BRONCHIOLITIS RESULTING FROM THE HANDLING OF BAGASSE

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Bagasse is sugar cane after the sugar has been extracted. Because of the toughness of its fibres and their good insulating properties it is used for making boards for interior decorating and for thermal insulating purposes. The sugar cane which causes for this purpose is grown in Louisiana and shipped to this country in bales. When the bales are packed in America the bagasse contains 4 per cent. of glucose, but on arrival in England it is sugar free.

Until the outbreak of war the bales were broken by a wet process: they were placed under water in a soak pit, broken, and then carried by a conveyor belt from the small house in which this process was carried out to the main factory for processing. At the end of 1939, in order to save shipping space, the bales were packed more firmly and it proved no longer possible to break them in this wet process. A machine, known as a shredder, was therefore introduced to break the bales. This consisted of a wheel with multiple blades which revolved inside a case. The bales were first broken with a pick-axe and the sections then thrown into the case. The bagasse was thus shredded by this dry process and the small pieces were carried by the conveyor belt as before. This process proved to be very dusty. Bagasse, as found at the factory, consists mostly of cellulose fibre through some of the cellulose is present in crystalline form. It also contains 1 per cent. of protein. Nagelschmidt found that bagasse gave 3 per cent. of ash on ashing at 400° C. By alternating treatment of this ash with cold dilute acid and hot dilute alkalies, over 80 per cent. could be dissolved. This dissolved portion contained the amorphous silica which came to about 50 per cent. of the total ash. The residue contained much quartz. Microscopic and x-ray diffraction analysis put the quartz at about 3–5 per cent. of the total ash. This amorphous silica formed roughly 1–2 per cent. of the bagasse and quartz 0.1–0.2 per cent.; many of the quartz particles were 20–30μ in size. It was most improbable, therefore, that any risk of silicosis could arise in handling bagasse. Bagasse was also found to contain many fungi. J. T. Duncan examined samples and also dust from the shredder room. He found many fungi present, and estimated that one gramme of the airborne dust contained 240 million fungal spores. These consisted of the uncultivable teliospores of fungi of the order Pucciniales (Rusts), as well as the conidia of many species of saprophytic fungi. About 20 different species were isolated in culture, including Paecilomyces variotii, Aspergillus fumigatus, A. niger, A. terreus, and A. candida, Trichoderma lignorum, Monilia sitophila, Aleurisma sp. and species of Penicillium, Mucor, Rhizopus, etc.

In 1940, after the installation of the dry bale breaking machinery, cases of respiratory illers began to appear among employees working the shredding machine and breaking the bales. These cases were observed by Lloyd (1940) at the Brompton Hospital. He considered that he was dealing with an unusual kind of pneumonia which was related to bagasse, but did not think that there was sufficient evidence to establish the aetiology. He consulted E. L. Middleton, H.M. Medical Inspector of Factories who advised that the machinery should be modified by a jet of water being directed on to the wheel to allay the dust. This advice was carried out in 1941, and until 1944 no further case of respiratory disease which could be attributed to the bagasse occurred in England.

Review of Literature

Jamison and Hopkins (1941) described the case of a young negro labourer who was employed in 1940 unloading bagasse from a ship at a board-making factory in New Orleans. This was a very dusty occupation. He developed an acute febrile illness with purulent diabetes, cough and scanty blood-stained sputum. Physical signs showed râles scattered throughout both lungs. X-rays showed 'miliary mottling' through both lung fields, but these cleared in the course of 2 months, leaving a normal healthy picture. The patient recovered completely. From a concentrate of a 24-hour specimen of his sputum a fungus was grown on two occasions. The authors, however, while stating that in their opinion this was the cause of the illness, fail to state the nature of the fungus.

