*Glyn R. Fox,*[1] *Ph.D. and John S. Hayward,*[1] *Ph.D.*

# Effect of Hyperthermia on Breath-Alcohol Analysis

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**ABSTRACT:** Mild hyperthermia to the extent of a 2.5°C increase above normal body temperature was produced by immersion of ethanol-intoxicated subjects in a warm water bath. Hyperthermia did not influence the blood-alcohol decay curve of the subjects. Hyperthermia did cause a significant distortion of the breath-alcohol decay curve, up to as much as a 23% increase above blood-alcohol concentration. The magnitude of this distortion effect was calculated to be a 8.62% increase in breath-alcohol concentration over blood-alcohol concentration for each °C increase in core body temperature. The forensic relevance of these results is that further support is given to previous recommendations that temperature monitoring be included in procedures for breath-alcohol analysis. This leads to the recommendation that mouth temperature be measured before breath sampling to screen for abnormal body temperature and to allow for potential use of a "temperature correction factor." This modification to existing analytical procedures would optimize the reliability of breath-ethanol analysis for prediction of blood-ethanol concentration.

**KEYWORDS:** pathology and biology, hyperthermia, breath-alcohol testing devices

Accurate and reliable inferral of blood-alcohol concentration (BAC) from measurement of breath-alcohol concentration (BrAC) is subject to interference by temperature conditions [*1-3*]. Variation in core body temperature modifies the initial blood:breath ethanol partition at the alveolar site according to the relationship determined in vitro by Harger et al. [*4,5*]. Second, the temperature of expired breath varies during exhalation, resulting in end-expiration BrACs that are dependent on mouth temperature [*6-8*]. Finally, the temperature of ambient air can alter BrAC by altering expired breath temperature [*9,10*].

Current methods for sampling breath for BrAC analysis require that the breath be sampled only at the end of a deep expiration and only after the test subject has equilibrated with ambient air of normal temperature for at least 15 min. These procedures negate to a large extent common and expected temperature influences caused by variable mouth or ambient air temperatures. However, they do not necessarily eliminate influences produced by altered core body temperature on the initial partition of ethanol between blood and breath at the alveolar site. Although this latter effect of temperature on BrAC analysis has been recognized for some time as a possible significant "source of error" when using BrAC

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[1]Research associate and professor, respectively, Department of Biology, University of Victoria, Victoria, B.C., Canada.

to infer BAC, [2,11] it has been virtually ignored in forensic determinations of BAC from BrAC [12].

We have recently reported that mild hypothermia in humans produces a serious distortion of the breath-ethanol decay curve of intoxicated subjects [3]. This distortion results in underestimation of BAC predicted on the basis of BrAC values. In light of this result, we have extended the examination of this temperature effect to *hyperthermic* humans by investigating the relationship between rectal temperature, mouth temperature, BrAC, and BAC.

## Methods

The subject group consisted of 9 young men (mean age 22.3 years, mean weight 75.3 kg). Each subject arrived at the laboratory in a substantially post-absorptive condition (minimum of 6 h). The subject donned bathing trunks and inserted a thermistor rectally to a depth of 15 cm, for continuous recording (Thermalert® monitor, Bailey Instruments, Model TH-6D) of his rectal temperature.

A 1.15-mL · $kg^{-1}$ dose of 95% (v/v) ethanol : distilled water, (0.853 g · $kg^{-1}$ absolute ethanol), designed to produce a BAC of approximately 80 mg · $dL^{-1}$, was mixed with unsweetened orange juice of a volume equal to three times that of the dose of ethanol. One hour before warm water immersion, the subject consumed this experimental drink while sitting quietly for a 20-min period. Room temperature was maintained at 22 to 23°C. Following an additional 40-min period to allow further absorption of the ethanol, the subject was immersed to the level of his lower neck in a stirred bath of 40°C water. After 5 min of immersion, the bath temperature was raised to 42°C for the remaining 45 min of immersion. At the termination of the warm water immersion (that is, at 50 min) the bath temperature was lowered to 35°C. This temperature was maintained for 15 min, after which the bath temperature was further lowered to 32°C. This bath temperature was maintained until the completion of the experiment at 90 min. At this time, the subject's rectal temperature had returned to near the normothermic level of approximately 37°C.

At seven times during the experiment, breath samples were obtained from the subject for determination of BrAC using a Breathalyzer® (Stephenson Corp., Model 900). Concurrent with these breath samples, 3-mL samples of blood were withdrawn from the antecubital vein. The blood was collected in sterile, Vacutainer® tubes (Becton-Dickinson and Co., Catalog No. 6387) containing 45 USP units of sodium heparin. No blood preservative (for example, sodium fluoride) was required since samples were refrigerated immediately and assayed for BAC by gas chromatography within 10 h of sample procurement. In addition, at these times, mouth temperature of the subject was measured using an electronic digital fever thermometer (Becton-Dickinson and Co.). This thermometer is unbreakable, inexpensive, and readily available at retail pharmacies. Both the Thermalert system used for measuring rectal temperature and the digital fever thermometer feature stable calibration within 0.2°C of the absolute temperature over their range of measurement.

