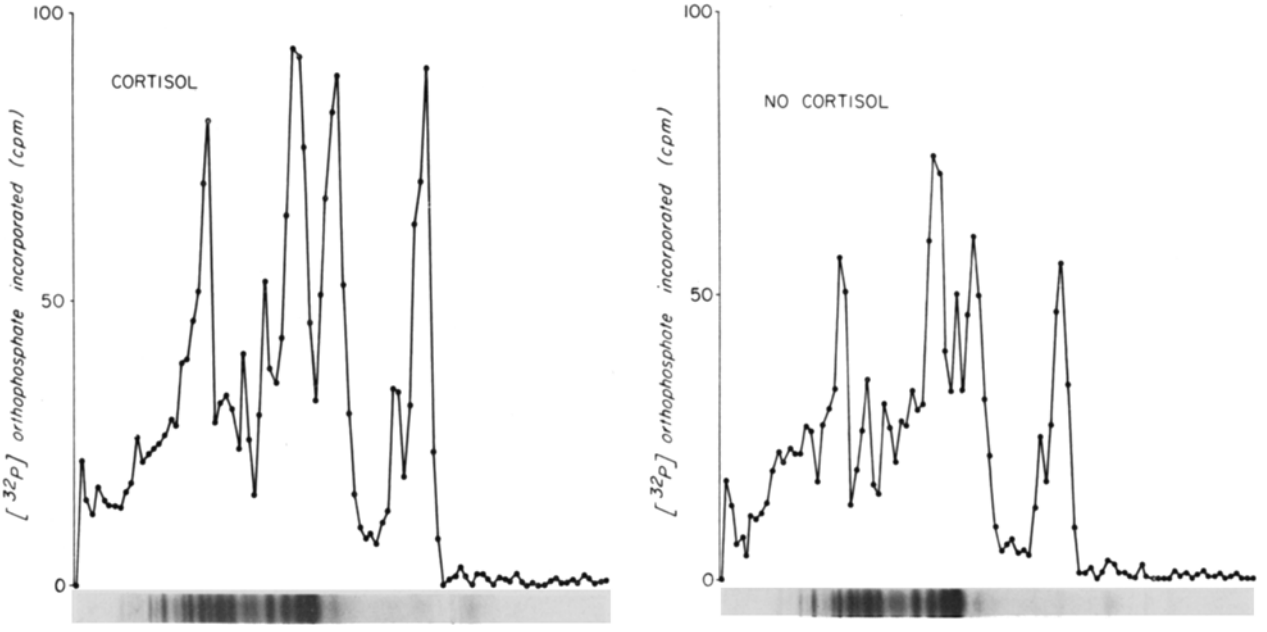

Comparisons of the effect of cortisol, 11 h after administration, on the electrophoretic pattern of phenol-soluble nuclear acidic proteins from rat liver with amounts of ^{32}P incorporated in them. Results with cortisol on the left portion and without cortisol on the right portion. For each electrophoretic analysis radioactivity is plotted as a function of the distance of migration and is aligned with the banding pattern shown immediately below.

via phosphorylation is a likely mechanism by which cortisol might influence genetic control, thereby affecting enzyme induction and liver growth. However, the biological significance of increased phosphorylation of nuclear proteins during times of gene activation is still unclear.

Cortisol did influence, with a lag period of about 7 h, phosphorylation of histones and the bulk of acidic proteins, i.e. preferential phosphorylation of single acidic nuclear proteins did not occur. Therefore, the cortisol-mediated enzyme induction and/or liver growth does not seem to be initiated by the specific phosphorylation of a single acidic nuclear protein. Further investigations are needed for clarification of the role of phosphorylation of nuclear acidic proteins occurring during hormone-induced increase in genetic activity.

Zusammenfassung. Nach Gabe von Cortisol war in adrenalektomierten Ratten ein gesteigerter Einbau von ^{32}P-*Ortho*phosphat in saure Kernproteine und Histone der Leber nachweisbar. Diese gesteigerte Phosphorylierung erfolgte mit einer Verzögerung von mehr als 5 h post injectionem gleichförmig in den verschiedenen sauren Proteinen. Das elektrophoretische Muster (Polyacrylamid) saurer Proteine blieb durch Cortisolgabe unbeeinflusst.

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How Well does Man Thermoregulate During Sleep?

Baker and Hayward have suggested that in active (REM) sleep thermoregulatory and other autonomic control is inhibited[1]. Panting and shivering in cats are indeed suppressed during active sleep[2,3]. In men sleeping at 'comfortable' environmental temperatures, sweating measured over small areas of the hand or chest is depressed or absent during the active phase[4]. If the sweat depression is general over the entire body and if it persists in warm environments then it would seem that active sleep and precise thermoregulation are mutually exclusive phenomena in normal human function.

