Exp Brain Res (1988) 70: 463–469

Experimental Brain Research

Sustained excitatory synaptic input to motor cortex neurons in awake animals revealed by intracellular recording of membrane potentials

M. Matsumura[1], T. Cope[2], and E. E. Fetz

Department of Physiology and Biophysics, and Regional Primate Research Center, University of Washington, Seattle, WA 98195, USA

Summary. 1. Most of the intracellular electrophysiological data on cortical neurons has been obtained in anesthetized or reduced preparations, and differs from observations in awake, intact animals. To determine whether these differences are due to experimental techniques or physiological factors, we recorded membrane potentials intracellularly from motor cortex neurons in chronically prepared cats and monkeys under Nembutal-anesthetized, Halothane-anesthetized, and unanesthetized conditions, or during transitions between anesthetized and awake conditions. 2. Resting membrane potentials were found to depend on the anesthetic state of the animal. Membrane potentials of neurons recorded in awake animals were more depolarized than those recorded in the anesthetized state. In the awake state membrane potentials were all less than –65 mV. 3. The input resistance of neurons recorded in awake animals were significantly smaller than those measured in the anesthetized state. Action potentials recorded in awake animals typically showed an undershoot (i.e. negative values at peak), implying that voltage-dependent conductances may be altered. Undershoot of the action potential was more prominent in pyramidal tract neurons (PTNs) than non-PTNs. 4. These data suggested that in awake animals motor cortex neurons, especially PTNs, receive sustained excitatory synaptic input or neuro-modulatory activities.

Key words: Motor cortex – Sustained excitation – Intracellular recording – Membrane potential

Introduction

Membrane potentials recorded from neurons in the CNS of higher mammals appear to be significantly different for anesthetized and unanesthetized animals. Levels of anesthesia have been suggested to affect neuronal firing threshold as well as membrane potentials (Sasaki and Otani 1962). In deeply anesthetized cats the membrane potentials were reported to be –70 mV or more in spinal motoneurons (Eccles 1964) and in Betz cells of motor cortex (Phillips 1956). Recording in chronic awake animals, experimenters have rarely encountered such large membrane potentials, either in cortical neurons (Brons and Woody 1980; Woody and Gruen 1978), or in motoneurons (Chase et al. 1980, Glenn and Dement 1981, Matsumura and Woody 1986). These observations could indicate that in awake animals neurons receive sustained excitatory or disinhibitory input from presynaptic sources; alternatively, the tonic conductance of specific ionic channels may be affected by anesthetics. However, membrane potentials recorded in these different studies could not be compared directly because different investigators have used different types of electrodes and/or different acceptance criteria for successful intracellular recording. To eliminate these sources of variance, we compared membrane potentials in the same population of motor cortex neurons with identical recording conditions and acceptance criteria, under different anesthetic states. Preliminary results were reported elsewhere (Matsumura 1981).

Material and methods

Relevant data was obtained in two different series of experiments, in cats and monkeys.

Present addresses: [1] Department of Neurophysiology, Primate Research Institute, Kyoto University, Inuyama, Aichi 484, Japan

[2] Department of Cell Biology and Anatomy, University of Texas, Health Science Center at Dallas, Dallas, TX 75235, USA

Offprint requests to: E. E. Fetz (address see above)

Experiment I

Seven cats (weighing 2.8–4.0 kg) were used for a comparative study of membrane potential of motor cortex neurons in different states. In preparation for chronic recording sessions cats were anesthetized with Nembutal (sodium-pentobarbital, 35 mg/kg, i.p.) and craniotomized. A concentric bipolar stimulating electrode was implanted into the medullary pyramidal tract (PT) to identify PT-neurons. Two tubes for head fixation were cemented to the skull. The bone covering motor cortex (area 4 gamma) was exposed by opening the frontal sinus, and covered by dental acrylic cement or bone wax. Details of this surgery has been reported previously (Woody and Black-Cleworth 1973).

