

CHEST[®]

Official publication of the American College of Chest Physicians



Optimal Cutoff Level of Breath Carbon Monoxide for Assessing Smoking Status in Patients With Asthma and COPD

Susumu Sato, Koichi Nishimura, Hiroshi Koyama, Mitsuhiro Tsukino, Toru Oga, Takashi Hajiro and Michiaki Mishima

Chest 2003;124:1749-1754
DOI 10.1378/chest.124.5.1749

The online version of this article, along with updated information and services can be found online on the World Wide Web at:
<http://chestjournal.chestpubs.org/content/124/5/1749.full.html>

Chest is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2003 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook, IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder.
(<http://chestjournal.chestpubs.org/site/misc/reprints.xhtml>)
ISSN:0012-3692

A M E R I C A N C O L L E G E O F
 C H E S T
P H Y S I C I A N S[®]

Optimal Cutoff Level of Breath Carbon Monoxide for Assessing Smoking Status in Patients With Asthma and COPD*

Susumu Sato, MD; Koichi Nishimura, MD; Hiroshi Koyama, MD; Mitsuhiro Tsukino, MD; Toru Oga, MD; Takashi Hajiro, MD; and Michiaki Mishima, MD

Study objectives: To assess the optimal cutoff level of breath CO concentration to distinguish actual smokers from nonsmokers among patients with asthma and COPD.

Setting: Kyoto University Hospital outpatient clinic.

Subjects and methods: Three hundred thirty-one consecutive outpatients (161 with asthma and 170 with COPD) were examined cross-sectionally by self-reported smoking status, breath CO monitoring, and serum cotinine concentration. Actual smoking status was verified by serum cotinine concentration.

Results: Mean serum cotinine concentrations of never smokers, former smokers, and current smokers with asthma were 6.0 ± 5.2 ng/mL, 12.1 ± 25.0 ng/mL, and 198.3 ± 181.7 ng/mL, respectively (\pm SD). Mean serum cotinine concentrations of former smokers and current smokers with COPD were 23.2 ± 69.2 ng/mL and 191.1 ± 109.8 ng/mL, respectively. Mean breath CO levels of never smokers, former smokers, and current smokers with asthma were 6.1 ± 2.4 ppm, 7.7 ± 3.2 ppm, and 19.9 ± 17.3 ppm, respectively. Mean breath CO levels of former smokers and current smokers with COPD were 7.7 ± 4.3 ppm and 13.5 ± 6.5 ppm, respectively. The optimal cutoff level of breath CO to discriminate between actual smokers and nonsmokers was 10 ppm in patients with asthma and 11 ppm in patients with COPD, giving 85.0% and 73.1% sensitivity, and 85.8% and 84.7% specificity, respectively.

Conclusion: The optimal cutoff level of breath CO to assess actual smoking status was 10 ppm in patients with stable asthma and 11 ppm in patients with stable COPD. In patients with asthma and COPD, breath CO levels were potentially influenced by underlying airway inflammation, suggesting misclassification in the assessment of smoking status by breath CO.

(CHEST 2003; 124:1749–1754)

Key words: asthma; breath carbon monoxide; COPD; smoking status

Abbreviation: ROC = receiver operating characteristic

Cigarette smoking is the primary risk factor for COPD¹ and increases asthma severity.² Smoking cessation is important in treatments of these patients.³ In a clinical setting, it is necessary to monitor patient smoking status exactly and to advise patients to abstain from smoking.

Self-reporting of smoking habits is widely used to

estimate the prevalence of cigarette smoking, although it has been shown to underestimate smoking status.^{4,5} It has been suggested that the increasing social unacceptability of smoking may result in underreporting.⁶ Since self-reported smoking status may not be reliable in a clinical setting, a number of biochemical markers have been used to evaluate smoking status, including measures based on thiocy-

*From the Departments of Respiratory Medicine (Drs. Sato, Tsukino, and Mishima) and General Medicine and Clinical Epidemiology (Dr. Koyama), Graduate School of Medicine, Kyoto University, Kyoto; Respiratory Division (Drs. Nishimura and Oga), Kyoto-Katsura Hospital, Kyoto; and Department of Pulmonary Diseases (Dr. Hajiro), Kobe Nishi City Hospital, Kobe, Japan.

