TB is appropriate, particularly in high-prevalence countries. The study by Lee et al performed in a high-prevalence Asian region and involving a very high number of untreated contacts demonstrates that the risk of progression is at its highest directly after *Mycobacterium tuberculosis* infection and that more than one-half of those subjects who progressed to TB did so within the first 2 years. Thus, the statistical power of a progression study will be greatest when the period of high risk of progression is studied, that is, the first couple of years after exposure to infectious patients. Furthermore, the problem of participant dropout will be minimized if the focus is on the first few years following recent infection. From randomized studies on the preventive treatment of latent TB infection, such as the large-scale International Union Against Tuberculosis Eastern European study published in 1982, we learned that following infection, there is a natural decline in the risk of developing TB over time.

Therefore, we agree with Drs Khurana and Khurana that a follow-up of people for 2 years at a given date makes little sense if infection with MTB occurred decades earlier (eg, in early childhood), unless there is impaired immunocompetence. Without any knowledge of the specific conditions of a previous MTB infection among the people tested, a positive result, irrespective of whether a tuberculin skin test (TST) or an interferon-γ release assay (IGRA) is used, cannot, per se, lead to the conclusion that there is a high risk of the person developing TB in the future. Thus, it follows that decisions on chemoprophylaxis should not be based on IGRA or TST results alone. As neither the IGRA nor the TST distinguishes old from recent infection, the history of exposure needs to be analyzed to decide whether old or recent infection is likely.

This also pertains to the screening of health-care workers. The high prevalence of positive TST or IGRA results and the low rate of progression for health-care workers indicate that old infections with a low risk of progression prevail. Of particular note is the fact that in the small study (n = 48) by Park et al, a considerable variation in IGRA results is observed, especially with concentrations around the cutoff. Again in agreement with Drs Khurana and Khurana, the meaning of these phenomena needs to be elucidated by generating data on risk of progression depending on IGRA variation.

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**Air-Splinting Maneuver During Flexible Fiber-optic Bronchoscopy**

To the Editor:

Flexible fiber-optic bronchoscopy is a well-established procedure and is regularly taught to trainee medical professionals in teaching hospitals. While doing this procedure via the transnasal route, I have noticed that in some patients the soft palate and posterior pharyngeal wall are in contact, and there may be no lumen visible when the scope has reached the junction of the nasopharynx with the oropharynx. This may cause difficulty in orienting and advancing the bronchoscope further ahead, particularly for a beginner.

I have found that making the patient breathe through his or her nose with mouth closed opens up the lumen much more than when the patient is breathing with mouth open. In addition, the operator gets a clear orientation to move the bronchoscope ahead. This maneuver, which I call Magazine’s air-splinting maneuver, has been successful in opening up the lumen in 20 such patients. All the patients were given IV midazolam for sedation and could be aroused to follow the verbal instructions for performing the maneuver. If the level of sedation is deeper, the patient may not be expected to do the same. The head-tilt chin-lift technique alone fails to open the lumen in these patients. However, the combination of both is useful only if there is additional obstruction due to the tongue falling back against the posterior pharyngeal wall. In patients in whom the lumen of the oropharynx is visible, this maneuver opens it up further and, thus, enlarges the field of vision in some cases.

The principle behind the maneuver is simple to understand. Nasal breathing with the mouth closed will force the entire inhaled or exhaled air to flow via the nasopharynx. Hence, air will act like a splint and keep the lumen open. If the patient is asked to breathe deeper and, hence, increase the tidal volume, then, as expected, the splinting action is further enhanced. The pharyngeal splinting effect of CPAP was described by Rothlisbech et al and McGrath et al to enhance the visualization of the upper airway. The air-splinting maneuver I describe will be of help to bronchoscopists, particularly beginners, who use the transnasal route when performing flexible bronchoscopy.

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**References**


From Sleep-Disordered Breathing to Increased Left Ventricular Wall Stress in Heart Failure

To the Editor:

Sleep-disordered breathing (SDB) frequently occurs in patients with congestive heart failure (CHF). Both entities seem to be inherently associated, but mechanisms causally linking SDB with CHF still remain uncertain. Thus, in an issue of CHEST (March 2012), Carr et al1 reviewed acute cardiopulmonary failure resulting from SDB.

Central sleep apnea is associated with increased circulation time in CHF due to a reduced stroke volume and is closely linked to the sympathetic tone. We showed that the severity of SDB is associated with increased end-diastolic and end-systolic left ventricular wall stress, but not with pump function.2 Thus, the question arises of whether cardiac function is indeed the key determinant.

Left ventricular wall stress is predominantly influenced by ventricular volume, myocardial mass, and the transmural pressure gradient. Ventricular wall stress is frequently increased in dilative CHF, since cardiac hypertrophy is not appropriate to compensate for ventricular dilatation.3,4 Increased ventricular wall stress was associated with an altered autonomic tone.5 In obstructive sleep apnea, upper airways obstruction increases the negative intrathoracic pressure, which transmits increased distending forces on the left ventricle and thereby increases wall stress. Increased left ventricular wall stress was identified as the missing link between CHF and SDB and should not be underrated.2 These considerations also provide a rationale for the benefits of positive airway pressure ventilation therapy in CHF.2

It has been suggested that repetitive nocturnal hypoxias interfere with an adequate ventricular hypertrophy in SDB, which enhances further wall stress and ventricular dilatation with worse prognosis.6 Increased left ventricular wall stress is associated with decreased heart rate variability with unfavorable consequences.7 Increased wall stress leads to the opening of stretch-activated cation channels, which increases the risk of sudden cardiac death. Wall stress is also associated with myocardial oxygen consumption, that is, increased wall stress can worsen wall motion abnormalities, further deteriorating cardiac function. We recommend, therefore, that ventricular wall stress should be considered as a diagnostic criterion in SDB and CHF.2 Normalization of high wall stress remains, however, an as-yet unmet therapeutic target.