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severe asthma continues uninterruptedly for days or weeks and may end in fatal exhaustion, can always be arrested by the method of continuous adrenaline injection which I first used about ten years ago. The needle is kept in position with a full syringe, and after an initial injection of a dose which is known to cause no unpleasant symptoms, one or more minims are injected every 15, 30, or 60 seconds, according to the patient's reaction, the rate being varied until it is found how frequently the injection can be made without tachycardia or other symptom arising. The injections are continued, if necessary, for even half an hour or more; relief always follows and generally manifests itself by the patient falling into a deep sleep, which may be the first he has had for several days.

Anaphylaxis can also be most efficiently treated by this method of injecting adrenaline, which is far more effective than occasional injections of larger doses. If adrenaline is always at hand when a patient is given serum or other treatment which may cause anaphylactic reaction, the latter should never prove fatal.

# PULMONARY ASBESTOSIS

## A REVIEW OF ONE HUNDRED CASES

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During the last few years we have from time to time reported the result of our observations of the effects of the inhalation of asbestos dust as seen in patients attending a chest hospital or revealed at autopsy. Our studies commenced in the year 1928, and a preliminary paper dealing principally with radiographic appearances was published by one of us in the following year. In 1930 we reported our findings in a series of 37 asbestos workers who had been examined at Victoria Park. As we now have had the opportunity of studying 100 cases, the time seems opportune for a brief survey of the whole group.

### ANALYSIS OF CASES

Our cases may be divided into four chief groups.

#### I.—PULMONARY ASBESTOSIS

Fifty-three cases (19 males, 34 females); 12 deaths (4 males, 8 females), including 2 with carcinoma of the lung and 1 with a few deposits of growth in the pleura. In the cases still alive and under observation diagnosis rests upon (*a*) typical appearances of the radiogram, (*b*) symptoms and physical signs of fibrosis, and (*c*) presence of asbestosis bodies in the sputum. These three diagnostic points are arranged in order of merit. Twenty-four cases (9 males, 15 females) were positive on all three counts; 7 (all females) had positive radiograms and clinical symptoms and signs, but no sputum was available—or if available no asbestosis bodies were found; 9 (5 males, 4 females) gave positive clinical signs and asbestosis bodies were present in the sputum, but radiograms were either not available or failed to show definite evidence of disease; 1 (male) had a positive radiogram and asbestosis bodies in the sputum but no clinical signs or symptoms.

#### II.—PULMONARY ASBESTOSIS WITH ACTIVE TUBERCULOSIS

Twenty-one cases (4 males, 17 females); of these 11 (3 males, 8 females) were fatal. One had a tuberculous empyema, one a pyopneumothorax and lardaceous disease, the others pulmonary tuberculosis. Ten cases (1 male, 9 females) are still alive. All have tubercle bacilli and asbestosis bodies in the sputum.

#### III.—PULMONARY ASBESTOSIS WITH OBSOLESCENT TUBERCULOSIS

Nine cases (4 males, 5 females). This group emphasises the fact that obsolescent tuberculosis is not necessarily reactivated by asbestosis. Three cases have been fatal (1 male, 2 females); of those still under observation 2 (females) have radiograms exhibiting typical asbestosis and healed apical tuberculosis, with asbestosis bodies in the sputum; and 4 cases (3 males, 1 female) have asbestosis bodies present in the sputum and radiograms showing obsolescent tuberculosis but indeterminate or negative as to asbestosis.

#### IV.—DOUBTFUL CASES

Seventeen cases (6 males, 11 females) under observation. All but one have suggestive symptoms and signs but the radiograms are indeterminate, and with two exceptions the sputum, where available, contains no asbestosis bodies. Of these exceptions one has indefinite symptoms, signs of emphysema, and a negative radiogram, but asbestosis bodies are present; the other has no physical signs or symptoms and a negative radiogram, but has asbestosis bodies in the sputum.

