Hypersynchronous Slow Delta, Cyclic Alternating Pattern and Sleepwalking

Comment on Pilon M; Zadra A; Joncas S et al. Hypersynchronous delta waves and somnambulism: Brain topography and effect of sleep deprivation. SLEEP 2006;29(1):77-84.

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CHRONIC SLEEPWALKING IS AN ABNORMAL BEHAVIOR SEEN PREDOMINANTLY IN CHILDREN AND IN YOUNG ADULTS EFFECTING 2.5% OF THE GENERAL population 15 years and older of the United Kingdom.¹

Hypersynchronous slow delta (HSD) has been recognized as present in recordings of sleepwalkers since 1965.² It significance has been questioned as different authors have reported variable times of incidence preceding sleepwalking events. It was Zucconi et al³ who associated the presence of bursts of delta waves not only in sleepwalking but several parasomnias, including bruxism, with cyclic alternating pattern (CAP) analysis. This analysis of sleep EEG is not necessary well recognized or understood. For example, Pressman⁴ recently looked at delta bursts in different sleep disorders, and published a clear example of a CAP cycle and never mentioned the term or the possible need to analyze sleep using the CAP atlas when such bursts are seen.

Pillon et al. in this issue⁵ have performed a further-analysis of EEG data obtained from sleepwalkers at baseline and after sleep deprivation. They looked at bursts of high amplitude slow delta (>150µV HSW) during NREM sleep. These authors concluded that HSD shows a clear fronto-central gradient across all subjects, that is more evident following sleep deprivation. This is not too surprising considering prior publications on delta wave generation. But this is of interest considering prior publications on delta wave generation.⁶

Pillon et al⁵ bring up the issue of the relationship of their findings with those observed with CAP scoring, indicating that “HSD events may be viewed by some in relation to the CAP which expresses the organized complexity of arousal-related phasic events in NREM sleep. Specifically, one CAP subtype (A1) could overlap with HSD, as it is characterized by synchronized EEG patterns (e.g., delta bursts), by low delta power and a frontal predominance.” They refer to Ferri et al⁶ but dismiss the possibility that they essentially scored CAP. There is, however, much evidence today to refute this assertion and to indicate that they were indeed measuring CAP A1 events in sleepwalkers. Some of the rationales these authors used to dismiss the case for CAP are results of studies performed on normals rather than those with pathological sleep as in their own study; and normal and pathological sleep, as seen in sleepwalking, cannot be compared. Also Ferri et al⁷ using a 19 scalp site EEG recording demonstrated a centro-frontal gradient in CAP as reported in the investigation of HSD. Further analysis using the low resolution brain electromagnetic tomography functional imaging to investigate the source analysis of the frequency components of phase CAP-A, indicates that the generator seems to be localized mostly over the frontal midline cortex for A1 and for the low frequency elements of A2 subtypes. It is difficult to determine where the EEG delta wave generator is located even with sophisticated techniques and more work will be needed combining EEG recording during sleep with imaging studies to confirm it, but the current available data are concordant and the findings of Pillon et al⁵ are in the same direction independently of the name given to the bursts of delta waves.

The lack of recognition that bursts of HSD are part of an abnormal CAP rate has consequences: Similar to Pressman,⁴ Pillon et al⁵ deny any informative value to these bursts of HSD, and we disagree on this point.

Bursts of HSD during NREM sleep can be a normal phenomenon. As has well been described by Terzano et al,⁸⁹ there is normally during sleep, passage from unstable to stable sleep, associated with progressive recruitment of controlling neurons in the thalamus-forebrain region and development of hypersynchronization of firing that will lead to the appearance of delta waves in the scalp EEG. But these EEG delta bursts are a transient element and normally there is occurrence of a continuous slow frequency (and high amplitude in younger subjects) EEG pattern. Here we see that a bursting pattern is persisting even during stage 3 and 4 NREM sleep as well described with abnormal persistence of CAP. What is abnormal is not the burst of HSD, but the reappearance of the background activity that interrupts the persistence of the slow delta (and this feature is missed in the analysis reported by Pillon et al⁵).

