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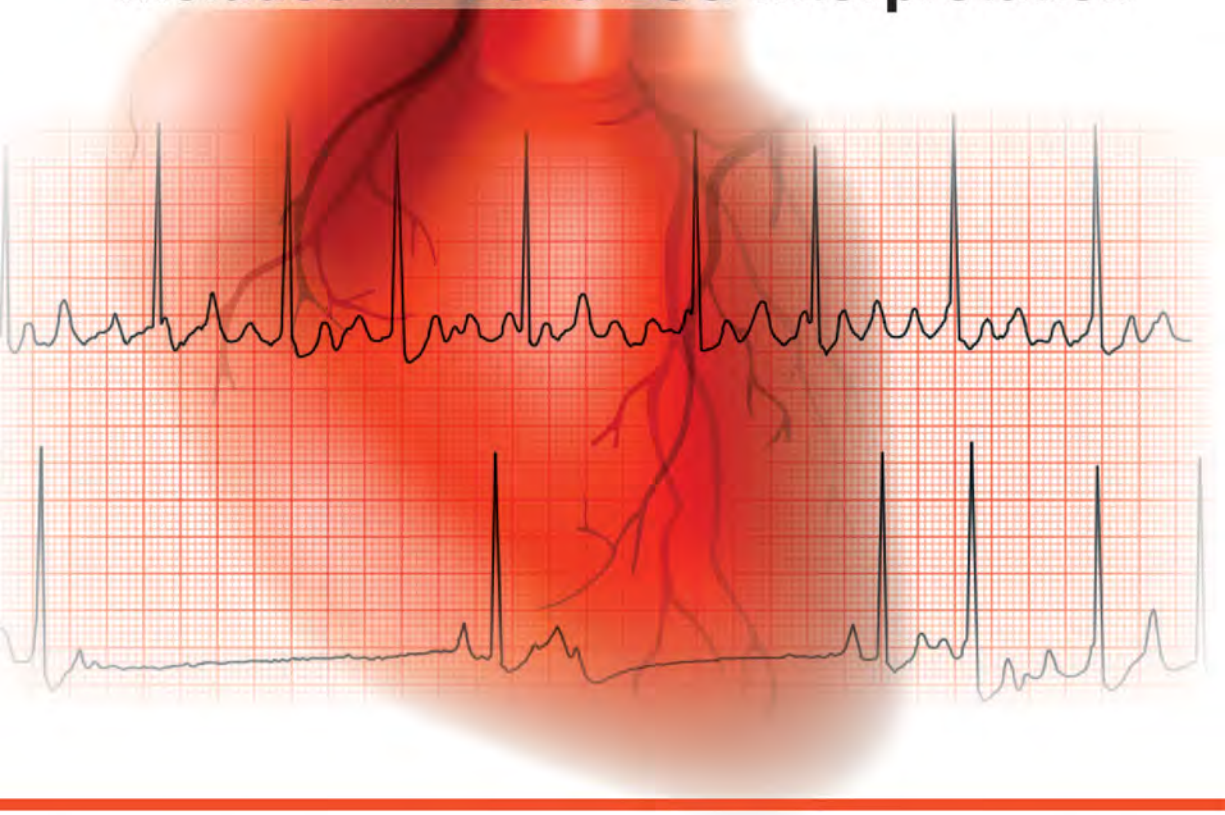
The Art and Science of Cardiac Physical Examination

With Heart Sounds, Jugular and Precordial Pulsations on CD

Includes 12-Lead ECG Interpretation



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Foreword
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Chapter

3 Blood Pressure and its Measurement

Snapshot

- Physiology of Blood Flow and Blood Pressure
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- Points to Remember when Making the BP Measurement
- Factors which Affect Blood Pressure Readings
- Interpretation of Blood Pressure Measurements
- Use of BP Measurement in Special Clinical Situations

PHYSIOLOGY OF BLOOD FLOW AND BLOOD PRESSURE

The purpose of the arterial system is to provide oxygenated blood to the tissues by converting the intermittent cardiac output into a continuous capillary flow, and this is achieved by the structural organization of the arterial system.

