that 'phthisiogenesis is more a problem of predisposition than of bacterial infection' (Fishberg, 1932).

Summary

The not infrequent incidence of metabolic diseases in family histories of tuberculous patients, as well as the drop of tuberculosis incidence in diabetes since the discovery of insulin, and observations on the 'curative' effect of tuberculosis on diabetes, has led us to investigate the relationship between pulmonary tuberculosis and the carbohydrate metabolism by means of sugar-tolerance tests. We carried out 62 of them on 50 unselected afebrile tuberculous patients. We used the classical method of giving 50 gm. glucose orally. We distinguished with this method two different types of curves, one with enhanced sugar tolerance with a very low descending limb, the other with impaired sugar tolerance, sometimes resembling those of potentially diabetic patients. Morphologically, the former curve corresponds with cases of exudative-ulcerative pulmonary tuberculosis, the latter curve with those suffering from a more chronic fibrotic type. Morphological transitions between these two extreme types of pulmonary tuberculosis correspond functionally with their respective transitional sugar-tolerance tests. By means of determinations of the blood density, undertaken simultaneously with the blood sugar estimations on 45 patients, we could show that the form of the sugar-tolerance curve is definitely independent of and uninfluenced by the fluctuations of the blood density. An explanation of our findings has been attempted on the ground of recent physio-pathological findings on the tuberculous lung. A path for further studies for the elucidation of the functions of the tuberculous lung is indicated.

REFERENCES

BARBOUR, H. G., and HAMILTON, W. F. (1926). *J. Biol. Chem.*, **69**, 625.

BILLIMORIA, R. B. (1940). *Transactions of the 2nd Tuberculosis Workers' Conference*, p. 63. Tuberculosis Assocn., India, New Delhi.

BOSE, J. P. (1939) .. *A Handbook on Diabetes Mellitus and Its Modern Treatment*. Thacker, Spink and Co. (1933), Ltd., Calcutta.

CORYLLOS, P. N., *et al.* (1932). *Amer. Rev. Tuberc.*, **26**, 153.

CURSCHMANN, H. (1928). *Beitr. Klin. Tuberk.*, **69**, 540.

FISHBERG, M. (1932) .. *Pulmonary Tuberculosis*. Lea and Febiger, Philadelphia.

GHON, A. (1926) .. *Zeitschr. ges. Exper. Med.*, **50**, 26.

GOLDBERG, B. (1939) .. *Clinical Tuberculosis*. F. A. Davis Co., Philadelphia.

HARRISON, G. A. (1937). *Chemical Methods in Clinical Medicine*. J. and A. Churchill Ltd., London.

JACOBY, H. (1930) .. *Zeitschr. ges. Exper. Med.*, **70**, 100.

JOSLIN, E. P., *et al.* (1940). *The Treatment of Diabetes Mellitus*. Lea and Febiger, Philadelphia.

LUNDBERG, E. (1925) .. *Acta med. Scandinav.*, **62**, 1.

PAGEL, W. (1935) .. *Amer. J. Med. Sci.*, **189**, 253.

POTTENGER, F. M. (1934). *Tuberculosis in the Child and the Adult*. Henry Kimpton, London.

SIEGEN, H. (1926) .. *Beitr. Klin. Tuberk.*, **63**, 143.

THAYSEN, T. E. H. (1935). *Quart. J. Med.*, **4**, 359.

TUNBRIDGE, R. E., and ALLIBONE, E. C. (1940). *Ibid.*, **9**, 11.

SIMULTANEOUS BILATERAL 'EARLY INFILTRATION' IN A MIDDLE-AGED PATIENT

By K. EISENSTAEDT, M.D., T.D.D.

State Hospitals, Jamnagar

IN this country, most cases of lung tuberculosis when diagnosed are in a more or less advanced condition. Early detection, unfortunately, is a rare exception. It has been shown in western countries that the beginning of adult tuberculosis, the tertiary stage according to Ranke or the re-infection stage according to others, is very frequently located in the posterior-superior sub-apical region. Pathologically, it is a more or less extensive, exudative infiltration. This observation challenged the earlier conception of an insidious tubercular onset in one or both apices, which is unfortunately still deeply rooted in the minds of many practitioners. Periodical and serial *x*-ray examinations of certain professional groups, especially of hospital staff, offered the opportunity to detect the very onset of the disease in persons who were proved to be healthy in previous *x*-ray examinations and appeared even so at the time of detection. Burrell warns us 'that no signs may be found even by an expert in a case of extensive disease, and that he may be faced with a legal action for negligence if he has based his negative diagnosis on the absence of physical signs and has not had an *x*-ray taken'. The statement that the onset of the disease might take place in the sub-apical region had already been made by Ewart (Fishberg, 1932), Fowler (Fishberg, 1932), Gekler (Fishberg, 1932) and Wessler (Fishberg, 1932). To Assmann (1923), however, the credit must be given for investigating these sub-apical exudative patches in periodical and serial *x*-ray examinations, emphasizing that, but for the *x*-ray detection, many of these cases would have been overlooked, as clinical signs and symptoms were completely absent. His term 'early infiltration' (Assmann focus) for this phenomenon has been accepted by phthisiologists. Since then, innumerable communications have dealt with this subject. If I venture to add the following case, I may put forward 4 reasons, *viz.* (1) The relative rarity of detection of an 'early infiltration' in this country with close following up for three months, (2) the exceptional late appearance, (3) the hereditary late susceptibility and (4) the simultaneous appearance of lesions on both sides.

