these differences are not reflected clinically. The proportions of normal lymphocytes, normal monocytes, and abnormal mononuclears seem to vary from case to case: sometimes the monocytoïd cells are prominent, sometimes the lymphoid cells. But it may be said that, if the clinical picture is suggestive, the presence of a mononucleosis which includes a significant proportion of the abnormal mononuclears described makes it very likely that the condition is infectious mononucleosis.

Apart from causing diagnostic difficulties it is of great interest that immature monocytes should appear in an apparently infective state as well as in a leukaemic state. It is well known that in septicaemia myelocytes may appear in the blood, and that whooping-cough often causes a lymphocytosis of leukaemic proportions. It is possible that the relation between the monocytosis of monocytic leukaemia and glandular fever may be similar, thus bringing the monocytic series into line with the other series of leucocytes. At present there is no evidence as to the nature of the relation between this type of response and that of leukaemia.

Summary

1. A case of monocytic leukaemia and two cases of infectious mononucleosis (glandular fever) presenting different clinical and haematological characters are described.

2. In infectious mononucleosis mononuclear cells of types not found in normal blood appear. These cells are described, and it is suggested (by comparison with the cells in monocytic leukaemia) that some of them are immature monocytes and that others are probably lymphoid. These cells are very typical, and are of considerable value in diagnosing the condition.

3. The differential diagnosis between monocytic leukaemia and infectious mononucleosis is considered.

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REFERENCES

- Baader, E. (1922). *Dtsch. Arch. klin. Med.*, **140**, 227.
 Baldrige, C. W., Rohner, F. J., and Hansmann, G. H. (1926). *Arch. intern. Med.*, **38**, 413.
 Bunnell, W. W. (1933). *Amer. J. med. Sci.*, **186**, 346.
 Dameshek, W. (1930). *Arch. intern. Med.*, **46**, 718.
 Doan, C. A., and Wiseman, B. K. (1934). *Ann. intern. Med.*, **8**, 383.
 Downey, H., and McKinlay, C. A. (1923). *Arch. intern. Med.*, **32**, 82.
 Forkner, C. E. (1934). *Ibid.*, **53**, 1.
 Friedmann, U., and Elkeles, A. (1928). *Internat. Zbl. Ohren.*, **29**, 285.
 Gittins, R., and Hawksley, J. C. (1933). *J. Path. Bact.*, **36**, 115.
 Hall, B. E. (1930). *Fol. Haematol.*, **43**, 206.
 Hartfall, S. J. (1934). *Lancet*, **1**, 620.
 Hopmann, R. (1923). *Dtsch. Arch. klin. Med.*, **142**, 196.
 Israëls, M. C. G. (1936). *Acta med. scand.*, **89**, 124.
 Levine, V. (1934). *Fol. Haematol.*, **52**, 305.
 Longcope, W. T. (1922). *Amer. J. med. Sci.*, **165**, 781.
 Orr, J. W. (1933). *Lancet*, **1**, 403.
 Paul, J. R., and Bunnell, W. W. (1932). *Amer. J. med. Sci.*, **183**, 90.
 Sprunt, T. P., and Evans, F. A. (1920). *Johns Hopk. Hosp. Bull.*, **31**, 410.
 Tidy, H. L. (1934). *Lancet*, **2**, 180, 236.
 Whitby, L. E. H., and Britton, C. J. C. (1935). *Disorders of the Blood*, London, 428.
 — and Christie, J. M. (1935). *Lancet*, **1**, 80.
 Wiseman, B. K. (1936). *Ann. intern. Med.*, **9**, 1303.

The tenth congress of oto-neuro-ophthalmology will be held at Geneva under the presidency of Professor F. Naville from May 14 to 16, when papers will be read on the central vestibular tracts and their disorders by Professor Van Gehuchten of Louvain, and on topographical syndromes of these tracts by Professor J. A. Barre of Strasburg. Further information can be obtained from M. Edmond Barbey, 20, Rue du Général-Dufour, Geneva.

