

Lorentz gas as a typical distribution of velocities for more than a moment. We may take the initial momentary distribution of velocities as conforming to Lorentz, but a moment later, owing to the finite distribution of the condensing wall, the ions there will have a radial distribution of velocity with $\mathbf{v} \cdot \nabla \mathbf{v}$ not equal to zero. With $\mathbf{v} \cdot \nabla \mathbf{v}$ not equal to zero at the points we cannot make the gas a Lorentzian throughout a region by any uniform transformation to uniformly moving axes. The Lorentz gas cannot be regarded as an example for any finite region, however well it may serve as an example for an infinite region.

The immediate appearance of $\mathbf{v} \cdot \nabla \mathbf{v}$ in the gas requires us to use our η' which is much larger than the η_L of the Lorentz gas.

¹ Spitzer, L., Jr., *Physics of Fully Ionized Gases* (New York: Interscience Publishers, Inc., 1956).

² Lehnert, B., *Nuovo Cimento Supplemento*, vol. 13, ser. 10, 59–107 (1959).

THE DIMINUTION OF THERMOREGULATORY SWEATING DURING COLD-RECEPTION AT THE SKIN*

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It was reported previously in these PROCEEDINGS¹ that thermoregulatory sweating of man is determined by *central* temperature, that it occurs in the *absence* of thermo-receptor impulses from the *skin* and that a central sensory receptor organ must exist which acts as *the human thermostat*.

The experiments from which these conclusions followed^{1, 2} were carried out on lean subjects with widely, and sometimes oppositely, varied skin and cranial internal temperatures. However, with these lean subjects, the experimental conditions did not permit a study of one particular paradoxical combination: cranial internal temperatures elevated beyond the "setpoint" of the individual thermostat with simultaneous skin temperatures below 33°C, in the *cold* range. At this skin temperature cutaneous cold-receptors begin to drive, with mounting impulse frequencies, the response of man to cold by increased metabolic heat production, unless a high internal cranial temperature prevents this response from occurring.

A seeming conflict with reports from other laboratories suggested an extension of our observations on sweating into this range. With a subject of much higher weight-to-height ratio than those of subjects used in our previous studies,^{1, 2} the paradoxical conditions could be produced with strenuous exercise in a cool environment.

Methods.—The methods were the same as those used in the previous study,¹ namely, gradient layer calorimetry for the measurement of sweating, tympanic membrane thermometry for measurements of cranial internal temperature, and properly located thermocouples for the measurement of average skin temperature. One subject, D. D. (nude, age 26, weight 88.6 kg, height 176 cm) was used for all the measurements presented. The tests on him were carried out between April 4

and June 5, 1961. In Figures 1 and 2, each plotted point represents a steady state of rest (○) or exercise (mechanical equivalents 6 cal/sec △ or 11 cal/sec △) with a steady cranial internal temperature, skin temperature, and rate of sweating established and recorded in the gradient layer calorimeter.

Because of the additional *internal* (metabolic) *heating* and the additional *external cooling* by increased sweating rates, skin temperatures, at any given level of internal cranial temperature, were lower during exercise than during rest. This difference amounted, on the average, to 1.5°C. It was thus ample to permit reliable conclusions on the presence, magnitude, or absence of effects exerted by skin temperature on rates of sweating.

Results.—Results have been divided into observations made in the *warm* range (skin temperatures 33 to 38°C, Fig. 1) and observations made in the *cold* range (skin temperatures 33 to 29°C, Fig. 2).

A basic difference between these two conditions is immediately apparent. In the cold range, rates of sweating are progressively diminished with decreasing levels of cutaneous temperature at any given cranial internal measurement. This diminution is of the order of 4 cal/sec in evaporating heat loss for every lowering of steady skin temperature by 0.1°C.

In the warm-range (Fig. 1), such an effect of skin temperature is conspicuously absent. If the effect observed in the cold range (Fig. 2) had extended into the warm range with similar intensity, this would have caused the observations of sweating during exercise (triangles) to be an estimated 60 cal/sec lower than the corresponding observations during rest (circles)—due to the average difference of 1.5°C between working and resting observations at any given level of cranial internal temperature. Instead, no indication of a downward or rightward shift of the triangles from the circles is seen in Figure 1. The distribution of triangles and circles around the “best line” is random and within experimental errors. It does not reveal any major changes in the “setting” of the human thermostat during a period of two months. The quantitative characteristics of Figure 1 prove that the influence of skin temperature on sweating was zero throughout the warm range, 33° to 38°C.

