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MCMXXX.

monia" of congenital syphilis. There is much tinous œdema, but no purulent exudation no softening. The trachea and bronchi are cted. The bronchial glands are not enlarged. spleen in both cases is slightly enlarged; sted but not soft. The other viscera show dy swelling and congestion. An intramuscular morrhage of recent origin is present in the ominal wall of Case 9. It is not at the site of a m injection. The bowel contents are fluid, and he cæcum are four small shallow acute ulcers, peritoneum over which is natural.

he *histological study* of the changes in the lungs ands more detailed investigation. Yet the liminary stage in the examination already shows ain peculiar features.

n both cases the main condition may be described a broncho-pneumonia, the cytology of the alveoli the actual areas of consolidation showing no sual characters. Neighbouring areas of lung œdematous. In addition there are small foci suppuration and in some places there are areas complete necrosis. In Case 3 (Figs. III. and IV.) one these areas is at the periphery of the lung and the sence of a thrombosed artery at its apex suggests t it may be an infarct. However, thrombosed ssels are seen in other parts of the lung, without jacent necrosis. A further peculiarity in this case (3) the presence in the lung of plugs of coagulated terial in many of the alveoli. These are of regular, cular, or oval shape, and contain no cells except d alveolar epithelium bordering them. No uctures exactly comparable with these have yet en seen in the second case.

General Remarks.

The study of the cases hitherto reported under provisional name of psittacosis, together with ese here recorded, makes the conclusion almost esistible that there is a septicæmic form of disease human beings closely associated in causative shion with disease in parrots and kindred birds. is conclusion is borne out alike by the clinical atures of the disease, by the peculiar morbid atomical findings—especially in the lungs—in tal cases, and by the evidence of contact, direct or direct, with sick or dead parrots.

Amongst the clinical features in the series of cases re noted the prevalence of epistaxis during the rly stage of the disease, the tendency to diarrhœa ith collapse, and the special liability to pulmonary mplications towards the end of the first week e noteworthy. If the cases are to be accepted s a group in which the infection is of the same order roughout, it is clear that the disease may be in me instances extremely severe, carrying a very ave prognosis, and in others quite mild. There also some suggestion that age is a factor in prognosis, that the severest cases have been in patients over 0 years of age. There appears to be no recorded se of a severe illness in a patient under 20 years of ge. Case 8 in the present group, a boy of 11 years, as very mild, as was the case in a boy of 7 years, he youngest in Dr. A. P. Thomson's series of 21 ases.[1]

In considering the *differential diagnosis* the acuteess of the onset with headache, general pains, norexia, and insomnia very naturally suggest nfluenza. In neither disease have we any diagnostic riterion either as regards physical signs, clinico-athological findings, or bacteriological tests. Absence f leucocytosis marks both diseases, at all events in heir early stage. Epistaxis or delirium occurring during the first few days should raise suspicion that he case is not influenza. The similarity between the diseases may obviously be so close that further knowledge can alone help us to decide how many cases labelled "influenza" properly belong to this more recently studied infection. Although it is no doubt true to say with Dr. Thomson that the outstanding features of psittacosis are consequent on the pulmonary involvement, it must be remembered that this is also true of the more severe cases of influenza. If, however, in a doubtful case, pulmonary signs appear during the first three days the diagnosis of influenza is the more probable. On the other hand, an influenza-like disease which persists for more than five days without pulmonary complications should lead to inquiries, *inter alia*, concerning sick birds. From the enteric group psittacosis is marked off in the main by its more abrupt onset, negative blood culture in the early days, the absence of an agglutination reaction in the second week, and the rarity in typhoid nowadays of pulmonary complications other than signs of slight bronchial catarrh. General tuberculosis will sometimes present a difficulty, but here again the onset is likely to be less acute, the pulse frequency raised more in proportion to the temperature, and the appearance of toxæmia, as expressed by the facies, anorexia, and delirium, less marked than in psittacosis.

In the matter of the incubation period of psittacosis no reliable evidence is afforded by a study of the cases here reported. They do suggest, however, that it is not less than ten days.

