

peared in many cells in the basal layer. These cells were provided with delicate dendrite-like processes running between the epithelial cells. Their shape and distribution strongly suggested that they were melanocytes. It is at present not possible to identify the compound(s) responsible for this reaction.

In a thin sub-epidermal layer the autofluorescence was weak and confined to fine smooth fibre structures. No varicose fibres were detected here although the conditions for observing their possible presence seemed favourable. Nor were any nerves observed in the non-fluorescent spaces between the connective tissue fibres in the immediate vicinity of or within hair follicles, eccrine and apocrine sweat glands, or sebaceous glands.

In the skin of cat and rabbit a nerve supply similar to that in human skin was found in arterial vessels and in the arrectores pilorum muscles. Although there seems to be no doubt that the fluorescence reaction demonstrates noradrenaline in adrenergic nerves<sup>10,13</sup>, animals that were reserpinized (5 mg/kg subcutaneously) 24 h previous to the biopsy were included in the investigation. No fluorescent nerve fibres were seen in the skin specimens from these animals. Nor did any specific fluorescence develop in skin of the hind leg obtained from cats 3 days after lumbar sympathectomy<sup>14</sup>.

There thus seem to be good reasons for believing that the epidermis, hair follicles and sebaceous glands are not supplied with adrenergic nerves. It seems improbable that this can be the case with the sweat glands. In the coiled portion of these there was no autofluorescence (except in

small granules in the secretory epithelial cells) and no nerves could be seen. On the other hand, fluorescent vascular nerves in the neighbourhood of the glands could be well observed in spite of the autofluorescent connective tissue fibres.

The results obtained in this study show a more limited distribution of the adrenergic nerves in the skin than most previous studies have suggested<sup>4,5,6,9,15,16</sup>.

**Zusammenfassung.** Adrenerge Nerven in der Haut werden mit einer spezifischen und sensiblen histochemischen Methode für gewisse Monoamine nachgewiesen.

Adrenerge Nerven wurden in den Mm. arrectores der Haare und arteriellen Gefässen, nicht aber in der Epidermis, den Haarfollikeln, Talgdrüsen oder Schweißdrüsen gefunden.

B. FALCK and H. RORSMAN

*Department of Histology and Department of Dermatology, University of Lund (Sweden), December 27, 1962.*

<sup>13</sup> A. CARLSSON, B. FALCK, and N.-Å. HILLARP, *Acta physiol. scand.* Suppl. 196 (1962).

<sup>14</sup> B. FALCK and H. MÖLLER, unpublished experiments.

<sup>15</sup> H. J. HURLEY and W. B. SHELLEY, *The Human Apocrine Sweat Gland in Health and Disease* (Charles Thomas, 1960).

<sup>16</sup> This investigation has been supported by a grant from the Alfred Österlund Foundation.

## Cerebral Projection of Group I Afferents in Fore-Limb Muscle Nerves of Cat

Stimulation of group I afferents from stretch receptors in hind-limb muscles does not evoke any surface potentials at the cerebral cortex<sup>1</sup>. On the other hand, some years ago AMASSIAN and BERLIN<sup>2</sup> reported that group I afferents in fore-limb nerves evoke positive potentials in the contralateral somatic area I after a latency of 6–8 msec.

We have now analysed the responses in the somatic receiving areas I and II (SI and SII) that are evoked by stimulation of muscle nerves in the fore-limbs of cats under pentobarbitone anaesthesia (Figure). Monophasic recording from the severed C8 dorsal root at the end of the experiment showed that the group I volley became maximal at 1.5 times threshold and that the group II volley appeared at 1.6 times threshold. In record A, a cortical potential was evoked at a strength eliciting a barely visible ingoing volley. The amplitude of the surface positive potential grew to a maximum with the group I volley (A–D). In some experiments additional activation of group II afferents increased the amplitude of the positive potential, and in the Figure there was an increase of the following negative potential (D and E). The latency of the surface potential was 4.3 to 5.1 msec in the various experiments, when measured to the initial positive deflection signalling the incoming volley (arrow in Figure, C). Similar observations were made on stimulation of the nerve to the long head of the triceps, the biceps, and other muscles.

