certainly possible that in conditions of partial coronary occlusion modest hypothermia could prove beneficial.

Résumé. On a étudié la tension isometrique des fibres trabéculaires isolées du myocarde du rat, en fonction d'une température de 38–44 °C. On a trouvé que la contractilité était nettement affaiblie aux températures supérieures à 38 °C. Après une courte durée, l'effet est en partie reversible.

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Excitability Changes in Spinal Cutaneous Primary Afferent Terminals Induced by Acoustic Stimuli

Heterosensory presynaptic inhibition at the level of the spinal cord was first demonstrated by Mallart[1] when he recorded lumbar dorsal root potentials (DRPs) in the chloralose-anesthetized cat after acoustic as well as photic stimulation. Recently, Chu[2] studied the effects of auditory and visual inputs on lumbar DRPs and spinal reflexes and found that a conditioning stimulus in either one of these two special senses, which in itself did not evoke lumbar DRPs, inhibited segmentally evoked DRPs while producing a biphasic effect on spinal reflexes. This biphasic effect consisted of an early facilitation of mono- and polysynaptic reflexes at 30–80 msec followed by a depression which reached a maximum at about 300 msec. It became of interest to study the effects of a conditioning auditory input on the excitability of cutaneous primary afferent terminals in the lumbosacral spinal cord. In the following experiments we tested the excitability of sural presynaptic terminals by the method of Wall[3].

Six adult cats were anesthetized by i.p. administration of α-chloralose (40–50 mg/kg), immobilized with gallamine triethiodide (Flaxedil), and artificially respired (4% CO_2 in expired air). The lumbosacral cord was exposed by laminectomy and covered with mineral oil maintained at a constant temperature of 37 °C. Body temperature was also maintained close to 37 °C by a heating pad placed under the cat. Ventral roots of L_6, L_7 and S_1 were sectioned on one side. A branch of the ipsilateral sural nerve was isolated, crushed peripherally, and placed on platinum hook electrodes in a mineral oil pool. DRPs were recorded from a cut dorsal L_7 filament. The excitability of the sural nerve afferent terminals was tested by delivering strong pulses of 0.1 msec duration and 10–50 volt strength through a low resistance 4 *M* NaCl glass microelectrode introduced into the spinal cord just medial to the entry of dorsal L_7 and directed laterally following the technique of Wall[3]. The best response was obtained at a depth of about 1.25 mm. The acoustic stimulus consisted of a train of clicks of various frequencies (300–1000 cycles/sec) and duration (10–40 msec) delivered through a loudspeaker placed about 10 cm away from the ipsilateral ear. The higher frequencies were usually more effective.

A train of clicks produced negative DRPs in a lumbar dorsal rootlet as originally reported by Mallart[1]. When acoustic stimulation preceded sural nerve stimulation, the conditioning auditory input inhibited segmentally-evoked DRPs. This inhibition reached a peak around 50 msec and persisted for over 400 msec. Its time course suggested the presence of a presynaptic inhibitory pathway.

Since cutaneous presynaptic inhibition is effected by depolarization of afferent fibers at their terminals[4], auditory conditioning should increase the excitability of these terminals when tested by direct electrical stimulation. The test pulses delivered through a microelectrode were always submaximal to allow for the recruitment of more fibers in the stimulated area as a result of their depolarization by the conditioning input, and only the fastest, synchronized group of antidromic impulses was considered. Several tracks and depths of sural terminations were explored. At some sites within certain tracks, little or no increase in excitability was observed. But at most sites,

Maximal increase (%) in excitability of sural terminals after acoustic stimulation at conditioning-test intervals of 50, 58 and 75 msec

Experiment	50	58	75
1	67	85	56
2	40	51	36
3	29	80	61
4	14	53	53
5	78	300	210
6	80	204	111

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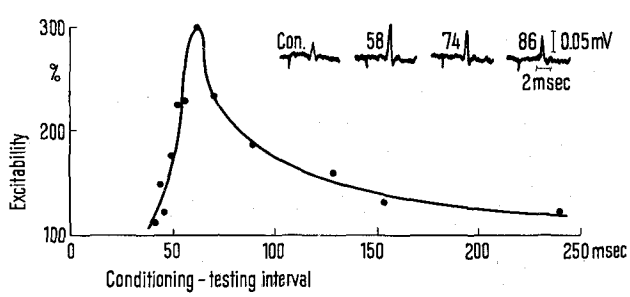

