unter Kontrollbedingungen «aufgeladen». Figur 1 zeigt das Ergebnis dieser Versuche: unter der Einwirkung von Adrenalin wird die ^{45}Ca-Aufnahme gegenüber den Kontrollen signifikant gesteigert. Die Abgabe der ^{45}Ca-Aktivität erwies sich unter Adrenalin ebenfalls deutlich gesteigert (Figur 2). Bei diesen Versuchen wurden die Präparate zunächst 60 min in einer ^{45}Ca-haltigen Badlösung «aufgeladen», anschliessend 5, 10, 20 und 30 min in inaktiver K-Tyrodelösung mit und ohne Adrenalinzusatz inkubiert.

Bei den geschilderten Versuchen fand sich keine signifikante Veränderung der Ca-Konzentration im Gewebe, sie lag im Mittel um 0,32 μÄq/0,1 g F.G.

Auf Grund unserer Ergebnisse und neuerer Angaben in der Literatur[2,3,6] möchten wir annehmen, dass die beschriebene Aufhebung der Kalium-Lähmung des Myocards durch Adrenalin mit einer Änderung der Membranpermeabilität für Kationen unter Bevorzugung der Kalzium-Ionen erklärt werden kann[7].

Summary. The effect of adrenaline on the cellular calcium exchange during the K-depolarization of myocardium was studied in isolated left auricles of guinea-pigs by means of the ^{45}Ca. Increasing the extracellular K-concentration from 2.7–30 m*M* without corresponding reduction in sodium concentration results in a depression of contraction strength of the myocardium when current is applied. The depression is immediately reversible by using adrenaline. This was associated with a significant increase of the ^{45}Ca exchange.

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Activation of Purkinje Neurons Through Climbing Fibres After Chronic Lesions of the Olivo-Cerebellar Pathway

In extracellular recording, a discharge evoked in a cerebellar Purkinje neuron by monosynaptic activation through climbing fibres can easily be distinguished from the one elicited in the same neuron through the disynaptic pathway of mossy fibres and granule cells[1] (Figure 1A). Whereas the latter appears as a succession of action potentials, the former is characterized by an initial spike potential followed by a burst of smaller spikes superimposed on a slow wave, and the whole complex is all or none in nature because of the one-to-one relationship between climbing fibres and Purkinje neurons.

Both types of discharges can be elicited in a single Purkinje neuron of the posterior vermis through either peripheral stimulation (somesthetic, visual and acoustic) or electrical stimulation of the cerebral cortex[2]; those due to the climbing fibres have a longer delay than those elicited by the mossy fibre-granule cell system.

According to SZENTÀGOTHAI and RAJKOVITS[3], in the cat almost all climbing fibres originate in the inferior olive, all other cerebellar input arriving via mossy fibres. Fibres leaving the olive on one side cross the midline, traverse the contralateral olivary nuclei and finally reach the cerebellar cortex through the inferior cerebellar peduncle[4]. One would therefore expect that a midline section between the 2 inferior olives or a bilateral section of the inferior cerebellar peduncles would destroy all olivo-cerebellar fibres and thus suppress those responses of the Purkinje neurons which are due to the climbing fibres. The present investigations, based upon such sections, suggest however that all climbing fibres do not originate in the inferior olive.

Chronic lesions of both types were performed electrolytically on 15 cats; 9 animals had a midline section (Figure 1) and 6 a bilateral section of the inferior peduncles. Only cats with complete lesions were taken into consideration in this report. The acute experiments were performed 8 days to 6 months after the section(s). Purkinje cells discharges were recorded from the posterior vermis of the cerebellar cortex (simplex, folium and tuber).

As a control, 8 intact animals were also explored. All preparations were performed under chloralose-nembutal anaesthesia (35 mg/kg of chloralose and 10 mg/kg of nembutal), immobilized with Flaxedil and maintained under artificial respiration. Platinum iridium microelectrodes, made according to the method of WOLBARSHT et al.[5] and insulated with Insulex, were used for recording the extracellular unitary activity. Purkinje cells were identified by their antidromic response to electrical stimulation of the underlying white matter through bipolar stereotaxic electrodes[6]. Only units responding with latencies of less than 1 msec and following the antidromic stimulation up to at least 200/sec were taken into consideration. The responsiveness of these Purkinje cells to activation of both climbing fibres and mossy fibre-granule cells was tested to electrical stimulation of the motor cortex and of one or other posterior paw, to tone (clicks) and to light (brief flashes).

