Heart Failure and the Lung
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Heart failure (HF) is a highly prevalent disease that leads to significant morbidity and mortality. There is increasing evidence that the symptoms of HF are exacerbated by its deleterious effects on lung function. HF appears to cause airway obstruction acutely and leads to impaired gas diffusing capacity and pulmonary hypertension in the longer term. It is postulated that this is the result of recurrent episodes of elevated pulmonary capillary pressure leading to pulmonary oedema and pulmonary capillary stress fracture, which produces lung fibrosis. It is likely that impaired lung function impairs the functional status of HF patients and makes them more prone to central sleep apnoea. (Circ J 2010; 74: 2507–2516)

Key Words: Acute decompensated heart failure; Heart failure; Positive airway pressure; Pulmonary disease; Sleep apnoea

Heart failure (HF) affects 23 million people worldwide. Rates of hospitalisation for HF increased 4-fold between 1971 and 1999, corresponding with an aging population and greater survival for acute coronary syndromes, valvular disease and other related conditions. Shortness of breath and fatigue are 2 of the cardinal symptoms of HF and it is likely that impairment in lung function and sleep disturbance play a role. There is good evidence that lung function is significantly and permanently impaired in HF and that this may influence both functional status and mortality. It is also likely that impaired lung function contributes to the development of sleep disordered breathing (SDB) resulting in further deleterious effects for the patient. This review summarizes the evidence for the pathogenesis of lung disease in heart failure and its effects on both morbidity and mortality. We begin with a brief description of fluid homeostasis in the lung.

Fluid Balance in the Lung

The alveolar wall consists of epithelial cells through which run the alveolar capillaries. Separating the alveolar epithelium and the capillary wall endothelium is the interstitium. The interstitium is often narrow on 1 side of the capillary where it is formed by the fusion of alveolar and capillary basement membranes and thicker on the other side of the capillary where it contains type 1 collagen fibres. The thin section of interstitium facilitates gas exchange whilst the thicker section is involved in fluid exchange (Figure 1).

The capillary wall endothelium is permeable to water and solutes but restricts the movement of proteins. The alveolar epithelial cells are far less permeable to water and solutes and also actively pump water from the alveoli into the interstitium via a Na⁺/K⁺-ATPase pump.

Starling’s forces dictate the movement of water between the capillary and the alveoli. Hydrostatic forces push fluid out of the capillary whilst osmotic forces keep fluid in. In health there is a net outward flow of about 20 ml/h, which is taken up by the lymphatics.

The fluid leaving the capillaries enters the interstitium and tracks into the perivascular and peribronchial regions via the interlobular septae. These regions, which normally form a thin sheet around the arteries, veins and bronchi contain lymphatics, which actively pump lymph back to the bronchial and hilar nodes.

If excessive leak occurs, fluid begins to accumulate in the interstitium (Figure 2). This results in increasing hydrostatic forces and falling osmotic forces within the interstitium and helps to prevent further leak. As well as this, a gel-like protein in the thick component of the interstitium serves to absorb this excess fluid, keeping it away from the thinner gas exchange component.

Ongoing capillary leak results in pulmonary oedema, of which there are 2 stages. The first, interstitial oedema, results in the engorgement of the perivascular and peribronchial interstitial tissue (“cuffing”), septal thickening and increased lymphatic drainage but has little effect on pulmonary function. During this phase, lymphatic drainage may increase 10-fold and up to a quarter of total lung water may accumulate in the pleural space. Once enough water has entered the interstitium, it begins to leak through the alveolar epithelium into the alveoli resulting in reduced surface tension and alveolar collapse (Figure 3). There is loss of alveolar ventilation, which results in shunting of blood and thereby hypoxaemia. Alveolar fluid is removed actively by the alveolar epithelium as well as through the large airways (resulting in frothy, pink sputum).

