Cardiac Diagnosis from Examination of Arteries and Veins

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Inspection of the Neck

Distinction between Arterial and Venous Pulsation

The examiner may acquire much valuable information pertaining to cardiac diagnosis by careful inspection of the major vessels in the neck with proper tangential lighting. It is important that the quick-rising carotid arterial pulsations be not confused with the more gradually rising internal jugular venous pulsations, which also originate beneath the sternocleidomastoid muscle. Distinction between the two may be made in the following manner. The carotid arterial pulse is single; ordinarily two or three venous pulse waves can be seen with each cardiac cycle. Internal jugular venous pulsations are readily obliterated by light pressure over the sternocleidomastoid muscle just above the medial end of the clavicle, whereas carotid arterial pulsations are unaffected. Venous pulsations can be altered by changing the position of the patient. The veins usually become more distended as the patient lies in a horizontal position and less distended as he sits upright. If the venous pressure is low, or normal, the venous pulsations are more evident when the patient is lying flat. In general, with higher venous pressures, the venous pulse is best seen with the patient more upright. If the venous pressure is elevated because of congestive heart failure, sustained abdominal compression will cause the venous pressure to rise and the venous pulsations to ascend higher in the neck.

Neck Vein Distention

The degree of distention and the quality of the pulsations in the external and internal jugular veins should be examined in detail. The external jugular veins, which are often distended abnormally in patients with congestive heart failure, may at times be invisible because of increased venous tone. As a result failure to appreciate the degree of distention and the level of pulsation in the internal jugular veins beneath the sternocleidomastoid muscle may lead to the erroneous conclusion that the venous pressure is normal. Distention and pulsation in both external and internal jugular veins are normal when the patient is in the recumbent position and the veins are below the level of the manubrium sterni. When the head and chest are elevated 45° from the horizontal, distention of these veins and their pulsations should extend not more than 1 or 2 cm. above the manubrium sterni. If they ascend higher, the venous pressure is elevated. In some patients with congestive heart failure in whom the venous pressure elevation is borderline or questionable, valuable information may be obtained by sustained compression of the abdomen. The compression may be made in the right upper quadrant; however, if the patient has a congested, tender liver, abdominal pressure should be exerted elsewhere. At this time it is essential that the patient not hold his breath and thereby perform a Valsalva maneuver, which will distend the neck veins in the absence of congestive heart failure. If one observes that abdominal pressure exerted during normal breathing causes a rise of the vertical level of pressure or pulsation in the neck veins, failure of the right heart is strongly suggested. This sign is called the hepatojugular reflux. Unilateral or bilateral distention of the neck veins without pulsations in the sit-
ting patient may reflect obstruction of the superior vena cava or the innominate vein rather than heart failure. Collateral veins over the upper chest, absence of heart disease, and lack of hepatic engorgement are confirmatory evidence of superior caval obstruction.

**Neck Vein Pulses**

It is important that the examiner analyze the pulsations in the neck veins. Ordinarily three positive pulses can be seen for each cardiac cycle (fig. 1). These are the a, c, and v waves. The a wave is related to atrial contraction. There is a negative wave or trough following the a wave, which is called the x descent and is related to atrial diastole and perhaps to downward movement of the tricuspid valve during ventricular systole. The second positive wave is the c wave, produced by bulging of the tricuspid valve at the onset of ventricular contraction. The c wave is invisible in many normal subjects in the right atrial pressure pulse record, but in the neck is often exaggerated by the transmitted carotid arterial pulse. The third wave is the v wave, which is produced by passive atrial filling. The v wave is followed by a negative wave, the y descent, which is produced during diastole as blood flows from the right atrium to the right ventricle. When a normal person inhaled, the vertical level of pulsations in the neck veins falls slightly with the decrease of intrathoracic pressure. When the level of pulsations in the veins rises during inspiration, this is an abnormal phenomenon (Kussmaul’s sign). Although generally regarded as a sign of constrictive pericarditis, this physical finding is not diagnostic of constrictive pericarditis, since it may be found in some patients with congestive heart failure.

A disproportionate increase in the amplitude of the a wave is a valuable physical sign that occurs in certain disorders in which outflow from the right atrium is impeded. Among these are tricuspid stenosis and tricuspid atresia (fig. 2). Very large a waves are found in patients who have moderately severe or severe pulmonic valvular stenosis. In these patients there are right ventricular hypertrophy and decreased right ventricular compliance, which render right atrial emptying more difficult.
The a wave is increased for a similar reason in patients who have moderately severe, or severe, pulmonary hypertension. As a result, a prominent a wave may be observed in patients who have severe mitral stenosis with pulmonary hypertension and in patients with congenital heart disease with left-to-right shunting complicated by pulmonary hypertension (Eisenmenger's complex).\textsuperscript{1} Large a waves may be found in patients with other varieties of pulmonary hypertension such as primary pulmonary hypertension, repeated pulmonary embolism, and cor pulmonale related to pulmonary disease.

