Disintegrations per minute of ^{3}H-thymidine per mg of DNA

Tissue	Group Control	CCK-OP	BPP	BPP + CCK-OP
Pancreas	9.4 ± 1.41	18.7 ± 4.25[a]	17.9 ± 2.61[c]	19.7 ± 2.91[d]
Liver	15.4 ± 2.83	12.9 ± 1.79	15.7 ± 2.98	14.1 ± 2.19
Duodenum	107.4 ± 16.62	154.0 ± 24.95	146.3 ± 20.94	152.2 ± 16.18
Stomach	17.7 ± 3.01	12.8 ± 1.79	15.4 ± 1.97	15.0 ± 1.83

[a] Results expressed as mean ± SEM; n = 12 for each group. p-values were determined using Student's t-test: [b] < 0.05, [c] < 0.01, [d] < 0.005 when compared to control values.

Results. The experimental observations appear in the table. In the pancreas BPP significantly ($p < 0.01$) increased DNA-synthesis from control values of 9.4 ± 1.4 dpm/μg, DNA to 17.9 ± 2.6 (mean ± SEM). Similarly, a significant ($p < 0.05$) 2fold increase in DNA-synthesis was observed with CCK-OP. BPP when given in combination with CCK-OP neither augmented nor inhibited the effect observed with CCK-OP alone. The 2fold increase in DNA-synthesis was comparable to that observed with the individual hormones, and was significantly above control values ($p < 0.005$).

Although in the duodenum CCK, BPP and the combination, resulted in levels 50% higher than those observed in NaCl-injected rats, the difference did not reach statistical significance. BPP, CCK-OP, or the combination did not increase the rate of DNA-synthesis in the stomach (oxyntic gland area) or the liver.

Discussion. Whereas protein and RNA-synthesis are measures of cell hypertrophy, DNA-synthesis is held to be a specific index of cell division and hence cell growth[9]. In the present study, bovine pancreatic polypeptide was found to increase the rate of DNA-synthesis in the rat pancreas. This hormone, together with gastrin and CCK therefore becomes the third peptide demonstrated to have a trophic effect on this organ. In contrast, BPP had only a weak effect on the duodenum and failed to stimulate DNA-synthesis in the stomach or the liver. Pharmacological studies in the dog have demonstrated that low dose infusions of BPP relax the gall bladder and inhibit CCK-mediated pancreatic enzyme production[7]. Because CCK stimulates DNA-synthesis in the pancreas a similar inhibitory effect by BPP might also have been predicted. However, in this study, the 2fold increases in pancreatic DNA-synthesis observed with CCK was neither inhibited or augmented by the addition of BPP.

More recently, it has been demonstrated that PP is released by CCK-like peptides[11]. In this study, the increase in pancreatic DNA-synthesis by PP was identical to that observed for CCK. As CCK causes release of endogenous PP, it is conceivable that its trophic action is mediated entirely through PP release. This hypothesis is supported by the fact that the reported trophic response pattern of CCK in the pancreas, duodenum and stomach[3] confirmed in this study exactly parallels that observed for PP.

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The effects of topical succinylcholine on single unit and electrocorticographic activity

Ü. Tan, C. Marangoz and F. Senyuva

Atatürk Üniversitesi, Fizyoloji Enstitüsü, Erzurum (Turkey), 15 March 1977

Summary. Topical succinylcholine induces massive epileptiform discharges in the electrocorticogram, coinciding with the bursting or tonic activity of single cortical cells.

The neuromuscular blocking agent succinylcholine (SCh) consisting of 2 acetylcholine molecules is known to stimulate the muscle spindles eliciting massive afferent discharge in relatively small dosages[1]. This drug also depolarizes the end plate free part of the muscle membrane[2] and reacts with the cholinoceptive site at the first node of the motor nerve terminal[3]. In addition to these SCh effects, it has recently been found that it may act as a potent convulsant when applied topically to the cerebral cortex[4]. It was assumed that topical SCh may strongly depolarizes the cortical cells. An attempt was made to verify this hypothesis in the present work.

