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Intracellular Analysis of Trigeminal Motoneuron Activity During Sleep in the Cat

Abstract. *Intracellular recordings were made from trigeminal motoneurons of normally respiring, unanesthetized cats during naturally occurring sleep. The transition from quiet to active sleep was accompanied by tonic motoneuron hyperpolarization. Stimulation of the reticular formation induced a depolarizing potential in trigeminal motoneurons during quiet sleep and a hyperpolarizing potential during active sleep. The results provide a synaptic explanation for the phenomenon of reticular response reversal and insights into the basic mechanisms controlling motor activity during the sleep states.*

Considerable effort has been made over the last 20 years in obtaining indirect indications of the synaptic processes that occur during the sleep states (*1*). However, the mechanisms controlling neuronal activity can be determined only by direct intracellular monitoring of neuronal processes. Unfortunately, there has been, to our knowledge, no intracellular recording in any neuron during sleep (*1*).

We have developed a method of recording intracellularly during sleep, and we now report the results obtained by recording the membrane potential of trigeminal motoneurons in cats during natural sleep. Since the animals were unanesthetized, completely undrugged, and unrestrained (with the exception of head fixation), we were able to study the spontaneous synaptic influences exerted upon these motoneurons during quiet [that is, non–rapid eye movement (NREM)] and active (REM) sleep. In addition, we will present evidence concerning the synaptic mechanisms underlying the state-dependent control of the trigeminal myotatic reflex by the reticular formation during sleep and wakefulness (*2*). This phenomenon of reticular response reversal is well suited for study by intracellular methods during the sleep states, for the pattern of reticular modulation of the masseteric reflex that occurs during quiet sleep and wakefulness (reflex facilitation) is diametrically opposite that induced during active sleep (reflex inhibition) (*2*). Thus, with the reticular site and level of stimulation remaining constant, the state of the animal determines the direction of effect. A resolution of the mechanisms underlying this state-dependent response and the importance of this reticular site in the control of motor activity during sleep must reside in an intracellular analysis of the synaptic events that result in motor facilitation and inhibition.

Seven animals were anesthetized with sodium pentobarbital (35 mg per kilogram of body weight), and standard electrodes were implanted for recording the electroencephalogram (EEG), electrooculogram (EOG), and electromyogram (EMG) (*2*). Stimulating bipolar strut electrodes were permanently placed in the mesencephalic nucleus of the fifth nerve and in the nucleus reticularis pontis oralis (*2*). A small circle (diameter, 5 mm) of occipital bone was removed by trephination in order to permit the subsequent penetration by a microelectrode through the cerebellum to trigeminal motoneurons. The trephined hole was filled with bone wax, which was removed during experimental sessions. The animals were allowed at least 1 week to recover from surgery (*3*).

During experimental sessions, after the penetration and identification of a trigeminal jaw-closer motoneuron with a micropipette filled with 3*M* KCl (tip resistance, 8 to 20 megohms), the spontaneous membrane potential of the motoneuron was correlated with polygraphic data indicating the state of the animal as one of quiet or active sleep. For certain cells, a short train of one to three pulses (interpulse interval, 2 msec) was delivered to the nucleus reticularis pon-

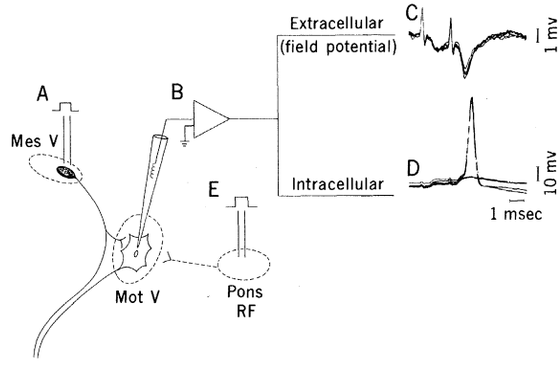

Fig. 1. Diagram of the basic stimulation and recording paradigm. The trigeminal motor nucleus (*Mot V*) was identified by monitoring its extracellular field potential (C) induced by stimulation (A) of the mesencephalic nucleus of the fifth nerve (*Mes V*). Jaw-closer motoneurons were identified by intracellular recording of the monosynaptic EPSP's and spike potentials of mesencephalic V origin (A, B, D). (E) Stimulation of the nucleus pontis oralis (*Pons RF*) (*4*).

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tis oralis at a rate of one pulse per second in order to examine the intracellular response of the motoneurons to reticular stimulation (Fig. 1) (*4*).

