Gas Exchange Efficiency in Congestive Heart Failure II

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It has become increasingly apparent that congestive heart failure (CHF) affects not only the cardiovascular system, but every organ system involved with oxygen transport, including the respiratory system, skeletal muscles, and the hormonal and neural feedback control systems for breathing, cardiac output, blood pressure, blood volume, and distribution of blood flow. One segment of this transport system cannot be isolated from the rest. The ventilatory response to exercise in patients with CHF is augmented despite normal arterial O$_2$ saturation and a normal or low end-tidal P$_{CO_2}$.. The augmented ventilatory response is measured as a steep slope of the increase in ventilation with respect to CO$_2$ output (ΔVE/ΔVCO$_2$) or as a high VE/VCO$_2$ ratio at peak exercise. The source of this ventilatory augmentation has been controversial, but its pathophysiological significance is clear. A high slope at submaximal exercise or a high VE/VCO$_2$ ratio at peak exercise is a powerful index of poor prognosis in patients with CHF. As indicated by Ponikowski et al in the current issue of Circulation, this prognostic power is retained in patients with CHF, even when the maximal O$_2$ uptake (VO$_2$ max) is near the normal range. Is the augmented ventilation during exercise an integral part of the deranged cardiorespiratory reflex controls in CHF? From the same laboratory provide indirect support for a high ventilatory drive related to increased chemoreceptor gain and ergoreceptor drive in skeletal muscle. However, if present, such an increased ventilatory drive should force the PaCO$_2$ below expected levels during exercise and generate a negative correlation between PaCO$_2$ and VE/VCO$_2$ at peak exercise.

A high VE/VCO$_2$ ratio has 2 possible sources: (1) increased ventilation, which is required to overcome a large dead space to maintain a normal arterial CO$_2$ tension (PaCO$_2$), or (2) increased central drive to ventilation, which drives the PaCO$_2$ below what is normally expected. Ponikowski et al present convincing evidence that the augmented ventilatory response to exercise in CHF is significantly correlated with other markers of abnormal cardiorespiratory reflex control (ie, central and peripheral chemoreceptor control of ventilation, ergoreceptor drive to ventilation, and both autonomic and baroreceptor control of the circulation). Thus, the high VE/VCO$_2$ seems related to altered chemoreceptor gain and ergoreceptor drive to ventilation, as well as to impaired reflex control of the heart and circulation. Impaired autonomic and baroreceptor control become manifest in severe heart failure by an abnormally reduced variability in heart rate and an increased variability in blood pressure, with predisposition to arrhythmias and sudden death. These observations provide a major link between augmented exercise ventilation in CHF and poor prognosis.

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A high VE/VCO$_2$ ratio is available at http://www.circulationaha.org

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Paco₂ at peak exercise in the CHF patients studied by Franciosa et al² with that in the normal subjects studied by Martin et al.¹⁵ The normal subjects had different chemoreceptor gains for P₀₂ and Pco₂ at rest, which were augmented at exercise; those normal subjects with high chemoreceptor gains had higher ratios of Ve/Vco₂ and a lower Paco₂. The point of the graph is to illustrate from the regression lines that that ventilation had to be about twice that in the normal subjects to achieve the same Paco₂ because of the inefficient gas exchange (ie, the high Vd/Vt ratio). This means that ventilatory drive had to be, on average, twice as high in the CHF patients than in the normal subjects studied by Martin et al.¹⁵ It is hard to explain this increased drive by a simple increase in chemoreceptor gain, however, because chemoreceptor gain does not represent a unidirectional drive; rather, it represents the strength of feedback control to minimize any deviation of arterial P₀₂ and Pco₂ in either direction from their respective set points. This is like the gain of the thermostat in a home air-conditioning system. Exercise must alter the set point of the control system, perhaps by increased sympathetic stimulation or from increased stimulation from skeletal muscle ergoreceptors, both of which are augmented in CHF. A high chemoreceptor gain would then tighten the control and ensure a smaller error signal at full response. It would be of interest to know whether normal subjects who have a high chemoreceptor gain and a high ventilatory response to exercise also have a high ergoreceptor drive from skeletal muscle.

The augmented ventilatory response to exercise in CHF correlates with control and reflex abnormalities and with hemodynamic alterations. The latter relationships can also be illustrated from the data of Franciosa et al² (Figure 3). There is a strong inverse correlation of Ve/Vco₂ with cardiac index (Figure 3A) and with pulmonary artery pressure (Figure 3B). Hence, there are multiple reasons why this simple ratio of Ve/Vco₂, or the slope of the increase in Ve with respect to Vco₂ during exercise, provides a powerful prognostic index in heart failure. It seems to reflect the severity of derangement in almost all aspects of CHF; it is also an objective measurement that can be made easily.

References

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