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Breathing strategy to preserve exercising cardiac function in patients with heart failure

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SUMMARY

The heart and lungs are closely linked as they lie in series, share a common surface area and compete for space within the thoracic cavity. The heart and lungs are exposed to the similar changes in intrathoracic pressure, and reflexes within one organ can influence the other (i.e. vagal influence of lung inflation on heart rate). In patients with heart failure, these cardiopulmonary interactions may be altered due to decreased lung and left ventricular compliance, increased cardiac size, high cardiac filling pressure and altered receptor sensitivity to neural activation. Exercise further affects the cardiopulmonary interactions by stimulating an increase in the depth and frequency of breathing which accentuates the fluctuations in intrathoracic pressure, and by requiring large increases in stroke volume and heart rate in order to respond to the increased metabolic demand. Previous work from our laboratory suggested that patients with heart failure avoid high lung volumes during exercise, often at the expense of unnecessary large positive expiratory intrathoracic pressures resulting in significant wasted effort. Moreover, we also observed that voluntarily increases in lung volume in patients with heart failure induced a mild relative bradycardia, a response not observed in similar aged healthy individuals. Thus, we hypothesized that the rapid shallow low lung volume breathing, in combination with positive expiratory intrathoracic pressure, often adopted by patients with heart failure during exercise is an attempt to preserve, or even enhance, the cardiac response to exercise.

Introduction of the problem

Exercise induces an increase in ventilation through increases in respiratory rate and tidal volume [1]. In healthy individuals, the increase in tidal volume is achieved through a progressive increase in end-inspiratory lung volume while end-expiratory lung volume falls. With aging and in highly fit individuals, end-expiratory lung volume falls with light to moderate intensity exercise but may increase with heavy exercise when expiratory flow limitation is present at high levels of ventilation [2] (Fig. 1). Patients with heart failure often exhibit excessive ventilation for a given workload, which is characterized primarily by an increase in respiratory rate rather than tidal volume [2,3]. It is usually believed that the exercising tidal volume of patients with heart failure is limited by an excessive elastic load to breathing, which restricts the increase in end-inspiratory lung volume and by expiratory flow limitation which prevents a further decline in the already reduced end-expiratory lung volume [4,5] (Fig. 1). In fact, end-expiratory lung volume has been observed to remain near residual volume throughout exercise despite increasing expiratory flow limitation in patients with heart failure [2]. This

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Conflicts of interest statement

None declared.

appears to occur despite room to dynamically hyperinflate and increase tidal volume by encroaching on the inspiratory reserve volume as is often observed in healthy adults with high levels of ventilatory demand. Although it is believed that exercise limitation in patients with heart failure is largely the result of cardiac dysfunction, it remains unknown whether alterations in lungs mechanics and/or abnormal cardiorespiratory interactions affect the cardiac response to exercise in this population [6].

Hypothesis

Patients with heart failure avoid breathing at high lung volumes and with deep lung inflations in order to preserve cardiac output during exercise.

Within-breath variation in stroke volume during resting spontaneous breathing

Breathing-induced changes in intrathoracic pressure and lung volume are believed to influence cardiac function [7]. Changes in lung volume and the accompanying changes in intrathoracic pressure have independent effects on cardiac performance [8,9]. It has been widely reported that inspiration results in acute reductions in stroke volume with decreases ranging from 7% to 18% [8,10–13]. The mechanism responsible for this transient inspiratory-induced reduction in stroke volume is dependent on changes in intrathoracic pressure but largely independent of lung volume changes [14]. Indeed, reductions in stroke volume during chest wall restriction appeared to parallel alterations in gastric and intrathoracic pressure rather than changes in lung volume [15]. However, there is some evidence that changes in lung volume partly influence stroke volume as inspiration without changes in lung volume triggers small changes in stroke volume relative to inspiration accompanied by changes in lung volume in healthy man [14]. Therefore, it is suggested that intrathoracic pressure, and not lung volume, is the main respiratory determinant that influences stroke volume in healthy individuals [7]. Possible mechanisms for this acute inspiratory-induced reduction in stroke volume are either a decrease in left ventricular (LV) diastolic filling (decreased LV preload) [13,16] or an increased impedance to LV ejection (increased LV afterload) [8,17,18].

