Approccio diagnostico e terapeutico alle epilessie morfiche: a consensus of the International Sleep & Epilepsy Task Force

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Sleep, sleep deprivation and sleep disorders (frequent in patients with epilepsy) may affect epilepsy, favouring seizure occurrence; in turn seizures during sleep, drugs and interictal epileptic activity may fragment sleep interfering with its restorative functions.

Some epilepsy syndromes (Sleep Related Epilepsies-SRE) are strongly or exclusively associated with sleep.

There are no standardized criteria regarding the procedures to be adopted for the diagnosis (and differential diagnosis) of SRE.

Sleep Related Epilepsies

- Benign Epilepsy with Centro Temporal Spikes (BECTS)
- Panayiotopoulos Syndrome (PS)
- Juvenile Myoclonic Epilepsy (JME)
- Epilepsy with Generalized Tonic Clonic Seizures alone (GTCSa) (previously known as Idiopathic Generalized Epilepsy with Generalized Tonic Clonic seizures on awakening)
- Sleep-related Hypermotor Epilepsy (SHE)
- Lennox-Gastaut Syndrome (LGS)
- West Syndrome (WS)
- Continuous Spike-Wave during Slow Wave Sleep (CSWS)
- Landau-Kleffner Syndrome (LKS)
Rolandic Epilepsy

Lennox Gastaut syndrome

Courtesy Drs. Sartori, Dr. Terzaghi, Mondino Institute, Pavia

Lennox Gastaut syndrome
Lennox Gastaut syndrome

Clinical features of sleep-related hypermotor epilepsy (SHE) in relation to the seizure onset zone

Nocturnal paroxysmal dystonia
The description of NPD triggered a debate on whether it constituted an epileptiform manifestation or a new type of sleep disorder

<table>
<thead>
<tr>
<th>TABLE 2. Summary of polygraphic data</th>
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<tbody>
<tr>
<td>Polygraphic variable</td>
</tr>
<tr>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Number of recorded seizures</td>
</tr>
<tr>
<td>Onset</td>
</tr>
<tr>
<td>EEG during transitions</td>
</tr>
<tr>
<td>EEG during seizures</td>
</tr>
<tr>
<td>EEG during episodes</td>
</tr>
</tbody>
</table>

Note: Seizures were clinically similar in all patients.
Nocturnal paroxysmal dystonia

The description of NPD triggered a debate on whether it constituted an epileptiform manifestation or a new type of sleep disorder.

Lugaresi and Cirignotta Sleep 1981; Lugaresi et al. JNNP 1986

Nocturnal paroxysmal dystonia

... wide and disordered purposeless movements, which assumed a choreoathetoid and/or ballic aspect; dystonic posturings, localised to only one limb or involving the entire trunk and all limbs, were usually associated or alternated with the dyskinetic movements (Lugaresi and Cirignotta 1986).

Complex Partial Seizures of Frontal Lobe Origin

Complex partial seizures of medial or orbital frontal origin were documented in 10 of 90 patients with intractable epilepsy who were studied with depth electrodes. The clinical features that, in part, served to distinguish these seizures from complex partial seizures originating elsewhere included brief, frequent attacks, complex motor automatisms with kicking and thrashing, sexual automatisms, vocalization, and frequent development of complex partial status epilepticus. The constellation of clinical characteristics was often bizarre, leading to the erroneous diagnosis of hysteria. Stereotyped attack patterns helped establish the diagnosis of epilepsy. Intracranial and ictal scalp electroencephalograms were often not helpful and were sometimes misleading.


