EEG Normale in VEGLIA

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Dichiarazione sul Conflitto di Interessi

Dichiaro che negli ultimi due anni ho avuto i seguenti rapporti anche di finanziamento (compensi per relazioni/moderazioni a Congressi, FAD, Expert Meeting, etc) con soggetti portatori di interessi commerciali in campo sanitario:

- Arvelle/Angelini
- BIAL
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O. Mecarelli (22.11.2021)

S.I. 10-20, Jasper 1958

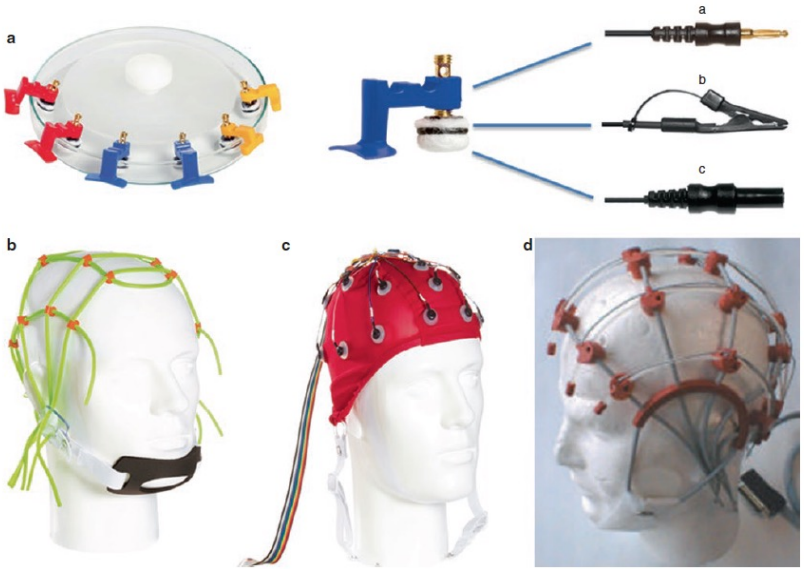

Fig. 3.1 (a) Bridge electrodes and their connectors: (*a*) spring plug; (*b*) alligator clip; (*c*) touch proof. (**b**) Cap with rubber bands for positioning bridge electrodes. (**c**) Prewired cap for standard EEG recording. (**d**) Elastic cap with electrodes pre-inserted in junctures

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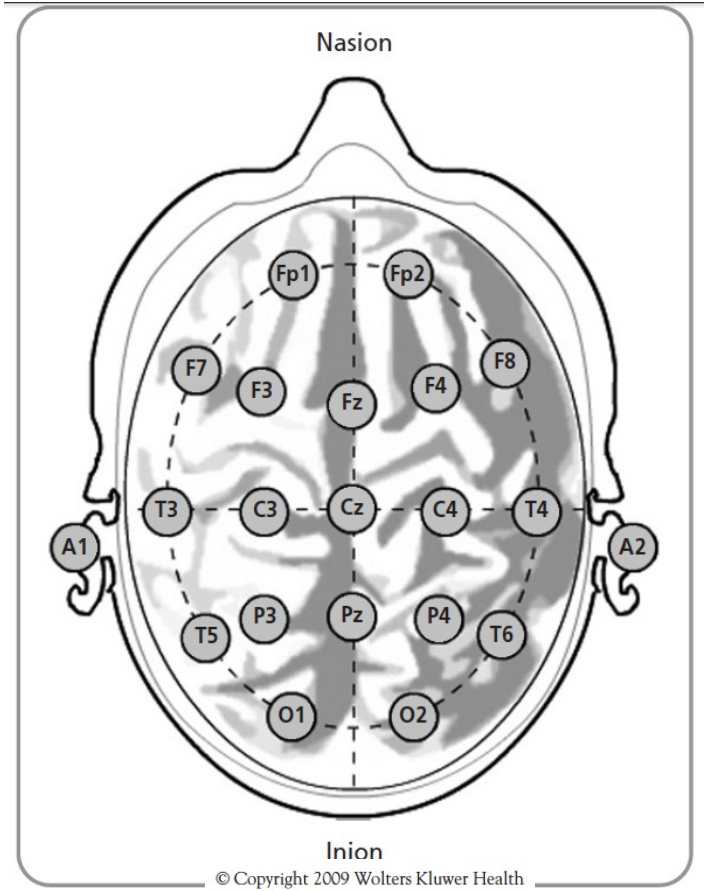

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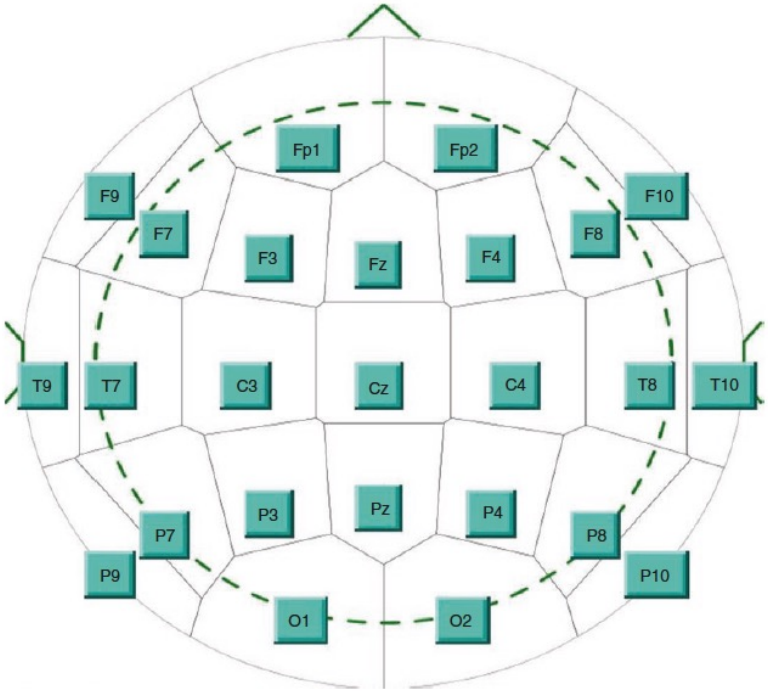
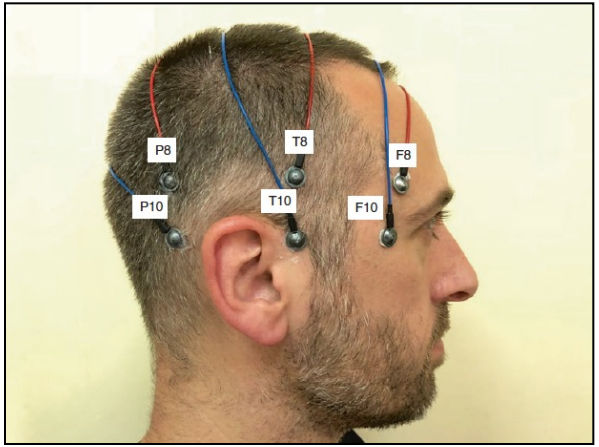
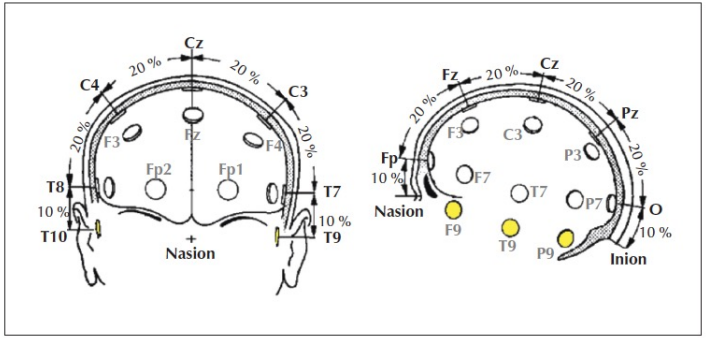
Fig. 4.3 New standard montage with additional coverage of the inferior and anterior brain regions, according to the recent recommendations of International Federation of Clinical Neurophysiology (from ref. [7], with permission)



Seeck M, Koessler L, Bast T, et al. The standardized EEG electrode array of the IFCN. Clin Neurophysiol. 2017;128:2070–7.





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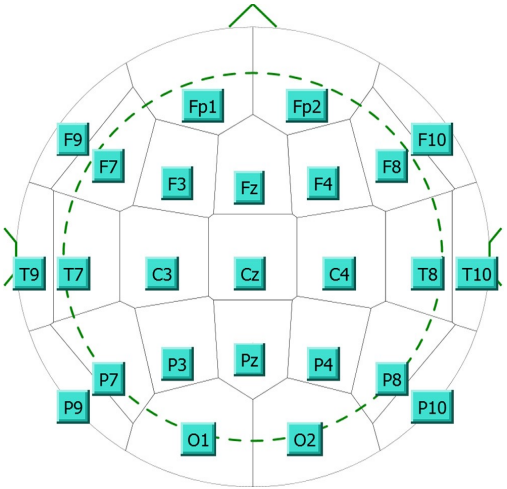
Clinical Neurophysiology 128 (2017) 2070–2077

Guidelines

The standardized EEG electrode array of the IFCN

Margitta Seeck [a,*], Laurent Koessler [b], Thomas Bast [c], Frans Leijten [d], Christoph Michel [e], Christoph Baumgartner [g], Bin He [f], Sándor Beniczky [h]



Recommended standard set-up

The standard 10–20 system did not include electrodes in the inferior chain (at the level of the preauricular point). Thus the inferior-basal and anterior part of the temporal lobe, which preferentially picks up activity originating or propagating from the mesial temporal structures, was not sampled (Rosenzweig et al., 2014; Koessler et al., 2015). Given that several diseases (e.g. temporal lobe epilepsy due to hippocampal sclerosis, autoimmune epilepsy, Alzheimer's disease) are characterized mainly by mesial temporal pathology, this region needs to be targeted through additional scalp electrodes in standard recordings. Thus, derived from the 10–10-system, we propose to add T9/T10 (10% inferior to T7/T8), F9/F10 (20% anterior to T9/10, or 10% inferior to F7/F8) and P9/P10 (10% inferior to P7/P8 or 20% posterior to T9/T10). The new basic array for clinical practice includes these six electrodes of the inferior temporal chain, which results in a total of 25 positions (Fig. 3). *For the reasons outlined above, we strongly recommend to use these 25 electrodes as a minimum for all standard recordings.*

Clinical Neurophysiology 129 (2018) 291–295

Added clinical value of the inferior temporal EEG electrode chain

Anders Bach Justesen [a], Ann Berit Eskelund Johansen [a], Noomi Ida Martinussen [a], Danielle Wasserman [a], Daniella Terney [a], Pirgit Meritam [a], Elena Gardella [a,b], Sándor Beniczky [a,c,*]

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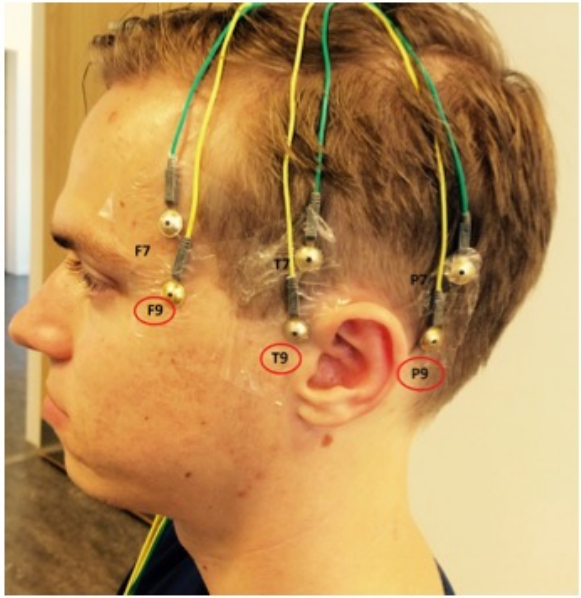

Fig. 1. Placement of the electrodes in the left inferior temporal chain (F9, T9, P9) compared to the neighboring electrodes of the 10–20 array (F7, T7 = T3; P7 = T5).

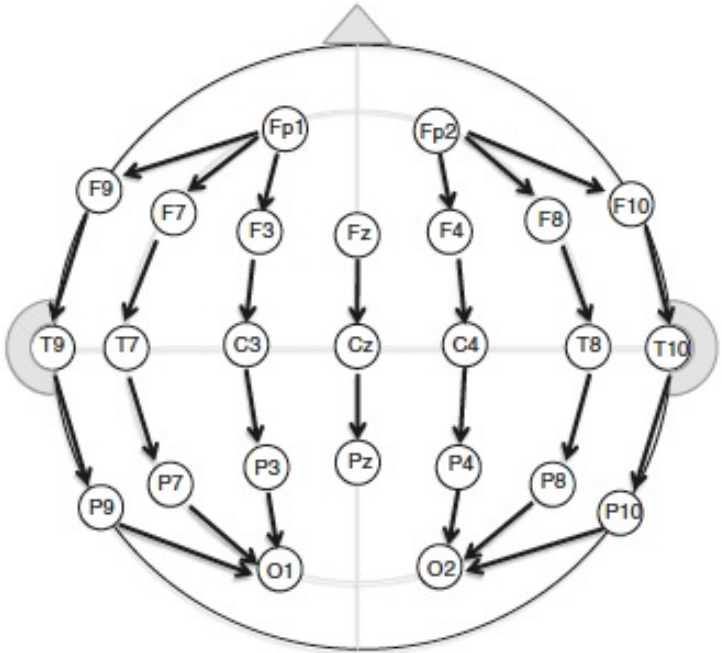

Fig. 4.19 New longitudinal bipolar montage proposed by International Federation of Clinical Neurophysiology (IFCN) [7]

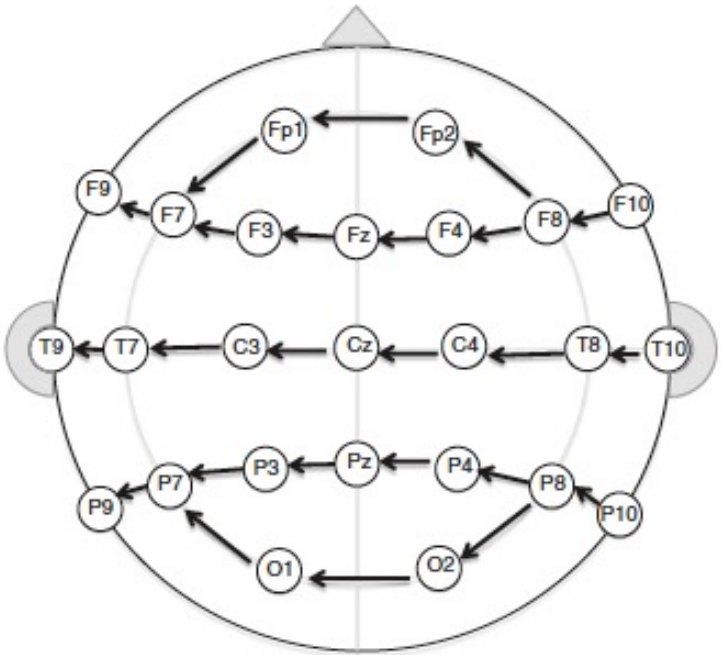

Fig. 4.20 New transverse bipolar montage proposed by International Federation of Clinical Neurophysiology (IFCN) [7]. Note that the interelectrode distance between the inferior and superior temporal electrodes is shorter (10%) compared to the other interelectrode distances

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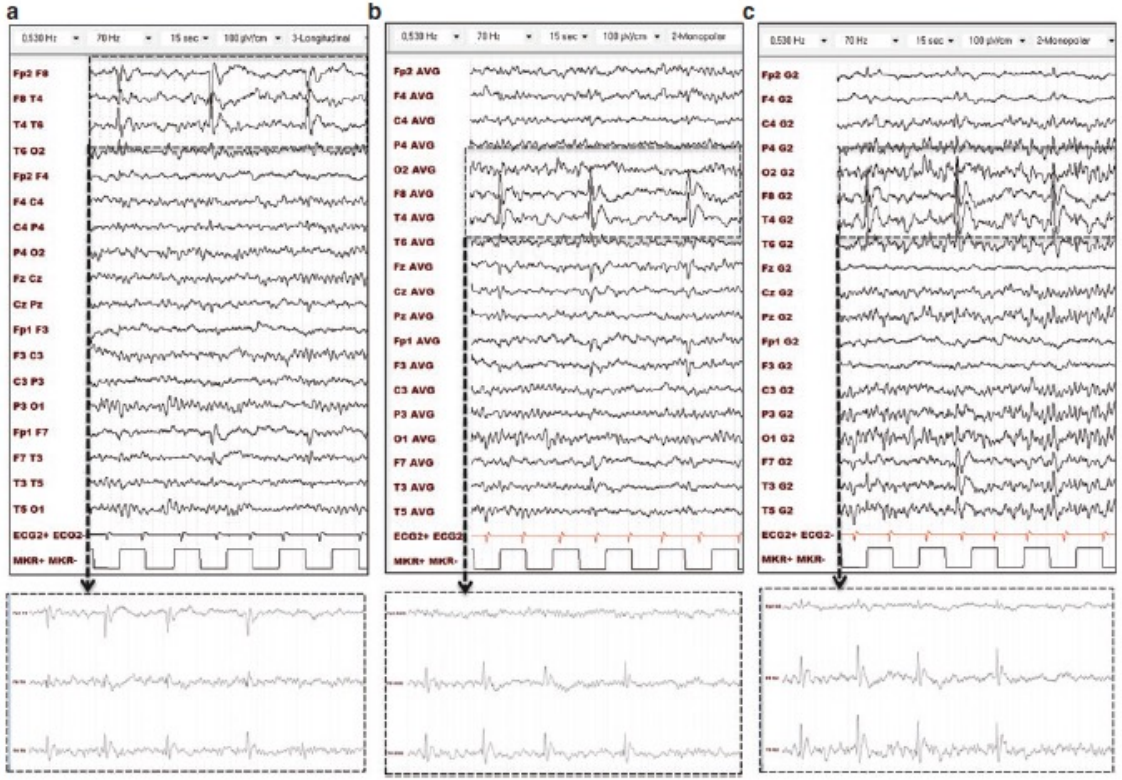
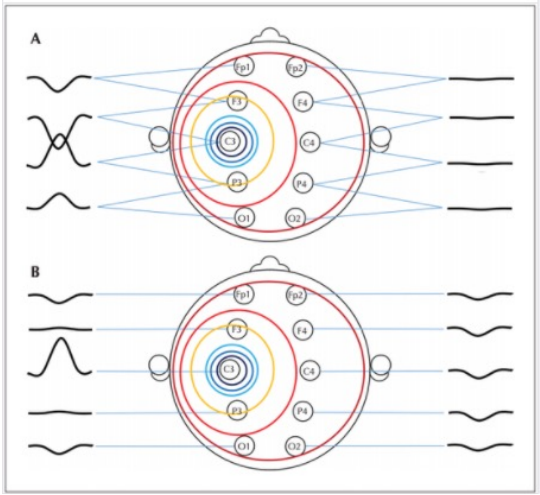


Fig. 4.17 The same EEG epoch of 5 s shows a right temporal epileptic focus in bipolar, AVG reference and common active reference derivations. In (**a**) (bipolar derivation) the phase reversal phenomenon in the first and third channel is evident, with almost total cancellation of the spikes in the intervening channel F8–T4 (these electrodes are placed over the focus and their potentials presumably have the same polarity and voltage as input to the differential amplifiers); note the poor spread of spikes to the homologous contralateral areas. The AVG reference derivation (**b**) confirms the higher negative signals at F8 and T4 electrodes; note, however, that positive signals are present also in Fz, Cz, Fp1 and F3, and negative in F7 and T3. When a common active electrode of reference is used (**c**) (G2, placed on midline in Fpz), the signal shows the same negative higher voltage in F8 and T4, with the evidence of synchronous lower negative signals contralaterally in F7, T3 and T5. However, in this practical example, all three derivations allow to localize the epileptogenic focus with good reliability (T4, T6, T3, T5 = T8, P8, T7, E7 according to the new nomenclature)



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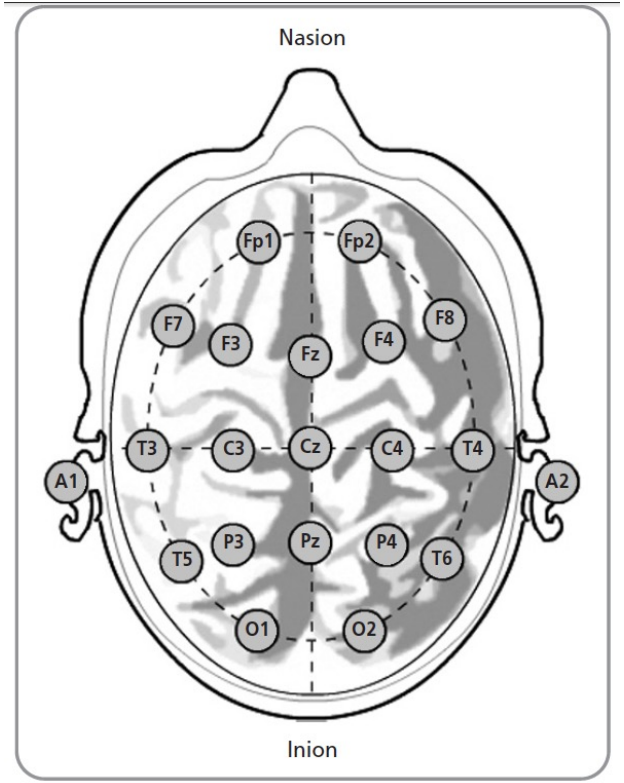
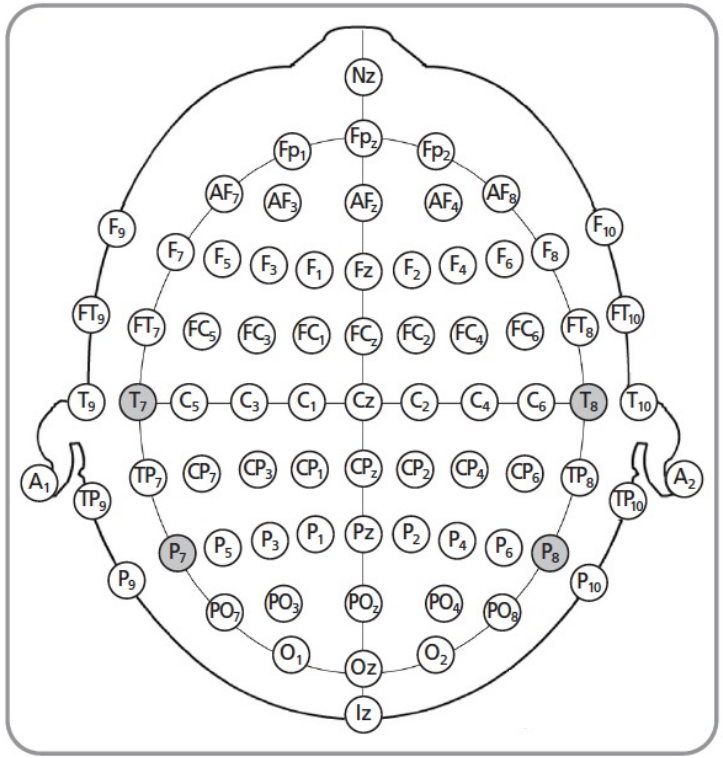
S.I. 10-20



