# CONSIDERATIONS FOR THE MEASUREMENT OF DEEP-BODY, SKIN AND MEAN BODY TEMPERATURES

Nigel A.S. Taylor[1], Michael J. Tipton[2] and Glen P. Kenny[3]

**Affiliations:**

[1]Centre for Human and Applied Physiology, School of Medicine, University of Wollongong, Wollongong, Australia.

[2]Extreme Environments Laboratory, Department of Sport & Exercise Science, University of Portsmouth, Portsmouth, United Kingdom.

[3]Human and Environmental Physiology Research Unit, School of Human Kinetics, University of Ottawa, Ottawa, Ontario, Canada.

**Corresponding Author:**

Nigel A.S. Taylor, *Ph.D.*

Centre for Human and Applied Physiology,

School of Medicine, University of Wollongong,

Wollongong, NSW 2522, Australia.

*Telephone:* 61-2-4221-3463

*Facsimile:* 61-2-4221-3151

*Electronic mail:* nigel_taylor@uow.edu.au

**RUNNING HEAD:** Body temperature measurement

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## Abstract

Despite previous reviews and commentaries, significant misconceptions remain concerning deep-body (core) and skin temperature measurement in humans. Therefore, the authors have assembled the pertinent ***Laws of Thermodynamics*** and other first principles that govern physical and physiological heat exchanges. The resulting review is aimed at providing theoretical and empirical justifications for collecting and interpreting these data. The primary emphasis is upon deep-body temperatures, with discussions of intramuscular, subcutaneous, transcutaneous and skin temperatures included. These are all turnover indices resulting from variations in local metabolism, tissue conduction and blood flow. Consequently, inter-site differences and similarities may have no mechanistic relationship unless those sites have similar metabolic rates, are in close proximity and are perfused by the same blood vessels. Therefore, it is proposed that a gold standard deep-body temperature does not exist. Instead, the validity of each measurement must be evaluated relative to one's research objectives, whilst satisfying equilibration and positioning requirements. When using thermometric computations of heat storage, the establishment of steady-state conditions is essential, but for clinically relevant states, targeted temperature monitoring becomes paramount. However, when investigating temperature regulation, the response characteristics of each temperature measurement must match the forcing function applied during experimentation. Thus, during dynamic phases, deep-body temperatures must be measured from sites that track temperature changes in the central blood volume.


**Keywords:** calorimetry, core temperature, mean body temperature, muscle temperature, skin temperature, thermoregulation

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## INTRODUCTION

Given existing reviews on body-temperature measurement (Woodhead and Varrier-Jones, 1916; Selle, 1952; Vale, 1981; Togawa, 1985; Brengelmann, 1987; Sawka and Wenger, 1988; Fulbrook, 1993; Ogawa, 1997; Moran and Mendal, 2002; Ring, 2006; Byrne and Lim, 2007; Pušnika and Miklaveca, 2009; Wartzek *et al.*, 2011; Langer and Fietz, 2014; Werner, 2014), another contribution might seem unwarranted. However, following a presentation designed for students (Taylor, 2011), and arising from a debate on the cooling of hyperthermic individuals (Casa *et al.*, 2010), it became apparent the assumed common knowledge on temperature measurement was not quite so common, nor could its existence be presumed. Therefore, in this communication, the authors aimed to draw together the relevant first principles, along with older and more recent physiological evidence that must be understood and considered when measuring body temperatures.

### First principles in thermodynamics

Homeothermic species employ sophisticated autonomic and behavioural temperature regulatory mechanisms to maintain body temperature within a somewhat narrow range. A vast circulatory network, with counter-current heat exchange capabilities (Bernard, 1876; Forster *et al.*, 1946; DuBois, 1951; Scholander and Schevill, 1955; He *at al.*, 2003), transports and distributes metabolically derived and exogenous heat among the body tissues. These are enclosed within a membrane that permits energy and particle exchanges with the environment. As a consequence, homeotherms are open thermodynamic systems, yet they adhere to the same physical principles governing non-biological energy exchanges, and these first principles moderate physiological processes.

> *"A great deal of misconception could have been cleared by an application of the simple laws of heat flow."* (DuBois, 1951; P 476).

The ***Laws of Thermodynamics*** define energetic relationships within thermodynamically closed (no material exchange) and isolated structures (no material or energy exchange). Whilst humans are rarely (if ever) in those states, these laws still apply, and provide the scientific foundation for understanding temperature measurement. Moreover, they define the principles of heat transfer. Therefore, several salient concepts, and their physiological implications, are highlighted below; readers are also directed to other treatments (Quinn, 1983; Narasimhan, 1999).

The energy possessed and exchanged by animals is made up from dynamic (kinetic) and static forms (potential: mass-, chemical-, nuclear- and force-related energies). This energy cannot be created, nor can it be destroyed. Instead, it may be converted into another form (***First Law of Thermodynamics***), and within a thermodynamic system, it can be used to perform work on another system (external work), transferring energy to that system (Joule, 1850). The total amount of energy possessed by an object is known as its enthalpy, which is minimal (but not absent) at temperatures approaching absolute zero (***Third Law of Thermodynamics***). It varies with the pressure and volume (mass) of each system, and its kinetic component causes sub-atomic and cellular movement and collisions, releasing thermal energy. Thus, heat content is a function of this collision frequency (Worthing, 1941), and it is quantified using temperature measurements and calorimetry.

Consider a closed (inanimate) system with an outer membrane (diathermal wall) permissive to energetic, but not to material exchange. If that system was placed within a stable environment, the collision frequency of its particles would eventually stabilise, and a state of thermal equilibrium (steady state) would exist. The temperature of that object would now be constant, whilst particle motion continued. If another system with a lower enthalpy comes into physical contact with the former, energy will be exchanged across their contacting walls towards the latter. That is, thermal energy moves down energy gradients (***Second Law of Thermodynamics***), either through a change of state (solid, liquid, gas) or via conductive (molecule to molecule), convective (mass flow) or radiative transfers. This establishes thermal gradients within and between these systems, with both systems eventually attaining a common thermal equilibrium. For homeothermic species in a steady state, thermal equilibration among tissues and organs is imperfect. This is because of the continuous and widely variable metabolic heat production and mass (convective) transport of heat that occur throughout the body.

*First principles in a physiological context*

To illustrate the implications of these principles for physiological measurements, a thermodynamically closed system is used (a steel sphere). Its enthalpy was changed from one steady state to another on three occasions. Each trial commenced from a different thermal steady state (stirred water baths: 15°, 25° and 35°C). Following equilibration, the sphere was placed in a water bath regulated at a higher energy level (38.5°C). At its centre

was a temperature sensor, with Figure 1A showing output from that sensor. On each occasion, energy was conducted down a thermal gradient within the sphere, which gained kinetic energy. Eventually, a higher thermal steady state was achieved.

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INSERT FIGURE 1 ABOUT HERE

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During the initial and final thermal steady states only, the sensor was in thermal equilibrium with both the water and the sphere. Since all three systems had equilibrated thermally, then the sensor provided a valid, instantaneous temperature measurement for both the sphere and the water, as long as the latter was precisely clamped. This is the principle of the ***Zeroth Law of Thermodynamics*** that forms the basis of thermometry: when three or more systems are in thermal equilibrium, they have the same temperature. In these circumstances, one can assume all parts of the sphere were at this temperature, although this is the only time this assumption is valid for either inanimate objects or living organisms. Furthermore, because the sensor was at the centre of the sphere, it was not immediately influenced by external energy transfers. These concepts are essential to understanding deep-body temperature measurement. Nevertheless, due to their relatively high resting metabolic heat production, thermal equilibration for mammals pertains only to the internal structures and not to the ambient environment.

During the dynamic phase of each trial, where the sphere asymptotically approached a new thermal equilibrium (Figure 1A: 80-450 s), the sensor furnished no meaningful information regarding either the initial or the final steady state. Instead, it only provided data about its own temperature, and that of the molecules in direct contact with the sensor. From 80-150 s, the coolest sphere gained heat most rapidly (Figure 1A), in accordance with ***Newton's Cooling Law*** (heat-transfer law [Newton, 1700]; heat conduction equation [Fourier, 1807]). That is, temperature change rates are proportional to the size of existing thermal gradients, as illustrated in Figure 1B where these curves are superimposed. This concept is also represented in Figure 1C, where the average rate of temperature change during these dynamic phases (right grouped bars) matches the corresponding thermal gradient (left grouped bars). These rates are also determined by the characteristics of the object (shape,

dimensions, specific mass [density], thermal conductivity, specific heat capacity). These principles help one to interpret temperature measurements during dynamic phases.

Another consequence of Newton's heat-transfer law (Newton, 1700) and the heat conduction equation (Fourier, 1807), was that the final steady-state temperature (38.5°C) was achieved at the same time (Figure 1A). This must always be so, regardless of the initial temperature (Figure 1B). Thus, pre-cooling (or pre-heating) has no influence on either the final temperature or the time at which it was obtained. In humans, a reduction in the pre-exposure, deep-body temperature, induced either through heat adaptation (Taylor, 2014) or pre-exercise cooling (Booth *et al.*, 2004), does not provide a protective advantage by prolonging the time to reach some terminal tissue temperature, as some have indicated (Houmard *et al.*, 1990; Buono *et al.*, 1998; Marino, 2002; Reilly and Waterhouse, 2009; McLellan *et al.*, 2013). This was demonstrated by Booth *et al.* (2004) using pre-cooling, pre-heating and thermoneutral treatments prior to exercise in the heat. These treatments elicited pre-exposure muscle (*vastus lateralis*) temperature differences of 7.5°C, along with deep-body (oesophageal) temperature changes of 1°C either side of the thermoneutral state. Nonetheless, during subsequent exercise in the heat, muscle temperatures converged on a common point, with pre-cooled tissues gaining heat six times faster than pre-heated muscle (0.23°C.min$^{-1}$ versus 0.04°C.min$^{-1}$). This was also reflected in the oesophageal temperature increases (0-20 min: 0.09°C.min$^{-1}$ [pre-cooled] versus 0.03°C.min$^{-1}$). Thus, pre-cooling and pre-heating modified only the rate of heat transfer. These facts become self-evident as one's appreciation of first principles grows.

In some circumstances, skeletal muscle temperature will approximate that of the heart, as in the example above. This is a characteristic of tissues with high perfusion capacities. During cold exposures, however, tissue temperatures will differ markedly due to local heat losses exceeding heat production and delivery. These extremes highlight changes that occur whenever the enthalpy of an organism is moved away from a state of thermal equilibrium, and the further tissues are away from the centre of that organism, the less likely they are to possess the same thermal energy content; so their temperatures must be different. To model these situations, let us consider dynamic phases when temperature sensors are located at different depths within the same sphere.

From the preceding description, one may reason that the centrally located sensor (Figure 1) provided little meaningful information about the temperature of locations closer to the surface of the sphere during non-steady-state conditions. Accordingly, sensors were implanted at 33% (shallow) and at 66% (deep) of the distance between the sphere's surface and centre, and thermal equilibration was again established (38.5°C). Two dynamic phases were now induced in succession: transient cooling followed by protracted re-warming (Figure 2). The sphere was first immersed in cool water (15°C stirred) until the temperature of the central sensor first started to decrease (position A). At that time, the sphere was re-immersed in the warm water, where it remained until all sensors reached 35°C.

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INSERT FIGURE 2 ABOUT HERE

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Not surprisingly, the shallowest sensor cooled earlier and more rapidly to its lowest point (position B: mean cooling rates: $0.13^{o}C.s^{-1}$ [shallow] versus $0.07^{o}C.s^{-1}$ for both the deep and central sensors). This point was 2.7°C cooler than the thermal trough observed for the deep sensor (position C) and 1.9°C cooler than the central sensor (position D), and the times at which these minimal values were realised were dictated by the depth of each sensor. Moreover, once cooling was initiated, it continued for some time after the sphere was returned to the warm water. This is the classical afterdrop phenomenon (Currie, 1797; Alexander, 1945; Golden, 1979), for which, in living creatures, there are both conductive and convective (mass transport) elements (Golden and Hervey, 1981; Webb, 1986; Mittleman and Mekjavic, 1988), although the convective component is minimal at rest in some tissues during hypothermia (Caldwell *et al.*, 2014). It follows, therefore, that an after-rise phenomenon must also exist when hyperthermic individuals are cooled.

Heat loss, once initiated, continued until the thermal gradient no longer existed, or was reversed. For this to occur, the outermost layer of molecules must gain energy from the water, forming a warmer layer (shell). Thus, one can imagine a series of infinitesimally small concentric shells (lamina) being formed that inexorably moved, as thermal fronts, towards the cooler centre until thermal equilibration occurred, with the temperature of each lamina being determined by the energy content of both the upstream and downstream

lamina. Points B, C and D coincided with the arrival of a warm front at each sensor, changing heat losses to heat gains, and successively terminating each afterdrop.

Two additional and instructive characteristics can be observed in Figure 2. Firstly, the between-sensor phase delays from the time of cold-water immersion to the first evidence of sensor cooling (shallow 15 s, deep 25 s, centre 35 s), and from point of warm-water immersion to the end of the local afterdrop (shallow 40 s, deep 70 s, centre 85 s), were a function of the length of the conductive path to the surface of the sphere. These delays are intuitive, and were perhaps first described by Golden and Hervey (1981) using mathematical and physical models. Webb (1986) reproduced these outcomes using gelatin and beef models, with both projects increasing our understanding of the impact of physiological insulation in dynamic thermal environments. Moreover, these outcomes have important interpretative consequences for the measurement of deep-body temperatures when the thermal energy content of the measurement site is primarily dependent upon thermal conduction, as is the case at the rectum. This dependency can be extended to include tissues in which perfusion becomes impaired or arrested, and this was elegantly demonstrated by Golden (1979). Using cold-immersed pigs that were euthanased 20 min following immersion, Golden (1979) observed that the rapid pre-mortem cooling of central venous blood and the oesophagus ended with circulatory arrest. Conversely, rectal tissue cooling, and to a lesser extent gastric cooling, continued at the same rate post-mortem. These observations provide an interesting and surprising insight into the role of the circulation in heat loss in the presence of cutaneous vasoconstriction.

Secondly, points D, E and F indicate times when the temperature traces of two sensors crossed. At these points, the thermal energy content of one sensor changed from being greater to less than that of the second sensor. At position E, the most slowly responding central sensor became cooler than the deep sensor, whilst at position D its temperature fell below the shallow sensor. Similarly, at position F, the deep and shallow sensors crossed. This conductive phenomenon occurs because of the thermal phase delays observed at different depths within the sphere. This same pattern will re-appear later when temperature traces from different deep-body tissues are plotted during sequential cooling and heating treatments (Figure 9), and its significance for temperature measurement will be explored.

## WHY MEASURE TISSUE TEMPERATURES?

Perhaps as an extension of the ***Zeroth Law***, there arose the concept of compartmentalising body tissues into thermally stable deep-body (core) and more variable superficial (shell) structures (Benedict and Slack, 1911; Burton, 1935; Aschoff and Wever, 1958). In this application, thermal equilibration applies only to the deep tissues, and whilst this simplification lacks precision (Burton and Edholm, 1955; Snellen, 1966; Jay *et al.*, 2007a; Kenny and Jay, 2013), it does provide a point from which to introduce this topic.

Under truly basal conditions, the body core tissues are assumed to possess similar amounts of kinetic energy and to be in a state of thermal equilibrium, but not necessarily in equilibrium with the ambient environment. Accordingly, such sites should all be at similar, but perhaps not equal temperatures (Benedict and Slack, 1911; Du Bois, 1941; Bazett *et al.*, 1948; Eichna *et al.*, 1951). This state can also exist within a narrow range of thermal environments when thermal equilibration is achieved above and below the thermoneutral boundaries. Because deeper structures are insulated from the ambient medium by less thermally stable tissues that readily exchange heat with that environment, temperature gradients must exist within and among the deep and superficial tissues (Benedict and Slack, 1911; Bazett and McGlone, 1927; Burton and Bazett, 1936; Barcroft and Edholm, 1946; Pennes, 1948; Eichna *et al.*, 1951; Grayson, 1951). These gradients are parabolic in nature (Benedict and Slack, 1911; Bazett and McGlone, 1927) and dictate heat transfer (Figure 1). Indeed, some regions (*e.g.* hands and feet) have unique characteristics that enable them to behave as physiological radiators (Taylor *et al.*, 2014). Furthermore, thermal gradients are essential, since heat-producing organisms will eventually overheat and perish if insulated from the environment to the extent that all heat loss is prevented.

Beyond basal conditions, variations in the distribution of blood flow mean that the volume of the thermally less-stable body tissues changes. In the classical view, this was considered analogous to changing the dimensions of the body core, which could be created by postural shifts (Rowell, 1986), post-prandial hyperaemia (Granger and Kvietys, 1981), water immersion (Arborelius *et al.*, 1972; Johansen *et al.*, 1997), changes in metabolic rate (*e.g.* exercise: Rowell *et al.*, 1967; Rowell, 1977), ambient temperature manipulations (Rowell *et al.*, 1969, 1970; Caldwell *et al.*, 2014) and anaesthesia (Ingram and Smith, 1970; Deakin, 1998). Thus, neither the temperature nor the dimensions of the core compartment

remain fixed, and these changes influence temperature measurement validity. Furthermore, uncertainty arises when endeavouring to assign a physiological meaning to any one deep-body temperature or when validating surrogate indices. Thus, there is no gold standard deep-body temperature, although if one existed, it must surely be in the form of a central blood temperature:

> "*variations in temperature exist even within the body core and this precludes the temperature of a single organ as a measure of average deep tissue temperature*" (Eichna *et al.*, 1951: P 358).

Instead, the validity of each deep-body temperature measurement must be evaluated relative to one's research objectives, and not to its relationship with another deep-body index or to limitations imposed by unsophisticated ethics committees. In truth, it is more unethical to undertake research using methods unlikely to provide a valid test of one's working hypothesis, either in the laboratory or the field, regardless of measurement simplicity, convenience or subjective preferences. Thus, if one's interest lies with the temperature of a specific body tissue, then measure temperature from that site, or a valid surrogate, whilst ensuring that equilibration and positioning requirements are satisfied. If one has another purpose, then the strengths and limitations of each measurement must be evaluated against that purpose. In the sections that follow, the logic of these propositions is developed in the context of quantifying heat storage, studying clinical states, evaluating central and peripheral thermoafferent feedback and investigating body-temperature regulation. It will be shown that no single index can satisfy every research objective, although some measures are even less likely to achieve that outcome. Before entering that discussion, it must be recognised that changes in heat storage occur more slowly and are governed by the ***Laws of Thermodynamics***, whilst thermal (neural) feedback is provided through a three-dimensional array of receptors that instantaneously respond to stimuli that often act only at discrete locations. Furthermore, the subsequent control of thermoeffector function is often of a whole-body nature, but with site-specific variations in intensity. Therefore, one must expect mean-body temperature measurements to necessarily differ in type and relative importance (weighting) across these research objectives (Simon, 1974; Werner, 2010).

**Measuring heat storage**

All metabolic processes ultimately result in heat production which occurs via cellular

metabolism (bioenergetics) and cell work, with organisms constantly exchanging thermal energy with the environment. Much of our understanding of the exchange processes within an organism, and between it and its thermal environment, is based on data derived from classical calorimetric studies (Atwater and Rosa, 1899; Burton and Bazett, 1936; Kenny and Jay, 2013). When heat loss matches production, there is no nett heat storage, but when this balance is imperfect or disturbed, storage is altered. Thus, one reason for measuring tissue temperatures is to estimate variations in heat storage (partitional calorimetry), so before discussing specific tissue measurements, it is important to elaborate on some principles of calorimetry, measurement interactions with the ***Zeroth Law*** and estimations of heat storage.

*Direct and indirect calorimetry*

Calorimetry is the measurement of heat, which can occur directly using insulated devices (calorimeters: small rooms, boxes, baths, suits) or it can be approximated from changes in respiratory gas exchange (indirect calorimetry [respirometry]). The focus here is upon direct calorimetry. With this technique, the total amount of heat dissipated by the body is measured, including that yielded through aerobic and anaerobic pathways, although the latter is very hard to quantify. The resulting measurements represent the rate that thermal energy is exchanged between an organism and a calorimeter, in the form of a heat flux.

These measurements were introduced in the late eighteenth century by Lavoisier and Laplace (1780), who developed an animal calorimeter (an adiabatic device) from which they derived metabolic heat production from the melting of ice and a knowledge of its latent heat. However, only direct calorimeters measure heat exchange, and in the case of human calorimeters, this represents whole-body heat loss. In their simplest form, calorimeters only measure non-evaporative heat loss (sensible heat loss), and modifications are required for quantifying evaporative (insensible) losses. These devices come in several forms, such as flow, gradient, storage and compensating calorimeters (see: Webb, 1985; Kenny and Jay, 2013). Since our focus will soon turn towards partitional calorimetry, some background information becomes essential.

Unlike thermal conformers (*e.g.* reptiles), mammals are thermal regulators that endeavour to balance heat exchanges with heat production and, in so doing, regulate mean body

temperature (Pembrey, 1898; Cannon, 1929). When homeostatic mechanisms are challenged, regulatory processes that modify heat production and exchange are activated to restore the thermal status of the *milieu intérieur* (Bernard, 1876), or at least to regulate it within physiologically acceptable boundaries (Werner *et al.*, 2008). However, even in basal states, there are continual exchanges of matter and energy across cellular and vascular membranes. From an energetic perspective, this dynamic equilibrium is governed by the ***First Law of Thermodynamics***, as expressed in the heat balance equation (Gagge and Gonzalez, 1996).

