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STUDIORUM PROGRESSUS

The Relationship of Lateral Geniculate Activity to the Electrocorticogram in the Presence or Absence of the Optic Tract Input[1]

By G. B. ARDEN and U. SÖDERBERG[2]

A series of similar light-stimuli to the eye produce variable responses in the lateral geniculate body[3], and the same is true of the medial geniculate[4]. It has been claimed, from neuroanatomical evidence, that almost all the nerve fibres afferent to the lateral geniculate originate in the retina[5]. Therefore a study of the activity of the lateral geniculate body, in relationship to the activity of other parts of the brain, should yield information about the effects of the retinal centrifugal system described by several authors[6].

We have tried to test this hypothesis by recording the activity of the lateral geniculate body before, during and after increasing the intra-ocular pressure (IOP) to the point where the eye became ischemic: this causes a reversible block of all retinal activity[7].

Experimental arrangements. Rabbits, anesthetized with ether, have been spinalized at C_1 and allowed to recover (encéphale isolé). One eye was eviscerated and the optic nerve head locally anesthetized with Xylocaine (Lidocaine). The other eye was stimulated with a flickering white light (Col. temp. 2760° K, intensity approximately 100000 lux). No precautions were taken to dark-adapt the animal.

The electrocorticogram (EEG) was recorded from Ag-AgCl electrodes placed on the dura. Lateral geniculate spikes were recorded by a glass micropipette filled with 4 *M* NaCl, tip resistance above 5 *MΩ*. Techniques for such recording and for identification of lateral geniculate cells are described by ARDEN and LIU[3]. Retinal activity was abolished by raising the IOP. This was achieved by connecting the eye to a constant pressure bottle via a small needle inserted into the anterior chamber. A second larger needle was inserted for IOP measurement.

We recorded on an inkwriter (Offner) and a cathode ray oscilloscope running synchronously. The spike discharges were integrated by an EKCO rate meter. Intraocular pressure (IOP) and arterial blood pressure (BP) were measured with strain gauge manometers (ELEMA).

The resting discharge and eye intact. Many cells discharged in bursts. The changes in average frequency in some cells were well correlated with changes in the EEG, the activity being higher when the animal was 'aroused'. Whistling and other noises also increased the activity, but there was no direct response to sound, the magnitude of whose effect was correlated with the effect produced in the EEG (Fig. 1 *A*). Sounds had little effect on the firing rate of the cell if the EEG already contained much high frequency activity. The animals were very sensitive to noise and the correlation described above only became clear if the loudspeaker which monitored the spikes was disconnected.

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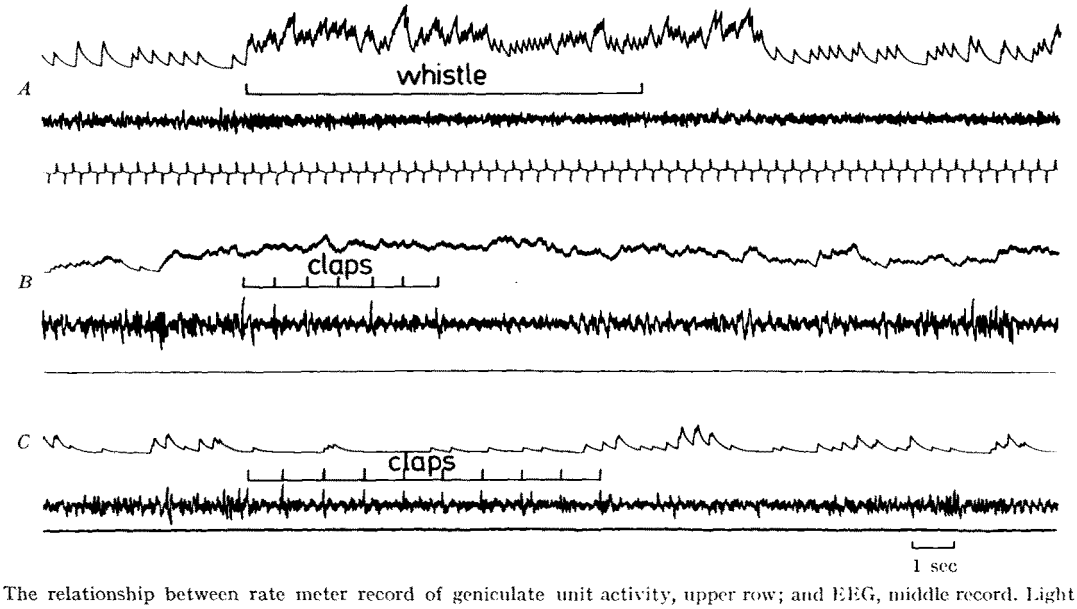