TERMINAL CASEATING TUBERCULOUS BRONCHOPNEUMONIA

IN WHICH THE DATE OF ONSET WAS KNOWN

BY

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(WITH SPECIAL PLATE)

In any subacute or chronic tuberculous lesion found at necropsy data are rarely available for fixing the time at which the pathological process began. As a rule this can only be inferred from the time at which signs or symptoms were first noticed. By how long the pathological changes had preceded these it is usually impossible to say. In the case which is here recorded it so happened that data were present for fixing within narrow limits the time of onset and consequently the rate of development of gross tuberculous lesions in the lungs.

Pathological Evidence

On August 20, 1936, a necropsy was made on a man, 45 years old, who had been a patient of the South Yorkshire Mental Hospital for nine years. The body was extremely emaciated. A large area of tuberculous bronchopneumonia was found occupying the upper and middle zones of the left lung, and there was a rather smaller area in the right lung in the same parts (see Special Plate, Figs. 1 and 2).

The presence of such gross and extensive lesions in the lungs came as a surprise, because although tuberculosis had been suspected in this man for some years, owing to his progressive wasting, yet at no time had any abnormal physical signs been found in the chest. Even as recently as July 2, 1936—that is, less than two months before death—not only had no abnormal physical signs been found on careful examination, but also an x-ray film of the chest had shown nothing definitely abnormal (Fig. 3). One must therefore suppose either that almost the whole of this extensive invasion of the two lungs had developed since July 2, 1936, or that there were at that date marked tuberculous changes in the lungs which not only gave rise to no obvious physical signs even when carefully sought for, but which also showed no evidence of their presence on x-ray examination.

It would be contrary to all our radiological faith to believe that any gross focus of tuberculosis could have been present in these lungs at the date at which this radiograph (Fig. 3) was taken. It therefore follows that almost the whole of the areas of solidification and cavitation found at the post-mortem examination must have developed during a period of some fifty days or less; how much less it is impossible to say.

Mention has already been made of the extreme emaciation of the body at death, and of the fact that this loss of flesh had been going on for six years, so as to cause repeated suspicion of tuberculosis. The explanation of this wasting is found in the records of the case. The man was a melancholic, with strong suicidal tendencies, and had acquired the habit of regurgitating a considerable part of every meal. Nothing could be done to prevent this. The wasting was thus due to gradual self-starvation, brought about by the psychosis.

Presumed History of Infection

The actual order of events would thus seem to be as follows. He probably received his tuberculous infection in early life, as most do, and was left with a latent focus which could not be recognized. There is a statement in his notes that he had influenza in November, 1918 (when one of the greater epidemic waves was present), and that after that, in May, 1922, he began to show mental symptoms, but no recognizable spread of tuberculosis took place either then or up to July, 1936.

It seems that when the wasting brought about by his self-induced starvation had reached a certain point the old original focus, no longer held in check, spread rapidly, involving a great part of both lungs within some fifty days. That there was a complete lack of resistance or reaction is shown by the fact that during the six months before his death the temperature, taken night and morning, was only above normal on four single occasions, and that during the last three weeks of life, when this rapid spread must have been taking place, it was never raised above normal at any time. This seems to be a rather striking example of an endogenous metastatic reinfection by the tubercle bacillus from within the body, due to severe loss of resistance. As Davidson (1935) states: "Such reinfection being commonly preceded by a lowered resistance on the part of the individual."

Indirectly the course of events in this man was of interest from another point of view—namely, as to whether he, like other persons with sufficiently lowered resistance, might have received a reinfecting dose from without. In mental hospitals and other institutions where numbers of persons are living together this possibility has always to be borne in mind, and special care is taken to remove all known cases of tuberculosis to a special block. This man had been bed-ridden for many months, and had not, during that time, been in contact with any known case of tuberculosis. The possibility, therefore, of reinfection from without was minimal. The most likely source of the sudden spread of tuberculosis in the lung seems to have been the caseating hilar gland found at the root of the right lung (Fig. 1). Possibly it had ulcerated through to a bronchus with a resulting "aspiration-pneumonia."

