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Interaction Between Sleep and Thermoregulation: An Aspect of the Control of Behavioral States

Pier Luigi Parmeggiani

Istituto di Fisiologia umana dell'Università di Bologna, Bologna, Italy

Summary: Studies on the interaction between sleep and thermoregulation are reviewed with regard to the processes underlying the ultradian evolution of behavioral states. The experimental evidence shows that thermoregulatory mechanisms influence the waking-sleeping cycle in both the absence or the presence of a thermal load. Such a control appears to be a functional necessity to maintain physiological homeostasis. **Key Words:** Thermoregulation—Behavioral states—Waking-sleeping cycle.

The relationship between the behavioral states of waking and sleeping and the circadian oscillations of body temperature has been known for a long time (1–5). During the last two decades, moreover, the study of the physiological interactions between waking-sleeping and thermoregulatory processes in several species has shown that much more than a temporal concomitance relates such behavioral events to body temperature changes (6–9). From a theoretical viewpoint, an important result of this study is also the demonstration that the regulation paradigm of physiological functions changes across behavioral states (10). The dominance of a general—that is, state-dependent—regulation over specific effector controls may be seen as a necessary expression of the integrative function of the nervous system. In this respect, however, a problem is raised by desynchronized sleep (DS or REM), for this sleep stage in several species is characterized by a dramatic impairment of homeostatic regulation (11). A good example is the suppression or depression of thermoregulatory effector responses such as shivering, panting, vasomotion, and sweating (6–9). Moreover, a loss of thermosensitivity in the majority of anterior hypothalamic–preoptic (AH-PO) neurons occurs in DS (Fig. 1) with respect to wakefulness (W) and synchronized sleep (SS or NREM) in both the cat (12,13) and the kangaroo rat (14). These results show that state-dependent processes override the basic functional organization of thermal ho-

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Address correspondence and reprint requests to Prof. P. L. Parmeggiani at Istituto di Fisiologia umana, Piazza Porta San Donato, 2, 40127 Bologna, Italy.

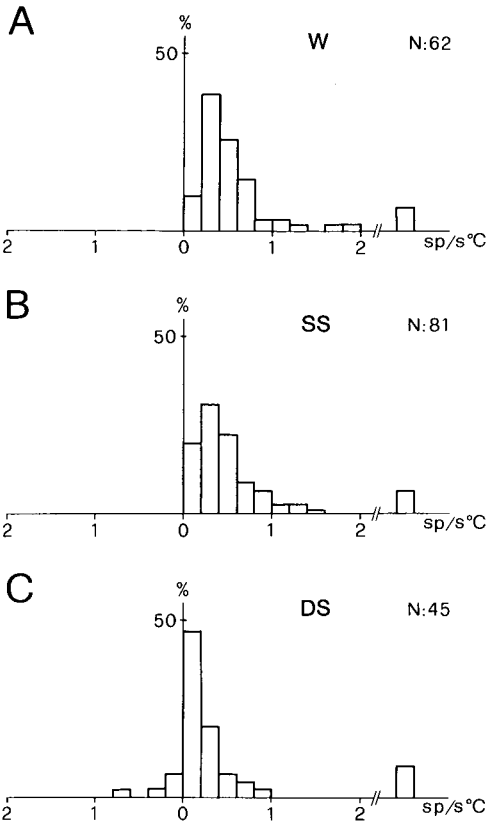

FIG. 1. Relative frequency distributions of the thermosensitivity (absolute values in spike/second and degree Celsius) of AH-PO neurons in wakefulness (A, W), synchronized sleep (B, SS), and desynchronized sleep (C, DS). The relative frequency of thermosensitivity above 2 sp/s°C is represented by the histogram on the right end of the abscissa. N, number of neurons. In W and SS the distributions are similarly shaped. In contrast, DS is characterized by the quantitative prevalence of small-value classes of thermosensitivity and by the encroaching of the distribution upon the prohibited left quadrant. This shows that some neurons display a thermal modality (cold- or warm-sensitivity), which is the opposite of that shown in both W and SS (right quadrant). (Data from 13.)

meostasis. Likewise, the paradigm of homeostatic regulation is also challenged by DS processes in the domains of circulation and respiration (15–17).