Stable intracellular recordings were made from 29 jaw-closer motoneurons for periods ranging from 5 to 20 minutes. Ten of the motoneurons were recorded during contiguous periods of quiet and active sleep; the remaining 19 were recorded during one or the other state. Resting potentials ranged from −40 to −70 mv.

The masseteric monosynaptic reflex is dramatically reduced in amplitude during active sleep compared with quiet sleep, and a further reduction in reflex amplitude occurs during the rapid eye movement periods of active sleep (*5*, *6*). These same patterns have also been reported for both monosynaptic and polysynaptic spinal reflexes (*7*). Thus, reflex reduction during active sleep is a pervasive motor pattern accompanying this state. The basis for this pattern of reflex modulation can be resolved by directly monitoring the level of membrane polarization, which we have now done in ten jaw-closer motoneurons during continuous epochs of quiet and active sleep. In all motoneurons, the membrane potential gradually became more polarized as the animal passed from quiet to active sleep (Figs. 2C and 4C). Tonic hyperpolarization (relative to quiet sleep) of 3 to 10 mv continued throughout the period of active sleep (Fig. 2C). This observation provides a final-common-pathway criterion for the muscular atonia characteristic of active sleep, first described by Jouvet and Michel in 1959 (*8*); it also presents direct evidence for the inference made by others that motoneuron hyperpolarization is the process responsible for motor suppression during active sleep (*7*).

Spontaneous subthreshold synaptic activity was also recorded (Fig. 2, D and E) and found to be similar in waveform to that of spinal motoneurons when homonymous and antagonistic muscles are stretched (*9*). In our cats, during quiet sleep, the motoneuron was constantly bombarded by a variety of spontaneous inputs whose frequency of occurrence decreased during active sleep (Fig. 2, D and E) (*10*). Therefore, active sleep is accompanied not only by tonic hyperpolarization of jaw-closer motoneurons but also by a concomitant decrease in subthreshold synaptic activity (Fig. 2, D and E) (*11*).

If subthreshold synaptic activity can be used as one possible criterion for determining synaptic bombardment in the region of the motoneuron soma (*9*), there

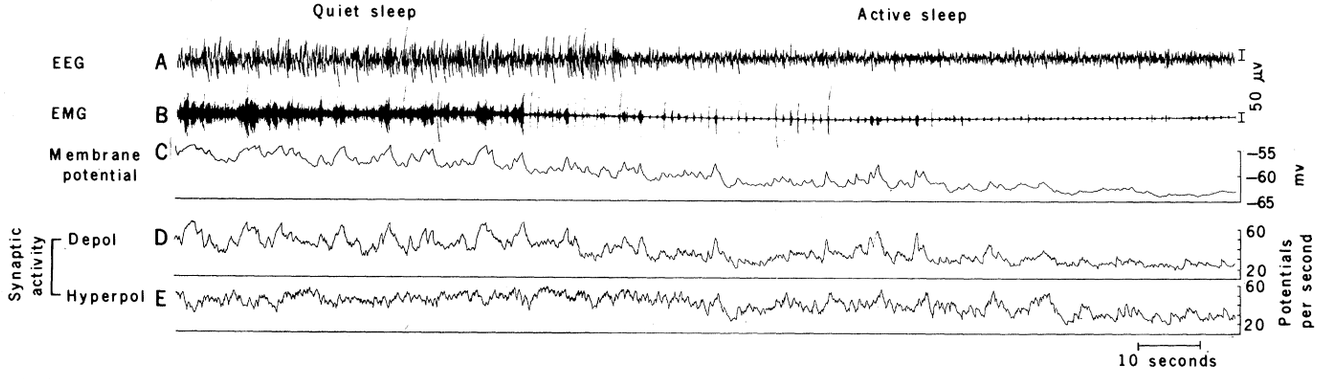

Fig. 2 (A through E). Development of motoneuron hyperpolarization as the animal passes from quiet to active sleep. Note the gradual increase in membrane polarization from a quiet sleep level of approximately −55 mv to −64 mv during active sleep. The frequency of subthreshold synaptic activity in the depolarizing (D) and hyperpolarizing (E) direction was less during active sleep than during quiet sleep. This decrease in synaptic activity in active sleep paralleled the increase in tonic hyperpolarization observed during this state.