In order to investigate whether such a conflict exists we exposed a sleeping man to 3 warm environments in a human calorimeter. The calorimeter[5,6] provided accurate control of the thermal environment and allowed direct measurements of sensible (radiant plus convective) and evaporative heat transfer with a precision of better than

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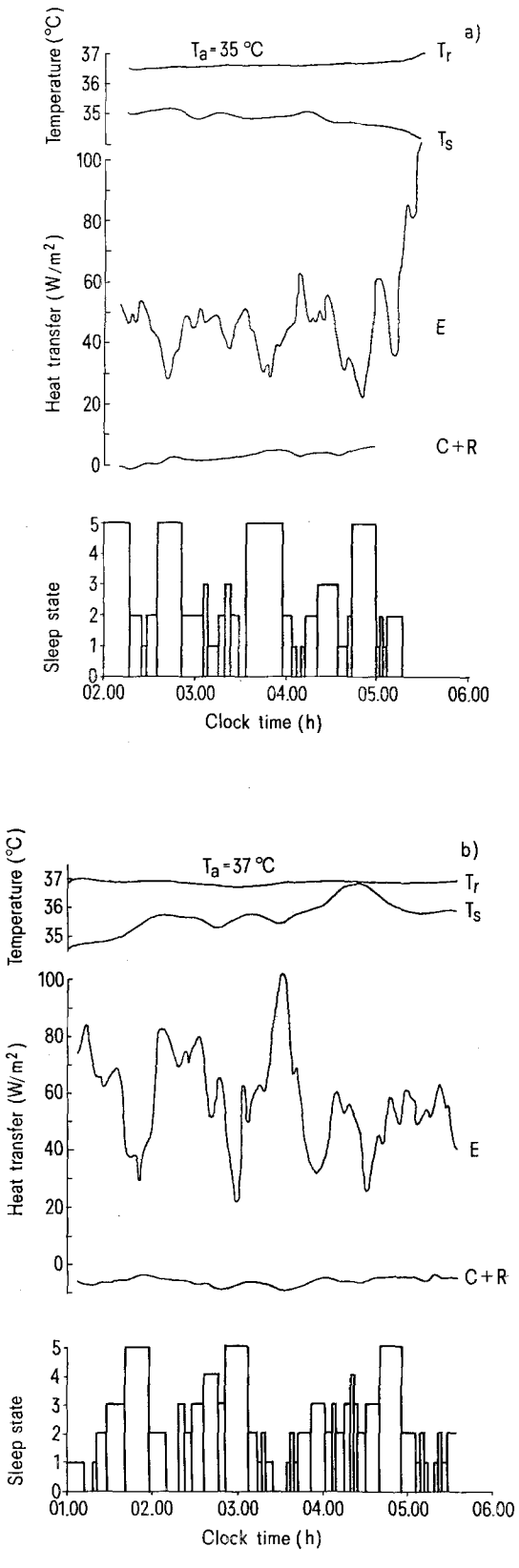

Evaporative (E) and sensible (C + R) heat transfer rate, rectal temperature (T_{re}), skin temperature (T_s), and sleep state (as defined by WILLIAMS et al.[8]) of a man sleeping in environments of 35 °C and 37 °C dry-bulb temperature. Active sleep (stage 5) is associated with depressed whole-body sweating, and wakefulness (stage 0) with augmented sweating.

5% and a response time of less than 30 sec. A 10 W light bulb gave a constant low level of non-glare illumination all night, and a low-pitched low-intensity hum emanated from the calorimeter machinery. An intercom system allowed communication between the subject and observers. Rectal temperature was measured with a flexible indwelling thermocouple probe, and mean skin temperature with 4 fine-wire thermocouples attached to the skin at the sites recommended by RAMANATHAN[7]. The electroencephalogram, oculogram and cardiogram were recorded continuously. The sleep state prevailing in each successive two-minute period was assessed by analysis of the record according to the technique of WILLIAMS, AGNEW and WEBB[8]. Sensible and evaporative heat transfer rates were averaged over the same two-minute periods.

The subject, a healthy 22-year-old volunteer who had participated in experiments in the calorimeter extending over several months, accustomed himself to sleeping in the experimental situation by spending 7 nights at a neutral temperature in the calorimeter with all measuring leads attached. He was then exposed to environments with a low wind speed (0.2 m/sec), constant low dew-point temperature (11.1 ± 0.1 (SD)°C), and 3 different dry-bulb temperatures of 32.9 ± 0.1 °C, 35.0 ± 0.1°C and 36.9 ± 0.1°C. After a meal at 18.30 h the subject rested awake in a neutral air-conditioned antechamber until 23.00 h, his normal retiring time. He then entered the calorimeter and lay, dressed in shorts only, on a plastic mesh bed. No medication was used to induce sleep: the subject went to sleep without incident on all 3 occasions. The transient period of adjustment to the thermal environment and entering sleep was excluded from the results.

