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Pulsus paradoxus

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Abstract

Systolic blood pressure normally falls during quiet inspiration in normal individuals. Pulsus paradoxus is defined as a fall of systolic blood pressure of more than 10 mmHg during the inspiratory phase. Pulsus paradoxus can be observed in cardiac tamponade and in conditions where intrathoracic pressure swings are exaggerated or the right ventricle is distended, such as severe acute asthma or exacerbations of chronic obstructive pulmonary disease. Both the inspiratory decrease in left ventricular stroke volume and the passive transmission to the arterial tree of the inspiratory decrease in intrathoracic pressure contribute to the occurrence of pulsus paradoxus. During cardiac tamponade and acute asthma, biventricular interdependence (series and parallel) plays an important role in the inspiratory decrease in left ventricular stroke volume. Early recognition of pulsus paradoxus at the emergency room can help to diagnose rapidly cardiac tamponade. Measurement of pulsus paradoxus is also useful to assess the severity of acute asthma as well as its response to therapy. Recent development of non-invasive devices capable of automatically calculate and display arterial pressure variation or derived indices should help improve the assessment of pulsus paradoxus at the bedside.

Introduction

The current definition of pulsus paradoxus is a fall of systolic blood pressure of more than 10 mmHg during the inspiratory phase [1] (Figure 1). The paradox described by Adolf Kussmaul in 1873 was a “pulse simultaneously slight and irregular, disappearing during inspiration and returning upon expiration” despite the continued presence of the cardiac impulse during both respiratory phases.

Pulsus paradoxus can be observed in cardiac tamponade and in conditions where intrathoracic pressure swings are exaggerated or the right ventricle is distended, such as severe acute asthma or exacerbations of chronic obstructive pulmonary disease (COPD).

There is no consensus on the underlying mechanism of pulsus paradoxus. It is likely that several different mechanisms can contribute to the occurrence of pulsus paradoxus. Their respective importance depends on the underlying aetiology (Table 1).

Heart-lung interactions during spontaneous breathing

To well understand the complex mechanisms of pulsus paradoxus, we review first the basic principles of heart-lung interactions during spontaneous breathing.

Spontaneous inspiration is associated with decrease in intrathoracic pressure (ITP), increase in abdominal pressure and increase in lung volume, which all interfere with circulation in a complex manner. Another important mechanism, which can play a role in the heart-lung interactions, is the biventricular interdependence phenomenon.

Inspiratory decrease in ITP

Effects on systemic venous return

Because the right atrium is located within the thorax, inspiration leads to a decrease in right atrial pressure relative to atmosphere. As right atrial pressure is the backpressure to systemic venous return, this should result in augmentation of the pressure gradient of systemic venous return (i.e. mean systemic pressure minus right atrial pressure) and

accelerate blood flow toward the right heart [2, 3]. In normal conditions and considering the steep part of the Frank-Starling relationship (stroke volume *vs*. ventricular preload), an increase in ventricular preload is associated with an increase in stroke volume (so-called preload-dependency condition). This latter phenomenon explains the increase in right ventricular (RV) stroke volume (SV) and pulmonary arterial flow [2, 4] that occur at inspiration in normal conditions. However, the augmentation of systemic venous return has its limitation: if right atrial pressure decreases below the atmospheric pressure, the systemic veins collapse as they enter the thorax, thus limiting blood flow [5]. It has to be noticed that in case of prior RV dilation or dysfunction, the right ventricle can operate on the flat part of its Frank-Starling curve, so that the increase in RV preload would not induce an increase in RVSV (preload-independency condition).

Effects on RV afterload

The negative ITP at inspiration may also impede RV afterload. Indeed, the pressure surrounding the right ventricle (i.e. ITP) decreases while the pressure surrounding the intra-alveolar vessels (i.e. alveolar pressure) is close to atmospheric pressure at end-inspiration. Therefore, the right ventricle, which must eject blood into the alveolar arterial bed, has to generate a higher pressure - i.e. transmural pressure - before blood can reach alveolar vessels. Thus, the right ventricle senses this condition as increased impedance to its ejection [6]. In fact, this phenomenon is only transient because at the same time, the increased driving pressure between alveolar vessels and left atrium (surrounded by a negative ITP) facilitates blood flow to left atrium and thus results in a reduction in blood volume and pressure in the alveolar vessels until the initial pressure was restored in this new steady-state [6]. It must be remembered that SV is much more sensitive to RV afterload increase than to left ventricular (LV) afterload increase in healthy subjects.

