Respiratory Influence on Skin Potential and Sweating (33710)

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(Introduced by C. M. Brooks)
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The existence of spontaneous fluctuations in D-C potential across the skin, occurring synchronously in the four paws of the cat have been described by Wang (14). He also noted the temperature dependence of such potentials (15), but did not directly demonstrate an increased sweat gland activity associated with the increased electrical activity of skin. Since it was stated, but not actually shown that such potentials are also present in human skin, I undertook this investigation to determine (a) whether or not spontaneous fluctuations of D-C potential do in fact occur in human skin, and (b) if they exist, to determine their relationship to sweating. The results of the experiments described below indicate that synchronous fluctuations in the potential of human skin do occur, and moreover, that such potentials are temperature dependent and can be correlated with changes in the rate of sweating. An unexpected finding, however, was that of the influence of respiration on both skin potential and sweating.

Methods. A total of 25 experiments were performed on two human subjects. In each experiment the subject's sweat rate, skin electrical potential, respiratory rate, and digital volume was continuously measured over a 5-hr period while he was exposed to a hot, cold, or neutral environmental temperature. Each experiment was begun at a neutral temperature with the subject sitting fully clothed in a room having a minimum of extraneous influences. Air temperature in the room was then adjusted to remain at levels between 21 and 39° while the subject's responses were measured. The actual order of heating or cooling for each experiment was deliberately made random so as to minimize conditioning. Air was continuously circulated through the room by means of an electric fan.

Electrical activity of skin. A pair of silver–silver chloride electrodes, 5 mm in diameter, were placed on the back of the hand and palm of the same hand (Type I recording), or on the back of the hand and forearm of that hand (Type II recording). The Type I and Type II pairs were then connected to separate Grass D-C amplifiers, the output of each giving the difference in potential between two active skin sites. In order to secure good and constant contact between electrodes and skin, the electrodes were not permitted to touch the skin directly, but instead, were separated from it by hollow cardboard spacers that had been filled with Grass EEG paste. The spacer-electrode assembly could be firmly taped to the skin and thus provided a fixed low resistance path between electrode and skin surface. At frequent intervals during an experiment, electrodes were moved to adjacent skin sites so as to minimize the occurrence of polarization potentials and to provide a check on the reproducibility of existing skin potentials.

Skin sweating. Static "prints" of palmar sweat gland activity were made by Randall's (11) method, while Wada's (13) method was used for the forearm. In some experiments atropine sulfate, 40 mg/100 ml, was iontophoresed (Teca Corp., model P6-1) into the skin directly beneath the electrodes. Subsequent sweat prints taken at these sites verified the resultant inhibition of sweating.

Dynamic changes in sweat rate were measured by means of a modified electric hygrometer (3). The instrument's transducer was transferred from its original wire mesh housing to a polystyrene chamber which measured $2 \times 1 \times 1$ cm and was open on one side. The open side was applied firmly to the skin surface. Care was taken to minimize the dead

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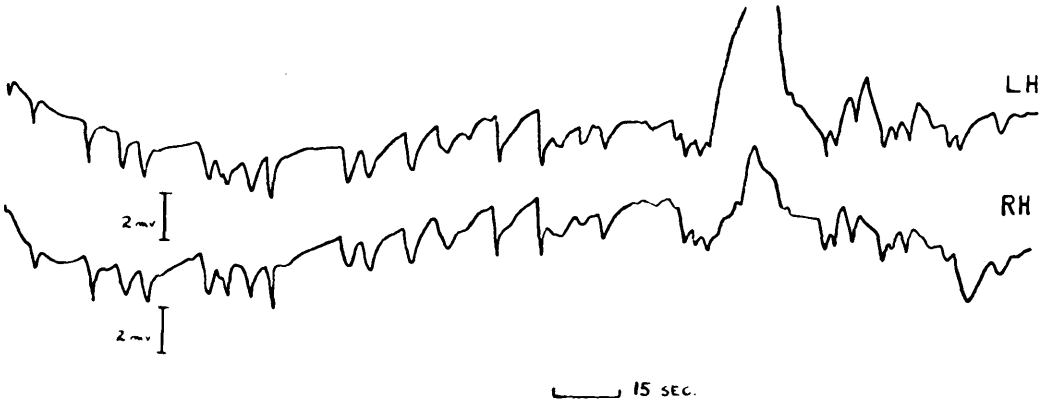


