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Commentary on: Hlastala MP. Paradigm shift for the alcohol breath test. J Forensic Sci 2010;55(2):451–6

Sir,

Hlastala's suggested paradigm shift of the physiology of breath alcohol testing (1) should probably be more accurately described as a model refinement, as one of the central tenets of his model, the interaction of breath alcohol with the mucosal surfaces of the respiratory passages, has been known since at least 1964 (2). His contention that variations in blood and breath alcohol concentrations can be mainly explained by his lung physiology model is myopic as there are numerous other factors not included in his model that a forensic scientist must assess and determine their relevance for the drinking drivers tested by the police. Indeed, most of the anomalies and variability on the blood breath alcohol ratio (BBR) he cites can be explained by the following factors:

(1) Low Blood Alcohol Concentrations (BACs)

As for all ratios, the BBR has greater variability at low concentrations (3). Emerson and colleagues evaluated three breath alcohol testing instruments (Intoxilyzer 4011A, CMI, Inc., Owensboro, KY; GC Intoximeter Mk IV, Intoximeters, Inc., St. Louis, MO; and Breathalyzer model 1000, Smith & Wesson Co., Springfield, MA), and it is explained as follows:

> At most levels the instruments showed a tendency to underestimate the BAC which increased at the higher BAC levels. At levels below 60 mg/100 mL, all the instruments produced wide variations from the ideal line, as small differences in the absolute values of analyses at these levels produced proportionately larger percentage differences than the same differences at higher BACs (4).

Hlastala incorrectly cited this study as having been published in the *Journal of Forensic Sciences*, whereas it was actually published in the *Journal of the Forensic Science Society*.

Although not cited specifically by Hlastala, failure to understand the importance of BAC on the variability of BBR is illustrated by Alobaidi and colleagues. This paper is often used as an example of anomalies in the BBR (5). For example, in this study, a BAC of 0.007 g/100 mL and a corresponding BrAC of 0.010 g/100 mL results in a BBR of 1470:1 and a BAC of 0.037 g/100 mL with a corresponding BrAC of 0.027 g/100 mL yields a BBR of 2878:1. Because the average BAC of drinking drivers tested by the police is typically about 0.170 g/100 mL, such studies have limited forensic relevance (6,7).

(2) Mouth Alcohol

Mouth alcohol can affect the BrAC to a much greater extent than any lung physiology model and is controlled in the field by appropriate waiting periods before breath alcohol testing, duplicate breath testing agreement, and instrument slope detectors (8). In laboratory studies, however, with rapid drinking in a short time interval and breath alcohol testing commencing shortly thereafter, without the precautions employed in the field, mouth alcohol can cause anomalous BBRs. For example, in one study of 21 male subjects who consumed undiluted whiskey (9), the author later explains the large variability in BBRs obtained:

> The first breath tests were therefore made 10 min after the subjects finished drinking undiluted whisky while fasting. This suggests that measurements of alcohol in breath might have been artificially high because of the mouth alcohol effect. This could explain at least in part some of the low blood/breath ratios obtained at the 30-min sampling point (range 990–2280, CV = 14%). I purposely included these data to test the significance of this phenomenon, because it represents unfavorable conditions of breath analysis (10).

(3) Skewed Distributions of BBR

Hlastala then cites two papers by Simpson in which a simple two standard deviations error analysis was conducted on previously published studies, to calculate 95% confidence intervals (11,12). Jones' comment on Simpson's error analysis is as follows:

> Much of the criticism he makes of my work is unfounded and the conclusions he draws from them are wrong…In conclusion I consider that Simpson has deliberately presented a generally negative picture of the potential usefulness of breath alcohol analysis when used for law enforcement purposes (10).

Normal error analysis cannot be calculated on BBR distributions as these distributions are typically skewed to the higher BBRs (13,14). Other statistical methods such as the natural log transformation should be employed (15). Using his error analysis paradigm, Simpson (12) has calculated an absurdly high error for breath alcohol analysis of up to +200,000%.

(4) Arterio-venous Lag

It is well known that in the rising absorption phase, the venous BAC will lag behind the arterial BAC as reflected by the BrAC, causing anomalous BBRs in experimental studies when breath and blood samples are collected close to the time of the completion of rapid drinking (16,17). Arterio-venous lag has limited practical significance in the breath alcohol testing of drinking drivers by the police, as the vast majority (>95%) of drinking drivers are tested by the police in the postabsorptive phase of the BAC curve (18).

(5) Analytical Variables

Numerous analytical variables can also occur and should be evaluated by the forensic scientist, which can cause anomalous BBRs. Fundamentally, the breath alcohol instrument used in the various studies should have been extensively evaluated. Inaccurate, unreliable instruments will yield inaccurate, unreliable BBRs. For example, the Alobadi et al. (5) study was based on a helium–neon infrared analyzer that apparently had not been compared with standard and well-established breath testing equipment (9).

In addition, one of the breath alcohol testing instruments used in the Emerson et al. (4) study—the Breathalyzer 1000—has been found to be unreliable and tended to produce spikes and excessive deviant test results (19).

Other analytical variables such as how the BAC was reported can affect the BBR. The Emerson et al. (4) study did not report the mean BAC but used the RTA Certificate of Analysis which subtracted 6% and would cause the BBRs to be skewed to the lower ratios.

Although the blood alcohol result tends to be accepted as the "gold standard," BAC analysis is also susceptible to forensic variables, such as the presence of preservatives, storage conditions, continuity, type of sample (plasma, serum, or whole blood), and method of analysis. One interesting case showing anomalous BBRs was attributable to a problem with the blood sample, not the breath analysis (20). A large discrepancy was noted in two separate suspected drink-driving offenses in which the roadside breath tests were not consistent with the BAC, and typically it would be assumed to be attributable to errors in the BrAC. It transpired though that the usual alcohol-free skin swabs in the forensic blood collection kits had been replaced with Medi-Prep swabs containing n-propanol. Because the blood alcohol analysis in the forensic laboratory used n-propanol as an internal standard, the swabs apparently had contaminated the blood samples causing an inaccurate BAC and anomalous BBRs.

Thus, the large variability in BBRs presented by Hlastala need not be explained by a new lung physiology paradigm but can be explained by other factors well understood by the practicing forensic scientist. Hlastala uses his paradigm to predict that human subjects with smaller lung volumes (typically women and lighter weight persons) would obtain higher BrACs and hence lower BBRs than subjects with large lung volumes. He then cites the study of Jones and Andersson (21) as supporting this gender difference, but they actually found no statistically significant differences in BBRs in male or female subjects. This lack of gender difference has also been found in other studies of the BBR (22–24). Yet, based on Hlastala's lung physiology model alone, there should be significant differences between male and female subjects owing to the differences in their lung sizes.

It is also curious that Hlastala cites a study by Lindberg et al. (25) that shows a strong correlation between BrAC and arterial BAC as support for his "new" lung physiology paradigm but fails to indicate that this same study also found no difference in BBRs between male and female subjects, which again is contrary to this paradigm.

The only study that Hlastala cited for the effect of the weight of the human subject on the BBR was an abstract and not a peer-reviewed publication (26). The study was based on low BACs (0.020–0.060 g/100 mL) as Norway had lowered its legal BAC to 0.020 g/100 mL. The low BACs would increase the variability of BBRs as discussed earlier.

In conclusion, it appears the lung physiology paradigm shift of Hlastala may be based in part on shifting theoretical sands and that many other factors can influence the variability of blood and breath alcohol concentrations.

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