Effects on left ventricular afterload

During inspiration, the pressure surrounding the left ventricle (i.e. ITP) decreases while the pressure surrounding the extrathoracic arterial compartment remains constant. As

the left ventricle must eject blood toward the extrathoracic arteries, it must generate a higher pressure - i.e. transmural pressure - before blood can leave the thorax. In other words, lowering ITP would be equivalent to raising the arterial pressure by a similar amount and both conditions are sensed as an increased LV afterload [7]. This hypothesis is well illustrated during a Mueller manoeuvre in healthy volunteers, where simultaneous increases in transmural arterial pressure, in end-systolic and in end-diastolic LV volumes, are observed [8]. Similar effects were reported in cardiac surgery patients [9] and in patients with coronary artery disease [8]. However, reduced LV preload rather than increased LV afterload was found by Brinkler et al. using 2D echocardiography during a Mueller manoeuvre in normal men [10].

Interestingly, in a series of dog experiments, Peters et al. demonstrated that a decrease in ITP confined to diastole could diminish the ensuing LVSV through reduction in LV preload (see below) [11] while a decrease in ITP confined to systole could increase LV afterload [12].

In animal experiments, Robotham et al. observed that during spontaneous ventilation, approximately 80 % of the time, the minimal and maximal integrated mitral flows preceded the respective minimal and maximal integrated aortic flows [13]. This suggests the dominance of preload effects on afterload effects in determining LVSV [13].

All these findings underline the difficulty to analyze cardiopulmonary interactions with respiration and partly explain some divergent results found in the literature.

Inspiratory increase in abdominal pressure

At the right side

During spontaneous inspiration, the abdominal pressure increases due to the active descent of the diaphragm. Takata et al. have proposed the concept of abdominal vascular zone conditions analogous to pulmonary vascular zone conditions [14]. In this connection, when the intravascular status is low, the increased abdominal pressure may collapse the inferior vena cava and hence decreases the systemic venous return from the inferior vena cava. When the intravascular volume is high, no critical closure pressure phenomenon is observed since the pressure surrounding the inferior vena cava does not exceed the

intraluminal pressure. In this condition, the increased abdominal pressure raises the gradient for systemic venous return. Thus, the net effect of increased abdominal pressure in presence of normo- or hypervolemia is an increased systemic venous return.

At the left side

Increased abdominal pressure during inspiration increases the intraluminal pressure of the abdominal aorta and, thus, the impedance to LV ejection, independently of the effect of decreased ITP [15].

Inspiratory increase in lung volume

Effects on RV afterload

The relationship between lung vessels resistance and lung volume is complex.

From a series arrangement viewpoint, the pulmonary circulation may be divided in extra-alveolar vessels and intra-alveolar vessels [6]. Inspiratory increase in lung volume compresses lumens of intra-alveolar vessels resulting in an exponential increase in intraalveolar vessels resistance [Figure 2]. By contrast, increased lung volume induces an exponential decrease in extra-alveolar vessels resistance from residual volume to total lung capacity [Figure 2]. Indeed, as lung volume increases, the radial interstitial forces increase, resulting in widening of extra-alveolar vessels diameters. Thus, the total pulmonary vascular resistances describe a U shape with a nadir corresponding to a lung volume equal to the relaxation volume or functional residual capacity [16] [Figure 2]. In healthy subjects, there is a slight increase in pulmonary vascular resistances during tidal inspiration. This effect can be more marked during exacerbation of COPD or asthmatic attack where the end-expiratory lung volume is significantly higher than the relaxation volume.

From a parallel arrangement viewpoint, the pulmonary circulation is distributed along a gravitational gradient of vascular-alveolar pressure difference [6]. By raising transpulmonary pressure (alveolar pressure minus ITP), inspiration may cause a larger proportion of the pulmonary circulation to behave as West's zone 2, especially when the

pulmonary venous pressure is low. This contributes to increase pulmonary vascular resistance and RV afterload, especially in cases of low blood volume states [17].

Effects on LV afterload

During spontaneous inspiration, a decrease in the LV septal-lateral dimension consistent with compression of the heart in the lateral direction was reported [18]. However, Scharf et al. showed that the compressive effect of lung inflation probably plays a less important role during spontaneous inspiration than during mechanical ventilation with positive end-expiratory pressure [19].

Biventricular interdependence

Biventricular interdependence can occur in a parallel or in a series manner.

Parallel biventricular interdependence

The two ventricles are surrounded by a poorly expandable membrane, the pericardium. During inspiration, the increase in RV end-diastolic volume increases the surrounding pericardial pressure relative to the ITP. This, in turn, increases the pressure in the left atrium and thus reduces the left atrial filling. In addition, if the RV filling pressure increases enough during inspiration, the septum shifts leftward [Figure 3]. This leads to a decreased compliance of the left ventricle, which further limits its filling. The presence of the pericardium, markedly enhances the degree of diastolic ventricular interaction [20].