FIG. 1. Type I recordings from left (LH) and right (RH) hands; see text for further details.

space through which water molecules had to traverse in order to reach the transducer element. To further reduce the time lag of the instrument, oxygen was circulated through the chamber at a steady rate of 3 liters/min. The response time of the transducer thus modified was on the order of 3 sec. For the purpose of these experiments it was sufficient to record relative rather than absolute changes in water vapor lost from the skin.

Digital volume and the chest wall excursions associated with respiration were monitored by means of a mercury in silastic tubing strain gauge (8). Skin potential, sweat rate, digital volume, and respiratory excursions were all recorded simultaneously on a Grass model 1 Polygraph.

Results. Presence of skin potentials. In all experiments, at all environmental temperatures, spontaneous fluctuations in skin potential were found to be present in type I recordings. Furthermore, type I recordings made simultaneously from both hands showed a striking similarity between the two records (Fig. 1). Typically, one saw "rapid" changes in potential, each of about 3-sec duration, superimposed upon "slow" changes of about 2–4 min in duration. The magnitude of these potentials was approximately 2 mV, with the palm negative with respect to the back of the hand. In a cool environment, type I and type II recordings made simultaneously from the same arm showed that the above potentials were present in the type I record but not in the type II record. Since the difference between the two forms of recording was

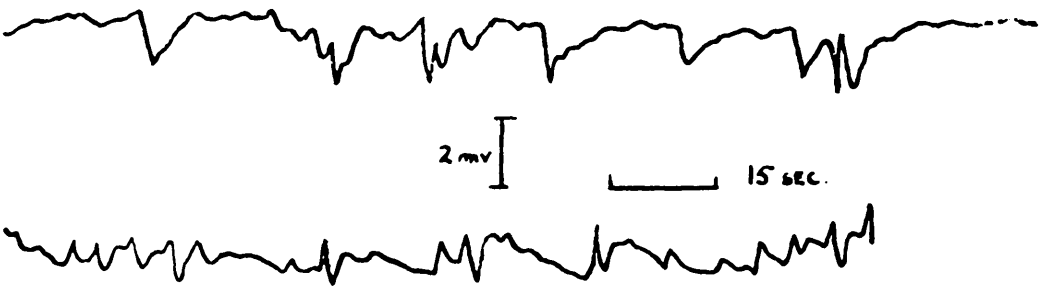
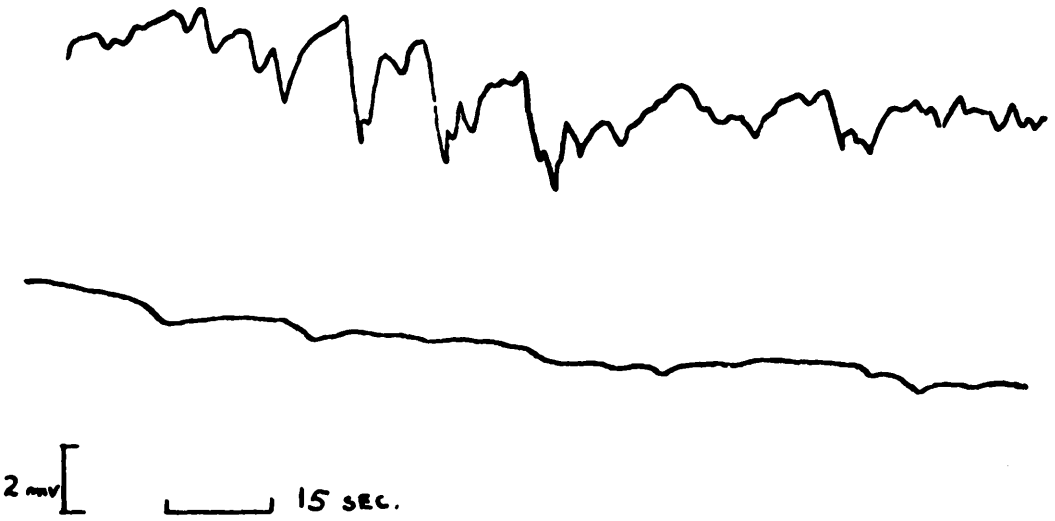


