A Developmental and Component Analysis of Active Sleep

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A wide variety of hypotheses have been put forth that address the functional significance of active sleep. Despite the well-accepted fact that active sleep expresses itself predominantly in the perinatal period, the vast majority of these functional hypotheses are applicable largely, if not exclusively, to the adult. We build on the developmental approaches of previous researchers and propose that the individual components of active sleep (e.g., myoclonic twitches, rapid eye movements) exhibit unique developmental and phylogenetic histories and may serve independent functions in the developing organism. This dynamic perspective leads to specific experimental approaches aimed at the developmental roles of these components in the neonate, their maintenance roles in the adult, and the means by which these various components coalesce temporally in what is commonly referred to as a behavioral state.

Introduction

Since its discovery over 40 years ago (Aserinsky & Kleitman, 1953), active sleep has presented a daunting challenge to neuroscientists. Although a small part of our adult sleeping and waking lives, active sleep exhibits such a curious combination of cerebral excitation, muscular paralysis, and autonomic irregularity that it could not help but fire the scientific imagination. Despite many years of research effort, however, a disparity of views continues among sleep researchers as to the significance of this form of sleep.

When considering the function of active sleep, researchers have placed primary emphasis on the adult animal. This is not surprising given that it was in the adult that active sleep was discovered. But active sleep is, we argue, a developmental phenome-

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non. It is a conglomeration of components, some of which are prevalent in fetuses and newborns, that are transformed and integrated during ontogeny. In other words, active sleep in the adult is a continuation of its earlier expression in the neonate (Corner, 1977).

In this review, we will not argue that active sleep has been neglected by developmental psychologists and physiologists. On the contrary, there has been an explosion of research on active sleep in the fetus and newborn. Despite such research, however, the neonate has been relegated to secondary importance when it comes to the formulation of functional hypotheses in that the majority of these hypotheses do not adequately address the significance of active sleep in neonates.

Thus far, we have used the word sleep as if its meaning is obvious. It is not. As a behavioral descriptor, sleep provides a useful shorthand for denoting the simultaneous or near-simultaneous expression of a group of defining components. But the word is also used to imply a deeper reality or an unseen factor that underlies the temporal cohesion of sleep-related components. This use of the word sleep is more than a shorthand: It denotes a *behavioral state* whose reality is presumed to exist regardless of the actual expression of the components that are normally used to identify it. In this article, we use the word sleep only in the former sense, for reasons that will become clearer below.

Phenomenology of Active Sleep in Adults and Neonates: Essences and Components

The behavioral and electrophysiological components of active sleep are well known (Table 1). One of the most prominent of these components in adult mammals is the appearance of an activated electroencephalogram (EEG), a wave pattern that is similar to that found in an awake animal and that reflects a cerebral cortex whose neurons are firing rapidly and independently of one another. In addition to changes in the EEG, muscle tone decreases to near-zero levels, the eyes begin darting, respiration becomes irregular, the hippocampus exhibits a theta rhythm, and twitching in the extremities can be observed (Vertes, 1984). Furthermore, animals in active sleep do not exhibit normal thermoregulatory responses (Parmeggiani, 1977). Other events can also be detected during active sleep, including the spontaneous activation of the muscles of the middle ear (Pessah & Roffwarg, 1972). Based on these multiple components, active sleep has

Table 1
Some of the Defining Components of Active Sleep in Adult Placental Mammals as Compared with Their Utility for Defining Active Sleep in Newborn Rats

Active Sleep Components in Adults	In Newborn Rats?
Activated EEG	No
Rapid eye movements	No
Muscle atonia	No
Inhibited thermoregulatory responses	No
Hippocampal theta	No
Irregular respiration and heartbeat	??
Myoclonic twitching	Yes

also been variously called rapid eye movement (REM) sleep and paradoxical sleep (due to the presence of an activated EEG in a paralyzed, sleeping animal).

In contrast to adults, active sleep in newborns must be defined on the basis of a reduced set of components. For example, Jouvet-Mounier, Astic, and Lacote (1970) were forced to rely on muscle twitching alone when monitoring "active" sleep in newborn rats (< 5 days of age) because none of the other characteristic components was observable or reliable (see also Gramsbergen, Schwartze, & Prechtl, 1970; Leblanc & Bland, 1979). Specifically, the newborn's undifferentiated EEG did not allow for discernible distinctions among behavioral states and rapid eye movements were not detected until the end of the 1st week postpartum; neck muscle tone also was not a useful indicator of active sleep in the newborn because baseline muscle tone was already very low. In addition, it recently has been shown that newborn rats are able to respond to cold exposure by increasing nonshivering thermogenesis even while they exhibit undisturbed levels of sleep-related twitching (Blumberg & Stolba, in press). Thus, many of the components used to define active sleep in adults are not observed or are not reliable in neonates.

Of course, in order to assess the developmental trajectory of active sleep, one must first understand how to define its presence or absence at any given time. As stated earlier, when Jouvet-Mounier et al. (1970) set out to describe the development of active sleep in neonatal rats, they were forced to rely on muscle twitching and also, toward the end of the 1st week, on ocular movements. Thus, for the earliest ages, pups were said to be in active sleep on the basis of a single indicator, that is, muscle twitching. Nonetheless, using twitching as their marker for the presence of active sleep, Jouvet-Mounier and her colleagues concluded that active sleep occupies 70–80% of the rat pup's time during the first 10 days postpartum.

The work of Jouvet-Mounier et al. (1970) raises the following question: how can we use only one or a few components to identify active sleep in neonates when it has been defined in adults using many components? There are a number of possible responses to this question, one of which is to deny the existence of active sleep in neonates when all the adult components are not present or detectable, perhaps designating neonatal sleep as a "proto-state" that is fundamentally different from that of the adult (e.g., Frank, Heller, & Dement, 1994). But merely renaming a behavioral state in neonates has its own problems, as Frank et al. (1994) illustrate when they ask "Is neonatal active sleep really paradoxical sleep?" Specifically, given that the many terms for active sleep are typically used interchangeably by sleep researchers, the above question cannot easily be distinguished from similarly posed questions such as "Is neonatal paradoxical sleep really active sleep?" or "Is neonatal active sleep really rapid eye movement sleep?"