The blood flow in a vessel is basically determined by two factors:

1. *The pressure difference between the two ends of the vessel that provides the driving force for the flow*
2. *The impediment to flow, which is essentially the vascular resistance*

This can be expressed by the following formula:

$$Q = \Delta P/R,$$

where Q is the flow, ΔP is the pressure difference and R is the resistance.

The pressure head in the aorta and the large arteries is provided by the pumping action of the left ventricle ejecting blood with each systole. The arterial pressure peaks in systole and tends to fall during diastole.

Briefly the peak systolic pressure achieved is determined by (*see* Chapter 2 on Arterial Pulse):

1. The momentum of ejection (the stroke volume, the velocity of ejection that in turn are related to the contractility of the ventricle and the afterload)
2. The distensibility of the proximal arterial system
3. The timing and amplitude of the reflected pressure wave

When the arterial system is stiff as in the elderly, for the same amount of stroke output, the peak systolic pressure achieved will be higher. The poor distensibility causes a greater peak pressure (Laplace relationship).

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In addition, a stiff arterial system results in faster transmission and reflection of the pressure wave, thereby adding to the peak pressure. The narrow and peaked pressure seen in the more peripheral muscular arteries is the effect of such reflection. The level to which the arterial pressure will fall during diastole is primarily dependent on the state of the peripheral resistance, which controls the runoff. Conditions with low peripheral resistance and vasodilatation will cause the diastolic pressure to fall to low levels.

The ***mean arterial pressure*** is the average of all the pressures obtained over an entire duration of a cardiac cycle. Since diastole is longer than systole, the mean pressure is estimated as the sum of 60% of the diastolic pressure and 40% of the systolic pressure. More accurate measurement will be by integrating the area under a recorded pressure curve. The ***pulse pressure,*** which is the difference between the systolic and the diastolic pressure, reflects not only the stroke volume but also the state of the peripheral resistance. Conditions associated with a large stroke volume and low peripheral resistance will be expected to give rise to a large pulse pressure and this will be reflected in the amplitude of the arterial pulse by palpation.

While the control of the cardiac output is usually determined by local tissue flow under physiologic states, the control of the arterial pressure is independent of these and is regulated through a complex system that involves nervous reflexes and neurohumoral mechanisms for short-term needs (such as "flight", "fright" and "fight" type reactions or in situations like those following acute loss of blood volume) and neuroendocrine, renin–angiotensin–aldosterone system and renal mechanisms for long-term adaptation. These control systems in the normal as well as their alterations in hypertension and in heart failure are well discussed in standard texts in physiology and medicine. In this chapter, our focus will be mainly in relation to the measurement of blood pressure (BP) by the sphygmomanometer and its use in special clinical situations.

PHYSIOLOGY OF BP MEASUREMENT

The indirect measurement of BP by the sphygmomanometer involves the application of a controlled lateral pressure by an inflatable cuff to occlude the artery by compression, thereby stopping the flow. The detection of the resumption of flow during slow deflation allows the determination of the pressure. The cuff is normally applied to the arm over the brachial artery. When the cuff pressure exceeds the systolic pressure, the brachial artery is fully occluded and the flow ceases. When the cuff is deflated to pressures just below the systolic peak, the flow begins to resume with each cardiac systole. The jet of blood coming through the partially occluded vessel is associated with tapping type sounds, which can be recognized using the stethoscope placed over the brachial artery just distal to the cuff. These sounds

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termed the Korotkoff sounds help in identifying the systolic and the diastolic pressures in the artery. As the cuff is being deflated, the pressure when the Korotkoff sounds first appear is noted. This corresponds to the systolic pressure. The disappearance of the Korotkoff sounds with further deflation will correspond to the diastolic pressure. Korotkoff sounds generally become muffled first when the cuff is being deflated before they totally disappear. It is always advisable to take the diastolic pressure to the level at which the sounds disappear, since it is less likely to introduce errors.[1]

The Mechanism of Origin of the Korotkoff Sounds

The exact mechanism of origin of Korotkoff sounds is not completely established. They have been thought to be due to turbulence of flow coming through the partially occluded artery. This is thought to be supported by the following.

