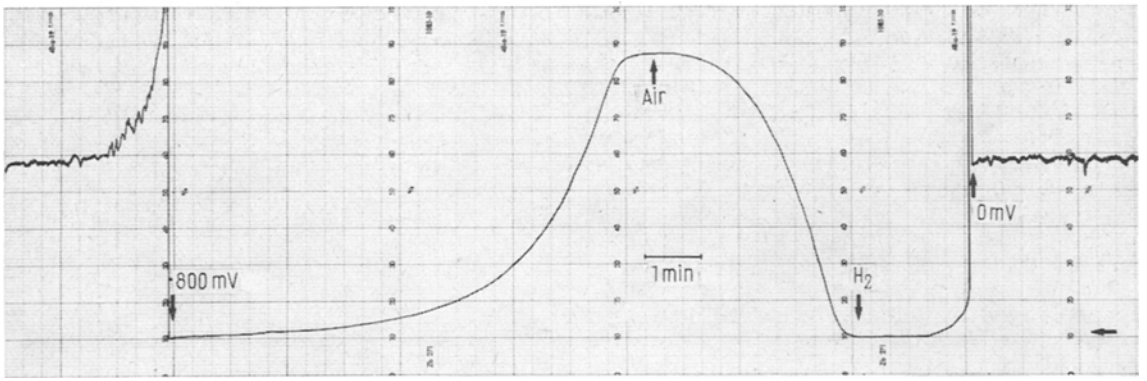

Fig. 3. Combination of Po_2- and H_2-clearance measurement. The change in polarization voltage, as well as in inspiratory gas mixtures, is marked by arrows. At the right- and left-hand corner of the Figure, the Po_2 registration and in the middle of the Figure the hydrogen registration can be seen.

The rats were intubated and breathed spontaneously. The micromanipulator was moved forwards 10 μ per step. At definite parts of the brain tissue, especially at Po_2 maximum and minimum values, polarization voltage was changed from 800 mV negative to 200 mV positive. Then the animals breathed a gas mixture containing hydrogen instead of nitrogen. After saturation of the brain tissue it was switched over to normal air. After that the hydrogen clearance was recorded.

Results and discussion. In this investigation the combined oxygen-hydrogen measurement with a single platinum microelectrode was examined. By means of the O_2- and H_2-polarogram (Figure 1) it can be shown that the current produced by the reduction of O_2 molecules at the platinum surface is zero if a positive polarization voltage of 200 mV is applied. For oxygen measurement a negative polarization voltage of 800 mV was applied. At 800 mV negative the current produced by hydrogen molecules is about 0.1% of that of the O_2 molecules. No interrelationship between oxygen and hydrogen measurements could be seen under these conditions. O_2 profiles as illustrated in the upper trace of Figure 2 could rarely be recorded in our experiments (material from about 25 rats). Typically, the Po_2-differences are smaller. Occasionally, a decrease in the regional oxygen partial pressure within the first 500 μ from the surface of the brain was observed when the platinum needles were moved centrally perpendicular to the brain surface (Figure 2, lower trace). Under these conditions it has to be taken into consideration that the big electrode shafts might diminish regional blood flow, especially that of the venous system which runs mainly on the surface of the brain tissue. Furthermore, FATT[6] observed a decrease of Po_2-recording when mechanical pressure is applied to the tip of the microelectrode. We suppose that the mechanical resistance of the pia caused the decrease of the Po_2-reading. The low Po_2-values agree well with the relatively high half-time values obtained for the fast component of the hydrogen curves.

In Figure 3 a combination of Po_2- and hydrogen-clearance measurements is illustrated. After a change in polarization voltage only a short time is necessary to obtain a constant H_2-value. After saturation of the brain tissue with hydrogen, the inspired gas was changed to air and hydrogen-clearance curve was recorded. The half-time necessary to reach half of the initial values of the hydrogen concentration amounted to about 2.15–4.45 min, the corresponding Po_2-values measures in about twenty Po_2–H_2-clearance combinations were 1–35 mm Hg, in a few cases the oxygen-tension values were greater.

In about 20 combined Po_2- and H_2-clearance measurements, the reproducibility of this kind of experiment was demonstrated (Figure 3). However, only 10% of the platinum needles which were suitable for oxygen measurement could be used for both oxygen and hydrogen measurements. A correlation between Po_2 and local blood flow values could not yet be proved by our results.

