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

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Abstract

Water immersion skin wrinkling appears to be the result of breaking the balance between secretory pressure of sweat glands and the pressure exerted by the surface tension of sweat droplet at the pore. When a hand is immersed in water, sweat droplet easily merge within the water causing pressure to drop at the pore. The resulted imbalance in pressure enables the sweat to flow freely into the water. Flow of sweat continues as long as there is a blood flow to hand. To prevent the loss of sweat from the body and to maintain homeostasis, sympathetic nerves trigger the reduction of blood flow to hand causing vasoconstriction. The overlying skin wrinkles due to loss of volume under the skin.

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

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Introduction

Water immersion skin wrinkling (WISW) is a well observed and documented phenomenon. Wrinkles appear on the skin if the hand is immersed in water for about 30 minutes. Surface of the skin reverses back to normal in about 10 minutes after removing the hand from water. The root cause of skin wrinkling, as observed by Wilder-Smith and Chow [1], is vasoconstriction and the reduction of blood flow to hand. Subsequent loss of volume under the skin results in wrinkling of overlying skin. Wrinkling of the skin is being used as a test for sympathetic nerve functioning [2-4] for a long period of time. However, the trigger mechanism that initiates the vasoconstriction remained elusive. In this article, we investigate a theoretical possibility for the trigger of such mechanism by taking earlier observations and different aspects of sweating into consideration.

Aquagenic wrinkling of palms (AWP) is a rapid formation of wrinkles with limited exposure to water. As the recent research indicates, AWP is occasionally associated with hyperhidrosis [5-7]. Kabashima et al. [6] reported that AWP, after 5 min of water immersion, was limited to iodine-starch positive areas; further suggesting, as one of the possibility, that the skin wrinkling is induced by hyperhidros following water exposure. Arkin et al. [7] observed an increase in transepidermal water loss, in the form of water vapor measured using Vapometer, immediately after skin

wrinkling in majority of cystic fibrosis subjects. Positive result for iodine-starch test and increased transepidermal water loss after immersion suggests the continuation of sweating in some cases even after skin wrinkling.

Existing Theory

There are many different theories on the cause of sympathetic trigger skin wrinkling. However, we will be discussing only the frequently referenced theory in this article. The leading theory on skin wrinkling [1,8] proposes that the skin absorbs water through many of the sweat ducts when the hand is immersed in water. The subsequent alteration of epidermal electrolyte homeostasis results in increased firing of the surrounding neurons. Increased firing of the sympathetic nerves results in the vasoconstriction and subsequent wrinkling of the skin due to loss of volume under the skin.

As indicated earlier, AWP is occasionally associated with hyperhidrosis. Due to excessive sweat during hyperhidrosis, sweat duct and sweat pore will be saturated with sweat. Any additional absorption of water into already saturated sweat duct needs to work against the pressure within the duct. The conditions, under which the AWP occurs, doesn't generate enough pressure to push the water into the sweat duct. Hyperhidrosis and the presence of sweat after skin wrinkling [6,7] leaves us with the assumption, according to the present theory, that sweat flows towards the pores before and after skin wrinkling but flows inward during immersion, which is very unlikely under the circumstances of AWP.

Even if there is a possibility of absorption of water and alteration of electrolyte homeostasis, that doesn't necessarily result in increased firing of neurons. There is no experimental evidence to support the triggering of sympathetic neurons via dyselectolytemia. Furthermore, skin wrinkling on hands sustains as long as the hands are exposed to water. It is not clear, in the existing theory, how the increased firing of neurons sustains over a long period of time. All these observations and analysis suggest the possible limitations to the theory.

New Hypothesis

Sweating appears to be a physical process involving the secretory pressure of sweat glands and the pressure exerted by the surface tension of sweat droplet at the pore. Sweat surfaces on the skin when the pressure within the duct is greater than the pressure exerted by the leading edge. As the sweat continues to flow, a droplet starts to form at the pore. Sweat droplet grows in size until the surface tension of the droplet and the pressure within the sweat duct are in equilibrium. If there is no further increase in the pressure within the sweat duct, no further sweat is formed; unless the sweat droplet is wiped off the skin, or evaporated. When the sweat droplet is wiped off the skin, a new droplet starts to form at the pore until equilibrium is reached again. A slow evaporation or reabsorption of sweat continuously gets replenished with new sweat by keeping the equilibrium all the time.

We could divide the process of sweating in a resting body into four different pressure components.

