

BLOCKAGE OF VIBRISSAL AFFERENTS: III. ELECTROCORTICOGRAPHIC EFFECTS

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INTRODUCTION

We have shown that bilateral vibrissae deafferentation produces depressive effects on behavior. After either bilateral infraorbital nerve (IO) section or vibrissal pad anaesthesia, we observed significant activity decreases in open field, motor deficits in a learned operant task (23) and increased thresholds for withdrawal in response to footshock (8). None of these effects was observed after cutting the whiskers at fur level. Thus, lack of tactile information provided by the vibrissae could not satisfactorily explain the generalized depressive effects of IO blockage. Instead, our results would seem to support the tonic or level setting hypothesis, i.e. the idea that some receptors play an important role in maintaining a central excitatory state, in addition to their sensory functions (2, 3, 10, 18). A close relation between this hypothesis and the sleep deafferentation theories has been postulated (4, 7).

Afferent activity and sleep-waking cycle control is an old neurophysiological problem (see ref 19 and 32 for reviews). Different arrangements of cutaneous and muscular afferent stimulation may produce either synchronized cortical activity with behavioral signs of sleep or desynchronized cortical activity with behavioral arousal (22). There are a great number of clinical and experimental results supporting the importance of afferent activity in maintaining the waking state. Perhaps one of the earliest and most dramatic experience was reported by Strümpel in 1887 (27). A patient had successive loss of sensory modalities, until finally only one eye and one ear were left. When these two input sources were blocked, the patient immediately fell asleep. Bremer (1) pointed out the relevance of sensory inputs in maintaining a "central tonus" supporting wakefulness. Galkine (11) and Vital-Durand and Michel (33) reported a significant decrease in motor activity after peripheral deafferentation. A polygraphic study of quasi-total deafferentation in cats (surgical section of olfactory, optic, statoacoustic and trigeminal nerves, a single vagal nerve and spinal cord posterior paths) mainly affected waking but not sleep mechanisms (33).

The aim of the present work was to study whether the effects of vibrissal pad anaesthesia on behavior are accompanied by electrocorticographic (ECoG) changes. These results have been partially presented (in abstract form) elsewhere (24)

METHODS

Male Wistar rats ($N = 20$, 300-320 gr) were implanted for chronic electrocorticographic (ECoG) and electromyographic (EMG) recording. Under general anaesthesia (Pentobarbital, 40 mg/Kg i.p.), recording electrodes were implanted. The two EMG electrodes (insulated nichrome wires, 0.4 mm diameter, 2 mm of the tips left bare) were implanted in the back of the neck muscles, with an interelectrode distance of 2-3 mm. The electrocorticograms (ECoG) were recorded by means of 3-4 pairs of electrodes (also insulated nichrome wire 0.4 mm, only tip left bare) placed through small holes in the skull, in contact with the dura. One or two pairs were implanted in both left and right parietal cortices (1 mm posterior to Bregma, 4 mm lateral to the sagittal suture), another pair was over occipital cortex (8 mm posterior to Bregma, 2 mm lateral to the sagittal suture); and the fourth pair was over frontal cortex (4mm anterior to Bregma, 2 mm lateral to the sagittal suture). The interelectrode distance of each pair was ≈ 1 mm. With such short interelectrode distances, the fine wires used, and differential amplification, we could be reasonably certain that the electrical activity recorded was mainly produced by the neocortex immediately underneath the electrodes. With the standard techniques commonly used (a large surface electrode and a distant indifferent), the close proximity of the hippocampus (often generating very large theta waves) made it impossible to clearly distinguish neocortical from hippocampal activity. A wire (same as previous, with a bare tip of 5 mm) was implanted in the temporal muscle and was used as ground. All wires were soldered to the different tips of a VGA socket. Three small stainless steel screws (0.5 mm diameter; 1.2 mm high) were attached to the skull and the whole set (screws and wires) were glued together with acrylic dental cement to the VGA socket.

The recording setup had a device (Slip Ring Capsule, AC6023-12, Poly-Scientific, Litton) allowing free rotation of the rat without inconvenience. Electrophysiological data was AC amplified (filters 0.3 Hz to 30 KHz, 3db); A/D converted (Digidata 1322A, © Axoscope) and stored in a PC compatible system (sampling rate at 1KHz). Usually 3-4 channels of ECoG and 1 of EMG were stored, along with comments about the animal's behavior.

