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# Are Microarousals Preceded by Electroencephalographic Slow Wave Synchronization Precursors of Confusional Awakenings?

P. Halász, J. Ujszászi, and J. Gádoros

*Department of Neurology, Postgraduate Medical School, and National Institute for Nervous and Mental Diseases, Budapest, Hungary*

**Summary:** The number of microarousals preceded by electroencephalographic (EEG) slow wave synchronization (MAS) and the number not preceded by EEG slow wave synchronization (K-complexes and/or delta groups) (MA) were analyzed during the first night of sleep in nine young patients with somnambulism and/or sleep terrors and in eight age- and sex-matched controls. While MAs peaked in REM and intermediate sleep, MASs appeared as a phenomenon of NREM sleep, peaking in stage 2. The number of MASs was significantly greater in all stages of NREM sleep in the patient group, but number and distribution of MAs did not differ between the two groups. In the patient group, the MASs occurred in slow wave sleep (stages 3–4 of each sleep cycle); in controls, MASs occurred infrequently. MASs were frequently associated with automatic chewing movements. The higher frequency of microarousals in the patient group did not result in an increase in time awake during the night. The increase in number of microarousals supports Broughton's hypothesis of the presence of some "arousal disorder" in somnambulism and/or sleep terrors. MASs may be predictive markers of ensuing confusional awakenings. **Key Words:** Microarousals—Sleep electroencephalogram—Somnambulism—Night terror.

In 1971 Schieber et al. (1) described spontaneous phasic arousal episodes under the name "les phases d'activation transitoire spontanées." The Strasbourg and San Diego sleep research groups (2–8) have described several physiological features of these transient activations.

These spontaneous microarousals are similar to the arousals elicited by external stimuli. The frequency of their occurrence is inversely proportional to the order of the stages of NREM sleep. They occur more frequently in stages 1–2 than in stages 3–4 NREM sleep. The frequency during REM sleep is comparable to the frequency observed in stage 1 of NREM. The frequency during REM sleep increases from evening to morning.

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Address correspondence and reprint requests to Prof. P. Halász at Department of Neurology, Postgraduate Medical School, Vöröshadsereg u. 116. H-1021. Budapest, Hungary.

**TABLE 1.** *Total sleep times*

| Sleep time (min) | |
|---|---|
| Patients[a] (n = 9) | Controls[b] (n = 8) |
| 486 | 419 |
| 522 | 390 |
| 371 | 430 |
| 468 | 456 |
| 432 | 382 |
| 405 | 407 |
| 336 | 379 |
| 397 | 316 |
| 354 | |

[a] $\bar{x}$ = 419, SD = 63.
[b] $\bar{x}$ = 397, SD = 41.

Previously, we analyzed (9–11) the obvious connection of phasic arousals with sleep cyclicity and detailed the presence of phasic activation in the ascending slope of cycles. We suggested that microarousals (MAs) be considered a standard measure of the level of arousal functions during sleep.

This article explores the characteristics of such phasic activation events in a group of patients with "disorders of arousal." We were surprised that little effort had been made to confirm Broughton's hypothesis (12) that attacks of somnambulism, enuresis nocturna, and pavor nocturnus or night terror "are best considered disorders of arousal and that the slow wave sleep arousal episode which sets the stage for these attacks is a normal cyclic event (12)." We decided to compare the sleep microstructure of patients suffering from somnambulism and/or sleep terrors to that of normal controls, with special reference to the frequency of phasic activation events.

Our results are in agreement with those of Naitoh et al. (8), i.e., that K complexes not only occur more frequently in the period before phasic arousals but also immediately precede them, thereby forming a close relationship with certain arousals, as seen in Fig. 4 of ref. 8. We have called this constellation a microarousal preceded by slow waves (MAS). Such events can be regarded as overlapping or identical with the K-α phenomenon of Raynal et al. (13). Based on careful study of phasic events in the sleep EEG, our group has elaborated some ideas about the dynamic relationship between phasic slow wave synchronization (K complexes, delta groups) and microarousals. In this conceptual framework, slow wave synchronization events are believed to represent sleep-protecting microshifts after transient shifts toward arousal, and microarousals are interpreted as transient breakthroughs of the arousal process against sleep-protecting forces. Therefore, the MAS is a possible candidate for a microevent modeling "awakenings accompanied by EEG signs of sleep," i.e., confusional awakenings.

