PULMONARY ASPERGILLOSIS

Short description.—A destructive disease of the lungs due to their invasion by a fungus, the Aspergillus fumigatus. The disease depends on the inspiration of the spores of the fungus, and occurs chiefly in those whose occupation brings them in contact with infected grain. Clinically the disease presents itself under two forms: (i.) like chronic pulmonary tuberculosis; (ii.) like emphysema and bronchitis.

Besides attacking the lungs primarily the aspergillus may become engrafted on pre-existing pulmonary lesions.

Historical.—Hughes Bennett, in 1842, described the first example of pneumomycosis, in which the sputum and cavities of a phthisical subject were found to contain a fungus. Gairdner, in 1853, showed a specimen of a tuberculous lung which had given rise to pneumothorax, with small circular white areas of fungoid growth on the pleural surface, penetrating very slightly (1/16 inch) into the lung substance, and measuring 1/4 inch in diameter. Rayer eleven years before, in 1842, had met with a very similar case. Bristowe in 1854 recorded the case of a woman who died with signs of chronic bronchitis; in the apex of the left lung there were two communicating vomices containing no secretion, but on the septum between them there was a powdery, velvety mass of mycelium; although there was no other evidence of tuberculosis: the vomices were regarded as being tuberculous. Virchow, in 1856, gave an account of several cases of aspergillary broncho- and pneumomycosis in patients dying from other diseases.

A number of other observers have recorded cases which, like the preceding cases, were regarded secondary infections of pre-existing pulmonary lesions.

In 1890 Dieulafoy, Chantemesse and Widal described clinically aspergillary pneumomycosis in persons engaged in stuffing and fattening pigeons for the Paris market, and struck out a new line in their view that it is a primary affection.

In 1897 Renon collected all the evidence bearing on the subject in his *Étude sur l'aspergillose chez les animaux et chez l'homme*, to which reference for an exhaustive discussion and account of the whole subject may be made.

At first and for a considerable time the occurrence of aspergillus was supposed to be no more than an accidental invasion of already diseased lung tissue, the fungus being merely saprophytic. Thus in Bristowe's case, although there was no sign of tubercle elsewhere in the lung, the lesions were regarded as tuberculous and not due to the activity of the fungus.
But lately the French school, and especially Renon, whose conclusions are based on extensive experimental research, have successfully argued in favour of primary pneumo-aspergillosis; whilst in England, Boyce and Arkle and Hinds have within the last few years described cases of the primary affection.

Aspergillar pneumomycosis may therefore be considered under the two heads—(a) primary; (b) secondary.

It is a difficult question, however, in many instances to settle whether the aspergillar affection be undoubtedly primary, and the cause of morbid lesions in a lung previously healthy; or whether it be a secondary infection only. In former times there was a strong and general impression that aspergillar occupation of the lung is essentially an accidental and secondary phenomenon.

Recently Max Podack has expressed doubts whether cases described as primary by the French observers are in reality of this nature; on the other hand, Renon regards Wheaton’s “case primarily of tubercle in which a fungus (aspergillus) grew in the bronchi and lung” as being an example of primary pulmonary aspergillosis. Thus different interpretations are put upon the same case.

Etiology.—Pulmonary aspergillosis is a trade disease in Paris; it occurs in persons whose calling is the artificial feeding of pigeons, and in those who comb and sort hair. The essential factor is the intimate relation to grain infected with the spores of the Aspergillus fumigatus. The pigeon-feeder fills his own mouth with a watery mixture of canary seeds and vetch seeds, and transfers the grain to the pigeon’s mouth. Spores of aspergillus attached to the seeds thus get into the trachea and are conducted to the air-vesicles, through the walls of which they easily pass. It is remarkable that the alimentary canal of man seems immune to Aspergillus fumigatus. According to Renon, there are only about ten persons engaged in this trade in Paris.

The hair-sorters employ the flour of rye to enable them to separate the hairs more easily; this process impregnates the atmosphere in which they work with dust, which may contain the aspergillus of the rye flour. The atmosphere of their working-rooms is so poisonous that birds die after being exposed to it for a fortnight.

Aspergillosis is a rare disease; it appears more likely to occur in millers, agricultural labourers, and those brought in contact with grain, than in any other class of the community. Apart from the Paris cases a few sporadic examples of the disease have been recorded.

Pulmonary aspergillosis belongs to a class of lesions which, though comparatively little known, has been more studied in animals than man. The lesions of the class pseudo-tuberculosis are granulomata, and resemble those of true tuberculosis, except in respect of the causal agent, which include bacilli other than those of tubercle, fungi of various kinds, and even worms (vide Distomum Ringeri, vol. ii. p. 1027). The close resemblance (to the naked eye) of the lesions of pseudo-tuberculosis to genuine tuberculosis renders it very probable that they are often re-
PULMONARY ASPERGILLOSIS

garded as such; and that, being rarely recognised, this form of lesion is not so infrequent as our present experience would suggest. Systematic examination of pulmonary lesions might prove that some conditions generally dismissed as tuberculous are in reality pseudo-tuberculous, and are due to quite a different cause. Flexner has recently described the condition of Pseudo-tuberculosis hominis streptotrichia in a man who died with the signs of pulmonary tuberculosis, and whose lungs showed consolidation with early excavation.

The aspergilli are true fungi, and belong to the family Perisporiaceae, order Ascomycetes. Of the varieties of aspergillus, two, A. fumigatus and A. niger, are parasitic, and produce morbid changes in the human body.

Pulmonary aspergillosis appears to be almost always due to A. fumigatus; A. niger has, it is true, been described in some instances, but Renon throws doubt on the accuracy of the observations, and regards them all as examples of A. fumigatus.

Both varieties have been described as attacking the external auditory meatus, and the skin.

It should be remembered that in order to determine the species cultures are necessary, and that without this no opinion as to the identity of the form of aspergillus is valid.

Aspergillus fumigatus flourishes best at the temperature 37°-40° C., while A. niger grows best at 25° C.; and this might be thought to explain the pathogenetic qualities of A. fumigatus; but in Renon's hands experiments on frogs do not support the simple view that it is merely a matter of the bodily temperature suiting the development of one species and not of the other.

* Primary pulmonary aspergillosis.—Symptoms.—The clinical features presented by the recorded cases of primary pulmonary aspergillosis may resemble either those of chronic pulmonary tuberculosis or those of emphysema.

When the disease takes the first of these two forms there is recurring haemoptysis, cough, expectoration becoming green and purulent, and signs first of bronchitis, and later of consolidation at the apex. Furthermore there is elevation of the temperature; and pleurisy may supervene. The resemblance, therefore, to pulmonary tuberculosis is so far exact; but if the sputum be examined, tubercle bacilli are absent, while the mycelium of Aspergillus fumigatus is present. The course of the disease is very slow and prolonged; recovery takes place eventually by expectoration of the aspergillus, but the affected portion of the lung undergoes marked fibrosis.

A patient affected with pulmonary aspergillosis offers a suitable soil for tubercle bacilli, and a secondary infection may take place, the aspergillus disappearing from the sputum and being replaced by tubercle bacilli. Renon and Sargent have recorded a case of primary pulmonary aspergillosis succeeded by tuberculosis, in which eventually both these infections became obsolete: but so much chronic pneumonia resulted
that death from failure of the right side of the heart terminated the case. In another and similar case related by Renon the sputum first contained the aspergillus alone; later very scanty traces of it were found, but plenty of tubercle bacilli, and eventually no bacilli or aspergillus, the patient surviving with evidences of chronic pneumonia.

In the emphysematous form the disease may run a rapid course, as in the case recorded by Arkle and Hinds. Hæmoptysis is infrequent, or may not occur at all; there is loss of flesh and strength, frequent cough and severe dyspnoea come on in attacks at night, and suggest spasmodic asthma. The physical signs are chiefly those of emphysema and bronchitis.

Intermediate forms between these two may occur, signs of apical consolidation supervening in the emphysematous varieties; and conversely cases which appeared like chronic phthisis may be marked by attacks of pseudo-asthma.

Morbid anatomy.—The data at our disposal are somewhat scanty, but so far as they go they tend to show that the morbid appearances in the lungs met with in the described cases of aspergillosis differ just as do the lesions of acute and chronic tuberculosis. This difference depends on the resistance offered by the lung tissue to the inroads of the fungus. It will be most convenient to describe the anatomical lesions in connection with the two chief clinical types of the disease to which attention has already been called.

1. In cases where the disease has run a very chronic course, resembling either chronic pulmonary tuberculosis or chronic pneumonia, the aspergillus may either (a) still be found on the lung tissue, or (b) it may have been entirely removed, and then have left behind it a chronic interstitial pneumonia which eventually proved fatal.

(a) Our knowledge of the lesions existing in primary aspergillosis when the aspergillus is still present in the lung tissue is particularly scanty. Renon bases his description on two cases, those of Boyce and Kohn. The lung tissue contains dilated bronchioles leading into cavities in pneumatic areas, in which there are pseudo-tubercles composed of hyphae so arranged as to resemble actinomycosis. There is much phagocytic reaction in the pneumatic areas, showing that very active resistance had been opposed by the lung tissue to the aspergillar invasion. Renon associates the actinomycotic form adopted by the aspergillus with the active resistance of the tissues, and considers it as an indication of defensive powers on the part of the tissue and of lowered vitality on the part of the aspergillus. Hence this form of pneumo-aspergillosis is called by Renon "abortive." The cavities also contain the aspergillus. The process is essentially the same as that in cases of aspergillosis; its clinical features are those of emphysema, namely, consolidation and destruction of pulmonary tissue; but it is a local process which has become arrested at an earlier stage.

(b) In a case of primary pulmonary aspergillosis, described by Renon and Sargent, in which true tuberculosis supervened with disappearance
of the aspergillus from the sputum, death took place from failure of the right side of the heart, and examination of the lungs showed chronic pneumonia; but no trace remained either of the aspergillus or of tubercle bacilli.

2. In cases where the symptoms have been those of emphysema and dyspnoea the lungs contain patches of consolidation breaking down into cavities, while there is compensatory emphysema which may be well marked. The lesions in Hind and Arkle’s case have some analogies with Tooth’s case of acute bronchiolectasis, though in the latter the causation had nothing to do with aspergillosis.

Microscopically the walls of the small bronchi are thickened, and both the lung substance and the alveolar cavities contain the aspergillus mycelium. In places the lung tissue is so disorganised as to be unrecognisable, and there is breaking down of the lung tissue leading to the formation of microscopic cavities. The mycelium is in extremely intimate relation with the lung tissue, and, as it is accompanied by phagocytic reaction, the aspergillar invasion of the lung tissue appears to be the direct cause of the lung lesions, not a merely accidental or post-mortem event.

Since no toxin has been obtained either from the media in which the Aspergillus fumigatus is grown (Kotliar), or from the fungus itself (Renon), it appears probable that the large quantities of the fungus in the lung tissue set up the inflammatory changes by mechanical irritation. The absence of any toxin explains the comparatively mild character of the disease; but it makes it somewhat difficult to understand why A. fumigatus is the chief if not the only variety of aspergillus pathogenetic for pulmonary tissue.

Generalisation of aspergillosis does not occur.

Diagnosis.—The physical signs are not in any way characteristic, and would point to bronchitis and emphysema or to chronic pulmonary tuberculosis. In Wheaton’s case there was a growth of the fungus at first white, later black on the tongue and palate. But this is the only help that ordinary methods of physical examination can be expected to supply, and, unfortunately as regards diagnosis, this coexistence of oral and pulmonary aspergillosis is almost unique.

The diagnosis depends on the presence of the fungus in the sputum, and the absence of the tubercle bacillus. In cases where tubercle becomes engrafted on primary pneumo-aspergillosis, both organisms might be found in the sputum; and, unless the patient had been under observation from the beginning when the aspergillus alone was present in the sputum, there would be no means at first of distinguishing the primary form complicated by tubercle from secondary aspergillosis occurring in the last course of pulmonary tuberculosis.

The fungus, derived from dust, is occasionally found in the mouths of healthy persons.

Cultures of the aspergillus in appropriate media, such as Raulin’s fluid, and inoculation of animals may be necessary to determine that the form
of aspergillus is the pathogenetic Aspergillus fumigatus, and not the other non-pathogenetic varieties, such as Aspergillus niger, glaucus, and so forth. It must be distinguished from the streptothrix form of the bacillus tuberculosis; and, lastly, the lesions must be distinguished from other forms of pseudo-tuberculosis due to different factors such as bacteria, streptothrix, actinomyces, or Distoma Ringeri.

The prognosis of pulmonary aspergillosis is less grave than that of pulmonary tuberculosis, since the lesion is usually much slower, never sets up a general infection comparable to generalised tuberculosis, and tends to undergo a gradual and spontaneous cure. But there are several reservations to this general statement. For, even if the aspergillus disappear, the lesions of chronic interstitial pneumonia may lead to dilatation of the right side of the heart, and so to a fatal result.

It need hardly be said that the development of genuine tuberculosis renders the prognosis much graver.

The prognosis of the emphysematous form does not, from the few recorded examples, appear to be nearly so favourable as that of the more chronic variety which has been likened to chronic tuberculosis.

Treatment.—Although there is no specific remedy for pulmonary aspergillosis, nor any drug that can be employed to kill the fungus outright in this situation, experiments on animals show that iodine, iodide of potassium, and arsenic increase the resistance of the organism to the invasion of Aspergillus fumigatus and inhibit its growth; their employment is therefore reasonable in this disease in man. The general strength should also be improved by good and generous feeding, cod-liver oil, tonics, and fresh air; thus we may guard against secondary infection of tubercle, and assist the tissues in their struggle against the aspergillar y infection.