Discussion and Conclusions.—(1) The data presented in Figure 1 confirm our previous observation (see Fig. 4 in ref. 1 and Fig. 1 in ref. 2) that the response of sweating is elicited *without* participation of impulses from skin thermoreceptors. Abundant peripheral *warm* impulses of widely different intensity in the resting as compared with the working subject must have been present in the experiments of Figure 1 where skin temperatures from 33 to 38°C were obtained using environmental temperatures from 27.5°C to 50°C (122°F). Nevertheless, *no* influence of skin temperatures on sweating was detectable throughout this warm-range in which the sweating mechanism physiologically operates. The human thermostat was acting as a terminal sensory organ with first neurons.

(2) With Figure 2, a contribution, previously observed by other authors, of skin temperature as a factor in human “physical” heat regulation has been confirmed. It has been identified as simultaneous with *cold*—not *warm*—reception. It has been described in quantitative terms of evaporative heat loss response (reduction by 4 cal/sec) to the stimulus of temperature (cooling by 0.1°C). Consistency is thereby ascertained with numerous published observations, old or recent. (Some

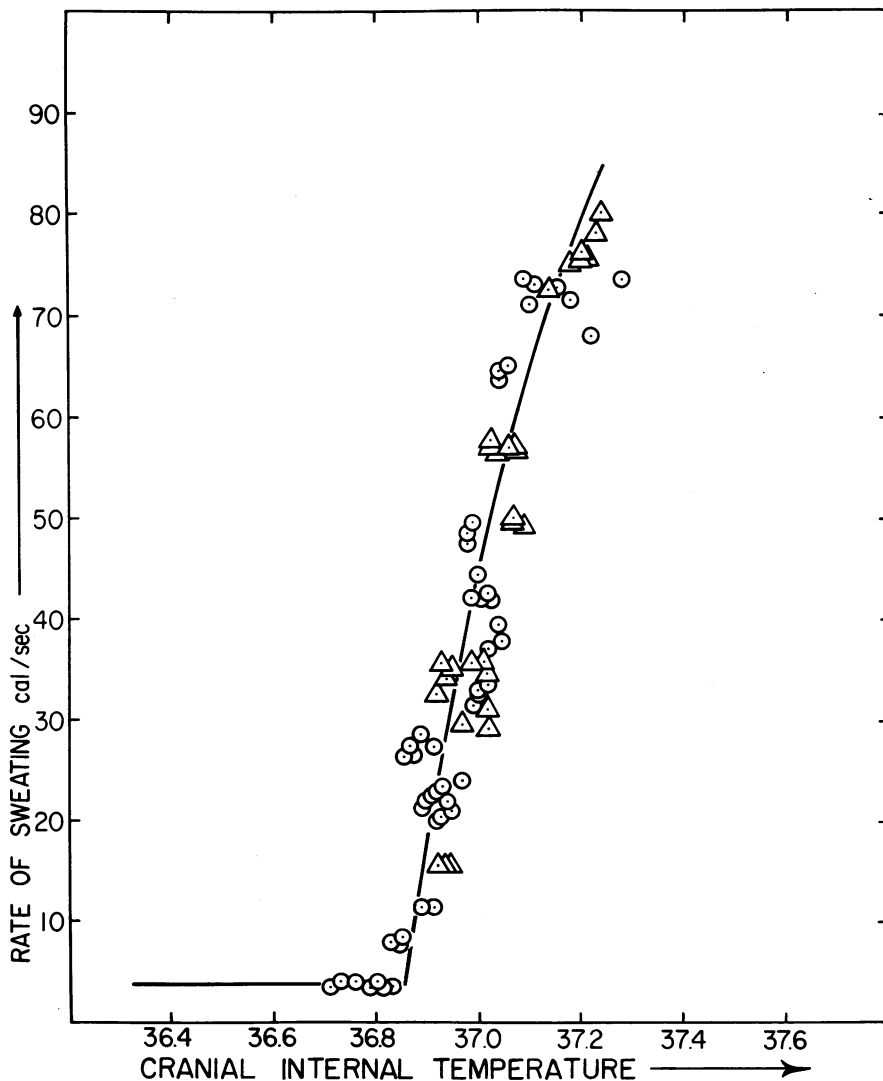


FIG. 1.—Intensity of thermoregulatory sweating during *warm* reception at the skin. Sweating rates were plotted against internal cranial temperatures in steady states or rest (O) or exercise (Δ 6 cal/sec and Δ , 11 cal/sec). Sweating begins at a sharply defined *internal* cranial temperature, the “setpoint” of the human thermostat, a result reproduced over a period of two months. The intensity of thermoregulatory sweating is inseparably related to the level of *internal* cranial temperature. There was no visible effect of the drastic differences in skin temperature (average, 2°C) between the resting and the working observations. This difference (average, 2°C) would have lowered the triangles by an estimated 80 cal/sec in comparison with the cycles measured at the same internal cranial temperature, if the inhibiting effect of cold observed in Figure 2 extended with similar intensity into the range of *warm* reception. (Experiments were carried out between April 4th and June 5th with one subject, D. D., nude, age 26, weight 88.6 kg, height 176 cm.)