Nocturnal Paroxysmal Dystonia with Short-Lasting Attacks: Three Cases with Evidence for an Epileptic Frontal Lobe Origin of Seizures

Paolo Tinuper, Angelina Cerullo, Fabio Cirignotta, Pietro Cortelli, Elio Lugaresi, and Pasquale Montagna

Institute of Neurology, University of Bologna, Bologna, Italy
Nocturnal Paroxysmal Dystonia with Short-Lasting Attacks: Three Cases with Evidence for an Epileptic Frontal Lobe Origin of Seizures

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Stereo-EEG allow to investigate the spatiotemporal evolution of epileptic activity and its relation to clinical signs

If signs and symptoms:
- Are not in agreement with MR or interictal EEG
- Identify the lobe but not the side
- Suggest a precocious diffusion in wide areas
- Are not pointing to an univocal origin of seizures
And Video-EEG doesn't resolve the doubtful points
Neuronal nicotinic acetylcholine receptor subunits (nAChR)

Missense mutations in KCNT1, (DEP domain-containing protein 5) Mutations of the CRH gene

<table>
<thead>
<tr>
<th>Gene</th>
<th>Location</th>
<th>Mutation</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRH</td>
<td>8q13</td>
<td>g.C475T*A</td>
</tr>
<tr>
<td>DEPDC5</td>
<td>23q2</td>
<td>g.4804C&gt;T</td>
</tr>
</tbody>
</table>


Guerrini et al. Neurotherapeutics 2014

Autosomal Dominant NFLE - ADNFLE

Nocturnal Frontal Lobe Epilepsy

• Prevalence 1.8-1.9/100,000 (Vignatelli et al. 2015)
• Seizures begin before the age of 20 years, with a peak during childhood-adolescence (Vignatelli et al. 2015, Nobili et al. 2014)
• Seizure frequency is usually high, and patients generally experience many seizures a night (Scheffer et al. 1995, Oldani et al. 1996, Nobili et al. 2014)
• Occasional seizures during wakefulness can occur (Provini et al. 1999, Nobili et al. 2007)
• Clinical neurologic examination is normal and intellect is usually preserved, but reduced intellect, psychiatric comorbidity, or cognitive deficits may occur (Oldani et al. 1996, Magnusson 2003, Heron et al. 2012)
• 30% of cases are resistant to carbamazepine and to other antiepileptic drugs (Hirsh et al., 1994; Provini et al., 1999, Nobili et al., 2007)
Nocturnal Frontal Lobe Epilepsy
Clinical features common to the genetic and symptomatic forms

• **Major attacks**: stereotyped movements of 20-30 seconds' duration characterized by asymmetric tonic or dystonic posturing, ballistic or other complex movements (Lugaresi and Cirignotta, 1981; Lugaresi et al. 1986; Oldani et al. 1996, Provini et al. 1999, Nobili et al. 2007)


• **Minor motor events**: short-lasting (2-4 seconds) stereotyped movements interesting the limbs, the axial musculature and/or the head (recurring periodically) (Sforza et al. 1993, Oldani et al. 1996, 1998, Provini et al. 1999, Terzaghi et al. 2007)

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**Minor events and Paroxysmal arousals**

---

**FRAGMENTS OF MAJOR SEIZURES**

...the increasing complexity of the ictal motor behavior may reflect a different duration and propagation of the discharge within the frontal lobe (Montagna et al. 1990, Montagna 1992)
Relationship of epileptic discharges to arousal instability

Brief arousals do not discriminate
Parasomnias

Epilepsy

Always a frontal onset?

Epileptic Nocturnal Wandering with a temporal lobe origin

Sleep-related hyperkinetic seizures of temporal lobe origin

Insular-opercular seizures manifesting with sleep-related paroxysmal motor behaviors: A stereo-EEG study

**Table 1. Main subjective and objective clinical features of motor manifestations**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Auras</th>
<th>Type of major seizures (SML classification)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&quot;Threatening inner voice&quot; followed by staring and freezing</td>
<td>Hyperkinetic automatisms (1.2.2), focal stereotypies</td>
</tr>
<tr>
<td>2</td>
<td>Feeling of floating in the air and screaming</td>
<td>Dysdiadochokinesia, hyperkinetic automatisms (1.2.2)</td>
</tr>
<tr>
<td>3</td>
<td>Feeling of electrical buzzing</td>
<td>Right temporal lobe seizures (1.2.1)</td>
</tr>
<tr>
<td>4</td>
<td>Feeling of pressure behind the forehead, followed by right hand</td>
<td>Right hemispheric seizures (1.2.1)</td>
</tr>
<tr>
<td>5</td>
<td>&quot;Unpredictable rigidity&quot;</td>
<td>Hypokinesia, blepharospasm</td>
</tr>
<tr>
<td>6</td>
<td>Feeling of being suffocated</td>
<td>Diffuse electroencephalographic suppression</td>
</tr>
<tr>
<td>7</td>
<td>Feeling of dizziness</td>
<td>Right temporal lobe seizures (1.2.1)</td>
</tr>
<tr>
<td>8</td>
<td>Feeling of smell or taste</td>
<td>Right temporal lobe seizures (1.2.1)</td>
</tr>
</tbody>
</table>

