

RELATIONSHIP BETWEEN OSMOTIC PRESSURE OF THE BLOOD AND SECRETION
OF SWEAT

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RELATIONSHIP BETWEEN OSMOTIC PRESSURE OF THE BLOOD AND SECRETION OF SWEAT

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I.

Reviewing the not-very-numerous studies intended to establish ^{/439*} the reason why humans and some other animals sweat in a hot enough thermic environment, one soon realizes the dearth of our knowledge on this point.

The relevant literature is well-enough collected in Metzner's monograph [1] and only one fact seems to be undisputed: The thermic secretion of sweat occurs essentially when the blood temperature tends to rise above normal. We do not know the connection between the two facts; only Kahn's research [2] shows that the increased blood temperature may be an adequate stimulus for the sweat secretion centers.

Some of my previous observations [3] partly fill in this gap in our knowledge of the mechanism of thermic secretion of sweat. In fact, I showed that in the blood of animals exposed to high temperatures, substances develop capable of provoking the secretion of sweat. The fundamental experiment was as follows: a large cat is submerged in a bath of hot water at 42-45°C, and one waits until the known acceleration of breathing occurs (thermic polypnea); after about 10 minutes a certain quantity of blood (60-80 cc) is collected, defibrinated and injected into the jugular vein of another small cat kept in a cool environment. After a few minutes, on the paws of this second animal one observes a more or less abundant secretion of sweat, lasting according to the quantity of

*Numbers in the margin indicate pagination in the foreign text.

blood injected.

The fact that this involves a specific action of the blood of the heated animal is shown in that even the injection of a triple quantity (150-200 cc) of defibrinated blood from another, normal cat, or of isotonic sodium chloride solution, is absolutely devoid of any sudorific effect.

The fact that the temperature of the injected blood does not matter is proved by the observation that the blood of the cat in thermic polypnea provokes sweating even when injected at quite a low temperature (28-30°C).

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The sudorific action of the blood from the heated animal is exerted in the sweat secretion centers. If in a small cat one cuts all the nerves to one of the forepaws and blood from another cat in a hot bath is injected, sweat secretion occurs in the whole paws; but not in the one with the cut nerves. On the other hand, if the axillary artery of one of the forepaws is tied and the same amount of blood is injected, there is secretion even in the anemic paw.

From these observations, which I thought it worthwhile to summarize at a certain length because they are not well enough known, one sees that in the body of heated animals there develop sudorific substances and one can logically deduce that the thermic secretion of sweat is principally due to the formation of such substances. These observations, however, do not say what mechanism the secretion is produced by; it is this point that I tried to illuminate in the research reported here.

II.

To resolve the problem of the action mechanism of the sudorific substances, I started out from other research of mine on the variation in the molecular concentration of the blood of animals exposed to high temperatures [3]. Against any theoretical expectation, I

found that the molecular concentration of the blood drops notably when a dog is immersed in a water bath at 45-46°C and kept there until polypnea develops, and I also found that injection of the blood of this dog is capable of lowering the molecular concentration in a second dog receiving the blood. Recently I. Sandelowsky [4] came to identical conclusions, demonstrating by refractometry that in experimental hyperthermia and also often in fever, there is a notable decrease in the blood concentration.

Correlating these facts, I began to find out how the molecular concentration of the blood varies in a cat in which secretion was produced by exposing it to a sufficiently elevated temperature.

The development of thermic sweating in cats is very characteristic. If one exposes a young cat to a rather high temperature (39-40°C) one notes that after a few minutes the animal presents a noteworthy secretion of sweat, especially in the bare part of /441 the forepaws, lasting for a time that varies considerably, depending on the height of the temperature and the hygrometric state of the air. At a certain point, however, secretion stops suddenly, nor does it appear again later even though the animal is exposed to the same ambient conditions. In other words, the same thing happens in the cat as often happens in humans when exposed to a high temperature: profuse sweating at first, cessation of secretion and heat stroke later.

I used this observation to establish the variations in the molecular concentration of the blood during three different periods: first, during the absence of sweat, while the animal is in a cool environment; second during sweating, while it is in a hot environment; and third at the time at which sweating stops, when the action of the elevated temperature has already lasted some time.

Observations were performed in the summer; making use of the various environments available at the Institute I could perform

them easily. With a small cat tied to the operating table, I prepared one of its carotids and left it for a certain time (1 to 2 hours) in a ground floor room with northern exposure, temperature 19-20°C.