The development of pulmonary oedema is multifactorial and can be the considered to result from:

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1. Increased hydrostatic capillary pressure (EG left ventricular failure)
2. Increased capillary permeability (EG acute lung injury)
3. Reduced lymphatic drainage (EG cancer with lymphangitis)
4. Decreased interstitial pressure (EG post pneumothorax)
5. Decrease colloid osmotic pressure (EG low albumin)
6. Unknown (high altitude, neurogenic, hyperinflation, heroin).

In HF the primary issue is increased hydrostatic capillary pressure.
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With progressive HF, there is increased left atrial pressure, which is transmitted into the pulmonary veins and capillaries, resulting in pulmonary hypertension. This can be assessed during cardiac catheterization by measurement of the “wedge” pressure—produced when the catheter has been wedged into a small pulmonary artery. Whether a rise in the pulmonary capillary wedge pressure (PCWP) results in pulmonary oedema seems to depend on the rate at which this pressure rises. Rapid rises in PCWP can result in pulmonary oedema at relatively low pressures, whilst those with long

Figure 3. Pulmonary oedema: capillary leak overwhelms the interstitial protective mechanisms as well as the lymphatic system. Alveolar fluid results in impaired gas exchange in hypoxaemia.

Figure 4. Chronic heart failure: in chronic heart failure fibrosis and lung compression as well as vascular changes result in impaired ventilation and gas exchange but reduce hydrostatic capillary leak.
standing pulmonary hypertension can tolerate remarkably high pressures without production of pulmonary oedema.

In hydrostatic pulmonary oedema, the alveolar oedema fluid has been traditionally thought of as having low-protein levels as capillary wall permeability remains intact. This was in contrast to pulmonary oedema due to disruption of the capillary wall in which protein levels would be relatively high. It is now apparent that during episodes of pulmonary oedema, some or the entire alveolar-capillary unit may be disrupted by elevated hydrostatic forces. This has been termed pulmonary capillary stress fracture and is visible on electron microscopy. Damage to the capillary endothelium and alveolar epithelium allows passage of red blood cells into the alveoli and also renders the alveolar–capillary membrane more permeable to fluid and protein. The resultant oedema fluid is of higher protein concentration than would be expected from a purely hydrostatic mechanism.

Pathological Changes in HF

There are a series of pulmonary vascular changes in those with chronic HF, which may explain their resistance to pulmonary oedema despite high pulmonary pressures. Most of the evidence for this comes from post-mortem or surgical biopsy studies of patients with advanced mitral valve disease. These studies have demonstrated pulmonary vascular abnormalities occur at and around the capillary bed including:

1. Increased capillary basement membrane thickness and capillary dilatation, whilst overall capillary density falls
2. Intimal thickening of the arteries and veins with muscularisation of the arterioles and venules
3. Circumferential fibrosis of both veins and arteries
4. Alveolar wall changes include; increased interstitial tissue, interstitial pericapillary oedema, haemosiderin deposition and alveolar wall thickening due to excess collagen, cuboidal (as opposed to flat) epithelium and increased type II pneumocytes
5. Compression of the peripheral airways by increased amounts of connective tissue
6. Bronchial smooth muscle hypertrophy.9-13

In patients with chronic mitral stenosis, pulmonary diffusing capacity falls with increasing peripheral artery intimal thickness and has been shown to be a good predictor of high pulmonary vascular resistance.13

In non-valvular HF, a small amount of animal and human data regarding pulmonary changes exist. A murine model demonstrated increased collagen formation and myofibroblast activity following myocardial infarct induced HF.14 Interestingly, in this study, these pulmonary changes did not occur in rats who were given an angiotensin II receptor inhibitor. Another study, in guinea pigs with HF induced by aortic artery ligation, demonstrated both vascular and septal thickening as well as increased dry lung weight.15