## METHODS

Nine adults, age range 15–35 years (mean 23.4 years), suffering from somnambulism and/or night terrors were investigated during the first night of sleep in the sleep laboratory. They kept their usual bedtimes and their total sleep times are shown in Table 1. All patients experienced more than one confusional awakening per week with automatic behavior of variable duration. The following variables were monitored polygraphically: 8–10 channel

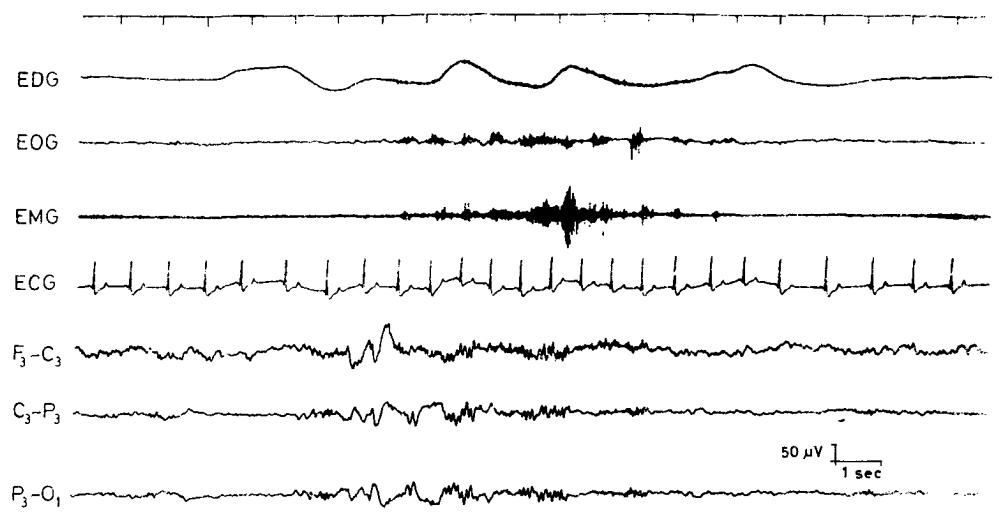


**FIG. 1.** Typical microarousal with slow wave synchronization in stage 2 sleep with transitory increase of pulse rate, electrodermal discharge, and chewing automatism. EDG, electrodermography; EOG, electro-oculography; EMG, electromyography; ECG, electrocardiography.

electroencephalogram, submental electromyogram, electro-oculogram, electrodermogram of the palm-to-forearm region, and electrocardiogram. Simultaneous video monitoring was also performed. A control group of eight age- and sex-matched healthy subjects underwent the same procedure.

The sleep records were visually analyzed for microarousals with synchronization (MASs) and those without synchronization (MAs). Sleep stages were scored according to the manual of Rechtschaffen and Kales (14), and intermediate stages, using the criteria of Lairy et al. (15). Sleep cycles, scored using the criteria of Feinberg (16), were divided into four parts: (a) descending slopes, estimated from the end of the awake state in the first cycle or the end of the previous REM stage to the beginning of the deepest NREM stage in the cycle; (b) trough, formed by the deepest NREM stage of the cycle (i.e., if stage 3 occurred more than once in the cycle, then the more superficial stages such as stage 2, bounded by the deepest stages such as stage 3, were also considered under this heading); (c) the ascending slope, estimated from the end of the last deepest stage to the next REM stage; and (d) the top of the cycle, usually an REM stage or REM stages interrupted by short intermediate and/or awake periods.

The criteria for phasic microarousals were those defined by Schieber et al. (1): (a) accelerated frequency of ongoing activity with decreased amplitudes; (b) transitional disappearance of sleep spindles and delta waves in slow wave sleep; (c) transitional disappearance of eye movements in REM sleep; (d) transitional appearance of awake EEG activity in the form of alpha spindles and occasionally of betas; (e) increase of EMG activity simultaneously with changes in EEG; (f) appearance of body movements either as elementary movements of the trunk or the limbs or as more complex movements, such as changes in posture; and (g) acceleration of cardiac rhythm.

Microarousals were scored as MASs when they did not intrude longer than 1 s and were preceded by a K complex, multiple K complexes, or a delta burst that could be clearly differentiated from background activity (Fig. 1).

We used the Student's two-tailed $t$ test for correlated means ($p \leq 0.05$).

**TABLE 2.** *Percentages of time spent in sleep stages in control and patient groups*

| Stage | Controls | | Patients | |
|---|---|---|---|---|
| | $\bar{x}$ | SD | $\bar{x}$ | SD |
| REM | 13.5 | 3.5 | 16.5 | 3.5 |
| 1-Int. | 10.7 | 4.1 | 9.9 | 3.7 |
| 2 | 47.6 | 7.1 | 48.8 | 6.3 |
| 3 | 11.5 | 4.8 | 14.8 | 3.7 |
| 4 | 13.8 | 5.2 | 8.9 | 5.3 |
| Aw | 2.9 | 1.7 | 1.1 | 1.3 |
| Total | 100.0 | | 100.0 | |

Aw, awake; Int., intermediate sleep.

