Pulsus paradoxus, a physical sign of tremendous diagnostic and prognostic significance can be seen in a variety of cardiac and extra-cardiac conditions. This article will discuss the mechanism, pathophysiology, detection and management of patient with pulsus paradoxus.

**Historical Aspects**
The reduction in pulse volume during inspiration was first described by Lomer in 1669 in constrictive pericarditis. A similar finding was described by Floyer and later by William in 1850 in bronchial asthma. Adolf Kussmaul coined the term “pulsus paradoxus” in 1873 in three patients with constrictive pericarditis. The “paradox” was: (1) the discrepancy between the absence of the pulse and present corresponding heart beat and (2) Amidst this irregularity, the regularity with which the pulse disappeared during inspiration.

**Respiratory Influences on the Pulse Volume**
Under normal conditions, arterial blood pressure fluctuates throughout the respiratory cycle, falling with inspiration and rising with expiration. The changes in the intrathoracic pressures during breathing are transmitted to the heart and great vessels. During inspiration, the right ventricle distends due to increased venous return, the interventricular septum bulges into the left ventricle reducing its size (reversed Bernheim effect), and increased pooling on blood in the expanded lungs decreases return to the left ventricle, decreasing the stroke volume of the left ventricle. Additionally, negative intrathoracic pressure during inspiration is transmitted to the aorta. The relatively higher negative pressure in the pulmonary circulation compared to the left atrium in patients with pericardial pathology causes back flow of blood from the left atrium into the pulmonary veins during inspiration.

Therefore, during inspiration the fall in the left ventricular stroke volume is reflected as a fall in the systolic blood pressure. The converse is true for expiration. During quiet respiration, the changes in the intrathoracic pressures and blood pressure are minor. The accepted upper limit for fall in systolic blood pressure with inspiration is 10 mmHg.

**Pulsus Paradoxus: What is the Paradox?**
The “paradox” refers to the fact that heart sounds may be heard over the precordium when the radial pulse is not felt. This is due to an exaggeration of the normal mechanisms mentioned above. Moreover, the clinical method of assessment of this “pulse” is by measurement of the “systolic blood pressure”. The mechanisms in various pathologies are discussed below.

**Causes of Pulsus Paradoxus**

- **Cardiac causes**
  1. Cardiac tamponade
  2. Pericardial effusion
  3. Constrictive pericarditis
  4. Restrictive cardiomyopathy
  5. Pulmonary embolism
  6. Acute myocardial infarction
  7. Cardiogenic shock

- **Extracardiac pulmonary causes**
  1. Bronchial asthma
  2. Tension pneumothorax

- **Extracardiac non-pulmonary causes**
  1. Anaphylactic shock (during urokinase administration)
  2. Volvulus of the stomach
  3. Diaphragmatic hernia
  4. Superior vena cava obstruction
  5. Extreme obesity

**Mechanisms and Pathophysiology**
As a general guideline, pulsus paradoxus will be a result of the following mechanisms operating alone or in combination:

1. Limitation in increase in inspiratory blood flow to the right ventricle and pulmonary artery
2. Greater than normal pooling of blood in the pulmonary circulation
3. Wide excursions in the intrathoracic pressure during inspiration and expiration
4. Interference with venous return to either atrium especially during inspiration

In this review the pathophysiology underlying only the major causes will be discussed.

**Constrictive Pericarditis and Cardiac Tamponade**
Pulsus paradoxus has great diagnostic significance in pericardial disease. The following mechanisms are proposed to operate:

1. Tense fluid accumulation within the pericardial sac impairs left ventricular filling causing an exaggerated reduction in systolic blood pressure during inspiration. Katz and Gauchat found that with pericardial tamponade,
intrapericardial pressure did not fall during inspiration. As a result, the pulmonary venous pressure would fall more than the left atrial pressure during inspiration tending to cause a fall in the left ventricular filling during inspiration due to incomplete transmission of the inspiratory fall of intrathoracic pressure to the left atrium. This has been documented in other studies as well. Dock proposed that the inspiratory traction by the diaphragm and mediastinum upon the taut pericardium further increased intrapericardial pressure thus interfering with cardiac filling. Shabetai found that both vena caval and pulmonary arterial blood flow velocity fail to increase normally during inspiration in patients with constrictive pericarditis.2

