

VESTIBULAR INFLUENCES DURING SLEEP

II. EFFECTS OF VESTIBULAR LESIONS
ON THE PYRAMIDAL DISCHARGE
DURING DESYNCHRONIZED SLEEP¹A. R. MORRISON² AND O. POMPEIANO*Istituto di Fisiologia dell'Università di Pisa, Centro di Neurofisiologia
e Gruppo d'Elettrofisiologia del C. N. R., Sezione di Pisa, Italia³*

INTRODUCTION

Experiments performed in unrestrained unanesthetized cats (13) have shown that the myoclonic twitches affecting skeletal muscles during desynchronized sleep: i) are produced by supraspinal volleys descending along the dorsolateral funiculi; ii) are generally related in time with the bursts of rapid eye movements (REM); and iii) affect mainly the flexor muscles. It is known that the pyramidal tracts course primarily along the dorsolateral funiculi. Experiments performed in the cat have shown, moreover, i) that the pyramidal discharge is phasically enhanced during REM (17, 18, 19); and ii) that cortically induced movements, mainly flexor in character, affect the spinal motoneurons through the same interneurons which are involved in the flexor reflex (15, 16). These data would suggest that pyramidal volleys are involved in the phasic excitation of the

spinal motoneurons underlying the clonic twitches. That these clonic movements are not dependent on the pyramidal tract is shown, however, by their persistence after interruption of the corticospinal pathway (17, 18, 19).

Recent experiments have shown that the REM are related in time with bursts of high frequency discharge originating from the second order vestibular neurons localized in the medial and descending vestibular nuclei (5, 6, 7). The REM are in fact selectively abolished by complete bilateral destruction of all vestibular nuclei, and even by a lesion limited to the medial and descending vestibular nuclei of both sides (26, 27, 28). Following these lesions the episodes of desynchronized sleep are still present, but they are characterized only by the usual desynchronized EEG pattern and by the disappearance of the spontaneous EMG activity in the posterior cervical muscles.

The present investigations were undertaken with the aim of finding out: i) whether the phasic enhancement of the vestibular activity occurring during desynchronized sleep is capable of triggering the sensory-motor cortex and the corticospinal discharge arising from it, and ii) whether the destruction of the vestibular nuclei abolishes the myoclonic twitches that occur synchronously with the REM.

METHODS

The experiments were performed in unrestrained, unanesthetized cats. Electrodes recording the electroencephalogram (EEG), the electro-oculogram (EOG), the electromyogram (EMG) of the posterior cervical muscles and of the tibialis anterior muscles of both sides were implanted chronically, under Nembutal anesthesia, following a previously described technique (29, 30). The integrated pyramidal discharge was led from the medulla rostrally to the *decussatio pyramidum* or at mesencephalic level and was recorded following a technique which has been proposed for studying the temporal variations of the average number of impulses which course along a bundle of nerve fibers (4, 3).

This activity, amplified by an A. C. pre-amplifier, was fed to a band-pass filter, whose critical frequencies were 500 cps and 10 Kcps with a selectivity of 12 db per octave. The output of this filter was measured by a Ballantine type 320 RMS voltmeter, whose output (proportional to the root mean square of the input voltages) was finally recorded in a D. C. channel of the EEG. A Grass D. C. 5 P 1 preamplifier was used and the driver amplifier low-pass filter was set at 3 cps, 6 db per octave. This technique has been already utilized for recording the pyramidal discharge in the intact cat (2, 17, 18, 19). EEG, EOG, EMG and the integrated pyramidal discharge were recorded on an ink-writer.

The pyramidal discharge was also routinely recorded, after the first stage of amplification, through one channel of a 502 Tecktronix C.R.O.; while the other channel was used for recording the ocular movements.

¹ Preliminary note in (23). An account of this work has been presented by one of us in Stockholm at the Nobel Symposium I on "Muscular afferents and motor control" (24), and in New York at the A.R.N.M.D. Meeting on "Sleep and altered states of consciousness" (25).

² Special Fellowship (1 F 10 NB 1204-01 NSRB) from the National Institute of Neurological Diseases and Blindness, Public Health Service. Present address: Department of Animal Biology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa., U.S.A.

³ This investigation was supported by PHS research grant NB 02990-04 from the National Institute of Neurological Diseases and Blindness, N.I.H., Public Health Service, U.S.A.

The experiments started at least two days after the chronic implantation of the electrodes, *i. e.* when the effects of the anesthesia were gone. When the phasic enhancements of the pyramidal discharge during the REM had been repeatedly confirmed, the animals were submitted to bilateral chronic lesion of the vestibular nuclei, following a technique described in the first paper (28) of this series. In several animals the vestibular lesion was made at the beginning of the experiment during the chronic implantation of the electrodes. The anatomical control of the vestibular lesion was performed at the end of the experiments following serial histological sections, stained alternatively with the Weil and the Nissl methods. For the anatomical localization and the delimitation of the vestibular nuclei, we followed the work of Brodal and Pompeiano (8; see also 9).