Materials and methods. The experiments were carried out in 6 adult cats spinalized at C_1 and maintained by artificial respiration. Under initial ether anesthesia, the trachea and the left femoral vein with the femoral artery were cannulated. The latter was used to monitor the blood pressure, which was always kept above 70 mm Hg. The left cerebral cortex was exposed by craniotomy, the dura was removed, and this area was filled with warmed

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paraffin oil at 37 °C. Pyramids were exposed by a ventral approach to identify the cortical pyramidal tract neurons by antidromic stimulation. All contact points were anesthetized locally with citanest®, after which the ether anesthesia was dicontinued. The cortical single cell activity was recorded by using glass micropipettes filled with 3 M KCl solution. Cortical surface activity was recorded by a silver ball electrode placed 1 mm near the microelectrode. Separate indifferent electrodes were placed on the pericranium. SCh chloride (Fako-Istanbul) was dissolved in saline to obtain a 20% solution. For the topical SCh application, a 2×2 mm² of an absorbent tissue was immersed into the SCh solution and placed over the cerebral cortex near the recording electrodes after removing paraffin oil which was refilled following completing the topical application. In initial experiments only the electrocorticogram (ECoG) was recorded bipolarly from the somato-motor (electrode 1), temporal (electrode 2) and parietal (electrode 3) cortex.

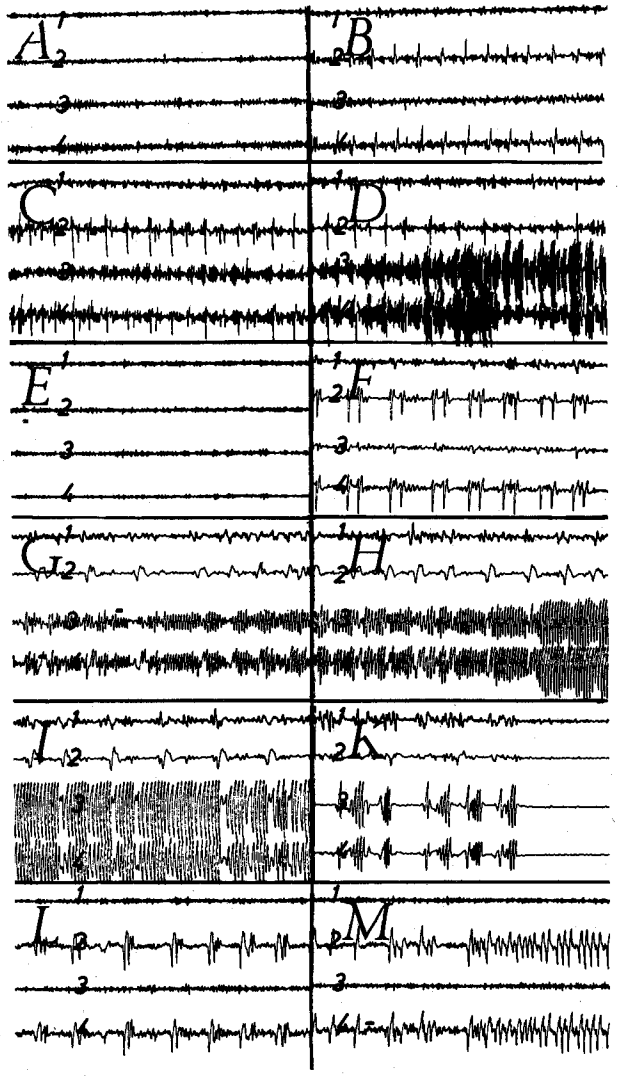

Fig. 1. The effect of topical SCh on ECoG. *A* Control; 1, EEG from the right scalp; 2, 3 and 4: ECoGs recorded between the electrodes 1–2, 2–3 and 1–3, respectively (1, somato-motor, 2, temporal, 3, parietal). *B* 2 min after 5% SCh under the electrode 2. *C* 30 sec later. *D* Within the following 5 sec. *E* Cessation of epileptiform activity, which reappeared within 6 min after SCh and developed to massive convulsive discharges as seen in the other cut-outs lasting for 6 sec in *A–E* and 3 sec in *F–M*.