Castleden and Hamilton-Paterson (1942) reported four case of 'bagassosis,' an industrial lung disease. The first was a boy of 19, who after working for six weeks in the spring of 1941, developed a bilateral pneumonia which radiologically showed an extensive consolidation in both lungs with enlarged mediastinal glands. The appearances did not show the typical miliary mottling of the syndrome associated with bagasse, but the process may have become confluent in this particular case, though it is impossible to distinguish from the atypical pneumonia which was prevalent in that year, and which has been well described by Drew, Samuel and Ball (1943), among others. Case 2 was an electrical mechanic aged 37, who was not himself engaged on
the work of breaking the bales. The machinery, however, was new and there were frequent breakdowns. He therefore spent considerable time in the house effecting repairs. X-ray of his chest in 1940 showed no abnormality beyond a catarrhal bronchitis. However, since that time he has developed an extensive progressive fibrosis of both lungs. He subsequently died in 1944 and an account of the necropsy is given later in this paper. (Case 12.)

Case 3 showed no radiological changes in the lungs, and case 4 was a man aged 44 who had previously worked for 17 years in the Durham coalfield and showed the x-ray appearances which have been termed reticulation by Hart and Aslett (1942). Castleden and Hamilton-Paterson also described skin tests which they had carried out on some of the workers. These showed that some developed a skin sensitivity to extracts of bagasse. Sodeman and Pullen (1944) repeated these tests in persons exposed to bagasse, and in a group who had not been exposed. They failed to find a negative reactor in either group. Castleden and Hamilton-Paterson therefore left the disease in a very obscure position, especially as the third and fourth cases show no disease that can be clearly attributed to bagasse and the fourth man had a pneumonokoniosis caused by his work as a collier.

A further case was reported from New Orleans in 1944 by Jamison, Bryan and Day in a negro man aged 32, who had been loading railway freight cars for a month. He developed dyspnoea and x-ray of his chest revealed infiltration with a miliary appearance which cleared completely in the course of four months. No fungi were found in this case.

Finally, Sodeman and Pullen (1943) gave an account of a fatal case and (1944) an account of 11 further cases. They say that shortness of breath was invariably the chief complaint, but that cough was an early and important symptom. In four instances haemoptysis occurred: it was variable in amount, but lasted only a few days. The sputum was scanty and mucoid. Intermittent fever with temperature rising to 102°F. was observed and persisted up to three or four weeks. X-ray examination of the chest showed a miliary mottling throughout both lungs, most dense in the hilar areas in all cases; White cell counts were over 10,000 in 10 instances with a polymorphonuclear leucocytosis. Needle biopsy specimens were obtained from the lung of one patient in the sixth week of the disease,
and sections were taken at necropsy from the lung of the patient who died. These showed pulmonary tissue with several 'spicules' of an irregular foreign material embedded in it. There was a fibroblastic reaction of the interstitial tissue of the lung. There were many large cells with a foamy cytoplasm in the alveolar spaces. The foreign bodies were microscopically similar to bagasse, and under the polarizing microscope these 'spicules' were seen to rotate the plane of polarized light; many smaller scattered pieces with an average diameter of 2–8\(\mu\) were also seen.

**Present Investigation**

An attempt has been made to ascertain what has happened to all the men who worked on the bagasse shredder at the factory in England. It was discovered that in 1940 14 men were employed on this machine for continuous periods of more than 3 days; in 1941 20 men, and in 1942 10 men. These men worked in pairs for two weeks by day, followed by a two weeks' night shift. The work was considerably more dusty at night since blackout regulations necessitated the closing of the large doors to the shed which housed the machine. Of the 14 men employed in 1940, 13 were traced; of the 20 in 1941, 18 were traced; and of the 10 in 1942, 8 were traced. As the machine was in an experimental stage in 1940 the assistant works manager, the chemist, the personnel manager, an electrical mechanic and a carpenter also spent a considerable amount of time in the shed. All five of these have been traced and examined. Of 13 men traced who worked on the shredder in 1940 7 showed a characteristic acute respiratory illness, similar to that already described by Lloyd at the Brompton Hospital and the American workers; and of the 8 men employed during the first three months of 1941 3 showed signs of this illness.

Thus, out of 21 men employed on the shredder in a period of 15 months, 10 (47.5 per cent.) developed the illness. The onset of symptoms usually occurred after the men had been working on the machine for 8 weeks. The disease manifested itself as an acute febrile illness with extreme shortness of breath, cough with scanty black, stringy sputum and occasional haemoptysis. Signs were scattered throughout both lungs, and x-rays of the chest showed miliary shadows throughout both lung fields. The appearances

![Figure 2](http://oem.bmj.com/first-published-as-10.1136/oem.3.2.64.on-1-April-1946.Downloaded-fromhttp://oem.bmj.com.on-January-3-2021.by-guest.Protected-by-copyright.)