Series biventricular interdependence

Blood pumped from the right ventricle travels through the pulmonary circulation to the left atrium. Thus, respiration-induced changes in RVSV affect LV filling. Because the pulmonary transit time is long (several seconds), the SV, which is maximal during inspiration at the output of the right ventricle can be maximal during expiration at the output of the left ventricle, and vice versa. The series ventricular interaction, which is operating in case of biventricular preload-dependency [Figure 4], likely plays a role in the changes in pulse pressure and LVSV variation observed during quiet spontaneous breathing in normal

subjects [21]. However, this could be different in cases of low respiratory rate or change in the inspiratory-to-expiratory time ratio. In addition, there is a great variability of the pulmonary transit time even in normal subjects at rest (between 5 and 10 seconds) [22]. The pulmonary transit time decreases when cardiac output increases and reaches its minimum value when cardiac index is about 8 L/min/m^2 [22]. The pulmonary transit time can be increased in congestive heart failure [23] or pulmonary hypertension [24], independently of the value of cardiac output. For all these reasons, it is difficult to predict which role the series ventricular interaction can play in the occurrence of LVSV respiratory variation. When the two ventricles are 180° out of respiratory phase, this can enhance the degree of LVSV variation. When the two ventricles are in phase, the LVSV variation due to other mechanisms could be attenuated or even absent.

Mechanisms of pulsus paradoxus

Respiratory changes in arterial pressure during normal breathing conditions

In healthy subjects, quiet inspiration results in a decrease in systolic arterial pressure [25], which can be related to either the direct transmission to the arterial tree of the decreased ITP [26] or to the decrease in LVSV [27-30], or more probably both. As detailed above, the inspiratory decrease in LVSV, which is associated with inspiratory increases in vena caval flow [3, 31], RV end-diastolic volume, RVSV and pulmonary arterial flow [2, 3, 29], can be the result of decrease in LV preload or LV afterload, or both. It is unlikely that the small decrease in ITP during quiet inspiration in healthy individuals (around 5 mmHg) can result in a significant decrease in LVSV related to increased LV afterload. In this regard, LV end-diastolic volume was shown to decrease rather than to increase during inspiration [27, 28]. The reduction in LV preload during inspiration, which is thus the most plausible explanation could be due: 1) either to series ventricular interaction (see above) making the right and left ventricles 180° out of phase, 2) or to reduction in LV diastolic time related to the increased heart rate accompanying inspiration 3) or to parallel ventricular interdependence phenomenon. It is unlikely however, that the latter mechanism is

significant in the absence of pericardial constraint or of RV overdistension during normal inspiration [32]. Another mechanism suggested by some investigators [25, 33] could be an inspiratory pulmonary venous blood pooling related to the fact that pulmonary vessels are supposed to be more compliant than the left ventricle.

Since in physiological conditions, the inspiratory decrease in ITP is low (< 5 mmHg) and the inspiratory decrease in LVSV is small (< 10%) [4, 25], the inspiratory decrease in systolic arterial pressure is physiologically < 10 mmHg (around 5 mmHg) [25].

Pulsus paradoxus in cardiac tamponade

Cardiac tamponade is a pathological condition where fluid accumulation in the pericardial sac surrounding the heart causes elevation and equilibration of pericardial and cardiac chamber pressures, reduced cardiac output, and pulsus paradoxus.

Different mechanisms leading to pulsus paradoxus (Table 1) can been involved.

In this situation, ITP decreases during inspiration less than in acute asthma but probably more than in normal conditions. Because the right atrial pressure is high at expiration, the decrease in ITP should increase more the inferior vena cava flow than in normal conditions as the inferior vena cava flow limitation is attenuated. The large inspiratory increases in vena cava flow [31, 34] and RV filling should result in a large inspiratory increase in RVSV as confirmed by experimental [2, 34] and clinical [4, 25] studies. In an experimental model of cardiac tamponade, Shabetai et al. found that when venous return to the right atrium was held constant throughout the respiratory cycle (right heart bypass), pulsus paradoxus did not appear anymore [31]. This confirms the important role of biventricular interdependence (parallel and/or series) in the development of pulsus paradoxus. Unlike normal conditions, cardiac tamponade should be associated with a marked parallel biventricular interaction because of space limitations imposed by the pericardial constraint. In accordance with this potential mechanism, Settle et al., using echocardiography, reported increased RV dimensions and decreased LV dimensions during inspiration. In each instance, this was caused by a posterior movement of the interventricular septum toward the left ventricle in inspiration [35]. Both parallel and series biventricular interdependence mechanisms should

contribute to decrease LV filling and hence to decrease LVSV at inspiration if the left ventricle is preload-dependent. Ramachandran et al. used a human cardiovascular-respiratory system model that simulates hemodynamic and respiratory changes associated with tamponade clinically [36]. They showed that both the parallel and series interactions have an equal and significant contribution to LVSV respiratory variation, and hence to pulsus paradoxus [36].

