suffered from 21 attacks in total during the polysomnography. Attacks were not related to specific sleep stages (N1: 2/21 attacks, N2: 6/21 attacks, N3: 5/21 attacks, REM: 7/10 attacks).
eCH patients had longer latency (18.9 vs. 11.7 minutes, $p<0.05$) and lower efficiency (84.4 vs. 86.5, $p<0.05$) compared with controls, but fewer PLMs (0.67 vs. 1.30 hour^{-1}, $p<0.05$). Finally, the sleep apnea index was similar in both groups (9.63 vs. 7.76 hour^{-1}, $p=0.7674$).
Conclusion: This is the first study that systematically investigates eCH patients with full polysomnography in both bout and remission and the largest study comparing eCH patients with controls. The observed sleep disturbances were not associated with the bout but rather seem to be the manifestation of a persisting, underlying pathology. Finally, the prevalence of sleep apnea was comparable in all groups and attacks were not related to specific sleep stages.

O42

Real-world preventative drug management of Chronic Migraine among Spanish Neurologists

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Background:
In migraine, the therapeutic preventive drug arsenal is varied. When prescribing both Guidelines and patient characteristics are taken into account. In Spain, the use of preventive therapies seems to be heterogeneous.
The objective of this study was to evaluate real-life clinical prescribing practice amongst neurologists in Spain
Methods:
Observational descriptive study done with a survey by Neurologists of the Spanish Neurological Society (SEN). Neurologists who participated were divided into Headache Specialists or not. The following data was collected: socio-demographic data,; preventive treatment and choices different migraine sub-types, and their personal perception of efficacy and tolerability to different drugs.
Results:
We analyzed 152 surveys from neurologists around our country. From them: 43.4% were female, 53.3% <40 years, and 34.9% were interested in headache .
In regards to preventive treatment choice; in chronic migraine topiramate (57%) amytriptiline (17.9%) and beta-blockers (14.6%), whereas in episodic migraine the preferred drugs were beta-blockers (47.7%), topiramate (21.5%) and amytriptiline (13.4%).
Regarding perceived efficacy, topiramate was considered the best option in chronic migraine (42.7%) followed by onabotulinumtoxinA (25.5%) and amitryptiline (22.4%). In episodic migraine, neurologist preferred topiramate (43.7%) and beta-blockers (30.3%).
Regarding the duration of preventive therapy when improvement was achieved, when treating episodic migraine 43.5% of the surveyed neurologists recommended 3 months and 39.5% preferred 6 months. When they treated chronic migraine, 20.4% of neurologists recommended 3 months, 42.1% 6 months, 12.5% 9 months and 22.4% preferred to maintain treatment during 12 months.
When considering onabotulinumtoxinA treatment, the number of prior therapeutical failures was cero in 7.2% of surveyed, one in 5.9%, two in 44.1%, three in 30.9%, and four or more in 11.9%. The increase of OnabotulinumtoxinA dose up to 195 UI was considered by 51% of neurologists after a first ineffective procedure, by 42.2% after two injections, and by 83% after a third infiltration. Surveyed colleagues admitted to take into account in their decisions mainly patient comorbidities (70.2%) rather than guidelines (13.9%).
Conclusions:
Initial management of Migraine among Spanish Neurologists is made with the preventative drugs which are considered as first choices in most of the guidelines. Management of episodic migraine differed from chronic migraine, both in the order or drugs and the perception of the most effective therapy.

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Effect of the H_1-antihistamine clemastine on PACAP38 induced migraine

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Objective To investigate the effect of the H_1-antihistamine clemastine on the migraine inducing abilities of pituitary adenylate cyclase activating peptide-38 (PACAP38).
Methods We conducted a double-blind, randomized, placebo controlled two-way cross-over study. Twenty migraine without aura patients were randomly allocated to receive bolus clemastine 2 mg (1 mg/ml) or bolus saline 2 ml intravenously over 2 min on two study days. Following each bolus injection 10 pmol/kg/min of PACAP38 was administered intravenously over 20 min. We recorded migraine/headache characteristics every 10 min until 90 min after infusion start and collected blood to investigate mast cell degranulation and the inflammation markers tryptase and tumor necrosis factor-alpha (TNF-alpha) before and after infusion of PACAP38.
Results After clemastine pretreatment 5/20 participants developed a migraine-like attack in response to a PACAP38 infusion compared to 9/20 after placebo pretreatment ($P=0.288$). Following clemastine pretreatment 15/20 participants reported headache in response to a PACAP38 infusion, whereas 19/20 participants did so following placebo pretreatment ($P=0.221$). We found no difference in area under the curve 12 h (AUC_{12h}) for headache intensity between the two experimental days ($P=0.481$). We found no difference in AUC_{180min} for tryptase ($P = 0.525$) or TNF-alpha ($P = 0.487$) between clemastine and placebo pretreatment days.
Conclusion H1-antihistamine, clemastine, failed to prevent migraine or headache after PACAP38 infusion thus making a role for histamine release or mast cell degranulation in PACAP38 induced migraine less likely.

O44

CSF pressure fluctuations as a marker of isolated CSF hypertension in headache sufferers

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Background
It is needed to identify the characteristics and the pressure-related features of isolated cerebrospinal fluid hypertension for differentiating headache sufferers with isolated cerebrospinal fluid hypertension from those with primary headache disorder.
Patients and Methods
In this prospective study patients with refractory chronic headaches suspected of having cerebrospinal fluid-pressure elevation without