RESULTS

1. *Pyramidal activity during physiological sleep in intact preparations.* — In the intact animal the integrated pyramidal discharge increases during the trains of large amplitude cortical waves (spindles) and decreases during the interspindle lulls. During transition from synchronized to desynchronized sleep the corticospinal activity becomes stabilized to a level equal to that reached during the interspindle lulls, provided the REM are absent. However, when isolated ocular movements appear at the beginning of the episode of desynchronized sleep, the baseline of the record gradually rises to a level equal to, or slightly higher than, that reached during the peaks accompanying the spindles in the synchronized phase.

The most remarkable changes in the pyramidal activity appears during the outbursts of REM. As soon as these phasic events occur, the pyramidal activity increases phasically, reaching a level higher than that occurring during the spindle trains. A clear relationship exists between increase in pyramidal activity and number, amplitude and frequency of rapid ocular movements. All these observations, already described in a previous work (18), have been repeatedly confirmed here. They have also been controlled on oscillographic records.

Fig. 1 illustrates the modulation of the pyramidal activity during synchronized sleep in the intact animal and the typical enhancement of the pyramidal discharge during the REM phases of desynchronized sleep.

2. *Pyramidal activity during wakefulness and synchronized sleep in cats with lesions of the vestibular nuclei.* — Following bilateral destruction of the vestibular nuclei the pattern of the integrated

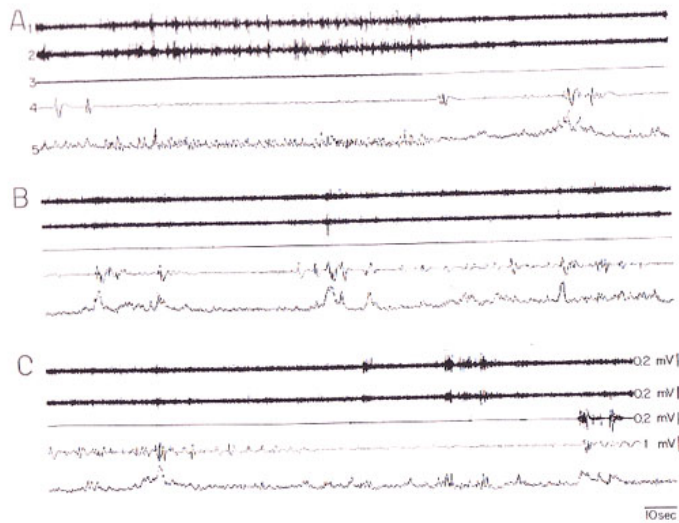


Fig. 1. — Increase in the pyramidal discharge occurring in the intact cat during the bursts of REM of the desynchronized sleep.

Unrestrained, unanesthetized cat. Experiment made 4 days after the operation. Bipolar records. 1: left parieto-occipital (EEG); 2: right parieto-occipital (EEG); 3: EMG of the posterior cervical muscles; 4: electro-oculogram (EOG); 5: integrated activity of the left pyramidal tract recorded at medullary level. Note the modulation of the pyramidal discharge during synchronized sleep and the large phasic enhancements in the pyramidal activity during the REM periods of desynchronized sleep.

pyramidal discharge is apparently not modified during synchronized sleep as compared with the intact preparation. Fig. 2 illustrates that during this phase of sleep the pyramidal activity still shows irregular oscillations in both amplitude and rate. As in the intact preparation the integrated pyramidal activity frequently increases during the EEG spindles, to reach the lowest values during the interspindle lulls. A clear-cut relationship between trains of synchronous waves and peaks in the pyramidal record is particularly evident also in the experiments where the pyramidal discharge

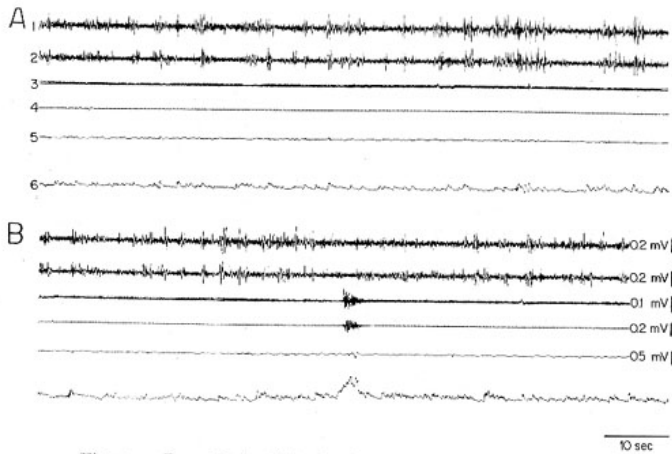


Fig. 2. - Pyramidal activity during synchronized sleep and wakefulness following bilateral destruction of the vestibular nuclei.

Unrestrained, unanesthetized cat. Experiment made 2 days after chronic implantation of the electrodes and complete bilateral destruction of the medial and descending vestibular nuclei (Fig. 5). Bipolar records: 1: left parieto-occipital (EEG); 2: right parieto-occipital (EEG); 3: EMG of the posterior cervical muscles; 4: EMG of the left tibialis anterior; 5: EOG; 6: integrated activity of the right pyramidal tract recorded at mesencephalic level.

