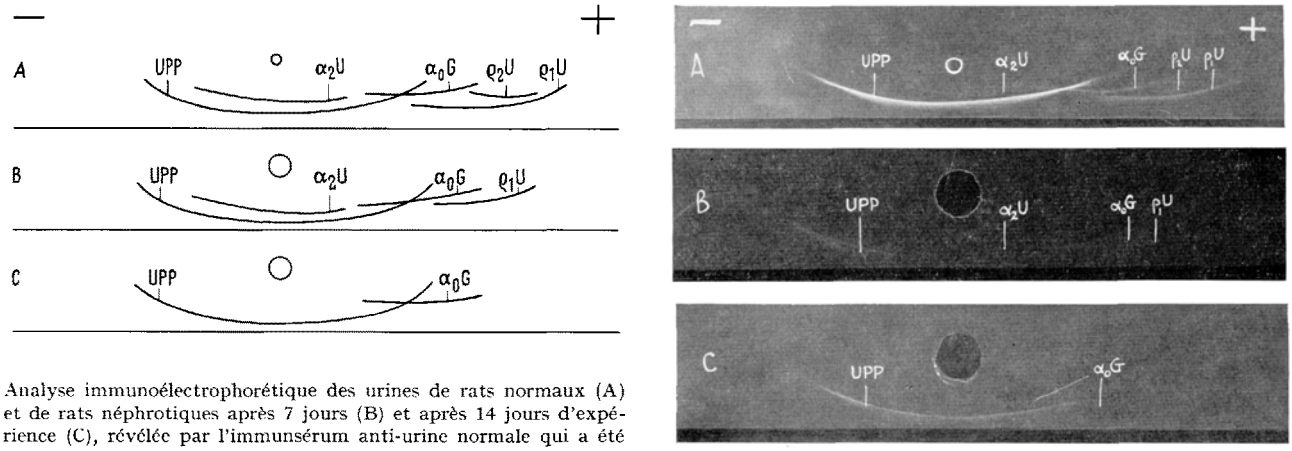

Analyse immunoélectrophorétique des urines de rats normaux (A) et de rats néphrotiques après 7 jours (B) et après 14 jours d'expérience (C), révélée par l'immunsérum anti-urine normale qui a été préalablement absorbé par le sérum normal de rat. ϱ_1 U, ϱ_1-protéine urinaire; ϱ_2 U, ϱ_2-protéine urinaire; α_0 G, α_0-glycoprotéine urinaire; α_2 U, α_2-globuline urinaire; UPP, uro-protéine principale. On remarque particulièrement la nette diminution en nombre et en quantité de protéines urinaires spécifiques dans les urines pathologiques.

bée[17]. Des lésions rénales qui en résultaient, pouvaient être aussi causées par une phosphorylation mitochondriale défectueuse, due à l'action de l'aminonucléoside[18]. La disparition de trois protéines urinaires spécifiques pourrait être expliquée soit par le mécanisme d'inhibition compétitive de la synthèse protéique soit par une modification structurale de la moule ARN (acides ribonucléiques) servant à la synthèse des protéines urinaires spécifiques, ou bien par la défection d'un système enzymatique responsable de la synthèse des protéines urinaires disparues, au cours de la néphrose à la Puromycine.

Il nous paraît assez clair que les protéines urinaires ont suivi parallèlement deux mouvements de sens inverse au cours du développement de la néphrose à la Puromycine chez le rat: d'une part l'augmentation des protéines sériques dans l'urine, et de l'autre, la disparition partielle des protéines urinaires spécifiques. Le premier phénomène présente peu d'intérêt à cause de son caractère commun avec plusieurs syndromes néphrotiques d'étiologies différentes, mais le dernier revêt une importance considérable au point de vue diagnostic si on tient compte de l'origine rénale des protéines urinaires spécifiques. Les modifications de ces dernières, s'effectuant en étroite relation avec les changements morphologiques du tissu rénal, nous informent indirectement sur la nature des lésions causées aux cellules rénales[19].

Summary. The disappearance of three specific urinary proteins has been demonstrated in relation to morphologic changes in renal structures during the development of amino-nucleoside nephrosis in rats.

