Sleep, 7(3):202–210

Phasic Activity of the Basolateral Amygdala, Cingulate Gyrus, and Hippocampus During REM Sleep in the Cat

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Summary: We analyzed the electrical activity of the basolateral amygdala (BLA), anterior and posterior regions of the cingulate gyrus (A-CG and P-CG), the dorsal hippocampus (DH), the anterior ventral thalamic nucleus (AVTN), and the sensory motor cortex during the rapid eye movements and ponto-geniculo-occipital (PGO) activity of REM sleep in cats in chronic preparation. Polygraphic recordings and computational perievent averages using the phasic contractions of the lateral rectus muscle (LR) of the eyeball as the triggering signal of the analysis were performed. We observed biphasic potentials (200–300 ms) of variable amplitude, related to the phasic phenomena of REM sleep, in the BLA, A-CG, P-CG, DH, and AVTN. The latencies of the potentials of these regions were always greater than those of the geniculate PGO activities. We propose that the recorded limbic potentials resulted from propagation of PGO activity and that this phenomenon may reflect the limbic structure of the hallucinatory, vegetative, and emotional components of REM sleep. **Key Words:** Sleep—Amygdala—Hippocampus—Gyrus cinguli—REM sleep—Ponto-geniculo-occipital activity.

Jouvet and Michel (1) demonstrated the existence of monophasic spikes in cats at the level of the pons during REM sleep. This electrical activity is propagated from the pontine region (2,3) to the cortical and subcortical structures of the visual system (4–6). Jeannerod et al. (7) were the first to call the phenomenon ponto-geniculo-occipital (PGO) activity. Ponto-geniculo-occipital activity has been studied thoroughly in the oculomotor system (8–12).

In man emotional changes, mnestic phenomena of a personal and conceptual type and vegetative changes accompany the rapid eye movements of REM sleep. Jouvet et al. (13), Snyder et al. (14), Baust and Bohenert (15), Welch and Richardson (16), and Fernández-Guardiola et al. (17) have described variations in the frequency of both cardiac and respiratory rates related to rapid eye movements.

The functions related to emotional and vegetative changes are integrated in the limbic system and in the hypothalamus. Electrical stimulation of these structures elicits emotional

Accepted for publication February 1984.
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and vegetative changes in the cat (18–21) and in the monkey (22). In man, in addition to these changes, hallucinatory and reminiscent phenomena are also provoked (23–28). It may be thought that during REM sleep PGO activity stimulates the limbic system to elicit these changes. Hobson (29) has demonstrated PGO-related potentials in various thalamic areas anatomically related to this system. The object of the present work was to test this hypothesis in cats by recording the bioelectrical activity of the basolateral amygdala (BLA), the cingulate gyrus, and the hippocampus during periods of PGO activity in REM sleep using a computational system of perievent analysis of the REM-sleep phasic phenomena.

METHODS

Eight adult male cats in chronic preparation were used. Bipolar concentric stainless steel enameled electrodes (cannula gauge, 0.7 mm; Teflon-insulated wire gauge, 0.17 mm; tip separation, 0.7–1.0 mm) were stereotaxically (30) implanted into the lateral geniculate body (LGB), the pontine reticular formation (PRF), the BLA, the anterior and posterior regions of the cingulate gyrus (A-CG and P-CG, respectively; coordinates: A: 17.0, L: 1.4, H: +10.7; and A: 3.0, L: 0.8, H: +11.0), the anterior ventral thalamic nucleus (AVTN), the dorsal hippocampus (DH), and the sensory motor cortex (SMC). Stainless steel electrodes, placed epidurally, were used to obtain the electrocorticogram of the visual cortex (VC). Electrodes were also placed to record the electromyogram (EMG) of the nuchal muscles and the electrooculogram (EOG). Fish hooks were used in recording the EMG of the lateral rectus muscle (LR) of both eyeballs (12).

After surgery conducted under sodium pentobarbital (33 mg/kg) anesthesia, the animals recovered for 15 days. Each cat was placed in a soundproof recording chamber under the same conditions as those used in the experiments for 8 habituation days. After habituation, sleep recordings were performed daily from 1000 to 1800 h.