The secretory pressure of sweat glands (Pg), the lost pressure due to reabsorption of sweat (Pr), pressure lost due to partial evaporation of sweat (Pe) at the pore and the pressure exerted by the surface tension of sweat droplet (Ps). By ignoring the friction within the sweat duct, we could equate the Pg to the sum of all other three pressure components in an equilibrium as follows.

$$Pg = Pr + Pe + Ps \quad \ldots \quad (1)$$

It is important to note that the atmospheric pressure (Pa) also works against the Pg on the sweat droplet. It means, the secretory pressure of sweat glands, in absolute terms, is the sum of Pg and Pa together. If the pressure components Pr and Pe are absent then the sweat droplet quickly grow in size because of the increased pressure on the droplet. If the pressure component Ps also absent then the sweat freely flows outward. Schulz [9] observed that the secretory pressure of sweat glands (Pg) in cystic fibrosis patients and control subjects measured up to 500 mm Hg. The absolute secretory pressure of sweat glands, with the addition of atmospheric pressure of 760 mm Hg, will be 1260 mm Hg. The pressure exerted water at the sweat pore, when the hand is placed under water at a depth of one meter, is 74 mm Hg. The combined pressure exerted by the water column and the atmospheric pressure will be 834 mm Hg.

Pressure within the sweat duct increases due to the generation of new sweat at eccrine sweat glands during a period of intense physical activity. The increased pressure pushes the sweat outward. The droplets grow in size and merge with adjoining droplets to form as drops, eventually sliding off the skin due to increased weight. When the body is in rest, the pressure within the duct and the other three pressure components will be in equilibrium. In these conditions, when a hand is immersed in water, sweat droplet break apart and quickly dissolve within the water. Evaporation of sweat cease to exist because of immersion of hand in the water. Reabsorption of sweat also probably will disappear because of the absence of counteracting pressure to hold the sweat inside the duct. When all the three components Pr, Pe and Ps disappear, the sweat flows freely into the water. As shown above, there will be a pressure difference of 426 mm Hg at the higher end between the pressure at the sweat glands and the pressure within the water at a depth of one meter. Sweat glands continue to generate sweat due to the pressure gradient. Increased sweat flow possibly causes initial vasodilation to feed the excess generation of sweat. The loss of water content from the blood, as excessive sweat, continues as long as there is blood flow to parts around the sweat glands. To control the loss of water and to maintain the homeostasis, sympathetic nervous system triggers the reduction of blood flow to palmar region, causing vasoconstriction [1]. The skin wrinkles due to loss of volume under the skin. A higher end pressure difference of 426 mm Hg might be a case for AWP. Approximately, a depth of 6.8 m water column will be equal to the secretory pressure of sweat glands at 500 mm Hg. Therefore, skin might not wrinkle in subjects with 500 mm Hg secretory pressure when hands are placed under water at a depth of more than 6.8 m. At that depth, both secretory pressure and the water column pressure will be equal.

It is possible that skin wrinkles on the hand faster when placed in the flow of running water than in a stationary water of same temperature. Flowing water strips away the sweat droplets on the pores faster than dissolving in stationary water. Moreover, the remaining sweat in the duct flows faster

into the water flow because of the low pressure created in the flowing water. According to the Bernoulli principle of fluid dynamics, faster the movement of the fluid, smaller the pressure it creates in the flow. Sweat glands produce more sweat due to the low pressure within the duct. This, continuous process of generation of sweat at the glands and merger of sweat into the water flow at the pores, depletes the water content of the blood faster than keeping the hands in a stationary water. A rapid depletion of water and excessive vasoconstriction causes the skin to wrinkle faster than the stationary water.

Discussion

Sweat on the surface of the skin continuously evaporates by absorbing the heat from the skin. At the same time, eccrine sweat glands continuously generate new sweat, exerting pressure on the sweat droplet. The mean secretory pressure of sweat glands observed by Schulz [9] in cystic fibrosis and control subjects is 300 mm Hg. Even at a depth of one meter, the secretory pressure will be 200 mm Hg more than the pressure exerted by the water column. Excessive pressure within the sweat duct combined with loss of pressure at the pore, when immersed in water, can only result in the flow of sweat towards the pore. Dilation of sweat ducts in AWP, as observed by Neri et al. [5], suggests a rapid flow of sweat. Stationary water in a container at shallow depth can't exert enough pressure to create a rapid flow of water from the container into the sweat duct. Furthermore, it was observed that skin wrinkling occurs, in some instances, even when the hand is briefly exposed to water. A flowing water also can't exert pressure at the pore, but still we could see the wrinkles form under a running tap water.

