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Alcohol Breath Test: Correcting for Bias

The alcohol[1] breath test ("ABT") has been touted for decades as an accurate measure of blood alcohol concentration ("BAC"). The explicit, and sometimes implicit assumptions are: (1) alcohol exchanges between the alveolar gas and blood in the surrounding capillaries; (2) exchange of alcohol between the blood and air is an equilibrium process; (3) end-expired alcohol concentration equals alveolar alcohol concentration, and (4) a correction factor (referred to as a blood-to-breath ratio) can be used to determine blood alcohol concentration from the end-exhalation alcohol concentration.

Research studies over the past 35 years have shown, with substantial experimental data and mathematical modeling, that *the above four critical assumptions are each false*. This article will discuss the research findings that are inconsistent with the assumptions, discuss the consequences implied by the research, and propose methods for minimizing the uncertainty resulting from using a misguided methodology.

Early Alcohol Breath Test: Theory

The ABT was developed as an indirect measurement of BAC.[2] Based on the awareness that oxygen and carbon dioxide exchanged in the alveolus between the air and the blood in the surrounding pulmonary capillaries, it was assumed that alcohol exchanged exclusively in the alveolus, that the exchange was complete, and that alcohol equilibrated between the pulmonary blood and alveolar air. In addition, it was assumed that the last part of the exhaled breath was equal in its composition to alveolar air. So, the design of the ABT was simple: Sample end-expired breath after a complete exhalation, measure the alcohol content, and multiply the value by a correction factor to determine the blood alcohol concentration.

Alcohol Breath Test: Practice

The location of gas exchange in the lungs depends strongly on the solubility of the gas in blood.[3] Respiratory gases (oxygen and carbon dioxide) exchange between the pulmonary blood and alveolar air because their intermediate blood solubility allows them to transit only the very thin membrane separating blood and air in the alveoli of the lungs. Highly soluble gases, like ethanol, can diffuse through both thin and thick tissue barriers.[4] So, alcohol can diffuse from the bronchial circulation (i.e., the blood that feeds the airway tissue) through the thick airway tissue and into the passing respired air (Figure 1).

Alcohol exchanges in both the airways and the alveoli.[5] Figure 2 illustrates the location within the airway tree and relative amount of alcohol exchange during both inspiration (black columns) and expiration (white columns). During inspiration, the passing airstream absorbs so much alcohol from the walls of the airway tree that less than 1 percent of alcohol comes from exchange within the alveoli.

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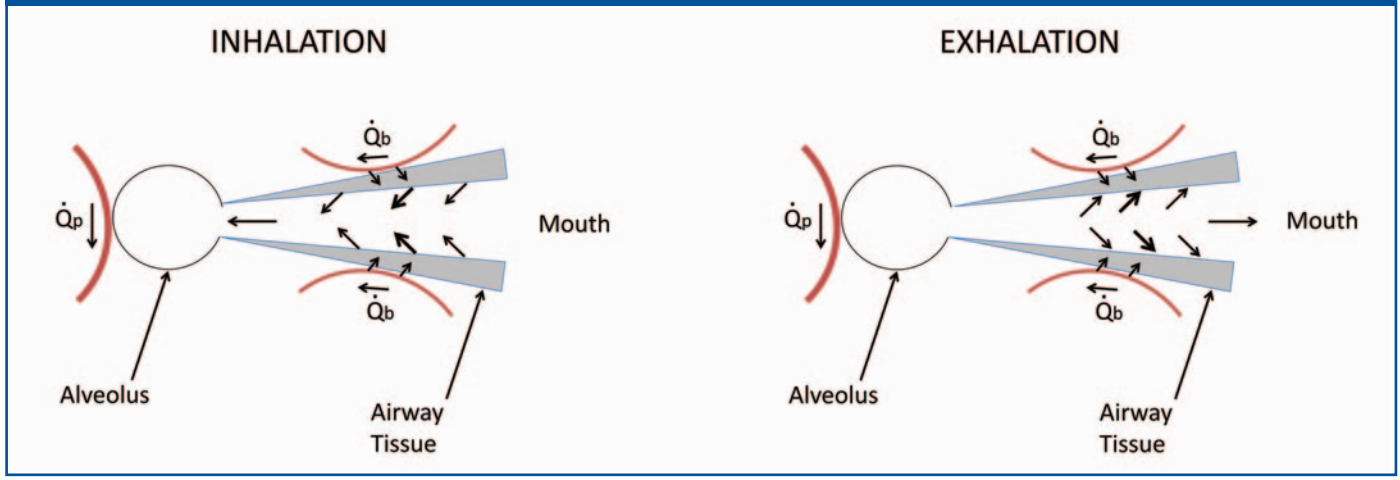
Figure 1



Schematic drawing illustrating alcohol exchange in the airways of the lungs during inhalation (left) and exhalation (right).

