Responses to both visual moving stimuli and to acoustic stimuli, simulating sound source movement, were compared in 23 neurones. Only 9 neurones exhibited responses to ΔT changing trains and responded to visual moving stimuli. Acoustical responses classified as 'asymmetrical' were characterized in 4 neurones (out of 9) by visual directional specifity and the preferred direction of movement for an acoustic stimulus agreed with the preferred direction for a visual stimulus.

It may be thus concluded that cerebellar units from area VI and VII respond to moving visual stimuli and similarly to acoustic stimuli, which simulate movement of the sound source. In comparison with neurones from specific auditory pathways[8,9], a remarkable number of neurones (31%) displayed an 'asymmetrical' type of reaction, with a certain preference for the direction of sound source movement. Moreover, 21% of neurones exhibited a 'symmetrical' type of response, i.e. without any preference for the direction of sound source movement.

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At the same time with this fact a large number of directionally non-specific visual neurones (51%) was found. Furthermore in units with preference for a certain visual direction, less expressed specificity was present, when compared with the properties of neurones from movement sensitive visual areas (e.g. the superior colliculus[10,11]). It is possible to assume from our experiments that a) the information about the movement of acoustic and visual stimuli seems to be most important for the cerebellum, b) responses to other characteristics of the stimulus (i.e. frequency, intensity, direction) are at least to some extent less expressed (e.g. [6]).

Zusammenfassung. Der Einfluss von simulierten bewegten Schall- und Lichtquellen auf einzelne Neurone im Kleinhirn wurde untersucht.

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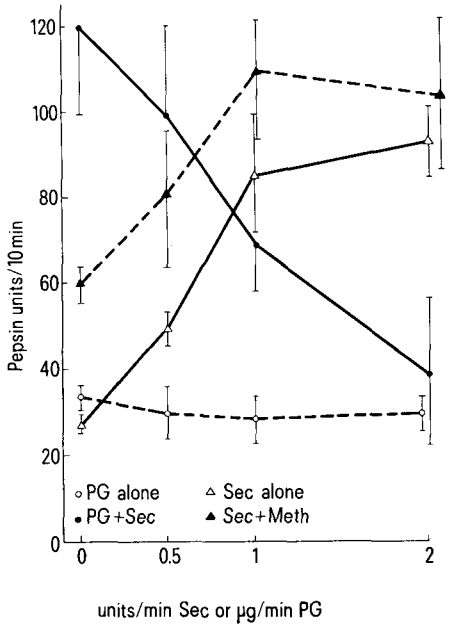
Does Pentagastrin Suppress Secretin Induced Pepsin Secretion in Heidenhain Pouch Dogs?

In 1956 Schofield[1] showed that pepsin secretion from Heidenhain pouches fell following feeding. A possible explanation for this is that gastrin depresses pouch pepsin secretion. Vagne and Grossman[2] have since found that pepsin stimulated by distension of the intact stomach through a fistula is depressed by exogenous gastrin. It is not clear in their experiments how they avoided antral stimulation. Olbe et al.[3] have noted that pepsin stimulated from Pavlov pouches by sham-feeding is also depressed by suprathreshold doses of gastrin. We[4,5] have found the same thing in distended Heidenhain pouches. Both secretin and methacholine are good stimulants of Heidenhain pouch pepsin in dogs. It was of interest to us to determine if pentagastrin diminished the action of either of these agents.



The effect of pentagastrin (PG) on secretin- (Sec) (1 unit/min i.v.) stimulated pepsin and of secretin (Sec) on methacholine-(Meth) (2 µg/min i.v.) stimulated pepsin compared with secretin (Sec) alone and pentagastrin (PG) alone in Heidenhain pouches. *n* in PG experiments = 6; in others = 10.

In reviewing data obtained 3 or 4 years ago and presented here we have found that if progressively increasing doses of pentagastrin (Peptavalon®, kindly supplied by Ayerst Laboratories) (PG) (0.5, 1 and 2 µg/min i.v.) are added to a continuous i.v. infusion of secretin, 1 U/min, the stimulated pepsin secretion is progressively depressed (Figure). In the methacholine experiments a continuous background i.v. infusion of 2 µg/min methacholine was given and then 0.5, 1 and 2 units of secretin/min was superimposed. At the lowest dose only, the secretin response was significantly augmented by methacholine. Beyond this there was no significant difference. Certainly secretin did not depress methacholine-stimulated pepsin or vice versa. In fact, the calculated maximal responses and the slopes of the dose response curves (reciprocal plot) for secretin alone and with methacholine are not significantly different.

We have noted previously that pentagastrin does not stimulate Heidenhain pouch pepsin and that in many respects the actions of cholinergics and secretin on pouch pepsin resemble one another[5].

In the PG experiments, 6 dogs were used and in the methacholine experiments 10 were used. The results shown (Figure) are the means of the last two 10-min collections at each dose level. Pepsin was estimated using Anson's[6] hemoglobin method. The units are mg tyrosine liberated per 10 min collection.

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It is concluded that in Heidenhain pouches pentagastrin does antagonize secretin-stimulated pepsin but secretin and methacholine do not antagonize one another.

