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INSENSIBLE SWEATING; AN INTERMEDIATE STAGE BETWEEN INSENSIBLE PERSPIRATION AND ACTIVE SWEATING

by

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ABSTRACT

Between insensible perspiration and sensible sweating a stage has been demonstrated which is characteristically distinct from either, being glandular in origin and therefore unlike the former, but non-cyclic in character and therefore unlike the latter. It appears to serve as an intermediate stage between vasodilation and active sweating in the heat in much the same way that an increase in muscle tone is intermediate between vasoconstriction and shivering. It is proposed that this should be termed Insensible Sweating.

RESUMÉ

Entre le stade de transpiration insensible et celui de sueur sensible, on a démontré qu'il existe un troisième stade qui, de facon caractéristique, ne ressemble ni au premier, ni au second.

Ce stade n'est pas comme le premier, étant donné qu'il est d'origine glandulaire, et n'est ni comme le second, puisqu'il est de nature non-cyclique. Il semblerait servir de stade intermédiaire entre le vaso-dilatation et la sueur active dû à la chaleur, de même façon que l'augmentation de tension musculaire sert d'intermédiaire entre la vaso-constriction et le frissonnement. Pour décrire ce troisième stade, on propose l'expression "sueur insensible".

TERMINOLOGY

This paper deals with the level of water loss above that of insensible perspiration but below that of manifest sweating. Such an intermediate level is clearly distinguishable from the former which is steady and the latter which is cyclic. Since it is customary to refer to transepidermal water loss through the skin in the absence of sweat gland activity as perspiration, and glandular activity as sweating, and since this intermediate stage appears to be glandular in origin but, like insensible perspiration, invisible to the unaided eye, it seems appropriate to coin the term "Insensible Sweating" to describe it.

INTRODUCTION

In 1946 Adolph (1) referred to a small increase in evaporation within five minutes of exposure to heat, and attributed this to local heating of the sweat glands. Kerslake (2), in view of the findings of Kuno and others that skin temperatures of 40°F or above are required to permit the initiation of sweat gland activity, suggested that Adolph may have failed to distinguished between insensible perspiration and sensible sweating, and that this small increase may represent only an increase in the rate of diffusion of water through the skin due to a rise in skin temperature. Experiments conducted recently suggested that Adolph may have been correct.

For a number of years we have observed that water loss from the skin is sometimes cyclic and sometimes smooth, depending upon the level of heat stress (3). These observations were made while working on heat stress problems of military interest, using the DREO-developed Sudorimeter (described elsewhere (4,5) to obtain sweating data. This instrument operates on the following principle. A capsule, with a small inlet and outlet port for flushing with dry air, is placed against the skin. The capsule contains a humidity sensing element the resistance of which in an atmosphere of 45% RH is exactly balanced by a similar resistance in the associated circuit. Any increase or decrease in element resistance upsets the balance of the circuit and is used to open or close the dry air control valve. As soon as sweating breaks out and the capsule atmosphere exceeds an RH of 45%, the lowered resistance causes a servo-mechanism to open the air control valve and admit dry air to the capsule to restore its null point RH of 45%. The level of sweating is thus monitored by a feedback system in which the amount of dry air passing through the capsule reflects faithfully the level of sweating at the time. The system is highly responsive and is capable of detecting and making appropriate adjustments for a change in sweating rate which, viewed as a total-body weight loss, is equivalent to as little as 160 mg of sweat in 1 min.

The instrument responds to this change in about 2 seconds. Measurements show that the element is detecting and responding to an increase or decrease of somewhere in the neighbourhood of .08 mg. of water evaporated from the skin covered by the housing. This was determined by bubbling the exhaust air from the capsule though sulphuric acid and measuring its increase in weight.

The instrument, for the experiments to be described, was intentionally adjusted to measure only sweat gland activity. Insensible water loss in the absence of glandular activity is discounted by allowing a very small flow of dry air (about 1 cc/min.) to pass continuously through the capsule in order to flush out the water vapour emanating from the area of exposed skin under the capsule. It was achieved by preventing total closure of the air control valve. This ensures that the sensing element of the Sudorimeter will not be triggered by a gradual accumulation of water vapour in the capsule prior to the onset of active sweating.

The character of glandular activity is cyclic provided that the heat stress is adequate. In the absence of cyclic sweating there is normally no instrumental response. However, we now have some evidence of activity which we believe to be glandular but intermediate between perspiration and frank sweating. This is observed in the early stages of, or subsequent to, a period of heat stress and takes the form of non-cyclic glandular activity which appears quite abruptly, occasionally changes level but without losing its characteristic smoothness, and then with equal suddeness assumes the form of active cyclic sweating as heat stress increases.

The smooth character and lack of cycling in the recording of insensible sweating might suggest that the Sudorimter is not capable of following very small fluctuations at such low levels of sweat gland activity. It would then be an instrument artifact and not, as proposed, a different kind of glandular activity.

There are several reasons for rejecting this alternative explanation. In the first place the Sudorimeter is perfectly able to monitor extremely small fluctuations as already pointedout in the Introduction. Moreover, it readily measures the very much lower levels of activity observed in the palmar region, especially on the whorls of the fingers where the sweat is quite invisible to the unaided eye. When two independent Sudorimeters are used the parallelisms between the records are so striking that the possibility of instrumental "hunting" is entirely excluded on almost all occasions. Now and then, the cycles are so regular on both records that one might suspect hunting is occurring at a similar frequency and magnitude in both Sudorimeters: but such occasions are very rare. Cycles are almost always uneven and random in character, quite unlike the phenomenon of hunting due to instrumental factors.

In the second place, the record from both Sudorimeters is concordant in pattern even when the two capsules are placed on different sites, such as the dorsum and the upper chest for example. In such cases parallelism is still observed, but the curves run at different levels due to differences in sweating rates at the two sites.

In Fig. 7. of the present series, at each low section of the sweating curve, the two Sudorimeters which were used throughout these experiments, indicated the same basic pattern. Similarly, in Fig. 10., at the lowest point of the curve (15 to 17 mins, approximately) the same pattern of sweating was observed on both Sudorimeter charts. This was cyclic sweating signifying glandular activity in phase, yet at as low a level or even lower than some of the non-phasic or insensible sweating records to be observed on other charts (Fig. 9., after 30 mins, for example).

It is quite possible that the smoothness of the insensible sweating curves would have disappeared wholly or in part by using a smaller capsule covering a much more limited area of the skin surface and therefore measuring the activity of far fewer glands. There is little doubt that the individual glands are all cyclic in action, but if they are random in frequency and magnitude at this low level of activity, their "individuality" is masked if enough of them are monitored together. What we have termed insensible sweating has, therefore, none of the cyclic character of manifest sweat gland activity measured over the general body surface where there is evidently a central drive imposing on all glands the same cyclic stimulus. In a very restricted area, a few glands may be active in a concerted manner during insensible sweating, but elsewhere the activity is not timed at the same frequency. The firing of the nervous impulses is apparently somewhat random, rather as it is in the intermediate phase between muscle relaxation in the warm and shivering in the cold, the intermediate phase being an increase in muscle tone but not phasic contraction and relaxation as occurs when shivering begins.

There is, therefore, evidence of a clearly marked distinction between the two kinds of sweating, insensible and manifest, as shown consistently in the present experiments. Above a certain level of heat stress, individual gland contractions are regimented to act in concert over the whole body surface in a way that is quite distinct from the low level response observed in insensible sweating.

EXPERIMENTAL PROCEDURE

Experiments were conducted with subjects in a hot chamber at rest (seated) or walking on a treadmill at speeds up to 3.5 mph, or exercising on a bicycle ergometer. None was performed within a period of less than one and one half hours after eating. Preparation of the subject, passively standing or sitting, took no more than five minutes, and evidence showed that it did not alter skin temperature for more than a minute or two.