The evidence of state-dependent regulation paradigms is consistent with the concept of predictive homeostasis (18). Predictive homeostasis entails functional changes controlled by the circadian timing system. Such an anticipatory adjustment to periodic qualitative and quantitative changes in ambient challenges improves the effectiveness of physiological mechanisms and decreases energy expenditures. The concept of predictive homeostasis applies well to the somatic and autonomic events of W and SS; in other words, the general regulation paradigms perfectly meet the functional requirements of the organism during these behavioral states (homeostatic operation modes) (11). In contrast, DS events cannot be reduced to such conceptual terms, although the impairment of homeostatic regulation also points out the dominance of state-dependent processes in this case. It is difficult, however, to infer a general regulation paradigm of DS from the functional events of this sleep stage (poikilostatic operation mode) (11).

DS is well characterized in only mammals and birds, which have developed efficient homeostatic mechanisms in association with the organization of clear-cut behavioral states. In terms of predictive homeostasis, such an organization entails the necessity of a controlled evolution of behavioral states, and particularly of DS. Since ambient and body temperatures are variables deeply affecting physiological and biochemical processes, the control of the ultradian waking-sleeping cycle—that is, of the single sequence of W, SS, and DS episodes—ought also to be dependent on thermoregulation.

INFLUENCE OF AMBIENT TEMPERATURE ON SLEEP

In the last two decades, investigations have been carried out in animals and humans to clarify the influence of different ambient temperatures on the waking-sleeping cycle. In these studies, the structure of the cycle at ambient temperatures close to (room temperature) or within thermoneutrality was conventionally considered as the normal reference. This article will show that the relationship of the "normal" waking-sleeping cycle with thermoneutrality is based on a more cogent reason than a mere convention. Thermoneutrality, which varies in the different species (19), is defined as the range of ambient temperatures within which the metabolic rate decreases to the minimum at rest and thermoregulation is actuated by physical mechanisms alone. Long-term adaptation to ambient temperatures without the limits of thermoneutrality induces shifts and/or modifications of the ambient temperature range well tolerated by the different species in terms of normal waking-sleeping patterns. Moreover, the amount of thermal load, which is positive and negative with respect to the body above and below thermoneutrality, respectively, does not depend only on ambient temperature and humidity. Several other factors (body size, thermal insulation, age, sexual cycle, feeding, season, etc.) consistently influence individual thermoregulatory responses in every ambient condition. Regional differences in body sensitivity to temperature should also be considered. Particularly, thermal comfort temperatures of airways and skin regions are different. The set of interacting variables is so complex that further investigations are necessary to dissipate some confusion in this matter and improve our quantitative knowledge. At any rate, the present experimental evidence shows that thermal loads elicit not only an increase in waking time but also an alteration in sleep structure, eventually bringing about total or selective sleep deprivation.

In nonadapted cats, the waking time increases as a function of the thermal load (20,21). Negative thermal loads are better tolerated than positive ones by these animals. Actually, the negative load can be experimentally graded within a range of >40°C, whereas sleep almost abruptly turns into maintained waking only a few degrees (~8°C) above the upper limit of thermoneutrality (27°C) (19). The prolongation of waking time elicited by increasing thermal loads is associated with important changes in the sleep structure. The duration of spindle sleep (NREM stage 2 sleep in humans) is scarcely modified, whereas that of slow wave sleep (NREM stages 3 and 4 sleep) at first increases and then decreases as a function of the thermal load. Such changes are less evident when spindle and slow wave sleep are not separately considered (21). In contrast, DS is only depressed by the increasing thermal load, being the slope of the decrease much steeper above than below thermoneutrality. In the cat, therefore, W and DS show reciprocal changes in response to thermal influences. This fact is of considerable importance, for the outcome of SS evolution towards W or DS, as a result of thermal influences, is specifically related to the control of the sleep structure by AH-PO thermoregulatory mechanisms.

