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Effect of Hypothermia on Breath-Alcohol Analysis

REFERENCE: Fox, G. R. and Hayward, J. S., **"Effect of Hypothermia on Breath-Alcohol Analysis,"** *Journal of Forensic Sciences,* JFSCA, Vol. 32, No. 2, March 1987, pp. 320–325.

ABSTRACT: Mild hypothermia, induced by experimental immersion of ten subjects in cold water, distorted the decay curve of breath ethanol of intoxicated subjects by as much as 22% while not altering overall ethanol clearance rate. The results provide in vivo verification of the in vitro temperature correction factor of 6.8% · $°C^{-1}$, and support previous recommendations that temperature monitoring be included in procedures for breath-ethanol testing. We recommend that mouth temperature be obtained 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 help to optimize the reliability of breath-ethanol analysis in predicting blood-ethanol concentration.

KEYWORDS: criminalistics, breath-alcohol testing devices, alcohol, hypothermia, ethanol, BAC, breath analysis, exposure, temperature, cold

The accuracy and reliability of predicting blood-alcohol concentration (BAC) from measurement of breath-alcohol concentration (BrAC) continues to be a topic of vital interest to law enforcement agencies [*1,2*]. Current procedures for breath analysis of alcohol are believed to minimize or negate the effect of temperature and other factors which can invalidate the standard partition ratio between the concentration of ethanol in blood and breath. Consequently, forensic scientists are generally confident of their ability to infer accurately BAC from BrAC. The purpose of this report is to reemphasize the fact that although such confidence seems generally valid, one further temperature-related improvement of procedures warrants consideration. That improvement is to account for any possible alteration in BrAC resulting from a significant variation in core body temperature.

Temperature can influence BrAC in three ways. First, 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. [*3,4*]. Second, the temperature and BrAC of expired breath varies during exhalation, yielding end-expiration values that are dependent on mouth temperature [*5,6*]. Third, ambient air temperature influences BrAC through its effect on expired breath temperature [*7,8*].

As a consequence of these important temperature effects on the accuracy of the BrAC to BAC relationship, Dubowski [*9,10*] has been a strong proponent for the incorporation of breath temperature monitoring in procedures which infer BAC from breath analysis. Typically, this recommendation has been eschewed in current procedures by sampling breath under controlled conditions designed to negate the effects of temperature variation. This involves sampling of breath only at the end of a deep expiration and after the test subject has

Received for publication 21 Jan. 1986; revised manuscript received 12 April 1986; accepted for publication 14 April 1986.

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equilibrated with ambient air of normal temperature for at least 15 min. However, these procedures eliminate only two of the three ways that temperature can confound prediction of BAC from breath analysis. They do not eliminate the possible effect of abnormal core body temperature (hypothermia and hyperthermia). This point is ratified by the statement of Dubowski [*9*] that monitoring of breath temperature during analysis could be used "to signal the possibility of undetected abnormalities in deep-body temperature which might impair or vitiate the validity of an instrument calibration or result conversion dependent on biological factors."

The importance, with respect to the validity of current procedures of breath analysis, of failing to account for possible variation in core body temperature needs to be assessed. It is generally assumed that this factor is relatively unimportant, since most test subjects who are ambulatory are likely to exhibit only minor variation in core body temperature, such as may be related to individual variation in "set" temperature, occurrence of fever, or consumption of aspirin [*10*]. However, this ignores the fact that mild to moderate hypothermia is potentiated by certain types of accidents which occur in cold ambient environments. Consider, for example, the common occurrence of automobile accidents caused by icy, winter roads. There can easily be a considerable time delay in the arrival on the scene of police and other emergency personnel. Another possibility is that a suspect may be trapped or pinned in the vehicle until specialized rescue equipment (for example, "jaws-of-life") becomes available. Such situations represent a significant opportunity for cold exposure of the individual involved. Although the core temperatures of severely traumatized or unconscious victims would tend to cool rapidly, such individuals are not legitimate candidates for breath analysis, even if alcohol intoxication was suspected. On the other hand, an ambulatory individual, who is a candidate for breath analysis, might also be mildly or moderately hypothermic as a result of such cold exposure. Importantly, this possibility would be augmented if the individual's thermal defenses were impaired by alcohol intoxication [*11*]. By inference, therefore, mild or moderate hypothermia, in conjunction with alcohol intoxication, may be a much more frequent occurrence than is generally appreciated. This incognizance would account for the lack of substantive data on this issue.

