clumsy "smaller-than-dates" to describe the foetus in cases of placental insufficiency.

Above all this *Bulletin* shows how greatly obstetrics has developed from pure midwifery to cover a multiplicity of disciplines. Contributions from physiology and biochemistry are well illustrated in the papers on folate metabolism, on water and electrolytes, and on blood-coagulation failure in obstetrics; from haemodynamics in the review of cardiovascular changes in pregnancy and labour; from pathology in the consideration of post-partum pituitary necrosis; and from biophysics in the evaluation of diagnostic ultrasonics.

The three issues a year of the *British Medical Bulletin* are widely accepted abroad as representative evidence of modern work in British medicine. They are no less valuable to doctors in the home countries, and no British obstetrician can afford to overlook this January 1968 number.

Diffuse Fibrosing Alveolitis

Inflammation and fibrosis occurring diffusely in the peripheral gas-exchanging part of the lung, and affecting primarily the alveoli, give rise to a characteristic disorder. The principal symptom is progressive dyspnoea on exertion accompanied by cough, either dry or with scanty, mucoid sputum. Common signs include increased respiratory effort, rales at the bases of the lungs, and clubbing of fingers. Chest radiographs show diffuse mottling or patchy consolidation but may be normal. Tests of lung function reveal ventilatory impairment, defective gas transfer, and decrease of lung compliance without evidence of airways obstruction.

This disease has been given various names, including diffuse interstitial fibrosis of the lungs, interstitial pneumonia or pneumonitis, and Hamman–Rich disease. Recently the term diffuse fibrosing alveolitis[1] has been used as a convenient generic name for a broad category of disease having two essential features: cellular thickening of the alveolar walls with a strong tendency to fibrosis, and the presence of mononuclear cells within the alveoli.[2] Additional histological features sometimes found in the more acute cases include other forms of cellular exudate, fibrinous exudate, and hyaline membrane. More chronic cases may show fibrotic destruction of lung structure, honeycombing, and excess of smooth muscle. Occasionally hyperplasia of lymphoid follicles and epithelioid granulomas occur.

Reviewing the specimens taken at lung biopsy from 16 patients with diffuse fibrosing alveolitis, J. G. Scadding and K. F. W. Hinson[2] found that the degree of thickening of the alveolar walls was correlated inversely with the number of mononuclear cells in the alveoli. At one extreme were lungs with much thickening of the alveolar walls and few intra-alveolar cells, the changes being irregularly distributed throughout the biopsy. This appearance they termed the "mural" type of fibrosing alveolitis. At the other extreme were lungs with little thickening of the alveolar walls and many intra-alveolar mononuclear cells, mainly of the granular type, the changes being uniformly distributed. This appearance they termed the "desquamative" type of fibrosing alveolitis. Radiographic appearances varied widely among the patients, but widespread, well-defined mottling occurred more often in those with the mural type, whereas patients with the desquamative type most often had either opacities of fairly uniform density above an elevated diaphragm or irregular patchy consolidation at the bases with widespread, ill-defined mottling. Treatment with corticosteroid suppressed symptoms and signs in a few of the patients with the desquamative type but in none of those with the mural type.

A. A. Liebow and colleagues,[3] who first described the desquamative type under the name of "desquamative interstitial pneumonia," considered that it warranted separation from other forms of fibrosing alveolitis, because it differed from them in histology, radiological appearance, and response to corticosteroids. Scadding and Hinson's study, however, shows that there is in fact no sharp distinction, but a range of cases occur in which an inverse relationship exists between alveolar thickening of the walls and intra-alveolar mononuclear cells. Despite that, recognition of the differences between the two ends of the range represented by the mural and desquamative type is of practical importance.

Fibrosing alveolitis is occasionally found in patients suffering from certain diseases of unknown cause, notably those associated with autoimmune phenomena, including rheumatoid arthritis, systemic sclerosis, Sjögren's syndrome, and idiopathic pulmonary haemosiderosis. Some other patients with fibrosing alveolitis but without clinical evidence of these diseases have autoimmune factors in their blood.[4]

Allergic reactions in the alveoli to several sorts of inhaled organic dusts may cause illnesses with patterns of symptoms, signs, and disordered function similar to that of fibrosing alveolitis of unknown cause. And in some of these cases there may be similarities also in histological appearances, especially in the late stages. The term "extrinsic allergic alveolitis" aptly describes the pulmonary disorder resulting from these organic dusts.[5] The classical example is farmer's lung, due to thermophilic actinomycetes in mouldy hay.[6] Similar pulmonary reactions occur in bird-fancier's lung, due to inhalation of dusts of pigeon and budgerigar droppings,[7 8] bagassosis from sugar cane,[9] maple bark disease due to the fungus *Cryptostroma corticale*,[10] wheat weevil disease due to *Sitophilus granarius* in wheat flour,[11] and mushroom-worker's lung due to actinomycetes.[12] These pulmonary disturbances are usually accompanied by systemic reactions, including fever, chills, and malaise, occurring a few hours after inhalation of the dust. These responses are mediated by precipitating antibodies, and serological tests for specific circulating precipitins are a valuable diagnostic aid.

The differential diagnosis of fibrosing alveolitis includes pulmonary disease due to the inhalation of certain inorganic dusts, among which are asbestos, beryllium, and aluminium, and also pulmonary lesions due to tuberose sclerosis,[13] generalized neurofibromatosis[14] and eosinophilic granuloma.[15] These disorders can usually be distinguished from fibrosing alveolitis by their characteristic clinical and histological features.

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