

THE EFFECT OF OCCLUSION ON CARBON DIOXIDE EMISSION FROM HUMAN SKIN

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Abstract. The effect of occlusion on the carbon dioxide (CO₂) emission rate (CDER) of human skin was determined. Occlusive plastic tape elevated the CDER 4.5 times (90 nl/cm²/hr) over the normal CDER (20 nl/cm²/hr). This increase was noted within a 3-hour period. Non-occlusive paper tape had no effect on CDER. Quantitation of the amount of CO₂ under plastic tape revealed that CO₂ was present at a concentration of 8-10%. Removal of the plastic tape after 24 hours allowed the CDER to return to approximately normal values within 2 hours. The mechanism by which occlusive plastic tape mediates this dramatic effect on CDER as well as the significance of elevated CO₂ concentrations under occlusion are discussed.

Key words: Carbon dioxide; Occlusion; Emission, skin

Several studies (1-4, 6, 11, 14-18) document that the normal skin emission rate of carbon dioxide (CO₂) fluctuates between 11 and 100 nl/cm²/hr depending on the anatomical site measured. Due to the "state of the art" when these measurements were made their accuracy is unknown. None of these references document the effect of occlusion on the CO₂ emission rate through human skin (CDER). The present report considers this topic.

MATERIALS AND METHODS

Carbon dioxide emission from skin was measured in the manner reported by Frame and co-workers (4). However, there was a difference in the sampling device employed for measurement. Briefly, a plexiglass sample cup (Fig. 1) with an area of 9.6 cm² was attached (teflon tubing) to an infrared spectrophotometer designed to detect CO₂ (Lira Luft Infrared Analyzer, MSA Model 200, Mine Safety

Appliances, Pittsburgh, Pa., USA) on one side (exit port) and a source of ultra high purity nitrogen (N₂) carrier gas on the other side (entry port). As CO₂ was emitted from the skin surface it is mixed with carrier gas (N₂) and flowed through the exit port to the CO₂ analyzer. Identical flow rates on both sides of the sampling cup assured that leaks at the skin surface were not present. By employing CO₂-filled detector cells and appropriately filled filter cells the analyzer responded specifically to CO₂ contained in the sample mixture. The instrument can detect as little as 1 ppm of CO₂.

To measure CO₂ flux through various materials, circular pieces the size of the sampling cup were applied to the skin. Adhesive-backed plastic tape (Blenderm®) and porous paper tape (Micropore®) were used. CO₂ emission through the tape was measured above the tape surface.

Five subjects were used in this report. They were healthy male, white volunteers 20-25 years of age. Room temperature was maintained between 20-22°C and an average relative humidity of 50%. The upper back was the test site chosen, since this area is subject to little environmental insult and a relatively large area can be studied (forearms gave similar results). No measurements were made if the subject was sweating, since sweating is known to increase carbon dioxide emission (4). Skin temperature was monitored with a telethermometer (Yellow Springs Instrument Co., Yellow Springs, Ohio). CDER readings generally reached a steady state in 5-10 min after the cup was sealed, by finger pressure, to the skin surface.

To measure relative permeabilities of the occlusive materials a special chamber similar in size and shape was screwed into the sample cup. The two chambers could be separated by the material of interest. Normal sample flow was directed through the sample chamber (upper chamber) while various mixtures of CO₂/N₂ were directed into the lower chamber. CO₂ diffusion from the lower chamber through the test material and into the sample cup provided a means of assessing the permeability of different materials to CO₂. This method allowed one to predict the concentration of CO₂ that must be present under a material in order to reach the flux rates observed through that material *in vivo*.