The above conclusion indicates the need for greater attention to core body temperature as a variable that can impair the reliability of BAC estimation by breath analysis. The problem could be resolved by measuring breath temperature and applying a "correction factor" as Mason and Dubowski [*10*] have recommended. The most recent correction factor to be proposed from empirical, in vitro data is $6.8\% \cdot {}^{\circ}C^{-1}$ [*12*]. However, the validity of using such a factor to correct for variation in core temperature has not been demonstrated experimentally in humans. No study is available that relates core temperature to mouth temperature, breath temperature, BrAC, and BAC. Partial evaluation of this relationship is possible from data used in a previous publication from our laboratory [*13*] concerning the effect of alcohol intoxication on cooling rate of humans in cold water. In that study, BrAC and core temperatures were measured, but blood-ethanol level and breath temperature were not. In light of the importance of evolving maximal reliability of breath analysis for inferral of BAC [*1,10*], and stimulated by renewed interest in the temperature variable [*7,8*], we have conducted a new analysis of our previous data to obtain a preliminary indication of whether mild hypothermia actually does perturb the BrAC to BAC relationship and if a correction factor of $6.8\% \cdot {}^{\circ}C^{-1}$ [*12*] is appropriate.

Methods

The subjects, apparatus, and procedures that yielded the present data have been detailed previously [*13*]. A brief description is required here.

Ten young men (mean age 21.2 years, mean weight 71.6 kg) constituted the subject group. Each subject arrived at the laboratory in a 4-h, postabsorptive condition. He then donned

bathing trunks and inserted a thermistor rectally for continuous monitoring of core temperature.

One hour before cold water immersion, the subject consumed an experimental drink containing 95% (v/v) ethanol: distilled water, dose 1.15-mL 95% ethanol · kg^{-1} (0.853-g absolute ethanol · kg^{-1}), mixed with unsweetened orange juice of a volume equal to three times that of the dose of ethanol. The subject sat quietly while consuming the drink over a 25-min period. Room temperature was 22 to 23°C. Following an additional 35-min period to allow further absorption of the ethanol, the subject was immersed to the level of his lower neck in a tank of stirred, 10°C water. After 45 min in the cold water, the subject transferred to a rewarming bath, the temperature of which was 27°C initially and was raised to 40°C over the next 15 min and then maintained at that level. The experiment ended when core temperature had returned to the normothermic level of approximately 37°C.

At six times during the experiment, breath samples were obtained from subjects for determination of BrAC using a Breathalyzer® (Stephenson Corp., Model 900). Subjects were familiar with procedures for providing breath samples, and the analyzer was operated according to the standardized procedure described by the manufacturer. Only fresh, certified reagents were used in the determinations of BrAC.

Results

Figure 1 compares the responses of rectal temperature and BrAC during the three experimental phases of preimmersion, cold water immersion, and rewarming. Rectal temperature decreased slightly, but insignificantly (0.3°C), during the preimmersion period. Mild hypothermia (to the extent of a 2.2°C reduction) was induced by the 45-min period of cold water immersion. The rewarming phase demonstrated an initial "afterdrop" [*14*] of rectal temperature, amounting to 0.3°C, followed by a steady increase to normothermia by 50 min of warm bath treatment. The observed BrAC declined during preimmersion at a mean rate of 16.6 mg · 210 L^{-1} · h^{-1} (equivalent to a BAC decay rate of 16.6 mg · dL^{-1} · h^{-1}) which is very similar to Kalant's [*15*] standard BAC decay rate of 16.0 mg · dL^{-1} · h^{-1} for normothermic man. Figure 1 includes a plot of standard BrAC decay rate based on Kalant's standard BAC decay rate. It should reflect the actual decay rate of blood ethanol in our subjects, since mild hypothermia does not appear to alter significantly the net kinetics of ethanol metabolism [*16,17*].

