Diffusion of water vapor through human skin in hot environment and with application of atropine[1, 2]

KONRAD J. K. BUETTNER AND FREDERICK F. HOLMES
Departments of Meteorology and Climatology and of Physiology and Biophysics, University of Washington, Seattle, Washington

BUETTNER, KONRAD J. K. AND FREDERICK F. HOLMES. *Diffusion of water vapor through human skin in hot environment and with application of atropine.* J. Appl. Physiol. 14(2): 276–278. 1959.—At room temperatures between 20° and 40°C, vapor transfer through skin of human forearm was tested with four small heated bottles containing air of humidities ranging from 2 to 100% relative humidity. Exposure times ranging from 30 to 120 minutes had no influence on results. Water loss or gain of skin were observed for the different bottles. At very high humidities, liquid water deposit on the skin was measured by weighing a blotter. Skin vapor loss decreases systematically when bottle moisture increases. This increase is enhanced at room temperatures above 24°C, where total loss into a dry bottle increases more than fivefold. This increase seems only partially caused by sweat and partially by a decrease of the skin diffusion resistance. Tourniquet and locally applied atropine did not affect vapor transfer in a cool room. In a hot room, the tourniquet lowered the vapor loss by only 20%, whereas atropine drastically curtailed vapor loss.

WHEN HEAT, FEVER, EXCITEMENT OR WORK activates the sweat glands, the skin loses more water than in normal conditions. This increase of water loss is generally assumed to be identical with the sweat flow. In doing so, we tacitly assume, however, that the diffusive water exchange of nonsweating skin and sweating skin is equal. This assumption needs testing.

The crucial factor involved is the skin diffusion resistance R_s (1–4). According to an earlier report (1), the R_s of nonsweating skin is higher in locally dry than in locally moist air. R_s is also higher in skin immersed in brine than in that immersed in water. Since the outer milieu can change R_s, the inner milieu might also. This effect was already indicated in water exposure tests (1, 2), more water being taken in by feet exposed to water while active or warm than by feet at rest. This finding indicates a lowering of R_s by inner factors such as skin temperature and blood flow.

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[2] Contribution No. 40, Department of Meteorology and Climatology, University of Washington, Seattle.

METHODS

Tests with the heated four-bottle technique described before (3) were conducted in hot rooms. Four of these bottles were concurrently attached to the inner side of the forearm, usually for 30 minutes. Many of the tests were repeated, immediately, 2–3 times. Tests with bottle temperatures above skin temperature were also conducted in normal-temperature rooms. The room vapor pressure was generally about 7 mm Hg.

The bottle temperatures were higher than in our previous tests (3), namely, between 38° and 47°C. The local skin temperature was recorded with an attached thermocouple stretched over one of the four bottles. Condensation water deposit on the skin, if any, was sorbed in a piece of blotter and then weighed.

In some tests a tourniquet exerting 200 mm Hg pressure was applied above the elbow for up to 25 minutes. When used, atropine sulfate in a 1% solution was applied as a wet pack on the arm for a 2-hour period before the bottle test.

Two women and six men participated as subjects. They wore bathing suits in the hot room, which they entered at least 1 hour before the test.

RESULTS

The tests, the results of which are shown in figure 1, were conducted with bottle temperatures between 40° and 47°C and bottle humidities between 2% and 100%. At high bottle humidities, the skin gained water by diffusion, corneum sorption and liquid deposit. The data in figure 1 are not corrected by exclusion of the liquid water deposit which becomes increasingly marked above 50 mm Hg bottle vapor pressure. The room temperatures are written on the transfer curves. Skin temperatures were around 35°C and were always below bottle temperature. There is practically no correlation between the skin temperature and the room air temperature. This refers, however, only to the temperature of