After ample recovery time from the surgery (usually 7 days), the cat was restrained for recording by enclosing its body, except for the head, in a linen towel. The head was attached to a stereotaxic frame (David Kopf) by four rods attached to the implanted tubes. Under Halothane anesthesia, the bone wax or dental cement covering the bone over motor cortex was removed. A small hole was drilled in the skull, and the dura was incised by a fine hyperdermic needle.

Glass microelectrodes were made from 1.0 mm omega-tubing (W.P.I.) with a vertical pipette puller (David Kopf). The tips of the electrodes were beveled until the electrode resistance dropped to 12–25 MΩ with 3 M KCl. The recorded signal was led to a high input impedance amplifier (WPI) and stored on an FM tape recorder (0–3 KHz). A ten millisecond constant-current pulse was injected intracellularly to monitor the input resistance of the recorded neurons.

Recording sessions consisted of two or three successive stages of anesthesia and waking. In three cases recording was started with the Halothane anesthetized state (1% Halothane + 1 l/min nitrous oxide + 2 l/min oxygen). After 2 h of intracellular recording of cortical neurons, Halothane administration was stopped and the cat was allowed to recover from anesthesia for at least 30 min, and recording continued. In the remaining four cats, recording was started with the cat awake. After 2–3 h of intracellular recording, the cats were deeply anesthetized with Nembutal (35 mg/kg, i.p.), and additional cells recorded in the same cortical area.

Criteria for intracellular recording were identical for all recording sessions, as follows;

1. abrupt DC shift of more than 40 mV on penetration,
2. discharge without broadening of action potential,
3. stable resting potential for at least 30 s.

Procedures usually applied in acute experiments to maintain intracellular recording for long times, e.g., pneumothorax and artificial ventilation, were precluded in this experiment. All surgical procedures were performed under full anesthesia; restrains and recordings in awake cats were painless, and cats showed no signs of stress or discomfort. Animal treatment was supervised by professional veterinarians under the "Guide for the Care and Use of Laboratory Animals" by NIH (1978), and were in accordance in the current guidelines.

Experiment II

In two macaque monkeys (weighing 4.2 and 5.2 kg), intracellular recordings were obtained from precentral cortex neurons during transient periods of anesthesia; the animals were briefly anesthetized with doses of ultra-short acting barbiturate (Brevital) or Halothane gas.

Preceding the experiment, monkeys were trained to perform a behavioral task (alternate flexion and extension of the wrist), and to tolerate sitting in a primate chair. After recovery time of at least a week following surgery to implant a head fixation device, intracellular recording were made. All surgical procedures for recording were similar to Experiment I.

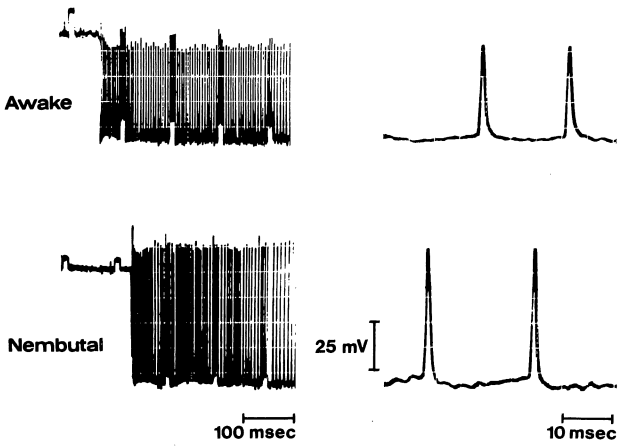

Fig. 1. Comparison of intracellular recordings from motor cortex neurons in awake (upper) and Nembutal anesthetized (lower) cat. Records show the moment of penetration (left) and membrane potential 30 s later (right). Note that in the awake condition the membrane potential is more depolarized and the action potentials are smaller. Depolarizing current pulses of 1 nA, 10 ms duration were applied every 100 ms. (Note: in these records the bridge was not balanced before the penetration)

After intracellular recordings were obtained in the awake condition, the monkeys were anesthetized with Halothane gas (1–1.5%), mixed with Nitrous oxide (1 l/min) and oxygen gas (3 l/min), to investigate the alteration of the membrane electrical properties during induction of anesthesia. In other cases, intracellular recordings were first obtained under Halothane anesthesia, and then Halothane was discontinued to document the changes during the recovery from anesthesia. In several cases, Brevital (50 mg/kg, i.p.) was administered instead of Halothane. Recording conditions and criteria for intracellular recording were the same as Experiment I.