This standard curve was utilized for production of the temperature corrected decay curve of Fig. 1 by accounting for the decline of rectal temperature from 37.2°C at −35 min, together with Dubowski's [*12*] temperature correction factor of 6.8% · $°C^{-1}$. The resulting temperature-corrected curve shows that a significant perturbation of the standard BrAC decay curve is to be expected as a result of the mild hypothermia. The observed BrAC values confirm this expectation by closely approximating (within 4%) the temperature-corrected curve. Observed BrAC values differed from the standard BrAC decay curve by as much as 22%. The extent of the reductions of the observed BrACs for all sample times are shown in Table 1. Based on these five samples, the grand mean value for the effect of temperature on the BrAC of our subjects was 7.3% · $°C^{-1}$.

Discussion

These results support previous warnings that altered core temperature can distort the BrAC decay curve of humans, leading to inaccuracy of prediction of BAC [*1,4,8,10*]. We acknowledge that this conclusion is, to some extent, circumstantial. That is, actual BAC was not measured as a result of the design of the original experiment [*13*]. A study in which both BrAC and BAC are measured during significant alteration of core temperature is certainly needed for ultimate verification of the effect of core temperature on BrAC. However, the

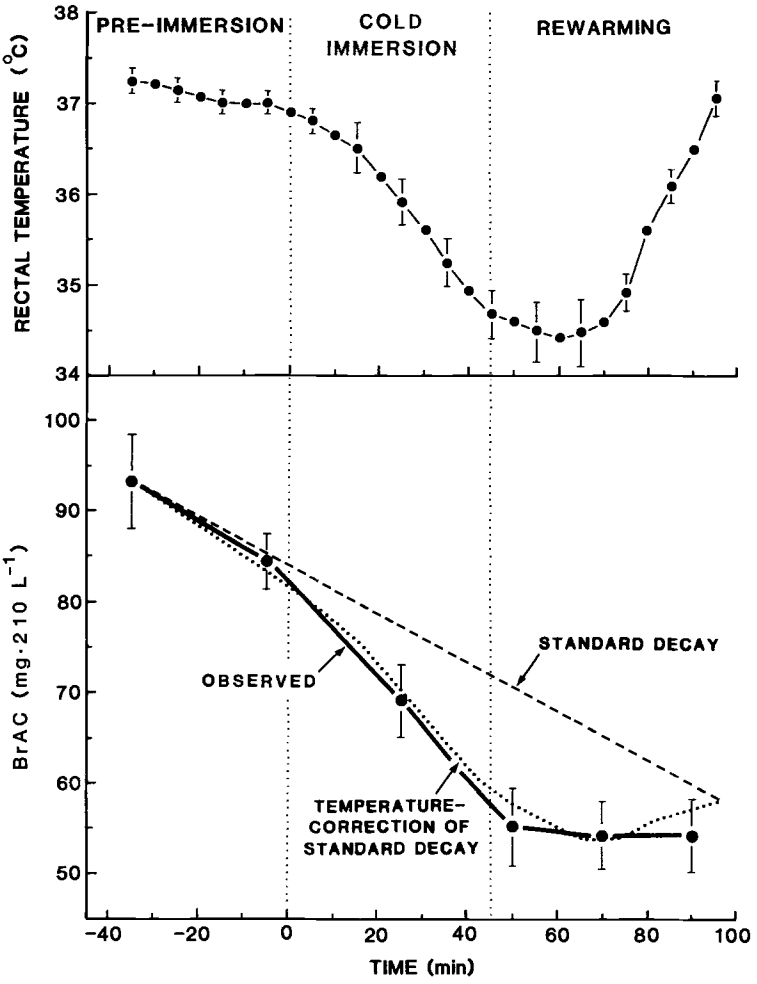

FIG. 1—*Relationship between changes in rectal temperature and breath-alcohol concentration (BrAC in units of mg · 210 L^{-1}, which is equivalent to BAC in units of mg · dL^{-1}). The temperature corrected curve for BrAC was calculated by application of the factor 6.8% · $°C^{-1}$ of rectal hypothermia to the standard BrAC decay curve based on 16 mg · 210 L^{-1} · h^{-1}. Note the close approximation of observed values to the temperature-corrected curve. Values are means ± s.e.m. for ten subjects.*