A: pyramidal activity during synchronized sleep. B: increase of pyramidal discharge during a short-lasting arousal associated with an orienting reaction and movements.

had been conventionally recorded, after the first stage of amplification, through one channel of the C.R.O.

Even in this series of experiments, just as in the intact animal, the pyramidal discharge increased phasically during spontaneous or induced arousal, to reach a level higher than that achieved during the spindle trains (Fig. 2B). This enhanced pyramidal activity was associated with EEG desynchronization. It then gradually decreased to reach a steady level similar to that occurring during the stage of quiet waking.

3. *Pyramidal activity during desynchronized sleep in cats with lesions of the vestibular nuclei.* — As pointed out in the introduction,

a bilateral lesion of the vestibular nuclei does not prevent the appearance of the episodes of desynchronized sleep. These phases of sleep are still characterized by desynchronized EEG patterns and by the abolition of neck muscular activity. However, the REM are completely abolished. Only slow ocular movements or isolated jerks of the eyes can be observed. During these episodes of desynchronized

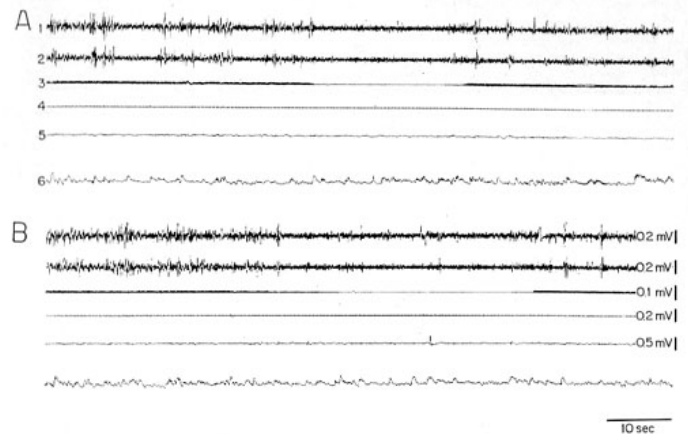


Fig. 3. - Pyramidal activity during short-lasting episodes of desynchronized sleep following bilateral destruction of the vestibular nuclei.

Same animal as in Fig. 2, same bipolar records. A, B: during the two short-lasting episodes of desynchronized sleep there are slight fluctuations of the integrated pyramidal record. However, bursts of REM and related enhancements of pyramidal discharge are lacking.

sleep the integrated pyramidal discharge is not markedly different from that recorded during the synchronized periods.

Fig. 3 shows the integrated pyramidal record during two short-lasting episodes of desynchronized sleep, while Fig. 4 illustrates the full development of an episode of desynchronized sleep following bilateral destruction of the vestibular nuclei. Note in both instances the complete abolition of the bursts of REM. In these cases, as well as in those in which oscillographic records of the pyramidal discharge were taken, the pyramidal record is stabilized. Peaks

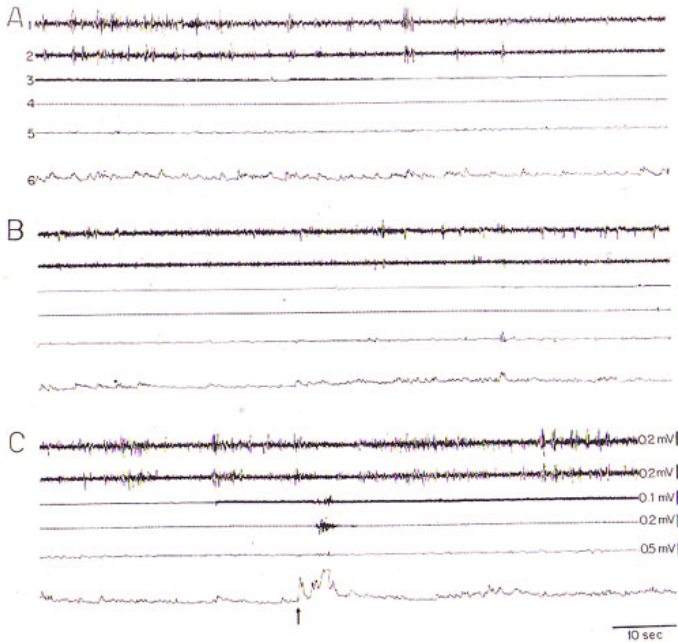


Fig. 4. — Integrated pyramidal discharge during desynchronized sleep after bilateral destruction of the vestibular nuclei.

