species[11–13], none have been made of the tissues near joints. It was therefore necessary to study some quantitative aspects of the capillaries of tissues near the human knee, in order to provide the data necessary for the model for understanding and predicting the occurrence of bends[9].

Specimens were obtained from 5 healthy young men, who had to have menisectomies due to recent sports-injuries. The tissues studied were: the deeper layers of the synovial membrane, the synovial capsule, fat, and tendon. They were processed by the normal methods[5,6] used for quantitative stereological electron microscopical morphology of capillaries. (These include attention to the colloidal osmotic pressure of the fixative and the standardization of the magnifications.) 4 separate, random blocks were taken from each piece of tissue and only 1 random section of each examined. Separated, random micrographs were made of each section with total mean areas of 3190 (SE 81.5) μm^2.

The results are shown in the Table and illustrated in the Figures. Poissonian distributions were used to determine the standard errors. The surface areas were calculated using the mean measured luminal circumference $\times$the lengths, adjusted as described elsewhere[5,6]. The intercapillary distances[5,6] were estimated using the square root of the reciprocal of the mean numbers of vessels per cm^2.

It can be seen that the numbers of capillaries/cm^2, and hence their lengths ($2\times$this[2–4]), are very much less than those found[11] in the human ventricle ($\sim 5\times10^5/cm^2$), in the muscles of various species[11–13] ($1–5\times10^5/cm^2$), or even in the fat of rats[11] ($2–10\times10^4$). While it must be remembered that in the muscles listed[11–13] it is likely that the sections were transverse to the fibres[5] so that the estimated lengths per cm^3 would be nearer to $1\times$the values for capillaries/cm^2, rather than $2\times$this as in random sections[2–4], nevertheless it is evident that the lengths, and the associated capillary surface areas are much less in the present tissues than in the muscles, or indeed in the other regions of the body of a number of species[11]. It is unfortunate that we do not have human skeletal muscle to compare with these findings – since the larger the animal, the less its vascularity[11–13]. Still it is very evident that the tissues we have examined are much less vascularized than muscles and other metabolically active regions. (It should be noted that we have here examined only the deeper layers of the synovial capsule; this is the part most relevant to diving, since air in the superficial layers, which are likely to have more vessels, could easily pass into the joint space).

The intercapillary distance is only approximately estimated by our method[5,6], but a more exact one does not yet seem to be available. It is evident that the distances found here are some 3–10 times greater than those which can be derived from the values for the other sites, quoted earlier. Also the distribution is very wide so that an appreciable proportion of the vessels must be very widely separated indeed. This must have a very great effect on the slow removal of gas from the tissues around the knee, and hence indicates why this region is more prone to develop the bubbles which cause 'the bends' during decompression. It is hoped that the present values, plus those for the capillary surface area, will be used in a model of this disease[9].

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Dog Behaviour as Related to Spinal Cord Temperature

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Summary. 3 dogs could behaviourally modify their own spinal cord temperature ($T_{spin.\ cord}$). In a hot environment, 2 dogs did not cool their spinal cord, 1 dog warmed it. The higher the environmental temperature, the higher the chosen $T_{spin.\ cord}$. These results seem to imply that this latter dog tended, in warm environment, to behaviourally reduce: $T_s > T_{spin.\ cord}$ (T_s mean skin temperature). Data obtained previously support this explanation.

CORBIT[2] has shown that direct thermal intracranial self-stimulation was possible in rats. Rats placed in a warm environment were able to cool their brains by self-stimulation. We considered that it would be interesting to offer the possibility of thermal spinal cord self-stimulation to animals, because: 1. Spinal thermal sensitivity has been shown to be of the same magnitude and efficiency as the hypothalamus in temperature regulation[3,4]. 2. Heating of the spinal cord was shown to be followed by an adequate corrective behavioural response in frogs[5] as well as in dogs[6]. Cooling of the spinal cord was followed by a corrective operant response in pigs[7] and by an adapted posture in pigeons[8].

Methods and results. We used 3 dogs which were previously trained to interrupt a light beam in order to obtain environmental infra-red heat or cool air. Each of the 3 dogs was chronically implanted with a U-shaped spinal thermode made of PE tubing (external diameter: 1.5 mm, internal diameter: 1 mm), through which water was circulated. This thermode was implanted in the epidural space, under general anesthesia, from C_2 down to the caudal end of the vertebral canal. These dogs had been implanted and trained for another experiment, the results of which have been published[6]. The technique consisted in an attempt to transfer the operant behaviour heat or cold reward from the skin to the spinal cord. A

