SLEEPENING: THE PROCESS OF FALLING ASLEEP

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The process of falling asleep has not received much attention in recent years from physiologists or psychologists. The fact that there isn't a word in the English language for this process may be part of this neglect. I propose to use the term sleepening. I want to review the important features of sleepening, with a plea for further investigation.

The relaxation phase of sleepening begins with behavioural modifications under voluntary control, such as the decision at a particular time to seek and lie down in a physically and psychologically "comfortable" environment (i.e. warm, dark, quiet and non-threatening). Voluntary movements are then inhibited with relaxation, particularly of postural muscles, and closure of the eye The next stage of sleepening involves a complex series of physiological and psychological changes with altered perceptual and cognitive function and less focused attention. Heart rate and blood pressure fall, skin temperature rises by vasolitation and sweating increases, apart from on the palms and soles where sweating is Core temperature falls and cortisol secretion is inhibited. inhibited temporarily, regardless of what phase of the circadian rhythm sleepening occurs. The pupil constricts and oculomotor control is reduced, with slow, non-conjugate eye movements occurring for a while before ocular quiescence. The auditory, pain, and other sensory thresholds are then increased progressively by active inhibition of sensory pathways in the thalamus.

Alpha waves in the EEG of the relaxed state are blocked quite suddenly as sleepening progresses. There is also a progressive decrease in the occurrence of beta waves, slowing in the frequency of theta waves and the appearance of low-amplitude delta waves in Stage 1 sleep. Vertex sharp waves and a burst for 2 or 3 seconds of high-amplitude theta waves (saw-tooth waves) often precede the first K-complex and spindle of Stage 2 sleep. Sleepening may end here or, more likely, continue for a few minutes until sleep is more established. The cyclical variations of NREM and REM sleep stages then proceed, probably involving different neuronal systems from that of sleepening. The inhibitory processes, begun during sleepening, are maintained and augmented.

Consistencies and variations in the sleepening of different subjects are clearly seen in the graphical summaries of the EEG, EOG and EMG produced by computer analysis at Epworth Hospital. These provide a means of studying sleepening and its failure in insomnia.