Oxidative Stress and Cardiovascular Complications in Sleep Apnea

To the Editor:

A recent letter in CHEST (May 2004) by Teramoto and colleagues1 on the role of interleukin (IL)-6 in the pathogenesis of obesity and obstructive sleep apnea (OSA) supported our findings suggesting this measurement for screening and monitoring OSA.2 More importantly, their data show a good correlation between plasma concentrations of proinflammatory cytokines (IL-6 and tumor necrosis factor-α) and C-reactive protein (CRP) as markers of cardiovascular complications. This supports the idea that inflammatory processes are activated in atherosclerotic lesions in patients with OSA. We demonstrated that in these subjects, 8-isoprostanone levels were elevated either in plasma or in exhaled breath condensate, indicating increased oxidative stress in OSA.2

Several mechanisms may be responsible for the oxidative-antioxidative imbalance in OSA patients, including recurrent hypoxia and reoxygenation during sleep, the increase in adrenergic activity, the marked reduction in rapid eye movement, and the continuous damage of upper airway mucosa as a result of recurrent mechanical obstruction during the sleep.3 In addition, we showed a good correlation between IL-6 and 8-isoprostanone. Proinflammatory cytokines, CRP, and oxidative stress could act in synergy to explain the acceleration in atherosclerosis progression, and the high frequency of cardiovascular consequence in OSA patients. Because continuous positive airway pressure treatment reduces the levels of IL-6, CRP, and, as we showed, 8-isoprostanone, this therapy may suppress the systemic and local inflammatory and oxidative-antioxidative imbalance in OSA, and thus reduce the progression of atherosclerotic lesions and reduce cardiovascular complications in OSA patients.

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Using Barrier Filters To Protect Spirometer Sensors From Droplet Deposition

To the Editor:

I read with great interest the recent article by Townsend et al1 (May 2004), who detail the problems of zero errors and sensor obstruction with flow-type spirometers during lung function tests. It is very astonishing that in those screening programs, a dozen of “grossly overestimated” FVC results (160 to 316% of predicted) had been accepted by some professionals. The authors alert the relevant spirometer users to the potential errors of flow-type spirometers and highlight the importance of quality control in lung function tests.

I agree with most of the authors’ recommendations, and would like to comment on recommendation 6. I think their recommendation, that if “one sensor is used for all subjects, it should be cleaned frequently,” is somewhat ambiguous, because neither the authors nor most spirometer manufacturers have actually specified the cleaning frequency of spirometer sensors (eg, being cleaned between subjects, daily, or weekly). With my experience, it is relatively easy to identify zero errors, but it is very difficult (if not impossible) to identify the problems of subtle partial blockage by droplets or water vapor condensation in pneumotachometers, just as pointed out by the authors as “elevating a subject’s 90% of predicted to 110% or inflating 70% of predicted to 90% is unlikely to cause suspicions of equipment errors.” I believe we should do everything possible to protect spirometer sensors from droplet deposition in lung function testing. The most practical and effective way would be the application of a single-use barrier filter (with high filtration efficiency rate, low resistance, and small dead space) in every spirometer for each subject. If so, we could easily solve the partial blockage problems in a spirometer with a heated flow-sensor, because a subject’s droplets of saliva and mucus could be completely captured by the barrier filter and the condensation of water vapor would not be formed. For a spirometer with an unheated flow sensor, flushing air through the sensor by a calibration syringe or an unheated hairdryer after each subject’s use could remove condensation. More importantly, cross-infection risks can be minimized during lung function tests by using the single-use barrier filter for each subject.

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Eosinophilic Pneumonia and Arthritis

To the Editor:

We read with great interest the recent case report in CHEST (September 2004)1 by Norman et al, in which eosinophilic...
pneumonia preceded the establishment of rheumatoid arthritis (RA). We would like to add some comments to that precious case report. RA, a polarized disease of representative T helper (Th) type 1 cells, is less compatible with the coexistence of allergic disorders with a Th2 cytokine pattern than with other inflammatory diseases. However, previous studies have demonstrated the coexistence of Th2 diseases, such as bronchial asthma and atopic dermatitis, and Th1 diseases, such as RA, insulin-dependent diabetes mellitus, and celiac disease. Kero and colleagues have indicated that the presence of Th2 diseases in subjects with Th1 diseases may be related to environmental factors. The most recognized and important effector cells in Th2 diseases are eosinophils. We are, therefore, interested in the chemotactic factors of eosinophils in the patient reported by Norman and colleagues. Immunohistochemical examinations of Th2 cytokines, including interleukin (IL)-4 and IL-5, and chemokines such as eotaxin in lung tissue specimens from the patient would provide further important information.

We are also concerned about the character of the arthritis in the patient. There are few case reports of eosinophilic synovitis in the literature. If Norman and colleagues collected synovial fluid from the patient, was eosinophilia detected in the fluid samples? Finally, we think that Figure 2 in the article by Norman et al cannot clearly identify the infiltration of eosinophils. We often stain the bronchoalveolar fluid and lung tissue specimens (Diff-Quik; International Reagents; Kobe, Japan) to determine the differential cell counts. Thus, staining of the lung specimens might have yielded a different image.

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Noninvasive Imaging for the Postoperative Assessment of Aortic Coarctation Patients

To the Editor:

We read with great interest the article by Hager et al (October 2004), who report comparable usefulness of helical CT (HCT) scanning and cardiovascular MRI (CMR) for the noninvasive evaluation of the thoracic aorta in patients with aortic coarctation. However, CMR not only allows detailed imaging of the entire aorta, but it also allows quantification of parameters of left ventricular function, aortic valve function, and collateral circulation in aortic coarctation patients. Steady-state free precession CMR is the most accurate imaging modality for measuring ventricular volumes, owing to its high accuracy and good reproducibility. It allows calculation of left ventricular systolic, diastolic, stroke volumes, and ejection fraction. Assessment of left ventricular mass—a parameter with prognostic significance—by CMR has also been shown much more reproducible and accurate than echocardiography, and has an excellent correlation with postmortem ventricular weights. CMR velocity mapping can be used to quantify the degree of stenosis and/or regurgitation of frequently found bicuspid aortic valves. Insight in the functional significance of native aortic coarctation or residual aortic stenosis can be gained by assessing the recruitment of collateral circulation by comparing the flow volume through the aorta just distal to the stenosis with the flow volume through the descending aorta at the level of the diaphragm. So, in agreement with Therrien et al, we believe that for the postoperative assessment of aortic coarctation patients, CMR is very much the preferred imaging modality and that aortic coarctation patients should be cared for in a specialized center with experience and appropriate CMR imaging facilities.

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