**FIG. 2.—**Case 1, three years after the acute illness.
were therefore those of an acute bronchiolitis. The patient's symptoms gradually improved over a period of six weeks, at the end of which time he had recovered and skiagrams showed the lung fields to be quite clear. One patient in 1941 died after 25 days' illness, but unfortunately no necropsy was performed. Except for the electrical mechanic, the men showed no evidence of disease in their lungs at this time. The electrical mechanic (case 2 Castleden and Hamilton-Paterson, case 12 this paper) had repeated acute respiratory illnesses which progressed to a chronic condition. He finally died in 1944, and necropsy revealed chronic bronchiolitis and bronchiectasis. These cases give strong evidence that a specific disease manifesting itself by acute bronchiolitis, collapse, and pneumonia has occurred in the lungs of men who handle bagasse. The case histories of the ten men described above who showed specific disease follow.

Case 1. G. J., aged 28. He had worked for one year in the South Wales coalfield, three years in a rubber factory, and six years in the regular Army before joining the firm handling the bagasse in June 1938 as a labourer. He worked on the shredding machine from December 1st, 1939, to January 29th, 1940, when he was taken ill. He was admitted to the Brompton Hospital on March 16th, 1940, under Dr. W. E. Lloyd, complaining of shortness of breath, cough with purulent sputum, which was streaked with blood on one occasion, sweating and loss of weight. On examination he had a fever to 101° F., which persisted for three weeks. There were impaired percussion note and many coarse rales scattered throughout both lungs. X-ray of his chest showed a diffuse mottling with some confluent areas scattered throughout both lungs. There was considerable hilar enlargement (fig. 1). Further x-rays taken on April 15th, May 6th and July 20th showed that the process rapidly cleared. On July 15th, 1943, he was well, but still suffered from some shortness of breath and cough. He had no abnormal physical signs and x-ray of his chest revealed no abnormality (fig. 2).

Case 2. W. D., aged 65. He had worked as a newspaper roundsman, a dock labourer, in the Merchant Navy, in the Army, and as a builder's labourer before joining the firm in September 1937. He worked as a labourer until December 1939, but in that month and January 1940, when he was taken ill, he worked on the shredding machine. He was admitted to the Central Middlesex Hospital under Dr. H. Joules on April 25th, 1940, complaining of shortness of breath, cough with a
considerable amount of sticky sputum, and progressive loss of weight. On examination his apex beat was not displaced, his pulse rate 88, blood pressure 150/105. There was slight thickening of the peripheral arteries and his aortic second sound was accentuated. He was cyanosed and dyspnoeic; the percussion note was normal and the breath sounds vesicular. Crepitant râles and occasional rhonchi were heard all over the chest. His sputum was negative for tubercle bacilli and lung fibres. The blood sedimentation rate was 40, and his blood count showed no abnormality. X-ray of his chest on April 27th, 1940, showed a diffuse miliary mottling scattered throughout both lung fields. By May 24th, 1940, this had substantially cleared, and on June 4th, 1940, he was discharged. Further x-rays taken on September 5th, 1940, and July 15th, 1943, revealed no abnormality, although he still complained of a severe winter cough with thick black sputum, and on examination there were scattered crepitations throughout both lungs.

Case 3. T. H., aged 29. He had worked as a pit-head worker in Scotland for 5 years, and had then been unemployed until he joined the firm on January 21st, 1940. He worked as a labourer until March 16th, 1941, and then worked on the shredding machine until he was taken ill on April 27th, 1941. He was extremely short of breath, and had a cough with black stringy sputum; no haemoptysis and no pain. He was in bed for 3 weeks with a temperature up to 103°F for 3 days. An x-ray was taken of his chest at Wembley Hospital on May 29th, 1941, and this showed mottled opacities scattered throughout both lungs, their frequency being greater on the right side. On September 23rd, 1943, he was quite well, working at the degreasing of metal sparking plugs. He had no symptoms, no abnormal physical signs, and an x-ray showed no evidence of disease in the lungs.

