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**Chapter 30 Examination of the Extremities: Pulses, Bruits, and Phlebitis**

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**Definition**Go to: 

Assessment of the peripheral vascular system is done to determine the characteristics of the pulse, to ascertain the presence of an arterial bruit(s), and to detect the occurrence of venous inflammation with possible secondary thrombosis of that vein.

Increases in pulse rate (tachycardia) may suggest hyperthyroidism, anxiety, infection, anemia, or arteriovenous fistula. Slowing of the pulse rate (bradycardia) may be seen in heart block, hypothyroidism, or with the use of certain drugs (e.g., propranolol). Irregularities in the pulse suggest the presence of premature beats, and a completely irregular pulse implies the presence of atrial fibrillation. Diminished or absent pulses in the various arteries examined may be indicative of impaired blood flow due to a variety of conditions.

Bruits are rushing sounds heard over large and medium-sized arteries as a result of vibration in the vessel wall caused by turbulent blood flow. The sound may originate from a local narrowing or dilation of the vessel itself, or it may be transmitted along the artery from a more proximal lesion in the vascular system. The intensity and duration of the bruit relate to the degree of vessel wall distortion. In general, bruits are not audible until an artery is approximately 50% occluded. The sound increases in pitch as the lumen becomes more narrowed to a critical size. Thereafter, the sound may no

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longer be detectable as the volume of blood flow becomes greatly reduced.

## Technique

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## Pulses

A complete physical examination includes the assessment and recording of arterial pulses in all locations. While examining the pulse, the observer should note its intensity, rate, rhythm, and if any blood vessel tenderness, tortuosity, or nodularity exists. It is unreliable to attempt to estimate blood pressure via arterial palpation without the use of the sphygmomanometer.

The patient should be examined in a warm room with arrangements made so that the patient's pulses can easily be examined from both sides of the bed. A cool environment may cause peripheral vasoconstriction and reduce the peripheral pulse. Palpation should be done using the fingertips and intensity of the pulse graded on a scale of 0 to 4 +: 0 indicating no palpable pulse; 1 + indicating a faint, but detectable pulse; 2 + suggesting a slightly more diminished pulse than normal; 3 + is a normal pulse; and 4 + indicating a bounding pulse.

The student examiner must be alert to the possibility that the pulse he or she feels may be due to digital artery pulsations in his own fingertips; this source of confusion can be eliminated by comparing the pulse in question to his own radial pulse or to the patient's cardiac sounds as determined by auscultation over the precordium. In general, it is inadvisable to use the thumb in palpating for peripheral pulses. The thumb carries a greater likelihood of confusion with the examiner's own pulse and generally has less discriminating sensation than the fingers. Frequently, inspection will be an aid to pulse location. The examiner may be able to see the skin rise and fall with each pulsation along the course of an extremity artery, particularly if a bright light is aimed tangentially across the surface of the skin.

To examine the *brachial artery* ([Figure 30.1](#)) in the right arm, the examiner supports the patient's forearm in his left hand, with the subject's upper arm abducted, the elbow slightly flexed, and the forearm externally rotated. The examiner's right hand is then curled over the anterior aspect of the elbow to palpate along the course of the artery just medial to the biceps tendon and lateral to the medial epicondyle of the humerus. The position of the hands should be switched when examining the opposite limb ([Figure 30.1](#)).



**Figure 30.1**

## Brachial artery.

For the *radial artery* ([Figure 30.2](#)), the patient's forearm should be supported in one of the examiner's hands and his other hand used to palpate along the radialvolar aspect of the subject's forearm at the wrist. This can best be done by curling the fingers around the distal radius from the dorsal toward the volar aspect, with the tips of the first, second, and third fingers aligned longitudinally over the course of the artery.



[Figure 30.2](#)

## Radial artery.

The *abdominal aorta* ([Figure 30.3](#)) is an upper abdominal, retroperitoneal structure which is best palpated by applying firm pressure with the flattened fingers of both hands to indent the epigastrium toward the vertebral column. For this examination, it is essential that the subject's abdominal muscles be completely relaxed; such relaxation can be encouraged by having the subject flex the hips and by providing a pillow to support the head. In extremely obese individuals or in those with massive abdominal musculature, it may be impossible to detect aortic pulsation. Auscultation should be performed over the aorta and along both iliac vessels into the lower abdominal quadrants.



