of 2 Hz frequency and even higher ones resulted in potentiation of late components of the focal discharges (i.e. after-discharges; Figure 1), a transition from the interictal to ictal phase of activity was often seen at stimulation frequencies of 4 to 10 Hz. The ictal activity usually started during stimulation series and continued after cessation of stimulation. It was often restricted to the hemisphere where the PNC was applied and spread to the opposite hemisphere when seizures reappeared. Once started, the ictal activity could be observed recurrently, also without any stimulation. In a seizure it was possible to distinguish 2 phases: the 'tonic' one with rhythmic spike activity, which transgressed into the following 'clonic' one with spike-and-wave or polyspike-and-wave electrocorticographic pattern. Stimulation during this second phase could revoke the first 'tonic' pattern.

Stimulation of the specific relay nuclei (in 2 rabbits) never elicited transition into the ictal phase. Low frequencies of stimulation triggered focal discharges at 1:1 rate, 'cyclical spike driving'[14], was also observed.

Hippocampal stimulation (2 cases) did not trigger individual focal discharges. Transition into ictal activity with higher stimulation frequencies was possible, but repeated stimulations were necessary; the seizure was sometimes found to start only after the stimulation was over.

Our results confirm the conclusions of previous authors[6,7,9] that the non-specific thalamic stimulation cannot trigger individual discharges of the cortical epileptogenic focus. The rhythmic stimulation in our experiments was found to transform the interictal activity into ictal phase. The results of MIHAILOVIĆ[15], with two independent cortical foci synchronized during non-specific thalamic stimulation, are based on the same mechanism – the synchronization of cortical bioelectric activity with a rhythmic stimulation of these thalamic nuclei creates favourable conditions for the spread of epileptic activity[16,17].

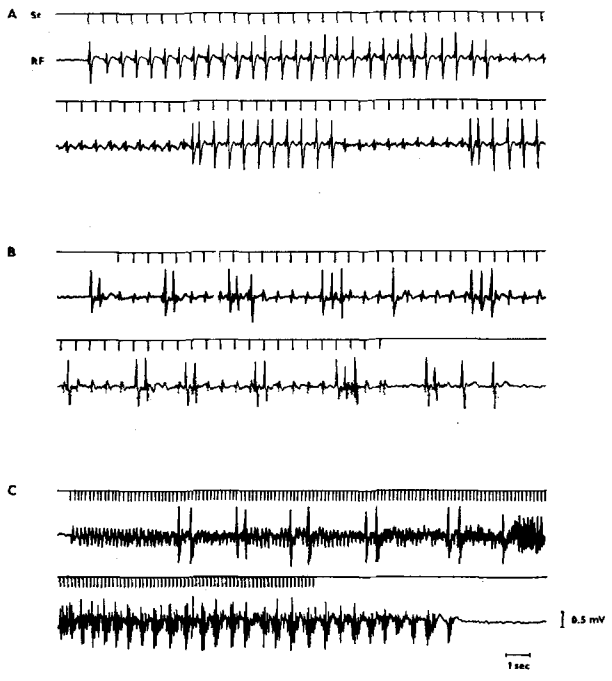


Fig. 2. (A) Stimulation of the right ventral posterolateral thalamic nucleus with frequency of 1.6 Hz; (B) stimulation of the right lateral anterior nucleus with frequency of 1.6 Hz; (C) Stimulation of the right lateral anterior nucleus with frequency of 7 Hz. In all pairs the upper row – stimulation marks, the lower row – activity of the right somatosensory cortical area. Both pairs in each section represent continuous recordings. Time mark 1 sec, amplitude mark 0.5 mV.

[14] D. A. PRINCE, Epilepsia *6*, 226 (1965).
[15] LJ. MIHAILOVIĆ, Experientia *15*, 119 (1959).
[16] M. L. WOODRUFF, F. H. GAGE and R. L. ISAACSON, Electroenceph. clin. Neurophysiol. *35*, 475 (1973).
[17] The authors wish to express their thanks to F. VOŽEH, M. D. for histological control and A. RŮŽIČKOVÁ for excellent technical assistance.

Influence of the Visual Cortex upon Collicular Evoked Responses in the Rabbit

S. MOLOTCHNIKOFF, M. DUBUC* and J. R. BRUNETTE

Département de Sciences biologiques, Université de Montréal, C.P. 6128, Montréal 101 (Québec, Canada), 17 June 1975.

Summary. In rabbit, the depression of the visual cortex by KCl showed an enhancement of the collicular evoked potentials. This enhancement revealed that the visual cortex had exerted a tonic type influence upon the superior colliculus.