A second response is, for example, to allow muscle twitching to *stand for* active sleep in the neonate and proceed *as if* the absence of other components does not matter. In other words, this approach assumes that there is an *essence* of active sleep and that any individual component is a mere front for this deeper reality. Essences have played important roles throughout philosophical history, but they are as enticing and dangerous in the realm of biology as they are in the historical sciences, as explained by Fischer (1970) in his treatment of fallacies of historical reasoning:

> The *fallacy of essences* begins with the old idea that everything has something deep inside it called an essence, some profound inner core of reality. . . . The fallacy of essences is, nevertheless, very common in historical writing. It is psychologically gratifying, for its supplies a sense of completeness and it encourages a sense of certainty. But these are illusions which an empiricist must learn to live without. (p. 68)

When applied to adults, the essentialistic view takes the more reasonable form of positing a single, unitary mechanism that is responsible for activating the individual components of active sleep and causing them to cohere temporally. There are many known features of active sleep, however, that are not easily explained by this single-mechanism perspective: That individual active-sleep components arise independently during development (Jouvet-Mounier et al., 1970); that the temporal coherence of individual components can break down in adults due to pathology or experimental manipulation (Morrison, 1983); and that the activity of individual components can be modified independently as a result of waking experiences (Herman & Roffwarg, 1983).

The tension between explanations in terms of essences or components is illustrated by Corner's (1985) metaphorical description of active sleep as a rope composed of multiple strands. When normal adults are considered, the rope is intact. As we go back in developmental time, however, the rope begins to unravel until, in the neonate, only one strand may remain as an index of active sleep. Corner's metaphor is useful for the way it depicts active sleep as a behavioral–physiological state constructed of multiple components. Like all such metaphors, however, it must be used appropriately. Specifically, we can treat active sleep in the adult as a cohesive rope that unravels as we go backward in developmental time. (We will ignore for the moment that development proceeds in the opposite direction.) But, as the rope unravels and we are left with a single remaining strand, should we consider that strand to contain within it the essence of the entire rope?

We think not. Rather, we should treat each strand as an independent entity with its own individual characteristics. Thus, we argue that our challenge is to explain each of the individual components of active sleep in developmental time and investigate the processes by which these multiple components coalesce, cohere, and self-organize during ontogeny. In other words, sleep is not the product of any single, essential controller but an emergent property of the dynamic interactions among individual components.

Hypothesized Functions of Active Sleep

The reader may already be familiar with the diversity of functions that have been proposed for active sleep. In general, these theories can be categorized broadly as relating to physiological restoration, behavioral adaptation, cognitive functioning, or brain maturation.

An example of evidence supporting a role for active sleep in physiological restoration is provided by the work of Rechtschaffen, Gilliland, Bergmann, and Winter (1983), who selectively deprived adult rats of active sleep by placing them on a platform that rotated if the rats entered active sleep; if an experimental rat did not wake up when the platform began its rotation, it would be forced into a shallow pool of water. Within weeks these active-sleep-deprived rats were severely debilitated compared to yoked controls and, within a month, died if the deprivation schedule was not terminated. Recent work suggests that such deprivation procedures suppress immune responding, making these animals especially susceptible to infection from normally indigenous bacteria (see Blakeslee, 1993). It is difficult, however, to determine from such experiments whether one is investigating the function of active sleep or the physiological consequences of an unusual, and perhaps stressful, experimental procedure.

As another example of a possible role for active sleep in the maintenance of physio-

logical function, Vertes (1984) has suggested that active sleep serves as a state of neural activation interspersed within the other, quieter states of sleep. This neural activation, it is hypothesized, is essential for preventing the brain from shutting down during sleep and thus leading to death.

A series of active sleep hypotheses have also been put forth that focus on this state as a behavioral adaptation. Snyder (1966) suggested the "sentinel" hypothesis which proposes that active sleep, with its heightened neural arousal and decreased sensory threshold, serves the animal in the wild by allowing for brief periods of environmental scanning embedded in the less engaged and deeper states of nonactive sleep. Similarly, Jouvet (1975) has suggested that the dreams that often accompany active sleep in adults act as a practice forum for species-typical behaviors. Such mental practice is proposed to be especially useful during active sleep because of the accompanying muscular paralysis, a condition that would reduce the risk of animals acting out their dreams at inappropriate times.

Cognitive hypotheses for active sleep also have been proposed. For example, Winson (1993) has proposed that active sleep helps us to remember and integrate species-specific behaviors. More generally, a number of empirical studies have argued that active sleep is necessary for the consolidation of memories; for example, Rose and Smith (1992) deprived adult rats of active sleep between 5 and 8 hr after the rats had been trained on the Morris water maze task and found that the sleep-deprived rats had greater difficulty finding the underwater platform. Alternatively, Crick and Mitchison (1983) have argued that if the brain can be compared to a computer-generated neural network, then active sleep may help the brain to remove spurious connections formed during our various waking experiences. In other words, active sleep may help us to forget, or "unlearn," as Crick and Mitchison prefer.

The functional hypotheses described earlier are largely, if not exclusively, concerned with active sleep in adult animals. The predominance of active sleep in newborn mammals, however, requires explanation, and Roffwarg, Muzio, and Dement (1966) were the first to attempt such an explanation. These investigators drew attention to the newborn's high levels of active sleep and focused on the physiological aspects of active sleep that could aid the developing animal. Specifically, they conjectured that the intense neural activity indicative of active sleep could assist in "neuronal differentiation, maturation, and myelinization in higher centers" (p. 616). Roffwarg and his colleagues, however, felt that their hypothesis, while perhaps effective in explaining active sleep in the newborn, was less effective in explaining active sleep in the adult. Thus, they posited a separate and distinct function for active sleep in the adult that, very similar to Snyder's sentinel hypothesis, suggests that active sleep maintains the animal in a state of readiness during an otherwise vulnerable period of behavioral inactivity.

Roffwarg et al. (1966) recognized that high levels of active sleep in neonates could reflect the satisfaction of particular developmental needs, as they suggest, or merely reflect the neonate's special *inability* to control the amount of active sleep exhibited. This latter possibility suggests that active sleep in neonates has no particular developmental function—it simply cannot be suppressed. In fact, theoretical treatments of the function of active sleep generally reflect this latter view that active sleep does not serve a developmental function. This reading of the literature is supported by the following two observations: first, even after nearly 30 years, the article by Roffwarg et al. (1966) remains as the preeminent citation by sleep researchers when discussing the issue of the developmental function of active sleep; and second, functional hypotheses of active sleep are generally stated in the context of adult functioning and only in some cases,

and then as only an afterthought, are the hypotheses shaped in a *post-hoc* fashion to fit the developmental data. We address these issues in more detail in the next section.

Do Current Functional Hypotheses Fit the Developmental Data?