The electrophysiological activities were amplified and polygraphically recorded. These signals were also recorded on magnetic tapes and were analyzed on-line. Perievent averages of 200 ms before and 800 ms after a phasic contraction of the LR during REM sleep were analyzed. Thirty averages of 256 activations of the LR and of the concomitant activity of the VC, PRF, LGB, AVTN, A-CG, P-CG, DH, BLA, and SMC were performed.

A total of 7,680 phasic events during REM sleep were computed. Four to five stages of REM sleep were necessary to complete the 256 phasic activation averages. Therefore, 120 to 150 REM stages over approximately 20 successive days were studied in each animal.

The latencies were calculated at the first negative (downward) component (N_1) of each potential, taking the N_1 of the PRF-PGO potential as reference. The Student's t test was used to evaluate the statistical significance of the differences between latency average differences.

At the end of the experiments, the animals were sacrificed by an overdose of pentobarbital, and a 10 mA DC current of 5 s duration was passed through the electrodes to mark the location of the tip. The brains were perfused with 20% formol and serial histological sections (80 μm) were made to verify the location of the electrodes.

RESULTS

During REM sleep, biphasic potentials of variable amplitude (50–150 μV) and shape interrupted the basal electrographic activity of the BLA. The biphasic potentials were related to phasic contractions of both LRs and PGO activity (Fig. 1). The A-CG, P-CG,

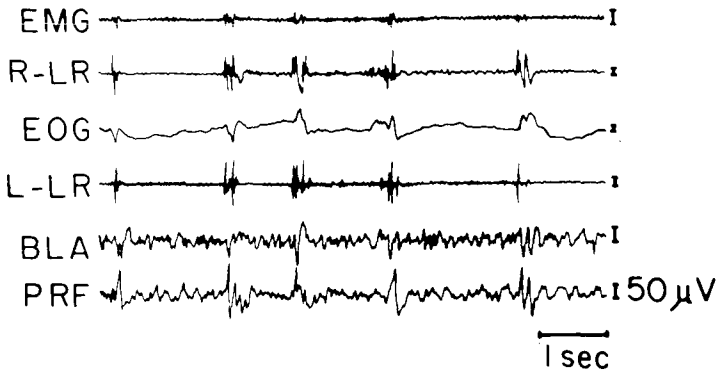

FIG. 1. Polygraphic recording during REM sleep. Note the appearance of slow potentials of variable shape and amplitude in the basolateral amygdala (BLA) related to the phasic contractions of the lateral rectus muscles of each eyeball (R-LR and L-LR, respectively) and to the ponto-geniculo-occipital potentials of the pontine reticular formation (PRF). EMG, electromyogram of the nuchal muscles; EOG, electrooculogram.

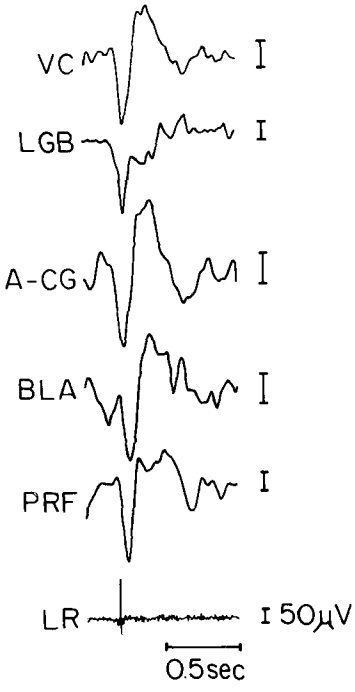

FIG. 2. Average of 256 ponto-geniculo-occipital and related subcortical potentials during REM stage. A-CG, anterior cingulate gyrus; BLA, basolateral amygdala; LGB, lateral geniculate body; LR, lateral rectus muscle of the eyeball; PRF, pontine reticular formation; VC, visual cortex. Note the presence of slow biphasic potentials in A-CG and BLA.

and DH also exhibited PGO activity related to biphasic potentials. In the SMC, potentials related to the REM phasic phenomena were not observed.

The perievent averages of BLA and A-CG activities indicated a relationship between the limbic potentials and PGO activity, revealing slow biphasic potentials (200–300 ms) of an amplitude between 150 and 200 μV (Fig. 2).