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When it was completely calm and showed no trace of sweat, I collected enough blood from the carotid to determine the point of congealment in a Beckmann cryoscope. Then I took the animal outside on the terrace, in full noon sunlight, where the air had been heated to 39-40°C. Then I collected a second blood sample for a second cryoscopic determination. I waited until secretion stopped (on the average about an hour) and just when the animal showed the first symptoms of intolerance to the environment (dilation of pupils despite the bright sunlight, signs of convulsive phenomena), without moving the animal I collected a third blood sample for a last determination. At the end of the experiment. I performed observations with the cryoscope on three blood samples, already defibrinated and kept in a cool place in closed containers to prevent any evaporation.

In the following table I present the data from the four determinations. They show that compared to normal, the molecular concentration of the blood (deduced from the congealment point) decreases considerably during secretion and increases notably when secretion stops.

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a		II		III
1 ^o	Catto peso g 1950	$\Delta = -0^0,670$	$-0^0,639$	$-0^0,711$
2 ^o	" " " 2000	$\Delta = -0^0,650$	$-0^0,620$	$-0^0,659$
3 ^o	" " " 1900	$\Delta = -0^0,633$	$-0^0,621$	$-0^0,672$
4 ^o	" " " 2200	$\Delta = -0^0,610$	$-0^0,611$	$-0^0,651$

Key: a. Cat, weight

N.B. The columns indicate, respectively, the Δ of the defibrinated blood of the cat: Normal (I), during secretion (II) and at the cessation of secretion (III).

This relationship, difficult to ascribe to coincidence, between the variation in blood concentration and development of sweating seemed to me to be highly important. One would suppose that the thermic secretion of sweat represents a means by which

the organism rids itself of the relative excess of water accumulated by the direct action of heat, a means that indirectly helps keep the thermic level constant because the sweat, caused by the lowered molecular concentration of the blood, helps lower body temperature by evaporating on the skin. In this way two groups of facts were brought together, formerly isolated in my previous observations: the production of sudorific substances and the lowering of the molecular concentration of the blood during heating.

It was, however, necessary to put the relationships seen in this first series of experiments to a more rigorous experimental examination, simplifying as much as possible the research conditions.

III.

The first simplification was to study the development of the secretion of sweat and the molecular concentration of the blood in some cats into which the blood of other cats heated in a hot water bath had been injected. As said, I had previously found that the injection of the blood of a heated animal caused secretion of sweat and lowering of the molecular concentration of the blood, but it was necessary to verify whether, in the same animal, the injection of the blood of another heated cat would at the same time produce both phenomena, sweating and a drop in the molecular concentration of the blood.

In the case of a positive result the connection between the two facts would be more evident, in that they could be produced not directly by the action of heat, but in fact even at a low temperature, by the action of specific substances formed in the organism by the action of the high temperature.

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For this purpose I immersed three large cats, suitably tied to a board, in a hot water bath at a temperature of 45-46°C, waited until polypnea developed, and after leaving them another ten minutes or so, collected about 100 cc of blood from the central

stump of one of the carotids, previously prepared. Defibrinated, filtered through paper and heated to 35-36°C, 60-70 cc of this blood was injected into the jugular of another kitten already kept tied to the operating table for an hour, in a cool environment (19-20°C) to keep it from sweating. Before injection I took a certain quantity from the peripheral trunk of the jugular, enough for a cryoscopic determination; after the injection I watched until sweating appeared in the bare part of the forepaws and collected a second blood sample for another cryoscopic determination. The results of two experiments performed were in complete accord and showed that in fact the injection of the blood of another, heated cat causes both secretion of sweat and lowering of the molecular concentration of the blood. I present the reports on the two tests:

1. Kitten, 1.200 kg, tied for an hour and exposed in a room at 19°C. No sweating. Point of congealment of defibrinated blood from the right jugular $\Delta = -0^{\circ}.668$. In the central stump of the right jugular, 50 cc of defibrinated blood was injected, at a temperature of 36°C, coming from another cat heated in a bath at 46°C. After 7 minutes, sweating occurred; after another two minutes a second blood sample was taken from the jugular, it was defibrinated and the congealment point was determined: $\Delta = -0^{\circ}.652$.

2. Cat, 1.520 kg. Ambient temperature 21°C; no sweating. Normal blood. $\Delta = -0^{\circ}.671$. Injection of 60 cc of blood from another cat heated as above. Secretion of sweat after 10 minutes. A second blood sample collected, $\Delta = -0^{\circ}.650$.