Pulsus paradoxus in acute asthma

Presence of large inspiratory decrease in ITP and breathing at high lung volume are the main characteristics explaining the occurrence of pulsus paradoxus during acute asthma. The large decrease in ITP during inspiration (- 20 to - 30 mmHg) [37] can affect systolic arterial pressure through both direct mechanism (passive transmission along the arterial tree) and indirect mechanisms (decrease in LVSV). Jardin et al., studying nine patients with acute asthma, reported a mean inspiratory decrease in systolic arterial pressure of 41 mmHg, in arterial pulse pressure of 23 mmHg and in diastolic arterial pressure of 18 mmHg [37]. The decrease in pulse pressure suggests that LVSV decreases at inspiration while the large decrease in diastolic pressure suggests that passive transmission of ITP along the arterial tree does not play a minor role.

It is far to be certain that pulsus paradoxus in acute asthma is only the exaggeration of the inspiratory decrease in systolic arterial pressure in healthy subjects. In both conditions, the right atrial pressure is generally not high at expiration. As the inferior vena cava flow is limited during deep inspiration when the right atrial pressure falls below the atmospheric pressure, the increase in systemic venous return is thus not exaggerated. At the same time, RV afterload increases because of both the deep decrease in ITP (see above) and the effect of lung inflation of the pulmonary vascular resistance. As detailed above, in acute asthma where functional residual capacity is markedly increased [38], the inspiratory increase in lung volume should result in an increased pulmonary vascular resistance. The ensuing increase in RV afterload was well illustrated in seven patients with acute asthmatic attack

[39]. Using 2D echocardiography examination in the short axis, Jardin et al. measured significant increases in RV end-systolic and end-diastolic areas at inspiration [38], findings that were not reported in healthy volunteers [40]. Moreover, RV stroke area and pulmonary artery pulse pressure decrease during inspiration, findings that are opposite to what is expected in normal subjects [4]. However, divergent findings were reported in a clinical study including asthmatic patients with cold air bronchial hyperreactivity before and during a bronchial challenge with cold air mimicking an asthmatic attack [41]. In this study, RV and LV volumes and ejection fractions were measured using radionuclide angiography during inspiration and expiration [41]. The bronchial challenge produced pulsus paradoxus and changes in esophageal pressure (measuring ITP) that were significantly greater than those during quiet breathing [41]. These changes were accompanied by a decrease in LV diastolic volume and in LVSV during inspiration, and an increase in these variables during expiration; RV diastolic volume and RVSV demonstrated changes reciprocal to those seen in the left ventricle [40]. Although these two clinical studies [39, 41] found divergent results in terms of RVSV during inspiration, both reported a marked inspiratory increase in RV end-diastolic volume and suggested that this mechanism is responsible for the occurrence of pulsus paradoxus. It is thus likely that in acute asthma, the increased systemic venous return and presumably the increased RV afterload during inspiration result in an inspiratory RV overdistension [39, 41] and thus in a reduced LV filling [37, 41] at the same time through parallel biventricular interdependence. If the left ventricle is preload-dependent, this could result in a decrease in LVSV at inspiration [37, 41].

Other mechanisms of pulsus paradoxus (Table 1) can be discussed.

Inspiratory increases in LV afterload could play a role. Although at the right side, a marked fall in ITP does not induce an exaggerated increase in RV preload because of inferior vena cava flow limitation, it can result at the left side in a marked increase in LV afterload. Even if it exists, this effect is difficult to identify because of the probable predominant biventricular interdependence effect.

Series biventricular interdependence could play a role in the development of pulsus paradoxus because the two ventricles are 180° out of phase, if respiratory rate, inspiratory-

to-expiratory ratio and pulmonary transit time are normal. This could be different in some instances during acute asthma, for example if the respiratory rate is abnormally low and the pulmonary transit time reduced in relation to a high cardiac output.

Studying patients with airway obstruction during normal respiration, and after a brief period of apnea, Ruskin et al. brought arguments against a major role of biventricular interdependence (series or parallel) [25]. Indeed, the fact that the inspiratory decrease of LVSV following a period of apnea was identical in magnitude with that observed during continuous respiration indicated that any contribution to respiratory-related alterations of LVSV by preceding alterations of RV output during continuous respiration must have been minimal [25]. They suggested that the inspiratory fall in LVSV during inspiration resulted from transient inspiratory pooling of blood in the pulmonary veins with a resultant decrease in LV filling [25].

Finally, the role of volume depletion was raised by Squara et al. in a clinical study performed in 10 patients with acute asthma and pulsus paradoxus [42]. Pulsus paradoxus decreased during military antishock trouser (MAST) inflation and returned to baseline values after MAST deflation [41]. Interpretation of these results is speculative. A first possibility is that MAST inflation made the heart (right ventricle and/or left ventricle) preload-independent such that any change in RV or LV end-diastolic volume did not anymore result in parallel change in RV or LVSV. A second possibility is that volume loading has reduced the extent of West's zone 2 and thus the degree of inspiratory increase in pulmonary vascular resistance and RV afterload and hence the degree of parallel biventricular interdependence. Although this study did not indicate with certainty what the major mechanism of pulsus paradoxus is in acute asthma, it clearly indicates that the direct and passive transmission of decreased ITP on the arterial system cannot be the sole mechanism.