**Venous Pulse Patterns in Cardiac Arrhythmias**

The a wave is absent in patients with atrial fibrillation. In some patients with atrial flutter it is possible to discern very rapid a waves occurring at a rate of approximately 300 per minute, with slower c and v waves at a rate of 150 or 100 per minute, or at an irregular interval, which depends upon the degree of atrioventricular block. In patients

![Figure 2](image)

*Figure 2*

*Jugular venous pulse recording showing giant a waves in a patient with rheumatic heart disease and tricuspid stenosis.*

![Figure 3](image)

*Figure 3*

*Jugular venous pulse recording, demonstrating cannon a waves in a patient with complete atrioventricular block. The cannon waves occur when the a waves fall within the QT interval of the electrocardiogram.*
with complete atrioventricular block the diagnosis may be made or strongly suspected by inspection of the neck veins. The atrial waves, or a waves, usually occur at a normal rate of 60 to 100 times per minute. The c and v waves occur less often, usually about 40 times per minute in the adult. The atrial rate, determined from the neck veins, is usually more rapid than the ventricular rate, which may be confirmed by palpation of the pulse or auscultation of the precordium. The most significant clue to complete atrioventricular block from inspection of the neck veins are cannon waves (fig. 3). The cannon waves are intermittent giant a waves produced when atrial systole occurs at a time when the tricuspid valve is closed. Thus, if atrial systole occurs at a time corresponding to the QT interval of the electrocardiogram, namely, between the first and second heart sounds, very large a waves will be produced. Cannon a waves occur regularly in nodal rhythms and in first-degree atrioventricular block with very long atrioventricular conduction time, but occur irregularly in complete atrioventricular block.

When there is organic or relative tricuspid insufficiency, a positive regurgitant venous wave precedes the v wave. The c and v waves tend to merge with this regurgitant wave, so that they are in effect replaced by one large positive pulsation. By far the commonest cause of this physical finding is relative tricuspid insufficiency. Regurgitant venous waves are a common observation in patients with right ventricular failure regardless of the cause, and are often found in patients with systemic hypertension or coronary disease with both left and right ventricular failure. Regurgitant waves may be found when there is no murmur of tricuspid insufficiency. In patients with constrictive pericarditis, inspection of the neck veins yields valuable information. The level of the venous pressure is determined by the distention of the veins and by the height of the pulsations. The venous pressure is almost always increased in constrictive pericarditis. The Kussmaul sign may be present, namely, the vertical level of venous pulsations may ascend with inspiration. The a and v waves tend to be increased but the trough produced by the y descent tends to be the predominant venous event. In pa-

Figure 4
Simultaneous recording of normal carotid pulse, with phonocardiogram and electrocardiogram.
tients with tricuspid stenosis who have prominent a waves, and at times large v waves, the y descent is gradual.

**Carotid Arterial Pulse** (fig. 4)

Normally the carotid arterial pulsations may be invisible if the patient is stocky or moderately obese. They may be readily visible in a thin person. Bounding carotid arterial pulsations suggest that the systemic arterial pulse pressure is increased and are usually associated with bounding radial and femoral pulses. Most commonly such exaggerated carotid arterial pulsations are caused by apprehension. The commonest pathologic cause is aortic valvular insufficiency. However, they may be associated with other causes of increased systemic arterial pulse pressure, including patent ductus arteriosus, other forms of congenital communication between the systemic circulation and the right heart, with severe anemia, with systemic arteriovenous fistula, and with thyrotoxicosis. A bounding carotid arterial pulse is strong evidence against severe uncomplicated aortic valvular stenosis but brisk carotid pulsations often occur with idiopathic hypertrophic subaortic stenosis. The carotid arterial pulse is often increased in coarctation of the aorta. Carotid arterial pulsations may be quiet or invisible in patients with severe aortic stenosis, and when there is systemic hypotension. It is important to call attention to unilateral increase of carotid arterial pulsations. These are most commonly observed just above the right clavicle and are often associated with an apparent widening of the carotid artery. This finding is often misinterpreted as an aneurysm of the carotid artery or of the innominate artery. It is most commonly caused by tortuosity or kinking of the common carotid artery in association with arteriosclerosis or hypertension but without true aneurysm formation. This disorder is most common in hypertensive women of middle age and beyond.