Symptoms should be treated as they arise. When hemoptysis occurs the treatment is the same as in pulmonary tuberculosis. Attacks of asthma may be relieved by iodide of potassium, tincture of lobelia, and other appropriate remedies; while creasote, terpene, turpentine, may with other drugs be given for bronchitis.

Removal from the poisonous atmosphere is an important essential, both in prophylaxis and in treatment.

When tuberculous infection has taken place, the course of treatment is that of chronic pulmonary tuberculosis.

Secondary pulmonary aspergillosis.—Here the Aspergillus fumigatus develops as a result of the inhalation of its spores; and finds a suitable nidus in lung tissue the resistance of which has been already much lowered by pre-existing disease, or has actually undergone necrosis.

It has been found in the bronchi and in the lung substance. Thus the aspergillus may be engrafted on bronchiectasis of old standing; or may take root on the walls of vomices due to tuberculosis; or in the lung under other conditions, such as malignant disease, pulmonary apoplexy, chronic bronchitis, broncho-pneumonia, and gangrene of the lung.

In some of the cases where it has been described as secondary, it may,
 EMPHYSEMA OF THE LUNGS

as already hinted with regard to Dr. Bristowe’s case, in reality have been primary.

In cases where there are multiple bronchiectases or vomicia in the lungs, the absence of the fungus from some of them and its presence in others are strong evidence in favour of the secondary nature.

It is remarkable that in gangrene of the lung associated with the presence of aspergillus there is no fæctor. It seems that the growth of the micro-organisms of putrefaction is prevented by the aspergillus.

The actinomycotic form of the mycelium appears to occur where there is considerable reaction and resistance on the part of the tissues, and it is probable that it does not occur in secondary or terminal aspergillosis.

Clinically speaking, secondary aspergillar pneumomycosis, like thrush in the mouth of adults, is probably a precursor of death, and is not likely to be suspected or discovered unless the mycelium be found in the sputum. It is in fact a terminal complication.

The treatment is that of the primary disease on which the aspergillosis has been engrafted.

H. D. Rolleston.

REFERENCES


H. D. R.

EMPHYSEMA OF THE LUNGS

Definition.—A disease of the lungs characterised by over-distension of the alveoli and atrophy of the alveolar walls.

It has been the custom to describe under this heading two essentially distinct morbid conditions; the one, corresponding in anatomical details to the definition above given, having nothing in common with the other but the name. An account of this latter affection, interlobular or interstitial emphysema, will be found at the end of this article.

The description of emphysema of the lungs given by Laennec, accurate though it was as regards both anatomical characters and clinical history,
remained incomplete until supplemented by the microscopical researches of Rokitansky and the clear exposition of its pathology which we owe to Sir William Jenner. Our knowledge of the disease has been mainly derived from their writings, and few additions of importance have been made to it in recent years.

Pathogeny.—Various hypotheses have been advanced to explain the origin of emphysema, some of which meet with but little support at the present time. It would serve no useful purpose to enter upon a detailed discussion of the problem, as it is exhaustively dealt with in the original papers of Sir William Jenner, to which reference may be made. It will be sufficient to mention those views which have at any time received considerable support, and to discuss in greater detail that which is now generally adopted.

Primary degeneration hypothesis.—The view that the general cause of emphysema is a primary fatty degeneration of the alveolar walls was first stated by Rainey, and subsequently received support from Villemain. The latter writer describes the changes as beginning in an excessive proliferation of the intercapillary nuclei, followed by secondary fatty degeneration of the nuclei and other structures, the result of pressure upon the capillaries. It is now generally considered that the degenerative changes in the alveolar walls are secondary to the distension of the air-vesicles and interalveolar spaces, and to the diminution in the blood-supply thereby induced.

It is possible, however, that in the form of emphysema met with in old people, primary degenerative changes may play a more important part. Reference will be made to this point subsequently.

Inspiratory hypothesis.—The hypothesis that emphysema is due to distension of the lungs during inspiration was really first advanced by Laennec. He believed that the air drawn into the lung in inspiration was retained, being unable to escape during expiration, owing to the obstruction caused either by catarrhal swelling of the mucous membrane of the bronchi or by accumulation of mucus in the tubes; and that as a consequence the lungs became over-distended with air.

Dr. Gairdner, in 1850, stated the inspiratory hypothesis in a different form. According to his view, some change in the lungs, such as collapse or retrocedent tubercle, leading to a diminution in size in one part, preceded the establishment of emphysema. As the air-vesicles within the area of disease or collapse did not expand during inspiration, an undue strain was thrown upon those in the immediate neighbourhood by the incoming air, and in consequence they became enlarged.

This opinion, as regards the general disease, has been completely displaced by that to be next mentioned; and as an explanation of the conditions found around patches of collapse or of fibroid tubercle—compensatory emphysema—it is believed that the distending force of inspiration, although possibly not without effect, is subordinate to that of forced expiration.

Expiratory hypothesis.—In 1845 Mendelssohn first advanced the opinion
that emphysema is produced during a forced expiration. He believed that the air is prevented from escaping from the upper lobes by the compression of the lungs during forced expiration; that consequently the pressure within the lung is increased, and the air-vesicles undergo dilatation. In 1857 Sir William Jenner stated the above-named hypothesis in the following terms: "The lung during expiration is compressed at different parts with different degrees of force. The parietes of the thorax, in consequence of their anatomical constitution, yield to the same force at different parts with various degrees of facility. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure."

In a footnote to his paper on "Emphysema of the Lungs," in Reynolds' System of Medicine, Sir William Jenner stated that he was unacquainted with Mendelssohn's paper when he advanced this hypothesis in 1845; and that, so far as he was aware, the existence of that paper was unknown in this country until 1867, and rarely, if ever, referred to abroad until that date.

Having regard to the above facts, to the singular completeness of Jenner's papers, and to his demonstration of the exact sites of emphysema, we may fairly regard him as having been the first to make known the true mode of origin of the disease.

The increased pressure in the air-passages, which we have seen to be a common antecedent of emphysema, may be induced in various ways.

Cough.—The almost invariable association of some degree of emphysema with chronic bronchitis points to cough as the most frequent cause of the disease. The chest having first been filled with air, the glottis is closed, a violent expiratory effort is made during which the tension within the air-passages is enormously increased, the glottis then relaxes, the air passes rapidly through the narrow orifice, and a cough results. It is the frequent repetition of this act which eventually induces a permanent dilatation of the air-vesicles and interalveolar passages. The effect of the compression of the lungs during a violent expiratory effort, such as that above described, is to drive the air in all directions from the central to the peripheral part of the lungs; the result is the distension of those parts which are least supported. As pointed out by Sir William Jenner, these parts are the apices, the anterior margin of the upper lobes, and the margins of the bases of the lungs. These are the sites of the primary lesions; but, in the course of the enlargement of the thorax which they entail, the relative position of a given area of lung and the chest wall gradually changes, fresh portions being brought into contact with the intercostal spaces, the resisting power of which is less than that of the ribs, and thus in course of time the change may become general throughout the lungs.

Muscular effort.—It is probable that next to cough violent muscular effort is the most common cause of emphysema. The mechanism is as
follows:—the lungs having been completely expanded by a deep inspiration, the glottis is closed; any severe and sustained muscular effort with the thorax in this position necessarily subjects the lungs to strong compression, the increase in pressure within the air-passages being most effectual in distending the lung in those situations where the organ meets with least support. Straining in constipation may have the same effect.

Further reference will be made to causes of over-distension in describing the etiological factors of the disease.

It will be convenient here to refer to those conditions of a temporary nature which lead to over-distension of the air-vesicles. In such cases when the cause is removed the effect may disappear; but whether it does so or not depends upon the duration of the exciting cause and the integrity of the elastic tissue of the lung.

The best example which can be given of this temporary over-distension of the lungs is the condition observed during a paroxysm of asthma. At the height of the attack the lungs may be found distended with air to a degree equal to that present in the most advanced cases of emphysema; but when the attack has passed off, the organs may return to their previous size. It is rare, however, to meet with patients whose asthma is of long standing who are not also the subjects of emphysema.

The mechanism by which this state of over-distension is produced appears to be a matter of doubt; the explanations vary with the hypotheses concerning the cause of the asthmatic paroxysm. If the hypothesis of a spasm either of the diaphragm or of the muscles of inspiration be held, there is little difficulty in understanding why the chest is in a condition of extreme inspiratory distension; if, on the other hand, we reject both these views and accept that now generally received, namely, that the asthmatic paroxysm is due to bronchial obstruction, the result either of a spasm of the muscular fibres of the bronchi or of a fluxionary hyperæmia of the bronchial mucous membrane, the explanation of its mode of occurrence is not quite so obvious.

It is, as a rule, gradual in onset and also in decline, and is apparently brought about in the following manner:—

(i.) The bronchial obstruction induces increased inspiratory effort.

(ii.) The entering air passes the obstruction with difficulty, but the gradually increasing prolongation and force of the expiratory act shows that the air meets with still greater difficulty in escaping from the lungs.

(iii.) Expiration, although prolonged, is not sufficiently so to produce an equilibrium between the incoming and outgoing air; a fractional addition is therefore made to the residual air by each completed act of respiration, and in time the lungs become over-distended.

It may be objected that, as the force of expiration is greater than that of inspiration, the obstruction should be more easily overcome by the outgoing than by the incoming current of air; but it would appear that experience teaches us to rely upon forced inspiratory efforts to
remedy a defective circulation of the blood, whereas the condition really requires for its relief forced efforts limited to the period of expiration.

Another possible factor in the production of this state of extreme distension is the compression of the smaller bronchi by the distended alveoli, an effect necessarily more felt during expiration.

Other causes of temporary over-distension of the lungs are laryngeal obstruction, from whatever cause arising, whooping-cough, acute bronchitis in children, and severe muscular strain.

Causation.—Age.—It is a matter of common experience that the disease may be met with at any age. Some of the most marked examples are seen in young children. The atrophic form of the affection (see Varieties of Emphysema, p. 269) is most often met with in old people.

Sex.—Men are naturally more subject to the disease than women, as they are more exposed to the conditions which favour its development.

Occupation.—Any occupation involving severe muscular effort, especially if performed with the lungs distended and the glottis closed, tends to produce emphysema. In all such efforts the chest is forcibly compressed by muscular contraction, and the act is equivalent to one of forced expiration. The classical example of an occupation involving the latter condition is that of a cornet-player. Smiths, hammermen, and porters engaged in lifting heavy weights are all liable to emphysema. Omnibus and cab drivers, and all persons whose occupations involve exposure to inclement weather, are prone to attacks of bronchitis, whence comes emphysema. The inhalation of dust, a condition almost inseparable from many occupations, necessarily induces catarrh of the bronchi; upon this cough and emphysema follow.

Diseases such as whooping-cough and chronic bronchitis present the conditions essential to the production of emphysema to the fullest extent. The violent respiratory acts in many forms of dyspnœa may lead to extreme over-distension of the lungs, which may be either temporary or permanent. The same is true in cases of extensive collapse of the lungs as regards those parts into which the air is free to enter. The mode of production of emphysema in asthma and allied conditions has been considered above.

The onset of emphysema will naturally be favoured by any conditions, such as chronic congestion from valvular disease and chronic bronchitis, which tend to diminish the natural elasticity of the lungs. Advancing age is a factor which operates in a similar manner.

Hereditary predisposition.—It has been suggested that there exists in some individuals and families an hereditary tendency to the disease; but this view is rarely insisted upon at the present time. Various observers have investigated this matter, the result being the supposed discovery of the hereditary tendency in a proportion of cases varying from 12 per cent (Lebert) to about 60 per cent (Fuller, Jackson) in adults, and 100 per cent of cases in children (Jackson). It is probably true, as pointed out by Sir William Jenner, that the tendency is not to the disease itself, but to conditions which dispose to it.
Although, however, we may not admit heredity in its most absolute sense to be a cause of emphysema, it does not follow that what, in the absence of precise knowledge, we call the "constitution" of the patient has no influence in determining its occurrence. The tone of muscle and its capacity for energy vary enormously in different persons, though no structural differences can be demonstrated; and the same may be true of the elastic tissues. That such is the case is certainly possible, and in my opinion probable; if so, the occurrence of dilatation of the pulmonary alveoli may well be brought about in certain persons by a degree of increased pressure within the air-passages, such as accompanies ordinary straining efforts, which we are not accustomed to regard as adequate to the production of emphysema, and which, in persons of firmer fibre, are not adequate.

All who have studied the subject of emphysema from a clinical standpoint must have met with cases in which the ordinary proximate causes of the disease seemed to be absent. In many of these the absence has, it is true, been but apparent, for it is difficult to realise how slightly a chronic winter cough impresses itself upon the memory of some patients; hospital patients, indeed, rarely mention such an ailment unless directly questioned about it. But due weight having been given to this source of error, there undoubtedly remains a certain small proportion of cases in which no adequate exciting cause can be discovered. This lack of resisting power on the part of the elastic tissues of the lung may certainly be acquired, it may possibly be inherited, and is probably a common result of the degenerative processes incidental to advanced age. A case recorded by Hugner proves clearly that after recovery from an attack of pneumonia emphysema of the affected part may ensue upon the resumption of an occupation, such as that of a cornet-player, which favours the occurrence of the disease, but which had been previously followed without injury to the lungs.