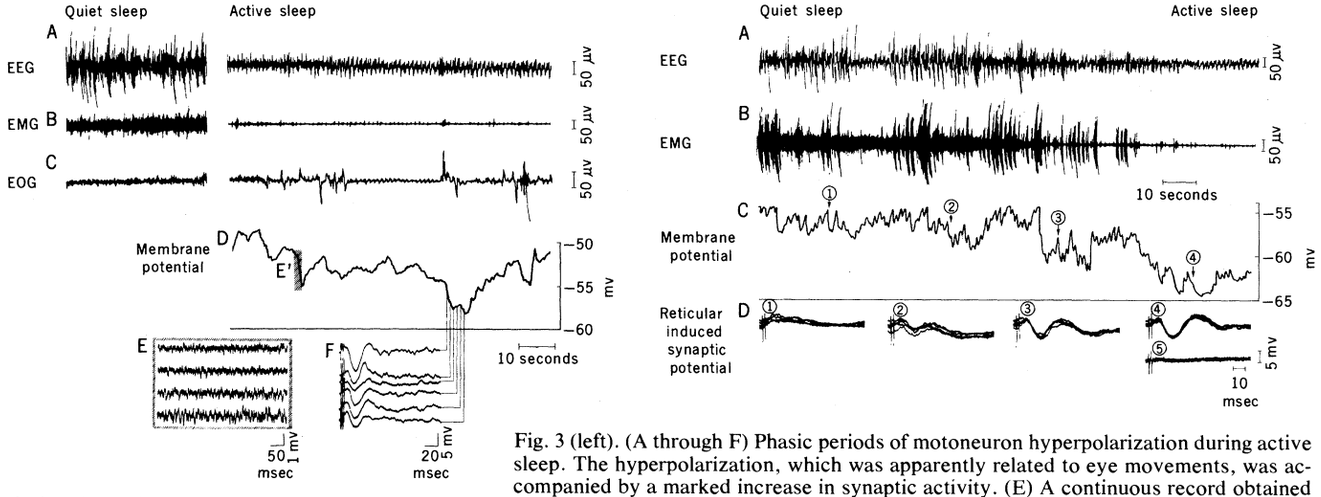

Fig. 3 (left). (A through F) Phasic periods of motoneuron hyperpolarization during active sleep. The hyperpolarization, which was apparently related to eye movements, was accompanied by a marked increase in synaptic activity. (E) A continuous record obtained during the periods of time indicated by the shaded area of *E'*; the intracellular record was filtered and amplified (*10*). During these periods, the hyperpolarizing response to reticular stimulation during active sleep was reduced in amplitude (F). Reticular stimulation: three pulses; 0.2-msec duration; 5 volts. Fig. 4 (right). (A through D) Effects of reticular stimulation. Note the tonic hyperpolarization of the motoneuron membrane as the animal passes into active sleep. (D) Reticular stimulation induced a depolarizing potential (D1) that was superseded by a gradually increasing hyperpolarizing potential (D2 through D4). The advent of the hyperpolarizing potential preceded the onset of active sleep EEG desynchronization. The record D5 represents the corresponding extracellular potential obtained during active sleep after the record D4 was obtained. Reticular stimulation: two pulses; 0.2-msec duration; 4 volts.

are at least three possible explanations for the mechanisms responsible for this decrease in activity during the tonic hyperpolarization of active sleep: (i) disfacilitation due to a decrease in excitatory synaptic bombardment of the soma; (ii) an increase in inhibitory dendritic synaptic bombardment, which could shunt dendritic excitatory postsynaptic potentials (EPSP's) and thereby decrease synaptic activity recorded in the soma (*12*); and (iii) an increase in somatic inhibitory input that impinges on the soma more asynchronously in active sleep than in quiet sleep and thus might not summate sufficiently to be observable as subthreshold synaptic potentials. However, an increase in such inhibitory inputs through axo-somatic inhibitory synapses is unlikely because tonic hyperpolarization of jaw-closer motoneurons resulting from sustained asynchronous input (for example, natural stimulation of the tongue) is accompanied by an increase in synaptic activity, which is chiefly induced through inhibitory synapses on the soma (*13*). The observed decrease in subthreshold synaptic activity, therefore, suggests that the tonic hyperpolarization occurring during active sleep may result from mechanisms involving disfacilitation, dendritic inhibitory postsynaptic potentials (IPSP's), or both.

A striking change in membrane activity also occurred phasically during active sleep. This change consisted of periods of phasic hyperpolarization of 1 to 3 mv superimposed on the tonic hyperpolarization (3 to 10 mv) of active sleep (Fig. 3D). In contrast to the decrease in synaptic potentials during the tonic hyperpolarization of active sleep, this phasic hyperpolarization was accompanied by an increase in subthreshold synaptic activity (Fig. 3, E and *E′* in D). On the basis of this increase in subthreshold synaptic potentials, somatic IPSP's may actively participate and contribute to these phasic periods of membrane hyperpolarization.

