experiment was capable of producing an increase in the weight of both the kidney and heart. In addition, the exposure of the animals to chronic, medium intensity (83 db) audiogenic stress produced a significant increase in the weight of the heart. A number of studies have clearly shown that noise is a potent stressor agent[20,21]. Ethanol has also been suggested as another type of stressor[8,9]. Certainly in the present investigation, as far as the heart and kidneys are concerned, this appeared to hold true. Yet, as the data clearly indicate, the ingestion of ethanol was an effective means of blocking or inhibiting the increase in heart weight due to audiogenic stress alone. In addition, ethanol's own weight increasing influence on the heart was nullified. The combination of ethanol and audiogenic stress also resulted in elimination of the individual effects of the 2 factors on the weight of the kidney. Thus, ethanol in the concentration employed in the present study was apparently functioning as an antistressor agent in the presence of another stressor with different characteristics[22].

Zusammenfassung. Gruppen weiblicher jungfräulicher Albinoratten wurden 2 Wochen lang periodischem Lärm- und Geräuschhintergrund ausgesetzt, während welcher Zeit sie entweder Leitungswasser oder Äthanol trinken durften. Kontrollgruppen wurden bei Abwesenheit des Lärmhintergrunds geprüft. Herzhypertrophie konnte in den Kontrollgruppen mit Äthanol und bei den Ratten (mit Lärmhintergrund) beobachtet werden. Nach Behandlung mit Äthanol war die Herzhypertrophie nicht mehr vorhanden.

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Conduction Block in Capsula Interna Fibres Caused by Striatal Spreading Depression in Rats

Depolarization of neural elements[1,2] with the consequent release of potassium is a necessary prerequisite of the self-maintained spreading of LEAO's[3] spreading depression (SD). On the contrary, SD does not penetrate across layers of white matter[4], probably because the myelinated axons do not release enough potassium ions to maintain the spreading of the process. It remained an open question whether myelinated fibres in the depressed region are passively depolarized or whether they continue conducting. The present paper shows that even gross bundles of myelinated fibres may be blocked by SD invading the adjacent grey matter.

Method. Experiments were performed on 12 albino rats aged 3 months. Tracheotomy and trephine openings were made under light ether anaesthesia. The animals were then immobilized with D-tubocurarine (2 mg/kg) and placed into a stereotaxic apparatus. They were maintained under artificial respiration (open system 60/min). Striatal spreading depression (StSD) was elicited by microinjection of 0.2–0.3 μl 25% KCl into the head of the caudate nucleus through the injector part of an electrode-cannula assembly. The glass capillary (300 μ outside diameter) connected to a calomel cell electrode, was used to lead off the slow potential changes from a point 1.5 mm remote from the cannula orifice. The stereotaxic coordinates of the injection and recording points were AP – 2.0, L 2.0, V 5.0 and AP 0.0, L 2.5, V 4.5 respectively (according to the atlas by FIFKOVÁ and MARŠALA[5]). Cortical EEG and steady potentials were picked up with wick calomel cell electrodes applied onto the brain surface exposed by trephine openings (5 mm in diameter) above the somatosensory or visual cortex. Somatosensory evoked responses were elicited by sciatic nerve stimulation (1 msec, 0.5/sec), visual evoked responses by light flashes (0.5/sec). A conventional 8-channel EEG apparatus was used to record both the EEG and the chopped DC potentials.

Results. Examples of typical experiments are shown in Figure 1. StSD did not significantly influence the spontaneous EEG, unless it invaded the overlying cortex[6]. As this occurs in only 50% cases and always after a considerable delay (usually 3–5 min, Figure 1A), at least 2–3 min were available for observing the pure striatocortical effects. Visual evoked responses were unaffected by ipsilateral StSD (Figure 1C) during this interval, while somatosensory evoked responses were severely depressed (Figure 1A, B). The decrease of the evoked responses started when the slow potential change in caudate attained maximum. The depression culminated after 10–30 sec, the maximum effect lasted for 20–30 sec and full recovery was reached 30–40 sec later. Both positive and negative components of the evoked response were influenced in the same way. Results of 29 experiments are summarized in Figure 2 showing the average changes of the surface positive components of the somatosensory or visual evoked responses during StSD. The predepression amplitude corresponds to the 100% level and the average curves are synchronized either to the maximum of the slow potential (full line) or to the onset of the maximal depression of the evoked response (dashed line). Synchronization of individual recordings in the latter way made the depression more pronounced. The displacement of the average curve along the time axis corresponded then to the mean delay between the maximum

