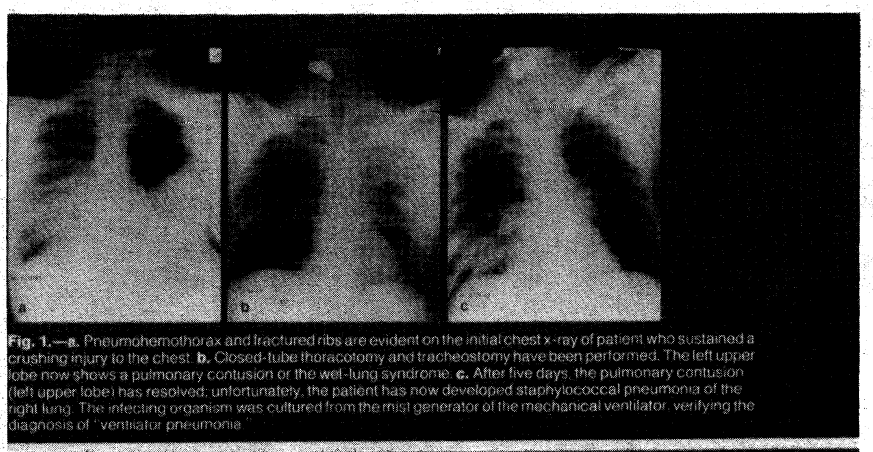

Fig. 1.—a. Pneumohemothorax and fractured ribs are evident on the initial chest x-ray of patient who sustained a crushing injury to the chest. **b.** Closed-tube thoracotomy and tracheostomy have been performed. The left upper lobe now shows a pulmonary contusion or the wet-lung syndrome. **c.** After five days, the pulmonary contusion (left upper lobe) has resolved; unfortunately, the patient has now developed staphylococcal pneumonia of the right lung. The infecting organism was cultured from the mist generator of the mechanical ventilator, verifying the diagnosis of "ventilator pneumonia."

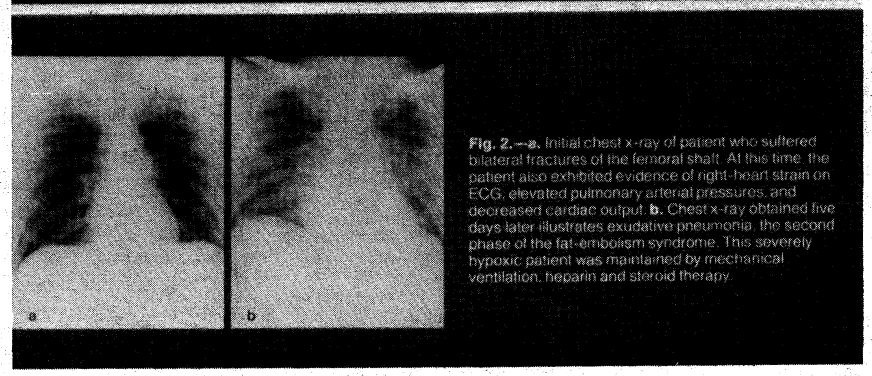

Fig. 2.—a. Initial chest x-ray of patient who suffered bilateral fractures of the femoral shaft. At this time, the patient also exhibited evidence of right-heart strain on ECG, elevated pulmonary arterial pressures, and decreased cardiac output. **b.** Chest x-ray obtained five days later illustrates exudative pneumonia, the second phase of the fat-embolism syndrome. This severely hypoxic patient was maintained by mechanical ventilation, heparin and steroid therapy.

Early in the clinical course of a patient with pulmonary burns, the chest x-ray usually reveals a diffuse alveolar exudate with local areas of atelectasis due to the presence of edema fluid in the alveoli and to bronchial blockage by exfoliated epithelial debris. On arterial blood-gas analysis, one finds that the pH is lowered while Pco_2 is increased. Hypoxia is caused by the inability of oxygen to diffuse into alveoli and across the edematous alveolar-capillary membrane. A "physiologic shunt" effect may ensue if the alveolar-capillary units are perfused adequately without being ventilated. A lack of oxygen diffusion and the resulting shunt effect can be identified on pulmonary function testing, ie, nitrogen or helium "washout," and by determining the pulmonary diffusion capacity of carbon monoxide.[5] In the presence of reduced ventilatory function, nitrogen or helium washout tests reveal a definite "slow space" effect related to those areas of alveolar hypoventilation. Diffusion capacity is reduced by interstitial and intra-alveolar edema.

INTERMEDIATE ONSET

Fat Embolism in Lung Following Injury

Soon after a severe injury, fat may be liberated into the blood from fractured bones. It has been suggested that fat globules may also be formed in blood by the aggregation of chylomicrons. Fat emboli are entrapped by small pulmonary vessels, leading to the *first* stage of pulmonary fat embolism which is characterized by the following: elevated pulmonary arterial pressures; right-axis deviation demonstrated on the electrocardiogram; decreased cardiac output and low arterial pressure; enlargement of the right heart and the right pulmonary artery (observed on x-ray, Fig. 2a); and an apparent increase in physiologic "dead space" detected by pulmonary function testing.[12] The latter is due to the lack of perfusion of alveoli, which may or may not be ventilated.

At this stage, the diagnosis may be confirmed either