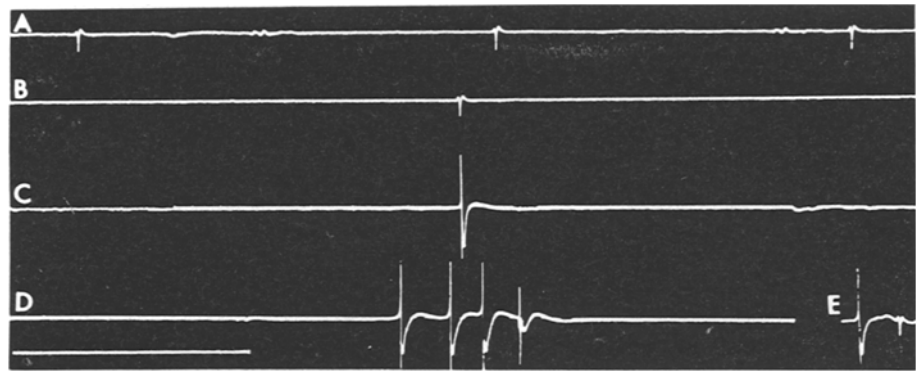

Influence of the level of tonic spasticity on the recruitment order of motor units in the exteroceptive reflex. Recordings from the soleus muscle. Reflexes obtained by skin stimuli on the heel. A: Tonic activity. B: Phasic activity on strong tonic spasticity. C and D: Phasic activity on weak tonic spasticity. E: The first recruited motor unit potential in phasic and the first recruited motor unit potential in tonic activity discharging together, proving that they belong to different motor units. Time bar 100 msec.

Muscles with strong tonic spasticity have an almost exclusive type II fibre atrophy. Only in one case with atrophy of nearly all type II fibres is a significant fraction of the type I fibres also affected (case 6). This selective atrophy of type II muscle fibres has earlier been described both from upper motoneurone lesions and parkinsonism[9,10].

In the muscles with weak tonic spasticity, a significant atrophy is found both within the type I and type II fibre group and there is no evidence for a selective involvement of one of the fibres types.

The present electromyographic study of paralytic muscles indicates that strong tonic spasticity causes a stereotyped order of recruitment of motor units, low frequency units being recruited before high frequency units irrespective of the mode of activation. This is in agreement with findings in decerebrate rigidity in cat, that small tonic motoneurones are always recruited before large phasic ones[11]. As maximal contractions hardly occur in paralytic muscles, some high frequency units should be continuously inactive.

The present histochemical study of paralytic muscles shows a selective type II muscle fibre atrophy in muscles with strong tonic spasticity but a non-selective atrophy in muscles with weak tonic spasticity. This difference in atrophy pattern can hardly be due to any other factor than tonic spasticity itself. We assume that the selective atrophy of type II muscle fibres is due to selective disuse of high frequency units discussed above.

The results also support the hypothesis that slow twitch units tend to discharge at a lower and fast twitch units at a higher frequency.

Zusammenfassung. Bei einer Läsion der zentralen motorischen Bahnen hat die tonische Spastizität eine stereotype Rekrutierungsordnung der motorischen Einheiten und eine selektive Typ II-Faser-Atrophie zur Folge.

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Hippocampal Lesions Produced by Prolonged Seizures in Paralyzed Artificially Ventilated Baboons

Patients having suffered from epilepsy often show hippocampal lesions described as Ammon's horn sclerosis[1,2] or mesial temporal sclerosis[3]. Such lesions resemble those seen after episodes of cerebral anoxia or vascular insufficiency, and it has been suggested that in epileptics they are commonly the result of systemic or local hypoxia occurring during generalised seizure activity[2,4,5]. Experimental studies in cats employing focal injections of alumina cream or tungsten have emphasised the possible role of local vascular factors and of local oedema[6,7]. Meldrum and Brierley[8] have recently shown in adolescent baboons that seizures (induced by bicuculline) lasting 82 to 300 min produce ischaemic cell change selectively in some neurones of the cortex, cerebellum and hippocampus. These animals showed severe systemic disturbances during the seizure (Meldrum and Horton[9]), but it was not possible to evaluate separately the contributions of the different systemic changes and the cerebral discharges themselves. We have therefore produced prolonged convulsions in adolescent baboons that are paralysed and artificially ventilated so that any contribu-