## RESULTS

The distributions of sleep stages in the patient and control groups (Table 2) were not significantly different.

The number of microarousals (Table 3) was significantly larger in the patient group than in the controls. This could be attributed to an elevation in the number of MASs; the number of MAs did not differ significantly between the two groups (Fig. 2).

The number of phasic arousals (MAs and MASs) in the control group and their distribution according to sleep stage agreed with the data of Schieber et al. (1), Ehrhart and Muzet (5), and Halász et al. (11), supporting the concept that transient arousal can and should be accepted as a standard sleep measure.

Chewing movements were associated with MASs (Fig. 1). Although their appearance in sleep is common knowledge, the connection with MAS has not been described before.

The distribution of MAs and MASs according to sleep stage was similar in the control and patient groups: MASs peaked in NREM sleep, most prominently in stage 2, whereas MAs occurred predominantly in stage 1 intermediate sleep and in REM (Fig. 3). The only difference between the two groups was the significantly higher frequency of MASs in all of the NREM sleep stages in patients. The distribution of MASs from cycle to cycle was the same in the two groups, with the greatest number in the middle part of the night. The patient group had more MASs in all cycles of sleep (Fig. 4).

**TABLE 3.** *Number of microarousals per minute in different sleep stages*

| Stage | Controls | | | | Patients | | | |
|---|---|---|---|---|---|---|---|---|
| | MAs/min | | MASs/min | | MAs/min | | MASs/min | |
| | $\bar{x}$ | SD | $\bar{x}$ | SD | $\bar{x}$ | SD | $\bar{x}$ | SD |
| REM | 0.21 | 0.10 | 0.01 | 0.02 | 0.20 | 0.08 | 0.00 | 0.00 |
| 1-Int. | 0.22 | 0.13 | 0.01 | 0.03 | 0.25 | 0.21 | 0.03 | 0.03 |
| 2 | 0.07 | 0.03 | 0.05 | 0.04 | 0.10 | 0.10 | 0.19 | 0.08 |
| 3 | 0.07 | 0.09 | 0.02 | 0.03 | 0.01 | 0.02 | 0.09 | 0.06 |
| 4 | 0.06 | 0.05 | 0.02 | 0.02 | 0.05 | 0.06 | 0.05 | 0.04 |

Int., Intermediate sleep; MA, microarousal without slow wave synchronization; MAS, microarousal with slow wave synchronization.

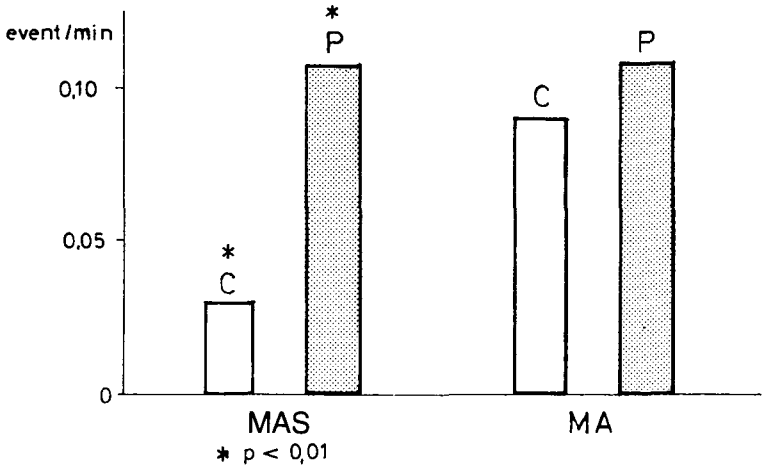


**FIG. 2.** Frequency of phasic arousals with (MAS) and without (MA) slow wave synchronization in event-per-minute-of-night means of the patient (P) and control (C) groups.