The hot chamber, just large enough to contain a treadmill, has a controlled temperature range from 70° to 125°F and has continuous air movement. By opening all ports at once, its temperature can be brought from 125° to 70°F within two minutes. The chamber air temperature can be cycled

upwards or downwards slowly or rapidly to approximate either a sine wave pattern or a step function as indicated in some of the following diagrams. These changes in temperature do not in any way disturb the operation of the Sudorimeter.

Occasionally involuntary shifting of the subject produced an anomalous spike in the curve. These spikes were rare, because the men were careful not to move unnecessarily. The spikes are easy to identify.

Three healthy male subjects were used. Before each experiment the men, dressed only in shorts and socks, were seated in an open-web garden chair for one hour in a room held at temperature of 70° - 75°F with slight air movement. They were balanced on an automated scale described elsewhere (6), and fitted with skin thermocouples at 3 sites (medial thigh, chest above the nipple, and forearm) for continuous temperature measurement. Rectal temperatures were also recorded continuously at a depth of 10 cm. Two Sudorimeter capsules were mounted against the skin on the dorsum at the level of the 3rd and 4th lumbar vertebrae and about 10 cm to each side of the midline. Total weight losses were determined by rebalancing after each experiment.

RESULTS

The nature of the evidence for an intermediate stage between insensible perspiration (which the Sudorimeter ignores) and active cyclic sweating, is shown in Fig. 1. This stage is also observed in the cooling stage as indicated in Fig. 2. It may continue virtually unchanged, for 50 minutes or more after manifest phasic sweating has ceased. The oscillations over the final 2 minutes were deliberately induced by reducing the feedback of the instrument until it was unstable. The object was to ensure that the system had not become insensitive for some reason. All responses were found to be normal.

Under cool conditions an inactive subject will normally maintain body temperature at an appropriate level without any measurable increase in insensible perspiration. Between 60° and 65°F the hourly loss is found to be about 35 grams. When the ambient temperature is raised to between 70° and 75°F, the hourly loss rises to about 50 grams as shown in Fig. 3, for one subject. On the basis of this man's weight and height, his transepidermal water loss ranged from .068 to .136 mg/cm^2/min. At this rate of loss the capsule of the Sudorimeter, which covers 14 cm^2 of skin surface, will respond and record within 10 to 6 seconds after being positioned against the skin. At higher rates of water loss, when active sweating is occurring, the delay in response is less than 1 second. The suddenness of the onset of insensible sweating is therefore a genuine phenomenon, as is its reversion at the 37 min mark in Fig. 2. to a prior level when room temperature declined again.

In the experiment of Fig. 4. the enviromental temperature fluctuated but held within a range low enough to prevent sensible sweating. It is clear that insensible sweating was responding in some measure to the corresponding fluctuations in skin temperature, though with little evidence of positive correlation.

In Fig. 5 are shown the results of an experiment conducted to see whether the pattern of insensible sweating could be disturbed artificially while temperature conditions remained unchanged. After about 7 minutes in an environment warm enough to induce frank sweating, the temperature was dropped rapidly, causing sweating to decline until only insensible sweating remained. Environmental conditions were then held constant for approximately 20 minutes, at which point the gain and feedback of the Sudorimeter were adjusted to cause hunting at various frequencies. It is evident that the mean level of this was approximately that of the stable condition which preceded. At (a) the capsule was lifted away from the skin, without otherwise disturbing things, for about 15 or 20 seconds. The record accordingly showed a null reading. When the capsule was repositioned the previous insensible sweating was picked up once more though at a slightly higher level since no air had been passing through the capsule in the interval to carry away the residual accumulated moisture on the skin. At (b) the capsule was again removed for five minutes and the Sudorimeter showed a no-sweating response. At (c) it was replaced in its former position and the record was recovered as before. Once more at (d) it was removed and then replaced nine minutes later, at (e), were insensible sweating was shown to be occurring at virtually the original level.

The persistence of this insensible sweating seems to be associated with a certain minimum skin temperature. In this and other experiments, so long as skin temperature was not allowed to fall below 86°F, there was evidence of this form of glandular activity, judging by the Sudorimeter response. In an effort to determine at what temperature insensible sweating ceased altogether until only insensible perspiration remained, the enviromental temperature was sometimes allowed to fall slowly until skin temperature was below 86°F. However, in no such experiment was the complete cessation of insensible sweating actually observed. One difficulty is that after about 90 minutes of inactivity subjects became sufficiently restless that the metabolic rate tends to rise once more above resting level, with the consequent renewal of active sweating.

On the other hand, insensible sweating will often decline to zero after exercise. When activity ceases the sweat rate declines much more rapidly and the intermediate stage is often by-passed through it and can occasionally be detected briefly. The temperature control mechanism in these circumstances seems to shift directly from one stage to the next one in a step-wise fashion, in either direction.

In Fig. 6. three such cooling curves are shown after activity. It will be noted that in (c) frank sweating did cut out completely within a few minutes, in (a) and (b) nearly so.

If the heat stress is imposed in a series of short cycles as in the experiments recorded in Fig. 7., the successive segments of the square-wave sweating pattern show that the rate of sweating declines slightly less each time until the heat which is stored in the body (in spite of a steady decline in mean weighted skin temperature) has accumulated sufficiently to obliterate the insensible sweating stage entirely during the final two cycles.

It was evident from other such experiments that insensible sweating was not entirely dependent upon mean weighted skin temperature (MWST), since if radiation losses were facilitated (to cooler walls outside the hot chamber, for example) the subject could, as shown in Fig. 8., sit for 35 minutes or more while skin temperature rose to 93.7°F without any evidence of insensible sweating. Once moved into the hot chamber the long delay seems to have rendered the body more sensitive and within 1½ minutes active sweating of a characteristic phasic nature broke out without the intermediate rise in insensible sweating.

As shown in Figs. 1 and 2, with a rising enviromental temperature in the hot chamber, insensible sweating is initiated at a MWST of about 89° - 90°F. In Fig. 9. the onset is again seen to be at about 90°F but this is followed by active sweating when MWST reaches 95.0°F.

This compares well with the onset of active sweating in Fig. 1. which also occurred with a MWST of 95.4°F. In a series of such experiments the average onset MWST of insensible sweating was found to be 90.3°F, and of sensible sweating 95°F. During activity the MWST of sensible sweating is lowered to 90°F and the insensible sweating stage is bypassed as already noted.

In Fig. 10. is shown the pattern of sweating of an active subject in a rapidly fluctuating environment. The subject walked on a level treadmill, dressed only in shorts and socks, at 3.5 mph for 17 mins. and rested for 5 mins. There is no evidence of insensible sweating during the cooling periods whether they coincided with periods of rest or exercise. The fluctuations in room temperature were achieved by turning on all heaters (radiant) to warm the chamber, and turning them all off and opening all ports to the laboratory environment to cool the chamber. It seems possible that insensible sweating is an adjustment which normally occurs only when the thermal challenge is presented slowly.

DISCUSSION

Water is continually evaporated from the lungs and the skin. In a cool environment this amounts to about 35 grams per hour for an inactive man. This total is accounted for by a combination of transepidermal water loss and by the expiration of nearly saturated air from the lungs. The former accounts for about 20 grams of the total, the latter for the balance of 15 grams. A number of factors influence both the total loss and the

proportion via each avenue.

When room relative humdity is very low (say 15% or less) the inhalation of comparatively dry air which is then exhaled nearly saturated, when combined with other avenues of heat loss such as conduction, convection, and radiation, provides a sufficient level of evaporative cooling to balance metabolic heat production so that there is a decline in transepidermal water loss.

When the room relative humidity is high (say 80% or more) respiratory loss is insufficient and insensible perspiration increases. Between 15% and 80% there appears to be little change in the total loss or the proportions by each route.