Instead of denying value to these bursts,⁴⁵ one should determine if they are in the range expected for age and gender matched controls, and if they are not, to find out what is interrupting the normal progression of NREM sleep in the studied cycle. Robert et al⁴ have used such an approach to affirm that they had reached an appropriate nasal CPAP pressure in their sleep apnea patients after eliminating apnea and hypopnea: the EEG patterns used in their approach is based on the disappearance of bursts of delta waves (i.e. CAP).

Our study in adults sleepwalkers shows a prominence of CAP during the first 2 sleep cycles and an important decrease during the last cycles.¹¹ Interestingly the Montreal group and ourselves²³¹¹ have shown that despite presence of these bursts of HSD, there is an overall decrease in delta power during the first 2 NREM-REM cycles in chronic sleepwalkers, the difference between Gaudreau et al¹² and our¹¹ findings was that we did not reach the same level as normal controls in the last sleep cycle compared to their find-

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ings, but in both cases the differences were non significant. In fact these findings are in favor of the possible improvement of the homeostatic function of sleep with sleep deprivation, due to the lower delta power initially seen in sleepwalkers as found by Pilon et al.5

We agree with the finding that controlled sleep deprivation may improves the sleep of sleepwalkers: All night sleep deprivation in our sleep lab followed by day time EEG and nocturnal polysomnography, was standard for over 10 years: we never elicited a sleepwalking event in this secure environment, despite reports in same subjects that stressful sleep deprivation outside our laboratory (and with somewhat different sleep due to polysomnographic equipment) leads to increase in sleepwalking. We always had better sleep after sleep deprivation than the 1st night recording.

But the important question is why is it important to place these bursts of HSW in the context of CAP analysis: In the two different CAP analyses performed in children and adult sleepwalkers,11,14 we showed presence of abnormal CAP rates even on nights without sleepwalking events. Delta waves are the essence of CAP A1 and A2 subtypes.

We have emphasized that presence of abnormal amount of CAP with changes in phases A1 and A2, i.e. HSD bursts, indicated the presence of another subtle sleep disorder- mostly upper airway resistance syndrome.11,14 We specifically mention “subtle” sleep disorder as for years we had monitored sleepwalkers and yet missed the presence of UARS. With implementation of more sophisticated equipment during polysomnography (such as esophageal manometry), better clinical evaluation, and systematic search for clues, we have identified subjects with discrete breathing abnormalities during sleep.11,15,16 Since these adjustments, despite same referral base and referral only for chronic sleepwalking, we have not had a single adult patients out of the last 100 sleepwalkers without presence of an associated subtle sleep disorder, mostly UARS.

The questions for us are thus, what are the reported EEG changes related to, sleepwalking, or a continuous abnormality of NREM sleep related to a continuous and discrete nasal flow limitation? Are bursts of HSD just an indication of the difficulty of the subject to reach a normal delta sleep, and should we enter these bursts within the CAP count? Instead of denying them any value, should we not ask ourselves why they are there? Are they part of a pattern? If so, are they within the normal distribution of age-matched subjects? And should we look at all bursts of slow delta frequencies independently of their amplitude?

One of the problems of the recognition of delta bursts is that it is still often based on visual recognition, despite development of computerized analysis. The CAP system has one advantage: it considers a “phase B” that follows the delta burst, and thus raises the question of why the delta phase I was interrupted, a question never otherwise addressed. Studies in sleepwalkers,11,14 in UARS patients without sleepwalking,11,14 and the study of Thomas et al.10 of OSAS patients, show that an abnormal CAP rate is present. Bursts of delta do not need to be related to the beginning of sleepwalking as they are seen on non-sleepwalking nights, but their presence is indicative of pathological NREM sleep instability with which different parasomnias may be associated. It is a background for occurrence of the pathology and we cannot ignore valuable clues. We have shown that treating the subtle associated sleep disorder eliminated abnormal CAP rate associated HSD, and controlled sleepwalking.11,14,16 The performance of studies on HSD bursts is not where investigations should be aimed, the real question is to understand why the normal transition to a continuous delta state does not occur.

REFERENCES