The effect of reticular stimulation was studied in 12 cells. Stimulation of the pontine reticular formation (in the vicinity of the nucleus reticularis pontis oralis) during quiet sleep induced a depolarizing potential in jaw-closer motoneurons with a latency of 2.5 to 5.0 msec and a peak from 10 to 15 msec (part 1 of Fig. 4D). The amplitude of the potential ranged from 1 to 3 mv. During the transition from quiet to active sleep, the depolarizing potential gradually became reduced in amplitude and was superseded by a hyperpolarizing potential with a latency of 9 to 15 msec and a peak at approximately 20 msec (part 2 of Fig. 4D). The hyperpolarizing potential gradually increased in amplitude and reached a plateau after the animal was well into active sleep (parts 3 and 4 of Fig. 4D). The peak amplitude of the potential was 5 to 7 mv. Coincident with the hyperpolarizing potential, spike potentials monosynaptically evoked from the trigeminal mesencephalic nucleus were inhibited. In conjunction with the phasic hyperpolarization of the motoneuron during active sleep, the hyperpolarizing potential induced by the pontine reticular formation was depressed (Fig. 3F). During the descending phase of the spontaneous hyperpolarization, subthreshold synaptic activity increased (Fig. 3, E and *E′* in D).

The findings indicate that the previously reported reticular-induced reflex facilitation of quiet sleep is due to the advent of a depolarizing potential. During active sleep the process becomes one of membrane hyperpolarization, which accounts for the reflex suppression observed during this state (*5*). Thus, the pattern of membrane potential modulation represents the basis for the paradoxical phenomenon of reticular response reversal (*2*).

These studies have allowed us to examine the synaptic mechanisms underlying two basic sleep phenomena—somatic reflex depression during active sleep and the control of motor reflex activity by the reticular formation. We have found that intracellular analyses can provide pivotal data for the resolution of basic properties of sleep behavior. These studies set the foundation for further detailed analyses of the phenomenon of response reversal as well as other aspects of sleep and behavior as revealed by the intracellular analysis of neuronal circuitry and membrane activity in behaving animals.

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3. In order to perform these experiments in animals that were unanesthetized and that exhibited normal physiological patterns of activity, trigeminal motoneurons had to be identified and penetrated by a microelectrode, and stable recordings had to be obtained for contiguous periods of quiet and active sleep. During surgery, while the animal's head was stereotaxically positioned, four receptacles for stereotaxically mounted calibrated bars were affixed to the calvarium with dental cement. During the experimental session, bars were inserted into the receptacles and attached to the stereotaxic instrument according to the calibration determined at the time of implantation; the animal's head could thus be rigidly held in a stereotaxically defined plane without any pain or pressure. After each animal had recovered from surgery, it was placed in the stereotaxic apparatus by means of the "false" head holder described. The bone wax was removed from the trephined hole, and a glass micropipette was lowered through the cerebellum to the motor nucleus of the fifth cranial nerve. Activity (EEG, EOG, and EMG) was recorded polygraphically and on magnetic tape along with the intracellular potentials.
4. Trigeminal motoneurons that innervate the jaw-closing muscles (jaw-closer motoneurons) were identified by the following criteria. (i) They were located in the region estimated stereotaxically to be the dorsal part of the trigeminal motor nucleus where motoneurons that innervate the masseter and temporal muscles are situated [N. Mizuno, A. Konishi, M. Sato, *J. Comp. Neurol.* **164**, 105 (1975); C. Batini, C. Buisseret-Delma, J. Corvisier, *J. Physiol. (Paris)* **72**, 301 (1976); S. Landgren and K. A. Olsson, *Exp. Brain Res.* **26**, 299 (1976)]. In this region, usually 16 to 18 mm below the cerebellar surface, stimulation of the ipsilateral trigeminal mesencephalic nucleus evoked a monosynaptic negative field potential with a latency of approximately 0.6 msec and an amplitude of 2 to 6 mv (Fig. 1C). (ii) Monosynaptic EPSP's evoked by stimulation of the mesencephalic nucleus were recorded in these cells with latencies of 0.5 to 0.7 msec [Y. Nakamura, L. Goldberg, C. Clemente, *Brain Res.* **6**, 184 (1967); Y. Kidokoro, *J. Neurophysiol.* **31**, 695 (1968)]. For all studies, extracellular as well as intracellular recordings were made in the same region in the trigeminal motor nucleus during quiet and active sleep.
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11. This is in direct contrast to the observation that

tonic hyperpolarization of the jaw-closer masseteric motoneuron in the decerebrate cat, associated with asynchronous activation of cutaneous trigeminal afferents, is accompanied by an increase in synaptic activity (*12*). Hyperpolarization of jaw-closer motoneurons induced by this pattern of stimulation of mucosal afferents is accomplished by a postsynaptic inhibitory mechanism, in which the synapses that mediate the inhibitory postsynaptic potential are located in the region of the soma (*4*, *12*).