Manuscript received November 15, 2002; revision accepted June 26, 2003.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (e-mail: permissions@chestnet.org).

Correspondence to: Susumu Sato, MD, Department of Respiratory Medicine, Graduate School of Medicine, Kyoto University, 53, Kawahara, Shogoin, Sakyo-ku, Kyoto, 606-8507, Japan; e-mail: ssato@kuhp.kyoto-u.ac.jp

anate, nicotine, cotinine, and CO. These measures differ widely in availability, cost, and ease of administration.

The measurement of breath CO level may provide an immediate, noninvasive method of assessing smoking status.⁷ The development of relatively inexpensive portable CO monitors enables breath CO levels to be measured in a range of clinical settings. A breath CO level of 10 ppm is usually taken as the cutoff between smokers and nonsmokers. However, several studies^{8–11} have shown that a cutoff level of 6 or 8 ppm is more appropriate. Accordingly, breath CO monitoring devices have been updated, and the cutoff level has recently been lowered to 8 ppm.

It is known, however, that breath CO levels may be elevated due to airway inflammation. Exhaled CO levels in asthmatic patients have been reported to be high and to decrease with corticosteroid therapy.¹² As such, the breath CO level may reflect intrinsic generation of CO due to oxidative stress in patients with COPD.^{13,14} The aims of this study were to assess the sensitivity, specificity, and reliability of breath CO monitoring, and also to assess the optimal cutoff level to distinguish smokers from nonsmokers in patients with stable asthma and COPD.

MATERIALS AND METHODS

Subjects

Each patient who regularly attended the Kyoto University Hospital outpatient clinic and had a diagnosis of COPD or bronchial asthma was enrolled consecutively. The diagnosis was based on the definition provided by the American Thoracic Society.^{15,16} Entry criteria for patients with COPD included the following: (1) FEV₁/FVC ratio of < 0.7 and FEV₁ of < 80% of the predicted value for all measurements made after inhalation of prescribed bronchodilator during the previous 6 months, (2) smoking history of > 20 pack-years, and (3) no history suggestive of asthma. For patients with asthma, criteria included the following: (1) confirmation of the presence of airway hyper-responsiveness (AHR) on the initial visit, and (2) a best ratio of FEV₁ to FVC of > 0.7 after treatment including inhaled or systemic corticosteroids or bronchodilator when a subject was a current or former smoker to exclude COPD. All participants had > 6 months of outpatient management before entry, no exacerbations of airflow limitation over the preceding 6 weeks, and no changes in treatment regimen over the preceding 4 weeks. All eligible asthmatic patients had undergone treatment with inhaled beclomethasone dipropionate. Verbal informed consent was obtained from all patients.

Methods

All patients underwent the following examination on the same day. Smoking habits were assessed during the examination by interview. Current smokers were defined as subjects who reported current, regular use of cigarettes. Former smokers were defined as subjects who had refrained from smoking for \geq 6 months. Never smokers were defined as subjects who reported

never using cigarettes. "Nonsmokers" in the content of this study means never smokers plus former smokers. Furthermore, patients with a history of nicotine patch or gum use and patients using these aids at the time of the study were excluded.

Breath CO monitoring was performed using a MICRO III Smokerlyser (Bedfont Instruments; Kent, UK), an inexpensive, portable CO monitor that has previously been shown to be useful.¹⁷ The subjects were asked to exhale completely, inhale fully, and then hold their breath for 15 s. If the subjects were unable to hold their breath for 15 s, they were asked to hold it for as long as possible. Following breath holding, the subjects were asked to exhale slowly into the Smokerlyser and were encouraged to exhale fully in order to sample the alveolar air.

All subjects had blood drawn for measurement of serum cotinine concentration and measurements were performed by gas liquid chromatography. To determine "actual" smokers, the cutoff level used was a serum cotinine concentration of > 50 ng/mL.¹⁸ The sensitivity and specificity of breath CO monitoring were calculated on the basis of cotinine levels.