Left ventricular afterload

The pressure load against which the left ventricle contracts is equal to transmural pressure, also defined as LV intraluminal pressure minus intrathoracic pressure. However, because aortic pressure approximates intraluminal pressure, aortic pressure is considered as the pressure load against which the left ventricle contracts. Increases in intraluminal pressure can be achieved by either raising LV pressure or by reducing the pressure surrounding the left ventricle, i.e. intrathoracic pressure. Thus, decreasing intrathoracic pressure (more negative) would increase LV afterload while increasing intrathoracic pressure (more positive) would unload the left ventricle. Therefore, inspiration and the accompanying decrease in intrathoracic pressure results in an increased intraluminal pressure and consequently LV afterload [12,17–25]. This increased LV afterload could be partly responsible for the acute inspiratory reduction in stroke volume as there is an inverse linear relationship between stroke volume and LV afterload [26] (Fig. 2). However, the magnitude of the inspiratory-induced decrease in stroke volume does not necessarily reflect the magnitude of inspiratory variations in LV afterload [14].

Left ventricular preload

It is generally believed that a decreased LV preload is the primary cause for the transient inspiratory reduction in stroke volume [8,10,13]. Several mechanisms such as variations in right ventricular (RV) output [18,20], pooling of blood in the lungs [13] or deviation of the interventricular septum [17] have been proposed as possible explanations for the inspiratory-

induced decrease in LV preload. It is generally accepted that the negative intrathoracic pressure generated during inspiration paradoxically reduces right atrial pressure and increases the pressure surrounding the abdominal vessels, thereby increasing the gradient for systemic venous return and RV filling [7,8,27]. Indeed, a phasic increase in inferior vena cavae blood flow has been reported during inspiration [16,28]. Despite an increased resistance to blood flow in the pulmonary bed during lung inflation, this augmentation in RV filling results in a larger RV output during the subsequent cardiac cycle [28]. It has been suggested that a phase lag between fluctuations in RV output and LV output results in a decreased LV preload during inspiration [7,8]. However, in patients sedated, paralyzed and ventilated because of brain injuries, disconnection of the ventilator for one respiratory cycle demonstrated that the changes in stroke volume during lung inflation were an immediate consequence of inflation and not a delayed effect from a previous breath or a preceding fluctuation in RV output [7].

A second possibility for the acute inspiratory-induced reduction in stroke volume is that lung inflation enlarges both pulmonary extra-alveolar arteries and veins by creating a negative pressure around the vessel, which increases transmural pressure and produce an increased extra-alveolar vascular volume [29]. This inspiratory-induced pooling of blood expands the pulmonary vasculature, which acts as a capacitance reservoir, and results in reduced LV filling [7,16]. Finally, a last possibility is that the inspiratory increase in RV volume may cause a leftward displacement of the interventricular septum, which leads to decreased LV compliance and could limit LV filling [8,10,14,30–32]. The physical constraint of the pericardium appears to contribute to this interaction as it was demonstrated that ventricular interdependence is reduced following pericardectomy in dogs [19]. It is generally accepted that the acute inspiratory-induced decrease in LV filling results from transient pooling of blood in the pulmonary bed or from ventricular interdependence and not from variations in RV output [8,10,13].

Conversely, expiration results in an acute increase in stroke volume [14]. While the increased intrathoracic pressure elevate right atrial pressure which decreases venous return and result in a reduction in RV output [33], it also reduces transmural pressure and LV afterload [7,13,17]. Moreover, expiration diminishes extra-alveolar vessel blood volume and produces a transient increase in pulmonary venous outflow, resulting in increased LV filling. Therefore, it can be concluded that expiration transiently induces decreases in RV filling, RV output and LV afterload but increases LV preload which result in an increased stroke volume (Fig. 3). On the other hand, inspiration induces increases in RV filling, RV output and LV afterload while decreasing LV preload, which results in a reduced stroke volume (Fig. 3). In the healthy heart, it is typically suggested that changes in LV preload outweigh the influence of changes in LV afterload on stroke volume. However, the relative contribution of LV afterload and LV preload may depend on the magnitude of the changes in intrathoracic pressure as the effect of a decreased LV preload predominates during small decreases in intrathoracic pressure whereas the influence of increased LV afterload on stroke volume is mainly observed with large changes in intrathoracic pressure [20].

Effect of heart failure on breathing-induced variation in stroke volume

Heart failure is a complex clinical syndrome arising from any structural or functional cardiac condition which impairs LV filling or ejection [34]. In heart failure, the gradual increase in heart size, interstitial pulmonary edema due to high LV filling pressure, and respiratory muscle weakness lead to reduced lung volumes and decreased lung compliance, which contributes to a reduced capacity to increase air flow and lung volume [35]. Heart failure is also characterized by decreased LV compliance, possibly due to LV hypertrophy or alterations in the collagen network, indicating that a higher pressure is necessary to fill the left ventricle to the same volume [36]. Thus, patients with heart failure tend to be less sensitive to changes in LV preload

due to a decreased LV compliance, suggesting an inability to augment stroke volume by means of the Frank–Starling mechanism [36]. It is suggested that large decreases in intrathoracic pressure accompanying inspiration may reduce stroke volume due to an overriding influence of LV afterload in this population.