In other words, the concentration of alcohol in the inspired air is 99 percent of the alveolar alcohol concentration before entering the alveoli. During exhalation, some of the alcohol within the airstream deposits on the airway wall. As a result, end-exhaled alcohol concentration is lower than alveolar alcohol concentration ("AAC"). After exhalation of a vital capacity (a complete inhalation to total lung capacity and complete exhalation to residual volume), breath alcohol concentration ("BrAC") is always less than the alveolar alcohol concentration, which is in equilibrium with blood.[6] For breath to have an alcohol concentration equal to 99 percent of the alveolar air, the subject would need to exhale 10 times the amount of available air in his lungs (i.e., vital capacity) after a single inhalation to total lung capacity.[7] If less than a vital capacity is exhaled, then the BrAC will be less than it is after a vital capacity exhalation.

In effect, the end-expired BrAC is corrected by a single factor to account for two adjustments. First, the measured BrAC must be adjusted to a level that would be approximately equal to alveolar alcohol concentration. Second, this alveolar-adjusted BrAC is further corrected to approximate the alcohol concentration in blood. Under the original paradigm, it was thought that the correction factor should be equal to the partition coefficient ($\lambda_{b:a}$) between blood and air at 37°C because an adjustment to the alveolar alcohol concentration was thought to be unnecessary. The in vitro $\lambda_{b:a}$ for alcohol in human blood has been measured by Jones[8] as 1810.[9] Figure 3 shows three exhaled alcohol profiles with different correction factors. The thick solid black line is the measured alcohol profile using a correction factor of 1810. Note that BrAC continues to increase as the subject continues to exhale. The dashed profile is the actual profile corrected by 2100,[10] the correction factor used by breath test instruments in the United States and many other countries. The dotted line is the alcohol profile corrected with a factor of 2310. This larger correction factor is needed to correct the measured alcohol profile to the alveolar alcohol concentration and then correct the alveolar alcohol concentration to the blood alcohol concentration. The model used for these calculations was published earlier.[11]

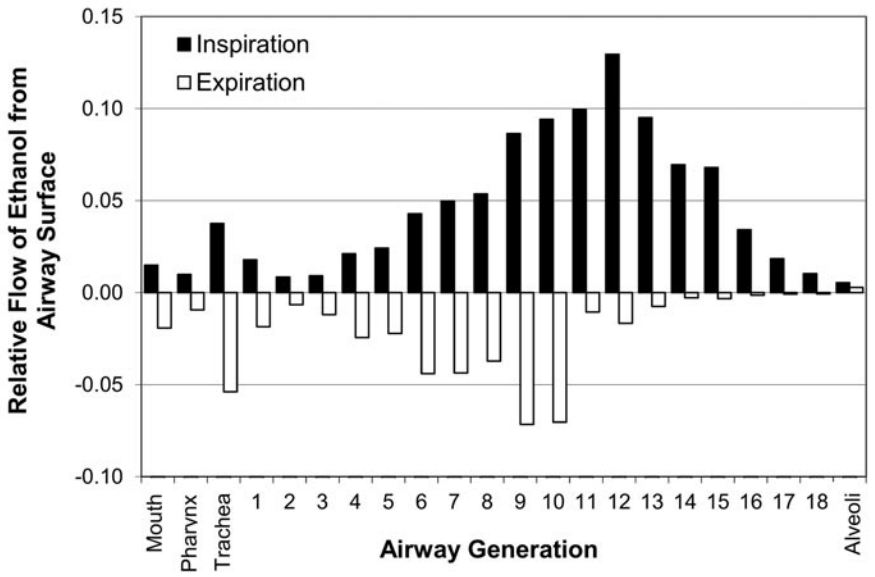
When BrAC is plotted against expired volume, there is no leveling-off at the end of exhalation. Leveling-off when plotting BrAC against time is simply an indication that exhalation has stopped. It is not an indication that alveolar air is obtained. Whether a person exhales 1.5 liters, a full exhalation or somewhere in between, the BrAC versus time plots will level off, but with very different BrAC values (Figure 4). This observation clearly shows that leveling-off of BrAC versus time does not indicate that alveolar alcohol concentration is obtained at the end of exhalation.