It is at this point that the unique contributions of calorimetry become apparent, with whole-body calorimeters providing concurrent measurements of the rates of total heat generation and exchange. These are illustrated in Figure 3A. Lags in the activation of heat loss mechanisms relative to heat gains result in transient thermal imbalances. One such lag is illustrated by the grey zone between the curves, with their difference determining the change in stored thermal energy or heat content (Figure 3B). This change modifies the enthalpy of the body which determines mean body temperature. Indeed, when heat storage is both positive and constant, there will be a continuous increase in mean body temperature. Thus, thermal equilibria require zero heat storage and temperature stability, and it is the peripheral temperatures that stabilise more slowly.

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INSERT FIGURE 3 ABOUT HERE

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*Partitional calorimetry, thermometry and mean body temperature*

Due to the limited availability of direct calorimeters, other methods are often used to estimate body heat storage and exchange. These involve partitional calorimetry and thermometry (Gagge, 1936; Winslow *et al.*, 1936; Hertzman *et al.*, 1952; Stolwijk and Hardy, 1966; Gagge and Hardy, 1967; Snellen, 1969; Vallerand *et al.*, 1992b). The former relies upon the partitioned estimation of dry and evaporative heat exchange avenues, with estimations of the dry exchanges incorporating skin temperature measurements (Winslow *et al.*, 1936; Gagge and Nishi, 1977), which can be obtained by subtraction. When used with indirect calorimetry to estimate heat production, heat storage can be quantified. However, it

can also be estimated using thermometric methods. These rely on the derivation of a mean body temperature, which is most frequently calculated as a weighted summation of deep-body and the average skin temperatures; the two-compartment thermometry model. The weighting (mixing) coefficients for each compartment were originally developed as a function of the parabolic thermal gradient found within the cutaneous tissues, and the assumption of more uniform temperatures within the core tissues (Burton, 1935).

This thermal surrogate of heat storage is typically computed as the product of the change in mean body temperature, total body mass and the average specific heat capacity of the body tissues (Gagge and Gonzalez, 1996). The validity of this approximation is determined by the integrity of the multiplicands. However, imprecision with mass measurements translate into proportional errors, although even when only the initial mass is used, the error remains small when experimental mass changes are moderate. Therefore, the two primary error sources relate to deriving mean body temperature and, to a lesser extent, to the assumption that a common specific heat capacity applies to all individuals, irrespective of body composition.

Since several temperatures are used to calculate mean body temperature, then the first possible error source relates to obtaining, or at least approximating, a thermal equilibrium at the sites of temperature measurement (***Zeroth Law***). Consider Figure 1A, which illustrates a deep-body (deep-sphere) temperature measured from one site only. There are two steady-state zones (0-40 s and beyond 450 s), and valid measurements for deriving mean sphere temperature can be taken in either of those zones. Now consider Figure 2, which has three temperature measures, each from a different location within the sphere. Imagine those sensors provided independent measures of deep-body temperature. Beyond 15 s, steady states did not exist, so which trace provided the most valid measurement? From 75 s to 110 s, the temperature of the shallowest sensor was rising whilst the others were falling. Which is the most valid measurement now? Beyond 150 s, all temperatures were increasing at similar rates. Are these now valid? In reality, correctly calibrated sensors provide valid local measurements, even though neither local nor whole-sphere thermal steady states existed beyond 15 s. However, validity must also be considered with respect to how the information will be used. If the objective was to derive a surrogate for body heat storage, then none of those measurements was valid. For this application, it is

essential to establish an internal thermal steady state for all deep-body measurement sites (Snellen, 1966), and this steady-state requirement also applies to the thermal and moisture characteristics of clothing worn at the time of measurement (Lotens and Havenith, 1995).

Unlike a rapidly responding direct calorimeter, in which dynamic phases are less problematic, the thermometric derivation of heat storage is frequently invalidated under non-steady-state conditions, especially when thermal fronts are moving in different directions. This is illustrated in Figure 2 between points A and D. Prior to point B, it appears as though thermal fronts were only travelling towards the surface of the sphere. Conversely, as soon as the sphere was immersed in warm water (35 s), a warmer front started to move inwards, whilst less-warm fronts continued travelling outwards; positioning sensors closer to the surface would have revealed that outcome. Point B was created by the arrival of the warmer front at the depth of that sensor. Beyond 75 s, that sensor was storing heat. Yet the deep and central sensors were still losing heat. Therefore, both position and time determined whether or not each sensor was gaining or losing heat.

Clearly, the validity of thermometric computations of heat storage are dependent upon establishing steady-state conditions. How long does this take in humans? In sections that follow, it will be shown that thermal equilibration for healthy individuals of normal body mass resting under thermoneutral conditions varies from 30-70 min. During constant-load exercise within thermally compensable conditions, this may take 30-45 min (Kenny and Jay, 2013).

A second error source accompanies the assumption the body can be sub-divided into two thermal compartments when determining mean body temperature; a stable core and a less-stable shell. It is well accepted that using only a deep-body temperature to compute heat storage is erroneous (Burton, 1935), so most investigators use temperatures obtained from two compartments. Yet despite its wide use, several groups have suggested this approach can result in significant underestimations of heat storage (Vallerand *et al.*, 1992b; Snellen, 2000; Jay *et al.*, 2007a, 2007b; Sawka and Castellani, 2007). Comparisons using direct calorimetry showed those errors increased when moderate intensity exercise (~40% maximal aerobic power) was performed in warmer conditions (*i.e.* 15.5% at 24°C versus 35.5% at 30°C). Adding more deep-body measurements improved the estimation (rectum,

oesophagus, auditory canal), as did changing the compartmental weighting coefficients (Jay *et al.*, 2007b, 2010). Nevertheless, those modifications still only accounted for ~56% of the variance in heat storage observed using direct calorimetry. Including skeletal muscle temperatures may prove helpful, since muscle constitutes a large fraction of the total mass (36-45%; female-male [healthy, active individuals]) and it has a high specific heat capacity (3.590 $kJ.kg^{-1}.{}^{o}C^{-1}$; González-Alonso *et al.*, 2000). Therefore, not only is muscle a significant heat source during exercise, but it represents an important heat sink. For instance, the combined relative mass of bone and adipose tissue (30-40%; male-female) would have an average specific heat capacity approximately half that of muscle (1.802 $kJ.kg^{-1}.{}^{o}C^{-1}$; Karmani, 2006; Foundation for Research on Information Technologies in Society, 2013). Thus, muscles can store almost twice as much heat. Collectively, the studies above show that the current thermometric models for estimating changes in body heat content and rates of heat storage are inaccurate, even when used under steady-state conditions.

These inaccuracies are likely to be accentuated when comparing subjects with diverse body compositions and morphological configurations (Livingstone *et al.*, 1987; Chudecka *et al.*, 2014), and even during different thermal environments (Burton and Edholm, 1955; Vallerand *et al.*, 1992a), where the weighting coefficients for the deep-body and shell compartments are necessarily modified. Attention here is directed only at variations in body composition, and it is accepted that the average specific heat capacity of humans is 3.47-3.49 $kJ.kg^{-1}.{}^{o}C^{-1}$ (Pembrey, 1898; Gephart and DuBois, 1915; Burton, 1935; Hardy and DuBois, 1938; Lipkin and Hardy; 1954). This varies among tissues, so errors must occur when individuals deviate from that standard, as is the case for contemporary populations. For example, for each $1^{o}C$ change in mean body temperature, changes in body composition among individuals of the same mass result in proportional changes in both the whole-body specific heat and heat storage. Thus, exchanging muscle for adipose tissue reduces the whole-body specific heat capacity and heat storage equally. The reverse also pertains, with these potential errors adding to those already described. Not surprisingly, considerable caution is required when deriving and interpreting body heat storage from mean body temperature measurements.

**Studying clinically relevant states**

Arnold (1840) was perhaps the first to investigate the relationship between body temperatures and illness, collecting data over 25 years from healthy and infirmed individuals. It is now appreciated that significant variations in both deep-body (Hunt, 1844; Wunderlich, 1869; Wunderlich and Seguin, 1871; Woodhead and Varrier-Jones, 1916; DuBois, 1941; Eichna *et al.*, 1951; Taylor *et al.*, 1994; Blatteis, 2007) and superficial tissue temperatures (Korpelainen *et al.*, 1995; Lavery *et al.*, 2004; Arora *et al.*, 2008; Eglin *et al.*, 2013) are associated with pathological states, and this is another reason why tissue temperatures are measured. Indeed, the extent of these thermal changes often reflects the severity of the condition and its prognosis.

Accordingly, relevant and accurate temperature measurement during the acute phase of some conditions is important. Unfortunately, when significant thermal gradients exist, deep-body measurement is challenging, and it can become very difficult for some of the most clinically relevant sites. Furthermore, depending upon the site chosen, its temperature can reflect whole-body heat storage or simply the temperature of blood irrigating those tissues and their local metabolic rate. Finally, temperature measurement may be compromised in emergency situations to facilitate treatment, resulting in the use of more remote measures (Barnason *et al.*, 2012).

Some clinical conditions are both instructive and relevant to this communication, and one such example relates to near-drowning in very-cold water. In this situation, aspirating cold water ($<6^{o}C$) into the lungs rapidly cools the heart, carotid artery blood and the brain. Using a live animal model, Conn *et al.* (1995) demonstrated that drowning in cold water ($4^{o}C$) produced a 7.5-8.5$^{o}C$ reduction in carotid blood, and therefore brain, temperature in as little as 2 min, whereas immersion without drowning resulted in a fall of $<1^{o}C$ over the same period. However, in both conditions, rectal temperature remained relatively stable. This very rapid reduction in brain temperature provides hypothermia-induced protection against hypoxia that can sometimes permit prolonged submersion ($>30$ min) with full recovery. In these situations, it is brain temperature, and certainly not that of the rectum, that is the clinically relevant and critical deep-body index, as it determines the likelihood of survival and the rationale for continued resuscitation (Tipton and Golden, 2011). The principle highlighted by this example applies to many scenarios including localised infections (Dellinger *et al.*, 2013), as well as to therapeutic, whole-body and regional

cooling (Bernard *et al.*, 2002) or heating (Alphandéry, 2014). In such circumstances, targeted temperature monitoring is essential (Werner, 2014).

**Quantifying thermoafferent feedback**

Tissue-temperature measurements are also used to estimate thermoafferent flow (feedback) from the deep and superficial thermoreceptors (Magoun *et al.*, 1938; Zotterman, 1959; Hardy *et al.*, 1964; Boulant, 1996; Pierau, 1996). Receptor feedback is integrated within the hypothalamus, with that arising from the deeper tissues generally exerting greater influence over organs participating in body-temperature regulation (Stolwijk and Hardy, 1966; Proppe *et al.*, 1976; Jessen, 1996; Werner *et al.*, 2008). However, feedback from the skin is important during thermal adaptation (Regan *et al.*, 1996; Tipton *et al.*, 2013; Taylor, 2014) and when its temperature changes very rapidly (Brown and Brengelmann, 1970; Nadel *et al.*, 1971; Libert *et al.*, 1978; Tipton, 1989). Nevertheless, direct measurement of afferent flow is challenging, so it is not unreasonable to assume that if one could measure the static and dynamic thermal characteristics of the tissues in which these receptors are positioned, then one may obtain a meaningful surrogate of thermoafferent feedback (Nadel *et al.*, 1973; Libert *et al.*, 1984; Cotter and Taylor, 2005).

There are several qualifications that must be addressed before this assumption can be accepted. Firstly, temperature sensors must be embedded within tissues that remain in equilibrium with the thermoreceptors. Those receptors must not be damaged nor can localised responses (*e.g.* hyperaemia) be initiated that will change local tissue temperatures (reactive error: modifying variables through the act of taking measurements). Secondly, each sensor must be equilibrated with the local tissue temperature. Thirdly, the sensors must not be influenced by conditions beyond those that elicit an equivalent modification to the energy content of the target tissues. Fourthly, sensors must be small enough and sensitive enough that response times do not lag far behind those of the thermoreceptors. Whilst sensors must be in thermal equilibrium with the surrounding tissues, the thermal status of the whole body will not necessarily be in the same thermal state, although this is not a requirement for research of this nature. Fifthly, chemicals (neurotransmitter analogues) exist that can stimulate thermoreceptors without a change in temperature (*e.g.* menthol [a monocyclic terpene alcohol]), and this interaction disassociates temperature from thermoreceptor feedback (Gillis *et al.*, 2010). Finally, the sensor distribution must be

sufficiently extensive to faithfully represent the thermoreceptive field(s) of interest. From these considerations, it is evident some temperature measurements no longer rely upon a strict application of the ***Zeroth Law***. Instead, dynamic phases become the primary focus.

These are very demanding qualifications. Whilst they are not unreasonable, in practice, they are rarely satisfied. For physicists, adherence is often paramount. For physiologists working with open thermodynamic systems with heat-producing elements and extensive, three-dimensional arrays of deep-body (Simon *et al.*, 1986; Villanova *et al.*, 1997; Boulant, 1996) and superficial thermoreceptors (Ivanov *et al.*, 1986; Pierau, 1996), the task would seem impossibly difficult, even in animal models. Nevertheless, all who take such measures should appreciate the significance of these qualifications, and their susceptibility to violation, if indices of thermoreceptor feedback are sought.

**Investigating body-temperature regulation**

The final topic of this section relates to investigating homeostasis. Since all tissues providing thermal sensory feedback are themselves parts of the regulated body mass (Simon, 1974; Jessen, 1996), then changes in those tissue temperatures provide essential information to those with an interest in body-temperature regulation. While the deep-body tissues dominate this feedback (Stolwijk and Hardy, 1966; Proppe *et al.*, 1976; Jessen, 1996; Werner *et al.*, 2008), it would be wrong to suggest that shell tissues are excluded from that regulated mass, as they too are components of the thermal feedback loops. Accordingly, one might also expect to see thermoeffector function tracking variations in shell temperatures. Thus, a weighted combination of simultaneously measured, rapidly responding deep-body and shell temperatures might provide insight into temperature regulation (Stolwijk and Hardy, 1966); the regulated mean body temperature. However, the weighting coefficients for its deep-body and shell constituents may differ from those used to describe body heat storage (Werner, 2014).

An example of the deeper-tissue relationships with effector function is illustrated in Figure 4, with sudomotor responses during a sinusoidal (24-min cycling) forcing function overlayed onto three deep-body temperatures and one active intramuscular temperature (*vastus lateralis*; Todd *et al.*, 2014). The sweat rate trace is the average of five local measurements (sweat capsules), and the merits of each temperature measurement will be

discussed later, but several features are noted here. Firstly, four variables tracked the sinusoidal function, although not all with equivalent precision. Secondly, the sweating response appeared to be tracking temperature changes reflected by both the oesophageal and intramuscular indices, whilst preceding those observed via the auditory canal. Thirdly, the intramuscular and oesophageal temperature peaks preceded those for sweating, with respective average phase delays of 26 s and 47 s (Todd *et al.*, 2014). Fourthly, two deep-body locations transiently stored and lost heat, returning to approximately equivalent peaks and troughs, while rectal temperature increased progressively over the trial ($>0.2^{o}$C). It is not unreasonable, therefore, to conclude that index provides less meaningful information concerning thermoeffector control during some dynamic phases, as reported by others (Benzinger and Taylor, 1963; Piironen, 1970; Brengelmann, 1987).

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INSERT FIGURE 4 ABOUT HERE

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Within another, non-steady-state experiment (Cotter *et al.*, 1995), four different deep-body temperatures appeared to provide similar, but not equivalent information (Figure 5), although the forcing function was not designed for teasing out differences among these variables. In this case, subjects were cycling at a constant work rate in the heat (36.5$^{o}$C), so neither the temperatures, which rose monotonically, nor the sweating responses, which increased asymptotically, were stable over the first 10-20 min. Indeed, local sweat rates began to plateau as each site approached its maximal flow. Thus, once those temperatures were uncoupled from the sudomotor responses, they ceased providing useful information concerning the neural control of sweating. Instead, they now reflected heat storage.

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INSERT FIGURE 5 ABOUT HERE

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Under steady-state conditions, another set of relationships exists, and an internal thermal equilibrium is approximated. This occurs within thermoneutral conditions, but it is also present during regulated hypo- and hyperthermic states when deep-body temperatures are

stable. In these circumstances, most deep-body temperatures provide the same information, even though local temperatures may vary. Thus, whilst rectal temperature is inadequate in certain dynamic phases (Figure 4), it is perfectly acceptable during steady states.

One may therefore conclude that, when investigating body-temperature regulation, the choice of a temperature index must be made after considering the forcing function used to provoke the homeostatic disturbance. During some manipulations, it is absolutely critical to measure temperature from a site that rapidly tracks changes in central nervous system and cardiac temperatures (Cooper *et al.*, 1964; Hayward *et al.*, 1984). In other, more slowly changing states, this requirement becomes less important.

## INDICES OF DEEP-BODY TEMPERATURE

The preceding overview provides a scientific rationale against which one may evaluate the methods for measuring deep-body temperature. Such measurements are taken by positioning sensors within, or in contact with, deep-body structures. Whilst some specialised organs have high metabolic rates (*e.g.* brain, liver, heart, kidneys) and are the primary heat sources, less-active tissues behave as short-term heat sinks (*e.g.* bone, adipose, skin), particularly during states of altered heat storage. Moreover, even under steady-state conditions, there is a non-stop flow and redistribution of thermal energy throughout the body via convective and conductive mechanisms, before its excess is dissipated to the environment. Consequently, the idea that the entire body can achieve a state of thermal equilibration is an over-simplification. During exercise, skeletal muscle metabolism can be dramatically elevated, altering heat production and the distribution of both blood flow (Saltin *et al.*, 1998; Tschakovsky and Pyke, 2008) and thermal energy. This complicates temperature measurement and its interpretation.

Not surprisingly, many body regions have different steady-state temperatures (Bernard, 1876 [animals]; Horvath *et al.*, 1950b [dogs]; DuBois, 1951 [humans]; Eichna *et al.*, 1951 [humans]; Minard and Copman, 1963 [humans]; Houdas and Ring, 1982 [humans]). Some temperatures will be equivalent due to similarities in heat production and removal, whilst others may be equivalent even though they produce vastly different amounts of thermal energy. Thus, these tissue temperatures are turnover indices resulting from changes in three variables (tissue conduction, local metabolism, local blood flow), each of which can be

independently modified. Accordingly, unless any two sites are in close proximity, are perfused by the same blood vessels and have the same metabolic rate, then it is highly unlikely that the temperature of one will bear any mechanistic relationship with that of the other, regardless of their numerical value. Nevertheless, each temperature may provide a valid reflection of local heat storage. In this section, intra-regional differences in how these qualifications apply to various deep-body temperature indices will be explored.

**Comparisons among measurement sites**

Body temperature measurement can be traced back to the eighteenth century. While the relationship between energy, heat and work (Joule, 1850) had not yet been recognised, others were exploring the temperature of the body, and its local and situational variations (Martine, 1740; Currie, 1797; Currie and Percival, 1792; Becquerel and Breschet, 1835; Beaumont, 1838). During the nineteenth century, the classical works of Wunderlich (1869) and Bernard (1876) led to the view that the resting body core temperature was typically 37°C (98.6°F), which it is not, while Liebermeister (1875) and Pembrey and Nicol (1898) advocated measuring deep-body temperature via the rectum due to its thermal stability.

From this legacy arose detailed descriptions of the variability in deep-body temperatures in resting, normothermic individuals. The most extensive contribution was that of Eichna *et al*. (1951), added to by Minard and Copman (1963) and Houdas and Ring (1982). In Figure 6, these temperature distributions are further embellished, although the message remains unchanged: deep-body temperature varies with location.

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INSERT FIGURE 6 ABOUT HERE

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The classical view was that the rectum is the hottest site (Bernard, 1876; Pembrey and Nicol, 1898; Haldane, 1905; Horvath *et al*., 1950a; Eichna *et al*., 1951), although most commentators only considered tissues positioned to the left of rectal temperature in Figure 6. From that perspective, the classical view is upheld, but when further sites were explored, a slightly different picture emerged. Notwithstanding the restrictions of comparing data collected from different population samples (Figure 6), with limited numbers for some sites

and with most studies contributing data for three or fewer sites, it is now recognised that the rectum is not the hottest site. Instead, brain tissues are the hottest (Hayward *et al.*, 1966; Shiraki *et al.*, 1988; Mellergård, 1994), with thermal gradients (Fusco, 1963; Mellergård, 1994) permitting conductive heat loss from the deeper to the cortical tissues. The critical point is that heat loss is essential to life. This heat transfer relies upon thermal gradients, and these dictate the existence of temperature variations throughout the body. Therefore, it is important that each deep-body temperature is described using site- and depth-specific identifiers.

**Sources of variability among deep-body temperature measurements**

> "*My hardest task during these last six months has been to find the average normal body temperature and the average diurnal range. All the best workers who have written on this subject have carefully avoided giving these figures.*" (DuBois, 1941; P 25).