Pulsus paradoxus in acute myocardial infarction can result from right ventricular infarction, cardiogenic shock or cardiac tamponade. However, it has been described in uncomplicated myocardial infarction due to the differences in the compliance of the left atrium, left ventricle and pulmonary circulation that is further exaggerated by an increase in the left ventricular stiffness due to myocardial ischaemia.16

Respiratory Diseases
Pulsus paradoxus is one of the ominous signs in acute exacerbation of bronchial asthma. This is the most common extra-cardiac cause of this physical sign. The main mechanism operartional in respiratory disease is the unusually great fluctuations in intrathoracic pressures that are transmitted to the aorta. The following theories have been proposed: 1. During increased airway resistance, there is an exaggeration in the inspiratory-expiratory difference in stroke volume mediated primarily by the effects of intrathoracic pressure on ventricular preload. Shim et al17 found that patients of asthma with pulsus paradoxus had greater air-flow obstruction than patients without. Also, it was often present in mild obstruction and absent in severe obstruction. In acute exacerbation of childhood asthma, pulsus paradoxus often correlates with both the severity and response to bronchodilators.18

2. Hyperinflation of the chest due to air trapping is also plays a role in pulsus paradoxus. Factors other than hyperinflation also contribute to the fall in systolic pressure that occurs at full inflation of the lungs. This is also observed in patients with chronic stable obstructive airway disease.19

Pulsus Paradoxus in Hypovolaemic Shock
Hypovolaemia may precipitate pulsus paradoxus in critically ill patients. Its occurrence may aid in the clinical recognition of the common syndrome of occult hypovolaemia in patients with shock and no obvious blood loss.20

Measurement of Pulsus Paradoxus

Cuff sphygmomanometry
The patient should be instructed not to breathe too deeply (enough to make the chest movements easily visible). The sphygmomanometer cuff is inflated above systolic pressure. Korotkoff sounds are sought over the brachial artery while the cuff is deflated at rate of approximately 2 to 3 mm Hg per heartbeat. The peak systolic pressure during expiration should first be identified and reconfirmed (when Korotkoff sounds are heard only during expiration). The cuff is then deflated slowly to establish the pressure at which Korotkoff sounds
become audible during both inspiration and expiration (when the Korotkoff sounds are heard during both inspiration and expiration). When the differences between these two levels exceeds 10 mm Hg during quiet respiration, a paradoxical pulse is present.

**Palpation**

Usually, palpation of the central pulses (carotid) is recommended for the evaluation of the character of the pulse. However, pulsus paradoxus is better appreciated in the peripheral pulses (radial). When the pulsus paradoxus is severe, it may be possible to palpate a fall (reduction in the pulse volume) during the phase of inspiration and rise during the expiratory phase.

**Arterial waveform analysis**

In the intensive care setting, where the arterial waveform is available, pulsus paradoxus can be diagnosed by visualising changes in the systolic blood pressure tracing during the inspiratory and expiratory phases of respiration.

**Pulse oximetry waveform analysis**

This technique has been found useful in the neonates with cardiac tamponade. In patients with obstructive airway disease since pulse oximetry is available in ICUs and emergency departments, it is a useful non-invasive means of continually assessing pulsus paradoxus and air trapping severity.

**What is Reversed Pulsus Paradoxus?**

Reversed pulsus paradoxus, a rise in systolic blood pressure during inspiration, was first described by Massumi et al. in patients with idiopathic hypertrophic subaortic stenosis, isorhythmic ventricular rhythm and patients of left ventricular failure on positive pressure ventilation. A rise in peak systolic pressure on inspiration by more than 15 mm Hg is considered significant. In a mechanically ventilated patient, positive pressure ventilation displaces the ventricle wall inward during systole to assist in ventricular emptying causing a slight rise in the systolic pressure during mechanical inspiration. A reverse pulsus paradoxus in mechanically ventilated patients is a sensitive indicator of hypovolaemia.