A week after implant surgery, recordings were obtained under control conditions during different rat behavioral states (awake, walking, resting or grooming; asleep). Then after brief ether anaesthesia (90 sec total), the vibrissal afferents were blocked bilaterally ($N = 9$) with local anaesthesia subcutaneously injected (1cc each pad, 0.5% procaine solution). Once the animal recovered from the ether (1-2 min after removal of the mask), ECoG records were obtained. At the beginning of each session, the recordings were carried out continuously (first 30 minutes) and afterward for periods of 30 seconds each minute. The session lasted for a total of 2 hours, after which the animals seemed completely normal (behavior and ECoG). In some animals the procaine concentration used was reduced by more than half (0.2%, $N = 2$) or injected unilaterally ($N = 2$, see results). In other animals ($N = 4$) the blockage of the vibrissae was carried out by injection of large amounts (1.5-2 cc) of physiological solution (see ref. 8 for a detailed description of this mechanical blockage).

On a different day (before or after the session with vibrissal pad blockage), control sessions without vibrissal anaesthesia, were carried out in order to examine the effects of the ether itself. The period of ether inhalation in these sessions was the same as in the experimental ones (90 sec). This recording session lasted 10-15 min. Usually 1-2 min after ether, the animal started walking normally and from then on, behavior and ECoG were completely normal.

A further control was made in two other rats, whose IO nerves were bilaterally severed just before the implantation surgery. In this case experimental sessions were carried out 7-10 days after operation and the general procedure and dose of procaine injection were the same as described above.

Finally, in three rats the brachial plexus was bilaterally blocked by subcutaneous injection of Procaine solution (0.5% in saline, 1.5-2 cc each side), under light ether anaesthesia.

RESULTS

Figure 1 shows a typical recording under normal conditions (without any treatment) during awake (1A) and sleep (1B) states, and 5 min after ether inhalation (1C). We observed only a few motor impairments after exposition to ether for 90 seconds. After this light anaesthesia, rats showed clumsy walking for the first three minutes; afterwards, the behavior appeared perfectly normal. As is shown in Figure 1C, there are no changes in cortical or muscular electrical activity 5 minutes after ether exposition. At this time, both ECoG and behavior were always identical to those observed in a normal awake rat.

On the other hand we found striking ECoG changes after afferent vibrissal blockage. Following bilateral vibrissal pad anaesthesia there were large cortical slow waves and a nearly complete lack of EMG activity in the neck muscles. Interspersed with these slow waves, the ECoG in frontal and parietal regions displayed bursts of spindles (6 to 14 Hz, 300 to 400 mV) Both the slow waves and the lack of EMG started 5-10 min after local anaesthesia injection and lasted 30-60 min (Figure 2). The mechanical blockage of the vibrissal system by injection of 1,5-2 cc of saline bilaterally produced the same results as the chemical (procaine) blockage. The only noticeable difference was the longer duration of the period of slow waves in the local anaesthesia case (as expected). Figure 3 shows typical electrophysiological records after this mechanical blockage.

It is important to note that during the occurrence of slow waves (with or without spindles) the rat behaved like an awake rat, even if depressed and clumsy. Under normal conditions, when the rat had its eyes open, we never observed cortical synchronization. After vibrissal pad anaesthesia, cortical slow waves were always seen in animals whose eyes were open and often even when the animal was walking or moving its head. In some cases there were a few short (10-20 sec) periods of ECoG activation but these sporadic activations were not always accompanied by animal movements nor by EMG activity increments.

Although ECoG synchronization was observed bilaterally in all cortical areas studied, there were many differences among them. After vibrissal pad anaesthesia, parietal ECoG synchronization was more pronounced and lasted longer than in the other regions.

For the occipital cortex, a predominance of theta activity was observed in waking and sleeping states under normal conditions. Complete synchronization was clearly observed only between 25 and 40 min after vibrissal afferent blockage.

The ECoG effects of vibrissae anaesthesia on the frontal cortex were less clear. During the first 15 min we observed an alternation between fast and slow activity (periods with a duration of 5-10 seconds). After that, there was a period of 15-20 min with almost no fast activity. This cortical zone was always the earliest in which activation appeared after the afferent blockage: between 30-40 min after pad anaesthesia the ECoG again displayed predominantly fast activity.