Spirometric testing to determine FEV₁ and FVC was performed in accordance with the method recommended by the American Thoracic Society,¹⁹ using a spirometer (AUTOSPIRO AS-600; Minato Medical Science; Osaka, Japan), which was calibrated with a 3.0-L syringe. The predicted values for the pulmonary function indexes were those proposed by the Japan Society of Chest Diseases.²⁰

Statistical Analysis

Statistical analyses were done using StatView (SAS Institute; Cary, NC). The results are expressed as mean \pm SD, unless otherwise stated. Comparisons between groups classified according to smoking status were made by the Fisher protected least-squares difference test. A discriminant analysis was performed to determine appropriate cutoff values, using receiver operating characteristic (ROC) curves.²¹ Spearman rank correlation coefficient was used to test the relationships between breath CO and other variables; $p < 0.05$ was considered statistically significant for all analyses.

RESULTS

A total of 331 eligible patients (161 with asthma and 170 with COPD) were consecutively enrolled (Table 1), representing 250 men and 81 women. None of the patients enrolled in the study had used nicotine gum or nicotine patches. The mean serum cotinine concentrations of never smokers, former smokers, and current smokers with asthma were 6.0 ± 5.2 ng/mL, 12.1 ± 25.0 ng/mL, and 198.3 ± 181.7 ng/mL, respectively (\pm SD). The mean serum cotinine concentrations of former smokers and current smokers with COPD were 23.2 ± 69.2 ng/mL and 191.1 ± 109.8 ng/mL, respectively. The mean breath CO levels of never smokers, former smokers, and current smokers with asthma were 6.1 ± 2.4 ppm, 7.7 ± 3.2 ppm, and 19.9 ± 17.3 ppm, respectively. The mean breath CO levels of former smokers and current smokers with COPD were 7.7 ± 4.3 ppm and 13.5 ± 6.5 ppm, respectively. Compared with nonsmokers (*ie*, former smokers and never smokers), current smokers showed

Table 1—Characteristics of 331 Outpatients With Asthma and COPD*

Characteristics	Asthma (n = 161)	COPD (n = 170)
Age, yr	46.5 ± 16.1 (15–78)	68.0 ± 6.9 (48–89)
Male/female gender, No.	84/77	166/4
FEV ₁ , L	2.54 ± 0.86	1.07 ± 0.51
FEV ₁ , % predicted	88.7 ± 19.4	40.3 ± 18.2
Self-reported smoking status, No. (%)		
Current	22 (13.6)	48 (28.2)
Former	55 (34.2)	122 (71.8)
Never	84 (52.2)	0

*Data are presented as mean ± SD or mean ± SD (range) unless otherwise indicated.

higher levels of both breath CO and serum cotinine ($p < 0.0001$). Among former smokers, there were no differences between asthma and COPD patients in either breath CO or serum cotinine levels.

The distributions of breath CO and serum cotinine levels for each group are shown in Figure 1. None of the never smokers showed serum cotinine levels exceeding 50 ng/mL. Among current smokers, 59 patients (84.3%) showed serum cotinine levels > 50 ng/mL but 11 patients (4 with asthma and 7 with COPD) had levels < 50 ng/mL. Among former smokers, 164 patients (92.7%) had serum cotinine levels < 50 ng/mL, but 13 patients (2 with asthma and 11 with COPD) had levels > 50 ng/mL.

The sensitivity and specificity of breath CO monitoring were calculated at various cutoff values, and ROC analyses were performed. Among all patients with asthma, a cutoff value of 10 ppm for breath CO was optimal, giving 85.0% sensitivity and 85.8% specificity (Fig 2). Among all patients with COPD, 11 ppm of breath CO was optimal, giving 73.1% sensitivity and 84.7% specificity.

DISCUSSION

Ten parts per million is the traditional cutoff level to discriminate between current smokers and nonsmokers, but previous studies^{8–11} have shown that 6 to 8 ppm was optimal. The American Thoracic Society statement in 2001 suggested that 6 ppm was the optimal cutoff level and that 8 ppm or 10 ppm may be relatively higher cutoff points.²² In the present study, among stable asthma or COPD patients, the optimal cutoff level of breath CO to assess smoking status was 10 ppm or 11 ppm, respectively.