Given all these divergent findings, it is difficult to propose a unique theory to explain pulsus paradoxus during acute asthma. It is likely that several mechanisms are involved.

Mechanisms of pulsus paradoxus during acute exacerbation of COPD could be a little different from acute asthma. First, unlike in asthma, right atrial pressure is generally high at expiration in COPD patients during acute exacerbation, so that the degree of vena cava flow

limitation during the ensuing deep inspiration should be attenuated. In this connection, a large inspiratory RV filling is expected to occur. In addition, lung hyperinflation and gas trapping could result in a positive intrathoracic pressure at expiration, in relation to the development of intrinsic positive end-expiratory positive pressure. This should result in a decrease in systemic venous return and RV preload at expiration, which is expected to be more marked in low central venous pressures states [43]. Combination of these two effects (decrease in systemic venous return at expiration and enhanced systemic venous return at inspiration) should result in a marked respiratory change in systemic venous return and RV stroke volume and thus in the development of pulsus paradoxus through series biventricular interdependence. In addition, RV afterload should increase during inspiration (breathing at high lung volume). During acute exacerbation of COPD, where the right ventricle can be already enlarged, combination of increased RV preload and increased RV afterload during inspiration should result in a marked parallel biventricular interdependence phenomenon, as demonstrated by Settle in an echocardiographic study [32]. This can contribute to the development for pulsus paradoxus during acute exacerbation of COPD. As in acute asthma, direct transmission of decreased ITP and increased LV afterload during inspiration can also play a role in the development of pulsus paradoxus [26].

Finally, pulsus paradoxus may scarcely be observed in massive pulmonary embolism, large compressive pleural effusion, profound hypovolemia and tricuspid atresia.

Clinical implications

Measurement of pulsus paradoxus

Pulsus paradoxus is traditionally measured using a sphygmomanometer. The brachial cuff must be inflated above the presumed value of systolic arterial pressure and then deflated slowly to find the highest pressure at which the first Korotkoff sound is heard, normally during expiration. The cuff must be further deflated until the pressure at which the Korotkoff sounds are heard during both inspiration and expiration. Pulsus paradoxus is present if the difference between the latter and the former pressures is greater than 10

mmHg. In general, the procedure is repeated two or three times to improve the accuracy of the method. However, this method is cumbersome and time-consuming (between 2 and 5 minutes) [44]. In addition, in some patients, audibility of sounds is poor because of tachypnea and noisy clinical environment (emergency room). In a great number of cases, experienced physicians failed to measure reliably the amplitude of pulsus paradoxus [45]. Because of all these drawbacks, more than 98% of care providers do not use the manual measurement at the bedside in acute asthma [46]. Alternative methods have been proposed to measure pulsus paradoxus, in particular methods allowing automatic and real-time measurements such as non-invasive blood pressure monitors [47], and pulse oximetry [48]. In a series of 26 patients with COPD, respiratory waveform variation of pulse oxymetry closely correlated with pulsus paradoxus measured using sphygomanometry [48]. In the context of intensive care unit environment, invasive monitoring of blood pressure using an arterial catheter provides an accurate real-time assessment of pulsus paradoxus.

Clinical significance of pulsus paradoxus

Cardiac tamponade

Guberman et al. reported that pulsus paradoxus is detectable in 98% of patients with cardiac tamponade even in the absence of hypotension [49]. Its early recognition can thus help to suspect rapidly the diagnosis, which must be confirmed by echocardiography. After pericardiocentesis, pulsus paradoxus and respiratory variation in transvalvular flow velocities decrease [4].

Acute asthma

Measurement of pulsus paradoxus during acute asthma exacerbations is currently recommended by international guidelines [50]. It is considered as a surrogate of airflow obstruction indexes in the emergency ward [51, 52] and is a key variable of the severity scores developed in acute asthma [50, 52-54]. Of note, in cases of severe respiratory distress with respiratory exhaustion, swings in ITP can be reduced and pulsus paradoxus can be absent.