**Palpation and Auscultation of the Neck Vessels**

The quality of the carotid arterial pulsation may be confirmed by palpation of the neck. Some patients with aortic stenosis have a systolic thrill and murmur over the carotid arteries. However, such a finding is not diagnostic of aortic stenosis. It may be observed in patients who have loud precordial murmurs and thrills of other cause, especially ventricular septal defect and pulmonic stenosis. A systolic thrill over the carotid arteries may on occasion be a normal finding in children and young adults in association with a supravclavicular arterial bruit. In such instances the thrill is often more intense over the subclavian artery, and especially so on the right side. A localized systolic thrill and murmur over the

![Figure 5](image)

*Figure 5*

Phonocardiogram demonstrating a continuous murmur over the right carotid artery in a patient with partial obstruction of the carotid artery demonstrated by arteriography.
carotid artery may be a reflection of partial obstruction of the carotid artery, most commonly caused by arteriosclerosis. In some such instances a more severe obstruction may produce a continuous thrill and murmur over the carotid artery (fig. 5). Bilateral systolic carotid murmurs are commonly found in high cardiac output states, such as anemia, beriberi, thyrotoxicosis, hepatic failure, and systemic arteriovenous fistula.

It is essential that the examiner be familiar with two common cervical murmurs found in normal children and young people. These murmurs are often misinterpreted.

Cervical Venous Hum
The first of these is the cervical venous hum. The cervical venous hum is a continuous murmur with diastolic accentuation. It is best detected just above the medial end of the clavicle over the sternomastoid muscle. Usually it is readily obliterated by light pressure with a stethoscope or the finger over the internal jugular vein. This murmur is usually detected with the individual in the sitting posture. It almost always disappears or becomes very faint when he lies down. It can be increased by turning the head away from the side being examined or by elevating the chin. The cervical venous hum is found in 95 per cent of children between the ages of 5 and 15 years, and somewhat less frequently below the age of 5 years. In young adults it may be found as often as 75 per cent of instances. Of especial importance is the fact that in 10 to 19 per cent of subjects between infancy and 49 years the murmur may be heard below the clavicle, and at times may be heard in the second right or left intercostal spaces. When the murmur is discovered adjacent to the upper sternum and is not traced to its origin in the neck, it may be confused on the left with the murmur of patent ductus arteriosus, and on the right with the murmurs of aortic stenosis and insufficiency. When heard in the neck alone the venous hum may be mistaken for the murmur of an arteriovenous fistula. The proper diagnosis may be almost always made by attention to the following points. (1) the murmur can be obliterated by light or moderate pressure over the internal jugular vein; (2) the murmur becomes faint or disappears when the individual lies down; (3) the murmur has diastolic accentuation rather than the usual systolic accentuation of a murmur of patent ductus arteriosus or arteriovenous fistula.

Supraclavicular Arterial Bruit
Another very common benign murmur found in the neck and the adjoining supraclavicular area is the supraclavicular bruit. We have observed this murmur in children as young as 2 years of age. It is also quite common in teenagers. The murmur occurs in early systole (fig. 6) and is usually loudest in
the supraclavicular fossa and over the carotid artery. The murmur may be of grade IV or V intensity and may be associated with a thrill. The murmur may be obliterated in most instances, but not all, by compressing the subclavian artery on the same side until the radial pulse on that side disappears (figs. 7 and 8). The murmur may be referred into the anterior thorax. On the right side, its detection over the aortic area may lead to a mistaken diagnosis of aortic valvular stenosis. On the left side, its discovery in the second left intercostal space may lead to an erroneous diagnosis of pulmonary valve stenosis.

**Figure 7**

Phonocardiogram showing the effect of subclavian artery compression upon a supraclavicular bruit. The murmur is accentuated as the artery is partially occluded, and then disappears when further pressure is exerted. The murmur disappears as the ipsilateral radial pulse becomes imperceptible.

**Figure 8**

Phonocardiogram demonstrating the reappearance of the supraclavicular bruit shown in fig. 7 as the compression of the subclavian artery is gradually released. The murmur is at first quite loud, and then returns to its usual intensity.
distinction may be made, as a rule, by careful attention to the following points. First, the murmur is louder in the supraclavicular area than in the thorax; secondly, the murmur is of short duration, usually being limited to the first half of systole; and thirdly, the murmur may usually be obliterated by compressing the subclavian artery on that side against the first rib until the radial pulse disappears. With light compression of the subclavian artery the murmur usually becomes louder (fig. 7).

