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HUMAN BRAIN HOMEOTHERMY DURING SLEEP AND WAKEFULNESS: AN EXPERIMENTAL AND COMPARATIVE APPROACH

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Abstract. To date human brain temperature has not been measured exactly. Limited published data indicate it to be about 37.5°C, which surprisingly is 1.0°C lower compared with placental mammals larger than the rat. Although the human brain is only 2% of body mass, it accounts for 20% of basal metabolism. Therefore, the removal of excess heat produced inside the brain is the main problem for its temperature regulation. The brain-arterial blood temperature difference in humans is probably twice that of larger mammals — 0.5°C. These two temperature factors play a crucial role for human brain homeothermy, particularly during motionless quiet waking and sleep. Low ambient temperature causes sleep deprivation. Moderate ambient heat allows sleep with negligible disturbances, and in humans induces sweating on the face and on the hairy (or bald) skin of the head. In passive hyperthermy human brain homeothermy depends on: (i) sweat evaporation from the skin surface of the face and whole head with face skin vasodilation, and (ii) enhanced venous return from the skin to the sinus cavernosus. This sinus is situated ventrally to the hypothalamus. Tympanic temperature reflects brain temperature fluctuations in humans.

INTRODUCTION

The human brain holds a particular position in the comparative anatomy and physiology of mammals. Compared with all other mammals, including primates, it is exceptionaly large, not only with regard to the rest of the body, but also with reference to the relatively small splanchnocranial part of the head. This anatomical peculiarity implies special features of the mechanisms regulating the human brain temperature. Actual knowledge of this problem is scarce. Invasive techniques which

have yielded valuable results in animal experiments are not suited for brain temperature research in humans. Therefore, the aim of this article is only to outline the problem of human brain homeothermy, taking into account the limitedness of the available experimental data and show a necessity of further research in this clinicaly important field.

HUMAN BRAIN TEMPERATURE

Surprisingly, we have no exact knowledge on the temperature of the human brain. Aschoff at al. (1), and Dymond and Crandall (11) reported that rectal temperature in men is about 0.2–0.4°C lower than that of the hypothalamus and the temporal lobe. In resting man, in a state of thermal balance, rectal temperature readings are normally between 37.0° and 37.4°C, with the characteristic circadian oscillations. The temperature of the brain is probably the same during sleep as during wakefulness when the subject is laying immobile under conditions close to thermoneutrality. Only in the early morning is the brain temperature considerably but transitorily lower. It is therefore likely that human brain temperature falls in the range of 37.4–37.8°C, and is about one degree lower than the brain temperature of placental mammals having a body mass exceeding that of the rat (8, 11, 14). The considerable difference between brain temperature of animals and humans should have inportant physiological consequences. Relatively low human brain temperature seems to be the first specific factor for human brain homeothermy. Undoubtedly, low brain temperature is advantageous because it increases the safety span between: (i) the resting hypothalamic temperature — 37.8°C (being the true body core temperature), and (ii) the maximum tolerable temperature — 41°C when thermal damage of the brain tissue begins. However, this intriguing problem has not yet been studied by clinical neurophysiologists and anesthesiologists. The upper limit of brain temperature tolerance is an important factor for general thermal physiology, because it probably involves all mammalian and avian species. A growing body evidence shows that cerebral tissue, compared to other body tissues, is particularly prone to heat damage (4, 15, 23).

HUMAN BRAIN TEMPERATURE REGULATION IN REST

According to Sokoloff (26) the mass of the human brain is only 2% of the whole body in the adult, but accounts for 20% of total basal metabolism. Since the human brain is exceptionally large in comparison

to that of other mammals, it produces incessantly a great amount of metabolic heat. Consequently, the main problem for human brain temperature regulation is the elimination of excess metabolic heat produced by the cerebral tissue. In a thermoneutral environment in the resting state, muscular thermogenesis results in a negligible increase of blood temperature during its passage through the muscle mass. However, according to Hayward and Baker (14), Caputa et al. (8) and Chęsy et al. (9), in resting unrestrained animals, arterial blood temperature measured at the circle of Willis is always cooler than the brain tissue. This temperature difference in animals is about 0.2–0.3°C. These data for humans are unknown, because this important thermophysiological parameter remains unmeasured. The reason seems to be not only the real difficulty in performing this measurement, but rather a general lack of interest in this parameter. So it is only posible to determine the human brain-arterial blood temperature difference approximately, by using the just mentioned data of Aschoff et al. (1) which reveals a difference twice as great as that of other mammals. This brain-blood temperature