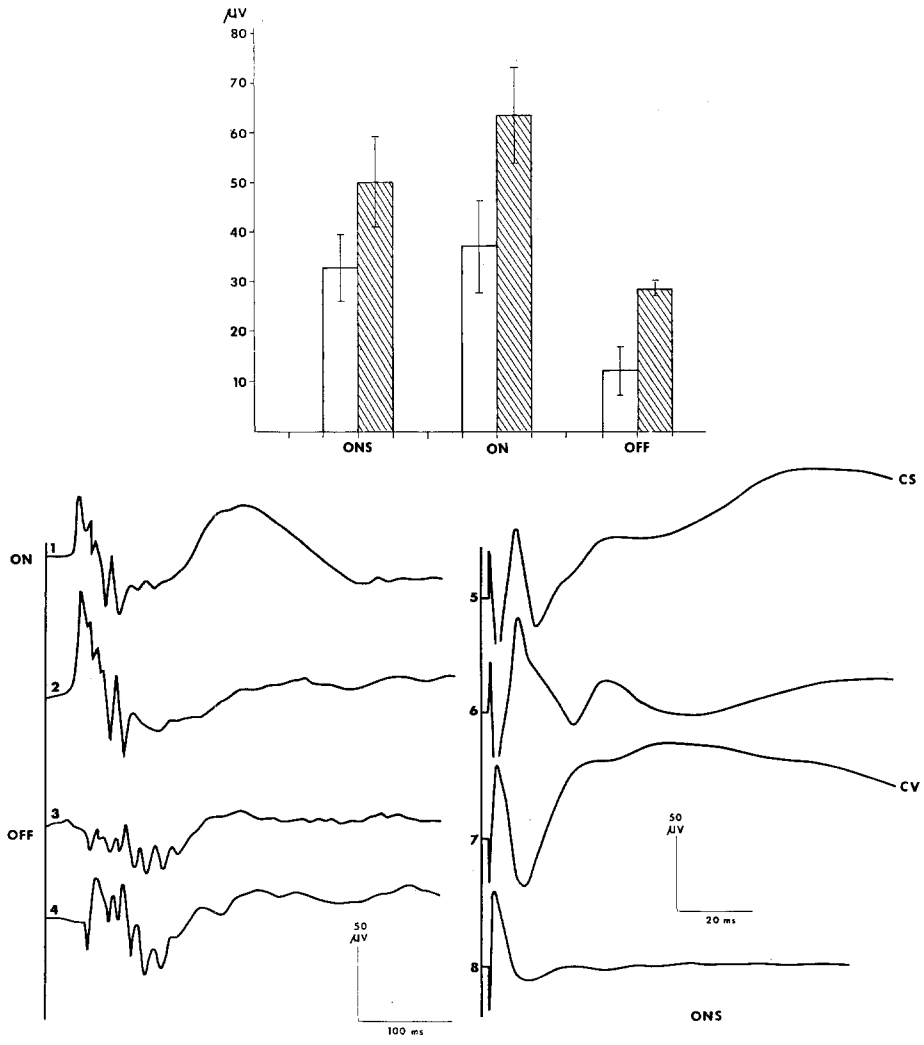
Electrophysiological studies have shown that receptive field properties of the superior colliculus (SC) cells depend to a considerable extent on cortical input in cat[1,2] and monkey[3]. Particularly the visual cortex seems to play a prominent role in controlling the responsiveness of deeper layers of the SC. In contrast to the cat, the ablation of visual cortex in the rabbit does not seem to affect the receptive field characteristics of superior colliculus neurons for either chronic or acute preparations[4,5]. However, the rabbit's SC receives inputs from visual area I and II[6]. Thus it seems that the role of the visual cortex upon the SC in rabbit is not yet established. The present results suggest that the visual cortex may have some tonic effect upon collicular responsiveness as revealed by an increase of evoked responses following a topical application of KCl upon visual cortex as a means for interrupting the cortical functions.

