Gas Exchange Efficiency in Congestive Heart Failure

Robert L. Johnson, Jr, MD

The lungs and heart are irrevocably linked in their oxygen and CO₂ transport functions. Functional impairment of the lungs often affects heart function, and functional impairment of the heart often affects lung function. In patients with chronic congestive heart failure (CHF), exertional dyspnea is a common symptom, and ventilatory effort is increased at a given exercise workload despite normal arterial blood gases. In this issue of Circulation, the increased exercise ventilation in CHF is reported to contain prognostic information that extends beyond that provided by maximal oxygen uptake (VO₂max), left ventricular ejection fraction, or the NYHA functional classification.¹ Their data indicate that the steepness with which ventilation increases relative to CO₂ production during incremental exercise, either alone or in combination with VO₂max, left ventricular ejection fraction, and NYHA classification, can be a sensitive tool for predicting event-free survival of patients with CHF. Such a tool can be important for evaluating the need for heart transplantation or for following the efficacy of therapeutic measures; it can be evaluated at submaximal work loads and is easier to measure than VO₂max.

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The high ventilation (Ve) with respect to CO₂ production (VCO₂) in CHF is not a new observation,²–⁶ but its potential usefulness as a prognostic tool to evaluate the severity of CHF is relatively new. Perhaps even more important, however, is what the studies of Kleber et al,¹ using this tool, tell us about impaired gas exchange in CHF and its relationship to impaired gas exchange in lung disease.

Because the high level of ventilatory drive in heart failure can predict survival, it must contain important information on how left ventricular dysfunction affects either the lung or ventilatory control. The first thing that we need to examine, then, is what basic information is contained in the slope of the relationship between ventilation (Ve) and CO₂ production (VCO₂). The modified alveolar equation⁷ concisely describes the determinants of the steepness with which Ve rises with respect to VCO₂:

\[ V_E = \frac{863}{P_{CO_2}(1-V_D/V_T)} \cdot V_{CO_2}, \]

where \[ \frac{863}{P_{CO_2}(1-V_D/V_T)} = \text{slope}, \]

\[ P_{CO_2} = \text{arterial CO}_2\text{ tension}, \]

\[ V_D/V_T = \text{fraction of the tidal volume} \]

\[ (V_T) \text{ that goes to dead space} (V_D). \]

The relationship between Ve and VCO₂ by Equation 1 is linear over a wide range, and its slope is determined by just 2 factors: (1) behavior of arterial CO₂ tension during exercise and (2) the VD/Vₜ ratio. If PaCO₂ is driven down by a high ventilatory drive from peripheral chemoreceptors or by ergoreceptors in skeletal muscle, the slope of the Ve/VCO₂ relationship will increase, or if VD/Vₜ is high, the Ve/VCO₂ slope will increase. Increased chemoreceptor gain is often seen in severe CHF,⁸ eg, in patients with Cheyne-Stokes breathing, but increased chemoreceptor gain alone will not drive the PaCO₂ down unless the set point about which PaCO₂ is controlled is depressed or unless hypoxic drive or ergoreceptor drive is high. Most studies suggest that blood gases are normal in patients with CHF⁴ and that PaCO₂ either stays the same or declines modestly from rest to peak exercise, no differently than in normal controls. There are 2 potential sources for a high VD/Vₜ ratio: (1) a low tidal volume (Vₜ) with respect to a normal anatomic dead space or (2) an abnormally high physiological dead space. Patients with CHF often have a reduced tidal volume at heavy exercise, which would increase the VD/Vₜ ratio; however, it has been estimated that only \( \approx 33\% \) of the increased dead space ventilation in CHF can be explained by a low Vₜ.⁵

Current information suggests that the major source for an abnormally steep Ve/VCO₂ slope in CHF is increased nonuniformity of ventilation-perfusion ratios (V/Q), causing inefficient gas exchange. However, a word of caution is still necessary. The above conclusion is based on indirect evidence. No direct comparisons have been made of PaCO₂ and dead space ventilation in CHF patients with and without a high Ve/VCO₂ slope during exercise. Such comparisons are needed.

What might be the source of an increased nonuniformity of pulmonary V/Q ratios in CHF and why would it provide prognostic information not provided by VO₂max? Lung volumes and ventilatory function in the CHF patients studied by Kleber et al¹ were relatively normal, and arterial blood oxygen saturation at peak exercise was normal, as is generally the case in CHF in the absence of coexisting lung disease. This pattern of a high VD/Vₜ ratio with normal arterial blood gases suggests that nonuniformity of V/Q ratios in the lung is

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Determinants of Gas Exchange at Maximal Exercise in Patients With CHF and With Primary Lung Disease

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<tr>
<th>CHF</th>
<th>COPD</th>
<th>IPF</th>
<th>Q_{max}</th>
<th>V_{Emax}</th>
<th>D_{LCO}</th>
<th>V_{D}/V_{T}</th>
<th>V_{E}/V_{CO_2}</th>
<th>Slope</th>
<th>D_{LCO}/Q</th>
<th>PaCO_2</th>
<th>SaO_2</th>
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COPD indicates chronic obstructive lung disease; IPF, interstitial pulmonary fibrosis; V, variable (can be high, normal, or low); N, normal; ↓, decreased; ↑, increased; and boldface arrow, a primary change. In CHF, the primary determinant of \( Q_{max} \) is a low \( Q_{max} \); in COPD, the primary determinant is \( V_{E}/V_{CO_2} \); and in IPF with alveolar capillary block, the primary determinant of \( Q_{max} \) is a low \( D_{LCO} \).

