may cause a lower airway instability. Grunting expiration is a breathing strategy to defend lung volume and lower airway patency by forcing gas into the peripheral airways. Infants with glossoptosis apnea/hypopnea rarely experience severe complications, including hypoxemia and hypercapnia, pulmonary hypertension, anoxic brain damage, cor pulmonale, and sudden death. They frequently experience a blood gas derangement characterized by a degree of hypoxemia greater than the degree of hypercapnia and a hypoxemia relatively resistant to oxygen administration. Increasing \( F_{O_2} \) required to maintain \( P_{O_2} \) between 60 and 80 mm Hg, results in a considerable increase in alveolar-arterial oxygen difference (Fig 1). No lung opacities or only small areas of collapse or consolidation are usually found on chest radiographs. Oxygen compared with other gases is more rapidly absorbed from the alveolus. Therefore, the most likely explanation for such large alveolar-arterial oxygen differences is that the high oxygen concentration in the inspired air converts in microatelectasis those segments of the lung with lower airways instability. Rapidly absorbed from the alveolus. Therefore, the most likely explanation for such large alveolar-arterial oxygen differences is that the high oxygen concentration in the inspired air converts in microatelectasis those segments of the lung with lower airways instability.

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The recently published article by Moua et al (February 2013) is timely but raises more questions about how to study patients with mediastinal lymphadenopathy (MLAD) and idiopathic pulmonary arterial hypertension (IPAH). It would be particularly helpful for clinicians who treat such patients to be able to perform a risk assessment of the need for mediastinal sampling in a patient group where even minimally invasive endobronchial ultrasound (EBUS)-guided transbronchial needle aspiration (TBNA) or conventional TBNA is likely to be tolerated less well compared with patients without IPAH. That said, EBUS-TBNA (or conventional TBNA) do provide less invasive alternatives to mediastinoscopy.

A plethora of guidelines exist regarding the approach to pulmonary nodules and MLAD in the context of lung cancer or malignancy. However, there seems to be a need for guidelines on the approach to idiopathic or unexplained MLAD (without the context of a known cancer) and prospective controlled studies of adequate size to clarify whether conservative monitoring of patients with IPAH and MLAD between 1 and 2 cm with an effusion misses any significant mediastinal pathology. For patients with MLAD > 2 cm with IPAH, a safer option might be to perform the less invasive neck ultrasound biopsy first before using EBUS-TBNA or conventional TBNA because it is known that there is a high incidence of pathologic supraclavicular lymphadenopathy in patients with MLAD, even though this may not be apparent on CT scan of the neck, as ultrasound is superior in this regard.

In addition, IPAH represents one of many nonmalignant disorders where MLAD occurs; others would include bronchiectasis, pulmonary veno-occlusive disease, congestive cardiac failure, interstitial lung disease (without granulomatous disease), and reactive infective adenopathy, to name a few. In many of these disorders, pulmonary hypertension may be present, and avoiding invasive EBUS-TBNA sampling might be preferable (unless a specific treatable diagnosis, eg, mycobacterial infection, could be achieved). Could it be that the predominant mechanism for MLAD in all these conditions is actually from elevated right-sided (rather than left-sided) pressures?

In summary, further guidance on a stratified, sequential approach to the investigation of MLAD in suspected benign disorders with pulmonary hypertension would be welcome, reserving EBUS-TBNA or conventional TBNA for situations where neck ultrasound biopsy is unhelpful. Mediastinoscopy should also be reserved for cases where EBUS-TBNA or conventional TBNA is nondiagnostic and the pretest clinical probability of malignancy or treatable mediastinal pathology outweighs the risk of performing mediastinoscopy in the presence of significant pulmonary hypertension.

**An Algorithm for Approaching Mediastinal Lymphadenopathy in Pulmonary Hypertension**

To the Editor:

The recently published article by Moua et al (February 2013) is timely but raises more questions about how to study patients with mediastinal lymphadenopathy (MLAD) and idiopathic pulmonary arterial hypertension (IPAH). It would be particularly helpful for clinicians who treat such patients to be able to perform a risk assessment of the need for mediastinal sampling in a patient group where even minimally invasive endobronchial ultrasound (EBUS)-guided transbronchial needle aspiration (TBNA) or conventional TBNA is likely to be tolerated less well compared with patients without IPAH. That said, EBUS-TBNA (or conventional TBNA) do provide less invasive alternatives to mediastinoscopy.

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**References**


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Response

To the Editor:

We are grateful for the thoughtful commentary by Dr Medford relating to our recent article in CHEST.1 We agree that because procedural risk in patients with idiopathic pulmonary arterial hypertension (IPAH) is high, pretest suspicion for a more ominous diagnosis is warranted prior to invasive studies of associated mediastinal lymphadenopathy (MLAD). Specifically, exclusion of malignancy (lymphoma, lung primary, metastatic primary, and so forth) should be the only immediate justification for invasive assessment in this setting.

Unfortunately, few studies have commented on MLAD associated with pulmonary hypertension. Our observed frequency of close to one in five patients with IPAH (18%) involved a selected cohort with both right-sided heart catheterization and chest CT scan for the purposes of correlating lymphadenopathy with severity of cardiac hemodynamics. This may underestimate the true prevalence of MLAD in all patients with IPAH. We did find a similar distribution and size of abnormal lymph nodes when compared with left-sided congestive adenopathy,2 and, although others have shown resolution or improvement in adenopathy following heart failure treatment,3 only one of nine patients with MLAD and pulmonary hypertension from most causes. For example, pulmonary hypertension associated with sarcoidosis may be a unifying diagnosis in some patients presenting with persistent MLAD.4 PET scanning and other studies may be suggestive in this setting,5 although sarcoidosis without pulmonary parenchymal findings is unlikely to be immediately life threatening, again justifying a period of observation prior to biopsy assessment if not regressing. There is a known frequent association of MLAD with interstitial lung disease,6 and, as such, diagnostic assessment without other clinical features of malignancy is likely unjustified.

We suggest and agree with a cautious approach to MLAD associated with pulmonary hypertension of any cause, particularly if pretest suspicion for malignancy is low. Further prospective and observational work to refine a safe approach to MLAD in this setting is needed, as noted by Dr Medford.

References


Air Pollution and Chronic Cough in China

To the Editor:

In a recent issue of CHEST (March 2013), Lai et al demonstrated that cough variant asthma, upper airway cough syndrome, eosinophilic bronchitis, and atopic cough (AC) constituted the

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