Results and discussion. Figure 1 illustrates the typical convulsant effect of topical SCh. In the control, the scalp EEG (A_1) and the ECoGs recorded between the electrodes 1–2 (A_2), 2–3 (A_3) and 1–3 (A_4) showed fast activity. 2 min after topical application of 5% SCh to the temporal cortex, intermittent spikes occurred at the 1st and 3rd electrodes ($B_{2,4}$). 30 sec later,

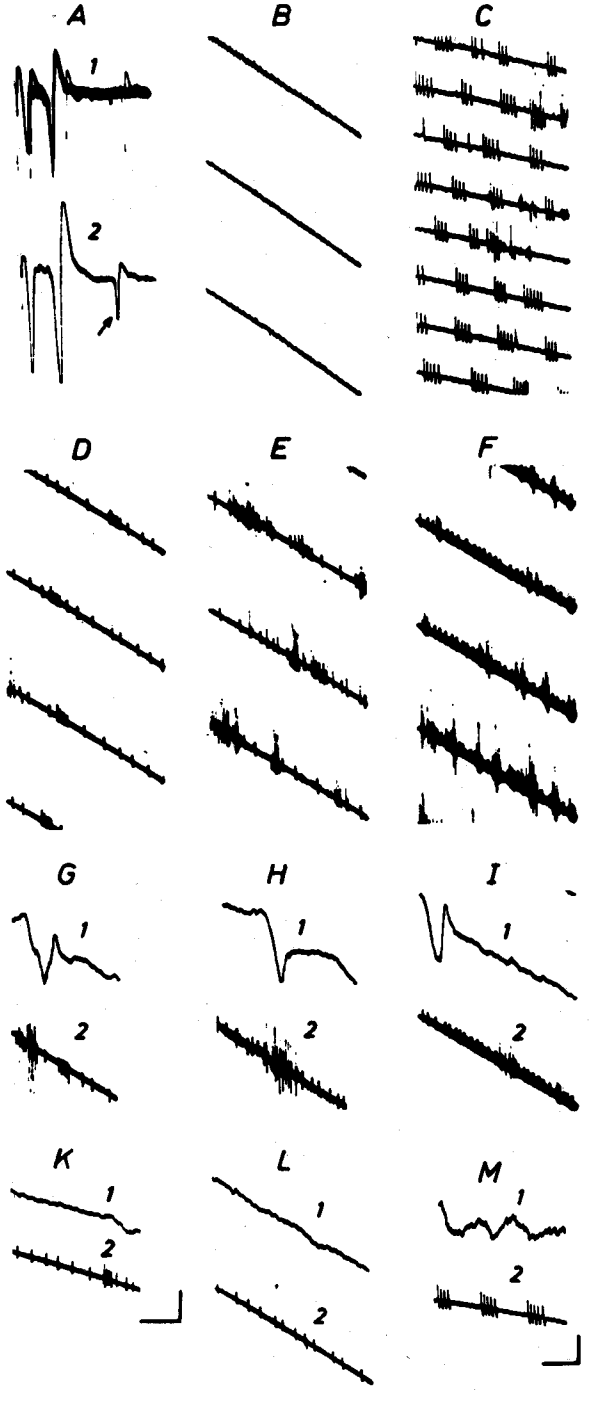

Fig. 2. The effect of topical SCh on the cortical neurons. $A_{1,2}$, Antidromic activation of a pyramidal neuron before and after SCh, respectively. Arrow indicates spontaneous discharge. *B–F*, Spontaneous, bursting and tonic activity of single units after topical SCh, as described in text. *G–M*, Correlation between ECoG (1) and unit activity (2). Horizontal line: 100 msec for *A–C* and *M*, 100 msec for *D–L*. Vertical line 100 μV for the ECoGs.

the amplitude of these spikes increased, and a fast conculsive activity began to appear ($C_{2,3,4}$), increasing in amplitude within the following 5 sec ($D_{3,4}$). This epileptiform activity disappeared 3 min later (E). Within 6 min of SCh application, the spikes reappeared in doublet form ($F_{2,4}$), which, however, developed gradually to massive epileptiform discharges in the following time period (G, H, I: 3,4). After cessation of the seizure activity (K) doublets reappeared ($L_{2,4}$) following an inactivation period lasting for about 1 min. Occasionally, the intermittent high amplitude sharp waves appeared and developed into the high frequency epileptiform activity ($M_{2,4}$). During seizure activities pupillary dilatation was observed in all experiments.

