Does This Patient Have Clubbing?

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Context The association between digital clubbing and a host of diseases has been recognized since the time of Hippocrates. Although the features of advanced clubbing are familiar to most clinicians, the presence of early clubbing is often a source of debate.

Objective To perform a systematic review of the literature for information on the precision and accuracy of clinical examination for clubbing.

Data Sources The MEDLINE database from January 1966 to April 1999 was searched for English-language articles related to clubbing. Bibliographies of all retrieved articles and of standard textbooks of physical diagnosis were also searched.

Study Selection Studies selected for data extraction were those in which quantitative or qualitative assessment for clubbing was described in a series of patients. Sixteen studies met these criteria and were included in the final analysis.

Data Extraction Data were extracted by both authors, who independently reviewed and appraised the quality of each article. Data extracted included quantitative indices for distinguishing clubbed from normal digits, precision of clinical examination for clubbing, and accuracy of clubbing as a marker of selected diseases.

Data Synthesis The profile angle, hyponychial angle, and phalangeal depth ratio can be used as quantitative indices to assist in identifying clubbing. In individuals without clubbing, values for these indices do not exceed 176°, 192°, and 1.0, respectively. When clinicians make a global assessment of clubbing at the bedside, interobserver agreement is variable, with κ values ranging between 0.39 and 0.90. Because of the lack of an objective diagnostic criterion standard, accuracy of physical examination for clubbing is difficult to determine. The accuracy of clubbing as a marker of specific underlying disease has been determined for lung cancer (likelihood ratio, 3.9 with phalangeal depth ratio in excess of 1.0) and for inflammatory bowel disease (likelihood ratio, 2.8 and 3.7 for active Crohn disease and ulcerative colitis, respectively, if clubbing is present).

Conclusions We recommend use of the profile angle and phalangeal depth ratio as quantitative indices in identifying clubbing. Clinical judgment must be exercised in determining the extent of further evaluation for underlying disease when these values exceed 180° and 1.0, respectively.

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CLINICAL SCENARIOS

Case 1

A respiratory therapist asks you to see her asymptomatic 76-year-old mother in consultation because she is concerned that her mother has clubbing. The patient has increased curvature of the nails, and you wonder whether other physical examination techniques can help you decide whether clubbing is present.

Case 2

While performing a routine physical examination on a 65-year-old female smoker with chronic obstructive pulmonary disease (COPD), you detect changes in the fingers suggestive of clubbing. You recall an association between clubbing and certain types of pulmonary disease, and you wonder whether any further diagnostic evaluation of this patient is warranted.

Why Is the Clinical Examination Important?

Clubbing is one of those phenomena with which we are all so familiar that we appear to know more about it than we really do.[1]
Samuel West, 1897

The association of clubbing with a host of infectious, neoplastic, inflammatory, and vascular diseases has captured the imagination of clinicians since Hippocrates first described clubbing in a patient with empyema in the fifth century BC.[2] Although clubbing can be a benign hereditary condition, the diagnostic implications in an adult are such that its detection should prompt consideration of the underlying etiology (TABLE 1).[3,4] In the pediatric population, clubbing usually represents the progression of established diseases, such as cystic fibrosis or uncorrected cyanotic congenital heart disease.

Digital clubbing is characterized by the enlargement of the terminal segments of the fingers and/or toes that results from the proliferation of the connective tissue between the nail matrix and the distal phalanx. Although most often symmetrical, clubbing can be unilateral or even unidigital.[5,6] Clubbing can occur in isolation or in association with hypertrophic osteoarthropathy.[7,8] Hypertrophic osteoarthropathy, a systemic disorder affecting bone and joints, is most commonly associated with bronchogenic carcinoma, but

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Table 1. Conditions Associated With Acquired Clubbing

Neoplastic intrathoracic disease
Bronchogenic carcinoma
Malignant mesothelioma
Pleural fibroma
Metastatic osteogenic sarcoma
Suppurative intrathoracic disease
Lung abcess
Bronchiectasis
Cystic fibrosis
Empyema
Chronic cavitary mycobacterial or fungal infection
Diffuse pulmonary disease
Idiopathic pulmonary fibrosis
Asbestosis
Pulmonary arteriovenous malformations
Cardiovascular disease
Cyanotic congenital heart disease
Infective endocarditis
Arterial graft sepsis*
Brachial arteriovenous fistula†
Hemiplegic stroke†
Gastrointestinal disease
Inflammatory bowel disease
Celiac disease
Hepatobiliary disease
Cirrhosis (particularly biliary and juvenile)
Metabolic disease
Thyroid acropachy

*Associated with clubbing distal to graft sepsis.
†Associated with unilateral clubbing.

it can occur in association with extrapulmonary malignancies as well as nonmalignant pulmonary diseases.[9] Pachydermoperiostosis is a rare, congenital form of hypertrophic osteoarthropathy. Congenital clubbing, which usually has its onset in childhood, may represent a limited form of pachydermoperiostosis.[5]

Unlike such physical findings as ascites and splenomegaly, the clinical impression of clubbing cannot be verified by simple imaging tests. Over the past century, many investigators have described possible reference standards for diagnosis of clubbing, including water displacement of the terminal phalanges, measurement of nail curvature using a device called an unguisometer, and measuring nail angles and ratios using plaster casts or shadow projections of fingers.[10-15] None has been accepted as a criterion standard of diagnosis, and all are cumbersome and impractical as a method of verifying the clinical impression of clubbing. Therefore, physicians must rely solely on their skills in clinical examination to detect clubbing.

