Respiratory muscle/circulation interaction in congestive heart failure

Die Wechselwirkung der Atemmuskulatur und des Blutflusses bei Patienten mit manifester Herzinsuffizienz


Schlüsselwörter Atemmuskulatur – Blutfluss – Herzinsuffizienz

Summary This paper reviews respiratory muscle/circulation interaction in congestive heart failure. Respiratory muscle dysfunction consisting of both reduced pressure – generating capacity and reduced endurance is well documented in these patients. The mechanisms underlying its development and the potential consequences especially in the development of dyspnea are then presented. Not only are respiratory muscles dysfunctioning in these patients, but under specific circumstances they can impede the circulation, such as during weaning from mechanical ventilation. The underlying pathophysiology and the theoretical model explaining this interesting interaction is finally discussed.

Key words Respiratory muscles – circulation – congestive heart failure

Introduction

Heart failure is the pathophysiological state in which an abnormality of cardiac function is responsible for failure of the heart to pump blood at a rate commensurate with the requirements of metabolizing tissues or to do so only from elevated filling pressures (1). The clinical manifestations of heart failure, i.e. fatigue and dyspnea which limit exercise/activity tolerance are well recognized by physicians. The consequences of heart failure on respiratory muscle function though significant are not usually appreciated. These consequences will be the subject of the first part of the present review.

There are also circumstances where the action of the respiratory muscles poses a significant burden on the cardiovascular function of patients with heart failure. This is exemplified by congestive heart failure (CHF) patients who fail to wean, and should be rather expected since the respiratory muscles are not only functionally but also mechanically linked to the heart and...
circulation within the thorax. This cardiovascular dysfunction will be the subject of the second part of this review.

Respiratory muscle dysfunction in patients with heart failure

It is well established that heart failure compromises the pressure generating capacity of the respiratory muscles (2–11). The reduction in the pressure generating capacity of the inspiratory muscles has been a consistent finding in all studies addressing this issue (2–10). Hammond et al. (2) reported that the maximum inspiratory pressure (P_{I,max}) measured at the level of functional residual capacity (FRC) was (mean±SD) 41±6 cmH\textsubscript{2}O (with individual values as low as 20 cmH\textsubscript{2}O), representing only 40% the values of age-matched controls, and Mancini et al. (3) found a mean P_{I,max} of 46±22 cmH\textsubscript{2}O. Later studies have confirmed these results, although the compromise reported was less severe with average values ranging from 60 to 80% of either predicted or age-matched controls (4–10). The results were similar when the maximum inspiratory pressure was measured at residual volume (RV) (5). Reduced muscle strength was also documented for the diaphragm, the main inspiratory muscle (4, 10). The reduction in the pressure generating capacity of the expiratory muscles has been less consistent, with some studies reporting a decrease in the maximum expiratory pressure (P_{E,max}) at all lung volumes measured (2, 9, 10), whereas McParland et al. (5) found a reduction in P_{E,max} only at total lung capacity (TLC) and not at FRC, and Nishimura et al. (6) were unable to confirm a decrease in P_{E,max} (reporting only a tendency that was not statistically significant). All these results, obtained in stable ambulatory heart failure patients, indicate that there is an impairment in the force output of the respiratory muscles, with the inspiratory muscles suffering more severe weakness than the expiratory muscles.

The respiratory muscle dysfunction could theoretically be part of the generalized skeletal muscle dysfunction frequently observed in these patients (2), since the respiratory muscles are skeletal muscles. However, when peripheral and respiratory muscle strength was concurrently addressed in heart failure patients, the impairment in contractility of the respiratory muscles was greater (2, 11) (Fig. 1), and existed even in the absence of peripheral muscle dysfunction (11), which implies that the reduced respiratory muscle force output is not simply a part of a generalized skeletal muscle weakness.

It is also unlikely that the reduced P_{I,max} and P_{E,max} values recorded in these patients represent submaximal or poor effort, despite the fact that both manoeuvres are volitional. Most of the studies mentioned have used sex and age matched controls and the same encouragement was given to either group during the manoeuvres. More importantly, when Mancini et al. (4) applied the twitch interpolation technique of Bellemare and Bigland-Ritchie (12) (i.e. they superimposed diaphragmatic twitches elicited by supramaximal bilateral phrenic nerve stimulation upon maximal voluntary diaphragmatic contraction) they failed to obtain increased transdiaphragmatic pressures with the superimposition of twitches. This means that most of these patients were able to maximally activate their diaphragm, and consequently the decreased pressure generating capacity recorded in heart failure patients does not result from submaximal effort, but represents true reduction in contractility. This is also supported by animal models where a reduction in twitch transdiaphragmatic pressure was recorded at all stimulation frequencies (13, 14).

Apart from weakness, patients with heart failure also exhibit decreased endurance of the respiratory muscles. Accordingly, Mancini et al. (4) reported that the tension time index of the diaphragm TTI\textsubscript{di} [which is the product of the mean transdiaphragmatic pressure expressed as a percentage of maximum and the duty cycle (i.e. the time spent in inspiration/total respiratory cycle duration, T_{i}/T_{T})] was higher in heart failure patients than in normal controls both at rest (0.03±0.02 vs. 0.01±0.01, p<0.05) and during exercise (0.10±0.03 vs. 0.03±0.02, p<0.04). Bellemare and Grassino (15) have shown that the value of TTI\textsubscript{di} is inversely related to the endurance time (i.e. the time that a given load can be sustained without development of fatigue). Furthermore, maximal sustainable ventilatory capacity was significantly reduced in these patients (53±22 liters/min vs. 90±23 liters/min in controls, p<0.05). Both results indicate that...