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Clinical neurophysiological studies in migraine were initially aimed at supporting diagnosis and then, more importantly, at the characterization of the migrainous brain.

There are some recent excellent reviews on these topics [1–3], and here we will summarize and update these reports. We will consider three aspects: neurophysiological testing in migraine diagnosis, neurophysiological features of the migrainous brain and, finally, neurophysiological testing in vestibular migraine.

2.1 Migraine Diagnosis

Recently, the European Federation of Neurological Societies (EFNS) published the guidelines for neurophysiological tests and neuroimaging procedures in non-acute headache [2]. For headache in general, and more specifically for migraine, there are little evidences that neurophysiological testing is a useful support for the diagnosis.

Table 2.1 summarizes the EFNS guidelines for neurophysiological tests, but also for neuroimaging procedure, in non-acute headache [2]. It is noteworthy that the recommendation did not change from the previous guidelines published in 2004, but some of them were supported by an increased level of recommendation.

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Table 2.1 Summary of EFNS guidelines [2]

Method	Routine evaluation	Level of recommendation [4]
EEG	Recommended for basilar/hemiplegic migraine and epilepsy-related migraine	IIB
Evoked potentials	Not recommended	IIB
Reflex responses	Not recommended	IV/IIIC
Autonomic tests	Not recommended	IIIC
Clinical tenderness test and surface EMG	Not recommended for diagnosis; manual palpation useful for classification	IIB
Neuroimaging	MRI recommended in patients with trigeminal autonomic cephalalgias, atypical headache, seizures or focal signs	

2.1.1 Electroencephalography (EEG)

During the attack the background rhythm may [5, 6] or may not [7] be slowed. Some migraineurs show the “H-response”, namely, an increased response to photic stimulation; however, a similar finding was reported in normal subjects [8]. The EEG still plays a valuable role when migraine shows epileptic features: unusually brief headache episodes, auras or aura-like phenomena, unusual aura symptoms and headache associated with severe neurological deficits.

2.1.2 Visual Evoked Potentials (VEPs)

The pattern reversal VEP more often proved to be normal, although amplitude could be either increased, between or close to attacks [9, 10], or decreased [11]. Increased VEP amplitudes have been reported with high contrast and spatial frequency stimuli, suggesting an impairment of the magnocellular system [12, 13]. VEP findings are very similar in migraine without and with aura [14].

2.1.2.1 Brainstem Auditory Evoked Potentials (BAEPs)

In contrast with the VEPs, the BAEPs do not originate from the cerebral cortex but from the inner ear, the acoustic nerve and from generators located within the brainstem. These same structures will be considered with more detail when we will focus on vestibular migraine. More frequently BAEPs proved to be normal [14], but a latency delay was reported in some papers [15].

2.1.2.2 Somatosensory Evoked Potentials (SEPs)

The standard SEPs obtained by electric stimulation showed very little abnormalities, but we can mention the cortical N19 latency delay and amplitude reduction reported in migraine with aura [16].

The CO₂ laser SEPs are obtained by selective stimulation of nociceptive A-delta and C fibres, and their amplitude was increased during a spontaneous [17, 18] or nitroglycerine-induced attack [19], and this increase was relieved by symptomatic treatment [20].

2.1.3 Habituation and Sensitization

Habituation consists in a reduction of a response when repeating a constant intensity stimulus. The response depends not only on the stimulus features but also on the “tonic” and “motivational” level within several structures including the monoaminergic nuclei of the brainstem [21]. Habituation is best evaluated by averaging blocks of responses (for instance, by splitting the responses in quartile depending on their trial number) rather than by analysing the single trials.

A reduced habituation occurs for many stimulation modalities: visual [12, 22–29], auditory [30, 31] and somatosensory [32, 33]. The initial response is usually lower than in normal subjects, and it is unlikely to be a factor to explain the lack of habituation as it could be for an increased initial response. Moreover, the “reduced” initial response suggests a lower pre-activation level and argues against sensitization (see also below). Interestingly, the excitatory 10 Hz repetitive transcranial magnetic stimulation over the visual cortex is able to increase the amplitude of the first block and to normalize habituation of VEP [34].

The habituation is reduced in the interictal phase, but normalizes during the attack. The thalamocortical cholinergic drive, and the cortical pre-activation level [35], plays a role in habituation, as pointed out by early high-frequency oscillations (HFO) that are reduced interictally, but normal during the attack [36].

For visual evoked potentials, habituation is related to the degree of short-range lateral inhibition, the behaviour of which is different in the interictal and ictal phases. In the interictal phase short-range latency inhibition is more than normal at the beginning and then becomes less than normal along the stimulation session; the opposite behaviour is observed during the attack.

Finally, a lack of habituation does occur also for nociceptive stimulation as detectable by blink reflex [37–40] or by laser SEP [17, 41–44].