Expired Volume

Because alcohol exchanges in the airways, the volume of expired air affects the breath alcohol concentration. This interaction begins during inhalation. Inhaled fresh air (i.e., zero alcohol concentration) picks up alcohol from the airway tissue as this respired air moves towards the alveoli. This movement of alcohol significantly diminishes the concentration of alcohol in the airway tissue. So much alcohol is absorbed into the airstream during the transit from mouth to alveolus that the air is saturated with alcohol by the time it reaches the alveolus. During a large exhalation, the depleted airway tissue is partially recharged with alcohol. As air from the alveolus moves towards the mouth, some alcohol from the respired air deposits on the airway tissue. The alcohol concentration in the air decreases while that in the tissue increases. The alcohol in the tissue builds up relatively slowly due to the very high solubility for alcohol in tissue and never reaches the concentration prior to the start of inhalation. Computer modeling shows that BrAC can never reach alveolar alcohol concentration within a complete vital capacity exhalation. Even with a full vital capacity exhalation, the end-exhalation alcohol partial pressure is approximately 20 percent lower than alveolar alcohol concentration[12] (see Figure 3).

It takes a considerable amount of effort to exhale a full vital capacity of air from the lungs. As people breathe normally, they inspire a tidal volume[13] (typically around 500 ml) of air using inspiratory muscles (external intercostal muscles between the ribs and the diaphragm, which separates the lungs and the abdominal organs). Exhalation results from relaxation of the inspiratory muscles. This results in expiration to functional residual volume ("FRC") (see Figure 5A). At FRC, respiratory muscles are relaxed. The outward pull of the chest wall just offsets the inward pull of the lung tissue. A vital capacity ("VC") exhalation is the largest air volume one can exhale. It requires considerable effort to inhale to total lung capacity ("TLC") and exhale to residual volume ("RV"). Inhalation of air to TLC requires full effort of the external intercostal muscles. Then complete exhalation of a VC to RV first requires relaxation of

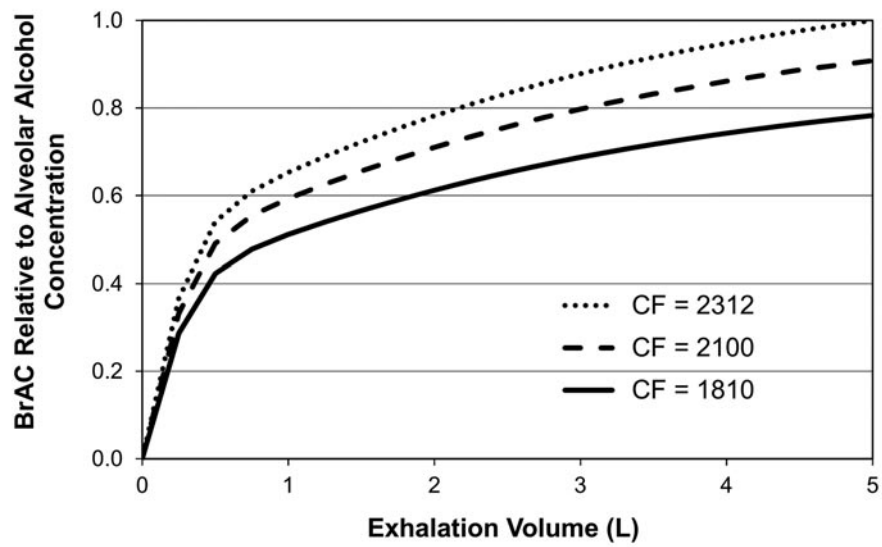
Figure 2



Alcohol movement for each airway generation over a single large breath. Solid columns show alcohol movement from the airway tissue into the passing airstream during inspiration. White columns represent deposition from the passing airstream onto the airway wall.

the inspiratory muscles followed by full effort by the expiratory muscles (internal intercostal muscles between the ribs). Exhalation is made more difficult at lower lung volumes because the airways narrow due to decreased tethering by the lung tissue, which decreases the size of airways and increases the resistance to flow. Individuals taking alcohol breath tests rarely have experience at performing respiratory maneuvers, and police officers administering the breath tests do not have adequate training in coaching subjects through these respiratory maneuvers. As it turns out, typical DUI subjects exhale an average of 50 percent of their available vital capacity.[14] Exhaled volume varies between 22 percent and 97 percent of vital capacity.[15] This variation in exhaled volume corresponds to an approximate 26 percent variation in BrAC.[16] Lack of control of exhaled volume contributes the greatest amount of uncertainty to the measurement of BrAC.[17] A typical breathing pattern for a subject taking an alcohol breath test is illustrated in Figure 5B.