Results were the same when IO blockage was carried out with a lower dose of procaine (0.2%). In these cases synchronization had a shorter duration (20-30 min).

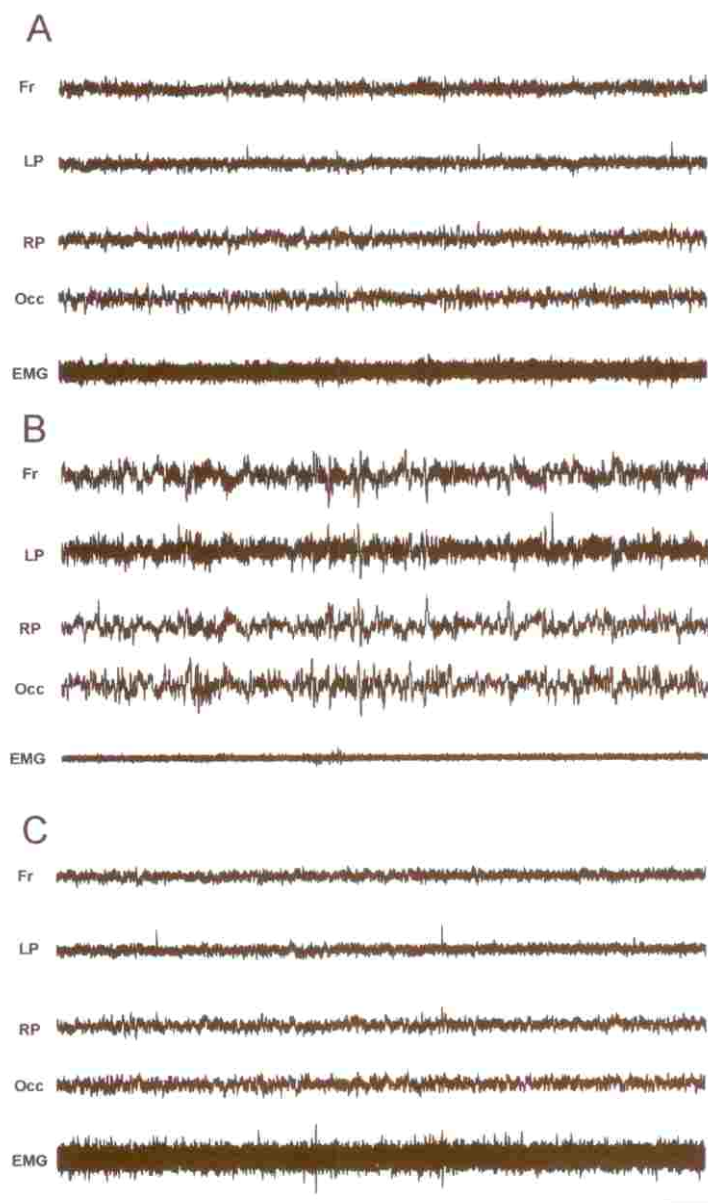


Fig. 1. - *Normal and control conditions.*

A: awake; B: asleep; C: 5 minutes after ether anaesthesia.

Fr: frontal zone, LP: left parietal, RP: right parietal
Occ: occipital

Calibrations: Horizontal, 2 sec. Verticals are 200 μ V in Fr, 150 μ V in LP, RP and Occ, 100 μ V in EMG

When procaine (0,5%, 1cc) was injected in only one vibrissal pad (right side) synchronization was clearly observed only in left parietal cortex 10 min after injection; it then lasted only 10-15 min. Frontal and occipital cortices during this period did not display synchronization and EMG activity was normal.

As in previous papers (8, 23), we observed a nearly complete akinesia in most rats and even catalepsia in many of them after vibrissal pad anaesthesia (even with lower doses of procaine or when only physiological solution was injected).

