Résumé

24 rats mâles castrés furent divisés en 4 groupes de 6. Ils furent traités à des doses physiologiques de benzoate d'œstradiol, de progestérone ou d'une combinaison des deux hormones. On constata que l'administration d'œstrogène augmente la concentration de l'hormone thyréotrope dans le sang des rats, tandis que la concentration de l'hormone gonadotrope en est diminuée. D'autre part l'administration de 0,4 mg de progestérone augmente la concentration des hormones gonadotropes et adrénocorticotropes dans le sang des rats. Le progestérone augmente aussi la quantité d'hormone thyréotrope dans l'hypophyse.

Wirkung des Neurotoxins *Shigella Shigae* auf die Krampfbereitschaft

Es wird der Einfluss des Neurotoxins der *Shigella Shigae* auf experimentelle Krampfanfälle geprüft. Während Dysenterieerkrankungen sind bei Kindern in der Klinik des öfteren Krampfanfälle zu beobachten[1].

Laboratoriumsmäuse erhielten intraperitoneal 0,4 ml dieses Neurotoxins auf 20 g Körpergewicht. Die Verdünnung war 1:100. Bei dieser Dosis wurden in 3 Tagen 20% der Versuchstiere getötet. Von den überlebenden Mäusen zeigten nur einzelne Zeichen einer Intoxikation (Diarrhöe, Benommenheit, Paresen der Extremitäten). 72 h später wurde die Krampfbereitschaft geprüft und zwar mit der Methode der Kardiazol-, Elektroschock- und reflektorischen audiogenen Krämpfe.

Krampfart	Anzahl der Versuchstiere	Davon Krämpfe %	Anzahl der Kontrolltiere	Davon Krämpfe %	Statistische Signifikanz *P*
Kardiazol (60 mg/kg intraperitoneal) . .	30	46,6	30	16,6	0,02
Elektroschockkrämpfe	49	83,7	49	53,1	0,001
Audiogene Krämpfe	26	61,5	26	23,1	0,01

Wie die Tabelle zeigt, war die Krampfbereitschaft bei allen verwendeten epileptogenen Reizen erhöht. Diese Erhöhung ist statistisch gesichert.

Dieses Ergebnis bestätigt den in der Klinik gewonnenen Verdacht, dass Shigellatoxin krampffördernd wirkt.

A. HRBEK

II. Kinderklinik der Pädiatrischen Fakultät, Prag, 13. Juni 1957.

Summary

The neurotoxin *Shigella Shigae* increases the readiness for cardiazol, electroshock and reflectory audiogenic cramps.

[1] W. D. DONALD, CH. H. WINKLER und L. M. BARGERON, J. Pediatr. *48*, 323 (1956).

Integrative Pattern of Reflex Actions by Impulses in Large Muscle Spindle Afferents on Motoneurones to Hip Muscles

Motoneurones are influenced by nerve impulses from various kinds of muscle receptors[1]. For example a slight stretch will cause a discharge in the large afferents (Ia fibres) from the muscle spindles. These impulses produce excitatory post-synaptic potentials (EPSP's) in the motoneurones on which they impinge monosynaptically and may thereby give rise to a reflex discharge of impulses.

The connexions which afferent Ia fibres make with different motor nuclei have been studied in detail on muscles operating at the knee and ankle joints by recording reflex discharges[2] and more recently by recording intracellularly the potential change occurring in individual motor nerve cells[3]. The excitatory action by Ia fibres coming from any particular muscle has been found to extend to motoneurones which subserve this muscle and to others which operate synergically at the same joint. One exception to this rule has been observed, namely the ankle extensor, soleus, which can be activated by Ia impulses from the knee extensor vasto-crureus[3].

Impulses in Ia fibres are known to exert inhibitory action on motoneurones of antagonist muscles evoking in them potential changes of opposite sign (so called inhibitory post-synaptic potentials, IPSP's). With knee and ankle muscles these inhibitory effects are strictly limited to the antagonists[4].

The present investigation has been concerned with the synaptic actions of Ia impulses onto motoneurones of hip flexors (iliopsoas and sartorius) and hip extensors (semimembranosus and adductor femoris). In Figure 1 intracellular records are shown from a typical iliopsoas motoneurone. The EPSP contributed by the nerve from iliopsoas itself (*A*) was of about the same size as that produced by an afferent volley from the synergist hip flexor, sartorius (*B*). In records *C–E* IPSP's are shown evoked as expected by afferent volleys from the antagonist hip extensors: adductor femoris (*C*), semimembranosus (*D*), and anterior biceps (*E*). In *F*, however, it is shown that a volley from the knee extensor, vasto-crureus, also contributed a similar IPSP. In other experiments in which it was possible to distinguish between fast and slow components (Ia and Ib) of the afferent group I volley[5], it has been ascertained that the IPSP's evoked by vasto-crureus volleys in hip flexor motoneurones are indeed produced by the Ia type afferents. This inhibitory action therefore represents an exception to the usual pattern of reciprocal innervation, and moreover, the inhibitory action contributed by the knee-extensor was larger than that coming from any single hip-extensor muscle.

Motoneurones of hip extensor muscles were found to receive excitatory effects from Ia afferents of other muscles than their synergists. Figure 2 shows EPSP's produced in a semimembranosus motoneurone by affe-

[1] C. S. SHERRINGTON, *The integrative action of the nervous system* (New Haven and London, 1906).

[2] D. P. C. LLOYD, J. Neurophysiol. *9*, 439 (1946). – Y. LAPORTE and D. P. C. LLOYD, Amer. J. Physiol. *169*, 609 (1952).

[3] J. C. ECCLES, R. M. ECCLES, and A. LUNDBERG, J. Physiol. *137*, 22 (1957).

[4] D. P. C. LLOYD, J. Neurophysiol. *9*, 439 (1946).

[5] K. BRADLEY and J. C. ECCLES, J. Physiol. *122*, 462 (1953). – Y. LAPORTE and P. BESSOU, C. r. Soc. Biol. (in press 1957). – J. C. ECCLES, R. M. ECCLES, and A. LUNDBERG, J. Physiol. *136*, 527 (1957).