[Figure 30.3](#)

## Abdominal aorta.

The *common femoral artery* ([Figure 30.4](#)) emerges into the upper thigh from beneath the inguinal ligament one-third of the distance from the pubis to the anterior superior iliac spine. It is best palpated with the examiner standing on the ipsilateral side of the patient and the fingertips of the examining hand pressed firmly into the groin. Auscultation should be performed in this area, as well.



[Figure 30.4](#)

Femoral artery.

The *popliteal artery* ([Figure 30.5](#)) passes vertically through the deep portion of the popliteal space just lateral to the midplane. It may be difficult or impossible to palpate in obese or very muscular individuals. Generally this pulse is felt most conveniently with the patient in the supine position and the examiner's hands encircling and supporting the knee from each side. The pulse is detected by pressing deeply into the popliteal space with the supporting fingertips. Since complete relaxation of the muscles is essential to this examination, the patient should be instructed to let the leg "go limp" and to allow the examiner to provide all the support needed.



[Figure 30.5](#)

Popliteal artery.

The *posterior tibial artery* ([Figure 30.6](#)) lies just posterior to the medial malleolus. It can be felt most readily by curling the fingers of the examining hand anteriorly around the ankle, indenting the soft tissues in the space between the medial malleolus and the Achilles tendon, above the calcaneus. The thumb is applied to the opposite side of the ankle in a grasping fashion to provide stability. Again, obesity or edema may prevent successful detection of the pulse at the location.

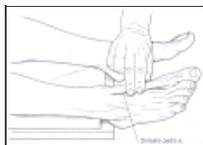


[Figure 30.6](#)

Posterior tibial artery.

The *dorsalis pedis artery* ([Figure 30.7](#)) is examined with the patient in the recumbent position and the ankle relaxed. The examiner stands at the foot of

the examining table and places the fingertips transversely across the dorsum of the forefoot near the ankle. The artery usually lies near the center of the long axis of the foot, lateral to the extensor hallucis tendon but it may be aberrant in location and often requires some searching. This pulse is congenitally absent in approximately 10% of individuals.



**Figure 30.7**

Dorsalis pedis artery.

### **Bruits**

After palpating the artery, auscultation for a bruit should be performed. Bruits are detected by auscultation over the large and medium-sized arteries (e.g., carotid, brachial, abdominal aorta, femoral) with the diaphragm of the stethoscope using light to moderate pressure. Excessive pressure may produce, intensify, or prevent a bruit from being detected by indenting the vessel wall or occluding blood flow in the artery. One should listen over the artery after palpation of the artery to avoid overlooking a significant lesion.

Occasionally, bruits are audible over the upper abdomen in young, healthy individuals. These sounds apparently originate from tortuous vessels and are of no clinical significance; if the subject has a normal blood pressure and is free of abdominal symptoms, such findings may be disregarded.

Frequently the examiner will detect a "thrill" or palpable vibratory sensation over a vessel in which a loud bruit is audible. The thrill is indicative of marked turbulence in local blood flow and suggests significant vascular pathology. If a thrill is noted during examination of the pulses, it should be recorded in the appropriate space on the data base.

### **Phlebitis**

With the patient supine, the veins of the extremities usually cannot be palpated but can be examined by inspection. Phlebitis most commonly occurs in the superficial arm veins and the deep veins of the lower extremity.

Arm vein phlebitis is suggested by noting erythema occurring along the course of the vein, which may be tender to palpation if secondary venous thrombosis has occurred; this may be palpable (i.e., a "cord").

Lower extremity thrombophlebitis can be clinically silent and may be very difficult to detect by physical examination. Changes secondary to the venous obstruction may develop and be observed as increased thigh or calf girth, erythema, warmth, tenderness, or palpation of a cord. Numerous laboratory

techniques have been developed to assist the physician in the search for lower extremity thrombophlebitis.