Pathophysiology

Normally, the nail-bed thickness is less than 2.0 mm. Clubbed fingers studied at autopsy show not only a thickness greater than 2.0 mm, but also a lower density of nail-bed connective tissue.[16] Morphologic findings include the presence of primitive fibroblasts, elevated numbers of eosinophils and lymphocytes, and increased caliber and number of blood vessels. Genetic predisposition, vagally mediated neural mechanisms, and the direct effect of tissue hypoxia or of circulating vasodilators that elude metabolism in the lung through right-to-left shunting have all been proposed to explain the morphology. While there is experimental and clinical evidence to support each of these hypotheses, it has not been possible to formulate a comprehensive theory of pathogenesis applicable to all clinical circumstances.[5,17-19]

Symptoms

Clubbing is almost always painless, unless it is associated with hypertrophic osteoarthropathy. Symptoms of hypertrophic osteoarthropathy include periarticular pain and swelling, most often in the wrists, ankles, knees, and elbows. Accordingly, the presentation of hypertrophic osteoarthropathy can be confused with such primary rheumatological disorders as rheumatoid arthritis.[5] Many patients with clubbing express unawareness of any abnormality in their fingers. In one series of patients with clubbing, only 32 of 116 patients were aware of the onset of the changes in their nails, and only 2 reported painful fingers or joints.[20]

Signs

Identification of advanced clubbing, which is characterized by so-called drumstick fingers poses little difficulty for clinicians. By contrast, the subtleties of the earlier stages of clubbing may lead to animated bedside debate among medical students, residents, and experienced physicians. The 2 approaches for identifying clubbing on physical examination are visual inspection and palpation of the cuticle for increased sponginess.[16,21-22]

Inspection

General Appearance. Inspection of the fingers for clubbing can reveal abnormalities in the nail-fold angles, and in the shape, depth, and width of the terminal phalanges. In addition to the obvious changes in the shape of the terminal phalanges in established clubbing (FIGURE A), close inspection of the cuticle may reveal a shiny and smooth appearance. Lovibond[23] described a lilac hue of the nail fold in clubbing, caused by increased vascularity in the connective tissue. Although the increased nail curvature seen in clubbed fingers has been studied extensively using chord-arc measurements and unguisometers, it is not easily measured at the bedside. Moreover, nail curvature tends to become more pronounced with age and can occur in the absence of other signs of clubbing.[5,24]

Nail-fold Angles. Inspection of clubbed fingers reveals a number of abnormalities in the angles made by the nail as it exits from the terminal phalanx. Lovibond[23] popularized this as the profile sign in his 1939 report on the diagnosis of clubbed fingers. He observed that in normal fingers, the nail projects from the nail bed at an angle of about 160°, but that this angle approached 180° in clubbed fingers (Figure, B). Later, the hyponychial angle was proposed as a more reliable sign than the profile angle in the assessment of clubbing (Figure, B).[11]

Phalangeal Depth Ratio. Estimation of the phalangeal depth ratio can be used to identify clubbing (Figure, C).[14] In the normal finger, the distal phalangeal depth is smaller than the interphalangeal depth. As connective tissue deposition expands the pulp in the terminal phalanx, this ratio becomes reversed. The phalangeal depth ratio appears to be independent of age, sex, and ethnicity in randomly selected populations.[14,25] A similar ratio using distal and interphalangeal width can be determined, but it has not been studied as extensively as the phalangeal depth ratio.

Although the phalangeal depth ratio was originally described using plaster casts and shadowgrams, subsequent studies have reported the use of cali-

pers on live fingers. To perform this measurement, the calipers should touch but not compress the tissue at the distal phalanx and the interphalangeal joint of the index finger during measurement. Baughman et al[26] estimated that this technique takes no longer than 1 minute to perform. Visual estimation for the reversal of the phalangeal depth ratio has been suggested as a simple bedside technique for clubbing, but the precision of this method has not been tested.

