Epilepsy Journal
ISSN: 2472-0895

Andrade-Machado, J Epilepsy 2016, 2:2
DOI: 10.4172/2472-0895.1000108

Review Article Open Access

Understanding Hyper Motor Seizures

René Andrade-Machado*

National Institute Neurology, CES University, Colombia

***Corresponding author:** René Andrade-Machado, National Institute Neurology, CES University, Colombia, Tel: 57 (4) 576 66 66; E-mail: reneandrade1970@yahoo.es

Received date: June 10, 2016; **Accepted date:** June 28, 2016; **Published date:** June 30, 2016

Abstract

Introduction: Hyper Motor Seizures [HMS] are characterized by complex high amplitude movements involving proximal segments of the body resulting violent and inappropriate to the context.

Objective: To review the possible ictal onset zones related to HMS and the cortical areas that would be covered if invasive recording are needed.

Development: Semiology can predict the localization of ictal onset zone. Two subtypes of HMS [type 1 and 2] have been described. HMS1 is associated with an epileptogenic zone on the ventromedial frontal cortex and HMS2 has been associated with a more dorsal epileptogenic zone than those resulting in HSM1. However, HMS can also be originated in temporal lobe [mesial, neocortical or in the pole], in insular cortex or even in parietal lobe. The origin of HMS can be suspected by the associated signs. Thus, HMS originating in the insula–operculum regions can be associated with various somatosensory auras; in parietal seizures, propioceptive sensations may precede hyper motor behavior [HM], whereas autonomic and emotional auras prompt to think in the temporal lobe origin.

Conclusion: Except in cases of lesional temporal lobe epilepsy with concordant prersurgical results, all patients with HMS should be evaluated through intracranial recordings. The auras, the HMS subtype, the hypometabolic areas showed by PET study, and the localization and lesion type seeing on MRI can help to decide what structures should be covered with depth electrodes during invasive recordings.

Keywords: Hypermotor seizures; Semiology; Invasive recording; Supplementary motor area; Temporal lobe epilepsy; Insular epilepsy

Introduction

Hypermotor Seizures [HMS] are characterized by complex high amplitude movements involving proximal segments of the body resulting violent and inappropriate to the context. These movements include proximal segments of the limbs or/and trunk with beating, kicking, creaming, boxing and pelvic thrusting and might or not be associated to dystonic posturing, head turning, or emotional symptoms [1].

Due to their bizarre characteristics, rapid postictal recovery, its occasionally preserved consciousness and the absence of a clear/cut ictal pattern in scalp electroencephalograms, they are frequently diagnosed as non-epileptic attacks. Hypermotor semiology has been associated to frontal lobe seizures; especially those originating from the mesial frontal or orbitofrontal regions but hypermotor seizures have also been described in seizures originating from the dorsolateral frontal lobe, mesial and neocortical temporal lobe, upper and anterior aspect of insular cortex and parieto-temporal cortex [2-10].

This review is focused in the ictal onset zones related to HMS, the subjacent networks and the cortical areas that should be covered if invasive recording are needed.

Semiology can predict the localization of seizure onset zone

Rheims et al. described two subtypes of HMS: HMS1, characterized by marked agitation including one or several of the following motor behaviors: Body rocking, kicking, or boxing, associated with a mimic automatism like a facial expression of fear. In this type of HMS tonic or dystonic posturing are rare presented. The motor behavior looks like semipurposeful, as if the patients were trying to protect themselves against an external threat, reactive automatisms. Autonomic signs, ipsilateral head deviation and auras as described in temporal lobe seizures are frequently found. The authors, primarily associated HMS1 with an EZ centered on the ventromedial frontal cortex [11].

HMS2 included horizontal movements or rotation of trunk and pelvis while the patients were lying on the bed. Otherwise to HMS type I, this type of seizure was usually associated with tonic/dystonic posturing and rare mimic automatisms. Rheims et al., found the HMS 2 in patients with an epileptogenic zone situated both dorsally in relation to mesial premotor cortex and the anterior cingulate cortex [11].

Otherwise, Alqadi et al. analyzed 16 patients who underwent epilepsy surgery after 2 year of resection. The authors did not find any statistic association between the sites of resection with either type of HMS. In addition, they described one patient with both, types of HMS semiology. What was relevant in their study was that HMS arising in the frontal lobe trend to be earlier during the seizures evolution than those arising outside the frontal lobe [12]. Nevertheless, an early onset of hypermotor behavior during the sleep has seen in some patients

with temporal lobe epilepsy and HMS. The early onset was considered an effect of sleep synchronization.