**Auscultation of the Thorax**

An important feature of cardiovascular examination is the careful auscultation of the thorax for murmurs that may originate outside the heart. Not infrequently such murmurs may be heard over the precordium. If the physician does not examine the remainder of the thorax, an erroneous diagnosis of cardiovascular disease may be made, when in fact none exists. An extracardiac systolic or continuous murmur suggests certain diagnostic possibilities. These include (1) coarctation of the aorta; (2) pseudotruncus arteriosus; (3) pulmonary arteriovenous fistula; (4) pulmonary branch stenosis; (5) anomalous pulmonary artery arising from the aorta; (6) internal mammary arteriovenous fistula; (7) other anomalous arterial branches arising from the aorta or the subclavian artery. We will discuss only the more common of these possibilities.

With coarctation of the aorta there is often a systolic murmur louder over the back of the chest, especially between the scapulae, than over the front of the chest. The studies of Spencer and associates have shown that this murmur often arises within the coarctation itself, although in some patients there is an additional murmur of aortic valvular disease. When the coarctation is severe, with an opening 2.5 mm. or less in diameter, there may be a continuous murmur over the back of the chest. Although in some instances a systolic murmur accompanying coarctation of the aorta may arise from dilated collateral intercostal vessels and scapular vessels, in most instances the continuous murmur arises from the coarcted area. The pulsation of enlarged collateral arteries may be palpable and occasionally visible in the interscapular spaces or beneath the angles of the scapulae. The diagnosis is confirmed by the demonstration of weak and delayed femoral arterial pulses.

Patients who have increased bronchial collateral circulation associated with pulmonary atresia (pseudotruncus arteriosus) commonly have a continuous murmur over the thorax. This is often loudest over the posterior thorax. Patients with pulmonary arteriovenous fistulae often have either a very long systolic murmur or a continuous murmur over the

**Figure 9**

Angiocardiogram revealing a pulmonary arteriovenous fistula in the left lower lung. The arrow indicates a pulmonary vein communicating between the fistula and the left atrium. The pulmonary artery, which supplies the fistula, is just above the vein. This patient had a long systolic murmur over the chest in the area of the fistula.
Thorax. The murmur may be increased in intensity and duration by inspiration. Many patients with this disorder have accompanying Osler-Weber-Rendu disease or hereditary hemorrhagic telangiectasia and may be found to have telangiectasia of the tongue and perhaps over the fingertips. This diagnosis is most clearly demonstrated by angiocardiography (fig. 9).

Pulmonary branch stenosis may be associated with either a long systolic murmur or a continuous murmur. Studies made by Eldridge and associates have shown that moderate compression of an artery causes a systolic murmur, but more severe compression causes the pressure proximal to the obstruction to exceed that distal to the obstruction during both systole and diastole. This observation explains the continuous murmur that may be associated with this disorder. Thus patients with pulmonary branch stenosis may be found to have a long systolic murmur or a continuous murmur that originates outside the heart (fig. 10). This observation, in the absence of the physical findings of coarctation of the aorta, in the absence of cyanosis, and in the absence of hereditary telangiectasia, strongly suggests pulmonary branch stenosis. Radiologic studies are desirable to confirm the diagnosis. Patients with pulmonary branch stenosis often have associated cardiac disease. Atrial septal defect and anomalous pulmonary venous drainage have been commonly found in our patients. Others have found that this disorder is not uncommon in association with tetralogy of Fallot. Here it is often missed unless the angiocardiogram is carefully studied.

**Systemic Arteriovenous Fistula**

This disorder has been referred to earlier as a cause of increased carotid arterial pulsations. Systemic arteriovenous fistulae that produce cardiac disability are most commonly single, large, acquired lesions, although they are occasionally multiple, small, congenital lesions. It is important to inquire concerning a history of trauma or of a surgical operation. The detection of a wide pulse pressure without a cardiac murmur suggests that careful auscultation and palpation be performed over all scars and areas of previous trauma or surgical operation. In typical instances such patients, in addition to a bounding pulse, have

![Figure 10](image_url)

*Figure 10*  
Phonocardiogram showing a long systolic murmur over the precordium in a patient with pulmonary arterial branch stenosis.
a continuous murmur and a palpable continuous thrill over the site of the lesion. If the fistula can be obliterated by manual compression there is characteristically a fall in systolic blood pressure and a rise in diastolic blood pressure and slowing of the heart, the latter being called Branham’s sign. Such physical findings are more likely if the fistula involves the larger arteries including the aorta, its immediate branches, or the femoral artery. As stated earlier, such fistulae may follow surgical procedures and rarely may follow a nephrectomy. The diagnosis may be confirmed by arteriography.

