Discussion. The data indicate that there are significant changes in arterial responsiveness to angiotensin II during the early neonatal period in lambs, just as has been found with α-adrenergic agents (KNIGHT and MCGREGOR[4]; DE CHAMPLAIN et al.[5]; GRAY[6]). The smooth muscle cells become more sensitive to this polypeptide molecule during early maturation, although at the end of the end of the 3 week period under study, the vessels are still less sensitive than comparable vessels from the adult animal and they are capable of developing only about $^1/_3$ the maximal tension. In contrast, norepinephrine sensitivity changes drastically during the 3 week period but does not change very much after that, even though there is still a large change in tension occurring after that period. In the Figure there is a progressive change in slope (except for group II which showed a large change in slope) of the curves depicting the relationship between threshold, ED_{50}, and tension. This is an indication that the threshold concentration (which stimulates the most responsive cells in the population) changes at a slightly different rate than the ED_{50} (which is related to the reactivity of the majority of cells) during the process of maturation, even though both are decreasing.

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The Effect of Intraventricular Thyroxine Administration on Body Temperature in Dogs at Rest and During Physical Exercise

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Summary. Infusion of 1 μg thyroxine into the left cerebral ventricle of the dog did not change body temperature at rest, but it caused significantly higher increases in T_{re} during physical exercise.

It has been found in this laboratory[1] that in dogs with excess of thyroid hormones temperature, responses to exercise are much higher than in control animals. It was also demonstrated that the thyroid-hormone-induced exercise hyperthermia cannot be attributed exclusively to the metabolic heat production[2]. Another possible explanation of this phenomenon is that thyroid hormones exert their action on central nervous structures involved in temperature regulation. An effect of thyroxine on the thermoregulatory centres has recently been described in the cat[3].

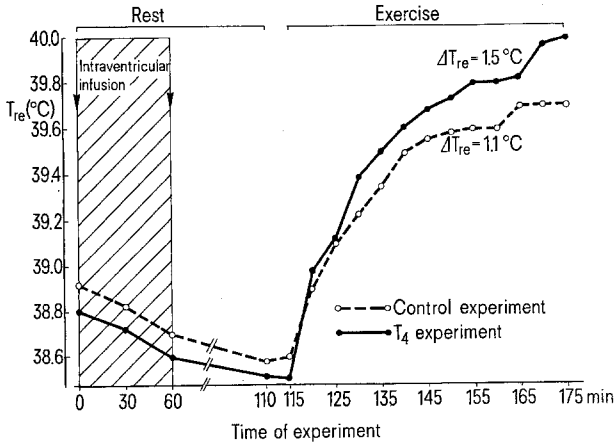
The purpose of the present work was to follow up the effect of intraventricular infusion of thyroxine on deep body temperature in dogs at rest and during physical exercise.

Material and methods. 6 mongrel dogs (18–22 kg b.wt.) were used. They were deprived of food for 18–20 h before the experiments, but had free access to water. At least 2 weeks before the experiments started each dog had a stainless steel guide cannula implanted into the left lateral ventricle. The operation was performed under Na-heksobarbital anaesthesia (45 mg/kg i.v.).

Control experiments. Dogs trained to stand quietly on a stand were infused intraventricularly with the artificial cerebrospinal fluid (ACSF) at a rate of 40 μl/min for 60 min. After the end of the infusion, the dogs remained on the stand for a further 50 min. Then, they were transferred to an electrical treadmill, where they began a 1 h physical exercise (slope of treadmill of 12°, speed 1.2 m/sec). A thermocouple (Ellab, Copenhagen) was inserted 12 to 15 cm deep into the rectum. Rectal temperature was read every 2 min throughout the experiment, and at 5 min intervals after termination of exercise until T_{re} returned to the pre-exercise value.

Experiments with thyroxine were performed on the same dogs according to the scheme described above, but thyroxine (Light and Co, England) in a total amount of 1 μg/dog was added to the ACSF infused.

In both series of experiments, venous blood samples were taken at the beginning of infusion, immediately before exercise, and after its termination, for hematocrit and plasma free fatty acid (FFA) level determinations[4,5]. The data were analyzed with the Student's *t*-test for paired samples.



Effect of intraventricular infusion of 1 μg thyroxine on rectal temperature (T_{re}) of a dog at rest and during 1 h physical exercise.

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Results. Results of a typical experiment are presented in the Figure. Thyroxine (T_4) added to the ACSF infused did not influence significantly T_{re} at rest. Both in the control and T_4-experiments, T_{re} declined by approx. 0.3 °C. At the beginning of exercise, T_{re} of the control dogs was 38.1 ± 0.2 °C (SE), and in the dogs infused with ACSF + T_4 38.05 ± 0.2 °C. At the end of the control exercise, T_{re} was 39.2 ± 0.1 °C while in the T_4 treated dogs it reached 39.7 ± 0.1 °C.

From the 6th min of exercise, T_{re} increases (ΔT_{re}) were significantly higher ($p < 0.05$ or $p < 0.01$) in the dogs treated with T_4 than in the same dogs infused with ACSF alone.