**Normal anatomy of a pulmonary lobule.**—A lobule of the lung may be regarded as a lung in miniature; a clear idea of the structure of a single lobule will therefore enable us without much effort to construct the whole organ.

Each lobule, more or less cone-shaped, is surrounded by areolar tissue; at its apex the lobular bronchus, the blood-vessels, lymphatics, and nerves unite to constitute it. The bronchus, after a short course within the lobule, divides and subdivides, with a first but slight diminution in size, forming passages which are termed the interalveolar or intervessicular passages. The course of the bronchus is at first fairly straight, but as the divisions increase in number and diminish in size the direction constantly changes. As the alveolar passages approach the surface of the lobule they cease to diminish in size. Each passage beyond the final division ends in a blind extremity, which, if not dilated, often appears to be so, from the fact, above stated, that the passages do not diminish in diameter. In some cases, however, the ends of the alveolar passages are really dilated, and from this appearance the name:"infundi-
bula" has been applied to them; but a distinctive name is scarcely necessary. As the bronchus enters the lobule rounded orifices appear upon its walls. These are the openings of the alveoli, which may be regarded as the radicles of the bronchial tree. They are at first but few in number, but gradually increase. As the air-channel passes onwards through the lobule, and the interalveolar passages are formed, their walls become more and more thickly studied with the orifices of the air-vesicles, until, by the time the surface of the lobule is reached, the blind ends of the passages are found to consist entirely of the orifices of these small recesses.

From the foregoing description it will be seen that the air-vesicles of the terminal passages open into a common space, adjacent vesicles being separated by incomplete partitions; and that all the air-cells of a single lobule are, to a considerable extent, confluent one with another. Adjacent interalveolar passages are separated by partitions formed at the site of branching of the air-vessels.

The interalveolar passages and their terminations are chiefly composed of unstripped muscular fibres, arranged circularly, and supported by a delicate fibroid tissue mingled with elastic fibres. The walls of the air-vessels consist of a delicate membrane crossed by a network of elastic fibres.

The capillaries on the terminal passages are covered by epithelium only on the surface looking towards the cavity; those in the septa project into the cavities on either side.

**Morbid anatomy.**—The primary lesion in emphysema consists in an enlargement of the terminal interalveolar passages, which increase in size at the expense of the alveoli opening into them. Sometimes, however, the alveoli appear to be the first to undergo dilatation. In any case the effect is, by pressure and stretching, to diminish the blood-supply to the epithelial and vascular structures in their walls. The alveolar epithelium undergoes fatty degeneration, the granules being aggregated round the remains of the nuclei. The septa between adjacent alveoli are reduced to small projections by a gradual process of wasting; subsequently the partitions between neighbouring alveolar passages are perforated, and they become fused into rounded spaces, the size of which tends to increase with the continued operation of the immediate cause of the disease. It is obvious that this process must be accompanied by a great destruction of the pulmonary capillaries, an important factor in determining some of the effects of the disease. According to Rindfleisch, wide communications are formed between the pulmonary artery and the pulmonary and bronchial veins, thus relieving the tension of the former vessel, but allowing the blood to pass through the lungs without undergoing proper aeration.

**Varieties of emphysema.**—Certain varieties of the disease may be recognised both clinically and pathologically; the morbid changes by which they are characterised will now be considered.

*Large-lunged emphysema (Chronic hypertrophic emphysema).*—The objection to the term hypertrophic as applied to this condition is that its
use connotes increased functional activity, whereas in emphysema the opposite condition prevails. The name here adopted, which was first suggested by Jenner, appears preferable, as it describes the condition and involves no hypothesis.

When the thorax is opened the lungs not only fail to collapse, but remain fully distended, and, when the smaller bronchi have been obstructed from inflammation, may even bulge forward. The apices fill the supraclavicular regions, and the enlarged anterior margins may be in contact beneath the whole length of the sternum, the precordial area being occupied by the distended auricular process of the left upper lobe. The diaphragm is depressed owing to the permanently inflated condition of the lungs. After removal, when the organs are held with the base upwards, the distended and rounded edges of the lower lobes form the sides of a deep cup.

The lungs in emphysema were likened by Laennec to a pillow of down, and the simile can scarcely be improved upon. They are soft and non-crepitant; when compressed a deep pit forms and remains. They are pale gray in colour, and are marked by black pigment, scattered over the surface in lines and spots, the lines in some cases mapping out the lobules. On close inspection the superficial portions have the appearance of a very fine froth, consisting of very minute air-bubbles covered by the pleura. This is rendered more obvious by the use of a hand-lens.

In some cases large rounded air-containing bulles are present, usually along the anterior margin of the upper lobes or around the bases, but they may be absent when the disease is advanced and widely disseminated. Some are attached to the lung by a narrow peduncle only, the auricular process of the left upper lobe being a common site of this particular lesion. They collapse when opened, and delicate fibrous bands, the remains of alveolar septa and obliterated vessels, may then be found crossing the interior.

These two forms, the "local" or "bullous" and the "general," are too frequently associated to justify a separation in nomenclature; but it is important to bear them in mind, as will appear when we come to consider the physical signs of the disease.

On section the lungs are bloodless and dry, except perhaps at the bases, where edema may be present. This, however, pertains more to some complication, such as bronchitis or cardiac failure, and is no necessary effect of the disease.

If the section be made from the extreme posterior margin forwards, the portion of the lung which occupies the hollow beside the spine will often be found in an advanced condition of emphysema; large spaces being present beneath the pleura, and extending for perhaps half an inch or more into the lung.

The smaller bronchi are in some cases dilated to a slight degree, but bronchiectasis is by no means frequently associated with emphysema.

Atheroma of the pulmonary artery is commonly present, and in advanced cases patches may be found throughout the vessel, not even the
smaller branches escaping; it is a result of the increased strain on the walls of the vessel from the obstruction to the passage of the blood through the lungs. There is very often a complete absence of pleural adhesions, a condition rarely observed in adults unless they are subjects of emphysema.

Small-lunged emphysema; Senile atrophic emphysema (syn., Senile atrophy of the lungs).—The most striking clinical and pathological characteristics of this condition of the lungs are indicated by its name. It appears to be primarily an atrophic change, incidental to advanced age, and shared by the lungs equally with the other organs of the body. Its title to be considered either as a substantive disease of the lungs or as a distinct variety of emphysema is doubtful. It never occurs apart from a general condition of atrophy; and the slight degree of emphysema which accompanies it is probably induced by the cough of a bronchial catarrh, from which the very aged are rarely quite free. It is, however, convenient and in accordance with custom to describe it as a variety of emphysema. The subjects of senile emphysema present a wasted, shrivelled, and withered-up appearance: the thorax is rigid, the space within is small, the lower ribs are almost in contact and very obliquely placed. On opening the chest the uncovered area of the heart is not diminished, it may even be enlarged; the lungs readily collapse, falling back towards the spine; they are smaller than normal, deeply pigmented, almost black in colour; light, dry, and easily compressible. On section they present a coarsely reticulated structure. The vesicles are enlarged by a process of fusion, the result of wasting of the septa; and this change may in places be so advanced as to involve adjacent lobules. Large bullæ are rare, but the margins are in some cases much dilated. The bronchi are thin-walled, and have undergone dilatation; the lining membrane is commonly inflamed, and the tubes contain puriform fluid. Collapse and oedema are often present, and are generally most marked on the posterior aspect of the lower lobes.

Local emphysema; Compensatory emphysema.—This form of the disease is invariably secondary to some pulmonary lesion, most commonly to tuberculosis which has undergone either complete or partial arrest. In the presence of a contracting lesion within the lungs—for instance, a cavity or an area of fibroid tuberculosis—either the surrounding tissue becomes emphysematous or the pleura thickened; the result being determined by the nature, site and extent of the lesion. In the case of a lesion situated close to the surface, if the lung intervening between it and the pleura be condensed, airless, and incapable of expansion, the visceral and parietal layers of the pleura, partially united by fine fibrous bands, tend to become separated. The space is at first filled with yellow serous exudation, which ultimately undergoes transformation into a thickened fibroid tissue almost cartilaginous in density. The apex of the lung, in cases of very chronic pulmonary tuberculosis, when the upper lobe is almost completely occupied by a contracted thick-walled cavity, shows such a thickening of the pleura as is here described. If, on the
other hand, the lung tissue around the lesion is not the seat of such advanced changes, and still admits of the entrance of air, the surface vesicles enlarge, coalesce, and form bullæ, sometimes of considerable size. Such a condition is commonly seen at the apex of the lung, and is a certain guide to a contracted lesion within. The surface may be scarred and puckered, and on section dense pigmented fibrous bands are seen surrounding old fibrous, caseous, or calcareous lesions, and extending into the neighbouring emphysematous tissue. The vessels and bronchi in such an area are usually obliterated, but on its confines the latter may be found dilated.

Another common site of local emphysema is the posterior and upper part of the lower lobe. Here the change is secondary to a contracting lesion, usually a cavity, at the apex of the lung; and may occupy a considerable area. In one such case observed by myself the posterior aspect of the contracted upper lobe was completely covered by the upper part of the lower lobe. No bullæ are formed, but on section a coarsely-reticulated structure is seen, replacing the normal tissue and reaching downwards along the posterior aspect of the lobe.

In cases of fibroid transformation of tubercle the densely pigmented contracting fibrous nodules are often found embedded in emphysematous lung; the whole presenting appearances which show unmistakably that the fibrosis has preceded the emphysema.

*Acute vesicular emphysema.*—The definition of the disease given at the head of this article does not include a lesion consisting merely in an over-distension of healthy alveoli, such as is present in the above-named condition. Atrophy of the alveolar walls is an essential part of the morbid anatomy of emphysema, and in its absence we cannot recognise acute vesicular emphysema as a true variety of the disease. It is sometimes found after death from acute bronchitis, or from asphyxia, which had been accompanied by violent inspiratory efforts; or when, from collapse or other cause, the air has been prevented from entering portions of the lung, thus throwing an increased strain upon the alveoli of other parts.

It may be demonstrated, however, by physical examination that a similar condition is present in cases which are not fatal; and also that after a time the lungs return to their normal size, a proof of the absence of structural damage.

The lung in such a condition of over-distension is large and pale, and with a hand-lens the increase in size of the surface alveoli can be readily seen.

*Lesions associated with emphysema.*—*Lungs.*—Although, in the majority of cases, bronchitis and emphysema stand related to one another as cause and effect, it is nevertheless true that when emphysema has become established it increases the tendency to bronchitis.

The over-distended air-vesicles compress and obstruct the capillaries and impede the circulation through the pulmonary and bronchial vessels. The bronchial mucous membrane becomes congested, and the condition
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thus established greatly increases the liability to inflammatory attacks. Rupture of dilated vesicles may lead to pneumothorax; but if the pleura overlying the site of rupture remains intact, interlobular emphysema results. Death is rarely due to pneumothorax so caused, but one such case has been observed by myself, and others are on record.

Bronchi.—As already described, the bronchi are often found obliterated and forming thin fibrous bands in large emphysematous bullae; they are, however, occasionally, but not commonly, found dilated to a moderate degree in less advanced cases of general emphysema, and more often in localised emphysema. In the atrophic form the bronchial walls are usually thin; in other forms they may be somewhat thickened, as may also be the walls of the vesicles and interalveolar passages.

Heart.—The obstruction to the flow of blood through the capillaries of the lungs naturally increases the pressure within the pulmonary artery and requires a more forcible contraction of the right ventricle. This leads to hypertrophy of the ventricle, and thus for a time equilibrium may be restored. But when, from any cause, the structural integrity of the new muscular tissue is impaired, particularly if at the same time greater stress is thrown upon the right ventricle, dilatation follows, the tricuspid orifice enlarges, and the valve becomes incompetent.

The right auricle, probably already somewhat enlarged, now undergoes still further dilatation, and the superior and inferior venae cavae are similarly affected. Congestion of all the organs which are drained by the systemic veins necessarily follows. The portal system may become involved at a later period. This sequence of events is not uncommonly initiated by an attack of bronchitis.

The dilatation and hypertrophy of the right ventricle, including the sinus arteriosus—for the latter is always involved—are usually found on autopsy to be associated with similar but less advanced changes in the left ventricle; a result probably due, at least in part, to their intimate association both in structure and functional activity.

Degenerative changes are often observed in the heart in emphysematous subjects, and the impaired nutrition of the muscular walls may be due to obstruction to the return of blood by the coronary veins.

As a result of the enlargement of the lung and the permanently depressed state of the diaphragm, the position of the heart becomes altered. It lies lower in the chest, and its axis is more nearly horizontal. The front of the heart is formed entirely by the enlarged right ventricle and auricle. The altered position and size of the organ account for the pulsation commonly observed in the epigastrium in well-marked cases of emphysema; but of these two factors the change of position is the more important.

Secondary changes of a fibroid character are not infrequently found in the tricuspid and mitral valves; and, more rarely, in the aortic valve also.

Liver.—The changes in the liver resulting from chronic venous congestion are too well known to require complete description. The organ...
is enlarged and the hepatic veins are dilated. The section presents the "nutmeg" character, and there is some degree of induration; but emphysema alone is as powerless as chronic mitral disease to produce a true cirrhosis.

- The kidneys may be enlarged and cyanotic, but in a considerable proportion of cases they are granular from the presence of chronic interstitial nephritis, a disease with which emphysema is not uncommonly associated. The spleen is as a rule enlarged and hard, but its condition, varies.

Chronic venous congestion of the stomach may give rise to catarrh and hæmorrhage into the mucous membrane. The brain also shows evidence of venous congestion.

