who point out that the previously accepted fact that vitamin-B deficiency, as such, decreases the inactivation of oestrogens by the liver has been shown to be untenable. Drill and Pfeiffer (1946) have shown that "the effect of acute vitamin-B-complex deficiency in producing a failure of the liver to inactivate oestrone seems to be due to the concomitant inanition."

If we may assume that cervical carcinoma is comparatively infrequent in the better-nourished members of a community, then there must be some factor or factors which lessen its occurrence in this group. Alternatively, in the undernourished group there must be some factor or factors which tend to cause this lesion. As cancer is an active condition it would seem more logical to suppose that poor economic conditions in some way produce this disease. Our present knowledge would suggest that cervical carcinoma may be produced in the poorer economic class by some unknown hormonal imbalance or by an unsatisfactory reaction, associated with poverty, to irritative lesions of the cervix.

Summary

Figures are given for the relative incidence of endometrial and cervical carcinoma as seen in a selected population.

These figures are at variance with the incidence usually accepted, and it is suggested that the incidence of the various forms of uterine cancer vary with the socio-economic group.

The possible implications of these findings are briefly discussed.

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REFERENCES

- Ayre, J. E. (1947). *Amer. J. Obstet. Gynec.*, **54**, 363.
 Brunelli, B. (1935). Quoted by Ayre (1947).
 Clemmesen, J., and Nielsen, A. (1951). *Brit. J. Cancer*, **5**, 159.
 Corscaden, J. A., and Gusberg, S. B. (1947). *Amer. J. Obstet. Gynec.*, **53**, 419.
 Drill, V. A., and Pfeiffer, A. (1946). *Endocrinology*, **38**, 300.
 Gagnon, F. (1950). *Amer. J. Obstet. Gynec.*, **60**, 516.
 Greene, R. R., and Peckham, B. M. (1947). *Ibid.*, **54**, 611.
 Hall, J. E., Pepe, J., and Tortora, J. (1950). *Ibid.*, **59**, 634.
Irish Journal of Medical Science, 1949, No. 284.
 ——— 1950, No. 300.
 ——— 1951, No. 312.
 Lombard, H. L., and Potter, E. A. (1950). *Cancer*, **3**, 960.
 Novak, E. (1951). *Amer. J. Surg.*, **82**, 149.
 ——— (1952). *Gynecologic and Obstetric Pathology*, p. 239. Saunders, Philadelphia and London.
 Stock, P. (1947). *Studies on Medical and Population Subjects*, No. 1. London.
 Weiner, I., Burke, L., and Goldberger, M. A. (1951). *Amer. J. Obstet. Gynec.*, **61**, 418.
 Willis, R. A. (1948). *Pathology of Tumours*, p. 524. Butterworth, London.

The National Research Development Corporation exists primarily to develop and exploit in the public interest inventions (including medical ones) resulting from public research. It also has other powers. In June this year the Corporation's borrowing powers come to an end, and if the Corporation is to continue its normal activities new legislation will be necessary. The chance is therefore taken in the annual report for 1952-3, published recently, to review the results of the Corporation's first four years of trading. Starting in 1950 with costs of a little over £20,000 and a return by way of royalties of less than 1%, by 1953 the accounts showed 23% earnings on operating costs of £120,000. Several of the inventions being developed by the Corporation have a direct bearing on medicine. The pilot scale production of hecogenin from sisal juice in East Africa provides a starting material for the synthesis of cortisone. Prototypes of the Burns analgesic inhaler, designed for trileone and other volatile analgesics, have been prepared for clinical trial. The Corporation has underwritten the costs of the initial manufacture of an aural microscope and chick sexer. Other projects range from large electronic digital computers ("electronic brains") to a new machine for liquefying atmospheric gases.

DIABETES INSIPIDUS ASSOCIATED WITH PULMONARY DISEASE

BY

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Spillane (1952) described four cases in which diabetes insipidus was associated with pulmonary disease. The case here reported falls into the same category and exhibits diabetes insipidus and lung lesions as the only discoverable abnormalities.

