

BAGASSOSIS

AN INDUSTRIAL LUNG DISEASE

BY

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AND

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(WITH SPECIAL PLATE)

Bagasse is the term applied to broken sugar-cane after the sugar has been extracted. This formerly waste product of the sugar-cane industry is now employed in the manufacture of board which, besides being an easily worked and durable material, has the property of insulating any cavity lined with it against sound and temperature changes. The many possible applications of these properties in building are obvious. At the present time the material is in demand for war purposes. The bagasse arrives at the board-making factory in open bales, which are then "broken"—i.e., the raw material is further crushed into small fragments. When manufacture was first started in England the bale-breaking was carried out under water. The process was slow, however, and about two years ago machinery was devised for breaking the bales in a dry state that proved to be much more rapid but gave rise to a great deal of dust, some very finely divided, in the air near the machinery.

Bagasse, which is largely composed of fibre, contains about 1% of protein and 5 to 7% of silica. Some of its cellulose content is said to be present in a crystalline form. The rest of the processes through which the bagasse goes are wet, an initial washing probably removing all the protein content, until the finished board is dried in a heated press at a temperature of 200° F. in the centre of the board, which would destroy any remaining protein. It is then cut and trimmed by circular saws, a process which again gives rise to a small quantity of dust containing silica but no protein.

Directly after the installation of the dry bale-breaking machinery, cases of respiratory illness began to appear among the workers engaged in this part of the process—namely, the labourers who fed the bales into the machine and the engineer who supervised its construction and running. No cases of respiratory trouble had previously occurred in the factory. Those workers who are engaged in the cutting and trimming of the finished boards are never affected. As soon as it was realized that the dust liberated at the initial stage of manufacture was injurious, measures were taken to suppress it. These consisted of a water spray and a suction draught applied to the site of its generation, and since the application of them no further cases of illness have occurred among the workers. The clinical features of four of these cases seen and the investigations made upon them are as follows:

Case I

A labourer at the factory aged 19, this patient had always enjoyed good health. There was no history of any chest trouble or of tuberculosis in himself or his family. He began work in the factory in Feb., 1941, and was exposed to bagasse dust as he fed bales into the breaking-machine. Since the end of March, 1941, six weeks before admission to hospital on May 4, he had had increasing shortness of breath and for four weeks a cough with scanty sputum that was sometimes blood-stained. He had been unable to work for ten days before admission.

On examination he was very dyspnoeic at rest, being hardly able to speak, but had only moderate cyanosis. There was occasional cough with a scanty, frothy, sticky, muco-purulent sputum which was sometimes streaked with blood. There was no clubbing. The heart was normal and the blood pressure 140/60. The chest showed poor movement, with rather less on the right side than on the left. The trachea was central. The percussion note was impaired, with distant bronchial

breathing in small areas at both bases. Vocal fremitus and vocal resonance were diminished at the right base. A few rales were present at the right base and scattered fine crepitations in all other areas. The abdomen, central nervous system, and urine were normal. A radiograph taken on May 5 (Plate, Fig. 1) suggested bilateral bronchopneumonia of influenzal type.

Progress.—The temperature, which was 100.6° on admission, fell to normal in two days, at which it remained until the ninth day, when remittent fever began, lasting one week. The rest of the course of the illness was afebrile. The pulse rate varied between 104 and 144 and the respirations from 24 to 48 a minute until the patient was well. Cyanosis with extreme dyspnoea persisted for three weeks, then both gradually subsided over the next four weeks. A radiograph taken on June 19 showed an increase in the area of consolidation (Fig. 2). The dyspnoea, which never had an asthmatic quality, was markedly relieved by oxygen, which was kept up continuously by nasal catheter for 51 days. Sulphapyridine appeared to have no influence on the course of the disease. Signs of patchy consolidation persisted in both lower lobes, varying in location and intensity for 7½ weeks, with rales frequently in other areas also. Finally the dyspnoea gradually ceased, oxygen was dispensed with for increasingly longer intervals, the chest began to clear by July 5 (Fig. 3), and the patient became subjectively well. He was followed up as an out-patient, remaining symptom-free, with further resolution of the lung lesions (Fig. 4), and five to six months after this last film was taken was admitted to the Navy.

The sputum was always scanty, muco-purulent, and at times blood-stained. The bacterial flora was varied, containing *M. catarrhalis*, *Str. viridans*, and fusiform bacilli. *Staph. aureus* was grown on one occasion, and on another pneumococcus Type 14. Tubercle bacilli were not found. The cells in the sputum were epithelial, polymorphonuclear, and lymphocytic. Eosinophils were never present.