They become muffled ***(phase IV)*** and eventually disappear ***(phase V)*** generally when the flow resumes in diastole, i.e. when the cuff is deflated below the diastolic pressure allowing the artery to be open throughout the cardiac cycle. In addition, before they become muffled and completely disappear, they may sound like a short bruit (initially as soft bruit in ***phase II*** and louder bruit as in ***phase III).***

However, they do not always sound like murmurs and often sound as sharp sounds. The possibility of Water hammer theory had been previously suggested (not to be confused with the water hammer pulse in aortic regurgitation) to explain the presence of distinct sounds.[2] The sounds are thought to be produced by the deceleration of high velocity jet of flow coming through the vessel, as it opens up from an occluded state, against the stationary column of blood distal to the occlusion. The intensity of the sounds, however, may vary being loud and persistent to low diastolic pressures or throughout diastole in certain situations as in aortic regurgitation in children and in pregnancy. In aortic regurgitation, the stroke volume tends to be large with increased velocity of ejection. Children generally have hyperkinetic circulation, and pregnant women tend to have a high cardiac output with increased sympathetic tone. On the other hand, the Korotkoff sounds tend to be poor in low output states.

When an oversized cuff is used it may lead to underestimation of the systolic BP. It has been shown that the state of the distal vasculature in the limb where the BP is being measured affects the intensity of the Korotkoff sounds. Vasodilatation makes the sounds louder, and vasoconstriction softer.[3,4] When the peripheral resistance in the arm distal to the cuff was changed by interventions such as heating, cooling and by induction of reactive hyperemia, the amplitude of the Korotkoff sounds appeared to change. Thus, these effects may lead to either over or under estimation of both systolic and diastolic pressures.[5] In fact, when the Korotkoff sounds are poorly heard, they are best

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augmented by raising the arm (decreasing venous distension) and having the patient open and close their fist on the side where the pressure is being measured a few times.[6,7] This is thought to increase the forearm flow.

Others have proposed that the pressure pulse wave itself may be the source of the Korotkoff sounds. The sounds may be attributed to "shock waves" where the flow velocity of blood in the narrowed segment may exceed the pulse wave propagation velocity and give rise to vibrations in the audible frequency range. This would be analogous to the sonic boom heard when the speed of a jet plane reaches and surpasses the speed of sound.

It is also possible that the Korotkoff sounds are related to energy (vibrations) that results from sudden termination of the pressure pulse wave at the site of the inflated cuff, which leads to partial occlusion of the vessel, causing this to become a terminating site favoring reflection. Between the systolic peak and the diastolic pressures during which time the Korotkoff sounds are present there is a considerable degree of termination and reflection together with beginning onward transmission of the pressure pulse wave. Once the cuff pressure is lowered below the diastolic pressure there is no more termination or reflection at the site of the cuff application and the pressure pulse is further transmitted along the artery. Therefore, there is no sound to be heard. This may be supported by the fact that when an oversized cuff is used it may lead to underestimation of the systolic BP. In conditions where the Korotkoff sounds are loud and last longer to low levels of diastolic pressure such as aortic regurgitation, there is a more rapid and higher launching momentum to the pressure pulse wave because of the large stroke volume which is ejected with a rapid velocity. Thus, there may be more energy for dissipation at the termination sites. In significant aortic regurgitation, one often feels "pistol shots" over the femoral arteries, which are also often sites of reflection because of bifurcation. On the other hand, this mechanism does not explain why the Korotkoff sounds are heard distal to the cuff and not proximal to it.