As considerable differences exist in the symptoms and physical signs which characterise the various forms of emphysema, it is necessary to describe them under their respective headings.

**Symptoms of large-lunged emphysema.**—The symptoms strictly referable to emphysema are very few, the condition, apart from its complications, being one of which patients have little or no knowledge, and one of which therefore they rarely complain.

Dyspnœa is the most important symptom, but even this is seldom mentioned until it has become somewhat urgent: it is in proportion to the extent of the disease. At first slight, and only experienced on exertion, especially on walking up hill, it may gradually increase, until in the end not only exercise, but even movement becomes impossible.

It is always much increased during an intercurrent attack of bronchitis, and tends, as the disease progresses, to occur in paroxysms, a condition to which the term "bronchial asthma" is usually applied. The asthmatic element in such cases may either arise directly from the emphysema—the more common order—or the emphysema may be a consequence of asthma. The difficulty of breathing is increased by anything which interferes with the descent of the diaphragm, such as flatulent distension of the stomach or intestines, stooping, or sitting in a low chair after a meal. Orthopœna follows as the disease progresses, the patient sleeping either propped up with pillows or in a sitting position.

Cyanosis may be considerable, even whilst the patient is still capable of movement—a combination rarely met with except in this disease.

Cough.—Sufferers from emphysema are rarely free from cough for long intervals, although cough is, strictly speaking, due rather to the condition of the bronchi than to the change in the lungs. It is loud, harsh and wheezing, and, like the dyspnœa, may occur in paroxysms. It is always more troublesome in the winter, and particularly so when the weather is cold and damp, or when fog is present.

Expectoration.—Emphysema does not of itself give rise to secretion, but it is by no means uncommon for patients to expectorate a small quantity of mucus to which the descriptive word "peary" is usually applied. When bronchitis occurs, expectoration becomes profuse, and passes through the various phases usual in this disease.
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Hemoptysis, although an unusual complication of emphysema, may occur, and may even prove fatal. It is generally small in amount. Having regard to the frequent association of atheroma of the pulmonary artery with emphysema, it is perhaps surprising that rupture does not more often happen.

The appetite is often poor; complaint may be made of flatulent distension of the stomach and intestines, and constipation is not uncommon. The deficient aeration of the blood may give rise to drowsiness and headache.

The arteries are badly filled owing to the distension of the venous system, and consequently the pulse is small and weak. The blood-pressure is low, but may be observed to rise during the act of coughing (Jenner). In the later stages, when the muscular tissue of the heart has undergone degenerative changes, its action often becomes irregular and intermittent.

The veins of the neck are usually distended, and they may pulsate and fill from below. Filling from below is a sign that the valves at the orifice of the jugular veins are incompetent. Forcible pulsation usually indicates that the tricuspid valve is incompetent, but a slight impulse may be the result of the impact of the blood against the tricuspid valve being transmitted through a distended right auricle to the over-filled jugular vein, or it may possibly be due to the systole of the auricle.

An impulse may also be produced in a distended jugular vein by the systolic wave in the underlying carotid artery.

The physiognomy of emphysema is characteristic. In the earlier stages of the disease the face is full, the lips are thick, and the mucous surface is congested. At a later stage, when emaciation has occurred, the appearance alters. The lines of the forehead are now deep, the brows knit, the naso-labial folds distinct, the expression careworn. The face is of a faintly bluish tint, the colour being well marked in the lips, which are thickened; the eyes are prominent, and the conjunctivæ injected. At a still later stage there may be well-marked cyanosis of the face. The signs of venous congestion always become more obvious on exertion.

Clubbing of the fingers and toes is often well marked, especially when emaciation has occurred.

The abdomen is usually somewhat distended; the liver and spleen are enlarged from congestion, and assume a lower position than normal; catarrh of the stomach and intestines is apt to cause dyspepsia and flatulent distension. Edema of the lower extremities is often present in the latest stages of the disease, and dropsy of all the serous cavities with anasarca may occur when there is pronounced failure of the heart. All the symptoms above described become more marked during intercurrent attacks of bronchitis; some, indeed, are present only at such times.

Physical examination; inspection.—The chest tends to undergo enlargement in all its diameters, but particularly in the antero-posterior,
owing to exaggeration of the dorsal curve, and to the curvature of the sternum in the opposite direction.

The angulus Ludovici, marking the junction of the manubrium with the body of the sternum, is prominent, and the costal angle is much widened. The vertical measurement is increased by the downward displacement of the diaphragm, and the "oblique diameters" by the ribs becoming more nearly horizontal and the interspaces wider. This form may be modified by the presence of any of the deformities of the chest due to rickets or other causes, to which reference has already been made; but otherwise the general tendency of the chest is to assume a rounded form—the so-called "barrel-shaped chest" of emphysema. The rounded outline is often more marked in the upper part of the chest, whilst in the lower the increase of the transverse diameter is more obvious.

The clavicles are thrown forward, and the sterno-mastoids and other muscles of the neck are tense, giving the neck a short and thick appearance. The supraclavicular hollows may be deep; but if the apices of the lungs are markedly affected the normal depressions here may have disappeared. The curvature of the spine causes the shoulders to be round, and in extreme cases the shoulder-blades may assume almost an horizontal position.

The upper intercostal spaces may present an even surface, but the lower are often depressed. This becomes more marked on inspiration owing to the non-expansion of the emphysematous lung. Bulging of the spaces may be well marked when the patient coughs. The respiratory movements are restricted, and the expiratory act is much prolonged notwithstanding the forcible contraction of the abdominal muscles. The gradual expansion of the chest during inspiration, which is characteristic of health, tends to be replaced by a uniform upward lift, during which the accessory muscles of inspiration stand out in strong relief. In some cases, however, the infra-axillary regions are drawn "inwards and the sternum projected forwards, whilst at the same time the epigastric region, instead of bulging during inspiration, may be visibly depressed. This recession of the lower ribs during inspiration is often well marked, and may accompany the deformity of the chest called the "transversely-constricted" thorax, which is usually a relic of infantile rickets. The downward and axial displacement of the heart, combined with the hypertrophy and dilatation of the right ventricle, to which reference has already been made, are jointly the causes of the epigastric impulse commonly observed in emphysema. A horizontal sulcus is observed in some cases to extend across the body from side to side about the level of the lower part of the costal arch. A broad line of dilated venules is often seen in emphysematous subjects tending obliquely upwards on either side along the line of the lower costo-chondral junctions, and across the base of the ensiform cartilage, and therefore corresponding roughly with the attachment of the diaphragm. It is rarely complete posteriorly.

**Palpation.**—The vocal fremitus is diminished.
The impulse in the precordial area is generally feeble owing to the cushion of lung intervening between the heart and the chest wall; but the hypertrophied right ventricle, in the absence of much enlargement of the lung, may cause a heaving impulse in the lower sternal region.

Percussion.—A hyper-resonant note will be found in regions such as the precordial and hepatic, which are normally dull; or dulness may still be present, but over a much diminished area; whilst behind it is by no means uncommon to find well-marked resonance as low as the twelfth rib. Inspiration and expiration make but little change either in the area of resonance or in the pitch of the note on percussion.

Auscultation.—The character of the respiratory murmur varies with the form of the predominant lesion, whether this be of the bullous type or general in its distribution. If “bullous,” the breath-sound is weak over the sternum and along the margins of the upper lobes, but harsh beneath the outer half of the clavicle; whilst in the “general” form the breath-sound over the upper lobes is everywhere feeble. It is right to state, however, that the opposite opinion is held by some authors. In place of the normal vesicular murmur audible on inspiration the continuous low-pitched rumbling sound produced by the contraction of the muscles is often very distinct.

When the disease is fully established the expiratory sound is almost invariably prolonged, often very markedly so; in fact, during an intercurrent bronchial catarrh its duration may be so prolonged as to be nearly four times that of inspiration.

These changes in the respiratory sounds are usually most obvious over the upper part of the chest; but when the posterior aspect of the lower lobes is affected the breathing will be weak at the bases, and fine crackling râles may be present there also. These signs are important both as evidence of advanced disease and of oedema of the affected parts of the lung.

At the apex of the heart the sounds are feeble, the characters of the first sound being determined by the relative preponderance of hypertrophy or dilatation of the right ventricle. In the former case it is low-pitched and prolonged, in the latter short and sharp, but weak. The point of maximum intensity of the sounds at the base is lower than normal, and, owing to the increased tension in the pulmonary artery, the second sound is accentuated, and may be reduplicated.

A rough murmur is often audible in cases of emphysema about the sternal end of the sixth left interspace and over the seventh rib, close to the base of the ensiform cartilage. It is systolic in time, usually short, sharp, localised, and superficial, and it often more nearly resembles a rough reduplication of the first sound than a murmur. It may be due to a “white patch” on the anterior surface of the right ventricle, a condition often present in emphysema. The effect of change of position of the body on this sound is variable. It may disappear or remain un-
changed. The only importance of the sign arises from the fact that it is very likely to be mistaken for the murmur of mitral regurgitation.

**Symptoms of small-lunged emphysema.**—In this form of the disease the symptoms are much less pronounced. The most important change in the lungs—the atrophy—is but a part of a general process of wasting in which all the tissues of the body, including the blood, share alike. The respiratory needs are therefore less, and they may be adequately met by a smaller pulmonary area. The capacity for exertion is limited because of the feebleness of muscular power; and, in the absence of effort, there may be little or no dyspnea.

Another point of difference from the variety just considered is that atrophic emphysema is rarely complicated by attacks of bronchial asthma; but intercurrent bronchitis may induce dyspnea which, although differing in its mode of onset, is hardly less in degree than that which characterizes the asthmatic paroxysm.

**Physical examination; inspection.**—The emaciated and withered appearance of the subjects of this form of the disease has already been mentioned. The evidences of venous obstruction, such as cyanosis and clubbing of the fingers, are absent; as also are the effects which that condition produces in the size, shape, and position of the heart. The chest assumes the barrel shape as a result, not of a process of enlargement, but of "shrinkage" in all its diameters, and especially in the lateral. The gradual diminution in the size of the lungs is necessarily accompanied by a recession of the ribs, which assume a more oblique position. The interspaces from the first to the fourth on the front of the chest are often both wide and deep; but the increased obliquity of the lower ribs tends to approximate them, so that the interspaces may be obliterated, or adjacent ribs may even overlap each other.

Inspiration is shallow, the rigid thorax moves as a whole, the upper interspaces recede, and descent of the diaphragm is restricted.

**Percussion.**—The note is hyper-resonant, but it tends to be clearer in tone and more tympanitic in quality than in the large-lunged variety. The area of precordial dulness is not diminished and may possibly be increased. The former statement applies also to the hepatic dulness.

**Auscultation.**—The breath-sound is weak, but the expiratory sound is not prolonged to nearly the same extent as in large-lunged emphysema. Adventitious sounds are not necessarily present, but the coexistence of chronic bronchitis is so common as to make their complete absence very rare; fine and medium bubbling râles may be heard over the bases of both lungs. Fine crackling râles may be audible over the same area if œdema is present.

Other pulmonary complications will give rise to the auscultatory signs by which they are usually characterised, modified to some extent by the presence of emphysema.

**Symptoms of localised emphysema.**—On reference to what has been stated as to the mode of production and common sites of this variety of emphysema, it will be seen that the symptoms must necessarily
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depend upon the condition to which it is secondary. It may, however, be repeated that it is frequently a sequence of tuberculosis, and its presence at the apex of a lung should suggest the possibility of such a connection.

An enlargement of one lung or of a portion of it, consequent on disease and contraction of the opposite lung, is not necessarily due to emphysema; it may be a true hypertrophy. The test by which the two conditions are distinguished is that of functional activity. If this is increased, the enlargement must be regarded as hypertrophy; if diminished, as probably due to emphysema: in the former case the breathing is puérile, in the latter it is usually feeble with prolonged expiration.

Symptoms of acute vesicular emphysema.—As already stated this condition is only recognised as a form of the disease in deference to tradition.

It originates during a state of extreme dyspnoea, the urgency of which it doubtless increases; but the result to the patient is probably determined almost invariably by the nature of the exciting cause and not by the effect produced upon the lungs. Cyanosis may very likely be observed during the attack.

The chest will be in a condition of extreme inspiratory distension. The nature of the breath-sounds and adventitious sounds will vary with the exciting cause.

The diagnosis of the large-lunged form of emphysema rarely presents much difficulty. It is suggested by a history in which cough and dyspnoea are prominent features, or by the patient being engaged in some occupation known to involve severe muscular effort; it is confirmed on examination by the alteration in the form of the chest, the hyperresonance on percussion, diminished movement and feeble respiratory sounds—signs which are present on both sides of the chest.

Error has apparently arisen at times from pneumothorax being mistaken for this form of emphysema. In such cases the methodical examination of the chest has probably been neglected, and undue reliance placed upon one step in the process, possibly on percussion. In pneumothorax the enlargement of the affected side, the obliteration of the interspaces, the absence of movement contrasting with the increased movement of the healthy side—if it be healthy, the displacement of the heart to the sound side, the more amphoric note on percussion, and the absence of the breath-sounds, or their amphoric quality, are signs which combine to form a picture that, in well-marked cases, should be unmistakable.