Fig. 1. – The relationship between rate meter record of geniculate unit activity, upper row; and EEG, middle record. Light and sound signals marked. *A*. IOP normal, light on. Soft whistle causes EEG activation and increases the discharge rate. *B*. IOP normal, no light. Handclaps arouse EEG and increase spike rate. *Note* evoked potentials in EEG at each clap. *C*. IOP raised, no light, no light reaction. Handclaps arouse EEG, but cause decrease in discharge rate.

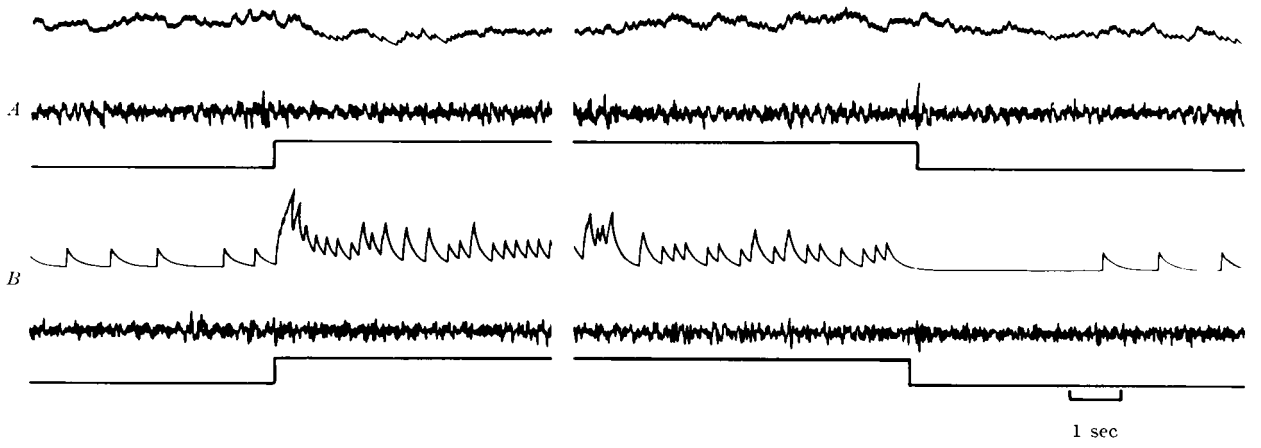

Fig. 2. – Records as in Fig. 1, from same rabbit. Both *A* and *B* show beginning and end of response to a flickering light-stimulus (as in Fig. 1*A*). *A*. IOP normal – slight decrease in firing-rate at light on. *B*. Recovery following period of ocular ischemia – light now causes great increase in firing.

Light and the intact eye. The cells responded to flickering light by burst discharges similar to those seen in the resting discharge, but now phased with the flickering light. At the slow rate of flicker employed, there was often little difference between the overall rate of discharge in flickering light and in darkness (Fig. 2*A*). At the beginning, and more commonly at the end of the flicker, transient changes in the average rate of discharge could occur. These were on the whole smaller than the 'spontaneous' changes in average rate. The average rate of discharge during a flicker was correlated with changes in the EEG, and with noise, in just the same way as was the resting discharge (Fig. 1*B*). Sometimes noise increased the firing rate during a flicker, but not when the animal was in the dark.