Résumé. La pression partielle de l'oxygène et la «clearance» de l'hydrogène ont été mesurées dans des régions circonscrites. Les deux valeurs sont enregistrées par la même microélectrode de platine (diamètre de la pointe 10 μ). Cette méthode nouvelle est appliquée dans des expériences faites sur le cerveau de Rats narcotisés et on est capable d'évaluer les inhomogénités locales de l'apport d'oxygène dépendant du flux du sang.

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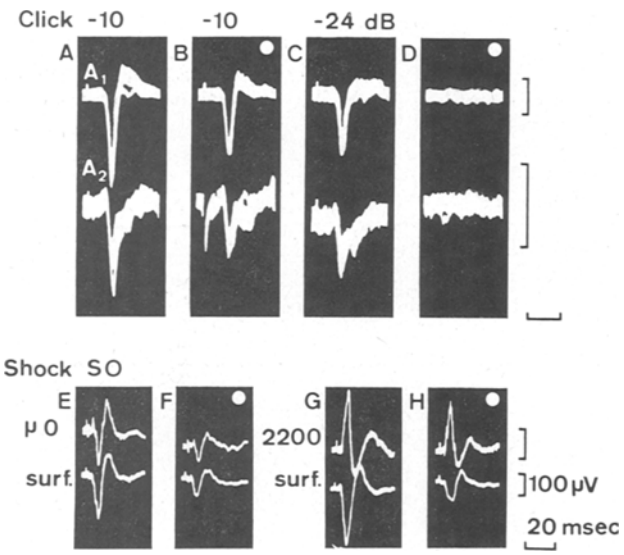
The Responsiveness of Acoustic Area after Stimulation of the Caudate Nucleus

Recent electrophysiological data seem to indicate that peripheral stimulation of various origin may activate the caudate nucleus[1–4]. The hypothesis has been suggested that the caudate nucleus participates in the control of specific and non-specific sensory afferents[5–7]. The present experiments were prompted by the relationship between caudate nucleus and EEG activity[8–11] to demonstrate the control of the sensory input in the neocortex by the caudate nucleus.

Material and method. The experiments have been carried out on 36 cats, curarized, with local anaesthesia of painful points or anaesthetized with pentobarbital or with chronically implanted electrodes. We recorded the evoked potentials in the acoustic area with or without conditioning stimulation of the caudate nucleus, periodically varying the intensity of the click sound energy (expressed in dB below the arbitrary reference level). We used conventional stereotaxis and electrophysiological

techniques (coaxial macroelectrodes and tungsten microelectrodes). Electrode locations were checked in serial paraffin sections through the fixed brain (Prussian blue marks).

Results and discussion. The conditioning stimulation of the head or the body of the caudate nucleus determines an inhibitory phenomenon on the responses evoked either in the primary (A_1) or in the secondary acoustic areas (A_2, A_3). The inhibition, induced by the caudate nucleus, reaches 14–15 dB equivalents for any degree of attenuation, as shown by the unconditioned responses, obtained by more attenuated clicks (Figure, A–C). The conditioning stimulation of the caudate nucleus alone (Figure, D) has never produced any response in the auditory cortex. We evidenced the inhibitory phenomenon on different areas of acoustic cortex employing well-defined stimulation parameters of the caudate nucleus. The optimal parameters of conditioning stimulation resulted as follows: 10 shocks at 200/sec; 1.5 msec; 10 msec interval between conditioning and test stimulus. The inhibitory phenomenon has been greatly diminished even by small changes in these parameters. An equal degree of inhibition in A_1 after conditioning of the caudate nucleus has been observed in barbiturized animals (50 mg/kg i.p.). The study of the potentials obtained with microelectrode at different layers of the cortex (A_1) showed that the inhibition of the caudate nucleus is exercized mostly in the superficial layers (0–1200 μ) and is less evident in the deeper layers (1400–2400 μ) (Figure, E–H). Even in the cat with chronically implanted electrodes, we observed the same effects as seen in the acute animal provided that immobility and somnolence were induced by small doses of pentothal (10 mg/kg i.p.). The most pre-eminent effect was obtained with 4–5 shocks at 150/sec; 1.5 msec; 16–20 msec interval between conditioning and test stimulus.