In the presence of high relative humidity, evaporative cooling is reduced at the skin surface and some rise in skin temperature occurs thereby increasing the vapour pressure and counteracting the high humidity to some extent. Burton et al. (7) and Pinson (8) have shown that the rise in skin temperature may also result from an increase in peripheral blood flow which serves the dual purpose of increasing the rate of heat transport from the core to the periphery as well as the rate of evaporation. The correlation between blood flow and insensible perspiration according to Robinson (9) is high and, expectedly, so also is the correlation between insensible perspiration and metabolic rate as demonstrated by Levine and Marples (10). Hildebrandt et al. (11) noted that during fever insensible perspiration varied directly with changes in regional skin temperature. Over a period of 24 hours, Burton (12) found that from 23.8 to 25.2% of the metabolic heat was eliminated by the evaporation of insensible perspiration, which therefore represents a remarkably uniform proportion of the heat loss in a non-sweating subject.

Kuno (13) views this transepidermal water loss as a process of diffusion outwards via intercellular spaces, the water being derived from the local blood supply. No glandular activity is involved in a cool environment since the sweat gland ducts appeared to be quite empty.

According to Taylor and Buettner (14) a reduced air pressure increases insensible perspiration by favouring the outward diffusion of body water. Whitehouse, Hancock, and Haldane (15) demonstrated that insensible perspiration rates can be as high in some areas of the body as the water loss of sensible sweating.

Newburgh and Johnston (17) showed that when the blood supply is reduced, as for example during vasoconstriction, insensible perspiration is significantly reduced, due possibly to the fall in skin temperature. Even when the rate of loss is high, it occurs as water vapour and is therefore normally quite insensible. Only in the palmar area does it occasionally become visible. The highest rates occur in desquamating areas, notably the palmar and plantar regions.

Pinson (18) found that if formaldehyde is introduced into the skin by iontophoresis to suppress local sweat gland activity, and the area is then heated artificially, there is an increase in insensible perspiration, which must be non-glandular in origin.

There is no evidence that an increase in blood pressure increases insensible perspiration according to Kuno (19), nor according to Hall and McClure (20) does the ingestion of a large quantity of water.

McQuarrie (21) found that where the number of sweat glands is largest per unit area insensible perspiration tends to be lowest, and vice versa. Scott and Bazett (22) observed that as soon as the skin is completely wetted due to active sweating, insensible perspiration probably ceases altogether.

By definition insensible perspiration is of non-glandular origin and therefore drugs which suppress sweating should have no effect upon trans-epidermal water loss unless they upset the diffusion process. But when a reduction is observed, even in the absence of any visible sweat gland activity, it might be argued that there is gland activity contributing somewhat to the insensible water loss. According to Rothman (23), ectodermal displasiacs whose sweat glands are quite inactive, show a normal rate of insensible perspiration after administration of atropine, whereas atropine administered to normal but apparently non-sweating children causes a measurable rise in skin temperature evidently resulting from the reduction of evaporative heat loss. This is presumptive evidence that some glandular activity was involved at the time of drug administration. In atropine fever, temperatures as high as 109°F have been observed according to Goodman and Gilman (24) whereas dogs which do not sweat show no fever after atropinization.

It is evident therefore that under certain environmental conditions, insensible water loss is not solely due to the diffusion process but is assisted by sweat gland activity of a very low order. List (25) expressed the belief that practically all evaporation below 88°F is an insensible loss of water. Possibly this is true, but even for the inactive man it seems that it is not all insensible perspiration but includes a glandular component hitherto largely overlooked or neglected.

Jurgensen (26) reported the presence of microscopic beads of water appearing momentarily at the ostia of sweat gland ducts, invisible to the unaided eye but clearly seen with a microscope. Their appearance was brief because, due to their minute size, they evaporated almost at once. Glands seemed to fill sporadically and overflow very slightly without regularity. The existence of this low level glandular activity which is properly termed insensible since it is normally invisible, has been acknowledged by Kuno (27) who argues that it constitutes only a very small element in insensible water loss because, relative to the surface area of the average male of 18,500 cm^2, the total area occupied by the 2,000,000 sweat glands is still small (about 90 cm^2).

Rothman (28) acknowledges the existence of this low level glandular activity at room temperature, the clear demonstration of it being found in the fact that when sweat gland activity is suppressed artificially there is a reduction in insensible water loss.

It thus appears that heat balance is achieved by a series of functional adjustments involving first, an increase in insensible perspiration due to a rise in skin temperature, followed by a further augmentation of insensible water loss by sweat gland activity of a special kind, and finally by frank sweating. Each stage has an identifiable and demonstrably specific character. The insensible perspiration is clearly a diffusion process only, the second stage involves a glandular activity but of a non-phasic kind, and the third stage is cyclic. These adjustments to a rise in body temperature correspond in an intriguing way to the successive adjustments of the body to a fall in temperature.

Between insensible perspiration and active sweating, therefore, there is an intermediate stage marked by a quite specific change in the Sudorimeter-record and apparently dependent on a combination of skin and environmental temperature. It is non-cyclic in character though showing some response to fluctuations in room temperature. It is quantitatively small, amounting in these experiments to about 10 to 15 g/h above the insensible perspiration water loss. It appears to be quite sudden in onset but is remarkably persistent and does not cut-out (as active sweating tends to do) with dramatic suddenness. It is suppressed by drugs acting on sweat glands. It is invisible to the unaided eye. Ectodermal displasiacs probably lack the facility. It bears striking similarity to the intermediate stage in a cooling body between vasoconstriction and shivering, by being the result of non-phasic gland muscle activity.

Pickering (29) has a descriptive paragraph in connection with shivering which by a substitution of terms where appropriate very neatly sums up the picture as it appears to us. He wrote:

"Electromyographic records show that at first the muscle units in the cold fire off at different frequencies and out of phase with each other: later the discharge becomes phasic. The former irregular contraction has been called thermal muscular tone: the phased discharge constitutes shivering".

Translating this into the proper terms of the present context we now have evidence that sweat glands in a warm (but not too stressful) environment may fire off at different frequencies and out of phase: later (as stress increases) the discharge becomes phasic. The former irregular contraction appears as insensible sweating: the phasic discharge constitutes frank sensible sweating.

There is therefore an interesting parallelism in the two compensatory mechanisms whereby the body adjusts itself to both increasing cold strain and increasing heat strain. Diagramatically it might be set forth as shown in Fig. 11.