Tavel et al. had previously shown using invasive pressure measurements that the Korotkoff sounds correspond to the steepened portion of the anacrotic limb of the pressure pulse.[8] More recently, Drzewiecki et al. have proposed an alternate origin for the production of the Korotkoff sounds. They relate it to the distortion of the pressure pulse under the cuff in the narrowed segment, and a change in both pressure and flow distal to the cuff resulting in a nonlinear pattern of pressure-flow relationship. This results in a steeper pulse slope distal to the cuff with higher frequency harmonic content of this steeper pulse slope reaching the audible frequency range. This interesting theory was proposed based on a mathematical model representing the structures involved, which was able to predict the range of features of the Korotkoff sounds previously reported.[9] However, it does not satisfactorily explain as to why in some people the Korotkoff sounds persist even when the cuff is fully deflated.

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It is clear from the short description above that the origin of the Korotkoff sounds is far from being completely established.

POINTS TO REMEMBER WHEN MAKING THE BP MEASUREMENT

The following points are worth noting when taking BP measurements:[10–12]

1. First, determine roughly the systolic pressure by palpation of the radial artery pulse, as the cuff is gradually being deflated so that ***an auscultatory gap*** *(during which Korotkoff sounds may disappear altogether and reappear again when the cuff is being deflated further) if present will not be missed. This sometime occurs in some hypertensive patients.[13] The auscultatory gap may also be due to venous congestion and decreased velocity of blood flow in the extremity where the BP is being measured.*[6] The systolic pressure estimated by the palpation of the distal radial artery (while compressing the proximal brachial artery) is usually 10 mmHg lower than the systolic pressure as assessed by the auscultatory method. Some studies have shown that assessment of central aortic pressure by measurement of the brachial BP by cuff method underestimates the systolic pressure by 5–20 mm Hg and overestimates the diastolic pressure by 12–20 mm Hg. This has been related to interobserver variability. Specially, for the diastolic pressure some may take phase IV and others phase V as the true pressure. This discrepancy is even more exaggerated during vasoconstriction.[14–16]
2. Keep the position of the antecubital fossa at the level of the heart to avoid the hydrostatic effect of gravity on the column of blood in the arm.
3. Deflate the cuff slowly. The recommended deflation rate is usually 3 mm Hg/s. This rate should actually vary according to the patient's heart rate (relatively slow deflation rate during bradycardias and relatively faster deflation rate during tachycardias).
4. Avoid venous congestion that tends to muffle Korotkoff sounds, by raising the arm and by inflating the cuff faster.[6]
5. When the BP is first determined in a new patient it should be measured in both arms. If a difference is noted then the higher reading of the two must be considered as the patient's BP. Slight variation between the two arms is common. The normal difference should not exceed 15 mm Hg. The difference may be due to the fact that the innominate on the right side and the subclavian on the left side may arise from the aorta at different angles. The relative suction due to a Venturi effect of the flow through the aortic arch between the two arms may be different for this reason. When the difference is abnormally high then it will be indicative of obstruction on the side with the lower reading. Conditions such as coarctation of aorta (one proximal to the origin of the left subclavian), atherosclerotic or embolic obstruction (rare in the upper extremities), and aortic dissection

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are some of the conditions that may have to be considered depending on the clinical situation. In coarctation of aorta proximal to the left subclavian, the right arm pressure will be naturally higher than the left. In supravalvular aortic stenosis, the direction of the jet of flow tends to be directly directed into the innominate artery. This often results in the direct impact pressure of the central jet being transmitted to the right arm, thus making the right arm pressure higher than the left due to a Coanda effect.[17]

