Tele-Encephalic Versus Cerebellar Control upon Ponto-Geniculo-Occipital Waves During Paradoxical Sleep in the Cat[1]

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Summary. Studying the effects that removal of the cerebellum and the frontal lobes had upon the phasic activities (PGO waves) of paradoxical sleep in the cat, it is shown that in this phase of the sleep-wakefulness cycle the cerebellum exerts an inhibitory action upon the amplitude of the GPO, while the frontal lobes influences the pattern of their discharges.

Ponto-geniculo-occipital (PGO) waves and concomittant activity in lateral rectus muscles of the eye appear particularly during paradoxical sleep episodes (PS)[3]. The frontal lobes are among other supratentorial mechanisms which influence oculomotor activity[4]. The cerebellum is a complicated neuronal network, which controls movements[5], and recently it has been proposed as an important step in the pathway which influences rapid (saccadic) eye movements[6]. We investigated the functional control that these two structuras have upon the PGO waves and rapid eye movements during paradoxical sleep.

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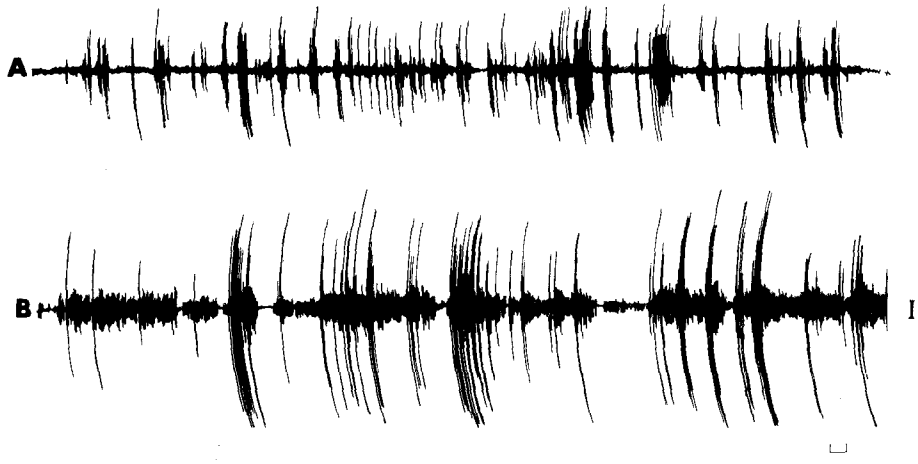

Fig. 1. PGO discharges during paradoxical sleep in lateral rectus muscles of the eye before (A) and after (B) cerebellectomy in the same cat. Vertical calibration = 50 μv; horizontal calibration = 1 sec.

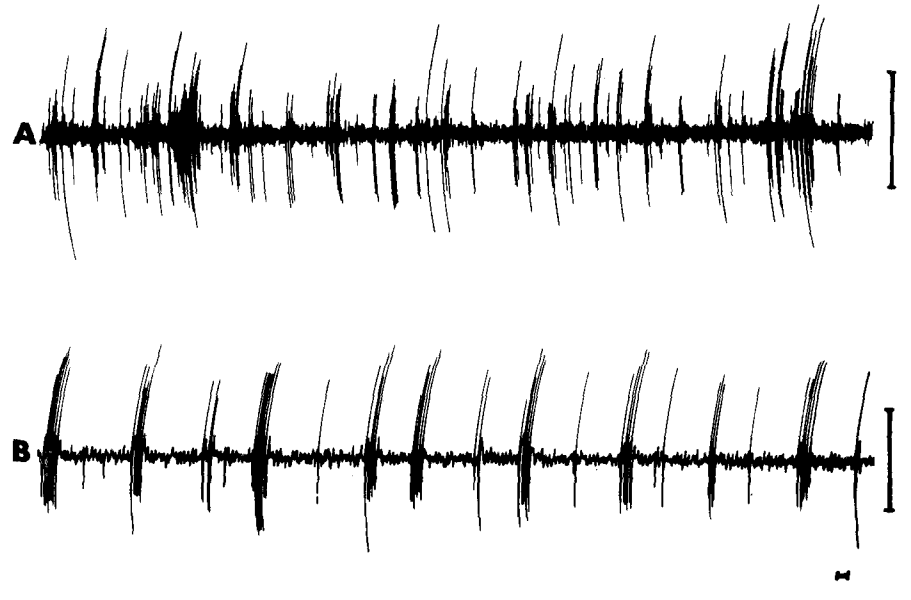

Fig. 2. Pattern of PGO discharge during paradoxical sleep in the 6th nucleus in a normal cat (A) and in another animal with a bifrontal lobectomy 1 day previously (B). Vertical calibration = 50 μv; horizontal calibration = 1 sec.