The blood count on admission was: red cells, 4,610,000 per c.mm.; haemoglobin 78%; colour index 0.87; leucocytes 11,300 per c.mm.—neutrophils 83%, lymphocytes 14%, monocytes 3%, and no eosinophil cells. Subsequent white counts gave 9,200 and 9,300 cells, with normal differential counts containing 3% and 1% of eosinophils respectively.

Case II

This patient, an engineer aged 37 who had previously enjoyed good health except for an attack of influenza in 1936, had no history of chest illness in himself or his family, none of whom had had tuberculosis in any form. In March, 1939, he started work in the factory, where from Aug., 1939, to June, 1940, he was in intermittent contact with bagasse dust. From June, 1940, to Sept., 1940, he was in continuous intimate contact with the dust, superintending the construction and running of machines for the dry crushing process. In Sept., 1940, he became ill with a chest complaint which was called pneumonia, and was off work for 10 weeks. The symptoms of his illness corresponded to those of Case I—namely, shortness of breath, cough, and scanty sputum. During convalescence he was sent by his doctor, under whose care he had remained at home, to see the Tuberculosis Officer, who reported no P.T. In Nov., 1940, he returned to work, and in December began to be short of breath again. This shortness of breath rapidly and steadily increased so that by Jan., 1941, he was out of breath at the least exertion and was able to work one day only in every three. On Jan. 17, 1941, he attended hospital as an out-patient. On examination there was seen to be early clubbing of the fingers. The chest was kyphotic, with many fine rales in all areas of both lungs anteriorly and at both bases. Physical examination was otherwise normal. A radiograph taken on Jan. 18 (Fig. 5) showed a few apical scars and a little fibrosis in the right midzone. By May, 1941, his shortness of breath was extreme and painful, and he would collapse in the street from walking slowly. In one of these attacks of collapse he was admitted to another hospital, where a radiograph was thought to reveal bilateral apical tuberculosis. After this he continued at home with troublesome shortness of breath until he was admitted to hospital on Nov. 22, 1941.

On examination there was marked dyspnoea at rest with orthopnoea but no cyanosis. Clubbing of the fingers was present. The spine was kyphotic and the thorax rigid, and upon comment being made on this the patient stated that he had been becoming more "round-shouldered" since Jan., 1941. The trachea was central and the percussion note impaired at the left apex both in front and behind, with bronchial breathing and bronchophony. Crepitations were present throughout all areas of the left lung and at the right apex. The heart was

L. I. M. CASTLEDEN AND J. L. HAMILTON-PATERSON: BAGASSOSIS, AN INDUSTRIAL LUNG DISEASE

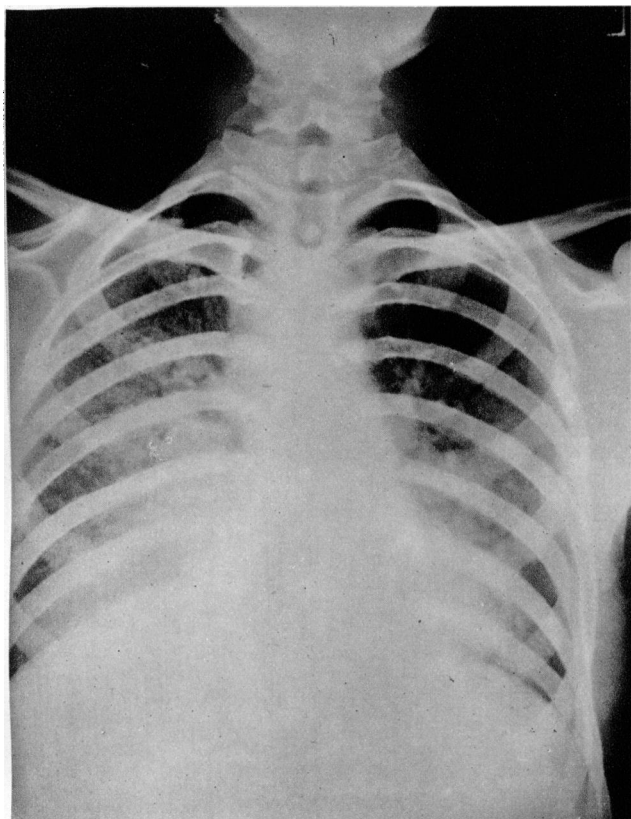


FIG. 1.—Case I (May 5, 1941). Bilateral basal mottling of bronchopneumonic type with enlarged right hilum gland.