To test the existence of a relay structure in the propagation of PGO activity from the LGB to the cingulate gyrus, perievent averages of the AVTN together with the activity of the LGB and A-CG were made. The results in the AVTN showed slow biphasic potentials related to PGO activity. The PGO potentials of LGB were always the first to appear and

were followed successively by the potentials recorded in the VC, AVTN, A-CG, and BLA (Fig. 3)

The activity of the DH and P-CG was analyzed concurrently to elucidate the shape, amplitude, duration, and latencies of the PGO-related potentials in these areas. The averages demonstrated the presence of slow biphasic potentials (200–300 ms) with an amplitude of between 150 and 200 μV in the P-CG and DH. These were related to the A-CG and VC-PGO potentials (Fig. 4). The amplitude of the DH potentials was greatest when the cannula was in the CA 2 area and the wire tip in the fascia dentata. Analysis of the SMC did not demonstrate the presence of PGO-related potentials.

When quantifying the temporal distribution of the potentials, we observed that the latencies of the beginning of each potential varied greatly and occasionally were difficult to determine accurately, whereas the N_1 peak latencies of all the potentials were more constant. We therefore determined the temporal relation between the potentials using the latencies of their N_1 peak, taking the PGO potential of the PRF as a point of reference. Figure 5 shows the average latencies and the standard deviation of the potentials recorded in each structure. As can be seen, the P-CG potential preceded that of the A-CG. There was also a delay between the hippocampal and the amygdaloid PGO-related potentials. The latencies of the potentials recorded from VC, AVTN, P-CG, A-CG, DH, and BLA were significantly longer ($p < 0.001$) than the latency of the LGB-PGO potential.

The latency of the BLA potential was also significantly longer ($p < 0.001$) than the latencies of DH, A-CG, P-CG, AVTN, and VC potentials. The latency of the DH potential was significantly longer than those of the A-CG ($p < 0.005$), P-CG ($p < 0.005$), and AVTN ($p < 0.001$). There were no significant differences between the latencies of the potentials of AVTN, P-CG, and A-CG. Table 1 shows the latency values ± SD in each animal.

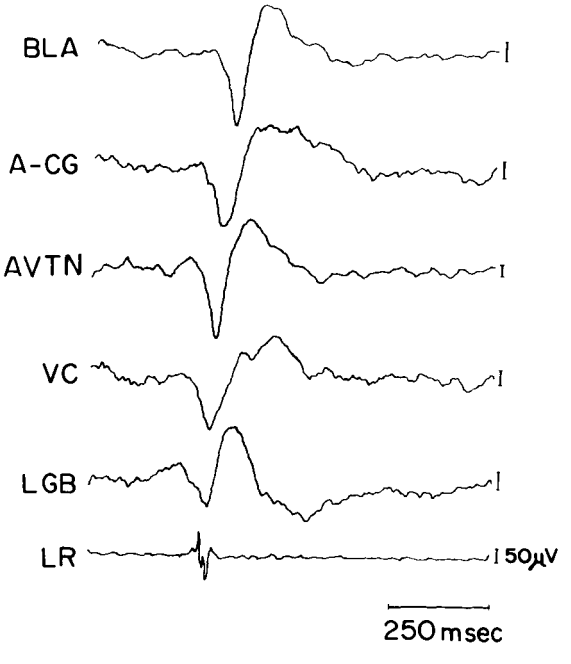

FIG. 3. Ponto-geniculo-occipital propagation to thalamic and limbic structures. Expanded sweep allows the appreciation of latency differences. AVTN, anterior ventral thalamic nucleus; for other abbreviations see Fig. 2. Note that A-CG and BLA display the longest latencies.

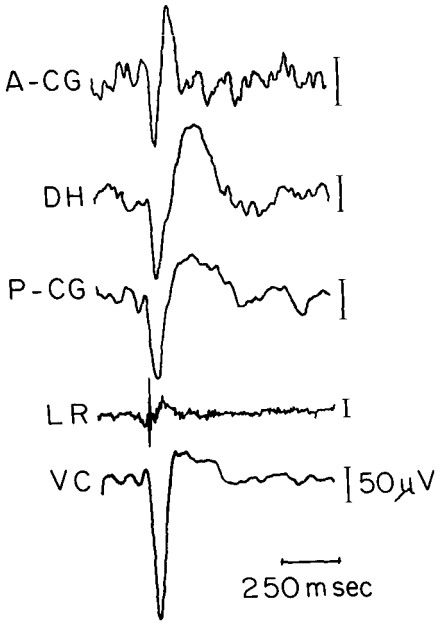

FIG. 4. Average of the hippocampal and cingular potentials during REM phasic phenomena. DH, dorsal hippocampus; P-CG, posterior cingulate gyrus; for other abbreviations see Fig. 2. Note the presence of slow biphasic potentials in DH and P-CG related to the VC-PGO and to the A-CG potential.

