**Expert Review** Examination for Finger Clubbing

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Abstract: Examination for the presence of finger clubbing is an important skill for medical students and doctors. This article describes a comprehensive, concise and evidence-based approach for assessing for the presence of finger clubbing which is consistent with The Principles of Clinical Examination[1]. We describe the signs seen in finger clubbing and, based on a review of the literature, the precision and accuracy of these signs is discussed.

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Introduction

Finger clubbing is a deformity of the fingers in which focal and bulbous swelling of the distal phalanges is accompanied by changes to the angles of the nail bed. This important clinical sign is associated with a number of diseases involving multiple organ systems (Table 1). First described by Hippocrates in 400BC, the underlying pathophysiology and clinical significance of finger clubbing are still subject to debate, as is the best way to examine the fingers. This article presents the available information on the causes, examination and relevance of one of medicine’s oldest clinical signs.

Literature search

The MEDLINE database was searched using PubMed with the Medical Subject Headings (MeSH) ‘Hypertrophic Osteoarthropathy’, ‘clubbed fingers’ and ‘Bamberger-Marie Disease’, as well as the non-MeSH search terms ‘finger clubbing’ and ‘digital clubbing’.

Clinical prediction rules were searched for clubbing of the fingers, although none were found. The Rational Clinical Examination Series of the Journal of the American Medical Association[2], clinicalevidence.com[3] and Evidence Based Medicine Online[4] we also searched with the same terms.

Selection criteria were relevant papers written in English and available through University of Bristol library services or South West Information Management System.

What causes finger clubbing?

The pathophysiology behind finger clubbing is not completely understood but the ‘platelet hypothesis’ [5] offers the most complete explanation. Platelets are derived from megakaryocytes which differentiate from haematopoietic stem cells in the bone marrow and enter into the venous circulation. Under normal physiological circumstances megakaryocytes enter the right side of the heart and subsequently the pulmonary circulation. Upon entering the pulmonary capillary bed, megakaryocytes are thought to fragment into platelets as they pass through the small calibre vessels.[6]

The role of megakaryocytes and platelets:

Platelet clusters and megakaryocyte fragments can be found in the distal digital vessels of clubbed samples but not in controls.[7] Certain pathological changes must therefore occur to allow platelet clusters or whole megakaryocytes to enter the systemic circulation. Being relatively large, they tend to stream axially which distributes them distally in the limbs and eventually they become impacted in the finger tips.[5] There are several possible explanations for how this may occur. A right to left intra-cardiac shunt would probably allow the greatest number of Megakaryocytes to remain intact within the systemic circulation, and indeed we do see a strong association with intra-cardiac shunts and finger clubbing. Inflammatory processes within the lungs, such as occur in the presence of neoplasm, might also produce less fragmentation of
megakaryocytes due to dilatation of the blood vessels. Other disorders are accompanied by chronic platelet excess, such as inflammatory bowel disease,[8] and some promote the formation of platelet clumps in the left side of the heart or systemic circulation, such as infective endocarditis or a subclavian aneurysm. All of these conditions also have known associations with finger clubbing. Liver disease is sometimes accompanied by pulmonary arteriovenous malformations, which might explain why clubbing is also sometimes associated with cirrhosis of the liver.

*The role of VEGF and PDGF:*

Megakaryocyte and platelet clumps impacted in the digital vessels release Platelet Derived Growth Factor (PDGF) and vascular endothelial growth factor (VEGF). Both PDGF and VEGF are hypoxically regulated. The hypoxic environment created when the capillaries become occluded further promotes VEGF and PDGF release from vascular endothelial cells, along with a number of other Hypoxia Inducible Factors (HIFs).[7] PDGF promotes growth, vascular permeability and chemotaxis, explaining the increased vascular smooth muscle cells and fibroblasts seen in clubbed fingers.[7] VEGF promotes neovascularisation and connective tissue changes, and is also known to increase vascular permeability leading to oedema.

There are a number of reports that the changes seen in finger clubbing may be at least partially reversible if treatment for the underlying cause is delivered early enough, for example in cystic fibrosis patients who have undergone lung transplantation.[9] However, longstanding finger clubbing is likely to be accompanied by a greater degree of chronic tissue changes and is therefore less likely to be completely reversible.