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Thermode self-control allowed	Dog	Trials	T_a (°C)	T_{th} perfused (°C) ± SE	T_{th} chosen (°C) ± SE
Raising of T_{th}	Buf	6	0–25	36.0 ± 1.2	38.6 ± 0.5
(warming perfused water)		6	25–60	36.0 ± 1.6	40.4 ± 2.2
Lowering of T_{th}	Kis	4	25–60	37.0 ± 1.1	Extinction of the behaviour
(cooling perfused water)		12	25–60	40.7 ± 1.4	
	Kad	3	25–60	38.5 ± 0.2	

solenoid resistor was placed around the afferent portion of the thermode at the entrance into the dogs' bodies. Warming of the spinal cord was obtained when the dog interrupted the light beam with its snout, thus turning on the solenoid. The temperature of the water entering the thermode (T_{th}) could increase by up to 6 °C in less than 1 sec. Cooling of the spinal cord was obtained by permanently heating the inflowing water, the dog's behaviour determining when to turn off the solenoid. The inflowing water would, thus, drop by as much as 6 °C in less than 1 sec.

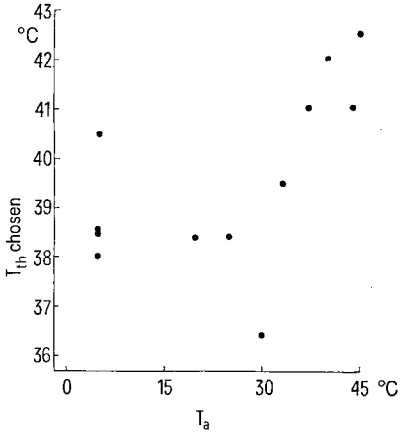

Fig. 1. Spinal thermode temperature chosen by dog Buf in various environmental temperatures.

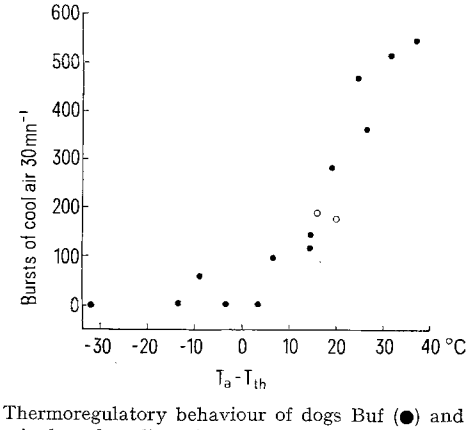

Fig. 2. Thermoregulatory behaviour of dogs Buf (●) and Ami (○) during spinal cord cooling. Ordinate: number of bursts of cool air triggered by each dog during a 30 min period. Abscissa: ambient temperature (T_a) minus spinal temperature (T_{th}). When [T_a — T_{th}] < 0, the relationship with behaviour is linear.

Each behavioural transfer trial lasted 20 min and was preceded and followed by a 20 min period, during which the dog's behaviour was rewarded by skin heating or cooling. The alternation of these periods was necessary to prevent extinction of the behaviour.

Dog Kad and Kis had their spinal cord thermode perfused by water at a cool (35.5 °C < T_{th} < 38.0 °C), a neutral (38.5 °C) or a warm (38.5 °C < T_{th} < 42.5 °C) temperature. They were allowed to cool their spinal cords by their behavioural adjustment during 19 trials in a warm or hot environment (25 °C < T_a < 60 °C).

Dog Buf had its spinal cord thermode perfused by water at a cool temperature (35 °C < T_{th} < 37.5 °C). It was allowed to warm its spinal cord by behavioural response during 6 trials in a cold environment (0 °C < T_a < 25 °C) and 6 trials in a warm or hot environment (25 °C < T_a < 60 °C).

The results shown in the Table represent thermode temperatures resulting from each dog's behaviour.

Discussion. Contrary to what was expected, dog Kis and Kad, when placed in a warm environment did not use the thermode to cool their spinal cords. This can be explained by two possibilities: either the dogs had no temperature sensation in the spinal cord, or they had a sensation but did not find it rewarding. We would consider the latter to be correct. The operant response was inhibited after each trial, because it was necessary to recondition each dog between trials. Only dog Buf used operant behaviour to prevent cooling of its spinal cord or to warm it: in a cool environment, its behaviour was sufficient to maintain T_{th} around 38.5 °C; in a warm environment, the higher the environmental temperature, the higher the chosen thermode temperature. This paradoxal result is not shown in the Table, where results are averaged, but becomes apparent in Figure 1, where thermode temperature (behaviourally selected by the dog) is plotted against environmental temperature. The result imply that the dog had tried, by means of its behaviour, to reduce the difference between its ambient and spinal cord temperatures (T_a – $T_{spin.\ cord}$), when its spinal cord thermode was being perfused with cool water. The signal for thermoregulatory behaviour would, therefore, not be absolute temperature, but rather a temperature gradient. These results led us to re-examine the data obtained previously by measurement of behaviour during spinal cord cooling[6]. In this previous experiment, the dogs were able to obtain external heat or cold by means of their behaviour. Spinal cord cooling, in this previous experiment (although accompanied by skin vasoconstriction) did not result in increased physical behaviour for infra-red skin heat but rather in unexpected skin cooling-motivated behaviour.