Results

Experiment I: cat

Membrane potentials under different anesthetic conditions. A total of 115 neurons (35 in awake, 21 in Halothane anesthetized and 59 Nembutal anesthetized conditions) which met the criteria for intracellular recording were obtained from 10 hemispheres of 7 cats. Figure 1 shows representative examples of intracellular recording from the same cat, when awake (top) and anesthetized with Nembutal (bottom). Typical records at the moment of cell penetration, at left, show an abrupt DC potential shift, followed by steady initial discharge for up to 30 s. The right records show subsequent action potentials on a faster sweep. These records illustrate the two major differences observed under these conditions. 1) The neuron recorded in the awake state had a smaller (i.e. more depolarized) membrane poten-

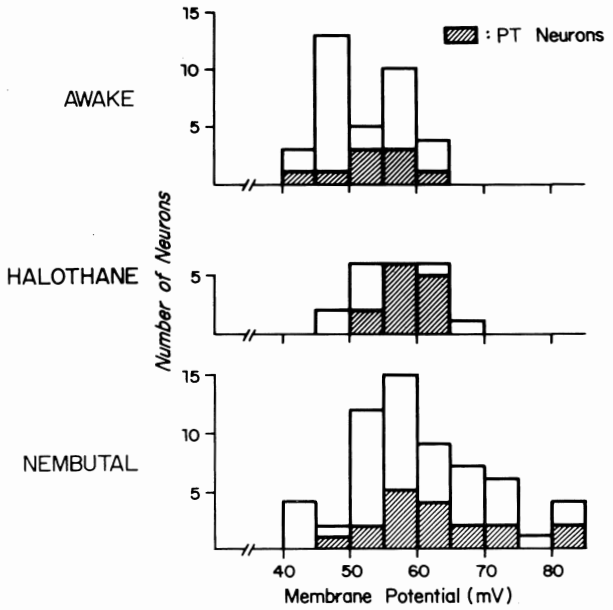

Fig. 2. Comparison of resting membrane potentials of motor cortex cells under three different anesthetic conditions: awake (upper), Halothane anesthetized (middle) and Nembutal anesthetized (lower). PT-neurons are indicated by hatched bars. In all cases membrane potential was measured within 30 s after penetration. Note absence of neurons with membrane potentials above 65 mV in the awake group

tial than the neuron recorded under Nembutal anesthesia (in this case, –51 mV vs. –57 mV). 2) The neuron in the awake condition exhibited smaller action potentials than that recorded under Nembutal anesthesia (–6 mV vs. +11 mV).

Some neurons recorded under Halothane or Nembutal anesthesia showed further increases in resting membrane potential (becoming more hyperpolarized) after stable recording over several minutes, whereas membrane potentials observed in neurons penetrated in the awake condition stayed at the same level. This increase of membrane potential might be attributed to recovery from an injured state, or other factors that could not be controlled in this experiment. However, to compare the membrane potential of neurons recorded in different anesthetic conditions under the same circumstances, and avoid any bias from such long term changes, the values of membrane potential were measured during the same period of recording, approximately 30 s after the moment of penetration, just after initial discharges had ceased.

Figure 2 compares the magnitudes of membrane potentials obtained under these 3 different anesthetic conditions. The difference of the mean membrane potentials during awake and Nembutal anesthetized conditions (8 mV) was statistically significant ($t = 4.18$, $p < 0.001$). The difference between awake and Halothane anesthetized conditions (4 mV) was also significant ($t = 2.63$, $p < 0.02$).

One striking difference in the membrane potential distributions among these conditions was the lack of larger membrane potentials in the awake condition, in contrast, the distributions of shallower potentials were quite similar among these three conditions. In particular, no neurons of more than –65 mV could be observed in the awake animals, whereas one-third of the neurons recorded in Nembutal anesthetized cats had membrane potentials exceeding –65 mV.

