Exhaled Carbon Monoxide in COPD

To the Editor:

I read with interest the article by Montuschi et al (August 2001). However, their study design might not be appropriate. The number of ex-smokers (n = 15) is too small. According to Figure 1 in their article, only 2 of 15 ex-smokers showed high exhaled carbon monoxide. It is well known that some ex-smokers deceive doctors about their smoking. Montuschi et al mentioned that smoking habit was checked by the measurement of urinary cotinine levels (data not shown). But it is not enough.

The measurement of carboxyhemoglobin (COHb) concentration in blood is an effective means of ascertaining the smoking status of patients. They should have checked the blood COHb concentrations of the subjects. If those two ex-smokers with high exhaled carbon monoxide levels showed high COHb, they were deceivers.

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REFERENCES


To the Editor:

Regarding the comment by Hiroshi Kawane, I would like to make the following points:
1. Measurement of urinary cotinine is a well-accepted method to assess smoking exposure. Measurement of carboxyhemoglobin may be helpful, but it is not necessary for excluding smoking exposure.
2. Patients with the highest exhaled carbon monoxide values had a negative urinary cotinine test result, indicating that they were not exposed to smoking. The high carbon monoxide values are likely to reflect the interindividual biological variability.
3. Even taking out the two highest exhaled carbon monoxide values, the difference between healthy nonsmokers and COPD patients who were ex-smokers is still significant (3.1 ± 0.3 ppm vs 4.7 ± 0.4 ppm, p < 0.01).
4. We do not agree that the study design was not appropriate. It was a cross-sectional study including 15 patients who were ex-smokers. The number was small, but sufficient to detect differences.

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REFERENCES


Vasopressin and Cardiac Performance

To the Editor:

We read with interest the review by Holmes and associates in CHEST (September 2001). Their article prompted us to review our experience with this agent. We retrospectively identified patients treated with vasopressin for septic shock at our institution from January 2000 to June 2001. These seven patients had a median age of 63 years and a mean (± SD) APACHE (acute physiology and chronic health evaluation) II score of 28.2 ± 11.7. All patients demonstrated a drop in cardiac index with use of vasopressin (mean dosage, 0.08 ± 0.06 U/min). The cardiac index was 3.9 ± 2.4 L/min/m² prior to start of the infusion and 2.7 ± 1.9 L/min/m² 4 h after initiation (p = 0.014). The stroke volume index declined similarly (31.6 ± 17.1 mL/beat/m² vs 22.9 ± 17.3 mL/beat/m²; p = 0.032). No significant changes in heart rate were observed. Notably, six patients subsequently required the addition of inotropic agents (dopamine in four patients and milrinone in two patients). As the article suggests, while vasopressin appears to be an effective vasoconstrictor, the potential for a decrease in cardiac output should be anticipated with use of this agent. However, as our data show, such decreases may not be solely rate related.

In addition, we submit that the literature on cardiac performance with vasopressin in patients with septic shock is not entirely as uniform as the authors imply. For example, the authors describe a study by Malay et al, in which vasopressin exerted no significant effect on cardiac index. Tsuneyoshi et al recently reported 16 patients with septic shock treated with continuous vasopressin infusion in whom no significant change in cardiac index was noted. Both of these trials utilized “physiologic” (0.04 U/min) doses of vasopressin, which might theoretically minimize the potential for coronary ischemia, and excluded patients with baseline myocardial dysfunction. While these are small studies, the variable response of cardiac output to vasopressin demonstrated thus far in human trials likely indicates that septic shock is sufficiently heterogeneous that predicting individual cardiac responses to such infusions is not straightforward. In particular, while use of vasopressin appears to allow for withdrawal of other vasoactive medications, in patients with sepsis-associated myocardial depression, addition of inotropes may be required. Randomized, controlled trials will be instrumental in assessing both the efficacy of and specific indications for vasopressin in patients with septic shock.

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