Figure 3



Computer generated Breath Alcohol Concentration normalized by Alveolar Alcohol Concentration versus Exhaled Breath Volume. BrAC curves using correction factors ("CF") of 2312 (dotted line), 2100 (long dashed line), and 1810 (thick solid line) are shown.

Comparing BrAC to BAC

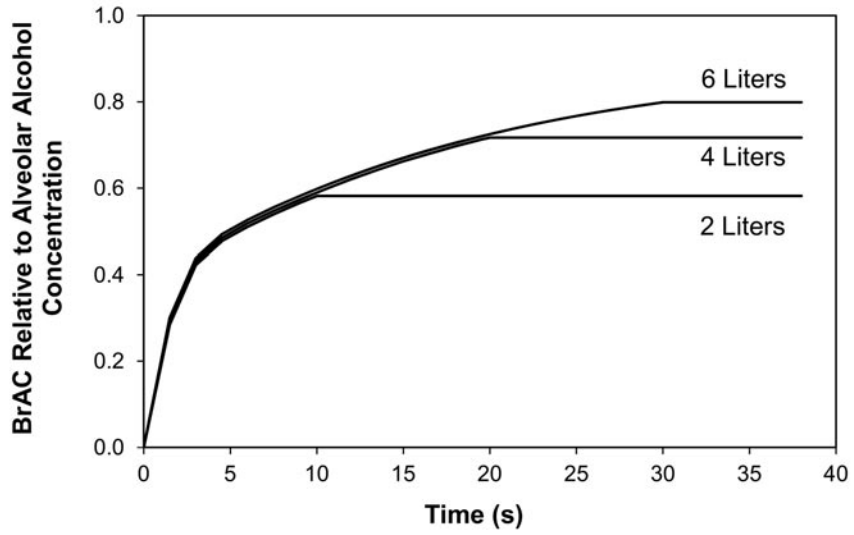
In order to validate the ABT, a number of studies have been performed to compare BAC (blood alcohol concentration) to BrAC (breath alcohol concentration). Many of these studies have been reviewed before,[18] so they will not be reviewed here. Each of these correlation studies found a much greater variation than would be expected if all the original assumptions were true. The variation in the ratio of BrAC to BAC was as much as ± 40 percent in the Emerson study.[19] However, it is more likely due to uncertainty in BrAC related to physiological uncertainty due to variation in exhaled breath volume, body temperature, and breathing pattern variation.[20] Additional uncertainty results from the use of venous BAC rather than arterial BAC (see below).

Venous BAC vs. Arterial BAC

BAC is not the same in all blood vessels in the body. During the absorption phase, alcohol is absorbed from the intestines, passes into the inferior vena cava and on to the heart. In the heart, the blood from the vena cava with higher alcohol concentration mixes with venous blood from other regions of the body, which creates a mixed venous blood that is lower in alcohol concentration than the blood coming from the intestines. This blood then passes through the pulmonary arteries and into the lungs, where a small amount (approximately 1/1810) passes out into the ventilated air if all alcohol exchange occurred in the alveolus. However, as some alcohol is retained due to airway exchange, the amount of alcohol exchanged is reduced by more than 20 percent depending on the subject's lung volume and the volume expired into the breath test instrument. The blood leaving the lungs via the pulmonary vein has an alcohol concentration that is essentially the same as the blood in the pulmonary artery. This blood distributes around the body. As it passes through the peripheral tissues, it loses some alcohol to the tissue and the systemic venous blood has a lower alcohol concentration than arterial blood. During the absorptive phase, venous BAC is lower than arterial BAC.

