

<i>Rp</i>	1.37	1.43	1.47	1.50	
2,4-D treatment	0	320 ppm	80 ppm	20 ppm	5 ppm
Mean (mg)	2.3	2.8	3.9	4.0	4.3

<i>Rp</i>	5.79	6.08	6.24	6.34	6.44	
2,4-D treatment	10 ppm	0	200 ppm	100 ppm	20 ppm	40 ppm
Mean	11.00	12.60	14.00	14.60	17.60	19.20

The results of this investigation demonstrate that increased risks of attack by insects and disease on corn may result from herbicide treatments. Additional

*Department of Entomology and Section  
of Ecology and Systematics, Cornell  
University, Ithaca, New York 14853*

recurrent discharge occurs in the absence of observable fluctuations in either sensory stimulation or motor activity. The existence of spontaneous activity in the neurons of behaving animals has been assumed in most theoretical formu-

	Cells (No.)	Duration (seconds)		
		Waking	SWS	REM sleep
NSA	27	166.8 ± 19.6	383.4 ± 57.9	321.4 ± 39.4
Midbrain raphe	10	5.8 ± 2.0	16.5 ± 3.3	44.1 ± 7.5
Pontine FTG	10	48.0 ± 8.1	91.2 ± 20.3	21.4 ± 8.7
Duration of state	27	179.4 ± 20.0	428.2 ± 58.7	480.0 ± 40.3

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lations of brain function (2, 3). In the course of recording brainstem units in unrestrained cats, we discovered a group of phasically active neuronal units that did not exhibit spontaneous activity in either waking or sleep. Further investigation revealed that such units were relatively common in a large region of the brainstem.

We located neurons that were not spontaneously active (NSA cells) by applying systematic sensory stimulation while advancing microwire electrodes in search of unit activity. The 32- $\mu$ m microwires were grouped in bundles of seven, attached to mechanical microdrives, and positioned in pontine or midbrain tegmental sites (4) in eight female cats. Macroelectrodes were also implanted, and records were scored for sleep state according to standard criteria (5). During recording the cats were entirely unrestrained in a shielded cubicle. Only units with signal-to-noise ratios greater than 4 : 1, good isolation from other units, and stable spike amplitudes were studied. To ensure that recordings were stable, we measured spike amplitude and wave-shape at the beginning and end of each recording period. All cells were recorded for at least one complete sleep cycle [waking, slow-wave sleep, rapid eye movement (REM) sleep, waking].

Of the 104 cells recorded, 27 were classified as NSA neurons, defined as those cells which (i) showed no activity when not appropriately stimulated during waking and (ii) remained silent for intervals exceeding 60 seconds during both REM and slow-wave sleep (Table 1). For comparison, Table 1 also includes measurements on two other groups of neurons. The first group consists of ten dorsal raphe neurons, a cell type whose slow discharge during waking and slow-wave sleep has been stressed (7). The NSA units show considerably longer silent periods than either of these cell types. Five of the NSA cells exhibited silent periods that extended over two or more slow-wave and REM sleep cycles and lasted up to 43 minutes without a single spike discharge. Those NSA cells with shorter silent periods typically had successive silences separated by a single spike discharge.

When activated by appropriate stimuli, the cells discharged at rates exceeding 50 spikes per second (Fig. 1). With continuous stimulation, spike trains lasting more than 90 seconds were observed. A detailed analysis of the correlates of dis-

charge was made in ten of the pontine NSA cells (8). Five responded only to vestibular stimuli, four only to somatosensory stimuli, and one responded only during tongue extension. Seven of the ten cells showed rapid habituation if their adequate stimuli were repeated. The heterogeneity of response correlates and the anatomical distribution of NSA cells suggest that they are not constituents of a single neural network.

Virtually all neurons described pre-

viously showed considerable activity during sleep (1). The nearly complete lack of spontaneous discharge during sleep by the NSA cells is unique. In two cases, somatosensory NSA units were activated during sleep when their receptive fields were in contact with a part of the cat's litter box. This observation suggests that the total lack of tonic sleep activity in NSA cells is a passive consequence of reduced stimulation. Thus, while the reduction in the activity of the NSA units dur-