It is possible, however, for a collection of air, confined by firm adhesions to a very small part only of the pleural cavity, to give rise to signs which may be mistaken for those of emphysema. Such a case, due to the rupture of an emphysematous bulla near the base of the lung, came under my own notice. It is sufficient to mention it as a possibility to be borne in mind without discussing in detail the diagnosis of a condition of such rare occurrence.
Aneurysm of the transverse part of the arch of the aorta compressing the trachea may be mistaken for emphysema with bronchitis. The tracheal stridor and brassy cough, the dulness, or at any rate the absence of increased resonance over the manubrium, and the loud tracheal breathing over the same area usually suffice to prevent error.

"Emphysema and bronchitis" is occasionally the diagnosis on admission to hospital of cases in which the primary disease is really stenosis of the mitral orifice; cardiac failure, pulmonary engorgement and oedema have supervened, and the murmur has disappeared. After a few days of rest and treatment considerable improvement as a rule takes place, the murmur again becomes audible, and the true nature of the case is then obvious.

True cardiac dyspnoea is distinguished from that accompanying emphysema by its "panting" character; but failure of the right heart often follows upon long-standing emphysema, and the dyspnoea is then the resultant of the two conditions and partakes of the characters of both.

An examination of the sputum for tubercle bacilli should always be made in cases of emphysema and bronchitis, particularly in such as are accompanied by marked emaciation. In the fibroid form of pulmonary tuberculosis, which is often associated with emphysema (not so-called "fibroid phthisis"), bacilli may be absent and the true nature of the disease may only be discovered on autopsy. The absence of pyrexia in such cases is not a distinguishing symptom of much value; fibroid tuberculosis being often unaccompanied by fever, at any rate for intervals of considerable duration.

The diagnosis of the atrophic form of emphysema is but rarely attended with difficulty.

Prognosis.—True emphysema, that is, dilatation with atrophy, is a permanent condition, with a decided tendency to advance. But whether it increase, and if so, at what rate, depends chiefly upon the continuance of the exciting cause, which, in the great majority of cases, is the cough of catarrh or bronchitis. If the patient is able, by change of residence or in other ways, to shield himself from adverse conditions of climate, the disease may remain stationary. Under any circumstances its course is chronic, and life only becomes endangered when complications arise.

The extent of the lesions will naturally influence the prognosis; but the effect produced upon the heart and circulation is a far more important factor in determining the probable duration of life. As dyspnöea is the chief evidence of this effect, its degree during rest and on exertion becomes one of the main elements in prognosis. The condition of the veins of the neck as to over-distension, pulsation, and filling from below, is an important guide to the state of the right side of the heart.

The existence of enlargement of the liver, oedema of the legs, ascites, and albuminuria marks an advanced stage of cardiac failure.

The presence of renal complications, particularly chronic interstitial nephritis, is of especial importance in prognosis.

Treatment.—Sufferers from emphysema rarely ask for advice on this
ground alone, the disease being one of the existence of which the laity may be said to be ignorant. As a rule, no complaint is made of the accompanying dyspnoea; the patient has become so habituated to it that he has ceased to regard it. In the majority of cases the condition is discovered when an intercurrent attack of bronchitis leads to an examination of the chest. Atrophy of the alveolar walls, destruction of the capillaries, and wasting of the elastic tissues are changes which cannot be repaired; and a return to the normal state is only possible in the cases of temporary over-distension which occur for the most part in young subjects, as a result either of laryngeal obstruction, spasm, or whooping-cough, or of bronchitis accompanying an acute disease, such as measles.

Much, however, may be done to stay the progress of the disease by shielding the patient from further attacks of bronchitis, or by advising a cessation of any occupation which necessarily involves a strain upon the respiratory organs. Treatment may also be usefully directed towards the relief of the secondary effects upon the heart and circulation.

Emphysema once established undoubtedly disposes to bronchitis; it is therefore of the first importance that all known causes of catarrhal inflammation should be carefully avoided. Those whose means permit will be well advised to spend the winter and spring in a warmer climate than is to be found in this country at such times; many sufferers, however, although they know this full well, are prone to delay their departure unduly, and an early November fog finds them still here; the result too often is a severe attack of bronchitis and much increase in the emphysema. Persons who are unable to leave home, if they hope to escape an attack of bronchitis, must exercise the greatest care in avoiding cold north and east winds, foggy and damp air, over-fatigue, or sitting in draughty rooms, and anything likely to give rise to a chill. Notwithstanding its unsightly appearance, a respirator, or woollen "comforter" covering the mouth, by warming the incoming air is of real service in warding off attacks of bronchial catarrh.

The conditions which give rise to increased pressure within the air-passages have already been described; it will be sufficient, therefore, to state that it is absolutely necessary for the sufferer from emphysema to avoid them if he wishes to escape an increase of his disease.

The effect upon the respiration is a useful test as to whether any form of exercise is harmful either in kind or degree; if it causes dyspnoea it should be avoided. The bowels should not be allowed to become confined, as, in addition to the gastro-intestinal derangements likely to ensue, much harm may be done by straining efforts in defecation.

In the article on "Aerotherapeutics" (vol. i. p. 315) a full description is given of the various forms of apparatus used in the application of condensed air to the body as a whole, and of condensed or rarefied air to the respiratory surface in emphysema. Notwithstanding that much has been done in recent years to render our knowledge of this branch of treatment more exact it is still but little used in this country. This is doubtless due to the fact that patients are rarely under treatment for
emphysema apart from its complications; and also to the small number of compressed air baths available for use.

The condition of the lungs in emphysema indicates that expiration into rarefied air should afford relief. This proceeding causes a diminution in the amount of residual air, and an increase in the volume of inspired air; thus a partial retraction of the lungs and a rise in the position of the diaphragm are brought about. These changes are accompanied by a lessened circumference of the chest, and by an increase in the vital capacity and of the force of inspiration and expiration. The apparatus of Waldenburg, of which a description will be found in the article on "Aerotherapeutics" (loc. cit.), is most suited for this form of treatment. Expiration into rarefied air produces a sense of extreme constriction within the chest and certainly diminishes the amount of residual air. The "vital capacity" of patients with emphysema under treatment by this method undoubtedly increases; but this result cannot be accepted as an absolute proof of its value, as it also follows the use of the apparatus by those whose lungs are structurally sound, practice enabling the individual to obtain a better result.

The results obtained from expiration into rarefied air are, however, much less satisfactory than those which attend the use of compressed air applied to the body as a whole.

I have given a prolonged trial at the Brompton Hospital to the use of the compressed air bath in the treatment of emphysema associated with bronchitis, and am able to support the favourable opinions expressed by Dr. C. Theodore Williams and others as to its great value.

Patients almost invariably state that they breathe more freely whilst in the bath and after a considerable number of baths (from 20 to 30 or more) have been taken; this feeling becomes continuous, and has remained whilst the patients have been under treatment. The greater capacity for exertion which follows the use of compressed air baths in emphysema has been tested by observation of the gradually increasing facility with which patients thus treated have been able to mount a flight of steps which leads from the basement, where the bath is situated, to the "gallery" (wards) occupied by them. Patients who at first were obliged to use the lift to return to their ward, or were only able to climb the stairs with many halts to take breath, have been enabled gradually to reduce the number of stoppages on the ascent; and many have at length been able to return from the basement to the uppermost floor without stopping once.

In addition to the greater freedom of respiration and increased capacity for exertion, the cough becomes less frequent and the quantity of expectoration is reduced.

It is not quite clear how these favourable results are produced. In a healthy person the effect on the respiratory organs of submitting the body as a whole to air gradually condensed to the extent of three-sevenths or one-half an atmosphere is to cause diminished frequency of respiration, enlargement of the lungs, increase of the vital capacity,
and probably also an increase in the amount of oxygen absorbed. The change is attributed to the greater density of the air, and consequently to the increased amount of oxygen supplied to the lungs. The respiratory power and the elasticity of the lungs, both during and after the bath, are increased; the chest is enlarged in all its measurements, and the diaphragm assumes a lower level. In the subjects of emphysema, however, the effect of the bath is to cause a reduction in the size of the chest, as ascertained by measurement of the circumference; and also in the amount of distension of the lungs, as proved by the reappearance of dullness in the precordial and hepatic regions. The diaphragm is raised instead of being lowered, and epigastric pulsation may be replaced by an impulse more nearly in the normal situation of the apex beat of the heart.

It appears probable that the condensed air penetrates into parts of the lungs which have been long unused in respiration, and in which air has been, so to speak, imprisoned at a high pressure; the escape of this air is facilitated and contraction of the lung follows.

In some cases the improvement following the use of the bath is but temporary, and in cases of emphysema accompanied by asthma I have observed very severe attacks of dyspnoea to follow very shortly after a bath. If this should occur after the second bath, it is generally better to discontinue its use. Many cases of asthma are, however, greatly benefited by this method of treatment.

For the details of this method the reader is referred to the article on "Artificial Aerotherapeutics," vol. i. p. 310.

The treatment of an attack of bronchitis occurring in a patient the subject of emphysema is not materially modified by the latter complication; but the duration of the attack is sensibly prolonged, and the danger to life is much greater, owing to the loss of power of expectoration which results from the diminished elasticity of the lungs.

Spasmodic dyspnoea often accompanies an attack of bronchitis, and requires the use of such remedies as stramonium, lobelia, belladonna, grindelia, or iodide of potassium in large doses, in addition to the ordinary drugs used in the treatment of bronchitis. The desirability of employing morphia in such cases will depend chiefly on the relative preponderance of the spasmodic or the catarrhal factor. The nearer the attack approaches in character to one of true asthma the greater is the probability of relief from a subcutaneous injection of morphia; whilst, on the other hand, if the dyspnoea be chiefly due to the accompanying bronchitis, the use of morphia may be attended with the greatest danger. The history of previous attacks, the mode of onset, the presence of pyrexia, the character of the adventitious sounds—for instance, the presence of fine or medium bubbling râles, indicative of an affection of the smallest bronchi or of the alveoli—and particularly the condition of the bases of the lungs, are some of the points to be considered in determining such a question. In the treatment of the attacks of wheezing, so often met with in emphysema, apart from any serious bronchial attack, a stimu-
lating liniment containing turpentine and iodine rubbed into the chest is often of much service. Iodide of potassium in doses of five, eight, or ten grains three times daily, in combination with extract of stramonium and carbonate of ammonia, generally affords relief. In the intervals of comparative freedom from such attacks, and often throughout the winter months, the administration of cod-liver oil is hardly of less service than in cases of pulmonary tuberculosis. It is of special benefit when nutrition is failing, as is commonly the case in advanced stages of the disease, and in the atrophic emphysema of the aged. Iron in combination with spirits of chloroform is often taken by patients with emphysema with much benefit.

Turpentine, terebene, and balsamic remedies are of service where expectoration is excessive; this symptom is, however, due to the accompanying bronchitis, and its treatment is described in the article on that subject.

Cyanosis is an indication for venesection, and the necessity is urgent when there is evidence of great over-distension of the right side of the heart, with tricuspid regurgitation, pulsation in the jugular veins, and oedema of the feet. Digitalis should be given as soon as the blood has been drawn; and its use may be necessary in cases which are not so advanced as to require venesection.

When, as is not uncommonly the case, emphysema supervenes on bronchitis of gouty origin, the existence of this factor in the case must not be overlooked in the treatment. The same statement applies to the coexistence of chronic interstitial nephritis. It must not be assumed at once that the presence of a small quantity of albumin in the urine is due merely to renal congestion; search should be made for casts.

It is of great importance in cases of emphysema accompanied by attacks of dyspnoea, occurring at night, that the patient should not take a heavy meal at seven or half-past and retire early to bed; by so doing he is very likely to induce an attack. Full time should be given for digestion, and the lighter the evening meal the better; such patients should dine in the middle of the day.

Few conditions apart from bronchial catarrh are so likely to induce an attack of dyspnoea as flatulent distension of the stomach. This is chiefly to be avoided by attention to diet; and these patients are nearly always well aware what food suits them and what does not. A mixture containing bicarbonate of soda, tincture of nux vomica, compound tincture of cardamoms or tincture of ginger, with a bitter infusion, taken half an hour before meals, may prevent such an attack. A dose of blue pill, taken twice a week at bedtime and followed in the morning by a saline purge, is often beneficial in middle-aged subjects of the disease who are well nourished and have a tendency to gout.
INTERLOBULAR OR INTERSTITIAL EMPHYSEMA

The escape of air into the connective tissue of the lung produces a condition to which the above name is applied.

As stated in the previous section, it has nothing in common with emphysema of the lungs but the name. The air appears as rows of beads beneath the pleura and in the substance of the lung. Wounds of the lung or rupture of the air-vesicles from over-strain during violent cough are the most common causes of the affection.

I have specially observed it in connection with laryngeal diphtheria, generally after tracheotomy had been performed; but it may occur independently of that operation. The air, as pointed out by Dr. Champneys, passes from the tracheotomy wound downwards into the thorax behind the deep cervical fascia. From the mediastinum it may spread along the connective tissue surrounding the bronchi and vessels, and may appear on the surface of the lung as small beads of air beneath the pleura.

Mediastinal and interlobular emphysema may occur in diphtheria when tracheotomy has not been performed, probably from rupture of vesicles upon the surface of the lung; and pneumothorax, from perforation of the pleura, may follow.

Pathology.—The following extracts from the post-mortem register of the Middlesex Hospital (2) illustrate the changes met with in cases of interstitial and mediastinal emphysema:

Case 1.—Male, age 3½ years. Diphtheria; tracheotomy. Extreme subcutaneous emphysema of the face, neck, and trunk; collapse of both lungs; mediastinal and subpleural emphysema.

Case 2.—Female, æt. 5. Diphtheria; tracheotomy. Lungs fully distended; no collapse; air in anterior mediastinum; membrane on fauces and in larynx, trachea, and bronchi.

