



Carbon Monoxide Meter: The Essential Clinical Tool – the ‘Stethoscope’ – of Smoking Cessation

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The first steps in any science involve observation and measurement. Our profession has long acknowledged the importance of independent assessments of smoking status. The tool most widely used to do this is the carbon monoxide (CO) monitor. Carbon monoxide monitors measure expired breath CO (and by calculation, the percentage of blood hemoglobin bound to carbon monoxide molecules) in an easy and noninvasive way (Jarvis, Russell, & Saloojee, 1980). CO is a gas produced during combustion of all organic matter (e.g., tobacco, petroleum products, home/office furnishings). CO is produced along with the approximately 4,000 other chemicals in tobacco smoke when cigarettes burn or combust. During each inhalation CO transverses across the pulmonary alveolar-blood capillary membrane where it binds with the hemoglobin in the red blood cells (erythrocytes). The resultant molecule is called Carboxyhemoglobin (COHb). High concentrations of COHb are fatal, lower concentrations are known to cause compensatory haemoconcentration, elevating erythrocytes, risking clotting and lowering oxygen carrying capacity. Expired breath CO correlates well with the percentage of blood COHb, 0.98 (Jarvis et al., 1980).

The topography of smoking (e.g., number of puffs, depth of inhalation, breath-holding, puff volume, inter-puff interval and velocity, and so on) contributes greatly to the expired CO level and blood nicotine concentrations. Indeed, smokers are able to titrate or ‘customise’ their smoking behavior to deliver just the level of nicotine needed.

Expired breath CO measurements have been used by researchers and clinicians for decades to validate self-reported abstinence in smoking cessation (SRNT, 2002) and exposure to second-hand smoke (Jarvis, Russell, &

Feyerabend, 1983). It is also used as a motivational tool — it is an inexpensive, tangible, personal and easily understood measurement of current smoking. Normal values found in non-smokers and ex-smokers differ greatly from active smokers (SRNT, 2002).

Studies also show that smoking topography changes with different types of cigarettes smoked and that compensatory smoking occurs while smoking (incorrectly labelled) ‘mild’ or ‘light’ cigarettes (Russell, 1990; Benowitz, Zevin, & Jacob, 1998). In the real world, ‘light cigarette’ compensatory smoking can precipitate puff dynamics that actually produce higher CO levels.

Carbon monoxide measurements are a useful teaching tool to disabuse smokers of the notion that these cigarettes are a safer or healthier option for them. In addition, baseline expired CO measurements are a valuable clinical tool in judging severity of dependence and likelihood of cravings during abstinence (West, 1985). There is some evidence that expired CO measurements correlate with levels of plasma nicotine and the severity of tobacco dependency (Lee, Malson, Waters, Moolchan, & Pickworth, 2003).

Genetic variation may also play a role in smoking topography. Strasser has recently shown that slower metabolisers of nicotine have significantly lower baseline expired CO levels. This implication is important for both treatment and risk assessment, as fast metabolisers convert tobacco specific procarcinogenic nitrosamines to carcinogens. These smokers are at greater risk of cancers (Strasser, Pickworth, Patterson, & Lerman, 2007).

Even prior to complete abstinence, clinicians may be able to assess efficacy of tobacco treatment medications by not only the decrease in cigarettes per day (CPD) but also perhaps the change in smoking topography that

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occurs while a smoker is still actively smoking but using pharmacotherapies. Often, reduction to cessation pharmacotherapy protocols (i.e., using medication(s) to gradually reduce their tobacco consumption prior to complete abstinence) will produce reductions that are measurable by CO monitoring. Benowitz (1998) has shown that nicotine intake is reduced when smoking concomitantly with NRTs.