Whilst homeothermic species evolved with a capacity for body-temperature regulation (Grigg *et al.*, 2004), there is no fixed temperature (set-point) that regulatory systems are striving to achieve (Brown and Brengelmann, 1970; Mekjavic and Eiken, 2006; Werner *et al.*, 2008). Indeed, there is no anatomical location in which resides some reference thermal state against which all instantaneous temperatures are compared. Instead, every stable mean body temperature is merely the result of a balance between heat transfer and the autonomic regulatory processes that defend mean body temperature (Werner *et al.*, 2008). Consequently, across population samples, one finds a wide distribution of basal, deep-body temperatures measured at the same site (Figure 7: DuBois, 1941; Ivy, 1944; Eriksson *et al.*, 1985; Sund-Levander *et al.*, 2002), the average of which can give the false impression of a common target. However, such apparent normality is a "*statistical abstraction*" (Minard and Copman, 1963; P 527), with dispersal about the mean reflecting instantaneous differences in feedback and effector activation, in combination with variations in body dimensions, specific mass, thermal conductivity, specific heat capacity and diurnal fluctuations in heat production and exchange. Thus, chronic neural disturbances can modify the normal temperature distribution among and within the deep tissues. For example, in resting (supine), spinal-cord injured individuals ($N$=9; C4-L5), the inter-site temperature variations normally observed in able-bodied people are reduced: oesophagus 36.7°, auditory

canal 36.6º and rectum 36.6ºC ($P > 0.05$; Wilsmore, 2008). Gass *et al.* (1988) reported similar changes in the oesophageal and rectal temperatures of paraplegics during wheelchair propulsion, and these outcomes presumably resulted from an altered balance between heat production and exchange (Pembrey, 1898; Sawka *et al.*, 1989).

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INSERT FIGURE 7 ABOUT HERE

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Depending upon the temperature index used, its distribution among individuals can vary in width and skewness (Figure 7), and the variation within a site can be as much as 1.5-2.4ºC (DuBois, 1941; Ivy, 1944; Eriksson *et al.*, 1985). A narrow distribution might be considered advantageous, even though it can also be a characteristic of sites that are less responsive to thermal transients. In some circumstances, this too may be desirable. The second feature of these distributions (skewness) has implications for the assumptions of some statistical procedures. Indeed, it may even lead one to question the merits of normality testing for data obtained from relatively small population samples.

Apart from this inter-individual variation, most deep-body temperatures undergo significant changes throughout daily and, in women, monthly menstrual cycles. Before pursuing these cyclical changes, it is necessary to return briefly to first principles. Within deep-body tissues, radiative and phase change heat transfers do not occur. Instead, each local temperature is the result of local heat production, and convective and conductive exchanges. For inanimate objects, the metabolic and convective elements do not exist. For poorly perfused tissues, convective heat flow may also be negligible. In this circumstance, the balance between local metabolism and tissue conduction determines local tissue temperatures. For instance, during protracted cold exposure, hand and foot blood flows can fall below those necessary to support metabolism (Abramson, 1965; Caldwell *et al.*, 2014). Thus, deep-hand or -foot temperatures bear little resemblance to intrathoracic temperatures. This physiological amputation highlights the need to consider tissue blood flow for every site from which one endeavours to measure deep-body temperature. Indeed, that blood flow is dictated by each organ's physiological role and metabolic rate. Therefore, the consequences of low perfusion in conduit structures used for measuring deep-body

temperatures (*e.g.* gastrointestinal tract, auditory canal) must be considered, and this qualification is relevant to most sites in Figure 6, with half of those representing well-insulated tissues with local blood flows that vary inversely with exercise intensity and changes in deep-body temperature (Rowell *et al.*, 1967, 1969, 1970). Consequently, it is theoretically possible for the temperature of such sites to increase in parallel with, but not due to, a rise in central blood temperature. This can be explained simply on the basis of reducing heat removal from a well-insulated, heat-producing organ (Molnar and Read, 1974), and this point arises again in a subsequent section.

Our first source of temperature variation relates to the time taken for thermal equilibration. For those investigating whole-body metabolism, the requirements for attaining a true basal state are rigorous (*e.g.* 10-h fast, overnight sleep, extended supine rest in thermoneutral conditions), as variations due to extraneous influences need to be minimised. Similarly, although much less demanding, thermal equilibration to each new steady state requires time. For instance, in thermoneutral conditions, the whole-body equilibration time for resting (seated) individuals varies from 30-70 min (Cranston *et al.*, 1954; Vallerand *et al.*, 1992b), during which deep-body temperatures approach stability. This is generally observed as a temperature reduction, and it dictates sufficiently long preparatory times before data collection. For laboratory research, this is easily satisfied. In the field, it is more frequently infringed than it is observed. Moreover, when exercising at a fixed work rate, and therefore at a constant rate of heat production, equilibration time will vary with both exercise intensity and the rate of dry heat exchange. These also have experimental implications, and when heat storage is the focus, equilibration is critical.

Within individuals, the nychthemeral (circadian) variations in brain temperature (parahippocampal gyrus) can be in excess of 0.5°C (Landolt *et al.*, 1995). Cyclical patterns in deep-body temperature have also been described, peaking around 17:00 h with a trough observed close to 04:00 h (Pembrey and Nicol, 1898; Richet and Langlois, 1898; DuBois, 1941; Smith, 1969; Reilly and Waterhouse, 2009). This pattern is evident across species and is linked to nocturnal behaviours (Simpson and Galbraith, 1905). The thermal trough occurs during sleep, with deep-body temperature falling while skin blood flow rises before sleep commences (Campbell and Broughton, 1994; Baker *et al.*, 2001). Variations of a similar magnitude are associated with the menstrual cycle (Rubenstein, 1937; Buxton and

Atkinson, 1948), with a lower temperature observed prior to ovulation (follicular phase), rising at ovulation and remaining elevated during the luteal phase (Coyne *et al.*, 2000; Baker *et al.*, 2001). These cyclical variations in naturally cycling, pre-menopausal women (20-45 y) have a significant and time-dependent impact on all deep-body temperatures.

Lower basal, deep-body temperatures are also observed in endurance-trained (Holmgren *et al.*, 1960; Shvartz *et al.*, 1974; Baum *et al.*, 1976) and heat-adapted individuals (Dreosti, 1935; Ladell, 1951; Buono *et al.*, 1998; Patterson *et al.*, 2004). Those variations are slightly smaller (0.3-0.4°C), and are hypothesised to be due to reduced body heat storage. Since this varies inversely with heat loss, then an elevated resting cutaneous blood flow will lower the basal mean body temperature (Werner, 1994). During exercise, a lower mean body-temperature threshold for sweating, as well as an elevated sudomotor sensitivity and peak sweat flows, combine to lower body temperature at the same fixed work rate following adaptation (Werner, 1994; Kenny and Jay, 2013; Taylor, 2014).

Postural variations have also been described, with liver, rectal, sublingual, gastric and tympanic temperatures all rising when moving from supine to standing rest, and decreasing when reclining (Cranston *et al.*, 1954; Graf, 1959; Ogawa *et al.*, 1993), although these changes are not necessarily observed during exercise. For instance, during continuous upright cycling (70 min), Greenleaf and Castle (1972) reported higher auditory-canal temperatures relative to similar supine exercise, whilst rectal temperatures were lower.

**Blood temperatures**

> "*Not only is the tissue temperature variable even in homoiotherms, but important variations in arterial blood temperature are common*" (Bazett *et al.*, 1948; P 18).

Much to the chagrin of cardiovascular physiologists, thermal physiologists often consider the heart as a thermal mixing chamber. Indeed, the heart accepts and mixes venous blood from all regions before delivering it to the lungs, where its temperature change is negligible due to counter-current heat transfers (Eichna *et al.*, 1951; Mather *et al.*, 1953; Bligh, 1957b; Walker *et al.*, 1961; Jaeger *et al.*, 1980), although not all hold this view (Kiyatkin, 2007). It then distributes this thermally homogenised fluid to all parts of the body.

Therefore, the temperature of the blood leaving the left ventricle is close to that encountered by the downstream deep-body tissues, though it loses heat *en route*, and the time constant for dynamic thermal changes at those sites is a function of local blood flow (Piironen, 1970).

Notable exceptions to this generalisation occur within metabolically active organs, in which blood behaves as a physiological coolant, increasing in temperature as it removes heat, or in tissues where perfusion is transiently impaired or arrested, with heat exchanges now becoming conduction dependent (Golden, 1979). Examples of such vascular and tissue heat exchanges are presented in Figure 8, with carotid arterial blood being 0.3-0.4°C cooler than that within the jugular veins. This gradient across the brain obtains during rest and exercise (Baker *et al.*, 1972; Nybo *et al.*, 2002), although this relationship can differ in species displaying selective-brain cooling (*e.g.* sheep: Maloney *et al.*, 2001). Similarly hot venous blood from the liver is observed (Figure 8), with high local blood flow preventing hepatic overheating. In fact, it was suggested that the liver is cooler than the rectum simply due to local differences in heat balance (Graf, 1959). Foetal temperatures are also 0.3-0.4°C warmer than the uterus (Houdas and Ring, 1982). This must be so since the foetal and maternal circulatory systems are not connected, and conductive heat heat loss must occur down the thermal gradient and across the membranes separating the placenta and the uterus. Conversely, the limbs, and particularly the hands and feet (Taylor *et al.*, 2014), facilitate heat dissipation in resting individuals, as illustrated by the 0.3°C reduction in blood temperature between the femoral artery and vein (see Davy [1814] and Pembrey [1898]).

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INSERT FIGURE 8 ABOUT HERE

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It has been suggested that the intra-cardiac temperature (Eichna *et al.*, 1951) and pulmonary arterial blood (Bligh, 1957b, 1973; Brengelmann, 1987) provide a good indication of the average temperature of the body core. Since minimal heat exchange occurs across human lungs, then so too would aortic blood, along with that in its immediate branches: the carotid arteries. Most accept these propositions, and this is certainly the view of the current authors, although few have the capability to undertake these temperature measurements,

forcing reliance upon surrogate indices. These indices will now be discussed with respect to both first principles and hypothesis testing.

**Sublingual (oral) temperature**

Sublingual temperature has historical precedence. Arnold (1840) undertook extensive research (25 y), noting "*there are generally one or two degrees of heat* [°F] *between experiments under the tongue and in the axilla, which was always found the lowest*" (Arnold, 1840; P 76). From his afebrile subjects, Arnold (1840) observed a mean sublingual temperature of 36.8°C ($N$=107), whilst Whiting (1915) subsequently recorded an average of 36.9°C ($N$=601). Both values seem high relative to Figure 6, but not Figure 7, and although the latter procedure appeared valid ("*under tongue with closed mouth for three minutes*" P 2), neither basal conditions nor thermal equilibration could be assured.

The primary limitation of the sublingual index is the evaporative cooling of the buccal cavity accompanying breathing (Pembrey, 1898; Haldane, 1905), the after-effect of drinking (Woodman *et al.*, 1967) and heat exchange through the cheeks (Sloan and Keatinge, 1975). Heat exposures seem not to be a problem (Mairiaux *et al.*, 1983; Amano *et al.*, 2013), and nasal breathing of sufficient duration allows the sublingual mucosa to equilibrate with deep-body temperatures.

The tongue receives an abundant blood supply (Hellekant, 1976) via the bilateral lingual arteries which branch from the external carotids, giving rise to the suprahyoid, dorsal lingual, deep lingual and sublingual arteries (Standring, 2008; Hansen and Netter, 2010). This ensures the sublingual space is potentially supplied with blood at the same temperature as that delivered to the brain. But since the lingual veins (dorsal lingual, deep lingual, sublingual) are in intimate contact with those arteries (Standring, 2008; Hansen and Netter, 2010), then arterial blood may be cooled (counter-current exchange). Nasal breathing minimises this effect, but the bias introduced by cold exposure (Sloan and Keatinge, 1975) is harder to counteract. Moreover, ensuring nasal-only breathing during longer-duration exercise can create difficulties.

Under ideal conditions, sublingual temperature might provide a valid surrogate of deep-body temperature. Indeed, Cranston *et al.* (1954) reported this index to be more responsive

to dynamic phases than rectal temperature, as did Piironen (1970) and Daanen (2006). Edwards *et al*. (1978), Mairiaux *et al*. (1983) and Brengelmann (1987) suggested it was on par with oesophageal and tympanic-membrane temperatures, although Mairiaux *et al*. (1983) did report a slightly sluggish response. Accordingly, it is important to know how long nasal breathing must continue to achieve sublingual thermal equilibration among the mucosa, deeper tissues and thermometer (***Zeroth Law***).

This question was perhaps first investigated by Benedict and Slack (1911), finding that 7 min was necessary for equilibration. In a more extensive experiment, Nichols and Kucha (1972; $N=390$) reported the time to achieve the highest sublingual temperature in 90% of subjects was 11 min. Whilst others have used longer equilibration times (>20 min: Cranston *et al*., 1954; Sloan and Keatinge, 1975), some sampling periods are far too short (3 min: Strydom *et al*., 1965; Togawa, 1985). Surprisingly, some investigators undertaking validation experiments have neglected reporting equilibration times (Edwards *et al*., 1978; Mairiaux *et al*., 1983; Mackowiak *et al*., 1992; Ganio *et al*., 2009), rendering the validity of those observations uncertain.

**Axillary temperature**

Historical precedence also applies to axillary temperature (Arnold, 1840). However, the further one moves away from the deep-body tissues, the more variable is the tissue temperature (Benedict and Slack, 1911; Burton, 1935), and the less likely it is to track the thermal status of the core. As a consequence, axillary temperature is often the lowest index (Arnold, 1840 [equilibration time not reported]; Machino, 1959; Ilsley *et al*., 1983 [equilibration time not reported]; Erickson and Woo, 1994; Figure 6). When seeking surrogate indices, it is best to avoid values at either end of a data range, for, according to error theory (Beers, 1953), such data are least likely to be correct. Whilst the strict application of this theory is inappropriate here, one must ask whether or not it is wise to choose a representative temperature index that differs markedly from most deep-body sites.

Most accept this criticism. Nevertheless, the axilla has features that support its use. For instance, the axillary arteries are continuations of the left subclavian and right brachiocephalic arteries that arise from the aortic arch (Standring, 2008; Hansen and Netter, 2010). These vessels carry blood from the deep tissues, and since they are well

insulated, this blood will initially have similar thermal characteristics, but will be modified by counter-current exchanges. Of greater concern, however, is the possibility that lower axillary temperatures may result from inadequate equilibration. Machino (1959) showed equilibration time was ~20 min, at which point axillary temperature was 0.2°C higher than measured at 10 min. Clearly, data collected within this 20-min interval are invalid as they do not conform with the requirements of thermometry. To the current authors' knowledge, systematic research has not been undertaken using this index when all sources of variability were minimised.

**Rectal temperature**

It is perhaps appropriate to commence this section with opposing views, separated by about 50 years, since 50 years further on, some still passionately defend the former opinion.

> "… *rectal temperature, which under ordinary conditions gives the highest readings, undoubtedly gives the truest indications of the internal body temperature* …" (Haldane, 1905; P 495).
>
> "… *it seems no longer justified to substitute a temperature in the lower pelvis or intestine for the central temperature actually sought*" (Benzinger and Taylor, 1963; P 111).

Similarly high brain and rectal temperatures (Mariak, 2002) could be considered a justification for using the latter as a surrogate index. However, the brain is hot because of its high metabolic rate, whilst the rectum is hot due its thermal inertia, which results from a low perfusion and heat removal; it is not due to bacterial activity (Rubin *et al.*, 1951; Durotoye and Grayson, 1971). Indeed, the dissociation between these temperatures was illustrated by Tanabe and Takaori (1964: rabbits), who modified carotid blood temperature, showing brain and tympanum temperatures to be similarly influenced, whilst rectal temperature changes were negligible. Thus, any similarity between brain and rectal temperatures is not mechanistically linked. Therefore, unless a mechanistic connection exists, then it is very misleading to suggest that thermal equivalence, or the lack thereof, between any two indices validates, or invalidates, either measurement (Roth *et al.*, 1996; Gass and Gass, 1998; Craig *et al.*, 2000, 2002; Hissink Muller *et al.*, 2008; Ganio *et al.*, 2009; Childs and Lunn, 2013), particularly when equilibration requirements may have been violated.

Whilst blood arriving at the rectum comes from the abdominal aorta via the inferior mesenteric (superior rectal), internal iliac (middle rectal) and internal pudendal arteries (inferior rectal: Standring, 2008; Hansen and Netter, 2010), this flow serves only its metabolic requirements. Of those vessels, it is the superior rectal (haemorroidal) artery that supplies tissues from which rectal temperatures are typically measured (12-15 cm beyond the anal sphincter), and these vessels are small relative to the mass of the rectum, so heat is not easily exchanged. The haemorroidal veins have similar dimensions (inferior, middle, superior: Standring, 2008; Hansen and Netter, 2010), so venous heat removal is also quite slow. Therefore, the rectum possesses the thermal characteristics of an under-perfused site. The bladder is similarly perfused, and it too is hot (Figure 6), as is the vagina (Benedict and Slack, 1911), although its blood flow is higher and more variable (Emmanuel *et al.*, 2000). In fact, the lower abdominal cavity is so well insulated that many mammals evolved with the male genitals below the pelvic floor to optimise heat exchange and testicular function (Waites and Moule, 1961). Against this background, mechanisms affecting heat exchanges in the rectal tissues will be explored.

Firstly, the rectal canal contains a thermal gradient, diminishing towards the anus, and permitting heat flow to the ambient medium (Benedict and Slack, 1911; Mead and Bonmarito, 1949; Nielsen and Nielsen, 1962; Lee *et al.*, 2010). Indeed, longitudinal gradients exist throughout the gastrointestinal tract (Cranston *et al.*, 1954; Rawson and Quick, 1972; Caputa, 1980; Jaeger *et al.*, 1980). In perhaps the most detailed project involving the rectum, depths from 4-19 cm beyond the anal sphincter were studied in resting and exercising individuals ($N$=19; Lee *et al.*, 2010). From 8-13 cm, rectal tissue had a consistent temperature, varying $<0.1^{o}$C. This was also apparent at 19 cm, but at 16 cm (ampulla), there was an inexplicable rise of about $0.5^{o}$C. Most recently, Buono *et al.* (2014) demonstrated this gradient to be modified by ambient conditions; increasing in the cold and narrowing in the heat. Consequently, standardisation of measurement depth is important if absolute temperature is of interest, yet some have not recognised this (Darowski *et al.*, 1991; Mariak *et al.*, 1993, 1994; Mellergård, 1994). Furthermore, movement of faecal matter within the bowel is challenging, as this lengthens the conductive path when a sensor becomes lodged within the stool. Various practices can minimise this possibility (*e.g.* timing of meals relative to experimentation, pre-experimental bowel evacuation).

Secondly, heat exchanged with neighbouring tissues is modulated by variations in heat production and perfusion. Several factors modify blood flow to, and the temperature of, the lower abdomen, and three have relevance to this discussion. In women, changes in uterine and vaginal blood flows accompanying menstrual cycling will modify heat flux (Emmanuel *et al.*, 2000). During upright immersion, there is an hydrostatically driven, yet temperature-dependent (Pendergast, 1988), displacement of venous blood into the upper thorax. This involves all dependent tissues (Arborelius *et al.*, 1972), but primarily the abdominal structures (Johansen *et al.*, 1997), and it elevates the role of conduction in organs so affected. Lower-body exercise elevates leg heat production, some of which is transported centrally (femoral veins), with the balance dissipated peripherally (Gisolfi and Robinson, 1970; González-Alonso, 2012). The femoral veins are not, however, in contact with the rectum (Standring, 2008; Hansen and Netter, 2010), although it has long been held that this blood flow was an important influence on rectal temperature (Asmussen and Nielsen, 1947). Instead, Mittleman and Mekjavic (1988) demonstrated, following an extended arterial occlusion of all limbs during cold-water immersion (15°C), that re-establishing limb blood flow produced an 8.8-fold reduction in oesophageal, but only a 1.8-fold change in rectal temperature. This reinforces the interpretative difficulty of temperatures recorded from sites that are more heavily dependent upon conductive transfers, as those tissues have a considerable thermal inertia. Most recently, Todd *et al.* (2014) highlighted this dissociation during sinusoidal cycling (Figure 4), and each of these outcomes is more consistent with a conductive heat-flow dominance at the rectum.

In addition, rectal temperature exceeds most intravascular temperatures in humans (Bazett *et al.*, 1948; Pennes, 1948; Eichna, 1949; Eichna *et al.*, 1951; Ilsley *et al.*, 1983; Mariak *et al.*, 1993) and animals (Horvath *et al.*, 1950b; Bligh, 1957a). Aulick *et al.* (1981) demonstrated this for femoral venous blood across metabolic rates from 173-401 $W.m^{-2}$ ($N$=3). This state resists heat influx until the thermal gradient is reversed, although the blood need not be hotter than the rectal tissues to elevate rectal temperature, it just needs to reduce heat removal. In this regard, vessels serving the gluteal muscles (inferior gluteal arteries and veins, internal iliac vein; Standring, 2008) may be sufficiently close to alter nearby tissue temperatures, although Aulick *et al.* (1981) suggested another reason for the rise in rectal temperature during endurance exercise; altered rectal-tissue perfusion.

Although rarely investigated, rectal (mucosal) blood flow varies with daily activities, and with hormonal and skin temperature fluctuations. For example, blood flow rises after eating, but decreases during the follicular phase of the menstrual cycle, and during head-up tilting (Emmanuel *et al.*, 2000) and smoking (Emmanuel and Kamm, 1999). It appears to be higher in men than women, and lowest in postmenopausal women (Emmanuel and Kamm, 1999). During physical exercise, visceral blood flow, and presumably that of the rectum, is reduced as the intensity rises (Rowell *et al.*, 1967; Otte *et al.*, 2001). Abdominal venous volume also decreases during upright immersion (Johansen *et al.*, 1997). Furthermore, when skin temperature is manipulated, both intestinal and rectal blood flows seem to change in the opposite direction (Grayson, 1950, 1951).