*Data from Ryvlin et al. Epilepsia 2006; Dobesberger et al. Epilepsia 2008; Proserpio et al. Epilepsia 2011; Proserpio et al. Sleep Med 2011.*

Immediately before the episodes, the patient occasionally felt a vivid sensation of levitation.

**Sleep Related Hypermotor Seizures with a Parietal Onset**

*Data from Ryvlin et al. Epilepsia 2006; Dobesberger et al. Epilepsia 2008; Proserpio et al. Epilepsia 2011; Proserpio et al. Sleep Med 2011.*
Hypermotor seizures arising outside the frontal lobe

Consensus Conference on NFLE

Aims of the conference
- Define the clinical features of the disorder
- Define the anatomo-electro-clinical characteristics of the seizures
- Establish diagnostic criteria with levels of certainty
- Assess evidence for etiology
- Define research need

Conference Methodology
- Participants included experts in epilepsy, sleep, and epidemiology
- Literature review completed before the Conference and working groups were assembled to address specific questions

Sleep-related Hypermotor Epilepsy (SHE)

- Sleep-related
  - Sleep, rather than time of day, is critical
  - Seizures may originate outside the frontal lobe

- Stereotyped, abrupt, hypermotor seizures
  - “Hypermotor” includes seizures with vigorous hyperkinetic features and seizures with asymmetric tonic or dystonic posturing

Timper et al Neurology 2016
Frontal vs. Extra-frontal SHE

• **Review of our surgical database of drug-resistant SHE including:**
  - Clinical features
  - Stereo-EEG when available
  - Seizure onset zone
  - Post-operative outcome

• **From October 1997 to July 2015**
  - Patients with Hypermotor Seizures (HMS) occurring more than 90% during sleep
  - Subdivided according to seizure onset zone: frontal, temporal, insular-opercular and posterior

**Semeiology Pattern 1**
early elementary motor signs, which included early clonic signs, asymmetric tonic postures and/or an asymmetric facial contraction.

**Semeiology Pattern 2**
unnatural hypermotor movements, which included non-integrated or anarchic gestural hypermotor movements with axial tonic postures and/or symmetric facial contractions.

**Semeiology Pattern 3**
integrated hypermotor movements, which included hyperkinetic behaviours (pedalling, kicking, rocking), distal stereotypies and/or manipulation/utilisation movements in the absence of clear a goal-directed purpose.
Semeiology Pattern 4

gestural behaviours with high emotional content, which include integrated gestural behaviours of fear, fight or flight behaviour, frightened facial expression and/or autonomic signs.

Patient selection

1433 patients
157 patients (sleep-related epilepsy)
136 SHE
1276 patients (not sleep-related)
19 NTLE*
2 unspecified

Patient selection

1433 patients
157 patients (sleep-related epilepsy)
136 SHE
1276 patients (not sleep-related)
19 NTLE*
2 unspecified