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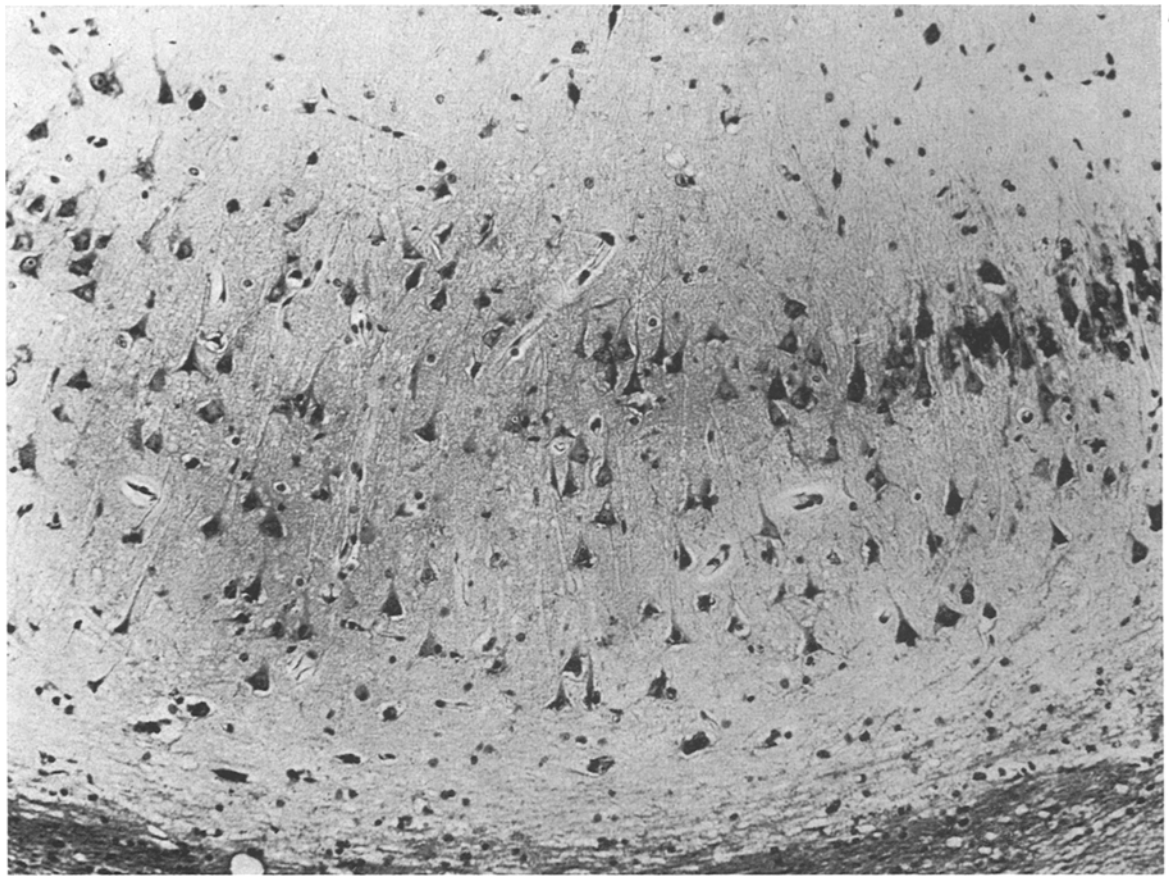

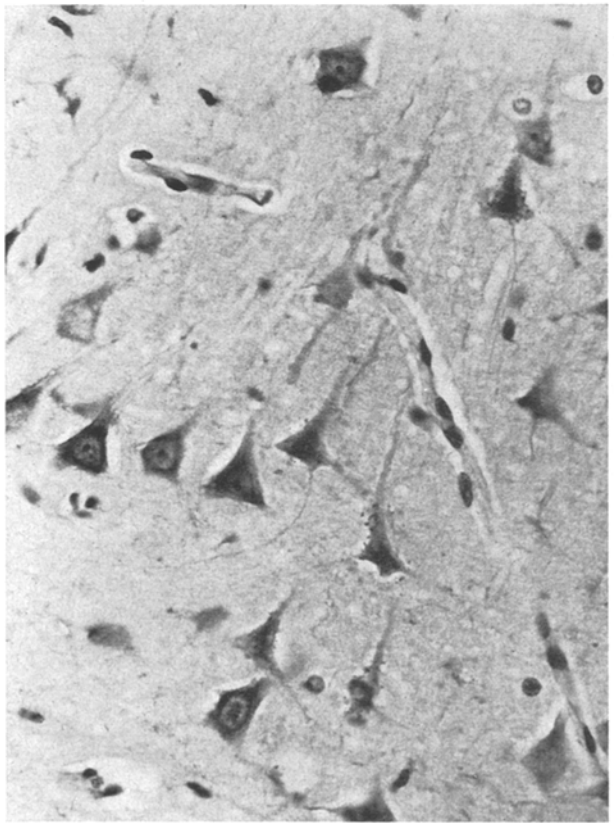

Paraffin sections, stained with luxol fast blue and cresyl fast violet of left hippocampus from baboon No. 706. Bicuculline (0.5 mg/kg) produced seizure activity which was sustained for more than 7 h 30 min; perfusion-fixation of the brain at 8 h. A) Junction of zones h_1 and h_2; ischaemic cell change in h_1. ×135. B) 5 neurones from zone h_1 show ischaemic cell change with incrustations. ×450.

tion of systemic hypoxia, hyperpyrexia and lactacidosis to the genesis of neuronal pathology is largely eliminated.

