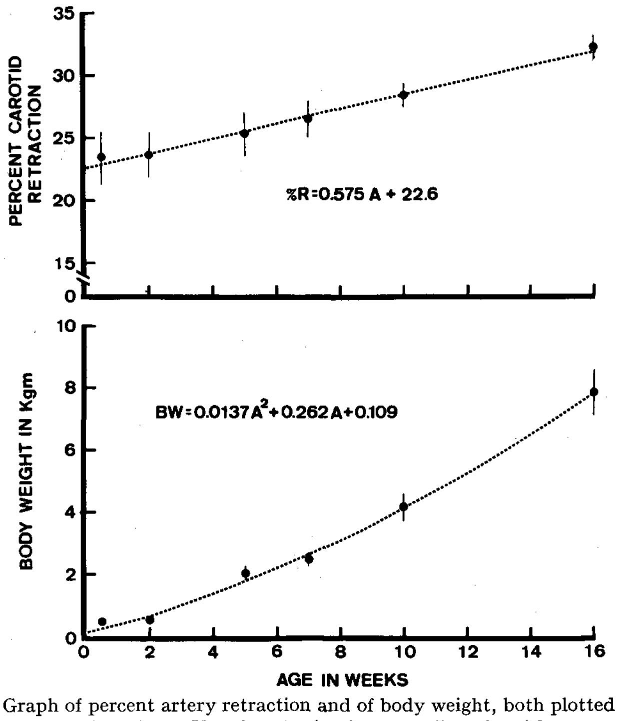
chronological age. The circles indicate means, and the vertical bars indicate standard errors. The numbers over each point are the number of vessels examined at that age. The upper graph indicates a remarkably linear increase in vessel retraction with age, from 23.4 ± 2.2% in the 1-week-old animals to 32.2 ± 0.9% in the 16-wek-old animals. These data were described by the linear equation shown, and predict a 22.6% artery retraction at the time of birth.

Body weight was used as an index of overall body growth. The lower curve in the Figure indicates that the animals body weight increased nonlinearly during the same developmental period. These data were fitted with the quadratic equation shown. The increase in body weight during this period implies that the unstretched carotid artery, as most other body tissues, probably grew in length. However, the increasing magnitude of retraction observed when the vessels were transected indicates that the vessels were stretched even more than they had grown.

Discussion. The present data show that longitudinal retraction develops as arteries gradually are extended during growth. A second determinant of vessel retraction may be developmental changes in the connective tissue composition of the vessel walls. Studies in various species report that during late fetal development artery walls thicken and exhibit increased medial collagen[9]. In the neonatal period there is further deposition of fibrous proteins in the subendothelium[10] and in the media[11,12]. During this period the ratio of collagen to elastin in the artery wall increases[13], and this correlates with increasing circumferential and longitudinal wall stiffness[13]. Thus, both increased stretch and increased collagen content may play a role in the development of longitudinal retraction. However, with aging in adults, there is still further deposition of collagen in the vessel wall[14-18], but this is associated with *decreased* longitudinal retraction[6-8]. The decline in retraction which occurs with age actually may result from the continued accumulation of collagen; fibres deposited after vessels have achieved a stable length in the mature animal resist both retraction and extension. In addition, the development of stiff athero-sclerotic plaques also tends to fix the vessels at their mature length. Both of these processes result in decreased longitudinal retraction. Decreased retraction has clinical importance, for the length of these arteries is maintained by the rigidity of their walls instead of by the length-stabilizing interaction of traction and arterial pressure. One might expect these vessels to buckle or bend with pressurization, and indeed, severe tortuosity of carotid and innominate arteries in aged patients has been described[19-21].



Graph of percent artery retraction and of body weight, both plotted as a function of age. Vessel retraction increases linearly with age.

Summary. Longitudinal retraction of carotid arteries, was examined in 105 neonatal puppies as a measure of longitudinal traction. Percent vessel retraction increased linearly with age. This was attributed to stretching of the vessels by growth and to changes in connective tissue composition. The mechanical significance of artery retraction was discussed.

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Seasonal Changes in the Circadian Variation of Oral Temperature During Wakefulness

The circadian variation of body temperature is an entrained endogenous rhythm which in the total absence of time cues (Zeitgebers) is of approximately 24 h periodicity (circadian). With man, a visually oriented animal, an important Zeitgeber is light and darkness[1-4]. In view of the seasonal changes in length of natural daylight the circadian rhythm of body temperature may also show seasonal changes, particularly with peak times. The only previous study of this type was of Eskimos[5] where a seasonal change in peak time of about $2^1/_2$ h was found. The extreme conditions of the Arctic preclude comparisons with people from lower latitudes. The present study investigated whether the circadian rhythm of oral temperature in European subjects would show any similar seasonal changes. Sampling was oriented towards times of the year when daylight length was 1. minimum

(December), 2. maximum (June), 3. midway between 1 and 2 (March).