Until now, pulsus paradoxus is not frequently measured because of the cumbersomeness of the manual method. Development of continuous non-invasive methods should make more frequent the measurement of pulsus paradoxus at the emergency room, and thus easier the clinical assessment of severity and response to treatment. In asthmatic patients presenting at the emergency room, arterial tonometry was used for automated measurement of inspiratory decrease in systolic pressure [45]. A value of 11 mmHg measured after 60 minutes of standardized asthmatic treatment allowed discriminating patients who were eventually discharged from those who were eventually hospitalized (triage use) [45]. Non-invasive finger blood pressure monitors can also assess pulsus paradoxus and its response to treatment [45]. Importantly, a variable derived from pulse oximetry respiratory waveform variation was demonstrated to correlate with airflow obstruction indexes in patients with acute asthma [52]. Incorporation of such variables into pulse oximeters has been proposed to improve clinical assessment of asthma severity and to easily follow the response to treatment in particular in patients who cannot perform spirometry because of young age or severity of illness [52].

Pulsus paradoxus can be present in other acute or chronic airway obstruction diseases. Pulse oximetry respiratory waveform variation was demonstrated to correlate with the degree of air trapping in patients with COPD [48]. In children who presented to the emergency department with signs of croup, the amplitude of pulsus paradoxus was correlated to the Westley croup score, and the degree of change in this amplitude in response to epinephrine was correlated to the change in the Westley croup score [55]. In obstructive sleep apnea [56], the presence of pulsus paradoxus is also a marker of severity since it indicates a marked inspiratory decreased ITP [56]. Its disappearance after continuous positive airway pressure application can be an indicator of treatment efficiency [56].

It is noteworthy that respiratory changes in arterial pulse pressure can occur during mechanical ventilation. The so-called pulse pressure variation is considered as a marker of biventricular preload-dependency and as such, a reliable indicator of fluid responsiveness in mechanically ventilated patients [57, 58]. The purpose of the present article is not to review

the mechanisms, the significance and the limits of pulse pressure variation, since they have been extensively detailed in the recent literature [58, 59].

Conclusion

Biventricular interdependence (series and parallel) and passive transmission to the arterial tree of the inspiratory decrease in ITP, play an important role in the appearance of pulsus paradoxus during cardiac tamponade and acute asthma. Recent development of non-invasive devices capable of automatically calculate and display arterial pressure variation or derived indices, should make easier the assessment of pulsus paradoxus at the bedside.

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Table 1: main mechanisms contributing to the decrease in systolic blood pressure during inspiration

Mechanisms	**Normal subjects**	**cardiac tamponade**	**acute asthma**
Passive transmission of decreased ITP during inspiration	**+**	**+**	**++**
Blood pooling in the pulmonary veins during inspiration	**+/-**	**+/-**	**+/-**
Series biventricular interdependence (pulmonary transit time)	**+**	**++**	**+**
Parallel biventricular Interdependence (septum and pericardium)	**0**	**++**	**+++**
Decreased LV afterload during inspiration	**0**	**+**	**+**

Legends of Figures

Figure 1

Recording of arterial pressure measured using an arterial catheter in a patient with acute exacerbation of airway obstruction. Pulsus paradoxus is present as the decrease in systolic arterial pressure is > 10 mmHg. Note that the arterial pulse pressure also decreases at inspiration suggesting that LV stroke volume decreases at inspiration.

The grey areas indicate the inspiratory phases (Insp). The arrow indicates the amplitude of pulsus paradoxus in this example (about 17 mmHg)

Figure 2

Relationships between pulmonary vascular resistances and lung volume (see text for detailed explanations).

FRC: functional residual capacity; TLC: total lung capacity; RV: residual volume.

Figure 3

Parallel biventricular interdependence

In some pathologic conditions, inspiration results in a marked dilation of the right ventricle (RV). Because expansion of the pericardium is limited, this will result in a leftward shift of the interventricular septum and thus, in impediment of the left ventricle (LV) filling. This in turn, results in a decrease in the left ventricular stroke volume during inspiration.

Figure 4

Series biventricular interdependence

Inspiration results in increase in filling and stroke volume (SV) of the right ventricle (RV). Because of the long pulmonary transit time, there is a phase lag between the RV and the left ventricle (LV) so that LV SV is maximal during expiration. All these mechanisms are fully operating in cases of biventricular preload-dependency (i.e. the RV and LV operating on the steep part of their Frank-Starling curve).