Effect of sustained changes in intrathoracic pressure on stroke volume in heart failure

The relative contribution of LV preload and LV afterload on stroke volume can be determined by manipulating intrathoracic pressure using a ventilator and/or resistors. While positive pressure ventilation, or unloading, reduces stroke volume in healthy man [7,37,38], a different response has been reported in animals with heart failure and patients with LV dysfunction. Indeed, it has been previously reported that reducing the negativity of inspiratory intrathoracic pressure by one half reduced stroke volume in healthy dogs but increased stroke volume and cardiac output in dogs with heart failure [39]. Moreover, while doubling inspiratory intrathoracic pressure had no effect on stroke volume and cardiac output in healthy dogs, it reduced stroke volume and cardiac output in dogs with heart failure [39]. Thus, the normally produced inspiratory intrathoracic pressure is required for an optimal stroke volume in healthy dogs but is detrimental to stroke volume in dogs with heart failure. This is further supported by the observations that increases in intrathoracic pressure may increase cardiac output in patients with LV dysfunction [40]. Indeed, the application of positive end-expiratory pressure increases cardiac output in patients with elevated filling pressures. The authors suggested that the augmentation in cardiac output may result from reduction in LV preload as a reduction in LV filling in patients with elevated filling pressures may decrease effective filling to a level more acceptable for LV performance. We are suggesting that reducing the negativity of intrathoracic pressure and producing positive end-expiratory pressure may reduce the negative effects of an increased LV afterload on stroke volume in patients with heart failure.

Effect of exercise on cardiorespiratory interactions

Exercise further affects the cardiopulmonary interactions by accentuating the fluctuations in intrathoracic pressure and altering the depth and frequency of breathing. Indeed, there is an increase in the swings in intrathoracic pressure with peak inspiratory intrathoracic pressure becoming more negative (-8 to -30 cm H₂O) and peak expiratory intrathoracic pressure becoming more positive (from -5 to $+5-30$ cm H₂O) with increasing exercise intensities [41]. The greater swings in intrathoracic pressure result in augmented breathing-induced changes in stroke volume [1,16]. Although it is not the primary cause for the inspiratory-induced reduction in stroke volume during spontaneous breathing, the mechanical influence of greater lung volumes could also induce greater variations in stroke volume during exercise.

Patients with heart failure typically have an enhanced ventilatory demand for a given metabolic demand [2,3]. Exercise tachypnea in chronic heart failure was suggested to be either the most economic breathing adaptation to the high lung elastic load due to heart disease (decreased lung compliance) [2,5,42] or the consequence of increases in cardiac pressures resulting in higher pulmonary vascular pressures which have been proposed to cause mild chronic subclinical pulmonary edema which may in turn stimulate receptors in the lungs that can augment breathing [43]. With increased exercise intensities, despite significant room to increase the tidal volume by encroaching further on the inspiratory reserve volume, the rise in exercising tidal volume of patients with heart failure appears blunted [2]. It is also believed that the exercising tidal volume of patients with heart failure is limited by expiratory flow limitation which prevents a further decline in the already reduced end-expiratory lung volume [4,5]. Therefore, patients continue to breathe at extremely low lung volume so that the majority

of the expiratory flows produced during tidal breathing meets or exceeds the maximal available expiratory flows even when this results in wasted expiratory effort [2,44] (Fig. 1).

Heart–lung interdependence

Another potential contributor to the cardiorespiratory interactions during resting spontaneous breathing is cardiac size in relation to thoracic cavity size. Increases in cardiac volume within a closed thoracic cavity pose significant constraints on the lungs and there is a strong inverse correlation between cardiac size and lung volume [44]. The increased cardiac size within the thoracic cavity commonly observed in heart failure may contribute to the reductions in lung volume and could play a role in the restrictive breathing pattern observed in these patients [44]. Changes in heart size increases competition for space which influences breathing pattern and subsequently interdependence between the heart and lungs. Moreover, pulmonary function in patients with heart failure is improved after cardiac transplant suggesting that pulmonary limitations are partly caused by the greater intrathoracic space occupied by the heart [45]. This increased heart–lung interdependence in patients with heart failure suggests that lung inflation could influence LV filling through mechanical compression of a less compliant left ventricle [30]. This possibility is also supported by findings that chest wall strapping of healthy individuals, in order to increase the competition for intrathoracic space between heart and lungs, resulted in a reduced stroke volume [15]. The increase in heart size and accompanying reduction in lung volumes in patients with heart failure likely contribute to the inspiratory load and limit the encroachment on the inspiratory reserve volume during times of increased ventilatory demand [46]. In fact, heart–lung interdependence is amplified during exercise and likely contributes to the rapid and shallow breathing patterns.