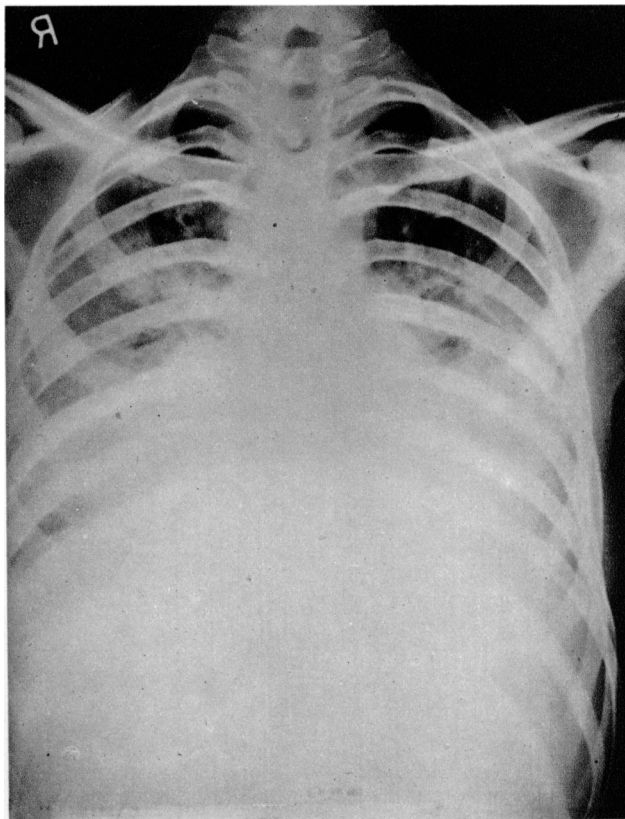


FIG. 2.—Case I (June 19, 1941). Extension of the lesions.

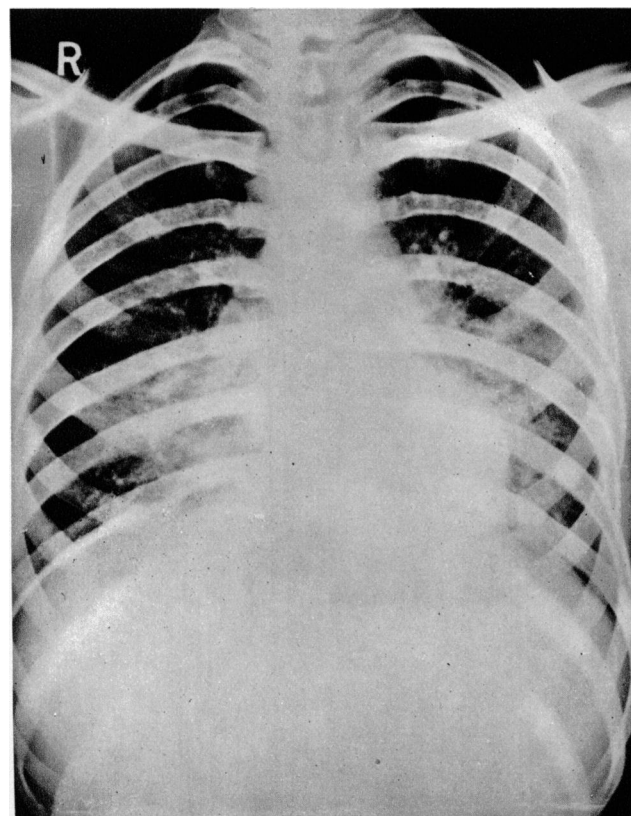


FIG. 3.—Case I (July 5, 1941). Resolution starting.

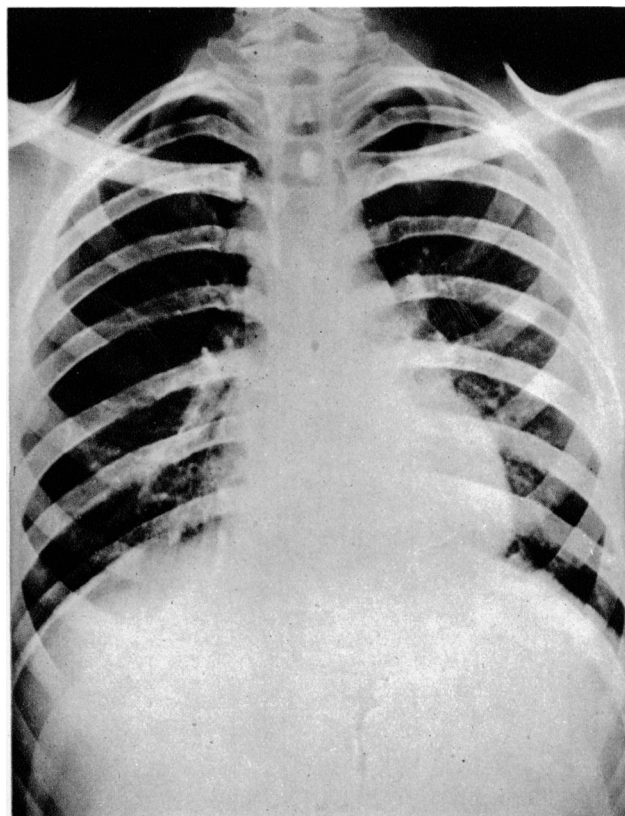


FIG. 4.—Case I (Aug. 29, 1941). Complete resolution of lung lesions except for a little basal striation.

