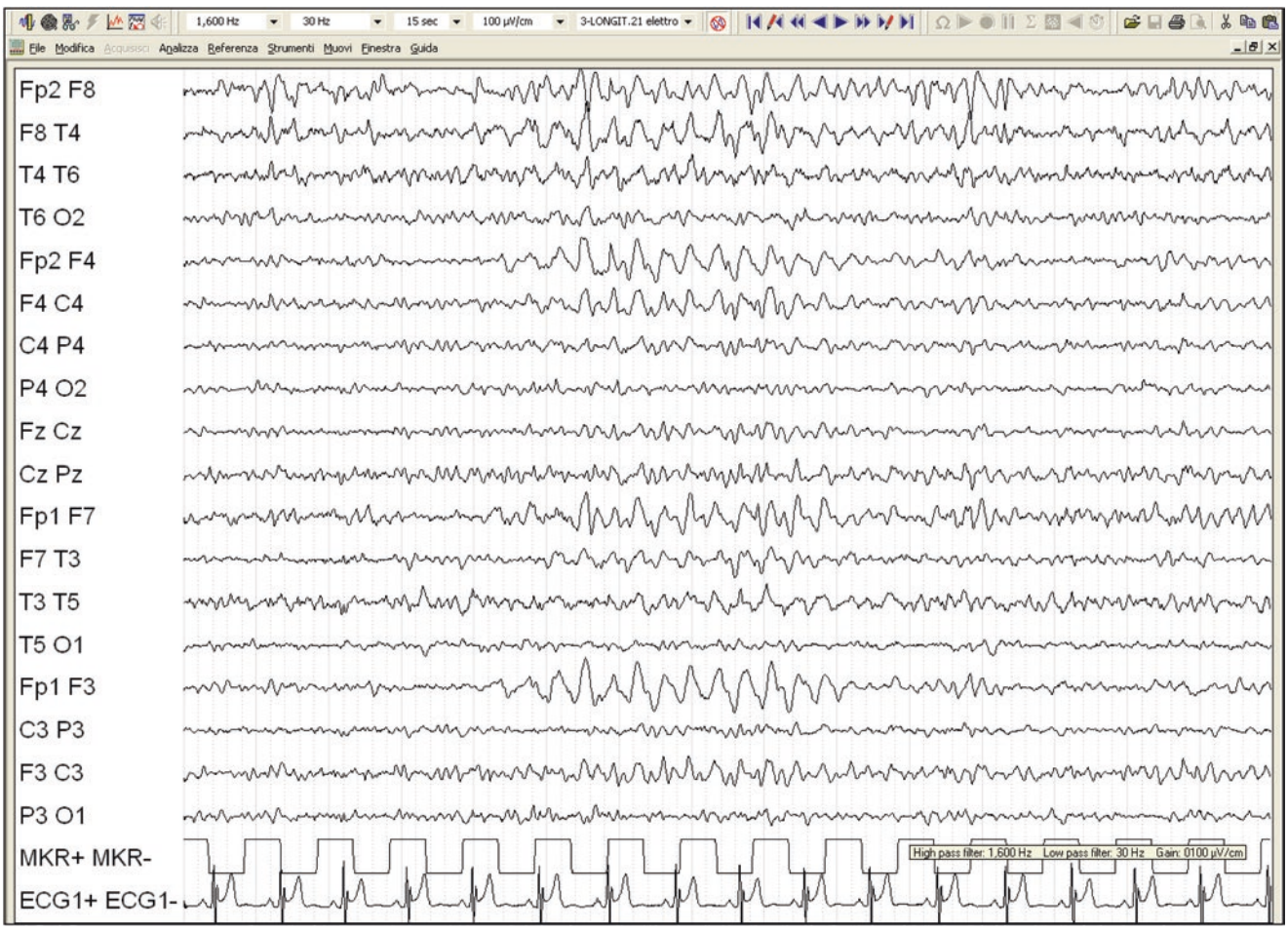

Fig. 48.10 A 27-year-old male patient, admitted to the emergency department for sudden onset of headache, dizziness, and confusion. EEG showed sequences of monomorphic slow waves (3 Hz) in right frontotemporal regions with a tendency to the contralateral transmission and associated with some spikes in phase opposition on F8 electrode. Magnetic resonance angiography showed a cavernous angioma in the same site

