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ochronosis—a word coined by Virchow,[14] who first described the pathological findings in alkaptonuria and wished to distinguish the disorder from melanosis.

THE CAUSE OF POSTURAL PROTEINURIA

A FEW weeks ago we said that the mechanism of postural proteinuria was still unknown.[15] An important contribution has since been made by Greiner and Henry.[16] These workers criticise the view[17] that the proteinuria is directly due to increased pressure in the inferior vena cava and renal veins, and refer to an experimental study[18] in which increase of caval pressure to 150–220 mm. saline, maintained for thirty minutes, caused no proteinuria.

Carefully controlled observations in six young men with postural proteinuria indicated that the most important aetiological factor is pooling of blood in the extremities. Advantage was taken of the fact that postural proteinuria is particularly liable to occur in hot conditions,[17] and all the observations were made at a temperature of 122°F. Lordosis in the supine position produced proteinuria once only, whereas venous-occlusion cuffs applied at a pressure of 80 mm. Hg to all four limbs resulted in *proteinuria in all the subjects*. Proteinuria, which regularly occurred after tilting the subjects to a 70° head-up position, was prevented by compression of the legs, which effectively inhibited peripheral pooling of blood.

Greiner and Henry point out that their observations explain the facts that postural proteinuria is commoner *in hot conditions producing peripheral vasodilatation*, and in motionless standing[17] which is particularly liable to cause pooling of blood in the legs. They suggest that deviation of blood from the heart and lungs causes arterial vasoconstriction which is proportionately greater in the renal arterial system. The erect lordotic posture greatly reduces the renal plasma flow,[19] but this fall is *no greater in cases of postural proteinuria than in* non-proteinuric controls.

LUNG CANCER IN ASBESTOSIS

THE causes of cancer of the lung are probably mainly exogenous,[19] but each of the definitely known carcinogenic substances—e.g., ores in Schneeberg and Joachimstal mines, bichromates—accounts for only a minute proportion of all cases. Doll[21] now confirms suggestive evidence that lung cancer is an occupational hazard of asbestos workers. Examination of the cause of death in 105 persons who had been employed in one asbestos works showed that 18 had had lung cancer, associated in 15 cases with asbestosis. The mortality of 113 men who had worked for at least twenty years in places exposed to asbestos dust was compared with the mortality experience of the whole male population. There were 39 deaths in the first group, whereas 15·4 were expected: the excess was due to deaths from lung cancer (11, compared with 0·8 expected) and from other respiratory and cardiovascular diseases (22, compared with 7·6 expected). *The average risk among men* employed for twenty *or* more years has been of the order of ten times that experienced by the general population. This risk has become progressively less since the precautions introduced as a result of the Asbestos Industry Regulations, 1931, to prevent dust dissemination in the works became effective.

Cloyne[22] found in his series of 1205 specimens of lungs with dust diseases, including 121 of asb[illegible], that the rates for primary neoplasm of the lung in male and female asbestos workers were 20% and 10% respectively. Barnett[23] found 31 deaths with carcinoma of lung in 235 deaths associated with asbestosis during the years 1924–46, with a distribution of 17·2% male and 8·4% female deaths. Adjustments for length of exposure to the dust and age at death would scarcely account for the discrepancy in the figures between the two sexes. The same discrepancy exists in the figures for all cases of carcinoma of the lung: Kennaway and Kennaway[24] showed that the rates of female to male deaths in England and Wales had changed from 1 : 1·9 in 1921 to 1 : 4·0 in 1945; between 1935 and 1945 the increase was 250% for males and 175% for females. If the causes of cancer in the lung are the same for both sexes (as in asbestosis and possibly in smoking), can the sex discrepancy be related to the baffling fact that, while the total "amount" of tumour in both sexes is approximately equal, 40% of the cancer deaths in females are from carcinoma of organs peculiar to the female,[25] leaving correspondingly smaller numbers for other sites? If so, what internal factor determines this organ selectivity?

Unlike asbestosis, the main causes of dust diseases in the lungs are not associated with an increase in carcinoma of the lungs; and Kennaway and Kennaway[24] showed that bronchial carcinoma is less common in coal-workers than in the general population. James[27] has confirmed this very neatly by comparing a series of South Wales coalminers with a series of non-miners in the same area, obtaining figures of 3·3% for the former and 5·4% for the latter; but he points out that the relatively low incidence of tumour in the miners may be partly due to dust disease causing death before the average cancer age. *The dust diseases generally* act on the connective tissues, and there is no satisfactory explanation of why the asbestos fibres should affect the epithelium as well.

EPILEPSY AND THE ELECTRO-ENCEPHALOGRAM

IMPROVED techniques of electro-encephalography (E.E.G.) have confirmed some earlier correlations of electrical with clinical activity, such as that of bilateral 3-per-second wave-and-spike patterns with petit mal; have led to the modification of some, such as that of *square-topped* waves and a sharp down peak with psychomotor epilepsy; and have cast renewed doubt on others, such as the identification of the cortical spike focus with the site of origin of an epileptic discharge.[28]

Gibbs and his colleagues[29,30] have described valuable information about the natural history of E.E.G. abnormalities which has important clinical implications. By *following changes* in E.E.G. patterns as infants and children develop, they have obtained a "longitudinal section" of electrical brain activity to supplement the brief "cross-sectional" view of isolated E.E.G. recordings. They point out that the immature brain tends to react to various possibly epileptogenic traumata with a generalised electrical abnormality. Moreover the pattern of reaction differs at different stages of maturation. In infancy "hypsarhythmia" (continuous slow-wave high-voltage discharge with diffuse spiking) is common;

14. Virchow, R. *Virchows Arch.* 1886, 37, 212.
15. *Lancet*, May 7, 1955, p. 958.
16. Greiner, T., Henry, J. P. *J. Amer. med. Ass.* 1955, 157, 1373.
17. Bull, G. M. *Clin. Sci.* 1948, 7, 77.
18. Farber, S. J., Becker, W. R., Elchna, L. W. *J. clin. Invest.* 1953, 32, 1145.
19. King, S. E., Baldwin, D. S. *Proc. Soc. exp. Biol., N.Y.* 1934, 86, 634.
20. Gough, J. In Recent Advances in Pathology. Edited by G. Haubeld. London, 1953; p. 199.
21. Doll, R. *Brit. J. industr. Med.* 1955, 12, 81.
22. Cloyne, S. R. *Lancet*, 1951, 1, 810.
23. Barnett, G. P. Annual Report of the Chief Inspector of Factories for the year 1917. H.M. Stationery Office, 1919.
24. Kennaway, E. L., Kennaway, N. M. *Brit. J. Cancer*, 1947, 1, 260.
25. Kennaway, E. L. *Brit. med. J.* May 7, 1955, p. 1167.
26. Kennaway, E. L., Kennaway, N. M. *Brit. J. Cancer*, 1953, 7, 10.
27. James, W. R. L. *Brit. J. industr. Med.* 1955, 12, 57.
28. Meyers, R. *Epilepsia*, 1954, 3, 1.
29. Gibbs, E. L., Gibbs, H. W., Gibbs, F. A. *Amer. J. Dis. Child.* 1954, 88, 506.
30. Gibbs, E. L., Fleming, M. M., Gibbs, F. A. *Pediatrics*, 1954, 13, 66.

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