Case 4. A. W., aged 54. He had worked as a farm labourer and in the Army until he joined the firm on July 9th, 1940. He then worked on the shredding machine until August 22nd, 1940, when he was taken ill. He was admitted to St. Charles Hospital, Ladbroke Grove, on August 28th, 1940, complaining of severe cough with purulent sputum, shortness of breath and night sweats for two weeks. On examination there were scattered râles over both bases and rhonchi at the left apex. He had clubbing of the fingers. During his first week in hospital he had a fever up to 100.5°F. Examination of his sputum showed no tubercle bacilli. X-ray of his chest on August 28th (fig. 3) showed very extensive mottling with some confluent areas throughout both lungs. X-ray examination on September 30th, 1940, showed that this process had almost cleared, and he was
discharged from hospital on November 3rd, 1940. On
July 15th, 1943, he was well, but still complained of
some shortness of breath. On examination there were
no abnormal physical signs. X-ray of his chest revealed
no abnormality (fig. 4).

Case 5. S. B., aged 40. He had worked as a building
labourer until he joined the firm on July 16th, 1940.
He worked on the shredding machine from July
16th to September 1st, when he was taken ill. He was
admitted to Willesden General Hospital under Dr.
Pearse Williams, complaining of cough with a small
amount of muco-purulent sputum, shortness of breath and
sweating for 2 weeks, and slight recent loss of weight.
There was no history of haemoptysis. On examination
there was impaired percussion note and many râles throughout
both upper zones. Sputum contained pus and showed
numerous acid-fast bacilli. On culture streptococcus
viridans, staphylococcus aureus and micrococcus
catarrhalis were present. Dr. Rohan Williams
reported on an x-ray taken on September 12th, 1940, as follows:—
Bilateral diffuse parenchymal inflammatory changes in
both mid and lower zones, the upper zones being rela-
tively clear. Probably a diffuse bronchogenic tuber-
culosis. Dr. Rohan Williams suggested the possibility
of an occupational cause for the appearances. The
patient was seen by the Tuberculosis Officer who agreed
that he was suffering from pulmonary tuberculosis, and
he was admitted to Clare Hall Sanatorium in November
1940. His symptoms had disappeared by then. His
sputum contained no tubercle bacilli, and an x-ray taken
on November 20th, 1940, revealed no abnormality. He
was examined on July 15th, 1943, when he was quite
well, had no abnormal physical signs, and an x-ray of
his chest revealed no abnormality.

Case 6. J. S., aged 45. He had worked on the roads
and railways in Ireland, and for 10 years as a window
cleaner in England before he started working on the
shredding machine on October 12th, 1940. He con-
tinued to work on the machine until November 20th,
1940, when he was taken ill. He attended the Brompton
Hospital on December 11th, 1940, complaining of
severe shortness of breath, cough with much sticky
purulent sputum, sweating, and loss of weight. On
examination there were many scattered râles through-
out both lungs. X-ray showed a diffuse soft mottling
throughout both lungs with some confluent areas. He
was admitted to the hospital from January 3rd, 1941
to February 19th, 1941, when he was found to have a
low grade fever to 100° F., which quickly subsided.
X-ray examination on February 19th showed that the
changes had completely disappeared. On July 15th,
1943, he was well with no symptoms, no abnormal

Fig. 5.—Case 8 showing scattered mottling, confluent areas, and enlarged hilar glands.
physical signs, and there was nothing abnormal on x-ray examination.

Case 7. W. H., aged 38. He worked for 5 years as a collier's assistant in the South Wales coalfield, and for 15 years as a platelayer on the London, Midland and Scottish railway before joining the firm in October 1940. He worked from then until December 23rd, 1940, on the shredding machine when he was taken ill with fever, shortness of breath, cough with a small amount of sticky sputum, and loss of weight. He attended the Central Middlesex Hospital on February 27th, 1941, when he was found to have no abnormal physical signs. An x-ray was taken, and this revealed slight miliary mottling scattered throughout both lungs with enlargement of the hilar glands. On September 21st, 1943, he was in the Army. He had no symptoms, no abnormal physical signs, and x-ray examination of his chest showed no abnormality.