Materials and methods. Three female adolescent baboons (wts 3.3–3.6 kg) were anaesthetized with halothane and a carotid and femoral artery exposed to permit determination of arterial pressure, blood gas tensions and cerebral blood flow (by isotope clearance[10,11]). Needle electrodes in the scalp were used to record the EEG. Animals were given gallamine triethiodide (10–15 mg) and mechanically ventilated on air (pump volume adjusted to give moderate hyperventilation as judged by arterial Pco_2). Bicuculline (0.5–1.1 mg/kg) was given i.v. and physiological changes recorded for 4 to 8 h. Finally the brain was perfusion-fixed with formalin, acetic acid, methanol (in ratio 1:1:8) and processed histologically[8].

Results. EEG seizure activity began within 10 sec of the injection and was sustained for 3 h 25 min-3 h 45 min and, in baboon No. 706 (Figure) for more than 7 h 30 min. Arterial pressure rose immediately after seizure onset, systolic pressures transiently rising to 180–260 mm Hg. Subsequently blood pressure was midly elevated or normal. Control arterial pH showed moderate alkalosis but tended to fall throughout the seizure, so that late values were in the range 7.40–7.43. In 2 animals body temperature had fallen before seizure onset to 33.8–34° and rose during the seizure to 36.0–36.5°, in the third baboon (No. 714) temperature rose to 38.1 °C late in the seizure. Control oxygen tensions (measured on an IL polarographic analyzer model 125 A, at 37 °C and corrected to original body temperature) were in the range 75–93 mm Hg; subsequent values fell within this range except for a single value of 72 mm Hg at 4 h in No. 706. Control Pco_2 values were in the range 17–27 mm Hg; during the seizure Pco_2 rose slightly (range 23–42.5 mm Hg). Blood flow in the cerebral grey matter (calculated from the half-times of the rapid exponential component of the clearance curve) showed control values in the range 59–66 ml/100 g/min. During the seizure flow was increased 2–3 fold for up to 2 h; later values were above control values except at 7 h in No. 706 (56 ml/100 g/min compared with a control value of 61 ml/100 g/min).

Histological examination revealed, in all 3 animals, ischaemic cell change involving hippocampal neurones in the Sommer sector (particularly at the junction of h_1 with h_2; Figure A). Progression to the incrusted stage was seen in all animals but was prominent in baboon 706 (Figure B). Some neurones showed scalloping of the cytoplasmic outline, probably due to swelling of astrocytic processes. A lack of staining of Nissl substance was seen in some neurones with normal nuclei. Ischaemic cell change was also seen in occasional neurones in the endfolium and in the amygdaloid nuclei (2 animals). Some pyramidal neurones of the 3rd, 5th and 6th cortical layers, particularly in the occipital cortex, also showed ischaemic cell change. The cerebellum was normal in all 3 animals.

This pattern of cerebral pathology differs from what MELDRUM and BRIERLEY[8] have described after prolonged seizures in non-paralyzed babbons, in the lack of cerebellar damage and the greater importance of the hippocampal lesions. The most significant cause of hippocampal damage may be the excessive local discharges during the seizure. However, vascular disturbances related to the intracarotid injections may have contibuted to the lesions in the territories supplied by the internal carotid artery. The absence of cerebellar damage in these 3 animals strongly suggests that the hyperpyrexia, systemic hypoxia and late mild arterial hypotension observed in non-paralyzed animals are significant factors in the production of cerebellar epileptic pathology.

Résumé. Des états de mal, durant de 3 à 7 h 30 sont provoqués par la bicuculline chez 3 babouins adolescents, immobilisés par de la gallamine et ventilés artificiellement. La tension artérielle et le débit cérébral sont augmentés pendant la première partie de la crise et redeviennent normaux secondairement. Des lésions ischémiques sont observées dans la zone fragile de l'hippocampe (zone de Sommer) et dans le cortex mais le cervelet reste indemne.

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Effect of Sino-Aortic Denervation of the Venous Coronary Reflex in Rabbits

Increase of venous pressure in the coronary sinus of the anaesthetized dog causes reflex hypotension through vagal afferents[1–4]. This venous coronary reflex was found to be highly sensitive to general anaesthesia. The barbiturate sensitivity of the reflex is especially greater than that of the carotid sinus reflec[3]. The capricious elicitability and slight effects of the venous coronary reflex in barbiturate anaesthesia give rise to the possibility that these phenom are associated with a powerful opposing effect exerted by the classic buffer afferents on the coronary afferent input. It may be surmized that after elimination of the buffering role of the arterial baroreceptors, the veous coronary reflex could be successfully elicited even in barbiturate anaesthesia, since there is no longer occlusion of the coronary afferent input by effects through the baroreceptors converging on the same central vasomotor neurons or any opposing effect through other vasomotor neurons receiving an independent arterial baroceptor projection. The purpose of present study was to test this possibility.

Investigations were carried out on 14 adult rabbits of both sexes, lightly anaesthetized with pentobarbital. After insertion of a tracheotomy tube, artificial ventilation was maintained with room air. Both carotid sinus regions, the vagi and the aortic depressor nerves were carefully dissected free in the neck for subsequent sectioning. The chest

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