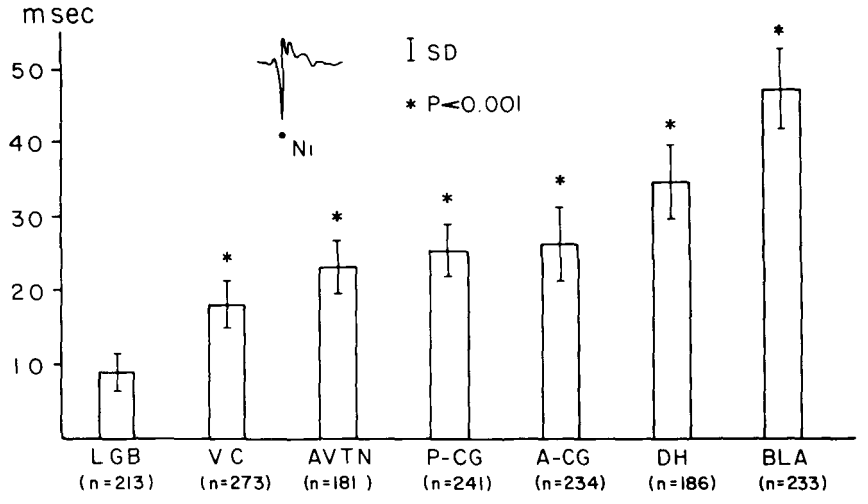

FIG. 5. The N_1 mean latency of PGO and related potentials, taking PRF-PGO as reference. The mean latencies of all the structures were compared with LGB mean latency. Statistical analysis was carried out with the Student's *t* test. See previous figures for abbreviations.

DISCUSSION

The appearance of potentials in the BLA, DH, A-CG, P-CG, and AVTN, related to PGO activity and LR phasic contractions, confirmed that these limbic and thalamic structures are phasically influenced during REM sleep.

In addition to receiving somesthesic, auditory, and visceral information, the BLA receives visual information, probably through the thalamic relay nuclei and, among them, the LGB (31–35). Impulses from the reticular formation of the brain stem (36) also arrive at the amygdala. The hippocampus, the cingulate gyrus, and the amygdala also receive polysensory

TABLE 1. *First negative component peak (N_1) latencies in ms ($\bar{x} \pm SD$) measured from the PRF-PGO potential in each cat*

Cat no.	LGB	VC	AVTN	P-CG	A-CG	DH	BLA
1	—	17.0 ± 2.2 (30)	—	25.2 ± 2.6 (49)	30.2 ± 3.3 (48)	39.0 ± 2.9 (48)	52.5 ± 3.2 (48)
2	8.1 ± 2.2 (40)	20.2 ± 2.9 (40)	—	27.1 ± 3.5 (47)	22.5 ± 3.7 (50)	37.1 ± 3.5 (45)	—
3	—	20.1 ± 2.7 (30)	25.2 ± 2.1 (47)	—	24.0 ± 4.2 (46)	—	50.0 ± 4.5 (45)
4	10.0 ± 1.9 (40)	16.0 ± 1.6 (40)	—	—	29.1 ± 3.3 (45)	31.1 ± 3.0 (47)	42.5 ± 3.6 (47)
5	7.2 ± 2.0 (30)	15.1 ± 2.6 (30)	—	24.0 ± 3.3 (45)	26.1 ± 3.6 (45)	—	—
6	11.2 ± 2.3 (35)	17.0 ± 2.2 (35)	19.1 ± 2.4 (47)	27.1 ± 3.1 (50)	—	—	48.0 ± 3.5 (50)
7	8.0 ± 2.2 (33)	21.0 ± 3.0 (33)	23.0 ± 3.4 (45)	—	—	30.0 ± 3.3 (48)	43.0 ± 3.6 (43)
8	10.1 ± 2.8 (35)	19.2 ± 2.2 (35)	24.3 ± 2.4 (46)	23.1 ± 3.1 (50)	—	—	—

A-CG, anterior cingulate gyrus; AVTN, anterior ventral thalamic nucleus; BLA, basolateral amygdala; DH, dorsal hippocampus; LGB, lateral geniculate body; P-CG, posterior cingulate gyrus; PGO, ponto-geniculo-occipital activity; PRF, pontine reticular formation; VC, visual cortex.
Number in parentheses is number of latencies.

information through the thalamus (37,38) and through fibers from the pontine region, the locus coeruleus, the parabrachial nucleus, and the raphe nuclei (38–41).