Many of the favored active-sleep hypotheses described in the preceeding section cannot account for the high levels of active sleep in neonates. For example, as described earlier, Vertes (1984) proposed the view that active sleep serves to maintain periodically high levels of brain activity without arousing the animal from sleep. Borrowing both from the developmental hypothesis of Roffwarg et al. (1966) and the sentinel hypothesis of Snyder (1966), Vertes proposes that "it may be detrimental for the brain to remain 'off' or quiescent for long periods of time as it is in slow-wave sleep and that REM prevents this from happening by providing endogenous stimulation to the brain at regular intervals throughout sleep" (p. 277). As support for this hypothesis, Vertes notes that the fact that "the sleep cycle is shorter in the infant . . . than in the adult . . . suggests that the immature brain may be even less tolerant of lengthy periods of inactivity" (p. 278). In fact, however, one can actually make the case that newborns are much more resilient to periods of sustained inactivity. For example, consider the fact that newborns can literally be frozen (a common and useful form of anesthesia in newborns; Phifer & Terry, 1986) but will recover hours later with no discernible negative effect, while older animals do not survive this procedure. Thus, newborns are more tolerant than adults of extreme hypothermia and hypoxia, not less tolerant as Vertes' hypothesis requires.

Newborns also display a variety of other features that suggest some problems for Vertes' notion that neonates are intolerant of decreased brain activity. For example, a number of neonatal organs (e.g., heart, adrenal medulla), in contrast to these organs in adults, function without direct neural connectivity (e.g., Seidler & Slotkin, 1986; Tucker, 1985). In addition, the neonatal spinal cord is capable of exhibiting many motor functions independent of descending influences, as indicated by spinal transection (Stelzner, Ershler, & Weber, 1975). Moreover, the transection procedure itself does not produce spinal shock as it does in older animals (Stelzner, 1982), again suggesting that the neonatal nervous system is more, not less, tolerant of insult and injury than the adult nervous system.

With respect to the notion that active sleep is a behavioral adaptation, it is hard to imagine how a newborn rat, whose survival depends upon its mother's behavior, could benefit from active sleep performing a sentinel function. Similarly, it is difficult to imagine that newborns are practicing species-typical behaviors in their heads at a time when their cerebral cortex is not even developed enough to exhibit recognizable signs of active-sleep activity. Thus, the notion that active sleep is a behavioral adaptation does not seem to capture the full flavor of active sleep as exhibited by newborn mammals.

Active sleep has also been hypothesized to play a role in cognitive functioning, such as in the learning processes that accompany daily life (e.g., Winson, 1993). These hypotheses have been applied almost entirely to adults while the implications of active sleep for cognitive processes in newborns have been laregly ignored. But, consider the possibility that newborns do require a mechanism such as active sleep to aid the learning process. Further consider that newborn rats are in active sleep 80% of the time while adult rats are in active sleep 5% of the time (and exhibit nonactive or quiet sleep approximately 30% of the time). This would then suggest that newborns are in active

sleep 80% of the time to remember what they learned during the remaining 20% of the time that they are awake, while adults are in active sleep 5% of the time to remember what they learned during the remaining 65% of the time that they are awake. This would suggest an enormous increase in the *efficiency* of active sleep that proponents of these "learning" hypotheses have yet to try to explain.

Winson (1993) builds on a memory-processing hypothesis by suggesting that active sleep aids in the "integration of individual experience into a strategy for further behavior" (p. 245). Specifically, he aruges that "experience gained during . . . species-specific waking behaviors is reaccessed and integrated into an animal's behavioral strategy during REM sleep" (p. 245). Winson's theory "centers on theta rhythm" (p. 245), an electrophysiological correlate of active sleep. This rhythm, Winson argues, is vitally connected to the expression of species-specific behaviors, thus leading to his hypothesis. In humans, he modifies his hypothesis so that active sleep in our species integrates "all waking experience that pertains to psychological survival." And what about newborns? Winson's answer is that active sleep may have a separate developmental function such that his memory hypothesis is not applicable until 2 years of age, when "the mnemonic function of REM sleep takes hold" (p. 246).

The majority of hypothesized functions of active sleep that have been proposed are not developmental in perspective. At best, a hypothesis conceived to account for adult phenomena may be stretched to apply to the neonate; at worst, a hypothesis cannot be applied at all. We contend, however, that we will never develop a coherent view of active sleep as long as we fail to consider its developmental aspects. A theory of active sleep that does not explain its features in the neonate explains very little.

The shortcomings of these adult-centered hypotheses were recognized by Crick and Mitchison (1983) when they proposed their "reverse learning" hypothesis. They write, "Any purely psychological theory (such as Freud's) is hard-pressed to explain the large amount of REM sleep in the womb, and any purely developmental theory must account for the quite appreciable amount of REM sleep in adult life" (p. 113). We agree, but it must also be recognized that active sleep is more than merely a cortical phenomenon, as Crick and Mitchison propose. Thus, although their hypothesis attempts to explain active sleep in both neonates and adults, it unnecessarily restricts its focus to a single component (i.e., the removal of "parasitic modes" from the cerebral cortex) whose significance for the neonate is unclear.

Active Sleep as an Extension of Spontaneous Behavior in Embryos

As described earlier, as we push our observations further back in developmental time, we find that active sleep in newborns must be identified on the basis of fewer and fewer components. As we have seen with newborn rats, myoclonic twitching is the one active sleep component that can be used to identify the presence of active sleep. But, it is at this point that the traditional conceptualization of active sleep begins to fray. To explain why, we must jump even further back in developmental time and, working our way forward, review the early behaviors of embryos and neonates.

Neuroembryological studies of chicks (Hamburger, 1973) and rats (Narayanan, Fox, & Hamburger, 1971) have revealed clear similarities and generalizations in the organization of early behaviors. Specifically, these early behaviors can be categorized on the basis of their spontaneity, their coordination, and their localization. For example, one basic kind of behavior (designated Type I behaviors by Hamburger, 1973) has the following characteristics: (a) It is spontaneous, by which is meant that it is elicited

independently of sensory stimulation (i.e., it is nonreflexogenic); (b) it is intermittent; and (c) the various limbs and appendages that exhibit this behavior do so in an uncoordinated fashion. The movements themselves, which can be described as "convulsive-type jerks and twitches" (Narayanan et al., 1971, p. 101), are easily distinguished from startles (Hamburger's Type II behaviors) which are characterized by a jerky tremor throughout the whole body. It is also easy to distinguish spontaneous twitching from coordinated movements of multiple limbs and body segments such as kicking, stretching, and, in the case of chicks, hatching movements (Hamburger's Type III behaviors).