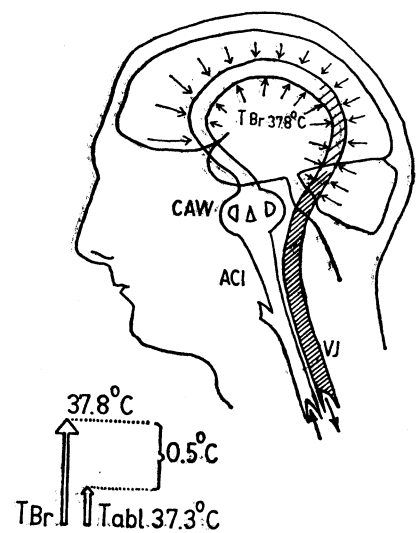

Fig. 1. Schematic presentation of the human brain-arterial blood temperature difference. ACI, arteria carotis interna; CAW, circulus arteriosusus Willisi; VJ, vena jugularis; TBr, brain temperature; Tab. 1, arterial blood temperature. Arrows denote direction of heat flow from brain tissue to the perfusing blood.

gradient is the second important factor (beside low brain temperature) playing a crucial role in the regulation of human brain temperature during rest and immobility. This suggests that under physiological conditions, when blood circulation is not disturbed, the half-degree cooler blood flow may be the main mechanism for the removal by convection of the brain metabolic heat excess (Fig. 1).

HUMAN BRAIN TEMPERATURE REGULATION IN PASSIVE HYPERTHERMY

In the humans sweat evaporation from the skin is the major channel of heat loss in hyperthermy. Its heat dissipating efficacy depends on the rate of both sweat secretion and evaporation. Evaporation from the upper respiratory pathways plays only a neglegible role. As Hertzman et al. (17), and McCaffrey et al. (22) have shown, the skin of the human face, and probably the skin of the whole head is the region of maximal sweating in comparison with the rest of the body surface. Sweating in hyperthermy always takes place together with skin vessels vasodilation (19). But powerful cooling of the skin covered by sweat drops out of low air humidity depends mostly on the convectional removal of the vapor from the immediate vicinity of the body surface through air movement (5, 6). However, such situation depends considerably on the rate of air flow over the face, particularly outdoors and through running. During sleep in a room of usual ambient microclimate, without a ceiling fan, this evaporating cooling is minimal. Caputa et al. (7) have shown that in hyperthermic human there is a considerably augmented venal blood flow passing from the facial skin to the sinus cavernosus surrounding

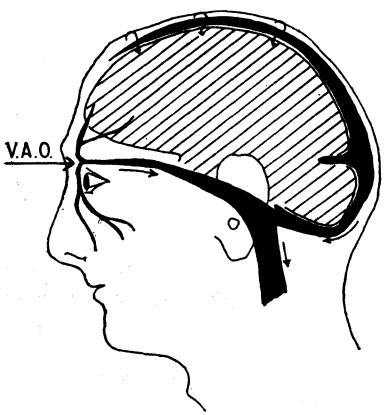

Fig. 2. Schematic design of the venous return from dilated skin vessels of the human face. V.A.O, vena angularis oculi outlet. Arrows on the top of the skull denote the probable venous return from the hairy (or bald) scalp covered by sweat drops. It is impossible to use the ultrasonic Doppler-effect probe for blood flow measurement here, since scalp venous return has of "field", and not of "point" flow.

the ventral hypothalamus. The main route of this flow is the vena angularis oculi, where it is also convenient to perform flowmetry (Fig. 2). It is a fairly big vessel in humans, and its outlet is easy to find (7). Anotomists have shown that in mammals deep facial veins are devoid of valves (7, 18, 21). It is a particular adaptation and evolutionary achievement enabling blood flow in either direction depending of the thermal state of the body. It also facilitates the blood flow from the face during hyperthermy. In consequence, brain metabolic heat excess may be directly removed in thermometrically measurable amounts from the basal part of brain by cooled venal blood flowing from the facial skin

(5, 6). This cooling is accompanied by a change in psychological self-rating from hyperthermy to normothermy or even hypothermy (5, 6).

