Eur J Appl Physiol (1995) 70: 207–212

ORIGINAL ARTICLE

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Nasal mucosal vasodilatation in response to passive hyperthermia in humans

Accepted: 7 August 1994

Abstract The present study was conducted to measure nasal mucosal blood flow (NMBF) during body warming. Five subjects [mean (SD) 24 (2) years], wearing only shorts and a thick felt hat with ear flaps, were immersed to the neck in a bath at 40 (0.5)°C. Tympanic (T_{ty}), esophageal (T_{es}), mean unweighted skin (T_{sk}), nose skin and ear pinna skin were recorded at 1-min intervals. NMBF on the lower septal wall was estimated using a laser Doppler flow meter. At rest T_{ty} and T_{es} were both 36.5°C. T_{ty} dropped significantly below T_{es} during body warming, despite impeded heat loss from the head due to the felt hat. T_{ty} increased to 37.3°C and T_{es} increased to 37.5°C during the immersion. During the immersion all skin temperatures were steady or increasing, ruling out the possibility of a contamination of T_{ty} from T_{sk}. Body warming significantly ($P = 0.001$) increased NMBF by approximately three times from resting values at the end of immersion. During the period of increasing core temperatures NMBF was significantly correlated to T_{ty} ($r = 0.93$, $P = 0.0001$) and T_{es} ($r = 0.97$, $P = 0.0001$), suggesting the blood flow change in this tissue was a thermoregulatory response. The increased NMBF during hyperthermia supports the hypothesis of respiratory cooling involvement in selective brain cooling of humans.

Key words Selective brain cooling · Respiratory heat loss · Hyperthermia · Tympanic temperature · Esophageal temperature

Introduction

In several species the nasal mucosal blood flow (NMBF) increases during heat stress (Caputa 1979; Pleschka et al. 1979; Baker 1982; Johnsen et al. 1985) at the same time as a thermally induced tachypnea (Pleschka et al. 1979; Baker 1982). The increase in NMBF together with thermally induced tachypnea can be used as a heat loss mechanism to selectively cool the brain. Cooled venous blood flows via the angularis oculi to the cavernous sinus where a counter-current heat exchange cools the warmer arterial blood arriving to the brain and this gives a selective brain cooling (SBC) relative to trunk core temperatures (Baker and Hayward 1967; Baker and Hayward 1968; Eekhawad et al. 1990; Magilton and Swift 1968; Johnsen et al. 1985).

To a smaller extent than other panting species, humans are known to hyperventilate relative to their metabolic needs during passively induced (Haldane 1905; Gaudio and Abramson 1968; Hanson 1974; Cabanac and White 1995) and exercise-induced hyperthermia (White and Cabanac 1993). This thermal hyperpnea gives an increased respiratory evaporative heat loss that is correlated to increased ventilation and core temperature seen during passively induced hyperthermia (Hanson 1974). Hanson (1974) showed that ventilation increases as a function of core temperature during hyperthermia, and the magnitude of the respiratory heat loss is a function of increasing minute ventilation. Unlike in animals (Hales and Dampney 1975; Pleschka et al. 1979) NMBF has not been measured during heat stress in hyperthermic humans. The advent of small laser Doppler probes for NMBF (Druce et al. 1984) has allowed such measurements.

The hypothesis tested in this study was that NMBF increases relative to rest during passively induced hyperthermia by hot bath immersion. Knowledge of nasal mucosal vasomotion in hyperthermic humans is valuable to understand three aspects of thermal physiology related to respiratory heat loss in humans. Firstly, it will provide evidence if vasodilatation in the mucosa of the upper respiratory tract is underlying increased respiratory heat loss in hyperthermia. Secondly, since core temperatures during bath immersion are increased

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while skin temperatures are clamped, the results will provide evidence of whether the NMBF might be influenced by efferent signals arising due to increased temperature of the central nervous system. Thirdly, results will provide information related to the possible involvement of respiratory heat loss in SBC of humans (for reviews, see Brengelmann 1993; Cabanac 1993).