Case 8. E. P., aged 31. He worked in the motor engineering trade, in brickyards, and as a builder's labourer, before joining the firm as a labourer in October 1939. He worked on the shredding machine from September 1940 to January 25th, 1941, when he was taken ill. He attended the Northampton General Hospital under Dr. Eric Shaw on February 25th, 1941, complaining of cough with thick phlegm, shortness of breath, fever, and loss of weight. There were signs of consolidation at the base of the right lung. An x-ray showed scattered mottling throughout both lungs with some confluent areas and enlargement of hilar glands (fig. 5). He again attended on June 18th, 1941, when x-ray showed no abnormality. On July 4th, 1943, he still complained of much shortness of breath, cough and sputum, but an x-ray of his chest showed no abnormality in the lungs (fig. 6).

Case 9. W. W., aged 30. He had worked for a few months as an assistant collier in the South Wales coalfield, and then as a builder's labourer before joining the firm. He worked on the shredding machine from January 20th, 1941 to February 22nd, 1941, when he was taken ill. He was admitted to Wembley Hospital on March 6th, 1941, with severe shortness of breath and cyanosis. There were scattered patches of consolidation with bronchial breathing and coarse rales at both bases. On March 11th, 1941, there was a pleural friction rub over the right lower lobe. He was treated with oxygen at 6 litres a minute through a BLB mask, but the disease gradually spread and his condition deteriorated. He
was considered to be too ill for x-ray examination, and died on March 19th, 1941. There was no necropsy.

Case 10. S. N., aged 21. (Case 1, Castleden and Hamilton-Paterson, 1942.) He had worked as a fisherman and milk roundsman before joining the firm as a labourer on September 11th, 1939. He worked on the shredding machine from March 21st, 1941, to April 21st, 1941, when he was taken ill. He was admitted to Redhill County Hospital under Dr. Castleden on May 4th with increasing shortness of breath and a cough with scanty sputum, sometimes blood-stained. His temperature was 100·6° F. on admission and he was febrile for 19 days. He was orthopnoeic and cyanosed: chest movement was poor, the percussion note was impaired, and there was distant bronchial breathing in small areas at both bases. The sputum contained M. catarrhalis, streptococcus viridans and fusiform bacilli. Staphylococcus aureus and pneumococcus Type 4 were each grown on one occasion. No tubercle bacilli were found. Blood count showed 4,610,000 red cells per c.mm., 78 per cent. haemoglobin, 11,300 leucocytes per c.mm., 83 per cent. neutrophils, 14 per cent. lymphocytes, 3 per cent. monocytes, and no eosinophils. X-ray on May 5th, 1941, showed extensive consolidation in both lower lobes with marked enlargement of the hilar glands. X-ray on June 15th showed some extension of the process, but on July 5th there was marked clearing, and by August 29th the lungs were completely clear. On August 27th, 1943, he was well and serving at sea with the Royal Navy. He had no symptoms, no abnormal physical signs, and x-ray of his chest showed no abnormality in the lungs.

In 1943 all the workers in the factory were interviewed and occupational histories taken. It was found that out of 163 men employed 22 had worked in the coal mines. A portable x-ray apparatus was taken to the factory and a skiagram (15 in. × 12 in.) was taken of the chest of all the workers. Of the 22 men who had previously been employed in the coal mines 19 showed evidence of chronic pulmonary disease, ranging through all stages of fibrosis, reticulation, nodulation and massive shadows. Three of these men, two showing fibrosis and one reticulation, had also worked on the shredding machine. These appearances could be superficially confused with those caused by bagasse, but whereas the latter is an acute febrile illness which clears in weeks or months, the former is a chronic state with no change over many years. Eight cases of pulmonary tuberculosis were also discovered.

Specimens of sputum obtained from the four men, who were working on the shredding machine at this time, were submitted to Duncan who isolated from them species of fungi common on the
bagasse and in the dust of the shredder room. Microscopical examination of the sputum did not reveal evidence pointing to mycosis, but showed the ingredients of the airborne dust. The fungal elements present, from which the cultures were obtained, represented merely the result of recent inhalation of dust.