The importance of sweat evaporation from the facial skin for human brain temperature regulation was shown conclusively by Cabanac and Caputa (5). The cooling effect measurable on the head surface and at tympanic membrane (which reflects brain temperature) was caused in these experiments by face fanning producing an air speed of 21 km/h. Data presented in that paper (5) clearly demonstrate a drop of temperature at the skin of the forehead and above the angularis ovuli vein outlet, and moreover, a decrease in tympanic temperature which reflects fairly well the brain temperature in humans. It should be stressed that fanning of the face had no effect on deep trunk temperature because oesophageal temperature was risings continuously as the subject was immersed in a bath at 38.6°C. Nevertheless, it demonstrates selective brain cooling caused by face fanning in passive hyperthermy (Fig. 3).

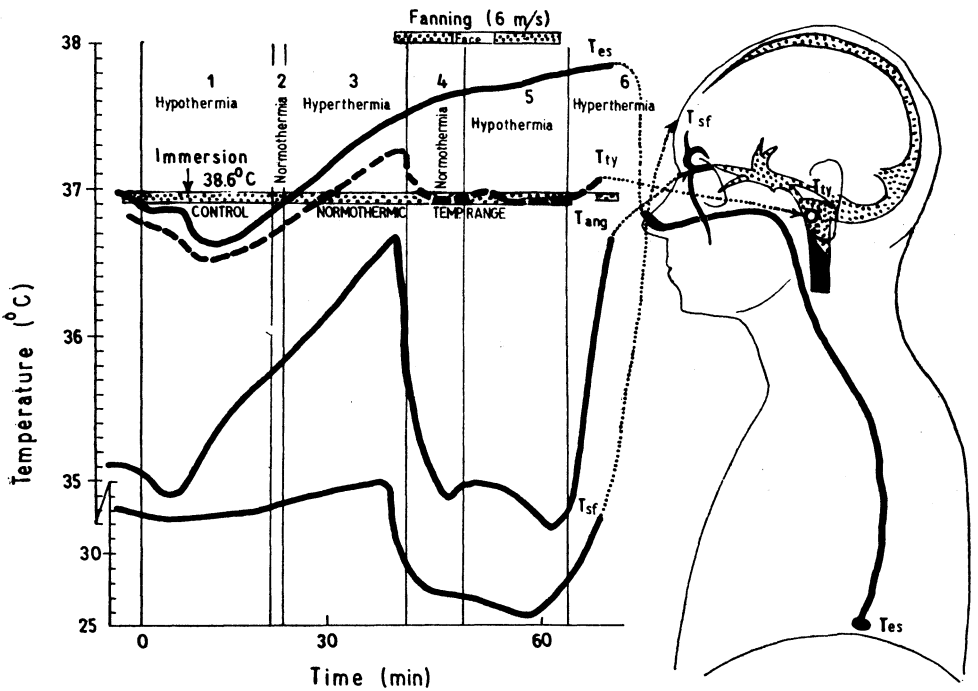

Fig. 3. Time course of temperature changes of the human body immersed in a warm bath of 38.6°C. Sites of temperature measurements: T_{ty}, membrana tympani; T_{sf} forehead (skin frontal); T_{ang}, angularis oculi vein outlet; T_{es}, oesophageal temperature. Ordinate: two temperature gauges; top, high accuracy; bottom, ten times lower accuracy. Horizontal gray band across temperature curves — 37°C — a control of normothermic temperature range (4).

The comparative approach gives some important contribution to the mechanisms discussed, which concern splanchnocranial vasomotor responses to hyperthermy. Winquist and Bevan (28) have discovered recently, in the rabbit, a thermically triggered venal sphincter in the external facial vein. Its role is to cause a shift of venal blood stream from the buccal skin covered by fur to the thermoregulatively active arterio-venal anastomoses ample (10) the moist and strongly evaporating nasal mucosa. Figure 4 schematicaly demonstrates this mechanism. In many mammals the respiratory evaporative cooling is acomplished by the moist nasal mucosa which covers the complex surface of the maxillary turbinate bones (2). But the necessary condition of the enhancement of this cooling by more than one order of magnitude is panting. One may suppose that this venal sphincter triggered by higher blood temperature, my operate analogically in all medium sized and large placental mammals which pant. All panting mammals which do not sweat or sweat poorly are equiped with efficient selective brain cooling mechanisms.