Methods

Subjects

Five college-aged [24 (2) years] male volunteers with a body mass of 66.5 (2.4) kg and height of 1.73 (0.02) m participated as subjects. The experimental protocol was approved by the Laval University ethics committee. All subjects were aware of the risks associated with the experiment and signed an informed consent.

Protocol

The experiments took place between 10:30 a.m. and 12:30 p.m. The subject was asked to fast and refrain from exercise for 3 h before the experiment. Before each experiment the subject was given approximately 40 min to adjust to the room temperature of 24.8 (0.2)°C. The session started with a 5-min rest period when pre-immersion data were collected. Then the subject was seated in a 40 (0.5)°C stirred bath with the shoulders immersed, wearing nylon shorts and a thick felt hat with earflaps tied under the chin, to prevent falls in head skin temperature. The hat covered an estimated two-thirds of the head including all the hairy scalp, part of the forehead, and the temples including the ears to a level under the mandible. The experiment was terminated if esophageal temperature (T_{es}) reached 1.5°C above resting levels or if NMBF reached a plateau value.

Instrumentation

T_{es} and tympanic (T_{ty}), unweighted mean skin (T_{sk}, arm, stomach and thigh), nose bridge skin (T_{ns}) and pinna lobe surface (T_p) temperatures were monitored at 1-min intervals using thermocouples. Verification of tympanic probe placement and insulation was as described previously (Brinnel and Cabanac 1989). The depth of the esophageal probe was determined using the subject's sitting height and the mean insertion length was 0.39 (0.01) m, a position corresponding to the level of the left ventricle (Mekjavic and Rempel 1990). Measurement of the temperatures was with an Omega data acquisition system controlled by a Macintosh computer.

NMBF was estimated using a laser Doppler flowmeter (Vasamedics, USA) with a soft flexible probe (model P440, Vasamedics, USA). This technique has been recently employed in the measurement of human NMBF in normothermic subjects (Druce et al. 1984). The probe was positioned between 10 and 20 mm in the nasal vestibule and placed on the septal wall separating the nasal vestibules. The probe was placed in the same position throughout each experiment since the probe insertion depth was marked.

Analysis and calibrations

ANOVA at four levels (minutes 4, 10, 15 and 20) was employed to evaluate changes in NMBF and temperatures over the immersions. Simple correlations between NMBF and temperatures were made and the level of significance was set at 0.05. For comparisons of means of NMBF the level of significance was adjusted by the Bonferroni statistic. All physiological data are presented as mean (SE). All thermocouples were calibrated in a temperature-regulated water bath.

Results

Figure 1 gives the mean time course of core temperatures over the experiments. At rest, T_{ty} and T_{es} were equal at 36.5°C. Following immersion in the bath T_{ty} decreased less than 0.1°C reaching a minimum at minute 9. T_{es} following immersion decreased to 36.3°C by minute 8. Both temperatures increased until minute 10 when both temperatures were again equal at 36.5°C. From minutes 10 to 20 both temperatures increased in an approximately linear manner. By minute 20, T_{ty} increased to a maximum of 37.3°C and T_{es} increased to a maximum of 37.5 °C. Over the course of this mild hyperthermia T_{ty} dropped significantly below T_{es} ($F = 4.09$, $P = 0.03$).

Resting values of mean NMBF were approximately 4 arbitrary units (AU) before the immersion (Fig. 2). The subject was immersed and the probe repositioned from minutes 4 to 6. Following immersion the value increased abruptly to a value of 6 AU at minute 7. Then the level remained steady at 6 AU until minute 10. From minutes 10 to 20 the value increased in an approximately linear manner until minute 18 where the maximum mean value of 12.9 AU was observed. For minutes 19 and 20 the value stabilized at close to this level. The values of NMBF at minutes 15 ($F = 10.6$,