Schamroth Sign. In 1976, Schamroth[27] reported a new clinical sign that incorporated 2 of the clinical features of clubbing (Figure, D). Normal fingers create a diamond-shaped window when the dorsal surfaces of terminal phalanges of similar fingers are opposed. In the clubbed finger, the diamond becomes obliterated because of the loss of the profile angle and the increase in the soft tissue at the cuticle. Since its original description, this technique has become popular with physicians as a quick test to establish the presence of clubbing. The precision and accuracy of this sign, however, have not been formally tested.[28]

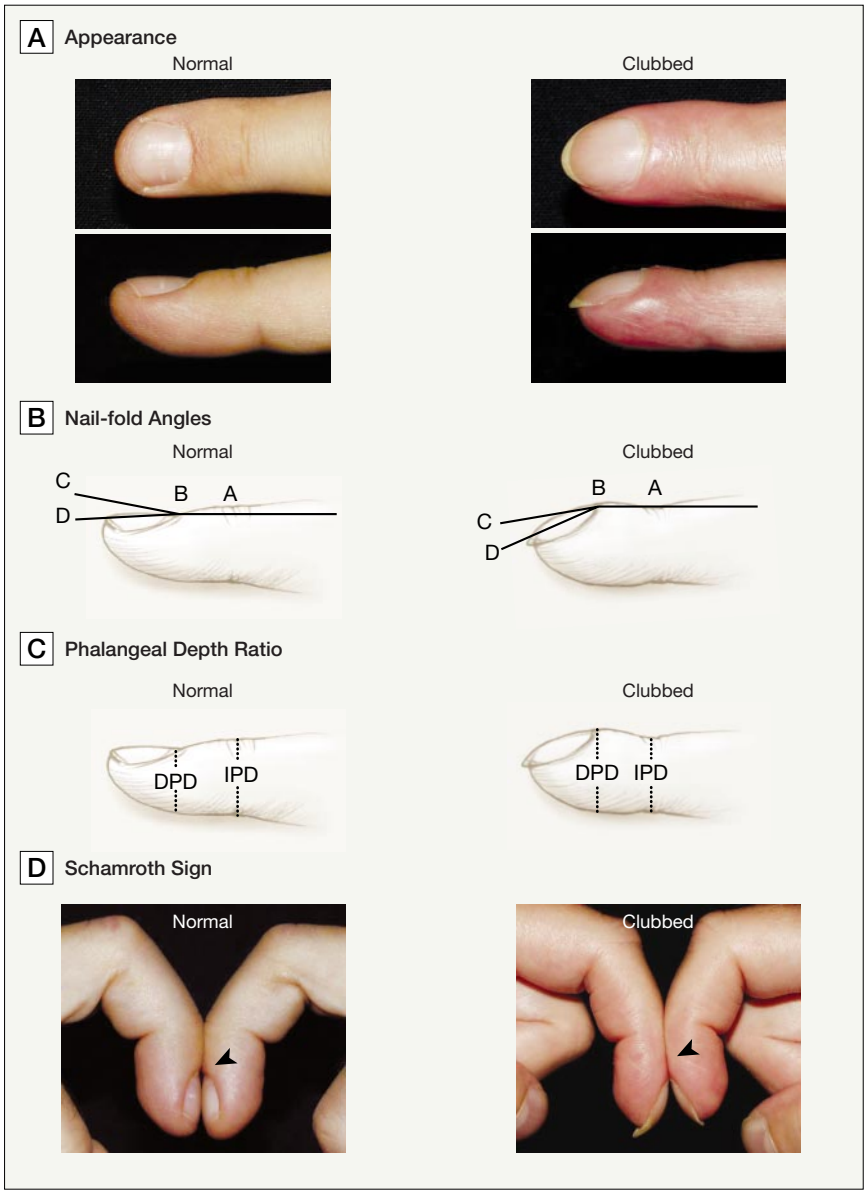
Palpation. On palpation of the base of the nail bed, the examiner perceives that the nail is "floating" within the soft tissue, and in advanced cases may even be able to feel the proximal edge of the nail. This sign is best elicited by gently rocking the nail. The examiner grips the sides of the subject's finger between the thumb and middle finger of each hand. Exerting downward pressure with his/her own index fingers, the examiner then rocks the distal and proximal ends of the subject's nail, using the nail bed as a fulcrum.

METHODS

We used the MEDLINE database to search for English-language articles related to the clinical evaluation of clubbing that were published between January 1966 and April 1999. The MeSH heading *hypertrophic osteoarthropathy*, followed by the textword *clubbing*, were used in the following search strategy: *physical examination/ or physical exam$*; *medical history taking*; *professional competence*; *sensitivity* and *specificity* or *sensitivity and specificity*; *reproducibility of result*; *observer variation*; *diagnostic tests*, *routine*; *decision support techniques*; and *Bayes theorem*. This strategy resulted in a limited number of articles.

