This hypothesis is supported by the observation that the left upper arm fits perfectly into the chest deformity with the abrasion corresponding to the position of the left elbow. The abrasion developed into a hypopigmented macule.

The chest wall depression is small compared with the shift in cardiac position. It is possible that at some time during intrauterine development, the depression of the chest wall was relatively much greater. The chest wall abnormality may somehow have interfered with the normal leftward migration of the cardiac apex that follows the initial rightward bending of the primitive cardiac loop.

The term dextrocardia simply means that the heart occupies the right side of the thorax. Although there is no unanimity of opinion about the nomenclature for various types of dextrocardia, the terms dextroposition, dextrorotation, pivotal dextrocardia, isolated dextrocardia, and secondary dextrocardia are each generally used to describe a right-sided heart without positional abnormalities of other organs. Dextroposition implies a rightward shifting or translation of the heart by external forces. Eventration of the left hemidiaphragm, congenital lobar emphysema, pneumothorax, pleural effusions, chest wall deformities, and hypoplasia or collapse of the right lung are examples of external forces that can result in the heart occupying the right hemithorax. When dextrocardia is due to anomalies of the lungs, diaphragm, or chest wall, the heart is usually intrinsically normal.

Key words: clubbing; lung transplantation; pulmonary fibrosis

Digital clubbing (DC) occurs in association with various lung diseases, but its pathogenesis and its clinical significance remain uncertain. However, it is generally accepted that the degree of acquired clubbing may vary and that it might regress following the resolution of the associated illness; this is often observed when the underlying disorder is a malignant neoplasm that has been partially or totally removed. However, this is not the case for idiopathic pulmonary fibrosis, a common cause of clubbing, in which by being a progressive and lethal disease in the short term, it is unusual to observe the regression of DC. In this communication, we report a case of reversible clubbing after lung transplantation in a subject with pulmonary fibrosis.

**Case Report**

A 51-year-old man was admitted to our hospital in April 1988 because of shortness of breath during mild exercise and cough. Physical examination revealed resting cyanosis, bibasilar crackles, and digital clubbing. The chest radiographs showed a predominant reticulonodular pattern with honeycombing. Pulmonary function tests revealed a severe restrictive disease with hypoxemia at rest worsening during exercise. The patient was submitted to open lung biopsy and the morphologic study results exhibited a pattern compatible with usual interstitial pneumonia and according to clinical, radiographic, functional, and histologic characteristics, the diagnosis of idiopathic pulmonary fibrosis was established and a course of prednisone (1 mg/kg/d) plus oxygen therapy was started. The prednisone dose was gradually tapered during the following months. Despite treatment, however, clinical and functional conditions worsened progressively, and because his expectancy of life was assumed to be very short, a lung transplantation was planned. The patient underwent a successful unilateral lung graft in January 1989.

To analyze DC, molds of the right index digit were made with an irreversible hydrocolloid material (Jeltrate) before, and 1, 2, and 3 years after the lung transplantation. The ratio of the distal phalangeal depth and the interphalangeal depth (DPD/IPD ratio) was measured by using a vernier caliper, and this was used as an indicator of the degree of clubbing. Additionally, the hypochondrial and profile angles were obtained as reported by Regan et al. For further explanations of the construction of the DPD/IPD ratio and angles, see Figure 1A.

To know the interobserver variability of the measurements, the hypochondrial and profile angles as well as the DPD/IPD ratio were measured by two different observers during 10 different days. The concordance of their measurements was evaluated with the intraclass correlation coefficient. The intraobserver variability was obtained by meaning the ten measurements made by each

**References**


**Reversal of Digital Clubbing After Lung Transplantation**

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Digital clubbing is a common sign in a variety of lung diseases. Although its pathogenesis remains unclear, it is known that the degree of clubbing may vary and even disappear, particularly when the underlying dis-

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DC=digital clubbing; DPD/IPD ratio=ratio of distal phalangeal depth and interphalangeal depth; P(A-a)O2=alveolar-arterial oxygen pressure difference

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The photographs of the four fingers, the respective way of measuring the DPD/IPD ratio, and the hyponychial and profile angles are shown in Figure 1A, B. The concordance (intraclass correlation coefficient) of the observers for each of the four fingers was higher than 0.9 for the DPD/IPD ratio and higher than 0.84 for both the hyponychial and the profile angles. The intraobserver variability was lower than 5% for all the fingers. We observed a statistically significant decrease in clubbing by all methods (p<0.001). The DPD/IPD ratio decreased from 1.155 to 1.048, 1.025 and 1.020, the hyponychial angle from 193 to 188, 186 and 178, and the profile angle from 226 to 208, 206 and 205 (for the first, second, and third year, respectively). Lung mechanics and gas exchange improved considerably. It is particularly appealing to note the improvement of the PaO2 (from 45 to 59 mm Hg) and the alveolo-arterial Po2 gradient (from 28 to 11 mm Hg), as well as the remarkable increase of FVC (percent of predicted) that showed an inverse correlation with the annual decrease of clubbing (Fig 2). No skeletal changes compatible with periostosis were found in the radiographs of the bones at any time.