Because the latencies of the potentials recorded in the BLA, P-CG, A-CG, DH, and AVTN are much greater than those of the PGO potentials, the activation of these limbic and thalamic structures may be the result of the propagation of PGO activity starting in the pons or in the thalamus (LGB) in a multisynaptic manner. The temporal difference between these potentials and the phasic contractions of the LR eliminates the possibility that they are due to an electrotonic propagation of the muscle contraction.

A possible propagation pathway may be through the fibers of the supraoptical decussation to the contralateral LGB (10). These fibers send collaterals to the supraoptical and tuberal regions of the hypothalamus, which are connected to the mammillary body, which in turn sends fibers to the AVTN through the mammillary-thalamic bundle. The AVTN sends fibers to the cingulate gyrus (38,42,43), which is in turn connected to the amygdaloid nuclei. Starting in the thalamus, the propagation of the PGO activity to the amygdala, the cingulate gyrus, and the hippocampus may take diverse pathways. In fact, Hobson (29) recorded PGO-related potentials in several thalamic nuclei.

Another possible mechanism of propagation may be the pontine-limbic system described by Nauta and Kuypers (44). The ascending fibers originate in the mesencephalic limbic area and run through two systems: the ascending component of the dorsal longitudinal fascicle of Shutz and the mammillary peduncle, which terminates in the hypothalamus. Some of these fibers reach the amygdala. From this circuit, impulses are sent to the intralaminary nuclei of the thalamus and then to the cingulate gyrus. Fuxe et al. (45) demonstrated the presence of catecholaminergic and indolaminergic fibers that connect the pontine nuclei (locus coeruleus and raphe nuclei) to the amygdaloid nuclei complex. Kuhar (46), Jones and Moore (47), Ottersen and Ben-Ari (48), and Cedarbaum and Aghajanian

(49) have demonstrated the existence of fibers that connect the BLA and the hippocampus to the pontine regions and the locus coeruleus.

White and Jacobs (50) reported that the cells of the lateral amygdaloid nucleus exhibit bursts of increased discharge frequency during slow wave sleep and REM sleep. Nevertheless, these authors did not find any relation between the amygdaloid bursts and the rapid eye movements of REM sleep. This may be due to their method of analyzing electrical activity or because the activity of the lateral, as opposed to the basal, amygdaloid nucleus does not reveal changes during rapid eye movements. Ravagnati et al. (51) reported that in man the unit activities of the hippocampus gyrus and of the amygdala show an increase in their discharge frequency during REM sleep, these frequencies being greater than those observed during slow wave sleep or wakefulness. Velluti and Monti (52) found that in the cat there was a direct relation between the activity of the reticularis pontis caudalis nucleus and that of the basal nucleus of the amygdala during the rapid eye movements of REM when they recorded the changes in Po_2 in those structures. The electrical stimulation of the temporal lobe amygdala, the cingulate gyrus, and the hippocampus elicits changes in some vegetative functions. Likewise, visual and auditory hallucinatory phenomena and mnestic phenomena, including those of the sexual type, have been elicited. These hallucinatory, mnestic, and vegetative phenomena have also been described during REM sleep (14,53–58). In addition, electrical stimulation of the amygdala, hippocampus, and cingulate gyrus of man evokes the sensation of dreaming (24,26,28,59,60).

In the cat elaborated hallucinatory or oneiric behavior (orientation, predatory and aggressive attack, rage and flight) during atonia-suppressed REM sleep has been demonstrated (61–64). The ascending PGO activity impinging on the visual system has been involved in visual phenomena, but no explanation has been found for the other components of oneiric behavior. Penfield and Rasmussen (65) and Penfield and Jasper (66) showed that electrical stimulation of the primary visual cortex of conscious patients produces only gross light, shadows, outline, and color sensations. Responses to stimulation in the auditory area are also elementary (clicking, humming, etc.). No elaborated visual phenomena are produced from these areas at any time. Since the activation of visual areas produces only elementary sensations, the emotional and elaborated sensory phenomena of REM sleep could be better explained by the PGO propagation to the limbic areas evidenced in the present study.