Group characteristics

<table>
<thead>
<tr>
<th></th>
<th>Frontal (n=91)</th>
<th>Extra-frontal (n=44)</th>
<th>Temporal (n=14)</th>
<th>Operculo-insular (n=21)</th>
<th>Posterior (n=9)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age of onset - years</td>
<td>6.1 ± 4.5</td>
<td>5.1 ± 4.0</td>
<td>5.0 ± 4.4</td>
<td>4.7 ± 3.7</td>
<td>6.4 ± 4.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>Mean duration of disease - years</td>
<td>18.1 ± 9.9</td>
<td>17.3 ± 11.0</td>
<td>17.6 ± 8.8</td>
<td>17.5 ± 11.8</td>
<td>16.6 ± 13.2</td>
<td>n.s.</td>
</tr>
<tr>
<td>Seizure frequency (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily</td>
<td>62 (68)</td>
<td>25 (57)</td>
<td>4 (29)</td>
<td>15 (71)</td>
<td>6 (67)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Weekly</td>
<td>22 (24)</td>
<td>13 (29)</td>
<td>7 (50)</td>
<td>5 (24)</td>
<td>5 (67)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Monthly</td>
<td>5 (6)</td>
<td>4 (9)</td>
<td>3 (21)</td>
<td>0</td>
<td>1 (11)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Sporadic</td>
<td>2 (2)</td>
<td>2 (5)</td>
<td>1 (5)</td>
<td>1 (5)</td>
<td>1 (11)</td>
<td>n.s.</td>
</tr>
<tr>
<td>EEG (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interictal activity</td>
<td>72 (79)</td>
<td>39 (89)</td>
<td>14 (100)</td>
<td>18 (86)</td>
<td>7 (78)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Localizing</td>
<td>40 (44)</td>
<td>30 (68)</td>
<td>13 (93)</td>
<td>11 (52)</td>
<td>6 (67)</td>
<td>0.03</td>
</tr>
<tr>
<td>Ictal activity</td>
<td>73 (78)</td>
<td>39 (89)</td>
<td>14 (100)</td>
<td>17 (81)</td>
<td>8 (89)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Localizing</td>
<td>47 (52)</td>
<td>27 (61)</td>
<td>10 (71)</td>
<td>11 (52)</td>
<td>6 (67)</td>
<td>n.s.</td>
</tr>
<tr>
<td>MRI-identifiable lesion</td>
<td>58 (64)</td>
<td>30 (68)</td>
<td>9 (64)</td>
<td>13 (62)</td>
<td>8 (89)</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

*according to Bernasconi et al. Neurology 1998
Early seizure semiology

Table 2: Frequency and types of early nonmotor manifestations in SHE

<table>
<thead>
<tr>
<th>SHE subgroups</th>
<th>Frontal</th>
<th>Temporal</th>
<th>Op-insular</th>
<th>Posterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>49/74 (66%)</td>
<td>8/14 (57%)</td>
<td>15/18 (83%)</td>
<td>8/9 (89%)</td>
</tr>
<tr>
<td>Type of nonmotor manifestations (n)</td>
<td>Sensory (10)</td>
<td>Epigastric (2)</td>
<td>Sensory (11)</td>
<td>Sensory (5)</td>
</tr>
<tr>
<td></td>
<td>Emotional (12)</td>
<td>Epigastric (2)</td>
<td>Epigastric (2)</td>
<td>Visual (2)</td>
</tr>
<tr>
<td></td>
<td>Epigastric (8)</td>
<td>Autonomic (2)</td>
<td>Auditory (1)</td>
<td>Undefinable (1)</td>
</tr>
<tr>
<td></td>
<td>Autonomic (6)</td>
<td>Cognitive (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cephalic (4)</td>
<td></td>
<td></td>
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</tbody>
</table>
Stereo-EEG studies of SHE

- Seizures analysis from 2-year post-operative Engel I patients
- Frontal SHE (40 pts, 108 seizures)
- Extra-frontal SHE (14 pts; 39 seizures)

Electrographic seizure duration in both groups divided into 20 second bins.

Elapsed time from stereo-EEG seizure onset to the start of the HMS seizure in both groups divided into 5 second bins.