### THE CLINICAL PICTURE

The picture of pulmonary asbestosis is that of a pneumonoconiosis occurring in a factory in which few precautions had been taken to protect the workers from a danger, the gravity of which was not realised. Happily these conditions are now a thing of the past and elaborate precautions have been taken to protect the workers. There is thus good reason to believe that the disease is now under control, though it seems probable that workers exposed to the dust under the old conditions will continue to present themselves for examination for some time to come. There is no need to amplify the descriptions of pulmonary asbestosis already given elsewhere. The symptoms are indeed those common to all forms of pulmonary fibrosis uncomplicated by bronchial dilatation or pulmonary excavation—stubborn cough with little expectoration, discomfort or dull pain in the chest, occasionally perhaps a streak of blood in the phlegm, and dyspnœa. It is this last symptom of which the patient complains. The distress occasioned by constant breathlessness is so great that the disease might almost be described as monosymptomatic. It is the patient's first and last complaint. In the earlier stages dyspnœa is only apparent during exertion—"I found I could not hurry to work in the morning!"—in the later stages the act of undressing or even the effort required for speech may cause the patient to pant for breath. When asbestosis is complicated by tuberculosis, bronchopneumonia, or other thoracic disease, the symptoms of these will be added, but the dyspnœa of pneumonoconiosis can still as a rule be recognised. Malnutrition is very evident in the terminal months when the patient may pass into a condition of extreme cachexia.

### DIAGNOSIS

When a new disease is under consideration the salient features are first observed, and diagnosis depends on the recognition of gross symptoms. Thus phthisis was formerly only diagnosed when symptoms and signs of advanced disease were evident. We now diagnose incipient phthisis with confidence at a stage when the patient is healthy in appearance, well nourished, and little troubled by symptoms. In the diagnosis of asbestosis we have still to depend upon the symptoms and signs of more or less gross disease. We have no means adequate to discover during life the existence of the finer degrees of

pulmonary fibrosis which must precede graver developments, and it is not surprising that the pathologist sometimes discovers post-mortem evidence of fibrosis not recognised during life.

In diagnosis at the present time we have to rely on a history of exposure to asbestos dust as our starting point. The symptoms described by the patient may supply a clue, for the malingerer does not as a rule emphasise dyspnœa as his chief disability. In the physical examination the patient's appearance should receive special attention. In advanced cases the "earthy" character of the complexion and a slight violet tinge in cheeks or lips are noteworthy. The physical signs supply suggestive but never conclusive evidence. They are those of a basal fibrosis, and the fine nature of the pulmonary scarring is indicated by fine dry crackles not dissimilar to those often heard over the bases of emphysematous lungs. More superficial crackles produced by the movements of a damaged pleura are often mingled with the fibrotic râles.

The radiographic picture is that of a fibrosis of fine type affecting the lower two-thirds of the lung fields; the scarring is as it were etched in. Attention may be drawn to a difficulty constantly encountered when a delineation of the basal portions of the lungs is required. The diaphragmatic shadow censors the bases of the lungs, and in a radiogram taken with the ordinary antero-posterior technique as much as two inches of the lung may be blotted out. The extent of this "silent" area can be demonstrated in the normal lung by a lipiodol injection, the result of which will surprise those accustomed to regard the supra-diaphragmatic area as the lung base. Again, a lateral view of the lung, if the interlobar septum is thickened or if the middle lobe is opaque as the result of, for example, an epituberculous exudate, shows that the lower margin of the middle—i.e., of the lower portion of the right upper—lobe corresponds approximately with the shadow of the cupola of the diaphragm. At a recent meeting of the Röntgen Society Dr. L. A. Rowden pointed out that a more extended view of the lung bases can be obtained by the simple expedient of placing the patient in the dorsal position and centring the tube over the brow. We have tried this method and verified its efficacy.

The exact diagnostic significance of asbestosis bodies in the sputum is doubtful. As pointed out by one of us (S. R. G.) the formation of an asbestosis body is evidence of tissue change. We cannot assume, however, that the expectoration of a few asbestosis bodies implies that pulmonary fibrosis has already occurred. If we could make this assumption the problems of diagnosis and claims for compensation would be simple. Nevertheless the microscope will sometimes reveal commencing fibrosis in lung sections where ante-mortem radiograms failed to reveal evidence of disease. The clinical methods at present available do not enable us to determine incipient disease. The asbestosis body is simply evidence of exposure to asbestos dust and of tissue reaction thereto. This tissue reaction may be accompanied by fibrosis, but it does not necessarily follow that this is sufficiently extensive to produce symptoms or clinical signs or abnormal shadows in a radiogram—or we might add to justify compensation.