S.I. 10-10



Oriano Mecarelli

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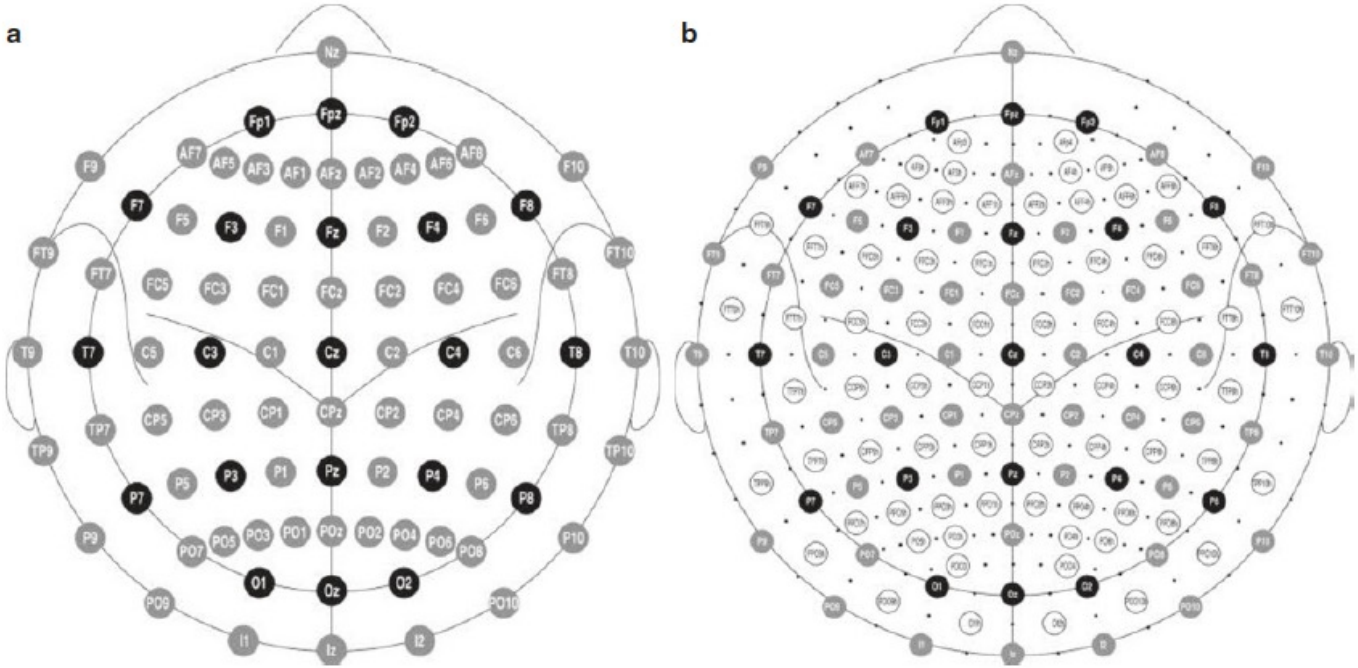

Fig. 4.5 The 10-10 and 10-5 extension of traditional 10-20 system. In (**a**) black circles indicate positions of the original 10-20 system and grey circles indicate additional positions introduced in the 10-10 extension. In (**b**) electrode positions in the proposed 10-5 system: additional positions to the 10-10 system are indicated with dots; a selection of additional positions useful for a 128 channel EEG system is indicated with open circles (from ref. [9], with permission)

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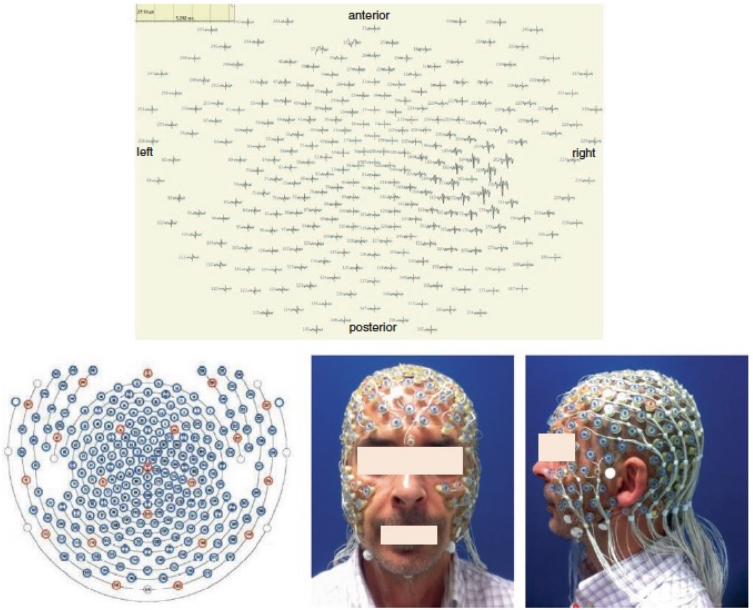

Fig. 4.6 An example of 256-channel high-density EEG, with projected locations of the electrodes on the scalp (courtesy from: Paolo Manganotti, Clinical Neurology Unit, University of Trieste—Italy)

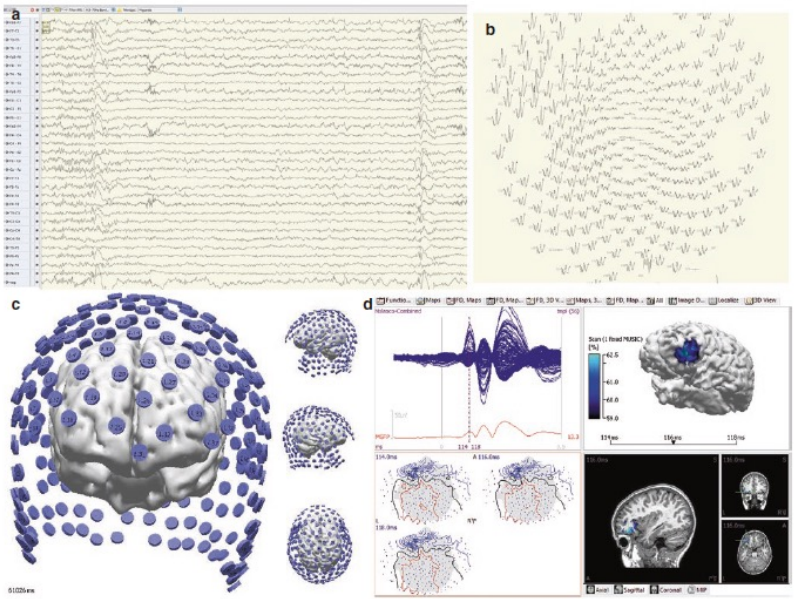

Fig. 4.7 Comparison of standard and high-density EEG in a patient with epileptic left temporal focus; (**a**) standard EEG recording with placement of electrodes according to 10-20 system; (**b**) recording of a single spike by 256 electrodes placed on the scalp; (**c**) the 256 placed electrodes projected onto a 3D image of the patient's brain, obtained by MRI; (**d**) source analysis of epileptic focus (56 spikes average) (courtesy from: Annalisa Rubino, Lino Nobili, Epilepsy Surgery Centre—Niguarda Hospital, Milan, and Child Neuropsychiatry, Department of Neurosciences, University of Genoa)

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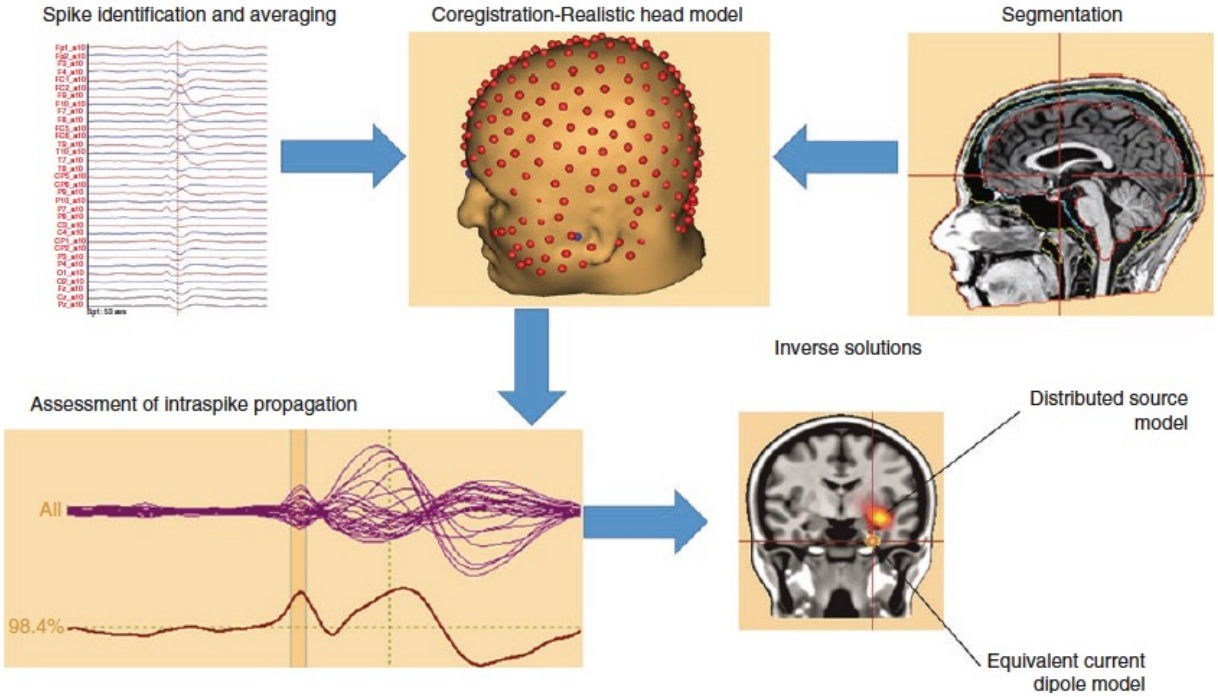

Fig. 20.11 Flowchart of the methodological steps of source imaging

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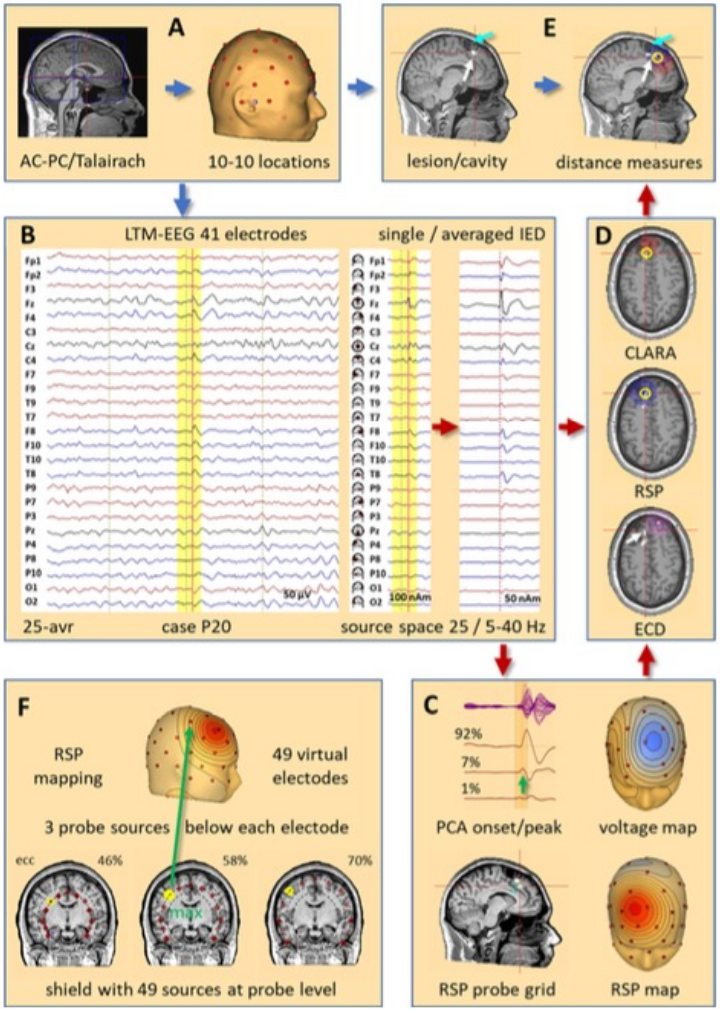


Fig. 1. Workflow of IED processing. Blue arrows show the workflow of MRI, red arrows show EEG processing. A: MRI transformation to AC-PC and Talairach space; automated placement of the 41 recording electrodes onto the individual MRI surface by 10–10 rules in case P20. B: EEG in virtual 25 channel montage; single marked IED segment/average of 60 selected IEDs in source space 25 filtered for pattern search/analysis (5–40 Hz). C: Peak definition by PCA at onset (green arrow at 2nd PCA component) for voltage/RSP mapping and peak localization (few RSP grid points illustrated). D: Source regions localized by CLARA (red), RSP (blue) and ECD (pink). Lesion marked by yellow circle where congruent with source region. E: The marked lesion (white arrow) was next to the cavity of the first resection (cyan arrow). Distance measured from center of source region to closest part of lesion (marked in yellow if < 20 mm). F: Illustration of RSP mapping. RSP calculated for three probes at different depths/eccentricities with their corresponding shields (here shown for C4; three layers illustrated by dashed concentric circles; sources in nearby coronal slices appear in the translucent display). Maximum RSP projected to each of the 49 virtual electrodes for mapping. Abbreviations: IED, Interictal epileptiform discharge; AC-PC, anterior-posterior commissure; PCA, principal components analysis; RSP, regional source power; CLARA, classical LORETA recursively applied; ECD, equivalent current dipole.

Basal EEG - Duration

The duration of standard EEG recordings varies widely among laboratories. European guidelines recommend at least 30 min of EEG recording. Results from studies conducted on epileptic patients suggest that in epilepsy-related indications the shortest duration of standard EEG should be 20 min. Also ACNS has recently established that standard EEG recordings under baseline conditions should consist **at least of 20 minutes of reliable and without artifacts recording, to which activation procedures (Intermittent Photic Stimulation and Hyperventilation) must follow.**

1. Caratteristiche generali dei segnali EEG

- **Morfologia** (**morphology/**wave shape)
- **Voltaggio** (**amplitude**)
- **Frequenza di ripetizione** (**frequency** of repetition)
- **Distribuzione spaziale** (**location/**spatial distribution)
- **Modalità di comparsa** (**rhythmicity, continuity, sinchrony,symmetry**)

Variabilità Inter- e Intraindividuale:

- **età**
- **stato mentale**
- **livello di attenzione/coscienza**
- **caratteristiche individuali**
- **etc,**

Anche in condizioni fisiologiche di tutte queste variabili occorre tenere conto quando si deve valutare un EEG, normale o patologico

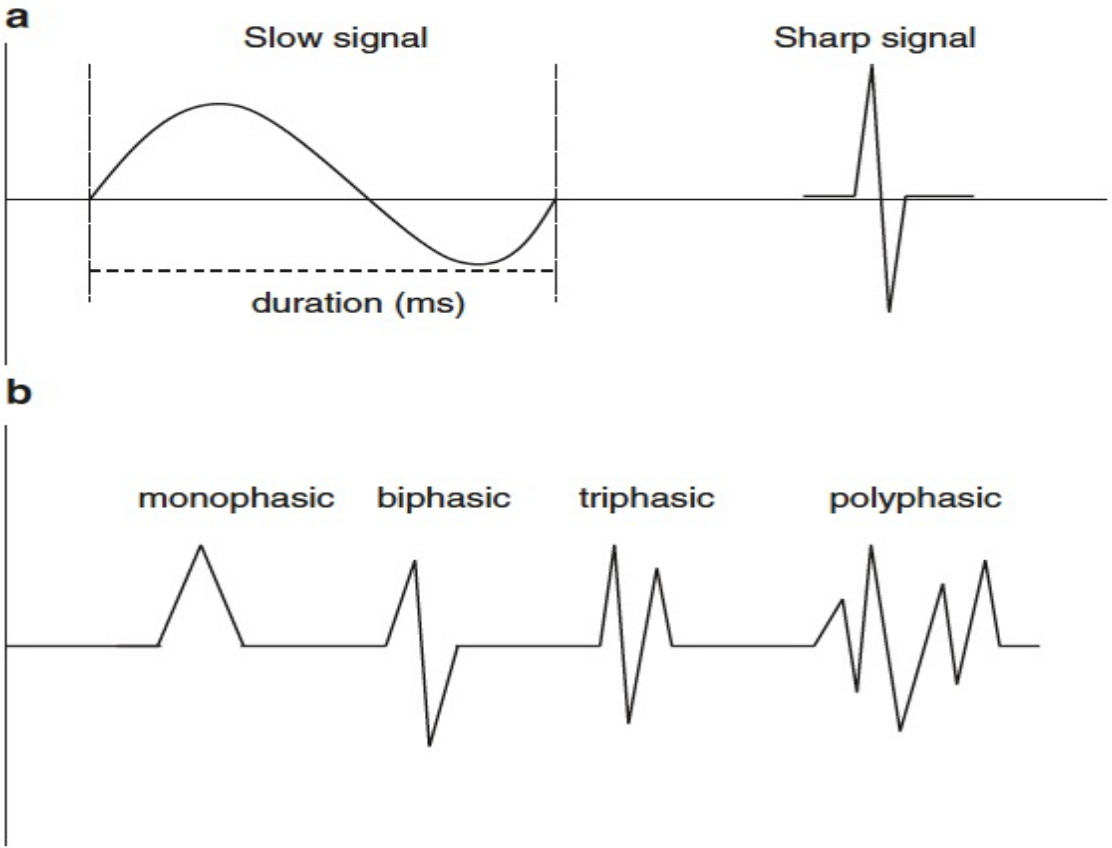


Fig. 9.1 Morphologic characteristics of EEG signals (a) Signal with slow or sharp shape. (b) Various types of signals, differentiating by their phases

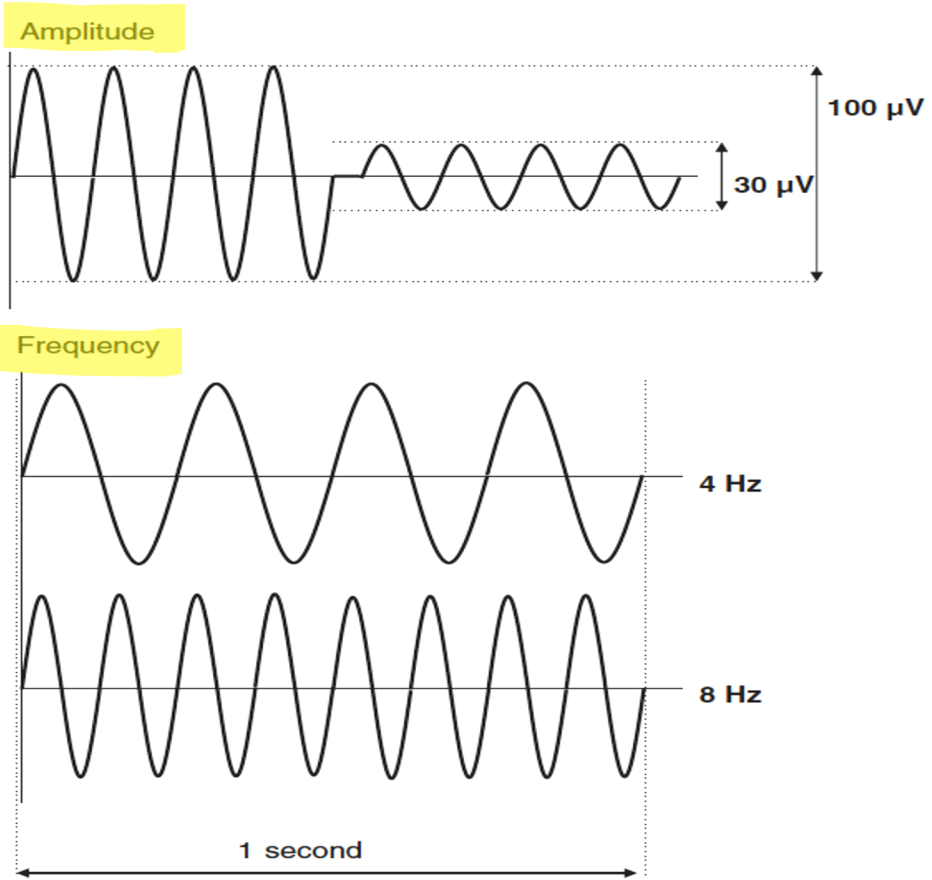


Fig. 9.2 Different amplitude and frequency of EEG rhythm

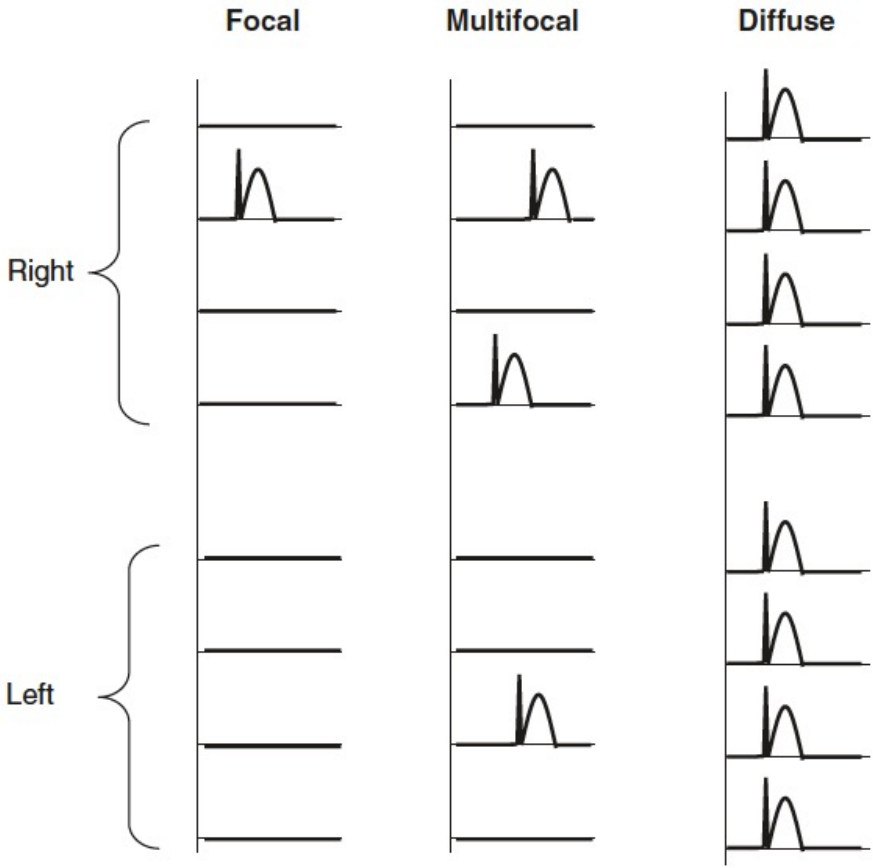


Fig. 9.3 Different localization of EEG signals on two cerebral hemispheres

2. Frequenze EEG

L' ampio spettro delle frequenze EEG è raggruppato in bande, storicamente denominate con alcune lettere greche
(Berger; Gray Walter).