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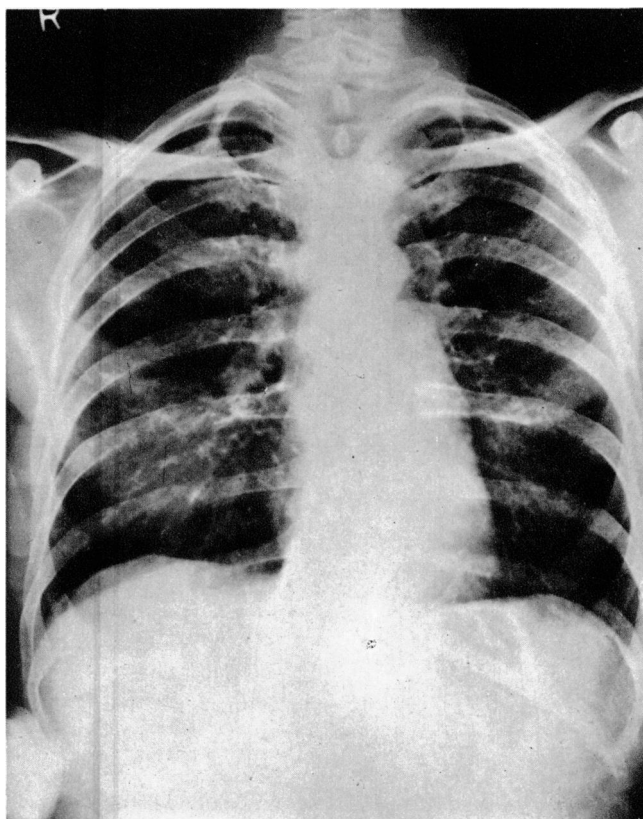


FIG. 5.—Case II (Jan. 18, 1941). A few apical scars and a little fibrosis in the right midzone.

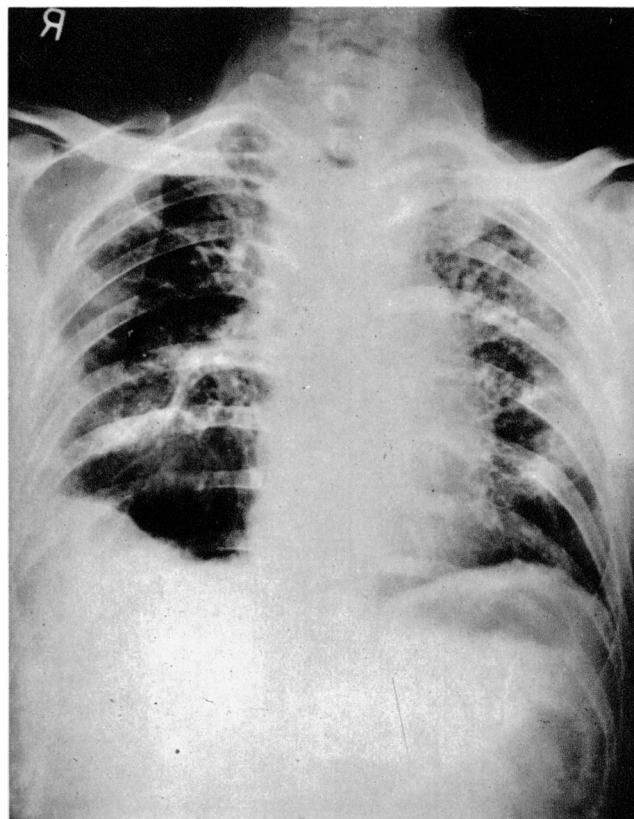


FIG. 6.—Case II (Dec. 5, 1941). Wide upper mediastinal shadow; irregularity of contour of right diaphragm with pleural thickening in costophrenic angle; streaky opacities of fibrotic appearance in both lungs.