**DISCUSSION**

A significant decrease of the degree of clubbing was recorded in this patient after lung transplantation. The variation in observer and the concordance interobserver for the DPD/IPD ratio, profile, and hyponychial angles were acceptable. In addition, the casts of the fingers allowed us to keep the contour of the digits without changes in the time.

Many theories have been postulated to explain clubbing of the fingers, most of them suggesting a circulating bioactive substance which, by the underlying pathologic disorder, is not inactivated in the lungs. A current hypothesis by Dickinson and Martin suggests that the impact of the megakaryocytes and platelet clumps in the arterial blood would be greater in the fingertips because large particles tend to be carried in the axial stream. Such impact should release platelet-derived growth factor, a potent growth

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**Figure 1.** A (top), Construction of the DPD/IPD ratio, and the profile and hyponychial angles. The angles were made with lines joining the points A, B, C and D. Point A is at the distal digital crease on the dorsal surface. Point B is at the cuticle. Point C is on the nail, about one third of the axial distance from the cuticle to fingertip. Point D is at the hyponychium. The hyponychium is the thickened stratum corneum of epidermis under the free edge of nail. The hyponychial angle is A, B, D; and A, B, C is the profile angle. B (bottom), Digits of the patient before, and 1, 2, and 3 years after lung transplantation. Note the decrease of the DPD/IPD and the profile and hyponychial angles.

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**Figure 2.** Correlation between the forced vital capacity (FVC) and the degree of clubbing obtained from annual measurements of three consecutive years after the lung transplantation. The changes before and after the lung graft were of 45 to 59 mm Hg for the PaO2 (normal values in México city at 2,240 m altitude are between 64 to 70 mm Hg) and from 25 to 11 for the P(A-a)O2.
promoter factor. For the hypothesis to be complete, a right-to-left shunt is required, so the giant cells may pass through to the systemic arteries. Pulmonary fibrosis exhibits both pathologic conditions; it is often accompanied by right-to-left shunts, particularly at the end stages of the disease, and by an overwhelming disarrangement of the parenchymal architecture so that the responsible substance might not be inactivated by the lung cells. In this regard, our patient had a significantly higher alveolar-arterial oxygen pressure difference (PaO$_2$) that almost normalized after lung transplantation. Intrapulmonary shunt could have contributed to the abnormal alveolo-arterial gradient. However, P(A-a)O$_2$ is influenced by other elements such as the mixed venous PO$_2$ and inspired PO$_2$, as well as by factors affecting the oxygen dissociation curve. Therefore, with our results, we can just speculate about the possible role of shunt in the pathogenesis and decrease of clubbing in this patient.

Likewise, regression of clubbing could be associated with the fact that the normal donor lung adequately inactivates platelet-derived growth factor and other presumably pathogenetic bioactive molecules. However, there was a remarkable improvement in pulmonary mechanics and gas exchange, and the decrease of the degree of clubbing was consistent with an improvement of PaO$_2$ and FVC.

The prevalence of clubbing in Mexico City in patients with interstitial lung diseases is extremely high in comparison with other reports. Whether the altitude (2,240 m) has some influence for the presence of clubbing is unknown. However, our patients with interstitial lung disease are usually more hypoxemic compared with the patients described by others, in part due to altitude and to severe lung restriction. Nevertheless, for the reversal of clubbing, altitude was not a variable in our patient, a resident of Mexico City.

Some hepatic disorders are associated with the presence of DC. However, patients with liver enlargement related to right-sided heart failure do not develop clubbing. The best example of this are the patients with COPD and cor pulmonale in whom clubbing is never recorded. Therefore, it is unlikely that the presence and/or reversal of clubbing in this patient is related to hepatic congestion. In addition, results of liver tests were normal both before and after the transplantation.

In summary, we report here in a significant decrease of digital clubbing in a patient with idiopathic pulmonary fibrosis after lung transplant. Organ transplantation as a cause of reversal of clubbing has been previously reported only after liver transplantation. To the best of our knowledge, this is the first description in which reduction of clubbing is documented after lung transplantation.

**References**


**Survival in a Case of Massive Paraquat Ingestion**

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Serious exposure to the herbicide paraquat usually results in death, either due to gastrointestinal caustic lesions, shock, and acute respiratory distress syndrome or related to the progressive development of pulmonary fibrosis associated with refractory hypoxemia. We report a case of suicidal parquat ingestion in a 59-year-old man. Most of the indicators of poor prognosis were encountered in this patient. Treatment consisted of early digestive decontamination and hemodialysis, followed by antioxidant therapy, including the administration of deferoxamine (100 mg/kg in 24 h) and a continuous infusion of acetylcysteine (300 mg/kg/d during 3 weeks). The patient only developed a nonoliguric acute renal failure, a mild alteration of liver tests, and an impairment of CO transfer factor without any respiratory complaint. Renal and hepatic disturbances completely resolved within 1 month, whereas CO transfer factor remained altered 14 months later. This observation suggests that early administration of an antioxidant therapy, including deferoxamine and acetylcysteine could be usefully associated with measures that prevent digestive absorption or enhance elimination to limit systemic toxicity in potentially fatal parquat poisoning.

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