FIG. 2A. Type I recording in a cool environment before and after atropinization of skin: (top) before atropine; (bottom) after atropinization of skin under electrode on dorsum of the hand. (B) As in (A), but with atropinization of skin under palmar electrode. Note that spontaneous skin potentials were abolished by atropinization of the palm but not by atropinization of the dorsum of the hand.



merely the location of one of the electrodes, it followed that palmar skin was giving rise to the observed potential changes. This hypothesis was tested by separately atropinizing dorsal and palmar skin and noting the effect on a type I recording. Figure 2 shows that atropinization of the skin under the electrode on the dorsum of the hand had no effect on the appearance of the skin potentials, while atropinization of skin under the palmer electrode abolished these potentials completely.

To test the hypothesis that the potentials were a manifestation of changes in skin blood flow, circulation through the arm was temporarily occluded by means of a sphygmomanometer cuff. From the record shown in Fig. 3, it can be seen that the spontaneous potentials persisted, undiminished, in spite of stoppage of blood flow through the arm.

The above findings indicate that in a cool environment with minimal activity of the dorsal "thermal" sweat glands (9), palmar skin nonetheless shows spontaneous changes in potential which, since they are independent of regional blood flow changes and are abolished by the antisudorific agent atropine, most likely arise as the consequence of palmar sweat gland activity.

Influence of respiration. No consistent relationship could be found between the frequency of the spontaneous skin potentials and the frequency of quiet respiration. When the subject sighed, however, or otherwise markedly increased the depth of his breathing, a pronounced change in skin potential occurred together with an increase in sweating (Fig. 4). This change in potential typically consisted of (a) a diphasic wave appearing within 3 sec of the initiating deep breath, (b) a pronounced positive change in

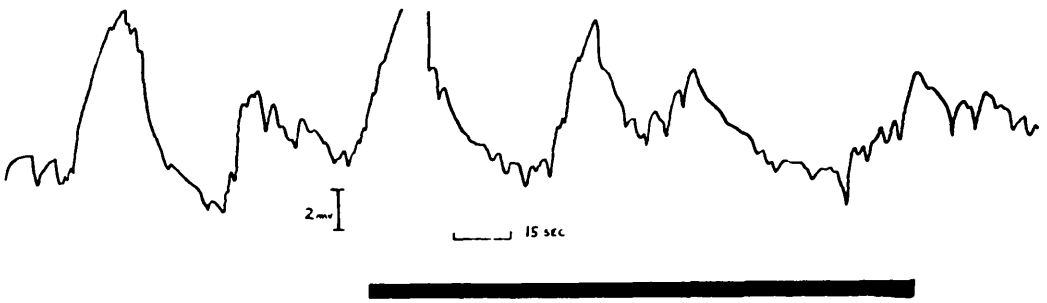


FIG. 3. Type I recording: showing the presence of skin potentials before and during inflation of a cuff around the arm to 180 mm Hg pressure; bar indicates the duration of inflation.

FIG. 4. Sweating and skin potential as influenced by the taking of a sudden deep breath: type I recording, with the sweat transducer on the subject's palm; arrow indicates the taking of the breath.

skin potential lasting about 1 min, and (c) disappearance of the rapid fluctuations in skin potential, with their reappearance at roughly the same time as skin potential returned to its former negative level. In Fig. 5 seven such responses were plotted from a typical experiment. It can be seen that the positive change in skin potential following a deep breath was most pronounced when the preinspiratory level of potential was most negative. (It should be recalled that periodically the skin became more negative due to the

