effect of ouabain on unchilled oocytes pretreated with ethacrynic acid[1].

Abolition of active Na^+ transport by an environmental temperature of 0 °C is shown to be rapid and reversible. These features are suggestive of an enzymic process or interruption of the energy supply to the Na pump, or both. The observation that a temperature of 4 °C fails to almost entirely stop Na efflux is in keeping with the idea that cold of this degree neither completely interrupts the Na^+-K^+-ATPase[5,6] nor the phosphorylation of ADP[7].

The Na efflux is drastically reduced by ethacrynic acid when chilled oocytes are treated sufficiently long with the inhibitor before rewarming. The size of the effect, roughly 90% in the majority of cases, is comparable to that produced by ethacrynic acid when unchilled oocytes are treated for more than 3 h[1]. It is thus evident that the use of cold shock is one simple way by which information obtained at room temperature can be verified.

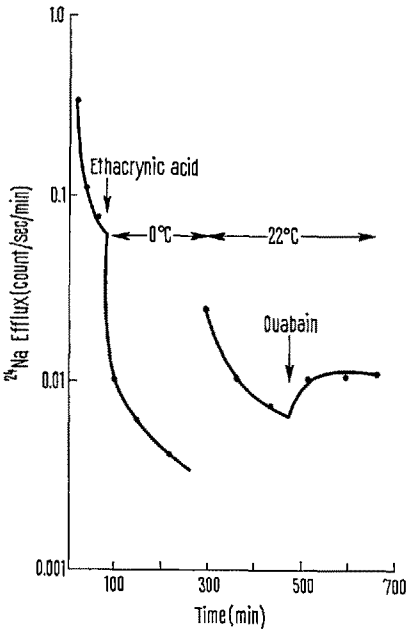

Fig. 4. The stimulating effect of $10^{-4}M$ ouabain on the residual efflux following rewarming of a chilled oocyte poisoned with ethacrynic acid. Second vertical arrow indicates time at which ouabain was applied.

A puzzling feature of the experiments with ouabain is that the inhibitor stimulates the residual efflux from oocytes poisoned with ethacrynic acid before rewarming. One may speculate that ouabain like oligomycin[8] has the power to mobilize the sequestered fraction of Na in the oocyte. Evidence favouring such an explanation comes from unpublished experiments showing that the Na efflux into K-free Ringer is frequently stimulated by ethacrynic acid or ouabain. The kinetics of Na efflux under these experimental conditions clearly indicate that the liberation of the sequestered Na caused by ouabain and ethacrynic acid leads to partial saturation of the pump[9].

Zusammenfassung. Die infolge der verzögerten Wirkung von Ethacrynsäure nur näherungsweise erfassbare Hemmung der Abgabe von Na^+ durch Oozyten der Kröte (*Bufo bufo*) lässt sich exakter bestimmen, wenn die chemische mit einer Temperaturbehandlung kombiniert wird.

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[5] N. GRUENER and Y. AVI-DOR, Biochem. J. *100*, 762 (1966).
[6] M. W. SMITH, Biochem. J. *105*, 65 (1967).
[7] H. W. HELDT and M. KLINGENBERG, Europ. J. Biochem. *4*, 1 (1968).
[8] E. E. BITTAR, Life Sciences *9*, 773 (1970).
[9] Acknowledgments. This work was carried out in collaboration with Dr. D. J. FRY and was supported by the Wellcome Trust.
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Effect of Heavy Physical Training on the Catecholamine Content of the Heart and Adrenals of the Guinea-Pig

In recent studies[1,2], a significant reduction in heart catecholamines after prolonged physical training has been described. In these studies a comparatively low training load was used, furthermore the animals were exercised only 3 times a week. In the present study the effect of a comparatively intense daily training program on the catecholamine contents of the heart and the adrenals was studied.

Materials and methods. Young male guinea-pigs were used. The animals were trained on a treadmill with gradually increased intensity throughout 5 months. The initial training consisted of running for 60 min at a speed of 22 m/min. In the last month, the animals had to run for 80 min at a speed of 37 m/min and a subsequent period of 15 min at a speed of 57 m/min. After 4 days rest, the animals were sacrificed by a blow on the head. The heart and the adrenals were taken out, cleaned and weighed. The noradrenaline content of the heart was extracted, as previously described[3], and determined according to CHANG[4]. The adrenal catecholamines were extracted[5] and determined according to EULER and LISHAJKO[6]. The catecholamine values are given as μg free base.

Results. The results are shown in the Table. The trained animals show a significant increase in heart ratio.

[1] C. DE SCHRYVER, P. DE HERDT and J. LAMMERANT, Nature, Lond. *214*, 907 (1967).
[2] C. DE SCHRYVER, J. MERTENS-STRYTHAGEN, I. BECSEI and J. LAMMERANT, Am. J. Physiol. *217*, 1589 (1969).
[3] N. O. SJÖSTRAND and G. SWEDIN, Acta physiol. scand. *72*, 370 (1968).
[4] C. C. CHANG, Int. J. Neuropharmac. *3*, 643 (1964).
[5] T. BRUNDIN, Acta physiol. scand., *70*, Suppl. 290 (1966).
[6] U. S. VON EULER and F. LISHAJKO, Acta physiol. scand. *58*, 1 (1963).