Hypermotor seizures and sublobar onset

Carreño et al. has suggested that HMS in patients with temporal lobe epilepsy arisen in temporal pole [13]. Nevertheless, Yu et al. published 9 patients with temporal lobe epilepsy in whom HMS were reported. At least 21 seizures were recording in 7 patients. These patients were assessed with intracranial electrodes. They found that HMS arose either from medial (6 patients) or lateral (one patient) aspect of temporal lobe. The authors also reported long interval from EEG origination to the beginning of hypermotor behavior. It is interesting that prior to hypermotor behavior all patients reported some ictal signs that put the seizure onset zone outside from frontal lobe. These signs included but are not limited to: auras, mimic, hands and vocal automatisms [4].

According to above mentioned reports both type of HMS can be seen in patients with temporal lobe epilepsy, although HMS type 2 appears more frequently and the temporal pole can be, by far, the seizure onset zone.

HMS arising from the insula–operculum regions are usually associated with somatosensory auras; most of them are reported to occur in the larynges and/or the throat. Autonomic, gustatory, and emotional auras are also reported. Among autonomic signs a feeling of suffocation could be frequent seen. Opercular signs such as hypersalivation accompanied, ictal semiology in the majority of cases. Ryvlin et al. hypothesized that different clinical features in insular epileptic patients reflect the area of the insular cortex involved at ictal onset [14]. Thus, seizure arising from the upper and superior aspect of the insular cortex could mimic the seizures seen in nocturnal frontal lobe epilepsy, whereas seizures involving the posterior or anteroinferior aspect of the insular cortex show clinical features that mirror to those of temporal lobe epilepsy [9].

It has been reported independently an unusual cases of a patients presenting with a history of drug-resistant epilepsy with HMS arising from the mesial or lateral parietal regions [posterior cingulate cortex, supramarginal gyrus or mesial aspect of superior parietal gyrus]. The seizure onset zones were all confirmed by stereo-electroencephalography investigation (SEEG). The presence of an infrequent feeling of levitation, fear, falling sensation, blurred vision, or dizziness before the HM behavior were key to suspecting parieto-occipital involvement. None of the cases presented ictal dystonia or mimic automatisms. It is interesting to note that an intense grasping of the bed railing associated to movements of the trunk was seen in 2 patients with a posterior cingulate seizure onset zone [12,15-17]. Grasping involved the two hands, synchronously and it was directed to homologous sites with respect to the longitudinal body axis and last until the end of the seizure. This semiology differs from the ictal grasping reported in frontal lobe seizures. Frontal grasping is characterized by brief, repetitive, forced prehension of an object or a body segment during the whole ictal behavior.

The above mentioned analysis about HM behavior and their associated signs may give some useful clues to determine the area of the cortex involving on it.

Neural network subjacent to HMS

The mechanisms resulting in ictal hypermotor behavior remain speculative. It is known that an inhibitory connection from orbitofrontal cortex to the amygdala inhibits the innate and physiologically violent motor behavior usually associated to emotional arousal. Thus, HMS1 may be caused by the direct deactivation of these inhibitory inputs from limbic frontal-temporal network while seizures involve cortical areas related to the violent motor behavior [18,19]. Also, some of the signs associated to hypermotor behavior appearing when discharges involving the anterior cingulate cortex, the orbitofrontal cortex, and the temporal neocortex. In the cases of patients with nocturnal HMS of temporal lobe origin, the clinical manifestations start while discharges propagate to the cingulate and frontal cortical areas [20]. Thus, the same networks are involving in the generation of both motor behavior and non-motor signs observed in HMS type 1 [11]. Supporting this view some studies have described that ictal discharges in HMS type I follows different pathways: frontopolar, mesiotemporal, to premotor cortex and also to SMA.

Otherwise, in HMS 2, the dissemination of the ictal discharges follows a unidirectional pathway. Hence, some patients may remain responsive during their seizures. Dystonic posturing occurs when epileptic discharge involve the supplementary motor area and the dorsal anterior cingulate cortex [8,14,15]. Dorsal pathway related to HMS 2 ends in the cingulate and SMA cortex. That is why, this type of seizures is frequently associated to dystonic posturing. Some ictal activation of the basal ganglia during HMS could explain also the presence of abnormal posturing. This conclusion could be extrapolated from our knowledge about movement disorders [21-23]. Theses hypothesis seem to be plausible due to direct projections from both premotor cortex and dorsal anterior cingulate cortex to the dorsolateral striatum are being describing in primates [24-28].