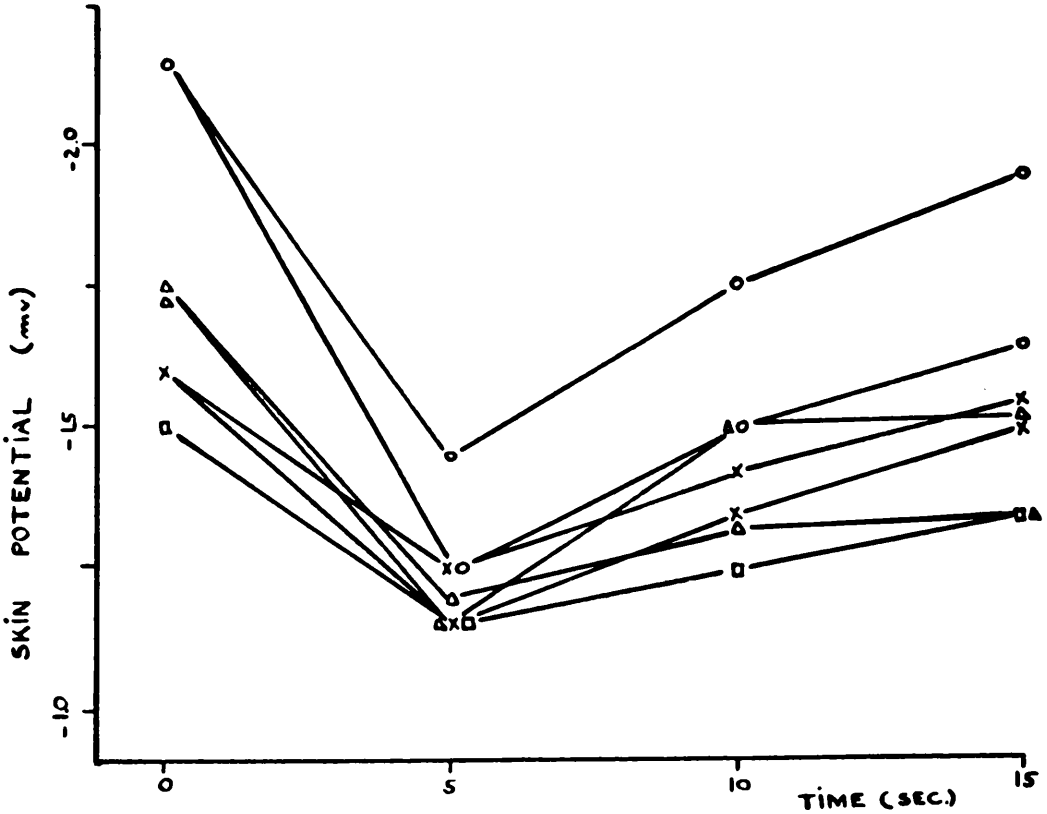


FIG. 5. Skin potential vs time in a subject initially breathing quietly: type II recording; graph shows the effect on skin potential of taking a deep breath (time zero); points are discrete values of skin potential measured immediately before and 5, 10, and 15 sec after the deep breath.