To expand the search, the titles and abstracts of all articles retrieved using the MeSH heading *hypertrophic osteoarthropathy* or the textwords *clubbing* and *Hippocratic fingers* were evaluated by each author independently. Based on this review, relevant publications were

Figure. Appearance on Inspection for Clubbing



A, Normal finger viewed from above and in profile, and the changes occurring in established clubbing, viewed from above and in profile. B, The finger on the left demonstrates normal profile (ABC) and normal hyponychial (ABD) nail-fold angles of 169° and 183°, respectively. The clubbed finger on the right shows increased profile and hyponychial nail-fold angles of 191° and 203°, respectively. C, Distal phalangeal finger depth (DPD)/interphalangeal finger depth (IPD) represents the phalangeal depth ratio. In normal fingers, the IPD is greater than the DPD. In clubbing, this relationship is reversed. D, Schamroth sign: in the absence of clubbing, opposition of the index fingers nail-to-nail creates a diamond-shaped window (arrowhead). In clubbed fingers, the loss of the profile angle due to the increase in tissue at the nail bed causes obliteration of this space (arrowhead).

retrieved and their bibliographies were evaluated for additional material. We also examined standard textbooks of physical diagnosis for information on the physical examination for clubbing. We attempted to contact the authors of articles in which more than 1 observer made a determination of clubbing to obtain additional data about precision of the examination for clubbing. Studies selected for data extraction were those in which quantitative or qualitative assessment for clubbing was described in a series of patients. Although our expanded electronic search identified 567 articles related to clubbing, only 16 studies met the criteria for inclusion in our analysis.

Study Characteristics

Clubbing differs from other physical signs evaluated in the Rational Clinical Examination series in that the lack of an accepted objective diagnostic criterion standard precludes meaningful assessment of the accuracy of clinical examination. However, our review of the literature on clubbing permitted us to evaluate quantitative indices used to distinguish clubbed from normal fingers; precision of physicians' bedside clinical examination for clubbing; and accuracy of clubbing as a marker of selected diseases. We chose to limit our review of the quantitative indices of clubbing to studies of nail-fold angles and the phalangeal depth ratio, because of their potential applicability at the bedside.

Data Analysis

Pooled weighted averages were calculated for quantitative measurements of nail-fold angles and phalangeal depth ratios from data in studies of normal and diseased populations. Using data available in 2 articles on the precision of clubbing, we calculated κ statistics using the Stata statistical package (version 3.0, Computing Resource Center, Santa Monica, Calif). Sensitivities, specificities, and likelihood ratios of clubbing as a marker of specific underlying disease were calculated from original data when possible.

RESULTS

Quality of the Evidence

By consensus and using criteria previously developed for this series, we appraised the quality of the evidence contained in the articles that we retrieved.[29] For reasons of selection bias, small sample size, and lack of an independent, blind comparison of the physical sign with a criterion standard, we classified all of the included studies as level 4, leading to grade C recommendations.[29]

Quantitative Indices of Clubbing in Normal and Disease States

Using plaster casts, shadowgraphs, and calipers, nail-fold angles and the phalangeal depth ratio have been measured in normal populations and in subjects with diseases associated with clubbing. The precision of these quantitative techniques is high. Using the shadowgraph method, Kitis et al[30] examined the precision of measuring nail-fold angles. Duplicate measurements of 51 subjects showed a difference of 0.2° in the mean of both the hyponychial and profile angles, with SDs of 4.6° and 4.3°, respectively. Although Waring et al[15] found that the measurement of the phalangeal depth ratio with calipers on live fingers rather than plaster casts resulted in a loss of precision, Baughman et al[26] investigated intrarater reliability and found an SD of only 0.0008. In the same study, 2 observers independently measured the ratio in 20 subjects, and the maximal difference in phalangeal depth ratio was 0.03.

Published data pertaining to the measurement of nail-fold angles and phalangeal depth ratios in disease-free individuals are summarized in TABLE 2. The pooled weighted mean values for the profile and hyponychial angle are 167.2° and 179.0°, respectively. The pooled weighted mean phalangeal depth ratio is 0.900. Do these measurements help distinguish those with from those without clubbing? The range was available for only 45 of the 161 disease-free subjects in whom the profile angle was measured, and none exceeded 176°. In studies of hyponychial angles, none of the 171 disease-free subjects had angles above 192°. The phalangeal depth ratio has been reported in 359 disease-free subjects, and in only 1 did it exceed unity.

TABLE 3 shows the nail-fold angles and phalangeal depth ratios in patients with diseases associated with clubbing. In such chronic diseases as cystic fibrosis and cyanotic congenital heart disease, the nail-bed angles and the phalangeal depth ratios are significantly higher than those found in disease-free populations. In case series of asthma and COPD, phalangeal depth ratios are slightly higher than normal values. However, it is impossible to exclude the possibilities that these series may have included patients with other pulmonary disorders associated with clubbing or that some patients were selected because they had clubbing.