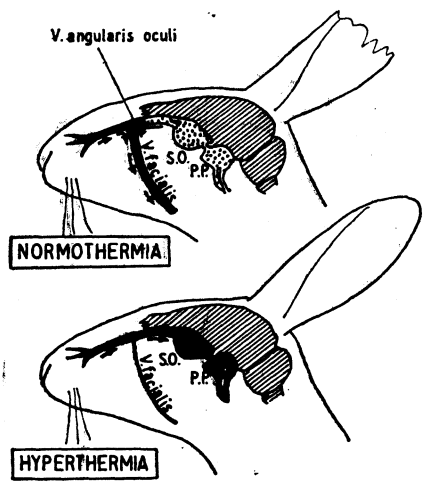

Fig. 4. Schematic arrangement of the rabbit cranial venal blood convectional mechanism removing brain metabolic heat excess during hyperthermy. Thermically triggered closure of the external facial vein causes the shift of the blood stream from vena facialis externa through nasal mucosa, and successively; sinus ophtalmicus — S.O. and plexus pterygoideus — P.P. lying below the ventral brain surface (28).

Baker and coworkers (3) have published pioneering research concerning the influence of both passive and exercise hyperthermia on two parameters: (i) common carotid blood flow, together with, (ii) upper respiratory water loss. The value of this work is the chronic conditions of experimentation. The study was performed on large mongrel dogs carefully adapted to the laboratory situation. Exercise hyperthermy was conducted at two work loads, and at two ambient temperatures, 25°C and 35°C. The dogs were forced to run on a treadmill for 15 min at a speed of 7.5 km/h on the level or on a 20% slope. The most important finding was that the rate of evaporation from the nose and the mouth

showed a striking temporal correspondence with the common carotid blood flow rate. Another finding was that during both passive and exercise hyperthermy, as well as during heating of the hypothalamus, common carotid blood flow considerably increased. This artifically induced rise of the dog brain temperature was strongly correlated with an enhanced rate of evaporation from the upper respiratory tract, accompanied by panting. Since it is well known that blood flow through the brain is rather stable, irrespective of the thermal state of the animal (12), the increase of the common carotid blood flow in hyperthermy must be through the splanchnocranium (the nasal and mouth mucosa in dogs), not through the brain. This is the surface through which the excess heat in the dog brain is dissipated. Quantitative data presented by these authors (3) revealed that the parallel rise of common carotid blood flow and upper respiratory tract water loss increased by three to four times. A smaller rise in these measures was obtained in dogs running at horizontal level, whereas maximal rise during running at 20% slope and ambient temperature of 35°C. It should be noted that readings of the rectal temperature in dogs shows clear inertia in response to exercise, expressed both during thermal load as well as when it was ended (3). This is why rectal temperature changes during exercise hyperthermia in large mammals may be considered as having limited importance, particularly when attention is focused on brain temperature regulation.

Therefore, one may conclude that a substantial enhancement of the splanchnocranial circulation is essential for brain homeothermy during

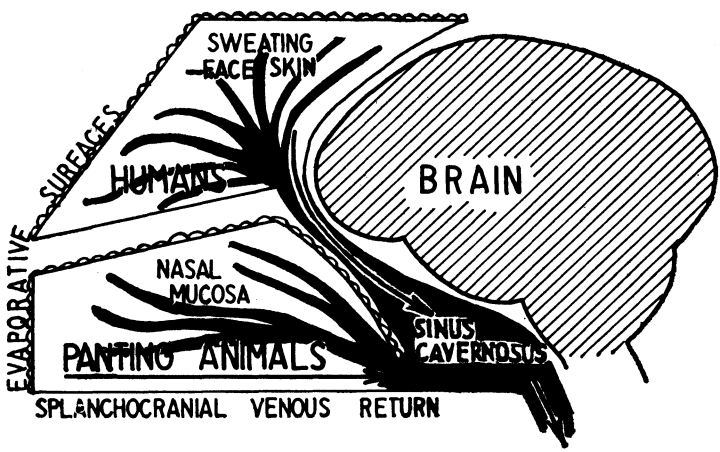

Fig. 5. Schematic picture of the selective cooling of the brain in the body hyperthermy of humans and larger placental mammals by venous return from splanchnocranial evaporating surfaces.

heat stress in the dog. This principle has some similarities in humans (5–7) and in rabbits (8,28) and enables selective brain cooling in all three species. In dogs (14) selective brain cooling does not operate during sleep (Fig. 5).