The mechanism that produces twitching was determined by Hamburger and his associates in the 1960s. For example, Hamburger, Wenger, and Oppenheim (1966) isolated the lumbosacral spinal cord of 2-day-old chick embryos by removing thoracic segments from the neural tube. In a second procedure in the same animals, they then surgically removed the dorsal, sensory half of the lumbosacral spinal cord. Despite these surgical procedures, embryos still exhibited twitching leg movements, thus demonstrating the spontaneous, nonreflexogenic nature of these movements. Subsequent electrophysiological investigations confirmed that twitching movements are produced by the activation of neurons within the lower half of the ventral spinal cord (Provine, 1986); that is, they are produced neurogenically. (A final possibility—that these movements are produced by the spontaneous contraction of muscles—was also disproved.)

Spinal transections that isolate the lumbosacral spinal cord also do not shut down hindlimb activity in fetal rats (Narayanan et al., 1971; Robertson & Smotherman, 1990). Thus, the notion that local neural circuits that generate spontaneous activity exist in the spinal cord is now widely accepted as a reliable phenomenon. Interestingly, in their initial article describing the spontaneous movements of fetal rats, Narayanan et al. (1971) made the following observations: "It is pertinent to mention here that spontaneous motility, especially local and regional motility and 'startles' continue after birth. However, they were observed only during activated sleep. . . . There may therefore be some neurophysiological relationship between spontaneous motility *in utero* and the uncoordinated spontaneous, unsolicited movements that occur during activated sleep postnatally" (p. 129). This comment is interesting because, once again, it raises the issue of how one knows whether a newborn rat is in active sleep *independent* of the observation of twitching. Even more interesting, however, is the recognition by these investigators of a basic similarity between the spontaneous movements studied by neuroembryologists and the myoclonic twitching studied by sleep researchers.

Michael Corner has, more than any other researcher, appreciated the significance of the basic similarity and, more importantly, the continuity between prenatal and postnatal spontaneous behaviors. Citing phylogenetic and ontogenetic data, Corner (1977) goes so far as to suggest that motor activity during sleep "is nothing less than the continued postnatal expression of primordial nervous functional processes" (p. 292). Elsewhere, he states: The working hypothesis here would be that the pontine regions which have been implicated in [active sleep] regulation . . . are not, after all, specialized products of ontogeny or phylogeny but rather a 'neotenic' vestige of an originally *diffuse* endogenously bursting system. (Corner, 1985, p. 182)

Myoclonic Twitching in Neonatal Rats with Spinal Transections

When a newborn rat is placed in a warm, thermoneutral environment, it soon adopts a relaxed posture and begins exhibiting twitching movements of the forelimbs, hindlimbs, and tail. Moreover, the twitching movements seem highly synchronized

such that, for example, a twitch of the right forelimb may occur nearly simultaneously with a twitch of the tail. It is also seemingly apparent that such an animal is in a cohesive behavioral state. But is this behavioral state generated by a single, unitary mechanism or does the state result from the temporal cohesion of multiple, independent components?

We were led to ask this question because of the apparent contradictory observations of neuroembryologists and sleep researchers regarding the mechanisms underlying spontaneous behaviors and myoclonic twitching, respectively. One possibility was that both groups of researchers observed qualitatively different phenomena. Another possibility was that the spontaneous behaviors as studied by neuroembryologists disappeared before the myoclonic twitching of active sleep made their appearance. Yet a third possibility was that both spinal and brain twitch mechanisms existed in the same animal at some point in development.

We examined this last possibility by transecting the spinal cords of newborn rat pups in the midthoracic region (Blumberg & Lucas, 1994). Such a transection has the effect of separating the motor control of the forelimbs from that of the hindlimbs without damaging the motor neurons subserving either limb group. Moreover, by performing the spinal transections in 1- to 2-day-olds, we knew the local neuronal circuits below the level of the transection would be spared and would retain their function (Stelzner, 1985).

When we observed the twitching movements of nontransected control pups at 5 days of age, we found that the forelimbs and hindlimbs twitched at similar rates. Moreover, log-survivor analysis of the temporal distribution of twitching in these limbs indicated the presence of two generating mechanisms, one producing high-frequency twitches (i.e., with intertwitch intervals less than 5 s) and another, apparently random, mechanism producing twitches over a broad frequency spectrum (i.e., with intertwitch intervals from 0 to 30 s).

The reality of these two mechanisms became apparent when we observed the twitch behaviors of the transected pups. First, hindlimb twitching was retained despite the surgery and, in fact, was only reduced by 50%, thus indicating the presence of twitch-producing mechanisms in the lumbar spinal cord. Second, log-survivor analysis of the temporal distribution of hindlimb twitching was altered from that of the normal pups (as well as from that of forelimb twitching). Specifically, hindlimb twitches were distributed randomly throughout the range of intertwitch intervals—the high-frequency component of twitching had been removed by the transection. Thus, we concluded from these observations that there existed in newborn rats two independent twitch-producing systems: One consisting of motor neurons in the spinal cord that fires randomly over a broad frequency range and one consisting of more rostral motor neurons, perhaps in the brainstem, that fires rapidly in a pattern, random or nonrandom, that remains to be determined. Finally, these results are consistent with work on rat fetuses showing that early (Days 16–19 of gestation) motor activity is produced by a random mechanism (see Figure 7 in Smotherman & Robinson, 1986).

Perhaps what was most enlightening was how the intact animal, asleep and exhibiting twitching among all its limbs, presents a picture of a coherent behavioral state. There is a significant degree of synchrony among all the limbs which can easily give the impression of a single mechanism controlling all of the observed phenomena. In fact, however, there exist a multiplicity of independent twitch-producing mechanisms throughout the spinal cord and brain whose firing is coordinated as a result of descending, and perhaps also ascending, information.

The illusion of a behavioral state generated by a unitary mechanism is shattered by a simple observation: First, we allowed a spinally transected pup to acclimate to a warm environment and fall asleep; then second, we pinched its tail. Although pinching the tail of an intact pup elicits kicking and squirming throughout its body (i.e., the animal "wakes up"), the transected pup exhibits stereotyped kicking in the hindlimbs while the forelimbs continue to twitch as if nothing had happened. This discordance between the "awake" hindlimbs and the "sleeping" forelimbs again calls into question the labels and concepts currently used to describe and explain the behavior of sleep.