The changes in the waking-sleeping patterns induced by thermal loads were also studied in the rat and the golden hamster with particular regard to the effects of adaptation to low or high ambient temperatures (22–25). The results are consistent with those obtained in the cat, except for the differences depending on both the species (body size, physiological characteristics, thermoneutral range, etc.) and the experimental conditions. Interesting from the viewpoint of the interaction between sleep and thermoregulation is the result that sleep time peaks at ambient temperatures around the upper limit of thermoneutrality (22–26). A positive correlation between DS total duration and ambient temperature is evident within the thermoneutral range in the rat; in other words, DS time increases from the lower to the upper limiting temperature (27). The relationship between DS processes and the thermoneutral range is also pointed out by the fact that in rats, maximal DS time defines a narrower thermoneutral range than does minimal metabolic rate (27).

In conclusion, the experimental data show that sleep time peaks at thermoneutrality, wherever the placement of thermoneutrality may be in the temperature continuum, depending on the species and the adaptation. Sleep time declines above and below the thermoneutrality range as a function of ambient temperature. The rate of decline is smaller on decreasing than on increasing ambient temperature. A negative correlation exists between W and DS. In contrast, the changes in slow wave sleep are more complex; its time may even increase under moderate thermal loads. This phenomenon is a result of the interaction between sleep and thermoregulation at the AH-PO level (28).

The waking-sleeping cycle in humans is also affected by ambient temperature (29–40). Deviations of ambient temperature from thermoneutrality increase the waking time and modify the structure of sleep. In particular, the frequency and duration of NREM stages 2, 3, and 4 sleep, and/or of REM episodes, may be selectively affected. As in the cat (21), a mild negative thermal load may increase the total duration of NREM stage 4 sleep (40). Differences in experimental conditions underlie fairly variable results. Moreover, thermal comfort or stress are weighty subjective factors, which consistently influence the waking-sleeping cycle in the civilized human being. At any rate, cold disrupts the structure of sleep more than heat (36).

The possibility that temperature effects on the waking-sleeping cycle may be the result only of an unspecific stress has been suggested for humans (34,36) and rats (41). This hypothesis correctly applies to the effects of thermal loads inducing W and total sleep deprivation. However, selective increases and decreases in frequency and duration or suppression of single sleep stages are changes in the structure of sleep also elicited by thermal influences on AH-PO thermoregulatory mechanisms, which control the evolution of the ultradian waking-sleeping cycle.

INFLUENCE OF THERMOREGULATORY MECHANISMS ON SLEEP

The onset of SS in the cat is characterized by a decrease in AH-PO temperature with respect to W (28). The amount of such a decrease at the end of the SS episode is related to both ambient temperature and the occurrence of either DS or W. When SS is followed by DS, the decrease of AH-PO temperature is greater at low (0°C) than at room (20°C) temperatures. When SS is followed by W, instead, the decrease is the same at both ambient temperatures. These results demonstrate that the decrease in AH-PO temperature during SS is dependent on sleep processes also affecting AH-PO thermoregulatory mechanisms so as to induce heat loss by decreasing the set temperature of

the thermostat regardless of ambient temperature. In the absence of a thermal load—that is, at thermoneutrality—heat loss is easily achieved, since sleep and thermal influences do not interact at the AH-PO level. In this case, the probability of DS occurrence is related only to the ultradian need of DS and to the circadian rhythm of body temperature (42). Under a thermal load, however, the probability of DS occurrence—that is, the normal evolution of the ultradian waking-sleeping cycle—is determined not only by the DS need but also by the effect of such a load on AH-PO thermoregulatory mechanisms. In other words, the sleep-specific adjustment of thermoregulation inducing heat loss (thermolysis) may be supported or counteracted by central or peripheral thermal influences. The outcome of this interaction, occurring during SS, is eventually DS or W.

