The Science Behind Polar Breeze®

The Research and Supporting References for Clinical Scientists

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Excessive Heat Events (EHE) is the term coined by the Environmental Protection Agency[i] to describe episodes of environmental heat stress that occur now and are likely to increase in the coming decades with the advent of global warming[ii,iii]. Such an increase in these EHE's makes serious environmental heat exposure events leading to the continuum of heat illness - heat exhaustion, heat injury, and heat stroke - all the more likely.

Heat Illness

Heat Exhaustion is a moderate form of heat illness characterized by a moderate-to-severe increase in body temperature, dehydration, and anhydrosis. A drop in cardiac output frequently underlies this condition and may result in fatigue, dizziness, headache, nausea, emesis, tachycardia, sometimes hypotension, and a potential for collapse[iv].

Heat Injury is a more severe form of heat illness with high body temperature, organ or tissue dysfunction/damage that tends to be reversible with proper treatment. However, the organ damage is frequently not apparent at the time of symptoms and so may be difficult to discern from *Heat Exhaustion*[4]. Unless properly treated and cooled, this condition may progress to *Heat Stroke*.

Heat Stroke is a life-threatening condition of profound CNS dysfunction, organ and tissue damage, and high body temperature. The organ and tissue damage may not be reversible even with treatment if not rapidly administered at onset of symptoms. Onset may be acute in *exertional heat stroke* (EHS) or gradual and progressive in *classic* (passive) *heat stroke* (CHS) where the prodrome may be nonspecific with weakness, loss of appetite, dizziness, fainting, nausea, vomiting, headache, restlessness, and confusion. The critical symptoms of *heat stroke* are mental status changes of delirium, seizures, and coma.[4,v]

Classic Heat Stroke is usually seen in passive individuals during a heat wave and is a disease of the sick and immunocompromised (including the very young and the elderly).

Exertional Heat Stroke is usually seen in young and fit individuals that collapse during high physical exercise or labor in excessive heat and/or humidity. These individuals rarely have anhydrosis, but their metabolic heat production exceeds their ability to dissipate that heat, their body temperature climbs, and *heat illness*

ensues. If cooling, rest, and fluids in these conditions are not instituted immediately and maintained, *exertional heat stroke* can develop.

Thermoregulation

The pre-optic (or supraoptic) nuclei, of the anterior hypothalamus (SONAH) in the human brain, have been recognized as the center of thermoregulation for the body at the crux of the hypothalamic-pituitary-adrenal endocrine control axis.

In laboratory animals, subjected to uncompensated heat stress, whether exercise-induced or passive, and hypothalamic temperature rises into the heat stroke range, damage to the hypothalamic-pituitary-adrenal axis occurs. Serologic markers of neuroinflammation are released. This has led to isolation of markers such as Heat Shock Factor 1[vi], monoamine oxidase, glutamate, and others[vii] from neuroinflammation as a result of the thermal insult to the hypothalamus. Multiple studies confirm laboratory data of hypothalamic damage in heat stroked rats that further advance the recognition of neurological changes during hyperthermia and heat-stroke in humans.

This is just part of the rapidly growing international consensus that damage to the hypothalamus is the probable cause of heatstroke. In laboratory work with rats, Shen-Hsien et.al.[viii] were able to show heat stroke syndrome (hyperthermia, hypotension, intercranial hypertension, hypothalamic ischemia and hypoxia) results in hypothalamic neuronal necrosis, apoptosis, and autophagy while secreting neuroinflammatory hypothalamic markers of cellular ischemia and damage, proinflammatory cytokines, oxidized radicals, and pro-oxidant enzymes. They summarized, "Taking these findings together, we conclude that ischemic, hypoxic, and oxidative damage to the hypothalamus is involved in the pathogenesis of heatstroke".

In human clinical practice, the importance of keeping the brain and SONAH below severe hyperpyrexia is used daily around the globe. *Therapeutic Hyperthermia* is the heating of the body in the 40°C to 45°C (104.0°F to 113.0°F) with chemotherapeutic agents in the treatment of cancer.[ix] As human protein begins to denature at 40.8°C, becoming irreversible by 41.6°C, with disastrous effects on the CNS structures. But this is ablated by packing the patient's head in crushed ice to keep the brain cool during these periods of hyperthermic treatment. Patients awake without apparent loss of CNS function after periods of severe therapeutic hyperthermia lasting up to an hour.

Temperature

There is no single body temperature, nor is there a single temperature that is 'normal'. This is variable individual-to-individual and dependent upon where that temperature is taken.