6. When Korotkoff sounds persist, take the point where they become muffled as the level of the diastolic pressure.
7. When Korotkoff sounds are poor, have the patient exercise their hand as pointed out earlier, thereby augmenting the intensity of the Korotkoff sounds.[7]
8. Always have a cuff of proper size for the arm especially for very obese individuals. The width of the bladder in the cuff should be roughly 40% of the arm circumference. In most patients (normal size), a 5-inch wide cuff would be the appropriate choice. Too wide a cuff may lead to erroneous recognition of the onset of the Korotkoff sounds for the site of auscultation may be too distal to the site of actual occlusion of the artery. This may make the Korotkoff sounds to be too soft in intensity to be heard and may result in underestimation of the pressure. A narrow cuff will allow overdistension of the bladder of the cuff before actual occlusion of the brachial artery can occur. Some of the pressures applied may be spent in distending the bladder rather than compressing the artery. This will lead to an overestimation of the systolic and even the diastolic pressures. Larger size cuff (8 inches wide) is needed in obese patients where the arm circumference is >35 cm.[18,19] The bladder of the cuff should cover at least 50% of the circumference of the limb where the BP is being measured.
9. Support the arm so that patient's arm muscles are relaxed. If not, isometric contraction may occur that will raise the peripheral resistance, thereby raising the diastolic pressure. This would also adversely influence the BP reading by muffling the Korotkoff sounds, thereby raising the diastolic pressure and most likely lowering the systolic pressure.
10. Avoid aneroid manometers, which can have problems in calibration. If an aneroid manometer is used it is recommended that it be calibrated, against a mercury manometer, yearly in fact probably more frequently.[20]
11. Local changes in the limb are known to affect BP readings. If the patient has washed his/her hand with cold or hot water just before getting the BP measured, the reading obtained may be higher or lower, respectively, and may not correctly reflect the central aortic pressure. When BP is taken more than once, they should be done at least 5 minutes apart to avoid reactive hyperemic vasodilatation during the second measurement. This lowers the recorded BP locally leading to false estimation of central aortic pressure.[5]

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FACTORS WHICH AFFECT BLOOD PRESSURE READINGS

1. Anxiety, stress and/or pain. Sometimes the mere fact of having the BP taken by a physician can excite a nervous and anxious patient to cause significant rise in the BP of the patient. (The so-called white coat syndrome.)[21,22]
2. Exercise such as climbing a couple of flights of stairs, and even mild activity involving bending or stooping in the elderly could raise the pressures. It should be noted that the transfer of energy of pulse pressure wave to the periphery is greater at low frequencies (<2.5 Hz) in patients at rest because of slower heart rates and longer ejection time. With exercise and the associated faster heart rates and shorter ejection time, the energy transfer occurs at higher frequencies (>2.5 Hz). Since higher frequencies are amplified more, the systolic brachial pressure will be measured to be elevated and may not necessarily reflect the true central aortic systolic pressure.[23–25]
3. Exposure to cold by causing sympathetic vasoconstriction will raise the pressures.
4. Postprandial state will tend to lower the pressure. In addition to increased vagal tone necessary for increased peristalsis, the lower pressure is likely related to vasodilatation in the mesenteric vessels diverting more blood to the gut for the purpose of proper digestion. This probably also relates to the increase in angina or postprandial decrease in exercise tolerance in patients with exertional angina. The increased demand of blood supply to the gut, increasing the work load of the heart coupled with the decreased diastolic pressure (coronary perfusion pressure), leads to mismatch between the oxygen demand and the coronary blood supply resulting in angina.
5. Alcohol acutely will lower the pressure while chronic alcohol consumption raises the pressures.
6. Both chronic and acute cigarette smoking tend to cause elevated pressures.
7. Illicit drugs such as cocaine use may cause significant elevations in BP.
8. Excessive use of liquorice can lead to sustained hypertension.
9. Distension of viscus organ such as the urinary bladder, gall bladder and bowels can cause severe increase in pressures.
10. Significant drop in BP may be noted on postural change from a supine to erect position ***(postural hypotension)*** in patients with hypovolemia, following use of certain antihypertensive drugs and in patients with diabetes with autonomic dysfunction. This is more common in type I diabetics.
11. Since pregnant uterus can compress the inferior vena cava in the supine position and cause decreased venous return, thereby lowering the BP, it is always advisable to measure the BP in a pregnant woman in the sitting position.