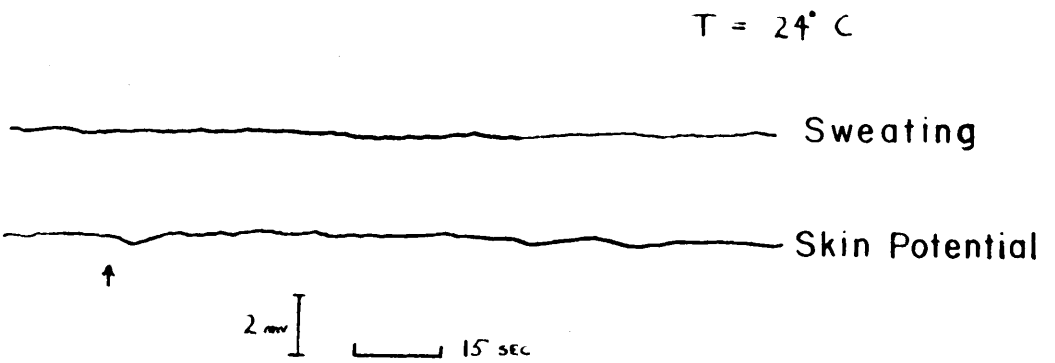
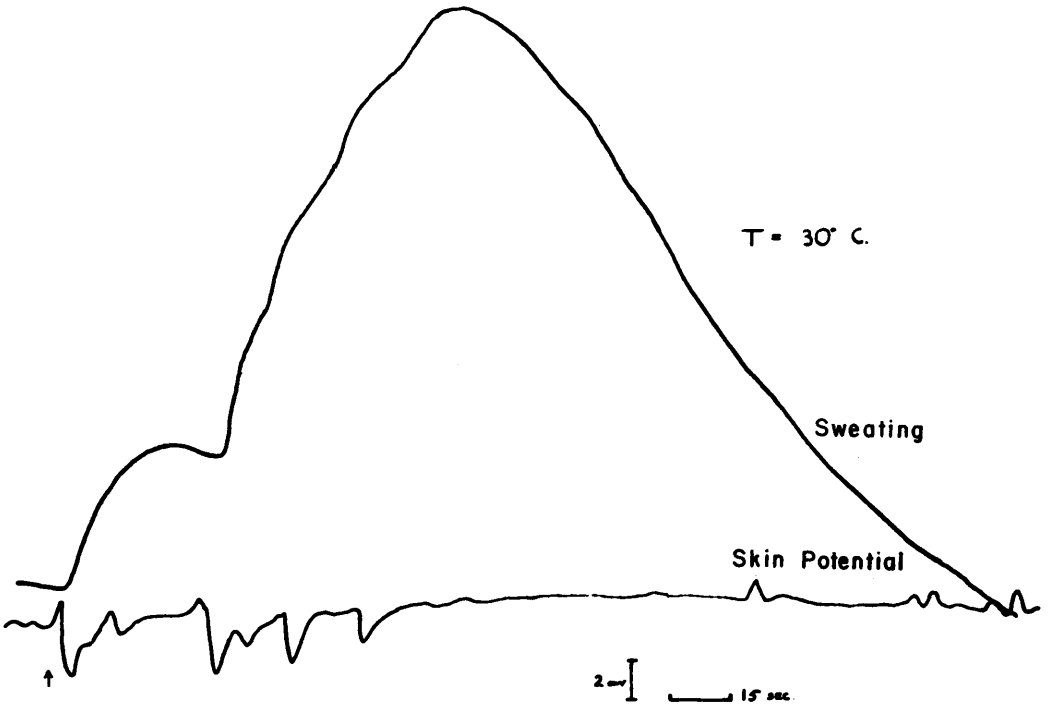


FIG. 6A. Sweating and skin potential in a cold environment: at the arrow the subject took a deep breath; type II recording; sweat transducer on dorsum of the hand. (B) As in (A), but room was warmed to 30°.

"slow" waves previously described.) Thus, the relationship between deep breathing and skin potential was such that the most pronounced effect of a single deep breath occurred when skin potential was at its most negative value. Although active sweating was going on, it was not possible by either the sweat print or the electrohygrometer technique to demonstrate definite cyclical changes in sweating while the slow changes in skin potential were taking place.

Influence of temperature. Fast and slow potential waves were always present in palmar skin, independent of the level of the environmental temperature. However, in the case of forearm skin or the skin of the back of the hand, spontaneous potential waves were forthcoming only when the environmental temperature reached the high end of the neutral range or beyond. Figure 6A and B shows a portion of an experiment in which a subject was exposed to two different environmental temperatures. In the cool environment, spontaneous changes in skin potential



