potential role as a biomarker, both in terms of absolute mean jitter value or as a percentage change in those values. It does suffer in terms of specificity, though. Although SF-EMG was unable to correctly distinguish a true case of MG from a case where the patient developed Myasthenia gravis-like symptoms due to a rare mitochondrial mutation in some of the studies, SF-EMG was of primordial importance in confirming the disease when the associated AChR, striational muscle and MuSK antibodies were altogether absent from the serum.

Conclusions

Single-fiber electromyography offers significant practical and analytical flexibility compared to more invasive techniques, such as muscle biopsy. As evidenced, SF-EMG can provide the missing confirmatory link between a myriad of symptoms and MG.

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118509

Neurophysiological features of the atypical truncal myoclonus preceded by epileptic seizures

Hiroya Ohara[ab], Hironori Shimizu[ab], Masami Yamanaka[c], Naokuni Iguchi[b], Keiko Tanaka[d], Kazuma Sugie[a], Masako Kinoshita[e], *[a]Nara Medical University, Neurology, Kashihara, Japan, [b]Minami-nara General Medical Center, Neurology, Yoshino, Japan, [c]Minami-nara General Medical Center, Clinical Laboratory, Yoshino, Japan, [d]Niigata University, Department of Animal Model Development, Brain Research Institute, Niigata, Japan, [e]National Hospital Organization Utano National Hospital, Neurology, Kyoto, Japan*

Background and aims

Etiology of truncal involuntary movement is difficult to differentiate. Here, we investigated the neurophysiological features of truncal myoclonus subsequent to epileptic seizure.

Methods

A 48-year-old female patient with spontaneous jerks was investigated. One month ago, she suddenly felt nausea and developed clonic seizures during her inspection work using flash lamps. Thereafter she had frequent involuntary jerks of the neck, upper limbs, and trunk. The clinical features and simultaneous records of EEG and surface electromyography (EMG) polygraph were analyzed.

Results

The involuntary movement was observed both in awake and in sleep, and elicited by stimuli. EEG showed slow waves in bilateral temporal areas. EMG revealed 107 jerks; the duration of burst of sternocleidomastoid (SCM) was 606.2 ± 185.6 ms (mean ± SD) and that of rectus abdominis (RA) was 490.1 ± 203.1 ms. In 55 jerks (51%) with more than 4.0 ms difference between the EMG onset of the muscles, the interval from SCM down to RA was significantly longer than that from RA up to SCM (185.5 ± 143.6 vs 77.4 ± 66.5 ms: $p < 0.001$, *t*-test). No pre-movement potentials were detected by jerk-locked averaging. Giant SEP and C-reflex were absent. Laboratory examinations including glycine receptor antibodies and MRI of head and spine were unremarkable.

Conclusions

Induction of involuntary movement by acoustic stimuli of this patient is compatible with the reticular reflex myoclonus, whereas characteristics of long burst duration, faster ascending than descending propagation in the spinal cord, and occurrence during sleep are compatible with the propriospinal myoclonus.

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118510

Neurodegeneration and neoangiogenesis in the cerebellar cortex in pentylenetetrazol (PTZ)- kindled rats treated with axitinib and rapamycin

Leonid Godlevsky[a], Mykhailo Pervak[a], Olesya Poshyvak[b], Kirill Latypov[a], Kseniya Prybolovets[a], *[a]Odesa National Medical University, Department of Biophysics, Informatics and Medical Devices, Odesa, Ukraine, [b]Danylo Halytsky Lviv National Medical University, Pharmacology Department, Lviv, Ukraine*

Background and aims

Morphological changes induced in pentylenetetrazol (PTZ)-kindled rats create the basis for the resistance of chronic brain epileptization. Cerebellar contribution to kindling seizures development was shown (Godlevsky L.S.et al., 2020). To investigate histological characteristics of the cerebellar paleocortex in PTZ-kindled rats under conditions of axitinib and rapamycin administration.

Methods

For kindling induction, we used PTZ administrations (35.0 mg/kg, i.p.) for three weeks. Axitinib (2.0 mg/kg, i.p.) and rapamycin (0.56 mg/kg, i.p.) administered during ten days. Light microscopy was performed on hematoxylin and eosin painted slices (x200 magnification). Data presented as +S.E., ANOVA Fisher test used for statistics and differences at $p < 0.05$ are presented.

Results

The thickness of the molecular layer decreased in kindled rats from 238.5 + 18.3 to 168.6 + 15.6 μM. The number of PC cells per 1 mm in lobule VI reduced from 65.0 + 2.2 to 38.0 + 1.5. The square of stellate cell nuclei reduced from 16.43 + 0.93 to 10.27 + 0.72 μM2. The thickness of granular cells decreased from 230.2 + 17.7 to 156.8 + 13.4 μM. The density of micro vessels increased in kindled rats from 25.5 + 2.2 to 40.7 + 2.8 per 2500 μM2. Treatment with axitinib and rapamycin prevented kindled generalized seizure in 8 out of 11 rats. After treatment, the thickness of the molecular layer was 185.0 + 19.3 μM, the number of PC per I mm of length was 53.0 + 3.2 per 1 mm, the number of micro vessels was 34.7 + 3.0, which was not different from the control ($P > 0.05$).

Conclusions

PTZ kindling resulted in neurodegeneration in all layers of the cerebellar cortex and increased neoangiogenesis. Treatment with axitinib and rapamycin prevented kindling-induced morphological deteriorations.

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118511

Blink reflex recovery cycle distinguishes patients with idiopathic normal pressure hydrocephalus from elderly subjects

Alessandro Mechelli[a], Andrea Quattrone[a], Rita Nisticò[b], Marianna Crasà[c], Domenico La Torre[d], Basilio Vescio[e], Aldo Quattrone[bc], *[a]University Magna Graecia, Institute of Neurology, Department of Medical Sciences, Catanzaro, Italy, [b]National Research Council, Neuroimaging Research Unit, Institute of Molecular Bioimaging and Physiology, Catanzaro, Italy, [c]University Magna Graecia, Neuroscience Research Centre, Catanzaro, Italy, [d]University Magna Graecia, Institute of Neurosurgery, Department of Medical Sciences, Catanzaro, Italy, [e]Biotecnomed S.C.Ar., Catanzaro, Italy*