A 42-year-old man with chronic kidney disease and a history of childhood repair of Tetralogy of Fallot was admitted with pneumonia. Examination of his extremities revealed clubbing of his fingers (Figure 1) and toes (Figure 2).

Clubbing may be primary, known as pachydermoperiostosis, or secondary, due to a variety of neoplastic, pulmonary, cardiac, gastrointestinal, and infectious diseases. Examination reveals softening of the nail bed with loss of the normal angle between the nail and the proximal nail fold, an increase in the nail fold convexity, and thickening of the distal phalange with eventual hyperextensibility of the distal interphalangeal joint. Diagnosis is based on various criteria, such as the profile angle (Lovibond’s angle) or distal phalangeal to interphalangeal depth ratio. The loss of the normal diamond-shaped window created by placing the back surfaces of terminal phalanges of similar fingers together, also known as Schamroth’s sign, was noted by Dr. Leo Schamroth when he developed endocarditis and is one of the few eponyms named after both a physician and the patient in whom it was found (Figure 3). Recent literature suggests that vascular endothelial growth factor (VEGF), a platelet-derived factor induced by hypoxia, may play a role in digital clubbing. Processes that alter normal pulmonary circulation disrupt fragmentation of megakaryocytes in the lung into platelets. Consequently, whole megakaryocytes enter the systemic circulation and become impacted in the peripheral capillaries, where they cause stromal hypoxia and release of platelet-derived growth factor and VEGF, leading to the vascular hyperplasia that underlies clubbing.

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