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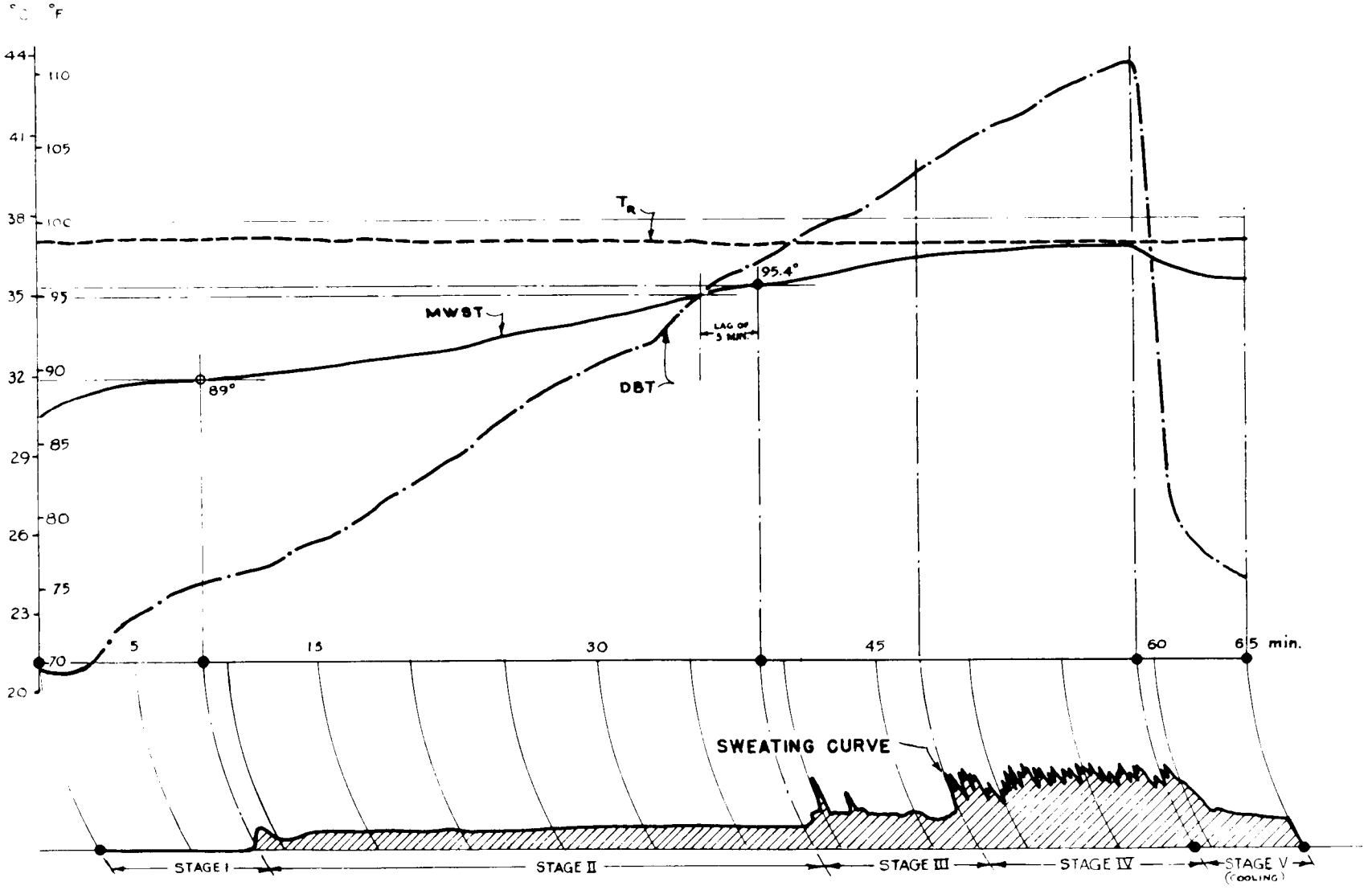

Fig. 1. Sweating curve for inactive nude subject seated in hot chamber with rising ambient temperature. Stage I, period of unrecorded insensible perspiration: Stage II, insensible sweating: Stage III, active sweating at low level with room temperature below MWST: Stage IV, active sweating at higher level as room temperature rises above MWST: Stage V, cooling curve, with reappearance of insensible sweating. All curves in Figs. 1 to 10 are redrawn to arbitrary scale.

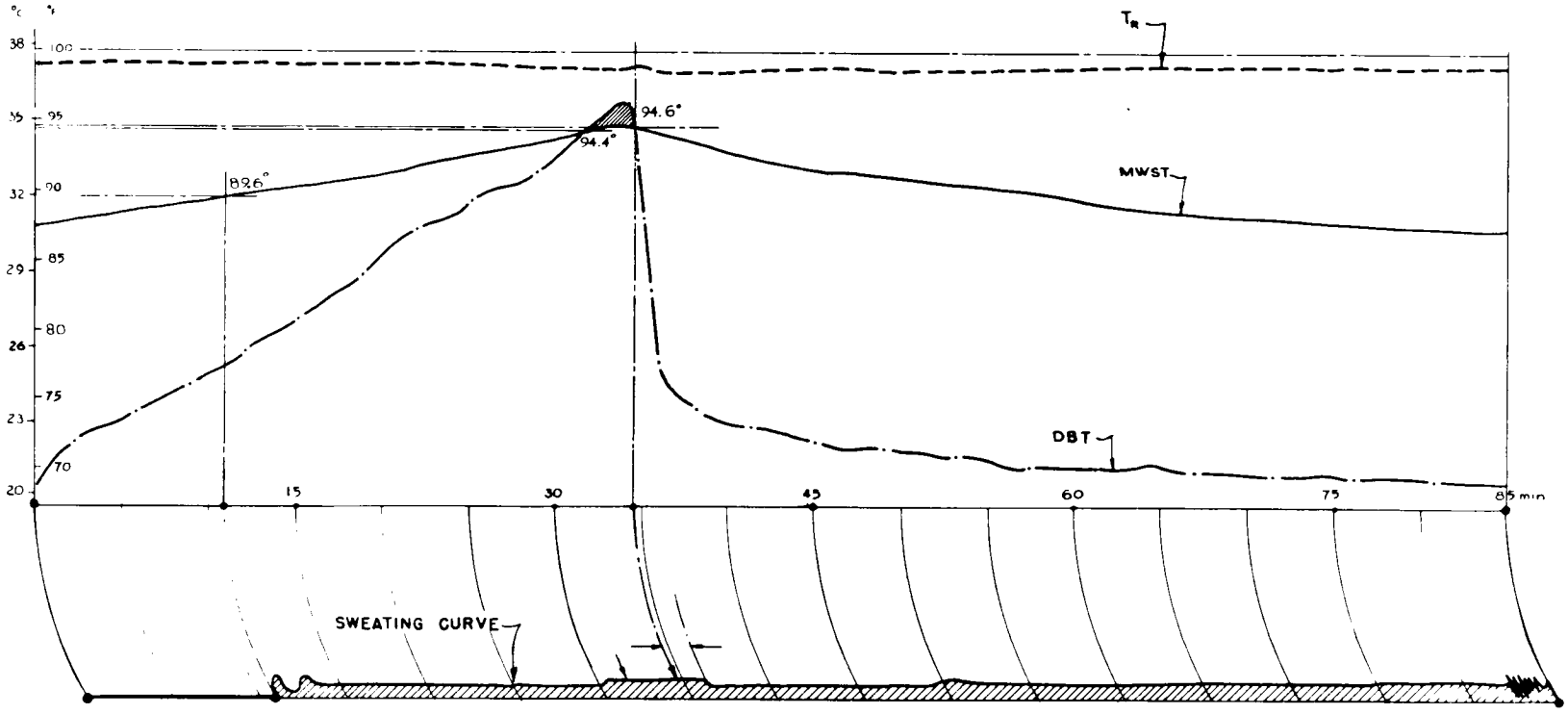

Fig. 2. Sweating curve for inactive seated subject showing insensible sweating pattern with slight rise when RT exceeded MWST.