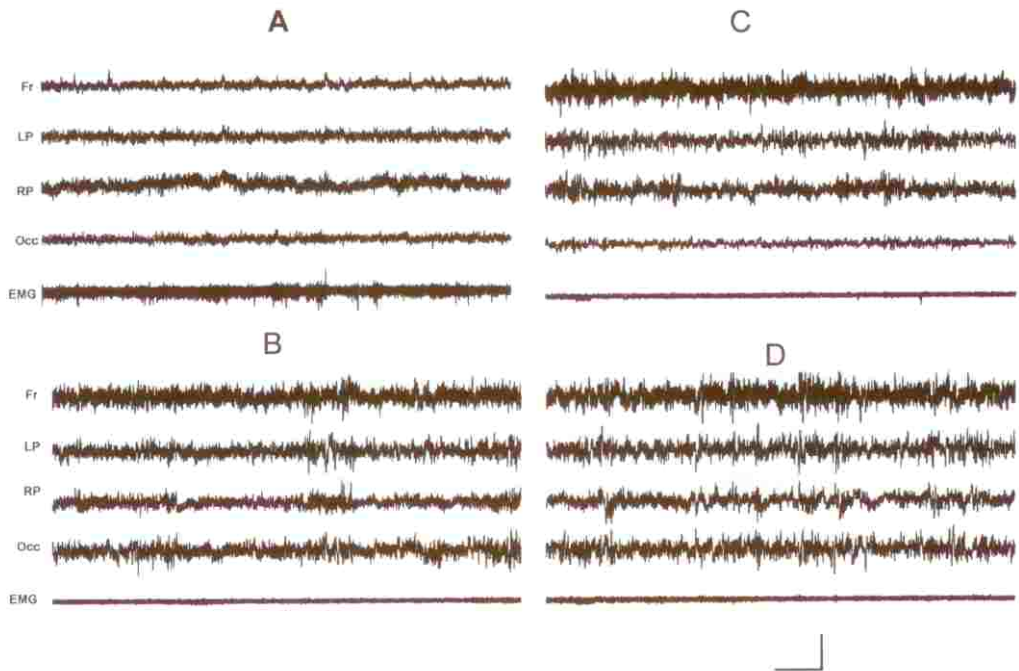


Fig. 2. - After bilateral vibrissal pad blockage (procaine injection).

Typical electrophysiological records before (A) and at different times after injection: 15 min (B), 25 min (C) and 35 min (D).

Calibrations: Horizontal, 2 sec. Verticals are 200 μ V in Fr, 150 μ V in LP, RP, Occ, and EMG. Abbreviations as in Fig. 1.

The effects of brachial plexus blockage are shown in Figure 4. Besides the expected and obvious motor impairments in the forelimbs, ECoG synchronization was observed in all zones studied without any behavioral sign of sleep. Slow waves and spindles displayed the same features and duration as in vibrissal pad anaesthesia.

Finally, in the group with chronically deafferented vibrissae, the local injection of the procaine solution did not result in either ECoG or any behavioral effect (Figure 5), except for the obvious swelling in the pads

DISCUSSION

We have shown that vibrissal pad anaesthesia produces a striking cortical synchronization. Slow waves and spindles observed were indistinguishable from the records in normal sleeping rats. However after IO blockage this cortical synchronization was always observed in the absence of behavioral signs of sleep (rat with eyes open, sometime moving the head or walking). It is important to note that closed eyes are an almost universal sign of sleep and have been considered as an essential feature of this state (16). Another interesting effect of this treatment was the nearly