Rectal temperature has long been the 'gold standard' of temperatures used in physiology as being the most accurate core body temperature. Yet now we understand metabolism in the gut elevates rectal temperature above other body temperatures in normothermic conditions. Intestinal temperature, as measured by radiotelethermometry capsules (CorTemp®, VitalSense®, etc.) are variable depending upon their position in the gut at the time of reading but are generally proportional to rectal temperatures and stable. These correlations are well known[x,xi,xii,xiii].

Oral temperature is considered accurate in a cooperative passive subject who has not engage in food nor drink within 30 minutes, but this measurement is effectively useless in exertional heat stress where panting subjects cannot close their mouths and intake of cold electrolyte solutions is essential.

Esophageal temperature probes (thermistor in the esophagus behind the heart, between the lungs, and anterior to the descending aorta and inferior vena cava) are the most accurate core body temperature. The esophageal temperature has long been held as closest to aortic arch blood temperature[xiv], the accepted 'core' temperature in the laboratory. But these esophageal devices cause gagging of the conscious subjects in whom they are inserted, and so have very little use in awake patients with exertional heat illness.

Tympanic Membrane (TM) or aural temperatures were given a bad reputation by Marcus[xv,xvi] when he concluded TM-temperatures were lower than rectal temperatures attributed to environmental artifactual cooling of the ear canal, causing the lower temperature discrepancy when compared to rectal. Indeed, this dismissive view of aural temperature has persisted to this day and is reflected in athletic training journals[xvii,xviii]; firefighter[xix]; and associated[xx] literature. Thus rectal or intestinal temperature became the accepted standard for core body temperature measurement, known in the literature as T_{core}.

This bias against aural temperature is not without basis as it has inherent problems capable of skewing temperature readings. Careless placement of an aural thermometer can result in reading the temperature of the ear canal instead of the tympanic membrane[31.] The ear canal can be prone to lose or gain temperature, depending on the temperature of the external environment, and this can bias readings from the aural thermometer.

Cerumen impactions within the ear canal can also skew aural temperature readings and cause increased influence of the external environment on that reading.

Increased deviation of the aural temperature from the rectal temperature with increasing hyperthermia[11], Huggins et. al. had noted aural temperature (T_{tm}) lower than rectal (T_{re}) by only 0.27°C. pre-exercise, but T_{tm} could deviate as much as 1.91°C. lower when T_{re} was greater than 39°C. This has been a primary reason aural temperature (T_{tm}) fell into disfavor and the rectal (or intestinal) temperature (T_{re}) rose into preference for hyperthermia studies.

But it hasn't stayed that way.

Controversies in Selective Brain Cooling and Tympanic (T_{tm}) vs. Rectal/Gut (T_{re}/T_{gi}) Temperature

Rectal temperature (T_{re}) has long been the "gold standard" for core body temperature measurement in many studies, preferred over the tympanic membrane temperature of the ear (T_{tm}) as too unreliable a measure of core body temperature[xxi,xxii,xxiii,xxiv,xxv,xxvi]. Difficult positioning of the infra-red T_{tm} sensor and cerumen obstructions of the ear canal account for much of the problems associated with T_{tm}. These problems remain a limitation to T_{tm}'s accurate use in hyperthermic individuals[10].

However, thermal physiologists are very aware there is no one "core" body temperature as simultaneous tympanic, oral, esophageal, and rectal temperatures all read differently[xxvii]. Variations in these temperatures are magnified when taken during exercise-induced hyperthermia, with tympanic membrane (T_{tm}) temperature frequently reading the lowest. In passive conditions, rectal temperatures (T_{re}) appear higher than other "core" temperatures, attributed to the metabolism involved in digestion[15,xxviii]. This higher temperature is isolated to the gut by strenuous exercise when blood flow is shunted from the gut to the voluntary muscles, heart, and brain, with the subsequent divergence of brain and gut temperature.

This lower T_{tm} during hyperthermia and exercise has been attributed to automatic bodily mechanisms to lower temperature of the brain and, especially, the *supra-optic nucleus of the anterior hypothalamus* (SONAH), the putative thermal regulatory center of the body[xxix,xxx,xxxi,xxxii,xxxiii,xxxiv,xxxv,xxxvi]. And while the T_{tm} does not track T_{re}, there is work[xxxvii] supporting T_{tm} as tracking brain temperature, and thereby the risk of severe heat illness after loss of thermoregulation in thermal stress disease.