The mean resting membrane potentials recorded under different conditions are summarized in Table 1. PT-neurons seemed to exhibit a greater difference between mean membrane potentials in awake and anesthetized states than that for non-PT-neurons or

Table 1. Mean resting membrane potentials and action potential peaks (±SD) of motor cortex neurons in cat in three different states of anesthesia

		Awake	(n)	Halothane		Nembutal	
Resting	potential						
	PTNs	–53.4 ± 5.2	(9)	–57.8 ± 3.8*	(13)	–62.7 ± 9.5**	(18)
	non-PTNs	–51.4 ± 4.6	(10)	–54.1 ± 7.3	(8)	–54.2 ± 9.7	(11)
	unidentified	–52.2 ± 6.9	(16)		(0)	–60.5 ± 9.9**	(30)
	Total	–52.3 ± 5.7	(35)	–56.4 ± 5.4*	(21)	–60.1 ± 9.9***	(59)
Action	potential (note 1)						
	PTNs	– 5.6 ± 6.5	(9)	– 1.2 ± 6.8*	(13)	+ 4.1 ± 7.0**	(18)
	non-PTNs	– 1.1 ± 7.9	(10)	– 2.2 ± 11.2	(8)	+ 2.2 ± 6.6	(11)
	unidentified	+ 1.6 ± 7.0	(16)		(0)	+ 4.8 ± 11.0	(30)
	Total	– 0.9 ± 7.4	(35)	– 1.6 ± 8.5	(21)	+ 4.5 ± 8.3*	(59)

(Note 1) Membrane potential at the peak of action potential
* difference from awake condition was significant at $p < 0.05$ by t-test
** difference from awake condition was significant at $p < 0.01$
*** difference from awake condition was significant at $p < 0.001$

unidentified neurons. For PT-neurons the differences of membrane potential between awake and Nembutal anesthetized conditions (10 mV) and between awake and Halothane anesthetized conditions (5 mV) were statistically significant ($p < 0.01$ and $p < 0.02$, respectively).

Similar differences were observed for recordings obtained under different anesthetic conditions from the same cortical area (within 2 × 2 mm), using the same micro-electrode on the same day. In two cases when intracellular recordings were obtained for more than 20 neurons on a same day, the mean values for Nembutal anesthetized and awake conditions differed significantly ($p < 0.02$).

Conductance changes of motor cortex neurons in different anesthetic conditions

Input resistance of the cortical neurons were measured by the bridge balance method, using 1 nA, 10 ms depolarizing current pulses applied through the recording electrode. Mean resistances were 6.0 ± 3.9 MΩ in awake, and 9.6 ± 4.7 MΩ in Nembutal anesthetized conditions. The difference between input resistances for awake and Nembutal anesthetized conditions was statistically significant ($p < 0.02$).

A conductance difference between the two states is also suggested by the difference in the peak of the action potential (Fig. 1). Table 1 gives the absolute membrane potentials at the peaks of the action potentials observed in awake, Halothane and Nembutal anesthetized conditions. The majority (75%) of neurons recorded in the Nembutal anesthetized condition showed classical features of 'spike overshoot', i.e. action potential peaks greater than 0 mV. However, neurons recorded in awake cats more frequently exhibited undershoots. This tendency was more striking in PT-neurons. For PT-neurons peak potentials obtained under Halothane anesthesia showed a distribution intermediate between those for awake and Nembutal anesthetized conditions. The difference of peak potentials between PT-neurons in awake and Nembutal anesthetized conditions was statistically significant ($p < 0.01$).

Experiment II: monkeys

Intracellular recording from 8 motor cortex neurons were obtained in two monkeys during the transition periods between anesthesia and waking, both for induction and recovery. In both directions of anesthetic transitions, slow rhythmic bursts of action potential (0.2–2 Hz) were observed, as previously observed with extracellular recording (Deschenes and Domich 1983).