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[17] B. A. BOROWSKY, D. M. KESSNER, W. S. HARTROFT, L. RECANT et M. B. KOCH, J. lab. clin. Med. *57*, 512 (1961).
[18] P. BARTLETT, J. KEEGAN et H. SCHAEFER, Proc. Soc. exp. Biol. Med. *112*, 96 (1963).
[19] B. L. DINH, A. TREMBLAY, A. BRASSARD et D. DUFOUR, in preparation.
[20] Nous remercions Mademoiselle P. GAGNON pour sa collaboration technique.

Descending Pathways with Monosynaptic Action on Motoneurones

Alpha motoncurones in the cat receive monosynaptic EPSPs not only from Ia afferents but also from axons descending in the ipsilateral ventrolateral funiculi[1]. The present investigation was undertaken in order to disclose the origin of the descending pathway having a monosynaptic connection with motoneurones. In unanaesthetized anaemic decorticate cats, paralysed with Flaxedil, intracellular recordings were performed from about 500 motoneurones, identified by antidromic stimulation of their axons at a peripheral level.

Three different series of experiments were performed. In the first series on acute spinal cats, the dissected ventrolateral funiculi were stimulated in the lower thoracic region. Single shocks, 0.05 msec in duration, produced a descending volley which was conducted at 100 m/sec and evoked monosynaptic EPSPs in both flexor and extensor motoneurones (central latency 0.4–0.5 msec). 83% of the motoneurones received monosynaptic impingement. The average amplitude of the maximal supraspinal monosynaptic EPSP was about 25% of the average amplitude of the maximal monosynaptic Ia EPSP recorded from both extensor (Figure 1, A–D) and flexor (Figure 1, E–H) motoneurones. In one animal with hemisection of the cord

[1] E. EIDE, A. LUNDBERG, and P. VOORHOEVE, Acta physiol. scand. *53*, 185 (1961).

at C3–C4 level preceding the acute experiment by 27 days, these monosynaptic effects were abolished, thus indicating their supraspinal origin.

In the second series of experiments, stereotaxic stimulation of the lateral vestibular nucleus was performed. The electrode was placed in the hindlimb region of the lateral vestibular (Deiters') nucleus by observing the ipsilateral extensor response due to stimulation before curarization[2]. This stimulation produced a descending volley recorded in L7 after a latency indicating a conduction velocity of about 100 m/sec. 82.5% of the extensor motoneurones received a monosynaptic EPSP on stimulation of the ipsilateral Deiters' nucleus. The average amplitude of the maximal supraspinal EPSP corresponded to 21.7% of the average amplitude of the maximal monosynaptic Ia EPSP in the same cells (Figure 2, A–D). By contrast, flexor motoneurones did not receive any monosynaptic EPSP from Deiters' nucleus even at high stimulus intensities (Figure 2, E–H).

Finally, in the third series of experiments, the animals were submitted to stereotaxic lesions in the vestibular nuclei on one side, performed 12–33 days before the acute experiments, which were carried out as in the first series described above. In those animals which had Deiters' nucleus destroyed, a descending volley in the ipsilateral ventrolateral funiculi did evoke a monosynaptic EPSP of normal amplitude in flexor motoneurones, but there was no effect in extensor motoneurones. A comparison of the findings from animals with different extent and location of the chronic vestibular lesion has revealed that the hindlimb region of the Deiters' nucleus must be intact if the descending volley is to produce monosynaptic EPSPs in extensor motoneurones; lesions in the other vestibular nuclei, on the other hand, do not abolish the effect.

In summary, this study shows a monosynaptic excitatory connection from the ipsilateral Deiters' nucleus to extensor but not to flexor motoneurones. There is another monosynaptic pathway to flexor motoneurones of supraspinal origin. This work provides a further criterion for distinguishing between flexor and extensor motoneurones showing that this classification, which so far has been only referred to the segmental organization, is valid also for effects from supraspinal structures.