A current academic controversy over selective brain cooling was started in 1979 by Cabanac and Caputa[xxxviii] to build on the argument the human brain has *selective brain cooling* mechanisms to lower brain (and SONAH) temperature during severe hyperthermia, helping the body maintain thermoregulatory control. Many studies have followed in support of this thesis[xxxix,xl,xli,xlii,xliii,xliv,xlv,xlvi,xlvii,xlviii,xlix,l,li,lii]. However, many other physiologists[38,liii,liv] equally oppose.

This argument has fostered considerable controversy between North American[lv,lvi] and European[lvii,lviii] physiologists over the last 40 years, resulting in a flurry of point/counterpoint discussions[lix,lx,lxi,lxii] in 2011. By whatever mechanism, cooling of the face, scalp, and neck with *transpulmonary cooling*[9], results in rapid thermal relief. This cooling blunts the rise of deep body temperature and lowering of the critical brain/SONAH temperature upon which control of thermoregulation depends.

Even Cabanac & Caputa[31] doubted pulmonary cooling of the bloodstream in 1979,"The local effect of face cooling on the human brain temperature during exogenous or endogenous heat stress is comparable to the local effect on brain temperature of evaporation from the upper respiratory tract of panting mammals in which a *rete* structure allows local cooling of the arterial blood supply to the brain… Because he does not pant and lacks a *rete* structure, it was assumed, hitherto, that no such brain cooling (through the lung) could occur in humans." Others researchers[lxiii,lxiv] further denied the possibility of cooling core body temperature through the lung.

But subsequent research and clinical findings now forward a different concept.

Thermal Hyperpnea - Body Cooling Through the Lungs

White and Cabanac[lxv] noted (1996) that exercising hyperthermia subjects, once they reached 70% of an individual's maximal workload, exhibited a divergence of esophageal (T_{es}) and tympanic (T_{tm}) temperatures. This was taken as evidence of *selective brain cooling*. Further studies by these authors[lxvi,lxvii] found this breathing pattern of thermal hyperpnea resulted in a respiratory alkalosis in which the normal chemoreceptors were overridden when a thermally driven increase in pulmonary ventilation is adopted.

"Hyperthermia and an elevated T_{core} give an increase of pulmonary ventilation and greater heat loss from the upper airways despite Pa_{CO_2} decreasing to values at which pulmonary ventilation is normally inhibited. Small increases in upper airway ventilation give a local selective brain cooling in humans."[52]

Further research[lxviii] expanded the theoretical basis for thermal hyperpnea and its resultant effects on selective brain cooling. This research is ongoing in the laboratory. But such research always has its skeptics and detractors[47,48].

But an unexpected confirmation of this occurred in 2014 with Kumar's[lxix] work inducing therapeudic hypothermia in resuscitated pigs after induced cardiac arrest. These intubated (thereby bypassing their *rete mirabilis* mechanism for *selective brain cooling*) animals were given a cooled refrigerant mixture for 90 minutes or

until a brain and body temperature of 32°C. (89.6°F.) was reached. T_{es} of 32°C. was achieved in an average of 56 minutes, the brain in 66 minutes. The pigs were normothermic at the time of arrest and resuscitation, without the hyperthermic drive normally invoking the *selective brain cooling* mechanisms. Thus effective whole body cooling through the lung does occur. Research on this mechanism continues[lxx,lxxi,].

Thus, Polar Breeze's hood channeling ambient air, cooled by 30°F, onto the head, neck, and upper torso may also be effecting core body cooling via the lung and the thermal hyperpnea mechanism. This core cooling via the lung is now being recommended as a means of therapeutic hypothermia in brain-injured patients and those status-post cardiac arrest.

Certainly the negative cognitive effects of hyperthermia are well known and documented[48,49,lxxii,lxxiii] as well as hyperthermia's adverse impact on decision-making. Given the danger of firefighting evolutions, this alone is *raison detre* for the most effective Thermal Rehabilitation possible for our firefighting elite.

Thermal Hyperpnea and the *Transpulmonary Cooling* Concept

Transpulmonary Cooling is a recent novel concept first identified by Kumar and Goldberg[lxxiv] in 2014 and refers to bloodstream (and thereby whole body) cooling through the lung. Logically, this makes sense in light of the anatomy of the lung. The human lung has approximately 59x the surface area of the skin[lxxv] without skin's insulating fat. Cold air into the lung is separated from the capillary blood flow only by the one-cell wall thickness of the alveolar air-sacs in the lung. The implication is cooling through the lung has far greater ability to achieve bloodstream and thus core body cooling faster than evaporative surface cooling through the skin and its insulating fat. This is a concept well known to mammalian physiologists as panting. Until recently, panting was a concept believed a constant in all mammals *except* humans.