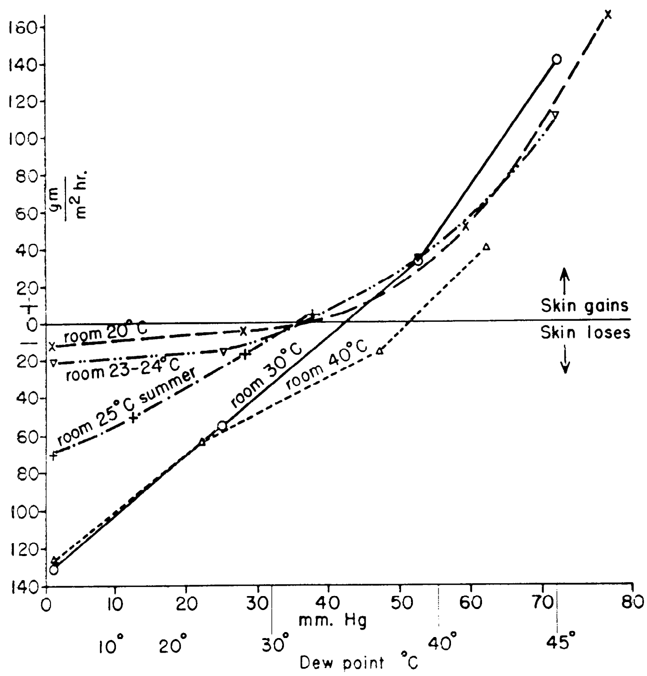


FIG. 1. Forearm skin vapor exchange from small heated cups containing different drying or wetting agents at their bottom. *Abscissa:* vapor pressure in cup (mm Hg) and dew point (°C). *Ordinate:* skin loss or gain in gm m^{-2} hr^{-1}. Above 50 mm Hg cup vapor pressure a substantial, but unmeasured, liquid deposit could be observed. Values on curves are temperature of chamber. Most tests were run in the cool season with the exception called 'summer.' Bottle or cup temperatures were 42°C in 40° room; 45°C in 23–24° and 30° room; 47°C in 20° room.

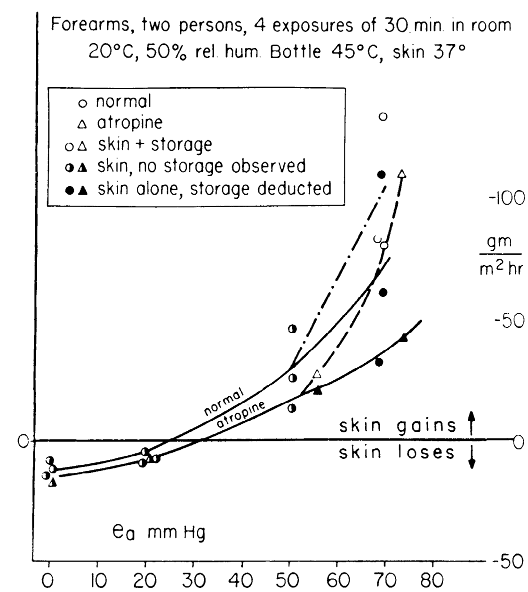


FIG. 2. Forearm skin vapor exchange from small heated cups containing different drying or wetting agents at their bottom. Normally clad people in a cool room. *Abscissa:* vapor pressure in cup. *Ordinate:* skin loss or gain. Circles: normal arm; triangles: atropinized arm. Half filled circles or triangles: no liquid deposit present at end of exposure. Open circles or triangles: transfer including liquid deposit. Filled in circles or triangles; net transfer through skin, i.e. observed transfer minus liquid deposit. Normal and atropinized arm were usually exposed concurrently.

the small skin areas exposed to the rather warm bottles.

General body sweat was observed when the room temperature was 30°C or higher. At 25°C sweat was never seen on the arm.

Each point in figure 1 is the average of about four series, each consisting of one to three consecutive 30-minute tests. No significant change of transfer between consecutive tests was seen.

In spite of bottle temperatures about 10°C higher than usual (1,3), transfers in the 20°C room (fig. 1) were the same as those recorded when the bottle temperature was the more usual 35°C (described elsewhere) if they were related to the same bottle vapor pressure. With increasing room temperatures, however, vapor losses into dry bottles increased gradually. This increase started under conditions in which no sweat could be detected with any means. This seems to show that this increase is only partly due to sweat, partly, however, caused by a lowering of skin resistance. Only ⅓ of the observed loss in a hot room seems to be sweat.

Up to 25°C room temperature, the point of no transfer or neutral point was constant at 35 mm Hg, but shifted to higher humidities when the room became warmer than 25°C. This shift was obviously caused by sweat.

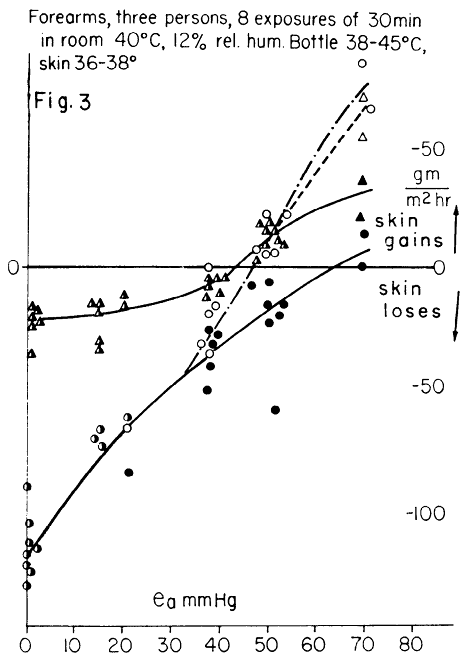
The 20°C and 40°C series were repeated (fig. 2, 3) with emphasis on liquid water deposit on the skin. Deducting this liquid water results in net transfer data, shown as filled in circles in figure 3. Liquid storage was restricted to humid bottles and no liquid water could be found on skin exposed to lower humidites. Consequently, all data in figure 1 for 30 mm Hg and less are true net transfers. Generally, the curve coincides with the 40°C room curve of figure 1.

