Pulmonary function laboratory to assist in the management of cardiac disease

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BACKGROUND

Pulmonary function tests (PFTs) are highly sensitive to derangements in the cardiopulmonary unit. Recognizing the effects of cardiac disease on PFTs is paramount to proper testing interpretation, a task particularly challenging in the presence of coexisting respiratory disorders.

OVERVIEW

A 72-year-old former smoker underwent PFTs due to out-of-proportion dyspnea after treatment optimization for heart failure with reduced ejection fraction (HFrEF). RV and functional residual capacity (FRC) were markedly increased (~160%) despite only a mild and proportional decrease in post-bronchodilator (BD) FEV_1 and FVC with preserved TLC. Given air trapping and smoking history, long-acting BDs were initiated with rapid improvement in dyspnea (case #1). An 81-year-old former smoker admitted to the hospital due to HFrEF decompensation showed "fixed" obstruction with a low DL_{CO} a week after discharge. The patient refused BD treatment for potential COPD. Two months later, all PFTs were within normal range (case #2).

Clinical interpretation of PFTs in cardiac disease associated with lung congestion and/or low cardiac output is full of pitfalls.[1,2] In chronic HFrEF, decrements in lung volumes (low lung compliance, alveolar filling, cardiomegaly) and, in some patients, inspiratory muscle weakness might cause restriction. If severe enough, this may "normalize" spirometry and counterbalance any hyperinflation in coexisting COPD.[3] Air trapping is not commonly seen in stable HFrEF and, in the proper clinical context, might signal underlying airway disease (case #1; Figure 1A). Exaggerated ventilation[4] coupled with poor muscle O_2 delivery on cardiopulmonary exercise testing might suggest that HFrEF contributes to exertional dyspnea in COPD. Overt obstruction with reduced mid-expiratory flows due to peribronchiolar cuffing, mucosal swelling, and vagal reflexes may occur in acute decompensated HFrEF ("cardiac asthma"; Figure 1B). These abnormalities may take weeks to resolve (case #2), frequently accompanied by airway hyperresponsiveness.[5] Although higher capillary blood volume may increase the "vascular" conductance to gas transfer, this is superseded by increases in the "membrane" resistance. Thus, mild-moderate decreases in DL_{CO}—worsening with HFrEF progression—may make it difficult to portion out the contribution of any underlying lung disease (e.g., COPD, interstitial lung disease) to a low DL_{CO} (Figure 1B).

In contrast with HFrEF, mild decrement in FEV_1/(F)VC and increases in RV are frequently seen in patients with stable moderate-to-severe mitral valve disease, likely because of the congested vessels in alveolar walls impeding their complete deflation. Congenital heart disease associated with left (L)-to-right (R) shunt and increased capillary blood volume may increase DL_{CO} and carbon monoxide transfer coefficient (K_{CO}). Mild hypoxemia and an increased alveolar-arterial O_2 gradient may occur due to ventilation/perfusion mismatch. Severe hypoxemia is the hallmark of patients with R-to-L shunt (e.g., tetralogy of Fallot, Eisenmenger syndrome), the shunted fraction being estimated by the 100% O_2 inhalation test.[6]

CLINICAL MESSAGE

Given that respiratory and cardiac disease often coexist and given the overlapping consequences to lung function, the pulmonologist should recognize the "shades of gray" when interpreting PFTs in these patients. Frequently, little is known about the pre-test likelihood of disease or disease severity/stability in patients with known cardiac disease. Thus, the report should cautiously state that testing results should be analyzed considering the individual's clinical context.

AUTHOR CONTRIBUTIONS

All authors contributed to conceptualization, writing, reviewing, and editing.

CONFLICTS OF INTEREST

None declared.