It is thus confirmed, as was a priori predictable, that even at low temperatures the injection of blood from a heated cat produces sweating and a rise in the congealment point of the blood. The coincidence of these two facts thus does not depend on the action of heat but on specific modifications of the blood due to heating.

IV.

Proceeding with the experimental analysis, I planned to discover the connection between the two phenomena observed, and since a priori it seemed improbable that the drop in the molecular concentration of the blood might be a consequence of sweating, I /444 began by verifying the opposite hypothesis, much more likely -- i.e., that the secretion of sweat can be a consequence of the decreased osmotic pressure of the blood.

I therefore tried to leave out of my research the factor of elevated temperature and to decrease the molecular concentration of the blood by other means to see if this condition alone was enough to cause the secretion of sweat.

Knowing that the endovenous injection of enough hypotonic saline solution will lower the molecular concentration of the blood for a certain time, I thought it worthwhile to make use of this simple artifice to see whether by lowering the molecular concentration of the blood in this way the production of sweat could occur.

The tests were performed on small cats, kept in a cool environment so as not to cause sweating. A small amount of blood was collected from one of the carotids, then defibrinated and kept aside in a closed container to be converted later for cryoscopic determination. The complete absence of sweat having been confirmed, enough 2 o/oo sodium chloride solution, heated to 38°C, was injected into the central stump of one of the outer jugulars. I then observed whether the animal sweated and during the secretion period collected a new blood sample from the carotid, to find the congealment point.

The experiment, ostensibly very easy, presented several difficulties. Sometimes the agitation of the animal caused spontaneous sweating due to the simple restraint on the operation table, a secretion that did not stop and thus made any continuation

of research useless. In other cases the amount of hypotonic solution injected was not enough to cause lowering of the molecular concentration of the blood. Finally, there were cases in which after injection the animal became agitated and thus it became difficult to establish whether the secretion of sweat depended on the lively movement or on the injection performed.

I therefore cite three of the experiments with the best results, which show that as osmotic pressure drops a notable secretion of sweat developed.

1. Cat, 2.000 kg. Tied to the operating table and the left carotid was prepared. It remained calm for half an hour in a half-dark room at a temperature of 17°C. A blood sample was collected, defibrinated and preserved for cryoscopy. The animal was still calm; then 130 cc of 2 o/oo NaCl solution heated to 37°C were /445 injected slowly over 6 min. At the end of injection the animal began to sweat and after two minutes one could observe large drops of liquid in the palms of the four paws, especially the forepaws. Another blood sample was taken. Congealment point of blood before injection $\Delta = -0^{\circ}.672$. After injection $\Delta = -0^{\circ}.610$.

2. Cat, 1.800 kg. Ambient temperature 16°C. Injection of 100cc 2 o/oo NaCl solution at 38°C over 5 minutes. Sweat manifested one minute from the end of injection.

Before injection $\Delta = -0^{\circ}.680$ in blood. After injection $\Delta = -0^{\circ}.620$.

3. Cat, 1.500 kg. Ambient temperature 16 °C. Injection of 65 cc of 2 o/oo NaCl solution at 38°C over 5 minutes. Very evident sweat only two minutes after the start of injection.

Before injection blood shows $\Delta = -0^{\circ}.631$. Afterwards $\Delta = -0^{\circ}.620$.

These observations thus show that even in a cool environment,

one needs only to lower the osmotic pressure of the blood artificially by injection of hypotonic liquid, to see the secretion of sweat increase.

Thus a fundamental fact was already established, i.e. that the secretion of sweat in the cases I previously observed is a direct consequence of the decreased molecular concentration of the blood. The main, if not sole, means by which high temperature causes sweating, must be sought in the lowering of the molecular concentration, which develops when the animal is heated. On the other hand, the sudorific action I already found in the blood of heated cats must not depend, as erroneously thought, on the presence of special substances that cause secretion, but rather on the special capacity of this blood to lower the molecular concentration of the animal into which it is injected.

One may object to these deductions that the known secretion of sweat I observed probably depends sic et simpliciter on the rather large amount of liquid injected and thus on the increase in arterial pressure caused transitorily by the increased volume of the circulating mass of liquid; but this objection falls aside if one considers: 1. Levy-Dorn [5] already demonstrated exhaustively that the secretion of sweat does not increase with blood pressure; 2. In my previous research already cited [3], I was able to inject significant quantities of defibrinated normal blood or /446 isotonic NaCl solution without seeing any sweating. The research already cited will serve to remove any other possible doubt on the point.