All studies have reported more or less prolonged delays between ictal onset pattern on SEEG and the time to see ictal HM behavior in videos. Such delays may be considered due to ictal dysfunction of cortical and/or subcortical regions located far from the seizure onset zone [4,6,11,14,16]. Accordingly to the above mentionated role of the mesial frontal structures, it has been thought that HM movements in extra frontal epilepsies are caused by propagation of the ictal discharges to the frontal lobe. Nevertheless, little evidence supports this view. For instance, SMA involvement in posterior epilepsies is equally frequent in patients with or without HMS. Additionally, Enatsu et al. could not found any ictal pattern in mesial or lateral aspect of frontal lobe and orbitofrontal areas during HMSs. By contrary, a fast activity discharge was observed in the anterior cingulate cortex in two patients although without close temporal relationship with the onset of hyperkinetic movements [29].

Recent neuroimaging studies suggest that HM behavior might result from the involvement of the brainstem and basal ganglia. Also, ictal single-photon emission tomography [SPECT] performed during HMSs of either frontal or temporal lobe origin showed clusters of significant hyperperfusion not only in the anterior cingulate gyrus but also in the brainstem and especially in the midbrain, the pons, and the lentiform nucleus. The involvement of caudate nucleus and the red nucleus appears to be independently to the seizure onset zone according to statistical parametric mapping analysis done in patients with HMSs originated in frontal, temporal, or parietal lobes. Therefore, HMSs may represent the activation of innate motor schemes from subcortical structures. The most accepted hypothesis suggests that the spreading of ictal discharges causes either deactivation or activation of central

generator of motor behavior, this phenomenon account for the release of subcortical structures. Thus, it is difficult to separate the role of each of these nervous structures in the generation of ictal hyperkinetic behaviors. Such complex neural behavior might be difficult to explain exclusively by subcortical mechanisms [25,30-32].

However, experimental data had shown that the stimulation of periaqueductal grey matter in brainstem induce a motor behavior seen during panic attack [33]. On the other hand, involuntary movements such as tremor and restless legs syndrome have been associated to red nucleus dysfunction. In these patients some alterations of the execution and planning of coordinate movements have been reported [34]. Then, these subcortical structures are supposed to be part of the central generator able of generate an organized pattern of motor activity involving trunk and limb movements [35]. These networks may be responsible for archaically motor sequences but also are implicate more complex behavioral manifestation. The existence of such organization in humans has been recently suggested [36-38].

Invasive recording when and how

With exception of lesional temporal lobe epilepsy with concordant prersurgical results [interictal and ictal EEG, neuropsychology assessment and MRI] all cases with HMS should be evaluated through intracranial recordings.

Take into account the available evidence we usually divided the cortical generator of HMS into two causative pathways: Ventral pathway and dorsal pathway. The ventral pathway is related to mesial aspect of fronto basal structures, mesial, lateral or polar areas of temporal lobe, anterior and basal insular cortex and ventral aspect of anterior cingulate cortex. The dorsal pathway include posterior and dorsal aspect of insular cortex, dorsal aspect of cingulate gyrus, supramarginal gyrus, posterior cingulate cortex, mesial aspect of parietal cortex, dorso-lateral parieto-occipital cortex and supplementary motor area. Connections from these cortical areas with the cingulate cortex explain both, the initial symptoms [auras] and the described time lag between the staring of ictal pattern on electrocorticography and the time for the beginning of ictal complex motor behavior, which has been reported during invasive recording. The activation of the cingulate cortex, head of caudate, red nucleus, rubrospinal and reticulospinal tracts could explain the activation of complex motor and also emotional behaviors. Thus, surgical plan would contemplate placement of intracranial electrodes, mostly in cases with normal MRI. Hence mesial structures of frontal, parietal and temporal lobes are involving in generation of HMS; depth electrodes are preferable over grids or strip. Anterior or posterior cingulate gyrus, ventromedial frontal cortex, SMA, temporal pole, mesial temporal structures neocortical temporal cortex, insular lobe and parietal lobe should be considered during the procedure.

