haut erst nach Versuchen mit Lichtreizen weitere Aussagen möglich. Immerhin ist schon jetzt zu vermuten, dass die verzögerte Leitungsgeschwindigkeit in der Netzhaut – wenigstens beim bewegten Auge – als ein zusätzlicher Mechanismus zu einer Umformung von Zeit- in Raumkoordinaten benutzt wird.

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Summary

The conduction velocity of the unmyelinated portion of the optic nerve fibres within the cat's retina was measured by shifting a microelectrode from the blind spot outwards and stimulating antidromically in the lateral geniculate body. The mean conduction velocities were 2·8 m/s for the fast, and 1·7 m/s for the slow group of optic nerve fibres, as compared with maxima of 70 and 23 m/s respectively for the same groups in the myelinated extrabulbar portion.

Crossed Fastigial Atonia[1]

The present experiments were concerned with the influence of localized lesions of the *Nucleus fastigii* on the extensor rigidity of the decerebrate cat. The transection of the brain stem was made just frontally to the anterior *Colliculi* («precollicular» cats), but preparations decerebrated at ponto-midbrain levels («postcollicular» cats) were also used. The cerebellar lesions were made electrolytically, after decerebration, through electrodes orientated with a Horsley-Clarke stereotaxic apparatus. 60 cats were studied while lying prone in a hammock, with the head held symmetrically in the Horsley-Clarke machine and with the limbs hanging free.

(1) Decerebrate rigidity was increased ipsilaterally and abolished or strongly decreased contralaterally after a lesion confined to the caudal pole of one fastigial nucleus. Serial Nissl and Weil slides showed that the other cerebellar nuclei and all brain stem structures were normal. The abolition of extensor rigidity in the contralateral limbs will be henceforth referred to as "crossed fastigial atonia".

(2) Crossed fastigial atonia is a deficiency symptom, since (*a*) it was observed up to 24 h after the lesion in the decerebrate preparation; (*b*) a marked reduction of postural extensor tonus follows chronic lesion of caudal pole of contralateral fastigial nucleus in the otherwise normal cat (Batini and Pompeiano[2]); and (*c*) electrical stimulation (300/s, 1 ms, 0·3 – 0·5 volts) of fastigial points, whose destruction is followed by crossed atonia, yielded the opposite postural asymmetry (i.e. ipsilateral flexion and crossed extension).

(3) Crossed fastigial atonia was observed after (*a*) postcollicular decerebration; (*b*) anterior lobe topectomy; (*c*) postbrachial transection of spinal cord, i.e. when decerebrate rigidity became very marked following different types of release from tonic inhibition. It was immediately and permanently abolished, and postural asymmetry was replaced by bilateral rigidity, when the caudal pole of the contralateral fastigial nucleus was destroyed. Apparently crossed fastigial facilitation is required for overcoming some inhibitory influence, and the postural imbalance is abolished as soon as symmetrical lesions are performed. Experiments aimed at identifying the inhibitory mechanisms underlying crossed fastigial atonia are in progress.

(4) The reverse postural asymmetry, characterized by ipsilateral disappearence or strong decrease of decerebrate rigidity, with increase of extensor hypertonus in contralateral limbs, occurred whenever the foremost $^1/_2$ or $^2/_3$ of fastigial nucleus was destroyed unilaterally. Since this operation involves unilateral severance of Russell's hook bundle after interfastigial crossing, as well as destruction of direct fastigio-bulbar systems, the origin of ipsilateral fastigial atonia must be left for further investigation. Similar mechanisms may underly Sprague and Chambers' phenomenon[3], which is characterized by ipsilateral flexor hypertonus and marked contralateral extensor rigidity following total destruction of one fastigial nucleus. We have confirmed these observations after total ablation of one fastigial nucleus and we found occasionally that slight flexor hypertonus paralleled disappearence of extensor rigidity even after partial lesions. Active flexion was not observed, however, following postcollicular decerebration and its midbrain mechanisms will be investigated with further experiments. In the present note we are concerned merely with unilateral abolition of extensor rigidity.

(5) Unilateral aspiration, cooling or local nembutalization (6%) of *Pyramis* and *Uvula* (Larsell's lobules[4] VIII and IX) were followed by a postural asymmetry similar in sign, but slighter in degree, to that elicited by a lesion of the underlying caudal pole of the *Nucleus fastigii*. This postural asymmetry was replaced by bilateral rigidity if contralateral folia of lobules VIII and IX were removed.

These physiological observations, combined with old and recent anatomical data (see Jansen[5]), suggest (1) that a tonic facilitatory discharge arises in the caudal part of each fastigial nucleus and is relayed to crossed brain stem mechanisms by Russell's hook bundle and (2) that the caudo-fastigial discharge is at least partially driven by corticonuclear volleys arising in the ipsilateral part of Larsell's lobules[2] VIII and IX.

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Riassunto

La distruzione unilaterale del polo caudale del nucleo del tetto del cervelletto nel gatto decerebrato è seguita da scomparsa o intensa riduzione della rigidità estensoria negli arti contralaterali. Diversi esperimenti suggeriscono che il fascio uncinato di Russell trasmette un'influenza tonica facilitatrice esercitata da neuroni fastigiali sui meccanismi estensori del lato opposto.

[1] This work was supported by a grant of the Consiglio Nazionale delle Ricerche.

[2] C. Batini and O. Pompeiano, Boll. Soc. it. Biol. sper. (in press).

[3] J. M. Sprague and W. W. Chambers, J. Neurophysiol. *16*, 451 (1953).

[4] O. Larsell, J. comp. Neurol. *99*, 135 (1953).

[5] J. Jansen, *Efferent cerebellar connections*, in: J. Jansen and A. Brodal, *Aspects of cerebellar anatomy* (J. Grundt Tanum, Oslo, 1954).