The platelet hypothesis is the most useful model we have for the pathophysiology of clubbing, however some questions remain unanswered. For example, unilateral clubbing may be seen in association with hemiplegia, and this remains unexplained.[10] Furthermore, there have been studies which show no vascular changes in clubbed fingers,[11] suggesting that in at least some cases there may be an alternative mechanism by which clubbing can arise.

*The clinical significance of finger clubbing*

Textbooks on clinical examination typically contain a brief list of diseases associated with clubbing. This list varies slightly from book to book, with little information on the likelihood of developing clubbing with any particular disease. Table 1 presents the current data on this subject based on an original literature search by Spicknall et al (2005),[8] with some additions. The sample size, research methodology and method used to assess clubbing status varied greatly between studies but rough comparisons can still be made.

Notably Martinez-Lavin (1982) reports that 100% of patients with congenital cyanotic heart disease develop finger clubbing.[12] This is a striking claim, and if true greatly supports the platelet hypothesis. There are also case reports of Systemic Lupus Erythematosus, CINCA syndrome and POEMS syndrome all presenting with associated clubbing.[8] Interestingly, when Hippocrates first described clubbing it was in a patient with what we assume was empyema, yet seemingly there are no recent reports in the literature of empyema-associated clubbing. Perhaps Hippocrates’ patient had other pathology as well.

*CINCA Syndrome:* An autoimmune inflammatory disease characterised by fever, rash, arthritic changes, eye problems and chronic meningitis.

*POEMS Syndrome:* A rare syndrome of Polyneuropathy, Organomegaly, Endocrinopathy/oEdema, M-protein (abnormal antibody) presence and Skin changes.

Unilateral finger clubbing:

Unilateral clubbing has also been reported, and this may be associated with ipsilateral hemiplegia. However, of 108 hemiplegic patients studied only two demonstrated unilateral clubbing, therefore the association is small.[10] Vascular pathology may also cause unilateral clubbing. For example there are case reports of unilateral clubbing associated with a dialysis fistula,[13] arteritis of the aorta or large arteries,[14] and an ulna artery aneurysm.[15]

*Hypertrophic osteoarthropathy:*

When investigating clubbing we must ascertain whether the patient has any other symptoms of Hypertrophic Osteoarthropathy (HOA), a syndrome in which clubbing is accompanied by arthralgia and periostial proliferation of the tubular bones. This may be primary (hereditary) or secondary to advanced disease states.[16] If any of these symptoms are present, does the patient have a family history to suggest primary HOA? HOA and advanced clubbing are generally considered to be markers of greater disease severity and increased risk of mortality.[8] If the presentation is likely secondary to underlying disease, rapid assessment for malignancy should be undertaken.
Idiopathic finger clubbing:
A proportion of finger clubbing is believed to be idiopathic. A Belgian study looked at all patients admitted to a general internal medicine ward over one year. They found that approximately 1% demonstrated signs of finger clubbing. Of those patients with finger clubbing, 40% were eventually found to have serious underlying disease over the one year follow up period. In the remaining 60% no indications of disease were found. It is therefore possible that as much as 60% of finger clubbing may be idiopathic, but we cannot be certain from this study alone since disease may simply not have been identified in the follow up period.[17]

Given the current gaps in our knowledge, how effectively can we use the discovery of finger clubbing to guide us towards a particular diagnosis? Figure 1 presents an algorithm suggested by Spicknall et al [8] to guide medical students in their assessment of a patient with finger clubbing.

Clubbing of the toe nails:
In some cases finger clubbing is accompanied by clubbing of the toes. Examination of the toes for evidence of clubbing is not routine and is beyond the scope of this article.

Examination of the fingers for signs of clubbing
This article describes how to assess a patient for the presence of finger clubbing. This is not usually done in isolation but as part of a wider examination.

Preparation
Introduce yourself to the patient, confirm their identity, obtain verbal consent and wash your hands. Sit opposite the patient with their wrists and hands exposed and placed, palms down, resting on a pillow in front of them.

Inspection
Look at the patient’s hands. Advanced finger clubbing may be immediately obvious with the terminal phalanges taking on an enlarged, bulbous appearance sometime referred to as ‘drumstick fingers’. Diagnosis of more subtle finger clubbing requires detailed examination (see Box 1 for the developmental stages of finger clubbing).