In the animals studies, light had little effect in 'arousing' the EEG. Therefore, we cannot assess whether a light-stimulus that produces 'arousal' in the EEG, has the same effect on the response to light in the lateral geniculate body as one that does not.

Resting discharge, optic nerve blocked. When the IOP was increased above BP, after 3–5 min the response to light in the geniculate cell vanished. Before this occurred complex changes in the spontaneous discharge and evoked responses could occur. However, when the optic nerve was completely blocked the resting discharge remained, though its rate might be slightly decreased. In those cells whose activity was previously correlated to the EEG, correlation was still found, but was sometimes entirely reversed, so that now periods of fast activity in the EEG correlated with periods of low firing rate in the lateral geniculate cell, and periods of slow wave activity were associated with high firing rates. Similarly, noises such as whistling or clapping might cause an abrupt decrease in firing rate, associated with the appearance of fast activity in the EEG (Fig. 1*C*). There were cells in which the correlation with the EEG only became apparent after blocking the input from the eye.

Recovery. When the IOP was returned to normal, the response to light rapidly returned. However, there was now a large increase in the average rate of firing during a period of flicker (Fig. 2*B*); when full recovery had occurred, the initial conditions returned, and the average rate of firing was again, in some cells, almost the same in darkness and during flicker.

During the recovery, the relationship between EEG and spike discharge rate could reverse from that obtained during ischemia of the eye, to the original picture described above.

Nociceptive stimuli such as pinching the nose, were effective in 'arousing' the EEG. They usually caused a profound decrease in the firing rate. However, since the spike size was never constant, after nociceptive stimulation, it is not possible to exclude artefacts – movements or volume changes in the brain due to vasomotor changes[8].

Discussion. There are great variations in the behaviour of the different lateral geniculate cells we have studied. We wish to emphasize the *changes* that occur in response to arousing stimuli when the optic nerve is blocked. Since the average firing rate of the geniculate resting discharge is but little affected by the complete removal of the retinal afferents, and can be more affected by alterations in other parts of the brain than by alterations in retinal activity, there must be another afferent pathway to the lateral geniculate, at least as important as the optic tract. The alteration in relationship between EEG and geniculate discharge which we observe on raising the IOP may be due to the removal of a loop involving centrifugal retinal fibres. The alternative possibility remains of interneuronal interactions between the optic tract and the 'second input' at a level prior to the cell from which we record. There is some evidence that the lateral geniculate contains interneuronal paths[9]. The 'second input' may be identical with the fibres of the 'lame blanche intermédiaire'[10].

Zusammenfassung

Am Kaninchen wurde die Beziehung zwischen Elektrokortikogramm und Impulsentladungen der Einzelzellen des lateralen Knickes untersucht und die Befunde bei normaler Netzhauttätigkeit mit denen am entkoppelten Auge verglichen. Die reversible Entkoppelung der Netzhaut wurde mit intraokularer Drucksteigerung hervorgerufen. Die Spitzenpotentiale, die schon im Dunkeln gruppiert auftraten, waren im Flimmerlicht reizabhängig. Die Durchschnittsfrequenz dieser Entladungen erwies sich nur wenig vom verwendeten Lichtreiz beeinflussbar. Elektroenzephalographische Weckreaktionen traten sowohl im Dunkeln, als auch im Licht, mit erhöhter Durchschnittsfrequenz der Zellenpotentiale gleichzeitig auf. Bei entkoppeltem Auge wurde während der Weckreaktion eine Erniedrigung der durchschnittlichen Entladungsfrequenz beobachtet. Aus diesen Befunden wird geschlossen, dass der laterale Knick auch von anderen Gebieten aus, nicht nur von der Netzhaut, beeinflusst wird.

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