This paints a complex picture. In a well-insulated region with a stable metabolic rate, convective heat removal primarily modifies tissue temperature. Thus, systematically high rectal temperatures are probably associated with its relatively low basal blood flow (Graf, 1959). This may also occur if its arterial supply is hotter, yet it is hard to conceive how this may eventuate without hyperthermia, as the rectum is one of the hottest deep-body sites during normothermia. Nonetheless, femoral arterial temperature is amongst the warmest (Figure 8), even though it may be cooled by companion veins. Thus, whilst rectal and mesenteric arterial temperatures are unknown, one assumes they are warmer, since those vessels arise from the abdominal aorta before passing through, and being warmed by, hotter visceral structures. Indeed, in four studies in which both rectal and gastrointestinal tract temperatures were simultaneously measured (Figure 6), two showed the latter to be higher (Lee *et al.*, 2000; McKenzie and Osgood, 2004), one recorded equal values (Easton *et al.*, 2007) while the fourth found higher rectal temperatures ($N$=4; Taylor, 2012: unpublished observations [Figure 9]). Furthermore, animal data have revealed the stomach, duodenum, ileum and large intestine are $\sim$0.5°C hotter than the aorta (Durotoye and Grayson, 1971). Thus, it is not unreasonable to suggest that variations in either the flow or temperature of blood within these well-insulated vessels are the likely reasons for high basal rectal temperatures.

If this is accepted, one must then consider what happens when rectal blood flow changes, as convective heat removal is flow- and temperature-dependent (He *et al.*, 2003). One outcome is a temperature change in the opposite direction if this blood removes locally

produced heat, as it does at the liver and brain. However, high hepatic and cerebral flows are dictated by the functions those tissues fulfil, but this does not apply to the rectum (Durotoye and Grayson, 1971). In fact, Grayson (1951) reported that as rectal blood flow increased, its temperature (16.5 cm deep) rose in synchrony, although that interpretation is not universally accepted (Wissler, personal communication). This response persisted regardless of whether the flow change was induced by a cutaneous thermal stimulus or an adrenaline infusion, and it was possibly an homeostatic reflex involving blood pressure regulation. The only scenario that could account for a synchronous rise in blood flow and temperature was a greater flow of warmer blood that reduced heat removal, although it was not necessary for this blood to have been hotter than the rectal tissues. Femoral artery occlusion during skin cooling failed to eliminate this change (Grayson, 1951), indicating the blood was of intra-abdominal origin. These paradoxical skin and deep-body temperature changes are observed at many sites, including the central arteries (Leslie, 1778; Burton and Bazett, 1936; Bazett *et al.*, 1948; DuBois, 1951; Ladell, 1951; Cranston *et al.*, 1954; Hardy, 1954; McCaffrey *et al.*, 1975; Edwards *et al.*, 1978). At the commencement of external cooling, for instance, cutaneous vasoconstriction transiently and instantaneously alters heat balance by modifying skin blood flow. As a result, deep-body heat storage occurs until conductive heat loss lowers peripheral tissue temperatures enough to re-establish the thermal gradient and heat loss from the body core. This same mechanism may explain simultaneous increments in rectal blood flow and temperature.

Whilst some rectal temperature variations may be tracking whole-body changes, in other circumstances (Figure 4), a temperature elevation may be explained by a different mechanism. For example, combined exercise and heat stresses may induce a powerful reduction in visceral blood flow, and in this circumstance, heat transfer becomes more heavily dependent upon conduction. The conductive model in Figure 2 is relevant here; the lower the flow, the greater is the conductive contribution and the longer is the phase delay (thermal inertia) for changes in thermal states. In Figure 4, intramuscular and oesophageal temperatures tracked sinusoidally varying heat production while rectal temperature climbed continuously, even after the other temperatures returned to pre-treatment baselines. This is consistent with a flow-dependence at those sites and thermal inertia within the rectum.

This phase delay of the rectal temperature response is frequently observed, varying between

6-60 min (Burton and Bazett, 1936; Bazett *et al.*, 1948; Eichna, 1949; Cooper and Kenyon, 1957; Gollan, 1959; Äikäs *et al.*, 1962; Benzinger and Taylor, 1963; Guidry and McDowell, 1966; Piironen, 1970; Saltin *et al.*, 1970; Molnar and Read, 1974; Edwards *et al.*, 1978; Hayward *et al.*, 1984; Savard *et al.*, 1985; Vallerand *et al.*, 1992b; Lee *et al.*, 2000; Lee *et al.*, 2010; Nagano *et al.*, 2010; Teunissen *et al.*, 2012). One consequence is that the offset between rectal and oesophageal temperatures can change from $<0.5^{o}$C to $>10^{o}$C during extreme states (Gollan, 1959), another is that thermoeffector activation may precede rectal temperature changes (Benzinger and Taylor, 1963; Snellen, 1969; Todd *et al.*, 2014), and a third is that steady-state rectal temperatures during exercise may take 50 min to be obtained (Greenleaf and Castle, 1972), depending upon the compensability of the conditions. Thus, "*rectal temperature appears to be a lagging and unsatisfactory index*" (Eichna *et al.*, 1951; P 353).

A further illustration of this delay is seen in Figure 9 (one participant), where deep-body cooling (cold-water immersion: 12.5$^{o}$C) to an oesophageal temperature of 35$^{o}$C was immediately followed by exercise in the heat (40$^{o}$C) until it reached 39$^{o}$C. The time for each site to fall 0.1$^{o}$C below its pre-immersion baseline was: 50 s (oesophagus), 285 s (auditory canal), 305 s (rectum) and 360 s (gastrointestinal tract). These variations reflect the contributions of convective and conductive losses, and were shorter when the former dominated. When conduction prevailed, trace cross-overs occurred following a change in the external temperature (also see Figure 2). These were also present at points A, B, C and D (Figure 9). Beyond point B, rectal temperature fell below oesophageal temperature, and remained so thereafter, as eventually did gastrointestinal (point C) and auditory-canal temperatures (point D). This cross-over phenomenon is also widely reported (Eichna, 1949; Bligh, 1957a; Cooper and Kenyon, 1957; Severinghaus, 1959; Benzinger and Taylor, 1963; Minard and Copman, 1963; Piironen, 1970; Keatinge and Sloan, 1975; Gibson *et al.*, 1981; Golden and Hervey, 1981; Hayward *et al.*, 1984; Savard *et al.*, 1985; Webb, 1986; Ash *et al.*, 1992; Teunissen *et al.*, 2012). Indeed, the current authors were unable to find exceptions, yet its implications have rarely been discussed.

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INSERT FIGURE 9 ABOUT HERE

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In many experiments, cross-over artefacts are irrelevant. In others, they may lead to misinterpretation. For instance, some normothermic, steady-state temperature offsets (Figure 6) are reversed during dynamic phases when heating follows cooling (Figure 9), or when cooling follows heating (Gibson *et al.*, 1981). Unawareness of this can have adverse clinical and scientific consequences when warming hypothermic, or cooling hyperthermic individuals (Severinghaus, 1959; Molnar and Read, 1974; Hayward *et al.*, 1984; Ash *et al.*, 1992; Taylor *et al.*, 2008; Weingart *et al.*, 2009; Casa *et al.*, 2010; Pearson *et al.*, 2012). In such instances, one cannot substitute a conduction-dependent surrogate index for the temperature of a critical and temperature-sensitive tissue, unless the former has been shown to faithfully track the dynamic response of the latter.

**Oesophageal temperature**

> "*A temperature-measuring device located in the oesophagus at the level of the heart would, by reason of its close proximity, seem to provide a more useful index of heart temperature than would a similar device introduced into the opposite end of the alimentary canal*" (Cooper and Kenyon, 1957; P 619).

Depending upon the distance above the cardiac sphincter, the oesophagus is sandwiched between the spinal column, trachea and the left main bronchus, as well as the heart and several major blood vessels (thoracic aorta, left common carotid, right pulmonary arteries, superior *vena cava*; Standring, 2008; Hansen and Netter, 2010). Whilst the oesophagus is merely another conduit, its intimate contact with these structures, in combination with its relatively low heat capacity, means that its temperature is readily influenced by convective heat exchanges. There is also a longitudinal temperature gradient within the oesophagus (Cranston *et al.*, 1954; Severinghaus, 1959; Whitby and Dunkin, 1968, 1969, 1971; Caputa, 1980; Jaeger *et al.*, 1980), but this is not merely a conductive gradient down to the mouth, but a gradient dictated by its contiguous structures.

Higher up in the oesophagus, the influence of ventilatory heat exchanges is most pronounced, so a sensor positioned here will not just have a lower absolute temperature, it will track ventilatory cycling (Nielsen and Nielsen, 1962; Whitby and Dunkin, 1969; Jaeger *et al.*, 1980). At the level of the carina, local tissue temperatures can be $>1^{o}C$

cooler than within the lower third of the oesophagus (Severinghaus, 1959), particularly when breathing cold air (Jaeger *et al.*, 1980). In its lower-third (fifth-tenth thoracic vertebrae), vascular exchanges dominate (Cooper and Kenyon, 1957; Severinghaus, 1959; Whitby and Dunkin, 1968, 1971; Brengelmann *et al.*, 1979; Mekjavic and Rempel, 1990), and oesophageal temperature is stable and independent of the inspired-air temperature (-40°C: Jaeger *et al.*, 1980). To ensure the correct positioning of the sensor within this vascular zone, it is necessary to standardise insertion depth, both absolutely and relative to stature (Brengelmann *et al.*, 1979; Mekjavic and Rempel, 1990). Nevertheless, in all positions, it is modified by swallowing saliva and drinking.

These powerful vascular influences were most clearly illustrated by Gollan (1959) during extracorporeal cooling (dogs). Right atrial blood was cooled before being returned via the descending aorta, partially bypassing the cardiopulmonary circulation. Therefore, cooled blood perfused the lower systemic vessels before those above the heart. Hence, the abdomen should have cooled earlier, and perhaps also deeper, than the mediastinum. Whilst intra-abdominal temperatures were not reported, myocardial temperature was reduced to $<5$°C. This was tracked by oesophageal temperature ($<10$°C), yet rectal temperature remained above 20°C (Gollan, 1959). Cooper and Kenyon (1957) reported similar outcomes in patients rendered hypothermic prior to aortic surgery. When aortic clamps were released at the completion of each procedure, blood flow to the legs returned, resulting in the cephalad displacement of cold blood. Just 15 s after release, oesophageal temperature dropped 0.7°C, while the corresponding change at the rectum was 0.1°C. Indeed, the authors found oesophageal temperature was never $>0.15$°C away from para-aortic temperature, yet rectal temperature deviated $>2$°C. This close oesophageal tracking of central blood temperatures has been reported by others (Lees *et al.*, 1980; Hayward *et al.*, 1984; Shiraki *et al.*, 1986; Nybo *et al.*, 2002).

This thermal responsiveness of the oesophagus relative to central blood temperature (80-s time delay: Shiraki *et al.*, 1986) demonstrates that it has not simultaneously equilibrated with poorly perfused deep-body tissues or tissues more distant from major blood vessels. Therefore, this index is unable to provide information concerning the temperature (or the heat storage) of those tissues, and by the same logic, the reverse state must also exist. Nevertheless, predictions of oesophageal temperature have been developed using other

deep-body locations or surrogates (Edwards *et al.*, 1975; Gibson *et al.*, 1981).

**Auditory-canal temperature**

Auditory-canal (aural) temperature measurement was perhaps initiated by Williams and Thompson (1948), who reported a close association with sublingual temperature. From Figure 4, it is clear that an insulated auditory-canal temperature can track variations in deep-body temperature, as reported by others (Cooper *et al.*, 1964; Greenleaf and Castle, 1972; Edwards *et al.*, 1978; Hayward *et al.*, 1984), albeit with an offset (Figure 6) and a phase delay (Figure 4). This raises an important question: is it more important to determine actual temperatures or temperature changes? The answer to this question dictates one's acceptance of data derived from the various indices described herein, and the acceptability of normalising body temperatures when presenting these data.

The auditory canal is supplied with blood from both superficial and deep sources. At its entrance (pinna, lobe, external meatus), the anterior auricular artery provides blood from the superficial temporal artery (Standring, 2008). This is a relatively shallow vessel coming from the external carotids. The posterior auricular artery, also arising from the external carotid, feeds tissues deeper within the auditory canal as well as the skin behind the ear (Standring, 2008). Facial skin receives arterial blood from the internal carotids (supraorbital and supratrochlear arteries). This, in combination with its proximity to the brain, explains why forehead temperature is among the warmest skin sites (Sheard *et al.*, 1941; Olesen and Fanger, 1973; Werner and Reents, 1980; Zaproudina *et al.*, 2008), why it varies during pathological conditions (Weinstein *et al.*, 1991; Ng and Chong, 2006) and why it is sometimes comparable to axillary and rectal temperatures (Ilsley *et al.*, 1983). Unfortunately, surface temperatures are powerfully influenced by ambient conditions, thus explaining why the temporal artery surface temperature cannot reliably track deep-body temperatures (Low *et al.*, 2007; Kistemaker *et al.*, 2006; Ronneberg *et al.*, 2008).

As a consequence of its vascular configuration, the auditory canal responds rapidly to changes in carotid artery (Cooper *et al.*, 1964) and cardiac temperatures (Hayward *et al.*, 1984). This was nicely demonstrated by Cooper *et al.* (1964), who infused the right internal carotid with warm saline (44°C) and observed an immediate rise in auditory-canal temperature, but not that of the contralateral ear. Since the tympanum, but not the auditory

canal, receives blood from this vessel, the authors suggested that retrograde flow at the bifurcation of the internal and external carotids might explain this observation. This is probable, as is the contribution from a direct influence on tympanic-membrane temperature.

Nevertheless, the auditory canal is influenced by variations in ambient temperature (Gibbons, 1967; Greenleaf and Castle, 1972; Morgans *et al.*, 1981; Sharkey *et al.*, 1987; Hansen *et al.*, 1993; Coso *et al.*, 2008; Nagano *et al.*, 2010; Teunissen *et al.*, 2011a) and wind (Teunissen *et al.*, 2011a), producing a thermal gradient down the canal (Cooper *et al.*, 1964). The cooler the air temperatures, the greater is the gradient, although it can be reversed. Thus, absolute temperatures are depth-dependent and lower than oesophageal temperature (Figure 6), and it is now known that this ambient bias also influences rectal temperatures (Buono *et al.*, 2014).

Under ambient temperatures not too different from deep-body temperature, this offset has minimal impact on tracking deep-body temperature changes (Greenleaf and Castle, 1972; Cotter *et al.*, 1995; Figure 4). Indeed, sites 0.3-1.7 cm within the canal track changes in oesophageal temperature with a faster response than sublingual temperature (Cooper *et al.*, 1964). Auditory-canal temperature also responds to postural variations (Greenleaf and Castle, 1972), being higher when supine, as observed in other deep-body sites (Cranston *et al.*, 1954; Graf, 1959; Ogawa *et al.*, 1993). Nevertheless, those using this index may face criticism from others who believe the external-temperature influence always invalidates this measurement, even though the bias disappears in the heat and can be minimised through insulation. Strangely, conduction- and ambient temperature-dependent rectal bias often appear more palatable.

Given that some investigators and clinicians consider absolute temperature to be critical, Keatinge and Sloan (1975) developed a servo-controlled, zero-gradient device that first heated the outer ear to that of the auditory canal (also see Moore and Newbower, 1978). Once equilibrated, the temperature of the pinna tracked auditory-canal temperature, removing both the thermal gradient and the ambient bias. Under these conditions, auditory-canal temperature is typically higher than oesophageal temperature, as is tympanic-membrane temperature (Figures 5 and 6; Keatinge and Sloan, 1975; Cotter *et al.*, 1995). Curiously, this device received little attention, yet its dynamic response characteristics

exceeded those of rectal temperature (Keatinge and Sloan, 1975; Maw and Taylor, 1992).

**Tympanic-membrane temperature**

To the authors' knowledge, the first recordings of tympanic-membrane temperature were made Benzinger (1959). From that work arose contributions of Cabanac and Caputa (1979), and controversy surrounding deep-body temperature indices. This was not so much about the utility of various indices, but using tympanic temperature to support the case for selective-brain cooling in humans (Nybo and White, 2008), and it was driven by passionate protagonists (Benzinger, 1969; Brengelmann, 1993; Cabanac, 1993; Simon, 2007). Whilst that discussion is avoided here, its root centred on the arterial supply for the tympanic membrane, with controversy relating to the certainty of membrane contact by the sensor and whether or not its temperature suffered from an ambient bias.

Arterial blood travels to the brain via the internal carotid and vertebral arteries, both of which branch off the subclavian artery, rejoining to form the cerebral arterial circle (circle of Willis: Standring, 2008; Hansen and Netter, 2010). Posteriorly, the basilar artery leads from the vertebral arteries to the circle, and from it arise vessels supplying the inner ear: the labyrinthine (auditory) arteries (Standring, 2008; Hansen and Netter, 2010). The tympanic membrane receives blood from two sources: the glaserian (anterior tympanic) artery, which leaves the maxillary artery soon after branching from the external carotid, and the caroticotympanic artery that arises from the internal carotid (Standring, 2008). The deep auditory canal, close to the tympanum, receives external carotid blood via the posterior auricular artery (Standring, 2008).

This anatomical configuration raised the possibility the tympanic membrane might faithfully track carotid arterial temperature, and it was hypothesised this blood would modify both hypothalamic and tympanic temperatures (Benzinger and Taylor, 1963; Brinnel and Cabanac, 1989). This was challenged by Brengelmann (1987, 1993). Nybo *et al*. (2002) subsequently found that, during rest and exercise, carotid arterial temperature was consistently cooler than jugular venous blood. Therefore, whilst true tympanic-membrane temperature can track blood and oesophageal temperatures (Figure 5: Tanabe and Takaori, 1964; Piironen, 1970; Molnar and Read, 1974; Shiraki *et al*., 1986, 1988; Brinnel and Cabanac, 1989; Cotter *et al*., 1995; Sato *et al*., 1996), that blood was acting as a brain

coolant (Hayward and Baker, 1968; Figure 8).

Brinnel and Cabanac (1989) suggested true tympanic-membrane temperature could only be measured via direct contact with its lower, anterior quadrant. The anatomical description above supports that proposition, although this requires otoscopic or possibly acoustic verification (Benzinger and Taylor, 1963; criticised by Brengelmann, 1987). Without verification, membrane measurements cannot be assumed. Nevertheless, while unverified measures are not tympanic, they are auditory-canal temperatures, which are similar but not equivalent (Figure 6). Indeed, due to the curvature of the auditory canal and the presence of cerumen (ear wax), infrared thermometers infrequently measure tympanic temperature (*e.g.* Shinozaki *et al.*, 1988; Daanen, 2006; Easton *et al.*, 2007; Lee *et al.*, 2011).

As with the auditory canal, there is evidence that ambient and facial-skin temperatures can bias tympanic-membrane temperature (Nadel and Horvath, 1970). Using the method of Benzinger and Taylor (1963: subject-determined sensor positioning), that group reported no apparent bias in air temperatures from 10°-28°C (Nadel and Horvath, 1969). This was subsequently verified, although from 22°-44°C, an elevation of 0.04°C for each 1°C rise in air temperature was described (Nadel and Horvath, 1970). Together, these studies showed two thermal plateaux, separated by a less-stable range (22°-34°C). The first was a possible negative bias in air from 10°-22°C (tympanic temperature 36.4°C) with the second appearing to be without bias (34°-44°C; tympanic temperature 37.1°C).

McCaffrey *et al.* (1975) tested this evidence by simultaneously heating (45°-50°C water) and cooling (3°-4°C water) the circum-orbital facial surfaces whilst recording tympanic temperature bilaterally. These very powerful treatments highlighted this skin temperature influence. Yet, whilst this study is often cited as proof this index is flawed, close examination reveals the maximal difference was just 0.4°C, which occurred because both skin temperatures were driven in opposite directions by ~45°C. Thus, the unilateral effects obtained when heating (45°-50°C) and cooling (3°-4°C) were half that value. While still present, the extreme conditions under which those effects were induced means this bias will be less pronounced under typical laboratory conditions, although Nybo *et al.* (2002) reported bias during face fanning.

One must also entertain the possibility that, in several of these investigations, the sensors were not in intimate contact with the tympanum. This was tested by Sato *et al*. (1996) using a bespoke tympanic sensor. Neither ice cooling nor skin cooling elicited by facial fanning influenced membrane temperature. Although when membrane contact was lost, this bias appeared, and herein lies the critical point; if membrane contact has not been verified, then one is measuring auditory-canal temperature. Indeed, Sato *et al*. (1996) recommended that contact can be verified by testing for the presence of facial skin cooling interference. Moreover, Shibasaki *et al*. (1998) established this for infrared thermometry, when a flexible, optical-fibre probe was used to approach the tympanum.

**Gastrointestinal temperature**

Gastrointestinal temperatures were first measured by Beaumont (1838), well in advance of the experiments of Wunderlich (1869), with interest re-emerging and expanding following the miniaturising of sensors and data loggers (Byrne and Lim, 2007), although this is not a recent phenomenon either (Fox *et al*., 1961). Since intestinal thermoreceptors exist (Rawson and Quick, 1972; Villanova *et al*., 1997), there are mechanistic interests in those tissues.

The gastrointestinal tract is much more than a conduit structure. However, since radio pills used to measure temperature travel along the tract, their location is dependent upon gastrointestinal motility. This can be modified, along with thermal equilibration requirements, due to the nature of the experiment (Misiewicz *et al*., 1968; reactive error). Moreover, since convective and conductive heat exchanges vary among sites, the thermal inertia of the local tissues changes as sensors travel along the tract.