During the post-absorptive (or elimination) phase, the opposite relationships occur because absorption has charged the peripheral tissues with alcohol while depleting alcohol within the intestine. Furthermore, alcohol metabolism in the liver is where most alcohol is eliminated from the body. Thus, blood passing through the hepatic and mesenteric veins and through the vena cava has a low alcohol concentration. After pass-

Figure 4



Computer-generated breath alcohol concentration normalized by alveolar alcohol concentration is plotted against expiration time. Three exhalation profiles are shown with different expiration times (10, 20, and 30 seconds) that correspond to different exhalation volumes (2, 4, and 6 liters).

ing through the heart and lungs, this blood with a low alcohol concentration becomes arterialized. Arterial blood passes through tissue and picks up alcohol from this tissue. Thus, the venous blood alcohol concentration is greater than arterial alcohol concentration during the elimination phase.

Blood in the pulmonary circulation (the primary source of alcohol in the breath) is better represented by arterial blood alcohol concentration than it is by antecubital vein (inside of the elbow) alcohol concentration. Four separate studies have shown a stronger correlation of breath alcohol concentration with arterial BAC than with venous BAC over the entire absorption and elimination cycle.[21]

In order to understand the exchange mechanisms for alcohol by the lungs, it is necessary to compare breath alcohol concentration with arterial blood. The studies using venous blood are not helpful. Only those studies using arterial blood when comparing alcohol concentration can help in clarifying mechanisms of alcohol exchange. However, sampling arterial blood is more difficult (vessels are deeper within the tissue and surrounded by muscle) and requires a trained expert.

Volume Bias

During exhalation, the rising BrAC due to continuing airway tissue interaction results in two types of volume bias: exhaled volume bias and lung volume bias. A person who exhales just beyond the minimum required exhaled volume will have a lower BrAC than a person who exhales a full vital capacity, everything else being equal. There is bias against the person who exhales more volume.

When a subject exhales into an alcohol breath test device, he or she can exhale anywhere between the minimum volume and full vital capacity. A subject with a smaller vital capacity must exhale a greater fraction of available lung volume to provide the minimum air volume required for a valid breath sample. Thus, the average BrAC will be greater for a person with a smaller lung volume than one with a larger lung volume.[22]

Assumptions Used in Support of the Alcohol Breath Test

Let's examine the critical assumptions of the alcohol breath test.

1. Alcohol exchanges between the alveolar gas and blood in the surrounding capillaries.

Diffusion of gases is governed by Fick's second law, the mathematical description of gas movement from a higher concentration to a lower concentration via molecular diffusion:

$$\frac{dV}{dt} = \frac{\beta D A}{l}(P_a - P_b)$$

where dV/dt is gas flow, β is solubility of gas in the tissue barrier, D is diffusivity of gas through the tissue barrier, A is surface area, l is the distance for diffusion (i.e., thickness of the tissue barrier), P is partial pressure of the gas, a denotes region a and b denotes region b, where the gas diffuses from region a through the tissue and into region b. The airways are perfused by blood passing through the bronchial blood vessels. The thickness of the diffusion barrier is much greater in the airways than the thickness of the thin membranes in the alveolus. However, the key factor in the equation above is solubility (β). The solubility of alcohol is so much greater than oxygen or carbon dioxide, it is possible for alcohol to diffuse over a greater distance through tissue, making it easier for alcohol to exchange between the air passing through the airways and the bronchial blood. In fact, the vast majority of alcohol exchange occurs in the airways with the bronchial blood before the inspired air reaches the alveolus. For alcohol, assumption 1 is untrue.

2. Exchange of alcohol between the blood and air is an equilibrium process.

For alcohol exchange in the airway, the diffusion path through the tissue is greater than that in the alveolus. The airway tissue diffusion path is distributed along the airways and decreases in thickness as the airway diameters decrease from mouth to alveolus.[23] Because respired air passes quickly through each airway segment, the large diffusion barrier prevents equilibrium between blood and air. Thus, a global equilibrium for alcohol between blood and air is never achieved. Assumption 2 is untrue.