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INTERPRETATION OF BLOOD PRESSURE MEASUREMENTS

The following important points need to be kept in mind in the interpretation of BP recordings:

1. Diagnosis of hypertension requires demonstration of sustained elevations of BPs under normal resting conditions. *The normal upper level of BP for adults regardless of age is 140/80. In fact, some newer studies suggest that mortality and cardiac events are less frequent in those with BPs <125/75. This, in fact, may become the normal for the future. In fact, in diabetics present recommendations suggest the BP to be controlled to 130/80, and if renal disease or microalbuminuria is also present the pressure should be 120/70 or lower. It appears that lower is better as long as the patient does not suffer any symptoms or adverse effects of hypotension and remains asymptomatic. Documentation of sustained elevations of BPs, therefore, requires more than one observation. Sometimes self-recorded pressures at home and/or recordings using an ambulatory monitoring system may have to be resorted to particularly in nervous and anxious individuals. In addition, one may also have to look for evidence of end-organ damage such as the presence of retinal changes, electrocardiographic and/or echocardiographic evidence of left ventricular hypertrophy*.
2. *While one cannot make a diagnosis of hypertension in a patient with one isolated elevation in BP, significance of such elevations nevertheless should be interpreted in relation to the clinical problem. For instance, an elderly patient who has exertional angina or dyspnea, with a resting BP of 130/80 mm Hg may have been noted to have a BP reading of 180/90 soon after undressing himself, untying his shoe laces and getting on the examining couch. Although one may not label this patient as hypertensive on the basis of that one recording of high pressures, it nevertheless points to the fact that the elevations in BP in this patient was not only inappropriate to the level of exercise and most likely are the contributing factor to the exertional symptoms. The lowering of the pressure with the use of medications such as angiotensin converting enzyme inhibitor and/or a β-blocker will be the right management strategy for such a patient.*
3. In the young patients due to peripheral amplification secondary to reflected waves in the more muscular stiffer vessels in the extremities, the systolic BP obtained may not correctly reflect the systolic central aortic pressure and may in fact be 50% higher. In these patients, the diastolic pressure may more accurately reflect the diastolic pressure in the central aorta.

   In the elderly because of arteriosclerosis and stiffened aorta and arteries in general, the pressure pulse wave travels faster. The wave reflection, therefore, tends to arrive early in the central vessels, augmenting the systolic pressure. In these patients, no peripheral amplification is noted and the brachial systolic pressure more accurately reflects the central aortic systolic pressure.[26–30]

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In atherosclerosis, there may be significant differences between the upper and the lower limb pressures. Because atherosclerosis tends to involve the lower extremities more, one may actually find in these patients decreased pulse amplitude together with lower BP in the lower limbs compared to the arms. This is in contrast to the usual findings in the young and/or normal patients, where the pressures in the legs are in fact 10–20 mm Hg higher than the brachial pressures.

4. In some patients, without any evidence of hypertensive end-organ damage, significantly elevated systolic BPs may be obtained in doctors' offices. These high office BP readings at times are associated with normal BP readings throughout the day when 24 hours ambulatory BP monitoring is carried out. Not so surprisingly the first and the last readings (normally readings taken in the laboratory in the presence of the technician) during these recordings also show significant BP elevations. This is known as "white coat syndrome" and is likely related to anxiety reaction on the part of the patient. Generally, this condition is felt to be relatively benign, although there always is a concern that they may become hypertensive in the future.

White coat hypertension has been defined and classified[31,32] into three groups:

1. ***White coat hypertension:*** Abnormal office systolic BP >150 mm Hg and daytime average systolic BP <140 mm Hg. (Patients are not on antihypertensive therapy).
2. ***White coat syndrome—normotensive:*** Patients' BPs controlled on antihypertensives. Their daytime average systolic BP < 140 mm Hg and office BP reading of > 150 mm Hg.
3. ***White coat syndrome—hypertensive:*** Patients may be on or off antihypertensive medications with daytime average systolic BP of > 140 mm Hg and office systolic BP measurement of > 150 mm Hg which is at least 15 mm Hg higher than the average daytime systolic BP.

