119 Thermal Exchanges in Man

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This chapter was originally published under the ISBN 978-3-540-01771-4 with the following book title Measuring the Skin. The content has not been changed.

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P. Humbert et al. (eds.), *Agache's Measuring the Skin*,
DOI 10.1007/978-3-319-32383-1_69

Keywords
Skin thermal exchange • Heat transfer • Heat balance equation • Cold environment • Hot environment

Man regulates his internal body temperature within a normal range, with limits around 37 °C. This endothermia relates only to the internal part of the body (core), as the peripheral part, represented by the skin, can vary greatly (from a few degrees up to 44 °C).

Regulation of the body temperature at a constant level is under the control of the thermoregulatory system located within the hypothalamus. The hypothalamic centers, which are also sensitive to the circulating blood temperature, are constantly informed of the thermal state of the various parts of the body. For that purpose there are peripheral (cutaneous) thermoreceptors, which respond specifically to warm or cold stimulation. Some of these sensors operate in static mode (temperature level), while others operate in dynamic mode (rate of temperature change) or both. The various thermal information is integrated inside the thermoregulatory center, which is, according to the thermostat principle (with a set point at 37 °C), sensitive to any perceived deviation compared to the set point (error signal) and triggers the thermoregulatory reactions involved in the adaptation to increased cold or heat.

The thermoregulatory adjustments required for the maintenance of endothermia aim toward equilibrating the heat gains and heat losses of the human body, and obtaining a heat balance of zero (thermal equilibrium). This balance (B) corresponds to the sum of the internal heat production (M, metabolic) and various heat fluxes (occurring at the skin surface at a pulmonary level): convective (C), conductive (K), radiative (R), and evaporative (E)

$$B = M \pm R \pm C \pm K - E$$

The metabolic heat production (M) associated with cellular activity in a resting condition is minimal (50 $W.m^{-2}$), but can be greatly increased (500 $W.m^{-2}$) during intense physical exercise. Heat, which is almost exclusively produced at the core level, is then carried away towards the skin, the site of thermal exchanges with the environment. This transportation is carried out by either tissue conduction or blood convection:

– *Tissue conduction* ($H_{k,t}$), is the transfer of heat by contact between different tissues, but this is of little importance as this process accounts for only 10–15 % and is reliant upon the formula:

$$H_{k,t} = h_{k,t}\left(T_{co} - \overline{T}_{sk}\right) \cdot A_k \text{ in W}$$

where $H_{k,t}$ represents the coefficient of the tissue conductance, T_{co} the core temperature, $\overline{T}_{sk}$ the mean skin temperature, and A_k the tissue area where heat exchange occurs by conduction.

– *Blood convection* represents the most important means of the core–skin heat transfer, and is reliant upon the following equation:

$$H_c = F_{sk} \cdot c_b \cdot \varrho \cdot (T_{ar} - T_v) \text{ in W}$$

where F_{sk} represents the global skin blood flow, c_b the specific heat of the blood, ϱ the blood density, and $(T_{ar} - T_v)$ the temperature difference between arterial blood and venous blood. Therefore the arterial-venous network is a true heat conducting system, removing heat from the muscles and active organs toward the other parts of the body, although it is mainly at the skin level that heat can be lost.

1 Heat Transfer between the Skin and the Environment

Heat transfer between the skin and the environment occurs by conduction (K), convection (C), radiation (R), and evaporation (E).

– *Heat exchanges by conduction* (K) occur by direct contact between the skin and the environment and are reliant upon the formula:

$$K = h_k(T_s - T_{sk}) \cdot A_k \text{ in W}$$

where h_k represents the thermal conductivity of body parts in contact with each other, T_s the temperature of those parts, T_{sk} the skin temperature in contact, and A_k the contact area.

– *Heat exchanges by convection* (C), used for instance when warming the air of a room (by an electric resistance heater), are reliant upon the formula:

$$C = h_c\left(T_{db} - \overline{T}_{sk}\right) \cdot A_c \text{ in W}$$

where h_c represents the heat exchange coefficient by convection, T_{db} the dry bulb (air) temperature, $\overline{T}_{sk}$ the mean skin temperature, and A_c the area where the exchange takes place. The air velocity increases these convective heat exchanges significantly by modifying h_c according to the formula:

$$h_c = K_c \cdot V_a^{0,6}$$

where K_c depends upon the shape of the body and V_a is the air velocity. In the absence of wind, convection is said to be natural convection; in the presence of wind it is said to be forced convection. Note that the reading of the dry bulb thermometer gives us the exact value of the air temperature.