Not surprisingly, Pearson *et al*. (2012) reported gastrointestinal temperature to lag behind pulmonary artery temperature during passive, whole-body heating and cooling. Kolka *et al*. (1993) and Teunissen *et al*., (2012) observed a similar delay relative to oesophageal temperature, but not rectal temperature, which it generally exceeds (Figure 6). These delays introduce bias during dynamic phases. However, gastrointestinal and arterial temperatures were equivalent when thermal equilibration was approached (~45 min; Pearson *et al*., 2012). One may therefore question comparisons among gastrointestinal and other deep-body surrogates during dynamic phases, and when sampling did not occur from the same

intestinal location (technically very challenging).

To gain an appreciation for variations in gastrointestinal temperatures during transit, one author consumed seven radio pills over a 37-h period (Figure 10A), including consecutive nights (Figure 10B), whilst maintaining a normal routine (office work). The minimal transit time was 9.2 h, and all pills ingested before retiring were passed before breakfast. Times for Figures 10A, C and D are referenced to trial commencement (time zero), with Figure 10C emphasising an endurance run (commencing at 06:20 h [12.2 h after time zero], duration 50 min) and Figure 10D highlighting resistance exercise (commencing at 18:40 h [24.5 h after time zero], duration 40 min).

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During running, pill temperatures rose linearly following an initial lag (Figure 10C), as expected in an unregulated thermal state in which heat storage was constant and positive. The overall temperature changes were 3.0°C (sensor 2) and 2.9°C (sensor 3), with peak values occurring ~5 min after exercise ceased (39.6°C and 39.3°C respectively). This reflected a conductive heat transfer dominance. The maximal between-sensor difference was 1.1°C, which occurred during recovery, and immediately prior to the passing of sensor 2. It was assumed that sensor was recording low colonic (stool) temperature, whilst the other measured gastric temperature, as it was consumed 60 min before exercising. It was also assumed that running increased gastric motility (Keeling and Martin, 1987). During resistance exercise, the highest temperature was 38.0°C (sensor 5). At the same time, the other temperatures were 37.5°C (sensor 3), 37.5°C (sensor 4) and 37.4°C (sensor 6), with a maximal between-sensor difference of 0.7°C (sensor 5 versus 6). Thus, for this individual, exercise was associated with significant temperature variations along the gastrointestinal tract.

When these exercise data were removed, the within sensor temperature ranges during transit averaged 1.0°C (Figure 10A: range 0.7-1.6°C). Therefore, the variability during exercise was also evident during sedentary activities, reflecting local differences in tissue

metabolism and heat exchanges. When sleep data were compared across consecutive nights using sensors ingested just before retiring (Figure 10B: sensors 2 and 7; time referenced to pill ingestion), the classical temperature decline appeared during the first hour (Campbell and Broughton, 1994; Baker *et al.*, 2001), but the circadian trough at about 04:00 h was less apparent. This time is beyond the 6 h plotted, as the subject arose at 03:45 h. On the second evening (sensor 7), a trough was approached. Throughout sleep, peak temperatures of 36.9°C (sensor 2) and 36.8°C (sensor 7) were observed, with respective within-sensor transit ranges being 0.6°C and 0.7°C. Every sensor was recording during sleep on one of the two nights, although sensors 1, 3, 4, 5 and 6 were ingested at varying times before retiring. When these data were analysed, the average, within-sensor overnight temperature range was 0.5°C, with neither the temperature peaks nor the troughs falling outside the data shown in Figure 10B. The significance of this outcome can be appreciated when one considers typical circadian (Landolt *et al.*, 1995) and menstrual variations (Buxton and Atkinson, 1948) in deep-body temperature, as well as temperatures changes that elicit thermoeffector responses (Werner *et al.*, 2008; shivering: Tipton *et al.*, 2013; sweating: Machado-Moreira *et al.*, 2014). These temperature changes are 0.3-0.5°C, rendering the signal to noise ratio of gastrointestinal measurements unacceptable to many.

Recommendations for the delay between ingesting radio pills and data collection appear to be predicated on gastric emptying times (Goodman *et al.*, 2009), and avoiding artefacts introduced by food and liquid consumption. Perhaps the most detailed investigation of radio-pill transit times was conducted by Roach *et al.* (2010). Subjects ingested a new sensor as each passed, with 65 transits recorded over 7 d ($N$=11). The mean transit time for single pills was 27.4 h (range: 4.6-82.8 h), although the individual with the shortest single transit also had one passage exceeding 26 h, while another person had a difference between the shortest and longest transit times of 55 h. McKenzie and Osgood (2004) described a transit mean of 24 h (range: 12.5-134.4 h: $N$=10), whilst O'Brien *et al.* (1997) reported the overnight passing of pills in three individuals. Clearly, since defaecation is entrained and infrequently modified by daily routine changes, then such data are exceedingly difficult to interpret. However, this research emphasises that some individuals pass pills very rapidly (6.2% within 12 h: Roach *et al.*, 2010), and that pill location within the tract is time- and person-specific (Livingstone *et al.*, 1983; Kolka *et al.*, 1993; Goodman *et al.*, 2009). By implication, validation of gastrointestinal temperatures against

other indices seems to lack justification, particularly if the reference index was less than ideal for the experiment.

To evaluate the influence of drinking on data collection, Wilkinson *et al*. (2008) tracked gastrointestinal temperatures during the hourly ingestion of cold drinks (250 mL, 5-8°C, $N=10$). The thermal artefact accompanying drinking lasted 32 min (SD 10), decreasing over time and being absent in all individuals 11.5 h after ingestion. Under controlled conditions, these interferences can be prevented, firstly by ingesting pills with water at 37°C and then by preventing fluid consumption (Gibson *et al*., 1981; Kolka *et al*., 1997; Ducharme *et al*., 2001). However, under field conditions, the latter may be less feasible, with artefact prevention constituting an important design consideration.

From this discussion, one may conclude that rectal temperature, due to its inherent stability, is preferable to gastrointestinal measurements (consider radio-pill suppositories). Within thermal steady states, rectal temperatures faithfully quantify deep-body heat content, but with minimal measurement artefact. Whilst gastrointestinal temperature tracks changes in local heat storage, it is subject to the vagaries of localised fluctuations in heat production and exchange encountered *en route*, and these artefacts confound interpretation.

**Intramuscular temperatures**

The first investigations of muscle temperatures appear to have been performed by Becquerel and Breschet (1835). Researchers have now demonstrated muscle temperature to influence tissue oxygenation (Barcroft and King, 1909), with heating (Starkie *et al*., 1999; Ferguson *et al*., 2002), but not cooling (Booth *et al*., 2001), modifying substrate utilisation and energy turnover. In resting states, intramuscular temperatures are not homogeneous (Webb, 1992), but increase with sensor depth (parabolic profile) and vary with proximity to large arteries and bones (Kenny *et al*., 2003). Indeed, muscles reveal a radiating thermal gradient that supports conductive heat losses. In addition, distal muscle tissues, which are smaller and have less thermal insulation, are readily influenced by the environment (Bazett and McGlone, 1927; Barcroft and Edholm, 1946; Pennes, 1948; Booth *et al*., 2004). These characteristics render resting skeletal muscles ill-suited as deep-body surrogate sites.

During exercise, and immediately thereafter, active skeletal muscles become significant

heat sources (Figure 11: Pembrey, 1898; Buchthal *et al.*, 1945; Kenny *et al.*, 2003, 2006; Booth *et al.*, 2004; Kenny and Jay, 2007; Todd *et al.*, 2014). This becomes exaggerated in hotter conditions (Saltin *et al.*, 1972) due to impaired heat conduction. At the start of exercise, rapid intramuscular temperature changes are observed (Figure 4), reflecting variations in heat storage, with temperatures becoming more uniform as exercise continues. Among muscles, however, the inter-site variability expands as metabolic variations from one segment to another increase (Figure 11: Kenny *et al.*, 2003, 2006).

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INSERT FIGURE 11 ABOUT HERE

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In the quadriceps, for instance, the rate of temperature rise is a function of force generation intensity (Edwards *et al.*, 1975), and recent evidence has linked sudomotor responses to those thermal changes, possibly indicating the presence of thermosensitive elements within skeletal muscle (Todd *et al.*, 2014). In many cases, the temperature of active muscles may exceed some deep-body temperatures (Figure 4), although this is not a pre-requisite for a core temperature elevation, as it is the balance (turnover) between heat production and removal that explains local thermal variations. Indeed, data from Todd *et al.* (2014) revealed the intramuscular temperatures of three participants exceeded oesophageal temperature on $>90\%$ of their simultaneous measurement during exercise, with an average difference of 0.43°C. For another five individuals, the reverse obtained; mean difference -0.46°C. Nevertheless, there was a strong, positive correlation between oesophageal and intramuscular temperatures ($r=0.79$) across subjects (Todd *et al.*, 2014).

Taken together, this evidence highlights the dynamic nature of intramuscular temperature during exercise. Differences between active and inactive muscle temperatures, as well as variations in heat distribution between the muscles and deeper tissues, emphasise the thermal significance of blood. Not only does its specific heat capacity exceed that of skeletal muscle (3.61 versus 3.59 $kJ.kg^{-1}.{}^{o}C^{-1}$; Davy, 1814; González-Alonso *et al.*, 2000), but the relative perfusion of the active and inactive limbs (Bevegård and Shepherd, 1966; Nishiyasu *et al.*, 1992), the ratio of that flow to the muscle mass and the arteriovenous temperature differences (Aulick *et al.*, 1981) have profound affects on muscle temperature,

convective heat flow and deep-body heat storage during exercise.

During early recovery (15-20 min), and irrespective of exercise intensity, intramuscular temperatures decrease by values comparable to oesophageal temperature (Kenny *et al.*, 2003, 2006). Indeed, the thermal gradient from the deep muscle (*vastus medialis*) to the oesophagus remained constant for the final 45 min of recovery (Kenny *et al.*, 2003). This is consistent with thermal equilibration and a uniform heat distribution. More recent studies revealed this sustained tissue temperature elevation occurred in parallel with a marked and sustained increase in body heat content (Kenny *et al.*, 2010; Kenny *et al.*, 2008). It appears that, during an inactive and seated recovery, blood pooling in the dependent regions traps heat and thereby contributes to a sustained elevation in muscle temperature.

**Transcutaneous temperature**

Due to continual heat exchanges between the skin and the ambient medium, a temperature gradient exists within the latter; the boundary layer (McGlone and Bazett, 1927). When clothing is worn, that layer becomes warm and humid, impeding heat loss and reducing environmental influences on skin temperature (Benedict *et al.*, 1919). These changes reduce the transcutaneous thermal gradient, with skin temperatures becoming more uniform (Pandolf and Goldman, 1978; McLellan *et al.*, 2013) as the skin blood vessels become engorged (Fogarty *et al.*, 2004). This resembles an exteriorisation of the deep-body temperature, and when well-insulated clothing is worn, it may be possible to track deep-body temperature from changes in transcutaneous temperature.

Since the zero-gradient (heat-flux) technique for measuring auditory-canal temperature (Keatinge and Sloan, 1975) removes the thermal gradient within the auditory canal, negating external interference, Fox and Solman (1971), Fox *et al.* (1973a) and Togawa (1979) used this approach for the transcutaneous tracking of deep-body temperature. Clinical techniques of this form were evaluated by several groups (Ball *et al.*, 1973; Kobayashi *et al.*, 1975; Singer and Lipton, 1975; Togwa *et al.*, 1976 [*Togawa*]; Carter and Perry, 1977; Lees *et al.*, 1980; Smith *et al.*, 1980; Yamakage *et al.*, 2002; Yamakage and Namiki, 2003; Kitamura *et al.*, 2010; Teunissen *et al.*, 2011b), usually under resting, thermoneutral conditions with minimal or no clothing over the measurement site. An extension of this approach, albeit it of questionable validity, used plastic-strip thermometers

positioned over blood vessels and hotter skin surfaces (Lewit *et al.*, 1982).

Another variation, developed to find a technically less-demanding and more robust method for field use, used clothing insulation and insulated-skin temperature measurements (Dollberg *et al.*, 1993; Bogh *et al.*, 1994; Taylor *et al.*, 1999; Edwards *et al.*, 2002; Armstrong, 2006; Gunga *et al.*, 2008). Those studies yielded inconsistent outcomes that appeared linked to the quality of the thermal insulation and ambient conditions.

For each of these approaches to be successful, two pre-requisites must be satisfied. Firstly, two thermal gradients must be simultaneously minimised; the core-to-shell and ambient-to-skin gradients. It is not sufficient to achieve one without the other. Only when these heat fluxes have been removed, or at least dramatically reduced, can thermal equilibration among the skin, deeper tissues and temperature sensor be assured (***Zeroth Law*** requirements). Zero-gradient instruments adequately satisfy this criterion, whilst insulated-skin temperature measurements approach it to varying degrees, and in proportion to the thickness and surface area of the insulating cover.

The second, often overlooked, consideration relates to positioning the sensor over a suitable skin surface. It is recognised that skin temperatures in normothermic individuals are quite variable, with some sites being consistently warmer (Benedict *et al.*, 1919; Bedford, 1935; DuBois, 1941; Olesen and Fanger, 1973; Werner and Reents, 1980; Zaproudina *et al.*, 2008). The forehead is one such site, but it is not the warmest, just the warmest among sites commonly evaluated. Instead, the inner canthus of the eye (junction of upper and lower eyelids) provides the closest, surface approximation of deep-body temperature among resting (Barnes, 1967; Ring, 2007), but not exercising individuals (Teunissen and Daanen, 2011). Other potential sites for this approximation include the navel (Barnes, 1967; Olesen and Fanger, 1973) and the lower occiput, neck and upper-thoracic vertebrae (Olesen and Fanger, 1973; Zaproudina *et al.*, 2008; Marins *et al.*, 2014), or regions where body segments are naturally apposed (axilla, groin: Barnes, 1967; Ilsley *et al.*, 1983; Darowski *et al.*, 1991; Childs *et al.*, 1999).

Unless both of these requirements are satisfied, uncertainty must exist concerning the precision with which the surface index tracks deep-body temperature. For example, whilst

the inner canthus may provide the closest resting approximation, it is not viable to modify thermal gradients at that site, so its use is restricted to non-exercising, steady-state conditions that vary only slightly from thermoneutrality. Moreover, the zero-gradient devices, which can readily satisfy both criteria, are often ill-suited for field use.

Accordingly, Taylor *et al*. (1998, 1999) and Armstrong (2006) evaluated the feasibility of insulated-skin temperatures (forehead, jugular, upper spine [T2-T4], ventral wrist) for predicting changes in deep-body (oesophageal) temperature in clothed and exercising individuals in warm-hot conditions. The earlier investigation, conducted at 33° and 40°C, revealed the insulation largely, but not completely, removed the ambient-to-skin thermal gradient, so an air-temperature bias was evident (Taylor *et al*., 1999), as confirmed by Gunga *et al*. (2008). The strength of the correlation between sensor and oesophageal temperatures was site dependent, with the forehead being superior to the spine, followed by the wrist and jugular sites. Prediction errors were primarily influenced by inter-individual insulated-skin temperature variability, so it appeared that calibration for each user might be required (Taylor *et al*., 1999; Eglin *et al*., 2004; Armstrong, 2006). Finally, when rectal predictions of oesophageal temperature were compared with those derived from two insulated sites combined (forehead plus spine [T2-T4]), <50% of those comparisons yielded predictions superior to those obtained from the insulated-skin temperatures (Taylor *et al*., 1999), as observed by Teunissen *et al*. (2011b) using zero-gradient thermometry.

Armstrong then undertook a two-stage investigation. In the first, a retrospective analysis was performed using data from three experiments. These included 64 separate trials performed in temperate (25°C [Wilsmore, 1996: $N$=12], 27°C [Taylor *et al*., 1998: $N$=7]) and hot conditions (33°C [Taylor *et al*., 1998: $N$=7; Armstrong and Fogarty, 1999: $N$=5, unpublished observations], 40°C [Wilsmore, 1996: $N$=17; Taylor *et al*., 1998: $N$=6]). In every trial, oesophageal, rectal and insulated-skin temperatures (spine: T2-T4) were recorded. In all trials except those of Wilsmore (1996), participants wore military combat fatigues. Data were modelled (regression) to predict changes in deep-body temperature.

Armstrong (2006) then evaluated the validity of those models using two independent datasets (47 trials), in which oesophageal, rectal and insulated-skin temperatures were again measured (Taylor *et al*., 2001 [$N$=8, 29.8°C, five thermal protective ensembles]; Fogarty

*et al.*, 2004 [$N$=7, 39.6°C, thermal protective clothing]). For the cooler conditions, predictive models failed to explain 48% of the observed variance; this was unacceptable. In the heat, the unexplained variance dropped to <20%, yet even with this improvement, every model over-predicted oesophageal temperature, with errors exceeding 1°C in each case. Thus, oesophageal temperature changes were tracked with acceptable accuracy, but actual temperature predictions were less reliable, possibly due to convective and conductive phase delays in the delivery of central heat to the periphery. For individuals wearing thermal protective clothing in the heat (40°C), it was concluded that oesophageal temperature changes could be predicted using an insulated skin temperature measured over the upper spine. To achieve this, the following equation was recommended: oesophageal temperature = 5.4784 * 0.8569 * insulated skin temperature (Armstrong, 2006).

Recently, Xu *et al.* (2013) and Niedermann *et al.* (2014) predicted deep-body temperature from skin temperatures, transcutaneous heat fluxes and heart rate. Unfortunately, these models were validated against gastrointestinal and rectal temperatures, the thermal inertia of which limited their utility. Nevertheless, there is merit in the approach, which should be undertaken using a more dynamically responsive, deep-body temperature index.

## SUPERFICIAL-TISSUE TEMPERATURE MEASUREMENTS

Under basal (normothermic) conditions, the superficial (shell) tissues are not in a state of thermal equilibrium with either the body core or the ambient environment. Instead, the kinetic energy possessed by, and therefore the temperature of, those structures forms part of the core-to-ambient temperature gradient. Thus, those tissues are transient retainers (relays) of thermal energy, and as such, the shell compartment does not satisfy the requirements of the ***Zeroth Law*** under thermoneutral conditions, even when local skin temperatures are stable. That is, heat flux is constant.

The body shell does, however, constitute a significant portion of the thermally regulated mass (Simon, 1974; Jessen, 1996), but with much more variable temperatures than the deep-body tissues. That variability is location dependent, both among (Benedict *et al.*, 1919; DuBois, 1941; Olesen and Fanger, 1973; Werner and Reents, 1980; Webb, 1992; Niu *et al.*, 2001; Zaproudina *et al.*, 2008 [Figure 12]; Marins *et al.*, 2014) and within sites (Pennes, 1948; Livingstone *et al.*, 1987; Frim *et al.*, 1990). It is also time and body-

composition dependent (Hardy, 1934b; Frim *et al.*, 1990; Chudecka *et al.*, 2014), as the temperatures of the shell tissues are largely dictated by thermal exchanges between the body core and surrounding environment (Bedford and Warner, 1934; Snellen, 1966; Psikuta *et al.*, 2014). Therefore, these temperatures only provide point-specific thermal information. But since about 50% of the body is within 2.5 cm of the skin surface (Burton, 1935), then the shell tissues represent a significant mass and potential heat sink. Thus, a knowledge of shell temperatures is essential, as is an appreciation of their usefulness and limitations.

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INSERT FIGURE 12 ABOUT HERE

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**Subcutaneous temperatures**

The parabolic form of the body core-skin thermal gradient is well established (Benedict and Slack, 1911; Bazett and McGlone, 1927). However, few have explored subcutaneous temperatures, which represent the penultimate location along that gradient. Exceptions include Bazett and McGlone (1927), Pennes (1948), Smith (1962), Webb (1992), Cotter *et al.* (1996), Cotter (1998) and Zeyl *et al.* (2004). The initial work of Bazett and McGlone (1927) described subcutaneous temperature gradients within the initial 6 mm of tissue ($N$=2). When averaged across trials, these varied from 0.22-0.48°C.mm$^{-1}$. Subsequently, Smith (1962), revealed intramuscular-to-subcutaneous temperature gradients of 2.6°C (*gastrocnemius*), 2.0°C (*tibialis anterior*) and 1.7°C (intercostals) in afebrile patients prior to anaesthesia, but the ambient conditions were below thermoneutrality (20.7°C) with cutaneous vasoconstriction presumably accentuating each gradient.

The most extensive investigation was that of Webb (1992), who studied subcutaneous temperatures in normothermic, seated males ($N$=6; triceps, forearm, chest, back, calf, thigh). He reported an average temperature of 33.9°C, with simultaneous oesophageal and mean skin temperatures of 36.9° and 33.5°C. Intramuscular temperatures were measured at each of two depths (20 and 40 mm) from the back and thigh (unspecified locations). At the back, the muscle-to-skin gradient was 2.7°C, while the gradient to the subcutaneous site was 0.9°C. For the thigh, the respective gradients were 0.8°C and -0.1°C; the negative value was not explained, nor was there evidence to indicate that it might be incorrect.

**Sources of thermal variability within and among skin sites**

When exposed to very cold conditions, generalised cutaneous vasoconstriction maximises the core-to-shell temperature gradient and minimises the shell-to-ambient gradient. Since skin blood flow can decrease below the local metabolic requirement during cooling (Abramson, 1965), the primary avenue for heat exchange becomes conductive. In that state, local tissue temperatures move closer to the ambient temperature (Pennes, 1948), particularly when one is surrounded by a medium with a high thermal conductivity. Furthermore, skin temperatures become more heterogeneous (Benedict *et al.*, 1919; Bedford, 1935; Hardy and DuBois, 1938; Sheard *et al.*, 1941; Werner and Reents, 1980; Webb, 1992), particularly at the distal sites (physiological amputation). These protective mechanisms buffer heat losses, with both an elevated subcutaneous adiposity and its more even distribution (*e.g.* women) insulating the skin from its heat source; the body core.