The subject claimed to have 'slept well' except at 37 °C, when he was 'hot'. In the 33 °C environment he slept for 352 min during 12% of which the electroencephalogram indicated a state of wakefulness. At 37 °C he slept for 305 min of which 30% was wakeful. The Figure shows the heat transfer, body temperature, and sleep state analyzed over successive two-minute periods on the 2 warmer nights. Sensible heat transfer rate was almost constant throughout each night and varied from a net loss of about 10 W/m² in the 33 °C environment to a net gain of 10 W/m² in the 37 °C environment. Evaporative heat transfer, on the other hand, showed large fluctuations. In the 37 °C environment, for example, the rate of evaporation varied from 20 W/m² to 100 W/m². The Table shows the mean, standard deviation, and coefficient of variability of the total heat transfer rate measured in each environment. Also shown are equivalent measurements made during a series of daytime studies on the same subject. The variability of heat transfer about its mean value during sleep was about four fold larger than during wakefulness.

The fluctuations in evaporation, presumably the result of fluctuations in whole body sweating, were evidently related to sleep state: low evaporation was associated with active sleep and high evaporation with wakefulness during the sleep period. Periods of active sleep therefore were apparently associated with periods of depression of sweating activity over the entire body, and these periods of depression persisted at an ambient temperature of 37 °C.

The fluctuations in evaporation did not produce concomitant variations in rectal temperature nor mean skin temperature. One explanation of this situation would

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Net heat loss to the surroundings during the sleep period and during wakefulness in similar environments

Sleep state	Net heat loss rate	Dry-bulb temperature (°C)		
		33	35	37
Awake	Mean (W/m^2)	46	47	50
	Standard deviation (W/m^2)	2	5	5
	Coefficient of variability (%)	6	9	10
Asleep	Mean (W/m^2)	36	47	59
	Standard deviation (W/m^2)	12	18	21
	Coefficient of variability (%)	33	38	35

be that metabolic rate is depressed during active sleep, and the depressions in evaporation are reactions necessary if thermal balance is to be maintained. However, metabolic rate (as determined by measurements of oxygen consumption) is in fact higher during active sleep than during other stages[9]. A more likely explanation is that in these experiments, as in similar previous experiments with an awake subject[10], the fluctuations in mean body temperature which result from fluctuations in heat transfer simply were not reflected in rectal temperature and mean skin temperature. The poor correlation between mean body temperature measured calorimetrically and the temperature of individual anatomical sites is well known[11,12], and it appears to be mean body temperature with which human thermoregulation is concerned[11,13]. One can conclude therefore that, at least for this subject, thermoregulation is considerably less precise during sleep than during wakefulness even though the imprecision does not result in gross fluctuations of rectal or skin temperature.

If, as it would seem, active sleep and precise thermoregulation are mutually exclusive, sweating being depressed during the active state, then active sleep might disappear entirely in sufficiently warm environments. The ultimate consequences of the conflicting requirements of sleep and thermoregulation under heat stress remain a matter for further experimentation[14].

Zusammenfassung. Eine Versuchsperson, im Calorimeter für Menschen einer warmen Umgebung ausgesetzt, zeigte auffallend grosse Unterschiede in der Schweissrate im Schlaf gegenüber dem Wach-Zustand. Perioden mit unterdrücktem Schwitzen waren mit aktiven (REM) Schlaf verbunden.

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Stimulatory Effect of *Leonurus artemisia* (I-Mu Ts'ao) on the Contraction of Human Myometrium in vitro

Leonurus artemisiae (Lour.) S.Y. Hu is an annual herb of the Labiatae family. As its Chinese trivial name 'i-mu ts'ao'[1] implies, it is frequently used by the lay people as a cure in obstetrical and gynecological disorders, e.g. to stop postpartum hemorrhage and to expel dead foetus or placenta. As an emmenagogue, it is consumed as a simple decoction prepared from 10–20 g dry leaves in each dose. Usually 2-3 doses can be very helpful. The therapeutic values of 'i-mu ts'ao' are documented in a wealth of classical and modern medical literature. The consensus points to the fact that 'i-mu ts'ao' extracts can stimulate uterine activity. The effective compound is believed to be an alkaloid called leonurine[2–6].

It is well known that the effect of different uterotonic agents varies with the species studied, the dosage used and the hormonal regime of the experimental subject[7–9]. Thus it is desirable to confirm the stimulatory effect of leonurine preparations in in vitro studies of human myometrium. 'I-mu ts'ao' dry plants from local sources were extracted with acidic methanol according to Hayashi[10]. The crude extract contains several Dragendorff-positive substances of which two are identified as free choline and stachydrine[3].

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