The blotting technique was also applied, using hot bottles in a normal room (fig. 2). Deducting the liquid deposit yields net transfer data (half filled and filled circles in fig. 2). The scatter of data at 70 mm Hg is substantial, but a net inflow exceeding 50 gm m^{-2} $hr.^{-1}$ seems certain.

Tourniquet and atropine. These tests were also conducted in 20° and 40°C rooms with bottle temperatures of about 40°–45° C. The opposite arm was used as control, yielding normal data as discussed above (figs. 2, 3).

Tourniquet tests at 20°C showed no significant change. In a hot room, vapor losses from the arm to which the tourniquet was applied were about 30% less than for the normal arm. Sweating of the entire forearm was seen on the normal arm and stopped abruptly when a tourniquet was applied. The observed reduction in vapor loss (fig. 4) coincides approximately with the sweat rate mentioned above. This similarity might, however, be fortuitous.

In a 20°C room, atropine had no significant influence on the vapor transfer (fig. 2). At 40°C the atropine effect was profound (fig. 3).



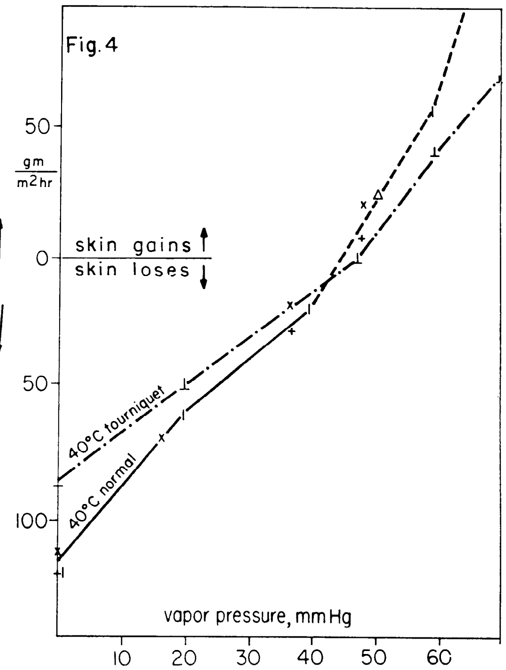


FIG. 3. Same as fig. 2, but hot room, people wearing bathing suits.
FIG. 4. Same as fig. 3, but one arm with tourniquet.

DISCUSSION

Temperature effects. The increase of water loss from the skin at higher room temperatures might result from an increase in sweat secretion or from an increase in vapor diffusion. The fact that sweat could not be observed shows only that local sweat secretion was either spread out into the adjacent horny layer or had evaporated before it could form a visible tiny water pool or was totally absent.

Sweat secretion, however, is not supposed to depend on local skin humidity. Principally, a sense of skin wetness could exist, but nothing is known about it. There certainly is no conscious sensory organ for skin wetness. We then can only conclude that, at higher room temperatures, sweat is transmitted equally into moist and dry bottles. This would, for example, shift the 20°C curve of figure 1 downward and parallel to itself. This explanation obviously is not valid, as shown by the 25°C curve which is oblique to and not parallel to the 20°C curve.

If sweat flow S is invariant for any curve in figure 1, the skin diffusion resistance, R_s, can be calculated with the formulas given previously (1, 2). It is convenient to make this calculation for the transfers at the driest bottle and at the neutral point. From figure 1, we find R_s = 2.5 m^2 hr. mm Hg/gm for a 20°C room, R_s = 1.3 for 23°–24°C, R_s = 0.15 for 25°C and R_s unmeasurably small for 30° and 40°C. If this is a real change of resistance with the environment or the ensuing body conditions, R_s changes of a comparable type should occur under other conditions.

Such changes do, in fact, occur. R_s for liquid water transfer (2) is higher if brine, rather than water, covers the skin. In a cool room, R_s is also larger on the bottle containing dry than on that containing moist air. As shown elsewhere (4), the stratum corneum conjunctum *in vitro*, as well as the horn, show similar changes of diffusion resistance between dry and moist air.

If this argument holds, the total loss of obviously sweating skin into a dry bottle (fig. 1, lower left points) is composed of diffusive vapor loss and of sweat loss. The diffusive loss seems to be the larger component. It certainly cannot be neglected.

Tourniquet and atropine. The tourniquet effect in a normal room is negligible; in a hot room loss into the driest bottle decreases by 27 gm m^{-2} $hr.^{-1}$, or about 30% of the total loss. Since the compressed arm becomes dry to touch and vision, one is tempted to assume that sweat flow rather than the diffusion might be altered. If so we would have one more proof of the hypothesis that a substantial part of the increase of vapor loss in the heat is an increased diffusion rather than sweat.

Atropine is without influence in a normal room. In a hot room, however, it changes transfer data in such a way that they resemble those of a normal room. One again is tempted to speculate; this change might indicate that sweat flow, as well as diffusion, is influenced by atropine.

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