Active Pulmonary Hemorrhage Localized by Selective Pulmonary Angiography*

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Massive hemoptysis in a young woman with negative chest film findings is presented. By using selective pulmonary artery angiography during active pulmonary bleeding, the following findings were demonstrated: (1) intraparenchymal hemorrhage, (2) clearing of blood from the lung and bronchial tree by coughing, (3) early filling of the inferior pulmonary vein. Following lobectomy, specimen angiography suggests the presence of a small arteriovenous fistula. This experience demonstrates that selective pulmonary arteriography may be a useful adjunct in the management of selected patients with massive hemoptysis of obscure etiology.

A case of massive hemoptysis in a young woman with a negative chest film is presented. Active pulmonary hemorrhage originating from a small branch of the right lower lobe pulmonary artery was demonstrated by selective pulmonary angiography. Following lobectomy, specimen angiography confirmed the presence of a small arteriovenous communication.

CASE REPORT

A 25-year-old woman was admitted to Prince George's General Hospital on May 23, 1972, because of massive hemoptysis. Except for tachycardia, the physical examination gave negative findings. The admission chest film and a perfusion lung scan were negative. Flexible fiberoptic photobronchoscopy performed during active bleeding revealed blood in every bronchial orifice, even after saline solution irrigations. After three more bleeding episodes within 24 hours, pulmonary angiography was performed revealing a peculiar prominent transverse arterial branch in the right lower lobe. Selective pulmonary arteriography was performed in order to more closely examine this questionable

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FIGURE 1. Selective right lower lobe pulmonary arteriogram. Early injection phase shows the arteriovenous communication (AVC). Contrast material has extravasated into the pulmonary parenchyma (pp).
FIGURE 2A (upper). Selective right lower lobe pulmonary arteriogram. Arteriovenous communication has become more clearly defined. The contrast material previously seen in the pulmonary parenchyma has been displaced into the bronchus due to cough. FIGURE 2B (lower). Line drawing composite of serial films of selective right lower lobe pulmonary arteriogram. RLLPA—right lower lobe pulmonary artery; IPV—inferior pulmonary vein; AVC—arteriovenous communication; Br—bronchus.

inferior pulmonary vein. These findings allowed the performance of a definitive lobectomy in a clinical setting in which reported mortalities are consistently above 50 percent. The most frequent causes for massive hemoptysis are said to be bronchiectasis, pulmonary abscess, pulmonary tuberculosis, and tumor. It has been demonstrated by numerous pathologic as well as angiographic methods, that these lesions are primarily, if not entirely, fed by the bronchial circulation. With the exceptions of Rasmussen’s aneurysm of cavitary tuberculosis and arteriovenous fistulae hemoptyses in general are attributed to leaks from the bronchial circulation into the bronchial tree. There remains, however, a group of patients in whom the specific diagnosis and mechanism of bleeding remains obscure, even after the most meticulous pathologic dissection, serial sections, bronchovascular cast injections, and injection studies. In Ehrenhaft’s series, 4 of 12 resected specimens failed to yield the exact origin of bleeding. This experience suggests that selective pulmonary arteriography may be a useful adjunct in the management of selected patients with massive hemoptysis of obscure etiology.

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Hyperventilation-induced T-Wave Changes in the Limb Lead Electrocardiogram

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Seventy-two healthy young individuals were subjected to controlled, moderate hyperventilation with room air and with 4.9 percent CO₂ in air, and monitored electrocardiographically. Significant summed frontal T-wave changes with hyperventilation (ΣT₁₋₃ ≥ 1.5 mm) were observed in 12 patients. Six subjects (8.3 percent) showed T-wave depression. It was reversed in five patients by hyperventilation with 4.9 percent CO₂ in air. T-wave elevation, observed in six subjects, was reversed in four patients by hyperventilation with 4.9 percent CO₂. A short period of hyperventilation with an air mixture containing 4.5 percent CO₂ is suggested as a means of screening patients under suspicion of ischemic heart disease exclusively on the basis of ECG changes.

Hyperventilation (HV) has been shown to produce flattening or inversion of T-waves in the electrocardiograms of healthy individuals without heart disease. In most previous investigations, however, hyperventilation has been simulated by a short (15 to 30 sec) period of quick, very heavy breathing—a situation rarely encountered in routine clinical practice. The purpose of this study was to examine the electrocardiographic changes produced by controlled, moderate hyperventilation, in order to 'mitate what occasionally occurs in the emotionally labile or neurovegetative patient. The incidence and magnitude of T-wave changes in a healthy young population was studied using this approach.

MATERIALS AND METHODS

Seventy-two volunteers were chosen at random from the house staff and employees of the Millard Fillmore Hospital, Buffalo, New York. This sample population consisted of 34 men and 38 women, aged 18 to 40 years. The subjects gave a negative history of heart or pulmonary disease and had no physical complaints at the time of the experiment.

All investigative procedures were carried out in the sitting position. Using a Siemens monograph 34, a baseline limb-lead electrocardiogram (leads 1, 2, 3—recorded simultaneously) was obtained. V leads were avoided because of the lack of any one specific "normal" pattern for the right precordial leads, and because their application would have made routine screening more difficult. Resting arterial carbon dioxide tension, PaCO₂, was estimated by infrared analysis of end-tidal CO₂, using the Beckman LB-1 Medical Gas Analyzer. Throughout the procedures, conventional mouthpiece breathing was employed. The subject was asked to hyperventilate for three minutes with room air, at a rate specially chosen to reduce his estimated PaCO₂ by 10-15 mm. In order to achieve this desired rate of breathing, the subject would voluntarily inspire and expire to the beat of a metronome, exchanging only a few breaths for each execution.

The ECG was again recorded. After a short rest interval, this hyperventilation procedure was repeated, at the same rate as previously established, using 4.9 percent CO₂ in air as the breathing medium. After three minutes of hyperventilation with this mixture, which normalized the PaCO₂, the electrocardiogram was similarly repeated. The three recordings thus obtained were scanned for S-T segment alterations and analyzed for T-wave changes, using the summed frontal T-wave value (ΣT₁₋₃) as parameter for comparison. T-wave amplitudes were estimated to the nearest 0.5 mm, after measuring at least five consecutive complexes to obtain a satisfactory mean. For purposes of analysis and interpretation, certain arbitrary standards for summed T changes (ΣT₁₋₃) were adopted. A ΣT₁₋₃ of 1.5 mm was chosen as the lower limit for significant T wave change. Changes equal to or