One at a time, lift the patient’s hands to eye level and view the index finger from the side. There are three parameters to consider from this position: the profile angle, the hyponychial angle and the phalangeal depth ratio. The profile angle is the angle between the proximal nail fold and the nail plate itself (Figures 2, 3A-B, and 4). A profile angle of greater than 180° is considered abnormal. The hyponychial angle is the angle between the proximal nail fold and the hyponychium (Figures 3C-D and 4). A hyponychial angle of greater than 190° is considered abnormal. If these parameters are both increased then the patient has early clubbing of the fingers.
Next assess the phalangeal depth ratio (PDR) which is a sign of more advanced finger clubbing. The PDR is the distal phalangeal depth (DPD) divided by the interphalangeal depth (IPD). The DPD is the depth of the terminal phalanx at the proximal nail fold and the IPD is the same measurement repeated at the distal interphalangeal joint (Figures 2 and 3E-F). These parameters can be assessed by visual estimate or measured using callipers. If used, callipers should touch but not compress the tissue, accurate measurements of the DPD and IPD should be taken and the ratio of DPD/IPD calculated. A value of less than 1 is used to define normality and a PDR greater than 1 therefore indicates the presence of finger clubbing. The use of callipers allows sequential recording of an accurate measurement, permitting the progress of suspected clubbing to be monitored if desired. This may be useful to determine if a patient has finger clubbing or simply unusual nail anatomy, since the natural history of finger clubbing is for it to progress, whereas non-pathological anatomical variation outside of the ‘normal’ range would not be expected to get worse over time.

In practice callipers are often unavailable and so visual estimation of the PDR may be preferable. This is achieved by evaluating whether the IPD is greater than the DPD, in which case the finger is normal. If the DPD is greater than the IPD then the PDR will be greater than 1 and therefore outside of the normal range. If the PDR is outside of the normal range and the profile and hyponychial angles are also abnormal, the finger is considered to be clubbed.

Assessment of profile angle, hyponychial angle and PDR can then be repeated in the other digits including the thumbs if desired, however it should be noted that the normal ranges for the profile angle, hyponychial angle and PDR are calculated on the basis of studies of the index fingers alone, or studies which do not specify which digits were assessed. Examination of other digits may therefore be useful for comparison, but the evidence base for the normal anatomical parameters is limited. The results of 11 studies into the normal anatomical parameters of the fingers are presented in table 2.

Figure 3 The profile angles in A) a normal finger and B) a clubbed finger. The hyponychial angles in C) a normal finger and D) a clubbed finger. Measurement of the distal phalangeal and interphalangeal depth in E) a normal finger and F) a clubbed finger.

Figure 4 The profile and hyponychial angles in a non-clubbed finger (ABC: profile angle, ABD: hyponychial angle)

These measurements are difficult to perform accurately without the use of shadowgraphs or plaster casts which are not used routinely in clinical practice. Gross abnormalities should be noted and
equivocal results interpreted in combination with other clinical tests. According to a recent JAMA review, when uncertain about a diagnosis of clubbing we should assess both the profile angle and the PDR. If these are both abnormal we should investigate for underlying disease.[23]

Figure 5 Palpation of the nail bed of the index finger

Palpation
Next palpate the nail bed of the distal phalanx of the patient’s index finger. Palpate between your own thumb and index finger, with the tip of the thumb placed directly over the proximal nail fold and the index finger supporting the digit from beneath (Figure 5). The thumb is then used to palpate the nail bed. Assess if the nail bed has a normal consistency or if you think it feels softer than normal. This is obviously very subjective but it is an important sign (Box 1). Repeat with the index finger on the patient’s other hand.

Special Tests: Schamroth’s test
The patient is requested to make a loose fist with both hands and then raise their hands to approximately mid-chest height with the dorsal surface of the hands facing upwards. They should hold their fists approximately 30cm away from their chest and, keeping their remaining fingers flexed, partially extend the index finger on each hand such that the dorsal surfaces of the terminal phalanges on each index finger can be brought together and opposed. (Figure 6) With the patient in this position, look at the opposed terminal phalanges from the ulnar side (the front of the patient). In non-clubbed fingers a diamond gap termed ‘Schamroth’s window’ is observed, made up of the nail and skin overlying the opposed terminal phalanges with the profile angle making up the apex on either side. The loss of Schamroth’s window implies a profile angle of $180^\circ$ or greater, which is suggestive of finger clubbing.