We desire to express our thanks to several of our colleagues—to Drs. James Maxwell, R. S. Johnson, and L. P. Garrod for pathological investigations; to Dr. E. R. Cullinan for his preliminary report on the histological appearances in the lungs; to Dr. E. L. Sturdee (of the Ministry of Health) for sundry communications; and especially to Dr. Desmond Urwick for his careful analysis of the group of eight cases recorded in the text, and his permission to make use of his notes.

PULMONARY ASBESTOSIS.

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ALTHOUGH asbestos and asbestos products have been used in industry for a long time, it is only during the last few years that pulmonary asbestosis has been recognised as a serious industrial disease.

Asbestos, which was known to the ancient world, is a silicate occurring in minerals in combination with iron, copper, calcium, or magnesium. It is quarried or mined in various parts of the world, Italy and the Mediterranean, South Africa, Rhodesia, Canada. In its natural state it occurs in strands of long silky fibres which are highly resistant to heat, strong acids, and alkalis. These masses are usually broken up into short lengths before exportation into this country. In this country the lengths are crushed and disintegrated and then either mixed with various substances for hardening into slabs, pipes, boards, &c., or carded, spun, and woven into various products like mats and mattresses, or used as an inert diluent or filling of a heat-resisting character. At certain stages in these industrial processes dust consisting of fine asbestos fibres is generated. When examined microscopically by dark-ground illumination the asbestos fibre gives the impression of a sharp, brittle metallic wire broken off at various angles and in different lengths, and, being highly refractile, has the appearance of the glowing filament of an electric bulb. These fibres can be found in the nose and mouth, and in the asbestos corns and minute skin lesions of the workers. On reaching the lung the fibres set up a pneumonokoniosis of a characteristic variety.

The first fatal case of pulmonary asbestosis recorded was that of H. Montague Murray,[1] who made a post-mortem examination of a case at Charing Cross Hospital in 1900. The next case recorded was by W. E. Cooke,[2] who published a short note in 1924 and reported the autopsy findings on the case in 1927. In the lungs of this case Stuart McDonald [3] noted the presence of certain curious golden yellow bodies, having the appearance of minute crustacean forms. These bodies did not stain with ordinary aniline dyes but preserved their golden brown colour. They were well seen in unstained sections and gave a characteristic prussian blue reaction with potassium ferrocyanide and dilute hydrochloric acid. Careful investigation led McDonald to believe that these bodies were not of vegetable or animal origin. In 1928 Simson [4] reported four cases with autopsy findings from South Africa, and noted also the golden yellow bodies.

In 1929 M. J. Stewart and A. C. Haddow [5] showed that these curious bodies could be found in the juice expressed, post mortem, from the lungs of cases of pulmonary asbestosis, and that they could also be detected in the sputum by digestion with equal quantities of concentrated antiformin.[6] They also suggested the name asbestosis bodies for these structures, a name which we think should now replace the former non-committal name—curious bodies.

Later in 1929 one of us (S. R. G.)[7] showed, by means of dark-ground illumination, that when the golden yellow asbestosis body was dissolved in concentrated sulphuric acid it had a central core consisting of a minute asbestos fibre. Asbestos fibres were also demonstrated in the nasal passages and mouth of workers.

A considerable number of asbestos workers have now been examined at Victoria Park, and a clinical record of 15 cases with radiograms was published in 1929 by one of us (W. B. W.).[8] Since this record was published we have been able to bring the numbers up to 37 cases including 4 asbestos workers with doubtful diagnosis, together with four records of post-mortem findings, two of which have been reported by Wood and Page.[9] An excellent résumé of the work done up to this point will be found in J. F. C. Haslam's [10] "Recent Advances in Preventive Medicine." It seems worth while, therefore, to place on record at this stage our findings up to date.