Case 3.—Female, æt. 5. Diphtheria; tracheotomy not performed. Emphysema of root of neck; mediastinal, interlobar, and interlobular emphysema; pneumothorax (R); pulmonary collapse.

Case 4.—Male, æt. 5. Diphtheria; tracheotomy. General emphysema of subcutaneous cellular tissue of neck, trunk, and arms; lungs almost completely collapsed from double pneumothorax; air in mediastina and around roots of lungs; membrane on tonsils and in larynx, trachea, and large bronchi.

Case 5.—Male, age 2 years. Diphtheria; tracheotomy. Larynx completely blocked with membrane, which extended throughout the trachea and main bronchi; lungs collapsed in patches; emphysema of anterior mediastinum.

Case 6.—Male, age 11 years. Diphtheria; tracheotomy. General emphysema; membrane in trachea and bronchi of left lung, latter collapsed; marked emphysema of anterior mediastinum.

Case 7.—Female, age 4 years. Diphtheria; tracheotomy. Interlobar emphysema on right side; air in anterior mediastinum; membrane as far as secondary divisions of bronchi; numerous areas of pulmonary collapse.
The preceding cases illustrate the lesions commonly found in association with interlobular emphysema when that condition occurs in diphtheria; the most important being general emphysema, pneumothorax, and pulmonary collapse.

*Symptoms.*—In all the cases above described in which tracheotomy was performed there would necessarily be urgent dyspnœa at the time the trachea was opened. The dyspnœa would then be relieved, but the occurrence of mediastinal and interstitial emphysema is accompanied by an increase in the dyspnœa. If pneumothorax supervenes, the dyspnœa becomes extreme.

Double pneumothorax is necessarily quickly followed by death.

The breath-sounds would almost certainly be weak or absent if the connective tissue of the lung were infiltrated with air. Pneumothorax would be characterised by its ordinary physical signs.

Interlobular emphysema is rarely recognised during life. It may be suspected when subcutaneous emphysema is present, or when pneumothorax occurs. The latter is a serious complication. It is probable that the condition here described is often present but is unsuspected, and that the air is absorbed when recovery takes place.

No definite treatment can be adopted for the condition.

J. K. Fowler.

REFERENCES


J. K. F.

ON ASTHMA AND HAY FEVER

Asthma is a paroxysmal dyspnœa which often manifests itself quite suddenly and from a great variety of causes; and which may subside again with like rapidity. The respiration in the intervals may or may not be normal.

It is usually divided into primary or idiopathic and secondary or spasmodic asthma. The latter kind appears to originate from more or less bronchial catarrh. In the management and treatment of the affection it will be necessary to take this distinction fully into consideration; but seeing that spasmodic asthma can, and certainly does, occur independently of local and chronic irritation, I shall first consider it in its simplest form as the primary disease. I shall first describe the features of an attack of asthma, and then discuss in natural sequence its causation, its pathology, and the general management of the asthmatic patient.
Hay fever is often a spasmodic asthma in its purest form, so that the two maladies will be considered in common.

Symptoms.—The asthmatic paroxysm may come on at any time. A susceptible or morbidly paroxysmal subject—to be paroxysmal more or less is a universal attribute of organic action—comes into contact with, say, some animal or vegetable exhalation; eats some indigestible article of diet, or something that, while innocent to the mass of mankind, is known to be in some way prejudicial to particular individuals; or in some other of many ways taxes his range of accommodation beyond the margin of its power, and within a few minutes an attack will begin. The beginning is said to be most frequent during the night, when the patient has had his first sleep; for instance, at two or three in the morning he suddenly awakes with a stuffy feeling in his chest, and within a short time he is in the throes of an attack of asthma. Thereupon he is compelled to sit up in bed, perhaps to rush to the window; the head is fixed, the shoulders raised, the hands are often planted well down upon anything firm upon each side to give purchase to the respiratory muscles, and so the sufferer sits labouring at his breath. Sometimes he bends forward, sometimes stands leaning upon some support; but the object in all cases is the same, to give the respiratory muscles better or more fixed support from which to act.

The pulse is usually but little affected, and the temperature is normal.

In bad cases the face is of an ashy pallor, or it is gray and leaden, or dusky from want of oxygenation of the blood; the skin is covered with perspiration, the eyes may look prominent, the nostrils may be dilated. Few diseases produce appearances so distressing and so grave, and yet it can fortunately be said that an attack of spasmodic asthma never kills. Probably it supplies its own corrective in this, that after a certain time of agony, or certainly of intense distress and anxiety to the patient, the irritated centres become exhausted, the spasm is gradually relaxed, and the patient sinks to sleep. In some cases the relief appears to be absolute; in the majority, however, it is only comparative, and more or less oppression is experienced for a day or two, sometimes for many days; or the malady may abide with the patient more or less continuously.

During an attack, although the patient is making violent efforts with all the respiratory muscles, the actual movements of the chest wall are little indeed. The chest may plunge, but there is no expansion of the thoracic cavity. On the contrary, as the chest walls are pulled outwards all the more yielding parts are depressed and thus the intercostal spaces become troughs. The epigastrium may be hollow or full; the supra-sternal and supraclavicular spaces are greatly exaggerated.

The actual condition of the chest during a paroxysm has been the subject of some discussion. The generally accepted description, following Salter, is that it is in a state of over-distension, the diaphragm being depressed and the upper part of the abdomen being full (Hyde Salter and Biermer). The movement is much restricted, and thus there is a very
short, abortive, suddenly pulled-up inspiration, and an expiration perhaps four or five times as long as it should be. The percussion of such a chest gives a hyper-resonant note. But I am sure that I have seen, as stated by Riegel and others, another form of chest, where the lower parts, if not retracted as some contend, were not unduly distended, and where dulness rather than hyper-resonance was detected. And I have always supposed that in these cases the obstruction in the smaller bronchia is so extreme as to lead to a state of collapse. Wilson Fox offered this explanation, and the whole subject will be found discussed in his posthumously published work, *A Treatise on Diseases of the Lungs and Pleura*.

The attack is, however, by no means always sudden in its onset, perhaps not generally so. More or less wheeziness and constriction of the chest may exist for a day or two beforehand; there may be a short, rather hollow cough, and, if the dyspnoea be at all pronounced, much weariness on exertion. Thus a mild or threatening attack may be recognised by the onlooker in the disinclination to all movement generally shown by persons thus affected.

If the chest of the asthmatic be auscultated during the paroxysm the chief feature is an almost complete absence of respiratory murmur. The chest, as I have said, plunges, but there is no corresponding inspiratory wave; there are sibilant rhonchi and muscular rumbles, and a variety of odd noises, but no real ingress of air; and with the expiration there may be, perhaps, a long, distant, soft sibilus, the sole evidence of the respiratory ebb. The disease is often unequally distributed: one side, or this or that portion of one lung is affected more than the other; the asthmatic storm flits about the lung, now here, now there, and when the disease is thus unilateral or partial it is liable to repeat itself thus! so that we surmise that there is some local disease in the form of bronchitis, emphysema, adherent pleura, and so on, which exercises a permanently determining effect.

It is said that when an attack is over the patient is free from liability to a recurrence for some time. But in all probability this depends upon the past duration of the asthmatic habit. Asthma, like gout, although in its earlier years markedly periodic in recurrence, tends, as the patient grows older, to become erratic in its manifestations, both as regards the time of its appearance and the form in which it comes. So that whereas in its earliest appearances it comes and goes, maybe with some special regularity, later in life it comes but it does not go, and the patient thenceforward is subject to a more or less chronic bronchitis. And in all old cases, in which the chest is damaged by the repetition of the paroxysms, emphysema is produced with its attendant chronic bronchitis.

As the attack ends, expectoration usually begins. In the earlier stages of the paroxysm the bronchial tubes—to judge from the character of the signs—are dry; the prevailing sounds in the chest being wheezing sibilus and rhonchus. Under the intense oppression the patient longs to expectorate, but is not able to do so. But when subsidence approaches, small gray pellets of mucus, of characteristic appearance, “like tapioca” (Salter),
"often filamentous in shape like boiled macaroni" (8), begin to appear and gradually increase. In association with the appearance of crepitation in the chest the sputum becomes more and more copious, thin and frothy, till it may reach some considerable quantity. Blood seldom comes, but in severe attacks it may, and, if so, generally in streaks.

The clinical history of spasmodic asthma is, however, by no means completed by this description of a characteristic attack. There are several other irregular states that to my mind are no less parts of the disease. First of all there is hay fever, when brow ague, coryza, a more or less general disagreeable stuffiness of the respiratory tract, rendering nasal breathing a difficulty, and producing a more or less chronic wheeziness and distress, last throughout the summer months. At times there is some slight febrile reaction, but it is not often great in degree; there is rather the subjective feeling of lassitude and heat than the objective evidence of actual pyrexia; and perhaps, on the whole, these rather indefinite symptoms are the more usual mode of its attack, although a definite attack of spasmodic asthma is by no means uncommon.

Paroxysmal sneezing is another way in which the asthmatic respiratory tract explodes. If we study asthma or the history of the asthmatic in any comprehensive way, we cannot but be sure that, either as a substitute or as part and parcel of the asthmatic attack, this sneezing must be taken into account. It is often found in asthmatic subjects and in asthmatic families, in which one member may have asthma and another exhausting paroxysms of sneezing; moreover, it often goes with asthma, the sneezing gives the impetus of origin to the asthma, the irritation in the upper air-passages gradually spreads down the bronchial tubes, and asthma more or less severe results. In the history of asthma cases of this sort are to be found in numbers; but they need not be more particularly described, for they are fully dealt with under an appropriate heading. I may say this, however, that most of the cases of sneezing I have met with have been in women, which is against the rule that prevails in asthma, in which case men are in the proportion of two to one. There are authors who attribute such cases largely to local disease in the nose, and believe that they are to be relieved, as also the asthma that accompanies them, by local treatment of the nasal mucous membrane. One case may be quoted that illustrates several of these points: a man, aged twenty-eight years, who came of healthy, non-asthmatic stock, fell off a bicycle and smashed his nose. Ever since that time, now twelve years ago, he has required for his daily use six or seven pocket-handkerchiefs, and now he has become asthmatic. His asthmatic attacks come on every month or six weeks, and last from half an hour to a day and a half. He has had his nose treated with decided benefit to his asthma, but he derives most benefit from smoking medicated cigarettes. With reference to the nose the experiments of Lazarus (7) are of much interest. This observer has demonstrated a certain relationship between the nasal mucous membrane and the bronchial muscles, so that, by the application of weak electrical

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currents to the nasal mucous membrane, he was able to register a distinct increase in the intra-bronchial pressure.

There is at least one other affection that I would include in the clinical history of asthma, namely, the paroxysmal bronchitis of infants and young children. It has always seemed to me that one of the most interesting features in the study of medicine is the modification that disease undergoes in the successive periods of life. It is not certain, perhaps, that disease is so modified, but there is plenty of evidence to point in that direction. For instance, when a man who in earlier years had acute rheumatism is attacked in middle age with well-marked gout, we may suppose that a common factor has been modified, so that what did produce acute rheumatism at a later date produces gout. Now, as regards asthma, I believe that something of this kind takes place. Asthma, as I shall presently show, is largely a disease of childhood, but it is not clearly present in the earliest years. Hyde Salter has seen two cases in infants of fourteen and twenty-eight days, but such instances are very rare. It frequently begins to appear at six, seven, or eight years of age, and there are a fair number of suspicious cases at earlier periods than this. In infancy, if asthma exists, as I believe it does, it shows itself as a bronchitis, so far as the physical signs go; but, if so, it is a strange and interesting bronchitis, apart from the physical signs. It comes on with remarkable suddenness; it is mostly associated with fever; it is generally attributed to chill by the relatives; but there are reasons for thinking that it owns a much greater variety of causes, such as over-excitement, errors in diet, dentition, and so on. It clears up with remarkable celerity and certainty; it often leaves the child no worse than it was before the attack. Such attacks as these occur in a particular class of children,—children that give conspicuous evidence either of coming of nervous stock, or of nervousness and excitability in themselves. The whole history of these cases is explosive and nervous; and it may well be that, in the early history of the child's life, the ribs and other parts of the respiratory apparatus are not sufficiently developed to produce asthma, as we expect to see it; so that the mode of the disease is atelectatic or bronchitic. Asthma, in its ordinary manifestations, requires certain conditions of respiratory power, which, in all probability, the thoracic walls do not readily supply at that early period. Moreover, there can be no doubt that in the seven stages of our existence—and this answers to some extent the question I have already mooted—our various viscera change places in their relative importance, not only in their several bearings upon the well-being of the organism, but also in the absolute degrees of their activities; now one, now another, becoming a centre of excitement and explosion, and thus of break-down in ill-balanced organisations. In infant life the stomach tends to play the part of the spoilt darling, and the lungs often have to pay the penalty for its caprices. However well it may be, it would appear that often, as with many another ill-bred person suddenly thrown into a position of trust and responsibility, it is not equal to the occasion; the household's teeth are
set on edge, and pulmonary catarrh or oedema or collapse is set up. It seems to me that these sudden storms, which so expend themselves on the lungs, or in the achievement of pyrexia, have much similarity to asthma in their sudden mode of outburst; they involve a similar area, and may therefore not inappropriately be considered in the youngest children as the representative of asthma. And having diverged from the immediate subject to introduce that of the correlatives, substitutes, or derivatives of asthma, I shall briefly indicate several other diseases that may in this respect be considered with the gastro-pulmonary fever that I have just mentioned. Some persons, for instance, have laid stress upon psoriasis in this connection. I have myself known of a case where asthma and psoriasis seemed to alternate in the same person, and I have also come across this curiously suggestive alternation as regards eczema. The mother of a family is the subject of spasmodic asthma. She has had four children. In the first and third pregnancies she had no asthma, and in each child bad eczema appeared, and death resulted in one from convulsions. In the second and fourth pregnancies the mother had bad asthma, and the children hitherto have been healthy. I find from a collation of my notes that no less than seven out of 125 cases of asthma were associated with severe eczema, and in two or three of these as the eczema went the asthma came. Carl v. Noorden is perhaps the most recent author who has drawn attention to the frequency with which asthma is associated with eczema, but the connection has long been noted.