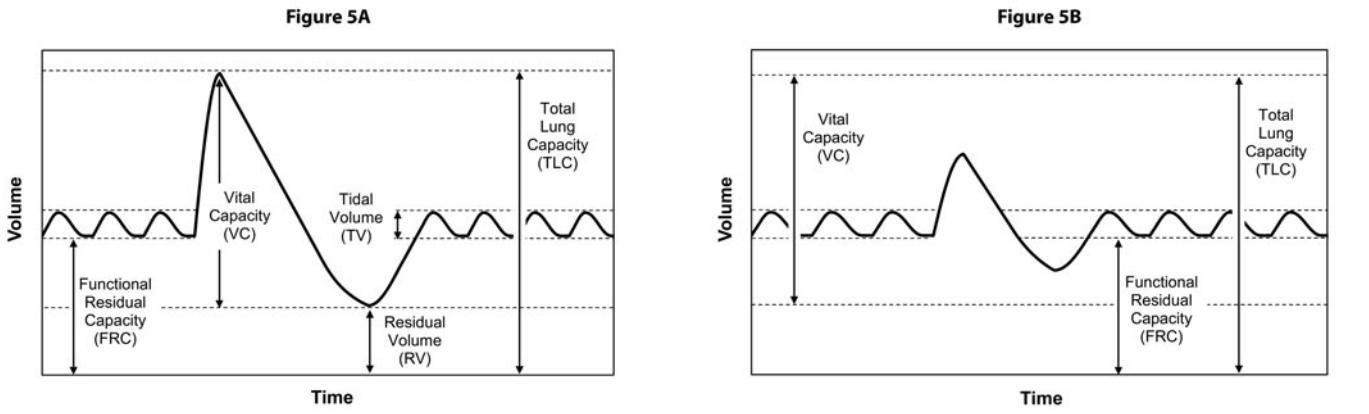
3. End-expired alcohol concentration equals alveolar alcohol concentration.

During exhalation, alcohol partially diffuses from the air to the airway tissue replacing the alcohol taken up from the tissue during inspiration. Thus, the alcohol concentration in the exhaled breath is lower than alveolar alcohol concentration. As exhalation continues, alcohol in the expired air alcohol gradually increases but never reaches alveolar alcohol concentration even after a full inspiration followed by a complete exhalation.[24] Assumption 3 is untrue.

4. A correction factor (partition ratio or blood-to-breath ratio) can be used to determine blood alcohol concentration from the end-exhalation alcohol concentration.

The term "partition ratio" does not apply because there is no equilibrium

Figure 5



Schematic illustration of lung volumes and capacities (more than one volume) during vital capacity measurement during spirometry (left) and typical alcohol breath test (right).

in the airway exchange process. Alcohol concentration increases with continuing exhalation. For a person stopping exhalation just beyond the minimum volume exhalation requirement, the BrAC will level off with a lower BrAC compared to a person who exhales a full vital capacity, everything else being equal. Because expired volume[25] is not controlled with today's alcohol breath testing instruments, there is no fixed ratio. The term blood-to-breath ratio does not apply because breath alcohol concentration changes with continued exhalation and the expired volume is not controlled. Assumption 4 is untrue.

Improving the Alcohol Breath Test

Bias in the alcohol breath test can be corrected with some modifications to the software of all alcohol breath testing instruments. Lung volume bias is caused by the practice of using the same minimum exhalation volume for all subjects. Lung volume can be predicted from standardized charts by entering age (or birth date), gender, race, and height.[26] The minimum expired volume can be tailored for each individual based on the individual's predicted lung size (e.g., vital capacity). This feature would not only correct the primary driver of lung volume bias, but it would also decrease the number of incomplete samples (not providing the minimum expired volume). In the past, others have proposed[27] that different minimum expired volumes be used for male vs. female subjects (reduced minimum volume for females) and for age (reduced minimum volume for older adults). However, there is no indication that this proposal has been put into use. This approach would provide a fairer minimum expired volume by eliminating lung volume bias and decreasing the number of incomplete samples. A potential minimum volume might be, for example, 10 percent or 20 percent of predicted vital capacity. Determining a subject-specific minimum should also reduce the number of incomplete breath samples, particularly for individuals with smaller lung volumes.