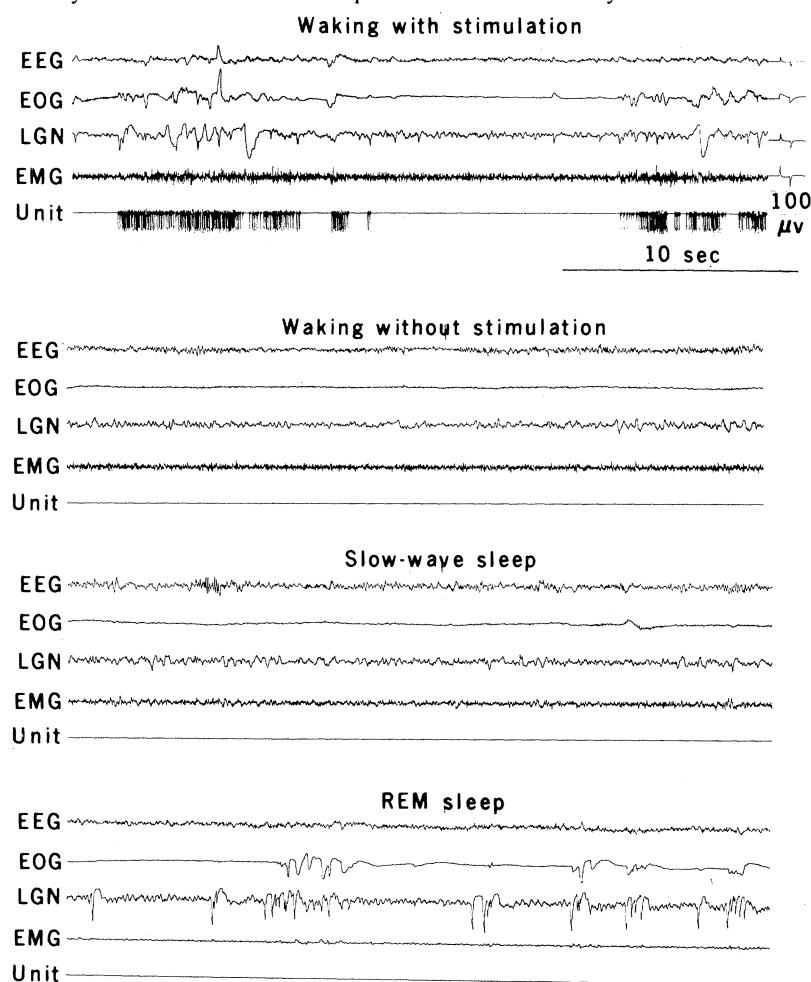


Fig. 1. Activity of a representative NSA unit during waking and sleep. During waking, when the proper stimulus is present, the cell discharges in sustained high-frequency bursts. During waking without stimulation, slow-wave sleep, and REM sleep, the unit is silent. Abbreviations: EEG, electroencephalogram; EOG, electrooculogram; LGN, lateral geniculate nucleus; and EMG, electromyogram. The unit channel displays the pulse output of a window discriminator.

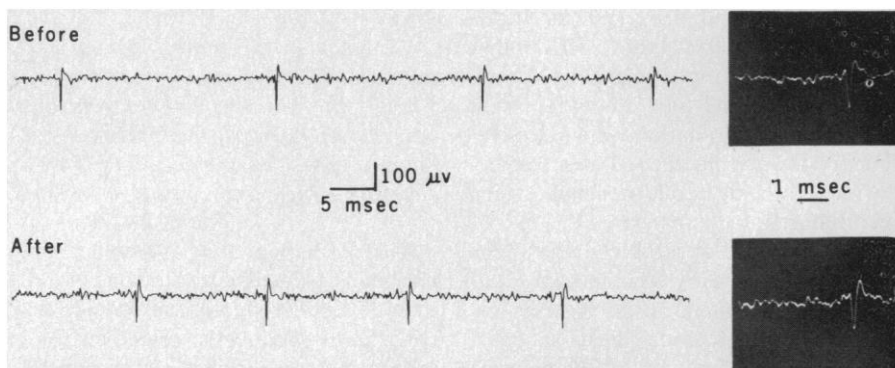


Fig. 2. Unit activity before and after a 23-minute silent period. Note the stability of spike amplitude and wave-shape. The low-speed tracings are computer plots of digitized data.

ing sleep is consistent with the occurrence of a recuperative process in this group of neurons (9), the lack of discharge during sleep seems to result from the behavioral inactivity imposed by sleep, rather than some specific neural inhibitory process.

One might hypothesize that the extended silences resulted from electrode movement that caused loss of unit recording. We can reject this hypothesis. (i) Our recording technique allowed stable recording for extended periods of time in a variety of brainstem areas (10). (ii) In every case, the NSA unit spike trains were recorded both before and after silent periods (Fig. 2). In 12 of these neurons, continuous recordings lasting more than 8 hours and including several sleep-waking cycles were obtained. Spike waveshape, signal-to-noise ratio, discharge patterns, and the unique behavioral correlates of discharge were always stable throughout the period of observation. (iii) All of the NSA units had large signal-to-noise ratios and stable spike amplitudes. In no case was a change in spike amplitude observed at the beginning or end of a silent period.

