How hot is the human body?

How much body heat is gained or lost during exercise and/or environmental exposure? This seems like a simple question that should be easily quantified; however, this is an unresolved issue that likely has produced flawed deductions concerning thermoregulatory control and treatment affects on body heat content (3, 8, 13, 15).

Body heat content is the product of mean body temperature (Tb) and body heat capacity (body mass × tissue specific heat), with the latter being constant for any given body composition (1, 9). Body heat content changes are usually estimated by measuring body temperature changes (thermometry), and values are rarely measured directly by calorimetry, as the latter approach is complicated and requires extensive sophisticated instrumentation (5). In addition, there is a paucity of data comparing Tb change values measured by calorimetry with values simultaneously estimated by thermometry. For calorimetry, Tb change is calculated from the difference between measured metabolic heat production (indirect calorimetry) and measured (direct calorimetry) heat exchange with the environment (11).

For thermometry, Tb changes are estimated from core and shell (skin and superficial tissues) temperature measurements (2). Core temperature and shell temperature measures are weighted by their anticipated size, which varies reciprocally with cutaneous vasodilation and cutaneous vasoconstriction (2). Traditionally, core/shell weightings of 0.9/0.1, 0.79/0.21, and 0.67/0.33 are used in hot, warm/temperature, and cool conditions, respectively. There is no one uniform “core” temperature, and relationships between internal tissue (e.g., organs and skeletal muscle) temperatures can change as a function of physical exercise and environmental exposure (2, 10). It is known that muscle, brain, and other tissue temperatures are often higher than core temperature during physical exercise and environmental heat exposure, but current thermometry equations do not make adjustments for these underestimated core temperatures. Likewise, there is a paucity of data regarding shell temperature gradients during exercise, heat, or cold exposure. Jay and colleagues (6) recently determined that a three-compartment (muscle, core, and skin temperatures) thermometry equation predicted Tb changes better than the traditional two-compartment (core and skin temperatures) thermometry equation during exercise in temperate and warm conditions; but even when including invasive muscle temperature measurements, this three-compartment thermometry equation accounted for only ~50% of the variance of Tb from calorimetry measurements.

In the present study in the Journal of Applied Physiology, Jay and colleagues (7) constructed a two-compartment thermometry model that employed “adjustment factors” to individually calibrate Tb values from calorimeter measures. Sixty subjects (men and women) were studied in one of the four environmental conditions (n = 10–23 per trial) where they performed cycle ergometer exercise (40% of peak O2 uptake) for 90 min in 24°C or 30°C in 30% or 60% relative humidity conditions. Core and skin temperature measurements were made, and Tb changes were calculated from calorimetry (direct and indirect) and thermometry. In addition, extensive statistical analyses were employed to develop an “adjusted” two-compartment thermometry equation that maximized prediction of simultaneously measured calorimetric Tb changes. The optimal adjusted two-compartment thermometry equations accounted for 56% of the variance when Tb change was calculated from calorimeter measurements. In addition, prediction was best during steady-state conditions and worse during transient conditions. It was concluded that thermometry provides an inaccurate estimate of Tb changes even when extensive customization is employed.

The implications of this study are that thermometry provides an inaccurate estimate of Tb changes, and therefore thermoregulatory models, forcing function analyses of thermoregulatory effecter responses and heat storage/heat debt values based on these measures, are likely flawed. For thermoregulatory models, thermometry measures and weighting factors have been used to determine the drive for heat loss and conservation (8); an example is deriving the sweating rate in the Pierce two-node thermoregulatory model (2). For forcing function analyses, thermometry is employed to determine the central (core) and peripheral (shell) temperature influences on control of skin blood flow, sweating, and shivering (3). Forcing function analysis, in brief, plots the thermoregulatory effecter response as a function of the body temperature changes that occur with some external perturbations (e.g., exercise, environmental temperature). In many cases, the temperature change plotted is mean body temperature, which uses “appropriate” weighting coefficients to determine Tb. From the results of the study by Jay et al. (7), it is of concern that these weighting coefficients are inaccurate and that the use of mean body temperature in forcing function analysis may lead to erroneous conclusions regarding drive and effector-response relationships.

Thermometry has been employed to calculate heat storage and heat debt. Vallerand et al. (14) calculated heat debt during sedentary exposure to cold air with either thermometry or partitional calorimetry and demonstrated a twofold higher calculated heat debt compared with calorimetry. One of the interesting findings from Vallerand et al. (14) is that the weighting coefficients that corresponded to the calorimetry-derived heat debt were 0.82/0.18, values typically assumed for temperate/hot environments and not in the cold. Similarly, the findings for heat storage from Jay et al. (7) reinforce the concept that calculation of mean body temperature for heat storage using weighted coefficients for core and skin temperature is flawed.

These inaccuracies for heat debt and heat storage calculations are accentuated when comparing between different subjects (e.g., lean vs. obese), environments (e.g., warm vs. cold), surface to volume ratios, and transient vs. steady-state conditions (14). To quote from Hardy and Dubois (4), “no one Tb formula can be used under all conditions”; however, we still may not have a suitable Tb formula for any condition. If thermometry is to be used, it should be employed with repeated-measures designs, where it will be adequate to determine treatment effects for heat storage and heat debt. However, it still may yield absolute numbers that are inaccurate. Thus it appears that the advice of Tikuisis (12) might be heeded: “changes in Tb are most accurately determined where the rate
of heat storage is measured via direct/indirect calorimetry,” and the true change in mean body temperature is given by heat storage/debt divided by the body’s specific heat. It is unclear what impact inaccurate Tb values have on thermoregulatory models.

Future research might develop three-dimensional models of Tb by employing modern whole body scanning approaches. If body tissue temperatures (region and depth) were measured by magnetic resonance spectroscopy, and local tissue composition (for specific heat calculations) were measured by dual X-ray absorptiometry, then perhaps accurate three-dimensional Tb formulas could be constructed for different experimental conditions. In a best-case situation, core, skin, and muscle temperatures could be measured by conventional thermometry and entered into a formula based on a three-dimensional model to calculate Tb for any given experimental conditions.

ACKNOWLEDGMENTS

The views expressed in this article are those of the author(s) and do not reflect the official policy of the Department of the Army, Department of Defense, or the U.S. Government.

REFERENCES


Michael N. Sawka
Thermal and Mountain Medicine Division
US Army Research Institute of Environmental Medicine
Natick, Massachusetts
e-mail: michael.sawka@us.army.mil

John W. Castellani
Thermal and Mountain Medicine Division
US Army Research Institute of Environmental Medicine
Natick, Massachusetts