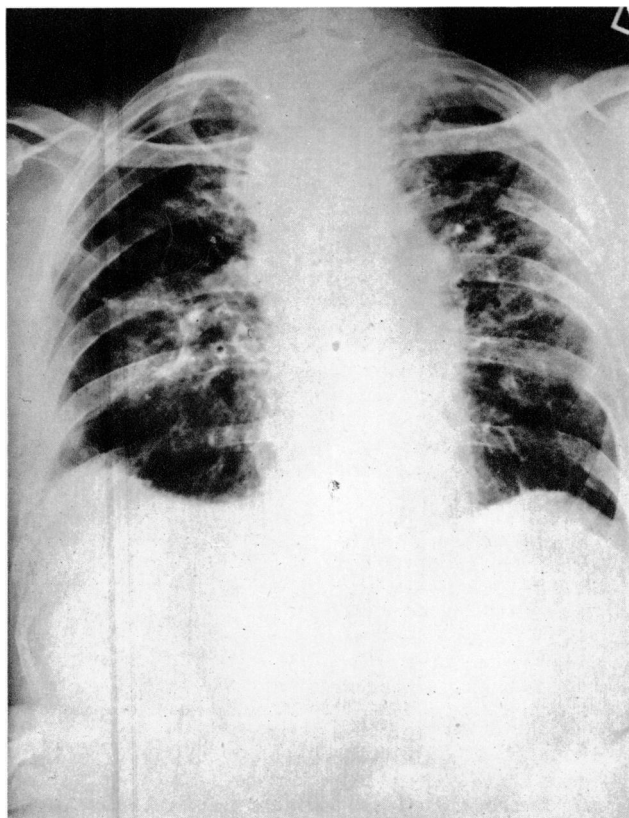


FIG. 7.—Case II (June 24, 1942). No radiological evidence of resolution.

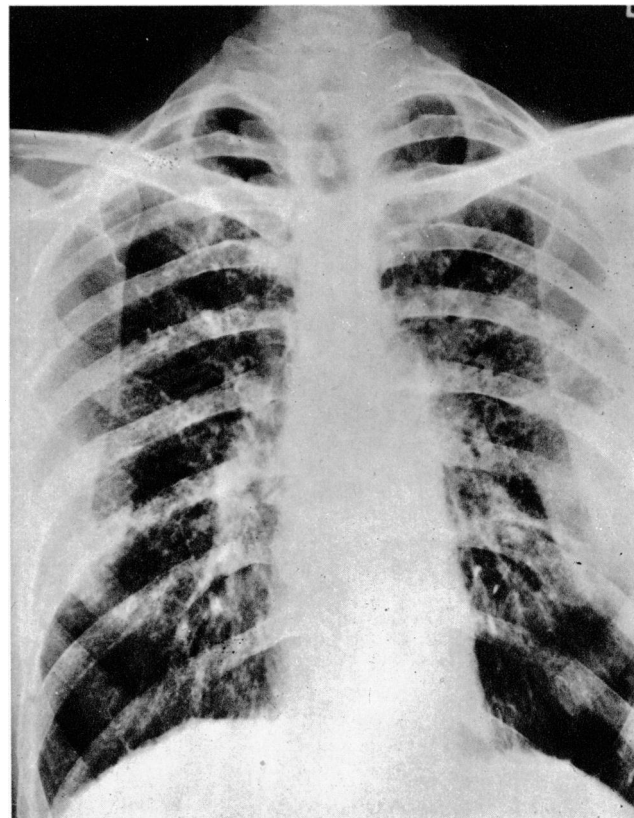


FIG. 8.—Case IV (June 15, 1942). General streaky opacity throughout both lungs.

normal and the blood pressure 110/70. The abdomen, central nervous system, and urine were normal. There were no signs of congestive cardiac failure.

The sputum was mucoid, viscid, scanty, but never blood-stained. The cells in it were polymorphonuclears and lymphocytes. Eosinophils were not seen. The bacterial flora was mixed, containing the usual mouth organisms, with occasionally haemolytic streptococci and pneumococcus Type 23. Tubercle bacilli were not found on repeated search. A special search was made for yeasts, moulds, and fungi, but none were seen. Radiographs taken on Dec. 5 (Fig. 6) showed appearances very much like bilateral pulmonary tuberculosis, and that the lung lesions had advanced since he had been seen as an out-patient. A blood count showed: red cells, 4,590,000 per c.mm.; haemoglobin 94%; colour index, 1.02; leucocytes, 10,200 per c.mm.—polymorphs 51%, lymphocytes 32%, monocytes 6%, eosinophils 11%.