Bande di frequenza classiche:

delta (δ): 0.1 to <4 Hz
theta (θ): 4 to < 8 Hz
alpha (α): 8 to < 13 Hz
beta (β): > 13 - 30 Hz

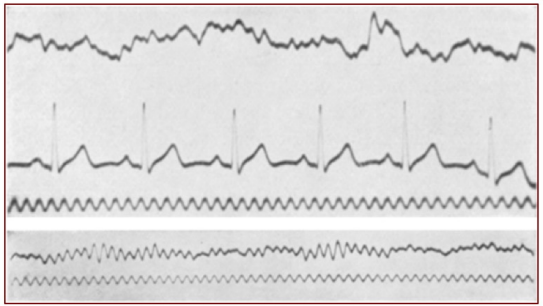
Le attività EEG registrate dallo scalpo durante uno S-EEG in genere escludono le frequenze al di sotto di 0.3-0.5 Hz e al di sopra dei 30 Hz

First paper: Berger H. Uber das Elektrenkephalogramm des Menschen.
Arch Psychiatr Nervenkr **1929**; 87: 527-570.

In his first scientific report Hans Berger described the characteristics of the **alpha rhythm** and defined also the **beta activity**.

In fact, it was Berger who identified with the Greek letter **"alpha"** the normal occipital background rhythm (reported in awake state, with eyes closed and with responsiveness to opening) and with the letter **"beta"** the more rapid frequency activity.

The first EEGs recorded by Berger on photographic paper lasted 1-3 minutes and they consisted of **1 EEG channel**, with frontal-occipital bipolar derivation, **1 channel for** simultaneous recording of the **electrocardiogram**, and **1 channel** for **time marking**



Always in England, from **1936 William Grey Walter** (1910-1977) became the **pioneer of clinical EEG**

Grey Walter built an oscilloscope able to record **three EEG channels** and this method began to be used for the ***diagnosis of cerebral tumours and as a rudimentary monitoring system***, both during surgery and to confirm the effects of anaesthesia.

It was Grey Walter who, following Berger's example, identified with the Greek letter **"delta"** and **"theta"** the **slow EEG activity** and who, as early as 1943-44, introduced the **first methods for automatic analysis** of the cerebral bioelectric signal.

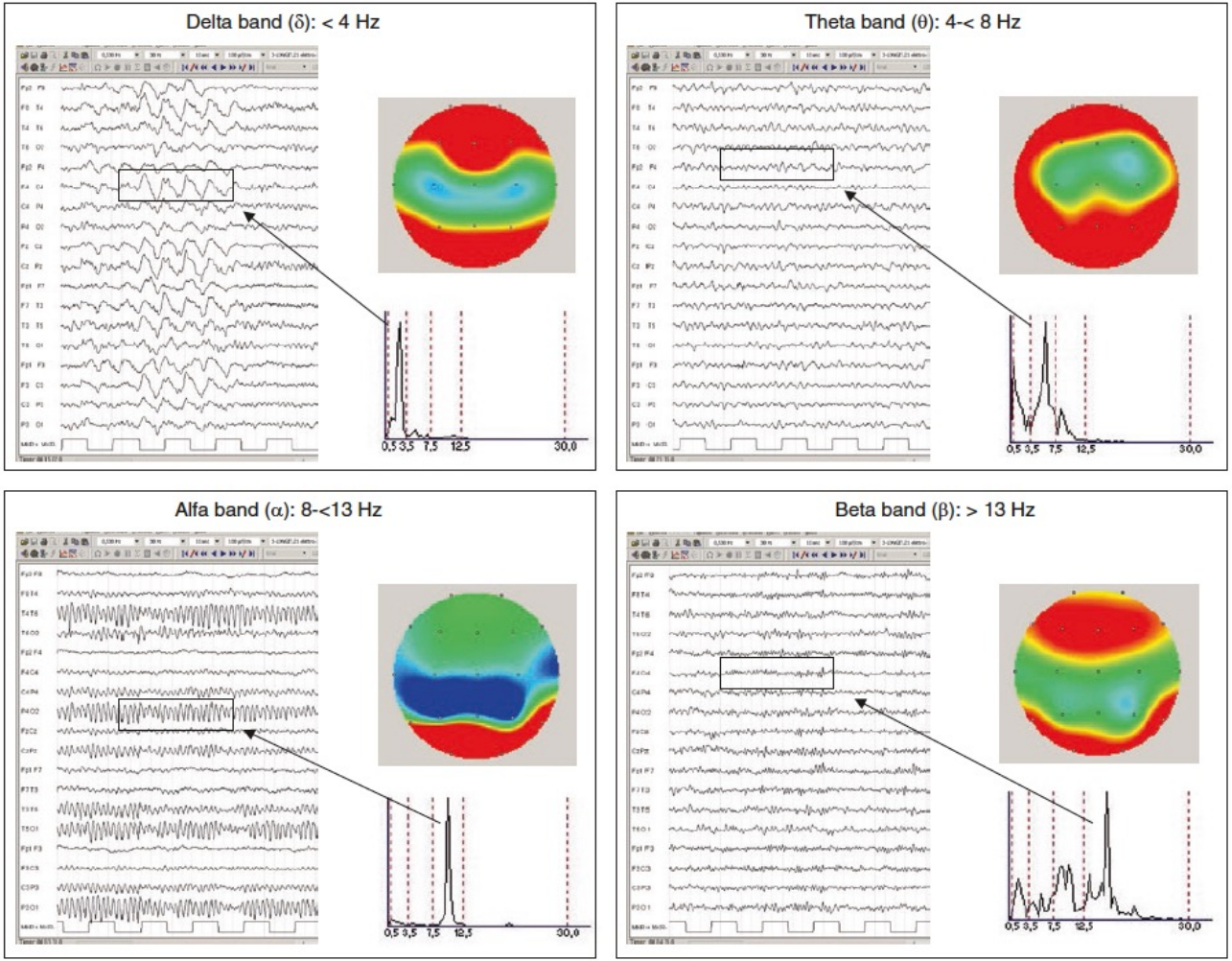
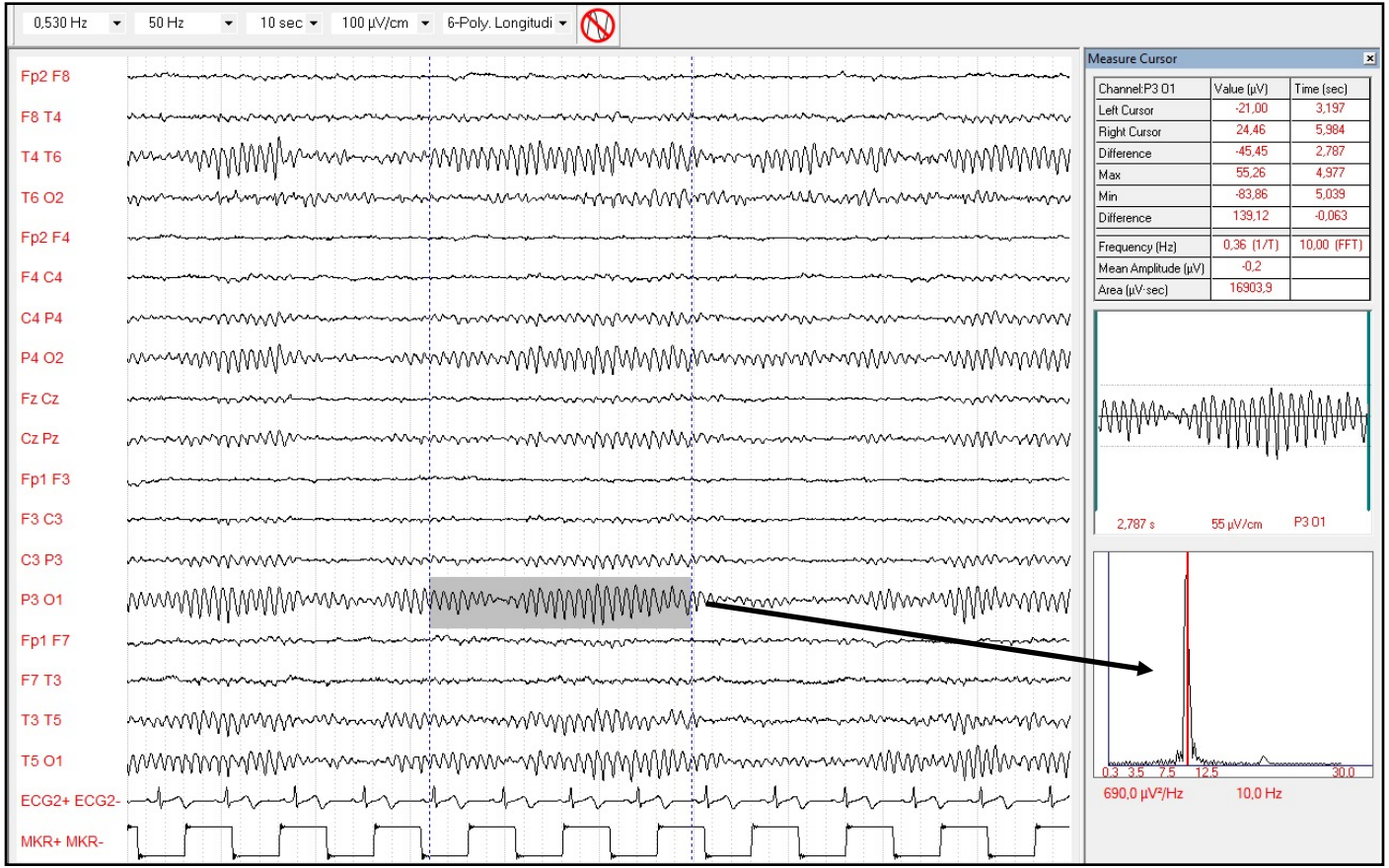


Fig. 9.4 Qualitative and spectral representation of the main EEG frequency bands

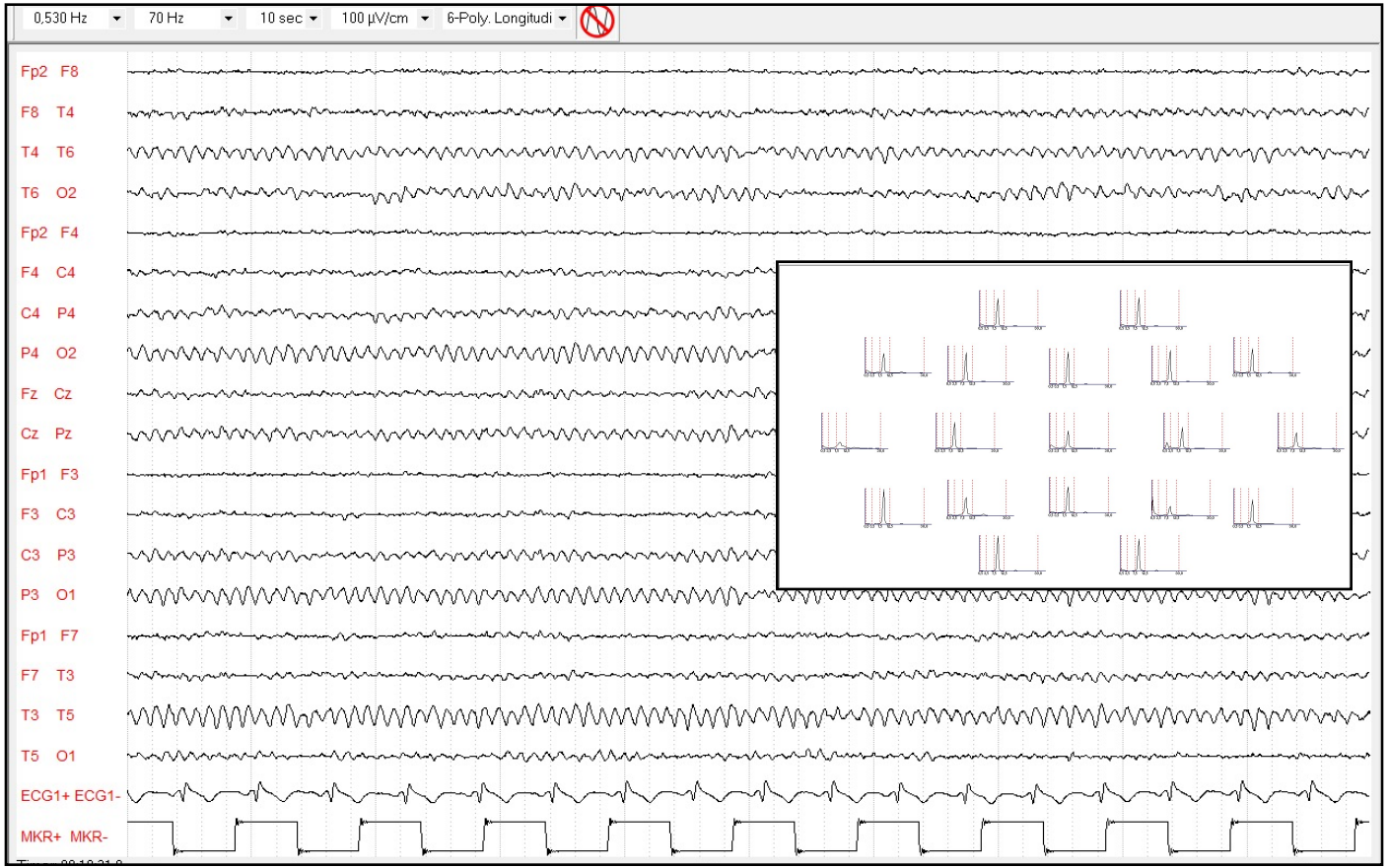
Hz	EEG bands
0 - 0.3	Ultraslow
0.3 - < 4	delta – δ
4 - < 8	theta – θ
8 - < 13	alpha – α
13- < 30	beta – β
30 - < 80	gamma – γ
	HFOs
80-250 Hz	Ripples
250-500 Hz	fast ripples
> 500 - 1000 Hz	ultrafast ripples

Ritmo di fondo ALFA (Alpha rhythm)

The *alpha rhythm* (or *posterior dominant rhythm*) is the hallmark of the awake EEG in older children and adults, with eyes closed or visually inattentive and during the psycho-sensorial relaxed condition.
The alpha rhythm has a **typical location** in the posterior head regions;
in some subjects the alpha rhythm is strictly confined to occipital areas, but in others spreads forward all the posterior quadrants (posterior temporal, parietal and occipital regions).
At times the posterior rhythm may even spread to the central and frontal areas (F4 and F3 electrodes), mainly in aged subjects or during drowsiness.



An example of alpha rhythm in an awake adult subject with eyes closed. The tract delimited by the dotted lines shows a background alpha rhythm with a frequency of 10 Hz, and with a peak of amplitude of 55 uV/cm. Alpha rhythm is typically visible on the posterior regions with a regular and sinusoidal morphology.

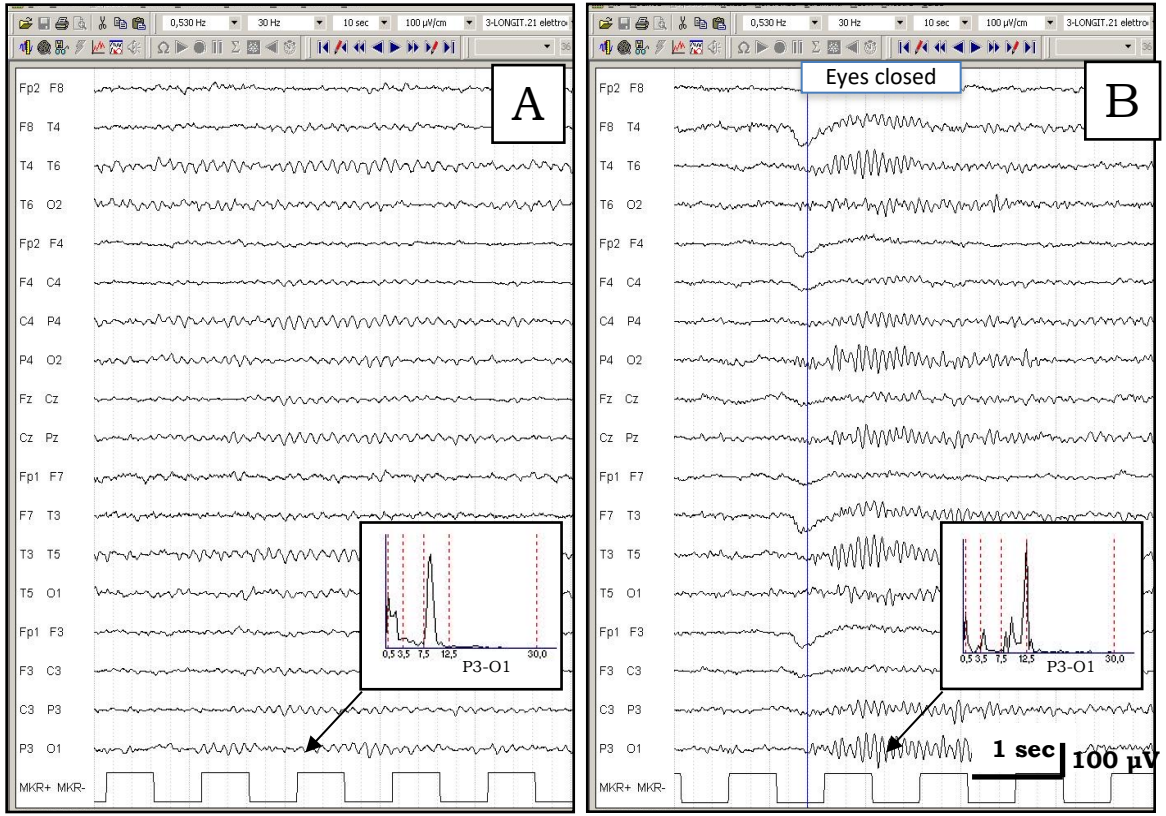


An example of alpha rhythm recorded in a normal adult subject with eyes closed, during drowsiness. Alpha activity spreads from the posterior regions to the central and frontal areas, as demonstrated by the spectrograms

The morphology of alpha rhythm is mostly regular (**monomorphic rhythm**) with sinusoidal or fusiform shape (increase and gradual decrease in amplitude, **"waxing and waning"**; peaks and throughs of the waveform rounded and regular). Rarely the alpha rhythm has a sharpened morphology (**"spiky alpha"**, with the negative component of wave sharp and the positive component rounded) and is considered a normal variant, especially in older children and young adults.

The alpha rhythm has a f**requency** within the range of alpha band (8 - <13 Hz) and in adults it generally ranges from 9 to 11 Hz. The frequency is generally the same in each hemisphere, but in the same individual it may vary in terms of 1 or even 2 Hz, while in a stable condition. Some frequency variations of the alpha rhythm may be dependent on the menstrual cycle (acceleration of alpha frequency in the premenstrual phase, with reduction of its amount or be related to the body temperature (increase of frequency during rising temperatures).
Recently has been demostrated in adult patients that the posterior alpha rhythm is significantly slower in the wake periods of sleep-recordings (before and after sleep), compared to standard wake recordings.

Brief faster alpha rhythm sequences than baseline can also be recorded immediately after the closing of the eyes (**squeak phenomenon**)



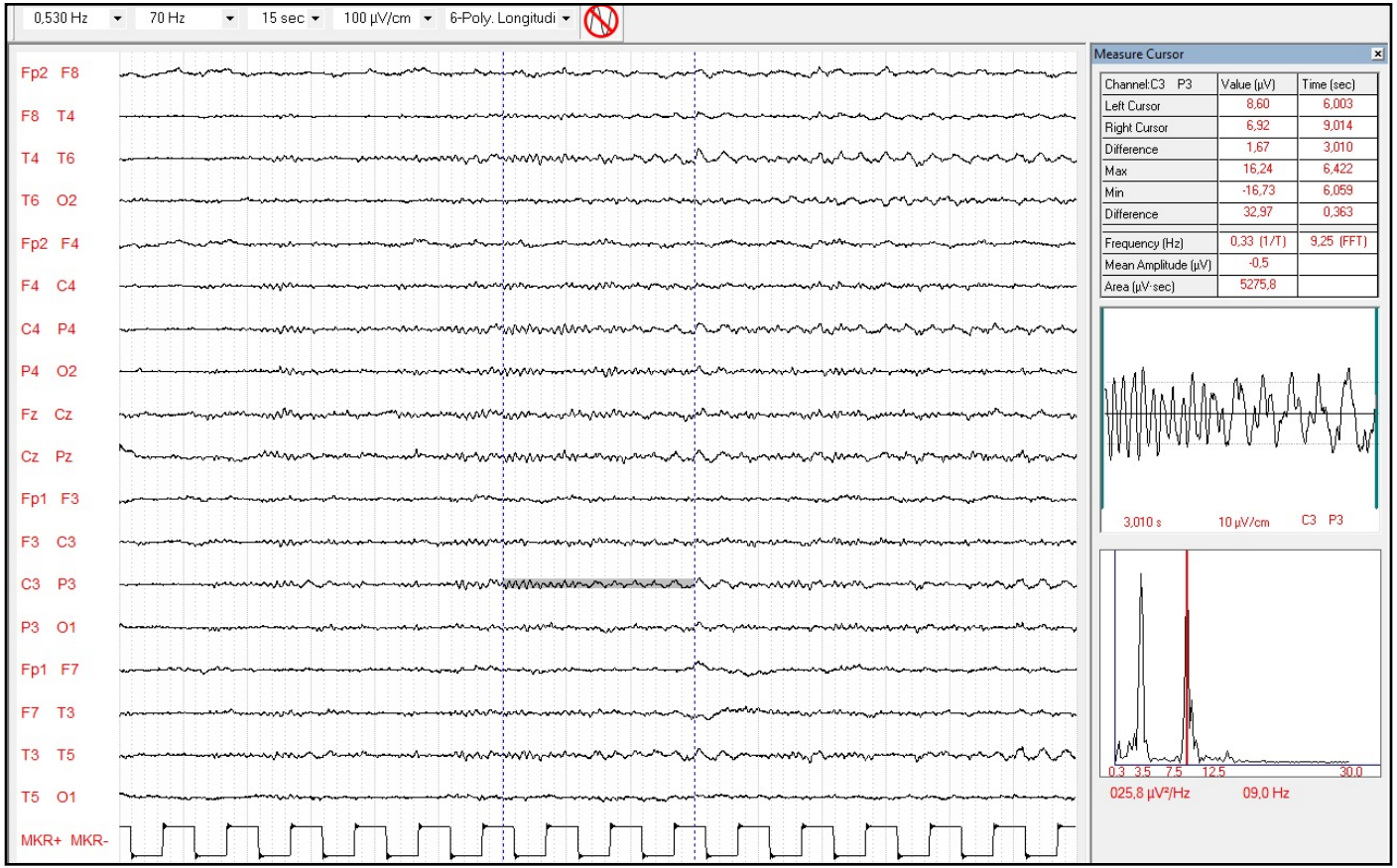
Squeak phenomenon

VARIANTI dell'ALFA (Alpha variants)

Sometimes the normal sequences of alpha rhythm are substituted by **slower** or **faster** frequencies (**slow or fast alpha variants**).

The term **"slow alpha variant"** is referred to the appearance of a subharmonic of the fundamental rhythm (with a ratio of 1:2 or 1:3; if for example the alpha rhythm has a frequency of 12 Hz its slow variants will have a frequency of 6 or 4 Hz respectively). The alpha slow variants have the same topography and reactivity as the fundamental rhythm but can also be unilateral or asymmetric.