Same animal as in Fig. 2, same bipolar records. The sharp peaks in the pyramidal record, occurring on a background of EEG synchronization (A) are still present, although reduced in number, during the desynchronized phase (B). The peaks of these pyramidal deflections, however, are generally never higher than those that previously occurred synchronously with the EEG spindles. They are sometimes, but not always, related with the residual isolated ocular jerks. The large bursts of REM and related phasic enhancements of pyramidal discharge are absent. Note in C the phasic increase in pyramidal activity that occurs during the arousal reaction produced by a strong auditory stimulus (arrow).

in the pyramidal record still appear during desynchronized sleep; however, they are never higher than those accompanying the EEG spindles during the synchronized phase. They are occasionally related in time with the slow ocular movements or with the isolated

jerks of the eyes which still remain after a complete bilateral destruction of the vestibular nuclei. As pointed out above arousing stimuli can still produce in these preparations a phasic enhancement in the pyramidal discharge (Fig. 4 C).

4. Vestibular structures responsible for the phasic enhancement in the pyramidal discharge during the bursts of REM. — The abolition

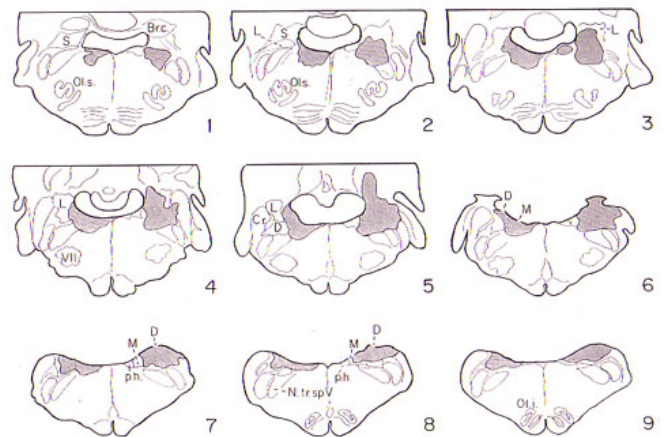


Fig. 5. — Bilateral destruction of the vestibular structures responsible for the REM and the related phasic increase in the pyramidal discharge during desynchronized sleep.

Same animal as in Figs. 2-4. Schematic representation of brain stem sections, taken at regular intervals and reproduced in a rostrocaudal direction. Shaded areas represent the extent of the electrolytic lesion.

Br. c.: brachium conjunctivum; C. r.: restiform body; D: descending vestibular nucleus; L: lateral vestibular nucleus (of Deiters); M: medial vestibular nucleus; N. tr. sp. V: nucleus of spinal tract of trigeminal nerve; Ol. i.: inferior olive; Ol. s.: superior olive; p. h.: nucleus praepositus hypoglossi; S: superior vestibular nucleus; VII: motor nucleus of the facial nerve.

of the phasic enhancement in the pyramidal discharge during the desynchronized episodes of sleep can be observed whenever the lesion is bilateral and affects the classical vestibular complex in its entire rostro-caudal extent. This effect has been followed for as

long as 12 days after the operation. Bilateral electrolytic lesions limited to the medial and descending vestibular nuclei are also equally effective (Fig. 5). As pointed out in a previous study (28) the medial and descending vestibular nuclei are responsible for the bursts of REM during desynchronized sleep (28). However, the electrolytic lesion of these vestibular nuclei must be complete in order to abolish the phasic increases of the pyramidal discharge. Fig. 6 shows the effects of a bilateral but incomplete lesion of the medial and descending vestibular nuclei on the pyramidal discharge. The bursts of REM are not abolished, although they occur rarely. They are represented, moreover, by few, low amplitude potential oscillations. The enhancement of the integrated pyramidal discharge is clearly present, though markedly reduced in amplitude (compare Figs. 1 and 6).

Finally a bilateral lesion limited to the superior or to the lateral vestibular nuclei, as well as a unilateral lesion of the vestibular complex, do not prevent the appearance of the REM and of the related pyramidal discharge.

5. *Myoclonic twitches during desynchronized sleep in cats with lesions of the vestibular nuclei.* — As reported in a previous paper (13), the myoclonic twitches that occur during the episodes of desynchronized sleep in a normal cat are frequently related in time with the bursts of REM. A relation with the isolated ocular movements or with the slow eye movements is by far less frequent.

The effects of vestibular lesion on the myoclonic twitches have been investigated by recording the EMG from the tibialis anterior of both sides. The records in Fig. 7 have been taken following a complete bilateral lesion of the vestibular nuclei. It appears that when the episode of sleep is fully developed in both its EEG and EMG aspect the large bursts of REM and the related myoclonic twitches are absent. Only isolated ocular movements can be detected. A few myoclonic twitches, isolated or grouped in trains, but generally unrelated with the ocular movements are observed. They occur at random, and are by far less conspicuous and frequent than those observed when the vestibular lesion is either absent or incomplete.