Progress.—The patient was afebrile throughout his stay in hospital except for very occasional temperatures of 99° to 99.6°. The pulse rate was 80 to 100 and respirations 20 to 24 a minute. The dyspnoea gradually decreased and the signs of consolidation in the left upper lobe disappeared, but rales and crepitations persisted throughout the left lung, and were usually present at the right apex or base. On discharge on Feb. 15, 1942, he was able to get about with less dyspnoea than formerly, and has attended as an out-patient since with decreasing dyspnoea but with increasing kyphosis and occasional dragging pain in the lower part of his chest on the left side. He continues to attend as an out-patient and is unable to work. Radiographs taken on Feb. 2, March 16, and June 24, 1942 (Fig. 7) are all identical. They show some resolution of left upper zone shadows between Dec. 5, 1941, and Feb. 6, 1942; but thereafter the widening of the mediastinal shadow in the upper part, the peculiar streaky opacities in both lung fields, and the peripheral basal pleural thickening have remained unchanged.

Case III

A labourer aged 40 attended as an out-patient, complaining of shortness of breath. He was working at the same machine as Case I, and after one month at the job began to be short of breath, had a cough with scanty sputum and chest pains. He was off work intermittently, and was finally transferred to an open-air part of the factory, since when he has had less cough but his shortness of breath has continued. He had had no previous chest trouble. There was no history of tuberculosis in his family.

On examination he was slightly dyspnoeic. There was no clubbing of the fingers. The chest movements were poor but equal, with impairment of the percussion note at the left base and in the left axilla. The breath sounds were diminished in these areas. There were no added sounds. The heart, abdomen, and central nervous system were normal. The blood pressure was 140/90. The blood count was: red cells, 4,560,000 per c.mm.; haemoglobin, 100%; colour index, 1.1; leucocytes, 3,000 per c.mm.—neutrophils 43%, eosinophils 3%, basophils 1%, lymphocytes 49%, monocytes 4%. A radiograph of his chest demonstrated no lung lesion.

Case IV

A labourer aged 44 attended as an out-patient, complaining of shortness of breath. Sixteen months ago he had begun work at the factory and was exposed to bagasse dust when feeding the bale-breaker. A year ago he began to be short of breath and to have a cough without any sputum. He was able to work for increasingly shorter times before difficulties with his breath compelled him to stop. Finally he could manage to work only one to three days in each week. He was then transferred to outdoor work, since when he had been getting better but was still very short of breath on exertion.

He had had pneumonia in 1914, from which he had made a good recovery, but had had no other illnesses. There was no family history of tuberculosis. On examination there was moderate cyanosis with dyspnoea at rest. There was no clubbing of the fingers. The percussion note was impaired at the right apex, with diminution of the breath sounds in this area and at the right base. The breath sounds were normal in character. A few rhonchi were present throughout both lungs. The heart, abdomen, and central nervous system were normal. The blood pressure was 110/70. The blood count was: red cells, 5,010,000 per c.mm.; haemoglobin, 108%; colour index, 1.08; leucocytes, 6,600 per c.mm.—neutrophils 55%, eosinophils 1%, lymphocytes 40%, monocytes 4%. A radiograph taken on June 15, 1942, showed a general streaky opacity throughout both lungs (Fig. 8).

Discussion

The clinical appearance of these patients suggests an acute inflammatory lung disease with urgent and extreme dyspnoea as the presenting symptom but with little or no febrile reaction. On admission Case I was thought to be suffering from bilateral bronchopneumonia, which appeared to be of the influenzal type, but there was no epidemic of influenza at the time and he had already been ill for six weeks before admission—two facts which were against this diagnosis. There had been no case of psittacosis in this country for several years, and he had had no contact with birds of the parrot species. The alternative seemed to be that the lesion was tuberculous or was a form of chronic pneumonia (Scadding, 1936). The radiological appearances were more those of an influenzal bronchopneumonia than of a tuberculous lesion: tubercle bacilli were never recovered from the sputum, and he had long periods when he was afebrile with persistence of his symptoms and signs, so that some form of chronic pneumonia seemed the most likely diagnosis; but here again the absence of pyrexia was difficult to explain. His history of being employed in a dusty occupation naturally led to further inquiry concerning this. He said that some of his workmates had complained of cough and shortness of breath; but the medical officer of the works stated that although several of the employees had had respiratory illnesses about that time he had no reason then to connect them with the work upon which the men were engaged. The eventual complete resolution of the lesions with the return of the patient to normal health seemed to indicate their inflammatory nature, the aetiology of which remained obscure. There the matter rested until Case II was admitted to hospital. He also had the presenting symptom of extreme dyspnoea, and on his physical signs and the radiological appearances was thought at first to be suffering from bilateral active tuberculosis, but tubercle bacilli were persistently absent from his sputum, and he also had long periods when he was afebrile in spite of the apparent activity of the disease. The presence of persistent extreme dyspnoea called to mind the appearance of Case I, and upon inquiry it was found that his occupation, which was given as that of an engineer, concealed the fact that he had not only been working in the same factory as Case I but also had been exposed to the same dust. Contact was then made with the Factory Department of the Ministry of Labour and National Service to ascertain if workers at this factory were subject to an industrial dust disease. Dr. E. M. Middleton, one of the medical inspectors, kindly came to see Case II in consultation; he said that the illness was similar to others which had occurred in workers at the factory, and was probably bagassosis (or, as we prefer to call it, "bagassosis"), a disease first recorded by Jamison and Hopkins (1941). These writers described an acute illness occurring in a young negro labourer who had been employed unloading bagasse at a board-making factory in America. His work was very dusty, and his illness was characterized by marked dyspnoea, cough with scanty sputum which was sometimes blood-stained, and physical signs and radiological appearances suggestive of bilateral bronchopneumonia. He made a complete recovery, and the lung lesions, after passing through a stage of "miliary mottling" radiologically, resolved, the clinical and x-ray appearances described being parallel to those of Case I.