Nineteen subjects with asthma claimed to be nonsmokers and yet had breath CO levels of > 10 ppm in the present study. Twenty-four subjects with COPD claimed to be nonsmokers and showed breath CO levels of > 11 ppm. Eleven of these patients (2 with asthma and 9 with COPD) had serum cotinine levels > 50 ng/mL. The latter would be strongly suspected to be actual current smokers who were denying their habits. Self-report classification misclassified them as nonsmokers. The inci-

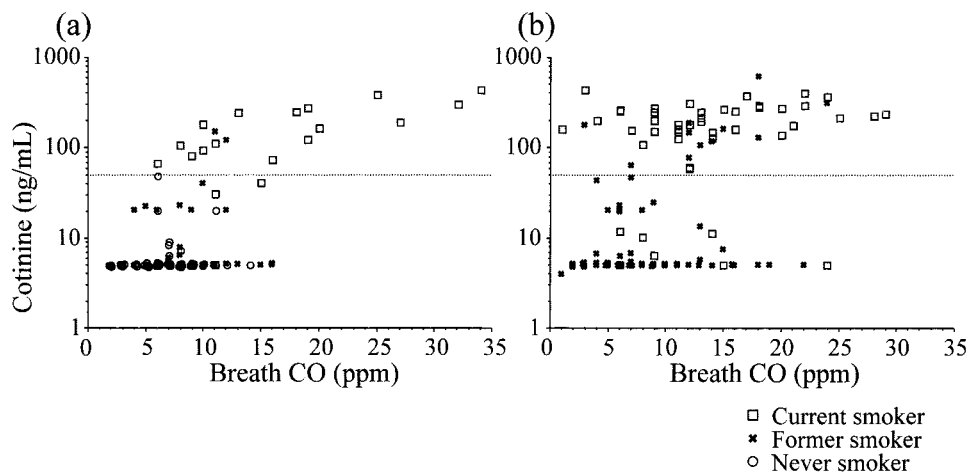


FIGURE 1. Distribution of breath CO and serum cotinine levels in all patients with asthma (left, a) and all patients with COPD (right, b).

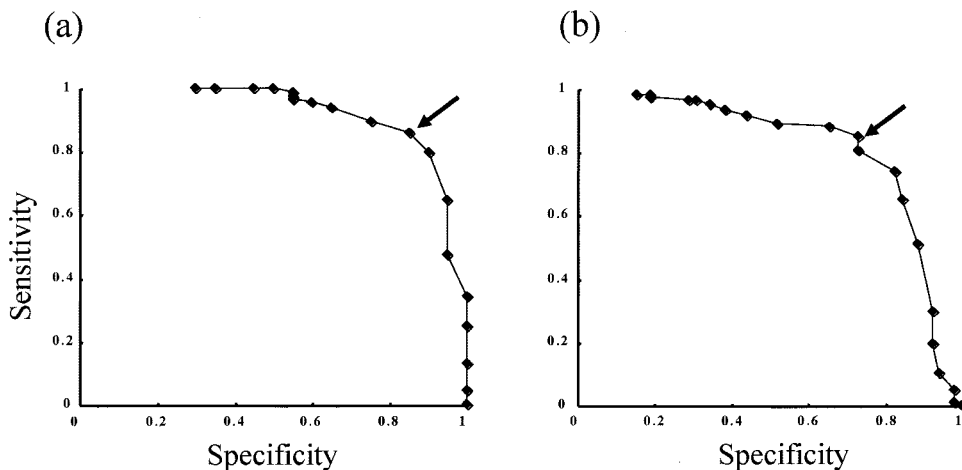


FIGURE 2. ROC curve that discriminates the optimal cutoff level of breath CO. Rhombuses indicate the cutoff level of breath CO to range from 0 to 21 ppm. In all patients with asthma (*left, a*), 10 ppm was determined to be the optimal cutoff level; in COPD patients (*right, b*), 11 ppm was optimal. Arrow indicates optimal cutoff points on each ROC curve.

dence of misreporting of smoking status was relatively low in the literature,^{4,5} and would be influenced by patients knowledge of biovalidation, such as breath CO or serum cotinine monitoring.