selected examples of references are Kuno,³ Bazett,⁴ Herrington,⁵ Robinson,⁶ Randall and Hertzman,⁷ Randall,⁸ Hertig and Belding,⁹ and Brebner and Kerslake,¹⁰ in which a correlation of skin temperature with sweating had been seen, while the conditions under which it occurred had not been finally delineated. A seeming

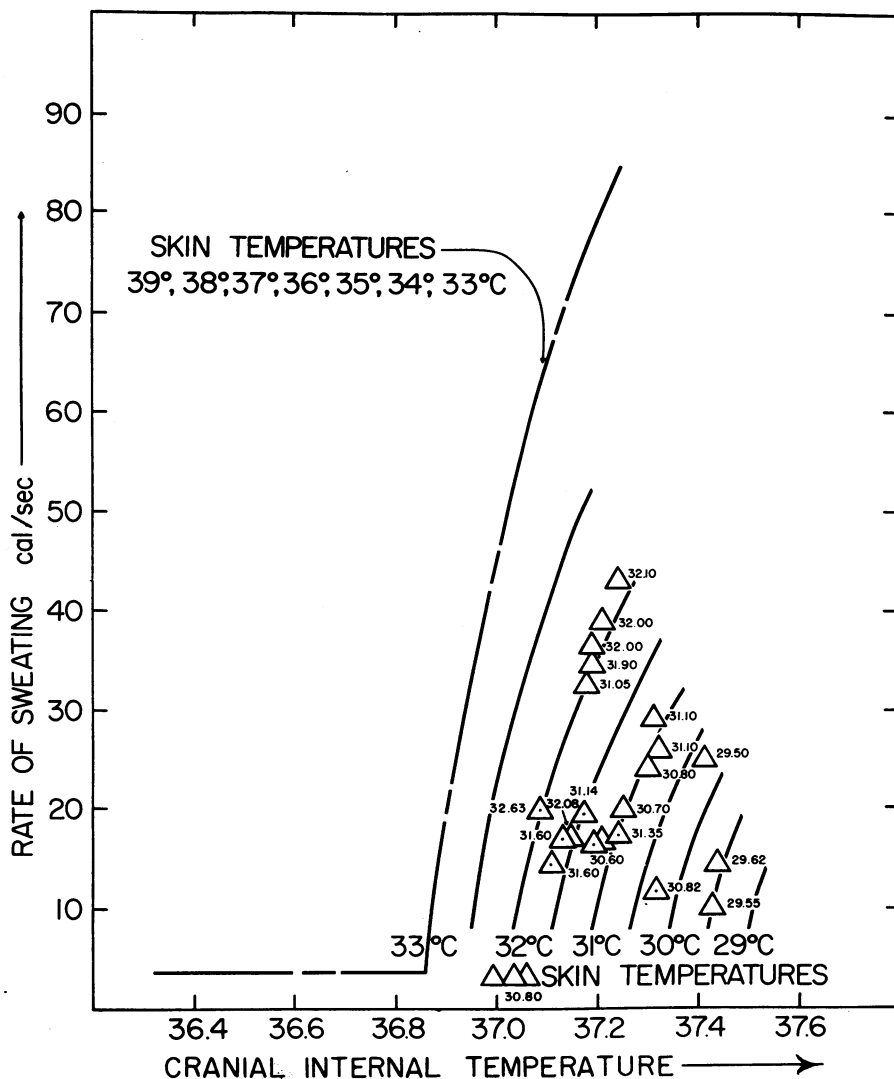


FIG. 2.—Intensity of thermoregulatory sweating during cold reception at the skin. Sweating rates were plotted against internal cranial temperatures. Measurements obtained at similar *skin* temperatures were connected with “best lines.” At any given cranial internal temperature, sweating rates are seen to be diminished by approximately 40 cal/sec for every degree C decrease in level of skin temperature. The line for 33° skin temperature was carried over from Figure 1. Figure 2 contains no resting observations as these paradoxical conditions cannot be produced in steady states at rest. Work rates were mechanically equivalent to 6 cal/sec (Δ) or 11 cal/sec (Δ), respectively. Increase in work rate enlarges the range of observations to the right (low skin, with high internal temperature). (Experiments were carried out between April 4th and June 5th 1961 with one subject, D. D., nude, age 26, weight 88.6 kg, height 176 cm.)

contradiction has been resolved by the results shown in Figures 1 and 2. The reliable and important observations of other investigators³⁻¹⁰—though not their conclusions—have been confirmed independently with a different method.

Additional conclusive evidence is, however, desired that the phenomenon described is neural and not caused by vasoconstriction, segmental reflexes, axon re-

flexes, or direct effects of cold on sweat glands. Those authors who made similar observations seem to be in agreement that they were due to a centrally relayed reflex.