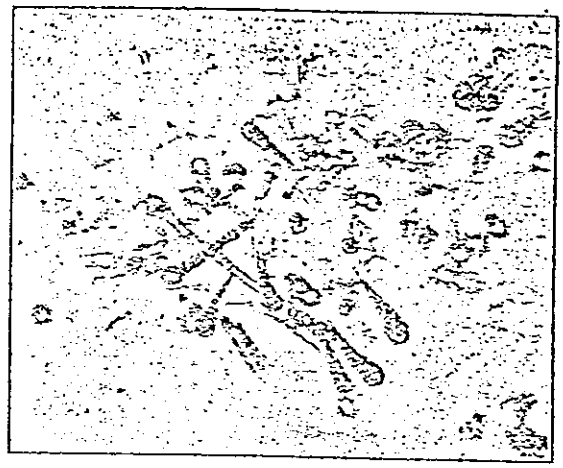
Symptoms.

The cardinal symptoms are dyspnœa and cough. The former is in many cases the earliest symptom noticed by the patient, who complains of slight breathlessness on hurrying or going upstairs or that his chest "feels stuffy." He therefore discards the respirator worn hitherto under protest. In late stages of the disease dyspnœa may be extreme. A "terrible tightness of the chest" may cause great distress and the slightest exertion give rise to laboured breathing. Cough is a variable symptom, and though usually present is seldom severe and may be absent for prolonged periods. It is either dry or accompanied by the expectoration of a little viscid phlegm. The sputum in this, as in other chronic pulmonary diseases, may on occasion be streaked with blood. This is, however, exceptional, and the only frank hæmorrhage noted in our series of cases occurred in an asbestos worker who was also suffering from adolescent phthisis with cavitation. Many patients complain of anorexia, lassitude, pains in the chest, and loss of weight. The last is a noteworthy feature, for wasting may be progressive, and in late stages of the disease is sometimes extreme.

Physical Signs.

When the disease is established the skin has an unhealthy leaden hue. Cyanosis may be absent, slight, or sufficiently evident to cause a dusky complexion. The chest is poorly covered and expansion is defective and may be reduced to one inch or less is often apparent. Corns due to the irritation caused by asbestos fibres embedded in the superficial layers of the skin of the hands are occasionally seen. The skin in other exposed positions—e.g., the legs of girl workers—may be similarly affected.

The physical signs in the lungs are those of pulmonary fibrosis, limited to, or predominant at, the lung bases. The coarse creakings and crepitations usually associated with fibroid change are, however, replaced by fine dry crackles. These may arise in the pulmonary parenchyma or be due to the movements of slightly roughened pleural surfaces. A definite friction rub



Asbestosis bodies in unstained section of lung.

may be heard over one or other axillary base. As the fibrosis is bilateral there is seldom any appreciable cardiac displacement.

Sputum.

The sputum is thick and mucoid—frequently so thick as to resemble semi-coagulated egg albumin. In the latest stages small nummules of pus appear.

Asbestosis bodies can be demonstrated by Stewart's technique. The sputum is mixed with an equal quantity of concentrated antiformin and allowed to stand. When the mixture has become digested it is centrifuged and the supernatant fluid pipetted off and a wet film of the deposit examined with a 1/6 objective for asbestosis bodies. To demonstrate individual asbestos fibres one of us (S. R. G.)[7] devised the following technique.

The sputum is digested with 10 per cent. antiformin which has been filtered through a candle to remove any foreign bodies, the digested mixture is centrifuged, the deposit calcined to destroy all cellular debris, and a wet preparation of the calcined deposit mounted in concentrated sulphuric acid and examined by dark-ground illumination. The asbestos fibre has been described above. In sputum it varies greatly in size, from 24 to 60 μ or more. It shows no branching but sometimes has a fine saw-like edge rather suggestive of the marks on a freshly cut plank. This saw-like edge does not extend along the whole length of the fibre. But the most marked characteristic of a single asbestos fibre, when examined by this method, is its marked refractility. Comparison with a control film of ground-up asbestos is essential. The sputum should always be examined for tubercle bacilli as well as for asbestosis bodies and asbestos fibres.

Asbestos fibres, but no asbestosis bodies, have also been found in nasal secretion and in the mouth-wash by the same method. We have also found asbestos fibres in the lacrymal secretion of one patient.