Jamison and Hopkins were able to grow a fungus on two occasions from a concentrate of a 24-hour specimen of sputum in their case, and advanced the view that it was a fungous disease of the lungs. They reported their case at a medical meeting, at which Fasting (1941) stated that he had seen three or four similar cases, which he thought were due to a form of silicosis—a view which is shared by J. Freeman in this country (personal communication).

In the present series Case I had an acute illness with complete resolution of the lung lesions clinically and radiologically. Case II had an acute illness which was called pneumonia, which resolved clinically, and which, although not studied radiologically, must have left few if any traces in the lungs for him to be passed as normal at a tuberculosis clinic soon afterwards. He returned to fresh exposure to the dust, becoming ill again with lung lesions, which advanced between the time of his first being seen and his admission to hospital (Figs. 5 and 6) and which to date show no sign of resolution (Fig. 7), although he has improved symptomatically. Case III, seen once only

as an out-patient, would appear to be in the stage of resolution ; and Case IV, also seen as an out-patient, may be going to resolve or might progress to the fibrotic type of lesion seen in Case II.

It would appear that exposure to bagasse dust is capable of initiating a pathological process in the lungs, the complete resolution of which is possible with a return to normal (Jamison and Hopkins, 1941) as in Cases I and III, or which may progress to a lesion the nature of which is probably fibrotic—Cases II and IV. In this connexion it is noteworthy that Cases I and III had least and Cases II and IV most exposure to the dust in point of time.

Although bagasse contains silica in an amount capable of producing silicosis, the acute pneumonic phase of the illness which these workers experience is unlike any known form of silicosis previously described. Further, the men who at the final stage of the process are also exposed to a silica-containing dust are never ill. A pathogenic organism has not been isolated from the sputum in the acute stage of the disease, and an examination of 15 consecutive 24-hour specimens of sputum from Case II failed to isolate any yeast or fungus. It seemed possible that an allergic factor might be responsible for the disease, the workers becoming sensitized to a protein present in the dust of bagasse. Extracts for skin tests were therefore made as follows:

Twenty grammes of coarsely crushed bagasse was placed in 200 ml. of menstruum with 0.25% tricresol added, and allowed to stand five days with occasional shaking. Four extracts were made in: (1) N/10 NaOH; (2) N/10 HCl; (3) normal saline; (4) 30% alcohol in distilled water. After five days all were passed through a Seitz filter. Extracts 1 and 2 were neutralized with equal quantities of N/10 H₂SO₄ and N/10 NaOH respectively. In both a flocculent precipitate formed. This was used as a suspension. The others (3 and 4) were used as clear yellow fluids as they came through the filter. For each antigen a control was made of menstruum with 0.25% tricresol. The antigens so prepared were tested on Case II by the scratch technique, when all gave negative results. They were then retested by intradermal injection of 0.2 ml. Extracts 1, 2, and 4 all gave a wheal maximal in half an hour and thereafter subsiding, and a flare maximal in 36 hours measuring 35, 30, and 40 mm. respectively. The normal saline extract No. 3 gave no wheal, but a flare developed which reached a maximum of 40 mm. in 36 hours. It was also painless, whereas Nos. 1 and 4 were painful. Four laboratory workers, all of whom had handled bagasse and crushed it with the liberation of a quantity of fine light dust, were then tested with the saline extract by intradermal injection, and all gave negative results. Nine volunteers from the nursing staff who had never been in contact with bagasse were also tested, with negative results. Six employees at the factory were also tested. Four of these gave negative results; they had worked at various places in the factory but not at the bale-breaking machine, and two were as strongly positive as Case II, and were Cases III and IV. Of 20 individuals tested, therefore, the only three who gave positive reactions were Cases II, III, and IV. A similar saline extract was then made of light dust which had settled on ledges in that part of the factory where the board is sawn. This, as stated above, contains no protein, which is removed in process of manufacture. Tests with this extract made on Cases II, III, and IV all gave negative results.

