Free flow of sweat due to loss of surface tension at sweat droplets causes water-induced skin wrinkling

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Abstract

Water immersion skin wrinkling has long been used as a test for sympathetic nerve function. However, the cause of underlying mechanism remained elusive. In this article, we theoretically investigate a possible cause of the phenomenon by taking various properties of sweating into consideration. The pressure exerted by the surface tension of sweat droplets counterbalances the secretory pressure of sweat glands at the pore. When a hand is immersed in water, sweat droplets easily merge with the water, causing the pressure to drop at the pore. Our calculations, using earlier measurements of secretory pressure, show that the water pressure at the sweat pore will be less than the secretory pressure of sweat glands when the hand is immersed at a shallow depth. The resulting pressure imbalance enables the sweat to flow freely into the water. We believe that there will be an initial vasodilation to feed the excess generation of sweat. Sweat flow continues as long as there is blood flow to the hand. To prevent excessive loss of sweat from the body and to maintain homeostasis, sympathetic nerves trigger vasoconstriction to reduce the blood flow to the hand. The overlying skin wrinkles due to loss of volume under the skin. It is possible that denerved fingers remain in the vasodilation state during immersion due to a lack of sympathetic nerve function.

Keywords: water immersion skin wrinkling, sweat droplet, surface tension, secretory pressure, homeostasis, sympathetic nerve function, vasodilation, vasoconstriction.

Introduction

Water immersion skin wrinkling (WISW) is a well-documented phenomenon. Wrinkles appear on the skin if the hand is immersed in water for approximately 30 minutes. The skin surface reverts back to normal approximately 10 minutes after removing the hand from the water. Aquagenic wrinkling of palms (AWP) is a rapid formation of wrinkles upon limited exposure to water. It has been reported that vasoconstriction and the subsequent loss of volume under the skin is the cause of skin wrinkling (1). Wrinkling of the skin has long been used as a test for sympathetic nerve function (2-4). However, the mechanism that initiates the vasoconstriction remained elusive. In this article, we theoretically investigate a possible cause of WISW by taking into consideration earlier observations and interaction of pressure components acting at a sweat pore.

Analysis of pressure components at the sweat pore

Eccrine sweat gland continuously generates new sweat and exerts pressure on the sweat droplet at the pore. The size of the sweat droplet will increase with the addition of new sweat. At the same time, part of the droplet continuously evaporates by absorbing heat from the skin. The remainder of the droplet acts as a barrier between the secretory pressure of sweat gland and the atmospheric pressure. The process of sweating in a resting body can be divided into three different pressure components acting at a sweat pore: (a) the secretory pressure of sweat gland (P_g), (b) the decrease in secretory pressure due to partial evaporation of sweat (P_e) and (c) the pressure exerted by the surface tension of the sweat droplet (P_s). In a resting body, these three pressure components will be in equilibrium. Therefore, the pressure component P_g equals the sum of P_e and P_s, or $P_g = P_e + P_s$. When taking the atmospheric pressure (P_a) into consideration as well, the absolute secretory pressure of sweat gland (P_G) can be calculated as the sum of P_e, P_s and P_a, or $P_G = P_e + P_s + P_a = P_g + P_a$. If sweat evaporation is absent, then the sweat droplet will continuously grow in size because of the increased secretory pressure on the droplet. If the pressure component P_s is also absent, then the sweat freely flows outward due to the pressure imbalance.

It has been observed that the secretory pressure of sweat glands in cystic fibrosis patients and control subjects measured up to 500 mm Hg (5). Assuming the measurement reflected the sustained secretory pressure of sweat gland at the pore (P_g), the absolute secretory pressure of sweat gland (P_G) in these cases can be calculated as 1260 mm Hg with the addition of atmospheric pressure of 760 mm Hg. The pressure exerted by water at the sweat pore when a hand is immersed in water at a depth of 10 cm is 7 mm Hg. Therefore, the combined pressure exerted by the water column and the atmospheric pressure will be 767 mm Hg, and the pressure difference at the pore will be 493 mm Hg.

