

The Morbid Anatomy of Tuberculosis in Old Age. ROULET, F. (1950). *Ann. Méd.*, **51**, 69.

In 270 necropsies carried out at the University of Basle on subjects over 60 years old, frank evidence of tuberculosis was present, although it was the cause of death in only 90 cases. The active focus was found in 245 cases around an old calcified or ossified nodule in the tracheo-bronchial lymph nodes; The lungs in these cases revealed no active primary focus, though an old, calcified, inactive lesion was often discovered. It is argued that active tuberculosis in old age is frequently caused by reactivation of an old lesion in the lymph nodes around the bifurcation of the trachea, possibly due to the chronic inflammation of the lower trachea and main bronchi, which is extremely common in old people. Tuberculosis of the tracheo-bronchial nodes frequently leads to complications: of the 245 cases of tracheo-bronchial-node infection, there was also frank tuberculous infection of the lungs in 35: in 12 cases there was a generalized haematogenous spread either from a focus in the tracheo-bronchial nodes or from an alternative site of active infection; in 13 cases there were foci both in the tracheo-bronchial nodes and elsewhere, but no generalized spread; and in 25 cases haematogenous spread had occurred with tracheo-bronchial nodes as the only possible primary source. In the remaining 160 cases the infection was confined to the tracheo-bronchial nodes.

Speaking generally, miliary tuberculosis develops more rapidly in the old than at other ages. The appearances are classical, though the tubercles may be rather large—as big as a pea or a hazel nut. In the whole series there were 16 cases of miliary spread amongst the 43 cases of frank pulmonary tuberculosis. Tuberculosis of the serous cavities is not uncommon in the elderly. Pulmonary tuberculosis may be of the chronic apical fibro-caseous type with cavities, or of the ulcerative caseous type of acute tuberculosis, the frequent occur-

rence of which in old people may possibly be due to reduced general resistance and antibody response.

Glomerulonephrosis. A Morphologic Manifestation of Renal Cortical Ischemia in Toxic Oliguria and Lower Nephron Nephrosis. FRENCH, A. J. (1950). *Arch. Path.*, **49**, 43.

The histopathological findings in the kidneys of 20 specially selected cases of glomerulonephrosis are described, special stress being laid on the glomerular changes. This study was undertaken in the hope of explaining the anuria or oliguria which accompanies lower nephron nephrosis ("tubular" or "toxic" nephritis), in which condition, on physiological grounds, there should be polyuria if a tubular lesion alone be present. For this purpose it was essential to eliminate cases of primary or secondary renal disease, eclampsia, and cardiovascular hypertension.

The changes found in the glomeruli, tubules, and medulla in sections of the kidneys (mostly stained with haematoxylin and eosin) are tabulated. Glomerular ischaemia, together with thickening of the capillary walls, was found in every case. The presence of granular material staining grey-blue in the glomerular spaces and proximal tubules was noted, this being regarded as the precursor of the protein casts seen in the distal convoluted tubules in all except one of these cases. R. B. T. Baldwin.

Corrections.—Dr. Varadi writes: I shall be obliged if you will print the following corrections to my article "Combined Fragility Test for Congenital Haemolytic Jaundice" (*J. clin. Path.*, **4**, 221). On page 222 in paragraph 3 read 0.021% instead of 0.21% NaCl, and, on page 224 paragraph 2 of the Appendix, marrow puncture instead of culture.

We much regret that in the description of a cell-suspension mixer by Dr. Matthews (*J. clin. Path.*, **4**, 383) the pipettes were described as 20 ml. and 50 ml. These should be 20 c.mm. and 50 c.mm.