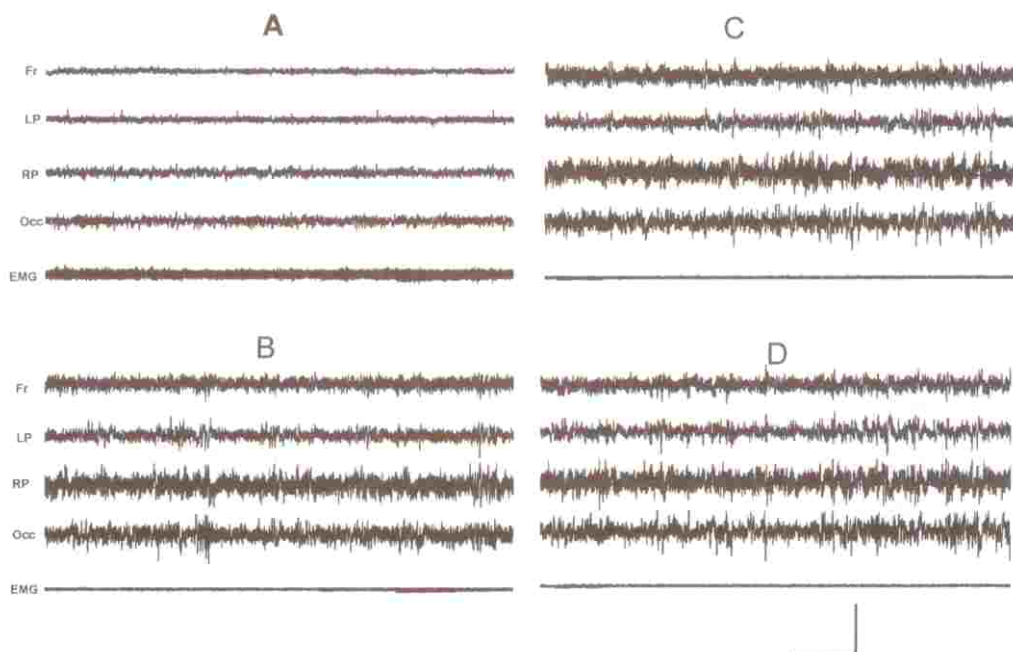


Fig. 3. - After bilateral vibrissal pad blockage (saline injection).

Typical electrophysiological records before (A) and at different times after injection: 15 min (B), 25 min (C) and 35 min (D).

Calibrations: Horizontal, 2 sec. Verticals are 200 μ V in Fr, RP and Occ, and 250 μ V in EMG. Abbreviations as in figure 1.

complete loss of muscle tone. These results would seem to support the sleep deafferentation hypothesis.

The interactions between afferent activity and the sleep-waking cycle have been extensively studied. The central nervous system processes sensory inputs during both waking and sleep states (including slow waves and paradoxical sleep), but the processing differs in each case (6,12). On the other hand, it was also shown that afferent activity (both nerve electrical stimulation [22], sensory stimuli [16, 17, 29] and deafferentation [11, 33]) influences the whole brain state, leading to changes in the sleep-waking cycle (see ref. 32 for a review).

Obviously, changes in the sleep-wake cycle after deafferentation should be studied in chronic preparations. In the present paper we only studied the acute effect of an important but reversible deafferentation, not a true change in the sleep-wake cycle. However, we think that ECoG synchronization and lack of EMG activity after IO deactivation could be explained by the sleep deafferentation hypothesis. As far as we know, acute ECoG effects of reversible afferent deactivation have never been used before as an approach to the sleep-afferent interaction. The combined use of reversible deactivation techniques with neurorecording and behavioral methods was postulated as a promising approach to the understanding of neural network operations (21). The striking changes in both motor behavior (23),