Diagnosis.

When the patient's occupation is known diagnosis should seldom give rise to difficulty except in the

inical picture. In the earlier stages of the disease, owever, a definite diagnosis may be impossible. ggestive symptoms may be the product of fear, ysical signs may be confined to a few fleeting ackles at a lung base, there may be no sputum ailable for microscopical examination, and the ray findings be equivocal. The difficulty of detectg the earliest radiographic signs is dealt with later.

Several of the cases in our series had been under eatment for suspected pulmonary tuberculosis. From e above description it will be realised that an uncomlicated case of asbestosis should not be mistaken r one of tuberculosis, nor is such an error likely to e made except by those who ignore the significance sputum tests. But the demonstration of tubercle acilli in the sputum of an asbestos worker may be a ource of difficulty. In three of our cases it seemed robable that tuberculosis had supervened in lungs lready damaged by silica. In two of these tubercle acilli were found in the sputum, and in one, reported lsewhere,[9] in the fæces shortly before death.

Radiographic Appearances.

In an advanced and uncomplicated case of pulonary asbestosis the radiogram is pathognomonic. he picture is that of a fine fibrosis affecting the ower half or two-thirds of the lung fields. At first ght the affected areas may exhibit a diffuse haze esembling ground glass. Closer inspection reveals reas of fine mottling and linear shadows which, owards the bases, may be draped like coarse cobwebs. he heavy granular shadows typical of certain other neumonokonioses—e.g., gold-miner's phthisis—are bsent. The root shadows radiating downwards are ery obvious, and where these cut the left edge of the ardiac shadow they give it a shaggy appearance. he costal angles are obliterated. The lateral margins f the lungs may show thickening of the parietal leura, and the dome of the diaphragm is obscured. bove the clavicles thickening and puckering of the pical pleura may be apparent. These are the ppearances of advanced disease, and all gradations re encountered between them and the first fine ottlings and striations of incipient mischief. Reference to control films is imperative, for the natural endency is to find abnormalities where no departure om the normal is present. The skiagram of a healthy dividual, if slightly out of focus or under-exposed, ay show faint mottling especially in the lower areas f the lung fields, and as it is necessary to allow for ossible imperfections in the films of chests of asbestos orkers, the control films should not be selected from e radiologist's show-case. Allowance must also e made for the haze produced by the mammary lands in the chest radiograms of healthy women.

Prognosis.

It is too early in the study of this disease to say ery much on this subject, but once the asbestosis odies appear in the sputum the course of the disease ould seem to be progressively downward. Nor does essation of exposure to further infection avail to heck its spread. Death ultimately ensues from roncho-pneumonia or tuberculosis.

Of the 15 cases reported by one of us (W. B. W.) in 929, three have now died, two of broncho-pneumonia nd one of pulmonary tuberculosis.

Treatment.

Symptomatic treatment is disappointing, for we ave no means of relieving the dyspnœa which is the atient's chief complaint. Prophylaxis is thus allmportant, and the only hope for the future of the sbestos worker lies in the adoption of proper means f protection against the risks attendant on the nhalation of the fibres.

Post-mortem Appearances.

Pleura.—The visceral pleura is generally uniformly

empyema cavity, containing dirty brown sero-pus, between the layers of the visceral pleura, with a similar empty cavity between the layers of the diaphragmatic pleura from the upper walls of which pus oozed. The interlobar fissures may be obliterated, adhesions to the pericardium have been described, and finally the whole pleural sac may be closed with adhesions.

Lungs.—The lesions are generally bilateral. The lungs are firm and contracted. On section they give the appearance of being hard and leatherlike and, to a certain extent, airless. The trabeculæ stand out prominently forming a fibrous network in the lung. The fibrosis is most marked at the bases, but although the apices may appear clear to the naked eye, on section they are generally found to contain a considerable amount of fibrous tissue. The basal fibrosis in one case seen by us gave rise to a striking honeycombed appearance. The bronchi are dilated and there may be small bronchiectatic cavities. Septic bronchitis, broncho-pneumonia, and lobar pneumonia are found as terminal events.

