Sweat suppression by forced breathing in man

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ALBERT, ROY E. *Sweat suppression by forced breathing in man.* J. Appl. Physiol. 20(1): 134–136. 1965.—Thermal sweating from the forehead was suppressed by forced air breathing in two normal male subjects. The decreased sweat rate was associated with symptoms of respiratory alkalosis. This sweat suppression was blocked by the introduction of excess carbon dioxide into the respired gas.

sweating and hyperventilation hyperventilation and sweating respiratory alkalosis and sweating

THIS PAPER describes the suppression of thermal sweating from the forehead that can be induced by hyperventilation of air. This effect was observed by chance during previously reported studies on the pulsatile character of sweating (1). The author, acting as subject, inadvertently hyperventilated to the point of dizziness while watching the continuous recording of his own sweat rate. It was noted that the reduction in sweating accompanied the symptoms of hyperventilation alkalosis both in regard to timing and magnitude.

The experiments described below were done to verify this observation and to determine whether it is a manifestation of respiratory alkalosis.

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METHODS

The technique for measuring sweat rates from small skin areas with an infrared gas analyzer has been described in detail (1, 3). Dry oxygen at a flow rate of 3 liter/min is passed through a cup held against the skin, and then to the infrared gas analyzer. The cup is 5 cm deep and encloses a 15 cm^2 skin area. The water vapor

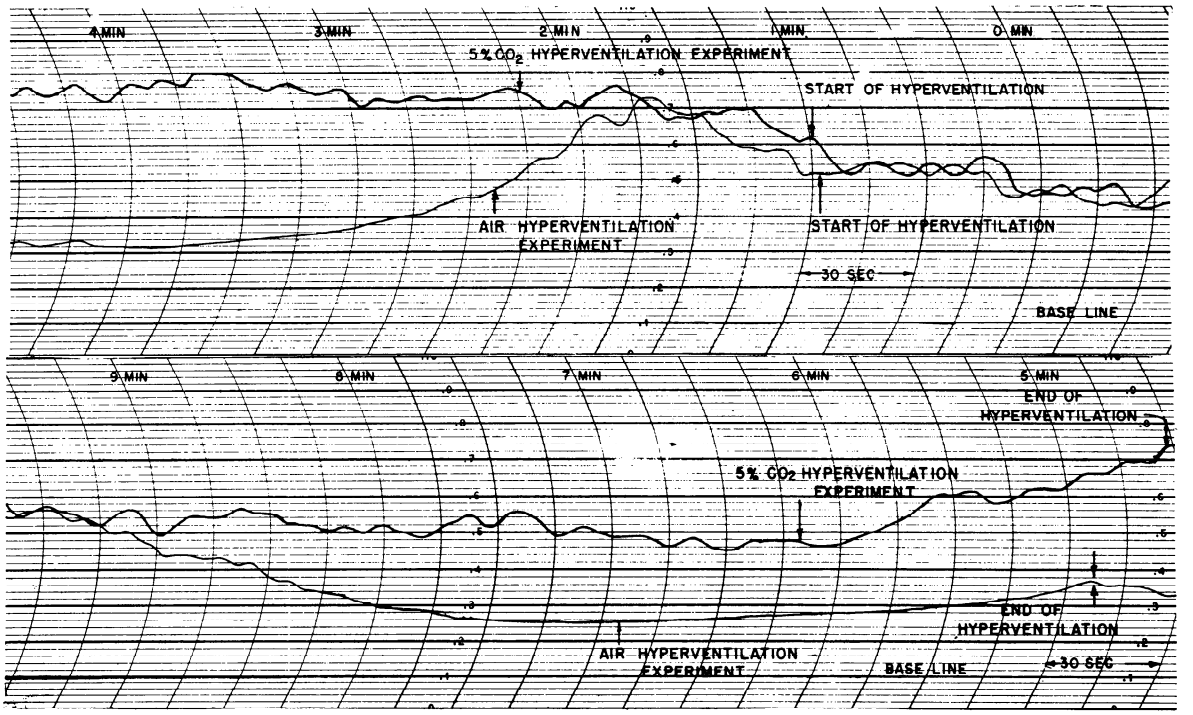

FIG. 1. Composite tracing of sweat rates from one subject during and after hyperventilation of air and oxygen plus carbon dioxide. The units on the ordinate are 0.025% water vapor per division; i.e., full scale deflection is equal to 2.5% water vapor concentration.