Proposed Functions of Spontaneous Behavior in Neonates

The transection experiment described earlier suggests that we face two issues in the study of active sleep. First, there is the question of the ontogeny and function of the individual components of active sleep, including myoclonic twitching and rapid eye movements. But second, there is the issue of coordination, that is, how these independent components come to cohere temporally. That myoclonic twitching in neonates is produced independently by motor neurons throughout the neuraxis suggests that, initially, we are justified in focusing our attention on the components as independent entities. This is not to say that the coordination of components is not a vital issue in the study of active sleep, only that any substantive examination of this issue requires a greater understanding of the individual components than is presently available.

Thus, to begin our attempt at a cohesive perspective of active sleep, we will first limit our focus. Specifically, we will develop a theory regarding the function of myoclonic twitching and will next argue that other components of active sleep have features that are analogous to muscle twitching. We focus on four processes known to be affected by spontaneous activity: Neuron cell death, muscle fiber differentiation, synapse elimination, and the formation of topographic maps. We suggest that the spontaneous neural activity underlying myoclonic twitching contributes to these four developmental processes.

Neuron Cell Death

Earlier, we briefly surveyed neuroembryological studies of rat fetuses and chick embryos in which investigators described the spontaneous movements of their subjects. Naturally, one of the questions that intrigued these investigators was "Do these spontaneous movements contribute to normal development?"

It is now well established that motor neurons are initially overproduced and that approximately 50% of these neurons die during development (Oppenheim, 1989). This process, called natural cell death, arises out of a competition between motor neurons for limited innervation sites on muscle fibers. That this competition is an active process was demonstrated by inactivating neuromuscular activity (e.g., with curare) in chick embryos. Such inactivation resulted in the survival of motor neurons that would have normally died (e.g., Pittman & Oppenheim, 1978). Although the search continues for the trophic factor or factors that promote neuron survival, there is little doubt that the process is activity-dependent.

Neuron cell death is not limited to the motor neuron population. In fact, there are few brain regions in which naturally occurring cell death has not been observed (Cowan, Fawcett, O'Leary, & Stanfield, 1984), and these nonmotor forms of cell death are also modulated by activity-dependent processes. For example, retinal ganglion cells in rats exhibit spontaneous activity in both the prenatal (Galli & Maffei, 1988) and

postnatal (Galli-Resta, Ensini, Fusco, Gravina, & Margheritti, 1993) periods. In the postnatal period, these ganglion cells form connections with neurons in the superior colliculus. When the activity of these ganglion cells is blocked by the sodium channel blocker tetrodotoxin, there is a 50% increase in cell death within the superior colliculus (Galli-Resta et al., 1993). Thus, neuromuscular activity is required to accomplish motor neuron cell death within the spinal cord, while retinal ganglion cell activity is required to prevent neuron cell death within the superior colliculus.

Muscle Fiber Differentiation

The role of activity in muscle fiber maintenance and differentiation has been investigated using a number of experimental approaches. For example, denervation of mammalian skeletal muscle has been shown to have numerous effects on the function, structure, and biochemistry of muscle fibers (e.g., Albuquerque & McIsaac, 1970). One obvious effect is muscle atrophy, a reduction in the mass of the fiber resulting from a decrease in the major muscle proteins actin and myosin (Harris, 1974). Another effect of denervation is supersensitivity of the denervated fiber to acetylcholine (e.g., Brown, 1937). Moreover, if nerve activity is blocked by the application of a local toxin (so that the nerve remains intact and is still capable of spontaneous release of acetylcholine and the release of neurotrophic factors), then even those fibers that do not demonstrate overt atrophy nonetheless exhibit supersensitity to acetylcholine (Lømo & Rosenthal, 1972). Clearly, nervous system activity is necessary for the functional maintenance of muscle fibers.

Activity also plays a role in muscle fiber type differentiation. Mammals have two main fiber types, the so-called slow-twitch fibers (found in postural muscles) and fast-twitch fibers (found especially in locomotion muscles). These two fiber types are differentiable based on the predominant form of myosin produced within the cell. Buller, Eccles, and Eccles (1960) first showed that cross-innervation of slow-twitch fibers and fast-twitch fibers by the opposite motor neuron type results in a change in the properties of the muscle fiber in accordance with the motor neuron innervating it. Thus, the activity pattern of the motor neuron can alter the type of muscle fiber produced.

Finally, it has also been demonstrated that the type of electrical stimulation a muscle fiber receives specifically alters the level and kind of myosin produced by the fibers (Goldspink et al., 1992). Because the fiber type is determined by the relative amounts of "slow" and "fast" myosin produced, which in turn is directly influenced by the activity pattern the fiber receives, it is clear that activity plays an important role in the determination of fiber type.

Synapse Elimination

At birth in a number of species including the rat, each muscle fiber is innervated by a multitude of synapses arising from many motor neurons. In the adult, however, each muscle fiber is innervated by only one motor neuron. Thus, during ontogeny, there is an initial overproduction of synaptic connections at each muscle fiber (just as there is an initial overproduction of *neurons* innervating target sites) leading to the "polyneuronal innervation" of muscle fibers by motor neurons. Moreover, just as there is a competitive reduction in the number of motor neurons during ontogeny, there is also a competitive reduction in the amount of polyneuronal innervation. But, it is not the case that this reduction in polyneuronal innervation, or synapse elimination, is merely the by-product

of neuron cell death. On the contrary, these two processes do not occur concurrently. For example, in rats, neuron cell death is largely a prenatal phenomenon while synapse elimination occurs 2–3 weeks postpartum (Brown, Jansen, & Van Essen, 1976; Purves & Lichtman, 1980).

As with muscle fiber differentiation and neuron cell death, synapse elimination is an activity-dependent process (Colman & Lichtman, 1993). For example, when alpha-bungarotoxin, a nicotine receptor antagonist, is applied to the soleus muscle of newborn rabbits so that postsynaptic activity is blocked, polyneuronal innervation at that muscle is retained (Callaway & Van Essen, 1989). A similar increase in synaptic survival was achieved by the application of tetrodotoxin to the spinal nerves travelling from spinal cord to soleus muscle (Callaway, Soha, & Van Essen, 1989). In contrast, synapse elimination can be accelerated by artificially increasing neuronal activity (O'Brien, Östberg, & Vrbová, 1978; Thompson, 1983).

As described earlier, each muscle fiber exhibits at birth a multitude of synaptic contacts; some of these contacts arise from the same motor neuron's axons, some do not. It is also true that the synapses arising from a single motor neuron and innervating a muscle fiber are either retained or eliminated as a group:

> It seems most reasonable then to view competition as pitting the *set* of synaptic terminals of one axon against the sets of synapses of other axons that also innervate the same junction. Such a set of synapses from one axon could be considered a "cartel," working together in an attempt to monopolize innervation of the muscle fiber [their italics]. (Colman & Lichtman, 1993, p. 3)

Thus, at the location of the muscle fiber, there is an identification problem: How does the muscle fiber "know" which synapses belong to the same cartel? As Colman and Lichtman (1993) state, "there must be some critical identifying characteristic that distinguishes the members of one synaptic cartel from members of other cartels innervating the same junction" (p. 3).

