

Effects of the mechanical ventilation on the cardiovascular system

DINKO TONKOVIĆ • ROBERT BARONICA •
DANIELA BANDIĆ PAVLOVIĆ • VIŠNJA NESEK ADAM •
ŽELJKO DRVAR • TAJANA ZAH BOGOVIĆ • SANJA SAKAN

DINKO TONKOVIĆ (✉) •
VIŠNJA NESEK ADAM
Department of Anaesthesiology
Reanimatology and Intensive Care
Clinical Hospital Sveti Duh, Sveti Duh 64
10000 Zagreb, Croatia
Phone: +38513712359
Fax: +38513712049
E-mail: dtonkovic@kbsd.hr

ROBERT BARONICA •
DANIELA BANDIĆ PAVLOVIĆ •
ŽELJKO DRVAR •
TAJANA ZAH BOGOVIĆ •
SANJA SAKAN
Department of Anaesthesiology
Reanimatology and Intensive Care
University Hospital Centre Zagreb
Zagreb, Croatia

ABSTRACT

Mechanical ventilation with positive pressure significantly influences cardiovascular system function. The main effects are a decrease of right ventricular preload and a decrease of left ventricular afterload. The initiation of mechanical ventilation in cases of hypovolemia with right ventricular dysfunction or severe lung disease adversely affects the cardiovascular system. Mechanical ventilation has beneficial effects on cardiovascular function in patients with left ventricular dysfunction and increased preload, afterload, oxygen consumption and metabolic disarrangements.

Key words: mechanical ventilation, heart failure, respiratory failure

Introduction

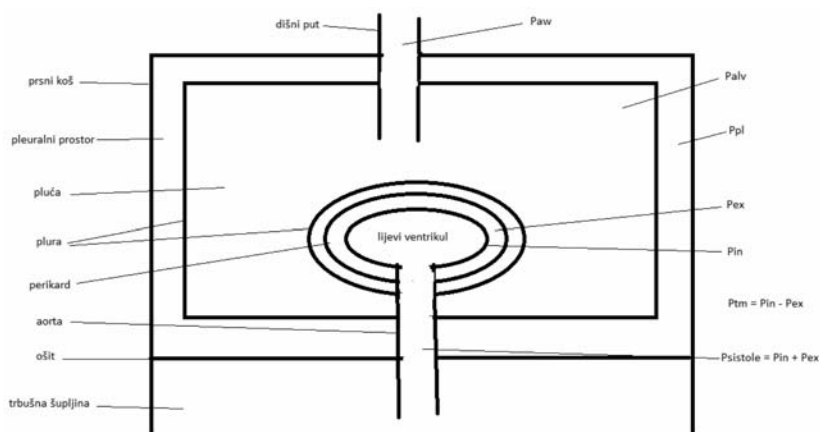
The main physiologic role of the cardiovascular and respiratory systems is oxygenation of tissues where they closely collaborate with permanent complementarity and interaction. Gas exchange is tasked to the lung, while the heart provides blood flow. A normal body spends 1-2 % of the total consumption of oxygen for gas exchange in the lungs. (1) In cases of respiratory failure this need increases up to 20 times. (1) The decrease in oxygenation and hypoxia rapidly lead to cell death, and the main task for the successful treatment of critically ill patients to maintain and support the functions of the heart and lungs. Today, modern intensive care medicine has many effective types of treatment of the

cardiovascular system (drugs, mechanical devices) and for respiration failure (artificial ventilation). Because of the tight physiological connection between the heart and lungs, medication therapy and therapeutic procedures to improve the functions of one system can often cause side effects to the other. Also, a number of heart and lung diseases significantly affect the interaction of the heart and lungs. The interaction of the heart and lungs is mentioned in 1733, when Stephen Hales described the reduction of blood pressure in spontaneous ventilation and again a century later when Kussmaul described the paradoxical pulse in patients with tuberculous pericarditis. (1) Today most of the physiological and pathophysiological processes in the interaction of spontaneous breathing and cardiac function are well known. (1,2,3). The effects of mechanical ventilation on cardiac function

are also well described. (2,3) An understanding of these interactions is fundamental for the proper diagnosis and therapy of cardiorespiratory failure with mechanical ventilation. (4,5)