REFERENCES

1. Jouvet M, Michel F. Corrélations electromyographiques du sommeil chez le chat décortiqué et mésencéphalique chronique. *C R Soc Biol (Paris)* 1959;153:422–5.
2. Hobson JA, McCarley RW, Pivik RT, Freeman R. Selective firing by cat pontine brain stem neurons in desynchronized sleep. *J Neurophysiol* 1974;37:497–511.
3. Sakai K, Jouvet M. Brain stem PGO-on cells projecting to the cat dorsal lateral geniculate nucleus. *Brain Res* 1980;194:500–5.
4. Mikiten T, Niebyl P, Hendley C. EEG desynchronization during behavioral sleep associated with spike discharges from the thalamus of the cat. *Fed Proc* 1961;20:327.
5. Bizzi E, Brooks DC. Functional connections between pontine reticular formation and lateral geniculate nucleus during deep sleep. *Arch Ital Biol* 1963;101:666–80.
6. Mouret J, Jeannerod M, Jouvet M. L'activité électrique du systeme visuel au cours de la phase paradoxale du sommeil chez le chat. *J Physiol (Paris)* 1963;55:305–6.
7. Jeannerod M, Mouret J, Jouvet M. Effets secondaires de la déafferentation visuelle sur l'activité électrique phasique ponto-géniculo-occipitale du sommeil paradoxal. *J Physiol (Paris)* 1965;57:255–6.
8. Michel F, Jeannerod M, Mouret J, Rechtshaffen A, Jouvet M. Sur les mécanismes de l'activité de pointes au niveau du systeme visuel au cours de la phase paradoxal du sommeil. *C R Soc Biol (Paris)* 1964;158:103–6.
9. Jeannerod M, Kiyono S. Décharge unitaire de la formation réticulée pontique et activité phasique PGO chez le chat sous réserpine. *Brain Res* 1969;12:122–8.

