The term digital clubbing is used to describe an enlargement of the distal segments of the fingers. The toes also may be affected by clubbing. Clubbing may be hereditary, but more often it is a sign of underlying disease. Clubbing is associated with a variety of pulmonary diseases, including idiopathic pulmonary fibrosis, lung cancer, bronchiectasis, lung abscess, and cystic fibrosis. Nonpulmonary conditions that may be accompanied by clubbing include cyanotic congenital heart disease, infective endocarditis, inflammatory bowel disease, and cirrhosis. Clubbing also may be idiopathic.

DETECTING THE PRESENCE OF CLUBBING

Digital clubbing is defined by structural changes at the base of the nails that results in a convex distal phalanx (Figure 1). The Schamroth sign can be used in the detection of clubbing. This sign is elicited by placing the dorsal surfaces of the terminal phalanges on opposing fingers together (Figure 2). Normally, a diamond-shaped window appears in a patient without clubbing, whereas in a patient with clubbing (Figure 1B) this window is obliterated.

Identification of advanced clubbing poses little challenge when the so-called “drumstick fingers” are obvious; however, identification of the early stages of clubbing can prove difficult. Two objective measures for determining the presence of clubbing have been proposed. The first, known as the digital index, measures 2 separate circumferences on each of the 10 fingers at the nail bed (NB) and the distal interphalangeal joint (DIP) (Figure 3). The sum of the 10 ratios (NB:DIP) determines the digital index. A digital index of 10.2 or higher signifies the presence of clubbing. Although a NB:DIP ratio of 1.0 or greater at any single digit is suggestive of clubbing, the sum of the 10 ratios is more specific for clubbing.

The second objective method for the detection of clubbing is calculation of the phalangeal depth ratio. In the normal finger, the distal phalangeal depth (DPD) is smaller than the depth of the interphalangeal joint (IPD). To perform this test, calipers should be used. The calipers should touch but not compress the skin. For clubbing to be present, the DPD:IPD ratio should exceed 1.0 (Figure 4). The index finger is the suggested digit used for measurement.

CLINICAL CORRELATIONS

Digital clubbing is a clinical syndrome with an unknown pathogenesis. Possible mechanisms include dilation of peripheral vessels and stimulation of connective tissue growth by platelet-derived growth factor (PDGF) or hepatocyte growth factor (HGF). Digital clubbing typically is a sign of underlying disease, usually of pulmonary or cardiovascular origin (Table 1). Pulmonary conditions often accompanied by clubbing include chronic lung infections, malignancy, and chronic interstitial lung diseases. Idiopathic pulmonary
fibrosis is one of the most common underlying causes of digital clubbing. Digital clubbing does not usually occur in chronic obstructive pulmonary disease or asthma. If clubbing is seen in a patient with chronic obstructive pulmonary disease, a work-up for underlying malignancy should be undertaken. Clubbing in patients with pulmonary diseases is usually accompanied by cyanosis.

Clubbing is not specific to pulmonary disorders and also can be seen in cyanotic congenital heart disease, infective endocarditis, cirrhosis of the liver, and inflammatory bowel disease. In fact, one study found that the highest incidence of clubbing occurred in patients with chronic liver disease. Clubbing without cyanosis is common in patients with infective endocarditis, inflammatory bowel disease, and cirrhosis.

Clubbing can be associated with hypertrophic osteoarthropathy. Unlike other forms of clubbing, clubbing associated with hypertrophic osteoarthropathy is a painful process. Hypertrophic osteoarthropathy results from subperiosteal formation of new bone in the distal long bones, and it occurs in patients with lung cancer, bronchiectasis, and cirrhosis. Clubbing in patients with hypertrophic osteoarthropathy is believed to be stimulated by PDGF leading to proliferation of connective tissue and periosteum. Several mechanisms have been proposed by which platelet clumps accumulate in the finger tips and release PDGF.

HGF is another potential stimulator of clubbing. One study found higher levels of HGF in lung cancer
patients with clubbing than in those without clubbing, suggesting that HGF may play a role in the formation of digital clubbing. Clubbing has been reported in 29% of patients with lung cancer and is more common in patients with non–small-cell lung cancer than those with small-cell lung cancer. Another study found higher levels of HGF in patients with idiopathic pulmonary fibrosis as compared to normal age-matched controls, suggesting that HGF may play a role in the pathogenesis of idiopathic pulmonary fibrosis in addition to the development of clubbing.

CONCLUSION

Digital clubbing is a clinical sign associated with a number of diseases. The most common causes of clubbing are pulmonary disease, cyanotic heart disease, and cirrhosis of the liver, but clubbing also may be idiopathic or congenital. Clubbing is often visually obvious. It can be confirmed by observation of the Schamroth sign, determination of the digital index, or by calculation of the phalangeal depth ratio. As it may be a sign of serious underlying disease, a comprehensive work-up should be performed when a patient presents with digital clubbing.

REFERENCES