At the other extreme, cutaneous vasodilatation (Rowell *et al.*, 1969, 1970) makes skin temperatures more homogeneous (Bedford, 1935; Hardy and DuBois, 1938; Sheard *et al.*, 1941; Werner and Reents, 1980; Webb, 1992). This reduces the core-to-shell and shell-to-ambient thermal gradients as blood circumnavigates the subcutaneous tissues to heat the skin. This too is protective, as it minimises heat gain while optimising the mass (convective) delivery of core heat. Heat dissipation must, however, now rely upon the evaporation of sweat. Thus, we have defined the thermally mediated, physiological outer limits for skin temperatures, with the regions between those extremes being most frequently investigated.

One of those states includes the transcutaneous temperature measurements described above, the focus of which was removing local thermal gradients. Those conditions provide an extreme situation that highlights an important fact, for it follows that, whenever anything covers the skin, the temperature immediately below that cover is altered due to modifications of the local heat exchanges (reactive error).

Even though the skin is rarely equilibrated with the body core, the ***Zeroth Law*** requires thermal equilibration with the sensor, and to achieve this, both intimate contact with the skin and insulation from ambient conditions are essential. Most often, these are both obtained (or approximated) using adhesive tape to cover and secure sensor contact, with

commercial skin sensors typically having a small insulating mass mounted on the rear surface. These characteristics modify local temperatures (reactive error), and this occurs with all contact thermometers (Hardy, 1934a, 1934b; Stoll and Hardy, 1950; Tyler, 2011). In addition, the size (heat sink) and temperature of the sensor can modify the local skin temperature when first applied (Hardy, 1934b; Sasaki and Kamada, 1952), as can the contact pressure (Stoll and Hardy, 1950; Guadagni *et al.*, 1972; Jirák *et al.*, 1975; Mahanty and Roemer, 1979). One must therefore not be surprised at the observation that contact and covered skin temperatures differ from each other (Buono and Ulrich, 1998; James *et al.*, 2014) and from the corresponding non-contact values (Buono *et al.*, 2007; Psikuta *et al.*, 2014), particularly if bias was introduced through atypical measurement practices (Buono and Ulrich, 1998). Moreover, comparing measurement devices using human skin introduces additional errors "*because its temperature is not known independently and its temperature is not constant either as to time or locality*" (Hardy, 1934b: P 613). Indeed, the above differences must always exist, and their artefactual impact must be considered for each experiment.

Therefore, if skin temperatures without bias are required, one should consider non-contact techniques that measure radiation emitted from the skin surfaces (Figure 12: Aldrich, 1928; Hardy, 1934a; Pennes, 1948; Nakayama *et al.*, 1977, 1981; Livingstone *et al.*, 1987; Torii *et al.*, 1992; Vallerand *et al.*, 1992b; Choi *et al.*, 1997; Zaproudina *et al.*, 2008; Costello *et al.*, 2012; Fournet *et al.*, 2013; Marins *et al.*, 2014). Unfortunately, this technique cannot be used to determine skin temperatures under clothing without significant modification. The method is based on the principle that all objects with a temperature above absolute zero emit electromagnetic radiation. Thermal-imaging cameras require information concerning the efficiency with which the target object emits thermal (infrared) radiation to calculate temperature. This defines the emissivity of that object, which is a ratio, and has a value between one (black body) and zero (white body). The emissivity of human skin is 0.97-0.98 (Hardy and Muschenheim, 1934; Mitchell *et al.*, 1967; Watmough and Oliver, 1968; Togawa, 1989), but errors in determining that value will be transferred to all calculations (Bernard *et al.*, 2013).

Hardy (1934c) determined that water vapour above the skin did not significantly influence skin radiation. However, recent research has shown that substances on the skin surface

produce emissivity changes (Bernard *et al.*, 2013). Of relevance here is the presence of moisture. Since water has a lower emissivity than skin, it lowers the overall emissivity, and in their investigation, where the emissivity was set at 0.98, it resulted in temperature errors >3.0°C. This has significant implications for measurements in sweating individuals.

**Skin temperatures**

As the skin represents the interface between the body and the environment, the accurate measurement of its temperature is important, yet several assumptions underlie the measurement of local and mean skin temperatures. The validity of those assumptions depends upon what is measured and why. For example, skin temperature is sometimes measured to quantify thermal gradients, and therefore heat exchanges between the body surface and the environment or the deep-body tissues. The pattern and dynamic nature of skin temperature changes following some perturbations (*e.g.* local cooling) are used in the diagnostic assessment of locomotor and vascular disorders and some malignancies. In this context, post-cooling increases in skin temperature are assumed, sometimes erroneously, to be an analogue of skin blood flow (Davey *et al.*, 2013). However, the actual temperature obtained for these differing functions depends on the location and number of sites measured, the ambient medium and its temperature, the metabolic state of the body (rest, exercise) and the stimulus applied to the thermoregulatory system (thermal, chemical).

Skin temperature has most often been measured using sensors attached to the skin surface, and as noted earlier, the method of attachment can change both the dynamic response and absolute temperature. For field use, wires can become limiting, and so other devices have been developed, including telemetry-based systems, iButtons®, indwelling subcutaneous sensors and infrared thermographic devices. Whilst these are not without limitations and logistical challenges, they offer alternatives, and may be the best option in some circumstances (Clark and Edholm, 1985; van Marken Lichtenbelt *et al.*, 2006; Dugay *et al.*, 2009). In very dynamic phases, such as sudden immersion into cold water, skin temperatures change very rapidly, driving the autonomically evoked cold-shock responses (Tipton, 1989). Even the psychophysical responses appear closely related to local skin temperature change rates (Guéritée, 2012). Clearly, it is critical that the dynamic response characteristics of the methods are fast enough to accurately track those rapid changes. Whilst seemingly obvious, this requirement is often overlooked, and it is sometimes

difficult to quantify.

Accurate skin temperature measurement is critical to measuring transcutaneous heat flux, but a frequently observed error is the assumption that the two are always synonymous. They are not. Indeed, a range of heat fluxes is possible at the same skin temperature depending, again, on the environment, physical activity and tissue insulation. For example, an increase in temperature is often taken to indicate cutaneous vasodilatation, and the corresponding and reciprocal changes in heat flux and tissue insulation. Whilst this can be the case, the quantity of heat flowing down the thermal gradient must also be considered. During exercise, for example, cutaneous vasodilatation is driven by an elevated mean body temperature (Werner *et al.*, 2008), yet the skin temperature may be prevented from rising by the similarly activated secretion and evaporation of sweat (Clark *et al.*, 1977; Torii *et al.*, 1992; Gagge and Gonzalez, 1996). Nevertheless, tissue insulation actually decreases, vasodilatation occurs and 2-3 times more heat is transferred down a similar thermal gradient. Therefore, skin temperatures (turnover indices) and heat fluxes both depend upon local heat production and the dry and evaporative exchanges (Burton and Edholm, 1955).

At exercise onset, during its progression and also at its cessation, changes in skin temperature are complex, regionally variable and often transient in nature. As one commences exercise, feedforward emanating from the rostral brain simultaneously activates motor and sympathetic neurons, producing a transitory reduction in skin blood flow whilst sweating increases (Christensen and Nielsen, 1942; van Beaumont and Bullard, 1963). Therefore, the delayed rise in skin temperature noted above can also be ascribed to a short-term reduction in the convective delivery of central heat to the periphery.

As exercise continues, there are concomitant increases in intramuscular and deep-body temperatures (Pembrey, 1898; Kenny *et al.*, 2003, 2006; Todd *et al.*, 2014), with the former occurring very rapidly (Figure 4). Accordingly, skin temperature directly over the working musculature rises by several degrees (Clark and Edholm, 1985), and these temperatures must deviate with regional variations in intramuscular temperature observed during exercise and recovery (Figure 11). A further complication relates to the presence of forced convective and evaporative cooling during exercise, particularly that which is performed under field conditions (Clark *et al.*, 1977; Saunders *et al.*, 2005).

Of course, individual skin temperatures are determined by local heat exchanges with the muscle and environment. Therefore, since muscles generate and loose heat at different rates, then some skin regions will experience faster temperature changes than others. Since blood pools and traps heat in the dependent zones during recovery (Kenny *et al.*, 2010; Kenny *et al.*, 2008), those skin sites will remain warmer for longer following the cessation of exercise. Finally, when exercise stops, the thermal influences of forced convection disappear and heat production returns more slowly towards resting levels. The implication of these interactions is that the reported independence of mean skin temperature from work rate can be meaningless when considered in terms of local skin temperature changes.

Instead, absolute and regional variations in resting skin temperature depend upon local heat production, the convective delivery of heat and local heat exchanges that are powerfully influenced by ambient temperature and, to a lesser extent, the method of measurement. During exercise, these variables are further modified, but not simply by its presence, but by the exercise mode and its duration, and whether or not vasodilatation and sweating have been evoked and to what level. Other factors that influence the absolute and dynamic profile of skin temperature may include: gender, age, ethnicity, subcutaneous adiposity and its distribution, and the presence of clothing and tissue damage (*e.g.* burns) or various chemicals applied to the skin (Colucci *et al.*, 1982; Graham *et al.*, 1989; Inoue *et al.*, 1992; Taylor *et al.*, 1995; Gillis *et al.*, 2010). All of these factors interact. It therefore becomes apparent that, quite apart from what may appear to be a very simple measurement, skin temperatures have very complex interactions that can always complicate, and can often confuse or defy interpretation.

**Combining skin temperatures into a meaningful mean**

Individual skin temperatures are normally combined to provide a mean skin temperature for the purposes described above. Therefore, to know that temperature with absolute precision, one must take measurements from every possible site. Since this is impossible, then the classical estimations used weighted summations of many sites, with mixing coefficients representing site-specific fractional contributions to the total body surface area (*e.g.* DuBois and DuBois, 1915; ISO 9886:2004). Notwithstanding this practice, the greater the number of skin sites sampled, the smaller is the fractional representation of the total surface area of each site and the greater is the accuracy of the ensuing estimation.

Perhaps the most demanding method remains that proposed by Winslow *et al*. (1936), with 15 measurement sites advocated. At the other end of the range, there are suggestions of three (Burton, 1934) and four sites (Ramanathan, 1964; Mitchell and Wyndham, 1969). Yet these too have their utility, providing the experimental conditions elicit thermal homogeneity across the skin surface (Teichner, 1958; Olesen, 1984), and providing one accepts reduced accuracy (Olesen, 1984; Choi *et al*., 1997). For example, Clark *et al*. (1977) reported a disparity of up to 4°C in mean skin temperatures between infrared and thermocouple measurements, with the size of the difference depending upon the number of sites used in the latter method. When 11-13 sites were used, the difference was $<1.5$°C.

Olesen (1984) compared ten equations for estimating mean skin temperature with the temperatures obtained from 14 unweighted skin sites chosen to represent an equal area of the body surface. It was determined that the number of sites necessary to obtain an accurate estimation of mean skin temperature was related to the extent of the intra-site variability. Thus, in warm conditions, 2-4 sites could be sufficient. For thermoneutral conditions, 4-8 sites were necessary, and in cold conditions, 8-12 sites were recommended. Olesen (1984) concluded that if one required a mean skin temperature estimate that was 95% certain to be within 0.2°C of its actual value, then it was necessary to measure 10-14 sites. However, an accuracy of 1°C could be achieved with just 2-6 sites. Finally, since large variations in skin temperatures are apparent for sites in close proximity, then precise sensor placement during repeated trials becomes essential, yet such precision is infrequently reported or missing.

One frequently finds mean skin temperature calculations developed for use in one environment being used in inappropriate and un-validated conditions; for example, the Ramanathan (1964) method is often used in cold conditions. Therefore, careful consideration should be given to the number of sites and locations sampled; these must vary with the nature of the environment, clothing worn and responses investigated. Whilst traditional surface area weighting methods are useful for determining heat exchange at the body surface, this approach assumes an homogenous and uniform distribution of cutaneous thermoreceptors; such distributions do not exist (Hardy and Oppel, 1938). If one is looking for a relationship between cutaneous thermoafferent feedback and a thermoeffector response, then weighing coefficients might be of greater utility if based on thermoreceptor density or regional variations in cutaneous thermosensitivity. For example, when examining

thermal sensation and comfort in transient and non-uniform thermal environments, one might expect an improved correlation between skin temperature and the psychophysical responses if a greater weighting was given to the hands and feet; which dominate thermal comfort in the cold (Zhang *et al.*, 2004). In hotter conditions, sudomotor function and the psychophysical responses display regional variations in thermosensitivity (Cotter and Taylor, 2005). In these circumstances, the skin temperature model used should perhaps be more related to the cortical homunculus than to a thermal manikin.

Perhaps unsurprisingly, many of the conclusions reached for the measurement of deep-body temperature also apply to the measurement of skin temperature: measurement validity should be evaluated relative to the research objectives, and the responsiveness of the temperature index must match the forcing function used to disturb homeostasis. It follows from the above that detailed consideration should also be given to the number and distribution of skin temperature measurements, and the precision and reproducibility of those measures with respect to the ambient medium, its temperature, the characteristics of the population sample (gender, age, ethnicity, adiposity), the interaction of exercise and the use of clothing. Compromise is inevitable, if only for logistical reasons, but measurement limitations should be recognised and considered when analysing and interpreting data.

## CONCLUSION

This review was aimed at providing comprehensive theoretical and empirical justifications for the selection and use of valid body temperature measurements that would best match the research objectives of, and the sensitivity of the forcing functions applied within, each experiment. Not all readers will accept every interpretation. However, it is hoped that sufficient material has been assembled to allow such decisions to be based upon objective evaluations of the available evidence. A further purpose was a possible restoration of the past common knowledge concerning temperature measurements, so that some contemporary misconceptions might gradually disappear. Since tissue temperatures are turnover indices modified by local metabolism, tissue conduction and blood flow, it has been posited that a gold standard deep-body temperature does not exist. Moreover, temperature similarities or differences among sites are proposed to lack a mechanistic relationship unless those sites are in close proximity, are perfused by the same blood vessels and have equivalent metabolic rates. It is therefore concluded that, for thermometric computations of whole-

body heat storage, the establishment of steady-state conditions is absolutely essential, as is the simultaneous measurement of several deep-body temperatures. For many clinical states, targeted temperature monitoring is critical. When investigating temperature regulation during dynamic phases, deep-body temperatures must either be measured from the central (cardiac or pulmonary) blood volume or from sites that reliably track those temperatures.

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**CONFLICT OF INTEREST**

There are no conflicts of interest.

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## FIGURE CAPTIONS:

**Figure 1:** Newton's Cooling Law (Newton, 1700) and the heat conduction equation (Fourier, 1807). The rate of temperature change of an object ($^oC.s^{-1}$) is dependent upon the size of the difference between its initial and final steady-state temperatures ($^oC$). **Figure 1A:** Temperatures were recorded from the centre of a steel sphere (mass 6.98 kg, radius 0.061 m). Following thermal equilibration to each of three steady-states (stirred water baths: 15°, 25° and 35°C), the sphere was transferred to a second regulated water bath (38.5°C stirred). Warming curves are shown for each of these three trials, with warming rates indicated for the period 80-150 s (shaded). Thermal equilibration was achieved at about 450 s in each trial. **Figure 1B:** To better illustrate the heat-transfer law and the impact of the heat conduction equation, these instantaneous temperatures were expressed as non-dimensional changes: temperature change = $(T_i-T_f) / (T_o-T_f)$; where: $T_i$ = instantaneous temperature, $T_o$ = initial temperature, $T_f$ = final temperature. **Figure 1C:** It is sometimes easier to visualise these concepts in terms of thermal gradients and change rates. The thermal gradient of the 25°C trial was 3.96 times that of the 35°C trial (left bars and ordinate), while the gradient for 15°C was 1.77 times larger than the 25°C condition. Accordingly, when the rates of temperature change at each 5-s interval were averaged over the entire warming phase (80-450 s: right bars and ordinate), identical between-trial ratios were observed for the thermal gradients and the resulting temperature change rates.

**Figure 2:** A steel sphere (mass 6.98 kg, radius 0.061 m) containing well-insulated thermistors embedded at its centre, and at 33% (shallow: 0.0203 m) and 66% (deep: 0.0407 m) of the distance from the centre to its surface, was heated to a steady state of 38.5°C (stirred water bath). Once a thermal equilibrium was established at all sites, the sphere was transferred to a second, but cooler stirred water bath (15°C: time zero) until the temperature of the sensor at the centre of the sphere first started to decrease (position A: 35 s). At this point, the sphere was moved back into the first water bath (38.5°C), and the temperature profile of each sensor was recorded (5-s intervals) until local temperatures had all risen to 35°C. Positions B, C and D identify points where heat loses changed to heat gains, whilst E and F coincide with temperature trace cross-overs. This simulation was inspired by Golden (1979) and Golden and Hervey (1981).

**Figure 3:** Whole-body calorimetry data illustrating total heat production rate (metabolic minus external work rates) and total (nett) heat loss rate (dry and evaporative) during seated rest, cycling at an external work rate of 70 W (0-60 min) and a 60-min seated, resting recovery (**Figure 3A**: air temperature 30°C, water vapour pressure 5.4 kPa). The difference between these heat production and heat loss rates (grey shaded zone) dictates whole-body heat storage (**Figure 3B**). Data are means with standard errors of the means ($N$=8). Modified from Kenny *et al*. (2008) and used with permission.

**Figure 4:** Oesophageal, auditory-canal (insulated), rectal and intramuscular (*vastus lateralis*) temperatures plotted with mean local sweat rate (five sites [sweat capsules]: forehead, chest, ventral forearm, bilateral anterior thighs) during three consecutive, sinusoidal work-rate waveforms (cycling: 8-min periods; 60 rev.min$^{-1}$; 25.2°C, 1.15 kPa), increasing from 30 W to 60% of each subject's peak power (mean: 206 W). This forcing function followed 20 min cycling at 35% of peak power (mean: 118 W) to establish thermal and thermoeffector steady-states. Data are average curves (sampled at 5-s intervals, $N$=8) extracted from Todd *et al*. (2014) and used with permission.

**Figure 5:** Tympanic membrane, rectal, oesophageal and zero-gradient auditory-canal temperatures during steady-state cycling at 40% peak power in hot, moderately humid conditions (36.6°C, water vapour pressure 2.62 kPa). Data are average curves with means and standard errors of the means at approximately 5-min intervals ($N$=6). Local sweat rates (sweat capsules) for the forehead (upper) and dorsal foot surfaces are also shown (grey dotted curves). Modified from Cotter *et al*. (1995) and used with permission.

**Figure 6:** Variations in resting, deep-body temperature among twelve measurement sites (ascending order). Data were extracted from the reports listed below, with means and 95% confidence intervals for each total sample. Numbers above the confidence intervals are the total number of subjects, whilst those below indicate the number of contributing investigations.

***Sources:*** **Axilla:** Ilsley *et al*. (1983), Darowski *et al*. (1991), Childs *et al*. (1999). **Sublingual:** Linder and Carmichael (1935), Ivy (1944), Barcroft and Edholm (1946), Consolazio *et al*. (1963), Fox *et al*. (1971), Fox *et al*. (1973b), Collins *et al*. (1977), Edwards *et al*. (1978) Erickson (1980), Kolanowski and Gunter (1981),

Ilsley *et al*. (1983), Mairiaux *et al*. (1983), Thatcher (1983), Baker *et al*. (1984), Eriksson *et al*. (1985), Keilson *et al*. (1985), Terndrup *et al*. (1989), Darowski *et al*. (1991), Marion *et al*. (1991), Mackowiak *et al*. (1992), Maw and Taylor (1992), Castle *et al*. (1993), McGann *et al*. (1993), Nakamura *et al*. (1997). **Auditory canal:** Edwards *et al*. (1978), Terndrup *et al*. (1989), Darowski *et al*. (1991), Chamberlain *et al*. (1995), Shibasaki *et al*. (1998), Childs *et al*. (1999), Easton *et al*. (2007), Jay *et al*. (2007b), Nagano *et al*. (2010), Lee *et al*. (2011), Taylor (2012: unpublished observations). **Liver:** Graf (1959). **Stomach:** Graf (1959). **Tympanic membrane:** Brinnel and Cabanac (1989), Mariak *et al*. (1993), Cotter *et al*. (1995), Shibasaki *et al*. (1998). **Oesophagus:** Saltin and Hermansen (1966), Keatinge and Sloan (1975), Edwards *et al*. (1978), Mairiaux *et al*. (1983), Brinnel and Cabanac (1989), Maw and Taylor (1992), Cotter *et al*. (1995), Kolka *et al*. (1997), Shibasaki *et al*. (1998), Lee *et al*., (2000), Booth *et al*. (2004), Jay *et al*. (2007b), Nagano *et al*. (2010), Taylor (2012: unpublished observations), Wilsmore (2008). **Zero-gradient auditory canal:** Keatinge and Sloan (1975), Maw and Taylor (1992), Cotter *et al*. (1995), Wilsmore (2008). **Urine:** Marion *et al*. (1991). **Rectum:** Linder and Carmichel (1935), DuBois (1941), Bazett *et al*. (1948), Pennes (1948), Eichna (1949), Horvath *et al*. (1950a), Graf (1959), Consolazio *et al*. (1963), Saltin and Hermansen (1966), Fox *et al*. (1971), Edwards *et al*. (1978), Kolanowski and Gunter (1981), Ilsley *et al*. (1983), Mairiaux *et al*. (1983), Terndrup *et al*. (1989), Maw and Taylor (1992), Chamberlain *et al*. (1995), Cotter *et al*. (1995), Lee *et al*., (2000), McKenzie and Osgood (2004), Easton *et al*. (2007), Jay *et al*. (2007b), Nagano *et al*. (2010), Lee *et al*. (2010, 2011), Taylor (2012: unpublished observations), Wilsmore (2008). **Gastriointestinal tract:** Kolka *et al*. (1997), Lee *et al*., (2000), McKenzie and Osgood (2004), Gant *et al*. (2006), Easton *et al*. (2007), Pearson *et al*. (2012), Taylor (2012: unpublished observations). **Bladder:** Ilsley *et al*. (1983).