Ma et al. [10] observed that topiramate reduced sweat secretion along with decreased aquaporin-5 expression by sweat glands in mice. Kabashima et al. [6] reported the excessive aquaporin-5 expression in sweat glands during AWP. Based on these observations, we suspect the excessive aquaporin-5 expression as a possibility for increased sweating during AWP. Increase in transepidermal water loss [7] immediately after skin wrinkling also points towards excessive sweating during the period of skin wrinkling. All these earlier observations, in conjunction with the proposed hypothesis, strongly suggest the case for flow of sweat towards the pores during skin wrinkling. The loss of sweat in a resting body triggers the vasoconstriction to maintain the homeostasis. Skin wrinkles due to vasoconstriction and loss of volume under the skin [1]. Wrinkles appear only on the palmar area because it has more sweat pores than any other part of the body [11]. More pores cause more sweat, and faster depletion of water from the body when immersed in water.

We could also possibly achieve the effect of skin wrinkling by dipping the hands into any material that could quickly absorb the sweat without letting it coalesce as a droplet at the pore. Wilder-Smith and Chow [12] observed that when vasoconstrictive cream, like eutectic mixture of local anesthetic (EMLA), is applied to distal digits, the degree of skin wrinkling was similar to the water immersion. EMLA cream possibly absorbs the sweat as soon as it forms at the pore. Sweat continues to flow at the rate of absorption of sweat by EMLA cream. Addition of sweat to EMLA cream either turns it into liquid form or gets evaporated by absorbing the heat from surface of the

skin. There is a possibility for excessive evaporation because the sweat occupies more space on the skin. To prevent the loss of sweat, body reacts by restricting the blood flow to the digits and causes skin wrinkling. It is important to note that aqueous cream blocks the sweat pores on the skin preventing the flow of sweat. Therefore, wrinkles wouldn't appear on skin when aqueous cream is applied [12].

Many physical aspects of the system influence the speed of sweat flow in the sweat duct when immersed in water. Surface tension of sweat droplet, along with the secretory pressure of sweat glands, increases with the increase in density of the sweat. Therefore, it is possible that increased tonicity of the sweat might generate more pressure within the duct. In this scenario, sweat rapidly depletes from the body if the hand is immersed in water, triggering skin wrinkling. AWP in cystic fibrosis [7,13] and Rofecoxib induced AWP [14] are possibly due to the increased tonicity of the sweat which generates more pressure and enables rapid loss of sweat when immersed in water. Tsai and Kirkham [15] observed the time taken to wrinkling as significantly slowed with the increase in tonicity of the water. Increased tonicity of the water exerts more pressure on the sweat at the pore, significantly slowing the flow of sweat.

Cales and Weber [16] reported that the optimal temperature for testing skin wrinkling on hands is 40^{o} C. Warm water stimulates the sweat glands and initiates the flow of sweat into the water similar to sweating in the proximity of hot environments like a furnace. Unlike hot environments, loss of sweat within the water will be more than normally required for thermoregulatory purposes. Abnormal loss of water from the body triggers the vasoconstriction and eventual skin wrinkling. Warm water also exerts less pressure on the sweat than cold water, enabling faster depletion of water from the body. Nagasaka et al. [17] suggested the vasoconstriction in fingers, when immersed in water of which the temperature was gradually increased, as a mechanism to reduce the heat gain from environment. Wilder-Smith and Chow [1] argued that the increase in blood flow when the hand was heated using hot air as an objection to the 'heat gain' and vasoconstriction theory. In the case of warm water, excessive cooling in the form of increased sweating caused 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]. In other case with hot air, blood flow increased due to lack of sufficient cooling mechanism at the hand. Therefore, vasoconstriction as a means of reducing the heat gain is not valid in all instances.

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 denereved fingers by Hsieh et al. [18], when immersed in water, supports the case for increased sweating. It was also observed that EMLA cream cause similar dilation when applied to denerved fingers [19]. As described earlier, either the sweat is absorbed by the EMLA cream or gets evaporated from the surface of the skin causing vasodilation in denerved fingers.

Prolonged exposure to water causes dehydration and expands the plaques to all of the exposed palmar skin. The resulting reduction in blood flow limits the supply of oxygen and nutrients to the fingers. Altogether, the process of wrinkling appears to be a protective mechanism of the body to maintain homeostasis. It is possible to control the formation of wrinkles using the temperature, tonicity and the depth of immersion. The theory of wrinkles as an evolutionary advantage as primate rain treads for handling objects in wet environments [20,21] seems implausible.

Conclusion

Analysis of the earlier observations suggest that the free flow of sweat due to loss of surface tension at sweat droplet, when immersed in water, as the possible cause of skin wrinkling. Other factors such as temperature and tonicity of both sweat and water, as well as the depth of immersion also influence the formation of wrinkles. The work presented in this paper coherently explains most of the observations. Due to the simplicity and testable nature of the hypothesis, it might emerge as a viable alternative to the existing theories in describing the underlying mechanism of water immersion skin wrinkling.

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