More rarely the appearance of **fast variants of alpha** can also observe (16-20 Hz), for the most part mixed with alpha activity and with the same topography and reactivity. The fast variants can be activated by Intermittent Photic Stimulation (IPS) and induced by benzodiazepines.



A combination of posterior alpha rhythm at 9 Hz and its subharmonic at 3 Hz (slow alpha variant).

The **amplitude** of posterior alpha rhythm in most adults is less than 50 μV.

In order to be able to measure the amplitude in an objective manner, a digital measurement cursor must be used, checking the amplitude values randomly at various points in the tracing and then calculating a mean value. This procedure is difficult to implement during a routine EEG inspection and therefore visual assessment of the alpha activity amplitude can be very arbitrary; it is then preferable to make a subjective quantification of the amplitude in terms of low, medium and high.

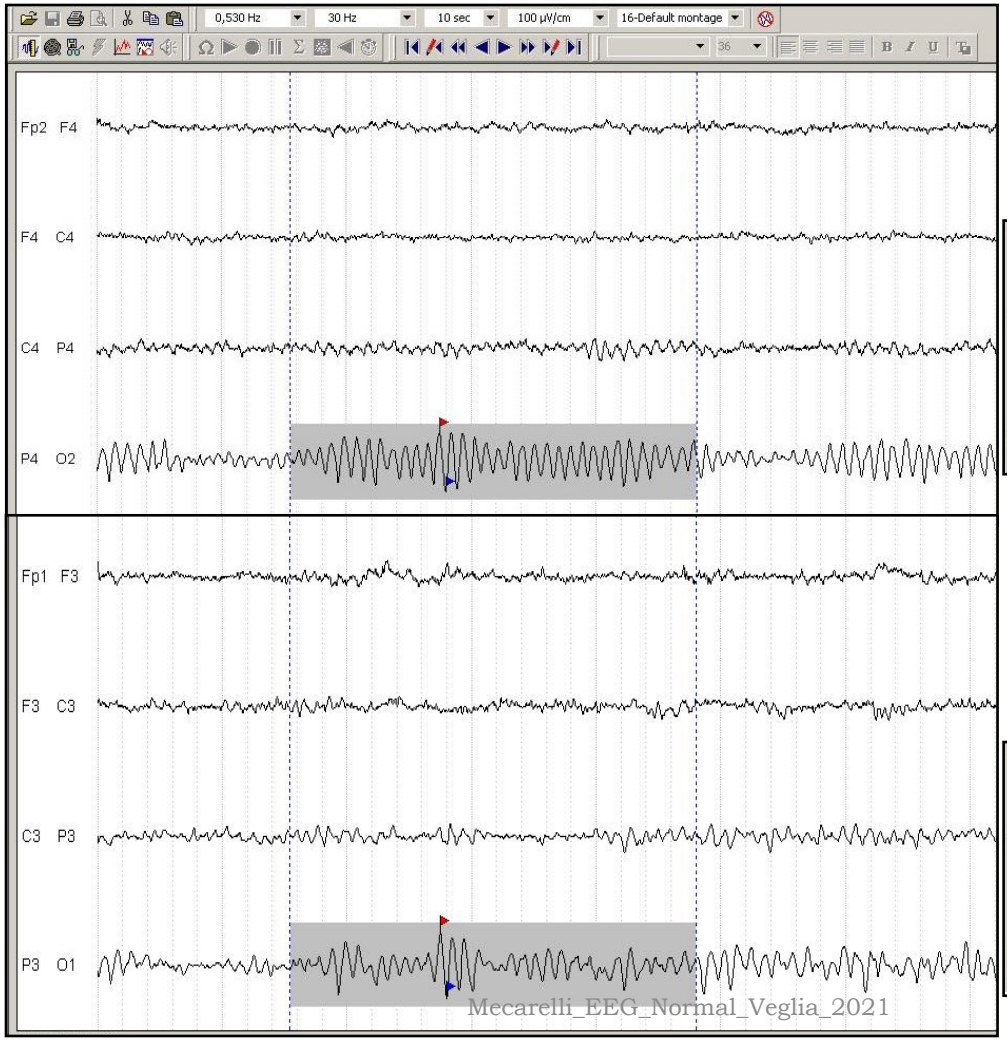
Usually the amplitude of posterior alpha rhythm is asymmetrical on the two hemispheres, with a **lower voltage over the dominant hemisphere** (the left in right-handed individuals and the right in the left-handed).

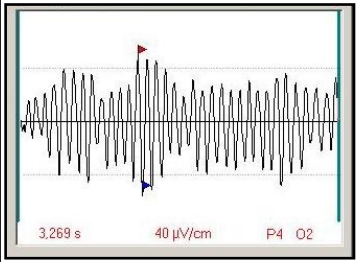
However, the difference in the posterior alpha rhythm amplitude between the two hemispheres usually not exceed 20 %.

An **asymmetry of posterior alpha rhythm is considered to be abnormal** when in the non-dominant hemisphere it is more than three-fold greater than that of the dominant hemisphere.

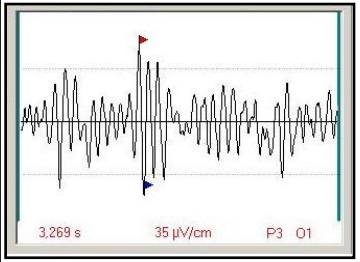
On the other hand, when the voltage of the alpha rhythm is higher over the dominant hemisphere the difference is considered abnormal if the amplitude is two-fold greater compared to contralateral side.

Differences of amplitude less than 50% are not, however, considered to be pathological.

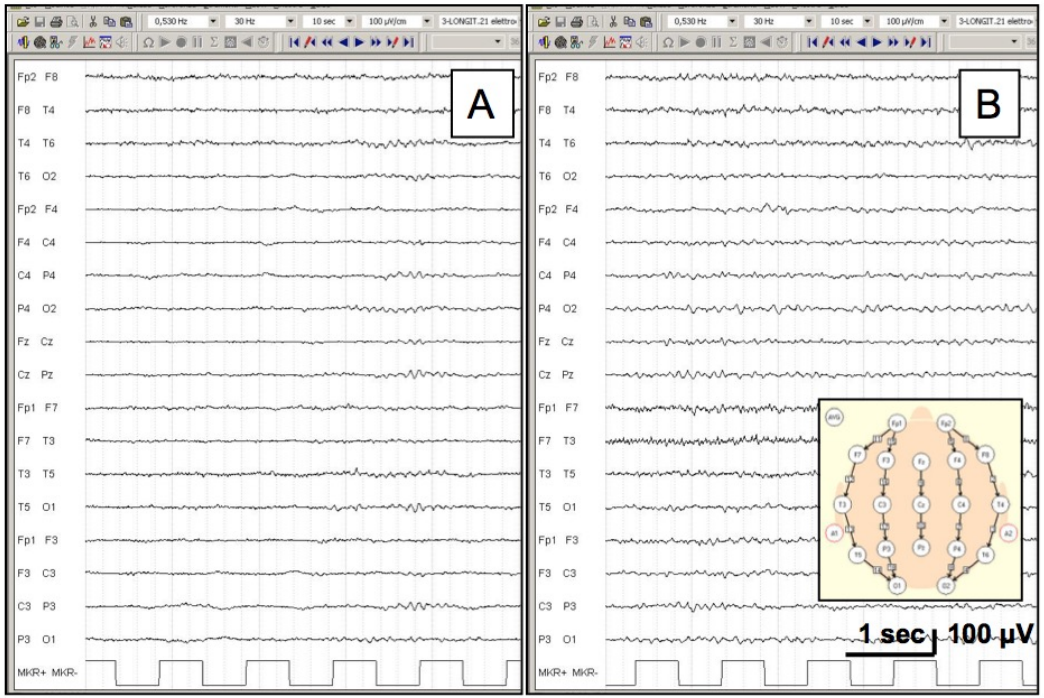




Normal asymmetry
(right > left)
of the posterior
alpha rhythm.



There are also normal EEG tracings in which the alpha activity is poorly appreciable (**low amplitude or desynchronised tracings**).
In these cases, often the **desynchronisation** of background activity is correlated to the state of **anxiety** due to examination or characteristic of the subject or is dependent on the non-relaxing registration environment.



A: a poorly appreciable alpha rhythm in an adult subject during basal condition;
B: during hyperventilation alpha activity appears better synchronized

Reazione d'arresto del ritmo alfa:

- completa
- parziale
- paradossa

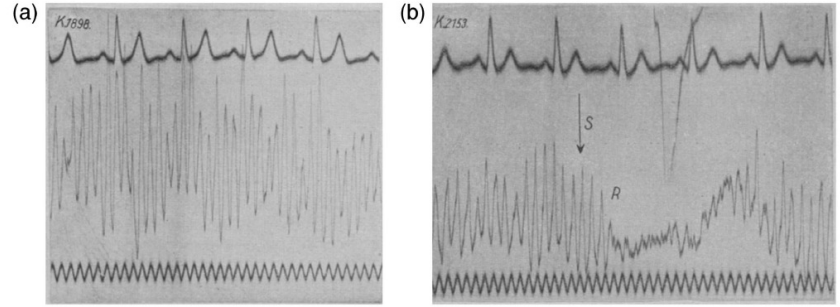


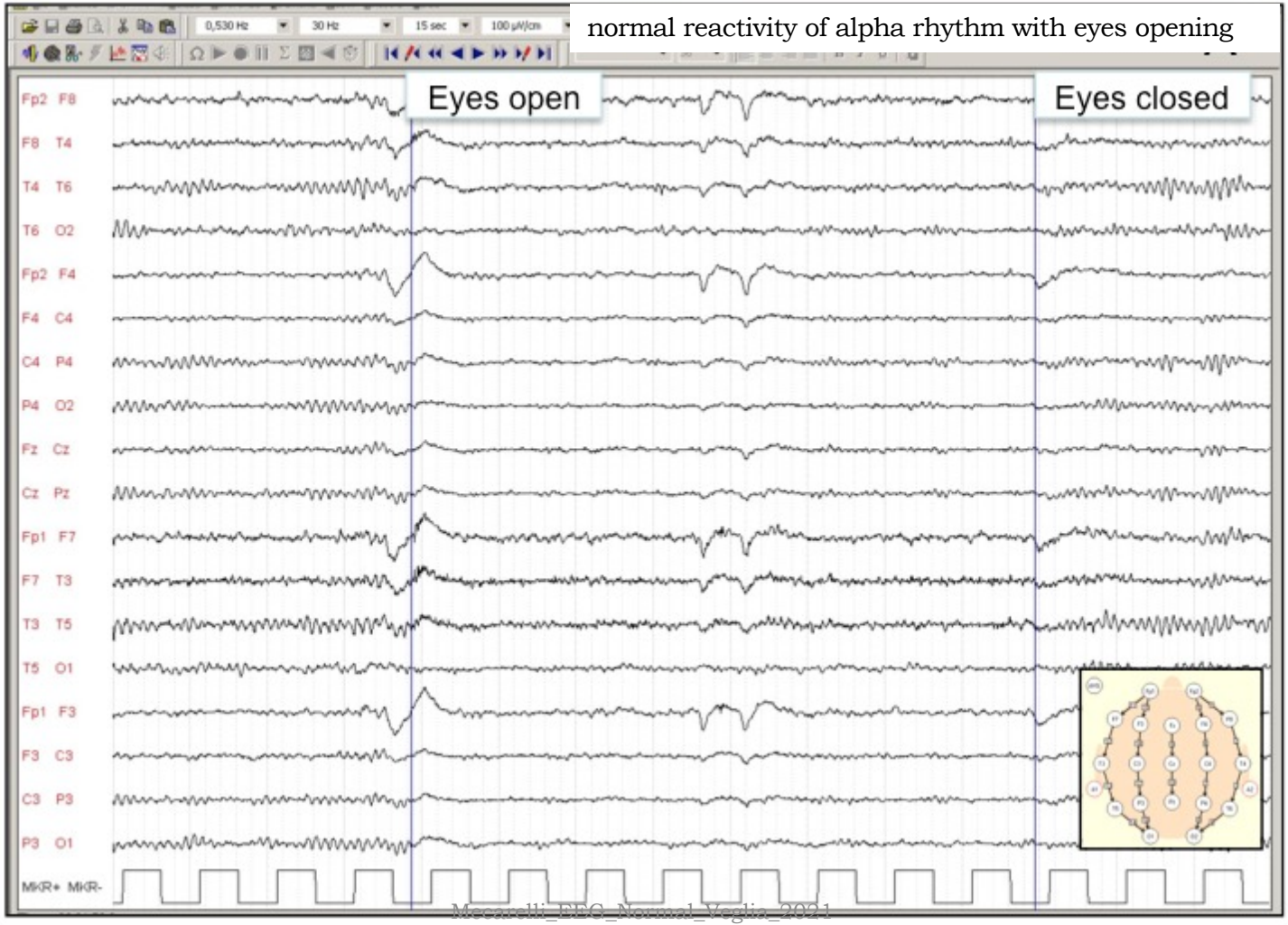
FIGURE 1 Sample recordings by Hans Berger from a family member and a patient. (a) Figure 3 in Berger (1935a): 'J.B., 14 years old'. Typical of his earlier work, the electrocardiogram is depicted in the top trace, and a time trace showing 1/10 s is shown below. The centre trace is the EEG, here recorded using silver chloride needle electrodes in a bipolar montage on forehead and occiput. (b) Figure 4 in Berger (1935a): 'M.M., 33 year old man. Large gap in the skull from the left forehead to the parietal region.' Here, the EEG was recorded epidurally using silver chloride needle electrodes placed 4.5 cm apart inside the gap in the skull. At the time marked with the arrow and S, a needle was briefly inserted into the patient's left index finger. R indicates the subsequent extinction of alpha. [Reprinted by permission from Springer Nature Customer Service Centre GmbH: Springer Nature, Naturwissenschaften (Das Elektrenkephalogramm des Menschen, Hans Berger), Copyright © 1969, Verlag von Julius Springer (1935)]

In addition to the **frequency**, the **location** and the **amplitude**, the other fundamental characteristics of the posterior alpha rhythm is its particular **reactivity**. This physiological posterior rhythm, very well **synchronised with closed eyes** and being in a relaxed state of wakefulness, **suppresses dramatically when the eyes are opened** and/or during heightened mental activity (such as arithmetic calculation), somatosensory stimuli, etc.

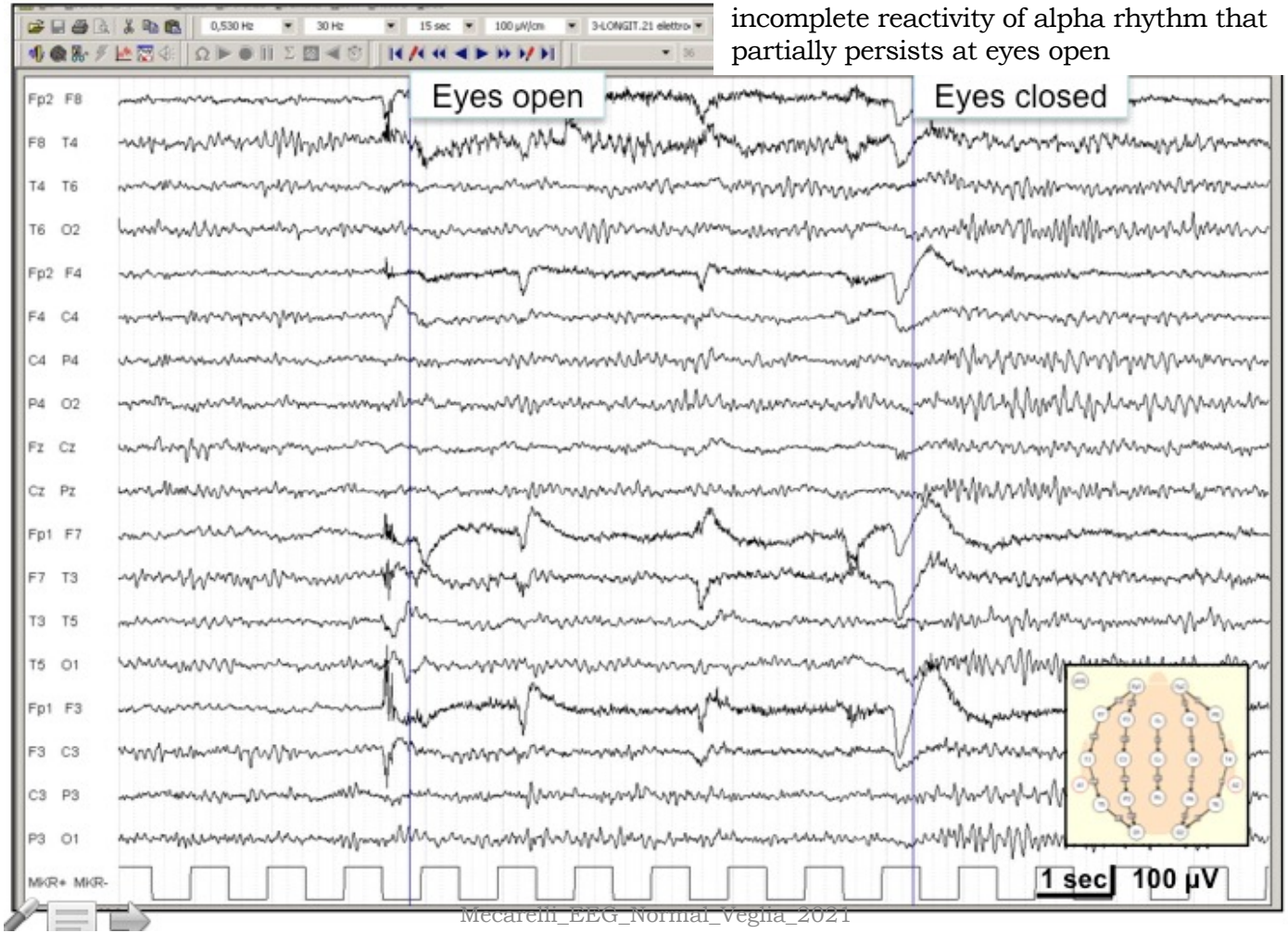
This phenomenon is what goes under the name of "**blocking arrest of alpha**", discovered in his first report by Berger: during the EEG recording with closed eyes is usually observed the synchronised alpha rhythm thath disappears immediatly as the technician ask the subject to open his eyes or to perform specific mental tasks. However, it must be emphasized that sensory stimulation and mental activity block the alpha rhythm in a less pronounced way than the opening of the eyes. **Sometimes the blocking reactivity is only partial**, and with open eyes persist an alpha rhythm poorly modulated.

On the other hand the blocking reactivity of alpha rhythm is instead defined as "**paradoxical**" when in subjects with closed eyes, there prevails a background activity of theta frequency (often related to drowsiness), in which the command of opening the eyes determines, as awakening reaction, the reappearance of alpha rhythm absent before.

normal reactivity of alpha rhythm with eyes opening



incomplete reactivity of alpha rhythm that partially persists at eyes open



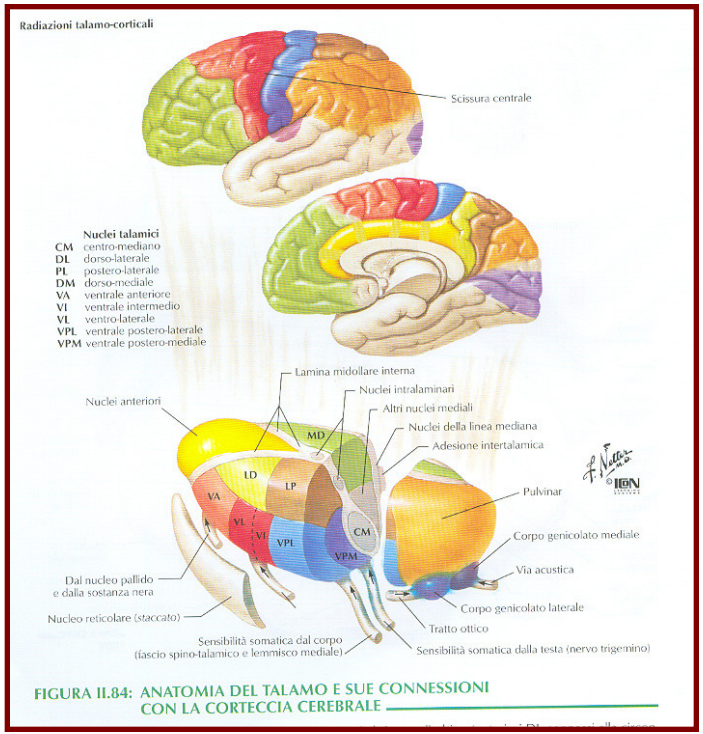
Genesis of the alpha rhythm

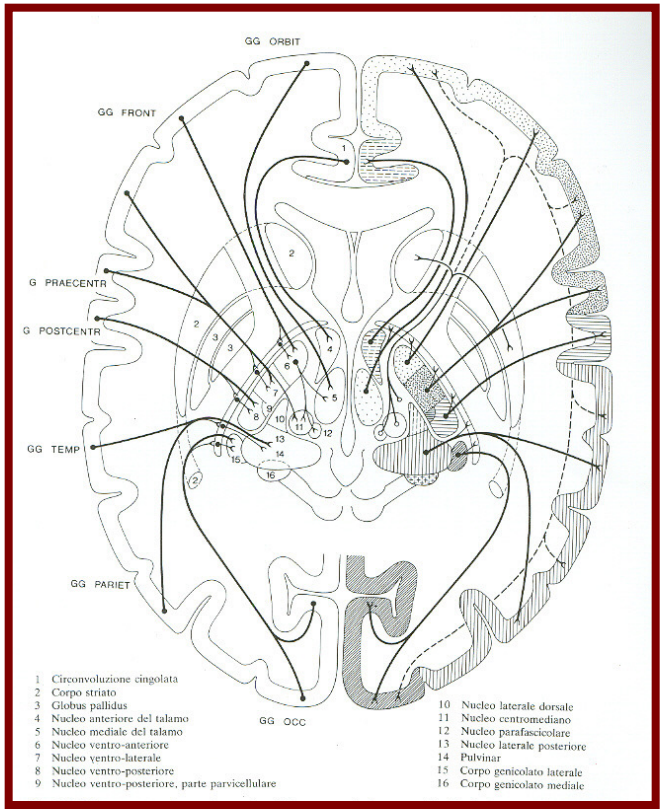
The majority of authors who have dealt with the genesis of the alpha rhythm have established that it is a rhythm with a **cortical origin but with an important thalamic governance**. Bishop in 1936 first proposed the concept of cortico-thalamic reverberating circuits, a concept that was later reaffirmed by many others. Andersen and Andersson in1968 proposed the theory of the ***pacemaker thalamic***, based on similitaries between human alpha activity and experimental splindle activity induced in animals by barbiturates. According to this theory, the alpha rhythm is generated on the basis of rhythmic inputs that come from the thalamic centers. These inputs, through specific thalamo-cortical fibers, excite the upper cortical centers, inducing a synchronisation in them. This type of **thalamo-cortical excitation** would also involve the activation of inhibitory interneurons acting only at the level of the thalamic circuits. Therefore, **according to this theory, in the thalamus an alternation of excitation/inhibition is then determined, wich would be the basis of the synchronisation and the rhythmicity of cortical activity.**

In recent decades the genesis of alpha rhythm has not been further clarified and demonstrated. From both clinical and experimental data it can be assumed that **cortical alpha rhythm is the result of cortico-cortical and thalamo-cortical systems interacting with each other in very complex way.**

The neurophysiological mechanisms underlying the reactivity of the alpha rhythm (i.e. the blocking of alpha with eyes open or during mental activities) are also not well elucidated, but it is supposed that **in the desynchronization** of the tracing **plays a fundamental role the ponto-mesencephalic reticular formation**, through its activating action towards the cerebral cortex.