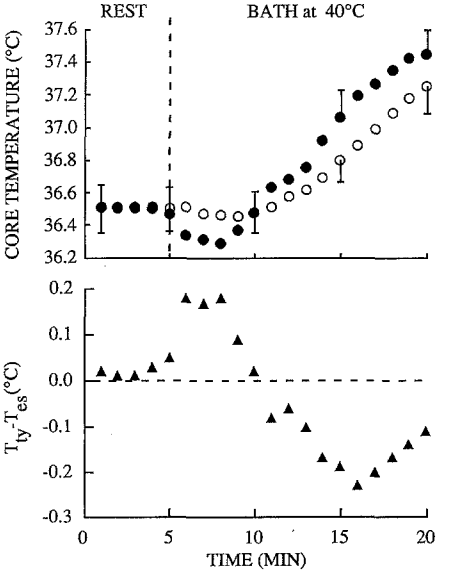

Fig. 1 Core temperature responses to 40 °C water immersion for 15 min. Each symbol represents the mean of five subjects. Hot immersion significantly lowered tympanic (T_{ty}) below esophageal (T_{es}) temperature. Error bars show SE. *Closed circle*, T_{es}; *Open circle*, T_{ty}

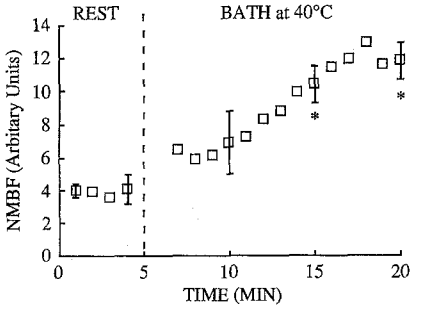

Fig. 2 Nasal mucosal blood flow (NMBF) during 4 min of rest, and during 15 min in a hot bath. Between minutes 4 and 6 the subject was transferred to the bath and the laser Doppler probe repositioned. Each symbol represents the mean of five subjects. Error bars show SE. Hot immersion gave significantly greater levels of NMBF and 15 and 20 min of immersion relative to resting values.*$P < 0.01$.

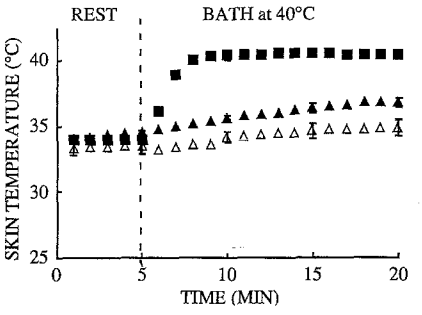

Fig. 3 Mean skin (*closed square*), ear pinna skin (*closed triangle*), nose skin surface (*open triangle*) temperatures before and during 15 min in the 40 °C bath. Each symbol represents the mean of five subjects. Error bars show SE. The SE is smaller than the symbol in the case of the mean skin temperatue. All values were steady or increased during immersion

$P = 0.005$) and 20 ($F = 15.9$, $P = 0.001$) were significantly greater than pre-immersion values.

All skin temperatures are presented in Fig. 3. T_{sk} before immersion was approximately 34°C. On immersion the value increased to 40.3°C by minute 9 were the value remained in an approximate steady state until minute 20. During the resting period T_p was at approximately 34.5°C. Following immersion at minute 5 T_p increased to a value of 36.7 by minute 16 where the value reached an approximate steady state for the remainder of the immersion. T_{ns} remained steady at approximately 33.3°C before immersion. A slight decrease of T_{ns} by 0.3°C was observed on immersion. T_{ns} nonsignificantly increased ($F = 1.5$, $P = 0.24$) to 34.6°C by minute 15, where it remained in an approximate steady state for the remainder of the bath.

From minute 9 when core temperatures began increasing until minute 20, significant correlations (Fig. 4) were evident between NMBF and T_{ty} ($r = 0.93$, $P = 0.0001$), and between NMBF and T_{es} ($r = 0.97$, $P = 0.0001$).

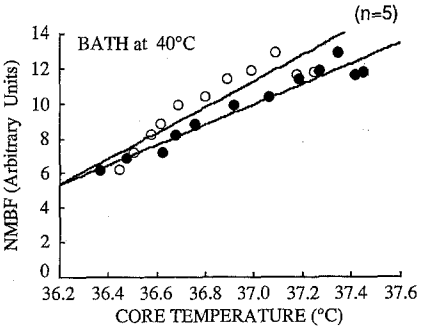

Fig. 4 Correlations between NMBF and core temperatures from minutes 9 to 20. Each symbol represents the mean of five subjects. NMBF increased proportionately to T_{ty} and T_{es}. *Closed circle*, T_{es}; *Open circle*, T_{ty}