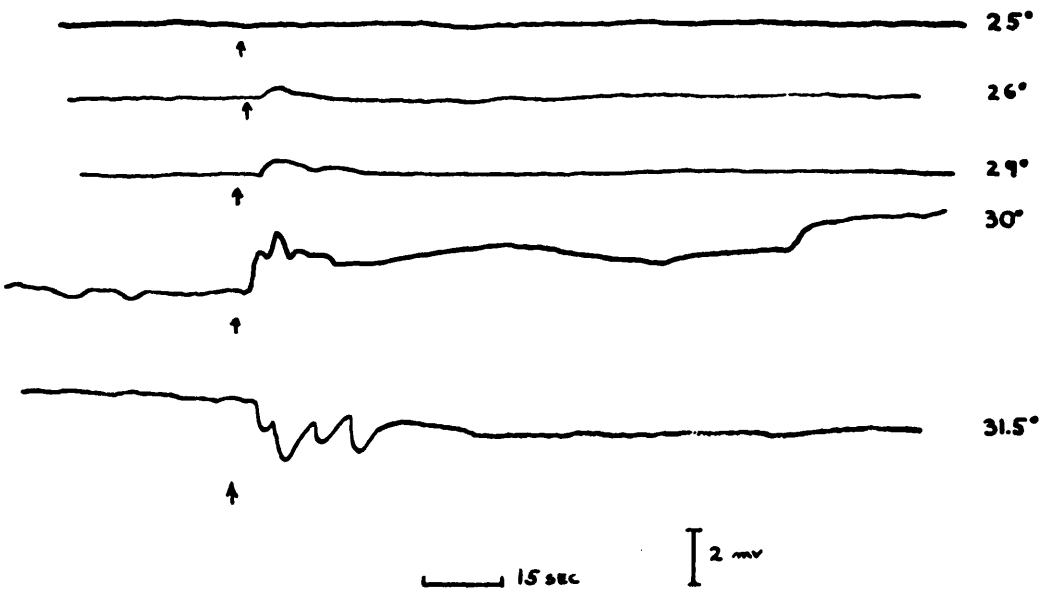


FIG. 7. Respiratory effect on skin potential, as influenced by the environmental temperature: each tracing shows a type II recording of the subject's skin potential, made at different room temperatures; at the arrow in each tracing a deep breath was taken. Note that the influence of deep breathing on skin potential depends upon the level of environmental temperature.

were almost completely absent, and sudden deep single breaths caused no detectable change in sweating. Thirty min later when the room had been warmed to 30°, skin potentials were present and deep breaths caused a marked increase in the rate of sweat production. This "threshold" effect of temperature on skin potential is also shown in Fig. 7, in an experiment performed on a different subject. Each tracing shows a type II recording made as room temperature was progressively increased from 25 to 31.5°. Spontaneous potential waves are not seen until a temperature of 30° is reached; however, a definite influence of deep inspiration–exhalation on skin potential can be seen beginning at 26°, and the magnitude of this response increases with increasing ambient temperature thereafter. It is interesting to note that in this experiment the "typical" positive deflection in skin potential in response to a deep breath was seen only at the highest temperature, while at lower temperatures a negative deflection was the response; yet in both cases an increase in sweating occurred. The complex nature of the relation between skin potential and sweating is borne out by the fact that positive, negative and diphasic skin potentials have been repoted to occur in association with an increase in sweat gland activity (17). In general, I have observed that initially, forearm skin potential gradually becomes more negative as room temperature increases and as the degree of sweating becomes more apparent. This increase is potential soon ends, however, and skin potential then remains relatively fixed in spite of further increases in sweat rate in response to the rising ambient temperature.

Influence of inspired CO_2. In a hot environment, hyperventilation by the subject resulted in an outburst of sweating that was greater and more prolonged than that seen when the subject took only a single deep breath. Following this period of hyperventilation, sweating gradually returned to its control value. To test whether this effect was in some way associated with changes in blood gas concentrations, the same maneuvers (single deep breath, hyperventilation, etc.) were performed with the subject breathing a 5% CO_2–95% O_2 gas mixture. Switching from room air to breathing the gas mixture, or once equilibrated, vice versa, had no consis-

tent effect on sweat production or skin potential. When the subject took a deep breath after a 5-min equilibration period of breathing the gas mixture, an outburst of sweating resulted which closely resembled that seen when room air was breathed. Similarly, when the subject hyperventilated while breathing the gas mixture, a prolonged outbreak of sweating resulted.