In 1944 the men working on the shredder again had skiagrams taken of their chests. One man complained of symptoms and the skiagram revealed the characteristic picture described in the previous cases. Attempts were made to persuade him to come into hospital but he declined, and being a citizen of Eire he returned to that country. As far as is known he was afebrile. It seems possible, therefore, that the x-ray changes may precede the onset of the acute illness and be caused by bronchial obstruction, giving rise to small areas of collapse.

Case 11. J. S., aged 50. Started work at the age of 15 as a messenger boy. Apart from 6 years in the Army he had worked in a bacon factory, and on buildings in Ireland until 1943 when he came to England and started work at the board-making factory. For the first 6 months he worked on the beater floor in the main building and for the 6 months previous to examination he worked on the shredding machine. For 3 months he had had shortness of breath on exertion, a severe dry cough which was worse at night and in the early morning. He had a poor appetite and was losing weight. On examination no abnormal physical signs were found. A skiagram of his chest showed a diffuse miliary mottling throughout the lung fields.

A man who had worked part of three shifts on the shredding machine but had otherwise worked for a year at the far end of the factory, was discovered on x-ray examination to have bilateral fibrosis and possibly cavitation of an unusual type in both lungs. The x-ray appearances were similar to those of the electrical mechanic already referred to as working in the shredder room in 1940. Both men were admitted to hospital for further investigation. Their case histories were as follows:

Case 12. T. M., aged 38. (Case 2, Castleden and Hamilton-Paterson, 1942.) He had worked as an electrician until he joined the firm as an electrical maintenance mechanic. In this capacity he was intermittently in close contact with the dry crushing process during the installation of the new shredding machine between August 1939 and September 3rd, 1940, when he first complained of shortness of breath, cough and scanty sputum. His only previous illness was in 1935, when he was in Salisbury Infirmary for a few days with acute bronchitis.

![Figure 8](http://oem.bmj.com/)

**Fig. 8.**—Case 13 showing extensive pulmonary fibrosis.
A sketchgram was not taken of his chest at this time. He attended the Willesden General Hospital in October 1940 and Dr. Rohan Williams reported on an x-ray of his chest. The appearances were consistent with a catarhal bronchitis, but no further pulmonary lesion was noted. He returned to work in the office on October 30th, 1940, but was away sick with similar symptoms from November 12th to December 3rd, 1940. In December 1940 he began to be short of breath at work, and in January 1941 he was out of breath on the least exertion. On January 17th, 1941, he attended Redhill County Hospital under Dr. L. J. M. Cass. Clubbing of the fingers and of the chest was kyphotic with many fine râles in all areas of both lungs. X-ray showed a few apical scars and a little fibrosis in the right mid zone. In May 1941 he was admitted to Wembley Hospital where an x-ray was reported on as showing bilateral apical tuberculosis. The shortness of breath increased and he was admitted to Redhill County Hospital on November 22nd, 1941, and did not return to work at the factory handling bagasse. He was orthopneic but afebrile; the percussion note was impaired at the left apex with bronchial breathing and bronchophony. Crepitations were present throughout the left lung, and at the right apex the sputum was mucoid, but no blood-stained. Polymorphs and lymphocytes were present. Haemolytic staphylococci and pneumococci (type 23) were present. Tubercle bacilli were not found in the examination of 18 plate cultures, but two-thirds of Martin smears showed no reaction. A blood count showed 4,590,000 red cells per c.mm., 94 per cent. haemoglobin, 10,200 leucocytes per c.mm., 51 per cent. polymorphs, 32 per cent. lymphocytes, 6 per cent. monocytes, 11 per cent. eosinophils. X-rays showed extensive fibrosis in both upper lobes with multiple cavities. He was discharged on February 15th, 1942, with less dyspnoea. His condition remained unchanged and he was admitted to the London Hospital on August 16th, 1943, with orthopnoea and markedly clubbed fingers. There was poor movement of the left side of his chest with many scattered highpitched rhonchi and râles. His sedimentation rate was 30 mm. in 1 hour (Wintrobe). His blood count showed 97 per cent. haemoglobin (alkaline haematin, photo-electric cell method) and 6,440 white cells per c.mm. X-ray of his chest (fig. 7) showed marked generalized fibrosis in both lungs and suggested the presence of cavities. His sputum showed no tubercle bacilli on smear, concentration, culture or guinea-pig inoculation. J. F. Duncan demonstrated the presence of Candida sp. in 1943. Mr. Vernon Thompson found a bronchoscopic record which revealed no abnormality. He obtained bronchoscopic swabs from the pus present in the bronchi. No tubercle bacilli could be demonstrated in this material either on smear, concentration, culture or guinea-pig inoculation. None of the fungi common in the sputum of men working on the shredding machine were found in the bronchoscopic specimens or any other specimens of sputum. The patient was treated with large doses of iodides without improvement, and discharged on November 27th.