In summary, in disease-free subjects, a phalangeal depth ratio above 1 is rare, the profile angle does not exceed 176°, and the hyponychial angle does not exceed 192°. To facilitate clinical use, we suggest accepting values of less than 180° for the profile angle (a straight line) and less than 190° for the hyponychial angle as describing normality.

PRECISION AND ACCURACY

Precision of the Clinical Examination for Clubbing

Four studies[35-38] have reported the precision of physicians' bedside examination for clubbing (TABLE 4). Although several of the case series describing the prevalence of clubbing in various disease states used multiple examiners, none reported interrater reliability. We have excluded from this section reports of precision that used only casts or shadowgraphs for determination of precision, since potentially important clinical information from inspection or palpation of the live finger was not available to the examiners.

In an attempt to challenge the prevailing wisdom that clubbing was easily recognized, Pyke[35] studied the precision of physicians' global assessment for the sign. He enlisted 12 physicians and 4 medical students to examine 12 patients for the presence of clubbing. He purposefully chose pa-

tients who exhibited the full range of findings from normal to advanced clubbing. Overall agreement was fair ($\kappa = 0.39$). From the reported data, it was impossible to determine the effect of training on the examiners' precision, but it was clear that the examiners used different criteria to identify clubbing. After completing their assessments, Pyke asked the examiners to define clubbing, and he received a wide variety of answers.

Rice and Rowlands[36] used several quantitative indices, including phalangeal depth ratios, to assemble 11 patients who exhibited a range of findings from normal to advanced clubbing. Nineteen clinicians, all internal medicine staff or resident physicians, examined the patients for clubbing. Clubbing was judged to be present in 103 of the 209 subject examinations. As with Pyke's findings, observer agreement was only fair ($\kappa = 0.36$).

Precision of physical examination for a variety of signs of pulmonary disease, including clubbing, was evaluated in a study in which 24 experienced physicians examined 4 patients each.[37] The precision of the examination for clubbing was moderate ($\kappa = 0.45$). While several signs showed marginally greater precision (eg, wheezes, $\kappa = 0.51$), most signs had significantly lower precision (eg, displaced trachea, $\kappa = 0.01$; whispering pectoriloquy, $\kappa = 0.11$).

A 1965 study[38] contrasted other reports of the precision of the physical examination for clubbing. Of 21 pulmonary signs, clubbing exhibited the highest rate of interobserver agreement among 9 experienced physicians examining 20 patients ($\kappa = 0.90$).[39] This high level of precision may reflect either the experience of the examiners or a selection bias, since the degree of clubbing in affected patients was not described.

In summary, the precision of the clinical examination for clubbing has been found to be fair to moderate, with 1 study showing very high precision. Although precision was higher in the 2 studies that used more experienced examiners, neither of these studies reported their selection criteria for patients. Use of cases of more advanced clubbing may have led to an overestimation of precision.

Accuracy of Clubbing as a Marker of Disease States

Determination of the accuracy of clinical examination techniques to detect clubbing has been confounded by incorporation bias that results when the clinical examination itself forms part or all of the diagnostic criterion standard. One example of such confounding is illustrated by the digital index of Vasquez et al.[40] This index, the sum of the ratios of the distal phalangeal finger depth and interphalangeal depth circumferences in all 10 fingers, has been reported to have a high sensitivity and specificity for clubbing. However, the index was evaluated in patients with cyanotic congenital heart disease, whose clubbing was so marked that it was "obvious by simple inspection."[40] Only 1 study[36] measured the accuracy of clinicians' bedside examination for clubbing against a priori diagnostic criteria derived from quantitative indices in disease-free populations and those with disease. Unfortunately, data were not given in sufficient detail to allow calculation of the sensitivity and specificity of the clinical examination. Hence, data on the accuracy of clinical examination compared with the quantitative indices to detect clubbing are limited.

An alternative approach is to consider the accuracy of the presence of club-

Table 2. Reported Values for Profile Angle, Hyponychial Angle, and Phalangeal Depth Ratio in Disease-Free Subjects

	Technique	Population	No. of Subjects	Mean (SD)
Profile angle				
Bentley et al,[13] 1976	Shadowgraph	Healthy subjects from a surgical clinic (age not specified)	25	168.3° (3.7°)
Kitis et al,[30] 1979	Shadowgraph	Healthy hospital employees	116	166.3° (4.3°)
Sinniah and Omar,[31] 1979	Shadowgraph	Healthy children (source population not specified)	20	171.4° (5.5°)
Pooled weighted mean			161	167.2° (4.4°)
Hyponychial angle				
Regan et al,[12] 1967	Plaster casts, planimeter	Healthy manual workers	10	186.1° (1.97°)
Bentley et al,[13] 1976[13]	Shadowgraph	Healthy manual workers	25	180.1° (4.2°)
Kitis et al,[30] 1979	Shadowgraph	Healthy manual workers	116	177.9° (4.6°)
Sinniah and Omar,[31] 1979	Shadowgraph	Healthy manual workers	20	180.7° (5.2°)
Pooled weighted mean			171	179.0° (4.5°)
Phalangeal depth ratio				
Waring et al,[15] 1971	Plaster casts, micrometer	Children and adults (source population not specified)	160	0.895 (0.041)
Sly et al,[25] 1973	Plaster casts, micrometer	Adults (medical center personnel and relatives of patients attending pediatric allergy clinic)	60	0.903 (0.043)
Paton et al,[32] 1991	Plaster casts, micrometer	Children and adults (random sample from people playing in nearby park)	85	0.890 (0.040)
Baughman et al,[26] 1998	Live fingers, calipers	Adults (medical center personnel)	54	0.920 (0.050)
Pooled weighted mean			359	0.900 (0.042)