– *Heat exchanges by radiation* (R) are a little more complex to understand. Any body, living or inert, emits a radiation called radiance (R), which relies upon the formula: $R_t = \varepsilon\sigma T^4$, where R_t is the body emissivity, a is the Stefan-Boltzmann constant (5.67×10^{-8} $W.m^{-2} \cdot K^4$), and T the absolute body temperature in Kelvin (K).

Any body surface which faces any other surface exchanges heat by radiation. Knowing that for infrared heat exchange the skin has an emissivity of almost 1 (0.97), the intensity of the heat exchange between the skin and the environment can be rewritten as follows:

$$R = h_r\left(\overline{T}_r - \overline{T}_{sk}\right) \cdot A_r \text{ in W}$$

where h_r is the linearized heat exchange by radiation, $\overline{T}_r$ the mean radiant temperature of the environment, $\overline{T}_{sk}$ the mean skin temperature, and A_r the exchange surface.

As we generally do not know the value of A_r exactly, we express it as:

$$A_r = \frac{A_r}{A_d} \cdot A_d$$

where A_d is the DuBois area (total skin surface area) given by the formula:

$$A_d = 0,2025 \ M^{0,425} \cdot T^{0,725}$$

where M(kg) = body mass = weight/9.81 $m.s^{-2}$, and T(m) = height; $\frac{A_r}{A_d}$ corresponds to the percentage of the skin area which exchanges heat with the environment, generally 70 % (the remaining 30 % corresponds to the human body parts which exchange between themselves: the inside of the legs, the inside of the arms, between the chin and torso, etc.).

– *Heat exchanges by evaporation* (E) occur when sweat evaporates from the skin surface: for every gram of sweat that evaporates 2.42 kJ are eliminated. This is reliant upon:

$$E = h_c(P_a - P_{sk})A_e \text{ in W}$$

where h_e is the heat exchange coefficient by convective evaporation, which is linearly linked to the heat transfer coefficient, A_e is the evaporating surface, and $(P_a - P_{sk})$ is the difference between the ambient water vapor pressure and the saturated water vapor pressure at skin temperature. P_a generally being unknown, one can write

$$Ac = A_d \cdot \frac{A_e}{A_d}$$

where A_d is the DuBois area (total skin surface area; see above for calculation) and $\frac{A_e}{A_d}$ is the skin wettedness expressed as a percentage. The concept of skin wettedness is interesting because it describes fairly well the severity of the heat strain. A 30 % skin wettedness is associated with discomfort; at 50 % skin wettedness sweat starts to drip from the body; at levels higher than 85 % skin wettedness corresponds to very severe exposure. For any given situation (he, A_e fixed), the heat exchange by evaporation will be proportional to the water vapor pressure difference $(P_a - P_{sk})$. As a consequence, the closer P_a is to P_{sk} (5.8–6.6 kP_a), the less evaporation takes place and the more sweat drips from the body without any efficient thermolytic effect.

In summary, the heat exchanges by K, C, and *R* are called dry heat exchanges, because they are essentially proportional to the difference between the skin temperature and that of the environment (T_{sk}, T_{dh}, and T_r). Heat exchange by evaporation, also called latent heat exchange, is essentially proportional to the water vapor pressure difference between air and skin. These exchanges are linked to the skin temperature and therefore under the control of the thermoregulatory system. They can be assessed for the overall body (because thermal equilibrium is required for maintenance of endothermia) but they can also be assessed locally (because they are at the origin of the thermal sensation and this may cause discomfort).

1.1 Additional Heat Exchanges Originating at the Level of the Respiratory System

Inspired air enters the respiratory tract at the dry bulb temperature, but leaves at approximately 35 °C, thus some heat exchange occurs by respiratory convection (C_{resp}). In the same way, inspired air enters the lungs at a humidity corresponding to the humidity level of ambient air. It leaves completely saturated at 35 °C; thus, some heat exchange also occurs by respiratory evaporation (E_{resp}). These heat exchanges (C_{resp} + E_{resp}) are not regulated because they are not under thermoregulatory control. Their intensity depends totally upon the ambient conditions and the respiratory ventilation rate. They are often negligible, except during intense physical exercise in dry environments.