A bilateral lesion of the superior and lateral vestibular nuclei, or a complete unilateral lesion of the entire vestibular complex does not alter the typical pattern of myoclonic activity. Bursts of REM

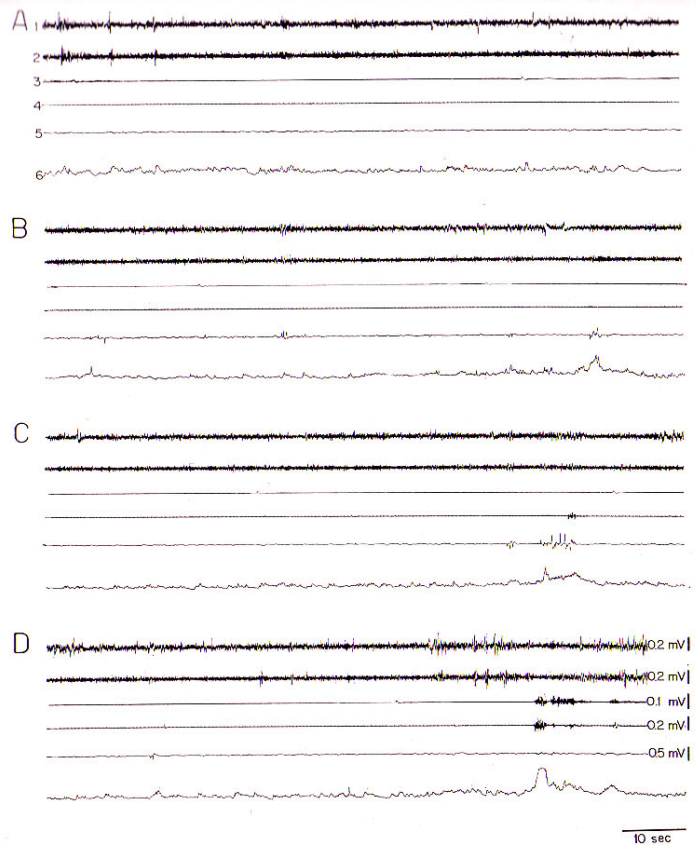


Fig. 6. — Phasic enhancements of the integrated pyramidal discharge related with the residual bursts of ocular movements during desynchronized sleep following bilateral incomplete lesion of the medial and descending vestibular nuclei.

Unrestrained, unanesthetized cat. Experiment made 4 days following bilateral lesion of the medial and descending vestibular nuclei, sparing the rostral part of these structures. Bipolar records as in Fig. 2. Note the enhancement of the pyramidal discharge during the residual bursts of ocular movements (B, C) and the phasic increase of the pyramidal activity at the moment of a spontaneous arousal (D).

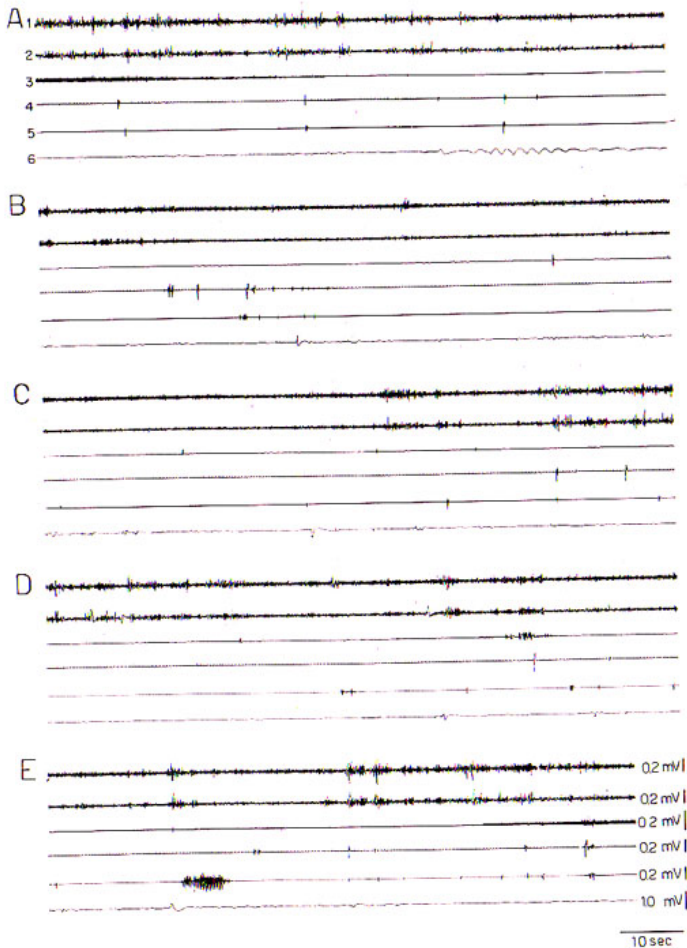


Fig. 7.

and groups of clonic twitches occur very frequently, and they are clearly related in time. Fig. 8 shows the persistence of bursts of REM and the symmetrical appearance of the myoclonic twitches in the tibialis anterior following unilateral lesion of the vestibular nuclei.

DISCUSSION

Evarts (11, 12) has clearly demonstrated that during desynchronized sleep there is a striking change in the pattern of discharge of single corticospinal neurons. Bursts of high frequency impulses suddenly appear, which are interrupted by prolonged periods of complete inactivity. The peculiar pattern of pyramidal activity affects not only the corticospinal neurons of the motor cortex (11, 12) but also the nerve cells lying in the somatosensory cortex (21, 22).