Table 3. Reported Values for Quantitative Measures of Clubbing in Disease States*

	No. of Subjects	Technique	Quantitative Measure	Mean (SD)
Asthma				
Waring et al,[15] 1971	45	Plaster casts, micrometer	DPD/IPD ratio	39/45 <1.0†
Sly et al,[25] 1973	119	Plaster casts, micrometer	DPD/IPD ratio	0.910 (0.050)
Bentley et al,[13] 1976	25	Shadowgraph	Profile angle; hyponychial angle	170.9° (4.1°); 185.4° (6.4°)
Paton et al,[32] 1991	20	Plaster casts, micrometer	DPD/IPD ratio	0.911 (0.046)
Chronic obstructive pulmonary disease				
Baughman et al,[26] 1998	54	Live fingers, calipers	DPD/IPD ratio†	0.94 (0.06)
Bronchogenic carcinoma				
Baughman et al,[26] 1998	109	Live fingers, calipers	DPD/IPD ratio‡	0.975 (0.099)
Cystic fibrosis				
Waring et al,[15] 1971	45	Plaster casts, micrometer	DPD/IPD ratio	38/45 >1.0†
Bentley et al,[13] 1976	50	Shadowgraph	Profile angle; hyponychial angle	179.0° (6.2°); 194.8° (8.3°)
Lemen et al,[33] 1978	18	Plaster casts	DPD/IPD ratio§	1.010 (0.016)
Pitts-Tucker et al,[34] 1986	73	Shadowgraph	Hyponychial angle	192°
Paton et al,[32] 1991	44	Plaster casts, micrometer	DPD/IPD ratio	1.033 (0.079)
Cyanotic congenital heart disease				
Waring et al,[15] 1971	27	Plaster casts, micrometer	DPD/IPD ratio	18/27 >1.0†
Bentley et al,[13] 1976	25	Shadowgraph	Profile angle; hyponychial angle	179.7° (4.8°); 195.5° (2.5°)
Asbestos exposure				
Regan et al,[12] 1967	50	Plaster casts, planimeter	Hyponychial angle	195.0° (9.6°)
Crohn disease				
Kitis et al,[30] 1979	200	Shadowgraph	Hyponychial angle	183.5° (7.8°)

*DPD/IPD indicates distal phalangeal depth/interphalangeal depth.
†Individual values not reported; proportion of patients with DPD:IPD of greater than 1.0 reported.
‡Value reported in table is for right index finger only.
§Pooled weighted average for right index finger only.

Table 4. Interobserver Agreement of Clinical Examination for Clubbing

Source, y	No. of Observers	Observer's Level of Experience	κ
Pyke,[35] 1954	16	4 Medical students 4 Medical registrars 4 Surgical registrars 4 Senior physicians	0.39
Rice and Rowlands,[36] 1961	19	Residents Fellows Staff physicians	0.36
Smyllie et al,[38] 1965	9	5 Medical registrars 4 Consultant physicians	0.90
Spiteri et al,[37] 1988	24	2 Senior house officers 14 Medical registrars 8 Consultant physicians	0.45

bing as a marker of underlying disease. Because many patients with clubbing have pulmonary disease, a relevant clinical question is whether clubbing separates those with COPD from those who have clubbing associated with pulmonary malignancy. In this way, 1 study[26] assessed the usefulness of the phalangeal depth ratio in distinguishing patients with documented lung cancer from control subjects and those with COPD. Using calipers, Baughman et al[26] measured the phalangeal depth ratio in both right and left index fingers in 109 patients with known lung cancer, 55 patients with COPD, and 54 control subjects. Of the 54 control subjects, none had a phalangeal depth ratio in excess of 1. In those patients who had a phalangeal depth ratio greater than 1, 40 had lung cancer and 5 had COPD alone (likelihood ratio, 3.9 [95% confidence interval {CI}, 1.6-9.4]). Seventy patients who had a phalangeal depth ratio of 1 or less had lung cancer, and 49 with the same depth ratio had COPD alone (likelihood ratio, 0.7 [95% CI, 0.6-0.8]). We reclassified 1 subject in the COPD group who had a pulmonary nodule detected on chest radiography at study entry, which was subsequently diagnosed as adenocarcinoma of the lung.