present results are compelling because of the high compliance of the observed BrAC values during hypothermia with the theoretical pattern of decay predicted from existing, in vitro knowledge of the effect of temperature on blood : breath partition of ethanol [*4,12*]. In addition, the magnitude of the distorting effect of core temperature variation (for example, up to approximately 20% with mild hypothermia) is too large to be ignored in breath-testing procedures. Although potential error would be to the advantage of the tested subject in the case of hypothermia, the principle should apply equally well to the case of hyperthermia, where such error would increase the subject's likelihood of being unjustly convicted [*1*].

Such potential errors are unacceptable, and for this reason, the major relevance of our results is to provide support for the recommendation of Dubowski [*9*], Mason and Dubowski [*10*], and Jones [*8*] that monitoring of breath temperature be incorporated into analyzers for

TABLE 1—*Percentage reductions of observed breath-alcohol concentrations (BrAC) from standard decay rate for various amounts of hypothermia below the initial temperature of 37.2°C at −35 min. Values are means for ten subjects.*

Time, min	Amount of Hypothermia, °C	Reduction of BrAC, %	Reduction/ Hypothermia, % · °C^{-1}
−5	0.25	0.35	1.40
25	1.29	10.38	8.05
50	2.63	21.73	8.26
70	2.62	16.58	6.33
90	0.75	9.38	12.50
		grand mean	7.31
		standard error of the mean	1.79

breath-alcohol concentration. An alternative strategy to such a modification of instruments would be to rely solely on blood analysis in cases of suspected abnormality of core temperature. We feel this is inappropriate for two reasons. First, a decision to use blood analysis on the basis of "suspicion" of abnormal temperature is inadequate. This is because mild or moderate levels of hypothermia produce behavioral symptoms such as impaired gait and balance, slurred speech, and drowsiness which are very similar to the symptoms of alcohol intoxication. Consequently, the occurrence of hypothermia might not be suspected because it might be masked by symptoms that led to initial suspicion of alcohol impairment. The second reason also relates to hypothermia. Sampling of blood from the periphery of cold-exposed individuals is very difficult as a result of strong vasoconstriction of all appendages. Furthermore, it is unlikely that any peripheral blood that could be collected would be representative of central blood because of the relative ischemia of the peripheral regions [*18*].

We propose a simple, inexpensive, and effective method to overcome the problem of possible inaccuracy of breath-ethanol analysis as a result of abnormal body temperature. Combined with the existing procedures of having the test subject wait in a room of normal temperature for at least 15 min, and collecting breath samples only at the end of deep expirations, we recommend the following. During the last 2 to 3 min before breath sampling, oral temperature should be measured in the standard clinical manner using a nonbreakable thermometer. This would serve a dual purpose. First, it would screen for departures from normothermia. Defined limits of departure could be set, a priori. For example, low and high limits of 36.5 and 37.5°C, respectively, would provide a "tolerance zone" of 0.5°C on either side of the normal mouth temperature of 37.0°C (98.6°F). Second, the recorded temperature would provide the opportunity for adjustment of the BrAC reading using a temperature correction factor. The in vitro factor of 6.8% · °C^{-1} [*12*] seems appropriate since our in vivo result (7.3% · °C^{-1}) closely approximates this value.

The importance of implementing the above recommendation hinges on the frequency of occurrence of abnormal body temperature in subjects undergoing breath analysis for alcohol intoxication. Studies to verify such occurrence certainly seem warranted. In the interim, our recommendation seems to be a worthy precaution, especially during conditions of winter cold.

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