Subjects were familiarized with procedures for providing breath samples, and the Breathalyzer was operated according to the standardized procedure described by the manufacturer. Only fresh, certified reagents (BDH Chemicals) were used in the determination of BrAC.

BAC was determined using a Micro Tek®, series 220 gas chromatograph (Tracor Inc.), fitted with a flame-ionization detector, according to the method of Cooper [13] using isopropanol as the internal standard. Chromatographic conditions were as follows: 2-m, 80-100 mesh, Super "Q"® column, (Mandel Scientific Co.); column temperature, 170°C; detector temperature, 220°C; carrier (nitrogen) flow rate, 45 mL · $min^{-1}$; input attenuation, $10^2$; output attenuation, 2; and bucking range, $10^{-8}$ $x$ +2.

Statistical analysis of differences between means was by Student's $t$-test with significance concluded if $P < 0.05$.

## Results

Figure 1 compares the responses of rectal temperature, mouth temperature, BrAC, and BAC during the three experimental phases of pre-immersion, warming, and cooling. During the warming phase, rectal and mouth temperatures increased to reach relatively stable maxima that were 2.6 and 2.8°C respectively, greater than initial temperatures observed at the beginning of warm water immersion. The temperatures of the two sites did not differ significantly up to time 0, or at 35, 50, and 90 min. Site temperatures did differ significantly at both 20 and 70 min of the experiment. These later times coincided with the periods of most rapid warming and cooling of the subjects and demonstrated a slight "lag" in the response of rectal temperature when compared to mouth temperature. This divergence in temperatures at the two sites was expected and is a consequence of temperature gradients established in the body in association with high rates of heat exchange during water immersion [*14*].

The following description of the results for alcohol concentration presents values in units of mg · $dL^{-1}$ of blood. BrAC values are actually estimates of BAC derived from breath analysis (where 1 mg · $dL^{-1}$ blood approximates 1 mg · 210 $L^{-1}$ of breath). As shown in Fig. 1,

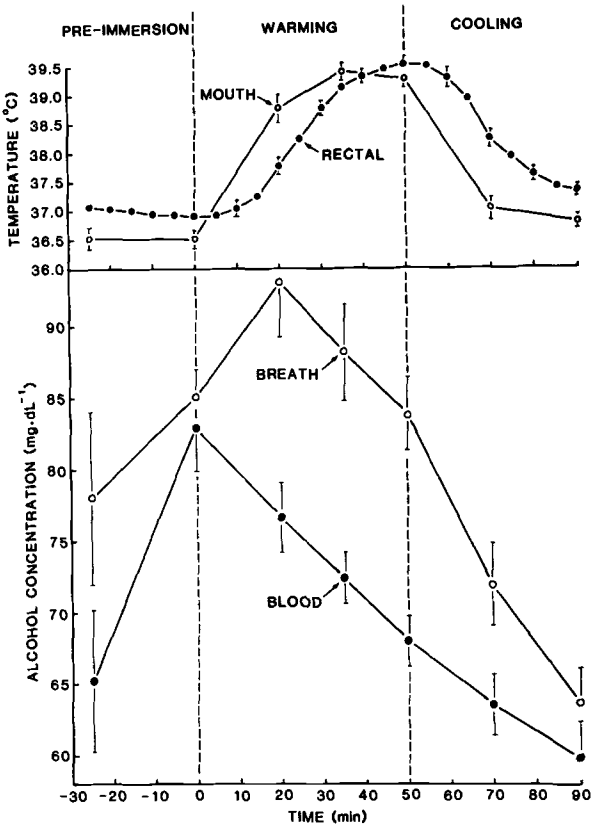


FIG. 1—*Relationship between changes in body temperature (mouth and rectal sites) and patterns of change in alcohol concentration of blood and breath of ethanol-intoxicated humans. Alcohol concentration is shown in units of mg/dL⁻¹ of blood. Values shown for BrAC were derived from the standard relationship that 1 mg/dL⁻¹ of blood approximates 1 mg/210 L⁻¹ of breath. Values are means ± standard error of the mean for nine subjects. Standard errors are not indicated for every mean rectal temperature, including those up to Time 0, which were too small for legible illustration.*

initial readings of alcohol concentrations occurred at 25 min before immersion (that is, 15 min after the cessation of alcohol consumption). Since absorption of alcohol was not complete at this time, considerable variation of both BrAC and BAC was observed. Also, as was expected on the basis of arterio-venous differences in ethanol concentration which occur during the absorption period [*15,16*], mean BAC was slightly lower (by 12.7 mg · $dL^{-1}$) than BrAC. Although this difference was not statistically significant, it did approximate the typical difference of about 10 mg · $dL^{-1}$ commonly observed between BAC and BrAC during the absorption period [*15*].