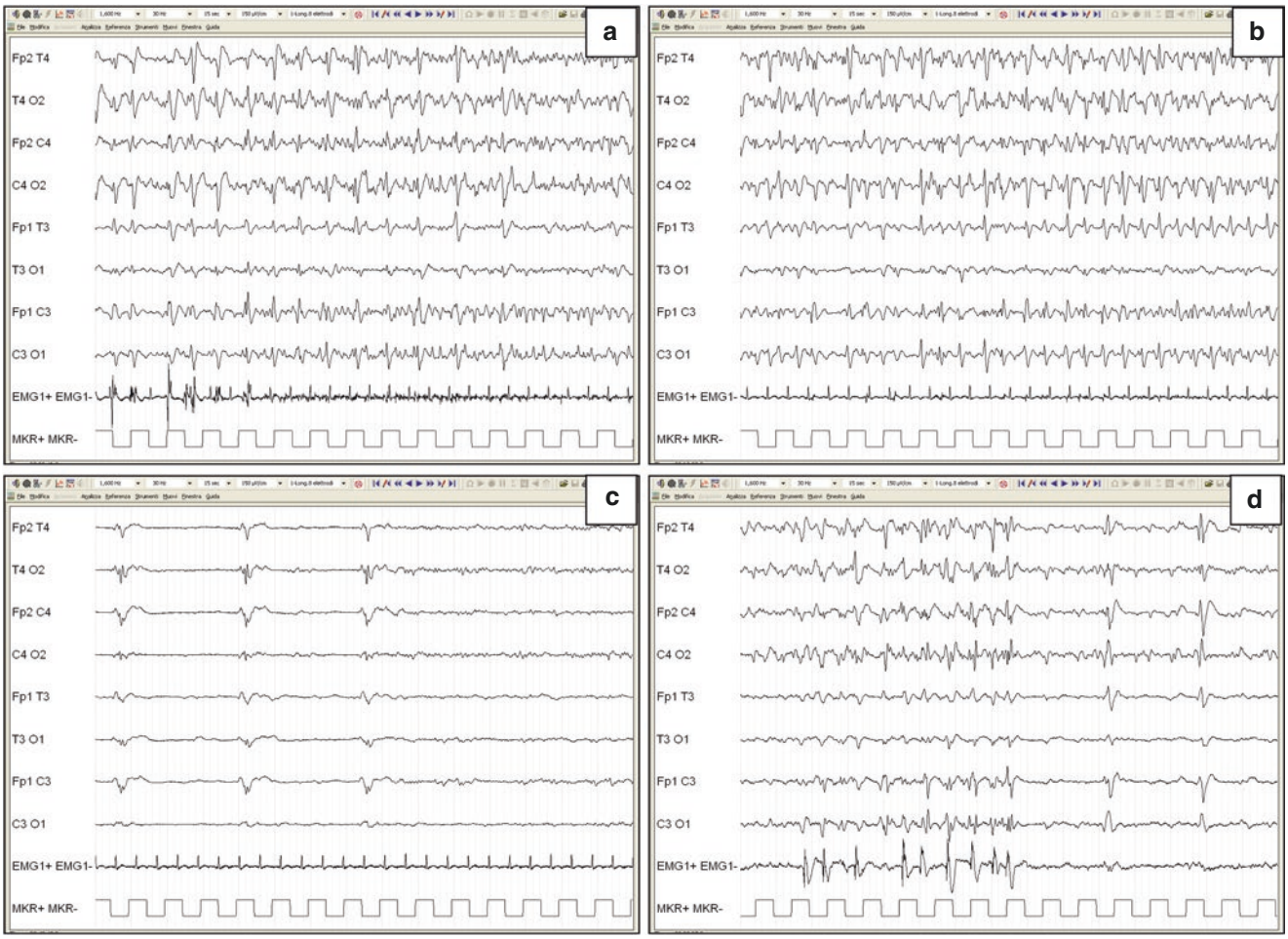

Fig. 48.11 A 55-year-old male patient after liver transplantation. Two days after surgical procedure, sudden onset of altered mental status worsening until coma and left hemisoma clonic movements. MR showed intracerebral right temporal hemorrhage. (**a**) EEG evidenced subcontinuous sequences of epileptiform graphoelements, more evident in the right hemisphere, sometimes related to muscular activity recorded by simultaneous EMG of left deltoid. (**b**) EEG monitoring, during and after administration of levetiracetam (1500 mg, i.v. bolus) showed, despite the disappearance of myoclonic jerks, the persistence of the epileptiform activity. (**c**) EEG abnormalities improved after the administration of Lorazepam (4 mg, i.v. bolus) and they were replaced by sporadic epileptiform abnormalities alternated with periods of electrocortical attenuation. (**d**) the day after, EEG showed more active right epileptiform activity, related to contralateral myoclonic jerks

eEEG is not indicated after a tonic-clonic seizure as it could evidence only minimal, specific, postictal slowings of background activity. However, sometimes, after a tonic-clonic seizure, symptoms as prolonged confusion, behavior disorders, and amnesia can be accompanied by interictal and/or ictal epileptiform abnormalities [46] (Fig. 48.12).

In their prospective study, Praline and colleagues confirm the value of an eEEG in the management of CSE and in the diagnosis of NCSE [47]. Even if a recording of at least 20 min is preferred, also a briefer (5 min) but promptly performed recording could have an adequate reliability [41].

Ziai et al. tested the availability and utility of EEG performed within 30 min in emergency departments in patients with AMS. For this purpose, a standard full-montage EEG lasting 20 min was performed within 30 min of referral by an emergency physician. The entire recording was also compared with abbreviated 5 min EEG. The study demonstrated that the rapid availability of standard full-montage EEG in emergency is feasible and useful to establish a diagnosis in about half of AMS patients; moreover, abbreviated 5-min EEG showed an adequate reliability [41].

