Die in Figur 2 dargestellte logarithmische Dosis-Wirkungskurve hat fast einen geradlinigen Verlauf, was für die Spezifität der Wirkung spricht. Die einzelnen Punkte entsprechen der maximalen Start-Minimum-Differenz, wobei die ausgezogenen Linien die Vertraulichkeitsgrenzen repräsentieren, berechnet mit dem t-Test bei $P = 0{,}05$. Die maximalen Start-Minimum-Differenzwerte der einzelnen Dosierungen wurden mit dem Leerwert auf die Signifikanz der Differenz der Mittelwerte mit dem t-Test untersucht. Dabei hat sich erwiesen, dass bei der Dosierung von 0,33 g/kg und 0,62 g/kg der Blutzuckergehaltsabfall noch nicht signifikant ist, dass aber der Abfall der Blutzuckerkonzentration bei einer Dosierung von 5,0 g/kg hoch signifikant ist ($P = 0{,}001$).

Ein gesondert durchgeführtes Experiment mit *Fructus poterii spinosi* und der Dosierung von 0,9 g/kg zeigte keine blutzuckersenkende, sondern eine blutzuckersteigernde Wirkung. Es ist daher anzunehmen, dass das wirksame Prinzip der Droge *Poterium spinosum* in Stamm/Rinde und Wurzel vorliegt, wie aus Figur 1 ersichtlich ist.

Weitere Untersuchungen sind im Gange.

Summary. The decocts prepared from the root and stem of the plant, when administred orally to rabbits, cause a fall in blood sugar level which is highly significant after the dose of 5 g/kg. The log dose/effect curve forms a straight line which confirms the accuracy of the effects observed. Preparations from the fruit were without any effect.

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α-Rigidity in Reserpinized Rats

The function of muscle spindles is said to be changed in Parkinsonian rigidity[1]. Depending upon the technique used, the results have been given exactly opposite interpretations: the one assuming low, the other high fusimotor activity. The methods available in clinical neurophysiology today do not, however, give reliable information on the function of human muscle spindles[2].

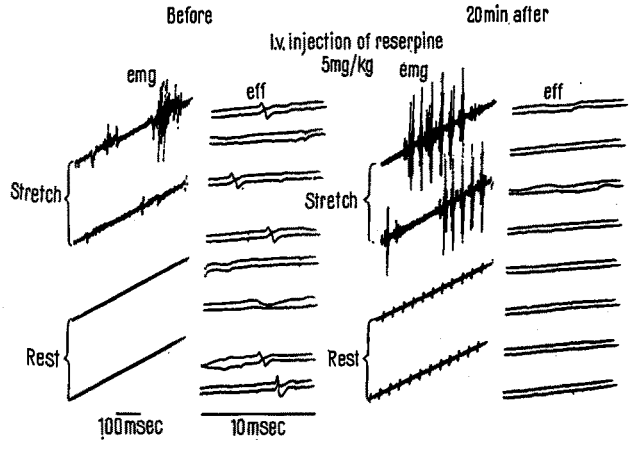

Fig. 1. In an intact rat the gastrocnemius electromyogram (emg) was recorded at rest (rest) and when the foot was dorsiflexed (stretch). The activity was recorded in the efferent fibres of a segmental tail muscle nerve crushed distally to the two pairs of recording electrodes placed 2.5 mm from each other, the proximal pair connected to the upper and the distal to the lower oscilloscope beam (eff). Before reserpine injection the emg was silent at rest and gave a weak response to muscle stretch. The last high amplitude asynchronous burst of activity was recorded during a movement of the limb. The time lag of the single γ-fibre potentials on the two beams corresponds to a 10 m/sec conduction velocity, compared to approximately 40 m/sec of the α-fibre potential also demonstrated. Reserpine induced an increased emg activity even at rest, and on stretch a synchronous discharge appeared. The fusimotor discharge was completely abolished.

A state of akinesia, rigidity and tremor simulating Parkinsonism can be induced in animals as well as in man by high doses of reserpine[3]. Reserpine injected into rats depletes the stores of monoamines in the brain[4], and thus also of dopamine which is concentrated in the nucleus caudatus and putamen[5]. The concentration of these substances is greatly reduced in the corpus striatum of Parkinsonian patients[6]. L-dihydroxyphenylalanine (L-DOPA), the dopamine precursor, abolishes the reserpine syndrome in animals[7] and has been demonstrated to reduce the Parkinsonian akinesia, rigidity and tremor in patients[8].

It is assumed that direct recording from single muscle efferents in this model syndrome in animals has relevance in the investigation of Parkinsonian rigidity.