Figures 1 and 2 are further proof that in spite of the continuity of the temperature scale of the *physicist*, the *physiologist* may discriminate objectively between a "warm" and a "cold" range. The discontinuity is near 33°C, where not only the cold-receptors of the skin begin to elicit the response to cold by increased metabolic heat production but where also the response to internal warmth by sweating begins to be inhibited from the periphery. A third phenomenon is the beginning of conscious cold-discomfort at this temperature in unpublished observations from this laboratory and another, the beginning of willful reactions to sensations of cold seen in experiments of Iberall.¹¹

(3) Some practical conclusions seem to be in order. With Figure 2, the resolution of the mechanisms of human temperature control has been extended into its least accessible area, where *central warm*-reception is colliding with messages of *cold* from the *skin*. Though paradoxical, such situations are not quite uncommon. The running athlete in cool weather, the ascending mountaineer, the skier on sunny slopes, all lightly clad, and perhaps the swimmer in tropical waters, experience skin temperatures low enough for cold-receptors to be active, while the athletic effort elevates internal cranial temperature above the setpoint. Cold-reception at the skin may then prevent or delay an immediate adjustment of elevated cranial internal temperatures through sweating. However, as a favorable effect, unnecessary, excessive, or prolonged evaporative cooling is prevented after termination of the muscular effort. The opposite condition, *central* temperature *below* the setpoint, combined with a warm skin, has been studied and clarified with experiments in water.² It is a transient state of tranquility and subjective comfort in which no thermoregulatory impulses seem to arise at either central or peripheral thermoreceptors.

(4) A unified and quantitative explanation free of contradictions can be given for the fact of human thermal homeostasis, for the ways in which it is achieved, and for the wealth of observations previously made, though not the conclusions previously drawn in this field. The classical experiments would have permitted by way of theory, but did not force by way of experimental argument, the simple conclusions listed below. These followed inevitably from more complete and more accurate measurements using the modern methods.^{1, 2}

The peripheral sensory component of the autonomic thermoregulatory system responds to *cold*, not warmth. Its functions are (a) to elicit (not to gradate and regulate) the metabolic response to cold and (b) to diminish or eliminate sweating when cold acts on the skin while central temperature is elevated. Both of these functions are not *per se* thermostatic. *Skin* temperature only, not *internal* temperature, can be regulated by *skin* thermoreception. The seat of homeostasis is the *central* thermoreceptive system. It elicits sweating and vasodilatation *without* receiving peripheral warm-impulses. It either depresses or releases the metabolic response to cold-receptor impulses from the skin precisely to such an extent as is required to maintain or restore homeostasis.² The temperature of the "thermostat" determines ultimately the rates of thermoregulatory heat production or heat loss in man.

(5) An addition is required to the component parts of the nervous system which

were postulated on calorimetric evidence,² namely, a central pathway connecting cold-receptors of the skin with effector neurons for sweating. The other postulated centers and pathways have been discussed.² It was found that the recent results of human calorimetry on one side and the discoveries of classical experimental surgery with animals on the other are consistent without exception.

Summary.—Certain correlations between temperature of the skin and intensity of thermoregulatory sweating, qualitatively observed before by others, have been clarified in quantitative terms. Evaporative (sweating) heat loss may be reduced by 4 cal/sec with every 0.1°C reduction in the level of skin temperature. This phenomenon occurs, however, *only* at skin temperatures *below* 33°C, where cold-receptors elicit the response to cold by increased metabolic heat production, unless a high internal cranial temperature prevents its occurrence. On the other hand, throughout the range of *warm*-reception (33 to 38°C skin temperature), where the sweating mechanism usually operates, *no* influence upon evaporative heat loss was detectable with drastic differences of skin temperature. It is thereby confirmed that sweating originates from a terminal sensory organ in a central, not a peripheral, position.

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³ Kuno, Y., *The Physiology of Human Perspiration* (London: J. and A. Churchill, 1934).

⁴ Bazett, H. C., in *Physiology of Heat Regulation and the Science of Clothing*, ed. L. H. Newburgh (Philadelphia: W. B. Saunders, 1949).

⁵ Herrington, L. P., in *Physiology of Heat Regulation and the Science of Clothing*, ed. L. H. Newburgh (Philadelphia: W. B. Saunders, 1949).

⁶ Robinson, S., in *Physiology of Heat Regulation and the Science of Clothing*, ed. L. H. Newburgh (Philadelphia: W. B. Saunders, 1949).

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⁸ Randall, W. C., "The physiology of sweating" (special review), *Am. J. Phys. Med.*, **32**, 229 (1953).

⁹ Hertig, B. A., and H. S. Belding, "Rates of adjustment of sweating and body temperatures following exposure to heat," *Fed. Proc.*, **20**, 211 (1961).

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