Ventilation during exercise in chronic heart failure

Abstract The ventilatory response to exercise in patients with chronic heart failure (CHF) is greater than normal for a given work or metabolic rate ($V_{O_2}$). The factors that determine the ventilatory response to exercise are: 1) the CO2 production ($V_{CO_2}$), 2) the arterial CO2 set-point (arterial PCO2 at rest), 3) the physiological dead space/tidal volume ratio (VD/VT), and 4) the change in PaCO2 during exercise. This report illustrates how each of these factors might influence the ventilatory response to exercise in CHF patients.

Thirty-one CHF patients (New York Heart Association, Classes 2 and 3) were studied, 18 from Harbor-UCLA Medical Center (cycle-ergometer exercise) and 13 from Queen's University at Belfast (treadmill exercise). A group of healthy subjects matched for size, age and gender served as control subjects. Minute ventilation (VE) was 48, 88 and 43% greater in the CHF groups compared to the control population at 6 min of the 25w and 60w cycle and low level (2.5 km h⁻¹ and 5% grade) treadmill exercise, respectively. $V_{O_2}$ kinetics were slower in CHF patients than the control group, the slowing being proportional to the lactate increase. However, the increase in $V_{O_2}$ above rest at 6 min of exercise was approximately the same for CHF and control subjects. $V_{CO_2}$ at 6 min increased in the CHF patients by 7% and 34% for 25 and 60 watts cycle and 19% for treadmill exercise, respectively, compared to the control group. Because PaCO2 was not measured in this study, neither CO2 set-point nor the VD/VT could be individually calculated. End-tidal PCO2 will decrease when PaCO2 decreases or VD/VT increases, the combined effect of PaCO2 change and increase in VD/VT could be assessed from the difference between the patient and the control group. Since PETCO2 was significantly reduced in the patient population at the end of 60w cycle exercise (32 versus 41 mm Hg), either the VD/VT was increased and/or the PaCO2 was reduced. Because the resting PaCO2 is generally normal in CHF patients, the increase in the ventilatory response to exercise in patients with CHF can best be accounted for by three physiological mechanisms: 1) an increase in $V_{CO_2}$ secondary to CO2 release from bicarbonate as it buffers lactic acid, 2) the reduction in PaCO2 secondary to the lactic acidosis-induced hyperventilation, and 3) an increase in the fraction of breath that is wasted (dead space). Mathematically, these factors interact so that relatively small changes in each cause large changes in VE.

Key words Dead space / tidal volume ratio – anaerobic threshold – lactate – CO2 production – CO2 set-point – $V_{O_2}$ kinetics

Introduction

The ventilatory response to exercise is often abnormal in patients with chronic heart failure (3, 8, 10, 14, 20, 26). However the mechanism(s) have not been systematically analyzed. The ventilatory response to exercise can be defined in quantitative terms (Fig. 1). By analyzing each of these terms, it is possible to determine the major
pathophysiology which contributes to the high ventilatory drive to exercise in patients with chronic heart failure (CHF).

\[ V_E = V_A + V(E(VD/VT)), \]  

where \( V_D \) is the physiological dead space and \( V_T \) is the tidal volume. By combining Eqs. (1) and (2), we can express \( V_E \) as shown in the following useful equation:

\[ V_E = V_{CO2}/863/[PaCO2(1-VD/VT)]. \]  

Figure 1 displays Eqs. (1) and (3) graphically and illustrates the effect of changing \( V_{CO2} \), \( PaCO2 \) and \( VD/VT \) on the ventilatory requirement.

\( V_E \) has been found to be highly consistent amongst normal subjects for a given \( V_{CO2} \) (21), when the work is performed below the anaerobic threshold (without a lactic acidosis). The reason for the consistency in the response is due to the relatively uniform values for \( PaCO2 \) and \( VD/VT \) in normal subjects (21). The purpose of this report is to analyze the factors that contribute to the abnormally high ventilatory response to exercise in CHF patients.

**Methods**

Two populations of CHF patients consisting of a total of 31 patients were used in this analysis. Eighteen patients with New York Heart Association (NYHA) functional classes 2 (\( N = 15 \)) and 3 (\( N = 3 \)) came from Harbor-UCLA Medical Center (Table 1). They were used in this report to determine gas exchange and heart rate dynamics, the change in ventilatory pattern and the change in end-tidal \( PCO2 \) (PETCO2) and blood lactate in response to 25w and 60w cycle ergometer exercise. Thirteen patients in NYHA functional class 2 (\( N = 7 \)) and 3 (\( N = 6 \)) were studied at Queen's University, Belfast (Table 2), during constant low level treadmill exercise (2.7 kph at 5 % grade) to determine the gas exchange and heart rate dynamics to this form of exercise. Both the Harbor-UCLA and Queen's University studies had size-, age- and gender-matched control subjects for reference (Tables 1 and 2).

Gas exchange was measured breath-by-breath at Harbor-UCLA (Medical Graphics CPX Metabolic Cart, St. Paul, MN) as previously described (29) and then interpolated second-by-second. The data were then time-aligned to the start of exercise and the average response was calculated for each 10 s before and after the start of exercise. At Queen's University, Belfast, a mixing chamber system (P.K. Morgan, Chatham, U.K.) was used as previously described (14).