Method. 12 male and 14 female subjects, 18–24 years of age, measured their oral temperature with calibrated mercury-in-glass thermometers. Each of the 3 seasonal sample periods lasted 3 weeks. For each day throughout every period oral temperatures were taken at approximately half hourly intervals commencing immediately upon awakening and ending at bedtime. Measurements were not taken during sleep. A high daily sample rate over many days was necessary to average out: a) error of fine measurement inherent with mercury-in-glass thermometers, b) daily individual fluctuations within the trend of oral temperature. Subjects retained their own thermometer. They were carefully trained in reading thermometer scales and were instructed to place the thermometer well under the tongue with mouth shut for 5 min. Measurements were not taken 1. during eating, drinking, smoking and changing environment, and within 20 min of completing these activities, 2. during exercise, and within 2 h of completing exercise. Subjects were not restricted in their normal daily activities, and kept diaries of sleeping and eating times.

At the end of each period, an averaged waking oral temperature change was compiled for each subject by dividing up the waking day into $^1/_4$ h epochs, making about 60 epochs in all, and averaging all measurements taken over the 3 week period which fell into each epoch. These epochs each contained about 4 readings.

A polynomial curve fitting programme (BMDO5R, 1966-Health Sciences Computing Facility, U.C.L.A.) using the sextic term smoothed each average curve. Peak times and mean daily temperatures were computed. A subroutine plotted the best fit straight line for: a) the rise of body temperature from 09.00 h to the peak, b) the decline from the peak to 23.00 h.

In Britain the clocks are advanced 1 h in the Spring. The December and March periods were performed during Greenwich Mean Time (GMT), whilst the June period was within British Summer Time (BST). All temperatures were recorded (and are presently displayed) at the clock time in force during each period.

Results. From Figure 1 results show that the peak time was significantly later, by about 70 min, in March compared with December, for men only. Both sexes displayed significantly later peak times, averaging 55 min in June compared with December. In Figure 2 the significantly higher mean daily temperatures in women compared with men can be seen for March and June, but there were no significant inter-month differences within sexes. Figure 3

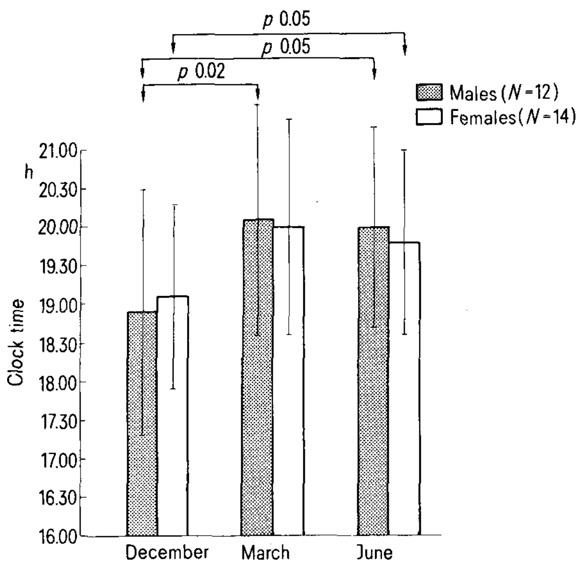

Fig. 1. Means and standard deviations of body temperature peak times, with significant differences.

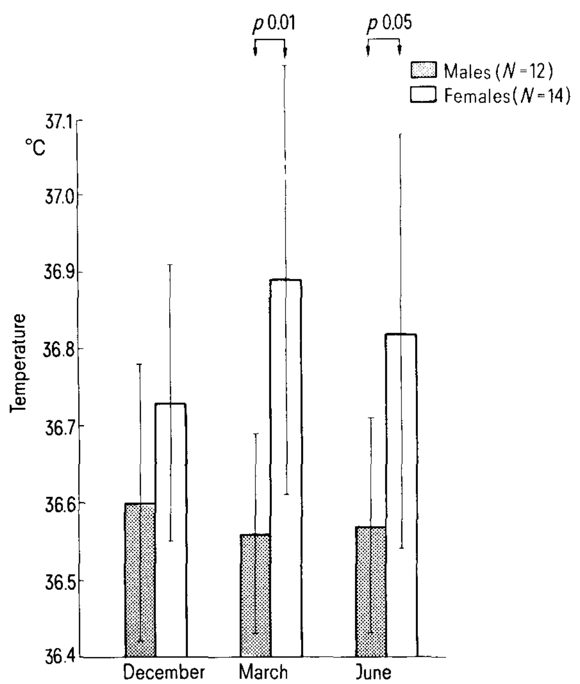

Fig. 2. Means and standard deviations of average waking temperatures, with significant differences.