The patient, a male Hindu, 46 years old, is a member of the medical staff of a general hospital. He had

Plate I

SIMULTANEOUS BILATERAL 'EARLY INFILTRATION' IN A MIDDLE-AGED PATIENT : K. EISENSTAEDT. PAGE 12.

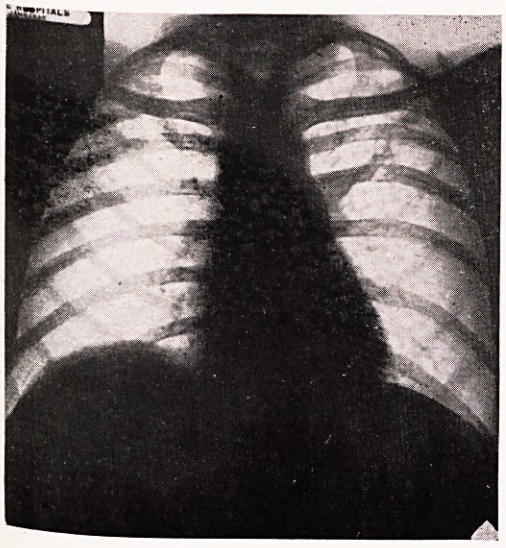

Fig. 1.

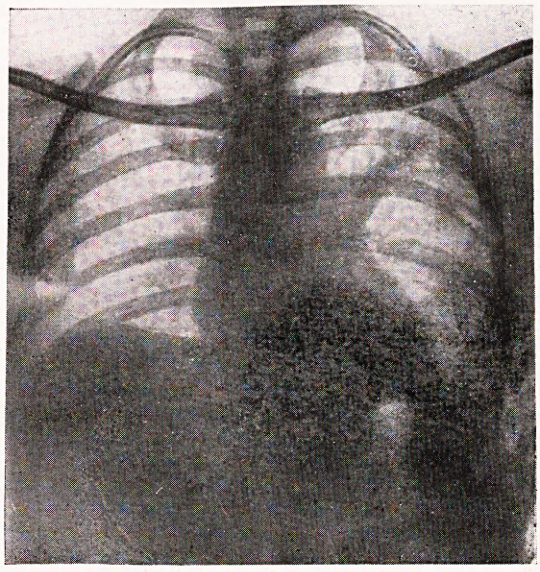

Fig. 2.

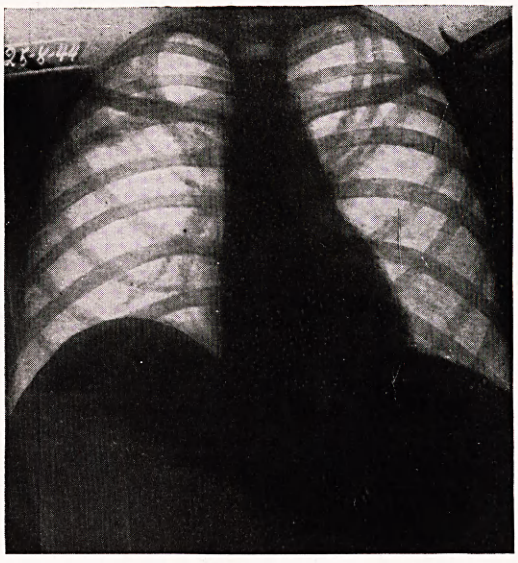

Fig. 3.

cervical adenitis at the age of 14 to 16 years; otherwise he enjoyed perfect health. The family history reveals the following interesting data: his father died at the age of 56 years after suffering from lung tuberculosis for about 6 to 7 years; his father's sister died at the age of about 50 years from lung tuberculosis; one brother died at the age of 38 years from lung tuberculosis. They all enjoyed perfect health but for the last period of their lives.