Despite the undeniable clinical indications and sensitivity of the eEEG, this is yet poorly utilized in emergency departments. An Italian nationwide epidemiological survey showed that 24 h/ 7 d EEG was available only in 27% of neurology units in the emergency setting [48]. The scenario is the same now as it was 10 years ago.

A more recent survey conducted in the United Kingdom on CSE management demonstrated that only 33% of ICUs have an access to early EEG monitoring, which is stated as essential by NICE guidelines in the management protocol of CSE [49].

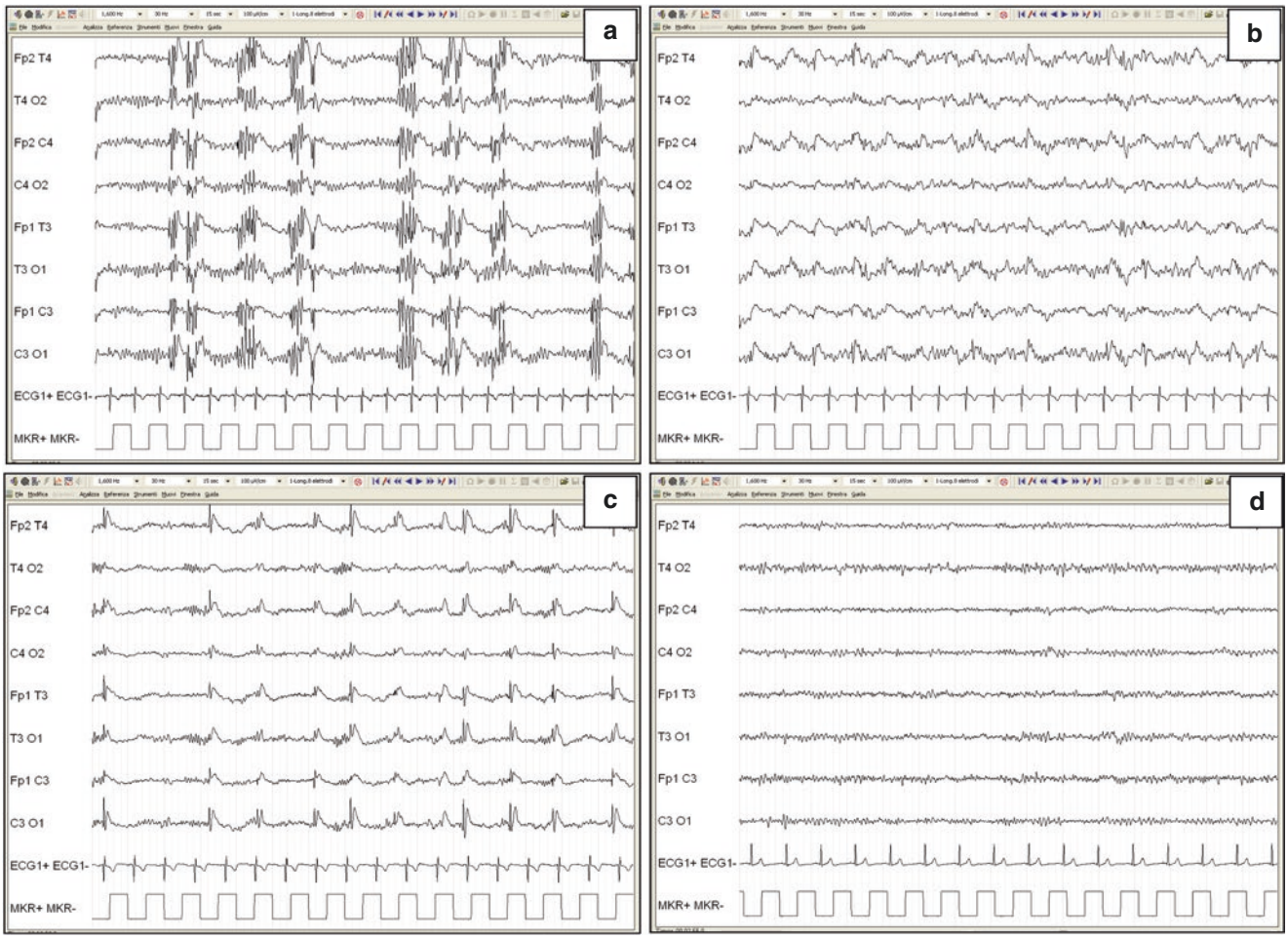

Fig. 48.12 A 66-year-old patient with generalized epilepsy syndrome, admitted to the emergency department because of confusion after two generalized tonic-clonic seizures. eEEG showed a critical pattern of nonconvulsive status epilepticus with diffuse, brief bursts of spikes at 10 Hz followed by a slow wave and alternated with alpha rhythm (**a**). 3 min after the administration of diazepam (10 mg i.v. bolus), EEG showed a diffuse delta activity with interposed spikes and spike-wave complexes (**b**). 20 min later, the alpha rhythm returned to be visible on the posterior regions, while periodic spike-wave complexes are detectable anteriorly (**c**). After 30 min, a second bolus of diazepam was administrated; the epileptiform abnormalities disappeared for the next 24 h (**d**)

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