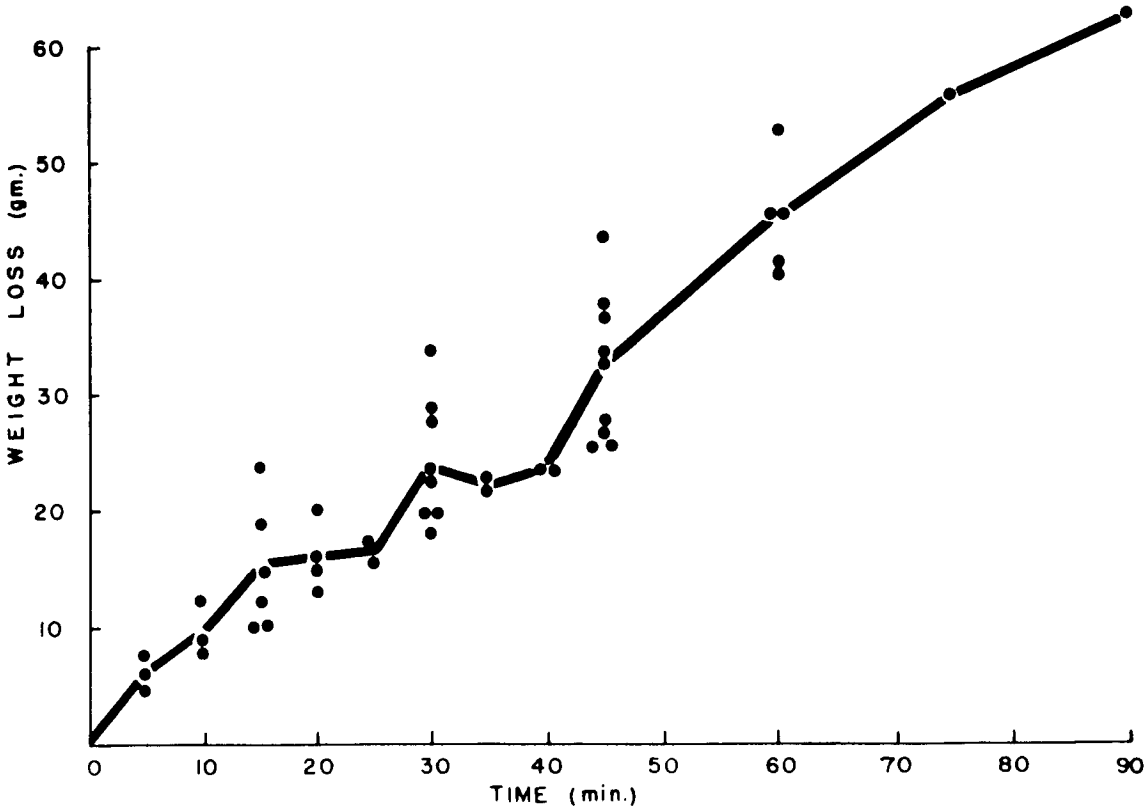

Fig. 3. Loss in weight via insensible perspiration for one subject at rest.

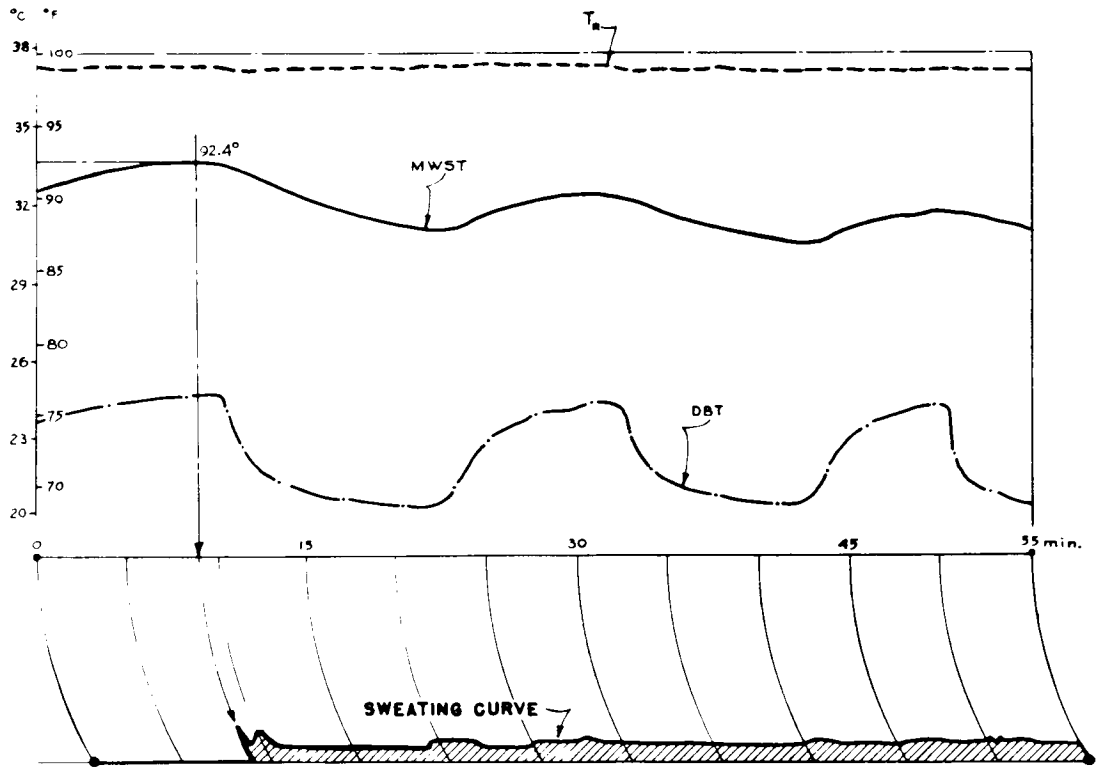

Fig. 4. Insensible sweating under conditions as in Fig. 2 with slight indication of response to successive heating cycles.

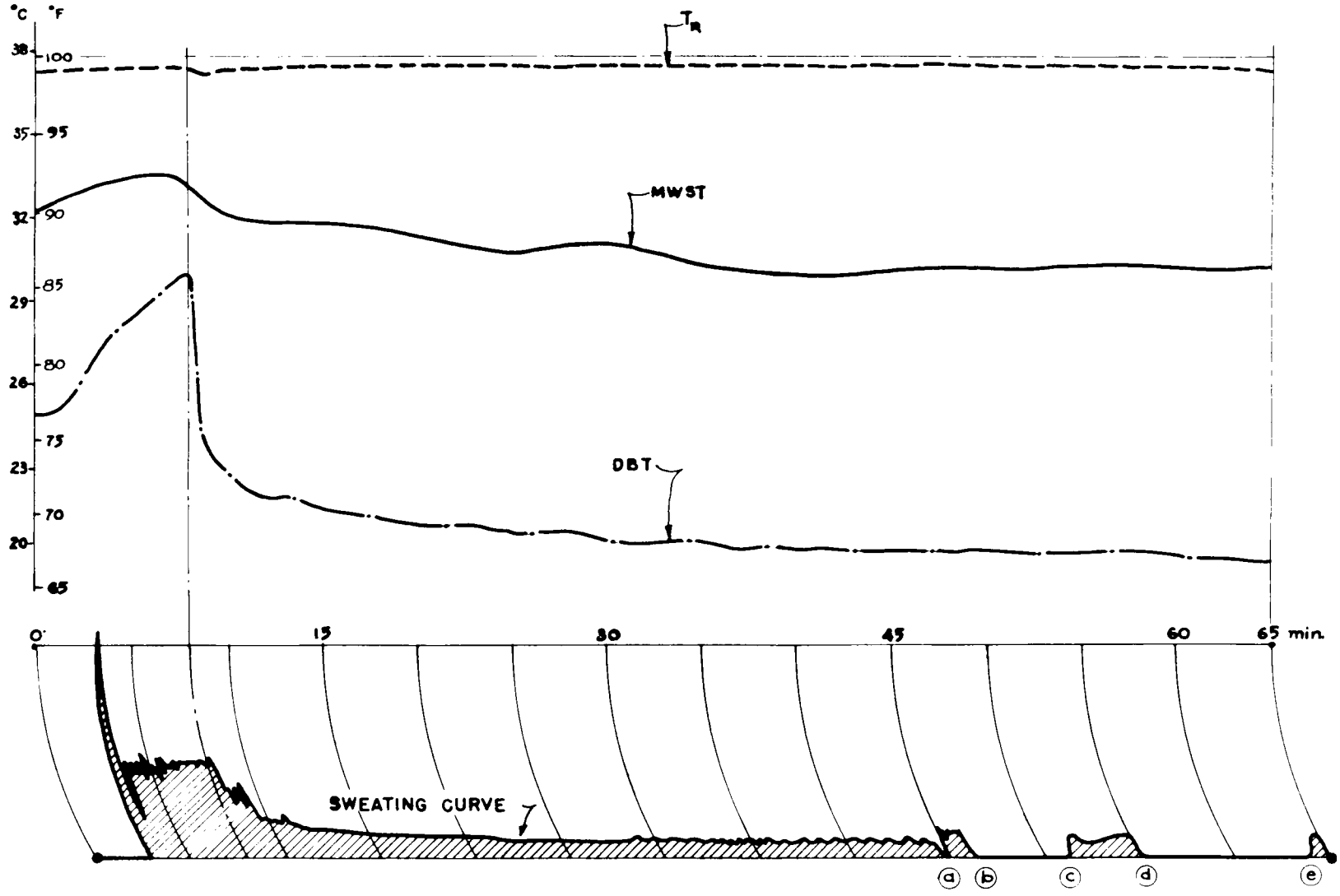

Fig. 5. Insensible sweating during extended cooling period (See text).