Methods. The studies were conducted on adult rabbits of either sex, anesthetized with pentobarbital sodium (30 mg/kg, i.v.), paralyzed with Gallamine triethiodide and kept under artificial respiration. The responses were evoked by applying, every 2 sec, a 900 msec pulse of light (45 F.C.) which provide a ON stimulus at its onset and a OFF stimulus at its offset. The optic nerve was electrically stimulated (ONS) through a fine platinum curved needle inserted in close proximity to the nerve at its exit

from the eye. Fine electrodes of stainless steel needle (100 μm diam.) insulated down to the tip were lowered stereotaxically toward the SC. The final position of the electrode tip was reached by observing the polarity of the evoked response. In all experiments reported here, the electrode was lowered until evoked response has inverted to a positive phase from surface negative[7–11]. The electrode was then held at a depth where the recorded response had the highest positive amplitude. Histological controls indicated that this depth corresponded to the edge separating the stratum opticum from the stratum griseum superficiale. The functions of the visual cortex were interrupted by applying topically a 3 *M* KCl solution covering area I and II[12]. The responses evoked were amplified with a Grass P511 amplifier (Bandwith 1–300 Hz) and averaged through a computer (Med 80).

Results. The analysis of the collicular evoked responses showed a significant increase in their magnitude following KCl application on ipsilateral cortex (Figure, upper part). The averaged amplitude of responses evoked by optic nerve stimulation (ONS), brightening (ON) and dimming (OFF) stimulations enhanced their magnitude by 52%, 71% and 137% respectively (shaded columns). The measurements were confined to the first positive wave which inverted its polarity. The lower part of this Figure illustrates examples of the responses recorded and the changes which occurred following cortical depression. All evoked responses shown in the Figure, lower part, were obtained following their polarity inversion from surface negative to positive as the electrode was advanced through the superior colliculus, in accordance with previous physiological data[9–11]. The collicular response to the ON stimulus was a complex polyphasic wave, where slow components and fast rythmic oscillations were observed in the initial part of the response (Tracing 1). This early response is followed by a smooth wave, the latency of which was 150 msec. The KCl application on the visual cortex resulted in an

[1] B. G. Wickelgren and P. Sterting, J. Neurophysiol. *32*, 16 (1969).
[2] J. T. McIlwain and H. L. Fields, J. Neurophysiol. *34*, 763 (1971).
[3] P. H. Schiller, M. Stryker, M. Cynader and N. Berman, J. Neurophysiol. *37*, 181 (1974).
[4] D. L. Stewart, D. Birt and L. C. Towns, Vision Res. *13*, 1965 (1973).
[5] R. Masland, K. L. Chow and D. L. Stewart, J. Neurophysiol. *34*, 148 (1971).
[6] R. A. Giolli and M. D. Guthrie, Brain Res. *6*, 388 (1967).
[7] M. Dubuc, M.Sc. Thesis, Montreal, in preparation (1975).
[8] S. Molotchnikoff, M. Dubuc and J. R. Brunette, Can. J. neurol. Sci. *1*, 66 (1975)
[9] M. Tamai and T. Ogawa, Tohoku J. exp. Med. *107*, 127 (1972).
[10] M. Tamai and T. Ogawa, Tohoku J. exp. Med. *109*, 189 (1973).
[11] M. A. Goodale, Expl. Brain Res. *17*, 75 (1973).
[12] J. M. Thompson, C. N. Woolsey and S. A. Talbot, J. Neurophysiol. *13*, 277 (1950).



Upper. Averaged amplitudes of the summated responses to optic nerve stimulation (ONS), light ON and OFF before (white columns) and after (dashed columns) KCl application upon visual cortex. The summation was accomplished over 64 successive presentations. Lower. Examples of evoked responses. Tracings 1, 3, 5, 7, controls; tracings 2, 4, 6, 8, following KCl application. Left hand, responses to light ON and OFF. Right hand, responses to electrical stimulation of the optic nerve (ONS). CS, superior colliculus; CV, visual cortex. Positivity upwards.

increase of the positive going wave without any change in fast rhythmic oscillations and latency, which remained identical: 35 msec. However, the late slow potential was abolished. The OFF stimulus, as expected[8], evoked a complex response the magnitude of which was smaller than that of the ON responses. (Also compare the averaged amplitudes of the summated ON and OFF responses in the upper part of the Figure). Nevertheless, the OFF response consistently presented more rhythmic oscillations than the ON response. This difference in shape cannot be related to a different electrode position since both responses were recorded from the same site and with the same electrode. The effects on collicular OFF responses, after KCl application upon cortical surface, were identical with those produced in ON responses, i.e. a general increase in magnitude of the evoked OFF response, without modification in latency: 35 msec (Tracings 3 and 4, Figure). It can be noticed that the amplitude of the rhythmic oscillations was enhanced but not their number. The KCl blockage of the cortical activity also augmented the amplitude of the early positive going wave evoked by electrical stimulation of the optic nerve (5 × above threshold), but abolished the latest slow wave (Tracings 5 and 6). The tracing 7 shows that the cortical evoked response, which has inverted its polarity from surface positive to negative in depth, is abolished following KCl application (Tracing 8). 30 min after the application of KCl on visual cortex, the cortical activity reappeared and the collicular evoked response decreased in magnitude. They reached approximately their original amplitude about 1 h after KCl application (recorded but not shown).