In humans, the known effects of non-valvular HF on lung histology is scant with 1 study assessing post-mortem patients with pulmonary oedema due to either renal or cardiac failure.16 Of the 16 patients studied, 10 had renal failure, 3 had cardiac failure secondary to ischaemic heart disease and 3 had valvular heart disease. The investigators found evidence of alveolar fibrosis and haemosiderin laden macrophages, but did not describe the vascular changes found in previous studies. The length of time of disease in these patients is unclear and therefore a relatively short duration of disease may explain the absence of these changes. This is demonstrated by another study of patients with various forms of valvular and congenital heart disease in whom capillary basement membrane changes were only found in those with a PCWP above 35 mmHg and a duration of heart failure over 6 years.17

The pathological changes described above may help to explain why patients with chronic HF appear far more resistant to acute pulmonary oedema than those with more recent disease onset and why some HF patients develop alveolar oedema whilst others develop ankle swelling (Figure 4). These changes appear to reduce capillary filtration rate at the alveolar level, increasing the hydrostatic pressure required to produce pulmonary oedema.15,18 This adaption, while protective in terms of pulmonary oedema, may lead to irreversible pulmonary hypertension and may explain why post-cardiac transplant patients with raised pulmonary vascular resistance...
have a poorer prognosis, especially if it is not reversible pharmacologically.19,20

**Effects of HF on Respiratory Function Testing**

This adaptation of the lung to HF means that any discussion of respiratory function testing in HF patients needs to take into consideration the duration of disease and the patient’s current clinical state. The degree of pathological change and volume status (or more specifically lung water) will have profound effect on the mechanical and gas exchange properties of the lung. The effect of posture on lung function is also important. Studies have shown increased flow limitation in patients with both acute21,22 and chronic, stable HF23 when placed in the supine position. This is likely to be caused by fluid shift from the lower limbs into the lung and upper airway resulting in increased airway resistance.24

There are few studies of patients with acute pulmonary oedema, most likely due to the difficulty involved in studying these patients (Table 1). A study in which 2L normal
saline was infused into 4 healthy subjects resulted in a reduction in total lung capacity (TLC) and vital capacity (VC), but did not affect flow volume loops. Conversely, infusion of 10 ml/kg of saline into 10 patients with a left ventricular ejection fraction (LVEF) <40% resulted in a fall in forced expiratory volume in 1 s (FEV1), forced expiratory ratio (FER) and diffusing capacity for carbon monoxide (DLCO). The same study also demonstrated lower baseline FEV1, FVC and DLCO in these subjects compared with a control group. Studies of patients undergoing acute hospitalisation for HF demonstrated increased pulmonary resistance and reduced compliance as well as reductions in FEV1, FVC and TLC but no change to DLCO compared to follow up testing. These latter 2 studies demonstrated rapid improvements in lung function following treatment for HF as did a study of patients presenting with acute myocardial infarction. Most recently, a multicentre Danish trial examining the effects of nolomizole in HF collected acceptable spirometry data in 527 of its 3,078 subjects. FEV1, FVC and FER was evaluated between 24 and 72h following admission with the diagnosis of HF at a time when investigators felt subjects no longer had pulmonary congestion. Mean FEV1 and FVC were reduced with an FER of 71%. In a follow up study, the authors were able to demonstrate that reduced FEV1, FVC and FER were independent predictors of mortality after 4.46 years of follow up. However, there was a high smoking rate in this cohort (~70%) as well as self reported chronic obstructive pulmonary disease (22%).

The changes in pulmonary function related to chronic HF have been more extensively studied, especially in those with mitral stenosis (Table 2). Patients with stable mitral valve disease demonstrate reduced FEV1, FVC and DLCO and increase residual volume (RV). These changes appeared to correlate with the severity of the valvular disease whether measured functionally or haemodynamically. There is also a significant inverse correlation between the cardiothoracic ratio and TLC and VC, suggesting at least some of the change is due to cardiac hypertrophy. A study that looked at patients following aortic or mitral valve replacement appears to confirm this, with patients TLC, function residual capacity, RV and compliance all improving post surgery in association with reducing cardiac size as determined by chest radiography. Interestingly, this study included both aortic and mitral valve replacements and demonstrated a greater degree of reversibility in those patients who had aortic valve disease. Within the mitral valve cohort, significant reversibility was only found in 3 of the 8 patients studied. Given aortic valve disease is often repaired quite promptly once symptoms occur and the 3 mitral valve patients had had shorter durations of disease than their peers, the authors postulated that chronic pulmonary congestion may have led to “irreversible lung damage”. The concept that restrictive lung disease in HF is caused by cardiac enlargement in a fixed thoracic cavity was further demonstrated by Olson and colleagues who measured cardiac, pulmonary and thoracic cage volumes using chest radiography. They found that cardiac size increased with increasing HF severity, but there was no change in the size of the thoracic cavity, thus resulting in reduced lung volume.