The principle for the pressure exerted by the surface tension of a droplet is, $P_s = 2s/r$, where s is the surface tension of the liquid and r is the radius of the droplet. The surface tension of a liquid increases with the density of the liquid. Therefore, the P_s will increase with the increase in the density of the liquid. Due to an inverse relation, the P_s will increase when the radius of the droplet decreases. The lower limit of the inner diameter of a sweat duct is 10 microns (6). The surface tension of water at 20° C is 72.8 dynes/cm. Therefore, the pressure exerted by the surface tension of a hemispherical water droplet with a radius of 5 microns can be calculated as 291200 dynes/cm^2, which is equal to 218 mm Hg. The calculated value of P_s for a minute droplet, in this particular

scenario, suggests the possibility of significant amount of pressure within the sweat duct without accounting for the pressure lost due to evaporation. In these circumstances, there will be an excess amount of sweat loss than the regular loss of sweat due to thermoregulatory process when both the pressure components P_e and P_s were absent.

When the body is at rest, the secretory pressure of sweat gland will be in equilibrium with the pressure components P_e and P_s. In these conditions, when a hand is immersed in water, the sweat droplet breaks apart and quickly dissolves within the water. P_s will be equal to zero in the absence of a sweat droplet. Evaporation of sweat ceases to exist because the hand is immersed in water. When these two pressure components, P_e and P_s, are absent then the sweat within the duct exerts its full absolute secretory pressure at the pore. As mentioned earlier, there will be a pressure difference of up to 493 mm Hg between the absolute secretory pressure of sweat glands and the water pressure at a depth of 10 cm. Therefore, sweat flows into the water continuously due to the pressure difference. Increased sweat flow may cause an initial vasodilation to feed the excess sweat generation. This loss of water content from the blood exceeds the normal sweat loss due to evaporation. The loss of sweat continues as long as there is blood flow to the region around the sweat glands. To control the loss of water and maintain homeostasis, the sympathetic nervous system triggers vasoconstriction, causing a reduction in blood flow to the area around the sweat glands. The skin then wrinkles due to loss of volume under the skin.

Hypothesis

Based on the preceding analytical observations, we summarize the cause of WISW with the following sequence of events as a hypothesis. First, the hand is immersed in water, causing a loss of surface tension at the sweat droplets due to the merger of the droplets with the water. This is followed by the free flow of sweat into the water due to a pressure imbalance between the secretory pressure of sweat glands and the pressure within the water at the pore. Vasodilation feeds the excess sweat generation. Next, sympathetic nerves are triggered to control the excess sweat loss, causing vasoconstriction and reduction of blood flow to the hand to maintain homeostasis. The resultant loss of subdermal volume causes wrinkling of the overlying skin.

The P_s needs to be a mere 10 mm Hg more than the water pressure, during immersion at a depth of 10 cm, to generate excess flow of sweat than the regular evaporation. As shown earlier, the value for P_s could exceed 200 mm Hg in a particular scenario. Therefore, we speculate that the time taken to form wrinkles will decrease with the increase in the P_s. A high value for P_s possibly causes faster depletion of sweat along with quick formation of wrinkles when immersed in shallow water and it could be a case for AWP. Similarly, a medium value for P_s is possibly a scenario of WISW. Sweat loss due to lower value of P_s increases the time taken to form the wrinkles. Wrinkles might not appear on the skin if the pressure within the water is equal to or greater than the P_s. Pressure exerted by the water increases with the height of the water column. Therefore, it is possible that the time taken to form wrinkles increases with the depth of immersion.