Case Report

A housewife aged 57 was first seen in January, 1952, having been referred for further investigation after attending a mass x-ray session. She had suffered from increasing dyspnoea on exertion for about four years, with morning cough and the expectoration of a small amount of yellowish sputum. For several years she had passed large amounts of pale urine at frequent intervals during the day and night. She was also troubled by excessive thirst and said that she could drink a pint of soda-water at a time and still be thirsty. Her sleep rhythm was unchanged and there was no somnolence by day.

Before her present illness she had been healthy and had when younger often cycled 50 miles (80 km.) a day without distress. Until seven years previously she had worked as a waitress in addition to looking after her home, but she had had to discontinue this in order to look after her husband, who was becoming severely disabled owing to post-encephalitic Parkinsonism. She had had pneumonia on three occasions, at the ages of 23, 29, and 37; erysipelas at 14; and scarlet fever in childhood. There was no history of skin disease.

She was the second oldest of a family of 13 of whom five of six brothers had died in infancy, the other being killed in adult life. Of her six sisters, five were healthy and one had died 48 years previously, the cause of death being unknown. The patient had had two children, both of whom were healthy; her son was 34 and her daughter 28.

Examination revealed a rather pale middle-aged woman, weighing 134 lb. (60.8 kg.), an excellent witness, though inclined to make light of her complaints. Her skin was somewhat dry and wrinkled but otherwise showed no abnormality. No lymphatic glands were palpable and there was no enlargement of the salivary glands. Venous congestion, ankle oedema, and finger-clubbing were not present. She was somewhat dyspnoeic at rest, but no other clinical abnormalities were detected in the chest or heart. The blood pressure was 150/100. The abdomen showed no abnormality and the liver and spleen were not palpable. The optic disks were normal and neurological examination was negative.

Further investigation showed that she passed 12 to 16 pints (6.8 to 9.1 litres) of urine daily and drank roughly equal amounts. The specific gravity of the urine was in the neighbourhood of 1005; there was no glycosuria or albuminuria and no casts were present. X-ray films of the chest showed a mixture of reticulation and miliary mottling throughout both lung fields without enlargement of the hilar lymph nodes. No cysts were detected and there was no pneumothorax. X-ray films of the hands, feet, and skull showed no bony abnormalities. The pituitary fossa was radiologically normal.

Sputum tests for tubercle bacilli were repeatedly negative. The Mantoux test was positive to 1:1,000 O.T. Blood examination showed: Hb, 66% (9.8 g.%); erythrocytes, 5,000,000. The E.S.R. was 12 mm. in the first hour (Wintrobe). The blood Wassermann and Kahn reactions were negative and the glucose-tolerance curve was normal. The

serum cholesterol was 305 mg. per 100 ml.; serum albumin 5 g. and globulin 3.5 g. per 100 ml.; albumin/globulin ratio 1.43:1. Liver-function tests showed serum colloidal gold reaction 3+, gamma globulin (zinc sulphate) 8 units, thymol turbidity 4 units, and thymol flocculation +.

Pituitary snuff immediately controlled her polyuria and allowed her to sleep well at nights. After starting this treatment, however, she stated that her breathlessness was somewhat increased. Because of the hypochromic anaemia she was given iron, which resulted in some amelioration of the dyspnoea.

A year later her condition was unaltered and a further x-ray film of the chest showed no substantial change.

Comment

The diagnosis of diabetes insipidus cannot be in doubt, there being adequate evidence to exclude other causes of polydipsia and polyuria—that is, diabetes mellitus, renal disease, and hysterical polydipsia. The only other discoverable lesions were those in the lung, manifested by exertional dyspnoea and radiographic changes.

Spillane (1952) summarized fully the literature relating to this combination of lesions. Following him the diagnoses to be considered are sarcoidosis, tuberculosis, xanthomatosis, tuberous sclerosis, syphilis, honeycomb lungs, and bronchogenic carcinoma.

The first of Spillane's four cases was an undoubted example of sarcoidosis, and this diagnosis could not be excluded in his Case 4. The other two cases were considered clinically to be examples of xanthomatosis. Necropsy in Case 3 later showed, in the hypothalamus and the lungs, the changes of eosinophilic granuloma which shares a morphogenesis in common with Hand-Schüller-Christian and Letterer-Siwe diseases, all three being secondary xanthomatoses associated with progressive reticular hyperplasia. It is therefore interesting to note that one of Cates and Garrod's (1951) cases of diabetes insipidus occurred in a patient with xanthomatosis and honeycomb lungs who also developed an eosinophilic granuloma of bone.