The potential evoked by stimulation of group I afferents remained after lesions in the lateral and ventral

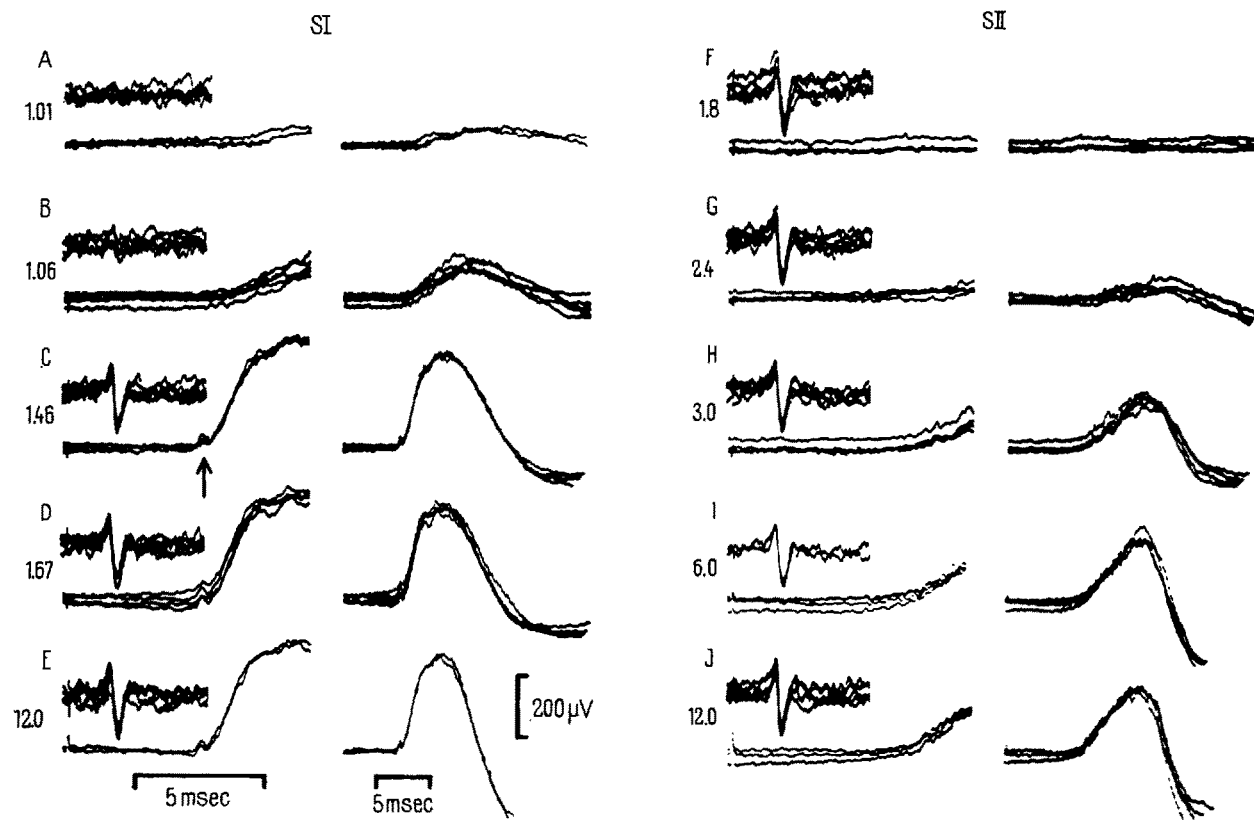
funiculi, but disappeared after lesions in the dorsal funiculi. Fibres in the region of the medial lemniscus were activated from contralateral group I afferents after a delay (the total latency being about 3.0 msec) indicating monosynaptic excitation, as disclosed by tracking with a needle electrode in the upper third of the medulla oblongata. It is concluded that the cortical potential evoked by group I afferents is mediated by the dorsal funiculus–medial lemniscus system.

Evoked potentials appeared in SII only when the stimulus strength had been raised to activate afferents of higher threshold than those belonging to group I (Figure, F–J).