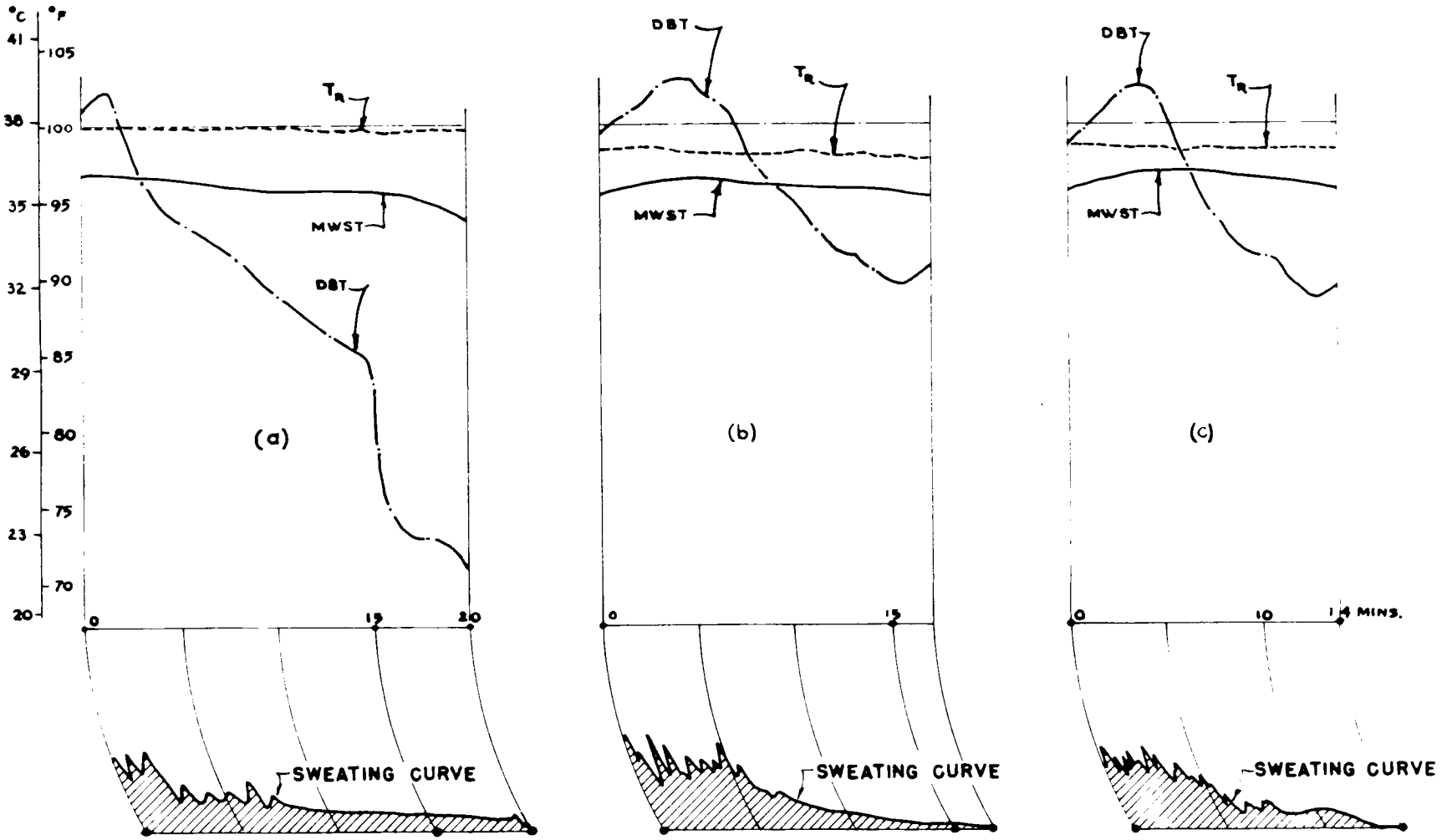

Fig. 6. Three cooling curves after exercise, showing that sweating (both active and insensible) will decline to zero under these circumstances.

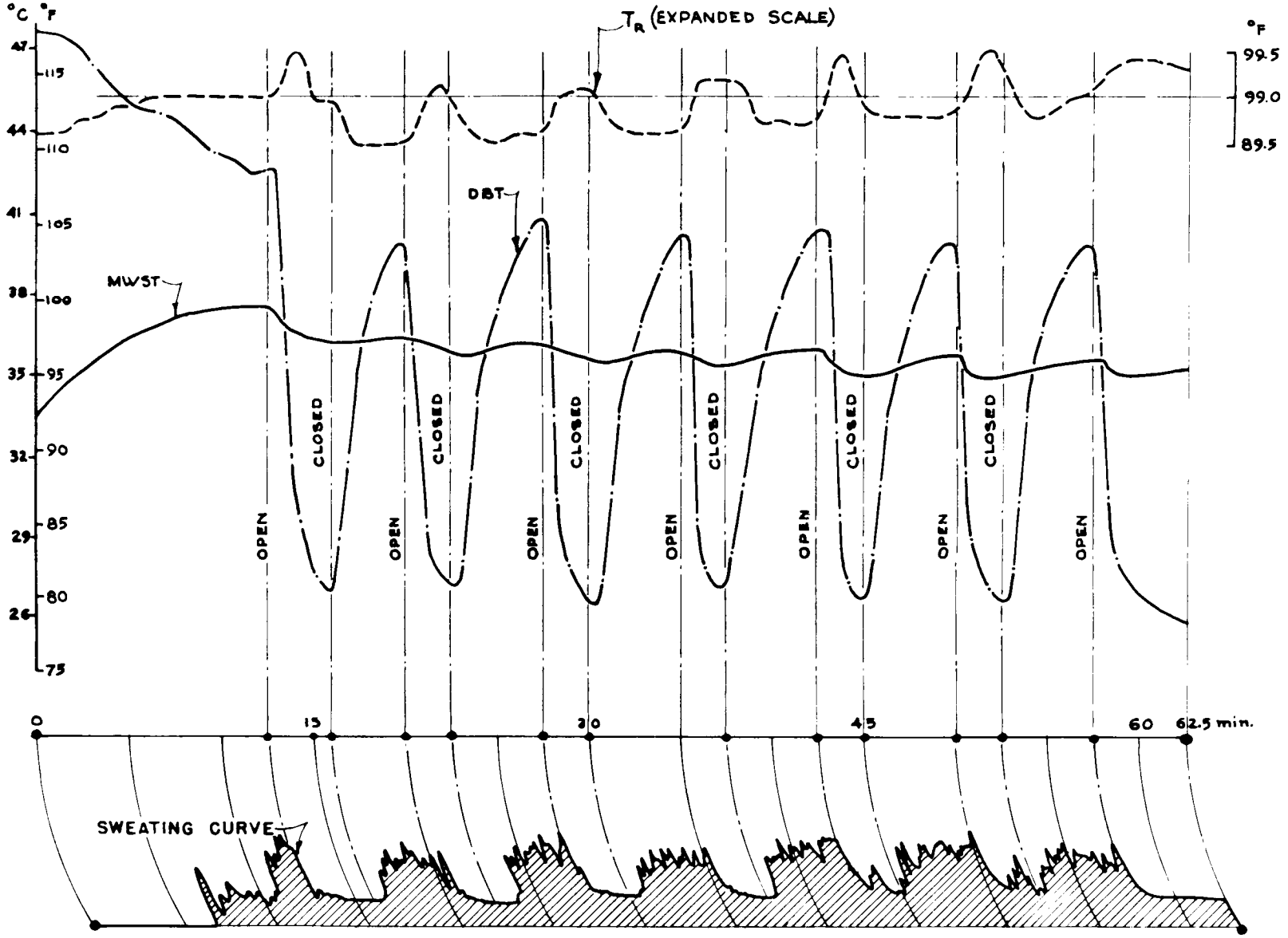

Fig. 7. Cycled heating of inactive nude subject, showing gradual elimination of insensible sweating stage in the cooling periods as body heat accumulated.