Arduini *et al.* (2), and Le Yaouanc and Rougeul (14), by recording the integrated pyramidal discharge, found an increase in the overall corticospinal activity during the desynchronized episodes of sleep. The increase, however, was characterized by a steady course; and phasic enhancements were not reported. Evarts (11, p. 168) pointed out that his single unit findings could be reconciled with those of Arduini *et al.* (2) by assuming that the high frequency outbursts of the individual corticospinal neurons might not occur synchronously during desynchronized sleep. Previous experiments (17, 18, 19, 21, 22) and the present observations indicate that a synchronization of the corticofugal discharges is actually present

Fig. 7. — Absence of bursts of REM and striking reduction of myoclonic activity during desynchronized sleep, following bilateral lesion of the medial and descending vestibular nuclei.

Unrestrained, unanesthetized cat. Experiment made 6 days after complete bilateral destruction of the medial and descending vestibular nuclei and chronic implantation of the electrodes. Bipolar records. 1: left parieto-occipital (EEG); 2: right parieto-occipital (EEG); 3: EMG of the posterior cervical muscles; 4: left tibialis anterior (EMG); 5: right tibialis anterior (EMG); 6: EOG.

Although the EMG silence started in A and ended in E, the EEG became desynchronized only at the end of A and the low voltage fast activity persisted throughout B and during the first part of C. Hence the cervical atonia heralded and markedly outlasted the EEG desynchronization (cf. 28). Note absence of bursts of REM; only slow ocular movements or isolated ocular jerks can be detected. Only a few myoclonic twitches, unrelated with the isolated ocular movements, occur throughout the period of cervical atonia (compare Figs. 7 and 8).

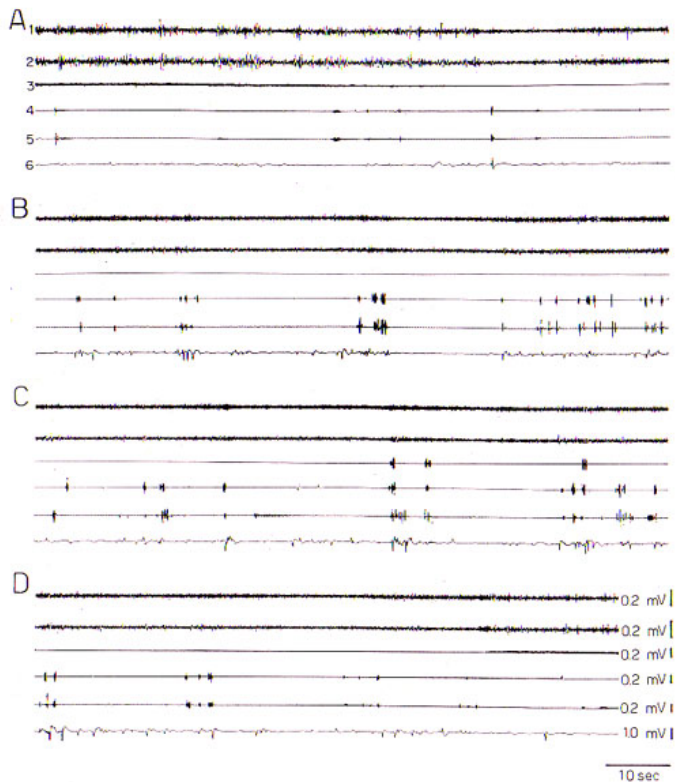


Fig. 8. — Persistence of bursts of REM and of related myoclonic twitches during desynchronized sleep, following unilateral lesion of the vestibular nuclei.

Unrestrained, unanesthetized cat. Experiment made 11 days following complete electrolytic lesion of the superior, lateral and descending vestibular nuclei of the left side and chronic implantation of the electrodes. Bipolar records as in Fig. 7.

This time EEG desynchronization and EMG silence appear simultaneously (A) and persist throughout B and C up to the end of D. Bursts of REM and related, bilateral groups of myoclonic twitches are conspicuously present (compare with Fig. 7).

during desynchronized sleep and appears to be related with the REM outbursts¹.

The present study was devoted to an analysis of the central mechanisms which are responsible for the phasic enhancement of the pyramidal discharge. Particular attention was devoted to the vestibular nuclei because it had been previously found that the rate of discharge of single vestibular neurons recorded from the medial and descending vestibular nuclei in the unrestrained, unanesthetized cat greatly increased during the bursts of REM (5, 6, 7). Furthermore electrolytic lesion of these same nuclei abolished the typical bursts of REM (26, 27, 28). The present experiments confirm and extend the previous findings (26, 27, 28). They show that not only the REM, but also the related phasic enhancements of the pyramidal discharge are abolished by the vestibular lesion. A tonic increase of the integrated pyramidal discharge (2, 14) could hardly be detected in these experimental conditions. As previously reported (26, 27, 28) the genuine tonic phenomena of the desynchronized phase, namely EEG flattening and cervical atonia, were unaffected by the vestibular lesion.