When analyzed according to distribution within sleep cycles, the MAs were least frequent in the descending slopes of cycles, somewhat more frequent in the troughs of cycles (due obviously to MAs at the turning points of cycles, i.e., at the beginnings of ascending slopes, considerably more frequent in the ascending slopes), and most frequent in REM and intermediate stages. MAS frequencies were highest in the ascending slopes (Fig. 5); in other parts of cycles their frequencies were low. The patient group had more MASs in the descending slopes and in the troughs of cycles, that is, in the deepening phases of the cycles, than did the controls. This greater number of MASs was not associated with more

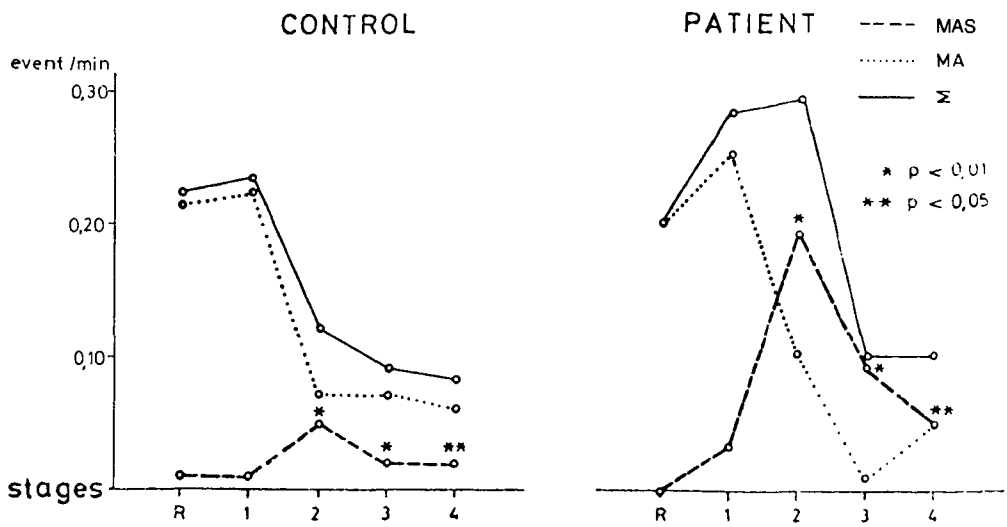


**FIG. 3.** Frequencies of phasic arousals in different stages of sleep in patients and controls. R, REM; 1, 2, 3, and 4, stages of NREM sleep. Other abbreviations as in Fig. 2. The Student's two-tailed *t* test was performed for comparison of values for patient and control groups at each stage of sleep. The solid line is the sum of the two lower curves.

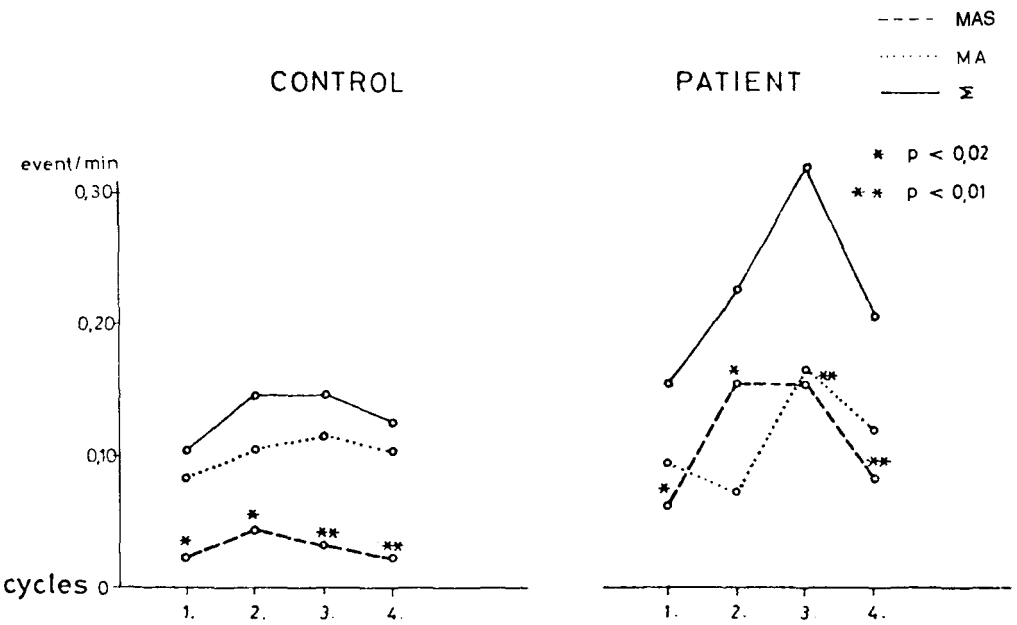


**FIG. 4.** Distribution of phasic arousals from cycle to cycle through the night in the two groups. Student's two-tailed *t* test was performed for comparison between patient and control groups values at each cycle of sleep. Abbreviations and conventions as in Fig. 3.