Cortex
Thalamus


Radiazioni talamo-corticali
Scissura centrale
Nuclei talamici
CM centro-mediano
DL dorso-laterale
PL postero-laterale
DM dorso-mediale
VA ventrale anteriore
VI ventrale intermedio
VL ventro-laterale
VPL ventrale postero-laterale
VPM ventrale postero-mediale
Lamina midollare interna
Nuclei anteriori
Nuclei intralaminari
Altri nuclei mediali
Nuclei della linea mediana
Adesione intertalamica
MD
LD
LP
VA
VL
VI
VPL
CM
VPM
Pulvinar
Corpo genicolato mediale
Via acustica
Corpo genicolato laterale
Tratto ottico
Dal nucleo pallido e dalla sostanza nera
Nucleo reticolare (staccato)
Sensibilità somatica dal corpo (fascio spino-talamico e lemnisco mediale)
Sensibilità somatica dalla testa (nervo trigemino)
FIGURA II.84: ANATOMIA DEL TALAMO E SUE CONNESSIONI CON LA CORTECCIA CEREBRALE

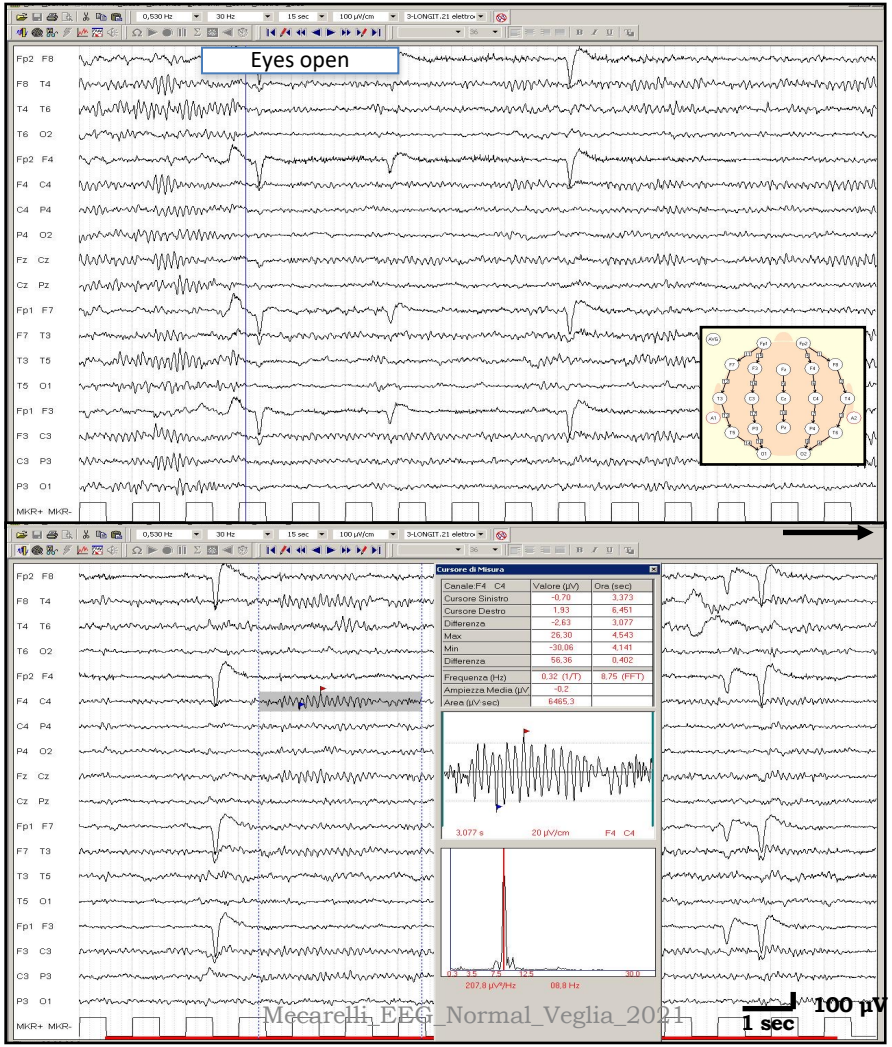

GG ORBIT
GG FRONT
G PRAECENTR
G POSTCENTR
GG TEMP
GG PARIET
GG OCC
1 Circonvoluzione cingolata
2 Corpo striato
3 Globus pallidus
4 Nucleo anteriore del talamo
5 Nucleo mediale del talamo
6 Nucleo ventro-anteriore
7 Nucleo ventro-laterale
8 Nucleo ventro-posteriore
9 Nucleo ventro-posteriore, parte parvicellulare
10 Nucleo laterale dorsale
11 Nucleo centromediano
12 Nucleo parafascicolare
13 Nucleo laterale posteriore
14 Pulvinar
15 Corpo genicolato laterale
16 Corpo genicolato mediale

Ritmo mu - Mu rhythm (μ)

Described for the first time by Gastaut in 1952 the **mu rhythm,**

(also known by discouraged terms such as rhythm en arceau, comb rhythm, rolandic rhythm, wicket rhythm and somatosensory alpha rhythm)

is characterized by brief and/or prolonged rhythmic sequences of waves with the frequency similar to that of an alpha posterior rhythm but with a different location and reactivity.
The most typical frequency of mu rhythm fluctuates **from 7 to 11 Hz**, and it is often mixed with beta activity.
This rhythm is typically seen in wakefulness but can also be seen in stages N1, N2 and R of sleep.
The amplitude of mu is generally less than 50 uV (usually lower than the amplitude of the alpha rhythm) and its **maximum spatial representation is in the central regions** of scalp (below the C4, C3 and Cz electrodes).
The waves which constitute the mu rhythm are **arciform** (with sharpened negative phase and rounded positive phase) and they appear **either at eyes open or closed**, in subjects with motor inactivity.



Mu rhythm, better evident with eyes open; the peak of frequency is at 8.8 Hz, and maximum spatial representation is on the central areas, bilaterally

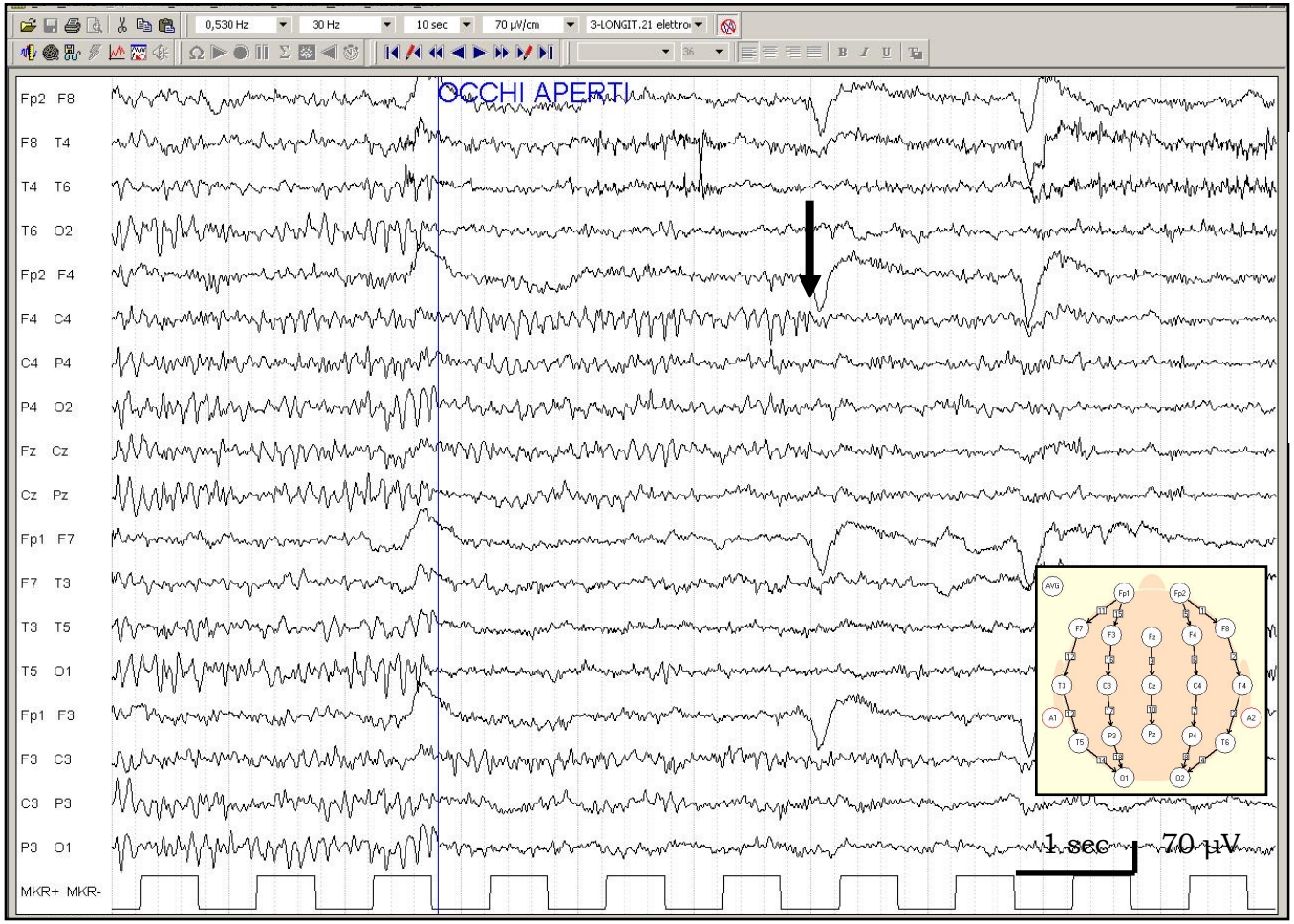
The prevalence of mu rhythm reported in the literature during the registration of EEGs in healthy subjects or patients is very variable with values of 10-20% in young adults and a lower incidence in children and the elderly.

Probably however the prevalence of mu rhythm is higher; in fact, during closed-eye recordings the mu rhythm can be confused with the alpha rhythm transmitted in the central regions. In addition, during routine EEG recordings the traits of tracings performed at eyes open are very short, and therefore the mu rhythm may not be highlighted. With more sophisticated quantitative analysis of the EEG could highlight the mu rhythm in almost all healthy adult subjects.

The **mu rhythm may be unilateral or bilateral, asynchronous and / or asymmetrical** in the two hemispheres, with no particular correlation with hemispheric dominance.

Isolated reports have suggested that an unilateral mu rhythm can be associated with a structural ipsilateral cortical lesion, even in the absence of additional EEG abnormalities.

The most important peculiarity of the mu rhythm is its reactivity. The mu rhythm is indeed blocked by the movements (active, passive or reflex), by the tactile stimulation of body areas or even by the mental planning of movement. If the mu rhythm is well evident only from one side the best manoeuvre to visualise its reactivity is to ask the subject to tighten the contralateral hand, or think about permorfing this action.



Sequences of mu rhythm, more evident on right central region, that are blocked by asking the subject to tighten the contralateral hand (at arrow).

The proof that the thought of performing the movement is sufficient to block the mu rhythm is shown by the fact that the rhythm stops a few millisecond before the beginning of the voluntary movement itself. In addition even subjects with an amputed limb show this reactivity of the contralateral mu rhythm, after thinking about the execution of the movement in the phantom limb.
Finally, it was noted that mu rhythm is enhanced or sometimes facilitated by Intermittent Photic Stimulation (IPS) and pattern vision, **validating the idea that for this rhythm the integration of sensory and visual function is relevant.**

The mu rhythm in the scalp's central areas was correlated with the beta activity (around 20 Hz) described in the motor cortex in patients during electrocorticographic recording. Also this activity could be blocked by contralateral movement or with thinking about the execution of movement.

Although the mu rhythm is present in a high percentage of healthy subjects and it represents a physiological normal EEG pattern, **in the past** it has often been attributed to a pathological significance or **it is interpreted as a "borderline pattern"**.

One of the historical theories on the genesis of the mu rhythm was in fact that it was an **expression of hyper-excitability of the rolandic cortex**.

More recent studies have instead propose to interpret the mu rhythm as an important information processing function that connects the perception with the action; specifically it would be the expression of the processes involved in the transformation of "seeing" and "hearing" in the "doing."

	alpha rhythm	*mu rhythm*
Frequency (Hz)	*8 - < 13*	*> 7 – 11*
Variants	*slow – fast*	*absent or rare*
amplitude (uV)	*< 50*	*< 50*
Location	*posterior areas*	*central areas*
Conditions of occurrence	*closed eyes*	*either closed and open eyes*
Reactivity	*opening of the eyes*	*movement, tactile stimulus, mental movement planning*

Characteristics of the alpha rhythm and the mu rhythm

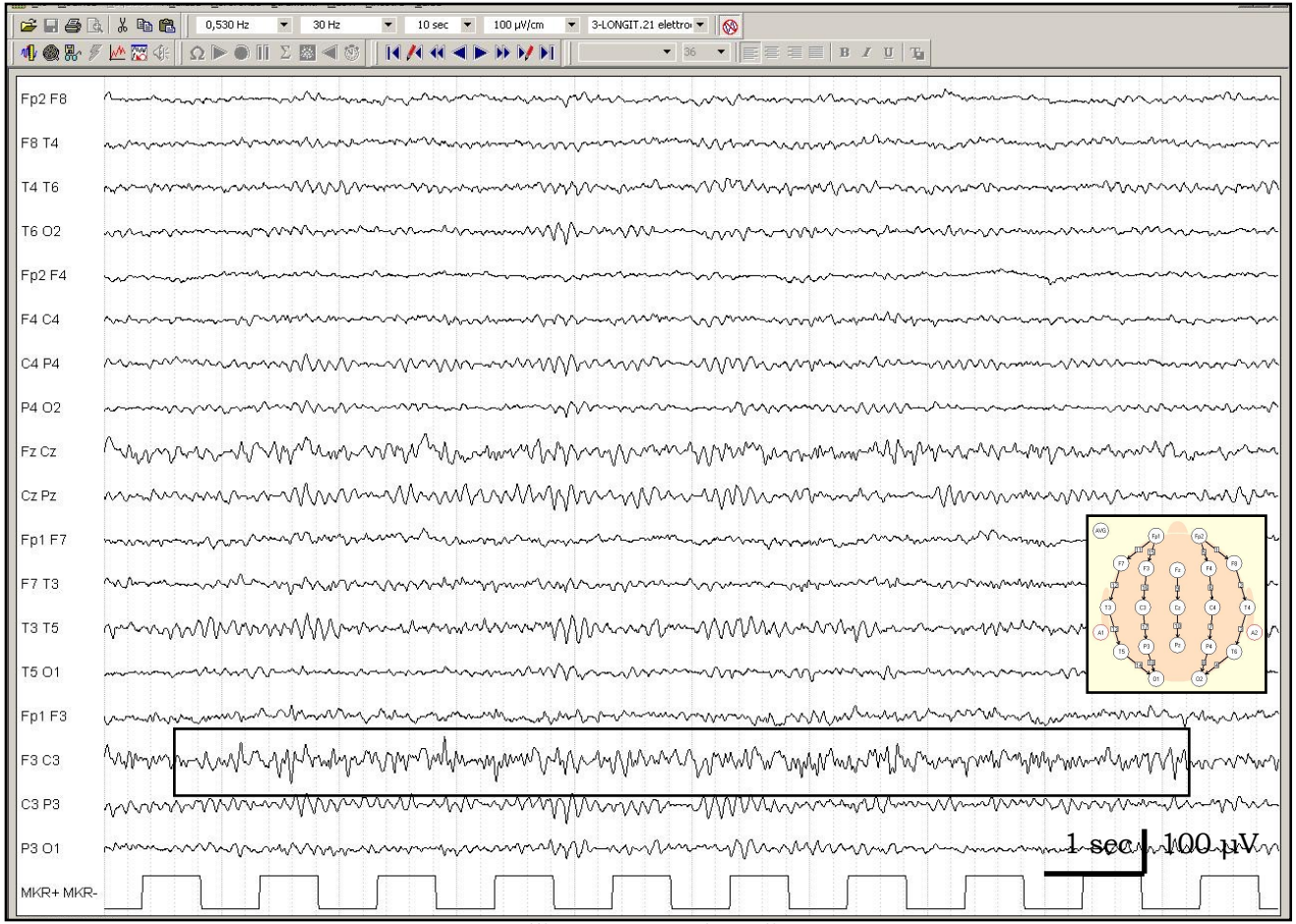
Ritmo di breccia (Breach Rhythm)

Breach rhytmh (BR) consists of a focal sequences of rhythmic or pseudorhythmic mixed activity (theta, alpha and beta), sometimes associated with focal slowing or sharp activity or isolated spikes. The breach rhythm develops over or near the area of bony skull defect, such as after craniotomy for neurosurgery or traumatic lesions, and it is not indicative of brain dysfunction.

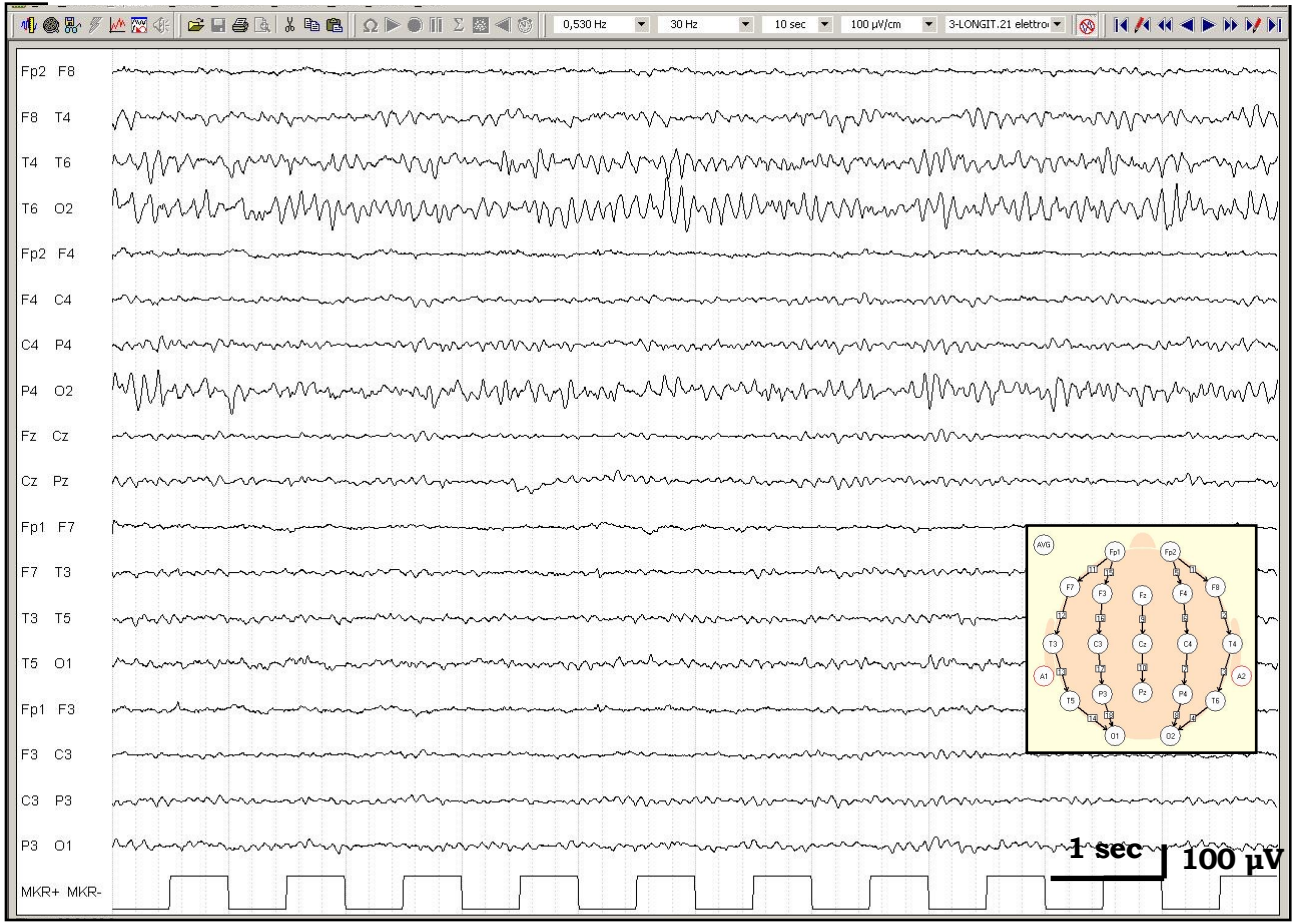
The breach rhythm may be considered as a consequence of a skull defect and not as an epileptiform abnormality.

The genesis of BR is not fully understood but the bone defects caused a reduced filtering effect and reduced electrical impedance. Sharpening and irregular morphology of BR may be misinterpretated, leading to a misdiagnosis of epilepsy. Therefore a correct differential diagnosis is important for the relevant clinical consequences. BR occurs in wakefulness but may persist also in sleep. If located over central region it may be blocked by contralateral movement, because of the coexistence of an underlying mu rhythm, but this reactivity is not observed over the temporal region.