Again, I have elsewhere thrown out the suggestion that some of the cases of paroxysmal sneezing, which, as I have said, are undoubtedly part of the complete picture of asthma, may also be a part of the history of Raynaud’s disease; for they go with weak peripheral circulation, with waxy fingers, with chillblains, and so on. All three are probably due to allied causes; and although in all the three the results are of different order (in the case of the nose, turgidity; in that of the extremities, cadaveric blanching or chillblains; in the case of the lung, a supposed spasm of the muscles of the smaller bronchi, leading to a temporary collapse of the affected part of the lung), yet the clinical history in all of these is not unlike. In all there is the same tendency to suddenness of onset, the same sort of rhythmical association between flux and its opposite, the same curious vagaries of onset from causes that seem quite inadequate.

Of other affections that surely belong to the same category is that form of looseness of bowels which is so common in nervous subjects, and in excitable children, where the mere ingestion of food seems sufficient to provoke a stomach-ache and a profuse liquid evacuation from the bowels. This is perhaps the very commonest of the kind. Another is urticaria, and it is not uninteresting to note that it is sometimes associated with or replaces asthma, as a case of asthma produced by contact with cats will show. I have records of three such cases.

To complete the clinical picture, it must be said that although a certain number of cases are inexplicable explosions, and all of them own
something of that character, yet many, perhaps most, have a local exciting cause—a cause inefficient, it may be, to produce any such disagreeable effects under healthy conditions of the nervous centres, but which under diseased or ill-regulated conditions becomes an active source of worry and excitement. Such things are pneumonia, bronchial catarrh, whooping-cough, and so on. Eighteen out of 125 are attributed to such a cause.

Causation.—As to sex, it is usually stated that asthma occurs twice as often in men as in women: 73 to 50 in my own cases. It might have been anticipated that the less stable centres of the woman would be the more likely to show a predominance, but it may well be that the instability of womanhood works off in other ways. Salter considers that this unexpected incidence of a nervous affection upon males may be an argument in favour of the existence of some organic change in the lung.

Of age, Hyde Salter remarks that it is a commonly received opinion that asthma is a disease of advanced life, but that it is not confined to any one time of life; so far, indeed, is it from being peculiarly a disease of the old, that a larger number of cases take origin in the first ten years of life than in any subsequent decade. This seems to me quite a correct statement of the matter, for I find that, of cases in which the point is noted, 50 began in children of ten years and under, 31 in males, 19 in females: it is interesting to note that the youngest case was in a little boy 3½ years old, whose father suffered from hay fever and asthma, and who was said to have been quite cured by local treatment of his nose with the cautery.

In 23 cases the disease began between ten and twenty, 13 being males and 10 females. In the period from twenty to thirty only 12 cases are reported, 4 males and 8 females. In 36 cases, 24 men and 12 women, the disease arose after the age of thirty. These figures indicate, too, that the excessive incidence of the disease upon males is all along the line, with the exception of the decade from twenty to thirty, in which perhaps the numbers are not sufficient to base any conclusions upon. Thus, in seventy-three cases out of 121 asthma began in subjects under full age.

Hyde Salter’s table of the age at which people have become asthmatic is as under:

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<thead>
<tr>
<th>Age Range</th>
<th>Number of Cases</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>1-10</td>
<td>60</td>
<td>31%</td>
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<tr>
<td>11-20</td>
<td>30</td>
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<td>21-30</td>
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<td>17%</td>
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<td>31-40</td>
<td>44</td>
<td>19%</td>
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<td>41-50</td>
<td>24</td>
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<td>51-60</td>
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<td>61-70</td>
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<td>71-80</td>
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<td><strong>Total</strong></td>
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Heredity.—Of 123 cases, 50 showed a well-marked neurotic inheritance of one form or another; in 25 it was the direct transmission of
Asthma or of hay fever. In 8 more one or other of the parents had had rheumatic fever; in other families there is a history of megrim; in others somnambulism and diabetes existed. And, indeed, in all this group of diseases—in asthma, hay fever, and paroxysmal sneezing—the number of the nervous phenomena that are to be found in the different members of the family is conspicuous. Borkart, in some carefully selected cases, found that in 16 per cent one or other parent suffered from asthma.

Of other remoter causes one must certainly mention an idiosyncrasy on the part of the subject—“individual constitution,” as Wilson Fox calls it. What this is we know no more than why certain foods, which for the majority of mankind are perfectly harmless, for a small minority are active poisons. For my own part I am inclined to doubt whether this constitution is ever wanting in the case of asthma, even though diseased conditions be actually present that seem so immediately provocative of an attack as naturally to be regarded as sufficient causes.

Immediate causes.—Of the two groups of cases into which we divided asthma at the outset, perhaps almost enough has been said incidentally about idiopathic asthma. Given a certain morbid sensitiveness of the nervous centres, anything seems capable of producing an attack. It may be a nervous shock, over-fatigue of mind or body, too monotonous a habit of living, too little exercise, too much food, indiscretions of one kind or another in diet, changes of temperature, changes of climate, a thunder-storm—changes in the weather seem to be particularly prone to induce asthma, microbial organisms in the atmosphere, or emanations of various kinds from animate (cat or horse asthma), inanimate bodies (hay), and so on.

Of some of these emanations one would not wonder that dust, fog, or pungent fumes of various kinds should now and again be responsible for the production of the disease. But the peculiarity of asthma would seem to be that it is evoked by irritants that under ordinary circumstances are no irritants at all (Salter). “One asthmatic is obliged to expatriate himself in the hay season and take a sea-voyage; another cannot stay in a room in which a bottle of ipaecuan is opened; a third cannot stroke or nurse a cat; another cannot go near a rabbit hutch; another is immediately rendered asthmatic by the neighbourhood of a privet hedge; another cannot sleep upon a pillow stuffed with feathers; another cannot use mustard in any shape, or bear it near her, so that she dare not even apply a mustard plaster; and one young lady I know who dare not pass a poulterer’s shop.”

I have myself knowledge of two cases of cat asthma. In one of them the existence of cats is the bane of life, for before accepting an invitation she is obliged, first to ask, “Is there a cat?” An attack of urticaria and coryza followed by asthma has been noticed to come on within ten minutes of having stroked a cat. At other times sitting in a room in which there was a cat, without any actual contact with it,
was sufficient to produce a bad attack, beginning within ten minutes of entering the room.

Professor Clifford Allbutt tells me of a little boy in whom horses work similar effects. He cannot, therefore, ride in carriages or cabs; and it has been necessary to let him run home and get wet through, rather than incur the greater evil of asthma, likely to be provoked by a ride in a cab with his mother.

Such statements as these, Salter truly says, one would hardly believe, were not their reality placed beyond doubt; there is neither invention, nor imagination, nor exaggeration about them.

Surgeon-Major Lethbridge Swayne, practising in Aurungabad, tells me that asthma is quite common there in association with malaria; and that asthma often ushers in an attack of malarial fever, and has done so in his own case several times.

Potain alludes also to the frequency with which amongst the infectious paludism plays the part of an exciting cause. The same thing has been noticed with regard to that malarial disease, influenza. Cases have, I believe, been recorded where influenza ushered itself in by provoking a severe attack of asthma; I have seen such a case myself, and shall record it later in this article. But it is seldom, perhaps, that this is the case; far oftener asthma comes on as a result of the post-influenzal exhaustion of the nervous centres. I have notes of six cases of the kind.

Of nervous shock, or strong emotion, I will only add that, as such impressions are well known to bring on attacks, so they may also remove attacks instantaneously and completely.

In all these cases examine the patient, in an interval of freedom, and there may be no evidence whatever of any disease. But of a large class of asthmatics this cannot be said. In many an asthmatic, for instance—80 per cent of all cases, according to Dr. Theodore Williams—there is evidence of permanent catarrh of the bronchial tubes (bronchitis). A little fresh accession of cold, and on comes asthma. Hyde Salter says much the same, namely, that 80 per cent of the cases of asthma in the young date from whooping-cough, bronchitis, or measles. There are other asthmatics who are gouty, and the gouty condition of blood seems to provoke a catarrh, in this respect occupying an analogous position to ague. The alterations of the ribs in old age lead to pulmonary obstruction and emphysema, and so favour an asthmatic paroxysm. The pulmonary congestions of chronic heart disease and renal disease bring about the same end. Hyde Salter describes a peptic asthma due to indiscretions in diet; but this seems to me to belong more properly to the idiopathic group, the stomach being a common point in the morbid circle from which the storm is set a-going. In many even of these secondary cases, however, it is still supposed, as I said at the outset, that some constitutional element or weakness allows the local disease to start the train of peculiar symptoms.

There is, however, one group of cases to which I am not sure that
this applies. 'Every now and then an asthmatic appears to have suffered, for the first time, in middle life, in whom there are no obvious tendencies to neurotic ailments, and no evidence of existing disease that might act as proximate cause. It is possible that a percentage of these may belong to the group already mentioned, where gout in the system or excessive vascular tension has been the cause; but I am not satisfied that these things explain all the cases of later appearance. To judge from my own experience, they are prone to be very severe, and to be but little amenable to treatment; and I have come to the conclusion that in certain cases there may be some rapid onset of emphysema, some process of degeneration in the tissue of the lung, such as was described some years ago by the late Dr. Greenhow.

Simple spasmodic asthma is very seldom seen in the wards of a general hospital. It is of course found often enough in the degenerate, in association with emphysema, chronic bronchitis, gout, and renal disease. But in the primary pure form it occurs seldom indeed. There are many reasons for this. Chief of them, perhaps, is that this disease comes and goes; and for maladies of that kind the working-man cannot afford to lie up. Indeed this applies to all classes of society. As Berkart truly says, "Asthmatics are not disposed to consider themselves as patients. Their suffering is forgotten as soon as it is over." But I cannot help thinking that the affection is one that belongs more peculiarly to the upper ranks of society. It may be, perhaps, that the angular condition of the nervous centres, to which the disease may be attributed, becomes rubbed down, so to speak, by the harder life of the labouring classes; just as such persons are less sensitive to noise, less sensitive altogether to what one may call the smaller ills of life.

Results of asthma.—When a man has been the subject of asthma for a long time, it is likely that he will present characteristic appearances in his general physique and gait. He is usually very thin; his back is rounded, his shoulders are high, and he walks lethargically, with a well-marked forward stoop. He sits, may it be said, turtle-like, with his neck dropped into his chest. In long-standing cases the face is a little dusky, the eye watery and perhaps congested; and there is often a cough of peculiar timbre, moist and hollow, not easy to describe, but evidently the product of feeble expiratory power. The asthmatic speaks, too, as he coughs; and for the same reason, that the tidal wave of the chronic asthmatic is exceedingly shallow: for the spine is rounded, the ribs stiffened and fixed, the chest elongated and depressed.

The morbid anatomy of asthma, saving perhaps one particular detail, is comparatively small in amount and simple in kind. It is obvious that all diseases, as they fall under the denomination of "functional," must proportionately be wanting more or less in those coarser changes in structure that we look for in the study of morbid anatomy; and so it is here. The leading departures are most of them certainly conditioned by, and secondary to asthma of long standing; they are the
results of the impaired respiration, not the cause of the asthmatic paroxysm. These are more or less chronic inflammation of the bronchial tubes, shown by injection, and thickening of the mucous membrane, thickening of the muscular coat of the bronchial tubes, dilatation of the tubes, emphysema of the vesicular structure, more or less thickening and atheroma of the branches of the pulmonary artery in the lung, and hypertrophy and dilatation of the right side of the heart. The changes in the skeleton that go with these have already been mentioned; these are the curved dorsal spine, the barrel-shaped chest, the stiffened ribs, the generally wasted frame.

But we have still to consider in more detail the state of the bronchial tubes, and the products that are shed from their mucous surface.

The most regular condition to be found in the asthmatic is more or less mucus or muco-pus in the smaller tubes. This may be considered perhaps to be a feature of the asthma itself, inasmuch as it is admitted that in 80 per cent of the cases there is some organic change in the lung.

Practically, all the controversies that have been waged over this subject have centred in this: which is cause, and which is effect? Medicine, so eager to find a cause for everything, is unwilling to accept anything as such that does not possess a definite basis of structural change visible to the naked eye or to the microscope; and is willing to attribute the phenomena to any change that is demonstrable, rather than incur the suspicion of going beyond the facts, of hasty generalisation, or of appropriating prematurely the possessions of the future.