In conclusion, the decrease in AH-PO temperature during SS indicates the active control of thermoregulatory mechanisms over the evolution of sleep stages according to the prevailing needs of thermal or sleep homeostasis. The restricted range of AH-PO temperatures, which is compatible with the occurrence of DS, the behavioral state characterized by an impaired thermoregulation, may be considered as a DS temperature "gate" controlled by AH-PO thermoregulatory mechanisms (28). Such a gate is less constraining to DS occurrence at 20°C than at 0°C ambient temperature (Fig. 2).

The previous results and conclusions are consistent with studies showing that experimentally induced changes in AH-PO temperature affect the waking-sleeping cycle. Cooling increases waking time (26). and warming promotes both SS and DS (26,42–46). As a matter of fact, the thermoregulatory activity elicited by central cooling is the opposite of the thermolytic adjustment of the AH-PO thermostat induced by SS processes. On the contrary, central warming elicits a change in thermoregulatory activity, which is synergic of SS influences on AH-PO structures (Fig. 3). SS processes are enhanced by the latter functional condition. In conclusion, the effect of small central thermal loads is specific at thermoneutrality, since cooling promotes wakefulness and warming promotes sleep. Heavy positive or negative thermal loads, however, exert only unspecific arousing influences.

The identification of warming with sleep and of cooling with wakefulness is possible

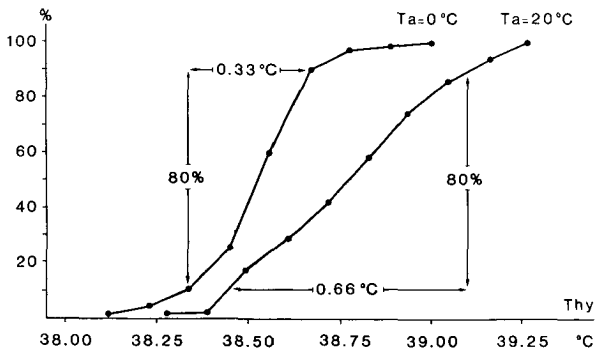

FIG. 2. Cumulative relative frequency distributions of DS episodes with respect to AH-PO temperature (Thy) at the end of preceding SS at 20°C and 0°C ambient temperatures (Ta). The left shift of the distribution at low ambient temperature shows the thermolytic (heat loss) adjustment of the AH-PO thermostat during SS. This thermoregulatory adjustment induces variable AH-PO cooling according to ambient temperature. The AH-PO temperature gate of DS occurrence is narrower at 0°C than at 20°C ambient temperature (0.33 vs. 0.66°C, respectively), and therefore, the probability of such an occurrence is reduced, apart from DS pressure. (Modified from 28.)

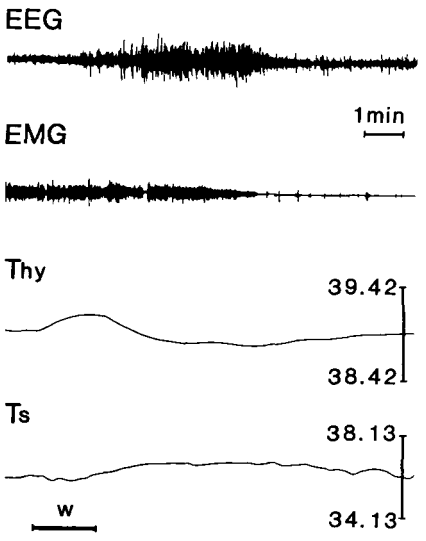

FIG. 3. Sleep-promoting influence of AH-PO warming. The rise in the AH-PO temperature (Thy) elicits an increase in the ear pinna temperature (Ts), which is indicative of the central thermostat adjustment inducing heat loss. As a result, Thy decreases steeply after the end of warming (w) in concomitance with EEG synchronization. A DS episode ensues only when Thy reaches the lowest level in SS. Ambient temperature, 25°C. (Unpublished observation, 13.)

also in respect to the influence of peripheral thermal loads on the waking-sleeping cycle. In this case, however, unspecific effects of thermal stimuli, related to the amount of thermal comfort or stress, may more readily affect the waking-sleeping cycle independently of thermoregulatory mechanisms.