These bursts were observed in transition periods with both Halothane and Brevital anesthesia. Figure 3 shows a representative example of intracellular recording from a PT-neuron during recovery from Brevital (150 min after administration). Although recovery was not monitored by EEG and EMG recordings, the monkey opened its eyes and occasionally raised its hand; however, recovery of motor activity was not sufficient to perform the movement task. As illustrated in Fig. 3a, b, action potential occurred in bursts. A characteristic phenomenon during this period was transient rhythmical cycling of membrane potential, between hyperpolarization and depolarization, with a consistent potential difference of 8 mV. As seen in the lower DC recording, burst activity was superimposed on the depolarized episodes of the membrane potential. In addition, more synaptic noise occurred during the depolarized phases (sections labeled A) than the hyperpolarized periods (labeled B). This alternation of the membrane potential was observed in all neurons exhibiting burst activity, and was not synchronized with the phases of respiration. These depolarization shifts ranged from 4 to 10 mV, valus comparable to the difference of mean membrane potential observed in awake and anesthetized cats.

In five of these eight neurons, intracellular recording were maintained for a sufficiently long period (more than 5 min) to observe the recorded membrane potential during recovery from Halothane anesthetized to awake states (n = 3) or induction of anesthesia (n = 2). Figure 3c, d illustrates sections from continuous recordings from the same cells, indicating that both the membrane potential and action potential were smaller when the monkey was awake. Starting from the awake state (Fig. 3c), the membrane potential and action potentials became larger after the monkey was anesthetized by Halothane. These neurons showed a consistent membrane potential shift of 3 to 8 mV towards depolarization in awake state, regardless of the direction of the transition between awake and anesthetized states.

Discussion

Depolarized levels of membrane potential in awake preparation

Membrane potentials of motor cortex neurons measured in deeply anesthetized preparations and in awake preparations, during the past three decades,