In the control group, almost all neurons identified as Purkinje cells showed the characteristic climbing fibre

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responses, at least to 1 but frequently to several of the 4 types of stimuli. These responses, as shown in Figure 2A, were usually preceded by 1 or several action potentials corresponding to the activation of the mossy fibres and granule cell system. Spontaneous climbing fibre responses were also a common finding. In a few cases, when no clear climbing fibre responses were obtained, afferent stimulation was repeated after i.v. injection of 0.10 to 0.15 mg/kg of strychnine. This method increased Purkinje cell responses to afferent stimulation (Figure 2 D–F). Only 2 from 52 units recorded failed to show spontaneous and evoked climbing fibre responses, but in these 2 particular units the strychnine test was not used. In the group with chronic lesions of the olivo-cerebellar pathway, 46 of the 80 Purkinje neurons indentified, still displayed both spontaneous and evoked climbing fibre activation and mossy fibre-granule cell responses as in the control group (Figure 2 B–C). In a few cases only, an injection of strychnine was needed to release responses (Figure 2 G–J). In the remaining 34 units it was by no means possible to obtain spontaneous or evoked climbing fibre responses while mossy fibre-granule cell activation invariably persisted. As shown in Figure 3, the strychnine test effectively shortened the delay and increased the number of spikes in response to mossy fibre-granule cell activation, but did not succeed in releasing the climbing fibre responses. Therefore it can be suggested that those particular Purkinje neurons were deprived of the monosynaptic excitatory input as a result of the chronic lesions performed. In this preliminary investigation, results could not be quantitatively differentiated according to the 2 kinds of lesions performed.

In one respect our experiments confirm the anatomical data[2] showing that climbing fibres originate in the inferior olive. But only 42% of Purkinje cells were affected by our lesions. It may therefore be postulated that more than half of the fibres projecting to the posterior vermis have an extra-olivary origin. Moreover it should be added

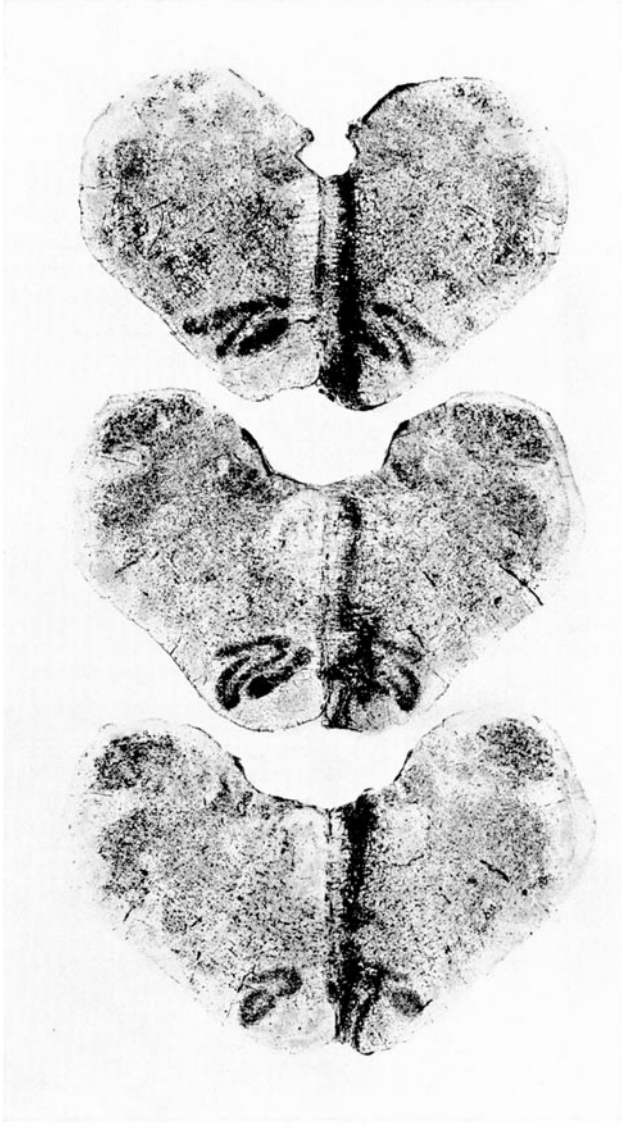

Fig. 1. Frontal sections of the medulla showing an electrolytic midline lesion made 20 days before. The anterior-most section is at the top, the posterior-most at the bottom. Interval between sections 800 μ. Nissl stain.