Dennis and Rosahn (1951) reported the case of a man aged 30 with diabetes insipidus and cystic lungs. They concluded from the necropsy findings that it represented a granulomatous disease involving the reticulo-endothelial system.

In the present case sarcoidosis does not appear to be the most likely diagnosis. Spillane (1952) collected from the literature 18 cases of sarcoidosis associated with diabetes insipidus. In none were the lesions confined to the pituitary region and the lungs. Similarly his own Case 1, which was an example of sarcoidosis, had an initial characteristic uveo-parotitis with facial palsy. His Case 4, in which no certain diagnosis was reached, and in which sarcoidosis could not be excluded, showed lesions in these two situations only. In this respect it is similar to the present case in which the Mantoux test (positive to 1:1,000 O.T.) is rather against the diagnosis of sarcoidosis. In addition, the dyspnoea was more intense than might be expected in a case of sarcoidosis with similar radiographical findings.

If we adopt a histological definition of sarcoidosis, as does Scadding (1950), diagnosis must await confirmation by biopsy. In the present case no superficial glands were available for biopsy and, owing to her commitments at home, the patient was unwilling to accept admission to hospital for liver biopsy.

Although, therefore, it is not possible definitely to exclude sarcoidosis, a granulomatous or xanthomatous process seems by exclusion to be a more likely diagnosis.

It is possible that when lung changes and diabetes insipidus occur together as the only discoverable disease they constitute a syndrome the pathological basis of which is a granuloma of some as yet undetermined and possibly variable type. The two conditions may, however, occur as a part of more widespread pathological processes such as those indicated in the papers cited.

Summary

The case is described of a woman aged 57 who had diabetes insipidus associated with a combination of reticulation and miliary mottling throughout both lung fields. A granulomatous or xanthomatous process is thought to be the most likely diagnosis, though sarcoidosis cannot be definitely excluded.

REFERENCES

- Cates, J. E., and Garrod, O. (1951). *Clin. Sci.*, **10**, 145.
Dennis, J. W., and Rosahn, P. D. (1951). *Amer. J. Path.*, **27**, 627.
Scadding, J. G. (1950). *British Medical Journal*, **1**, 745.
Spillane, J. D. (1952). *Thorax*, **7**, 134.

PULMONARY ACARIASIS IN SPAIN AN ILLUSTRATIVE CASE REPORT

BY

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Considerable interest has been created in many parts of the world by the association that has been postulated by workers in Ceylon between hypereosinophilic respiratory disorders and pulmonary acariasis. Carter Wedd, and D'Abrera (1944) were the first to detect mites (Acarina) in the sputum of asthmatic patients with high eosinophilia. This discovery was confirmed by Soysa and Jayawardena (1945), who also suspected that the syndrome might result after exposure to inhalation of airborne mites. Further investigations in Ceylon by Carter and D'Abrera (1946) and by Soysa (1949, 1952a) have verified the previous findings. The conclusions of the Ceylonese workers that pulmonary acariasis may be associated with respiratory symptoms and massive eosinophilic reaction have been supported by reports from England (Hall, 1946), the Netherlands West Indies (Van der Saar, 1946), East Africa (Wilson, 1947), etc.

Since 1946 my attention has been engaged by the occurrence of mites in the sputum of certain patients with respiratory disorders associated with eosinophilic leucocytosis, and cases have been reported in Spain (Figueroa Taboada, 1946). I have been able, in my studies of this curious syndrome, to verify the ideas that have been so carefully and clearly presented by Soysa, and the results of my investigations are in complete agreement with his views concerning the three-stage clinical course discernible in many cases; the remarkable eosinophilic leucocytosis that is the most prominent and constant pathological feature of this syndrome; the variability of the pulmonary radiological appearances; the presence of mites in the sputum; the possibility of bronchial infestation by airborne mites; the ambient air importance of environment in relation to the acquisition of the disease; and the dramatic response to arsenical therapy, which is often associated with a Herxheimer reaction.

As an example of our findings in Madrid I cite a typical case history.

Case Report

A male clerk in a food store, aged 39, was investigated for asthma of seven months' duration. His personal and family history revealed no indication of any allergic antecedents. A blood count showed a total leucocyte count of