Notes of Case

The patient was admitted to the South Yorkshire Mental Hospital on July 2, 1929. He had had influenza in November, 1918, and had been showing mental symptoms since May, 1922. His father was alcoholic, otherwise the family history was negative. No abnormal physical signs were found in the chest on admission. He was suffering from melancholia, was depressed and apprehensive, said he was unfit to live, and was actively suicidal. His desire for death found expression in a habit of regurgitating food. After his meals he used to put his finger in his throat and induce vomiting. If restrained from doing this he became very resistive and threatened to do harm to himself. He lost weight steadily. In June, 1931, he weighed 9 st. 2 lb.; in February, 1932, he weighed 8 st. 12 lb.; in March, 1933, 6 st. 11 lb.; in February, 1934, 6 st. 7 lb.; and in June, 1936, 5 st. 5 lb. (See Chart.) It was impossible to induce him to retain his food.

On June 7, 1936, a complete physical examination was made, and the note on his condition reads as follows: "Heart sounds regular—no bruits. No lesion found in chest or abdomen. Remarkably free from bedsores, despite extreme emaciation." He had been in bed since July, 1930. On sunny days he was nursed on the veranda. There was no cough or sputum. His temperature was taken morning and evening, and was charted from February 19, 1936, until his death in August of that year. On only four occasions was the temperature above normal. During the last three weeks

of his illness there was no pyrexia at all. His pulse rate was between 60 and 70 throughout, and his respiration rate ranged from 18 to 22 per minute.

The differential white cell count, taken on June 29, 1936, showed: polymorphonuclears, 82 per cent.; lymphocytes, 17 per cent.; and large hyaline cells, 1 per cent. A specimen of his urine on June 9 contained a trace of albumin and an occasional granular tube cast. The faeces were tested for tubercle bacilli on June 8 and 15, with negative findings. The chest was radiographed on July 2, 1936. The report reads as follows: "Heart normal in size and position. Hila both slightly enlarged. The apices light up fairly well, the left

better than the right. There is slight fibrosis present over the whole of both lungs, but no active mottling anywhere." A note on August 16 stated: "Is slowly degenerating both mentally and physically. Appears very feeble at times—still vomits after his meals."

He died on August 20, and a post-mortem examination was made on the same day. The body was emaciated.

The pleura over the left lung was normal. The lower part of the upper and upper part of the lower lobes were solid. This area was clearly demarcated; it involved almost the total horizontal section of the lung, and measured 5 in. vertically. Many areas up to 1/2 in. diameter of caseation were present in the consolidation. These areas had resulted in softening and cavity formation, and there was a marked absence of fibrotic tissue. The cavities contained thick green necrotic material; a film of this material, stained by the Ziehl-Neelsen method, revealed a profusion of tubercle bacilli. A section of the consolidated area stained with haemalum and eosin showed a diffuse caseation of the involved area of lung. There was no overgrowth of fibrous tissue, and no giant-cell systems were seen. There were numerous alveoli containing only fibrin. The section stained by the Ziehl-Neelsen method showed numerous tubercle bacilli.

In the right lung there were areas of trefoil necrosis in the middle lobe and the lower inch of the upper lobe, but there was considerable spongy lung substance still present in the affected zone. Over the diseased area of this lung there were small patches of thickened pleura and a certain amount of comparatively recent pleurisy. There were also three small areas the size of a pea in this lung which had a little fibrous tissue reaction and a few giant-cell systems, and only in these small areas of the widely involved area in both lungs was the disease probably as much as a few months old. At the root of the right lung there was a solitary caseous peribronchial gland, half an inch in diameter; it was darkly pigmented and distended with caseous material, and appears to have been the primary focus. The heart and kidneys were much atrophied. There was no gross lesion of the stomach. There was no dissemination of tuberculosis throughout the body.

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REFERENCE

Davidson, Maurice (1935). *Manual of Diseases of the Chest*, London, p. 281.

C. E. H. TURNER: TERMINAL CASEATING TUBERCULOUS BRONCHOPNEUMONIA

FIG. 1.—Right lung on August 20, 1936.

FIG. 2.—Left lung on August 20, 1936.

FIG. 3.—X-ray of chest taken on July 2, 1936.

PHILIP J. GANNER: THREE SEPARATE CAUSES OF
ANTE-PARTUM HAEMORRHAGE OCCURRING SIMUL-
TANEOUSLY

Uterus opened three months after removal.