Discussion

The mean secretory pressure of sweat glands in cystic fibrosis and control subjects was reported as 300 mm Hg (5). Even at a depth of one meter, the absolute secretory pressure will be 200 mm Hg more than the combined pressure exerted by the water column and the atmosphere. When a hand is

immersed in water, a continuous channel will be established between the sweat glands and the water in the absence of a barrier. Sweat therefore flows into the water due to the pressure difference. At a shallow depth, the sweat freely flows into the water driven by a pressure equal to the secretory pressure of sweat glands. The observed dilation of sweat ducts in AWP (7) suggests a rapid flow of sweat. Stationary water in a container at shallow depth cannot exert enough pressure to create such a rapid flow of water from the container into the sweat duct. Furthermore, skin wrinkling was observed, in some instances, even when the hand was only briefly exposed to water at a shallow depth. Therefore, the observed dilation of sweat ducts is likely due to the rapid flow of sweat out of the pore during immersion.

It has been reported that AWP, after 5 min of water immersion, was limited to the area where hyperhidrosis occurred (8). Hyperhidrosis occurs due to a higher sweat secretory rate, and it can be associated with higher secretory pressure of sweat glands. It is possible that the values for P_s and P_e will increase with the increase in the secretory pressure of sweat glands. Therefore, we can deduce that the wrinkles occurred in the areas where there was higher P_s prior to the immersion. A strong positive result for iodine-starch tests indicates the possibility of excessive sweating in wrinkled areas even after immersion. The observation of an increased evaporation immediately after skin wrinkling (9) also suggests an increase in the sweat rate after immersion. It is possible that the dilated sweat ducts enabled the increase in the sweat flow after immersion.

These earlier observations, in conjunction with the proposed hypothesis, strongly suggest that sweat flows out from the pores during water immersion. Excessive loss of sweat in a resting body, in addition to the normal evaporation, triggers the reduction of blood flow to the hand to maintain homeostasis. The skin then wrinkles due to vasoconstriction and loss of volume under the skin. The skin surface gradually returns to normal after immersion due to the formation of sweat droplets and the resumption of normal blood flow to the hand. Wrinkles appear only on palms and soles because they have more sweat pores (10). More pores cause more sweat and faster depletion of water from the body when immersed in water.

An alternative theory for the trigger mechanism of skin wrinkling proposed that increased firing of sympathetic neurons due to the alteration of epidermal electrolyte homeostasis by water absorption causes WISW (11). The absorption of water will be more in areas with less sweat compared to the areas with hyperhidrosis. Therefore, dense wrinkles should form around the areas with less sweat according to this theory. The observation of dense wrinkles in the areas with hyperhidrosis (8) does not explain how water absorption would be higher in these areas even after saturation with sweat. Skin remains wrinkled on hands as long as the hands are exposed to water. It is not clear, in the existing theory, how the increased firing of neurons is maintained over a long period of time. Moreover, the pressure difference between the sweat duct and the water suggests the flow of sweat towards the pore, not the absorption of water into the sweat duct.

The effect of skin wrinkling may also result from dipping the hands into any material that quickly absorbs sweat without allowing it to coalesce as a droplet at the pore. It has been observed that when vasoconstrictive cream, such as eutectic mixture of local anesthetic (EMLA), is applied to distal digits, the degree of skin wrinkling is similar to that resulting from water immersion (12). EMLA cream might absorb the sweat as soon as it forms at the pore. Sweat would continue to flow at the rate of absorption by the EMLA cream. Addition of sweat to the EMLA cream either turns

the cream into a liquid form, or the sweat evaporates by absorbing heat from the surface of the skin. The EMLA cream may increase the sweat evaporation rate because when in EMLA cream, the sweat occupies more space on the skin than as a droplet at a pore. To prevent the loss of sweat, the body reacts by restricting the blood flow to the digits, causing skin wrinkling. When aqueous cream is applied, it blocks the sweat pores on the skin, preventing the flow of sweat. Therefore, wrinkles would not appear on the skin when aqueous cream is applied (12).