HUMAN BRAIN HOMEOTHERMY DURING SLEEP

The body size of adult humans ranks it amongst larger homeothermic organisms having a small body surface to body weight ratio. Large organisms are relatively less affected by ambient temperature. Although human skin is naked, clothing and bedding help to maintain homeothermy in both wakefulness and sleep. Homeothermy during sleep is secured mainly by sleeping room and bed-preparing behavior, and to a lesser degree by the automatic working of the body thermostat. On the other hand, the body thermostatic mechanisms are of particular importance in sleeping animals, and this has been shown in the numerous papers of Parmeggiani and his group e.g. (25). The reason for the efficient human sleeping place preparing behavior for homeothermic purposes seems to be the high level of experience and common social habits for bed and bedroom thermal comfort. The exceptions are rare. For example, the sleeping habits of Australian aborigines in their natural surroudings require from them a particular resistance to cold during sleep (27). Studies on human sleep performed in laboratory and consisting in the exposition of subjects to unusual thermal conditions are undoubtedly valuable but rather for the progress in understanding the consquences of sleep deprivation. However, that type of research may by useful for studying the problem of men's resistance to severe sleep disturbances, which are unavoidable in long-lasting expeditions to the regions of extremal climate, as the Himalays as or Antarctic Continent.

Studies of human body temperature regulation during sleep are scarce. Moreover, they do not involve human brain temperature monitoring, which is understandable because of limitations of the unavoidably invasive technique. Tympanic temperature measurement during human sleep has been used only exceptionally (24).

Among more or less exhaustive and descriptive definitions of sleep repeated in numerous monographs and textbooks, one of the important, even crucial, feature of sleep is rarely stated. This is that sleep is a particular functional state of vigillance fully dependent on the central nervous system. This opinion is based on the principal symptom of sleep — the transitional but fairly deep and fluctuating loss of consciousness. The variable level of consciousness, which repeatedly and rythmically oscillates along nycthemeral periods during life, may be localized

exclusively inside the brain. The statement is evident, but it seems reasonable to stress here its fundamental significance, because it is rarely expressed decidedly. The rest of the body during sleep is certainly in a special state, but it differs only slightly from the habitual repose during wakefulness and immobility. Therefore brain temperature during sleep, not trunk or rectal temperature, is more important in sleep research, as the function of sleep lies in the brain, not in the other organs of the body.

Only three recent papers devoted to thermoregulation during human sleep will be choosen and discussed briefly or only mentioned in this concise review.

Haskell et al. (13) have published excellent research performed in two laboratories under the supervision of R. J. Berger and H. C. Heller. These studies concern the full night course of thermoregulation during sleep in nude subjects at three ambient temperatures: cold — 21°C, thermoneutral — 29°C, and warm — 37°C. These authors did not measure the tympanic temperature, with certainly reflects human brain temperature. Instead they measured the rectal and estimated the mean skin temperature, taken from seven sites on the human body surface: chest, abdomen, thigh, calf, upper arm, forearm, and forehead. Among these sites the only really valuable for the purpose of this article (human brain homeothermy during sleep) was the forehead temperature. The importance of this measure was shown by Cabanac and Caputa (5, 7) since they demonstrated that forehead temperature correlates with the intensity of sweat evaporation from the face skin, which in turn causes a decrease in human tympanic temperature (5–7). Unfortunately, the forehead temperature was incorporated into mean skin temperature. Regrettably, skin temperature at the point of angularis oculi vein outlet was not monitored.