Discussion. The present investigation showed that spontaneous fluctuations in potential occur in the skin of both hands, in a synchronous fashion. This is in accord with Wang's (14, 15) description of spontaneous, synchronized "potential waves" in the pads of the cat paws, and it confirms his assertion that such waves exist in man.

The results suggest that sweat gland activity is responsible for these fluctuations, yet I find no simple relationship between the form of the potential waves and the degree of sweating. It is well established, however, that monophasic and diphasic potentials do arise at the skin's surface as the consequence of sweat gland activity (6, 12, 16–18). Although the matter has been actively investigated, it would seem that the mechanism responsible for the generation of such potentials is still the subject of controversy (12, 16).

I found that deep breathing affects sweat gland activity, and since this effect is independent of skin circulation and is abolished by atropine, I concluded it to be mediated by a neural mechanism.

A phenomenon, perhaps related to the above, is the cutaneous vasoconstriction which followed the taking of a deep breath (4, 5, 7) or hyperventialtion (10). Bolton *et al.* (4), and Carmichael *et al.* (5) concluded that this effect also was mediated by a nervous pathway, whose afferent route lay within the thorax and whose efferent route traversed the peripheral sympathetic nerves. Gilliatt *et al.* (7) believed the inspiratory vasoconstriction to be purely a spinal reflex, while Carmichael and his group believed that central integrative mechanisms also played a role. Recently, it was suggested that vagal pulmonary stretch receptors may form part of the afferent route (10). One would expect that the phenomenon of cutaneous vasoconstriction, consequent to the taking of a deep breath, would change the skin's electrical resistance but such an effect cannot explain the fluctuations in skin potential found in the present study because such potentials were forthcoming even when the limb circulation was entirely occluded. This is consistent with the fact that short-term changes in the blood supply to active sweat glands do not alter their rate of secretion (9). Carmichael *et al.* (5) did find a change in electrical resistance of the skin following a deep breath and concluded that this effect was due to two separate events: cutaneous vasoconstriction and an increase in the rate of sweating. With regard to the former, I have not found cutaneous vasoconstriction to be an invariable consequence of deep breathing; rather, I observed it most often when subjects were in a cool environment and (presumably) some degree of vascular tone was already present. Insofar as one is permitted to relate changes in skin resistance with changes in potential and sweating (1, 17), the results of this study are consistent with the findings of Carmichael *et al.* in that a subject must first be warmed to the point of active sweating before a deep breath will have any effect on the rate of sweat secretion.

Albert (2) reported that hyperventilation diminishes sweating in the skin of the forehead, while in this study hyperventialation increased sweating in the forearm and back of the hand. The difference in results may lie in the chosen skin sites, since the latter two areas are known to respond primarily to thermal stimuli whereas forehead sweat glands can respond both to thermal and emotional stimuli (9). In addition, a quite different interpretation of Albert's data is possible; his Fig. 2 shows that initially in response to hyperventilation, sweat output in both of his subjects rose considerably before it decreased to below the control level. In one subject this increase lasted for about 1 min of hyperventilation and in the second subject it lasted for virtually the entire 3 min period of hyperventilation. An increased sweat rate also occurred when his subjects hyperven-

tilated while breathing a 5% CO_2–95% O_2 mixture in place of room air, as it did in our subjects.

Summary. The D-C potential at the surface of human skin has been measured in sweating and nonsweating subjects. In nonsweating skin a steady D-C potential was present which changed to a rapidly fluctuating pattern when the skin began to sweat actively. These fluctuating potentials occurred synchronously in the skin of both arms. The potential changes were independent of local hemodynamic events and could be abolished by atropine; which suggests they are a manifestation of sweat gland activity. An influence of breathing on skin potential and the rate of sweating was also found. In response to deep breathing, skin potential transiently became more positive while sweat output increased. These respiration-induced changes were not caused by a transient hypocapnia and could be observed only in skin that was already sweating actively.

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