## PROGNOSIS

As the disease has only been recently recognised as a clinical entity our present ideas on prognosis may require modification. In a previous article we stated that the cessation of exposure to asbestos dust did not avail to check the spread of disease. This is certainly often true, but we have now seen patients whose condition appears to have remained stationary since stopping work in the factory. That even serious damage to the lungs is not incompatible with many years of tolerably comfortable existence is proved by such an example as that of a patient who was still able to carry out administrative duties at a factory when nearly 40 years' exposure to the dust had caused a fibrosis involving all except the apical regions of the lungs. We recently examined a female patient, at the request of Dr. C. McShane, who after a period of two and a half years' employment as a spinner had retired to get married. Twenty years later symptoms, signs, and X ray pictures supplied convincing proof of pulmonary asbestosis of a moderate degree of severity, and asbestosis bodies were readily demonstrated in the sputum. But despite some disability the patient, whose period of service did not entitle her to compensation, described herself as fairly comfortable, and she was still able to perform her household duties. It is none the less obvious that any patient who betrays symptoms of fibrosis or in whom any evidence of fibrosis is discovered, whether by physical or radiological examination, should at once cease working in any dusty occupation. A quiet life, preferably in the country, and avoidance of the risk not only of dust inhalation but of catarrhal infection, provides the best safeguards against an aggravation of incipient disease.

Septic bronchitis, broncho-pneumonia, and pulmonary tuberculosis are the commonest terminal complications of the disease.

## COMMENTS

*Occupation.*—An elaborate analysis of occupational factors is not possible in this short article. All the 67 women in our series had been exposed to asbestos dust in the same factory. They were employed in the following departments: spinning, 24; weaving, 5; mattress making, 9; carding and opening, 11; and miscellaneous or unstated, 18. Twenty-two of them began their employment before the age of 18, which is now no longer permitted under Home Office regulations. The shortest exposure was six months (two cases) and the longest 15 years.

The 33 men came under several categories. About two-thirds of them were employed in the same factory as the women and had performed a variety of jobs. The others included an elderly man who was himself the owner of a small factory, a van boy, aged 18, whose spare time during two and a half years' employment had been spent in mixing powdered asbestos in an open yard, a middle-aged boiler riveter who had served his apprenticeship as a youth in a shop where asbestos was used for lagging pipes, and a man who had been employed handling asbestos mattresses in the open air at an aerodrome. Among the men the shortest exposure was 11 months (disintegrating department), and the longest about 40 years (clerical and administrative work). Four of the group began their employment under the age of 18.

*Pathology.*—In addition to the autopsies mentioned above, 10 post-mortem examinations have been made by one of us (S. R. G.) of cases we had not seen during life (3 males, 7 females), but these are not included in our 100 cases. Macroscopically the fibrosis is best seen in the lower lobes, and adherent pleura is very common, but silicotic nodules have not been encountered. Histologically the disease begins in the respiratory bronchioles and alveolar ducts, and is characterised by a cellular reaction of

mononuclear phagocytes and asbestosis giant cells followed by the formation of a reticular fibrosis.[1]

*Tuberculosis and Asbestosis.*—In a previous article[2] dealing with tuberculosis and asbestosis we recorded 11 cases of active tuberculosis in 57 cases of pulmonary asbestosis (17·5 per cent.). The figures have now reached 21 cases of active tuberculosis out of 100 (21 per cent.). It must be borne in mind, however, that these figures do not indicate the percentage of cases of asbestosis with tuberculosis in the industry. We have been dealing with a group, mostly from one factory, who were sufficiently ill to seek advice at hospital. In discussing this question at length, Merewether[3] considers, after a review of the whole industry, that the data are too fragmentary to be dogmatic at the moment, but that whatever "the added risk of tuberculosis may be in asbestosis, it is less than that associated with silicosis." We hope to publish our material on this subject at a later date.

*Complications.*—Apart from pneumonia and pulmonary sepsis and complications arising from tuberculosis, there have been none definitely attributable to the asbestosis with the exception of two instances of spontaneous pneumothorax diagnosed by X ray, and apparently unassociated with tuberculosis, which may have been due to the rupture of emphysematous bullæ.

