

Pulsus Paradoxus

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Under normal conditions of rest, arterial and venous blood pressures fluctuate throughout the respiratory cycle, falling with inspiration and rising with expiration. All anatomical structures within the thorax, including the heart and great vessels, are exposed to the pressure changes generated during breathing. Pulsus paradoxus (**Sidebar**) is an abnormally large inspiratory decline in systemic arterial pressure and pulse pressure. This fluctuation occurs during forced respiratory effort, heart failure, pericardial tamponade, severe asthma, hypovolemia, and mechanical ventilation.

HISTORIC PERSPECTIVE

In 1924, pulsus paradoxus was first defined as “a rhythmic pulse occurring in natural breathing, which shows a waxing and waning in size during respiration, evident on palpation in all accessible arteries.”¹ Relying on sphygmograms for diagnosis because there was no way to record blood pressure at that time, the irregular pulse was recognized to occur in patients with enhanced intrathoracic pressure variation or with cardiac tamponade. Clinical understanding of pulsus paradoxus has changed little since this original description.

PHYSIOLOGY

Under normal conditions of rest, an inspiratory fall of arterial systolic pressure less than 10 mm Hg occurs, accompanied by an inspiratory fall in venous pressure. The thorax can be schematically represented as a sealed box that contains a roller pump (ie, the heart) connected to a venous reservoir through collapsible tubings and to an arterial outflow through elastic tubings (**Figure 1**). Changes in intrathoracic pressure increase or decrease the height of the roller pump without affecting the venous reservoir or the arterial side of the thorax.

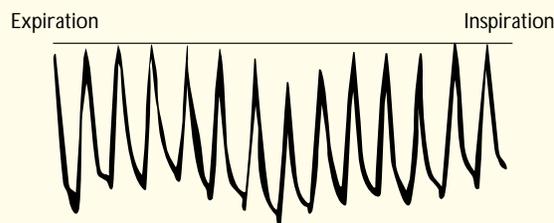
Consider the heart and lungs as a pump oxygenator located within the thoracic cage. This pump receives blood from a venous reservoir of low pressure, oxygenates the blood, and expels the blood with sufficient force to raise it to an arterial reservoir outside of the thorax. Because the walls of the heart are distensible, a reduction in intrathoracic pressure by inspiration is the equivalent of lowering the pressure within the heart and lungs relative to the systemic arterial and venous

PULSUS PARADOXUS

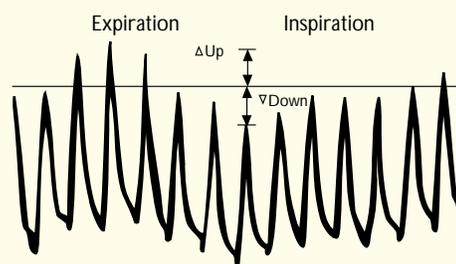
Normal pulse: Under normal conditions of rest, an inspiratory fall of less than 10 mm Hg in the arterial systolic pressure and an accompanying inspiratory fall in the venous pressure occur.

Paradoxical pulse: Pulsus paradoxus differs from a normal pulse in two respects: 1) the inspiratory diminution in arterial pressure exceeds 10 mm Hg, and 2) the inspiratory venous pressure remains steady or increases. The exaggerated waxing and waning in the pulse volume can usually be palpated and demonstrated with the sphygmomanometer or arterial catheter.

Normal arterial pressure trace:



Pulsus paradoxus:



pressures outside of the thorax. Then, an increase in the force that the left ventricle must develop in the next beat to sustain the same arterial pressure occurs; that is, the left ventricle experiences increased afterload¹ and empties incompletely, and systemic pressure falls.

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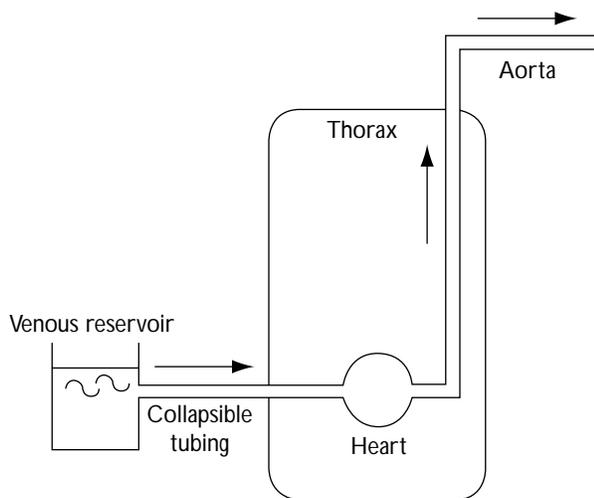


Figure 1. The heart and lungs are pump oxygenators located within the thoracic cage. The pump receives blood from a low pressure venous reservoir and expels the blood to a high pressure arterial reservoir outside of the chest.

PATHOPHYSIOLOGY

Two mechanisms seem to be involved in pulsus paradoxus.² The first is a direct consequence of the change in pleural pressure associated with breathing. Because all thoracic organs are exposed to changes in pleural pressure, organ function is unaffected except in locations where arteries leave or veins enter the thoracic cage. At these points, a change of intrathoracic pressure alters the pressure gradients along which blood leaves or enters the thorax. The second mechanism is a consequence of the intimate anatomic relationship between the two ventricular chambers (ie, distension of one influences the filling characteristics and distensibility of the other).

Reduction of pleural pressures also increases the gradient from the systemic venous reservoir to the right ventricle, contributing to accelerated venous return. However, negative pressures cannot be transmitted through the veins, which are collapsible. Thus, the increase in venous return is not proportional to the fall in pleural pressure as right atrial pressure falls from slightly negative to more markedly negative levels.³ The small increase in venous return causes a selective increase in the preload of the right ventricle without an equivalent increase of inflow to the left ventricle.⁴

ELICITATION

To elicit pulsus paradoxus, the sphygmomanometer cuff is inflated above systolic pressure. Korotkoff sounds are sought over the brachial artery while the cuff is deflated at a rate of approximately 2 to 3 mm Hg per

heartbeat. The peak systolic pressure during expiration should first be identified and reconfirmed. The cuff is then deflated slowly to establish the pressure at which Korotkoff sounds become audible during both inspiration and expiration. When the differences between these two observed levels reaches or exceeds 10 mm Hg during quiet respiration, a paradoxical pulse is present.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for pulsus paradoxus include asthma, cardiac tamponade, emphysema, hypovolemia, pericardial effusion, pericarditis, and pulmonary embolism. In both cardiac tamponade and constrictive pericarditis, inspiration is accompanied by abnormal decreases in systolic and diastolic pressures, reflecting an exaggerated decline in left ventricular volume. For example, inspiratory augmentation of systemic venous return in cardiac tamponade increases the volume of the right side of the heart at the expense of the left side.⁵ An inspiratory increase in the filling of the right side of the heart now causes encroachment on the left ventricular space through increased pericardial pressure and displacement of the septum.⁶ The compressed left ventricle thus accepts less blood from the lung, and systolic ejection is reduced and may be completely abolished in the next beat.

The most common noncardiac cause of pulsus paradoxus is pulmonary emphysema. The decrease in lung compliance (ie, stiff lungs) exaggerates the normal inspiratory fall in left ventricular volume and in systolic and diastolic pressures. When airway obstruction coexists, such as in patients with bronchospasm and asthma,⁷ the respiratory effects on the pulse are even greater because inspiration continues to cause an exaggerated fall in systolic and diastolic pressures, whereas exhalation is accompanied by an exaggerated rise in systolic pressure above normal. **HP**

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