Discussion

The main finding of this work is that NMBF significantly increased from the resting pre-immersion value during hyperthermia. As illustrated in Fig. 2 the value for NMBF increased from the pre-immersion value of approximately 4 AU to 12 AU by the end of immersion. The increase in NMBF did not appear to be attributed to a local temperature effect since T_{ns} increased nonsignificantly during the immersion (Fig. 3). Thus it appeared the main influence on NMBF was the increased core temperature. This contention is supported by NMBF being significantly correlated to both T_{ty} and T_{es} (Fig. 4). The results suggest that the increase in NMBF observed was a response arising from changes in core temperature, at constant levels of skin temperature on the nose surface and body. It should be emphasized that the laser Doppler technique provides a relative change and cannot be used quantitatively in calculations of heat exchange.

NMBF with core and peripheral temperature changes

NMBF has been shown to decrease with skin cooling (Spiesman 1936; Ralston and Kerr 1945; Cole 1954; Drettner 1961) and increase with skin warming (Spiesman 1936). In these previous experiments estimates of changes in NMBF were made by measuring thermoconductivity of nasal mucosal tissues (Drettner 1961), or by measuring nasal mucosal (Spiesman 1936; Ralston and Kerr 1945; Drettner 1961) or submucosal (Cole 1954) temperatures. The nasal mucosa contains arteriovenous anastomoses (Cauna 1970). These organs are specialized thermoregulatory organs. Local temperature on the septal wall decreased rapidly during cooling of the feet with ice (Drettner 1961). Also the temperature of the nasal mucosa (Spiesman 1936; Ralston and Kerr 1945) or submucosa (Cole 1954) decreased in response to cold stimulation on the skin of the back and feet (Spiesman 1936; Ralston and Kerr

1945; Cole 1954). Spiesman (1936) reported increased nasal mucosal temperature during heating of various regions along the spine and body surface. Therefore, the literature supports that NMBF responds to skin temperature changes in a thermoregulatory fashion with vasoconstriction during cold stress and vasodilatation during warm stress on a distal body surface. In these previous studies core temperatures were either normothermic (Drettner 1961) or not measured (Spiesman 1936; Ralston and Kerr 1945; Cole 1954). The present results support that at elevated and stable skin temperatures, NMBF increases proportionately to core temperatures (Fig. 4).

The temperature of the inhaled air seems to be able to modify NMBF, as indirectly judged from air pressure in the airways. Cold air did not modify the pressure, but hot inhalation was followed by increased pressure, indicating vasodilatation (Negus 1960). Although this result would seem irrelevant to the present work where the temperature of the inhaled air remained constant, it nevertheless shows that the nasal mucosa responds to heat by vasodilatation, a result that would support our view that heat loss from the nose is increased under heat stress.

Perspective: SBC in humans

These results could be considered to support the hypothesis of SBC in humans. The significantly smaller rise in T_{ty} relative to T_{es} during the 40°C immersion (Fig. 1) supports the occurrence of SBC in humans as reported previously (see Cabanac 1993 for a review). The smaller rise of T_{ty} than T_{es} was seen despite the main heat loss being only from the exposed face and upper airways due to the headgear, hot water immersion, and the fact that the hyperthermia was mild (about 0.8 °C). The "contamination" of T_{ty} by conductive cooling from skin (Shiraki et al. 1988) can be ruled out (Fig. 3) since all skin temperatures were increasing or stable during the immersion. The results suggest that the respiratory heat loss was contributing to the fall of T_{ty} below T_{es} during hyperthermia. During hyperthermia (Haldane 1905; Gaudio and Abramson (1968; Hanson 1974; Cabanac and White 1995) a thermally induced hyperpnea was observed in humans. Respiratory heat loss is thus hypothesized to increase during the simultaneous thermal hyperpnea and nasal mucosal vasodilatation (Fig. 2). This suggests that respiratory cooling is involved in SBC in humans in a similar mechanism as that seen in other animals demonstrating SBC (Baker 1982). During a heat stress nasal vasodilatation is evident in several animals (Baker and Hayward 1968; Blix and Johnsen 1983; Johnsen et al. 1985) together with a thermal induced tachypnea (Pleschka et al. 1979) both acting to give SBC.