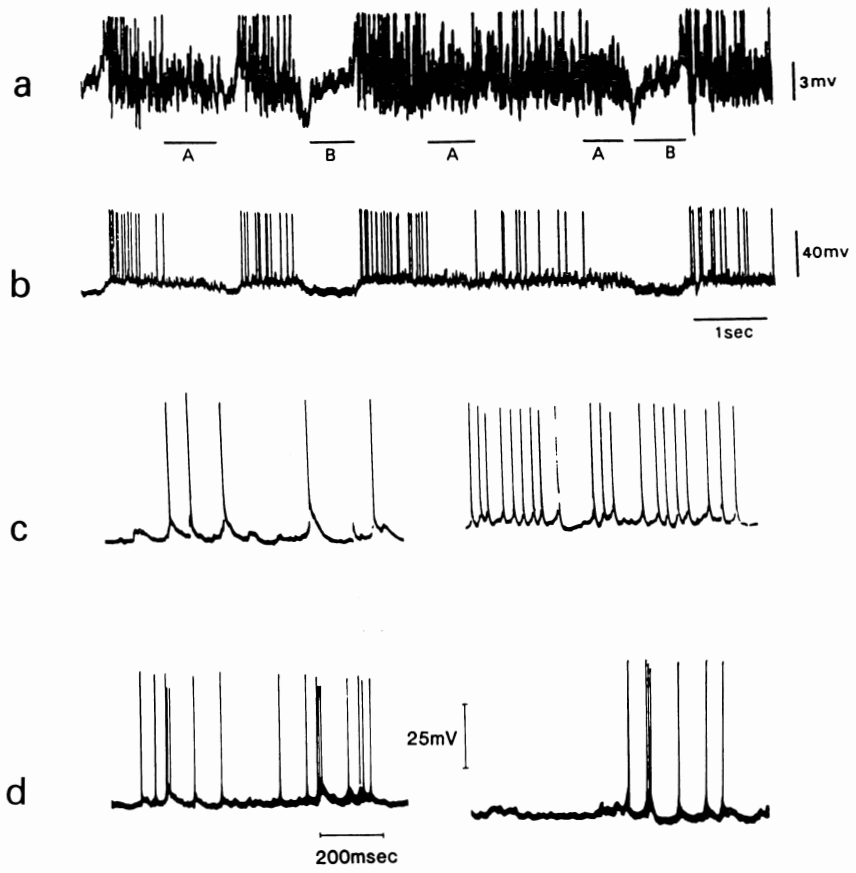

Fig. 3. a, b Intracellular recording of motor cortex neurons in the monkey during recovery from ultra-short acting barbiturate (Brevital). Higher magnification of AC recording (**a**) and lower magnification of DC recording (**b**) of same period are shown. Between bursting activity two types of membrane behavior are evident. Underlined portions labeled A show more depolarized membrane potential in DC recording and more synaptic activity in AC recording than the relatively hyperpolarized membrane potentials labeled B. **c, d** Examples of membrane potential shifts in the same motor cortex cells, recorded from Halothane anesthetized to awake state (**c**) and from awake to Halothane anesthetized state (**d**). In **c**, the membrane potential was recorded just before the Halothane was terminated (left), and 4 minutes later (right). In **d**, the membrane potential was at first recorded when the monkey was awake (left) and 3 minutes after onset of Halothane anesthesia (right). In both cells continuous recording showed larger membrane resting and action potentials under anesthesia

Table 2. Membrane potentials of motor cortex neurons reported in different studies

Author(s)	Year	Membrane potential	Animal	Type of neurons	Anesthesia
1) *Anesthetized*					
Phillips	1956	−70 (mV)	cat	Betz	barbiturate
Li	1959	−50 – −90	cat	PTN	barbiturate
Takahashi	1965	−58 – −62	cat	PTN	barbiturate
Lux and Pollen	1966	−50 – −70	cat	Betz	barbiturate
Koike et al.	1968	−60 – −80	cat	PTN	barbiturate
Deschenes et al.	1979	−63 ± 2	cat	PTN	barbiturate
Stafstrom et al.	1984	−70 ± 8	cat	Layer V	in vitro
Present results		−63 ± 10	cat	PTN	barbiturate
		−58 ± 4	cat	PTN	Halothane
2) *Awake*					
Woody and Black-Cleworth	1973	−50	cat	un-identified	
Woody and Gluen	1978	−50	cat	un-identified	
Matsumura	1979	−58 ± 12	monkey	PTN	
Present results		−53 ± 5	cat	PTN	

are summarized in Table 2. This table also includes the membrane potential in in vitro study, which are similar to the anesthetized preparations. The membrane potentials obtained from awake preparations tend to be consistently smaller than those from anesthetized animals. Interestingly, membrane potentials of motoneurons obtained from awake preparations also show similar lower values (Morales

and Chase 1978; Chase et al. 1980; Glenn and Dement 1981; Matsumura and Woody 1986; Whitney and Glenn 1986).

In the present study, we found that the mean membrane potential from awake preparations were significantly smaller than those from anesthetized preparations; moreover, neurons with membrane potential exceeding –65 mV were never observed in the awake preparations. It should be noted that these differences exist in the same animals recorded on the same day by same technique using the same criteria for intracellular recording. Moreover, records obtained during transition states (Fig. 3) indicate reversible shifts between depolarized and hyperpolarized levels in the same cells. It had been suggested that membrane potentials or membrane excitabilities in the motor pathways would be changed according to the levels of barbiturate or levels of consciousness (Sasaki and Otani 1962; Evarts 1964), though it is not known whether the shifts produced by sleep and anesthesia involve comparable mechanisms. Membrane potentials in the Halothane anesthetized state seemed to be intermediate between the awake and Nembutal anesthetized states, perhaps due to the characteristics of Halothane, or to the relatively light levels of Halothane anesthesia used in this experiment.

Conductance change in awake preparations

Membrane resistances of motor cortex neurons were significantly larger under Nembutal anesthetized than awake conditions. The mean value of membrane resistances of PT-neurons recorded under Nembutal anesthesia was comparable to those previously reported under anesthesia (Takahashi 1965). Moreover, our mean value for the awake cat was comparable to resistances previously obtained under similar conditions (Brons and Woody 1981).