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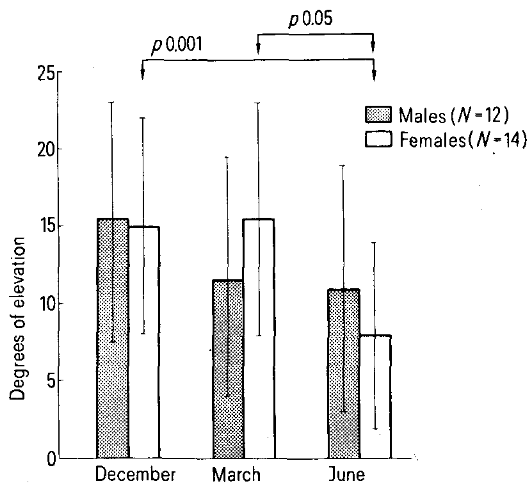

Fig. 3. Means and standard deviations of temperature rise angles (from waking to peak time), with significant differences.

shows that only for females were there any significant changes of rise angles, with a lower rise angle in June compared with both December and March. Finally, from Figure 4 it can be seen that there are no significant differences between sexes, or between months, for evening fall angles. Analysis of the sleep diaries produced no intra-subject monthly changes in sleeping times.

Discussion. The peak time change from December to June, being similar in size and direction to the clock change from GMT to BST, may have been due to the clock change between these months. However, the significantly later peak time from December to March, in the males, was within GMT; furthermore, there were no significant peak changes from March to June, across the clock change. In a small pilot study, 2 subjects monitored their temperatures during the GMT to BST change.

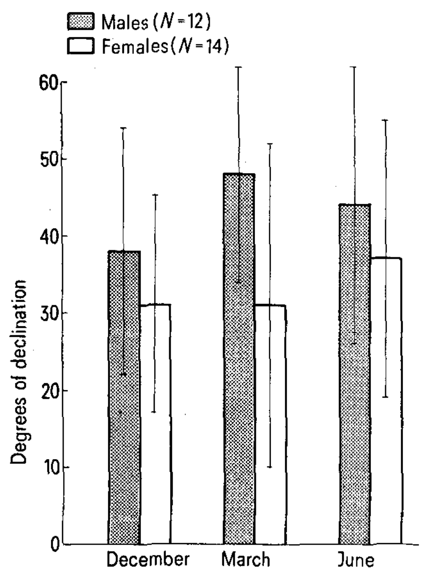

Fig. 4. Means and standard deviations of temperature fall angles (from peak time to 23.00 h).

Although both subjects had peak times in accordance with GMT on the first day of BST, within 2 further days the peak times had reverted to the same time on BST as for GMT previously. Thus it appears that the peak time changes from December to March/June are due to factors other than the clock change. It was found that the times of meals and sleeping in all subjects were remarkably constant against both GMT and BST scales.

The extent of daylight in June is 16 h/day, and for December, 8 h/day. If daylight-night time was an important Zeitgeber for the present subjects, then a larger peak time change between these months might be expected. But, artificial light was freely available to the subjects. Eskimos[5] are not so dependent upon the clock time as Europeans, and place much reliance upon the physical environment, particularly daylight and darkness, for time cues. Probably because of a dependence upon clock time, together with a way of life fairly independent of the physical environment, the present subjects may not be using daylight-night as a Zeitgeber to the same extent as Eskimos. This conclusion is supported by a laboratory study[6] where light and dark were systematically varied, however, subjects showed no substantial changes in circadian rhythms, and it was proposed that social Zeitgebers were sufficient to entrain circadian rhythms.

Summary. The circadian change of oral temperature in 26 subjects was compiled for December, March and June. Average peak time delays up to 70 min, and a reduced daytime temperature rise were found in June compared with December.

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Acute Effect of Hypophysectomy on the Natriuresis Following Saline Infusion in Dogs

It has been shown that after hypophysectomy the homeostatic ability to increase renal excretion of sodium following extracellular fluid volume expansion with isotonic saline in rats[1–3] is impaired. A similar effect was seen in the present experiments on anaesthetized dogs, suggesting that the former finding is not restricted to the rat. Thus, the role of the pituitary in the mechanism of the extracellular fluid volume regulation seems to be confirmed in another species.

Material and methods. 17 dogs of either sex (body wt. 7.5–15.0 kg) were anaesthetized by sodium pentobarbital, 25 mg kg^{-1} body wt. i.v. Both femoral arteries, a femoral vein and the ureters (approached by a suprapubic incision) were cannulated. Subsequently, hypophysectomy was performed by buccal route in 8 dogs, whereas only the appropriate incisions were made in a group of 9 sham operated animals. After completion of surgery, ^{51}Cr-EDTA infusion in isotonic saline was started to measure glomerular filtration rate and following a 20 min equilibration period 2 urine samples of 30 min each were taken. Then approximately 2 h after hypophysectomy, or sham operation, extracellular fluid volume was expanded with 0.9% saline at a rate of 0.5% of the body wt. per min. During two 10 min periods a total amount of 10% of the body wt. was infused. The expansion was followed by another 5 urine samples taken at 10 min intervals. Arterial blood samples were withdrawn at the beginning and at the end of the clearance periods.

The completeness of hypophysectomy was verified at autopsy.

Blood pressure was measured in a femoral artery by a damped mercury manometer, sodium concentration in urine and blood samples was determined by flame photometry, and ^{51}Cr activity of the samples was counted in a Nuclear Chicago gamma spectrometer. The experimental data were calculated for 100 g of kidney weight. Differences

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