They then suggest a solution to this problem:

> In addition to sharing the same cytoplasm, the synaptic boutons that make up one axon's cartel on a muscle do share a common activity pattern. Even though the separate boutons may have different probabilities of release . . . , the members of a single cartel are much more likely to be active synchronously than synapses belonging to different axons. . . . Thus, at the neuromuscular junction an axon's activity pattern is a potentially useful characteristic to differentiate its cartel from those of other axons. (Colman & Lichtman, 1993, pp. 3–4)

We hypothesize that the "critical identifying characteristic" necessary for the unique identification of each cartel could be provided by a simple random-firing mechanism. And significantly, recall that hindlimb twitching in 5-day-old transected pups was generated by a random mechanism. Thus, we suggest that the twitching movements commonly associated with active sleep in neonates contribute to the competitive interactions that give rise to synapse elimination. Specifically, synapses that belong to the same cartel will fire synchronously and thus will not compete with each other. In contrast, synapses that belong to different cartels will fire asynchronously and thus will compete with each other. As long as each motor neuron fires randomly, unique identification is possible.

The ontogeny of synapse elimination provides clues as to its relation to myoclonic twitching and behavioral development. Specifically, Brown and his colleagues (1976) investigated the ontogeny of synapse elimination in the soleus muscle of neonatal rats.

They found that while 100% of all soleus muscle fibers were polyneuronally innervated through Day 10 postpartum, very few such fibers were polyneuronally innervated by Day 15 postpartum. In other words, rapid synapse elimination was concentrated between Days 10 and 15 postpartum (see text-Figure 4 in Brown et al., 1976).

There also appears to be a rapid diminution in the occurrence of active sleep over this same 10- to 15-day period in rats. For example, Jouvet-Mounier et al. (1970) recorded a significant decrease in active sleep over this period. Specifically, they found that as active sleep decreased, quiet sleep increased in frequency while waking behavior was relatively unchanged.

This correlation between synapse elimination and the incidence of myoclonic twitching gains in apparent significance when one considers the changes in behavioral development over the first 3 weeks postpartum. Altman and Sudarshan (1975) investigated the development of locomotor behavior in neonates and found a dramatic alteration in locomotor abilities over this time span: Neonates that could only crawl at 10 days of age were walking by 15 days of age. Thus, although only correlational, these observations taken together suggest intriguing interactions at the neuromuscular and behavioral levels during a relatively brief period of development. Of course, only direct experimentation can uncover any causal connections between these various phenomena.

Formation of Orderly Connections

One of the basic questions in developmental neurobiology involves the organization of maplike, orderly representations in the nervous system. For example, ganglion cells that reside next to each other within the retina project to target cells in the lateral geniculate nucleus that also reside next to each other. In turn, these cells then project to target cells in the visual cortex that reside next to each other. Similarly, topographic relations are established between spinal cord motor neurons and their target muscles (e.g., Smith & Hollyday, 1983). The means by which such nearest-neighbor relations develop and are preserved has been the focus of intense experimental investigation (Udin & Fawcett, 1988).

Similar to synapse elimination, competitive interactions among active presynaptic neurons are an essential component of the formation and/or fine-tuning of topographic organization in a number of systems [It should be noted, however, that activity-independent processes are also important (Shatz, 1990)]. For example, as was originally demonstrated by Hubel and Wiesel (1963), visual information arriving from each eye is segregated within ocular dominance columns within the visual cortex. Subsequent work demonstrated that visual experience was necessary for the formation of these ocular dominance columns (Shatz, 1990).

But what kind of visual experience? In 1986, Stryker and Harris showed that ocular dominance columns develop in kittens even when they are reared in the dark. Two years later, Galli and Maffei (1988) were able to record spontaneous activity from the retinal ganglion cells of rat fetuses as early as Day 17 of gestation. Thus, nearly 3 weeks before a rat's eyes even open, its retinal ganglion cells are firing spontaneously and thus participating in the construction of its own visual system.

As with synapse elimination, the temporal patterning of retinal ganglion-cell activity appears to contribute to the formation of ocular dominance columns. Specifically, Stryker and Strickland (1984) blocked retinal ganglion-cell activity using the sodium channel-blocker tetrodotoxin and then stimulated the two optic nerves either synchro-

nously or asynchronously. Only when the two nerves were stimulated asynchronously were ocular dominance columns in the visual cortex established. Thus, it follows that the spontaneous retinal ganglion-cell activity recorded by Galli and Maffei (1988), if random and thus asynchronous, could provide sufficient stimulation for the formation of ocular dominance columns.

Ocular segregation is not the only example of orderly connectivity within the visual system. As described earlier, at a finer level of analysis, nearest-neighbor relations are also established such that neurons residing next to each other in the retina project to neurons that reside next to each other in the lateral geniculate nucleus and, in turn, in the visual cortex. Once again, the synchrony and asynchrony of neuronal firing play a role. For example, retinal ganglion cells that reside next to each other are more likely to fire synchronously than cells that are distant from each other (Maffei & Galli-Resta, 1990; Meister, Wong, Baylor, & Shatz, 1990); such synchronous firing of neighboring cells could be due to synaptic connections between neighbors or to electrotonic or gap junctions (Walton & Navarrette, 1991).

The foregoing discussion makes it clear that the nervous system generates much of its own "experience" and thus shapes its own development. It is also clear that many of the processes that give rise to ordered connections in the nervous system arise out of random processes. It remains to be determined, however, how these spontaneous behaviors relate to the behavioral state that we designate as active sleep. We will come back to this issue later in the article.

Are Rapid Eye Movements Just Another Form of Myoclonic Twitching?