The anatomy and physiology of the heart and lungs

The thoracic cavity contains the following organs: the heart, major blood vessels and lungs and pulmonary blood vessels. The heart and lungs are separated and wrapped with membranes, and the cavity is bounded by the ribs and diaphragm. (1,4) The mechanical properties of the organs in the thorax are variable and depend on the volume and elasticity. (1,4) Increasing or decreasing the volume of one organ changes the volume of another. The heart in the thoracic cavity works as two pumps (right and left ventricles)



Palv, alveolar pressure; Paw, airway pressure; Pex, extramural pressure; Pin, intramural pressure; Ppl, intrathoracic pressure; Psystole, aortic blood pressure; Ptm, transmural pressure

Figure 1. Schematic presentation of pressures in the thoracic cavity.

that are connected in series, between which stands pulmonary circulation. The blood squeezed from the right ventricle is the venous return of the left ventricle. The contraction of the left ventricle occurs with a delay of 1-2 heart beats because blood passes the pulmonary circulation. (1,2,4,5) The pressures that affect the heart, major blood vessels and lungs during spontaneous breathing and mechanical ventilation are listed in Table 1 and are shown in a Figure 1. (1,2,3)

Hemodynamic changes in spontaneous breathing

Due to the interaction of organs on each other in the thoracic cavity, changes in intrathoracic pressure have a direct effect on cardiac pressures. In spontaneous breathing the contractions of the diaphragm and intercostal muscles reduce intrathoracic pressure, leading to a greater pressure gradient according to the values of atmospheric pressure and air entering the lungs. (1,2,3) The decrease in intrathoracic pressure increases the pressure in the pericardial cavity, which directly increases the transmural pressure of the heart and increases the pressure load of the left ventricle, while at the same time increasing venous filling of the right ventricle. (1,3) Thus the main consequences of

the decrease in intrathoracic pressure during spontaneous breathing are an increase in afterload of the left ventricle and an increase in preload of the right ventricle. The hemodynamic result is a slight decrease in systolic blood pressure in spontaneous breaths. Other factors that influence the hemodynamic changes are interaction of the right and left ventricle and transposition of intrathoracic pressure on the large blood vessels (figure 1). Increased respiratory efforts during spontaneous breathing (asthma, pulmonary edema) or increased sensitivity to changes in transmural pressure in the heart (hypovolemia, tamponade, heart failure) leads to a decrease in systolic blood pressure during inspiration more than 10 mmHg, creating pulsus paradoxus. (1,4,5) The effect of spontaneous breathing on pulmonary blood vessels is negligible and rarely cause a clinically significant drop in systolic pressure. Also, lung volumes during spontaneous ventilation rarely affect the increase in pulmonary vascular resistance. (1,2,3)

Hemodynamic changes in mechanical ventilation with positive pressure

During mechanical ventilation, applying positive pressure forces the entrance of air into the lungs, increasing intrat-

horacic pressure. (3,6) This produces physiological effects that are directly opposite to normal spontaneous ventilation. The impact of the increase in intrathoracic pressure resulting from the application of non-invasive mechanical ventilation in the mask in healthy volunteers was described by the Cournad's group in the 1940s. (1) Similar changes have been described in the experiment of Valsalva where an increase in pressure in the airways with a closed glottis caused a decrease in venous return to the heart. (1) Hemodynamic effects of positive pressure ventilation includes the following: a decrease in venous return of the right and left ventricle, increases in the ventricles interaction, an increase in pulmonary vascular resistance, an increase in central venous pressure and a decrease in left ventricular afterload. (1,2,3,6) This leads to a drop in cardiac output and systolic blood pressure. The mentioned hemodynamic effects of mechanical ventilation are proportional to the amount of positive pressure, inspiratory volume and value of positive end-expiratory pressure. (1,4,5) A decrease in preload and blood pressure depends on the volume status of the patient and are more pronounced in conditions of reduced venous return (hypovolemia, vasodilation). (1,5) The decrease will also be greater with controlled modes of mechanical ventilation with high tidal volume and high airway pressure, thus the application of assisted mechanical ventilation modalities (BiPAP, CPAP, PSV) are favoured. (1,5) Hemodynamic changes caused by positive pressure produce compensatory sympathetic response with tachycardia, vasoconstriction, oliguria and retention of water and NaCl. (3,5)