Breach rhythm recorded on left fronto-central region in a man who underwent craniotomy in the same site

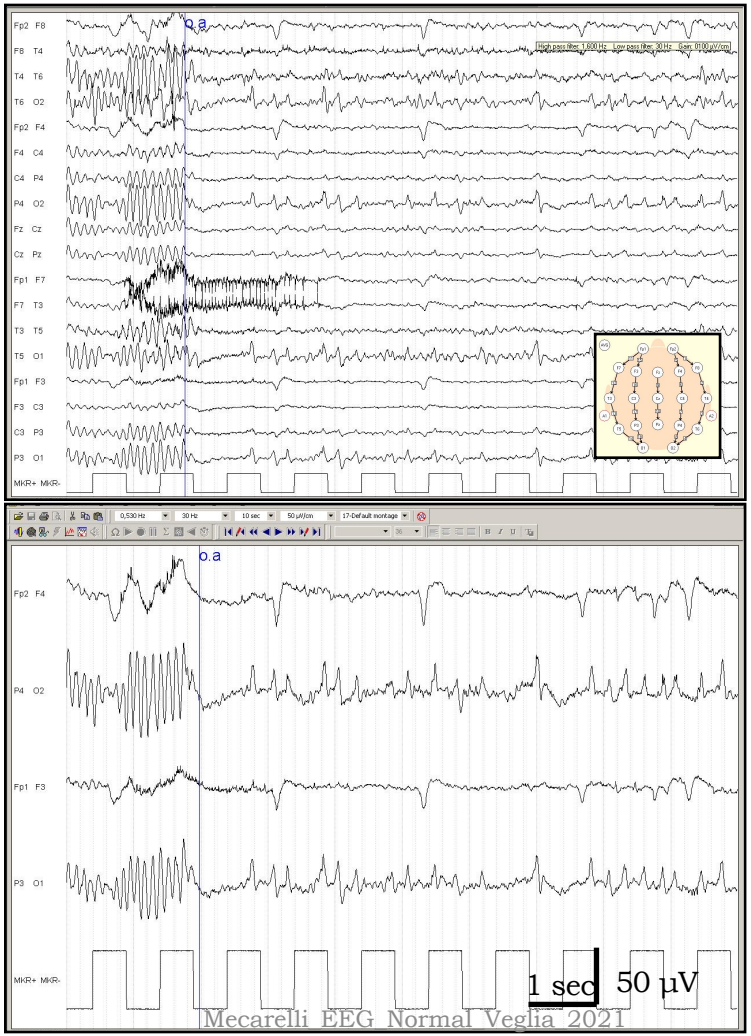


Breach rhythm on right posterior parieto-temporal region in a 48 years old patient who underwent craniotomy to remove a ganglioglioma



Onde lambda - Lambda Waves

Lambda (λ) Waves are physiological sharp waves occurring during **visual scanning in waking subjects**.
Their prevalence has been reported to vary between 2% and 88%, with the highest occurrence in children and young adults.
They appear in **occipital regions**, with minimal spread to parietal and posterior temporal areas.
The lambda waves morphology is biphasic or triphasic, and the most prominent phase is positive.
Their amplitude is usually low (20-40 uV), the duration lies between 200-300 msec and the frequency of repetition is at intervals from 200 to 500 msec.
They are mostly bilateral, synchronous and symmetrical, but they may also be unilateral or otherwise asymmetrical.



Bilateral, synchronous and symmetric lambda waves, with eyes open

Lambda waves are best elicited when the subject visually scans a textured or a complex picture, in a well-lit laboratory and are closely correlated with fast saccadic eye movements. Lambda wave appear after a saccadic movement with a latency of 70-80 msec.

The disappearance of lambda waves is obtained with closed eyes, the decrease of the ambient lighting level, or by placing a completely white sheet of paper in front of the eyes of the subject (*fixation off*). Therefore, from a neurophysiological point of view, lambda waves represent a sort of visual evoked responses and are the expression of oculomotor and visual integration.

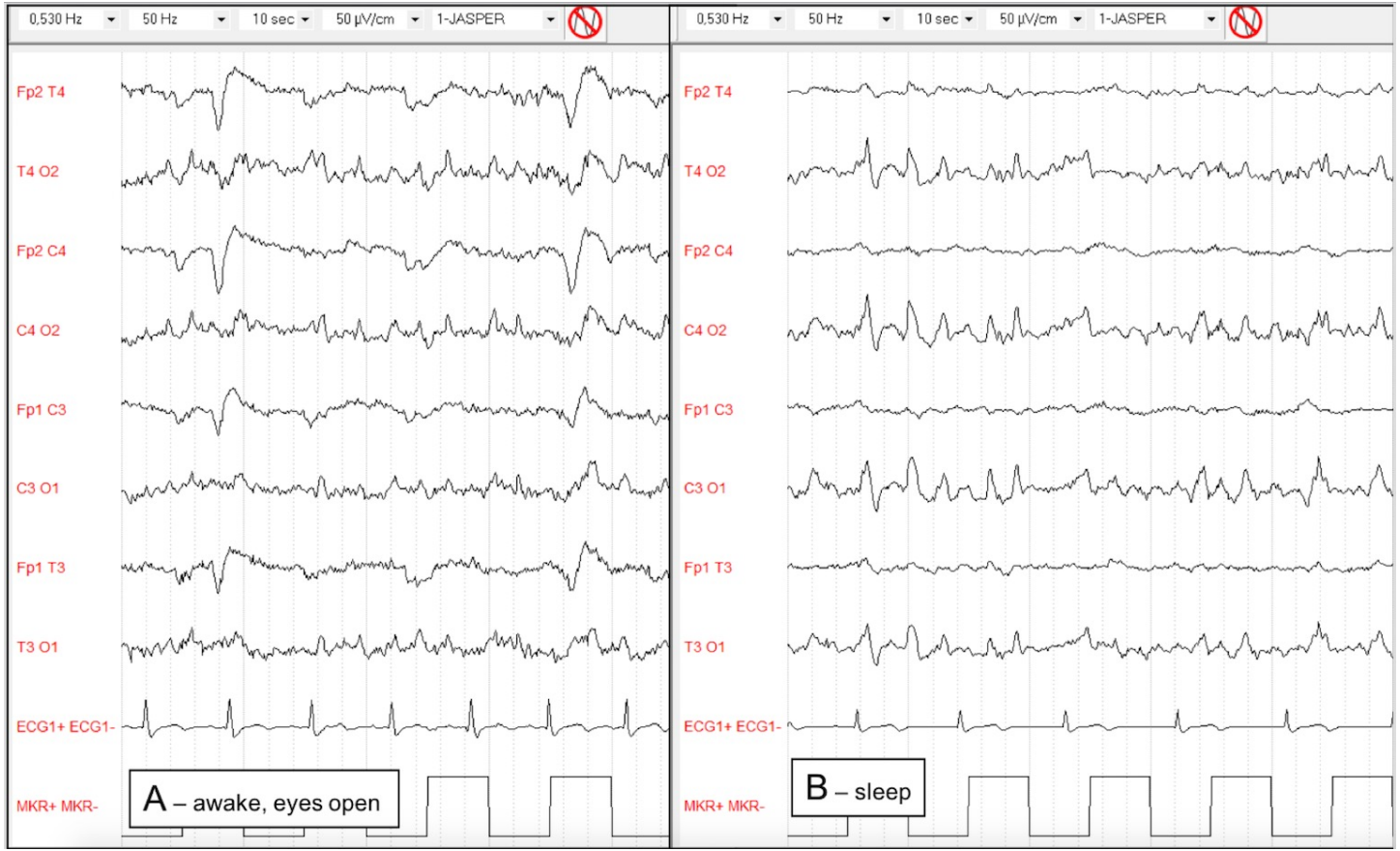
There are two different types of lambda waves. The **first type** would be generated by the faster conducting fibers of the optic nerve (Y-type fibers), immediately after the primary saccadic movement. The **second type** would instead be due to visual impulses coming through the slower optical fibers (X- fibers), with return to central vision during or shightly before the locking phase of the secondary corrective saccadic movement.

Recently Alvarez et al (2011) studied the occurrence of lambda waves in adult outpatients during prolonged EEG monitoring for unclear diagnosis of epilepsy.

Lambda waves were found in 32% of prolonged recordings, mainly during watching TV, in relation to normal EEGs. Furthermore, all recordings with lambda waves also had positive occipital sharp transients of sleep (POSTS).

This study showed that watching TV likely represent a powerful stimulus for lambda waves and confirms the physiological benign nature of this EEG pattern.

Finally, the lambda waves are of principal interest for the study of the visual system **and should not be misinterpreted as epileptiform abnormalities.**



In the same adult subject lambda wawes during awake, with eyes open (A), and POSTS during N1 stage of sleep (B).

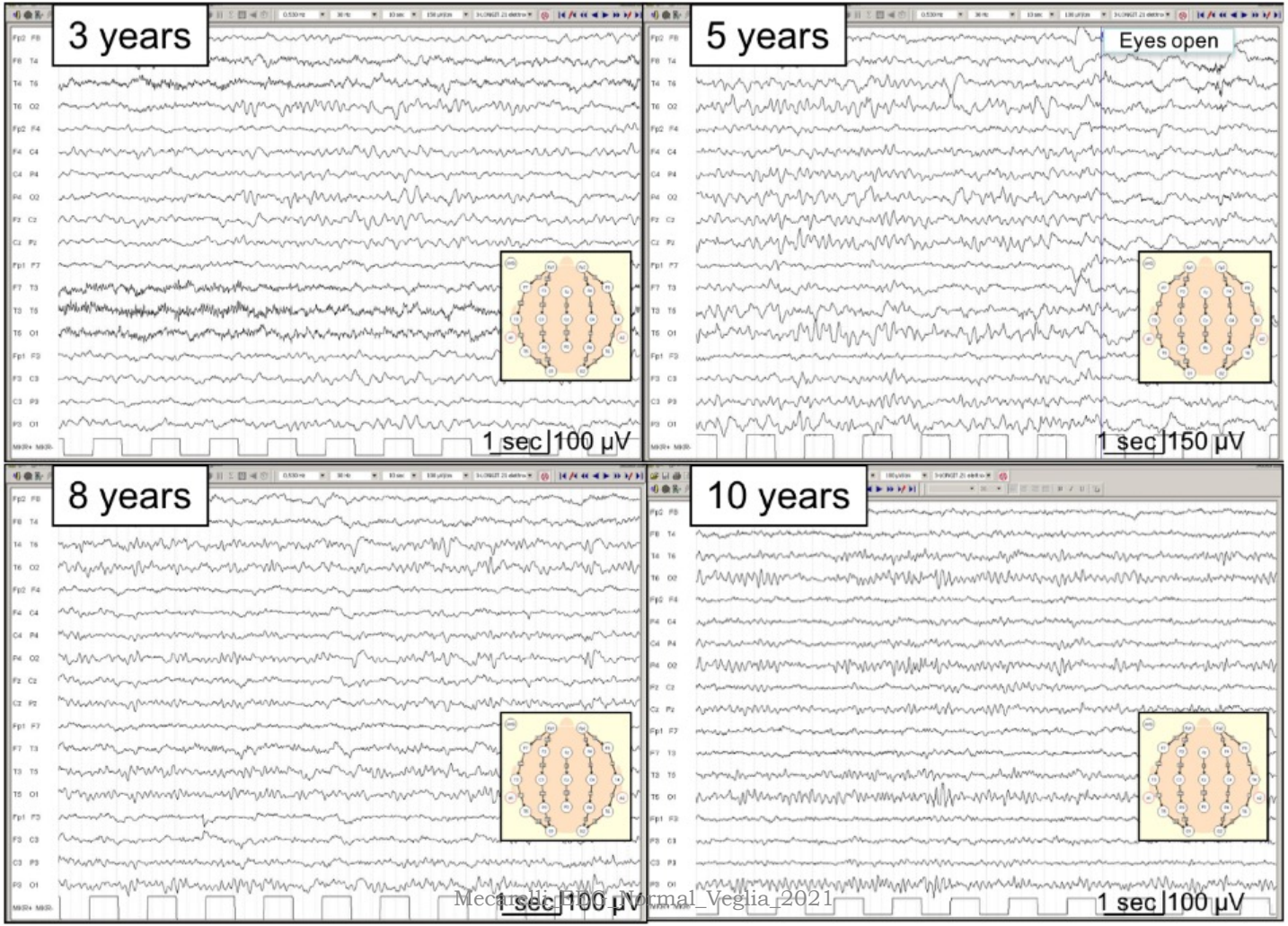
Age effect on the EEG of wakefulness

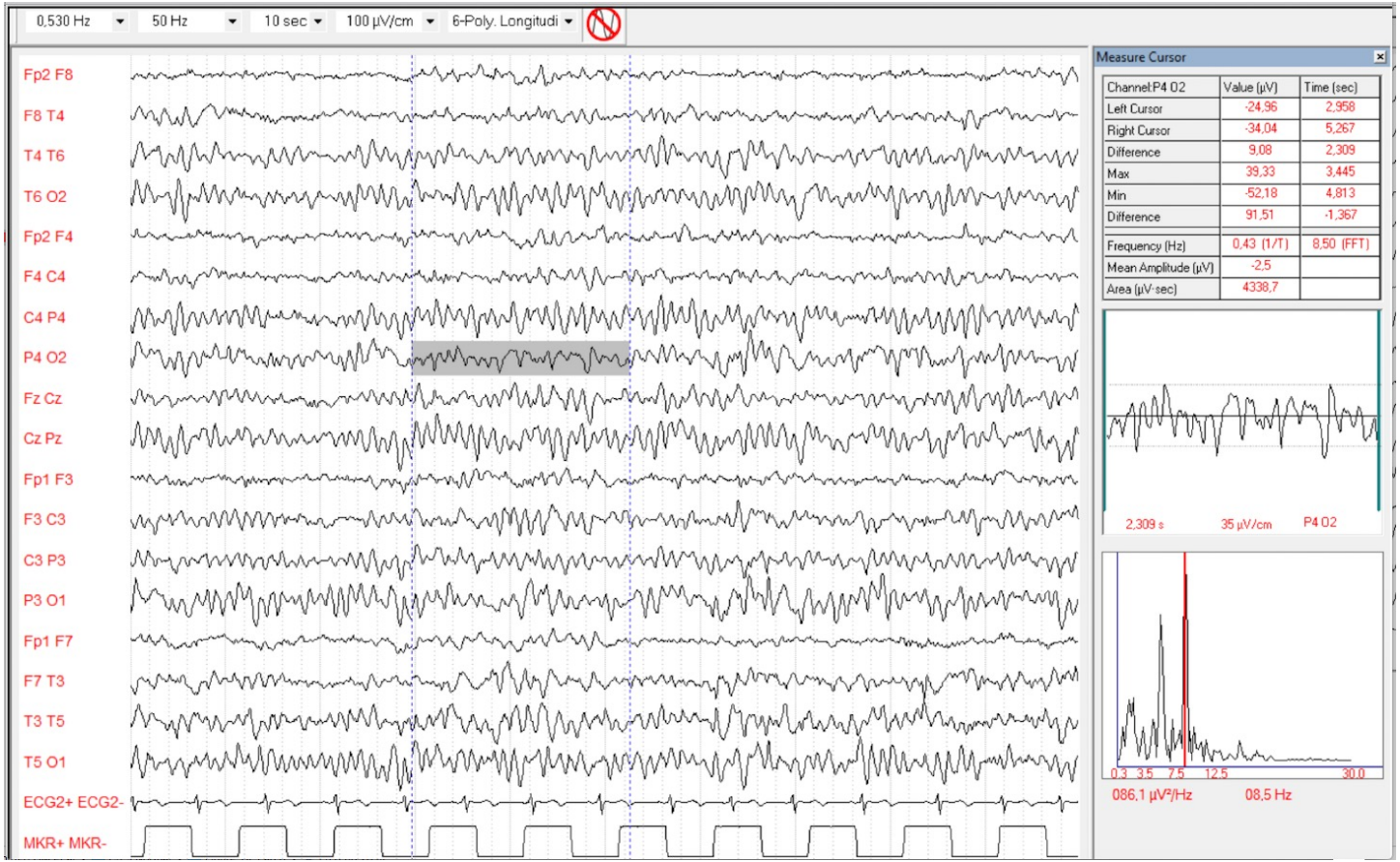
In the **first few years of life (1-3)** the awake EEG is dominated by diffuse slow delta-theta activity, with high amplitude. This slow activity gradually tends to become rhythmic and with a prevalence in the central and parietal regions.
In particular, **from 3 to 12 months** of life, the posterior background activity, considered a precursor of the subsequent alpha rhythm, has a frequency of about 4 Hz, is quite rhythmic, and partially reactive to the opening-closing of the eyes.
In the 2nd year of life this activity reaches the frequency of about 6-7 Hz and **in the 3rd** is around 7-8 Hz, with a more evident reactivity.

From the 3rd year of age, therefore, an alpha rhythm, wich we could define as *"immature"* (low frequency, partial posterior location) is already recognisable. The mid-posterior background activity in these epochs is of high amplitude and mixed with slower rhythms at 2-5 Hz.

From 3 to 5 years of age the posterior rhythm is maintained as a frequency around 8 Hz, interrupted at intervals by sequences at 1.5-4 Hz, and the tracing shows abundant theta activity in anterior regions and / or diffuse, at 5-7 Hz. Hyperventilation (HV, activation procedure that you can get in laboratory with a good cooperation already from 3-4 years) shows mostly a widespread, hypersynchronous, wide-voltage slow activity, and a photic driving response particularly at low frequencies is observed during the Intermittent Photic Stimulation (IPS).

F**rom 6 to 12-15 years**, the alpha rhythm progressively reaches the frequency of about 10 Hz, although it is always of high amplitude and mixed with slower theta activity (**"*posterior slow waves of youth*"**); sometimes this theta activity, confined in temporal posterior regions, is rhythmic and at subharmonic alpha frequency (slow alpha variant)


3 years
1 sec 100 µV
5 years
Eyes open
1 sec 150 µV
8 years
1 sec 100 µV
10 years
1 sec 100 µV



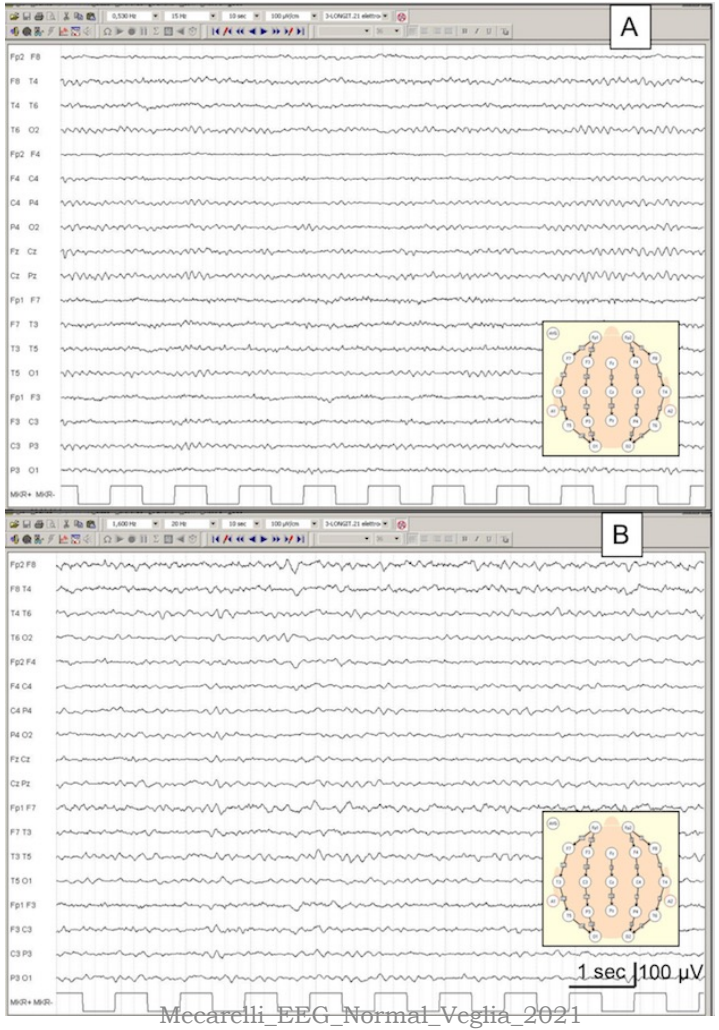
Posterior Slow Waves of Youth

Mecarelli_EEG_Normal_Veglia_2021

From 12-15 years onwards the EEG acquires progressively all the characteristics of the adult, both in basal and during activation tests, with attainment of a final mature pattern around 18-20 years.
In the EEG maturative process, however, the interindividual variability must always be taken into great consideration, since it has a decisive influence on the age of achievement of what can be considered a mature waking EEG pattern.
From about 20 years the physiological awake EEG maintains stable characteristics.
From the age of 50 onwards instead, a slight increase in theta activity and/or a slight decline in alpha frequency may begin to appear, although consistent with a normal EEG for the age.
According to some authors the alpha rhythm slows down by 0.08 Hz per year from the age of 60, but this fact has not be confirmed by others. A more recent review by Rossini et al describe resting EEG changes across physiological aging, with gradual amplitude decrease of alpha rhythm and a global slowing of the background activity.

However, an alpha rhythm at a frequency of 7.5-8 Hz in subjects over 80 years can be considered completely normal. Furthermore, in the elderly the posterior physiological rhythm is of lower amplitude and tends to spread to anterior regions .
Often the slow alpha variant are also found in posterior regions.

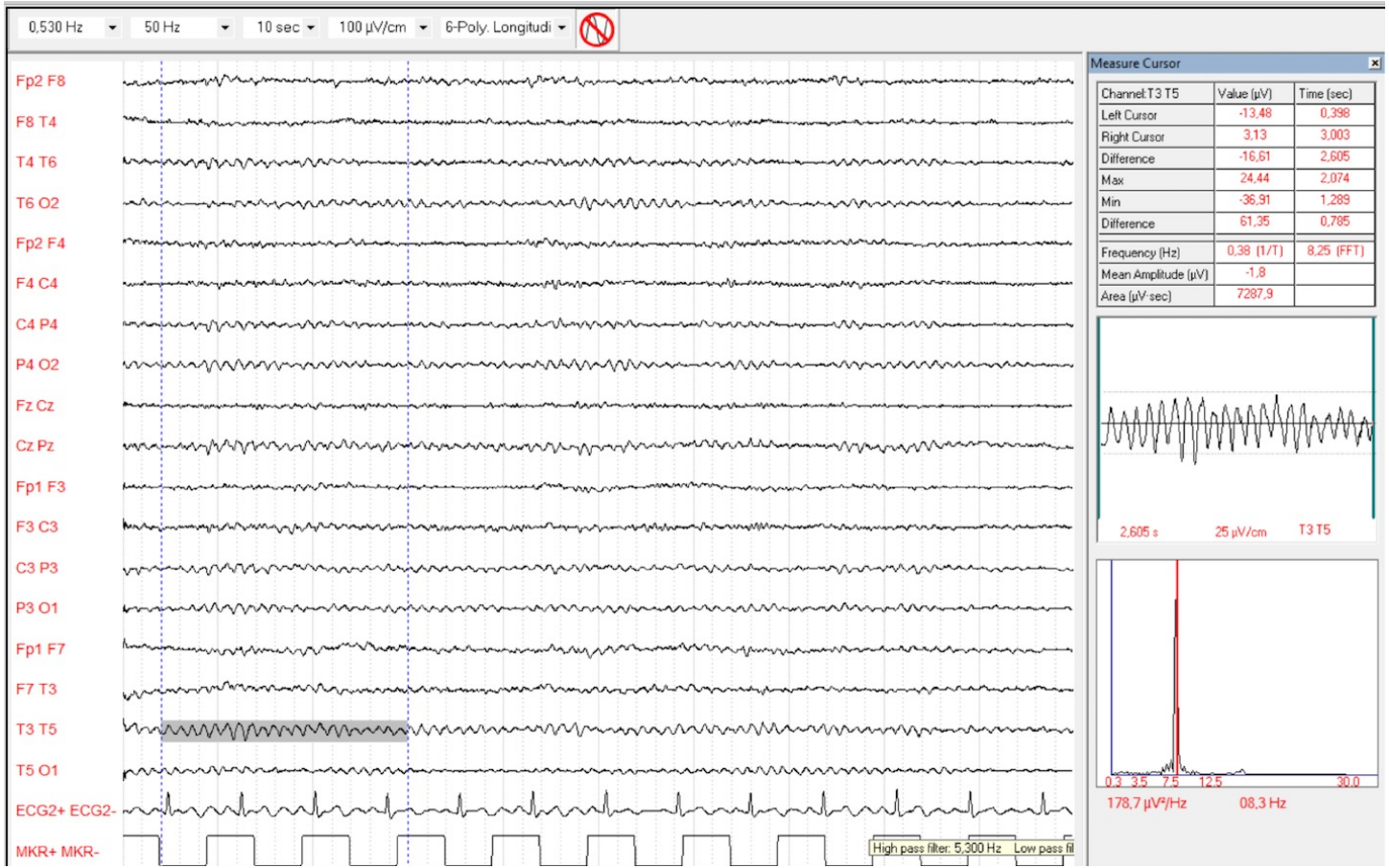
Examples of background activity with eyes closed in two old subjects (A, 83 y.o. and B, 95 y.o.).



