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Editorial

Arousals and anti-arousals

This month's issue contains an intriguing re-examination of previously published datasets investigating cyclic alternating pattern (CAP) in electroencephalographic (EEG) activity. The 'Parma Group' continues its pioneering work in this area by comparing CAP [1] and American Sleep Disorders Association (ASDA) arousal scoring [2]. Having CAP and ASDA arousal data side-by-side is instructive, especially when two age groups of normal volunteers are considered.

A decade ago, the ASDA (now the American Academy of Sleep Medicine) appointed a committee to create and define a standard for scoring 'arousals'. In accordance with the traditional delineation between cortical activation as 'arousal' and autonomic sympathetic outflow as 'activation', the committee sought an EEG measure. Essentially, the search was for a quantifiable biological marker for sleep disturbance that was less than an awakening but more than an activation. The rules, published in 1992, focused on 'EEG speeding', typically manifest as increased alpha activity. What is not well known, however, is that the committee wrestled with the question of whether to include isolated 'delta-like' slow EEG bursts and K-complexes as indices of arousal. There were long discussions of K-alpha, parasomnia-related hypersynchronous slow wave sleep, and slow wave bursting in response to environmental disruptors (for example, sounds). Ultimately, ASDA arousal scoring addressed only the fast component and deferred calling the slow activity 'arousal' until more data were available. It was unclear whether the slow activity indicated a central nervous system arousal response or the brain's attempt to preserve sleep.

By contrast, CAP was not necessarily developed to quantify sleep disruption but rather to characterize sleep process. Thus CAP scoring could include the full spectrum of all possible phenomena associated with arousal, and went one step further by considering rhythm and periodicity. Data in this paper [1] clearly demonstrate ASDA and CAP A3 to be nearly isomorphic (95% scoring agreement). By contrast, CAP A1s essentially do not overlap with ASDA arousals (3%) and A2s fall somewhere in between (62% agreement). Furthermore, the changes as a function of age are commensurate with traditional staging trends. Slow waves decline while awakenings (and arousals) increase. Thus, a composite score for CAP A1–A3 form a *U*-shaped function. The authors do an excellent job of demonstrating this pattern quantitatively and highlighting ASDA arousal and CAP differences.

Regardless of whether the slow wave component of CAP is considered to reflect a disturbance or preservation of sleep, CAP sequences identify episodic sleep instability. As such, CAP conceptualizations fit well with neurophysiological sleep models. The slow component may indeed represent a human correlate of higher brain reinforcement of subcortical gating mechanisms; one would expect that occasional success in preserving sleep would thereby produce a CAP A1 and, at the other end of the spectrum, failed attempts to preserve sleep would result in a CAP A3 (or ASDA arousal). Younger subjects with more synchronizable EEG should predictably have more CAP A1 and older subjects with more fragile sleep should have more CAP A3.

The potential for CAP analysis has yet to be exploited. Factors that increase and decrease sleep stability are waiting to be explored. Alterations may stem from endogenous, exogenous, homeostatic, autonomic, or circadian factors. We know sleep is often destabilized by specific pathophysiologies. CAP, as a central mechanism, can help us understand variations in the presentation of periodic phenomena (for example, the missing leg movement in a sequence [3] or periodic K-alpha [4]). Finally, the recently published CAP Scoring Atlas [5] and the aggressive development of computerized scoring can be expected to make CAP scoring a sensitive, useful, and promising polysomnographic technique in the years to come.

References

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Max Hirshkowitz*
VAMC – Sleep Center 116A, 2002 Holcombe Boulevard, Houston, TX 77030, USA

* Tel.: +1-713-794-7562; fax: +1-713-794-7558.
E-mail address: maxh@bcm.tmc.edu (M. Hirshkowitz).