2 Heat Balance Equation

The heat balance equation $B = M + K + C + R + E$ is defined as the sum of different components, which, by definition, will be allocated a positive (+) value if they correspond to a heat gain, and a negative (−) value in the case of heat loss. Given this, the equation is written:

$$B = M \pm K \pm C \pm R - E \quad \text{in W.m}^{-2}$$

So:

- M is always positive, as it is an internal heat production
- E is always negative, as it always heat loss (except in special cases where condensation could occur on the skin)
- K, R, and C can either be positive or negative depending upon the temperature difference between the skin and the environment.

Therefore, B can either be zero (thermoequilibrium), positive or negative.

In summary, when the heat balance equation is zero ($B = 0$), heat gains and heat losses are balanced and endothermia is maintained.

When heat gains and losses are unbalanced ($B \neq 0$), heat storage (S) can occur: this frequently happens during heat exposure and/or during physical exercise. On the other hand, heat debt (D) will take place especially during exposure to cold. In these two cases endothermia is at risk from the dangers of hyperthermia (heat) or hypothermia (cold).

2.1 Heat Balance in Cold Environments

The zero thermal balance in a cold environment dictates that $M = -R - C - K - E$, which means that the metabolic heat production Mmust offset all the heat losses (R, C, K, and E) to maintain endothermia. Therefore, two thermoregulatory reactions can be triggered against the cold:

- A reduction of heat losses by increasing the cutaneous insulation: peripheral vasoconstriction (diverting blood from the skin) leads to a decrease in the mean skin temperature, therefore reducing the temperature difference.
- An increase in internal heat production by either shivering, which corresponds specifically to muscular activity in the fight against the cold, and can reach between 200 and 250 W.m^{-2}, or by voluntary physical activity (up to 500 W.m^{-2}). Nevertheless, neither shivering nor physical activity can be maintained for long periods of time, rendering man particularly susceptible to the cold.
- Other heat sources may exist, notably the specific dynamic action of food intake, which has negligible importance. Regarding the non-shivering thermogenesis, well known in rodents, it is unlikely to occur and if so only in the case of developing adaptive mechanisms.

In summary, without the help of clothes or heated areas, in humans the cold climate often results in an unbalanced heat equation leading to a heat debt (hypothermia), the importance of which depends upon the intensity of the cold stress, its duration, and the type of environment the body is exposed to (water > air). The skin, thanks to the thermal insulation induced by peripheral vasoconstriction, plays a major role in the reduction of heat loss. It is the excessive physiological insulation that plays a part in the appearance of frostbite. Some adaptive mechanisms appear to reduce the intensity of the skin heat losses; for example, increased subcutaneous fat thickness which reduces peripheral thermal conductivity.

2.2 Heat Balance in Hot Environments

The balanced thermal equation in the heat is written $E = M \pm R \pm C \pm K$, which means that evaporation of sweat is the only means of heat loss compensating for the internal (M) and external ($R \pm C \pm K$) heat gains. Thus endothermia is maintained if $E = M \pm R \pm C \pm K$, and E represents the required evaporation (E_{req}) necessary to achieve zero thermal balance. The ambient evaporative capacity restricts the evaporation of sweat to an E_{max} value, which mainly depends upon the temperature and the humidity of the ambient air. Thus, when $E_{max} > E_{req}$ endothermia can be guaranteed, but when $E_{max} < E_{req}$, heat storage (S) will occur, leading to hyperthermia, the importance of which depends upon the severity of the climatic conditions, the activity level of the person, and the duration of heat exposure. Hyperthermia in the form of heat stroke is a serious condition which often results in death. A limitation of the thermolytic capacity depends upon the maximum sweating capacity of the subject. This can reach between 2 to 3 litres of sweat per hour, but this sweating rate can only be maintained for short periods of time. On the other hand, sweat losses, if not correctly counterbalanced by fluid intake, can lead to dehydration associated with hypothermia, tachycardia, and osmotic imbalance.

3 Conclusion

The skin is the site of heat exchanges between man and his environment. Its capacity to modify the intensity of these exchanges via vasomotor control means that it plays an essential role in the fight against environmental thermal stress. The development of millions of sweat glands allowing secretion and excretion of sweat makes it a unique location for heat loss in warm conditions.

References

General information on body temperature regulation will be found in the following books

Chapter 110 and 111: Cooper KE Basic thermo-regulation – Body temperature control and its disorders. In: Greger R, Windhorst U, editors. Comprehensive human physiology: from cellular mechanisms to integration, vol 2. Berlin/Heidelberg/New York: Springer; 1996. p. 2199–218.