Furthermore, our observations are that while using Varenicline for smoking cessation, decreases in expired CO may predict successful outcomes (Bittoun, Harrison, Baker, & Boukarim, 2008). Drops in weekly measurements of expired CO were significant predictors of future abstinence compared to no changes in weekly CO measurements. In our small pilot study of Varenicline-treated smokers, weekly expired carbon monoxide measurements demonstrated a statistically significant ($p > .074$) drop in expired CO in smokers who went on to spontaneously quit compared to smokers who did not. These results were unrelated to the numbers of cigarettes smoked per day at any time during the study. The difference between abstainers and nonabstainers occurred no earlier than two weeks after commencement of treatment and began to show significance at 3 weeks. These spontaneous abstainers began to quit at 4 to 7 weeks post treatment initiation. The use of serial carbon monoxide testing (more than numbers of cigarettes smoked per day) may be valuable as a clinical tool to assist in differentiating the Varenicline treatment resistant smoker from the smoker who may benefit from Varenicline.

Decreasing expired CO levels when smokers continue to smoke while using NRT may be encouraging to those smoking patients who may believe that this protocol may be detrimental. Motivation may improve when a smoker sees serial drops in expired CO as NRT is increased. This may explain why smoking while treated NRT may be a gateway to quitting (Fagerstrom, 2005).

In summary, with the rise of the professional status of tobacco treatment specialists (Hughes, 2007) carbon monoxide monitors should become the 'stethoscope' of our profession. Carbon monoxide monitors offers the tobacco treatment specialist an independent clinical tool that provides valuable evidence in identifying, educating, assessing and treating the tobacco dependent patient.

Disclosure or Conflicts of Interests

None.

References

- Benowitz N.L., Zevin, S., & Jacob, P. 3rd. (1998) Suppression of nicotine intake during ad libitum cigarette smoking by high-dose transdermal nicotine. *Journal of Pharmacology & Experimental Therapeutics*, 287(3) 958–962
- Bittoun, R., Harrison, D., Baker, J.J., & Boukarim, M.-L. (2008, October). *Serial drop in expired carbon monoxide, not numbers of cigarettes per day, may be a predictor of future abstinence with Varenicline*. Poster presentation at the SRNT, First Asian Regional Conference, Bangkok, Thailand.
- Fagerstrom, K.O. (2005) Can reduced smoking be a way for smokers not interested in quitting to actually quit? *Respiration*, 72, 216–220.
- Hughes, J. (2007). Tobacco treatment specialists: A new profession. *Journal of Smoking Cessation*, 2(Suppl.), 2–7.
- Jarvis, M., Russell, M.A.H., & Saloojee, A. (1980). Expired air carbon monoxide: A simple breath test of tobacco smoke intake. *British Medical Journal*, 281, 484–485.
- Jarvis, M., Russell, M.A., & Feyerabend, C. (1983) Absorption of nicotine and carbon monoxide from passive smoking under natural conditions of exposure. *Thorax*, 38(11), 829–833
- Lee, E.M., Malson, J.L., Waters, A.J., Moolchan, T.E., & Pickworth, W.B. (2003), Smoking topography: Reliability and validity in dependent smokers. *Nicotine and Tobacco Research*, 5(5) 673–679.
- Russell, M.A. (1990). Nicotine intake and its control over smoking. In S. Wonnacott, M.A.H. Russell, I.P., Stolerman, I.P. (Eds.), *Nicotine psychopharmacology: Molecular, cellular, and behavioural aspects* (pp. 375–418). Oxford: Oxford University Press .
- SRNT Subcommittee on Biochemical Verification. (2002). Biochemical verification of tobacco use and cessation. *Nicotine and Tobacco Research*, 4(2), 149–159.
- Strasser, A.A., Pickworth, W.B., Patterson, F., & Lerman, C. (2004) Smoking topography predicts abstinence following treatment with nicotine replacement therapy. *Cancer Epidemiology Biomarkers Prevention*, 13(11), 1800–1804.
- Strasser, A.A., Malaiyandi, V., Hoffmann, E., Tyndale, R., & Lerman, C. (2007). An association of CYP2A6 genotype and smoking topography. *Nicotine and Tobacco Research*, 9(4): 511–518.
- West, R.J., & Russell, M.A.H. (1985). Pre-abstinence smoke intake and smoking motivation as predictors of severity of cigarette withdrawal symptoms. *Psychopharmacology*, 87, 334–336.