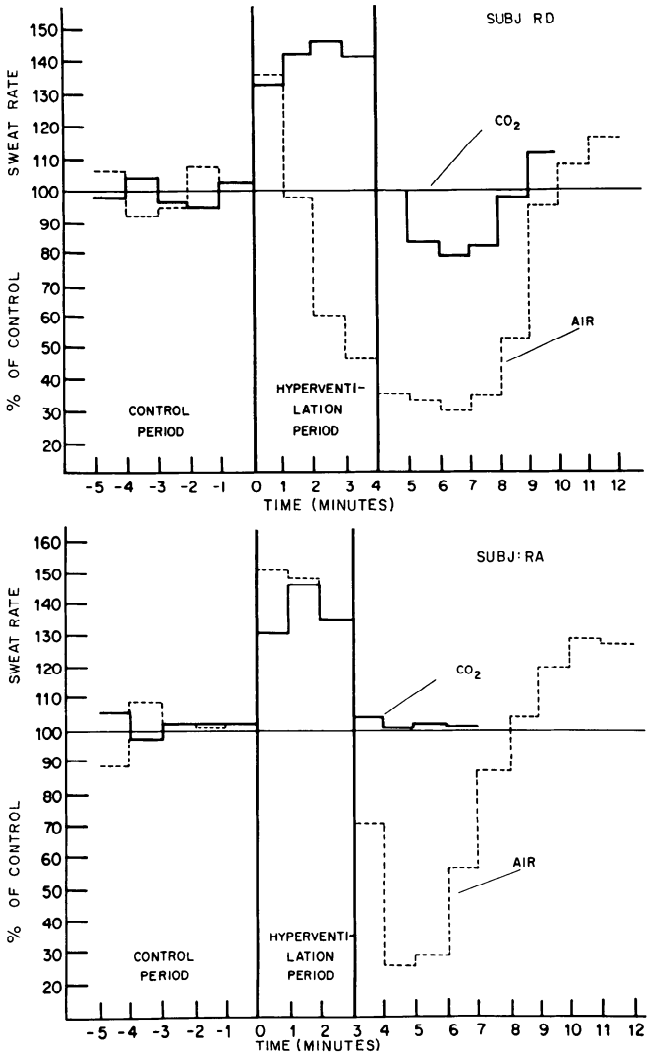

FIG. 2. Averaged results of all hyperventilation experiments on each of two subjects.

content of the oxygen is measured and recorded continuously and reflects the discharge rate of the sweat glands within the cup.

Sweat rates were measured on the foreheads of two healthy males, in their twenties, while reclining on a cot after an equilibration period of .5 hr to ambient conditions of 105 F and 20% relative humidity.

Each subject underwent a series of hyperventilation periods which involved breathing 170 liters of gas from a Tissot spirometer with rapid and deep respirations. A recovery period of 15–20 min duration was sufficient to restore the sweat rate to normal levels for at least 5 min.

The spirometer was alternately filled with room air and 95% O_2 + 5% CO_2. One subject (*RD*) underwent 9 periods each of air and O_2 + CO_2 hyperventilation and the other subject (*RA*) had 10 breathing periods of each gas mixture.

The sweat rate recordings were integrated planimetrically for consecutive 1-min intervals. For each breathing experiment, the average sweat rate for the 3- to 5-min control period was normalized and the 1-min consecutive average sweat rates were expressed as percentages of this control rate.

RESULTS

The sweating response from the forehead to forced hyperventilation of air was qualitatively the same for both subjects in every experiment.

There was an initial transient rise in the sweat rate beginning seconds after the start of hyperventilation; this was followed by a marked drop in sweating either during hyperventilation itself or in the subsequent apneic period. Recovery of sweating was associated with another overshoot.

When 5% CO_2 + 95% O_2 was hyperventilated, the initial transient rise was also observed, but the sweat suppression was either absent or much attenuated.

Figure 1 is a composite sweat-rate tracing from a typical pair of breathing experiments on the same subject.

There were quantitative differences in response of the two subjects. *RD*, on the average, took longer than *RA* to breathe the 170 liters (3.0 and 3.8 min, respectively). *RD*'s initial increase in sweat rate was of shorter duration and his sweat suppression phase was longer. The duration of the hyperventilation periods for the CO_2 + O_2 runs averaged 3.2 min for *RA* and 3.6 min for *RD*.

Figure 2 shows, for each subject, the averaged results of all the breathing experiments.

DISCUSSION

It was noted during the course of the earlier studies (1) that sweating from the forehead, in terms of its average rate or pulsatile character, is not affected by short periods of voluntary apnea during the course of normal breathing or by consciously induced irregularities in the breathing pattern that do not significantly increase the respiratory minute volume. However, sudden onset of vigorous muscular exercise, associated with forced breathing, is invariably associated with a quick rise in thermal sweating from the forehead. The same reaction is seen when the subject is startled. The relationship of anxiety reactions to sweating in areas of skin innervated by the sympathetic nervous system (palms and forehead) is well known, but these studies indicated that a similar response is produced by sudden muscular effort without any associated anxiety.

The presence of 5% carbon dioxide in the respired gas markedly reduces both the mental symptoms of respiratory alkalosis and the suppression of sweating associated with forced breathing. This indicates that reduction of sweat gland activity is one of the manifestations of respiratory alkalosis, as is the decrease of cerebral blood flow (2). It is not clear whether the sweat suppression is a consequence of depressed central nervous system function or is due to a direct effect of diminished carbon

dioxide tension on sweat glands. Although respiratory alkalosis is associated with diminished skin circulation, previous studies (1) indicate that blockage of the skin circulation produces only a delayed and slow reduction of sweat gland output.

The "hyperventilation syndrome" is one of the commoner anxiety reactions encountered in the general population and has also been observed in a particularly severe form (frank tetany) in soldiers under experimental conditions of severe heat stress (D. Bass, personal communication).

This raises the question of whether the sweat suppression of respiratory alkalosis is a contributory factor in heat stroke.

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