The technique used by NORBERG and SJÖQUIST for the identification of intraganglionic adrenergic terminals is identical with that described by FALCK[6]. This technique involves the use of frozen-dried specimens, subjected to formaldehyde vapours, resulting in a brightly fluorescent condensation product of norepinephrine (6,7-dihydroxy-3,4-dihydro-iso-quinoline). Recently we reinvestigated the norepinephrine-induced fluorescence of autonomic ganglia using cryostat sections instead of frozen-dried specimens. This modification[7] renders it possible to study large tissue sections instead of the small blocks preferable for freezing-drying.

With regards to the stellate, coeliac and inferior mesenteric ganglia, our results were more or less identical with those reported by the Swedish group. We have found, however, a fair number of adrenergic terminals also in the cervical superius (Figure 1) that originated from strongly fluorescent cells (Figure 2). At least some of these fibres correspond to axon collaterals (Figure 3). No real chromaffine cells could, however, be found in the cervical superius, though such cells were present in all the 3 other feline ganglia studied.

What is the reason for the apparent differences between our results and those of NORBERG and SJÖQUIST? It is improbable that adrenergic terminals were more completely preserved in cryostat sections than in frozen-dried specimens. A more likely explanation is the peculiar structure of the common cervical vago-sympathetic ganglion. In the cat, the superior sympathetic and the inferior vagal (nodose) ganglion are located in a common connective tissue sheath. When using small tissue blocks for freeze-drying, one is subjected to the pitfall of collecting samples from the nodose part of this joint ganglion. In our large cryostat sections, however, one distinctly sees the green-yellow fluorescence of the sympathetic ganglion and the virtually negative nodose ganglion, where the only fluorescent material is the lipofuscine pigment in the sensory ganglion cells.

The role of catecholamines in the modulation of ganglionic responses has been repeatedly discussed since the early observations of MARRAZZI[8]. ECCLES and LIBET[5] ascribed the catecholamine-induced P-wave to intraganglionic chromaffine cells. The observations reported above suggest that in the cervical superius ganglion, as well as in other sympathetic ganglia, the pericellular adrenergic nerve fibres furnish an adequate structural basis for the release of catecholamines within the ganglion.

Zusammenfassung. Fluoreszenzmikroskopische Untersuchungen beweisen die Anwesenheit adrenergischer Terminale im Ganglion cervicale superius der Katze. Die meisten von ihnen stammen aus Axon-Kollateralen. Eine intraganglionäre Hemmung scheint auch im Ganglion c.s. über adrenergische Nervenendigungen zu laufen.

B. CSILLIK, G. KÂLMÁN and ELIZABETH KNYIHÁR

Department of Anatomy, University Medical School, Szeged (Hungary), 13th January 1967.

[6] B. FALCK, Acta physiol. scand. *56*, Suppl. 197 (1962).
[7] B. CSILLIK and S. D. ERULKAR, J. Pharmac. exp. Ther. *146*, 186 (1964).
[8] A. S. MARRAZZI, Am. J. Physiol. *127*, 738 (1939).

Influence of Side Position on Hippocampal Afterdischarge (HAD) in the Rabbit

Both labyrinthine and optic nystagmus are due to asymmetric excitation of the pathways concerned. The asymmetry of these reflexes finds an expression not only in the way in which they interact with each other[1,2], but also in their influence on other responses of the CNS. Thus in the rabbit, labyrinthine stimuli, producing nystagmus to the left, inhibited only the hippocampal afterdischarge (HAD), following electrical stimulation of the left dorsal hippocampus. The same vestibular stimulus had no effect, or occasionally even produced enhancement of the HAD, when the right hippocampus was excited[3].

Another form of asymmetrical labyrinthine stimulation is provided by side position. Thus when the head of an animal rests on the right side, the right saccule is excited more intensely than the left one[4]. Therefore side position exerts again an asymmetric influence on optic nystagmus[5]. It was thus of interest to determine whether a given side position might produce a different effect on the HAD, evoked from the left or right hippocampus of the rabbit.

Method. Under local anaesthesia, one pair each of bipolar, concentric electrodes were placed into the left and right dorsal hippocampus of a rabbit. Square wave pulses of 2 msec duration were supplied at a frequency of 40/sec from a Tektronix pulse generator, which triggered a constant current source. Stimulation lasted always for 5 sec. The electrical activity of the hippocampus was registered bilaterally on a Schwarzer electroencephalograph. Control records were taken, while the animal was lying in a hammock in prone position. The hammock was then turned slowly into side position and remained there for 10 min before stimulation was started again. During the experiment, both eyes were protected from light.

[1] J. LACHMANN, F. BERGMANN, J. WEINMAN and A. WELNER, Am. J. Physiol. *195*, 267 (1958).
[2] F. BERGMANN and A. COSTIN, Israel J. Med. Sci. *1*, 1366 (1965).
[3] A. COSTIN, F. BERGMANN and M. CHAIMOVITZ, Progress in Brain Research *27*, (1966) (in press).
[4] J. FISCHER and L. E. WOLFSON, *The Inner Ear* (Grune & Stratton, New York 1943), p. 36.
[5] A. COSTIN, F. BERGMANN and M. CHAIMOVITZ, Acta oto-lar. *61*, 323 (1966).