"White coat hypertension/syndrome" may not be as benign as it once was thought to be.[33] There appears to be higher incidence of increased mean albumin levels in the urine of some of these patients. Some show increased albumin/creatinine ratios > 30%.

USE OF BP MEASUREMENT IN SPECIAL CLINICAL SITUATIONS

Determination of Pulsus Paradoxus

Effects of Respiration on the BP in the Normal Subjects

The effects of respiration on the level of BP in the normal must be understood in relation to the changes in the respiratory variations of the intrathoracic pressures as well as the venous return. On inspiration, there is generally a

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lower systolic pressure compared to the end of expiration. The intrathoracic pressure falls on inspiration, which helps to augment venous return to the heart. The inspiratory expansion of the lungs by increasing its pulmonary venous capacity accommodates for the extravolume of blood returned to the right side on inspiration and the consequent increase in right ventricular output. The increased venous return on inspiration is, therefore, not immediately available to the left heart. In fact, the return to the left heart may slightly decrease on inspiration, the expanded lungs holding the extravolume for at least a few cardiac cycles. By that time the expiratory phase usually occurs. The intrathoracic pressure on expiration becomes relatively more positive. The aorta being an intrathoracic structure, these changes in the intrathoracic pressures will also affect the aortic pressure. There is generally a fall in the systolic BP with normal inspiration and the magnitude of this fall is about 5–10 mm. This is due to both the fall in the intrathoracic pressure and the effect of the expanded lungs holding the extra venous return, thereby diminishing the return to the left heart and therefore its output. The opposite occurs on expiration, the intrathoracic pressure rises and the lungs contract in volume by exhaling air. This aids in increased pulmonary venous inflow to the left side increasing the stroke output. The net effect leads to an increased arterial pressure on expiration. In the normal, the expansion of the right ventricle on inspiration does not usually result in shift of the interventricular septum to the left, since the normal pericardium does not limit physiologic changes in ventricular volumes.[34]

In the normal, the effect of this ***inspiratory fall*** in the systolic BP is not detectable by palpation of the arterial pulse. However, the magnitude of the inspiratory fall in the BP can be easily assessed by the BP cuff at the bedside. *When the cuff is being slowly deflated to detect the onset of the Korotkoff sounds, careful observation will reveal that initially the Korotkoff sounds are audible only at the end of expiration. With each inspiration they will be seen to become inaudible. The level of the BP at the end of expiration when the Korotkoff sounds begin to be heard must first be noted. With further cuff deflation however, it will be observed that the Korotkoff sounds are audible throughout both inspiration and expiration. The level at which this begins to happen must be noted next. The difference between the two systolic levels namely the number of mm Hg to which the cuff was needed to be deflated (when Korotkoff sounds no longer remain inaudible on inspiration) gives the magnitude of the inspiratory fall in the BP.*

Pulsus Paradoxus in Cardiac Tamponade

In cardiac tamponade, there is an exaggeration of the normal inspiratory fall in the systolic BP leading to a truly definable ***"pulsus paradoxus".*** The fall in the stroke volume of the heart and consequently the systolic BP despite the

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increased venous return to the heart caused by inspiration is the paradox that has led to this term. In fact in significant cardiac tamponade, the palpation of the arterial pulse may reveal that its amplitude is less or the pulse may not even be felt on inspiration. This, of course, is not the case in the normal. The mechanism of this exaggerated fall in the BP on inspiration is attributable to the compressive effect of the fluid in the pericardial space, which is under a high pressure. In cardiac tamponade, all the four chambers of the heart are as if they were boxed in this tight pericardial space. In the absence of pre-existing cardiac disease, the pressures in the right and the left atria, the intrapericardial pressures as well as the right and left ventricular diastolic pressures are all elevated to the same level. In extreme cases of tamponade, the thinner walled structures like the right ventricle get compressed more completely than the thicker walled left ventricle. When inspiration increases the venous return to the right heart as in the normal, the expansion of the right ventricle within this enclosed tight pericardial space will of necessity push the interventricular septum to the left side, thereby further diminishing the left heart filling and its compliance. This will lead to decreased left ventricular output. The septal bulge on inspiration to the left side can in fact be demonstrated in echocardiograms of patients with tamponade.