Positioning is important for this test to be effective – the hands must be far enough away from the chest that adequate light can pass through and the window can be seen clearly if present, and the terminal phalanges must be appropriately opposed so that the window is occluded if the nails are clubbed. Demonstration of the required position is highly recommended. It may also be necessary to gently guide the patient’s hands and fingers to the correct position.

Completing the Examination
Finally, wash your hands and thank the patient for their cooperation.
References
Figure 1 Algorithm for the assessment of bilateral finger clubbing suggested by Spicknall et al[6]
### Table 1
The association of diseases with bilateral finger clubbing and the evidence to support this association. Adapted from information presented in Spicknall et al (2005) unless otherwise referenced.

<table>
<thead>
<tr>
<th>Diseases associated with clubbing</th>
<th>No. Subjects studied</th>
<th>% patients with clubbing</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.F.</td>
<td>61</td>
<td>64</td>
</tr>
<tr>
<td>Hypersensitivity pneumonitis</td>
<td>82</td>
<td>54</td>
</tr>
<tr>
<td>Interstitial pulmonary fibrosis</td>
<td>588</td>
<td>49</td>
</tr>
<tr>
<td>Asbestosis</td>
<td>167</td>
<td>43</td>
</tr>
<tr>
<td>Bronchial carcinoma (usually not small cell)</td>
<td>111</td>
<td>28</td>
</tr>
<tr>
<td>T.B.</td>
<td>426</td>
<td>15</td>
</tr>
<tr>
<td>Sarcoïdosis</td>
<td>90</td>
<td>12</td>
</tr>
<tr>
<td>Mesothelioma</td>
<td>90</td>
<td>7</td>
</tr>
<tr>
<td>I.B.D.</td>
<td>200</td>
<td>38</td>
</tr>
<tr>
<td>Liver cirrhosis</td>
<td>74</td>
<td>24</td>
</tr>
<tr>
<td>G.I. lymphoma</td>
<td>40</td>
<td>15</td>
</tr>
<tr>
<td>Malabsorption e.g. celiac</td>
<td>42</td>
<td>14</td>
</tr>
<tr>
<td>Cyanotic congenital heart disease</td>
<td>32</td>
<td>100</td>
</tr>
<tr>
<td>Endocarditis [24]</td>
<td>402</td>
<td>7</td>
</tr>
<tr>
<td>Atrial myxoma [25]</td>
<td>1 case</td>
<td></td>
</tr>
<tr>
<td>Thyroid acropachy</td>
<td>178</td>
<td>20</td>
</tr>
<tr>
<td>H.I.V.</td>
<td>155</td>
<td>6</td>
</tr>
<tr>
<td>Laxative abuse</td>
<td>2 cases</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2
The results of 11 studies into the normal anatomical parameters the finger nail [23]

<table>
<thead>
<tr>
<th>Anatomical feature</th>
<th>No. studies</th>
<th>No. subjects</th>
<th>Pooled weighted mean measurement of anatomical feature (SD)</th>
<th>Maximum limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Profile angle</td>
<td>3</td>
<td>116</td>
<td>167.2° (5.5)</td>
<td>None &gt;176°</td>
</tr>
<tr>
<td>Hyponychial angle</td>
<td>4</td>
<td>171</td>
<td>179° (4.5)</td>
<td>None &gt;192°</td>
</tr>
<tr>
<td>PDR</td>
<td>4</td>
<td>359</td>
<td>0.9 (0.042)</td>
<td>&gt;1 in only 1/359 subjects.</td>
</tr>
</tbody>
</table>
Box 1 The developmental stages of finger clubbing.[ii]

1) Periungual erythema and softening of the nail bed develops
2) The profile and hyponychial angles increase
3) The nail begins to develop convexity as it grows
4) The nail develops longitudinal ridging and the periungual skin begins to appear shiny.
5) The depth of the distal phalange increases and the distal interphalangeal joint may become hyperextensible.

This process usually takes years but clubbing can also develop subacutely