10. Laurent JP, Cespuglio R, Jouvet M. Délimitation des voies ascendantes de l'activité ponto-géniculo-occipitale chez le chat. *Brain Res* 1974;65:29–52.
11. Cespuglio R, Laurent JP, Jouvet M. Etude des relations entre l'activité ponto-géniculo-occipitale (PGO) et la motricité oculaire chez le chat sous réserpine. *Brain Res* 1975;83:319–35.
12. Cespuglio R, Laurent JP, Calvo JM. Organisation anatomique des activités phasiques provoquées par la réserpine au niveau du systeme oculo-moteur. *Electroencephalogr Clin Neurophysiol* 1976;40:12–24.
13. Jouvet M, Michel F, Mounier D. Analyse électroencephalographique comparée du sommeil physiologique chez le chat et chez l'homme. *Rev Neurol (Paris)* 1960;103:189–204.
14. Snyder F, Hobson J, Morrison B, Goldfrank F. Changes in respiration, heart rate and systolic blood pressure in relation to electroencephalographic patterns of human sleep. *J Appl Physiol* 1964;19:417–22.
15. Baust W, Bohenert B. The regulation of heart rate during sleep. *Exp Brain Res* 1969;7:169–80.
16. Welch AJ, Richardson PC. Computer sleep stage classification using heart rate data. *Electroencephalogr Clin Neurophysiol* 1973;34:145–52.
17. Fernández-Guardiola A, Calvo JM, Contreras C, Brailowsky S, Solís H. Heart rate and galvanic skin response variations during the REM and non-REM sleep phases in man. *Electroencephalogr Clin Neurophysiol* 1973;34:694–5.
18. Kaada BR. Somatomotor autonomic and electroencephalographic responses to electrical stimulation of "rhinencephalic" and other structures in primates, cat and dog: a study of responses from the limbic, subcallosal, orbito insular, piriform and temporal cortex, hippocampus-fornix and amygdala. *Acta Physiol Scand* 1951;24(suppl 83):1–285.
19. MacLean PD, Delgado JMR. Electrical and chemical stimulation of fronto-temporal portion of limbic system in the waking animal. *Electroencephalogr Clin Neurophysiol* 1953;5:91–100.
20. Ursin H, Kaada BR. Functional localization within the amygdaloid complex in the cat. *Electroencephalogr Clin Neurophysiol* 1960;12:1–20.
21. Kaada BR. Stimulation and regional ablation of the amygdaloid complex with reference to functional representations. In: Eleftheriou BE, ed. *The neurobiology of the amygdala*. New York: Plenum Press, 1972:205–81.
22. Reis DJ, Oliphant MC. Bradycardia and tachycardia following electrical stimulation of the amygdaloid region in monkey. *J Neurophysiol* 1964;27:893–912.
23. Heath RG. Pleasure response of human subjects to direct stimulation of the brain: physiologic and psychodynamic considerations. In: Heath RG, ed. *The role of pleasure in behavior*. New York: Harper and Row, 1964:219–43.
24. Brazier MAB. Stimulation of the hippocampus in man using implanted electrodes. In: Brazier MAB, ed. *RNA and brain function, memory and learning*. Berkeley: University of California Press, 1966:299–310.
25. Velazco-Suárez M, Fernández-Guardiola A. Fenómenos reminiscentes durante el estudio y el tratamiento de epilepsia temporal con electrodos de profundidad. *Gac Med Mex* 1968;98:1533–7.
26. Bancaud J, Talairach J, Geier S, Bonis A, Trottier S, Manrique M. Manifestations comportamentales induites par la stimulation électrique du gyrus cingulaire antérieur chez l'homme. *Rev Neurol (Paris)* 1976;132:705–24.
27. Fernández-Guardiola A. Reminiscences elicited by stimulation of the temporal lobe in humans. In: Druker-Colín R, McGaugh JL, eds. *Neurobiology of sleep and memory*. New York: Academic Press, 1977:273–80.
28. Halgren E, Walter RD, Cherlow DG, Crandall PH. Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain* 1978;101:83–117.
29. Hobson JA. L'activité électrique phasique du cortex et du thalamus au cours du sommeil désynchronise chez le chat. *Soc Biol (Lyon)* 1964:2134–5.
30. Snider RS, Niemer WT. *A stereotaxic atlas of the cat brain*. Chicago: University of Chicago Press, 1961.
31. Robinson R, Lennox MA. Sensory mechanisms in hippocampus, cingulate gyrus and cerebellum of the cat. *Fed Proc* 1951;10:110–1.
32. Bonvallet M, Dell P, Hugelin A. Projections olfactives, gustatives, viscerales, vagales, visuelles, et auditives, au niveau des formations grises du cerveau antérieur du chat. *J Physiol Pathol Gen* 1952;44:222–4.
33. Gloor P. Electrophysiological studies of the connections of the amygdaloid nucleus in the cat. Part II. The electrophysiological properties of the amygdaloid projection system. *Electroencephalogr Clin Neurophysiol* 1955;7:243–64.
34. Gloor P. Neurophysiology. In: Field J, Magoun HW, Hall VE, eds. *Handbook of physiology, vol 2*. Washington DC: American Physiological Society, Williams and Wilkins, 1960:1395–420.
35. Wendt R, Albe-Fessard, eds. *Sensory responses of the amygdala with special reference to somatic afferent pathways: physiologie de l'hippocampe*. Paris: Centre Nationel de la Recherche Scientifique, 1962.
36. Machne X, Segunda JP. Unitary responses to afferent volleys in amygdaloid complex. *J Neurophysiol* 1956;19:232–40.
37. MacLean PD. Some psychiatric implications of physiological studies on the fronto-temporal portion of the limbic system (visceral brain). *Electroencephalogr Clin Neurophysiol* 1952;4:407–18.