Since the pericardium is attached to the diaphragm, the descent of the diaphragm with inspiration may also pull on the pericardium, thereby altering its global shape to a more spindle shape. This physically can lead to further rise in the intrapericardial pressure. The effects of the inspiratory fall in the intrathoracic pressures as well as the effects of the inspiratory expansion of the lungs and pulmonary venous pooling leading to diminished left heart filling that were mentioned above in the normal are also still operative in patients with cardiac tamponade. The net effect of these changes on the left heart filling as well as the intrathoracic pressures will cause a greater fall in the left ventricular stroke output and the BP on inspiration. The opposite changes occur on expiration leading to a higher arterial pressure on expiration. The effect of the inspiratory pulmonary venous pooling is particularly more dramatic when the left ventricular stroke output is already diminished due to the tamponade.

The BP cuff is used in the same manner as mentioned above to determine the magnitude of fall in mmHg due to the pulsus paradoxus. The pulsus paradoxus by BP measurement must exceed 15 mm Hg to be considered significant.

In the presence of significant elevations of the left ventricular diastolic pressures due to pre-existing cardiac disease, cardiac tamponade does not cause pulsus paradoxus. The raised diastolic pressures offer greater resistance to the compressive effects of the intrapericardial pressures. Cardiac tamponade also does not lead to pulsus paradoxus in two other conditions, namely aortic regurgitation and atrial septal defect. In the former, the extrasource of left

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ventricular filling due to the aortic regurgitation keeps the left ventricular volumes from falling, thereby preventing the respiratory fluctuations in left heart filling. Similarly in atrial septal defect, the left-to-right shunt accommodates for respiratory changes in venous return, thereby preventing a fall in left heart filling during inspiration.[35–43]

Conditions Other than Cardiac Tamponade with Pulsus Paradoxus

Constrictive pericarditis: In constrictive pericarditis, pulsus paradoxus is much less common than in cardiac tamponade. It is more common in the effusive subacute type constriction than in the chronic cases. Pulsus paradoxus is absent in the chronic cases due to poor transmission of the intrathoracic pressures to the cardiac chambers due to the thickened and fibrosed and sometimes calcified pericardium. When pulsus paradoxus is seen, it is probably due to a combination of two factors, namely the septal shift to the left and the increased pulmonary venous pooling on inspiration. The diaphragmatic pull on the pericardium on inspiration is also unlikely to be operative on a thickened and scarred pericardium.[38]

Bronchial asthma and chronic obstructive pulmonary disease: The exaggerated swings in the intrathoracic pressures may be directly transmitted to the aorta, affecting the systolic and the diastolic pressures with very little change in the stroke volume or the pulse pressure. In the presence of significant airways obstruction, there is marked increase in the intrathoracic pressures during expiration caused by the intercostal muscles to overcome the airways obstruction. This elevated intrathoracic pressure is directly transmitted to the aorta raising the expiratory pressure in the aorta. Thus, there is an ***expiratory gain*** in BP rather than the usual inspiratory fall in the normal. However, the net effect on the BP is the same.

Hypovolemic shock and acute pulmonary embolism: In some patients with hypovolemic shock, pulsus paradoxus may be noted.[44] This is due to an exaggerated effect of the inspiratory pulmonary venous pooling on the already diminished stroke volume. The change in the percentage of the stroke volume is higher compared to the normal individuals with normal cardiac output.

Similarly, the reduced capacity of the pulmonary arterial bed due to embolic obstruction in the presence of the normal pulmonary venous bed may also lead to an accentuated effect of the inspiratory pulmonary venous pooling on the left ventricular stroke volume.[45]

Blood Pressure Response to the Valsalva Maneuver

The ***Valsalva maneuver*** is basically a forced expiration against a closed glottis. This maneuver is used in our daily life when we try to bear down or

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ISBN 978-93-5152-777-0