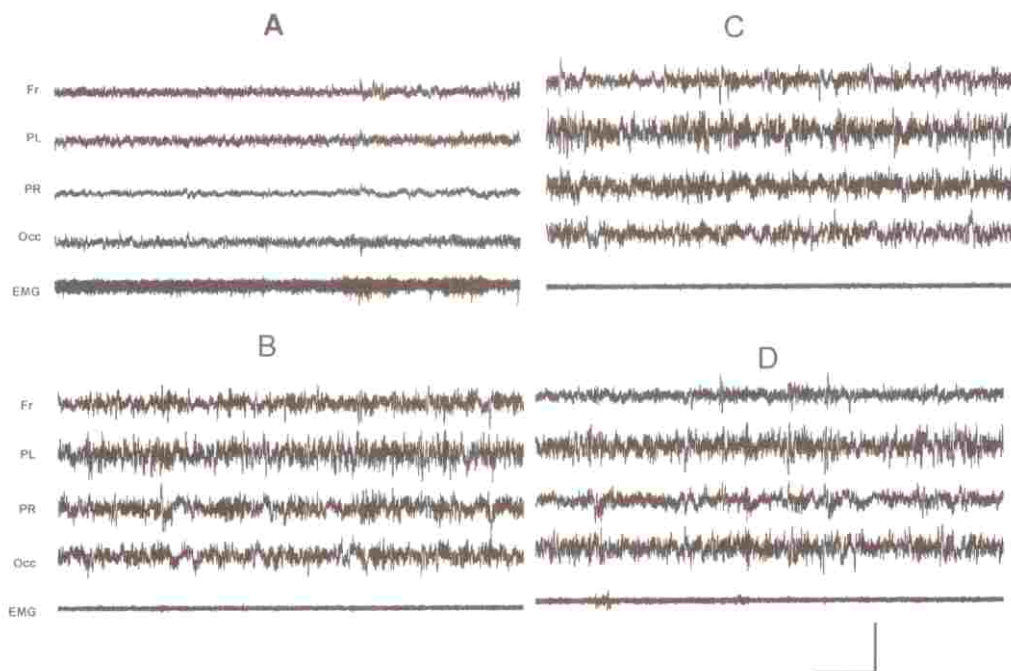


Fig. 4. - After bilateral brachial plexus anaesthesia.

Typical electrophysiological records before (A) and at different times after injection: 15 min (B), 25 min (C) and 35 min (D).

Calibrations: Horizontal, 2 sec. Verticals are 200 μV in Fr, LP, and Occ and 150 μV in EMG. Abbreviations as in Fig. 1.

sensory thresholds (8), with ECoG synchronization and loss in EMG activity (present results) after IO reversible deactivation agree with the importance of this type of study.

A dissociation between ECoG and behavior, like the one we obtained after vibrissal afferent blockage (either chemical or mechanical) is well known to be an effect of certain drugs. Both bulbocapnine (13) and reserpine (5) produce akinesia and cataleptic-like states. In this context it is interesting that Vanderwolf and coworkers (30, 31) have shown that atropine blocks cortical fast activity during "automatic" (type 2) behavior but not during "volitive" (type 1) behavior. Furthermore, combined administration of atropine and reserpine cancel all fast cortical activity, whatever the behavior of the rat happens to be (9). On the basis of these findings, plus electrical recording of unit activity in the basal forebrain, several authors (9, 28, 35) have postulated a double mechanism to explain cortical activation. The principal one would be cholinergic, with its neurons located in the basal forebrain and the other, would be serotonergic, provided mainly by Raphe neurons. On the other hand, Ramson and coworkers (15, 25, 26) showed cataleptic states in cats and monkeys produced by lesions in rostral areas of the hypothalamus, close to where the basal forebrain cholinergic neurons are located.

We have not explored the possible mechanisms by which deafferentation produced ECoG synchronization without true behavioral sleep. More studies are needed to test what encephalic structures (ascending reticular system, cholinergic basal forebrain neurons and/or thalamocortical neurons) are involved in the results we are presenting. Because the similitude between our results and those reported by Vanderwolf's group (9, 30, 31) and Ramson's papers (15, 25, 26), we think that the same basal structures may be involved.

Whatever may be the mechanism underlying our results, we think that two factors were very important, viz, the acute approach and the number of afferent elements blocked. Plasticity is a recognized feature of the nervous system that tends to minimize lesion induced effects over time. In the previous papers we have already discussed behavioral differences between local anaesthesia and IO nerve section.

As in previous studies (11, 33), the amount of deafferentation is probably important for the results obtained in our experiments: bilateral vibrissal pad anaesthesia involves the acute deactivation of more than 25% of each somatosensory cortical area (34). On the other hand, we have shown that unilateral vibrissal afferent blockage produced a more restricted and shorter-lasting ECoG synchronization. The importance of the amount of deafferentation is also clear in the results of other authors (11, 20, 33). As explained above, our ECoG effects were not limited to vib-