83 yrs

95 yrs

Poorly localized alpha activity with low amplitude and peak of frequency of 8.3 Hz in a 85 y.o. subject



Prove di Attivazione EEG

Text modified from: Mecarelli O. Activation Procedures.
In: Clinical Electroencephalography. Springer Nature, 2019 (in press)

Hyperventilation (HV)

Hyperventilation (HV) - or overbreathing (synonym) - is the EEG activation procedure described firstly and always used in clinical EEG laboratories; it is recommended as part of the standard EEG recording by the most prestigious international guidelines. The procedure is easy to perform, very safe and in most cases it is able to induce the appearance of interictal or ictal epileptiform discharges.

This method consists of deep and regular breathing, performed for a minimum of 3 minutes at a rate of about 18-20 breaths per minute, with prolonged recording for at least 1-2 minute after cessation of overbreathing.

In special cases it is necessary to continue the recording even for longer periods after stopping the HV. A recent large-scale study showed that HV prolonged for 5 min increased the diagnostic yield of HV (16% of seizures and 30% of interictal EEG abnormalities triggered by HV occurred after 3 min of HV) and that 99% of patients who are able to hyperventilate for 3 min can complete a 5 min HV, without additional adverse events.

Tabella 1. Iperventilazione. Sintesi metodica e pattern EEG attivati

Paziente	Posizione seduta o sdraiata, in ambiente tranquillo
Modalità esecuzione	Inspirazioni profonde e rapide, seguite da espirazioni prolungate, a bocca semiaperta (18-20 atti respiratori/min)
Durata prova	3-5 minuti
Modificazioni EEG fisiologiche	Rallentamento ritmo alfa Rallentamenti diffusi (> posteriormente nei bambini e > anteriormente nei giovani) – di entità variabile (a seconda dell'età, del soggetto ecc.) – con normalizzazione dell'EEG entro 5-60 s
Modificazioni EEG patologiche	Rallentamenti focali e/o diffusi non giustificati per l'età e di notevole entità Asimmetrie importanti Normalizzazione del tracciato non pronta Anomalie di tipo epilettico: > generalizzate (> scariche di complessi punta-onda a 3 Hz), ma anche focali, con o senza correlato clinico

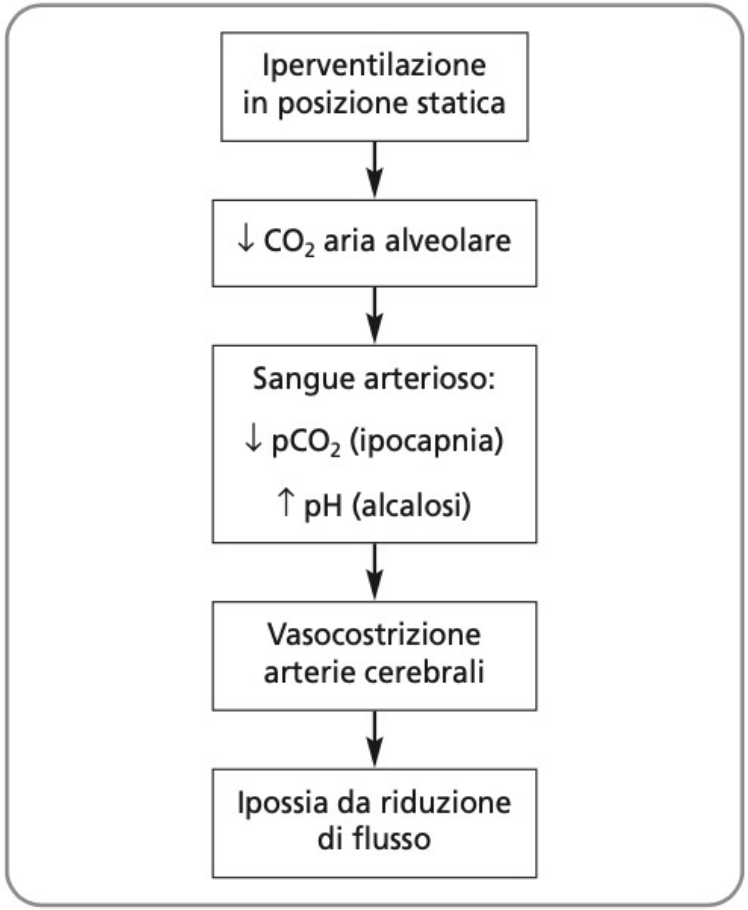


Figura 5. Sintesi delle modificazioni emogasanalitiche ed emodinamiche indotte dall'iperventilazione.

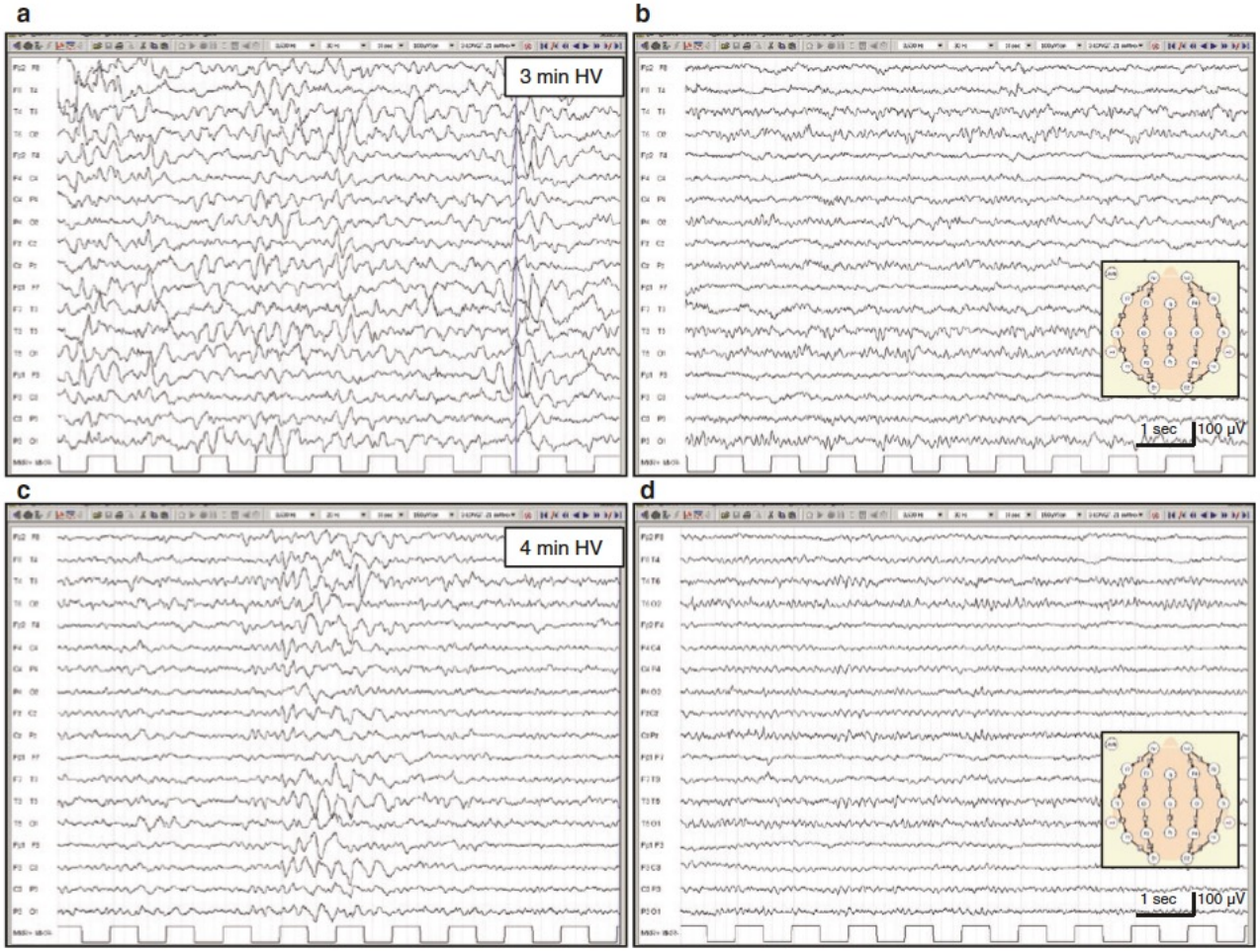


Fig. 14.1 Physiological EEG changes induced by HyperVentilation (HV). Sequences of delta slowings, dominant posteriorly, after 3-min HV, in a 7-year-old child (**a**); basal reverting of EEG recording 30 s after HV end (**b**). Burst of delta waves dominant anteriorly after 4 min of HV in an 18-year-old subject (**c**), with EEG return to basal condition after 20-s HV end (**d**)

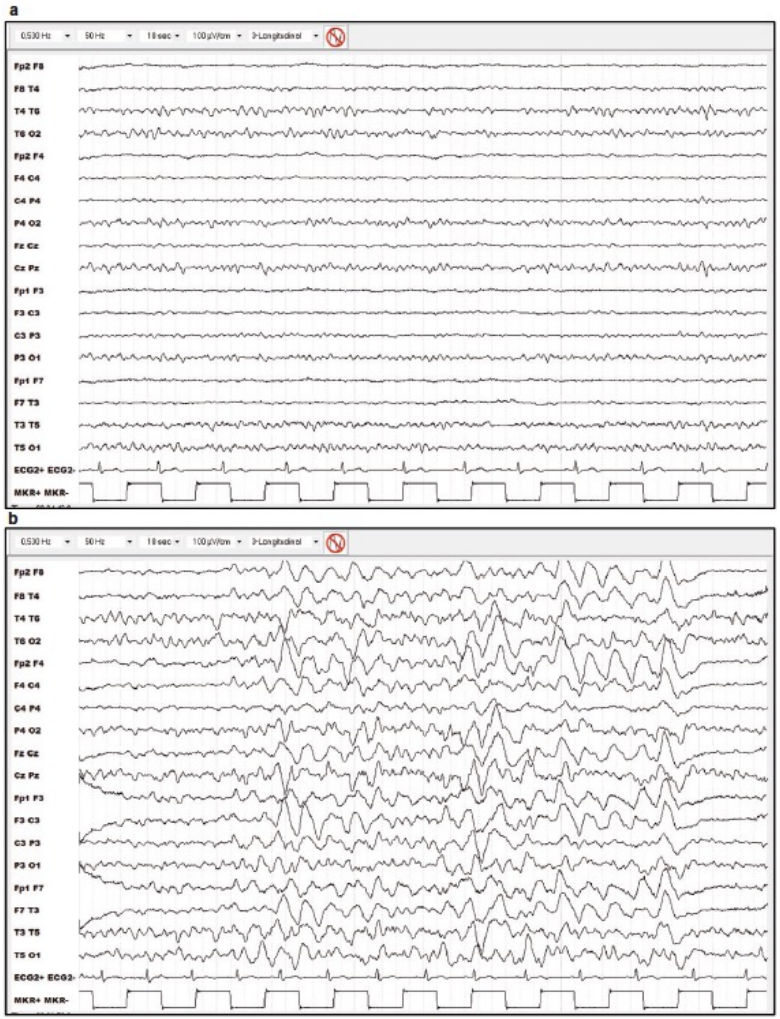


Fig. 14.3 Normal basal EEG (**a**) and slow diffuse hypersynchrony after 3-min hyperventilation (**b**) in an 18-year-old subject with recurrent neurally mediated syncopes

HIHARSs:
Hyperventilation
Induced
High
Amplitude
Rhythmic
Slowings

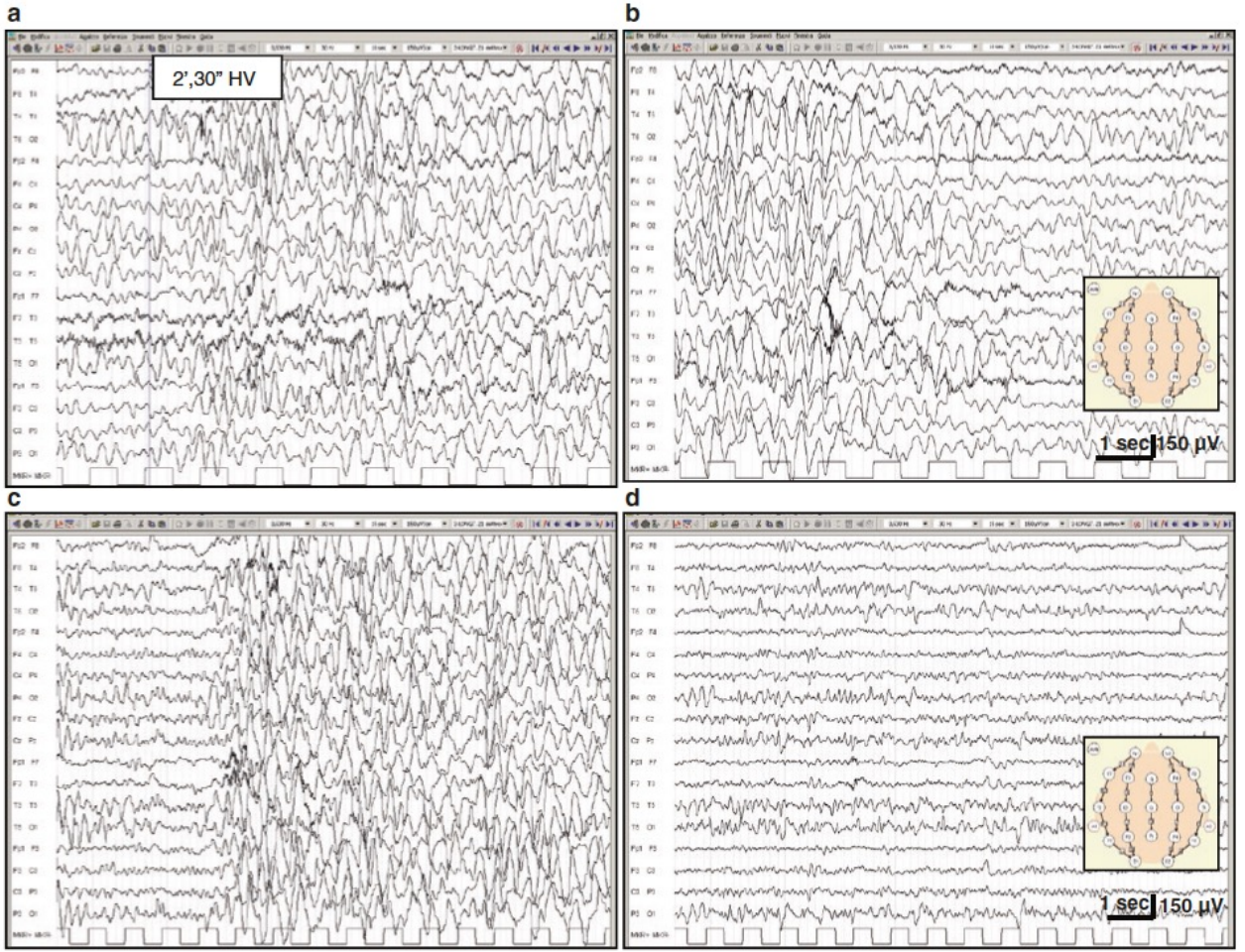
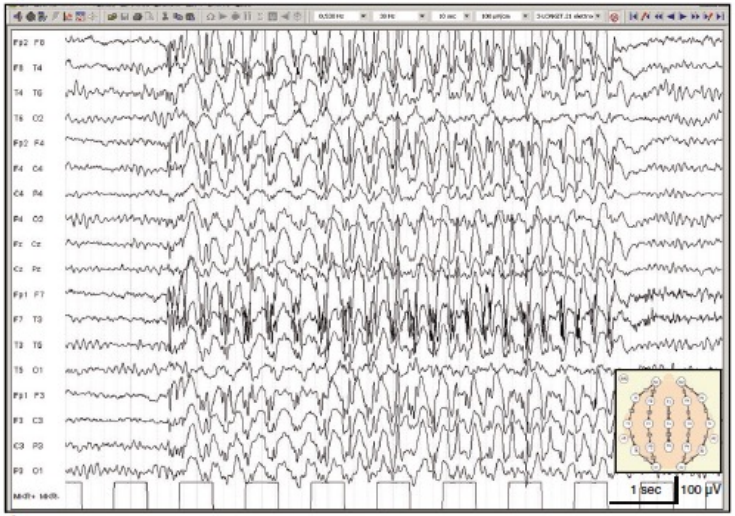
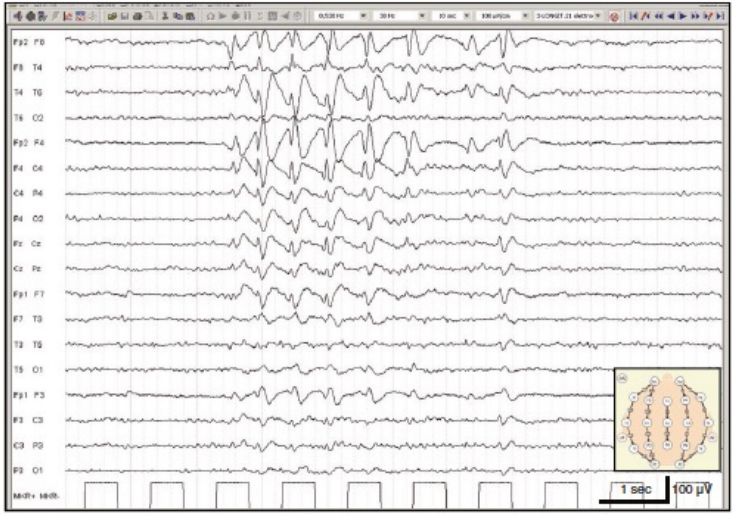


Fig. 14.2 Two examples of hyperventilation-induced high-amplitude rhythmic slowing, in a 6-year-old (**a**, **b**) and 7-year-old child, respectively (**c**, **d**). In (**b**) a gradual reappearance of normal background activity 40 s after the HV stop is observed, while in **d** the EEG normalisation is earlier, 30 s after the end of the activation procedure

a



b



2. Intermittent Photic Stimulation

Intermittent Photic Stimulation (IPS) must be carried out before Hyperventilation or at least 3 minutes after its conclusion.

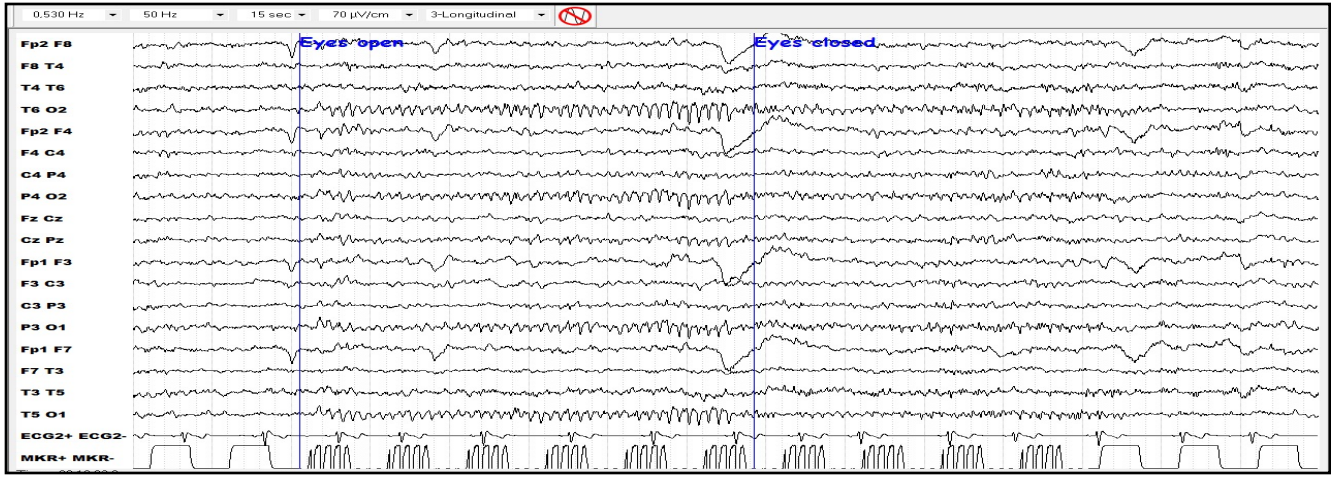
In fact, biochemical variations and eventual EEG modifications induced by hyperventilation can be prolonged for a few minutes after its interruption; because of this, performing this test before IPS can be confusing.

