[Topics in Practice Management]

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Use of Expired Air Carbon Monoxide Testing in Clinical Tobacco Treatment Settings

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Carbon monoxide (CO) testing is considered an easy, noninvasive, and objective contribution to the assessment of smoking behavior, as CO is rapidly absorbed into the bloodstream when lit cigarettes or cigars are inhaled. CO testing is a medically important billable outpatient service that can contribute to sustainability of face to face tobacco use treatment services by clinicians. This article reviews research on the clinical use of CO testing to provide biomedical feedback in assessing smoking behavior, educating smokers on tobacco health effects, assisting with treatment planning, and as a motivational tool to encourage people to become tobacco free. Further research can focus on how to best incorporate CO testing into clinical practice, including more research on outcomes and methods to ensure that insurers reimburse for testing and improved ways to use CO testing to initiate attempts to quit tobacco use, to maintain cessation, and to prevent relapse.

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Carbon monoxide (CO) is a clear odorless gas that reduces oxygen-carrying capacity in the blood. CO is produced by oxidation of carbon-containing compounds, such as those that occur with combustion of cigarettes or cigars. When smoke from combustible tobacco smoke is inhaled, CO is rapidly absorbed into the bloodstream. Although environmental sources of CO exist (eg, from incomplete combustion of carbon, such as motor vehicle exhaust, pollution, or malfunctioning furnaces during the winter[1,2]), tobacco smoking is the major source of elevated CO levels in the bloodstream. In contrast, CO_2 is the principal product of combustion of fossil fuels.

CO testing is considered an easy, noninvasive, and objective contribution to the assessment of smoking behavior.[3] CO testing measures the amount of CO in end-tidal expired breath as a marker for smoking status, and it is an increasingly important clinical tool for biochemically assessing and confirming smoking status and for use in counseling people who smoke cigarettes to quit smoking.[4,5] CO monitors measure the amount of CO expired in parts per million (ppm). The level of CO in ppm in the breath corresponds to the percent of carboxyhemoglobin or the percentage of blood cells carrying CO instead of oxygen.[4] Most CO in the blood is bound to

ABBREVIATIONS: CO = carbon monoxide; COHb = carboxyhemoglobin; %COHb = percent carboxyhemoglobin

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hemoglobin. The percentage of fetal carboxyhemoglobin (COHb)can also be calculated using maternal expired CO.[6] People who smoke cigarettes have higher levels of CO in expired breath, and CO monitors report both CO and COHb levels.[4]

As a billable outpatient service in the United States, CO testing can contribute to sustainability of face to face tobacco use treatment services. CO monitors can provide biomedical feedback to assess smoking behavior, educate smokers about tobacco health effects, assist with treatment planning, and serve as a motivational tool for people to become tobacco free. This paper reviews current use and research on CO testing in clinical tobacco treatment settings.

CO Assessment in Clinical Situations

CO testing is used in clinical and research settings as an objective noninvasive way to assess smoking behavior.[3] CO levels in expired air are correlated with levels of self-reported cigarette or cigar smoking.[3] CO has a half-life of 5 to 6 hours in the body,[7] and CO levels return to normal after 24 to 48 hours of not smoking.[1]

In research studies of people who smoke, CO levels $>$ 10 ppm in expired breath indicate current smoking, whereas CO levels $<$ 10 ppm will often be interpreted as evidence of smoking cessation.[7-9] CO measurements are also used clinically to predict severity of nicotine dependence and cravings for people attempting to quit.[4] One study found that CO levels in expired air could predict whether smokers with normal lung function had smoked in the past 8 hours.[7] CO levels at or $<$ 12 ppm predicted smoking abstinence in the previous 8 hours.[7] CO testing using these thresholds may not easily distinguish occasional smokers from nonsmokers.[8] Therefore, in some clinical settings, a threshold of 6 ppm provides additional assurance of complete abstinence.[1,10]

CO and COHb levels are also determined by several endogenous (eg, normal metabolism of heme proteins), environmental (eg, exposure to motor vehicle exhaust), product (eg, different types of combustible tobacco products), medical (eg, certain diseases), and individual factors. For instance, different methods of smoking tobacco products (eg, hookah, cigars, "little" cigars, and chillum [conical pipe] smoking) result in different and often higher levels of CO.[11] Smoking marijuana will elevate exhaled CO.[12] Patients with COPD, OSA, or asthma, as well as those who live in heavily urbanized areas, have higher CO levels, even if they do not smoke.[8,13,14] To assess smoking behavior in such patients, a cutoff point of 10 ppm for patients with asthma and 11 ppm for patients with COPD has been proposed.[15] Conflicting evidence exists about the presence of elevated levels of exhaled CO in patients with diabetes mellitus. One study found that exhaled CO levels were higher in people with diabetes and correlated with increases in blood glucose levels.[16] A more recent study found that this was not the case using newer CO monitors.[17] CO levels may be elevated in people with very high levels of secondhand smoke exposure (eg, in homes, cars, hookah bars), or exhaust from combustible materials (eg, a faulty furnace). This includes people whose occupations expose them to exhaust and smoke, such as firefighters, toll collectors, tractor drivers, road asphalt workers, and those who spend time along roads or in heavily polluted urban areas.[13,18]

Individual differences can also affect CO levels. For instance, a reduced effort (due to respiratory muscle fatigue or lack of desire, motivation, or ability to inhale and exhale deeply) will reduce CO levels.[19] Hyperventilation and exercise can also lower CO levels.[20] Although these factors may make it more difficult to make comparisons of CO levels across patients, consistently elevated levels within individual patients who use combustible tobacco products likely demonstrates higher exposure to the toxicity of these products.[8,10,18] Even if a clinician makes an adjustment for those with heavy environmental or endogenous exposure, the most frequent reason for a CO level to be high in someone who previously smoked is that they have returned to smoking, either socially or more heavily.[18] Some patients may be resistant to telling their providers they have returned to smoking out of fear of disappointment, judgment, or even shame.[21]

Higher levels of CO and nicotine in the blood may even correlate with the nature of smoking behavior. For example, depth of inhalation, number of puffs, and other measures of smoking topography can affect CO levels as much as the number of cigarettes smoked.[4,22] Some people reduce the number of cigarettes they smoke but smoke them more efficiently, resulting in similar CO levels.[4,22] CO levels fluctuate throughout the day, Q5 increasing after each cigarette and decreasing between cigarettes.[7,24] The effectiveness of some medications or products marketed as cessation aides can be assessed using CO testing. For instance, some have used CO testing to assess quitting of combustible tobacco products among groups of subjects who have switched to vaping and electronic cigarettes.[25] Even though a patient continues to smoke and use nicotine

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