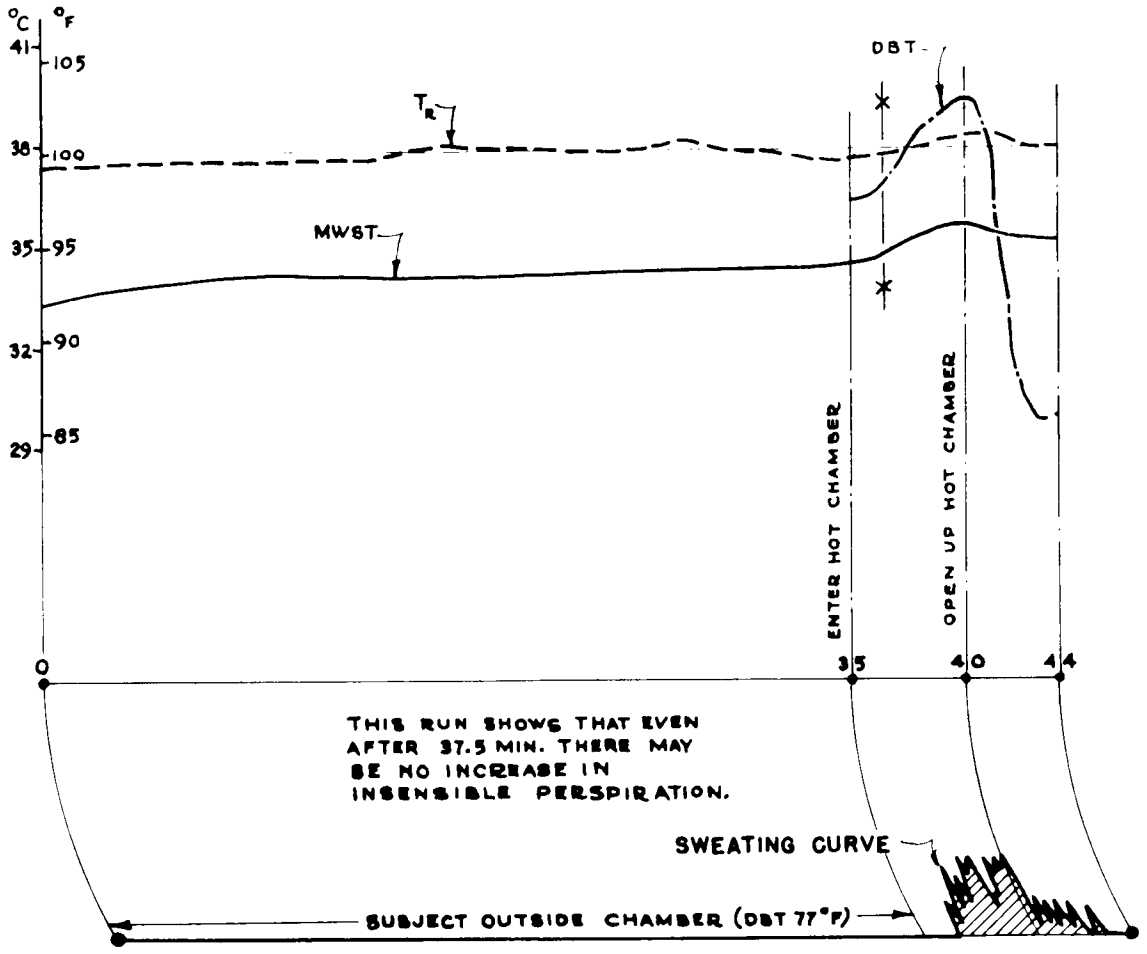

Fig. 8. Sweating pattern of nude subject before entering hot chamber and afterwards, showing absence of insensible sweating in these circumstances.

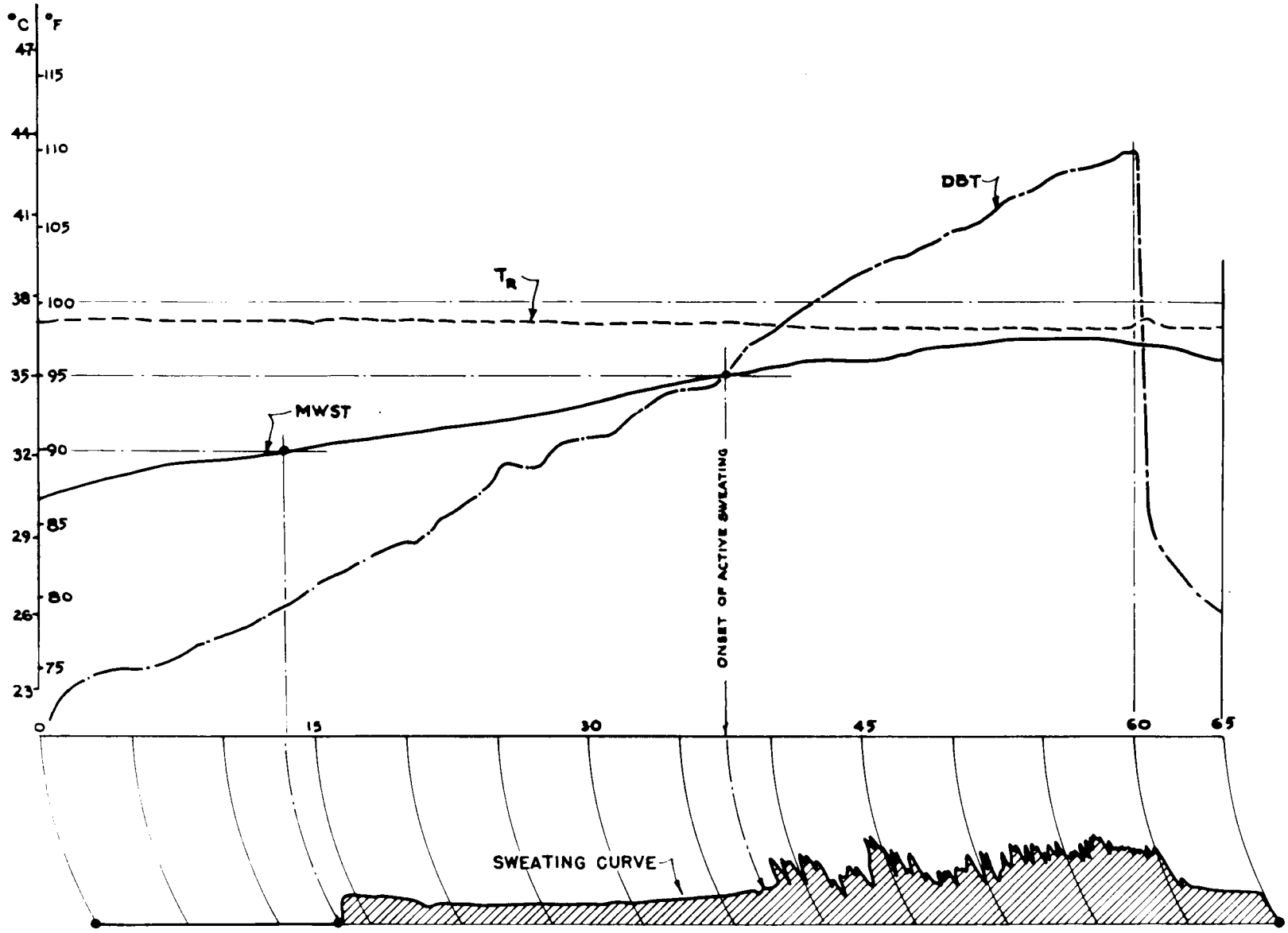

Fig. 9. Normal sweating pattern of nude resting subject as RT is raised, showing outbreak of insensible sweating at MWST of 90°F and active sweating at 95°F.