Elapsed time from the first video-EEG detectable movement, usually an awakening, to the start of HMS semiology.
Onset of hyperkinetic behaviors

Stereo-EEG studies of SHE

- Seizures analysis from 2-year post-operative Engel I patients

<table>
<thead>
<tr>
<th></th>
<th>Frontal SHE (40 patients, 106 seizures)</th>
<th>Extra-frontal SHE (14 patients, 39 seizures)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrographic seizure duration</td>
<td>31.49 sec ± 27.28 [range 7-233 sec]</td>
<td>52.65 sec ± 22.40 [range 17-62 sec]</td>
<td>0.0001</td>
</tr>
<tr>
<td>Clinical seizure duration</td>
<td>24.55 sec ± 23.96 [range 3-174 sec]</td>
<td>42.60 sec ± 18.47 [range 14-84 sec]</td>
<td>0.0001</td>
</tr>
<tr>
<td>Seizure onset to HNO discomfort</td>
<td>4.71 sec ± 4.30 [range 8-28 sec]</td>
<td>9.57 sec ± 7.56 [range 1-27 sec]</td>
<td>0.0001</td>
</tr>
<tr>
<td>Seizure onset to awakening</td>
<td>6.49 sec ± 5.32 [range 1-98 sec]</td>
<td>29.42 sec ± 15.50 [range 1-60 sec]</td>
<td>0.0001</td>
</tr>
<tr>
<td>Awakening to HNO</td>
<td>1.95 sec ± 2.20 [range 0.9 sec]</td>
<td>11.29 sec ± 6.55 [range 0-33 sec]</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

*More than 80% of extra-frontal SHE seizures had a 10 sec or longer delay between the electrical onset of the seizure and the emergence of hypermotor manifestations in contrast to 21% of frontal onset seizures.

FCD type II is the most common histopathological substrates of drug-resistant SHE (67% of the cases) with no significant differences in relation with the location of the SOZ.


FCD type II is the most common histopathological substrates of drug-resistant SHE (67% of the cases) with no significant differences in relation with the location of the SOZ.

Post-operative outcome in SHE at 2 years

(extra available in 120/136 SHE patients (88%)

<table>
<thead>
<tr>
<th>Post-op outcome (2y)[%]</th>
<th>Frontal (91 pts)</th>
<th>Extra-frontal (42 pts)</th>
<th>Temporal (15 pts)</th>
<th>Operculo-insular (21 pts)</th>
<th>Posterior (9 pts)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engel class I</td>
<td>72 (84)*</td>
<td>34/45 pts</td>
<td>13/15 pts</td>
<td>9/21 pts</td>
<td>6/9 pts</td>
<td>0.008</td>
</tr>
<tr>
<td>Engel class II</td>
<td>4 (4)</td>
<td>24 (70)</td>
<td>0</td>
<td>9 (60)</td>
<td>3 (50)</td>
<td></td>
</tr>
<tr>
<td>Engel class III-IV</td>
<td>10 (12)</td>
<td>7 (21)</td>
<td>1 (8)</td>
<td>5 (33)</td>
<td>2 (33)</td>
<td></td>
</tr>
</tbody>
</table>

Extra-frontal SHE

- Temporal onset tend to produce hyperkinetic organized behaviours with a high emotional content
- Most HMS arising from an insular-opercular or posterior onset feature elementary motor components with dystonic features (70%) but hyperkinetic movements can also be present
- Seizures with high emotional content seem to be rare in posterior and insular-opercular SHE
- Post-ictal confusion, alteration of consciousness are more frequent in Temporal SHE

Sleep and Epilepsy Task Force

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Sleep and Epilepsy Task Force

Standard procedures for the diagnostic pathway of sleep related epilepsies (SRE) and comorbid sleep disorders: an EAN, ESRS and EC-ILAE consensus review

……to define the standard procedures for the diagnostic pathway of patients with possible SRE and the general management of patients with SRE and comorbidity with sleep disorders

The document is a consensus review following the recommendations of the EAN for the production of EAN guidelines (Leone 2013, Leone 2015).
The following clinical scenarios were addressed:

1) **Scenario 1**: the diagnostic pathway for sleep-related paroxysmal clinical or neurophysiological manifestations.

   This scenario was conceptually devised by experts and filled with the answers to basic questions (PICO questions). PICOs were considered for each of the SRE and addressed the diagnostic accuracy of clinical history, daytime standard EEG, daytime sleep EEG, home-video recording and in-lab video-EEG-polysomnography (PSG).

1) **Scenario 2**: the management for SRE comorbidity with sleep disorders.