The total amount of noradrenaline in the heart, as well as the concentration in the heart, shows a probably significant increase in the trained guinea-pigs. In the other measured parameters, no overt differences are seen.

Effect of chronic physical training on the catecholamine content of the heart and the adrenal glands of the guinea-pig

	Controls (n = 14)	Trained animals (n = 13)
Initial body weight ± S.E.M. (g)	271 ± 5	274 ± 4
Final body weight ± S.E.M. (g)	846 ± 18	808 ± 16
Heart weight ± S.E.M. (g)	2.29 ± 0.07	2.39 ± 0.04
Heart ratio ± S.E.M. (g/100 g body weight)	0.27 ± 0.005	0.30 ± 0.003[a]
Adrenal weight ± S.E.M. (paired organs) (g)	0.46 ± 0.02	0.44 ± 0.03
Total noradrenaline in heart ± S.E.M. (µg)	3.42 ± 0.32	4.26 ± 0.17[b]
Noradrenaline concentration in heart ± S.E.M. (µg/g)	1.48 ± 0.12	1.78 ± 0.06[b]
Total adrenaline in adrenals ± S.E.M. (µg)	82 ± 5	88 ± 5
Adrenaline µg/kg body weight ± S.E.M.	100 ± 7	110 ± 7

[a] Different from controls $P < 0.001$. [b] Different from controls $P < 0.05$. n, number of animals.

Discussion and conclusions. From the present study it is evident that a prolonged physical training, giving rise to a cardiac hypertrophy as indicated by the increase in heart ratio, does not lower the cardiac noradrenaline content. On the contrary, a slight increase is seen. In the studies in which a decrease in amount of sympathetic transmitter of the heart was noticed[1,2], no cardiac hypertrophy was observed. This would indicate that a low degree of physical training, not inducing cardiac hypertrophy, has the opposite effect on the amount of sympathetic transmitter in the heart to a physical training giving rise to cardiac enlargement. The reason of this discrepancy, as well as its functional significance, is obscure and requires further investigation.

Zusammenfassung. Ein langdauerndes Training bewirkt keine Herabsetzung der Catecholamine in Herz und Nebennieren des Meerschweinchens.

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Topographical Investigation of Cortical Afferents to the Red Nucleus in the Cat

Anatomical and physiological studies have shown that the large cells of the red nucleus (RN) receive a topographically organized projection from the motor cortex[1–4]. In this electrophysiological study we have attempted to examine in more detail the characteristics of the corticorubral relation at the unitary level.

Techniques. Results were obtained from 41 cats anesthetized with Chloralose and paralyzed with gallamine triethodide (Flaxedil). Recordings from mesencephalic neurons were made with tungsten microelectrodes that had an impedance equal to or greater than 7 mΩ at 1.000 Hz. The rubrospinal tract was stimulated at a frequency of 0.15 Hz at both the cervical (C2) and thoracic (D9) levels to permit antidromic identification of RN cells. A discrete coagulation was made at the end of each microelectrode descent in order to localize the tip in histological sections.

The effect of stimulation of the pericruciate cortex was tested on cells yielding antidromic responses to stimulation of the rubrospinal tract. Cortical stimulation was achieved by means of 7 pairs of nickle needle electrodes that were varnished except at the tip. Each electrode was lowered through a small burr hole in the skull to a cortical depth of 1.5 mm before being cemented into place. Cortical stimulation, consisting of 3 shocks, was delivered at a constant current of 300 µA to each of the electrode pairs, with each member of the pair serving alternatively as the cathode. In this study, only spikes evoked by stimulation intensities of 150 µA or less and with latencies of 20 msec or less have been included. From a total of 173 antidromically activated cells only 72 achieved these criteria.

Results. The cortical areas explored by the stimulation were the anterior sigmoid gyrus, the anterior portion of the posterior sigmoid gyrus and the gyrus proreus (Figure 1, left). The cortical regions from which it was possible to induce spikes in red nucleus units are shown in Figure 1 right. The points are found in the pre- and post-cruciate cortex of area 4 (according to HASSLER and MÜHS-CLEMENT's division of cortical areas[5]). Stimulation of area 6 failed to evoke spikes in the red nucleus, which is in agreement with SCHMIEDT's evoked potential study of corticorubral projections[6]. The zones of greatest corticorubral projections are, according to WOOLSEY's motor homunculus for the cat[7], the somatomotor areas for the forelimb (i.e. the lateral portion of the anterior and posterior sigmoid gyri) and the hindlimb (i.e. the medial portion of the posterior sigmoid gyrus).

[1] O. POMPEIANO and A. BRODAL, J. comp. Neurol. *108*, 225 (1957).
[2] E. RINVIK and F. WALBERG, J. comp. Neurol. *120*, 393 (1963).
[3] M. MABUCHI and T. KUSAMA, Brain Res. *2*, 254 (1966).
[4] N. TSUKAHARA and K. KOSAKA, Expl Brain Res. *5*, 102 (1968).
[5] R. HASSLER and K. MÜHS-CLEMENT, J. Hirnforsch. *6*, 377 (1964).
[6] A. SCHMIEDT, Thèse 3e cycle. Fac. des Sciences. Orsay (1970).
[7] C. N. WOOLSEY, in *Biological and Biochemical Basis of Behaviour* (Eds. H. F. HARLOW and C. N. WOOLSEY; The University of Wisconsin Press, Madison 1958), p. 63.