These observations suggest that volleys ascending from the vestibular nuclei influence the corticospinal neurons. In cats under chloralose anesthesia a discharge of impulses may be recorded from the bulbar pyramid on single shock stimulation of the vestibular nerves (20). The present experiments show that this ascending vestibular connection to the sensorymotor cortex is particularly active during the desynchronized phase of sleep.

It is of interest that the excitatory influences exerted by the vestibular nuclei on the cerebral cortex involve the corticospinal neurons originating from both the motor (11, 12, 17, 18, 19) and the somatosensory cortex (21, 22). The pyramidal volleys originating from the motor cortex act mainly on flexor motoneurons, through the interneurons of the flexion reflex pathway (15, 16). On the other hand the pyramidal discharge originating from the somatosensory cortex exerts presynaptic inhibitory influences on the central endings of both the cutaneous and high threshold muscular affe-

¹ The synchronous high frequency discharges of several corticospinal units during the REM might be associated with potential oscillations in the electrocorticogram of the motor cortex. This aspect of the problem is now under investigation (O. P.).

rents (10, 1). Thus when the ascending vestibular volleys reach the frontal cortex, they excite two parallel corticospinal systems acting respectively i) on spinal motoneurons and ii) on primary afferents.

These experiments have some relevance also to the problem of the myoclonic twitches, which represent (with the REM) the major motor manifestation of the desynchronized phase of sleep (13). These twitches are strikingly reduced in both intensity and frequency after the bilateral vestibular lesion. This effect cannot be explained by the abolition of a phasic vestibulospinal barrage because the myoclonic twitches are still present following section of the ventral funiculi (13). It has been shown that the descending volleys which are responsible for the phasic excitation of the motoneurons course mainly along the dorsolateral funiculi (13), where most of the pyramidal tract fibers are also located. These observations, together with our findings on the phasic enhancement of the pyramidal barrage during the bursts of REM (17, 18, 19, 21, 22) suggest that the vestibular nuclei evoke phasic discharges of the motoneurons indirectly through the roundabout way of the motor cortex. It might be predicted, however, that descending extrapyramidal pathways are driven by the vestibular volleys since the myoclonic twitches are not abolished by pyramidotomy.

SUMMARY

1) Pyramidal activity has been recorded at the medullary level in unrestrained, unanesthetized cats before and after bilateral destruction of the vestibular nuclei.

2) Bilateral lesions of the vestibular nuclei do not substantially alter the rhythm of wakefulness and sleep, nor do they prevent the appearance of the synchronized and desynchronized phases of sleep. Previous findings showing that the bursts of REM are abolished by the vestibular lesion were confirmed. Only isolated ocular movements could be detected after this operation.

3) The changes in the integrated pyramidal discharge occurring during wakefulness and synchronized sleep were apparently unmodified by the vestibular lesion. This operation, however, abolished the phasic enhancements of the pyramidal discharge that occur synchronously with the bursts of REM. Also the myoclonic

twitches, usually related in time with the bursts of REM, were abolished after the lesion.

4) The abolition of the phasic enhancements of pyramidal activity was obtained only when the vestibular nuclei of both sides were completely destroyed. Bilateral lesions limited to the medial and descending vestibular nuclei were equally effective. Unilateral lesion of the vestibular nuclei or bilateral lesion limited to the superior and lateral vestibular nuclei, however, did not prevent the appearance of the REM, and the related phasic enhancement of the pyramidal discharge.

REFERENCES

- ANDERSEN, P., ECCLES, J. C. and SEARS, T. A. Cortically evoked depolarization of primary afferent fibers in the spinal cord. *J. Neurophysiol.*, 27: 63-77, 1964.
- ARDUINI, A., BERLUCCHI, G. and STRATA, P. Pyramidal activity during sleep and wakefulness. *Arch. ital. Biol.*, 101: 530-544, 1963.
- ARDUINI, A. and PINNEO, L. R. A method for the quantification of tonic activity in the nervous system. *Arch. ital. Biol.*, 100: 415-424, 1962.
- BEIDLER, L. M. Properties of chemoreceptors of tongue of rat. *J. Neurophysiol.*, 16: 595-607, 1953.
- BIZZI, E., POMPEIANO, O. e SOMOGYI, I. Attività spontanea di singole unità registrate dai nuclei vestibolari nei gatti integri non anestetizzati nel corso del sonno e della veglia. *Boll. Soc. ital. Biol. sper.*, 40: 138-141, 1964.
- BIZZI, E., POMPEIANO, O. and SOMOGYI, I. Vestibular nuclei: activity of single neurons during natural sleep and wakefulness. *Science*, 145: 414-415, 1964.
- BIZZI, E., POMPEIANO, O. and SOMOGYI, I. Spontaneous activity of single vestibular neurons of unrestrained cats during sleep and wakefulness. *Arch. ital. Biol.*, 102: 308-330, 1964.
- BRODAL, A. and POMPEIANO, O. The vestibular nuclei in the cat. *J. Anat.*, 91: 438-454, 1957.
- BRODAL, A., POMPEIANO, O. and WALBERG, F. *The vestibular nuclei and their connections. Anatomy and functional correlations.* Edinburgh, Oliver and Boyd, VIII-193 pp., 1962.
- CARPENTER, D., LUNDBERG, A. and NORRSELL, V. Primary afferent depolarization evoked from the sensorimotor cortex. *Acta physiol. scand.*, 59: 126-142, 1963.
- EVARTS, E. V. Temporal patterns of discharge of pyramidal tract neurons during sleep and waking in the monkey. *J. Neurophysiol.*, 27: 152-171, 1964.
- EVARTS, E. V. Neuronal activity in visual and motor cortex during sleep and waking. Pp. 189-212. In *Aspects anatomo-fonctionnels de la physiologie du sommeil.* Lyon, 9-11 Septembre 1963. Éditions du Centre National de la Recherche Scientifique, Paris, 1965.
- GANSEL, M. M., MARCHIAFAVA, P. L. and POMPEIANO, O. Phasic changes in muscular activity during desynchronized sleep in unrestrained cats. An analysis of the pattern and organization of myoclonic twitches. *Arch. ital. Biol.*, 102: 449-470, 1964.
- LE YAOUANQ, A. et ROUGEUL, A. Activités pyramidales recueillies chez le chat pendant le sommeil naturel. *J. Physiologie*, 57: 649-650, 1966.