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Pontine Reticular Formation Neurons and Motor Activity

The conclusion of Siegel and McGinty (*1*) that "activity in pontine reticular formation neurons is more closely related to motor output than to sensory input" is correct but incomplete. They overlook an obvious hypothesis which explains their findings, namely, that the pontine reticular formation (PRF) unit activity is related to eye movement. There is an extensive body of work which shows that the PRF contains the neural mechanisms for producing conjugate slow and rapid horizontal eye movements. This conclusion is based on analysis of PRF lesions in humans (*2*) and animals (*3*), on gross potential changes associated with rapid eye movements in the PRF (*4*), on stimulation studies of the PRF (*5*), and on the analysis of the activity of single nerve cells in the PRF of the alert cat, rabbit, and monkey (*6*). Henn and I (*7*) have shown that PRF cells which fire before and during rapid eye movements code the necessary activity to induce these eye movements. This activity is organized according to a polar coordinate system with individual cells coding a representation of overall amplitude of eye movement, of direction of eye movement, and the components of eye movement in the pulling planes of the individual eye muscles. "Blink" units are also found in the PRF and are probably similar to the units that Siegel and McGinty describe as "flinch" units.

Other areas of the brainstem including the vestibular nuclei (*8*), the prepositus nucleus, and the adjacent medullary reticular formation (*9*) are also known to have cells whose activity is related to eye movements. These regions project to neck motoneurons. Consequently, individual units might be expected which would fire in association with head movement.

In short, Siegel and McGinty appear to have reaffirmed previous work which shows that motor activity is widely represented in PRF neurons. What they apparently failed to realize is that this activity is predominantly related to eye movement. Representation of head movement in some PRF neurons might also be expected, as shown in figure 1D of their report; and perhaps that is a new finding. However, before accepting this conclusion, one would have to be certain that the cats were not looking at the body part they were grooming.

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12 May 1977

Activity in cat pontine reticular formation (PRF) cells is not, as Cohen suggests, "predominantly related to eye movement." We tested for eye movement relations in every cell we encountered, polygraphically recording and visually observing eye movements while monitoring unit discharge. We also tested for unit activity correlated with eye blinks elicited by corneal stimulation in all cells. A number of cells related to eye movement were observed, but histological analysis localized these cells to the region of the abducens nucleus, in agreement with previous studies in the cat (*1*). The gigantocellular tegmental field (FTG) units that we identified as head movement cells, the most common cell type, all showed intense discharge without any eye movement. Conversely, rapid eye movements (REM) and maintained eye positions in both the horizontal and vertical planes without accompanying unit discharge were observed in each of these cells. Since head movements tend to be associated with eye movements these cells do show a general correlation with eye movements. Cells specifically related to eye movement may exist in the PRF (*2*), but clearly they are not the predominant cell type in the FTG area, which comprises most of the PRF.

Several other findings illustrate the lack of relationship between unit activity in most FTG cells and eye movement. (i) During adaptation to head restraint, FTG unit firing decrement correlated closely with decrease in neck electromyogram (*3*, *4*), not electrooculogram (EOG). (ii) Most cells habituated to rapid head acceleration in conjunction with changes in neck muscle tone. However, EOG response to such stimulation does not habituate. (iii) Operant conditioning of increased firing rate in those FTG cells which appeared to discharge in relation to head movement was accompanied by repetitive head movements. In no case did we observe a conditioned increase in unit firing correlated only with increased eye movements. (iv) During REM sleep many of these cells discharge in long intense bursts. This firing does not result from increased numbers of eye movements (*5*, *6*). (v) Many FTG cells were found to be entirely unrelated to head or eye movement. We have observed cells which discharge in close relationship to directionally specific tongue movements. Other cells exhibited activity related to facial musculature and to specific postures (*4*). It would be difficult to reconcile such findings with the claim that FTG cells relate predominantly to eye movement.

In monkeys, eye movement cells are not uniformly distributed throughout the PRF, but tend to be restricted to dorsomedial regions (*7*). Similarly, neurons related to vestibular nystagmus in the cat are not distributed throughout the PRF, but rather are sharply localized to dorsomedial regions, especially the area caudal to the abducens nucleus (*2*). While some connections exist (*8*), horseradish peroxidase studies have not revealed a major projection from the FTG region to the oculomotor nuclei (*9*).

Stimulation in the PRF produces com-

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