Curarized cat with local anaesthesia in the painful points. (A–D) Superimposed (7–8) oscillograms of responses evoked simultaneously in the primary acoustic area (A_1) and in the secondary acoustic area (A_2). (A) Responses to a click, attenuated by − 10 db below the arbitrary reference level. (B) The − 10 db clicks are preceded by a conditioning stimulation of the homolateral caudate nucleus (10 shocks at 200/sec; 1.5 msec; 10 msec interval). (C) Unconditioned responses to a click attenuated by − 24 db showing satisfactory degree of inhibition when compared with the conditioned responses. (D) Oscillograms showing only conditioning stimulation. (E and G) Oscillograms of responses evoked in the primary acoustic area (A_1) through electrical shock on the cochlear nucleus; the top response of simultaneous recordings refers to the potential evoked by microelectrode at different depth (0 and 2200 μ); the bottom response refers to the potential evoked by superficial macroelectrode. (F and H) Conditioning stimulation of the homolateral caudate nucleus (10 shock at 200/sec; 1, 5 msec; 10 msec interval) inhibits to a greater degree the superficial potentials and to a lesser degree those evoked in the depth (2200 μ). In the figure negativity is directed upwards.

The results reported show that the high frequency and short duration stimulation of the head and the body of the caudate nucleus produces inhibition of the potentials in the primary (A_1) and the secondary (A_2, A_3) acoustic areas. The inhibitory process appears to depend critically on parameters of conditioning stimulation which must be chosen within a narrow range of values. The inhibition of evoked responses in the acoustic cortex is of 15 db equivalents, calculated with the equivalence in db method, with aural click stimulation. We have not observed any difference in the degree of inhibition between the primary and the secondary acoustic areas. Laminar analysis suggests that the degree of caudato-cortical inhibition is greater on the potentials evoked from the superficial layers and become smaller in the bottom layers. Our results, obtained in barbiturized animals with pharmacological blockage of reticular formation[12], and the bibliographic data about lesions of the same structure[5] seem to indicate that the inhibition induced by the caudate nucleus does not follow pathways via the reticular formation. The experiments on animals with chronically implanted electrodes indicate the possibility of obtaining equal effects in the primary acoustic area after conditioning stimulation of the caudate nucleus, when the animal is in a state of distraction and drowsiness[13].

Riassunto. La stimolazione ad alta frequenza e di breve durata del nucleo caudato determina inibizione dei potenziali evocati nell'area uditiva primaria e secondarie sia in animali preparati acutamente sia in animali portatori di elettrodi a dimora.

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[1] M. BONVALLET, P. DELL and A. HUGELIN, J. Physiol., Paris *44*, 222 (1952).
[2] D. ALBE-FESSARD, E. OSWALDO-CRUZ and C. ROCHA-MIRANDA, Electroenceph. clin. Neurophysiol. *12*, 405 (1960).
[3] C. ROCHA-MIRANDA, Tesi di Medicina, Rio de Janeiro (1961).
[4] V. LA GRUTTA, S. ABBADESSA, G. AMATO, S. AVELLONE, A. F. LA COMMARE and M. A. PADERNI, Archo. Sci. biol. *50*, 269 (1966).
[5] M. DEMETRESCU and M. DEMETRESCU, Electroenceph. clin. Neurophysiol. *14*, 37 (1962).
[6] G. KRAUTHAMER and D. ALBE-FESSARD, J. Neurophysiol. *28*, 100 (1965).
[7] V. LA GRUTTA, S. GIAMMANCO and G. AMATO, Archo. Sci. biol. *53*, 1 (1969).
[8] A. M. LAURSEN, Acta physiol. scand. *59*, suppl. 211, 1 (1963).
[9] F. E. HORVATH, S. SOLTYSIK and N. A. BUCHWALD, Electroenceph. clin. Neurophysiol. *17*, 670 (1964).
[10] V. LA GRUTTA, S. GIAMMANCO and G. AMATO, Archo. Fisiol. *65*, 238 (1967).
[11] V. LA GRUTTA, S. GIAMMANCO and G. AMATO, Archo. Sci. biol. *52*, 64 (1968).
[12] A. ARDUINI and M. G. ARDUINI, J. Pharmac. exp. Ther. *110*, 76 (1954).
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