Opposition to *transpulmonary cooling* has been assumed to be voiced by McFadden's[lxxvi,lxxvii] research of 1985. Looking at mildly exercising and normothermic hyperventilating volunteers, McFadden concluded cooling through the lung is minimally effective. In this population, he may be correct. Therefore, most thermal physiologists have taken this as proof transpulmonary cooling is impossible and assigned human panting behavior post-exercise to simple exercise-induced hypoxia.

But a closer inspection of McFadden's work[74] reveals a much ignored paragraph on p.10,¶2: "…If we had imposed a greater absolute work load, or duration, and/or had interfered with evaporative losses from the skin during exercise, it is probable that, as core temperature rose, the lungs would have been called on to eliminate the excess heat."

Here McFadden calls upon Mitchell's[lxxviii] work as reference of the existence of cooling through the lung in extreme body heat. Thus even McFadden knew the severely hyperthermic could utilize *transpulmonary cooling* when rising body heat becomes critical. Many thermal physiologists continue to believe cooling through the lung is impossible. But these same physiologists have no problems with this same transpulmonary concept under a different name, *thermal hyperpnea*.

Described by Mariak[lxxix] et. al. in 1985, Cabanac and White in 1996[64], and summarized by White[lxxx] in 2006, *thermal hyperpnea* is an increase in human pulmonary ventilation by 35% with a rise in venous blood pH from ~7.38 to 7.46 from an increase in rectal temperature of just 1°C. These are coupled with a decrease in plasma bicarbonate from ~25.5 to 22.3mEq/l and pCO2 from ~44 to 33 torr in evidence of hyperventilation, despite chemoreceptor input opposing these changes. At a T_{tm} of 39.3°C, blood pH =~7.6 and pCO2 to ~20 torr in resting volunteers who had no change in plasma norepinephrine or potassium. Collectively, this evidence points to *a direct increase in pulmonary ventilation as a result of core body temperature elevation, with an over-riding of normal chemoregulation.* Thus *transpulmonary cooling* does occur in humans subjected to extreme hyperthermia, whether at rest or exercise. Thus McFadden was correct as to no loss of temperature through the lung in the normothermic, but incorrect for *transpulmonary cooling* in hyperthermic humans.

Thus *transpulmonary cooling* in humans is not only real, it is an automatic response to an increases in core body temperature. Transpulmonary cooling directly cools the blood flow through the lung, resulting in cooled cardiac output, with the brain being the first organ to received this cooled blood supply. Thus not only can *selective brain cooling* be explained by this transpulmonary phenomena, the lower T_{tm} is also explained. Thus the bias attributed to McFadden's work must be debunked if we are to provide better therapy for the hyperthermic firefighter, athlete, and industrial worker.

Studying therapeutic hypothermia induction for brain preservation after cardiac arrest, Kumar and Goldberg[1] demonstrated efficient rectal temperature lowering by injection of low vapor-pressure refrigerant into the pulmonary circuit of anesthetized pigs. Here they achieved a lowering of core body temperature four times faster than skin surface cooling. Animal modeling has further indicated the optimum gas temperature to induce transpulmonary hypothermia is 4°C[lxxxi]. Thus the concept of *transpulmonary cooling* is well known to portions of thermal physiology. To this date, translation of this data to functional clinical use in humans has yet to occur.

But known clinical correlations for *transpulmonary cooling* do exist. General anesthesia is noted to rapidly lower esophageal temperature when high air/oxygen gas flows (>5 l/min.) are used during general anesthesia. Gases released from high-pressure tanks rapidly absorb heat and vaporize at a temperature in the 16°C range.

Upon breathing this cool gas in volume, much heat is absorbed from the lung and thereby the bloodstream, resulting in a cooling of the esophageal temperature ~1°C every 3-4 minutes.

While this is bad for patients, this rapid cooling through the lung can be very effective for the severely hyperthermic who employ every cooling mechanism available in an attempt to forbear thermal stress injury. This can be the saving grace for firefighters after fire suppression, athletes, and industrial workers subject to severe thermal stress.