These data confirm, as expected, that while a normal phalangeal depth ratio does not rule out lung cancer, an abnormal ratio implies an increased probability (likelihood ratio, 3.9; 95% CI, 1.6-9.4) of underlying lung cancer. Only 3 of the patients with COPD had a phalangeal depth ratio greater than 1.05, and none had a ratio greater than 1.1. Among those with lung cancer, there was no significant difference in the prevalence of clubbing (as defined by distal phalangeal finger depth/interphalangeal finger depth ratio >1) among the different histologic subtypes of lung cancer.

Kitis et al[30] investigated the association of clubbing with the activity of inflammatory bowel disease in 327 patients. Clubbing was defined as a shadowgraph-measured hyponychial angle greater than 186°, which corresponded to 1.65 SDs above the mean value found in a group of 116 healthy controls. Disease activity was determined using an index incorporating the

results of various laboratory investigations. The likelihood ratios for clubbing as a marker of active Crohn disease were 2.8 (95% CI, 1.8-4.1) and 3.7 (95% CI, 1.4-9.4) for ulcerative colitis. The sensitivity and specificity values were 0.58 and 0.79 for Crohn disease vs 0.30 and 0.92 for ulcerative colitis, respectively.

SCENARIO RESOLUTION

In the first case, you find that the patient appears to have increased nail curvature. You use calipers to estimate a phalangeal depth ratio of 0.90, and on inspection you estimate a profile angle of about 160°. Based on your knowledge of these values in disease-free subjects, you inform the respiratory therapist that her mother does not have clubbing. On the other hand, you find that the second patient has a phalangeal depth ratio of 1.1 and a profile angle of 180°, findings that are quite unusual for disease-free subjects or patients with COPD alone. You conclude that a search for bronchogenic carcinoma (or other causes of clubbing) should be undertaken.

THE BOTTOM LINE

For generations, medical students and residents have been quizzed at the bedside about the diagnostic features of clubbing. Confident though their inquisitors may be in their own ability to detect clubbing, the literature shows that interobserver agreement is only fair to moderate, and that the accuracy of techniques to detect clubbing has not been well established. Nevertheless, since nonhereditary clubbing is almost always a portent of serious disease, clinicians need to be as certain as possible about its presence.

Recognizing the limitations of the studies we have appraised, we recommend the following:

- In cases of diagnostic uncertainty, the phalangeal depth ratio may be helpful. This ratio can be measured using calipers at the bedside, and in disease-free populations rarely exceeds 1.0. An elevated ratio should prompt a search for underlying disease. Although patients with COPD have slightly higher ratios than do disease-free subjects, it is unusual for the ratio to exceed 1.05. A value in excess of this in a patient with COPD should prompt a search for bronchogenic carcinoma. Because most clinicians do not have calipers, visual estimation of reversal of the phalangeal depth ratio should be assessed.
- Although the accuracy of clinicians' bedside estimation of nail-fold angles has not been studied, the normal values for these angles have been established. A profile angle that approaches a straight line (180°) is rare in disease-free subjects, and in our opinion is easily identifiable at the bedside. Although the normal range of the hyponychial angle has also been defined, this angle is more difficult to estimate at the bedside.
- No published evidence exists as to the diagnostic yield or the optimal strategy for investigating a patient with clubbing. Therefore, after completion of a thorough medical history and physical examination, clinical judgment must guide the choice of investigations.