Other evidence supporting the involvement of respiratory heat loss in human SBC is that: (1) breathing of humid air caused a convergence of T_{ty} and T_{es} during the hyperthermia of exercise (White and Cabanac submitted), (2) humans hyperventilate relative to metabolic needs in hyperthermia induced by exercise (White and Cabanac 1993), and (3) dilatation of the nares during submaximal exercise gave slower rise of T_{ty} during submaximal exercise (White and Cabanac 1992). These results together with the vasodilatation taking place in the nasal mucosa in hyperthermic humans would suggest that nasal heat loss participates in SBC.

Two studies recently examined the change in the latency of acoustically evoked potentials in hyperthermic humans to examine for the existence of selective brain cooling (Jessen and Kuhnen 1992; Nielsen and Jessen 1992). These results have been discussed and refuted (Cabanac 1993) and are discussed briefly here. Jessen and Kuhnen (1992) as well as Nielsen and Jessen (1992) showed that the latency of auditory evoked potentials decreased due to a higher speed of conduction during hyperthermia, but that cold or warm air face fanning did not change the latency times of auditory evoked potentials in hyperthermic subjects. They concluded there was no SBC since with face fanning there was not effect on the latency of the auditory evoked potentials. However, the cold air face fanning Jessen and Kuhnen (1992) performed was just able to decrease the mean T_{ty} by 0.4°C and the warm air face fanning of Nielsen and Jessen (1992) decreased T_{ty} by only 0.15°C. These authors calculated that the limit of sensitivity to detect a change in the latency of auditory evoked potentials is 0.4°C. A small but significant change in T_{ty} does not obviate the need to show a change in temperature by 0.4°C or more to allow conclusions regarding the latencies of the auditory evoked potentials during hyperthermia with face fanning.

Brain temperature and tympanic temperature

Recently two studies simultaneously measuring T_{ty} and brain temperature have presented diametrically opposite result concerning the relationship between them (Shiraki et al. 1988; Mariak et al. 1993). The findings of Shiraki et al. (1988) were on one normothermic 12-year-old unanesthetized patient with a drainage catherter and thermocouples implanted into the right lateral ventricle to relieve intracranial pressure arising from a pineal tumor. In one of four experiments the T_{ty} was dissociated from the measurements in brain temperature and from this the authors concluded that T_{ty} was independent of brain temperature. Although the authors state that cerebral angiography revealed no abnormalities, the increased intracranial pressure is likely to have affected cerebral venous blood flow. During periods of increased intracranial hypertension Cushing's response acts to increase systemic blood

pressure to maintain cerebral blood flow. This increased intracranial pressure is likely to have influenced blood flow and temperature measurements of the tympanic membrane (Cabanac and Brinnel 1985; Caputa et al. 1978; Deklunder et al. 1991; Nagasaka et al. 1989). Chmielowa et al. (1980) have shown that T_{ty} of feverish hydrocephalic children was 1.3°C higher than feverish controls showing that intracranial hypertension influences T_{ty}. In direct contrast to Shiraki et al. (1988) a direct relationship between T_{ty} and brain temperature during local cooling of the mesencephalon in the interpeduncular fossa in six hyperthermic humans was evident (Mariak et al. 1993). The results confirmed the assumption that T_{ty} is an index of brain temperature (Cabanac 1993). In the same experiments T_{es} and rectal temperature gave no changes following the local cooling of the mesencephalon (Mariak et al. 1993) showing T_{ty} was the best index of brain temperature.