The effects of heart diseases on hemodynamics in mechanical ventilation

Heart diseases are frequent in patients requiring mechanical ventilation and can have important hemodynamic effects during mechanical ventilation depending on the type and severity of

Table 1. Pressures in the thoracic cavity

Pressure	Definition
Paw - airway pressure	Pressure in the airways
Palv – alveolar pressure	Pressure in the alveoli
Ppl - intrathoracic pressure	Pressure in the thoracic cavity
Pex –extramural pressure	Extramural stress
Pin - intramural pressure	Intramural stress
Ptm - transmural pressure	Difference between intramural and extramural pressures
Psystole = Pin + Pex	Aortic blood pressure

the disease. The main physiological determinants of cardiac output are preload, contractility, afterload and heart rate. (1) Changes in cardiac output are the result of an increase in intrathoracic pressure, which causes a decrease in preload and afterload. (1,6) In cases where the cardiac output is dependent on venous return (hypovolemia, restrictive cardiomyopathy, tamponade and valvular stenosis) positive pressure ventilation will cause its further reduction. (1,5,7) In heart diseases with reduced ventricular compliance (coronary heart disease, fibrosis, hypertrophy) increased intrathoracic pressure of positive pressure ventilation will reduce afterload of the left ventricle and increase cardiac output. (1,4,5,6,7) In left ventricular dysfunction, manifested by increase in preload and afterload, mechanical ventilation increases cardiac output. (2,3) The most common example is ischemic diastolic dysfunction of the left ventricle with pulmonary edema. (7,8) In left ventricular failure an increase in preload and afterload with compensatory sympathetic activity also increases oxygen demand of the myocardium. (5,7) Those lead to a negative myocardial oxygen balance. In cases of impaired left ventricular contractility and coronary artery disease, the heart will not be able to compensate for the increased need for oxygen and increased effort for bre-

athing, which can increase up to 20 times and will result in cardiorespiratory arrest. (1,7,8) The application of mechanical ventilation in such cases will have a favourable effect on preload and afterload reduction of the left ventricle, and will reduce the need for oxygen with the correction of hypoxia and metabolic acidosis. (6,7) The effect of positive pressure ventilation on the right ventricle is not so favourable. The increase in intrathoracic pressure and positive end expiratory pressure increases pulmonary vascular resistance and impaired right ventricular function by reducing preload and increase afterload. (1,2) In this example, mechanical ventilation with small or large tidal volumes increases pulmonary resistance and will have a deleterious effect on right ventricular function and should be avoided. (1) An increase in pulmonary resistance will also result from hypoxia and metabolic acidosis caused by hypoperfusion in cardiorespiratory failure. (3,5) The effect of mechanical ventilation in this case is positive as it improves oxygenation and reduces metabolic acidosis. In right heart failure an increase of right ventricular volume has a negative effect on ventricular interaction by increasing the septum excursion that impairs the left ventricular filling. (1) Without a severe increase in pulmonary vascular resistance, the effect of the interaction of

the right and left ventricle on cardiac output is small. (1) Upon the initiation of mechanical ventilation, in patients with hypovolemic or septic shock, a reduction in blood pressure should be expected immediately at the beginning of the application due to reduced venous return. (4,5) To avoid that scenario, treatment should include volume therapy, choice of anaesthetics and analgesics during intubation and support of vasoactive drugs if necessary. (4,5,7) After the initiation of mechanical ventilation for the treatment of cardiorespiratory failure (especially associated with lung hyperinflation), reduced sympathetic tone can sometimes cause an increase in parasympathetic tone. (1,2,6)