The health of the patient was checked as a matter of routine at periodical intervals. Fluoroscopies of the chest were done in December 1943, February 1944 and at the beginning of May 1944. None of the three screenings showed signs of parenchymatous lesions, although at the time of the last examination in May, the patient complained of pain at the back of the left side. Another screening on 5th June, 1944, suddenly revealed an infraclavicular infiltration on the left side with a cavity, and some small, hazy patches at the corresponding region of the right side, confirmed by a photo taken the next day (figure 1, plate I). There was no doubt that these infiltrations and the cavitation of the left side must have developed during the course of the previous month. The patient noticed only little symptoms of activity, some cough and expectoration (positive), slight occasional pain, rare subfebrile temperature, the general condition being quite good.

For personal reasons the patient paid little attention to medical advice and took only a little rest. The second photo (figure 2, plate I) taken on 8th July, 1944, showed slight resorption of the exudative patches of the left side, and replacement by apparently more linear shadows: the cavity was unchanged. The small indistinct patches seen on the right side of the first photo, however, had developed into an irregular, confluent infiltration with beginning cavitation in the centre. Again the patient preferred an expectant policy until 7 weeks later, on 25th August, 1944, a third photo (figure 3, plate I) revealed a cavity at the right side of about the same size as that of the left side, the latter being unchanged. Although a little higher, the picture of the right side appeared almost as if reflected by a mirror from the left side. Then at last the patient entered the hospital. (Other clinical data are omitted as irrelevant.)

The interesting points of this case are :—

(1) The sudden appearance and rapid cavitation of a typical 'early infiltration', a rare observation unless periodical and serial *x*-ray examinations are performed, particularly among professionally exposed persons.

(2) The appearance of an early infiltration at an advanced age of 46 years, whereas generally these forms are observed at the age of 15 to 25 years (Fishberg, 1932).

(3) A distinct familial taint evidenced by three other members of the patient's family who became victims of tuberculosis at a relatively advanced age. According to Burrell (1938) '. . . the disease undoubtedly tends to run a similar course in some families . . . in other families the disease may develop at about the same age in each patient. Thus, some families tend to have an early and others a late susceptibility to tuberculosis. In one family 2 brothers and 5 sisters died from pulmonary tuberculosis, their age at death being 30, 44, 65, 76, 76, 78 and 81. It is generally agreed that tuberculosis is not hereditary, but that some factor affecting resistance or susceptibility may be inherited'. According to Fishberg, 'family resemblance' in phthisis has been found in about 75 per cent of cases. According to Edel (Fishberg, 1932) and others, in 34.29 per cent of the families the disease began during the corresponding age.

(4) The simultaneous appearance of similar bilateral lesions. When the disease was detected, there was already a cavity on the left side, whereas the right side showed only slight infiltration. The difference in time, if there was any, between the onset on the two sides is negligible, notwithstanding the earlier appearance of the left cavity. The fully developed picture of the disease as seen in the third photo gives no clue as to the priority of cavitation (and even less of the very onset) on either side. The so-called daughter-infiltrations (Redeker), however, appear later, and are easily distinguishable from the original 'early infiltration', at least for quite a long time in the course of the disease. In the present case no such distinction is possible. In spite of the earlier cavitation on one side, the brevity of the developing period and the final picture of the fully developed disease point to simultaneous appearance on both sides.

Summary

A case of pulmonary tuberculosis is reported, showing a bilateral, simultaneous 'early infiltration' in a patient of 46 years with familial late susceptibility.

REFERENCES

ASSMANN, H. (1923) .. *Beitr. Klin. Tuberk.*, **60**, 526.
BURRELL, L. S. T. (1938). *The British Encyclopædia of Medical Practice*, **8**, 182. Butterworth and Co., Ltd., London.
FISHBERG, M. (1932) .. *Pulmonary Tuberculosis*. Lea and Febiger, Philadelphia.

AN ANTI-TUBERCULOSIS SCHEME FOR AN INDIAN CITY

By P. V. BENJAMIN, M.B., B.S., T.D.D.

Technical Adviser, Tuberculosis Association of India, Medical Superintendent, Union Mission Tuberculosis Sanatorium, Arogyavaram, Madanapalle

MANY are now busy with post-war planning for medical schemes, which include schemes for anti-tuberculosis work, and frequent requests are being made for plans and suggestions about how to proceed, and enquiries as to what is required. This paper is to suggest a scheme for a city of about one lakh population.

Estimate of the tuberculosis problem

Before we draw up a plan for an anti-tuberculosis scheme, we should have knowledge about the problem which has to be faced in the town. Some idea can be obtained from the mortality statistics, but this will represent the minimum only, as experience and investigations have shown that only a proportion of deaths from tuberculosis is reported as such. Probably for most Indian cities, it will be found that the tuberculosis death rate will vary from 200 to 500 per 100,000 population. The usual estimate