We have encountered these cells in histologically verified sites in midbrain regions (AP 1.0 to 3.0, ML 0.0 to 0.2, DV -0.8 to -3.2) and in the pontine reticular formation (AP 3.0 to 8.0, ML 1.0 to 2.8, DV -3.6 to -7.0). After we were alerted to the existence of NSA cells, about 30 percent of the cells that were encountered were of this type. However, the percentage of these cells in the brain is difficult to estimate accurately. Many of the cells fired only in sporadic bursts and could easily have been overlooked. Cells with more subtle sensory or motor correlates would not have been activated by our simple stimuli. Furthermore, the concentration of NSA units may not be the same in all brain regions, although the frequencies of encounter in the midbrain and pontine regions did not differ.

It was necessary to apply systematic stimulation while exploring for unit activity in order to find NSA neurons. If this were done in other brain areas, other types of NSA units might be found. Adams (11) found four otherwise silent cells in the midbrain that were selectively activated during elicited affective behavior, although he did not make sleep recordings.

The existence of NSA cells has been predicted in work by Vladimirova *et al.* (12), who calculated, on the basis of histological and electrical field analysis, that fewer than 5 percent of the neurons within range of their cortical microelectrodes showed spontaneous activity.

These findings, coupled with the results reported here, suggest that NSA cells constitute a large proportion of neurons in the brain.

The idea that neurons are spontaneously active is incorporated in a wide range of theories of brain function (2). The brain's information processing has been conceptualized as a system for extracting signals from the background noise of spontaneous activity (3). The existence of large numbers of NSA neurons allows alternative formulations of these theories. The specificity of discharge in these neurons suggests the existence of specialized neural systems operating phasically in relation to specific sensory or motor events. Such systems might have considerably less ambiguity in their output than systems solely employing neurons with high levels of spontaneous activity.

JEROME M. SIEGEL

*Neurophysiology Research, Veterans Administration Hospital, Sepulveda, California 91343, and Department of Anatomy, University of California, Los Angeles 90024*

DENNIS J. MCGINTY

*Neurophysiology Research, Veterans Administration Hospital, Sepulveda, and Department of Psychology, University of California, Los Angeles*

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8. Visual stimuli included a light directed into each eye, moved in the horizontal, vertical, and oblique planes, toward and away from the cat, and flashed on and off. Auditory stimuli included clicks presented above, below, and to the left and right of the cat's head. Vestibular stimuli included passive head acceleration at speeds ranging from 90 to 360 deg/sec in the vertical and horizontal axis. Units showing a response to vestibular stimulation discharged even at slower accelerations, although more rapid movement produced a brisker response. We found two units that responded solely to dorsal head acceleration and two that fired to either dorsal or ipsilateral acceleration. Kinesthetic stimuli included movement and maintained displacement of all four limbs and their joints, and of the head, neck, and jaw. Somatic stimuli included light and deep pressure applied to the ears, lips, tongue, vibrissae, neck, trunk, and limbs. We found somatosensory units that specifically responded to (i) deep pressure on the ipsilateral ear, (ii) deep pressure on the ipsilateral neck, (iii) light pressure on the ipsilateral vibrissae, and (iv) deep pressure in the region of the ipsilateral vibrissae. Unit activity was also monitored during spontaneous and elicited eye movements, eating, drinking, and other behaviors.
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## Memory: Proline Induces Retrograde Amnesia in Chicks

**Abstract.** *Intracerebral injection of L-proline, 1 minute after one-trial avoidance training of chicks, impaired memory 24 hours later. Chicks injected with proline 1 or 4 hours after training and controls injected with L-isoleucine at intervals after training, showed no impairment of memory 24 hours after training. The retrograde impairment of long-term memory induced by proline occurred without the convulsions or electrographic events usually associated with retrograde amnesic treatments.*

The formation of long-term memory has been assumed to include one or more time-dependent steps. Interference with early steps produces a selective loss of recent memory, called retrograde amnesia (1). The treatments commonly used to induce experimental retrograde

amnesia (2) are deleterious agents such as electroconvulsive shock (ECS), chem- oconvulsants, anesthetics, and protein synthesis inhibitors, which have multiple and widespread actions. A more physiologically selective chemical treatment would be desirable for tracing the dis-