The present experiments indicate that group I muscle afferents in fore-limb nerves project to the SI, but not the SII area of the cerebral cortex. The short latency of the volley reaching the cortex suggests mediation through a disynaptic pathway which is in agreement with the disclosure that the dorsal funiculus–medial lemniscus system is the afferent pathway. Very little summation was needed as indicated by the appearance of the cortical potential very close to the nerve threshold. Hence there is a very efficient synaptic linkage both at the cuneate

<sup>1</sup> V. B. MOUNTCASTLE, M. R. COVIAN, and C. R. HARRISON, *Res. Publ. Ass. nerv. ment. Dis.* 30, 339 (1952). – A. K. MCINTYRE, *Proc. Univ. Otago med. Sch.* 31, 5 (1953). – A. K. MCINTYRE, *Symposium on Muscle Receptors* (D. BARKER, Ed., University Press, Hong Kong 1962), p. 19.

<sup>2</sup> V. E. AMASSIAN and L. BERLIN, *J. Physiol. (Lond.)* 143, 61 P (1958).



Evoked potentials recorded from the somatosensory area I (SI) and II (SII) on stimulation of the contralateral deep radial nerve. The potentials were simultaneously recorded on a fast (left traces) and slow time base. The inset records (upper left traces) show, on the fast time base, the primary afferent volley triphasically recorded from the dorsal funiculus at the C3 level immediately after the cortical recording. Arrow in C indicates initial positivity signalling incoming volley. Stimulus strength in multiples of nerve threshold is indicated on each set of records. Positivity is signalled upwards. Voltage scale applies to cortical potentials (right traces). Superposed sweeps.

and thalamic relay comparable to that found at the group I relays to the cerebellar tracts in Clarke's column<sup>3</sup> and the external cuneate nucleus<sup>4</sup>. The finding that the whole threshold range of group I afferents, including afferents of the lowest threshold, contributed to the evoked potential suggests that afferents from muscle stretch receptors are responsible.

**Zusammenfassung.** Bei Reizung der Gruppe I afferenter Fasern der Muskelnerven des Vorderbeins erhält man positive Potentiale mit kurzer Latenz in der somatischen Area I der Grosshirnrinde. Die somatische Area II bleibt

reaktionslos. Die Leitung läuft über das System, Funiculus dorsalis–Lemniscus medialis.

O. OSCARSSON and I. ROSÉN

*Institute of Physiology, University of Lund (Sweden),  
December 20, 1962.*

<sup>3</sup> A. K. MCINTYRE, Abstr. XIX Internat. Physiol. Congr. (1953), p. 107. – B. HOLMQVIST, A. LUNDBERG, and O. OSCARSSON, *Acta physiol. scand.* **38**, 76 (1956). – A. K. MCINTYRE and R. F. MARK, *J. Physiol. (Lond.)* **153**, 306 (1960).

<sup>4</sup> B. HOLMQVIST, O. OSCARSSON, and I. ROSÉN, *Acta physiol. scand.*, in press.

### Influence of the Caudate Nucleus on Hippocampal Afterdischarges in the Rabbit

The nucleus caudatus inhibits many cortical and subcortical responses<sup>1,2</sup>. In a few cases, however, caudate stimulation in the cat produced motor effects such as head turning, licking, sniffing, swallowing and other autonomic reflexes<sup>3,4</sup>. Stimulation of this nucleus may also facilitate a response evoked from another part of the brain, such as vestibular nystagmus<sup>5</sup>.

As a new, striking example of such facilitatory action, we describe here the enhancement of hippocampal after-

discharge (HA). This effect is especially clear, when compared with the influence of caudate stimulation on the resting hippocampus (Figure 1). In the latter, caudate

<sup>1</sup> F. A. METTLER, H. W. ADES, E. LIPMAN, and E. A. CULLER, *Arch. Neurol. Psychiat.* **41**, 984 (1939).

<sup>2</sup> F. A. METTLER and C. C. METTLER, *Brain* **65**, 242 (1942).

<sup>3</sup> D. FORMAN and J. W. WARD, *J. Neurophysiol.* **20**, 230 (1957).

<sup>4</sup> N. A. BUCHWALD and F. R. ERWIN, *Electroenceph. clin. Neurophysiol.* **9**, 477 (1957).

<sup>5</sup> A. SCHEIBEL, C. MARKHAM, and R. KOEGLER, *Neurology* **11**, 1055 (1961).