BAC increased during the pre-immersion, normothermic period to reach a peak measured level of 82.8 mg · $dL^{-1}$ immediately before immersion in the warm water bath. BAC then declined steadily at a mean rate that was equivalent to 15.9 mg · $dL^{-1}$ · $h^{-1}$ (a decay rate essentially identical to Kalant's [*16*] standard BAC decay rate of 16 mg · $dL^{-1}$ · $h^{-1}$ for normothermic men). Thus, mild hyperthermia did not alter the net kinetics of ethanol metabolism in our subjects.

BrAC increased during the pre-immersion period to reach a value of 85 mg · $dL^{-1}$. This BrAC-derived value was insignificantly different from that of the BAC value (82.8 mg · $dL^{-1}$) observed immediately prior to immersion in warm water. Thereafter, BrAC-derived values were significantly greater ($P < 0.01$) than corresponding BAC values throughout the entire warm-water immersion period and during the initial 20 min of the cooling period. During these periods, significant hyperthermia was evident in the subjects. By the time normothermia had become reestablished in the subjects at 90 min, BrAC (63.3 mg · $dL^{-1}$) and BAC (58.9 mg · $dL^{-1}$) were again insignificantly different. Thus, mild hyperthermia elicited a significant distortion of BrAC from its expected congruence with BAC. Observed BrAC-derived values differed from BAC values by as much as 23% (at 50 min) during the period of hyperthermia. These differences were used, as follows, to calculate a "distortion index" for the perturbing effect of hyperthermia on BrAC. Values obtained at 35 and 50 min were used because at these times mouth and rectal temperatures were insignificantly different and relatively constant. Therefore, these temperatures would provide the most reliable assessment of core (for example, lung) temperature during hyperthermia. Accordingly, "distortion indices" (that is, the percent increase of BrAC over BAC divided by the amount of hyperthermia from Time 0) were calculated for mouth and rectal temperatures at 35 and 50 min. The mean of these four values (that is, the mean distortion index) was 8.62% · $°C^{-1}$ (SD = 0.504) and provides the most valid estimate for the perturbation effect of core hyperthermia on BrAC.

## Discussion

These results show clearly that mild hyperthermia in humans does not alter the standard decay curve of BAC (and therefore the net kinetics of ethanol metabolism), but does significantly distort the BrAC decay curve to an extent which would cause serious inaccuracy for prediction of BAC. The magnitude of this distorting effect of core temperature is too large (up to 23% with mild hyperthermia) to be ignored in breath-testing procedures. In contrast to the situation provoked by hypothermia [*3*], such error in the case of hyperthermia increases the likelihood of a suspect being unjustly convicted [*1*]. Ethanol intoxication may accompany several situations which can significantly elevate core temperature. For example, core temperature may be significantly elevated by fever, consumption of certain drugs (for example, amphetamines [*17*]), heavy physical exertion, or exposure to high ambient air or water temperatures as found in saunas or hot tubs. Consequently, it seems warranted, from a judical viewpoint, to insure that any possible variation in core temperature be detected during forensic assessment of BAC from BrAC. For this specific reason as well as others relating to temperature influences [*2, 10, 11*], we recommend that direct monitoring of breath temperature be incorporated into BrAC analyzers to provide automatic temperature com-

pensation. Breath temperature measurement would be suitable because it reflects mouth temperature [6], which in turn reflects core temperature. Until such instrumentation becomes routinely available, it may be necessary to rely solely on blood analysis in cases of potential abnormality of core temperature.

The foregoing solution, however, is impractical in the case of hypothermia due to the difficulty of blood sampling caused by cold-induced peripheral vasoconstriction [3,18]. Furthermore, the use of invasive sampling in cases of "suspected" hyperthermia is obviously undesirable. Rather than relying on direct blood-alcohol analysis, it seems more reasonable, using present BrAC instrumentation, to attempt to correct for possible error of BrAC resulting from altered body temperature by measuring the body temperature at some site and applying a "correction factor" to the measured BrAC value. This approach has been suggested for hypothermia, using the correction factor determined in vivo of $7.3\% \cdot {}^{\circ}C^{-1}$ decrease in rectal temperature [3]. This factor closely approximates the in vitro correction factor of $6.8\% \cdot {}^{\circ}C^{-1}$ determined by Dubowski [19]. Similarly, we suggest the expediency of utilizing the mean distortion index of $8.6\% \cdot {}^{\circ}C^{-1}$, determined herein, as a suitable correction factor for the perturbing effect of hyperthermia on BrAC. A suitable body site would be the mouth, a common and socially acceptable site at which to measure core temperature. In contrast to the present experimental situation, which used rapid body cooling by water immersion, correction of BrAC based on altered mouth temperature would have great validity under the conditions of slow body cooling typical of forensic science assessment. Thus, the measurement of a test subject's mouth temperature before breath sampling for BrAC offers two advantages. It would screen for possible departures from normothermia and would provide an opportunity to adjust BrAC by use of an appropriate temperature correction factor.

In summary, mild hyperthermia or hypothermia [3] can significantly distort BrAC and lead to serious inaccuracy of predicted BAC. Feasible methods now exist to remedy this problem.

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Address requests for reprints or additional information to
John S. Hayward, Ph.D.
Department of Biology
University of Victoria
Victoria, B.C. V8W 2Y2 Canada