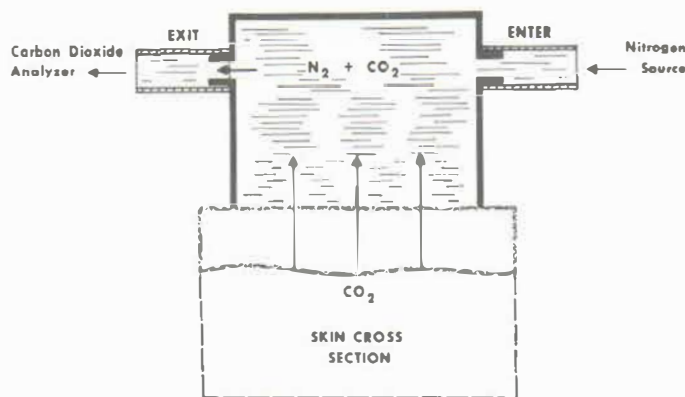


Fig. 1. Method of measuring carbon dioxide emitted from skin. (N₂=nitrogen; CO₂=carbon dioxide.)

RESULTS

The normal emission rate of carbon dioxide and the effects of an occlusive plastic tape on this rate are depicted in Fig. 2. The mean emission rate of carbon dioxide from the control site was 20 nl/cm²/hr. This rate was fairly constant in that subsequent readings over a 24-hour period were approximately equal. When an occlusive plastic tape was applied to skin, a dramatic effect on carbon dioxide emission ensued. Five minutes after application an increase in carbon dioxide emission was noted (statistically not significant). At 30 min the increase in carbon dioxide emission was statistically significant (two-tailed *t*-test: *t*=3.675; *p* (0.05-0.025). The emission of carbon dioxide was

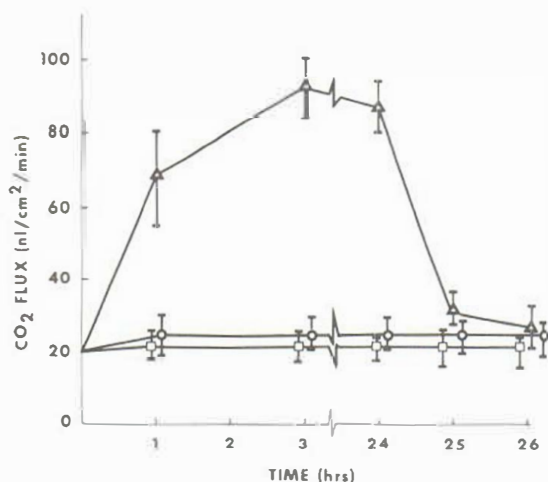


Fig. 2. Measurement of carbon dioxide from skin. All points represent the mean value of 5 subjects. ○—○, control site (no occlusion), □—□, paper tape site (Micro-pore), △—△, plastic tape site (Blenderm).

higher still at 3 hours; when measured at 24 hours the rate of emission was similar to the 3-hour reading. Statistically the mean value at 3 hours does not differ from 24-hour readings. Thus, it appears that at 3 hours the CDER reaches a plateau of approximately 90 nl/cm²/hr. Removal of plastic tape at 24 hours led to complete restoration of normal emission rates of carbon dioxide in 2 hours. At this time, carbon dioxide emission rates of control and occluded sites were statistically comparable.

To rule out the possibilities that these increased readings were due to: 1) the adhesive property of the tape, or 2) an artifact encountered when any material, occlusive or not, was placed on skin, the following data were obtained. Instead of occluding the skin with plastic tape, an adhesive but very porous paper tape was utilized. Increases in carbon dioxide emission were not detected at any time of measurement. All values were statistically equivalent to control sites. These data implied that the increase in carbon dioxide emission rate noted with plastic tape was attributable to its occlusive properties, and was not due to its adhesiveness nor to a random increase encountered when any material is placed against the skin surface.