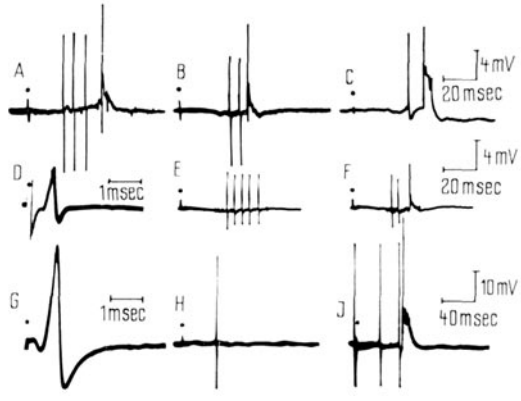

Fig. 2. A–C, responses of 3 Purkinje neurons to electrical stimulation of a posterior paw in A, in an intact preparation; in B, in a cat with bilateral section of the inferior cerebellar peduncles; and in C, in a cat with midline medullary section. Note that, in all 3 cases, mossy fibre-granule cell responses are followed by the climbing fibre responses (see text). D–F, intact preparation; responses of a Purkinje neuron to antidromic stimulation (D) and to posterior paw stimulation before (E) and after (F) i.v. injection of 0.1 mg/kg of strychnine. G–I, cat with bilateral pedunculotomy. Same successive tests as D–F.

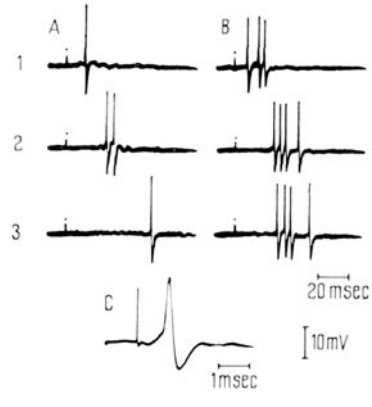

Fig. 3. Animal with bilateral pedunculotomy. Responses of a Purkinje cell identified by response to antidromic stimulation (C), to electrical stimulation of the motor cortex (1) of a posterior paw (2) and to clicks (3), before (A) and after (B) i.v. injection of 0.15 mg/kg of strychnine.

that a complete lesion of the inferior cerebellar peduncles suppress other afferent pathways to the cerebellar cortex. Our midline sections were not specifically limited to olivo-cerebellar fibres (see Figure 1), but extended to the neighbouring reticular formation which also projects to the cerebellar cortex[6]. Therefore, it is possible that the interruption of the olivo-cerebellar pathway is only in part responsible for the suppression of Purkinje neuron responses to climbing fibre activation.

Résumé. Les décharges évoquées dans les cellules de Purkinje lors de l'activation des fibres grimpantes ont été testées par stimulation afférente dans un groupe d'animaux intact et dans un groupe d'animaux avec lésion chronique totale et bilatérale de la voie olivo-cerebelleuse. On a démontré qu'une partie seulement des fibres qui atteignent la région vermienne du lobe postérieur du cervelet ont leur origine dans l'olive inférieure.

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Histochemical Changes in Upper Motor Lesions, Parkinsonism and Disuse. Differential Effect on White and Red Muscle Fibres

Histochemical methods for indication of the enzyme activity in striated muscle have permitted a differentiation of the fibres into groups with different metabolic prerequisites. Thus, in vertebrates, a largely reciprocal relation exists between the phosphorylase activity and the oxidative enzyme activity in the individual muscle fibres[1]. The white fibre has a high phosphorylase activity, whereas the myoglobinrich red fibre has a high activity of oxidative enzymes, such as cytochrome oxidase, DPN diaphorase and succinate dehydrogenase.

On the basis of a method for morphological indication of muscle activity[2], it has recently been shown in preliminary experiments that the motor unit is, to a high degree, uniform with respect to its enzyme pattern[3].

In man, the order of recruitment of the motor units is largely fixed, and is thus independent of the type of activation[4]. This implies that some muscle fibres are in a state of more or less continuous activity, whereas others are utilized only under special conditions, such as strong or rapid contraction. Against this background, it is of interest to ascertain how motor disturbances, associated with some inactivity and change in tone, respectively react on the functional anatomy of the skeletal muscle.

Three groups of patients were studied by means of muscle biopsy. (A) Central hemiparesis due to vascular cerebral lesion or cerebral tumour (9 cases), (B) parkinsonism (4 cases), (C) injury to the anterior cruciate ligament of the knee (11 cases).

Staining for myofibrillar A-band ATPase[5] allows a grouping into 2 distinct types of fibre, in which the activity level is largely reciprocal to the oxidative. The distinct differences in activity were maintained under the existing pathological conditions (Figures 1 and 2). This makes the method well suited for classification of the fibres in measurement of the 2 main types separately.

In the group of central paresis, the unaffected side served as a control. The other groups of patients were compared with a control material[6].

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