Summary. Secretin-stimulated pepsin secretion from Heidenhain pouches was significantly depressed by concomitant pentagastrin. Pentagastrin by itself was without effect on pouch pepsin. Methacholine, on the other hand, did not antagonize secretin-stimulated pouch pepsin.

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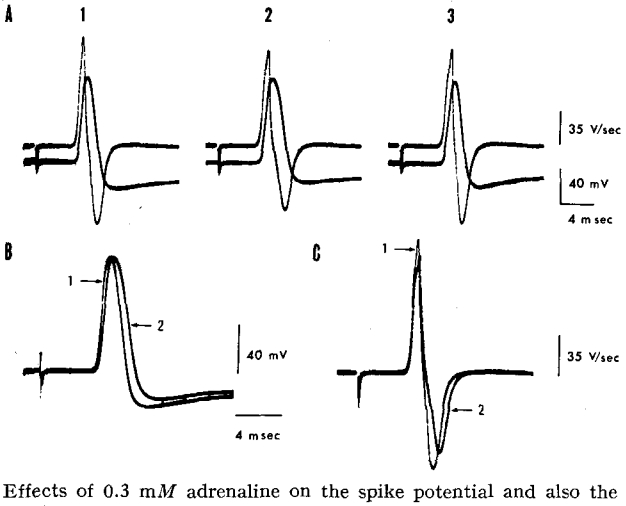
The Direct Action of Adrenaline on the Action Potentials of Bullfrog's (*Rana catesbeiana*) Sympathetic Ganglion Cells

Catecholamines play their roles as chemical transmitters at neuromuscular or interneuronal junctions. It is generally believed that catecholamines released from catecholaminergic neurones act on their target muscle or nerve cells and thereby regulate the excitability (or the resting membrane potential) of these cells. In the case of cardiac muscles, however, catecholamines are able to regulate not only the resting membrane potential but also the shape of the action potential by increasing the plateau amplitude[1,2]. The main effect of catecholamines on the action potential has been explained by an increase of the slow inward current, presumably carried by both calcium and sodium ions[1,2]. Another possible effect of catecholamines was the decrease of potassium conductance[2]. Such direct controls, by catecholamine, of the processes involved in generation of action potentials have not been observed in the nervous system. The present work, however, demonstrated that a catecholamine is indeed able to control directly the generation of action potentials of target nerve cells at interneuronal junctions.



Effects of 0.3 m*M* adrenaline on the spike potential and also the maximum rates of rise and fall of spike potentials in bullfrog's sympathetic ganglion cell. A) Suppression of the peak amplitude of the after-hyperpolarization of action potential and of the maximum rates of rise and fall in the presence of adrenaline. Upper recordings show the maximum rates of rise and fall of action potentials. Lower recordings are action potentials which were produced by applying antidromic stimulations to postganglionic nerve fibres at an interval of 2 sec. These records were taken before (1), 5 min after (2) an application of adrenaline and record 3 was taken 6 min after its withdrawal. B) Action potentials of records 1 and 2 in A) were superimposed in order to clarify the action of adrenaline. Number 1 and 2 are records 1 and 2 in A), respectively. A suppression of the peak amplitude of after-hyperpolarization and a prolongation of the duration of the action potential are clearly seen in 2. C) The maximum rates of rise and fall of action potentials taken from records 1 and 2 in A) were superimposed in order to clarify the effect of adrenaline. Number 1 and 2 are records 1 and 2 in A), respectively. Suppressions of both maximum rates of rise and fall are clearly demonstrated in 2.

The bullfrog's (*Rana catesbeiana*) sympathetic ganglion cells were used throughout. The intracellular recording technique of action potentials are essentially similar to that described elsewhere[3]. The ionic composition of Ringer's solution are as follows: NaCl 112 m*M*, KCl 2 m*M*, $CaCl_2$ 1.8 m*M* and $NaHCO_3$ 2.4 m*M*. Adrenaline bitartrate was used at the concentration of 0.3 m*M* throughout. The experiment was carried out at room temperature (20–23 °C).

When adrenaline was added to perfusate (Ringer's solution), the resting membrane potential of ganglion cells showed no change or slight depolarization, which never exceeded 5 mV, depending on individual cells. Changes in the membrane resistance (input-resistance) of ganglion cell observed in the presence of adrenaline were not detectable even when the membrane was depolarized. In the case of the depolarized membrane by the action of adrenaline, a slight increase of membrane resistance, however, was observed when the membrane potential was fixed at original resting potential level by anodal current. These results indicated that the membrane resistance tended to increase under the effect of adrenaline.

The effect of adrenaline on action potentials was tested by using ganglion cells of which resting membrane potentials showed no depolarization in the presence of adrenaline. Action potentials of these cells were generated by applying repeated antidromic stimulation to postganglionic nerves at intervals of 2 sec. These action potentials produced before, during and after an application of adrenaline were compared with each other, in order to examine the effect of adrenaline and its reversibility. A most significant effect of adrenaline on the action potential was found to be a decrease of the peak amplitude of after-hyperpolarization of action potential, and a maximum rate of fall and also a prolongation of the duration of action potential. These changes were often

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