Variability of SBC and respiratory heat loss

Since the early evidence of SBC was reported (Baker and Hayward 1967; Magilton and Swift 1968) this phenomenon has been found in a large number of animals (Baker 1982). In all cases SBC results from respiratory cooling. The rat (Caputa et al. 1991) showed a small but significant SBC, yet the antelope had a brain temperature 2.7°C cooler than carotid arterial blood during exercise (Taylor and Lyman 1972). As such, a large variability of SBC appears evident. Animals with a carotid rete and those that pant appear to have greater SBC but these are not essential characteristics for SBC, which is seen in the absence of a carotid rete (Gordon et al. 1981; Baker 1982; Fuller and Baker 1983; Caputa et al. 1991; Hales et al. 1993). Of these animals with no carotid rete the horse sweats to cool itself yet shows SBC (Hales et al. 1993). Human SBC appears from the present results and previous results (Rasch et al. 1991; White and Cabanac 1992; White and Cabanac 1993) to be in part due to respiratory heat loss although the large surface cooling from sweating on the head appears quantitatively to be of greater importance, as originally proposed (Cabanac and Caputa 1979a, b).

These results show that NMBF increases during the hyperthermia of a warm bath immersion and suggest that the increased respiratory heat loss in hyperthermia is in part due to airway mucosal vasodilatation. The results support the hypothesis of respiratory cooling as a mechanism of heat loss involved in human SBC.

Acknowledgements This work was supported by the Defense and Civil Institute of Environmental Medicine of Canada and by Natural Science and Engineering Research Council of Canada.

References

Baker MA (1982) Brain cooling in endotherms. Ann Rev Physiol 44:85–96.
Baker MA, Hayward JN (1967) Carotid rete and brain temperature of cat. Nature 216:139–141
Baker MA, Hayward JN (1968) The influence of nasal mucosa and carotid rete upon hypothalamic temperatures in sheep. J. Physiol (Lond) 198:571–579
Blix AS, Johnsen HK (1983) Aspects of nasal heat exchange in resting reindeer. J Physiol (Lond) 340:445–454
Brinnel H, Cabanac M (1989) Tympanic temperature is a core temperture in humans. J Therm Biol 14:47–53
Brengelmann GL (1993) Specialized brain cooling in himans? FASEB 7:1148–1153
Cabanac M (1993) Selective brain cooling in humans: "fancy" or fact? FASEB 7:1143–1147
Cabanac M, Brinnel H (1985) Blood flow in the emissary veins of the human head during hyperthermia. Eur J Appl Physiol 54:172–176
Cabanac M, Caputa M (1979a) Open loop increase in trunk temperature produced by face cooling in working humans. J Physiol (Lond) 289:163–174
Cabanac M, Caputa M (1979b) Natural selective cooling of the human brain evidence of its occurrence and magnitude. J Physiol (Lond) 286:255–264
Cabanac M, White M (1995) Core temperature thresholds for hyperpnea during passive hyperthermia in humans. Eur J Appl Physiol (in press)
Caputa M (1979) Temperature gradients in the nasal cavity of the rabbit. J Therm Biol 4:283–286
Caputa M, Perrin G, Cabanac M (1978) Écoulement sanguin reversible dans la veine opthalmique: Mécanisme de refroidissment sélectif du cerveau humain. CR Acad Sci Paris t. 287:1011–1014
Caputa M, Kamari A, Wachulec M (1991) Selective brain cooling in rats resting in heat and during exercise. J Therm Biol 16:19–24
Cauna N (1970) The fine structure of the arteriovenous anastomosis and its nerve supply in the human nasal respiratory mucosa. Anat Rec 168:9–22
Chmielowa M, Kielczewska-Mrozikiewicz D, Skuratowicz A (1980) Tympanic temperature in some diseases with fever (in Polish) Rocz Akad Med Poznam X25:157–160
Cole P (1954) Respiratory mucosal vascular responses, air conditioning and thermoregulation. J Laryngol 68:613–622
Deklunder GM, Dauzat M, Lecroart J-L, Hauser J-J, Houdas Y (1991) Influence of ventilation of the face on thermoregulation in man during hyper- and hypothermia. Eur J Appl Physiol 62:342–348
Drettner B (1961) Vascular reactions of the human nasal mucosa on exposure to cold. Acta Otolaryngol 166 [Suppl]:1–109
Druce HM, Bonner RF, Patow C, Choo P, Summers RJ, Kaliner MA (1984) Response of nasal blood flow to neurohormones as measured by laser Doppler velocimetry. J Appl Physiol 57:1276–1283
Elkhawad AO, Al-Zaid NS, Bou-Resli MN (1990) Facial vessels of desert camel (*Camelus dromedarius*): role in brain cooling. Am J Physiol 258:R602–R607
Fuller CA, Baker MA (1983) Selective regulation and body temperature in the squirrel monkey. Am J Physiol 245:R293–R297
Gaudio Jr, Abramson N (1968) Heat-induced hyperventilation. J Appl Physiol 25:742–746
Gordon C, Rezvani AH, Fruin ME, Ttautwein S, Heath JE (1981) Rapid brain cooling in the free running hamster (*Mesocricetus auratus*). J Appl Physiol 51:1349–1354
Haldane JS (1905) The influence of high air temperatures. J Hyg 55:497–513
Hales JRS, Dampney RAL (1975) The redistribution of cardiac output in the dog during heat stress. J Therm Biol 1:29–34
Hales JRS, McConaghy FF, Hodgson DR (1993) Limited selective brain cooling in the horse during exercise. In: Milton AS (ed)