V.

This research represents a checkup on the previous. If it is true that the secretion of sweat can develop by lowering the molecular concentration of the blood artificially, in a sweating animal raising this concentration should suffice to stop the secretion of sweat.

a) A first series of tests was performed by injecting a hypertonic sodium chloride solution into cats already sweating because they were exposed to a high temperature.

As in the other experiments, for this purpose I used the simple measure of exposing the animal to the rays of the summer sun on a terrace where the temperature was ca, 40°C.

Before exposure I collected a blood sample from the carotid, defibrinated it and kept it in a closed container, to determine the normal Δ . When, under the rays of the sun, the animal had already sweated 3 or 4 minutes, such that large drops of liquid fell from the palms of the front and back paws, without taking it from the hot thermic environment I injected into the previously prepared outer jugular a certain amount of 20 o/oo NaCl solution. Once the course of the secretion of sweat had been determined, I took a second sample of carotid blood to establish the new congelment point and check the effect of the injection.

Here are the results of two experiments:

1. Cat, 3.500 kg. From the carotid, 20 cc of blood were collected and defibrinated, $\Delta = -0^{\circ}.621$. Exposed to sun, air temperature 40°C. After 5 minutes, abundant sweating on all four paws. Without removing the animal from its position, 30 cc of 20 o/oo NaCl solution were injected into the jugular. After one minute the skin of the paws dried and there was no further secretion of sweat.

The congelment point of the carotid blood sampled at this point was $\Delta = -0^{\circ}.719$.

2. Cat, 3 kg. Normal Δ of blood = $-0^{\circ}.630$. Exposed at 39°C to the sun, sweats copiously. 5 minutes after start of secretion, 40 cc of 20 o/oo NaCl solution injected. Secretion stops even before

before the injection is over. Δ of blood 3 minutes after stop of secretion = $-0^{\circ}.721$.

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These observations show the stop of sweating in relation to the increase in the osmotic pressure of the blood so clearly that I thought it unnecessary to multiply them, and they prove among other things that the spontaneous stop of the secretion of sweat after prolonged exposure to heat, which I found to coincide with an increase in the molecular concentration of the blood, indeed depends on this factor.

b) A second series of analogous tests concerns the behavior of the secretion of sweat, provoked by the injection of hypotonic sodium chloride solution, when the blood is then rendered hypertonic by the introduction of highly concentrated saline solution into circulation.

In a cat kept in a cool environment, and thus not sweating, a certain solution of hypotonic sodium chloride solution was injected; hardly had the secretion of sweat begun when a highly hypertonic solution of the same salt was injected, and the development of the secretion was noted. Cryoscopic determination with carotid blood taken before injection, after the hypotonic injection, and after the last, hypertonic injection, indicates the variations occurring in the molecular concentration of the blood.

As can easily be understood, not all the experiments performed in this way can be sure of succeeding. In fact one does not always find the right proportions between the hypotonic and hypertonic solutions such as to make the value for the molecular concentration of the blood first drop and then rise. I therefore do not report all the experiments performed, some of which were demonstrative only in a certain sense, but cite only the following among the most successful:

Cat, 3.800 kg. A sample of normal blood is collected from the left carotid. $\Delta = -0^{\circ}.618$. Air temperature 19°C ; animal not sweating. 150 cc of 2 o/oo NaCl injected; duration of injection 5 minutes. After 2 minutes, small pearls of sweat appear on the bare skin of the paws; at this time a second carotid blood sample is taken, $\Delta = -0^{\circ}.600$. Immediately afterwards, 50 cc of 20 o/oo saline solution injected. Duration of injection 2 minutes. Injection was not yet terminated when sweating stopped. A third blood sample had $\Delta = -0^{\circ}.710$.

The secretion of sweat is therefore, at least in our case, a function of the osmotic pressure of the blood.

With this experiment we only reproduced artificially what happens in the cat when it is exposed to a high thermic ambience; there is sweating when the osmotic pressure drops, and sweating stops when this rises above normal.

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On the other hand the same experiment once again proves that the secretion of sweat cannot depend on the increase in blood pressure caused by the volume of liquid injected, otherwise it would not have stopped with the intromission of new liquid into the circulatory system.

VI.