Results. The influence of side position on HAD can be displayed only under special conditions. It has been stressed previously[6] that the duration of HAD depends on the intensity of the electrical stimulation when small currents are being used. However, at a certain intensity the HAD attains its maximal span and cannot be extended by a further increase in current strength (Figure 1). We have found that the inhibitory effect of side position on HAD can be demonstrated most easily by using the minimal intensity (marked A in Figure 1) that produces afterdischarges of maximal length. Under these conditions, the HAD evoked by right hippocampal stimulation was markedly shortened by placing the animal's head on the right side, but left side position had no visible effect (Figure 2, left half). Conversely, enhancement of HAD became manifest when the hippocampus was stimulated with a lower intensity (indicated schematically by line B in Figure 1). Under such circumstances, the afterdischarge in prone position lasted only for a few seconds, but was markedly prolonged by placing the head on the side contralateral to the location of the stimulating electrodes (Figure 2, right half). Other examples of the 'dual' influence of side position on HAD are summarized in the Table.

Remarks. Under special conditions, an asymmetrical influence of the labyrinth on the rabbit's hippocampus can be demonstrated in spite of the fact that unilateral stimulation of this structure always produces bilateral discharges of equal length[6]. Apparently, 2 arrangements determine hippocampal activity: (a) Each side has unilateral connections to other brain centres; (b) both sides are linked by commissural connections. Therefore, even when only one hippocampus is stimulated, the two sides respond as a functional unit. This type of organisation is frequently encountered in the central nervous system. The question arises whether an experimental situation can be found in which the discharges of left and right hippocampus will be dissociated[7].

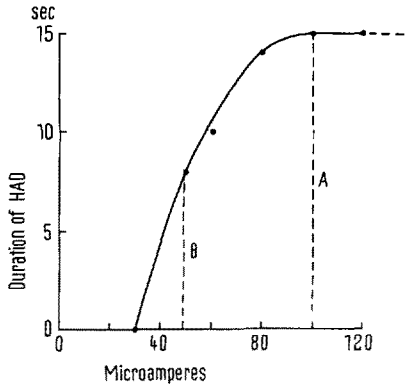

Fig. 1. Duration of HAD as function of intensity of stimulation. Note that beyond the intensity marked (A) no further prolongation of the period of afterdischarges can be obtained. (B) indicates the response to a submaximal stimulus.

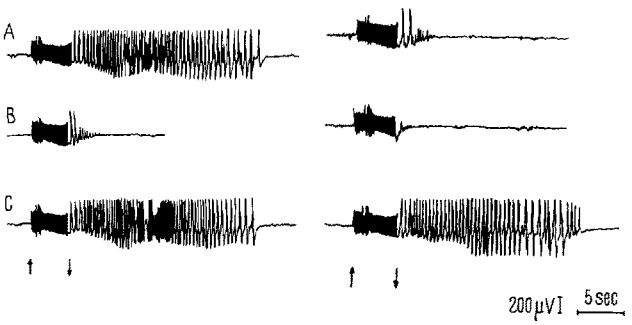

Fig. 2. Effect of side position on HAD. Male rabbit, 2 kg. Stimulation of left dorsal hippocampus at 40 pulses/sec, 2 msec, during 5 sec (between arrows). Time scale and voltage calibration in right lower corner. Left half: side position inhibits HAD, following stimulation of ipsilateral hippocampus with 0.52 ma. (A) prone position; HAD lasting 27 sec. (B) head resting on left side; HAD stops already after 2 sec. (C) head placed on right side; HAD now extends again over 27 sec, which is the maximal duration. Right half: facilitation of HAD, evoked by stimulation of contralateral hippocampus with 0.45 ma. (A) prone position; HAD lasts 4 sec. Note the brief duration of the afterdischarges, as compared with the experiment in left half. (B) head resting on the left side; HAD stops after 1 sec. (C) right side position; HAD extends over 24 sec, a period close to the maximal duration of the HAD on the left hand graph.

Influence of side position on the duration of hippocampal afterdischarges

Experiment No.	Side of hippocampal stimulation	Current strength (mA)	Duration of HAD (sec)		
			Prone	Right side	Left side
1	Left	0.45	4	24	1
		0.52	27	27	2
2	Left	0.18	4	13	0
		0.23	55	55	18
3	Left	0.45	3	55	3
		0.11	65	65	21
4	Right	0.2	5	3	13
		0.4	65	15	65

Résumé. Chez le lapin, la postdécharge hippocampique, provoquée par la stimulation maximale unilatérale de l'hippocampe, est inhibée par l'excitation du saccule ipsilatéral, mais elle est augmentée par l'excitation sacculaire contralatérale si l'hippocampe est stimulé avec une intensité submaximale.

A. Costin, F. Bergmann and M. Chaimovitz

Department of Pharmacology, The Hebrew University-Hadassah Medical School, Jerusalem (Israel), 16th December 1966.

[6] J. Gutman, A. Costin and F. Bergmann, Electroenceph. clin. Neurophysiol. *15*, 989 (1963).

[7] The authors wish to thank R. Knafo for skilful preparation of the Figures.