In 1969, Berger proposed his Oculomotor Innervation Hypothesis. This novel hypothesis suggests a relationship between the rapid eye movements of active sleep and binocular vision:

> It is proposed that REM sleep provides a mechanism for the establishment of the neuromuscular pathways involved in voluntary conjugate eye movements in both phylogenesis and ontogenesis; and that throughout mammalian life REM sleep furnishes periodic innervation of the oculomotor system during extended periods of sleep, in order to maintain facilitation of binocularly coordinated eye movement into subsequent wakefulness. (Berger, 1969, p. 146)

In support of his hypothesis, Berger examined the degree of binocular vision (as estimated by the partial decussation of the optic nerve at the optic chiasm) across a series of animals and compared it with the amount of active sleep displayed by each animal. He noted that the optic nerves from the eyes of turtles and hens project completely from each eye to the contralateral hemisphere and these species also do not exhibit rapid eye movements during sleep. In contrast, the optic nerves from the eyes of cats, monkeys, and humans project to both ipsilateral and contralateral hemispheres and these species also exhibit significant amounts of rapid eye movement sleep. Thus, Berger argues, the rapid eye movements of active sleep help to maintain the oculomotor system so that conjugate eye movements are possible. The initial statement of this hypothesis was subsequently supported by experimental testing in humans (Berger & Scott, 1971; Berger & Walker, 1972; Herman & Roffwarg, 1983).

When Berger (1969) first put forth his hypothesis, the role of spontaneous activity in the development of the nervous system was not yet known. Nonetheless, Berger's

hypothesis is consistent with the spirit of our current knowledge, even though he could not be more explicit about the exact mechanism that maintains binocular muscle control. Ontogenetically, Berger (1969) imagined a need for "intrinsic innervation" (p. 146) that the rapid eye movements of active sleep provided. Phylogenetically, he was able to demonstrate, albeit preliminarily, that species that lack binocular vision also lack rapid eye movements during sleep.

Interestingly, Berger's (1969) hypothesis originally stated that rapid eye movements perform both developmental and maintenance functions. In fact, Berger himself viewed his hypothesis as a modification of Roffwarg et al.'s (1966) brain maturation hypothesis:

> Newborn mammals spend large amounts of time in REM sleep, a phenomenon which progressively declines with maturation. . . . It is proposed that the function of REM sleep in ontogenesis is to provide intrinsic stimulation, as has been suggested by Roffwarg et al. (1966), *but specific to the oculomotor system, rather than the entire cortex* [italics added]. (Berger, 1969, pp. 149–150)

Berger perhaps undermined his hypothesis by suggesting that it was specific to the oculomotor system, thus unnecessarily narrowing the hypothesis' generalizability. On the other hand, he had little empirical or theoretical basis at the time for extending it beyond the oculomotor system.

As described earlier, we now know that spontaneous activity plays a vital role in the organization of neuromuscular and sensory systems. Thus, we are suggesting that Berger's initial insight can now be generalized to a wide variety of developmental processes. Specifically, we hypothesize that the rapid eye movements of active sleep are the result of myoclonic twitches of the ocular muscles, and that they perform the same function for the development and maintenance of oculomotor control as hindlimb twitches are being suggested to play in the development and maintenance of hindlimb control. That rapid eye movements, like retinal ganglion cell activity, precede eye opening in rats further supports the notion that this neuromuscular activity plays a role in the organization of ordered connections in this system (Jouvet-Mounier et al., 1970; Van Someren et al., 1990).

If the dynamic perspective promoted here is valid, then it should be possible to selectively increase or decrease the prevalence of a single component within an active-sleep period. This has been shown to be the case for rapid eye movements. Specifically, Herman and Roffwarg (1983) had human subjects wear goggles during the daytime that reduced their vision to a 5-degree field. At night, it was found that the amplitude and frequency of rapid eye movements increased as a result of this manipulation while the total amount of active sleep (as determined by standard measures) was unaffected. This finding is unique for its selective alteration of a single component of active sleep. As Herman and Roffwarg (1983) note, most active sleep hypotheses would not have predicted such selective changes.

The hypothesis that rapid eye movements are the result of myoclonic twitching of the ocular muscles places these movements into a broader context of spontaneous activity during active sleep. Other examples of such spontaneous activity includes, of course, twitching in the distal limbs as well as in the middle ear (Pessah & Roffwarg, 1972). It is also possible that spontaneous activity in the autonomic nervous system results in the irregularities in heart rate and respiration observed during active sleep. Moreover, spontaneous activity in the autonomic nervous system could reflect underlying neuromuscular processes similar to those outlined earlier, including synapse elimi-

nation (Lichtman, 1980). It is expected that detailed investigation of individual components, their development, and their specific design features will help to elucidate their functional similarities and dissimilarities during active sleep.

Comparing Active Sleep in Newborns and Adults: Continuity Versus Discontinuity

We have suggested that many of the sleep-related developmental processes that contribute to the development of the nervous system continue to play maintenance roles in older animals. Plasticity in muscle differentiation and synaptic organization (especially as a result of learning) suggests that many of the developmental processes described earlier could continue to benefit the organism into adulthood. After all, adults that continue to learn must restructure the local topology of their nervous systems. In support of this notion, evidence continues to grow that the adult nervous system is capable of sizable degrees of plasticity in response to a variety of experimental manipulations (e.g., Mæhlen & Njå, 1982).

It is possible, of course, that adult active sleep is qualitatively different from newborn active sleep, and that the spontaneous behaviors of neonates are only functional as hypothesized here in the young. We do not consider this discontinuity hypothesis to be parsimonious, especially given that there is no empirical basis to posit such a discontinuity. Nonetheless, such a discontinuity may exist, and we are not currently in a position to rule it out.

There are many examples of ontogenetic features that serve a developmental function and then disappear; such features have been called ontogenetic adaptations (Oppenheim, 1981) and represent an important feature of developmental theory. The umbilical cord is certainly one such example. Behaviorally, the suckling of newborn rats at the nipple has been shown to be qualitatively different from those eating movements that are the hallmark of independent ingestion (Hall & Williams, 1983). It should be noted, however, that earlier researchers had considered suckling and eating to be developmentally continuous based on their common function; that is, ingestion of nutrients. Even on a gross behavioral level, suckling does not "look" like eating. In contrast, newborn rats exhibiting myoclonic twitches "look" like they are asleep.

An alternative to the discontinuity position could hold that some of the developmental functions of active sleep (e.g., myoclonic twitches, rapid eye movements) continue on through adulthood but that additional components with independent functions are added on as the animal ages (recall Corner's rope metaphor). It is even possible that the same components perform different functions at different times during the life span. Such a position is perfectly consistent with the main theme of this article: If we are to understand active sleep we must study its components individually with regard to their development and function.

We acknowledge the influence of the dynamic systems perspective on our approach to active sleep (e.g., Kelso, 1995; Thelen & Smith, 1994). The dynamic systems approach denies causal priority to any single component of a system. Rather, this approach emphasizes how components act independently and in concert with other components to generate complexity. For example, in their analyses of the development of walking, Thelen and Ulrich (1991) stress that many components must all be in place for a child to exhibit independent walking, including postural control, synchronous motor control of the legs, and a vestibular apparatus to maintain balance. These and other components all must work together under normal circumstances for a child to walk, but

no single component can be said to *cause* walking. One can, however, structure the environment in such a way that a faulty component can be bypassed, such as by providing support to a child who has difficulty maintaining balance.