Measurements of membrane resistances revealed corresponding differences in conductance of motor cortex neurons under different anesthetic conditions. Whitney and Glenn (1985) observed similar increase of membrane resistances in cat spinal motoneurons after Halothane anesthesia, which was accompanied by a shift of membrane potential. Conductance changes were further suggested by the smaller amplitude of action potentials and by the undershoot rather than overshoot membrane potential at peak of action potentials in awake cats. If the membrane potential at the peak of action potential is taken to reflect Na+ equilibrium potential, this may suggest changes of membrane conductance of a voltage-dependent Na+ channel.

Direct effects of barbiturate on the neuronal membrane must also be considered. Direct application of barbiturates potentiates postsynaptic membrane responses to GABA (Lodge and Curtis 1977), and depresses putative excitatory transmitter action (Nicoll 1975), perhaps due to increased conductance of Cl– or K+ channels (Nicoll and Madison 1982). These observations are consistent with our membrane potential changes. Direct application of barbiturates also produced higher membrane conductance in those preparations, which seems to contradict our findings; however, other factors such as changes in synaptic inputs could easily counteract this direct effect of barbiturate on the membrane. Decreases in membrane conductance may simply be due to a decrease in total amount of synaptic inputs in the anesthetized animals.

Sustained excitatory synaptic inputs to PT neurons in awake condition

The present observations suggest an increase of excitatory inputs onto motor cortex neurons when the animals are awake; moreover, this input seems greater onto PT neurons. These findings might indicate that different types of cortical neurons respond differently to anesthesia, and would explain why the trend observed for PTNs was obscured by inclusion of unidentified cells.

Several mechanisms could produce excitatory inputs to motor cortex. One possibility is the well-known 'ascending reticular activating system' (Moruzzi and Magoun 1949) or 'diffuse thalamic reticular system' (Jasper 1949). Inubushi et al. (1978) found that stimulation of reticular formation evoked both hyperpolarization and depolarization in motor cortex neurons.

Another possible source of activation is the lateral group of thalamic nuclei, such as VA or VL, which send strong monosynaptic excitatory input to motor cortex neurons, and which usually exhibit a steady level of activity (Lamarre et al. 1971). This contribution is supported by findings that barbiturate anesthesia virtually silences the activity of VL neurons, and that spindle-like bursting in VA neurons are correlated with spindle activity of cortical neurons during recruiting or augumenting responses (Sasaki et al. 1975).

A third possibility is that levels of specific neurotransmitters or neuromodulators, such as monoamines, might contribute to activation of motor cortex neurons in the awake condition. Iontophoretic studies have shown that some types of monoamine receptors cause long-term effects on neuronal excita-

bility. Recent studies showing changes of serotonin or norepinephrine in CNS during different stages of sleep (Jouvet 1972), also suggest these monoamine systems are involved in sustaining consciousness.

Whatever the source of these synaptic inputs to motor cortex neurons, our data suggests that they are depressed by both Nembutal and Halothane anesthesia. Moreover, intracellular recordings during transition periods indicate alternating cycles in these synaptic effects.

Acknowledgement. This work was supported by grants from NIH (NS12542 and RR00166).

References

Brons JF, Woody CD (1980) Long term changes in excitability of cortical neurons after Pavlovian conditioning and extinction. J Neurophysiol 44: 605–615

Chase MH, Chandler SH, Nakamura Y (1980) Intracellular determination of membrane potential of trigeminal motoneurons during sleep and wakefulness. J Neurophysiol 44: 349–358

Deschenes M, Labelle A, Landry P (1979) A comparative study of ventrolateral and recurrent excitatory postsynaptic potentials in large pyramidal tract cells in the cat. Brain Res 160: 37–46

Deschenes M, Domich L (1983) Abolition of spindling rhythmicity in thalamocortical cells disconnected from the reticularis thalami nucleus. Neuroscience Abstr 9: 1213

Eccles JC (1964) The physiology of synapses. Springer, New York, pp 49

Evarts EV (1964) Temporal patterns of discharge of pyramidal tract neurons during sleep and waking in the monkey. J Neurophysiol 27: 152–171

Glenn LL, Dement WC (1981) Membrane potential, synaptic activity and excitability of hindlimb motoneurons during sleep and wakefulness. J Neurophysiol 46: 839–854