Many physical aspects of the system influence the rate of sweat flow in the sweat duct when immersed in water. The surface tension of a sweat droplet, along with the secretory pressure of sweat gland, increases with an increase in sweat density. Therefore, increased tonicity of the sweat may generate more pressure within the duct. In this scenario, sweat rapidly depletes from the body, triggering skin wrinkling. AWP in cystic fibrosis (13) and Rofecoxib-induced AWP (14) may be caused by the increased tonicity of sweat, which generates more pressure and enables rapid loss of sweat when immersed in water. Hyperhidrosis, in these cases, also enables the faster depletion of sweat. It was observed that the time for wrinkling to occur was significantly slowed by an increase in tonicity of the water (15). Water with increased tonicity exerts more pressure on the sweat at the pore, slowing the flow of sweat.

It has been reported that the optimal temperature for testing skin wrinkling on hands is 40°C (16). Warm water stimulates the sweat glands and initiates the flow of sweat into the water similar to sweating in the proximity of hot environments, such as near a furnace. Unlike in hot environments, the loss of sweat within water will be more than is required for thermoregulatory purposes. Abnormal loss of water from the body triggers the vasoconstriction and eventual wrinkling of the skin. Warm water also exerts less pressure on sweat flow than cold water, enabling faster depletion of water from the body. It has been suggested that vasoconstriction in fingers immersed in water in which the temperature was gradually increased is a mechanism to reduce heat gain from the environment (17). An increase in blood flow when the hand was heated using hot air was reported as an objection to the 'heat gain' and vasoconstriction theory (1). In the case of warm water, excessive cooling in the form of increased sweating causes the vasoconstriction. If the heat gain to the fingers is more than the heat loss through sweating, then blood flow will increase to the fingers as observed with higher temperatures (17). Increased blood flow further increases the loss of sweat. In another case with hot air, blood flow increased to increase the evaporation of sweat for thermoregulation. A common experience of skin wrinkling in cold environments is due to excessive cooling and reduction of blood flow to the hand, not the loss of sweat. Therefore, vasoconstriction is a result of excessive cooling, not a means of reducing heat gain.

It is possible that depletion of sweat in denerved fingers continues when immersed in water. Blood flow increases to feed the excess generation of sweat in these fingers, causing vasodilation. Because of the non-functioning sympathetic nerves, vasoconstriction and skin wrinkling will be absent in the denerved fingers. Observation of vasodilation in denerved fingers (18) immersed in water supports the case for increased sweating. It has also been observed that EMLA cream causes a similar dilation when applied to denerved fingers (19). As described earlier, either sweat is absorbed by the EMLA cream or evaporates from the surface of the skin, causing vasodilation in denerved fingers.

Prolonged exposure to water causes dehydration and excessive cooling of the body due to loss of

sweat. The resulting reduction in blood flow limits the supply of oxygen and nutrients to the fingers. Small whitish plaques gradually expand to all of the exposed palmar skin and likely hinder manipulation of objects. Therefore, the process of wrinkling appears to be a protective mechanism of the body to maintain homeostasis. It is also possible to control the formation of wrinkles using water temperature, tonicity and the depth of immersion. The theory of wrinkles as an evolutionary advantage for primates handling objects in wet environments (20,21) seems implausible.

Conclusions

Vasoconstriction and skin wrinkling associated with water immersion indeed appears to be a fight or flight response of the body to maintain homeostasis. Analysis of the earlier observations and our calculations suggest that the free flow of sweat due to loss of surface tension at sweat droplets is the possible cause of WISW. Excessive loss of sweat in a resting body, due to the pressure imbalance between the secretory pressure of sweat glands and the pressure exerted by the water at the pore, results in excessive cooling of the body. To maintain homeostasis, the sympathetic nervous system triggers a reduction of blood flow to the hand, causing the vasoconstriction and eventual wrinkling of the skin. Other factors, including the temperature and tonicity of both the sweat and water, as well as the depth of immersion, also influence the formation of wrinkles.

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