Peripheral and central sensitization mechanisms are able to explain some features of the migraine attack, and these correspond both to a reduced nociceptive threshold and to an increased nociceptive response both to noxious and non-noxious (for instance, tactile or mechanical) stimuli. If we consider sensitization as the counterpart of habituation [1], it should consist in an increased response or in an increased excitability, and in terms of modifications observed with increasing ordinal number of block of response as for habituation, sensitization should correspond to a larger than normal response in the first block.

Considering sensitization there are several aspects that should be considered, and a distinction should be made depending on stimulus features and on the interictal rather than the attack phase.

The reversal from lack of to normal habituation that we mentioned about the attack phase might be considered as a sensitization phenomenon [27, 45, 46].

Sensitization, in association with normal habituation, is detectable by SEP during the attack in episodic migraineurs in medication overuse headache [47].

If we consider noxious stimuli, migraine patients show a reduced pain threshold before and during the attack [48, 49], and this is related to attack frequency [48] and worsens in chronic migraine and in medication overuse headache [44, 50]. During the attack, the R2 component of the blink reflex [51] and the amplitude of the laser SEP [48] are increased in the affected as compared to the unaffected side, and laser SEP does not habituate during the attack [48], which is at variance with the behaviour observed with other, non-noxious, stimulation modality.

In medication overuse headache pain-related evoked potentials proved to be abnormally large after both cranial and extracranial stimulations, and this abnormality is no longer detectable after the discontinuation of medication overuse [52].

Transcranial magnetic stimulation (TMS) is another tool for the neurophysiological evaluation of the migrainous brain. The increased excitability of the visual cortices, usually expressed as a reduced threshold required to generate phosphenes, has been demonstrated in several [53–67], but not all [34, 68], studies. The literature about the motor cortex is small and reports both an increased [61] and a decreased [53, 60] excitability. TMS has been used both to treat [69–76] and to evaluate treatment efficacy in migraine [56, 67, 77–79].

2.2 Vestibular Migraine

For many years, the comorbidity of vertigo and migraine is regarded as not only a co-occurrence by chance [80] and codified by classification criteria [81, 82]. Very recently, Lempert et al. [83] updated the diagnostic criteria for vestibular migraine (VM, Table 2.2), and these criteria have been included in the ICHD3-beta [84].

VM is likely to be the third cause of migraine and to have a life prevalence in the general population of about 1 % [81].

Besides VM, the idea that subjects with migraine may have a vestibular dysfunction is suggested by the findings that about 50 % of them suffer from motion

Table 2.2 Diagnostic criteria for vestibular migraine [83]

A.	At least five episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h
B.	Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD)
C.	One or more migraine features with at least 50 % of the vestibular episodes: Headache with at least two of the following characteristics: one-sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity Photophobia and phonophobia Visual aura
D.	Not explained by another vestibular disorder

sickness and in those with VM motion sickness can be relieved by triptans [85]. The motion perception threshold is reduced in VM compared to normal and migraine subjects in the dynamic roll tilt paradigm, that is, in keeping with the idea that patients with VM may have enhanced perceptual sensitivity for head motions that dynamically modulate canal and otolith inputs together [86].

In the interictal phase, the occurrence of vestibular clinical and instrumental signs can be as high as 83 % [82, 87], and the occurrence of these signs can be the same [88, 89] or higher [90] in VM than in patients with migraine but not VM.

The vestibular and ocular motor abnormalities are in keeping with a dysfunction involving the peripheral and/or the central nervous system [80]. The vestibular dysfunction affects both the canal system, as detectable by caloric or rotatory testing [88–93], and the otolith system, as detectable by cervical or ocular vestibular evoked potentials (c- or o-VEMP) [94, 95] and by the evaluation of the subjective visual vertical that proved to be either normal [96] or abnormal [97]. Also the vestibulospinal [93] and the auditory [15] systems can be affected in VM.

Not surprisingly c-VEMPs are abnormal in basilar artery migraine [98] (currently migraine with brainstem aura) that we can be considered as a particular kind of VM. Finally, c-VEMPs by using 0.5 and 1 kHz tone bursts [99] can be used to differentiate VM from Ménière disease.

When VM patients are followed up for a time period of about 10 years, the number of subjects presenting interictal vestibular signs increases from 16 to 41 % [100] or from 20 to 63 % [101], and this progression seems to be prevented by prophylactic migraine treatment [101].

There are two papers on the evaluation of patients during an acute phase of their VM. In one paper the major finding was that all the patients, during but not outside the spell, showed a positional nystagmus [102]. In the other paper, the most interesting finding was that the attack-related signs suggested a central or a peripheral vestibular dysfunction in 50 and 15 % of the subjects, respectively, but in 35 % of them the location of the dysfunction could not be determined with certainty [103].

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