Effects of lung diseases on hemodynamics in mechanical ventilation

Lung diseases affect the interaction of the heart and lungs in several ways. They pathologically change in lung volume and elasticity, increase airway resistance, increase work of breathing and increase interaction between the right and left ventricles. (1,2,3) Conditions that alter lung volume are the result of inflammatory diseases of the lung, atelectasis, pulmonary edema, and almost always arise as a result of reduced functional residual capacity after anesthesia. (1,3,7) Any reduction or increase in lung volume increases pulmonary vascular resistance and increases the load on the right ventricle. It also increases dependence of left ventricle function on the right ventricle and their interaction. For the prevention of acute right heart failure due to excessive increase in pulmonary vascular resistance, ventilation with large volumes and high pressure should be avoided. (4,5) To restore normal hemodynamics after the initiation of mechanical ventilation in patients with severe lung diseases, assisted ventilation modes should be used with the complementation of volume therapy and vasoactive drugs if necessary. (1,4,5,7) Diseases that increase resistance in the airways (asthma, COPD) help maintain air in the lungs at end exhalation, creating intrin-

sic positive end expiratory pressure. (1,2) The increase in lung volume decreases venous return of the right and left ventricles. The increase in intrathoracic pressure at expiration increases left ventricle afterload and reduces venous return, producing pressure on the major blood vessels and increasing the possibility of paradoxical pulse. (4,5) Extreme expiratory effort with increase in work of breathing can cause respiratory arrest and sudden death (severe bronchospasm). (1,6) The initiation of mechanical ventilation in such patients requires the application of assisted modalities and, if possible, ventilation with smaller volumes and increased expiratory time to reduce auto PEEP. (3,5) Increased work of breathing is present in the majority of lung diseases. Cardiac compensation of increased oxygen demand for respiration needs creates an energy load on the heart. With the presence of coronary artery disease, accompanied with hypoxia and acidosis, a further reduction in cardiac output may cause heart failure with a reduction of blood flow through the respiratory muscles, ending in cardiorespiratory arrest. (1,2,5) Mechanical ventilation has therapeutically beneficial effects on work of breathing by reducing oxygen consumption and preserving cardiac function. (4,6,8) An increase in pulmonary vascular resistance increases the afterload of the right ventricle and decreases venous return of the left ventricle. Severe incre-

ases in afterload could result in acute right ventricular dilatation, which reduces systolic function of the left ventricle due to the interaction of the ventricles. Decreases in cardiac output and hypotension further jeopardize myocardial function. Mechanical ventilation impairs right ventricular function, especially cases of ventilation by large volumes and pressures that increase pulmonary vascular resistance and decreases cardiac output. (1,2,3) In such cases it is necessary to apply assisted mechanical ventilation modalities when possible, with careful airway pressure and volume adjustment. (5,7) Other therapeutic measures include volume therapy for the maintenance of optimal venous return and right ventricular function support with inotropic drugs, while maintaining adequate tissue perfusion with vasoconstrictors. Volume replacement and titration of vasoactive medications should be individualized and guided by goal directed therapy using invasive hemodynamic monitoring. (1,4,5,7) In acute lung injury (ALI), cardiovascular function is impaired in several ways. Inflammatory reactions cause a reduction of lung elasticity, an increase in airway pressure and airway resistance, atelectasis, vasoconstriction, pulmonary hypertension and increased work of breathing with increased oxygen consumption followed by acidosis and hypoxia. (1) Those pathological effects lead to a deterioration of right ventricular function. Left ventricu-

lar function is also impaired by reduced preload, negative ventricular interaction, systemic hypotension, metabolic changes, systemic vasodilatation and myocardial depression caused by systemic inflammatory mediators. (1,5) Mechanical ventilation in patients with ALI is very challenging. High intrathoracic pressure must be avoided by using assisted ventilation modalities, if possible, with PEEP up to 15 mbar. (1,5) Hemodynamic support for cardiac function should include volume therapy, inotropes and vasoconstrictors. It is often necessary to use muscle relaxants and heavy sedation to be able to achieve gas exchanges. (1,5) The beneficial effects of mechanical ventilation consists of reducing hypoxia and acidosis correction to improve cardiac function. (8,7,9)

Conclusion

Mechanical ventilation with positive pressure significantly influences cardiovascular system function. The main effects are decrease of right ventricular preload and decrease of left ventricle afterload. The initiation of mechanical ventilation in cases of hypovolemia with right ventricular dysfunction or severe lung disease adversely affects the cardiovascular system. Mechanical ventilation has beneficial effects on cardiovascular function in patients with left ventricular dysfunction and increased preload, afterload, oxygen consumption and metabolic disarrangements.

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