If chronic pulmonary congestion causes irreversible lung changes, then these findings should be consistent across all forms of congestive HF. A retrospective study assessed 132 patients being evaluated for heart transplant who had undergone pulmonary function testing. These patients had a mean ejection fraction of 19% and a cardiac index of 2.1 L·min⁻¹·m⁻². The mean duration of HF in this cohort was 3.4 years. The predominant finding was that of impaired diffusion, which affected 67% of the cohort. Reduced lung volumes were common but obstruction was absent except in those with a significant smoking history. Once again there was a correlation between heart size and degree of restriction, but no relationship was found between pulmonary function tests and other variables such as length or severity of disease. This contrasts with another study, which compared potential heart transplant recipients depending on maximum oxygen consumption (VO2max) and showed that those with a VO2max ≤14 ml·min⁻¹·kg⁻¹ (IE more severe HF clinically) had significantly poorer FER, TLC and inspiratory muscle strength compared to those with a VO2max >14 ml·min⁻¹·kg⁻¹. Another study has shown that as VO2max falls, so does DLCO. Siegel and colleagues found falls in DLCO correlated with reduced EF, but only in those patients found to have rales on clinical examination. Other studies have demonstrated similar reductions in lung volumes and gas transfer as well as reduced respiratory muscle strength, Alveolar–arterial oxygen gradient is also widened when compared with matched controls in patients with chronic, stable HF. Overall, it would appear that those with chronic HF have a restrictive ventilatory deficit with impaired diffusing capacity.

### Table 3. Studies of Pulmonary Function Following Cardiac Transplantation

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>n</th>
<th>Time post Tx</th>
<th>FEV1</th>
<th>FVC</th>
<th>FER</th>
<th>PEFR</th>
<th>TLC</th>
<th>RV</th>
<th>DLco</th>
<th>VA</th>
<th>KCO</th>
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<td>Mattauer</td>
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<td>21</td>
<td>Pre Tx</td>
<td>78</td>
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<td>73</td>
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<td>N</td>
<td>1.67</td>
<td>Mean duration of disease 7.3 years, *% predicted not given, result as (mmol/min/kPa) and (mmol/min/kPa/L)</td>
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<td>71</td>
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<td>14</td>
<td>Pre Tx</td>
<td>80</td>
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<td>1 month</td>
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<td>79</td>
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<td>N</td>
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<td>N</td>
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<td>17</td>
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<td>73</td>
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<td>15 to 10 months</td>
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Abbreviations see Tables 1, 2.
Mean results as: FEV1, FVC, TLC, RV, DLco, VA, KCO= % predicted, FER= %, PEFR=L/min.

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and that these changes correlate with reduced exercise capacity.