Figure 1

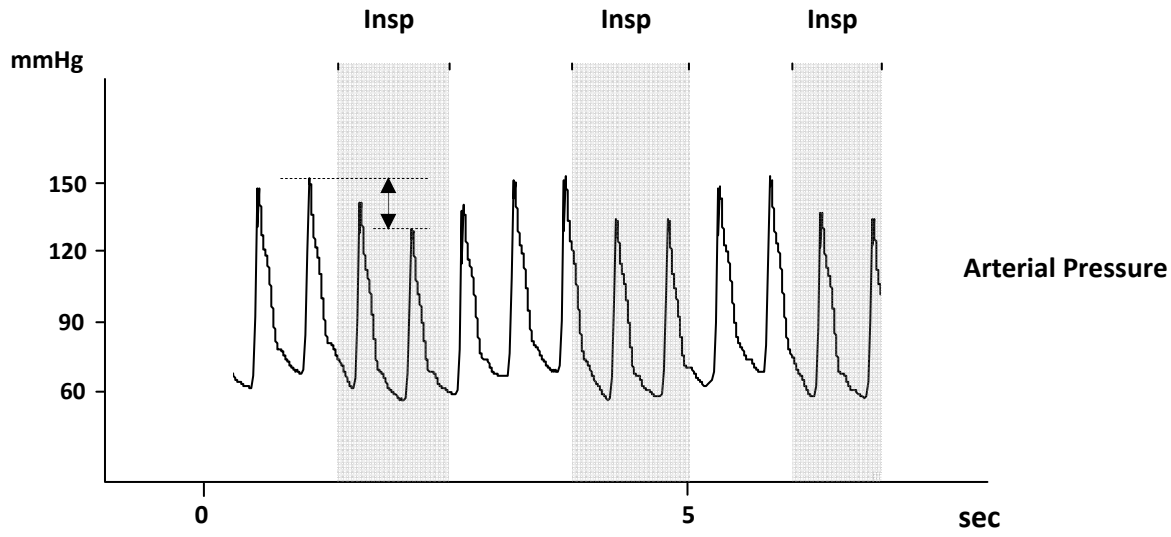


Figure 2

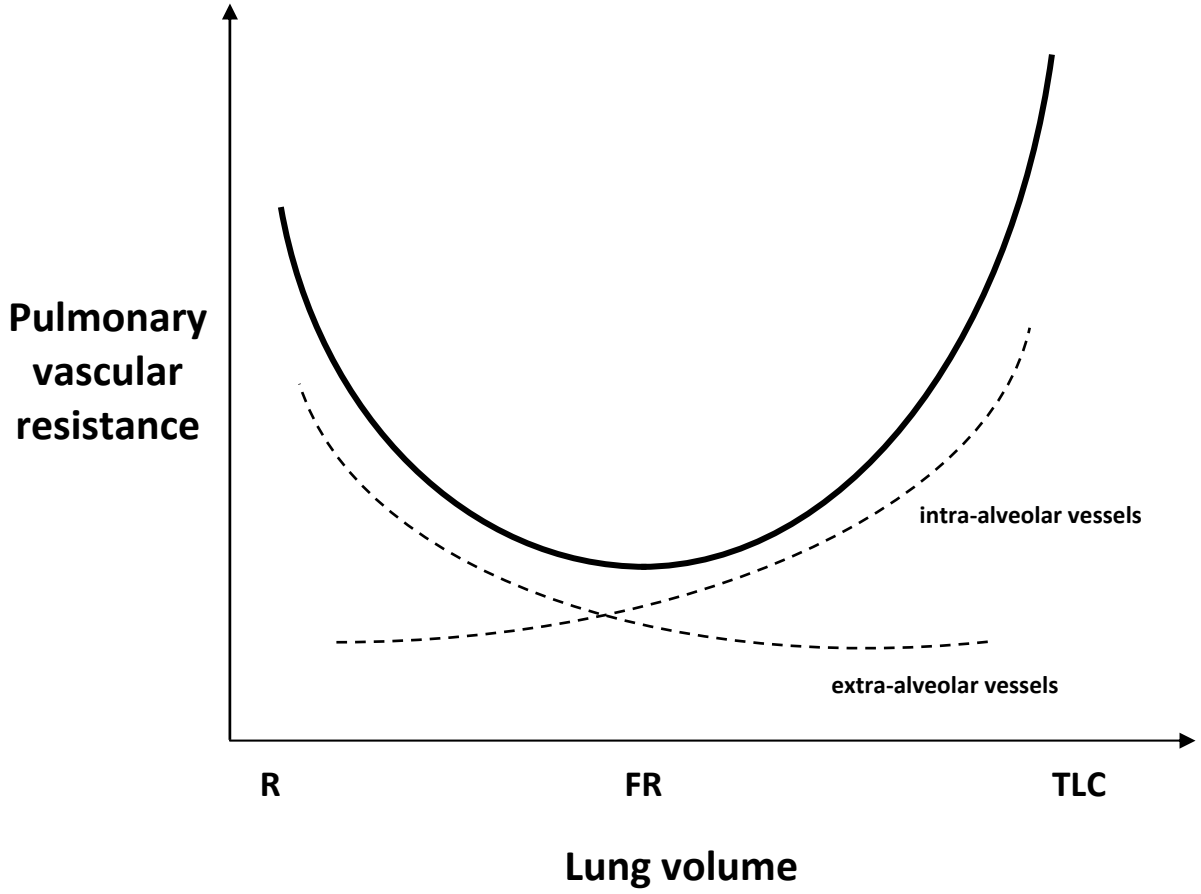
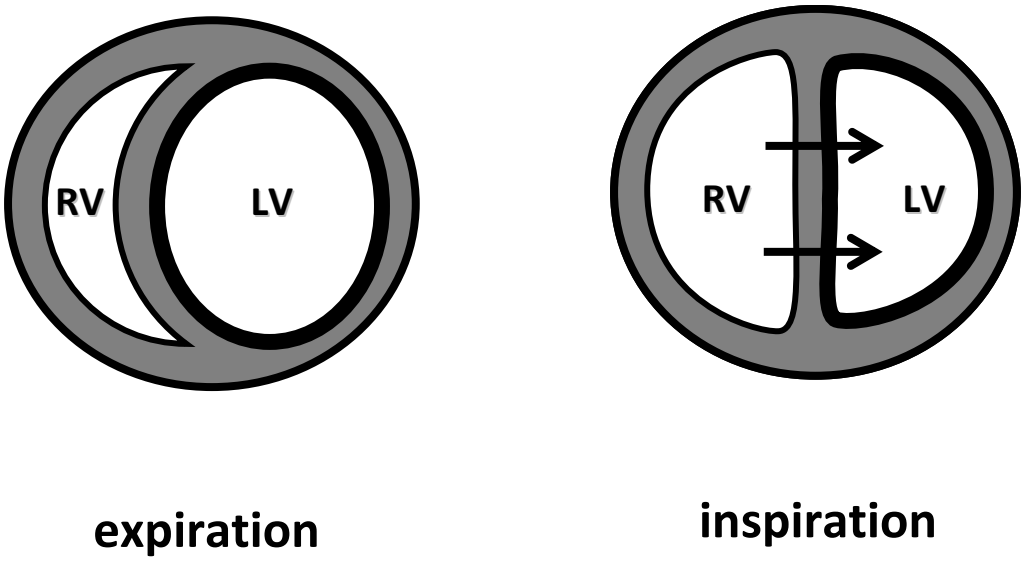
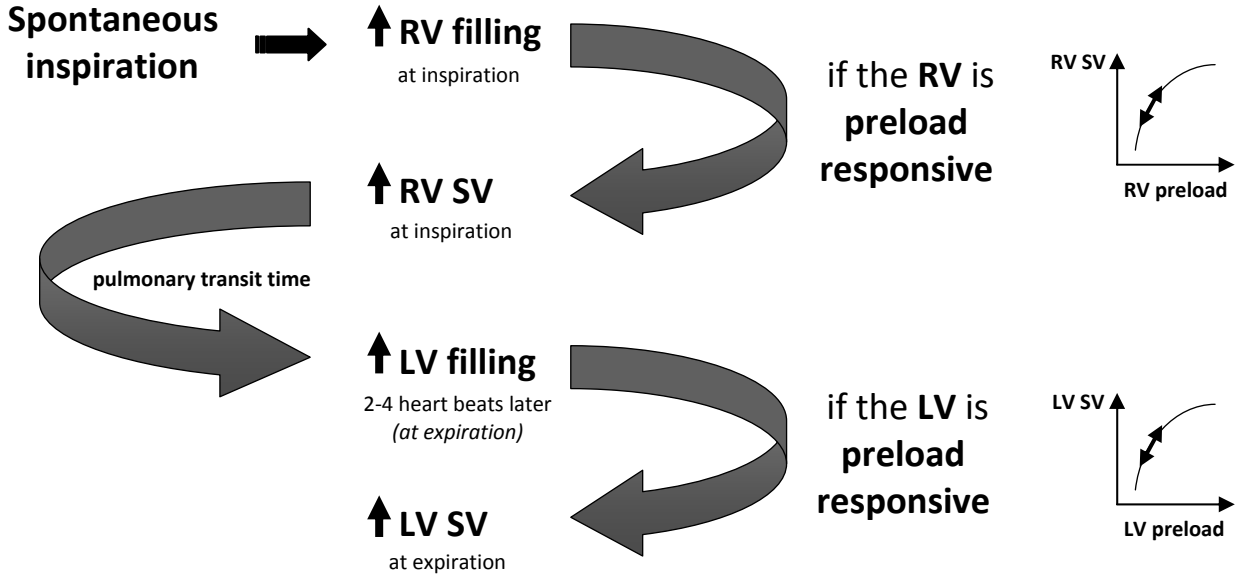

Pulmonary vascular resistance
intra-alveolar vessels
extra-alveolar vessels
R
FR
TLC
Lung volume

Figure 3



expiration **inspiration**

Figure 4


Spontaneous inspiration
RV filling
at inspiration
RV SV
at inspiration
pulmonary transit time
LV filling
2-4 heart beats later
(at expiration)
LV SV
at expiration
if the RV is preload responsive
RV SV
RV preload
if the LV is preload responsive
LV SV
LV preload