A failed IRB-approved study[lxxxii] at the Florida State Fire College Ocala in September 2018 noted a 0.55°C drop in T_{tm} every 3-minutes when firefighter instructors exiting fire suppression training were thermally rehabbed in cooling hoods supplied with a 15.6-18.3°C airstream within a hood. This cooled hood supplied surface cooling to the face, neck, scalp, as well as providing cooled air for inhalation. The instructor cooling was twice as fast as the younger and lighter recruits who only received water + fan skin surface cooling in 32.2-34.4°C ambient environment. Intestinal radiothermotelemetry capsules failed to reliably register and thus gut temperatures could not be established. Data remains unpublished.

Thus a comparison of *transpulmonary cooling* to standard thermal rehabilitation in firefighters, is one of the aims of this proposed study. But due to the design of the device used for *transpulmonary cooling*, another mechanism may also be at play to achieve rapid lowering of temperature.

Head and Neck Cooling

The thermal rehabilitation efficacy of head cooling in the hyperthermic has long been known. Williams et. al.[lxxxiii] noted "It is evident core temperature, and consequently body heat storage, is influenced strongly by cooling the head and neck area. This resulted in a 50% decrease over controls". They further noted " ..cooling hood in hot humid environment markedly reduces physiologic strain". Kissen's[lxxxiv] earlier work (1971) had noted "Ventilating the head, with 8% body surface area, more effectively reduces physiologic strain than ventilating 60% body surface area of the trunk under equivalent conditions". Thus we have known core temperature, as a storage reservoir of body heat[16], can be remarkably limited by simple fanning of the head and neck. The resultant evaporation of sweat results in remarkable cooling and thermal comfort, but also remarkably limits the heat storage normally accruing during repetitive burn evolutions in training or on-scene fire suppression.

While *transpulmonary cooling* appears to be the most likely method to effect *selective brain cooling*, other mechanisms may also play a role. The original work of Hertzman[lxxxv] and Fox[lxxxvi] noting the lack of vasoconstrictor motor tone in the face and scalp, and the work of Shvaartz[lxxxvii], Nunneley[lxxxviii], Brown[lxxxix]and McCaffrey[xc]

on the beneficial effects of head cooling both deep body temperature as well as thermal comfort in humans, all pointed to the highly beneficial effects of head cooling on rectal temperature and thermal comfort in human subjects. Caputa[xci] et. al. noted the normal outward flow of venous blood, from the brain to the face, can be reversed under the influence of face fanning in the hyperthermic, to flow from the *angularis oculi* veins to the *cavernous sinus.* Indeed, facial cooling and cold upper respiratory airflow has been successfully used to therapeutically cool brain-injured patients by induced *selective brain cooling*[xcii] and thereby improve their clinical outcomes.

Thus a combination of *transpulmonary cooling* and head/neck surface cooling appears to have the capacity to quickly cool firefighters and thereby effectively limit their thermal stress. As well, these methods may effectively limit accruing body heat storage in the gut. But the study of head and neck cooling to effectively lower core body temperature brings up another, and controversial, concept: *selective brain cooling*.

What is not controversial is that cooling of the head, neck, and upper trunk results in rapid removal of cerebral heat in hyperthermia with a relief of hyperthermic symptoms and return of comfort despite a continued hyperthermic environment[23]. Laboratory researchers have proven that rapid lowering of the cerebral temperature in critically hyperthermic laboratory animals ablates symptoms of heat stroke and prevents multi-organ system failure. They postulate similar advantages for humans.

Polar Breeze is a microenvironmental air-chiller that lowers ambient air-temperature by 30°F. in a singe pass through the machine and delivers it at 10 SCFM to a hood covering the head, neck, and upper torso. Firefighters in Thermal Rehabilitation during Burn House training have anecdotally reported rapid return of comfort with lowering of aural (T_{tm}) temperatures on the average of 1°F.(0.56°C.) every 2 minutes, an observation confirmed (independent of the manufacturers) in training centers across the nation.

The Polar Breeze hood covers not only the head, neck, and upper trunk, it also provides a cooled airstream to be taken into the lungs. As a 6'0" male at 170 lbs. has a body-surface area (BSA) of just under 2.0m^2, the most recent estimation of his lung-surface area is 118m^2 (+/- 11m^2)[xciii], despite more frequently quoted older work that placed the LSA at 50 m^2-75m^2 [xciv,xcv]. Unlike skin, with its varying degrees of insulating fat dependent on BMI, the lung has no insulation separating cool air from the bloodstream. In fact, that separation of cold air from the blood vessels is one cell layer thick - the wall of the pulmonary alveolus. Thus, it seems logical direct cooling of the bloodstream by cooled air is possible. After all, furry mammals without sweat glands achieve thermoregulation by panting. Is there an analogous function in humans?

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