Discussion. The results reported suggest that a cortical control upon collicular activity exists in rabbit. The responses with the shortest latency and the fast rhythmic oscillations show an enhancement of their amplitude following cortical depression. The latest component was abolished and thus is due to the activity of the cortico-collicular pathway. Comparing our results to those obtained in the cat, an interesting discrepancy emerges. In the cat, visual cortex ablation reduces the collicular evoked responses[13]. Studies carried out on receptive field properties of collicular neurons have shown that the most specific characteristics such as direction and orientation sensitivity, require cortical integrity in cats[1]. In rabbits, the above-mentioned properties are of retinal origine[4]. Consequently, the visual cortex ablation does not produce any loss of receptive field characteristics at collicular level. However, our results suggest that the cortex could exert upon collicular activity a tonic type of action which would modulate the collicular cell responsiveness. Such a tonic action has been already proposed for the cat, where cortical depression instead of enhancing, reduces collicular evoked responses[13].

[13] D. JASSIK-GERSCHENFELD, P. ASCHER and J. A. GUEVARA, Archo ital. Biol. sper. *104*, 30 (1966).

* M. DUBUC was supported by training grant No. 217-617-489 of the Ministère de l'Education du Québec.

Effect of Subdiaphragmatic Vagotomy on Production of Gastric Ulcers in Pylorus-Ligated Albino Rats

A. K. GANGULY and S. K. LAL

Department of Physiology, Jawaharlal Institute of Post-Graduate Medical Education and Research, Pondicherry-6 (India), 12 May 1975.

Summary. A reduction in volume and free and total acidity of gastric content was noted along with reduction in ulcer index, with a shift of the site of ulceration from fundus to the glandular part of stomach, following vagotomy in pylorus-ligated rats. Low volume and acidity explains the absence of ulcers in the fundus, but the increased involvement of glandular part in ulceration is possibly due to weakening of the mucosal barrier following vagotomy.

Our experiments[1,2] indicate that alteration of blood sugar level influences the production of stress-induced gastric, ulcers, a connection which involves the hypothalamus. Hypothalamus influences gastric secretion through vagal and adrenal pathways[3–5]. That the vagal and adrenal pathways are both important in producing stress-induced gastric ulcers is evident from our observation that, even after bilateral adrenalectomy restraint ulcers did develop, although with a low ulcer index in albino rats[6]. The present experiment was planned in order to study the effect of complete, subdiaphragmatic gastric vagotomy on production of gastric ulcers in pylorus-ligated rats.

Materials and methods. 16 albino rats, obtained from the central animal house, of either sex, weighing between 130 and 176 g, housed in separate cages, were divided into 2 groups of 7 and 9.

In the first group, pylorus ligation was done, and in the second, sub-diaphragmatic vagotomy was followed by pylorus ligation. The completeness of vagotomy was confirmed by electric stimulation test[7]. Oesophageal end of the stomach was occluded by introducing a polythene tube, closed and slightly dilated at its terminal end through the oesophagus. A ring electrode was positioned around the distended lower oesophagus and square wave pulses of 10 V, 5 msec duration and 10/sec frequency for 30 sec, used in order to stimulate vagal fibres, if any were left after vagotomy. Completeness of vagotomy was confirmed by absence of noticeable contractions of gastric wall, which is the basis for gastric pressure changes.

[1] A. K. GANGULY, S. K. LAL, S. D. NISHITH and S. K. SREEPATHI RAO, Indian J. med. Res. *57*, 295 (1969).

[2] A. K. GANGULY and S. K. LAL, Indian J. Physiol. Pharmac. *14*, 121 (1970).

[3] J. D. FRENCH, R. L. LONGMIRE, R. W. PORTER and H. J. MOVIUS, Surgery *34*, 621 (1953).

[4] R. W. PORTER, A. J. MOVIUS and J. D. FRENCH, Surgery *33*, 875 (1953).

[5] H. SHAY, D. C. H. SUN, B. DLIN and E. WEISS, J. appl. Physiol. *12*, 461 (1958).

[6] A. K. GANGULY and O. P. BHATNAGAR, Can. J. Physiol. Pharmac. *51*, 748 (1973).

[7] A. K. GANGULY and S. K. LAL, Indian J. Physiol. Pharmac. *15*, 151 (1971).