Figure 2 illustrates the effect of topical SCh on the cortical single cell activity and the correlation between ECoG and single cell discharge pattern in a typical experiment. Single shock stimulation of the left pyramid above decussation elicited two spikes recorded extracellularly from the left postcruciate gyrus in depth of 1.8 mm from the cortical surface (A_1). Spontaneous activity did not occur in the control (B). Within 2 min after topical application of a 20% SCh-solution to the postcruciate gyrus near the recording microelectrode, the antidromic response remained unchanged, but a small neuron began to discharge spontaneously (A_2), which lasted only for about 1 min (not illustrated). This cortical cell being not activated by antidromic stimulation (nonpyramidal tract neuron) was reactivated within 3 min and delivered bursts composed of 3–6 spikes at constant 2-msec-intervals (C). Within 7 min these bursts occurred less frequently, but a tonic discharge appeared (D), and the large pyramidal tract neuron also delivered high frequency bursts (E, F). ECoG showed no waves in the control because of low amplification. However, after topical SCh, small or large sharp waves appeared (G, H, I, K, M: 1). The large sharp waves coincided with strong bursts of a large neuron and the small waves with burst of a small neuron (G and H; respectively). Sharp waves without corresponding burst were also common ($I_{1,2}$). Synchronization of the unit discharge, i.e., tonic activity, usually associated with flattening of the ECoG (K and L: 1,2). In M, low voltage rhythmic waves (1) appearing in ECoG during bursting activity of a nonpyramidal tract neuron (2) was recorded in fast sweep.

The results of this work clearly showed that topical SCh is a potent convulsant, bringing the single cortical neurons to bursting activity and tonic discharge. The close correlation between ECoG and unit activity suggests, on the other hand, that a) the small waves in the ECoG may be produced by the bursting activity of the small cortical cells; b) the large sharp waves may occur either without participation of any spike activity, or during very strong bursting activity of many cortical cells in concert; c) the flattening periods in the ECoG may appear during tonic activity of the cortical cells without bursting.

Morphological appearance of fat in the epithelial cells of different portions of the intestines in mice

R. L. Snipes

Zentrum für Anatomie und Cytobiologie, Justus-Liebig-Universität, D-6300 Giessen (Federal Republic of Germany), 25 April 1977

Summary. Absorption of administered fat in the small intestine of mice as judged morphologically in semi-thin sections demonstrates a proximal to distal gradient, being greatest in the mid-jejunal area, but less in the duodenum and ileum. The criterion of the amount and size of fat droplets in intestinal epithelial cells, however, does not necessarily give a reliable indication of the efficiency of fat absorption in the different segments of the intestine.

Numerous investigations on the course of fat absorption have been performed in the past decade with the aim of elucidating the mechanism of fat uptake by the epithelial cells in the small intestine[1]. While much attention has been given to the ultrastructure and molecular levels, the course of fat absorption in the different portions of the intestine as a whole has been somewhat neglected. Most of the fat in a normal diet is sequestered in the duodenum and this process is so efficient that the more distal segments are not normally exposed to a substantial amount of fat. That the distal portions are capable of absorption is not doubted; even the cecum and colon of some species have been shown to have a fat-absorbing capacity[2]. This ability may be of medical importance, especially in extensive resection of the small intestine or in pathological conditions of the proximal small intestine. Recently, Sabesin et al.[3] demonstrated that distal portions of the intestines of rats perfused with fat were less efficient in absorbing lipids than proximal portions of the small intestine.

To date, a morphological definition of the role of each segment of the small and large intestine in fat absorption is lacking. It is the aim of this communication to offer a morphological picture of the process of fat absorption in the different segments of the intestine at the light microscopical level, using semi-thin sections of resin-embedded material stained specifically for fat.

In order to involve the different segments of the intestine in the process of fat absorption, a fasting period of 2–3 days is necessary to empty the gut of food content. Fasted mice were force-fed massive doses of commercial triglycerides (Mazola oil; 0.10–0.15 ml/g b.wt) over 2–3 days by gastric intubation. The various segments of the intestine were sampled measuring from the pyloric-duodenal junction (PDJ) distally. When the cecum and colon contained fat, these segments were also sampled. Small pieces were cut from each sample and fixed according to routine methods for electron microscopy. 1–3 µm thick sections were cut and stained with p-phenylene diamine[4]. Morphologically, fat appears as small droplets concentrated in the supranuclear region, presumably in the Golgi region, in the epithelial cells of the proximal-most portion of the small intestine (duodenum; first 4 cm from the PDJ). In addition, scattered, fine droplets occur in the apical cell area. Distal to the duodenum (4–20 cm from the PDJ) an abrupt increase in the content and size of fat droplets can be noted. The apical portions of some cells are completely filled with droplets of varying sizes. In the more distal portions of the small intestine (ileum; approximately 20 cm from the PDJ to the cecum), the amount of detectable fat decreases gradually. In the cecum and proximal colon a limited number of fat

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