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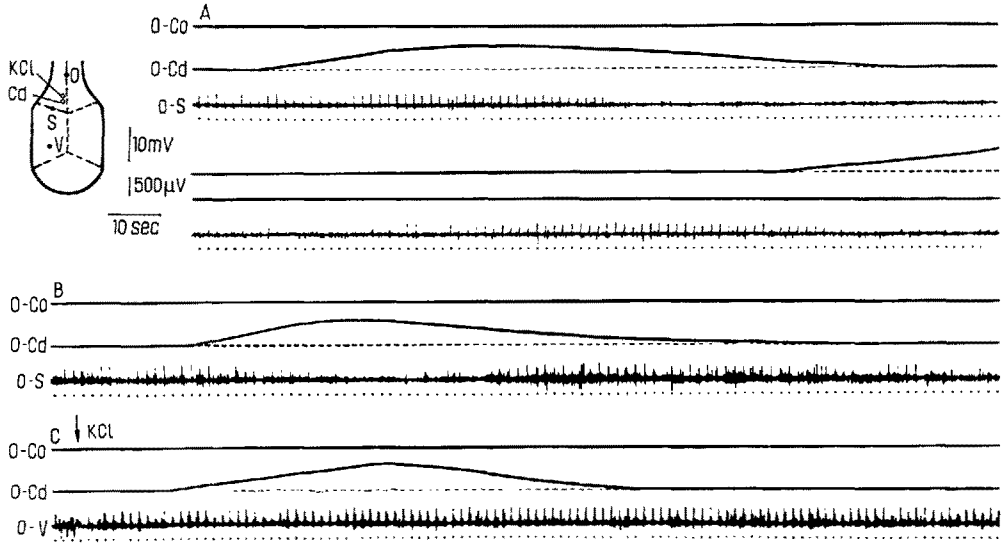

Fig. 1. The effect of striatal spreading depression on somatosensory (A, B) and visual (C) evoked responses in the cerebral cortex. Electrode position shown in the inset brain diagram. O, indifferent electrode; S, V, electrodes in the somatosensory and visual areas; KCl, injecting cannula; Cd, capillary electrode in caudate nucleus. O–Co, O–Cd: slow potential recording from the cortex or caudate respectively; O–S, O–V: EEG activity in the somatosensory or visual cortical areas. Dots indicate application of stimuli. Calibration: 10 mV for the slow potential recording, 500 μV for the EEG.

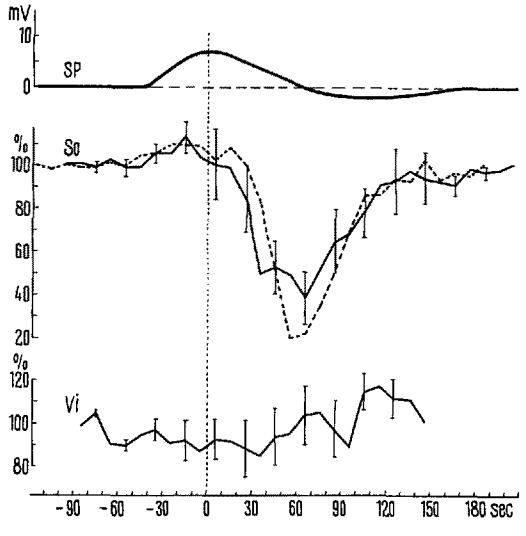

Fig. 2. Average amplitude (in % of the control value) of the positive component of the somatosensory (So) and visual (Vi) evoked responses during StSD (shown by the slow potential wave SP). Vertical bars denote standard errors of the mean values. For details see text.

slow negativity and maximum depression computed from individual recordings.

Discussion. As StSD does not significantly influence the spontaneous activity of cortical neurons[7], the above results can not be due to remote effects at the cortical level. Inhibition of non-specific thalamic nuclei, observed during StSD by BUREŠ et al.[8], would probably affect the somatosensory and visual evoked responses in the same way and would hardly account for such a deep impairment of impulse transmission in a primary sensory pathway. It seems most probable, therefore, that StSD induces a conduction block in the thalamo-cortical fibres forming a part of capsula interna, the scattered bundles of which penetrate through the caudal portion of striatum. During StSD in the surrounding grey matter, the K^+ concentration around these axons is raised to the blocking level. Assuming the potassium effect on central axons to be the same as in peripheral nerves, the peak extracellular K^+ concentration can be estimated as 30–50 mEq/l. KŘIVÁNEK and BUREŠ[9] and BRINLEY et al.[10] arrived at a similar value by measuring the K^+ outflux into the fluid washing the surface of cerebral cortex during SD.

The conduction block induced in capsula interna fibres by StSD resembles in many respects the mechanism of presynaptic inhibition. It seems that some forms of 'remote inhibition'[11] can be explained without assuming specific axo-axonal contacts, by simple depolarization of afferent fibres by excess accumulation of potassium or other depolarizing substances in the extracellular space.

Zusammenfassung. «Spreading depression» im Corpus Striatum kurarisierter Ratten beeinflusst weder spontane EEG noch die visuelle Reizantwort in der Hirnrinde; jedoch kommt es zu einer ungefähr 1 min andauernden, erheblichen Erniedrigung der kortikalen Primärantwort auf somatosensorische Reize. Die Resultate deuten darauf hin, dass die Faserbündel in der Capsula Interna, die in der Striatummasse zerstreut liegen, durch eine erhöhte Kalium-Konzentration blockiert sind.

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