38. Baleydier CH, Mauguiere F. The duality of the cingulate gyrus in monkey. Neuroanatomical study and functional hypothesis. *Brain* 1980;103:525–54.
39. Blackstad TW, Fuxe K, Hökefelt T. Noradrenaline nerve terminals in the hippocampal region of the rat and guinea pig. *Z Zellforsch* 1967;78:463–73.
40. Moore RY, Halaris AE. Hippocampal innervation by serotonine neurons of the rat. *J Comp Neurol* 1975;164:171–84.
41. Russchen FT. Amygdalopetal projections in the cat. II Subcortical afferent connections. A study with retrograde tracing techniques. *J Comp Neurol* 1982;207:157–76.
42. Powell TPS, Cowan WM. The origin of the mamilothalamic tract in the rat. *J Anat* 1954;88::489–97.
43. Locke S, Angeviene JB Jr, Yakovlev PI. Limbic nuclei of the thalamus and connections of limbic cortex. VI Thalamocortical projections of lateral dorsal nucleus in cats and monkeys. *Arch Neurol* 1964;11:1–12.
44. Nauta WJH, Kuypers HGJM. Some ascending pathways in the brain stem reticula formation. In: Jasper HH, Proctor LD, eds. *Reticular formation of the brain*. Boston: Little, Brown, 1958:3–30.
45. Fuxe K, Hakfelt T, Ungerstedt U. Distribution of monoamines in the mammalian central nervous system. In: Hooper E, ed. *Metabolism of amines in the brain*. London: Macmillan, 1969:10–22.
46. Kuhar MJ. Cholinergic neurons: septal-hippocampal relationships. In: Isaacson RL, Pribram KH, eds. *The hippocampus. Structure and development, vol I*. New York: Plenum Press, 1975:269–83.
47. Jones BE, Moore RY. Ascending projections of the locus coeruleus in the rat. II Autoradiographic study. *Brain Res* 1977;127:23–53.
48. Ottersen OP, Ben-Ari Y. Pontine and mesencephalic afferents to the central nucleus of the amygdala of the rat. *Neurosci Lett* 1978;8:329–34.
49. Cedarbaum JM, Aghajanian GK. Afferent projections to the locus coeruleus as determined by a retrograde tracing technique. *J Comp Neurol* 1978;178:1–16.
50. White TJ, Jacobs BL. Single unit activity in the lateral amygdala of the cat during sleep and waking. *Electroencephalogr Clin Neurophysiol* 1975;38:331–3.
51. Ravagnati L, Halgren E, Babb TL, Crandall PH. Activity of human hippocampal formation and amygdala neurons during sleep. *Sleep* 1979;2:161–73.
52. Velluti R, Monti JM. pO_2 Recorded in the amygdaloid complex during the sleep-waking cycle of the cat. *Exp Neurol* 1976;50:798–805.
53. Ramsey G. Studies of dreaming. *Psychol Bull* 1953;50:432–55.
54. Kamiya J. Behavioral, subjective and physiological aspects of sleep and drowsiness. In: Fiske D, Maddi S, eds. *Functions of varied experience*. Dorsey: Homewood, 1961:145–74.
55. Shapiro A, Goodenough D, Biederman I, Sleser I. Dream recall and the physiology of sleep. *J Appl Physiol* 1964;19:778–83.
56. Fisher C, Gross J, Zuch J. Cycle of penile erection synchronous with dreaming (REM) sleep. *Arch Gen Psychiatry* 1965;12:29–45.
57. Snyder F. The organismic state associated with dreaming. In: Greenfield N, Lewis W, eds. *Psychoanalysis and current biological thought*. Madison: University of Wisconsin Press, 1965:275–315.
58. Calvo JM, Contreras CM, Fernández-Guardiola A. Fluctuaciones de la frecuencia cardiaca durante la fase REM [Abstract]. *Bol Estud Med Biol* 1973;28:93.
59. Pampiglione G, Falconer MA. Electrical stimulation of the hippocampus in man. In: Field J, Magoun HW, Hall VE, eds. *Neurophysiology. Handbook of Physiology, vol 18*. Washington DC: American Physiological Society, 1960:1391–4.
60. Penfield WP, Perot P. The brain's record of auditory and visual experience. A final summary and discussion. *Brain* 1963;86:595–696.
61. Jouvet M, Delorme JF. Locus coeruleus et sommeil paradoxal. *C R Soc Biol (Paris)* 1965;159:895–9.
62. Jouvet M. Essai sur le reve. *Arch Ital Biol* 1973;111:564–76.
63. Henley K, Morrison AR. A reevaluation of the effects of lesions of the pontine tegmentum and locus coeruleus on phenomena of paradoxical sleep in cat. *Acta Neurobiol Exp (Warsz)* 1974;34:215–32.
64. Sastre JP, Jouvet M. Le comportement onirique du chat. *Physiol Behav* 1979;22:979–80.
65. Penfield W, Rasmussen T, eds. *The cerebral cortex of man*. New York: Macmillan, 1950.
66. Penfield W, Jasper H, eds. *Epilepsy and the functional anatomy of the human brain*. Boston: Little, Brown, 1954.