**Figure 7:** Rectal (A) and sublingual (B) temperature distributions in humans with means from Figure 6 shown as dotted vertical lines. Data for **Figure 7A** were extracted from DuBois (1941) for afebrile men and women under supine, basal conditions ($N$=252 [digitised from original]). The solid vertical line shows the derived sample mean (36.99ºC) based on the digitised counts. Modified and used with permission. **Figure 7B** was extracted

from Eriksson *et al*. (1985) for afebrile, seated men (57-75 years; $N$=760). The mean was provided by the original author (solid vertical line: 36.70ºC). Modified and used with permission. Ivy (1944) also provided a sublingual temperature distribution: seated medical students ($N$=276; mean: 36.73ºC), although the methods of Eriksson *et al*. (1985) were considered to be superior.

**Figure 8:** Temperatures and thermal gradients within the arteries and veins of normothermic humans, arranged to illustrate heat flow direction. Data are means obtained from the literature (below), with 95% confidence intervals. Data for all but three sites were taken from single experiments, and those sites are indicated by dataset numbers to the left of each mean. Numbers to the right are site-specific sample sizes.

***Sources:*** Bazett *et al*. (1948: brachial artery), Eichna (1949: femoral artery and vein), Pennes (1948: brachial artery), Eichna *et al*. (1951: all sites except brachial artery), Ilsley *et al*. (1983: pulmonary artery), Mariak *et al*. (1993: carotid artery), Pearson *et al*. (2012: pulmonary artery).

**Figure 9:** Deep-body temperatures during a sequential cooling and heating trial. Following preparatory thermal equilibration (phase 1: 20ºC; swimming costume only), the subject (an author) was positioned above an immersion tank (phase 2: 12.5ºC) and lowered into the water (12 min), remaining there until the oesophageal temperature reached 35ºC (phase 3). At this time (44.5 min), he was removed from the water (phase 4), dried and immediately transferred into a pre-heated climate chamber (40ºC), sitting at rest for 12 min (phase 5). Note the afterdrop at each site, but most pronounced for rectal and gastrointestinal temperatures. The rectal probe was partially and transiently dislodged during phase 5 (data omitted). Finally, steady-state cycling commenced (100 W: phase 6), and continued until oesophageal temperature rose to 39ºC. Data were sampled at 5-s intervals except for gastrointestinal temperature (1-min intervals). This trial was performed on four individuals, all of whom showed qualitatively similar responses, but the timing varied, as this was set by the oesophageal temperature targets. The aberration in gastrointestinal temperature (82-85 min) was possibly physiological in nature, and occurred in two participants.

**Figure 10:** Variations in gastrointestinal-tract temperature in one individual (an author) during the complete transit of seven radio pills (Jonah 500-0100-02, Respironics

Deutschland, Herrsching, Germany) over a 37.35-h period. Each pill was equilibrated in water at 37°C and consumed with that water, with ingestion sequence corresponding to the sensor numbers. **Figure 10A**: The complete transit data for seven sensors are shown following removal of ingestion- and exercise-induced artefacts as well as random errors, so that these data reflected sedentary activities. Times are referenced to ingestion time (0 h). Data logging commenced on day 1 at 18:27 h (sensor 1) and 21:36 h (sensor 2), and on day 2 at 05:07 h (sensor 3), 09:40 h (sensor 4), 13:25 h (sensor 5), 17:53 h (sensor 6) and 21:58 h (sensor 7). Data were smoothed (5-point averages) and reported at 15-min intervals. In **Figure 10B**, data from two sensors consumed immediately before retiring on each night are shown (sensors 2 and 7). Time zero signifies retiring. Sleep commenced within 30 min, with arousal occurring 6.1 h (sensor 2) and 7.2 h (sensor 7) after retiring. Data were smoothed (5-point averages) and reported at 15-min intervals. **Figure 10C**: Endurance running (shaded area) and recovery. Data were collected at 1-min intervals and were not smoothed. Legend times show the time from ingestion to the first data point on the graph. The time axis is referenced to the start of data-collection (Figure 10A). **Figure 10D**: Resistance exercise (shaded area) and recovery. Data were collected at 1-min intervals and not smoothed. Legend times show the time from ingestion to the first data point on the graph. Time is referenced to the start of data-collection (Figure 10A).

**Figure 11:** Intramuscular temperatures from three body segments recorded during cycling at an external work rate of 70 W (0-60 min) and during a 60-min seated, resting recovery (air temperature 30°C, water vapour pressure 5.4 kPa)). Data are means with standard errors of the means ($N$=8). Modified from Kenny *et al*. (2008) and used with permission.

**Figure 12:** Variations in regional skin temperatures (infrared thermography) obtained from resting (standing), normothermic males ($N$=16). Data are means with 95% confidence intervals taken from Zaproudina *et al*. (2008), and used with permission of IOP Publishing ().

**Figure 1:**

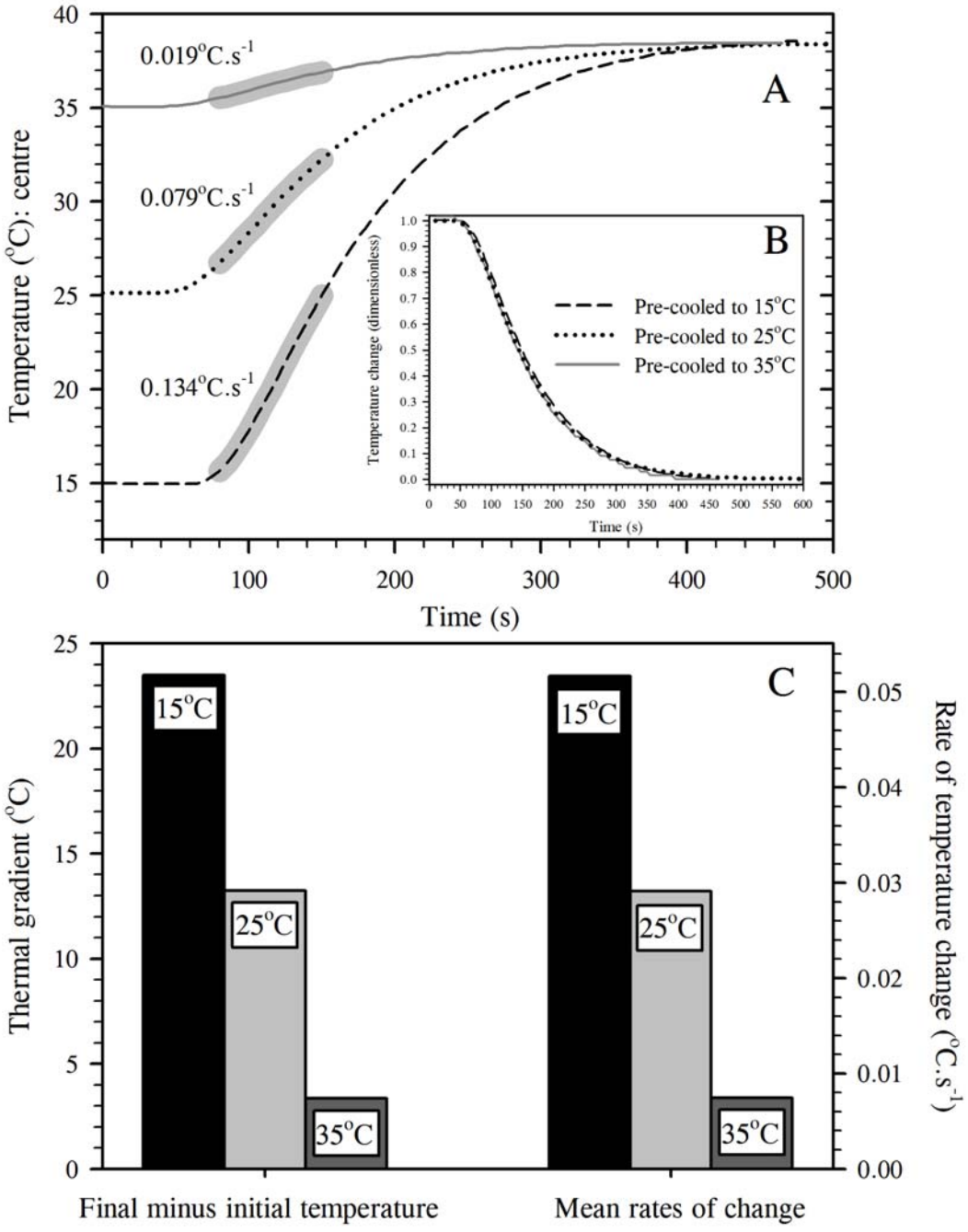
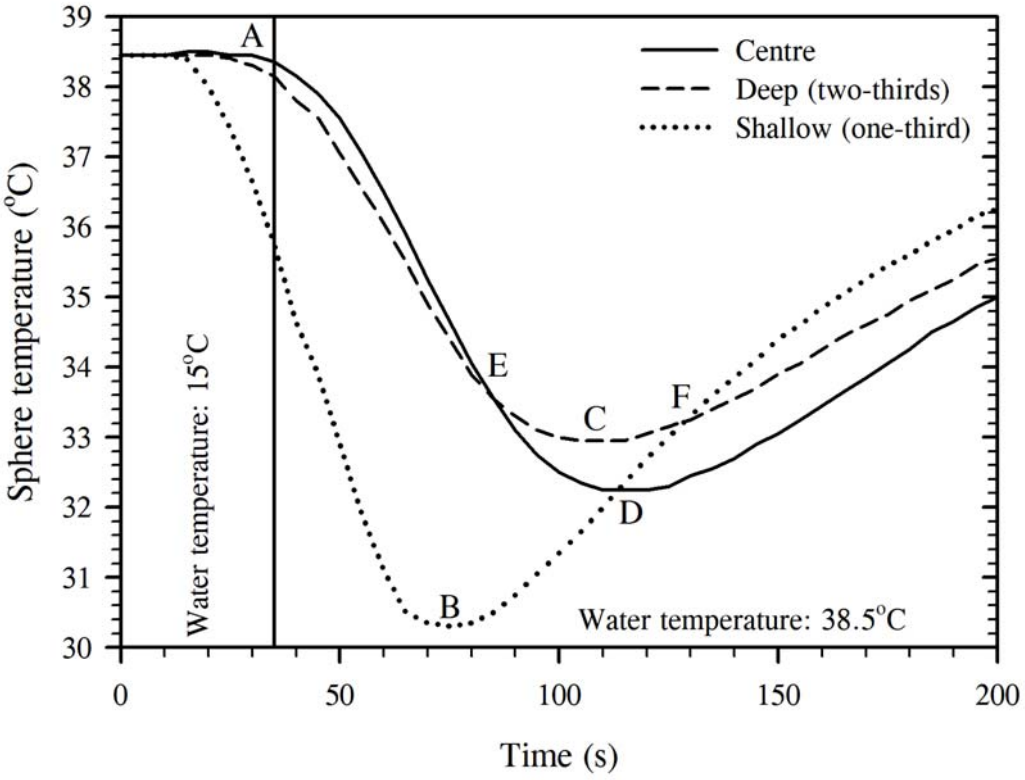

A
0.019°C.s-1
0.079°C.s-1
0.134°C.s-1
Temperature (°C): centre
Time (s)
B
Pre-cooled to 15°C
Pre-cooled to 25°C
Pre-cooled to 35°C
Temperature change (dimensionless)
Time (s)
C
15°C
25°C
35°C
Thermal gradient (°C)
Rate of temperature change (°C.s-1)
Final minus initial temperature
Mean rates of change

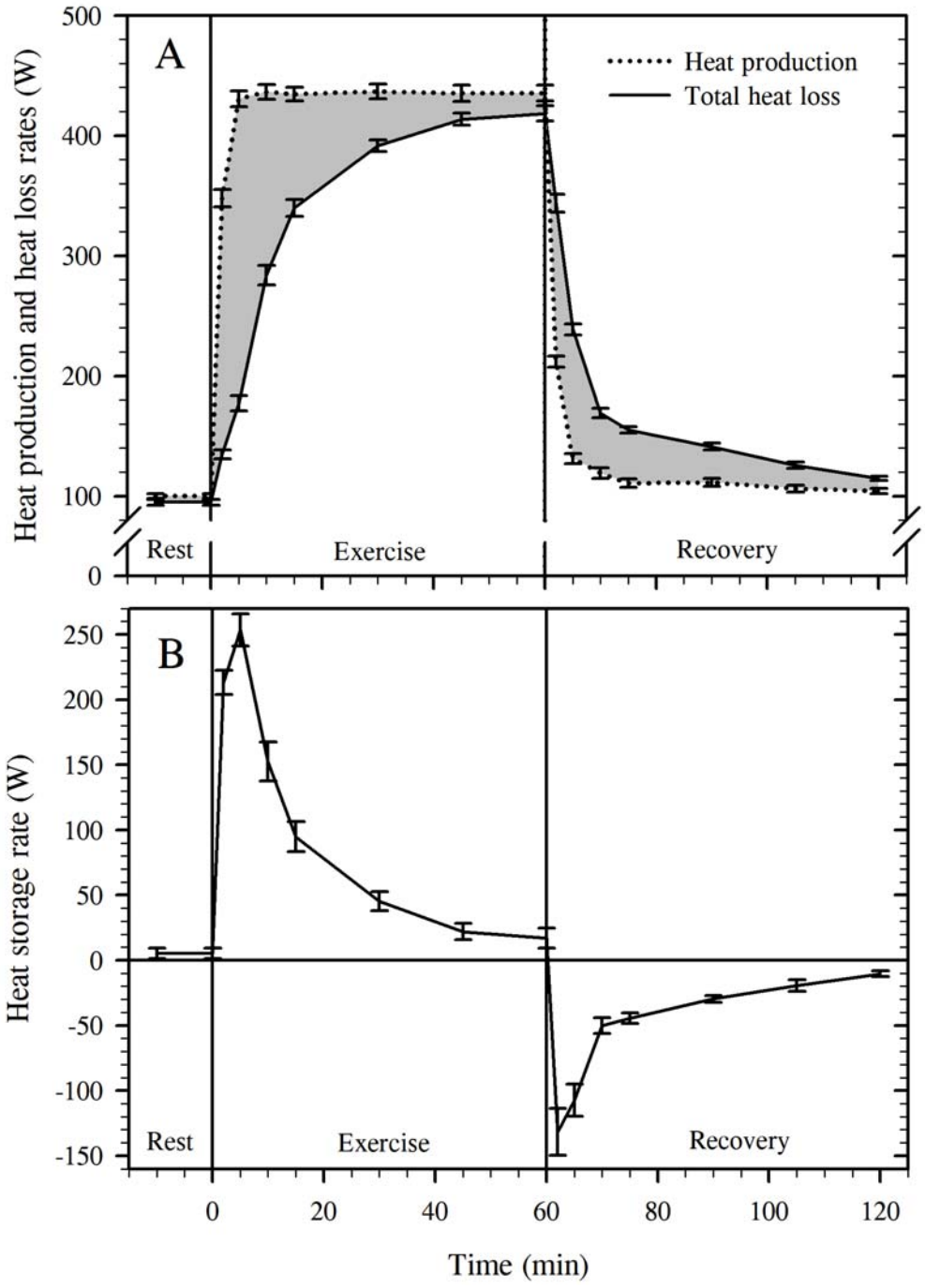
**Figure 2:**


Centre
Deep (two-thirds)
Shallow (one-third)
Sphere temperature (°C)
Time (s)
Water temperature: 15°C
Water temperature: 38.5°C
A
B
C
D
E
F
39
38
37
36
35
34
33
32
31
30
0
50
100
150
200

1 **Figure 3:**

2

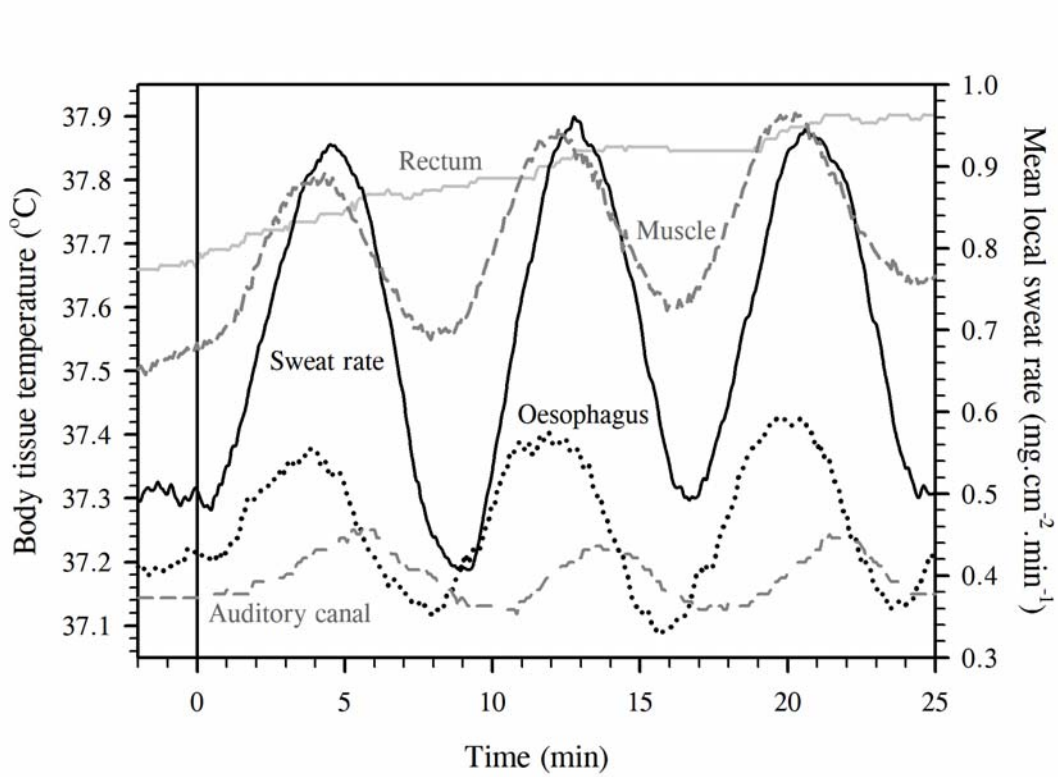
3


A
B
Heat production
Total heat loss
Heat production and heat loss rates (W)
Heat storage rate (W)
Rest
Exercise
Recovery
Time (min)
500
400
300
200
100
0
250
200
150
100
50
0
-50
-100
-150
0
20
40
60
80
100
120

1 **Figure 4:**

2

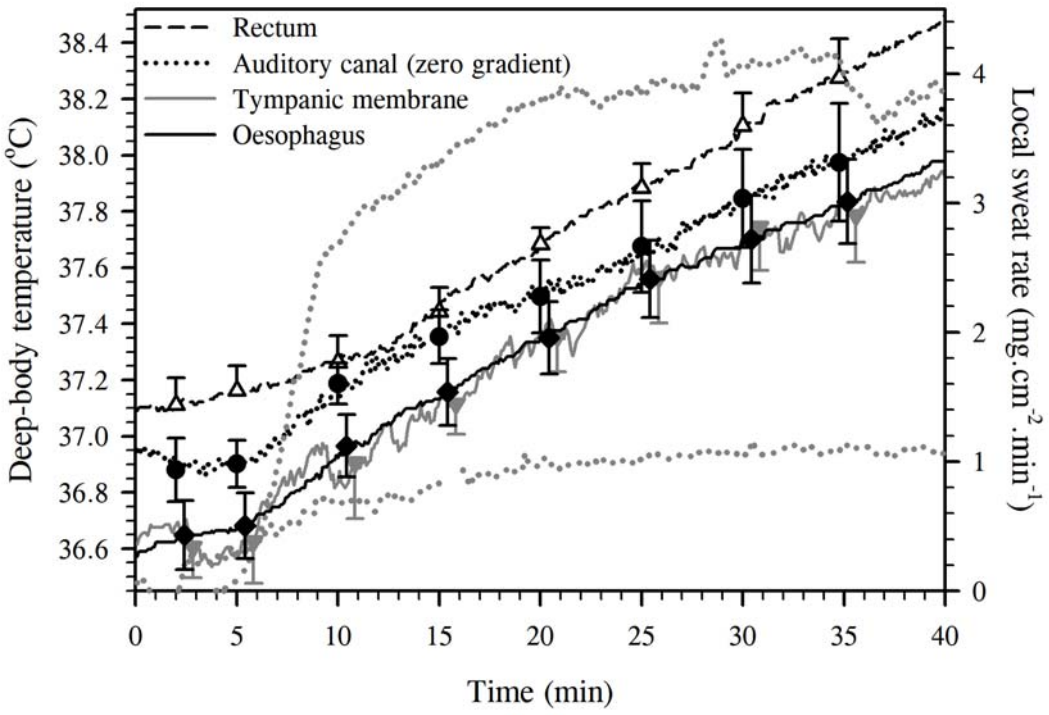
3


Body tissue temperature (°C)
Mean local sweat rate (mg.cm-2.min-1)
Time (min)
Rectum
Muscle
Sweat rate
Oesophagus
Auditory canal
37.9
37.8
37.7
37.6
37.5
37.4
37.3
37.2
37.1
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0
5
10
15
20
25