It seems certain, therefore, that whole bagasse contains an antigen soluble in normal saline to which workers who inhale the dust can become sensitized. The acute phase of bagassosis is possibly an allergic response in the lungs to this antigen with, but more probably without, an infective element. The nature of the chronic pathological process is obscure. It might be due to: (1) a form of silicosis which supervenes upon the allergic phase during or after the latter's resolution; (2) a response on the part of the lungs to crystalline cellulose; (3) a chronic process of fibrotic nature occurring in tissues oedematous from their allergic response to the antigen. The conception of chronic allergy is foreign to pathological thought at the present time, but fibrosis is known to occur in lymphatic tissue oedematous for some time from any cause, and this explanation of the lung changes in these cases might be subsequently found to have a parallel in some of the more

common diseases of chronic nature whose aetiology is at present obscure, such as rheumatoid arthritis.

Opportunities for the study of the morbid anatomy of the condition have not yet occurred, and attempts to reproduce the disease in experimental animals have so far failed.

Summary

An industrial lung disease is described and a theory of its causation which may link it with more common chronic diseases is advanced. It is suggested that the disease should be called bagassosis and not bagasscosis, as it has formerly been named.

Our thanks are due to Dr. J. N. Deacon, superintendent of the hospital, for permission to publish this paper, to the members of the pathological and nursing staffs who acted as normal controls, and to the management of the factory for details of their manufacturing process and facilities freely granted for visiting the factory and performing tests upon some of their employees.

REFERENCES

- Fasting, G. F. C. (1941). *New Orleans med. surg. J.*, **93**, 582.
Jamison, S. C., and Hopkins, J. (1941). *Ibid.*, **93**, 580.
Scadding, J. G. (1936). *Brit. J. Tuberc.*, **30**, 186.

INTRAMURAL INTESTINAL HAEMORRHAGE

BY

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The following is an account of an unusual case occurring in a woman who was operated on because of signs of an acute abdomen. The peritoneal cavity was found to be full of blood, the source of the haemorrhage being traced to a haematoma of the wall of the caecum which had ruptured.

Case Report

The patient, a married woman aged 51, was admitted to Newton Abbot Hospital on Feb. 1, 1941. There had been no former illness of note. On Jan. 30 she had had sudden acute pain over the lower abdomen, had vomited, and had had a little diarrhoea. The temperature and pulse were slightly raised, and there was some abdominal tenderness but no rigidity. On Jan. 31 the condition was unaltered except that no further vomiting had occurred and the diarrhoea had stopped. On Feb. 1 the pain increased, settling in the right iliac fossa, and the right lower abdominal quadrant showed both tenderness and rigidity. The temperature was 99° F. and the pulse rate 90. In the afternoon the temperature rose to 100° F. and the pulse rate to 120. Acute appendicitis was diagnosed (pelvic variety), and it was decided to do a laparotomy, a right paramedian incision being made on account of the patient's sex and the unusual features of the case.

Findings at Operation.—The peritoneal cavity was full of blood, as in a case of ruptured tubal pregnancy. On exploration, however, this cause was excluded, but in the right iliac fossa a lump was found which seemed to be attached to the caecum. As the latter was mobile it was possible to deliver it, along with the tumour, through the abdominal wound. It was then seen that the appendix was thickened and inflamed and that there was a considerable deposit of fat in and around the meso-appendix. A groove separated its base from the tumour, which was a plum-coloured swelling, measuring 2 by 1½ in., situated on the lateral wall of the caecum (see illustration). Its appearance was that of a subserous haematoma, and the site of escape of the blood into the peritoneal cavity was indicated by a ragged tear in the serous coat, 1½ by 1 in. in size. On section the interior of the mass was found to consist of blood clot, and careful examination revealed no perforation into the lumen of the bowel. Around the tumour and beyond its confines the caecal wall was normal.

Operative Treatment.—There was enough space between the ileo-caecal valve and the base of the appendix to allow of resection of that part of the caecal wall which contained the haematoma and the base of the appendix. This was done, and the caecal wall was repaired with layers of sutures, the abdomen being closed without drainage. Except for a slight rise of temperature for 3 days, convalescence was uneventful and the patient made a good recovery, leaving hospital on Feb. 19.