Mediastinal Glands.—In our cases these have been small, pigmented, and hard, with occasional small calcareous nodules. Enlarged mediastinal glands have been described by other writers. No lesions of importance have been found elsewhere in the body with the exception of tuberculosis and terminal broncho-pneumonia.

Microscopic Appearances of the Lungs.

The following are the chief changes:—

1. The bronchioles show desquamation of epithelium and sometimes complete sloughing of the mucous membrane. Beneath this there may be granulation tissue with newformed capillaries. The bronchioles are surrounded by thick layers of fibrous tissue which may occasionally compress and completely obliterate the lumen. Sometimes there is cellular infiltration of the peribronchial tissues.
2. Endarteritis of smaller branches of the pulmonary artery has been described, some of them being thrombosed. There is also a large increase of connective tissue around the arterioles.
3. Thickening of the interlobular connective tissue.
4. Thickening of subpleural connective tissue.
5. All these connective tissue areas show fibroblasts and often small areas of round cells.
6. The pulmonary alveoli may show desquamation of alveolar cells or may be filled with leucocytes; other alveoli may be obliterated by connective tissue, and others, again, show compensatory emphysema.
7. Occasional giant cells may be found, but it must not be assumed that these are tuberculous in origin because foreign giant cells form readily, and the asbestosis giant cell may easily be mistaken for the tubercle giant cell. This is a point of some importance.
8. Scattered throughout the lung, more or less indiscriminately, is a considerable amount of amorphous, brown pigment. This is often found phagocyted by alveolar cells.
9. The presence of the pathognomonic asbestosis bodies. These bodies are found in the lumina of the bronchioles with desquamated bronchial epithelium around them, in alveoli, again with desquamated alveolar cells around them, and also scattered indiscriminately in the fibrous tissue. Sometimes the bodies appear to be phagocyted.

The Asbestosis Bodies.

These are golden yellow bodies varying in length from 24 to 60 μ, and in breadth from 12 to 24 μ. At first sight they have a superficial resemblance to minute crustaceans with a bulbous head, segmented body, and a long tapering tail. The substance of this golden yellow body is apparently homogeneous except for a central linear thickening, suggestive of a fibre running through the length of the body. When the body is dissolved in concentrated sulphuric acid under dark-ground illumination the highly refractile asbestos fibre which forms the core can easily be seen. What the golden yellow material is which is deposited round this asbestos fibre in the tissues—for the asbestosis bodies have not been found in vitro

show subsidiary bosses on it or may be broken off altogether or splintered in small bits. The segments may be closely contiguous like those of a crustacean, or they may be separated by a clear interval, and they are not infrequently fragmented. They vary in size and may be rectangular or oval. Occasionally sausage-shaped forms of the asbestosis body without any constrictions are found. Also what appear to be early forms can be detected. These look like thickened fibres with a slightly bulbous head, and they often are of unusual length, rather suggesting that once the asbestosis body is fully formed it tends to get fragmented. The bodies do not stain with the ordinary aniline dyes, but can be faintly stained with the prussian blue reaction used for detecting iron in histological sections. They are resistant to antiformin and to weak acids, and we have found them, in sections, to resist weak decalcifying fluid for many days. We have also found them to resist desiccation and autolysis in sputum for at least a week.

Once seen the asbestosis body can be readily recognised, and we know of no other substance found in human histology with which it can be confused.

Suggested Classification of Stages of Pulmonary Asbestosis.

Pulmonary asbestosis falls into group B [1] of occupational diseases of Bridge's classification.[11] Three stages can be defined: (1) the presence of asbestos fibres in nasal secretion or in the mouth, merely indicating exposure but not necessarily disease; (2) the presence of asbestosis bodies which is a sign of a tissue response to the infecting dust; (3) the typical symptoms, signs, and X ray appearances which are, in addition, clear evidence of the disease irrespective of the finding of asbestosis bodies.