The auras, the type of HMS, the hypometabolic areas showed by PET study, and the localization and type of lesion on MRI can help to decide what of those structures should be covered with depth electrodes during invasive recordings.

Conclusion

The cortical generator of HMS can be divided into two causative pathways: Ventral pathway and dorsal pathway. Each one expressed with different semiological signs. This semiology helps to localize the epileptogenic zone and to plan the invasive recording. Except in cases of lesional temporal lobe epilepsy with concordant prersurgical results, all patients with HMS should be evaluated through intracranial recordings. The auras, the HMS subtype, the hypometabolic areas showed by PET study, the localization and the type of lesion uncovered on MRI can help to decide what structures should be covered. Due to most of demonstrated ictal onset zone in patients with HMS are depth and mesial brain structures, depth electrodes should be chosen, although some centers have used strip or grid to cover the hypothetic zones during invasive recording.

References

1. Kotagal P, Arunkumar G, Hammel J, Mascha E (2003) Complex partial seizures of frontal lobe onset statistical analysis of ictal semiology. Seizure 12: 268-281.
2. Yu T, Zhang G, Wang Y, Cai L, Zhou X, et al. (2013) Surgical treatment of hypermotor seizures originating from the temporal lobe. Seizure 22: 862-866.
3. Wang L, Mathews GC, Whetsell WO, Abou-Khalil B (2008) Hypermotor seizures in patients with temporal pole lesions. Epilepsy Res 82: 95-100.
4. Tao Y, Guojun Z, Yuping W, Lixin C, Wei D, et al. (2010) Surgical treatment of patients with drug-resistant hypermotor seizures. Epilepsia 51: 2124-2130.
5. Yu T, Zhang G, Li Y (2013) Surgical treatment of hypermotor seizures originated from temporal lobe. Epilepsia 54: 81.
6. Dobesberger J, Ortler M, Unterberger I, Walser G, Falkenstetter T, et al. (2008) Successful surgical treatment of insular epilepsy with nocturnal hypermotor seizures. Epilepsia 49: 159-162.
7. Marusic P, Elisak M, Krijtova H, Tomasek M, Mohapl M, et al. (2011) Hypermotor seizures in temporal lobe epilepsy patients. Epilepsia 52: 191.
8. Elisak M (2013) Temporal lobe epilepsy and hypermotor seizures. Epilepsy Behav 28: 328.
9. Proserpio P, Cossu M, Francione S, Tassi L, Mai R, et al. (2011) Insular-opercular seizures manifesting with sleep-related paroxysmal motor behaviors: A stereo-EEG study. Epilepsia 52: 1781-1791.
10. Staack AM, Bilic S, Wendling AS, Scholly J, Kraus U, et al. (2011) Hyperkinetic seizures in patients with temporal lobe epilepsy: clinical features and outcome after temporal lobe resection. Epilepsia 52: 1439-1446.
11. Rheims S, Ryvlin P, Scherer C, Minotti L, Hoffmann D, et al. (2008) Analysis of clinical patterns and underlying epileptogenic zones of hypermotor seizures. Epilepsia 49: 2030-2040.
12. Alqadi K, Sankaraneni R, Thome U, Kotagal P (2016) Semiology of hypermotor [hyperkinetic] seizures. Epilepsy Behav 54: 137-141.
13. Carreño M, Donaire A, Pérez Jiménez MA, Agudo R, Quilez A, et al. (2005) Complex motor behaviors in temporal lobe epilepsy. Neurology 65: 1805-1807.
14. Ryvlin P, Minotti L, Demarquay G, Hirsch E, Arzimanoglou A, et al. (2006) Nocturnal hypermotor seizures, suggesting frontal lobe epilepsy, can originate in the insula. Epilepsia 47: 755-765.
15. Montavont A, Kahane P, Catenoix H, Ostrowsky-Coste K, Isnard J, et al. (2013) Hypermotor seizures in lateral and mesial parietal epilepsy. Epilepsy Behav 28: 408-412.
16. Nishibayashi H, Ogura M, Taguchi M, Miki J, Uematsu Y, et al. (2009) Nondominant parietotemporal cortical dysplasia manifesting as hypermotor seizures. Epilepsy Behav 14: 691-695.
17. Gibbs SA, Figorilli M, Casaceli G, Proserpio P, Nobili L (2015) Sleep Related Hypermotor Seizures with a Right Parietal Onset. J Clin Sleep Med 11: 953-955.
18. Davidson RJ, Chapman JP, Chapman LJ, Henriques JB (1990) Asymmetrical brain electrical activity discriminates between psychometrically-matched verbal and spatial cognitive tasks. Psychophysiology 27: 528-543.