We are indebted to our colleagues at Victoria Park for permission to quote certain cases seen by them.

For a full account of pulmonary asbestosis in all its bearings the reader is referred to Dr. Merewether's exhaustive paper.[3]

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# SURGICAL ASPECTS OF THE SPASTIC COLON AND THE PREDIVERTICULAR STATE*

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MANY patients suffering from colon dysfunction, apart from proved examples of carcinoma, diverticulitis, and ulcerative colitis, are admitted to surgical as well as medical wards for investigation. Of this group the majority are discharged with intact abdomens and uneasy minds; others undergo fruitless laparotomy under general anæsthesia; a minority, however, operated upon under spinal anæsthesia, display a fascinating and important living pathology.

Neoplastic, ulcerative, and fully developed diverticulous diseases of the colon have no place in this paper, which concerns a group of cases suffering from abdominal pain, mainly left-sided, but with important references to other areas and sometimes associated with blood and mucus in the stools; constitative diarrhœa and constipation occur in equal proportions. Dyspepsia is often an important and misleading symptom. Concentrating mainly on those prediverticular conditions, until recently mainly of radiological interest, an attempt will be made to establish spasm as an important ætiological factor in the formation of diverticula in the colon. The disquieting thought that surgery, apart from dealing with complications such as obstruction, perforation, abscess-formation, and fistulous communications, only offers a dangerous operation for the cure of colon diverticulitis, makes investigation of the earlier prediverticular state of tremendous importance. Advances in the knowledge of the ætiology may enable effective medical treatment to be employed. If spasm be an important factor, the administration of belladonna in suspect prediverticular states will be of great value as a prophylactic measure.

*Based on a short paper read before the Association of Surgeons of Great Britain and Ireland at Birmingham on Dec. 3rd, 1934.

## SPASTIC COLON

Spastic colon is a well-recognised entity. Dr. Stacey Wilson first drew attention to it in his book "Tonic Hardening of the Colon"[1]; a tender, hard, contracted colon may be palpated in patients whom I have grouped into three classes.

(*a*) The commonest sufferer is described by Dr. Hardy[2] as being "the emotionally unstable type of individual whose colon is unstable also." Often the patient is a nervous female, thin and unhappy, perennially dyspeptic, ailing and constipated, through whose thin abdomen the racing aorta and a hard tender colon can be palpated with ease. Radiological and biochemical examination discover an active tonic stomach with pylorospasm and hyperchlorhydria. I have no evidence, nor do I think, that these patients proceed to diverticular formation. They are frequently met with in the surgical out-patient room; it is no place for them and they repay surgery with groans and a constantly increasing list of symptoms. More than one has undergone pyloroplasty, many appendicectomy, without the least relief of their dyspepsia.

It is important to remember this class of patient when investigating cases of pylorospasm. Cannon and Murphy[3] have produced pylorospasm by traumatising intestines and have declared its occurrence in these circumstances to be due to a viscero-visceral reflex. Clinically, suitable general treatment combined with the administration of medicine containing belladonna, the tincture of hyoscyamus, the perchloride of iron and of mercury, often leads to great improvement.

(*b*) Occasionally spastic colon occurs in the young athletic man who suffers from constipation; pain may be a prominent feature and may lead to operation for supposed acute abdominal conditions. No abnormality is seen other than generalised enterospasm.

(*c*) Far different is the last group represented by the rather fat middle-aged man, muscular and athletic in his younger days, sane and sanguine in outlook, but worried by an alteration in his bowel habit and because his ample meals, formerly enjoyed with relish, cause indigestion and discomfort; frequently defæcation is unsatisfactory and irregular. Small internal hæmorrhoids often develop and the descending colon is tender and hard. Rectal examination is more painful than usual, but once the sphincter has been passed the rectum itself is normal. We will consider later that the important pathological change in these cases lies in the muscle layer and not in the mucous membrane; mucus and blood are therefore not always present. The test-meal (in all the cases so far investigated) shows hyperchlorhydria. Outline in the radiological picture of the barium enema, as in the clinical picture, is indecisive