Several studies have been published that evaluate pulmonary abnormalities following cardiac transplantation (Table 3).\textsuperscript{43-45} These demonstrated that while lung function abnormalities persist for some time after transplant,\textsuperscript{43} most resolve after 12 months. The exception to this is gas transfer, which initially falls following transplant before improving, but does not return to normal.\textsuperscript{43} In an effort to delineate the reasons behind this permanent reduction in \(D_{lCO}\) 1 study divided the \(D_{lCO}\) into 2 components; membrane conductance and pulmonary capillary blood volume. The investigators found that in cardiac transplant recipients, membrane conductance remained low whilst pulmonary capillary blood volume fell (as left atrial pressures returned to normal).\textsuperscript{43} In this study, this resulted in a net fall in \(D_{lCO}\) compared with pre-transplantation. The authors also found that the reduction in membrane conductance and \(D_{lCO}\) correlated well with the duration of HF. Similar studies that examined the membrane conductance and pulmonary capillary blood volumes of 38 patients awaiting cardiac transplant\textsuperscript{42} and 191 patients in chronic, stable HF\textsuperscript{49} found both were significantly reduced. While a low pulmonary capillary blood volume appears counter intuitive given the high left atrial pressures in HF, the authors hypothesized that fibrosis, pulmonary vascular injury and perhaps small, recurrent pulmonary emboli may have played a role. This is further evidence that prolonged exposure to raised hydrostatic pressures damages the alveolar-capillary interface and results in irreversible impairment of gas exchange.

Cardiopulmonary exercise testing in patients who have undergone heart transplantation demonstrate that \(VO2_{\text{max}}\) does not reach normality and that about 40% of patients demonstrate reduced ventilatory efficiency.\textsuperscript{47} It is likely that reduced \(D_{lCO}\) contributes to this.

### Sleep Disordered Breathing

There is evidence that this reduction in diffusing capacity contributes to the development of SDB in HF. SDB is common in HF and is associated with increased sympathetic nervous activity,\textsuperscript{48} impaired function and increase mortality.\textsuperscript{49} SDB can be broadly divided into obstructive (OSA) and central sleep apnoea (CSA). For the purposes of this review we will concentrate on CSA and how changes in lung physiology in HF can influence its development. We have previously published a more detailed review of SDB in HF\textsuperscript{50}

CSA is present in 28–33% of those with advanced HF\textsuperscript{51-53} and is associated with increased morbidity and mortality. CSA is also known as Cheyne-Stokes respiration or periodic breathing and is characterized by alternating periods of hyper and hypventilation leading to fluctuating arterial \(CO_2\) levels. The underlying mechanism for this is that of an unstable (high gain) ventilatory control system and prolonged circulation time. It is well established that HF patients with CSA have prevailing hypcapnia\textsuperscript{54} awake and asleep with elevated ventilatory responses to \(CO_2\) awake\textsuperscript{55} and asleep (Arzt), which is likely due to elevated sympathetic activity\textsuperscript{56,57} related to the underlying HF. In addition, it is likely that impaired lung function contributes to this instability by reducing the lung’s ability to cope with changes in ventilatory requirements. Our laboratory has demonstrated that severity of CSA increases with reducing \(D_{lCO}\) and arterial \(O_2\) in HF patients.\textsuperscript{58} Nocturnal fluid shift also appears to have an effect. A series of studies have demonstrated that in healthy males and in those with HF, there is significant fluid shift from the lower limbs during sleep, which corresponds to an increase in neck circumference.\textsuperscript{59,60}

In HF patients, the degree of fluid shift from the legs appeared to correlate well with both increasing severity of OSA and CSA. While the change in OSA can be explained by increasing upper airway resistance due to neck oedema,\textsuperscript{34} how fluid shifts affect CSA is less clear. The authors postulated that fluid shifts into the lung parenchyma may result in afferent vagal nerve irritation, promoting hyperventilation, which leads to falling \(CO_2\) levels and eventual cessation of breathing.\textsuperscript{66} However, our laboratory has shown CSA in a patient with a vagally denervated lung secondary to lung transplantation.\textsuperscript{63} Thus an alternative explanation is that rostral fluid shift and associated increased lung water may increase CSA severity by impairing lung function.