19. Bartolomei F, Gavaret M, Hewett R, Valton L, Aubert S, et al. (2011) Neural networks underlying parietal lobe seizures: A quantified study from intracerebral recordings. Epilepsy Res 93:164-176.
20. Biraben A, Taussig D, Thomas P, Even C, Vignal JP, et al. (2001) Fear as the main feature of epileptic seizures. J Neurol Neurosurg Psychiatry 70: 186-191.
21. Kravitz AV, Kreitzer AC (2001) Striatal mechanisms underlying movement, reinforcement, and punishment. Physiology [Bethesda] 27: 167-177.
22. Presti MF, Watson CJ, Kennedy RT, Yang M, Lewis MH (2004) Behavior-related alterations of striatal neurochemistry in a mouse model of stereotyped movement disorder. Pharmacol Biochem Behav 77: 501-507.
23. van den Heuvel OA, Veltman DJ, Groenewegen HJ, Cath DC, van Balkom AJ, et al. (2005) Frontal-striatal dysfunction during planning in obsessive-compulsive disorder. Arch Gen Psychiatry 62: 301-309.
24. Rizzolatti G, Fogassi L, Gallese V (2002) Motor and cognitive functions of the ventral premotor cortex. Current Opinion in Neurobiology 12: 149-154.
25. Bonini F, McGonigal A, Wendling F, Reegis J, Scavarda D, et al. (2013) Epileptogenic networks in seizures arising from motor systems. Epilepsy Res 106: 92-102.
26. Weinrich M, Wise SP (1982) The premotor cortex of the monkey. J Neurosci 9: 1329-1345.
27. Gallese V, Fadiga L, Fogassi L, Rizzolatti G (1996) Action recognition in the premotor cortex. Brain 2: 593-609.
28. Passingham RE (1998) Premotor cortex and preparation for movement. Exp Brain Res 70: 590-596.
29. Enatsu R, Bulacio J, Nair DR, Bingaman W, Najm I, et al. (2014) Posterior cingulate epilepsy: clinical and neurophysiological analysis. J Neurol Neurosurg Psychiatry 85: 44-50.
30. Guedj E, McGonigal A, Vaugier L, Mundler O, Bartolomei F (2012) Metabolic brain PET pattern underlying hyperkinetic seizures. Epilepsy Res 101: 237-245.
31. Guedj E, Aubert S, McGonigal A, Mundler O, Bartolomei F (2010) Déjà-vu in temporal lobe epilepsy: Metabolic pattern of cortical involvement in patients with normal brain MRI. Neuropsychologia 48: 2174-2181.
32. Masuda H, Shariff EM, Tohyama J, Murakami H, Kameyama S (2012) Clinical patterns and pathophysiology of hypermotor seizures: An ictal SPECT study. Epileptic Disord 14: 32-40.
33. Moers-Hornikx VMP, Vles JSH, Lim LW, Ayyildiz M, Kaplan S, et al. (2011) Periaqueductal grey stimulation induced panic-like behaviour is accompanied by deactivation of the deep cerebellar nuclei. Cerebellum 10: 61-69.
34. Habas C, Guillevin R, Abanou A (2010) In vivo structural and functional imaging of the human rubral and inferior olivary nuclei: A mini-review. Cerebellum 9: 167-173.
35. Goulding M (2009) Circuits controlling vertebrate locomotion: moving in a new direction. Nat Rev Neurosci 10: 507-518.
36. Gerasimenko Y, Gorodnichev R, Machueva E, Pivovarova E, Semyenov D, et al. (2010) Novel and direct access to the human locomotor spinal circuitry. J Neurosci 30: 3700-3708.
37. Gorodnichev RM, Pivovarova EA, Pukhov A, Moiseev SA, Savokhin AA, et al. (2012) Transcutaneous electrical stimulation of the spinal cord: non-invasive tool for activation of locomotor circuitry in human. Fiziol Cheloveka 38: 46-56.
38. Harkema SJ (2008) Plasticity of interneuronal networks of the functionally isolated human spinal cord. Brain Research Reviews 57: 255-264.