   This scenario was conceptually devised by experts and filled with the answers to basic questions (PICO questions). Each PICO was considered for the comorbidity with the single sleep disorder (such as sleep-disordered breathing, restless legs syndrome, insomnia) of patients with SRE and addressed the specific management of these conditions.

**Scenario 1**: the diagnostic pathway for sleep-related paroxysmal clinical or neurophysiological manifestations.

The diagnostic pathway to be applied in case of sleep related paroxysmal manifestations/suspected SRE considers the following steps:

- 1.1 Clinical history
- 1.2 Tools for capturing the event at home: home-video recording
- 1.3 Tools for objective evaluation in lab
- 1.3.1 for capturing the event: overnight recording (Video-EEG-PSG)
- 1.3.2 for recording interictal abnormalities
  - 1.3.2.1 Daytime standard EEG
  - 1.3.2.2 Daytime sleep EEG
  - 1.3.2.2 Overnight recording (Video-EEG-PSG)

**PICO questions**

<table>
<thead>
<tr>
<th>P</th>
<th>I</th>
<th>C</th>
<th>O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population Patient Problem</td>
<td>Intervention Or Exposure</td>
<td>Comparison</td>
<td>Outcome</td>
</tr>
<tr>
<td>Who are the patients?</td>
<td>What do we do to them?</td>
<td>What do we compare the intervention with?</td>
<td>What happens? What is the outcome?</td>
</tr>
</tbody>
</table>

Types of outcomes

- Diagnostic PICO: reproducibility, sensitivity, specificity.
- Management/therapeutic PICO: epilepsy remission or 50% reduction in seizure frequency.

**BECTS, PS, JME, IGE-GTCSaw.**

Capturing the event

- Home Video Recording

Clinical History

Daytime EEG for recording interictal abnormalities

- Standard-EEG
- Sleep-EEG

Overnight recording: Video-EEG-PSG

Diagnosis
Clinical History

SHE

Capturing the event
Home Video Recording

Overnight recording: Video-EEG-PSG

Clinical History

Daytime EEG for recording interictal abnormalities

Standard-EEG

Sleep-EEG

Diagnosis

LGS, WS

Capturing the event
Home Video Recording

Overnight recording: Video-EEG-PSG

Clinical History

Daytime EEG for recording interictal abnormalities

Sleep-EEG

Diagnosis

ESES/CSWS - including LKS

Capturing the event
Home Video Recording

Overnight recording: (Video)-EEG-(PSG)

Clinical History

Daytime EEG for recording interictal abnormalities

Standard-EEG

Sleep-EEG

Diagnosis

Scenario 2: the management for SRE comorbidity with sleep disorders.

The diagnostic and management framework and pathway to be applied in case of SRE and suspected comorbid sleep disorders (sleep disordered breathing, insomnia, RLS/PLMD) include the following steps:

2.1 Clinical history
2.2 Further diagnostic workup
2.3 Management / treatment
Statements

• Any condition leading to sleep disruption and sleep deprivation should be recognized and treated according to standard procedures. Considering to high frequency of the disorders we highlight the following situations:

Sleep-disordered breathing (SDB)

✓ To treat comorbid sleep disordered breathing (SDB) in sleep-related epilepsy is beneficial. Treating SDB should be considered independently of its severity in non-seizure free patient.
✓ Benefit of OSAS treatment on seizure reduction and sleepiness must be controlled by follow-up
✓ Caution should be used in treating epilepsy patients with sedating and inducing weight gain AEDs or with VNS, in case of SDB comorbidity

Chronic Insomnia:

- Cognitive Behavioral Therapy (CBT) is considered as first choice for treatment of insomnia in subjects with sleep-related epilepsy, with caution in adopting sleep restriction procedure as it may induce daytime sleepiness and may be provoke seizure in particular epilepsy syndromes.
- BDZ, Z-drugs and low-dose histaminergic drugs should be taken in consideration as first choice drugs for pharmacological treatment.

RLS/PLMS:

- There is high degree of consensus among the members of the panel about the need for RLS/PLMs treatment, if they are associated with sleep fragmentation, even without RLS.
- Gabapentin and pregabalin are considered the first drug treatment choice to treat RLS and PLMs in the specific context of sleep related epilepsy.

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