The other important bias is the expired volume bias. This bias is caused by the continuing alcohol exchange with the airway tissue during exhalation. The further a subject exhales, the greater the BrAC. Ideally, it would be best if the BrAC versus expired volume profile could be extrapolated to the alveolar alcohol concentration. However, the BrAC vs. expired volume is a multi-exponential curve with an unknown pattern. In addition, expired volume size depends on inspired volume and many of the expired volumes are less than 50 percent of the vital capacity, making extrapolation problematic. As the average volume of breath exhaled into a breath test instrument is approximately 50 percent of vital capacity (range: 26 percent-97 percent), a significant improvement would be to correct all BrAC values to what they would have been if the individual had expired 50 percent VC using a correction factor, perhaps 9.2 percent per liter of breath.[28] This approach would normalize the ABT reading to a standard exhalation volume fraction and eliminate exhaled volume bias. For example, if a subject with a vital capacity of 6.0 liters exhaled 2 liters (1 liter less than half of 6 liters) into the breath test instrument with a BrAC reading of 0.075, the corrected reading would be = 0.075 + 0.075 x (3 - 2) x 0.092 = 0.075 + 0.0069 = 0.082. Similarly, if a person with a vital capacity of 4 liters exhales 3 liters and a BrAC of 0.085 gm/210 liters, the corrected BrAC would be (0.085 gm/210 liters — ((3-2) x 0.085 x 0.092)) = 0.085 - .0078 = 0.077 gm/210 liters. Correction for expired volume differences can make a significant difference in cases in which the breath test reading is close to the legal limit. An alternative method would be to use isothermal rebreathing to get a better sample of alveolar alcohol concentration.[29]

Because BrAC correlates more closely to arterial BAC than it does to venous BAC over the absorption and elimination cycle and arterial BAC carrying alcohol to the brain determines intoxication, Jones et al.[30] have suggested that BrAC (closely correlated with arterial BAC) could be a better measure of impairment than venous BAC. This would be true if the biases in the breath alcohol were to be eliminated using the simple modifications to data analysis procedures described above.

Conclusion

In summary, BrAC increases with continued exhalation. BrAC at the end of exhalation never reaches alveolar alcohol concentration within a single exhalation. As currently used, the alcohol breath test causes two types of bias: lung volume bias and expired volume bias. Two minor changes in the analytical procedures are proposed for elimination of the biases due to expired volume uncertainty.

The alcohol breath test has been used for decades with an inappropriate design and underlying assumptions. The current design incorporates two types of bias, which disfavor subjects who exhale a greater breath volume and those with smaller lung volume. It is imperative that the breath test instrument manufacturers modify the software to eliminate the biases as soon as possible.

Notes

1. The scientific term for drinking alcohol is ethyl alcohol or ethanol.

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6. M. Hlastala & J. Anderson, *Alcohol Breath Test: Gas Exchange Issues*, 121 J. Appl. Physiol. 367-375 (2016).

7. *Id.*

8. A. Jones, *Determination of Liquid/Air Partition Coefficients for Dilute Solutions of Ethanol in Water, Whole Blood, and Plasma*, 7 J. Anal. Toxicol. 193-197 (1983).

9. Determined by averaging Jones' data for males and females; It is usually assumed that the $\lambda_{b:a}$ in the alveolus is identical to the $\lambda_{b:a}$ measured in vitro. For alcohol, the in vivo $\lambda_{b:a}$ may actually be greater than it is in vitro due to the Fahraeus effect (decreased hematocrit within the capillaries). But this is a small effect.

10. The 2100 correction is used in all breath alcohol test devices after field comparison studies of venous BAC and BrAC (without expired volume correction).

11. J.C. Anderson, A.L. Babb & M.P. Hlastala, *Modeling Soluble Gas Exchange in the Airways and Alveoli*, 31 Ann. Biomed. Eng. 1-21 (2003).

12. *Id.*

13. The volume of air breathed in and out when relaxed.

14. J. Anderson & M. Hlastala, *The Alcohol Breath Test in Practice: Effects of Exhaled Volume*, 126 J. Appl. Physiol. 1630-1635 (2019).

15. *Id.*

16. *Id.*

17. M.P. Hlastala, *The Alcohol Breath Test - A Brief Review*, 84 J. Appl. Physiol. 401-408 (1998).

18. M.P. Hlastala, *The Alcohol Breath Test - A Brief Review*, 84 J. Appl. Physiol. 401-408 (1998); 15. A. Jones, *Physiological Aspects of Breath Alcohol Measurement*, 6 Alcohol, Drugs and Driving 1-25 (1990); and A. Jones & L. Andersson, *Variability of the Blood/Breath Alcohol Ratio in Drinking Drivers*, 41 J. Forens. Sci. 916-921 (1996).

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25. Correcting for variation in expired volume does not eliminate the need to correct for variation in other factors affecting the BAC/BrAC relationship. M.P. Hlastala, *The Alcohol Breath Test - A Brief Review*, 84 J. Appl. Physiol. 401-408 (1998).

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