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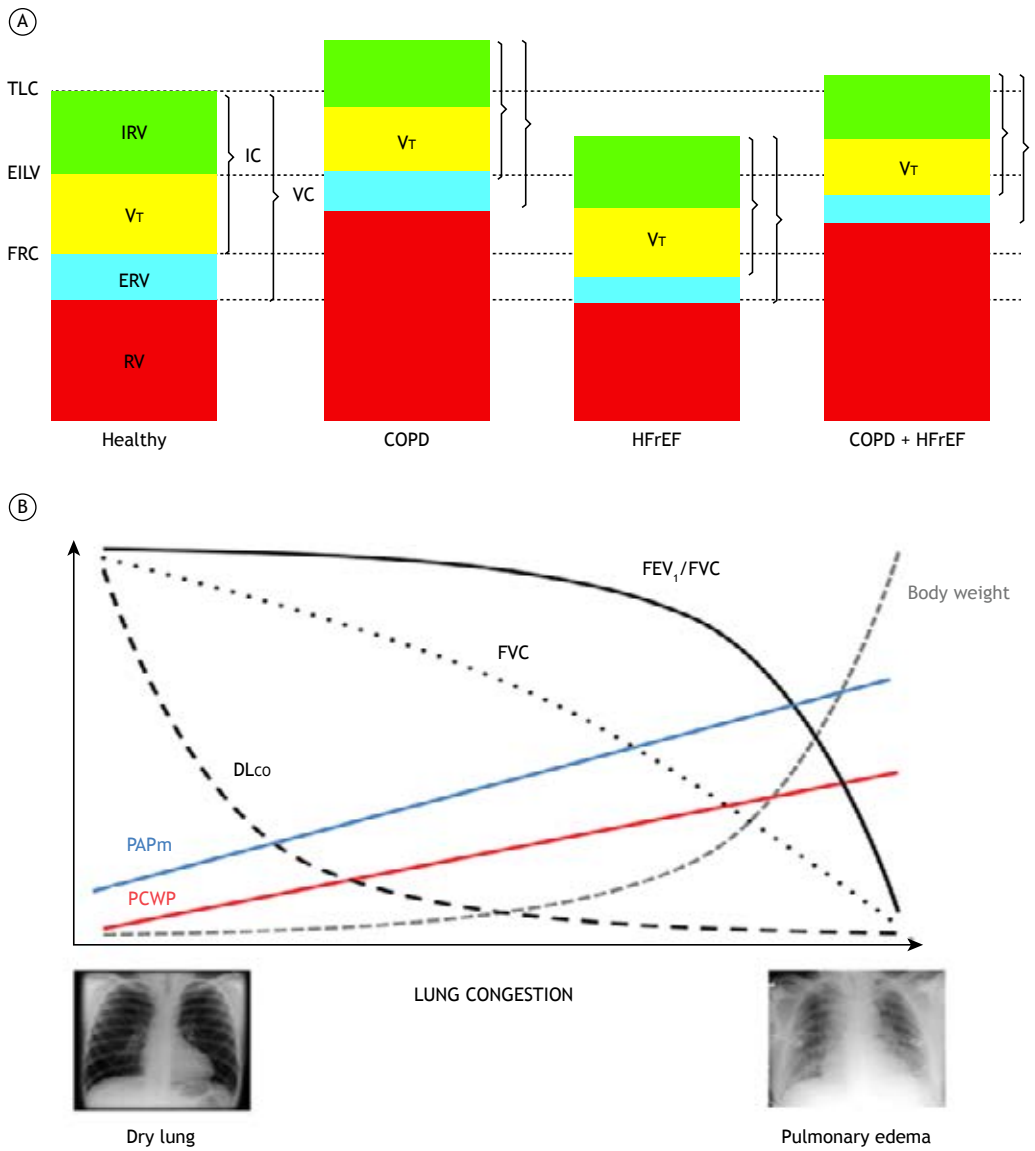

Figure 1. Respiratory functional consequences of heart failure with reduced ejection fraction (HFrEF) with the highest potential to influence the interpretation of pulmonary function tests. Panel A depicts the effects of COPD, HFrEF, and their combination on resting lung volumes and capacities in a non-obese subject. Variable degrees of thoracic hyperinflation (⇑ TLC), lung hyperinflation (⇑ FRC), and gas trapping (⇑ RV) result in low volume available (⇓ IC) for tidal volume (V_T) expansion in patients with moderate-to-severe COPD. This latter consequence is also observed in moderate-to-severe HFrEF in which the most consistent mechanical effect is a reduction in TLC that is also more pronounced than those found in FRC. In contrast to COPD, the IC decrement in HFrEF occurs predominantly due to a lower "ceiling" (TLC) rather than a higher "floor" (FRC). Lung volume measurements (e.g. body plethysmography) are particularly useful to suggest coexistent HF and COPD. HF may 'normalize' TLC in a COPD patient with previous thoracic hyperinflation. Conversely, high RV and RV/TLC may indicate the presence of COPD-related gas trapping as RV is more "resistant" than TLC to decrease secondary to coexistent HFrEF. Panel B provides a schematic representation of potential trajectories of lung function, hemodynamics, and body weight with progressing lung (and peripheral) congestion. As left ventricular filling pressure increases (⇑ pulmonary capillary wedge pressure; PCWP), lung congestion and interstitial edema develop, causing reductions in FVC and DL_{CO}, while FEV_1/FVC remains normal. Therefore, FVC and particularly DL_{CO} may decline with even moderate congestion, whereas an obstructive-like pattern (low FEV_1/FVC) may emerge with advanced congestion due to thickening of bronchial wall by edema and increased vascular volume, peribronchial edema, and vascular engorgement, as well as low radial distending forces on the bronchial wall due to low lung volumes and increased airway smooth muscle contractility elicited by neuro-humoral mechanisms. EILV: end-inspiratory lung volume; FRC: functional residual capacity; IRV: inspiratory reserve volume; ERV: expiratory reserve volume; IC: inspiratory capacity; and PAPm: mean pulmonary arterial pressure. Panel B reproduced with permission of the publisher.[1]

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