CONCLUSIONS

The problem of the interaction between sleep and thermoregulation has been approached in this article only from the viewpoint of sleep physiology. Therefore, the main issue to be addressed is whether the influence of ambient temperature on the waking-sleeping cycle is nonspecific (comfort or stress) or specific (involvement of thermoregulatory mechanisms also at thermal neutrality).

Thermal stimuli, for instance, nonspecifically affect the cycle by influences on encephalic mechanisms controlling behavioral states, as many other sensory modalities do (47). However, the experimental evidence shows that this is not the only effect that ought to be considered. More subtle changes in the waking-sleeping cycle are observed than an all-or-none increase in waking or sleeping time as a response to thermal stress or comfort. Selective depression or enhancement of sleep phenomenology, and changes in the duration and frequency of SS or DS stages, entail the existence of specific alterations in the mechanisms controlling the evolution of the waking-sleeping cycle.

In conclusion, the effects of thermal loads ought to be considered as specific whenever thermoregulatory mechanisms are involved. From this point of view, it is convenient to consider briefly the thermoregulatory effector responses, since the underlying somatic and vegetative processes may be consistent or inconsistent with a normal evolution of the waking-sleeping cycle.

Thermoregulatory responses to thermal loads may be classified as behavioral (posture and motility changes) and physiological (vasomotion, piloerection, shivering, metabolic heat production, sweating, tachypnea).

The full expression of behavioral thermoregulation implies the waking state. Manifestations of behavioral thermoregulation, as the changing sleep posture at different ambient temperatures, are observed in SS. DS, in contrast, is characterized by a stereotyped muscle hypotonia or atonia, which is unrelated to the thermal load. Concerning physiological thermoregulatory responses, changes in threshold and gain of the regulator are observed in the transition from W to SS. Such changes are specifically related to sleep processes. Most remarkable during DS is the disappearance of the physiological responses (vasomotion, shivering, tachypnea), which are driven by AH-PO mechanisms in furry animals (6–9). Recent studies also show that the depression of sweating in humans depends on a decrease of the central drive rather than on peripheral mechanisms (48–50).

At any rate, it is still uncertain, for lack of experimental evidence, whether humans fully conform to the model of thermoregulatory changes in sleep that is based on studies carried on in small furry mammals. With this restriction, however, it is possible to conclude that only W complies perfectly with the requirements of thermally stressful conditions. At less demanding ambient temperatures, thermoregulation and SS processes are not mutually exclusive, although their interaction affects DS occurrence. In contrast, the functional state of DS implies that thermoregulation as a teleologically integrated mechanism is temporarily inactivated. Heavy thermal loads (cold or warm) elicit arousal in every sleep state. This stereotyped effect is unspecific, but it is, nevertheless, biologically adequate. It underlies the reestablishment of a functional state, wakefulness, enabling the organism to optimize thermoregulation by means of the whole behavioral and physiological repertory of the species.