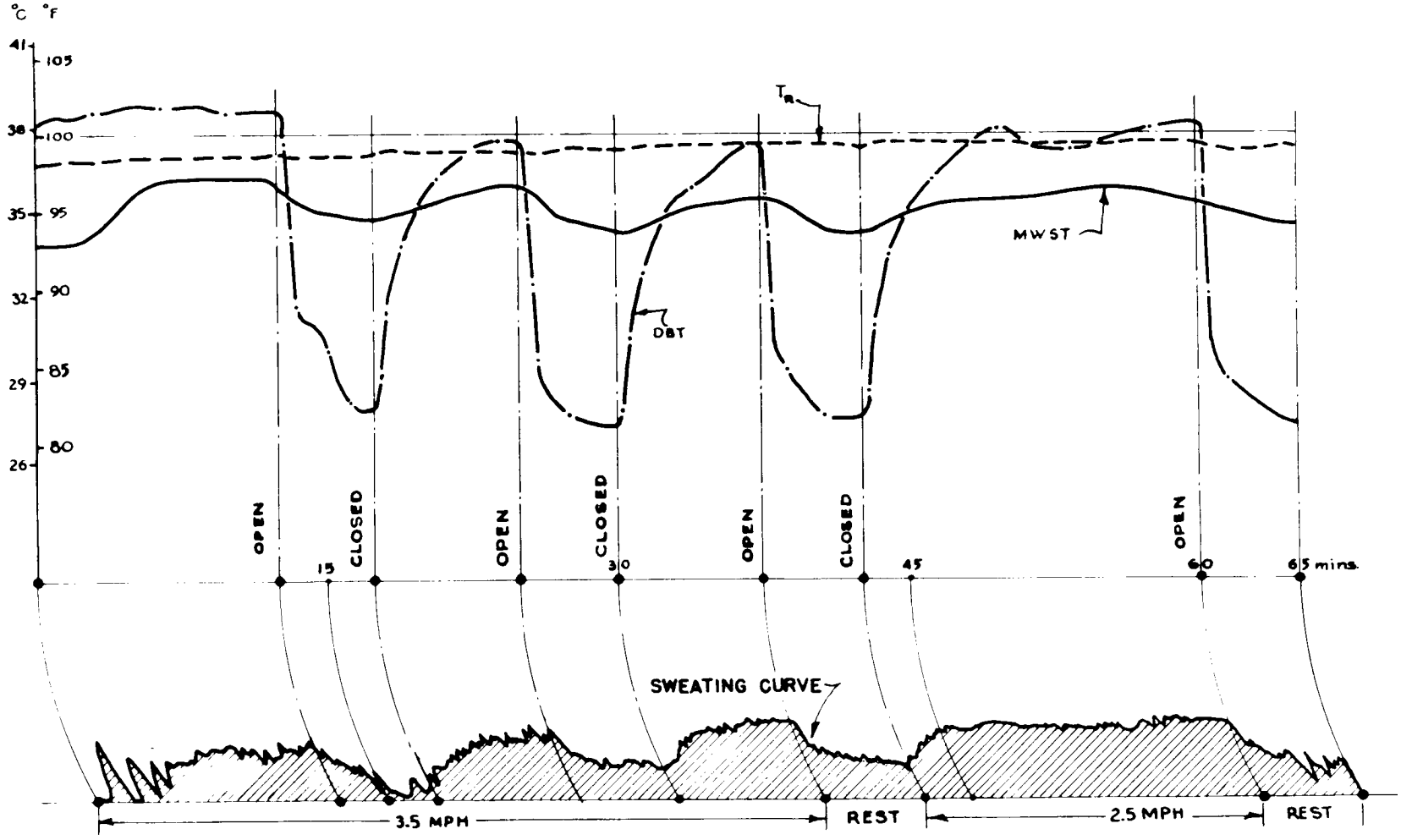

Fig. 10. Pattern of sweating of active subject, showing absence of insensible sweating after exercise.

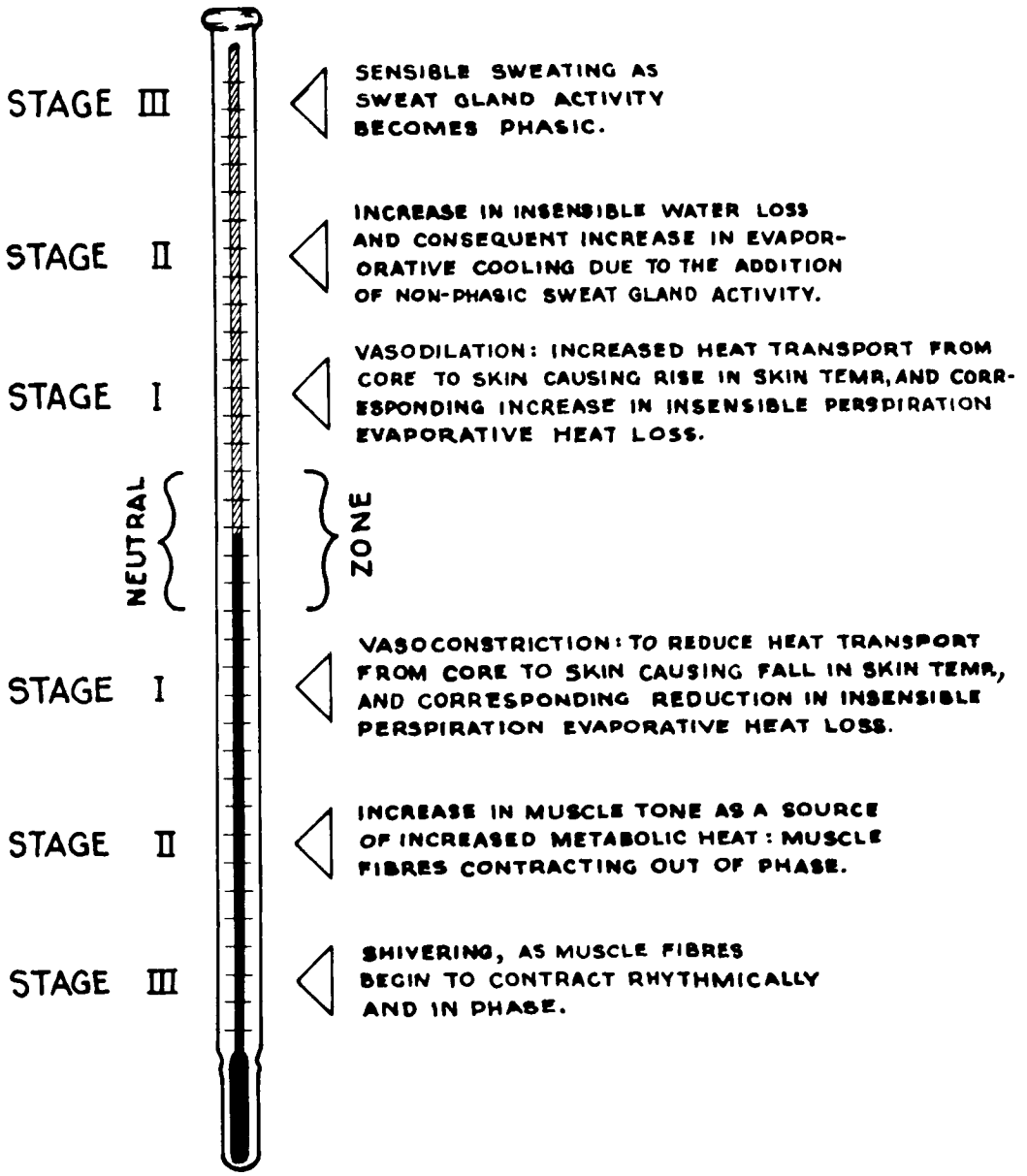

Fig. 11. Parallelism between stages in the mechanism of maintenance of thermal equilibrium in heat and cold stress. Neutral zone is the zone where vasomotor control is presumed to be in balance.

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