At the end of exercise, the mean difference in ΔT_{re} between the control and T_4 treated dogs amounted to 0.47 ± 0.07 °C ($p < 0.002$). During the run, Hct changed only slightly ($p > 0.05$) both in the control and T_4 treated dogs, and plasma FFA concentration increased by 142.7 ± 22.4 µEq/l and 144.7 ± 23.3 µEq/l respectively ($p > 0.05$).

Discussion. Contrary to the results obtained in cats[3], the data presented here indicate that in dogs infusion of 1 µg thyroxine into the lateral ventricle of the brain does not affect body temperature at rest. It does, however, cause significantly higher exercise-induced increases in rectal temperature in comparison with control runs performed by the same dogs following infusion of ACSF alone. This finding supports the previously made hypothesis that the exercise-hyperthermia described in the dogs injected s.c. with a single large dose of thyroxine or triiodothyronine (T_3)[1] may be partly due to the central action of thyroid hormones on the structures involved in thermoregulation. It has been shown recently[6] that T_3 and T_4 given by i.v. infusion to dogs penetrate both brain and CSF.

The mechanism of the hyperthermic action of intraventricularly administered T_4 during exercise is difficult to explain. In this case, peripheral calorigenic effects of thyroxine, being mostly due to potentiation of metabolic action of catecholamines[2], can be excluded, since no difference in blood FFA level at the end of exercise was found between the control and T_4 treated dogs.

It may be supposed that T_4, applied in a small dose into the brain ventricles, either acts directly on the activity of central neurons involved in thermoregulation, or it exerts its action by changing sensitivity to neurotransmitters[7] during physical exercise.

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How Gill Surface of *Saccobranchus fossilis* Facilitates Active Gas Exchange?

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Summary. The surface morphology of fish gill has been studied by scanning electron microscopy. The surface of gill epithelium shows a pseudoconcentric arrangement of arborizing ridges and channels. Further, at places on gill filaments the surface shows several infoldings labelled as micropits. The role of these morphological adaptations has been correlated with the gas exchange physiology.

There is no means other than diffusion by which the respiratory gases are carried across the surfaces separating water from the blood. In fish gills, the secondary lamellae of the gill filaments are the main centres of gas exchange. The fine structure of secondary lamellae shows that these are mainly composed of a pair of two-layered epithelial sheets (each about 3 µm thick) joined together by pillar cells[2] (Figure 1). The flanges of these pillar cells line most of the channels through which blood flows in the secondary lamellae; and due to this pillar cells come in a very close contact with the whole blood during circulation. The pillar cells thus form an important part of respiratory

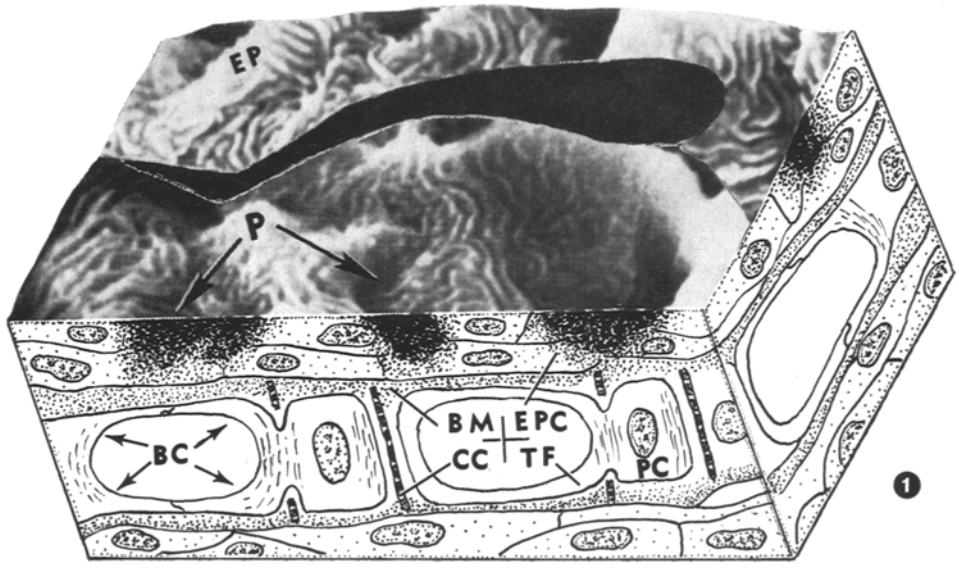

Fig. 1. Semi-diagrammatic representation of the structure of secondary gill lamella from a gill filament showing surface and sectional views. Note the engravings on the surface of the epithelium (EP); micropits (P) show their depth in sectional view (dark areas); BC, blood channel; BM, basement membrane; CC, collagen column; EP, epithelium; EPC, epithelial cell; PC, pillar cell; TF, thin flange of pillar cell. (modified after BETTEX-GALLAND and HUGHES[2]).