More likely caused by increased nonuniformity of perfusion than of ventilation. When ventilatory capacity remains normal, inefficient gas exchange caused by abnormal distribution of perfusion usually can be well compensated during exercise by raising ventilation enough to maintain a normal PaCO_2 and normal arterial blood O_2 saturation. This is not true in severe chronic obstructive lung disease, in which not only are ventilation and perfusion poorly matched, but also, compensatory increases in ventilation are restricted by the high resistance to air flow; during exercise, PaCO_2 rises and arterial satory increases in ventilation are restricted by the high ventilation and perfusion poorly matched, but also, compen-

(1) Maximal ventilatory capacity is well maintained in CHF and can compensate for the high V_{D}/V_{T}, bringing the PaCO_2 down to normal levels at peak exercise and maintaining a normal or high alveolar oxygen tension. (2) Maximal cardiac output (Q_{max}) in CHF is reduced more than is the D_{LCO}; hence, the ratio of D_{LCO}/Q never falls low enough during exercise to cause a fall of O_2 saturation of blood leaving the lung. It is the low maximal cardiac output and impaired peripheral O_2 extraction that primarily impairs oxygen transport in CHF, not pulmonary gas exchange; arterial blood gases remain normal. However, the reduced efficiency of gas exchange in CHF reflected by the steep relationship between V_{E} and V_{CO_2} is probably a major source of the exertional dyspnea with normal arterial blood gases.

Thus, left ventricular heart failure has important effects on lung function, just as lung disease has important effects on cardiovascular function. The application of a measurement that quantifies efficiency of gas exchange during exercise as an index of the severity of CHF and life expectancy in CHF emphasizes the important functional linkage between the heart and the lungs. The measurement used is simple and can be applied even at low levels of exercise. It must be emphasized, however, that the measurement, i.e., the slope of the relationship between V_{E} and V_{CO_2} during exercise, is nonspecific and is frequently abnormally steep in primary lung disease as well as in CHF, although usually associated with abnormal arterial blood gases in lung disease. Hence, the measurement used by Kleber et al must be interpreted in context. To emphasize this, a comparison of the primary determinants of impaired gas exchange in CHF, chronic obstructive lung disease, and interstitial lung disease with alveolar capillary block are shown in the Table.

In the Table, the arrows, pointing either up or down, indicate the change in direction of the key determinants at each step in oxygen transport for each condition. The Table is oversimplified but is conceptually useful. In CHF, the primary impairment of oxygen transport is imposed by a reduced maximal cardiac output (Q_{max}), indicated by a boldface arrow pointing down. In patients with chronic obstructive pulmonary disease, primary impairment of oxygen transport is imposed by a reduced maximal ventilation (V_{Emax}) with inefficient gas exchange, and in patients with interstitial lung disease with alveolar capillary block, the primary impairment is imposed by a reduced D_{LCO}. In all of these disorders, uneven V/Q matching increases the V_{D}/V_{T} ratio and impairs the efficiency of CO_2 excretion from the lung; if ventilation can be increased enough during increasing exer-
To prevent the $\text{PaCO}_2$ from rising, the $\text{Ve/VC}_{\text{O}_2}$ slope will be steeper than normal in lung disease as well as in CHF, as indicated by the bracketed term in Equation 1. In severe chronic obstructive pulmonary disease, $\text{PaCO}_2$ will rise as exercise load increases, and the $\text{Ve/VC}_{\text{O}_2}$ slope may become low even though $\text{Ve/VT}$ is high. Coexistent lung disease can significantly alter the expected pattern of gas exchange in CHF. Thus, it must be cautioned that if a patient with CHF has significant coexistent lung disease, application of the $\text{Ve/VC}_{\text{O}_2}$ slope to predict survival, as proposed by Kleber et al., becomes invalid.

In summary, available data suggest that chronic CHF induces structural changes as well as interstitial pulmonary edema in the lungs, which impair the efficiency of gas exchange; the extent of these changes reflects the severity of the CHF and probably its duration. Physiologically, these structural changes are manifested by an increased ratio of dead space to tidal volume ($\text{VD/VT}$), which causes an abnormally high ventilation during exercise. They are also usually manifested by a reduction in diffusing capacity of the lung (DLCO), which varies with the severity of CHF. Although the magnitude of these physiological changes in lung function can reflect the severity of CHF and be an important predictor of survival, inefficiency of gas exchange is not the primary cause of impaired exercise capacity. Reduced maximal oxygen transport in CHF is caused by a low maximal cardiac output and perhaps impaired peripheral oxygen extraction; arterial $\text{PaCO}_2$ and arterial $\text{O}_2$ saturation at peak exercise remain normal. Even though arterial blood gases remain normal, inefficient gas exchange can be a major source of exertional hyperpnea and dyspnea. The pattern of abnormal gas exchange during exercise in CHF clearly differs from that in primary lung disease; problems of interpretation arise when CHF and primary pulmonary disease coexist.

References


Key Words: Editorials ■ exercise ■ dyspnea ■ hyperpnea ■ blood gases
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