He attended the out-patient department until September 23rd, 1944, when his breathlessness became very much worse, and he felt he had not the strength to cough up sputum. He was therefore admitted to hospital. On examination he was orthopneic, temperature 98”, pulse 110, respiration 32 at rest in bed. He was a dry type with a bronchial cough and enlarged lymph glands in the upper cervical chain. There was a soft systolic murmur in the mitral area with a triple rhythm in the tricuspid area. His blood pressure was 105/70. Movement on both sides of the chest was poor and all accessory muscles of respiration were in play. There were palpable rhonchi over the front of the chest; percussion note was impaired at both upper and sub-apical areas, bronchial at both axillae, and vesicular elsewhere. Coarse râles and rhonchi were heard all over the chest. His liver was felt 3 finger-breadths below the right costal margin. The sputum contained multiple organisms with streptococcus viridans and pneumococcus predominating. Blood count: 91 per cent. haemoglobin (photo-electric cell), 13,900 leucocytes, 90 per cent. polymorphs, 2 per cent. eosinophils, 7 per cent. lymphocytes, 1 per cent. large hyalines. X-ray of chest showed no marked difference from previous x-rays. His condition steadily deteriorated and he died on October 18th, 1944.

Necropsy revealed much thick pus and injection of mucosa in trachea and main bronchi. The posterior two-thirds of the upper lobe of the left lung was greatly contracted, airless, anthracotic and fibrotic, showing smooth rubbery, mottled grey and buck cut surface and considerable bronchiectasis up to 0.5 cm. diameter with a few bronchiectatic cavities up to 1 cm. diameter. These cavities were empty and had smooth grey linings. There were thickened dilated bronchioles up to 0.3 cm. diameter throughout the anterior third of left upper lobe and throughout lower lobes of both lungs and right middle lobe, with much intervening well aerated emphysematous lung tissue. There were large areas of severe emphysema with emphysematous bullae in all borders of lower and middle lobes of both lungs. There were numerous irregularly shaped, grey and black mottled, tough mostly subpleural patches to 1 cm. breadth completely white lung tissue with a smooth rubbery cut surface throughout the upper lobe of the right lung affecting all the lung tissue in the apex and almost all of the posterior fifth of the lobe, but with a considerable amount of residual air. All the right upper lobe of the rest of the lobe. Similar areas of fibrotic pneumonia formed an interrupted irregular subpleural zone 12 cm. long with depth varying from 0.2 to 1.2 cm. in the posterior part of each lower lobe, and also patches 1.5 cm. broad near the hilum of each lower lobe. Thick white pus was present in many bronchiectases throughout both lungs. Numerous areas of fibrous thickening, up to 0.2 cm. thick, of the visceral pleura covered these lungs. Fibrous pleural adhesions obliterated both pleural cavities except over part of the left lower lobe.

Professor H. M. Turnbull is working on the pathology of the disease and when the work is completed will publish a full account of it in this journal.

Case 13. T. K., aged 42. He had worked on a farm and as a cellarman in Ireland before he joined the firm in July 1938. He worked in the packing department and on the ‘defibrator’ in the main factory, and on three occasions he worked part of a shift on the shredding machine. He complained of no symptoms, but in the mass radiogram performed on him on September 10th, 1943, he was found to have extensive fibrosis in the mid zone of both lungs (fig. 8). He was admitted to the London Hospital on November 23rd, 1943, and tomograms suggested the presence of cavities. Sputum examination revealed no tubercle bacilli on smear, concentration, culture or guinea-pig inoculation. Mr. Vernon Thompson performed a bronchoscopy on December 6th, 1943, and took swabs from the pus in the bronchi. Examination revealed no tubercle bacilli on smear, concentration, culture or guinea-pig inoculation. None of the fungi common in the sputum of men working on the shredding machine were found. The patient was discharged on November 8th, 1943, and his condition has not changed since then.