Measurement of calf circumference should be obtained in all patients with suspected lower extremity thrombophlebitis. This should be done with the patient standing and the feet 30 cm apart. The maximum circumference is recorded and a significant difference exists if the two sides differ by 1.5 cm in males and 1.2 cm in females.

Skin erythema or warmth may be noted in active phlebitis. A thrombosed segment of vein (i.e., a cord) may be palpable, especially in the superficial veins of the lower extremity.

Pain is a prominent feature of muscular, synovial, or vascular leg disease and various tests have been suggested to help identify the specific etiology.

Homan's test (dorsiflexion sign) is most popularly used to detect irritability of the posterior leg muscles through which inflamed or thrombosed veins course. A popular clinical misconception is that calf pain is the endpoint of the test; however, Homan clearly stated that "discomfort need have no part in this reaction." A positive sign is when dorsiflexion of the foot on the affected side is less complete or is met with more resistance than on the unaffected side. Resistance to dorsiflexion may also be manifested by involuntary flexion of the knee.

The Lowenberg cuff test is another helpful clinical maneuver for detection of calf vein thrombosis. Wrap a blood pressure cuff around the thigh just above the knee, taking care not to pinch the skin behind the knee. Close the valve and inflate the cuff gradually to 180 mm Hg. Ask the patient to tell you of any unusual discomfort. Minimal discomfort immediately under the cuff is common. Spontaneous complaint of calf pain at 20 to 80 mm Hg (that is, above venous pressure) is highly suggestive of local venous disease, particularly if 150 to 180 mm Hg contralateral thigh pressure is well tolerated.

Other symptoms and signs include Moses' test (calf pain greater with anteroposterior than side-to-side palpation), localized leg pain on coughing (Lawrence's sign), and tenderness to touch in the sole of the foot (Owane's sign).

## Basic Science

Go to:

The deep veins of the lower leg include the paired anterior tibial, posterior tibial, and peroneal veins that course alongside the tibia and fibula through the soleus and gastrocnemius muscles. Those veins join to form the deep popliteal vein behind the knee. The popliteal vein then drains into the superficial and common femoral vein and on to the external iliac vein, inferior vena cava, right heart chambers, and pulmonary arteries.

The superficial veins of the lower leg include the long (greater) and short

(lesser) saphenous. The long saphenous vein is usually visible just anterior to the medial malleolus of the foot. It courses superficially up the leg until joining the common femoral vein near the inguinal ligament. The short saphenous vein is often visible just posterior to the lateral malleolus of the foot. It courses up the lateral leg aspect to join the popliteal vein behind the knee.

On quiet standing, the venous pressure approaches 120 cm H<sub>2</sub>O in the lower leg veins. This is reduced to 20 cm H<sub>2</sub>O during walking. The high dependent venous pressure accounts for blood flows of only 4.0 cm/sec. Venous return up the leg is assisted by muscular contractions that squeeze blood far enough up the veins to be trapped by bicuspid venous valves, then spurted upward again by the next muscular contraction. These valves are typically present in both superficial and deep leg veins as far proximally as the external iliac vein.

Venous stasis and slow flow accelerate thrombus formation. It is therefore not surprising that various "hypercoagulable" clinical states are associated with thrombosis of the deep leg or other low-flow systemic veins.

Thrombosis is further accelerated by either traumatic or inflammatory injury to the venous endothelium such that platelet aggregation and fibrin formation are enhanced.

Since thrombosis in the calf veins is usually asymptomatic, an astute clinician should be aware of its potential presence given appropriately suspicious clinical settings. Examples would include the following:

1. Any recent leg trauma
  - a. Fractures of the tibia, fibula, femur, or hip
  - b. Soft-tissue leg injuries
  - c. Ankle sprains
2. Venous stasis and relative leg muscle immobility
  - a. Bed confinement
  - b. Postoperative
  - c. Postpartum
  - d. Varicose veins or chronic venous insufficiency
  - e. Prolonged car, truck, or bus trips, high strap boots, extended TV viewing with crossed legs
3. Certain drugs
  - a. Postdiuresis

b. Corticosteroids

c. Estrogen-containing oral contraceptives, particularly high-dose

4. Malignancy

## Clinical Significance

Go to: 

Examination of the pulse can provide clues to the presence of systemic diseases. Ancient physicians would diagnose heart, liver, renal, and gastrointestinal problems from assessment of the pulse. Today, other tests have been developed to assist in the work-up of systemic illness, but examination of the pulse is still an important part of patient assessment.