From the combination of research reported here, it is certain that the secretion of sweat occurs after a decrease in the osmotic pressure of the blood and stops when this pressure rises. The thermic secretion of sweat would represent a special case of this law, since a lowering of the osmotic pressure of the blood occurs precisely during the thermic secretion of sweat and a rise occurs when it stops. It was necessary to see by what mechanisms the various alterations of the osmotic pressure of the blood, whether caused by heat or not, affect the secretion of sweat. Various facts already known lead to the supposition that a central mechanism

must be involved.

Research to date does indeed tend to recognize the central origin of thermic sweating.

For details the reader is referred to the cited work of Metzner, but I think it useful to point out that in general one must speak of a central action because a cat in which one paw's nerves are cut, kept in a very hot environment, sweats in the three whole paws and not in the one with cut nerves; also, one must rule out a reflex action on the basis of the observation that by cutting a cat's dorsal spinal roots, corresponding to one of the sciatic nerves, and exposing the animal to heat, one finds sweat developing in all four paws even though one is rendered completely anesthetic and no reflex is possible.

However, it is not clear how high temperature provokes this central sweating action. The high temperature of the blood may in itself represent an adequate stimulus to the central sweat secretion organs. In this respect Kahn's research is very demonstrative; by surrounding the carotids with suitable heating equipment, he saw secretion of sweat develop in cats when only the temperature of the encephalon had risen, while that of the rest of the body was normal.

On the other hand, my own already-published research, cited at the start of this paper, tends to admit a central origin of the thermic secretion of sweat, because it shows that the sudorific substances that form in the body of heated cats act exclusively /449 on the secretion centers.

Now, recognizing that these hypothetical sudorific substances are really so because they can modify osmotic pressure of the blood, it remained to find out whether the drop in this pressure causes sweating by peripheral or central action.

Before starting any test on the possible central action, I thought it well to resort to the indirect measure of studying how the molecular concentration of the blood varies during the secretion of sweat caused by pilocarpine which, as is known, acts directly on the glands or their intrinsic secretory nervous apparatus.

The research was very simple; I injected pilocarpine into a cat in doses sufficient to provoke sweating, and determined the congealment point of the blood before and during sweating. The experiments I report demonstrate that the secretion of sweat due to pilocarpine generally does not coincide with any variation in the osmotic pressure of the blood. But sometimes, during secretion, the osmotic pressure rises and decreases just as happens in thermic secretion.

Here are the results of some experiments.

1. Cat, 1.500 kg. Normal carotid blood $\Delta = -0^{\circ}.621$. Sweat after injection of 1/4 cg of pilocarpine chlorhydrate in a 1% aqueous solution. Δ of blood during secretion = $-0^{\circ}.622$.

2. Cat, 2.300 kg. Normal carotid blood $\Delta = -0^{\circ}.618$. Profuse sweating after injection of 1/2 cg pilocarpine chlorhydrate. Δ of blood during secretion = $-0^{\circ}.618$.

3. Cat, 2.800 kg. Normal carotid blood $\Delta = -0^{\circ}.635$. Injection of 1/2 cg of pilocarpine chlorhydrate, abundant sweating. Δ of blood = $-0^{\circ}.642$.

I did not continue this line of study, which related indirectly to my theme, but it seems worth further investigation. For me it was enough for the moment to certify that the secretion of sweat, certainly provoked by a mechanism of direct stimulation of the glands, does not depend or at least does not coincide with appreciable variations of the osmotic pressure of the blood.

VII.

To determine directly a probable stimulation of the sweat secretion centers by the blood when its osmotic pressure has dropped below normal, I simply repeated the aforementioned experiment, from which I could conclude that the blood of the heated animals provokes secretion by way of action on nervous centers.

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In a cat I cut all the nerves of the right axillary cavus in such a way as to separate one of the front extremities from any contact with the nervous centers. I chose the nerves of the forepaw because here, as is known and as I confirmed in all my observations, the secretion of sweat is more abundant and easier to produce. The operation is a but more complicated than that of cutting the sciatic nerve, but presents no grave difficulties.

The animal was allowed to rest for about 4 hours to let it recover from the trauma and especially to avoid the possible effects of mechanical stimulation of the stump section of the cut nerves. First confirming that the animal was not sweating, I injected 150 cc of 2 o/oo NaCl solution at a temperature of ca. 38°C into the outer jugular. The liquid was injected rather slowly, for 6 minutes; two minutes after injection the usual sweating appeared, quite marked in the back paws and very evident in the left forepaw. In the right forepaw, the one with cut nerves, there was no trace of secretion. This observation, which coincides with the other analogous one made with the blood of a heated animal, represents a valid enough proof that a decrease in the osmotic pressure of the blood causes sweating by the intermediary of the central sweat secretion organs.