Rats became akinetic and rigid and developed tremor 20–40 min after an intravenous injection of 3–6 mg/kg of reserpine. A cog-wheel phenomenon was recorded electromyographically in the gastrocnemius on dorsoflexion of the foot. The activity was recorded in single γ-efferents in nerves to the segmental tail muscles. After the injection

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of reserpine, the fusimotor activity disappeared simultaneously with the appearance of the rigidity in the emg (Figure 1).

The spinal reflexes were studied by stimulation of the dorsal root of the 4th caudal segment and recording from the corresponding ventral root in rats lightly anaesthetized with Viadril (Pfizer) and nitrous oxide. Fusimotor activity in the ventral root and the gastrocnemius electromyogram were also recorded. After the reserpine injection, the monosynaptic reflexes were strongly increased at the same time that rigidity appeared and fusimotor activity disappeared (Figure 2)[9].

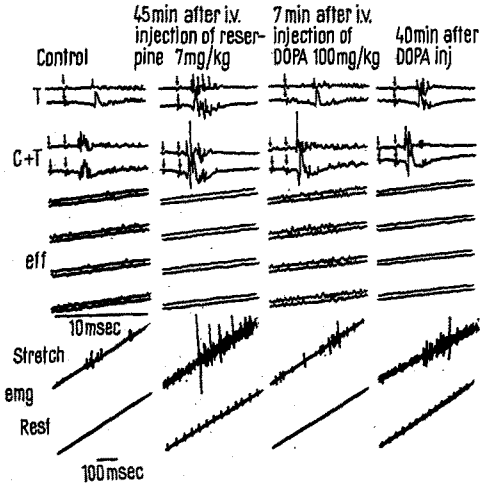

Fig. 2. The reflexes were recorded using two pairs of recording electrodes 9 mm apart on a coccygeal ventral root. The dorsal root was stimulated with a single test pulse (T) and with a conditioning pulse preceding the test pulse (C + T). Shock artefacts indicated by arrows. The spontaneous efferent discharge was also recorded from the ventral root (eff). Reserpine increased the monosynaptic reflex, abolished the fusimotor activity and induced rigidity. DOPA reversed the effects of reserpine.

In order to test whether the results had bearing upon the clinical state of Parkinsonan, DOPA (100 mg/kg) was injected i.v. in the reserpine rats. It caused a reversible disappearance of the rigidity, reappearance of the fusimotor activity and reduction of the monosynaptic reflexes[10].

Reserpine thus causes a shift from γ- to α-efferent-activity simultaneously with the appearance of rigidity. The similar effects of DOPA on reserpine rigidity in rats and Parkinsonian rigidity support the assumption that the results gained in the rat experiments reflect the pathophysiology of Parkinsonism.

All factors discussed above connect the Parkinsonian and the reserpine rigidity and indicate an α-rigidity in Parkinsonism.

Zusammenfassung. Ein parkinsonähnliches Syndrom von Akinesie, Rigidität und Tremor wurde in Ratten durch Reserpin induziert. Gleichzeitig mit dem Auftreten der Rigidität wurde die spontane Aktivität der einzelnen γ-Efferenen zum Stillstand gebracht, weil die monosynaptischen Reflexe gesteigert geworden waren. Das reserpininduzierte akinetische Syndrom stellt also eine α-Rigidität dar.

Die Ähnlichkeit der Wirkung von L-DOPA auf das reserpininduzierte Syndrom und das Parkinsonsyndrom bei Menschen lässt eine gemeinsame Pathophysiologie vermuten.

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Mast Cells and Monoamines

Studies on the human skin suggest that, besides the adrenergic nerves, there exist local tissue stores of some monoamine which may be a vasoconstrictor, and that these stores may be represented by chromaffin cells[1–5]. The cells may belong to the group known as 'tissue mast cells'[6,7]. The existence of chromaffin cells in the human skin has been questioned[8].

With the use of highly sensitive and specific fluorescence methods[9–13] for the direct demonstration of monoamines and some of their precursors (e.g. L-dopa) at the cellular level, we have started experiments to elucidate the existence and significance of local monoamine stores in the tissues – especially the skin – of mammals. Some of the results obtained are reported briefly in this paper. Unless otherwise stated, the tissues were freeze-dried, treated with formaldehyde gas, embedded in paraffin, sectioned and mounted for fluorescence microscopy according to the formaldehyde method[10–12]. Catecholamines and 5-hydroxytryptamine (5-HT) are in this way converted to intensely fluorescent 3,4-dihydroisoquinolines (green) and a 3,4-dihydro-β-carboline (yellow), respectively[13].

Only two monoamine-storing structures have been found so far in the skin of mouse, rat, guinea-pig, hamster, rabbit and cat: (1) Adrenergic nerves, which have in their terminals very high concentrations of noradrenaline[11], and (2) specific cells that on the basis of their staining with Astrablau (at pH 0.2 according to BLOOM and

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