**Palpation of the Pulse**

Space does not permit a discussion of the diagnosis of the various cardiac arrhythmias, which may be suggested from palpation of the radial pulse. Neither can we consider the several causes of weakness or obliteration of one or both radial or femoral pulses.

**The Paradoxical Pulse**

We should like to devote our attention to the paradoxical pulse. A paradoxical pulse is an abnormal decline in systolic blood pressure during inspiration. It is well known that systolic blood pressure normally falls several mm. of mercury with inspiration. Studies of experimental animals in our laboratory have shown that there is in fact a decrease in left ventricular stroke output during normal inspiration 10 (fig. 11). This decrease is believed to be related to the expiratory fall of right ventricular output and the delay in transmission through the pulmonary circulation. When systolic blood pressure falls as

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**Figure 11**

Pressure recordings from the aorta, pericardium, and pleural space, and flow recording in the descending aorta of a dog which was subjected to experimental cardiac tamponade. In the control record on the left, with the onset of inspiration, indicated by the decline of intrapleural pressure, there is a decrease of aortic flow and pressure. The record on the right is made after cardiac tamponade was produced by the injection of physiologic saline solution into the pericardial space. The pericardial pressure is increased approximately 10 mm. Hg from the control. There is a striking paradoxical pulse in the aortic pressure recordings. The inspiratory fall of blood pressure is accompanied by a considerable decline in aortic stroke flow during inspiration.
much as 8 or 10 mm Hg during quiet inspiration, a paradoxical pulse is said to be present. When the paradoxical pulse is moderate, it is best detected during blood pressure measurement while the patient breathes normally. Deep inspiration may cause 10 or 15 mm fluctuation in systolic blood pressure in a normal person but should not cause the radial pulse to disappear. The latter event suggests that there is a significant paradoxical pulse. Paradoxical pulse has been most commonly described in association with pericardial disease that compresses the heart either by fluid or scar tissue. It is important to state that a paradoxical pulse is more commonly caused by emphysema or bronchial asthma, and it may be found occasionally in patients with myocardial disease.

The mechanism of the paradoxical pulse in pericardial disease has been of considerable interest to us. Katz and Gauchat \(^{11}\) suggested in 1924 that the paradoxical pulse of pericardial disease was produced by a greater inspiratory fall in pulmonary venous pressure than in intracardiac pressure, thus producing a reduced pressure gradient for filling of the left heart during inspiration. Dock \(^{12}\) has stated that inspiratory traction upon the pericardium increases intrapericardial pressure and thus interferes with cardiac filling during inspiration. Dornhorst and associates \(^{13}\) postulated that increased right heart filling during inspiration compresses the left heart thus interfering with its filling. Decreased filling in turn leads to decreased left heart output during inspiration. Studies in our laboratory have demonstrated that there is inspiratory increase of right heart filling during cardiac tamponade.\(^{14}\) If right heart venous return is made constant during cardiac tamponade, the paradoxical pulse no longer occurs (fig. 12). Sudden increase in right heart filling during cardiac tamponade leads to temporary decrease in left heart output. Thus it seems that

**Figure 12**

Simultaneous recording of aortic pressure, intrapleural pressure, and intrapericardial pressure in a dog subjected to cardiac tamponade. In this animal, systemic venous return was drained into a reservoir and the blood pumped at a constant rate into the right atrium. The control record is shown on the left; on the right, the intrapericardial pressure has been increased approximately 10 mm Hg by the injection of physiologic saline solution into the pericardial space. The inspiratory decline of aortic pressure is no greater than during the control period and no significant paradoxical pulse is produced by cardiac tamponade. Compare with fig. 11.
the paradoxical pulse is related to increased inspiratory filling of the right heart which increases the pressure within the pericardial space. Increased intrapericardial pressure interferes with left heart filling during inspiration. Patients with myocardial disease may have a paradoxical pulse. This abnormality is more likely in patients with primary myocardopathy, myocardial fibrosis, and cardiac amyloidosis. Relative to this point, we have been able to show that acute cardiac dilatation in the experimental animal in effect causes the normal pericardium to compress the distended heart. A sudden increase of right heart return again leads to a temporary decrease in left heart stroke output. With experimental respiratory obstruction left heart output shows no greater-than-normal inspiratory fall. The abnormal inspiratory decline of blood pressure is caused by the increased variation of intrathoracic pressure.

References