None of the fungi common in the sputum of men working in the shredding room were found in these two men’s sputum. It may be presumed that they and the other men were exposed to the dust of the shredding room, their sputum and probably the larger bronchi contained numerous fungus spores. We have no evidence of survival of any of the fungi in the lungs of men after removal from exposure to the atmosphere of the shredding room. These two men were less exposed to bagasse than those who developed the acute infection described in cases 1–10; nevertheless they were exposed and
their histories and clinical investigations are not typical of any known disease of the lungs. Case 12 had an acute respiratory illness at the commencement and though no skiagrams are available of his lungs, at that time the natural chronic sequel to an acute bronchiolitis and pneumonia would be chronic bronchiolitis and bronchiectasis, and this is the condition that was found in his lungs at necropsy. Pathologically this chronic bronchiolitis and bronchiectasis does not differ from that arising in any chronic interstitial pneumonia, but the distribution in this case involved the upper lobe particularly posteriorly, and causing tough subpleural patches of fibrotic lung was most unusual except in the diseases caused by dust. It seems probable, therefore, that it was a chronic sequel to disease in the lung caused by bagasse.

The two cases reported by Jamison and his colleagues (1941 and 1944), the twelve cases reported by Sodeman and Pullen (1943 and 1944), and the ten cases reported here are all similar, and show almost conclusively that bagasse dust can give rise to acute bronchiolitis and pneumonia. Out of the 24 cases there were two deaths, a mortality rate of 4·3 per cent. Here, therefore, is an industrial disease with serious complications.

The exact nature of the disease, however, remains obscure. There are undoubtedly a great many fungi present in bagasse and Duncan has recovered similar fungi from men who have worked on the shredding machine, but there is no evidence that any particular fungus has caused the syndrome which is described; neither is there any evidence that the disease has been caused by any specific bacterium or virus. It is to be noticed that acid-fast bacilli were found in the sputum in case 5, and the case described by Jamison and Hopkins (1941). It is possible that the disease is caused by the irritation of the dust itself in the bronchioles, giving rise to an inflammatory reaction, but this hardly seems a satisfactory explanation.

Fungi may play an important role in breaking down the fibre into a very fine vegetable dust, and may possibly even render this toxic. Fawcitt (1938) has described a condition which he labelled 'Farmer's lung.' This disease is caused by inhalation of a fine vegetable dust from mouldy hay and in its symptomatology and radiological findings it closely resembles byssinosis as well as the condition described in this paper as caused by bagasse. A similar disease occurs in horses and is known as 'broken wind.' These four diseases so closely resemble each other that they may possibly belong to a single group with a common pathology.

Neal, Schneider and Caminita (1942) described an acute febrile illness occurring among workers exposed to high concentrations of stained cotton dust. Although these authors attributed this disease to the presence of a gram-negative rod shaped bacterium, it is possible that this disease also resulted from the fine vegetable dust created in the process, or from the breakdown of vegetable matter by the organism described.

**Prevention**

In the spring of 1941 the process of breaking the bagasse bales had been rendered wet by a spray of water to allow the dust. Exhaust ventilation was also installed. Since that period only one further case of the disease has been observed.

**Summary**

An account is given of 11 cases of acute bronchiolitis and pneumonia arising from the inhalation of bagasse dust. One of these cases was fatal. Severe dyspnoea, cough with scanty sputum and occasional haemoptysis were the usual symptoms. There was a characteristic x-ray appearance which showed a miliary mottling scattered throughout both lungs with heavy shadows at the hilum. Reference is made to 14 similar cases described in the literature, among which there was also one death. The aetiology is uncertain but the disease was most likely caused by fine vegetable dust. Two workers who had been in contact with the bagasse developed extensive fibrosis of lung, and one of these subsequently died. Necropsy revealed chronic bronchiolitis and bronchiectasis.

The method of prevention suggested is to allow dust and to handle bagasse in a moist state. Exhaust ventilation is also desirable.

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**References**


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