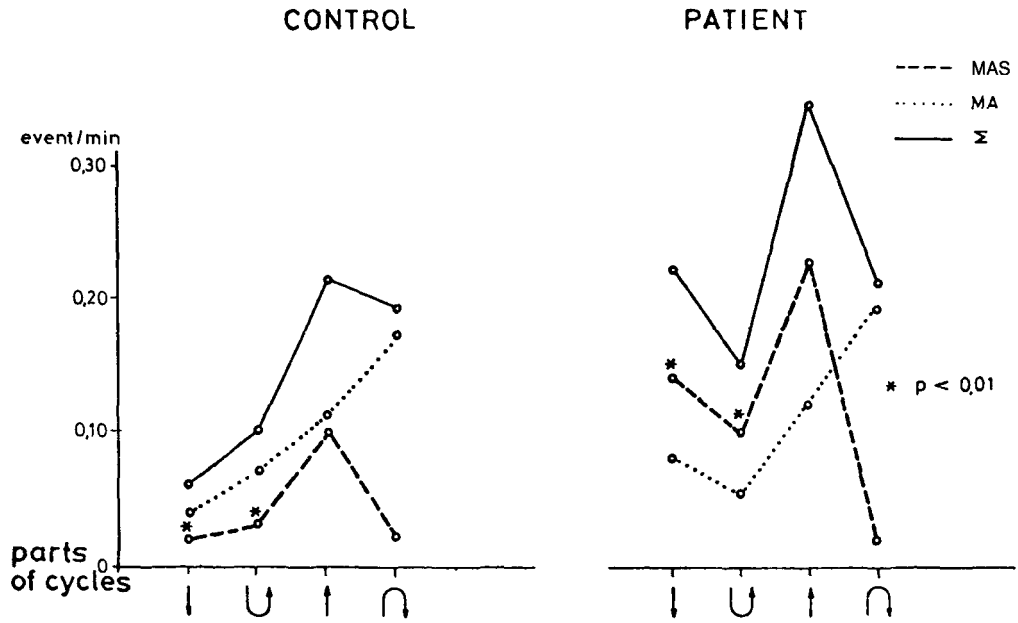


**FIG. 5.** Distribution of phasic arousals among the parts of sleep cycles. Student's two-tailed *t* test was performed for comparison between values for patient and control groups at each part of the cycle. An upward arrow represents ascending slopes of sleep cycles; an arrow curving down, the tops of cycles; a downward arrow, descending slopes of cycles; and an arrow curving upward, troughs of cycles.

awakenings. The differences between the patient and control groups were found only in the MAS values, not in the MA values.

## DISCUSSION

The MAS type of phasic arousal occurred in greater numbers in NREM sleep of patients with confusional awakenings than in matched controls. Although their distribution did not differ from that of the controls, this kind of activation invaded slow wave sleep (stages 3–4 NREM), where their frequency is normally low.

These findings appear to confirm the existence of some kind of arousal disorder, as proposed by Broughton (12), in our patients. However, important limitations on this study are set by the possible first-night effect. It is possible that the differences observed between the patient and control groups reflect differences only in adaptation to sleep under the new, sleep laboratory conditions. If so, this would mean that patients with histories of confusional awakenings react with more arousals in NREM in response to environmental changes than do controls.

The relevance of these results to the mechanism of pathological confusional awakenings is that the presence of MASs cannot be considered a pathological sign, because confusional awakenings can occur in healthy persons as well (17). Only their occurrence in very high numbers or their persistence into adulthood can be considered a disorder.

Raynal et al. (13) found the K-α phenomenon to be more frequent in the various forms of sleep disruption. Our findings, therefore, could be nonspecific and reflect only a disruptive component arising from either internal or external sources of activation. However, the same pattern of slow wave activity followed by arousal has been described in another group of patients subjected to confusional awakenings (18).

Mainly during slow wave sleep (stages 3–4), especially in the first cycle, increasing sleep pressure must counterbalance the phasic arousals. We are, therefore, inclined to interpret the MAS phenomenon as a phasic arousal occurring in the presence of sleep-protecting tendencies represented by slow waves. Increased phasic activation in the form of MASs, therefore, cannot be considered a pure agent of the psychological awakening process, but rather a precursor of confusional awakening. This interpretation is supported by the fact that an increase in the number of MASs did not result in an increase in time awake during the night.

We believe that the study of sleep microstructure, with special reference to the dynamics of phasic arousals, provides insight into the physiopathogenesis of arousal disorders. The association between a high frequency of MASs and fragments of automatic behavior seen in somnambulism and night terrors merits attention. Oral automatism in the form of chewing movements could be a part of MASs. MASs could also be regarded as representing the liberation of a microautomatism, a counterpart of the other automatisms that characterize all forms of confusional awakenings.

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