Besides measuring the rectal and computing the mean skin temperature, Haskell et al. (13) measured oxygen consumption during 8 hours' night sleep in the same subjects, and found significant changes depending on the ambient temperature. Whenever the rate of total body metabolism of the subject exceeded the basal value, it was directly correlated with the body movements, which means that each movement enhanced muscular metabolism and thermogenesis. Such events are typical for fairly quiet sleep, both in thermoneutral and warm environment. Instead, under cold ambient temperature O_2 consumption was often much more enhanced, by about 30%, and was correlated with shivering and with periods of wakefulnes in the subjects during the cold night.

In summary, it seems that in all three ambient temperatures the elevated O_2 consumption reflected not brain metabolism, but more peri-

pheral increases in body metabolism, mostly during insered waking periods rather than during sleep. During quiet sleep in thermoneutral or warm ambient temperatures, each body movement registred was accompanied only by about 5–7% increase in O_2 consumption. Such increase may have only a small influence on the human body temperature regulation.

The paper of Henane et tl. (16) supplies eivdence of the altered range of human body temperature regulation during REM sleep. These authors have shown that in warm ambient temperature (35°C) the inhibition of sweating appears in every REM sleep period. This means that during REM sleep sweat secretion is inhibited. A quantitative assay revealed that skin evaporation was diminished by as much as three to four times during each REM sleep period in comparison with N-REM sleep. But since REM sleep episode lasts only several minutes, and due to considerable thermal inertia of the human body, the REM accompanied cessation of sweat secretion and evaporation has little influence on deep body and also brain temperature (16).

On the other hand, subject sleeping in the cold do not demonstrate full inhibition of shivering during REM sleep (13). Such reaction in humans contrasts with the strong response to cold in animals during REM sleep. Full inhibition of shivering in cats during REM sleep was reported by Parmeggiani (23). However, the damped shivering of human subjects sleeping in the cold starts before the beginning of REM sleep periods, during the preceeding N-REM sleep (13). This is another difference between animal and human REM sleep symptoms in cold ambient temperatures. Consequently, the suspension of the body thermostatic function, previously described by Parmeggiani (25), appears to be perceptably weaker in humans than in animals during REM sleep. The same opinion was recently stressed by French authors of the Strassburg group, by Liebert et al. (20).

CONCLUDING REMARKS

Humans belong to the group of large placental mammals. Despite their naked skin they are resistant to cold, owing first to clothing and second, to a very effective thermoregulatory behavior, particularly when preparing for sleep.

Due to the exceptionaly large mass of the human brain, the removal of metabolic heat excess from this organ is a particularly important function of the body thermostat. Probably for this reason, the human brain is about 1°C cooler than the brains of other large and medium-sized placental mammals. The removal of heat excess is a continuos day

and night process both in humans and animals. In rest and in thermoneutral environment, this process is maintained by: (i) brain-arterial blood temperature difference which in humans appears to be twice (0.5°C) that of other mammals, and — (ii) the abundant perfusion of the brain tissue with slightly cooler arterial blood.

In hyperthermic humans, both awake and sleeping, another mechanism is included. It consists in enhanced venous return from the skin surface of the face cooled through sweating. This cooled blood reaches the cavernous sinus inside the basal part of neurocranium. It removes heat from the ventral part of the brain. This selective brain cooling occurs directly in humans and other species (e.g. rabbits (8)), or indirectly and more efficiently, by a counter current arterio-venal heat exchanger (carotis rete mirabile), in carnivora and artiodactyls. All these animals demonstrate evaporative heat loss from the upper respiratory tract mucosa during panting. But they pant mostly when awake and behaviorally active.

Ambient temperatures not much different from thermoneutrality are needed for undisturbed sleep. Deviation from this level in either direction, but particularly to the cold, causes awakening and, in consequence, sleep deprivation. This deviation motivates the subject to change the thermal microenvironment. Therefore, human physiological reserves during sleep for a possible defense against body hyperthermy or hypothermy are limited.

More work is needed to acquire further knowledge not only of the vasomotor activity of the facial and nasal arterio-venous anastomoses, but also of the venous blood flow shift mechanisms which operate inside splanchnocranium during hyperthermy. They are not the same in humans and in other mammals, but certainly are essential for thermal comfort of the subject both during wakefulness and sleep, and for effective defense against core and brain temperature deviations.

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