An experiment showing the time course of the increase in excitability of sural afferent terminals after conditioning acoustic stimulation. A low resistance microelectrode was inserted into the dorsal horn and used for test stimulation, the antidromic response being recorded in the ipsilateral sural nerve. This antidromic response was taken as control, and the increase in its size by conditioning acoustic stimulation is expressed as percentage on the ordinate, while the conditioning-test intervals are indicated on the abscissa. The inset shows at left the control antidromic response after stimulation of the sural terminals, followed by the response at the indicated intervals.

conditioning acoustic trains increased the size of the sural antidromic spike to an appreciable extent, with a maximum increase of 50–85% in 4 experiments, but up to 200–300% in 2 experiments (Table). This increase reached a maximum at conditioning-test intervals of 55–65 msec, and ran a prolonged time course lasting over 150 msec (Figure).

In two experiments, conditioning photic stimulation also produced an increase in excitability of sural afferent terminals, but this was much weaker than that produced by acoustic stimulation.

In conclusion, an auditory input depolarizes lumbosacral presynaptic terminals of cutaneous afferent fibers leading to an increase in their excitability. This suggests a presynaptic modulatory influence of acoustic stimuli on cutaneous transmission at the level of the spinal cord[5].

Résumé. L'excitabilité des terminaisons des fibres afférentes cutanées primaires spinales augmente après conditionnement par un stimulus sonore.

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Hormonal Rupture of Larval Diapause in the Tick *Rhipicephalus sanguineus* (Lat.)

In nature in India, eggs of the hard tick, *Rhipicephalus sanguineus* (Lat.) (Acari: Ixodidae) usually hatch during the winter or early spring. But the larvae diapause, they do not seek hosts or attach if they are placed on hosts during the subsequent spring or summer. In the laboratory, larvae exposed to a photoperiodic cycle of 16 h of light, 8 h of dark (LD 16 : 8) at a temperature of 25 ± 1 °C and 75% R. H. diapause in much the same way, but comparable larvae kept in LD 8:16 for 5 weeks attach and feed on rabbit and/or mouse[1].

It is well known that juvenile hormone or juvenile hormone analogues will induce feeding activity[2], yolk deposition[3], oviposition[4] and morphogenetic effects[5] in diapausing insects. Also, moulting hormones (ecdysones) will rupture diapause in several species of insects[6]. We have recently investigated the effects of the insect moulting hormones (α-ecdysone and β-ecdysone (ecdysterone) and an analogue of juvenile hormone (*trans, trans*-10, 11-epoxyfarnesenic acid methyl ester), on the termination of larval diapause in the hard tick, *R. sanguineus*, and the results are reported in this paper.

The ecdysones were dissolved in 10% methanol and the analogue of juvenile hormone was dissolved in acetone. Larvae were anaesthetized by exposing to Co_2 and/or in ice bath before treatment with hormones.

Groups of 30 diapausing larvae exposed to LD 16:8 from the time the adult mother tick dropped from the host were anaesthetized and were treated with either 0.1 or 1.0% solutions (1 µl) of one of the 3 compounds applied

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Table I. Effects of hormones on termination of diapause in *Rhipicephalus sanguineus* (Lat.) larvae as determined by the percentage of attachment to rabbits when treated topically with 1 µl per larva to 6 replicates of 30 diapausing larvae 30 days old

Concentration (%)	Larval attachment (%)						
	1	2	3	4	5	6	Average
	α-ecdysone						
0.1	10	12	6	8			9
1.0	20	19	21	32	30	44	29
	β-ecdysone						
0.1	48	28	28	20			31
1.0	32	28	38	30	25		31
	trans, trans-10, 11-epoxyfarnesenic acid methyl ester[a]						
0.1	0						
1.0	0						
	Long-day control						
	4	8	10	12	6	4	7
	Short-day control						
	52	43	56	45	32	31	43

[a] Methyl-10, 11-epoxy-3,7,11-trimethyl-2,6-dodecadienoic acid. A few larvae escaped or died. Therefore, the percentage of attachment indicates the ratio of the number of larvae which attached to the total number present and alive at the end of 48 h.