However, the other patients (17 with asthma and 15 with COPD) were apparently genuine nonsmokers, who would be potentially misinterpreted as current smokers on the basis of breath CO monitoring. Passive smoking may elevate serum cotinine levels, but breath CO can also be elevated in this situation. The low serum cotinine and high breath CO shown by same patients may also be attributable to underlying airway inflammation and oxidative stress.^{12,23} At the cutoff level of 10 ppm or 11 ppm, only two patients were misclassified, apparently denying their smoking habits but showing serum cotinine levels > 50 ng/mL. If the cutoff level was lower, more nonsmoking patients may be misclassified as smokers. Using breath CO monitoring, we could detect 11 subjects (84.6%) who “actually” misreported their current smoking habit; however, 32 subjects (12.9%) who were “actual” nonsmokers had been considered as current smokers. Breath CO monitoring is an easy, noninvasive, immediate, and quite sensitive method to detect current smoking; however, it tends to provide pseudo-positive results in assessing patients with asthma or COPD.

Previous studies showed that cutoff levels of 6 ppm or 8 ppm were valid. Edward et al⁸ reported that 6 ppm was appropriate. While the latter study examined respiratory patients, the underlying diseases were not represented. Crowley et al⁹ also reported that a breath CO level of > 8 ppm was strongly associated with a self-report of current smoking. They studied only patients with COPD,

however, and their survey included patients receiving home oxygen therapy, which might decrease breath CO levels. Moreover, determination of smoking status was dependent on self-reporting. Jarvis et al¹⁰ reported that the optimal cutoff was 8 ppm, giving 90% sensitivity and 89% specificity among outpatients. Terao et al¹¹ reported that the optimal cutoff points were between 6 ppm and 7 ppm for men, and 5 ppm and 6 ppm for women with a sensitivity and a specificity of approximately 90%. The subjects of these studies were not exclusively to patients with asthma or COPD. The present study showed that the optimal cutoff level of breath CO was higher than indicated in previous studies.^{8–11}

It has been reported that breath CO levels may be raised as a result of certain inflammatory lung diseases, such as bronchial asthma and bronchiectasis, with mean values of approximately 7 ppm.^{12,22} These elevations in breath CO levels due to the intrinsic generation of CO associated with oxidative stress, not only in respiratory diseases, but other diseases such as diabetes.²⁴ In the present study, the mean breath CO level of self-reported never smokers with asthma was 7.7 ppm, and 6 of these subjects showed levels of breath CO > 10 ppm. All of these patients had cotinine levels < 50 ng/mL. These results are consistent with other studies.^{12,14} In the evaluation of breath CO levels in asthma and COPD in a clinical setting, the influence of airway inflammation should be considered.

Although serum cotinine is considered to be the best marker in discriminating smokers and nonsmokers,²⁵ 11 current smokers were misclassified as nonsmokers on the basis of serum cotinine levels. Serum cotinine and breath CO may also be influenced by a patient’s smoking style. All of them reported smok-

ing > 10 cigarettes daily, but their smoking styles were unclear. They may smoke infrequently, and inhale little or may have stopped several days before examination, resulting in lowered serum cotinine levels. The half-life of plasma cotinine and alveolar CO are reported to be 6 to 16 h and 150 min, respectively.^{26,27} Six current smokers (three with asthma and three with COPD) who showed low cotinine levels and high breath CO might smoke only rarely but may have smoked recently.

The present study has certain limitations. We did not examine normal subjects, and did not obtain patient history regarding passive smoking, last cigarette smoking, or smoking patterns. These factors may influence breath CO levels and may decrease the sensitivity and specificity of breath CO monitoring. Moreover, airway obstruction may influence levels of exhaled CO. Togores et al²⁸ reported that exhaled CO measurements can be inaccurate in patients with severe airflow obstruction. In the present study, there was no significant negative correlation between breath CO and FEV₁ (percentage predicted) in nonsmokers.

Serum cotinine was used as a standard to determine smoking status. Cotinine is considered as the best choice for research protocols in which accurate categorization is essential.²⁵ However, 11 patients (15.7%) reported to be current smokers were misclassified on the basis of serum cotinine levels. Reasons for misclassification may include the time of day of sample collection, cigarette consumption for nicotine content, and the higher cutoff value of serum cotinine used in the present study. However, additional analyses were performed using lower cutoff value (20 ng/mL) of serum cotinine in accordance with the standard by the Foundation for Blood Research and showed quite similar result for the optimal cutoff value of breath CO, though the area under the curve of ROC was smaller than that obtained using the value of 50 ng/mL.