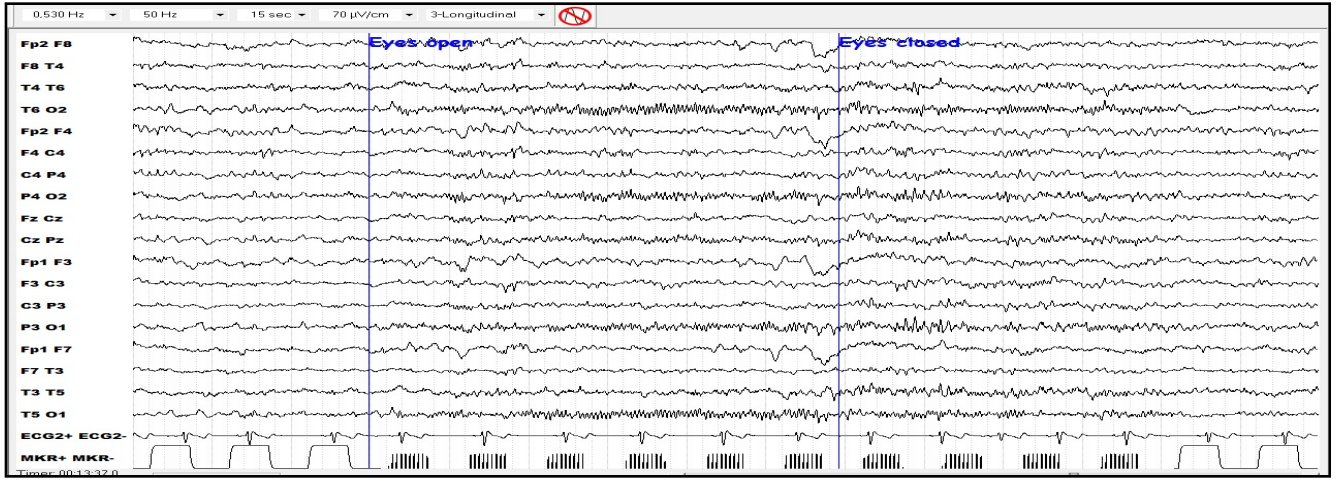
Table 2. Methodology for Intermittent Photic Stimulation (IPS)

IPS	Methods
Environment	Dim room lighting
Patient	sitting position, head slightly reclined fixing the center of the lamp
Photic Stimulator	Circular lamp (13 cm diameter) Intensity of flash: at least 0.7 Joule Viewing distance: 30 cm; angle: 13 degrees
Standard IPS procedure	Flash frequencies: 1-2-8-10-15-18-20-25-40-50-60 Hz eye conditions: eye closure - eyes closed - eyes open (5 sec IPS and 5 sec rest times, for each eye condition)
Appearance of PR	lower threshold: appearance of PPR at a certain frequency; in this case: skip the remainder of the series, start again with 60 Hz and go down (60-50-40-25..), until again PPR occurs (upper threshold)

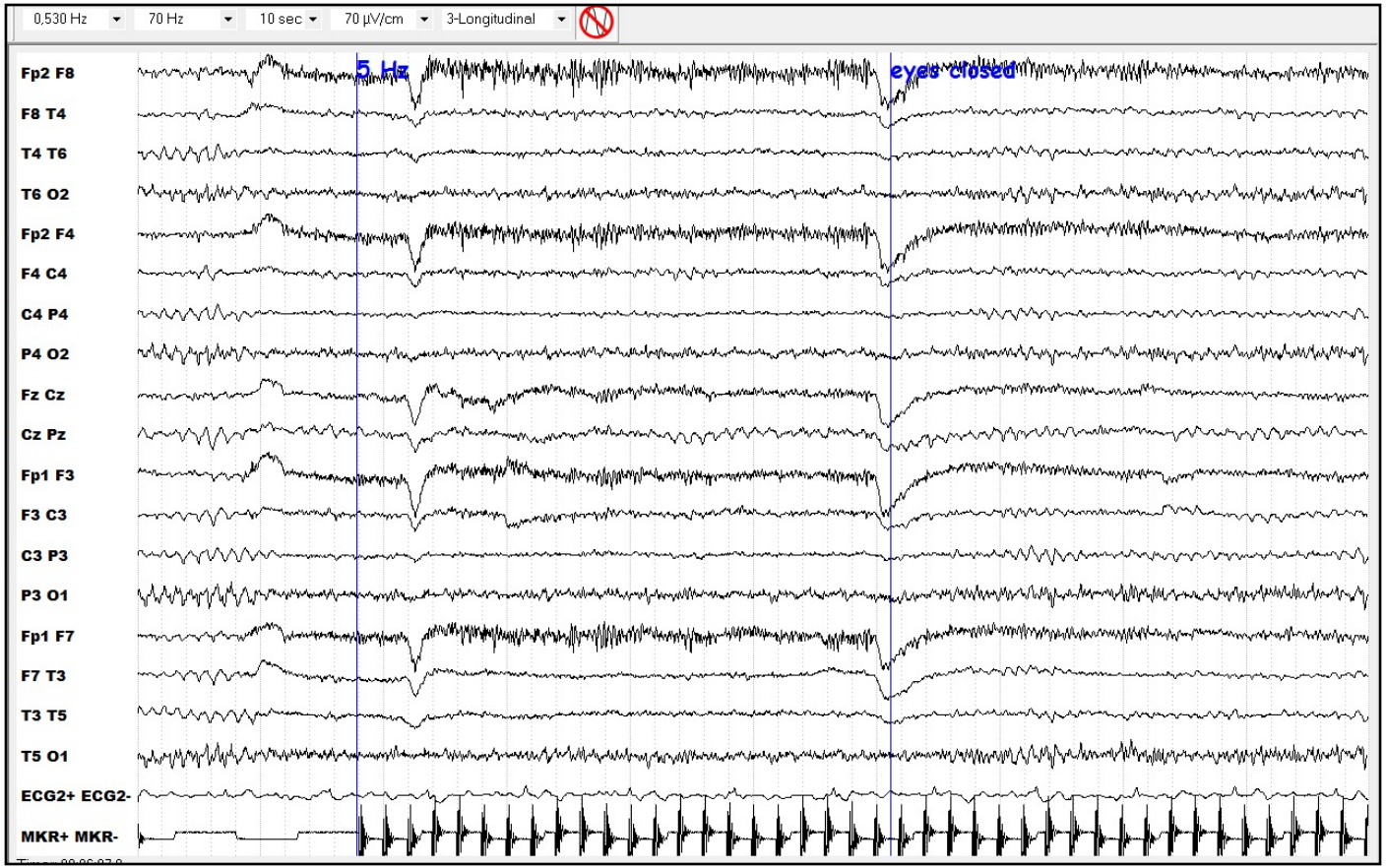
Table 3. EEG patterns induced by IPS

Photic Driving Response (PDR)	- time-locked to light stimulus - same frequency or harmonically related to stimuli
Photomyogenic Response (PMR)	Repetitive muscle spikes over anterior regions (electromyographic response)
Photoparoxysmal Response (PPR) *different types (Waltz criteria)*	Epileptiform Abnormalities induced by IPS 1. focal occipital spikes time-locked to light stimuli 2. parieto-occipital spikes and slow waves 3. parieto-occipital spikes and slow waves, spreading to frontal regions 4. Generalized spike- or polyspike-and-slow wave complexes - self-limited - prolonged/self-sustaining

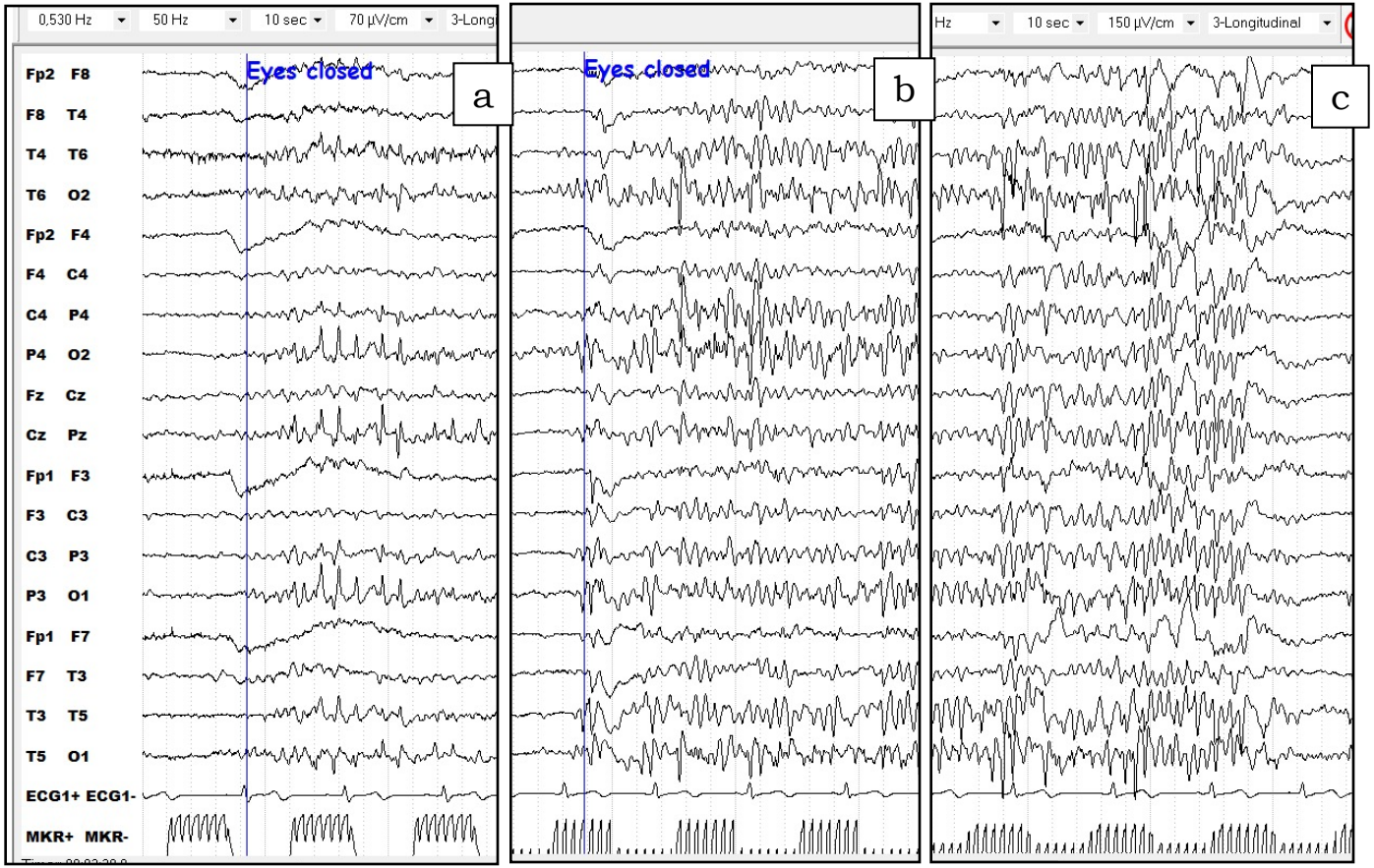




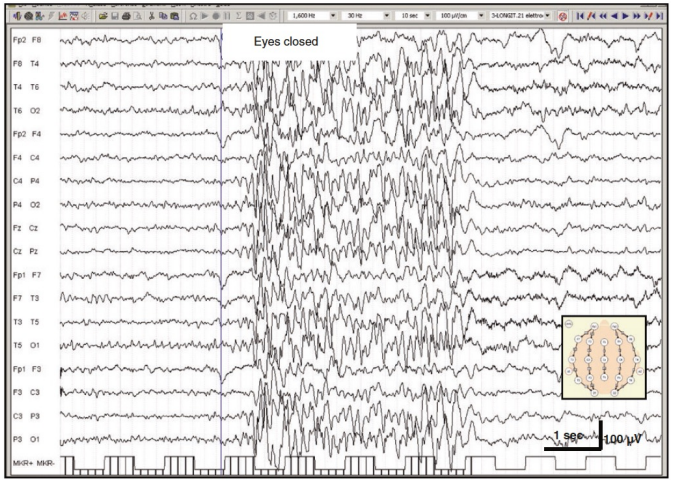
Occipital photic driving response (PDR) in a 19 yrs old subject, at 8 Hz (upper) and 18 Hz (bottom).



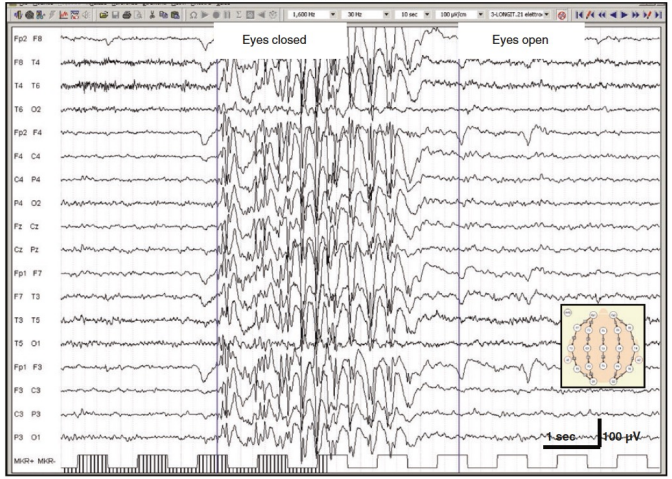
Frontally photomyogenic response in a 35 yrs old subject.



Photoparoxysmal response (PPR) of type 1 (a), type 2 (b) and type 3 (c), according to Waltz criteria



self-limited

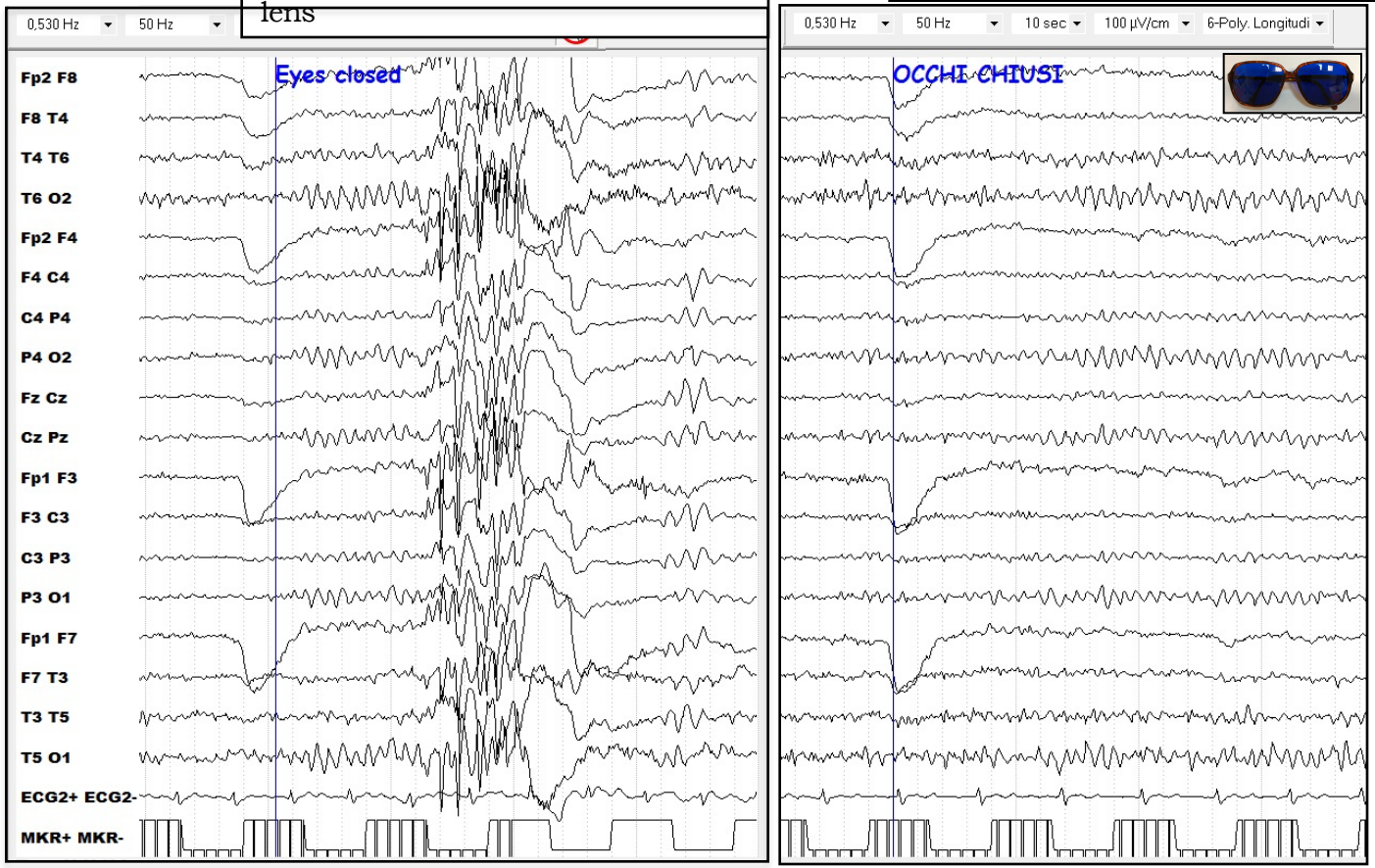


self-sustaining

Photoparoxysmal response (PPR) type 4

Eyes closed - IPS 10 Hz without lens

Eyes closed - IPS 10 Hz with lens

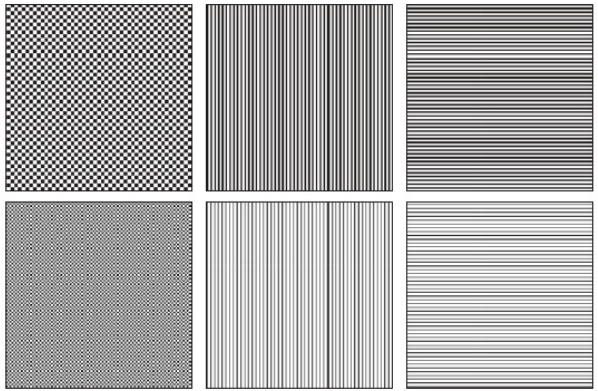


PPR with IPS at 10 Hz, suppressed by Zeiss-Claret 1.5 KF 133 lens

Other methods of visual stimulation: Pattern stimulation

A remarkable number of photosensitive patients are sensitive not only to IPS, but also to geometric patterns. The incidence of paroxysmal discharges induced by patterns varies widely across studies (from 5% to 72%) depending on the spatial and temporal characteristics of visual pattern and the stimulation protocol. However, there have been reported also patients responding to pattern, but not to IPS. The most activating geometric pattern consists of parallel lines or stripes with sharp edges, black-and-white and high-contrasted.

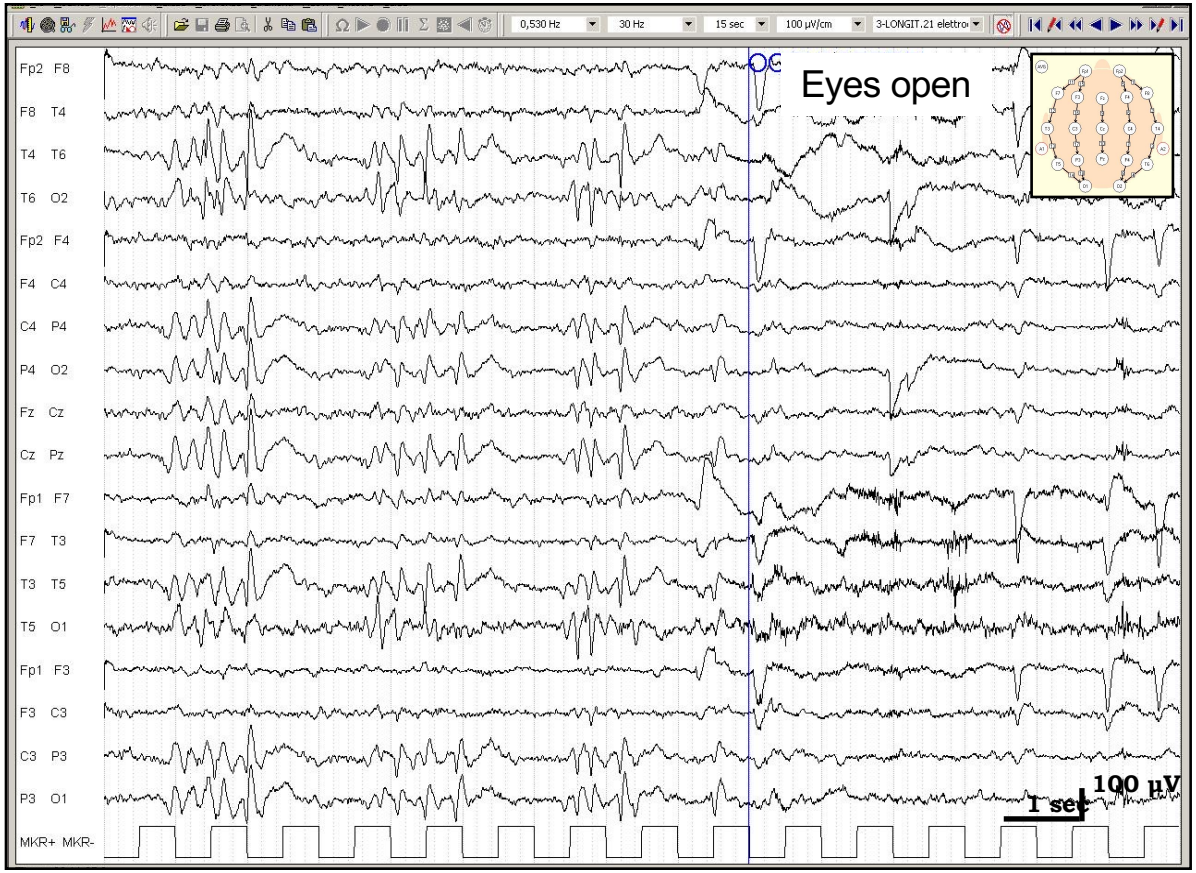
The stimulation can be performed with eyes open, in ambient lighting with a monitor connected to an electronic grating generator, positioned 1 meter from the patient's eyes. Each pattern is presented for 10 s, in oscillatory (optimal oscillation frequency: 15-20 Hz) or stationary modality, at spatial frequencies between 0.5 and 6 cycles/degree. Pattern sensitivity occurs in various epileptic syndromes, but it is a readily distinguishable subtype of the visually provoked reflex epilepsies.



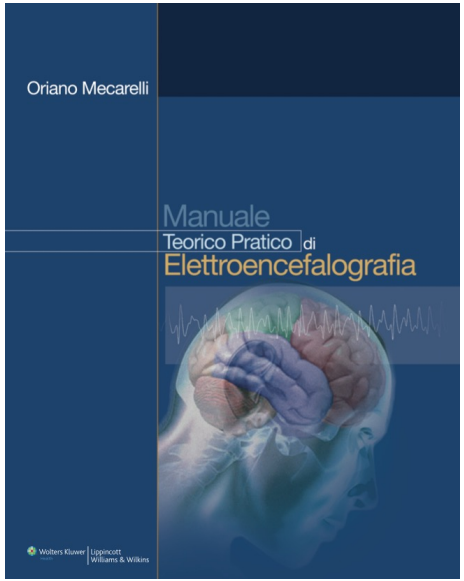
Fixation-off sensitivity

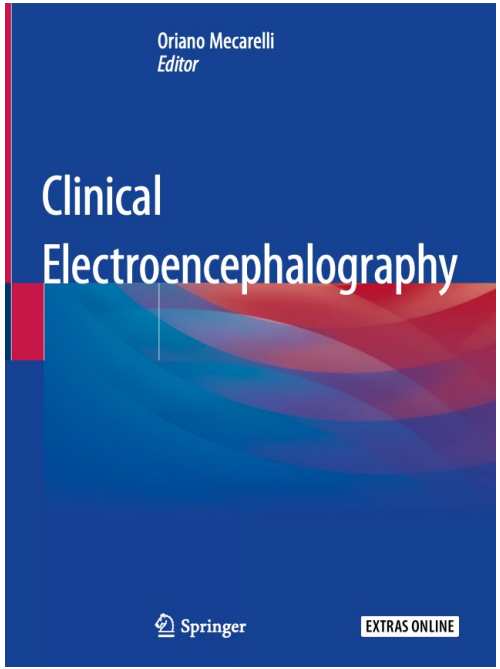
Fixation-off sensitivity (FOS) is a rare phenomenon induced by elimination of central vision/fixation.
FOS is characterized by continuous posterior or diffuse epileptiform discharges that appear 1-3 sec after eye closure, persist throughout the recording at eye-closed and disappear immediately with eye opening.
A definite diagnosis of FOS requires a demonstration that the epileptiform abnormalities also occur by impending central vision and fixation using Frenzel lenses or by placing a sheet of white paper 20 cm in front of the subject (at eyes open, but without fixation).

The epileptiform abnormalities (spike, spike and wave, sharp wave) can be registered in patients without clinical epileptic seizures, but most commonly FOS is associated with occipital idiopathic childhood epilepsies or with focal or generalised symptomatic epilepsies.
Based on the knowledge acquired so far, FOS seems to be the expression of the occipital hyperexcitability, but the exact mechanisms underlying this phenomenon remains somewhat obscure.



Fixation-off sensitivity (FOS) phenomenon in a 26 yrs old patient with focal epilepsy. With eyes closed, subcontinuos epileptiform abnormalities in the posterior regions of both hemispheres, without clinical correlates. The epileptiform activity immediately disappears with eyes opening.





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- Activation Procedures (Ch 14)

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