VIII.

To complement the preceding observation I planned another experiment to confirm indirectly what has already been presented.

As already said, Kahn, by heating the carotids artificially,

demonstrated that the encephalic sweat secretion centers are activated by the increased temperature of the blood reaching them. I performed an analogous experiment; consisting of allowing blood with low molecular concentration to reach the encephalon in isolation, without any variation in the rest of the circulatory system. This goal was achieved easily by tying off one of the carotids in a cat and injecting into the peripheral stump of the other a hypotonic sodium chloride solution heated to body temperature. It is clear that by this method one circulates blood with a low molecular concentration in the encephalon because the hypotonic liquid injected into the carotid must necessarily mix, in Willis's circulatory system, with the blood from the two vertebral vessels, and thus arrives diluted at various points in the encephalon. To limit the drop in osmotic pressure to the /451 blood of the encephalon alone it is necessary, as one can easily understand, to inject the hypotonic liquid in a suitably limited proportion and at a moderate rate; furthermore, the pressure at which the liquid is injected must exceed that of the animal's arteries by just the right amount.

Given these advance considerations, I conducted the experiment as follows: In a large cat weighing 3 kg the two carotids were isolated and a blood sample was taken from the stump of the left one, $\Delta = -0^{\circ}.618$. With the right carotid tied, the central stump of the left one, previously cut, was injected with a 2.5 o/oo sodium chloride solution heated to 39°C , at a pressure of 17 mm Hg. The injection was performed at the rate of $10\text{ cm}^3/\text{minute}$. Hardly had 30 cc of the liquid penetrated (i.e. 3 minutes from the start of injection) when the forepaws of the animal, which previously had been perfectly dry, began to become wet with sweat. Injection was suspended, the skin of the paws was dried with blotting paper, and after a few seconds large drops of sweat appeared. At this point a certain amount of blood was collected from the right crural for another cryoscopic determination; the congelment point was identical, $\Delta = -0^{\circ}.618$.

Secretion was evident for another minute and then stopped gradually during the next minute.

This experiment thus proves that it is enough to lower the osmotic pressure of the blood circulating only in the encephalon to produce secretion of sweat. One must therefore deduce that the decrease in the normal osmotic pressure represents a sufficient stimulus for the sweat secretion centers and thus that the secretion produced by heat or by the injection of hypotonic liquids is always of central origin. One may indeed object to this research that the secretion of sweat developed not due to the specific action of hypotonic liquid, but due to other conditions dependent on the introduction of the liquid per se; I will only note in passing that the experiment repeated with a NaCl solution isotonic with the blood of the cat gave negative results.

General Conclusions

From my research essentially the following facts result:

1. The thermic secretion of sweat coincides with a lowering of the osmotic pressure of the blood, as the cessation of sweating coincides with an increase in this pressure.
2. By injecting defibrinated blood from one cat previously heated in a hot water bath into another cat, one produces sweating and at the same time a drop in the osmotic pressure of the blood.
3. By injecting a hypotonic sodium chloride solution into a cat, one produces secretion of sweat when the injection reduces the osmotic pressure of the blood.
4. By injecting a hypertonic sodium chloride solution into sweating cats (both due to direct action of heat and due to the injection of hypotonic saline solution) one produces a cessation of the secretion of sweat with an increase in the osmotic pressure

of the blood.

5. The drop in the osmotic pressure of the blood, however it is produced, is a sufficient stimulus for the central nervous organs of sweat secretion.

These facts, as one can easily see, constitute a noteworthy advance in our scarce knowledge of the sweat secretion mechanism, and more especially of sweating due to heat.

They confirm what was known before, i.e. that this secretion is essentially of central origin, and demonstrate that the nervous sweat centers are very sensitive to the lowering of the molecular concentration of the blood perfusing them. The thermic secretion of sweat thus represents a special case of a general law: The central nervous apparatus that controls the secretion of sweat begins to function when the osmotic pressure of the blood drops below normal, i.e. when the relative quantity of water circulating in the body increases. In this way another of the organism's many devices to regulate its osmotic pressure is revealed.

With respect to the thermic secretion of sweat, this device also has a thermoregulatory significance. The drop in the osmotic pressure of the blood due to heat (as I showed in my "biothermic research" and confirmed in the present study) activates the sweat secretion centers, thus helping to lower the elevated temperature of the body.

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