Breath CO monitoring provides an easy, noninvasive, and immediate way of assessing patient smoking status, in contrast to the measurement of serum cotinine, which is invasive, expensive, and does not permit immediate assessment. Breath CO, however, is potentially influenced by many factors, including oxidative stress, air pollution, passive smoking, the subject's clumsiness in using the measuring device, the period of time after last smoking, the depth of smoke inhalation, the pattern of puffing, the butt length of cigarettes smoked, and so on.

CONCLUSIONS

In the present study, 10 ppm in asthmatic patients and 11 ppm in patients with COPD were determined

to be the optimal cutoff level of breath CO to distinguish actual smokers from nonsmokers. These cutoff levels are higher than those reported in previous studies.⁸⁻¹¹ These differences may be attributable to elevations in basal breath CO levels due to airway inflammation and oxidative stress. As such, adjustments in the cutoff level of breath CO for distinguishing current smokers or nonsmokers may have to be considered. Moreover, other methods of biochemical monitoring may be required to assess smoking status such as the measurement of cotinine, if patients with asthma and COPD show slightly higher breath CO levels but claim to be nonsmokers.

Breath CO monitoring is quite useful, but in patients with airway inflammatory diseases, such as asthma and COPD, we may be at risk of misclassification in the assessment of smoking status. As such, it may be necessary to consider other factors in addition to cigarette smoking.

REFERENCES

- 1 Pauwels RA, Buist AS, Calverley PMA, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. *Am J Respir Crit Care Med* 2001; 163:1256-1276
- 2 Siroux V, Pin I, Oryszczyn MP, et al. Relationship of active smoking to asthma and asthma severity in the EGEA study. *Eur Respir J* 2000; 15:470-477
- 3 Paul DA, John EC, Lance AW, et al. Smoking cessation and lung function in mild-to-moderate chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2000; 161:381-390
- 4 Coultas DB, Howard CA, Peake GT, et al. Discrepancies between self-reported and validated cigarette smoking in a community survey of New Mexico Hispanics. *Am Rev Respir Dis* 1988; 137:810-814
- 5 Colletti G, Supnick JA, Abueg FR. Assessment of the relationship between self-reported smoking rate and Ecolyzer measurement. *Addict Behav* 1982; 7:183-188
- 6 Sillett RW, Wilson MB, Malcolm RE, et al. Deception among smokers. *BMJ* 1978; 2:1185-1186
- 7 Nicholas JW, Marianne I, Jillian B. Carbon monoxide in breath in relation to smoking and carboxyhaemoglobin levels. *Thorax* 1981; 36:366-369
- 8 Edward T, Middleton B, Alyn H, et al. Breath carbon monoxide as an indication of smoking habit. *Chest* 2000; 117:758-763
- 9 Crowley TJ, Andrew AE, Cheney J, et al. Carbon monoxide assessment of smoking in chronic obstructive pulmonary disease. *Addict Behav* 1989; 19:121-126
- 10 Jarvis MJ, Tunstall-Pedoe H, Feyerabend C, et al. Comparison of tests used to distinguish smokers from non-smokers. *Am J Public Health* 1987; 77:1435-1438
- 11 Terao A, Konishi M, Baba S, et al. Exposure to tobacco smoke in a Japanese urban population: an analysis using biochemical markers of smoking [in Japanese]. *Nippon Koshu Eisei Zasshi* 1998; 45:3-14
- 12 Zayasu K, Sekizawa K, Okinaga S, et al. Increased carbon monoxide in the exhaled air of asthmatic patients. *Am J Respir Crit Care Med* 1997; 156:1140-1143