Inubushi S, Kobayashi T, Oshima T, Torii S (1978) Intracellular recordings from the motor cortex during EEG arousal in unanesthetized brain preparations of the cat. Jpn J Physiol 28: 669–688

Jasper HH (1954) Functional properties of the thalamic reticular system. In: Adrian ED, Bremer F, Jasper HH (eds) Brain mechanisms and consciousness, a symposium. Blackwell Scientific, Oxford, pp 374–401

Jouvet M (1972) The role of monoamines and acetylcholine-containing neurons in the regulation of the sleep-waking cycle. Ergebn Physiol 64: 166–303

Koike H, Okada Y, Oshima T, Takahashi K (1968) Accommodative properties of fast and slow pyramidal tract cells and their modification by different levels of their membrane potential. Exp Brain Res 5: 189–201

Lamarre Y, Filion M, Cordeau JP (1971) Neuronal discharges of the ventrolateral nucleus of the thalamus during sleep and wakefulness in the cat: I. spontaneous activity. Exp Brain Res 12: 480–498

Li CL (1959) Cortical intracellular potentials and their responses to strychnine. J Neurophysiol 22: 436–450

Lodge D, Curtis DR (1977) Pentobarbitone enhancement of GABA. Nature 270: 543–544

Lux HD, Pollen DA (1966) Electrical constants of neurons in the motor cortex of the cat. J Neurophysiol 29: 207–220

Matsumura M (1979) Intracellular synaptic potentials of primate motor cortex neurons during voluntary movement. Brain Res 163: 33–48

Matsumura M (1981) Sustained excitatory synaptic inputs to motor cortex neurons in awake animals; a comparative study of membrane potential in anesthetized and unanesthetized state. Neuroscience Abstr 7: 564

Matsumura M, Woody CD (1986) Long-term increases in excitability of facial motoneurons and other neurons in and near the facial nuclei after presentation of stimuli leading to acquisition of a pavlovian conditioned facial movement. Neurosci Res 3: 568–589

Morales FR, Chase MH (1978) Intracellular recording of lumber motoneuron membrane potential during sleep and wakefulness. Exp Neurol 62: 821–827

Moruzzi G, Magoun HW (1949) Brain stem reticular formation and activation of the EEG. EEG Clin Neurophysiol 1: 455–473

Nicoll RA (1975) Pentobarbital: action on frog motoneurons. Brain Res 96: 119–123

Nicoll RA, Madison DV (1982) General anesthetics hyperpolarize neurons in the vertebrate central nervous system. Science 217: 1055–1057

Phillips CG (1956) Intracellular records from Betz cells in the cat. Q J Exp Physiol 41: 58–69

Sasaki K, Otani T (1962) Accommodation in motoneurons as modified by circumstantial conditions. Jpn J Physiol 12: 383

Sasaki K, Matsuda Y, Oka H, Mizuno N (1975) Thalamo-cortical projections for recruiting responses and spindle-like responses in the parietal cortex. Exp Brain Res 22: 87–96

Stafstrom CE, Schwindt PC, Flatman JA, Crill WE (1984) Properties of subthreshold response and action potential recorded in layer V neurons from cat sensorimotor cortex in vitro. J Neurophysiol 52: 244–263

Takahashi K (1965) Slow and fast groups of pyramidal tract cells and their respective membrane properties. J Neurophysiol 28: 908–924

Whitney JF, Glenn LL (1985) Membrane potential and input resistance of motoneurons in the awake and Halothane-anesthetized cat. Neuroscience Abstr 11: 404

Whitney JF, Glenn LL (1986) Influence of pentobarbital on motoneuron function in awake, intact cats. Neuroscience Abstr 12: 884

Woody CD, Black-Cleworth P (1973) Differences in the excitability of cortical neurons as a function of motor projection in conditioned cats. J Neurophysiol 36: 1104–1116

Woody CD, Gluen E (1978) Characterization of electrophysiological properties of intracellularly recorded neurons in the neocortex of awake cats: a comparison of the response to injected current in spike overshoot and undershoot neurons. Brain Res 158: 343–357

Received April 16, 1987 / Accepted November 20, 1987