The authors were able to demonstrate that continuous positive airways pressure (CPAP) prevented the neck circumference increase and reduced OSA severity.\textsuperscript{68} Unfortunately, CPAP was not provided to those patients with CSA and so the effect on these patients remains unclear. There has been a large, randomized, controlled study of the use of CPAP for CSA in HF patients, which failed to demonstrate significant mortality benefit,\textsuperscript{62} although there was improvement in ejection fraction, nocturnal oxygenation and 6-min walk distance. Sub-analysis suggested that mortality was improved, but only in those in whom CSA had been effectively suppressed.\textsuperscript{68} Bilevel positive airway pressure (BPAP) provides a pre-determined level of expiratory pressure with an increased level of inspiratory pressure. It has been used in the management of CSA\textsuperscript{65} and demonstrated improvement in AH1 in patients unresponsive to CPAP. Other studies have produced improvements in AH1, arousals, LVEF and sympathetic outflow\textsuperscript{55,67} but there is no data no long term outcomes such as mortality.

An alternative form of ventilation assistance known as adaptive servoventilation (ASV) has been demonstrated to have a greater effect than CPAP in terms of reduced CSA severity, increased LVEF and improved quality of life\textsuperscript{68} after 6 months in a small, randomized, controlled trial of systolic HF patients. ASV provides “background” CPAP with a variable degree of ventilatory assistance during the central apnoeas without aggravating the hypcapnia. An uncontrolled study has also demonstrated the benefit of ASV in reducing CSA in patients with diastolic HF.\textsuperscript{69} Currently there are large, multi-centre, randomized, controlled trials underway to determine if ASV provides significant morbidity and mortality benefits. There are no studies of the effects of CPAP, BPAP or ASV on lung function in chronic HF.

### Effect of Positive Airway Pressure on Lung Function in Acute Pulmonary Oedema

Several trials and meta-analyses have demonstrated significant clinical benefit from the use of CPAP in APO.\textsuperscript{70-73} This clinical benefit includes improved mortality\textsuperscript{70,73} as well as reductions in intubation rate and improvements in physiological parameters (dyspnoea, hypcapnia, acidosis and heart rate).\textsuperscript{70,72} The changes in physiology that contribute to this clinical benefit have been explored but the full picture is incomplete. A canine model of APO has shown that positive end expiratory pressure (PEEP) reduces extravascular lung water and increases lymphatic drainage of the lungs.\textsuperscript{74} PEEP in hydrostatic heart failure also results in a reduction in left ventricular size,\textsuperscript{75} likely to be caused by lung expansion. Reduced venous return due to a more positive intrathoracic
pressure also plays a role and acts to reduce preload on the heart. Heart rate variability, a marker of prognosis in HF, has been shown to be improved with CPAP.

CPAP also causes bronchodilation, which may help overcome the obstruction caused by airway oedema. It also increases end expiratory lung volume in patients with stable HF, an effect that is likely to be duplicated in those with APO. Lung compliance improves and lung resistance falls with CPAP reducing the work of breathing (WOB). There is also a reduction in the intrathoracic pressure swings and respiratory rate, representing a fall in respiratory muscle effort and leading to reduced left ventricular transmural pressure. It has been previously demonstrated that as WOB increases, there is significant increase in blood flow to the respiratory muscles and an attendant increase in oxygen consumption. CPAP’s role in reducing this WOB explains why overall oxygen consumption falls in HF patients on CPAP. Finally, because diaphragmatic muscle fatigue is related to muscle tension produced and amount of time in spent in inspiration, CPAP by reducing both intrathoracic pressure swings and respiratory rate prevents respiratory muscle fatigue and the development of hypercapnic respiratory failure.

Conclusions

Given their anatomical proximity and physiological interdependence it is not surprising that pathology in either the heart or lungs results in downstream effects in the other. The concept of lung disease resulting in pulmonary hypertension and right heart failure is well established in the medical literature and part of every medical school curriculum. Damage to the lungs due to heart disease has been less well understood but there is an increasing awareness that left cardiac pathology produces irreversible changes in the lungs which result in demonstrable morbidity and mortality. It is also apparent that treatment aimed at correcting lung pathophysiology, such as positive airway pressure, can lead to dramatic improvements in HF outcomes. It is hoped that greater understanding of the effects of HF on the lungs will produce further advancements in its management.

References