Obviously (1) is no evidence of disease, but we can accept (2) as a sign that disease is present. In the absence of sputum diagnosis must be made on (3) alone. There remains, of course, the difficulty of a patient with pulmonary fibrosis of childhood origin, subsequently becoming employed in an asbestos factory. We consider the radiogram would be sufficient to distinguish this type of case from true pulmonary asbestosis.

Lastly, there is the question of pulmonary tuberculosis as a complication. Does the occupational disease light up quiescent tuberculosis or does tuberculosis become implanted on pulmonary asbestosis as a terminal event? It is impossible to decide this question, but it is worth while noting that when pulmonary tuberculosis does occur in these cases it is liable to be of an anomalous type.

We are indebted to the Medical Staff of the hospital for permission to use their cases, to the Staffs of the Radiological and Pathological Departments, and last but not least to Dr. C. Leonard Williams, medical officer of health for Barking, for much assistance throughout.

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YORKSHIRE REFRACTION HOSPITAL. — This institution is at present housed in temporary premises in Leeds owing to the difficulty of obtaining a more suitable building, but the Yorkshire Optical Society is now hoping to raise £30,000 for the erection of its own hospital. It has been equipped and maintained hitherto at the cost of medical

SHEEP-DIPPING AND THE SHEPHERD.

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SHEEP-DIPPING is a compulsory procedure, and practitioners in rural areas are acquainted with abdominal, cutaneous, and nerve troubles that have been attributed to the absorption of arsenic during the process. In the case we are now recording the symptoms were suggestive of arsenical or ptomaine poisoning and the medical attendant did not feel justified in giving a death certificate. We were asked to carry out the pathological and chemical investigations, and our findings may be found useful for future reference.

The sheep dips in common use are generally made up in the form of a powder containing about 20 per cent. of arsenious oxide, mixed with sodium carbonate and finely ground sulphur. When prepared according to the directions, the dipping solutions contain 0·2 per cent. of soluble arsenic. It is, however, said to be a solution of arsenic trioxide in sodium di-hydrogen arseuate, and its toxicity to be about one-half of what it would be were the arsenic trioxide dissolved without the addition of an alkali.

The actual details of the sheep-dipping vary in different parts. The conditions under which the deceased man was reported to have worked were as follows. When 500 sheep were to be dipped, 35 two-pound packets of sheep dip were obtained from the estate office stores. The ends of these were slashed open with a knife. The contents of three packets were emptied into a two-gallon bucket; water was added and the powder and lumps dissolved with the aid of a stick. The mixture was then tipped into a 200-gallon bath. The procedure was repeated until the necessary density of the bath was attained. As each packet was emptied it was placed on the ground. When a heap of packets had collected they were burned. One man drove the sheep through a trap-hurdle into the bath; another guided their way through the bath to its exit by means of pronged sticks. The dripping sheep drained off for half an hour in one pen, and were then transferred to another pen for further drying. If the day was hot the sheep dried off in 2–4 hours; if it was dull and showery 8–24 hours were required. The dried sulphur-arsenic solution could be seen and felt on the sheep for several days. At the close of the day's dipping, the arsenical bath was left overnight, and later made up again to the required volume and strength by the addition of more "dip" powder. If the bath was not wanted again the residue was washed out into drains. When the dip solution was first prepared a yellow scum appeared on the surface. This was removed by a broom. The broom, after use, was swilled out and left by the bath for future use. None of the men wore overalls, gloves, or special garments. There were no printed instructions about the precautions the men should take in order to protect themselves from the solution, but they were told individually not to allow any of the fluid to get on to any sores or into their mouths. They were enjoined to wash their hands before eating. Water was provided for the purpose, but there was neither soap, nailbrush, nor towel.

At this farm there was a man, aged 48, who had been a shepherd since boyhood. On June 27th, 1929, he and one helper dipped 125 sheep. The bath had been prepared for him by the farm bailiff. The dipping of the sheep took one hour. On July 11th he got up early to look after his sheep and had for breakfast some fried beef prepared from some ribs of beef supplied the previous day. The remainder of