To obtain a relative estimate of the concentration of carbon dioxide under occlusion the following experiment was performed. An *in vitro* chamber was designed (see Materials and Methods) which would allow passage of standard concentrations of carbon dioxide under plastic tape. Carbon dioxide emission through the tape was measured in the same manner as that described for skin. A standard curve, carbon dioxide concentration under the tape vs. emission of carbon dioxide over the tape, was constructed (Fig. 3). Taking the average value of

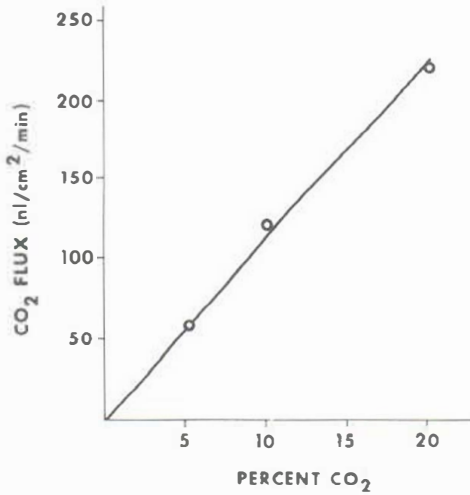


Fig. 3. Measurement of carbon dioxide flux through plastic tape as a function of carbon dioxide concentration (%) below the tape.

carbon dioxide emission rates shown in Fig. 2 and plotting them on this graph, a percentage of carbon dioxide necessary to produce this emission rate through the tape can be extrapolated. While a percentage for normal skin cannot be determined, since the emitted carbon dioxide would be quickly dissipated to the surrounding atmosphere, it is noted that under occlusion (24 hours) carbon dioxide levels reach 7-8%.

DISCUSSION

The data presented establish that occlusion of human skin by plastic tape increases the rate of emission of carbon dioxide. Plastic tape mediates increased carbon dioxide emission through its occlusive properties and not through adhesiveness. Carbon dioxide concentrations under the occlusive tape reach 7-8%.

The exact mechanism of increased carbon dioxide emission from occluded skin is not known. The increased amount of carbon dioxide under the tape is most likely due to this tape's relative impermeability to this gas. However, the fact that equilibrium of carbon dioxide emission over plastic tape is approximately 4.5 times that of normal, suggests that this gas must be emitted through skin at this elevated rate in order to maintain this static level. Thus, not only is there a trapping effect of the tape but also an increased CDER. It is doubtful that epidermal damage is responsible for the increased

CDER caused by occlusion, since removal of the occlusive plastic tape allowed for rapid restoration to a normal CDER. Rothman (14) attributed the increase in CDER at elevated temperatures to increased vascularity resulting in increased metabolism of eccrine sweat gland cells. Neither increased temperature nor vascularity (erythema) were observed on occluded sites. Also, we tested one subject having congenital anhidrotic ectodermal dysplasia and found that this subject also demonstrated an increased CDER, equivalent to a normal test subject. Thus, it is unlikely that increased vascularity with subsequent increased eccrine sweat gland metabolism is the mechanism by which occlusion increases CDER.

Hydration, a factor known to effect CDER, may be responsible for the increased CDER. Frame and co-workers (4) showed that application of water to normal skin doubled the rate of carbon dioxide emission. As under occlusion the skin becomes hydrated (a generally accepted fact) then increased emission of carbon dioxide may be expected. Whether or not this increase is sufficient to account for the 4.5-fold increase found under occlusion is not known. Further studies must be performed in order to gain a complete understanding of the effects of occlusion on carbon dioxide emission from skin.

Occlusion has been implicated as being involved in several cutaneous infectious diseases (7-10, 12, 13, 19-21), although the exact mechanism by which occlusion aids in the initiation of these diseases is unknown. In a recent report (5) which documents that carbon dioxide has a profound effect on dermatophyte growth and physiology, we question whether this gas plays any role (either positively or negatively) in the pathogenesis of dermatophyte infections. We extend this speculation not only to dermatophytes but also to other organisms that initiate their skin manifestations under occlusion. At present, direct evidence that carbon dioxide can influence the ability of an organism to cause disease does not exist. However, we feel that this speculation warrants continued investigation.

ACKNOWLEDGEMENTS

This work was partially supported by the Johnson & Johnson Baby Products Co.

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Received March 17, 1977

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