1 **Figure 5:**

2

3


Rectum
Auditory canal (zero gradient)
Tympanic membrane
Oesophagus
Deep-body temperature (°C)
Local sweat rate (mg.cm-2.min-1)
Time (min)
38.4
38.2
38.0
37.8
37.6
37.4
37.2
37.0
36.8
36.6
0
5
10
15
20
25
30
35
40
4
3
2
1
0

1 **Figure 6:**

2

3

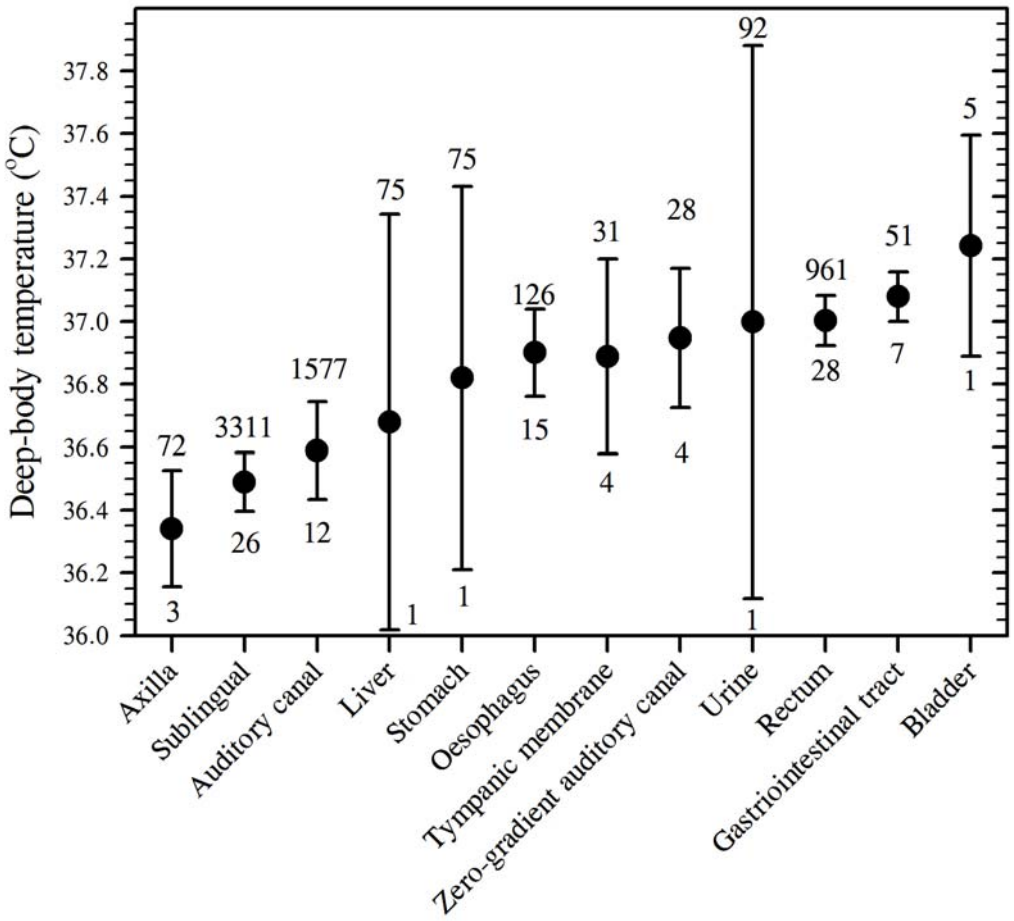

Deep-body temperature (°C)
37.8
37.6
37.4
37.2
37.0
36.8
36.6
36.4
36.2
36.0
72
3311
1577
75
75
126
31
28
92
961
51
5
3
26
12
1
1
15
4
4
1
28
7
1
Axilla
Sublingual
Auditory canal
Liver
Stomach
Oesophagus
Tympanic membrane
Zero-gradient auditory canal
Urine
Rectum
Gastriointestinal tract
Bladder

**Figure 7:**

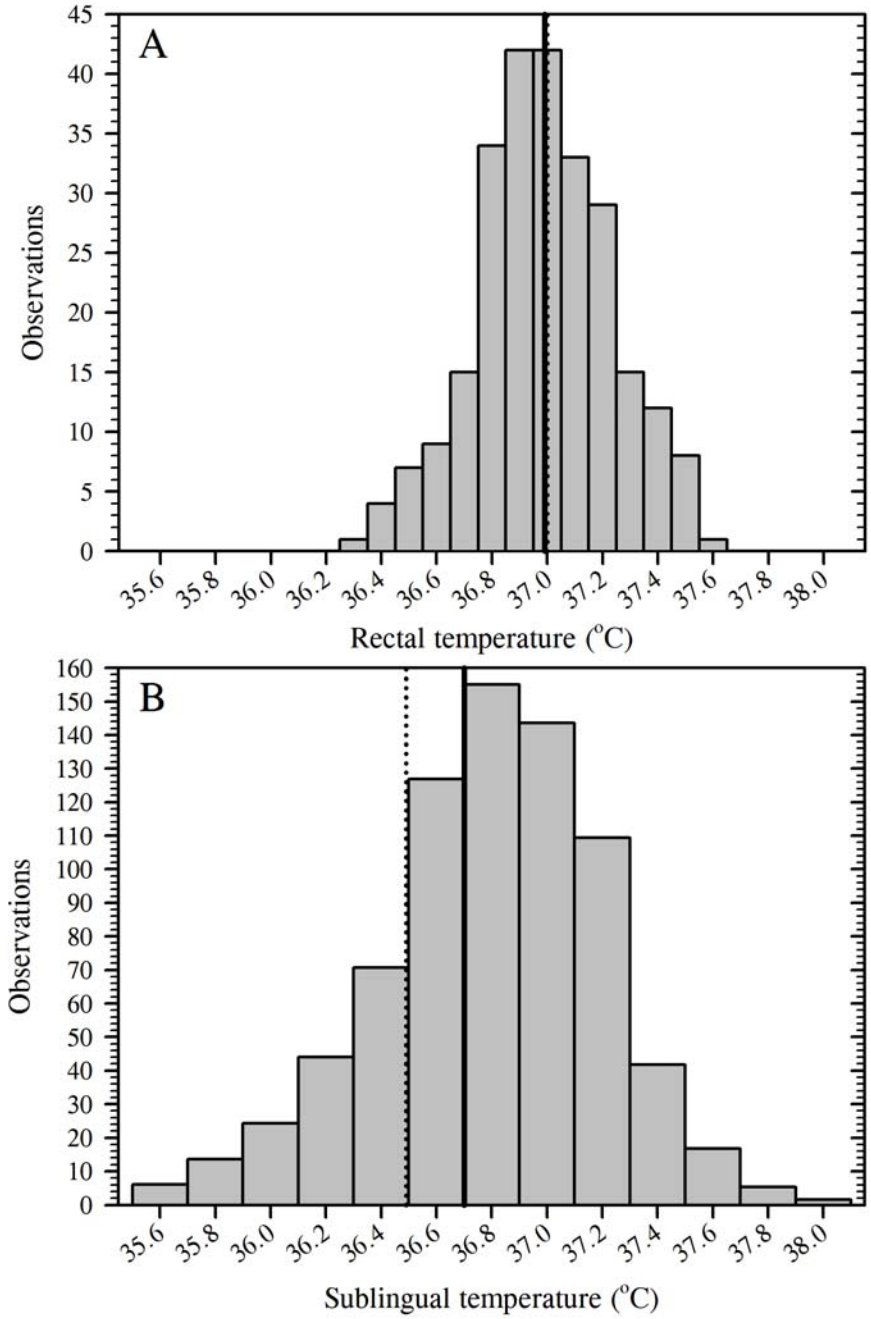

A
Observations
Rectal temperature (°C)
B
Observations
Sublingual temperature (°C)

**Figure 8:**

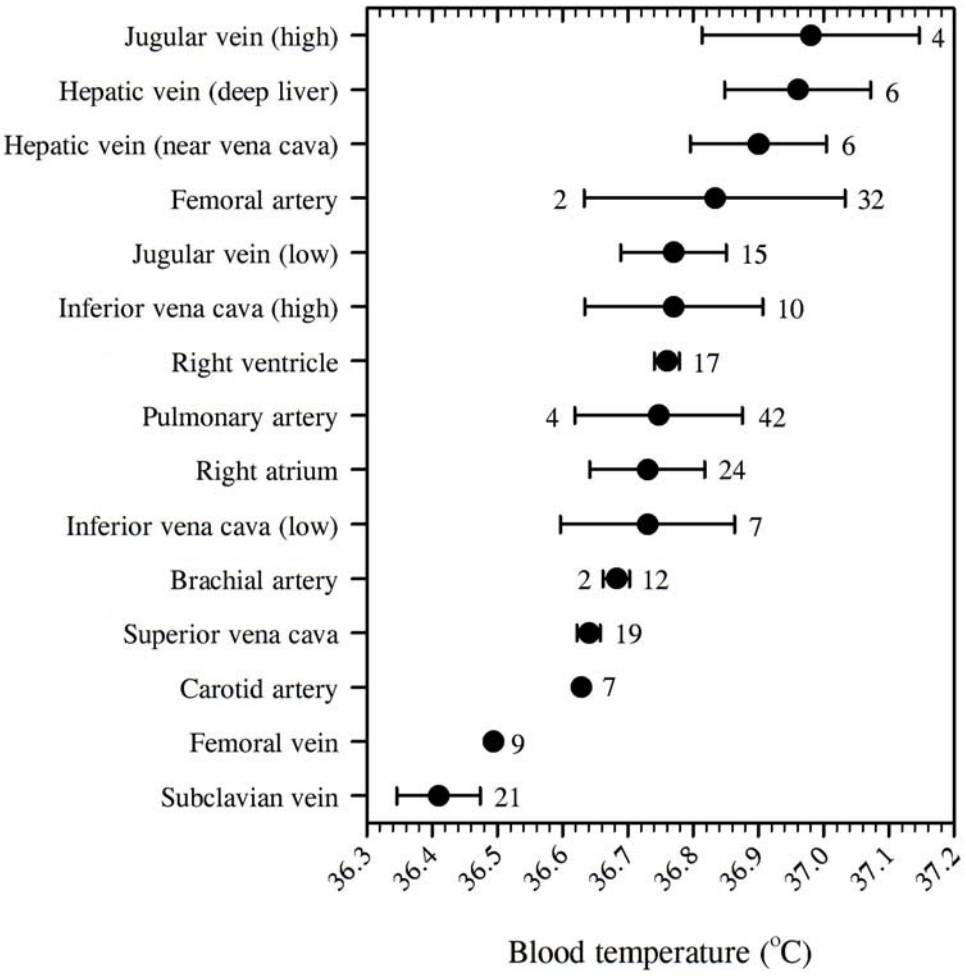
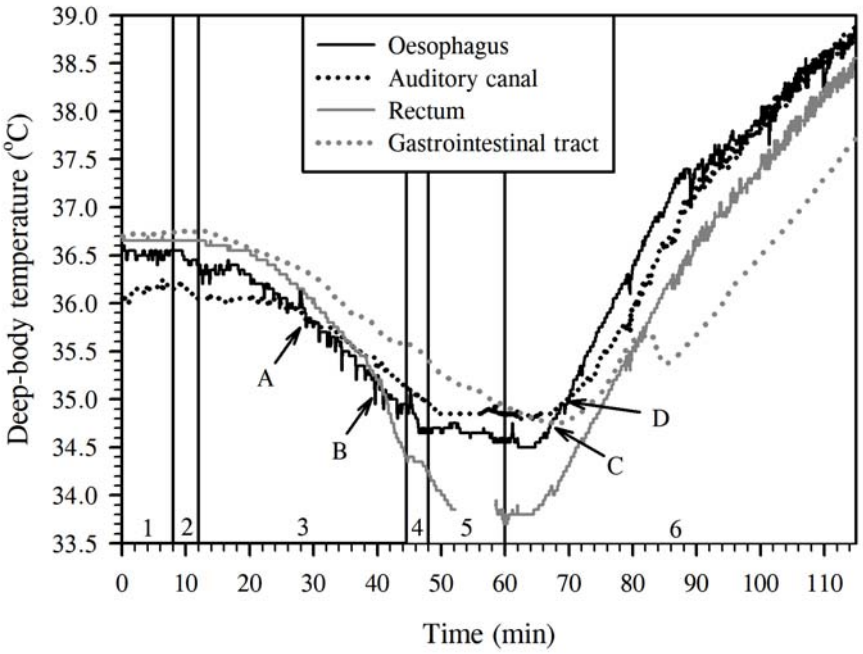

Jugular vein (high)
Hepatic vein (deep liver)
Hepatic vein (near vena cava)
Femoral artery
Jugular vein (low)
Inferior vena cava (high)
Right ventricle
Pulmonary artery
Right atrium
Inferior vena cava (low)
Brachial artery
Superior vena cava
Carotid artery
Femoral vein
Subclavian vein
4
6
6
2
32
15
10
17
4
42
24
7
2
12
19
7
9
21
36.3
36.4
36.5
36.6
36.7
36.8
36.9
37.0
37.1
37.2
Blood temperature (°C)

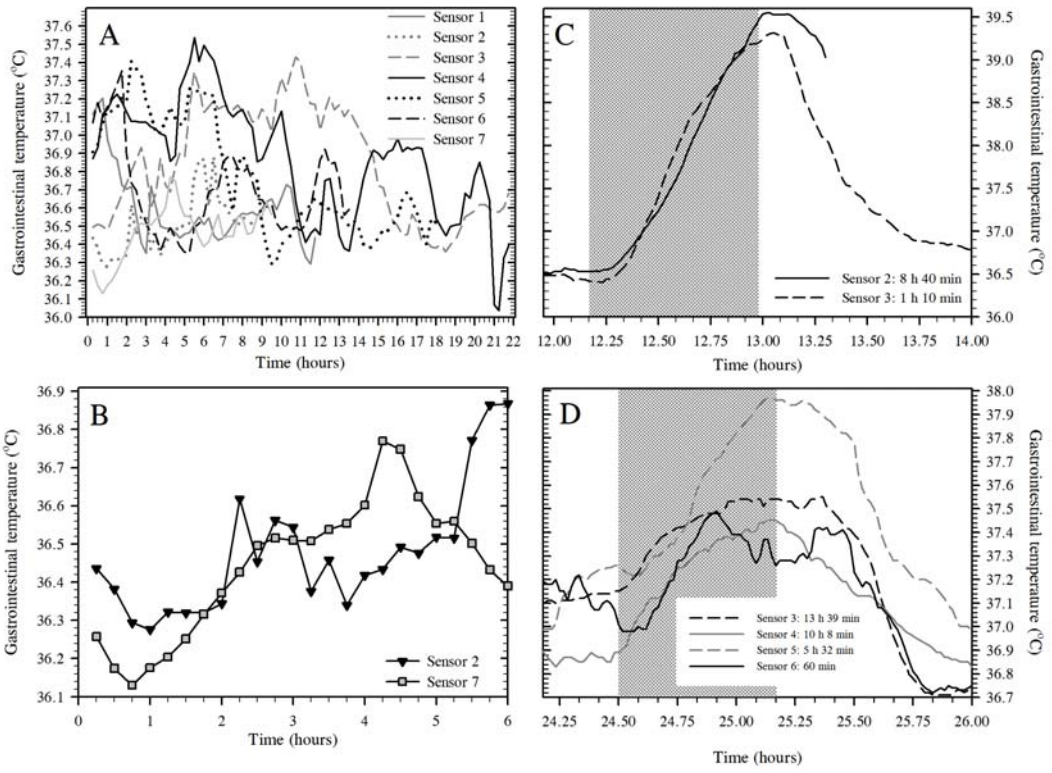
**Figure 9:**


Oesophagus
Auditory canal
Rectum
Gastrointestinal tract
Deep-body temperature (°C)
Time (min)
A
B
C
D
1
2
3
4
5
6

1 **Figure 10:**

2

3



1 **Figure 11:**

2

3

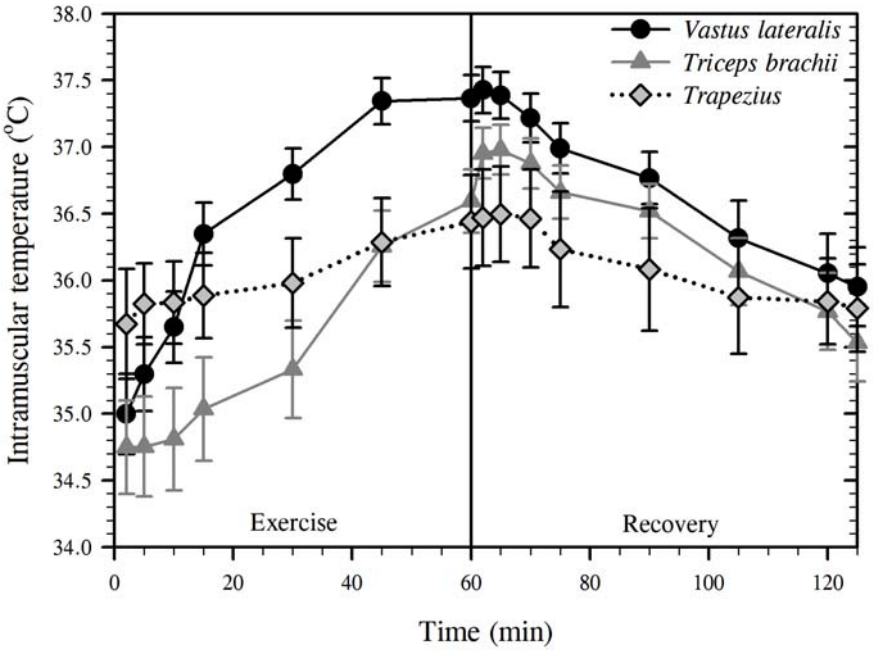
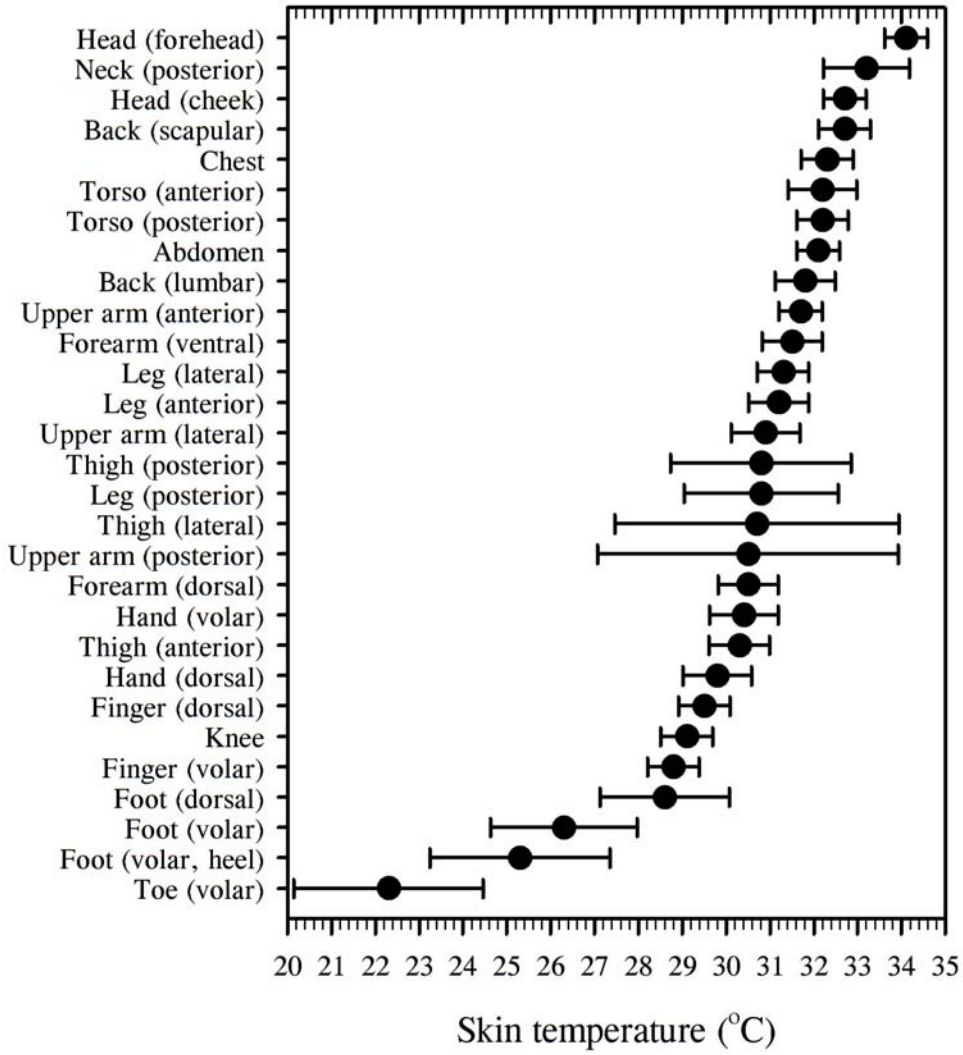


**Figure 12:**


Head (forehead)
Neck (posterior)
Head (cheek)
Back (scapular)
Chest
Torso (anterior)
Torso (posterior)
Abdomen
Back (lumbar)
Upper arm (anterior)
Forearm (ventral)
Leg (lateral)
Leg (anterior)
Upper arm (lateral)
Thigh (posterior)
Leg (posterior)
Thigh (lateral)
Upper arm (posterior)
Forearm (dorsal)
Hand (volar)
Thigh (anterior)
Hand (dorsal)
Finger (dorsal)
Knee
Finger (volar)
Foot (dorsal)
Foot (volar)
Foot (volar, heel)
Toe (volar)
20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35
Skin temperature (°C)