Methods. The experiments were performed in chronic cats. In 1st operation, the animals have electrodes implanted in the lateral rectus muscle of the eye and in the 6th nucleus of the pons. Cortical and neck electrodes complete the polygraphic study of the sleep-wakefulness cycle. PS episodes were monitored in an 8-channel EEG grass model 7A Polygraph. In a 2nd operation, the animals had either a total ablation of the cerebellum or an ablation of the frontal lobes by removing all the brain tissue in front of a plane passing through the bregma. After these two types of operation, PS episodes were monitored in the same way as in Control experiments.

Results and discussion. After cerebellectomy, the most striking result is the increase in amplitude of PGO waves (Figure 1). This increase in amplitude lasts all the time in all the animals studied (up to 2 weeks after cerebellectomy). It appears in isolated as well as in the PGO discharging in bursts and it affects the positive as well as the negative part of the PGO waves. It is noteworthy that not all of the PGO in the paradoxical sleep episode increase their amplitude. On the contrary, the most striking effect produced by frontal lobe lesions is the change in the pattern of discharges of the PGO, without a noticeable change in their amplitude (Figure 2). The change in pattern of the PGO is very marked in the first 2–3 days following the operation. After 5–6 days there is a new change in the pattern of PGO. It is modified in the sense of a great complexification of the PGO discharges related to control experiments and the bursts of PGO contain a greater number of waves.

It appears from our results that the cerebellum controls mainly the amplitude of PGO waves and that the frontal lobes act upon the pattern of PGO discharge. Recently the cerebellum has been proposed as being a very important relay relation in the control of rapid eye movements[6]. On the other hand, the output of the cerebellum has mainly an inhibitory character[5]. It was already proposed that the Purkinje cell discharges bore some relationship to eye movements[7]. The increase in amplitude of PGO waves clearly demonstrated here indicates that the cerebellum has an inhibitory action upon the phasic events of paradoxical sleep. This effect could be exerted through a cerebellum-locus coeruleus pathway recently discorvered[8], and the influence that this latter structure has upon paradoxical sleep[9] is well known. On the other hand, this inhibitory cerebellar influence could be exerted through anatomically demonstrated direct connections from the cerebellum with oculomotor neurons[10] or through the connections with reticular formation structures[11]. That the frontal lobes have an important influence upon the oculomotor apparatus is well known[4]. This action could also be exerted through their connections with the reticular formation[11]. Our results confirm the findings of other authors[12] that show an increase in the density of oculomotor activity of paradoxical sleep episodes and other oculomotor alterations[13] after frontal lobe lesions. Moreover we demonstrated that the frontal lobes control the pattern of PGO discharge. These early changes could be more easily explained through the connections of the frontal lobes with the reticular formation and the oculomotor neurons[4,11] than through retrograde degeneration of the pathway that project from the locus coeruleus to the frontal lobes[14]. Therefore it is concluded from our studies that the oculomotor neurons of the brain stem and/or the reticular formation surrounding them are, during paradoxical sleep, under the influence of several structures. During this phase of sleep-waking cycle, one of them, the cerebellum, controls mainly the amplitude of phasic PGO activity, while the other one (frontal lobes) controls mainly the pattern of their sequential discharges. There is conclusive anatomical data to support our functional findings.

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Weak Neuronal Accumulation of Octopamine in Dopaminergic Neurons of the Rabbit Retina

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Summary. Dopaminergic retinal neurons become weakly radioactive after the injection of tritiated octopamine into the vitreous, but no other neurons do so. This indicates the absence of any effective mechanism for the accumulation of octopamine in rabbit retina.

Releasable octopamine has been detected in high amounts in crustacean peripheral nerves[1,2], and the amounts present seemed to correlate with the number of perikarya present in nerve. The substance was suggested to have a modulating action on muscle contractions. It is also present in high amounts in certain other invertebrate neurons[3–5]. In insects, an adenylate cyclase system was described, which was more sensitive to octopamine than dopamine or 5-hydroxytryptamine[6]. In mammals, octopamine is accumulated by sympathetic nerves, from which it can be released by nerve stimulation, and it has been suggested as a kind of neurotransmitter[7,8]. It is also present in the rat brain, although in much lower concentrations than, for instance, the catecholamines[8,9].

Catecholamines are well known as CNS transmitters, and in the retina dopamine is the dominating one (see EHINGER[10]). The dopaminergic neurons as well as the ones presumably operating with glycine or GABA have an efficient mechanism for accumulating their transmitter and it seems that this ability is of importance for terminating the action of released transmitter. Octopamine is structurally related to dopamine, and it was therefore of interest to test to what extent it would be selectively accumulated by retinal neurons.