Thermal Physiology. Proceedings of the IUPS Thermal Physiology Commission symposium. BPCC-AUP, Aberdeen, p 41

Hanson R de G (1974) Respiratoru heat loss at increased core temperature. J Appl Physiol 37:103–107

Jessen C, Kuhnen G (1992) No evidence for brain stem cooling during face fanning in humans. J Appl Physuol 72:664–669

Johnsen HK, Blix AS, Jorgensen L, Mercer JB (1985) Vascular basis for regulation of nasal heat exchange in reindeer. Am J Physiol 249:R617–R623

Magilton JH, Swift CS (1968) Description of the two physiological heat exchange systems for the control of brain temperature. In: I.E.E.E. Conference record, 5th Annual Rocky Mountain Bioengineering Symposium, 6–7 May pp 24–27

Mariak Z, Lewko J, Luczaj J, Polocki B (1993) The direct relationship between human cerebral and tympanic temperatures as demonstrated with manipulation of brain temperature. In: Milton AS (ed) Thermal physiology, Proceedings of the IUPS Thermal Physiology Commission Symposium. BPCC-AUP, Aberdeen Ltd, Aberdeen, p 78

Mekjavic IB, Rempel ME (1990) Determination of esophageal probe insertion length based on standing and sitting height. J Appl Physiol 69:376–379

Nagasaka T, Brinnel H, Hirata K, Noda Y, Sugimoto N (1989) Increase in venous flow through opthalmic veins enhanced selective brain cooling in hyperthermic humans. In: Mercer JB (ed) Thermal physiology. Elsevier, Amsterdam, pp 205–210

Negus VE (1965) The biology of respiration. Livingstone, Edinburgh

Nielsen B, Jessen C (1992) Evidence against brainstem cooling by face fanning in severely hyperthermic humans. Pflügers Arch 442:168–172

Pleschka K, Kuhn P, Nagai M (1979) Differential vasomotor adjustments in the evaporative tissues of the tongue and nose in the dog under heat load. Pflügers Arch 382:255–262

Ralston HF, Kerr WMJ (1945) Vasular responses of the nasal mucosa to thermal stimuli with some observations on skin temperature. Am J Physiol 144:305–310

Rasch W, Samson P, Coté P, Cabanac M (1991) Heat loss from the human head during exercise. J Appl Physiol 71:590–595

Shiraki K, Sagawa S, Tajima F, Yokota A, Hashimoto M, Brengelmann GL (1988) Independence of brain and tympanic temperatures in an unanesthetized human. J Appl Physiol 65:482–486

Spiesman IG (1936) Vasomotor reactions of the mucosa of the upper respiratory tract to thermal stimuli. Am J Physiol 115:181–187

Taylor CR, Lyman CP (1972) Heat storage in running antelopes; independence of brain and body temperatures. Am Physiol 222:114–117

White MD, Cabanac M (1992) Physical dilatation of the nares lowers thermal strain in exercising humans. In: Lotens WA, Havenith G (Eds) Environmental ergonomics. Proceedings of the 5th International congress of environmental ergonomics. TNO Press, Soesterberg, Netherlands, pp 220–221

White MD, Cabanac M (1993) Exercise hyperpnea and selective brain cooling in humans (abstract). FASEBJ 7:A16