15. LUNDBERG, A., NORRSELL, U. and VOORHOEVE, P. Pyramidal effects on lumbo-sacral interneurons activated by somatic afferents. *Acta physiol. scand.*, 56: 220-229, 1962.
16. LUNDBERG, A. and VOORHOEVE, P. Effects from the pyramidal tract on spinal reflex arcs. *Acta physiol. scand.*, 56: 201-219, 1962.
17. MARCHIAFAVA, P. L. e POMPEIANO, O. Scariche cortico-spinali e inibizione di risposte motorie a stimolazione piramidale nel sonno desincronizzato. *Rend. Accad. naz. Lincei, Cl. Sci. fis., mat. nat., Ser. VIII*, 36: 547-549, 1964.
18. MARCHIAFAVA, P. L. and POMPEIANO, O. Pyramidal influences on spinal cord during desynchronized sleep. *Arch. ital. Biol.*, 102: 500-529, 1964.
19. MARCHIAFAVA, P. L. and POMPEIANO, O. Attività piramidale durante il sonno desincronizzato. *Com. Soc. italo-francese di EEG e Neurofisiologia*, Padova, 1964 (in press).
20. MEGIRIAN, D. and TROTH, R. Vestibular and muscle nerve connections to pyramidal tract neurons of cat. *J. Neurophysiol.*, 27: 481-492, 1964.
21. MORRISON, A. R. and POMPEIANO, O. Corticospinal influences on primary afferents during sleep and wakefulness. *Experientia*, 21: 660-661, 1965.
22. MORRISON, A. R. and POMPEIANO, O. Pyramidal discharge from somatosensory cortex and cortical control of primary afferents during sleep. *Arch. ital. Biol.*, 103: 538-568, 1965.
23. MORRISON, A. R. e POMPEIANO, O. Influenze vestibolari sull'attività piramidale durante i movimenti rapidi oculari nel sonno desincronizzato. *Boll. Soc. ital. Biol. sper.*, 41: 937-939, 1965.
24. POMPEIANO, O. Muscular afferents and motor control during sleep. Pp. 415-436. In Granit, R. (Ed.), *Nobel Symposium I: Muscular afferents and motor control*. Stockholm Almqvist and Wiksell, 1966.
25. POMPEIANO, O. The neurophysiological mechanisms of the postural and motor events during desynchronized sleep. In *Sleep and altered states of consciousness*. Meeting of the Association for Research in Nervous and Mental Disease, 3-4 December 1965, New York (in press).
26. POMPEIANO, O. e MORRISON, A. R. Abolizione dei movimenti rapidi oculari nel sonno desincronizzato prodotta da lesioni vestibolari. *Boll. Soc. ital. Biol. sper.*, 14: 935-937, 1965.
27. POMPEIANO, O. and MORRISON, A. R. Vestibular origin of the rapid eye movements during desynchronized sleep. *Experientia*, 22: 60-61, 1966.
28. POMPEIANO, O. and MORRISON, A. R. Vestibular influences during sleep. I. Abolition of the rapid eye movements during desynchronized sleep following vestibular lesions. *Arch. ital. Biol.*, 103: 569-595, 1965.
29. POMPEIANO, O. and SWETT, J. E. EEG and behavioral manifestations of sleep induced by cutaneous nerve stimulation in normal cats. *Arch. ital. Biol.*, 100: 311-342, 1962.
30. POMPEIANO, O. and SWETT, J. E. Identification of cutaneous and muscular afferent fibers producing EEG synchronization or arousal in normal cats. *Arch. ital. Biol.*, 100: 342-380, 1962.