The dynamic systems approach also provides a means by which to conceptualize the stagelike, discontinuous character of the developmental process. As Thelen and Ulrich (1991) state, "It is a major theoretical challenge to account for discontinuities in performance arising from processes that are themselves continuous" (p. 2). Such discontinuities, however, need not result from a qualitative change in the components of a system; on the contrary, there are many physical and biological examples of discontinuous transitions arising from changes in the interactions among existing components (e.g., gait transitions in horses; see Kelso, 1995). This is also the challenge for sleep researchers: To understand not only how the various components of active sleep are expressed and function, but also how their interactions throughout development account for those aspects of active sleep that we commonly observe. We believe that this approach helps move us toward a perspective that views the development of active sleep as a self-organizing process and away from the perspective that views active sleep as driven by an essential core that is causally and conceptually independent of its individual components.

Comparative and Evolutionary Issues in Active Sleep

The behavioral patterns of nonmammals have been extensively studied, and debates have arisen as to which animals exhibit active sleep and which do not. For example, although researchers are now generally agreed that reptiles do cycle through a sleep state, there is still some controversy as to whether this state can be equated with mammalian quiet sleep or active sleep (Romo, Cepeda, & Velasco, 1978; Warner & Huggins, 1978). Although most researchers deny the existence of active sleep in reptiles (Flanigan, 1973; Flanigan, Wilcox, & Rechtschaffen, 1973), others do argue for the existence of active sleep based upon the observation of a unique electrophysiological rhythm (Huntley, 1987) or rapid eye movements (Tauber, Roffwarg, & Weitzman, 1966). Interestingly, no one has reported seeing myoclonic twitching in sleeping reptiles (Huntley, 1987).

This debate highlights the pitfalls of essentialist thinking in that it has focused on whether animals *have* active sleep, once again demonstrating the accepted notion that active sleep is a unitary entity. But in reviewing these debates, it is clear that different investigators favor different components (or, in other words, assign causal priority to different components) in their assessments of the presence of active sleep. Some may require the presence of an activated EEG while others may emphasize rapid eye movements. Nonetheless, even while these investigators argue about different components, there is agreement that their argument pertains to a single subject, that is, active sleep.

Because the search for active sleep in reptiles has focused on adults, we wondered whether young reptiles would be more likely to demonstrate active-sleeplike behaviors. In our behavioral observations of newly hatched leopard geckos (*Eublepharis macularius*), however, we found few similarities to the sleep behaviors of neonatal rats (Lucas & Blumberg, 1994). The limb-twitching characteristic of active sleep in neonatal rats was never seen in the geckos. Our observations are, however, consistent with the ecological and behavioral demands placed on these animals. Specifically, at hatching they must be ready to function at a more mature level than neonatal rats, which are born in an altricial state. In contrast to rats, leopard geckos are born with their eyes

open, they exhibit defensive reactions (e.g., hissing) to threat, and they must independently forage for food. Thus, the precocial development of these lizards places different demands on the development of their neuromuscular systems.

This is not to say, however, that reptiles do not show any of the features that we associate with active sleep, only that they occur before hatching. Specifically, as discussed earlier, lizards, like all embryos, display the spontaneous movements of the limbs that similarly characterize the fetal and neonatal behavior of rats and other mammals (e.g., *Lacerta vivipara*, Hughes, Bryant, & Bellairs, 1967). This was exactly Corner's point when he wrote that these sleep movements in mammals are "nothing less than the continued postnatal expression of primoridal nervous functional processes" (Corner, 1977, p. 292). Once again, by focusing on the components of active sleep, we can initiate a truly comparative analysis that relates the developmental mechanisms that characterize various species to their specific ecological requirements.

Conclusions and Future Directions

The basic argument in this article is that active sleep is composed of a series of components, each of which can be studied independently with regard to their developmental and functional significance. We have focused primarily on two such components, myoclonic twitching and rapid eye movements, but there are many other components that can be similarly addressed. We are not proposing, however, that all active sleep components can be understood using identical conceptual constructs; each may exhibit a unique developmental trajectory and is very possibly reliant on the development of different subcomponents.

Just as the individual components are expressed independently through ontogenetic time, so can they be expressed independently through phylogenetic time. Thus, we should not expect the components of active sleep to be the same in, for example, a dog and a snake. Debates over which species *have* active sleep and which do not can be avoided by asking comparative questions that address the relationship between the presence or absence of an individual component and a particular species' developmental and ecological constraints.

The tendency to focus on specific components merely as markers for determining whether a particular species or neonate exhibits active sleep is no more justified an approach than defining the awake state by the occurrence of walking or talking. The awake state, like the sleep state, is composed of a number of components (i.e., locomotion, activated EEG, etc.) that are expressed differentially throughout development and throughout the day. But relying on a single component to define this state leads to dilemmas that are best avoided: The fact that individuals often walk and talk while "asleep" supports the contention that no single behavior or component can serve to define any period of animal activity.

We should stress that accounting for the coherence and coordination of individual components during active sleep relies on detailed examination of the ontogenies and dynamics of individual components. Once these details are available, we anticipate that understanding component cohesion can be accomplished without positing a unitary source of cohesion. One path to examining this issue is to determine those factors that provide a permissive environment for the expression of the components of active sleep. For example, ambient temperature influences the expression of active sleep in adults (as measured by EEG and EMG; Szymusiak & Satinoff, 1981) as well as in neonates (as measured by rates of muscle twitching; Blumberg & Stolba, in press). The identification

of those factors that mediate this effect may help direct us toward understanding the temporal cohesion of individual compqnents.

The approach presented here does not specifically negate the efforts of researchers who embrace some of the more traditional views of the function of active sleep. Taking a developmental perspective does not imply that the function of active sleep is entirely separate from other proposed roles for active sleep, such as in learning and memory processes. For example, because a traditional Hebbian view of learning and memory requires local circuit reorganizations, the significance of active sleep for learning and memory processes can be incorporated within a developmental framework. Thus, much of the work that has been done on active sleep is subsumed by our approach while the range of potential experiments is broadened.

It should be clear that the current article raises more questions than it answers. We must now seek to describe the individual developmental pathways for each component of active sleep (across mammalian and nonmammalian species), and determine how those components coalesce to suggest a seemingly cohesive and unitary behavioral state.

Notes

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