Conditions associated with tachycardia or bradycardia have been discussed, as have the causes of a pulse irregularity. Reduced or absent arterial pulses are a sign of impaired blood flow. The causes include: (1) congenital abnormalities (coarctation of the aorta, anomalous peripheral arteries); (2) intrinsic arterial disease (atherosclerosis, thrombosis, arteritis); (3) vasospastic disorders (Raynaud's phenomenon); or (4) involvement of the vessel by extrinsic compression (thoracic outlet syndrome, trauma, neoplasms). The resultant alteration of pulses, with or without accompanying bruits, may be indicative of either acute or chronic changes in a given patient. The vascular history, together with associated physical findings such as skin color, temperature, and neuromotor status of the extremity, should help to elucidate these points. More refined diagnostic techniques such as Doppler ultrasound examination and arteriography may be required to evaluate abnormalities suspected from the physical examination. In this regard, it is important to understand that significant arterial occlusive disease of the lower extremities may exist in a patient who has almost normal peripheral pulses in the resting state, since collateral circulation can produce pulsatile flow in the peripheral arterial bed in some patients. If such an individual is instructed to exercise to the point of claudication, however, pulse distal to the major vascular occlusion will diminish or disappear.

Significant widening of an artery to the examining fingers may be the best clue to an otherwise silent arterial aneurysm. The wary examiner will not be misled by tortuosity of the vessel giving a false impression of increased diameter. Careful palpation may also reveal the rock-hard vessel wall of calcified atherosclerosis, the harsh systolic thrill of a tight arterial stenosis, or the continuous thrill of a peripheral arteriovenous fistula. In the latter condition, auscultation should confirm a continuous, or machinery-like, murmur with systolic accentuation.

Much valuable information can be gained from examination of the peripheral pulses in addition to the status of the arterial system itself. The attentive examiner may detect variations in the rate, rhythmicity, intensity, and contour

of the pulse wave that yield insight into a variety of disease states. The rapid, thready pulse of hypovolemic shock is a well-known clinical sign, as is the rapid, snapping pulse characteristic of thyrotoxicosis, and the collapsing, "water-hammer" pulse of aortic insufficiency. (Also read [Chapter 17, Pulse](#), and [Chapter 20, Carotid Pulse](#).)

There are at least four reasons why it is important to determine if a patient does or does not have thrombophlebitis. These include the threat and prophylaxis of pulmonary embolism, the risk of septicemia, the use of certain drugs other than anticoagulants, and the occasional detection of any other primary disease processes.

Deep vein thrombophlebitis requires hospitalization and anticoagulation to prevent morbidity and mortality from associated pulmonary embolism. The efficacy of anticoagulant therapy in this setting was clearly demonstrated in control studies done many years ago. Evidence is not convincing that anticoagulant therapy is of benefit in phlebitis clearly isolated to superficial veins of the arm or saphenous system of the leg. However, there are communicating leg veins between the superficial and deep venous system, and the examiner bears the responsibility for determining that the aforementioned signs of deep phlebitis are absent.

Pelvic thrombophlebitis and thrombophlebitis associated with indwelling intravenous catheters are a surprisingly frequent source of septicemia. Recognition of either requires careful evaluation of the patient for evidence of bloodstream infection.

Thrombophlebitis and pulmonary embolism are more frequent in recently diuresed patients. One should therefore use therapeutic discretion in planning diuretic therapy for patients who belong to any of the previously described "suspicious clinical settings." In addition, patients receiving corticosteroid therapy are more susceptible to phlebitis. The same is true for women taking estrogen-containing oral contraceptives; other family planning measures should be substituted for such therapy in the presence of spontaneous thrombophlebitis.

Thrombophlebitis that is either recurrent, located in unusual veins (subclavian, jugular), or migratory to various superficial veins may be the first sign of an underlying disease process such as occult malignancy or systemic lupus erythematosus.

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