At this point, a distinction between sleep-promoting and sleep-regulating influences of ambient temperature is appropriate. During W, thermal loads may induce or oppose the occurrence of sleep, depending on whether sensory influences (EEG synchronizing or desynchronizing) (47) and thermoregulatory somatic and autonomic activities (posture, skin vasomotion, metabolic rate, circulatory and respiratory activity) are consistent or inconsistent with sleep processes. Thus, a moderately warm ambient temperature better promotes sleep than a cold one. In this case the onset of sleep may be considered as a synergic concomitant of thermoregulation in the adaptation to a positive thermal load (25,26,43–45,51). However, since sleep may also occur under adverse ambient conditions, the sleep-promoting role of thermoregulatory structures is only facultative in W, and its functional importance is inversely related to the degree of sleep pressure. In contrast, during SS, thermoregulatory structures temporarily become an essential part of the mechanisms regulating the further evolution of sleep. The changes in AH-PO temperature during SS are consistent indicators of such a regulation. The AH-PO temperature gate of DS episodes shows that DS occurrence depends on a specific functional level—that is, on a thermolytic adjustment—of the AH-PO thermostat. The loss of AH-PO neuron thermosensitivity during DS (12–14) is, therefore, a controlled event. The sleep-regulating function of AH-PO structures during SS is progressively overshadowed by their thermoregulatory responses to increasing thermal loads. As a result, the duration of SS may also be prolonged in comparison with total sleep time (20,21,26,40), and arousal instead of DS may eventually occur, since thermal stimuli also activate arousal mechanisms. In such conditions, DS is de-

pressed or suppressed for long periods until the accumulation of an increasing DS debt produces a sufficient DS pressure to overwhelm the AH-PO thermoregulatory drive (20,21,42). In this case, DS brain stem effector mechanisms literally escape from the normal AH-PO control.

The previous considerations concerning the AH-PO control of the ultradian waking-sleeping cycle also apply to sleep occurrence in relation to the circadian rhythm of body temperature in the absence of thermal loads. Given a need for sleep, SS duration varies inversely as the ease with which the AH-PO thermostat may be adjusted to induce the somatic and autonomic functional changes underlying heat loss. On this basis, DS probability is obviously greater around the minimum than around the maximum of the circadian oscillation of body temperature, taken as a reference of the functional conditions of the AH-PO thermostat. As a result, SS shows variable durations and may be followed by either a DS or an arousal episode, according to the phase of the circadian oscillation of body temperature. The need for DS particularly underlies the structure of the waking-sleeping cycle, since the DS debt accumulation is a continuous process that develops at a steady rate during day and night (42). Thus, sleep may occur at any time. However, the AH-PO control mechanism of sleep exerts a restricting influence on its occurrence in relation to the circadian rhythm of body temperature that is the expression of a timing system integrating many internal and external clues relevant for physiological regulations (42). Recent studies show that in humans, sleep and particularly DS occur, with rare exceptions, around the temperature minimum in conditions of both internal synchronization and desynchronization (4,5,52–54). The fact that body temperature and waking-sleeping rhythms cannot be easily dissociated is consistent with the concept developed in this article that stresses the basic control exerted by AH-PO thermoregulatory mechanisms on the waking-sleeping cycle in the absence or presence of a thermal load.

The evolution of sleep in mammals may be seen as a stepwise regulatory regression (Fig. 4), which attains, in a reverse fashion, the successive functional levels of the phylogenetic development of the encephalon (10). On this basis, the role of thermoregulatory mechanisms in the control of behavioral states conceptually appears as a functional necessity to maintain physiological homeostasis.

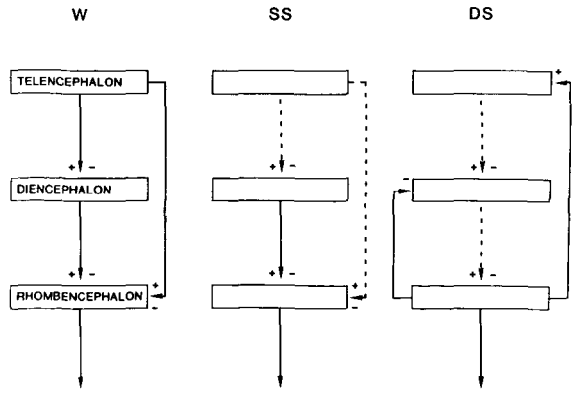

FIG. 4. Functional dominance of telencephalon, diencephalon, and rhombencephalon in wakefulness (W), synchronized sleep (SS), and desynchronized sleep (DS). The loss of hierarchical coherence between the morphological and functional organizations of the encephalon during DS underlies the impairment of homeostatic regulation (10).

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