**What is Pseudopulsus Paradoxus?**

Salel et al. described a patient of complete heart block who was misdiagnosed to have pulsus paradoxus. This was the result of forfituous synchronism of inspiration with the cyclic intermittent properly timed atrial contribution to ventricular filling characteristic of atrioventricular dissociation in this condition. This is termed pseudopulsus paradoxus. This error can be avoided by strictly adhering to the guidelines for pulsus paradoxus laid down by Gauchat and Katz: (1) The pulse must be felt in all the accessible arteries (2) There is no need for deep inspiration and (3) There must be no irregularity of cardiac action.

**Absent Pulsus Paradoxus in Cardiac Tamponade**

All cases of cardiac tamponade are not accompanied by pulsus paradoxus. The reasons for this are not clear in all cases, but it is likely that other compensatory mechanisms are brought into play in order to maintain a normal systemic blood pressure. The following are such conditions:

1. Aortic regurgitation (AR): In the presence of AR, the left ventricle can fill from the aorta during inspiration. Therefore, if aortic dissection produces both AR and tamponade, pulsus paradoxus may be absent.

2. Large atrial septal defect: The normal increase in systemic venous return on inspiration is balanced by a decrease in the left to right shunt, resulting in minimal change in the right ventricular volume.

3. Isolated right heart tamponade: This entity has been described in patients of chronic renal failure on hemodialysis.

4. Elevated left ventricular diastolic pressures

5. Severe rheumatoid spondylitis or disease of the bony thorax: Wide changes in intrathoracic pressure prevented by the relative immobility of the chest wall.

6. Coexistent condition producing “reversed pulsus paradoxus”

**Importance of Kussmaul’s Sign in Pulsus Paradoxus**

Kussmaul’s sign is a paradoxical increase in the peripheral venous distension and pressure during inspiration. The major mechanism is a change in the shape of the pericardium with a resulting increase in the intrapericardial pressure and obstruction to the venous return to the heart. Compare this with the marked exaggeration of the normal expiratory increase in venous pressure that accompanies patients with pulmonary disease. Note that pulsus paradoxus may be present in both groups of patients.

**Approach to a Patient With Pulsus Paradoxus**

Rule out common, important and life threatening causes first.

1. Careful history of the illness
2. Haemodynamic status of the patient
3. Meticulous examination of the jugular venous pulse – do not forget Kussmaul’s sign and abdominojugular reflux
4. Look for Beck’s triad (distended jugular veins, hypotension and muffled heart sounds) – suggestive of cardiac tamponade
5. Detailed evaluation of the respiratory and cardiovascular
whereas the two tests were equally valuable in hypovolaemic intrapericardial pressure during euvolaemia and hypervolemia specific than pulsus paradoxus in detecting increases in right ventricular diastolic collapse was more sensitive and more on echocardiography and pulsus paradoxus were compared, studies have shown that when right ventricular diastolic collapse has its limitations. The use of the term is not uniform and as it

tially treatable causes are likely to be missed by the examining physician.

Limitations of Pulsus Paradoxus

Although pulsus paradoxus is a valuable physical sign, it has its limitations. The use of the term is not uniform and as it is an exaggeration of a normal phenomenon, a cut-off value is difficult to provide. In patients of cardiac tamponade, studies have shown that when right ventricular diastolic collapse on echocardiography and pulsus paradoxus were compared, right ventricular diastolic collapse was more sensitive and more specific than pulsus paradoxus in detecting increases in intraopericardial pressure during euvoalaemia and hypervolaemia whereas the two tests were equally valuable in hypovolaemic states. As with other clinical signs, pulsus paradoxus must not be considered in isolation but in conjunction with the patient’s clinical state and with other indices of the severity of asthma. Finally, the absence of pulsus paradoxus does not rule out the presence of a significant periardial effusion. However, this important bedside sign must be elicited in indicated patients, foregoing which life threatening and potentially treatable causes are likely to be missed by the examining physician.

Khasnis A, Lokhandwala Y*  
Departments of Medicine and Cardiology*, Seth G. S. Medical College and K. E. M. Hospital, Parel, Mumbai - 400 012, India.

References