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REFERENCES

1. West S. Two cases of clubbing of the fingers developing within a fortnight and four weeks respectively. *Trans Clin Soc London.* 1897;30:60.
2. Adams F, trans. *The Genuine Works of Hippocrates, 1:206.* New York, NY: Wm Wood & Co; 1891.
3. Horsfall FL. Congenital familial clubbing of the fingers and toes. *CMAJ.* 1936;34:145-149.
4. Buchman D, Hrowat EA. Idiopathic clubbing and hypertrophic osteoarthropathy. *Arch Intern Med.* 1955; 97:355-358.
5. Mendlowitz M. Clubbing and hypertrophic osteoarthropathy. *Medicine (Baltimore).* 1942;21:269-306.
6. Alvarez AS, McNair D, Wildman J, Hewson JW. Unilateral clubbing of the fingernails in patients with hemiplegia. *Gerontologia Clinica.* 1975;17:1-6.
7. Marie P. De l'ostéo-arthropathie hypertrophiante pneumique. *Rev Med.* 1890;10:1.
8. Bamberger E. Über knochevneränderungen bei chronischen lungen und herzkrankheiten. *Atschr Klin Med.* 1891;18:193.
9. Coury C. Hippocratic fingers and hypertrophic osteoarthropathy: a study of 350 cases. *Br J Chest.* 1960; 54:202-209.
10. Cudowitz L, Wraith DG. An evaluation of the clinical significance of clubbing in common lung disorders. *Br J Tuberc Dis Chest.* 1957;51:14-21.
11. Stavem P. Instrument for estimation of clubbing. *Lancet.* 1959;2:7-8.
12. Regan GM, Tagg B, Thompson ML. Subjective assessment and objective measurement of finger clubbing. *Lancet.* 1967;1:530-532.
13. Bentley D, Moore A, Swachman H. Finger clubbing: a quantitative survey by analysis of the shadowgraph. *Lancet.* 1976;2:164-167.
14. Mellins RB, Fishman AP. Digital casts for the study of clubbing of the fingers. *Circulation.* 1966;33:143-145.
15. Waring WW, Wilkinson RW, Wiebe RA, Faol BC, Hilman BC. Quantitation of digital clubbing in children. *Am Rev Respir Dis.* 1971;104:166-174.
16. Bigler FC. The morphology of clubbing. *Am J Pathol.* 1958;34:237-261.
17. Dickinson CJ. The aetiology of clubbing and hypertrophic osteoarthropathy. *Eur J Clin Invest.* 1993; 23:330-338.
18. Racoceanu SN, Mendlowitz M, Suck AF. Digital capillary blood flow in clubbing. *Ann Intern Med.* 1971; 75:933-935.
19. Martinez-Lavin M. Digital clubbing and hypertrophic osteoarthropathy. *J Rheumatol.* 1987;14:6-8.
20. Onadeko BO, Kolawolw TM. The clinical and aetiological pattern of finger clubbing and hypertrophic osteoarthropathy in Nigerians. *Trop Geogr Med.* 1979;31:191-199.
21. Hansen-Flaschen J, Nordberg J. Clubbing and hypertrophic osteoarthropathy. *Clin Chest Med.* 1987; 8:287-298.
22. Rabin CB. New or neglected physical signs in diagnosis of chest diseases. *JAMA.* 1965;194:546-550.
23. Lovibond JL. Diagnosis of clubbed fingers. *Lancet.* 1938;1:363-364.
24. Carroll DG. Curvature of the nails, clubbing of the fingers and hypertrophic osteoarthropathy. *Trans Am Clin Climatol Assoc.* 1972;83:198-208.
25. Sly RM, Ghazanshahi S, Buranakul B, et al. Objective assessment for digital clubbing in Caucasian, Negro and Oriental subjects. *Chest.* 1973;64:687-689.
26. Baughman RP, Gunther KL, Buchsbaum JA, Lower EE. Prevalence of digital clubbing in bronchogenic carcinoma by a new digital index. *Clin Exp Rheumatol.* 1998;16:21-26.
27. Schamroth L. Personal experience. *S Afr Med J.* 1976;50:297-300.
28. Lampe RM, Kagan A. Detection of clubbing—Schamroth's sign. *Clin Pediatr (Phila).* 1983;22:125.
29. Holleman DR, Simel DL. Does the clinical examination predict airflow limitation? *JAMA.* 1995;273:313-319.
30. Kitis G, Thompson H, Allan RN. Finger clubbing in inflammatory bowel disease. *BMJ.* 1979;2:825-828.
31. Sinniah D, Omar A. Quantitation of digital clubbing by shadowgram technique. *AJDC.* 1979;54:145-146.
32. Paton JY, Bautista DB, Stabile MW, et al. Digital clubbing and pulmonary function abnormalities in children with lung disease. *Pediatr Pulmonol.* 1991;10:25-29.
33. Lemen RJ, Gates AJ, Mathe AA, et al. Relationship among digital clubbing, disease severity and serum prostaglandin F2 and E concentrations in cystic fibrosis patients. *Am Rev Respir Dis.* 1978;117:639-646.
34. Pitts-Tucker TJ, Miller MG, Littlewood JM. Finger clubbing in cystic fibrosis. *AJDC.* 1986;61:576-579.
35. Pyke DA. Finger clubbing. *Lancet.* 1954;2:352-354.
36. Rice RE, Rowlands PW. *A Quantitative Method for the Estimation of Clubbing* [thesis]. New Orleans, La: Tulane University Medical School; 1961.
37. Spiteri MA, Cook DG, Clarke SW. Reliability of eliciting physical signs in examination of the chest. *Lancet.* 1988;1:873-875.
38. Smyllie HC, Blendis LM, Armitage P. Observer disagreement in physical signs of the respiratory system. *Lancet.* 1965;2:412-413.
39. Armitage P, Blendis LM, Smyllie HC. The measurement of observer disagreement in the recording of signs. *J R Stat Soc A.* 1966;129:98-109.
40. Vazquez-Abad D, Pineda C, Martinez-Lavin M. Digital clubbing. *J Rheumatol.* 1989;16:518-520.