We had therefore concluded that the spinal cord did not possess a cold sensitivity capable of triggering a

corrective thermoregulatory behaviour. In view of the present result, it may be hypothesized that the signal for behaviour was the difference: $T_a - T_{spin.\ cord}$. One would then expect the dog behaviourally to reduce its ambient temperature, when its spinal temperature was maintained at a low value.

Such was the case in the previous experiment; Figure 2 summarizes the results then obtained for dogs Buf and Ami. The behavioural response was measured in terms of the number of fan coolings requested by the dogs during the 30 min trial period. When ambient temperature in the climatic chamber (T_a) was higher than spinal thermode temperature (T_{th}), the dogs' behaviour was proportional to $T_a - T_{th}$. This was true for all values of T_{th} and T_a. Here again, the dogs' behaviour may seem paradoxal, because with a cool spinal cord (20 °C), dog Buf behaved in such a way as to reduce its skin temperature. Two completely different circumstances: thermal spinal cord self-stimulation and environmental temperature adjustment seemed, therefore, to be motivated by the same signal, $T_s - T_{th}$. The dogs where working to reduce this signal, when $T_a - T_{th} > 0$ assuming that $T_s = f(T_a)$.

Role of Catecholamines in Thyroxine-Induced Changes in Metabolism and Body Temperature During Exercise in Dogs

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Summary. Blockade of beta receptors inhibited thyroxine-induced increases in T_{re}, blood FFA and LA levels during exercise in dogs.

Pretreatment with thyroid hormones markedly increases plasma free fatty acid (FFA) levels and body temperature during exercise[2–4]. A suggestion was made that this increase in the plasma FFA concentration is mostly caused by potentiation by thyroid hormones of the lipolytic action of catecholamines, which are released in increasing amounts during exercise in dogs[5].

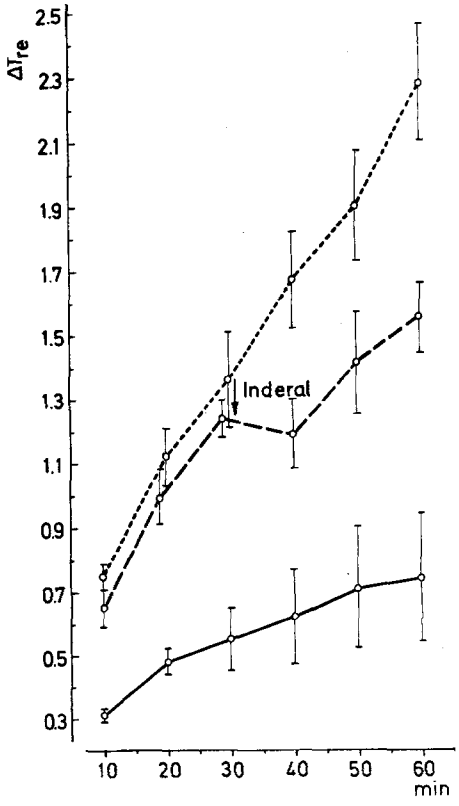

Fig. 1. Changes in rectal temperature (ΔT_{re}) during 60 min exercise. ○—○, control exercise; ○—○, exercise performed 72 h following T_4 + Inderal given at 30 min of the run; ○- - -○, exercise performed 72 h following T_4 without Inderal.

The thyroid hormone-induced exercise hyperthermia was supposed to be at least partly due to a greater heat production during exercise, since an increased FFA mobilization usually results in increased calorigenic action. An enhancement in anaerobic metabolism might also have had a part in this mechanism.

In the present study, an attempt was made to ascertain the role of catecholamines in the thyroxine-induced changes in metabolism and temperature during exercise. For this purpose propanolol, which inhibits the metabolic action of catecholamines, was administered during the exercise performed by thyroxine-treated dogs.

Material and methods. Experiments were carried out on 6 male, mongrel dogs weighing 18–25 kg. Before each experiment the dogs were deprived of food for 18–20 h, but had free access to water. At first, the dogs performed a 60 min control run on a treadmill. The slope of the treadmill was 12° and its speed ranged from 1.2–1.6 m/sec, according to the individual capacity of each animal. Then, after at least 1 week interval, the dogs were injected s.c. with L-thyroxine (Light and Co., England) in a single dose of 100 µg/kg body weight. 3 days after the injection the animals performed treadmill exercise of the same intensity and duration as in the control experiments. At 30 min of the exercise propranolol (Inderal, ICI) was given i.v. in a dose 0.25 mg/kg body weight, and after 10 min rest the run was continued for a further 30 min. In the same dogs thyroxine (T_4) injection was repeated after at least a 1-month interval, and the dogs performed an exercise run 3 days following the injection. In these additional experiments only T_{re} measurements were

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