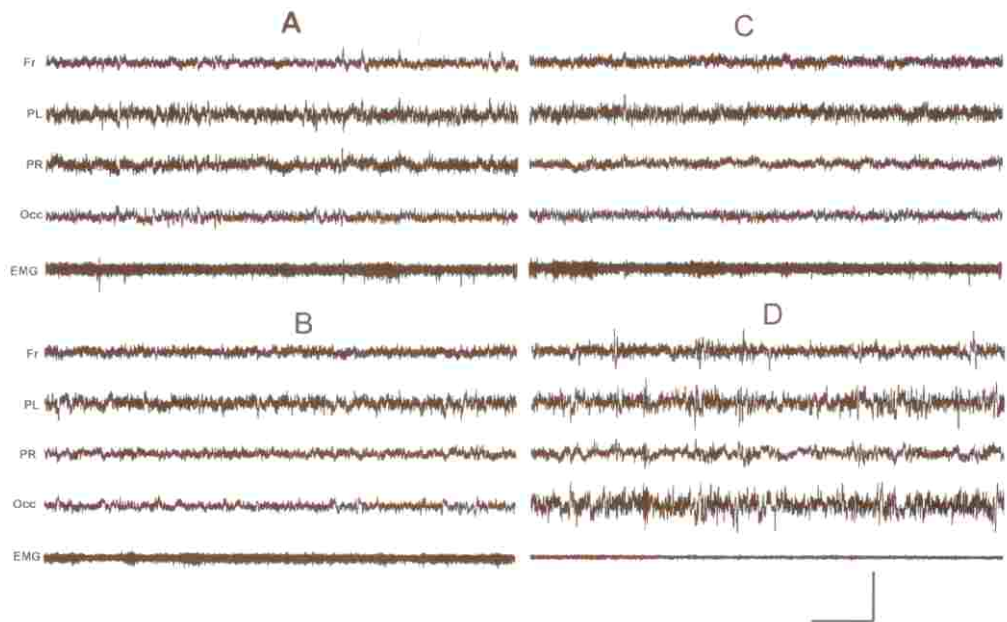


Fig. 5. - After procaine injection in deafferented vibrissal pads.

A: awake (just before procaine injection); B, C and D: 15, 25 and 35 minutes after injection respectively. D, rat sleeping with eyes closed.

Calibrations: Horizontal, 2 sec. Verticals are 200 μV in F and LP 150 μV in RP, Occ, and EMG. Abbreviations as in Fig. 1.

rissal block. Similar results were also produced by local anaesthesia of both brachial plexuses. Obviously, in the latter case behavioral analysis is not pertinent, i.e. locomotor impairment is fully explained by the paralysis and anaesthesia of the forelimbs. However the cortical synchronization (with open eyes) we observed had roughly the same characteristics as that described above for vibrissal pad anaesthesia. Therefore this result would suggest that afferents do not have different roles with respect to their control of the waking state but rather it is the total amount of deafferentation that is important.

Regarding the hypothetical direct effect of procaine on the brain, there are several facts which weigh heavily against the idea. First, it is well known that procaine applied directly to the cortex is a convulsant, not an anaesthetic (13). Second, we have shown that the general effects of vibrissal afferent blockage are the same with either procaine or saline solution injection. Finally the lack of ECoG and behavioral effects when procaine was injected in chronically deafferented vibrissal pads is a strong proof that our results are specifically due to the acute afferent blockage (vibrissae in this case).

The effects of IO nerve section on the ECoG were not tested because of the difficulties clearly separating the spindles and low frequency waves due to general anaesthesia from the possible synchronization produced by deafferentation.

In summary, we have shown that bilateral local anaesthesia of the IO nerves produces depression of motor behavior, both innate and learned (23). Furthermore, a general increase in threshold for a reflex response is also present under the same conditions (8). These changes thus seem to agree with the hypothesis formulated by Granit (14): "spontaneous" afferent activity has a general "energizer" effect on the central nervous system.

Our studies have also shown that acute bilateral deafferentation of the vibrissal system induces a high degree of electrocortical synchronization. Curiously enough, the animals were behaviorally awake in this situation. Thus the ECoG-behavioral dissociation, known to be induced by chemical means (parenteral administration of atropine plus reserpine), has also been obtained by an acute and massive sensory deafferentation, including mechanical deactivation of the IO nerves.

SUMMARY

We have shown signs of behavioral depression after vibrissal deafferentation. Locomotor slowing, motor impairments and footshock thresholds increment were demonstrated after vibrissal afferent blockages. Here, we study the electrocortical (ECoG) effects of vibrissal pad anaesthesia, also replicated by bilateral brachial plexus blockage. We found in both cases, that this acute and massive deafferentation produces synchronization over the entire neocortex accompanied by an important loss of muscular electrical activity. Slow waves observed in this condition were similar to those recorded in the sleeping rat without any treatment, but in our case, there were no behavioral signs of sleep. Thus a clear behavioral electroencephalographic

dissociation was obtained by acute deafferentation. These results would seem to support the sleep deafferentation hypothesis.

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