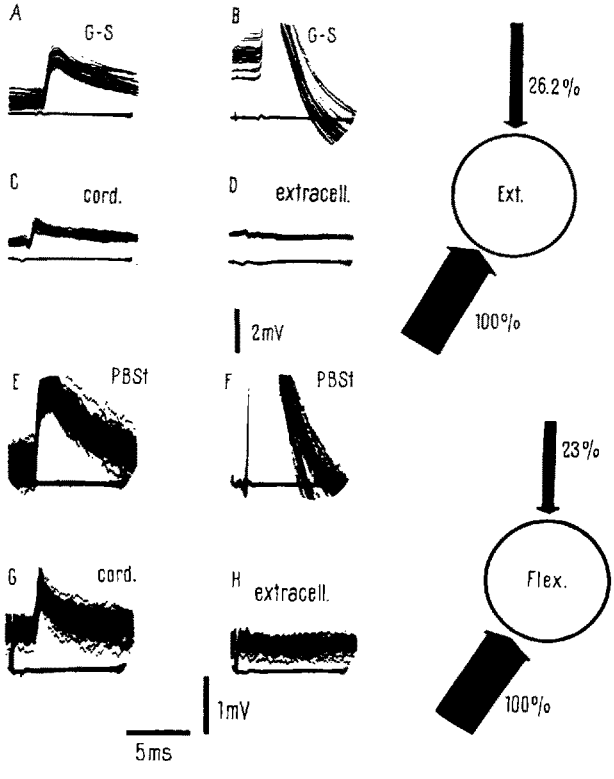

Fig. 1. A–D show effects on a gastrocnemius-soleus (G-S), E–H on a posterior biceps-semitendinosus (PBSt) motoneurone. Upper traces in each record are taken by microelectrode (negativity downwards), A–C, E–G are intra-, D, H are extracellular recordings. Voltage calibration see inset. Lower traces show records from L7 dorsal root entry zone (negativity upwards). A, E show the maximal monosynaptic Ia EPSPs; B, F antidromic spike obtained by slightly higher stimulus intensities applied to the homonymous nerves. C, D, G, H show effects of supramaximal stimulation of the ipsilateral ventrolateral funiculi. See text for explanation of the drawings.

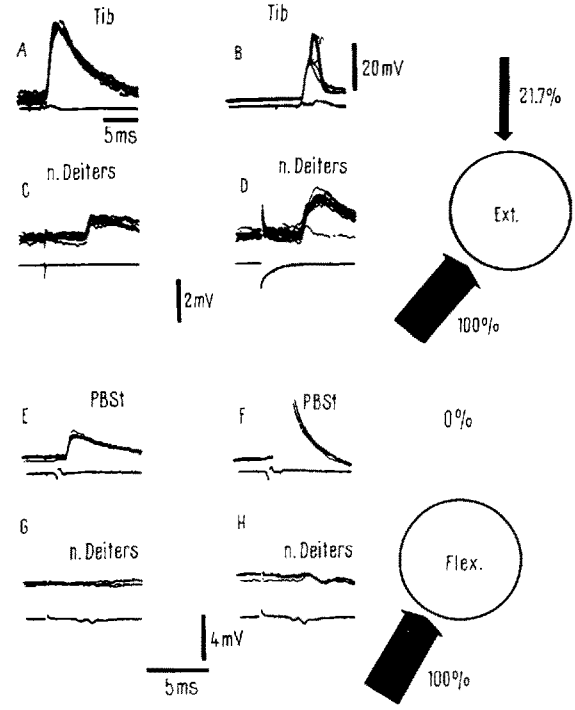

Fig. 2. A–D are from a tibial (Tib), E–H from a posterior biceps-semitendinosus (PBSt) motoneurone. Upper traces in each record are intracellular recordings with microelectrode. Voltage calibration see inset. Lower traces are records from L7 dorsal root entry zone. A, E show the maximal monosynaptic Ia EPSPs; B, F antidromic spike obtained by slightly higher stimulus intensities applied to the homonymous nerves. C, G show effects of stimulation of Deiters' nucleus with low and D, H with high stimulus intensities.

Zusammenfassung. Motoneuronen, sowohl zu Streck- wie Beugemuskeln, werden von absteigenden Bahnen des Rückenmarkes supraspinalen Ursprungs monosynaptisch beeinflusst. Es konnte gezeigt werden, dass die Wirkung an den Motoneuronen der Streckmuskeln aus dem Nucleus vestibularis lateralis (Deiters) stammt.

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Department of Physiology, University of Göteborg (Sweden), June 11, 1965.

[2] A. Brodal, O. Pompeiano, and F. Walberg, *The Vestibular Nuclei and their Connections, Anatomy and Functional Correlations* (Oliver and Boyd, Edinburgh 1962), p. 115.