Effect of lung inflation on heart rate

An increase in lung volume is also believed to affect cardiac output by generating cardio-deceleration through the activation of pulmonary and bronchial C-fiber receptors [47]. Because patients with heart failure have reduced lung compliance, a deep inflation of stiffer lungs could result in a greater activation of pulmonary stretch receptors, leading to a reduction in heart rate [48]. Preliminary data from our laboratory (unpublished) suggested that deep lung inflations reduced exercising heart rate in patients with heart failure but not in healthy individuals (Fig. 4). This could become a problem during exercise as patients with heart failure show minimal increases in stroke volume during exercise suggesting an increased reliance on heart rate to increase cardiac output. In conclusion, avoiding high lung volume, reducing the negativity of intrathoracic pressure and producing positive expiratory pressure through rapid and shallow breathing may reduce the negative effects of mechanical compression the heart by the lungs, greater swings in intrathoracic pressure and the accompanying effect on LV preload and afterload, heart–lung interdependence and heart rate reflexes on exercising cardiac output in patients with heart failure.

Evaluation of the hypothesis

We are proposing to test our hypothesis by studying the changes in beat-by-beat stroke volume and heart rate during different breathing maneuvers performed during steady-state submaximal exercise in healthy individuals and patients with heart failure. We will also measure lung volume and intrathoracic pressure to determine their independent effect on stroke volume. The effects of an increase in lung volume on exercising stroke volume will be determined by increasing lung volume voluntarily, with expiratory threshold loads and by continuous positive airway pressure during steady-state submaximal exercise. The effects of changes in negative intrathoracic pressure on exercising stroke volume will be determined through inspiratory unloading and loading using proportional assist ventilation and inspiratory resistance, respectively. The influence of deep lung inflations on exercising heart rate will be examined

during a brief voluntary inspiratory capacity maneuver. Inspiratory capacity maneuvers will also be performed in combination with proportional assist ventilation to determine the effects of a reduction of the negative swing in intrathoracic pressure on heart rate. The role of changes in lung volume on heart rate will be determined by performing an inspiratory capacity maneuvers against a resistance, which decreases intrathoracic pressure without significantly changing lung volume.

Consequences of the hypothesis

The proposed studies are novel and have not been previously performed in a systematic way in humans, particularly not during exercise. For the majority of patients with heart failure, symptoms do not occur at rest but while they are performing activities of daily living. Because many patients with heart failure perform exercise through a cardiac rehabilitation program, determining the effects of deep lung inflations on heart rate and the effects of changes in lung volume and intrathoracic pressure on exercising stroke volume will allow the design of rationale strategies to limit negative cardiorespiratory interactions in this population. For example, patients could be trained to volitionally alter breathing strategy or to alter breathing strategy through the use of positive pressure breathing during exercise.

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Fig. 1. Tidal flow-volume responses to exercise in controls (CTL) and patients with heart failure (CHF). Data shown are mean values for all subjects (exercise 1–3 = 50%, 75% and 100% of VO_{2peak}). EILV: end-inspiratory lung volume, EELV: end-expiratory lung volume [2]. Patients with heart failure tend to breathe at low lung volumes and avoid high lung volumes during exercise.

Fig. 2.

For any given LV filling pressure (end-diastolic pressure: EDP), the greater the afterload, the less the stroke volume. As filling pressure is raised, the flow and volume displaced from the chamber increases for any given ejection pressures [26].

Fig. 3.

Effects of inspiration and expiration on cardiac hemodynamics in healthy individuals and in patients with heart failure. Arrows represent RV filling, RV output, LV preload and LV afterload. In the healthy heart, inspiration increases RV filling, RV output and LV afterload and decreases LV preload, while expiration decreases RV filling, RV output and LV afterload but increases LV preload. Patients with heart failure tend to be less sensitive to changes in LV preload while LV afterload has an overriding influence on stroke volume. Therefore, it is suggested that the overriding influence of LV afterload during inspiration results in a reduced stroke volume while the reduction in LV afterload with expiration allows an increase in stroke volume.

Fig. 4. Influence of inspiratory capacity (IC) maneuvers on heart rate in patients with heart failure (CHF) and controls (CTL) during exercise (unpublished observation).