- 13 Repine JE, Bast A, Lankhorst I. Oxydative stress in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1997; 156:341-357
- 14 Rahman I, Morrison D, Donaldson K, et al. Systemic oxidative stress in asthma, COPD, and smokers. *Am J Respir Crit Care Med* 1996; 154:1055-1060
- 15 National Asthma Education and Prevention Program. Expert panel report 2: guidelines for the diagnosis and management of asthma. Bethesda, MD: National Institute of Health, 1997; Publication No. 97-4051
- 16 American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1995; 152:S77-S120
- 17 Waage H, Silsand T, Urdal P, et al. Discrimination of smoking status by thiocyanate and cotinine in serum, and carbon monoxide in expired air. *Int J Epidemiol* 1992; 21:488-493
- 18 Establishing a nicotine threshold for addiction: the implications for tobacco regulation. *N Engl J Med* 1994; 331:123-125
- 19 Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. Standardization of spirometry. *Am J Respir Crit Care Med* 1995; 152:1107-1136
- 20 Japan Society of Chest Diseases. The predicted values of pulmonary function testing in Japanese [in Japanese]. *Jpn J Thorac Dis* 1993; 31:Appendix
- 21 Zweig MH, Campbell G. Receiver-operating characteristic (ROC) plots: a fundamental evaluation tool in clinical medicine [published erratum appears in *Clin Chem* 1993 Aug; 39(8):1589]. *Clin Chem* 1993; 39:561-577
- 22 Kharitonov SA, Barnes PJ. Exhaled markers of pulmonary disease. *Am J Respir Crit Care Med* 2001; 163:1693-1722
- 23 Horvath I, Loukides S, Wodehouse T, et al. Increased levels of exhaled carbon monoxide in bronchiectasis: a new marker of oxidative stress. *Thorax* 1998; 53:867-870
- 24 Paolo P, Wojciech B, Giovanni I, et al. Exhaled carbon monoxide levels elevated in diabetes and correlated with glucose concentration in blood. *Chest* 1999; 116:1007-1011
- 25 Benowitz NL. Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev* 1996; 18:188-204
- 26 Darby TD, McNamee JE, van Rossum JM. Cigarette smoking pharmacokinetics and its relationship to smoking behavior. *Clin Pharmacokinet* 1984; 9:435-449
- 27 Russell MA, Wilson C, Patel UA, et al. Comparison of effect on tobacco consumption and carbon monoxide absorption of changing to high and low nicotine cigarettes. *BMJ* 1973; 4:512-516
- 28 Togores B, Bosch M, Agusti AG, et al. The measurement of exhaled carbon monoxide is influenced by airflow obstruction. *Eur Respir J* 2000; 15:177-180

NetWorks

NetWorks Make the Difference... You Can Too!

NetWorks are interdisciplinary, special interest groups providing the opportunity for personal involvement in the ACCP. NetWorks provide an outlet for action on a national level, establishing forums for advocacy, leadership, communication, and education. You can help make a difference by becoming involved in any one of the ACCP NetWorks. For more details on NetWorks, visit ChestNet at www.chestnet.org/networks/.

Are you NetWorked yet?
Join the NetWork of your choice today!
e-mail: networks@chestnet.org
phone: Marla Brichta: 847-498-8364
Ellynn Shapiro: 847-498-8332



Optimal Cutoff Level of Breath Carbon Monoxide for Assessing Smoking Status in Patients With Asthma and COPD

Susumu Sato, Koichi Nishimura, Hiroshi Koyama, Mitsuhiro Tsukino, Toru Oga, Takashi Hajiro and Michiaki Mishima

Chest 2003;124; 1749-1754
DOI 10.1378/chest.124.5.1749

This information is current as of January 6, 2012

Updated Information & Services

Updated Information and services can be found at:

<http://chestjournal.chestpubs.org/content/124/5/1749.full.html>

References

This article cites 26 articles, 18 of which can be accessed free at:

<http://chestjournal.chestpubs.org/content/124/5/1749.full.html#ref-list-1>

Cited By

This article has been cited by 3 HighWire-hosted articles:

<http://chestjournal.chestpubs.org/content/124/5/1749.full.html#related-urls>

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:

<http://www.chestpubs.org/site/misc/reprints.xhtml>

Reprints

Information about ordering reprints can be found online:

<http://www.chestpubs.org/site/misc/reprints.xhtml>

Citation Alerts

Receive free e-mail alerts when new articles cite this article. To sign up, select the "Services" link to the right of the online article.

Images in PowerPoint format

Figures that appear in *CHEST* articles can be downloaded for teaching purposes in PowerPoint slide format. See any online figure for directions.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]