From a very early time Lefèvre, himself an asthmatic as recorded by Börkart, had described the expectoration of a peculiar kind of sputum; but we may take up the matter at the later date when Curschmann re-observed and redescribed peculiar elongated plugs or spiral bodies in the expectoration of the asthmatic, to which he was inclined to attribute considerable importance. To the formation of these bodies—"Curschmann's spirals," as they have been called—spasmodic asthma has been attributed. Their nature is yet in doubt, some considering them to be inspissated epithelium, some fibrinous concretions from the smaller bronchia; I should myself suppose, although this view is combated by Curschmann, that in their nature they are allied to the plugs that form in the so-called plastic bronchitis; and that like these—although their formation is an acute process—they leave behind them some habitual morbid condition of the bronchial tubes, and possibly also some proclivity to the formation of such bodies in the individual attacked. There is no doubt, moreover, that although more common in asthma, these bodies are found in pneumonia and oedema of the lungs also; and in certain secretions from the conjunctiva (Gerlach). Altogether it seems impossible to consider their presence as satisfactorily explaining the onset of a condition so peculiar as that of spasmodic asthma.

As little can be said of the Charcot-Leyden crystals present in the
sputum and interior of the spiral plugs, and likewise of the eosinophile cells (Adolph Schmidt) in the blood and sputum of the asthmatic. I should agree with Müller and with Schmidt that the discovery of either of these bodies in the sputum does not definitely indicate the nature of the disease from which the patient is suffering: if this be so, the contention that they have any causal importance has little to support it.

Such, then, is the history of an attack of asthma; such, and it is but little, is the morbid anatomy that is associated with it. But these, and certain physiological experiments, are almost the only data upon which to frame a pathology of the disease.

Pathogenesis.—A disease that is, or may be, so sudden as to be well-nigh instantaneous in its onset—one produced under the influence of strong emotion, one which, under such and other circumstances, may subside as quickly as it came—can hardly be other than some functional aberration of normal structures. The changes that seem most competent to explain the phenomena of asthma are: (i.) A muscular spasm of the smaller bronchia; (ii.) Some rapid tumefaction of the mucous membrane of the bronchia; (iii.) A rapid production of collapse of parts of the lungs. All these, as has already been said, are hypothetical causes only, although each one of them can be defended by more or less cogent arguments. The one most generally accepted of recent years, it need hardly be said, is that the production of asthma is due to spasm of the muscular coat of the smaller bronchia. The late Dr. Hyde Salter, who was the chief and most able expositor of this view, makes use of the following arguments in its favour:

"In the first place, the sudden induction and remission of the asthmatic paroxysm is consistent with this supposition; in the second place, there is abundant proof that the air in the lungs is locked up, and can neither be got in nor out; there is evidently plenty of air in the chest, percussion is even over-resonant; yet the patient is as unable to drive air out as to draw it in; he can neither inspire nor expire; he cannot discharge breath enough to whistle, to blow out a candle, or to blow his nose. The muscles of respiration tug and labour to fill and empty the chest, but the chest walls remain almost immovable; the inspiratory muscles cannot raise them, the expiratory cannot depress them. On listening to the chest we find corroborative evidence of the stagnation of the air. The respiratory murmur is in a great degree lost. This absence of respiratory sound, accompanied by violent respiratory effort, is one of the most striking and suggestive of the facts of asthma. How can we explain it except by supposing that there is some bar to the ingress and egress of air; and what can this bar be, unless it be spasm of the bronchial tubes? It cannot be inflammatory thickening of the mucous membrane lining them; for the sudden, almost instantaneous establishment and remission of the dyspnoea is incompatible with this. It cannot be mucus plugging the tubes, for the attack will often come and go without any expectoration whatever. But we have still more positive and precise evidence of the circumscribed narrowing of the air-tubes in the
musical sounds that are present in asthmatic breathing. This symptom has all the certainty and precision that characterise physical phenomena; and it shows that the air-tubes are the seat of constrictions which throw the air passing through them into vibrations, and convert them into musical instruments: since these musical sounds are multitudinous, the points of constriction must be many; since they are constantly varying in locality and character, the constrictions of the tubes must be undergoing similar change. Lastly, the effects of remedies and their nature tell the same tale, and point to muscular spasm as the immediate essential condition. The most powerful remedies for asthma are what are called cerebro-spinal depressants, such as "emetics, tobacco, etc., remedies the direct effect of which is to relax muscular spasm" (13).

This view, originally affirmed by Reisseissen, who based it upon his discovery of the presence of circular muscular fibres in the bronchial tubes, has since been proved on experimental observation by numerous observers from C. J. B. Williams onwards, including Paul Bert, Riegel, Biermer, Lazarus, and others.

This spasm is held sufficient to explain a state of things round which a good deal of controversy has centred, namely, the over-distension of the chest that occurs in the asthmatic, associated with a dyspnea that is mainly an expiratory one. The obstruction in the tubes being incomplete, it is said that the air, under the labour of forced inspiration, enters the lungs, but that expiratory paralysis or obstruction prevents its getting out again. It is objected, however, by Wintrich and others, that as the expiratory force is greater than the inspiratory this solution is not satisfactory. The lungs under such circumstances should tend to collapse. Wintrich, accordingly, believes the attack to depend upon spasm of the diaphragm. But this explanation does not seem free from difficulty; for, as Wilson Fox says, the phenomena of "this condition are widely different from those observed in spasmodic asthma."

No doubt if one is to accept as absolute that doctrine which teaches that atelectasis is a necessary consequence of the collection of mucus in tubes that narrow progressively from the trachea to the periphery, because plugging of such tubes creates an inspiratory difficulty more than an expiratory, the air being able to get out of such tubes under the pressure of the expiratory force, but hardly to get in under the ordinary inspiratory act,—there is a difficulty. The distended chest of asthma contravenes the usual rule.

But the asthmatic state is a complex one, and it is to be explained by no simple and universal law. In the first place, granting the existence in bronchitic states of conical tubes and adapted conical plugs,—which, after all, is an imaginary description,—a spasmodic contraction of the bronchial muscles is not the same thing as a plug of mucus in the tube. In the one case the obstruction might, and probably would, to some extent be on the side of expiration; it could have no such effect in the other, unless indeed the spasm were regular and rhythmical from the periphery upwards towards the main tubes. A spasm of the tubes
must tend to prevent air getting both in and out; and the more in or out according as the inspiratory or expiratory force is the greater. But the expiratory force is said to be the greater, and so it may be for ordinary respiration, but no one who has seen the forced action of the ordinary and extraordinary muscles of respiration in the exceeding labour of inspiratory effort during an attack of asthma, can have any doubt that the natural order is completely destroyed, or have any difficulty in believing that the air is really sucked past the obstruction, so that the lung becomes over-distended. Nor is it unimportant in this regard to insist again that the obstruction flits about from one part of the lung to another; from one side to the other; a temporary relaxation of spasm which means a liability to compensatory over-distension of the unlocked part.

Some authors, unable to get over the difficulties which this assumption of spasm of the bronchia creates, have suggested a spasm of the respiratory muscles; others, again, a paralysis of the muscles of the bronchial tubes. As regards the latter, admitting that under ordinary circumstances the muscular coat of the bronchial tubes may be reckoned as one of the forces of expiration, it is hard to think that its share can be so great that its failure should constitute a departure from normal so grave as asthma. More might possibly be said in favour of a spasm of the muscles of respiration, for, if we run over the field of clinical medicine, we are not unfamiliar with several curious vagaries of breathing which are attributable to such a cause: Cheyne-Stokes respiration is one of these; the air-hunger of heart disease another; the asthma of uremia another. All these, in common with spasmodic asthma, are immediately conditioned by some convulsive or misapplied action of the respiratory centre, and perhaps give some colour to the suggestion that one of the factors in the production of spasmodic asthma may lie in aberrant action of the muscles of the thoracic walls.

It must be admitted, however, in the present state of our knowledge, or of our ignorance, that, although other explanations may seem to some to be as good, the theory of muscular spasm is at any rate fairly complete. It is a reasonable and satisfactory explanation of the facts, and it does not appear that there is much that is convincing to be said against it. Nevertheless, the hypothesis placed second in order is, I think, almost equally good, that which assumes some rapid turgidity or erectility of the bronchial mucous membrane. The capacity of such active congestion, even in parts that are not naturally erectile, is well seen in certain morbid phenomena—in some cases of Graves' disease, for example, where from some sudden shock the eyes have as suddenly become prominent; that this is a purely vascular turgescence is shown by its complete subsidence after death, and by the fact that the orbital or ocular structures show no morbid change: now if we look to the respiratory tract itself we all know only too well how near a common cold may come to an attack of asthma. There is the initial irritation of the nostrils, then the sneezing, then sore or dry throat, then some little
tracheal worry, and finally a definite, albeit slight, bronchial stuffiness and wheezing. Now in these cases the initial change is certainly turgescence of the upper air-passages, and so also is it in the cases of paroxysmal sneezing, and in certain cases of local disease of the nasal mucous membrane, of deflected septum, or of polypus.

It seems, therefore, a rational belief that what can be proved to exist in the upper air-passages, namely, a definite erection or turgidity of the nasal mucous membrane, may also take place lower down in the bronchial mucous surface; and, so doing, accomplish much of what we call spasmodic asthma. It may be thought, however, that swelling such as this is hardly adequate to explain the sudden origin and subsidence of the paroxysm; yet it is quite competent to do so, for paroxysmal sneezing comes on quite suddenly, and subsides as readily if, from any cause whatever, the attention be averted from the subjective discomfort. This hypothesis also is not without a considerable body of influential support, dating too from early times. Wilson Fox states that Bree, as early as 1807, held some such opinion, considering that asthma was a convulsive attempt to expel peccant material from the bronchial tubes. Traube considers asthma as a very acute catarrh. Blackley contends that the asthma of hay fever is the turgescence in the nose extending to the general bronchial mucous membrane. And Sir Andrew Clark considered the phenomena of an attack to be explained by a vaso-motor neurosis, by which changes analogous to those of urticaria upon the skin are produced.

To this it may be added, that Störcck actually observed with the laryngeal mirror that in certain instances of asthma the whole length of the trachea and part of the right bronchus were deeply congested.

Berkart, however, will have nothing to do with a neurosis of any kind. Although he admits the existence of a peculiar predisposition, he will not allow that the history of the asthmatic attack, as regards its sudden onset and sudden subsidence, is anything but vague and untrustworthy report. He sums up his opinion thus: “The conclusion, therefore, seems irresistible, that what is commonly described as bronchial asthma is an acute and progressive, nay almost erysipelasous, form of inflammation, which extends from the pharynx upwards and downwards, and is accompanied by a copious exudation.” But it is impossible thus to discard so large a body of evidence, vague though some of it admittedly be, as case after case of asthma supplies. And, if not, then the surmise of an initial inflammatory process is much less securely seated. Indeed, “an acute and progressive, almost erysipelasous form of inflammation” may well be thought to lend its advocate in even greater difficulties, seeing that the disease is seldom associated with fever, seldom with any pneumonia, and, as an attack, is never a cause of death.

I believe, on the contrary, that the explosive character of asthma is absolutely certain. Let two cases suffice:—A gentleman was seized with influenza, and it was ushered in by an attack of asthma. This condition was supposed to have gone on to broncho-pneumonia, and this proved intractable. At great inconvenience to himself, therefore, he obeyed the
order to take a long holiday; and it was my good fortune to see him as he passed through London, within two or three hours of his leaving home. I was to find a particular focus of disease at an indicated spot. The man told me, as so many asthmatics do tell us, that he felt his breath relieved after he had been twenty minutes in the train; and when I saw him an hour or two later, no one could say that he was other than absolutely healthy. The second case I owe to the editor of this work.

Dr. Allbutt was examining by auscultation the backs of the lungs of a gentleman of nervous habit, who was overworked and suffering from pains which were suspected to be of the nature of angina pectoris. The patient was sitting up in bed, and his face was under the observation of his own medical attendant. After hearing a few inspirations of a normal character, to Dr. Allbutt's surprise the inspiratory murmur began to diminish on the left side, and in a few moments ceased. Perplexed by this strange event, percussion was quickly applied to the left side of the chest, but with negative results. During this time the family attendant, Mr. Bowman of Ripon, saw the patient striving for breath; and attention being drawn to his state, it became manifest that he was in his first attack of asthma: respiration quickly became almost inaudible over both lungs, and then, after a definite interval, sibilus supervenien. The attack followed the usual course, and the asthma thereafter frequently recurred.

As regards the sudden occurrence of atelectasis pulmonum, alleged as a cause of the disease, there is perhaps less to be said. Nevertheless, it may be well to point out that, in infancy at any rate, there are cases, and these by no means infrequent, where atelectasis occurs very suddenly; and there are cases where the auscultatory evidence makes it probable that this condition flits about the lung in a manner almost comparable to that of the migratory passage of the asthmatic paroxysm. And for my own part, I believe it to be probable that acute collapse of the lung occupies a much more important place in the production of pulmonary affections than is supposed.

Diagnosis.—It is not necessary to linger long upon this section of the subject. It is true that many affections are called asthma that are not so regarded in this article. The short breath and the dyspnœa of chronic bronchitis, and the dyspnœa and orthopnœa of heart disease, are often thus designated; in both of these the dyspnœa is rather a subdued distress than the acute agony of spasmodic asthma; so with the air-hunger of some cases of renal disease, and of dilatation of the heart. The inspiration is free in such cases, but panting; it is not a dyspnœa. And yet it must be added that sometimes, in the early history of a granular kidney, the complaint of the patient may be chiefly of asthma of a mild kind; and without a general investigation of the case, without the hard pulse, the thick first sound, and perhaps the retinal changes, the real nature of the case might be overlooked. Of other conditions more likely, perhaps, to give rise to mistake I incline to place hysteria. I have certainly found myself occasionally in a
difficulty between the one and the other, more particularly when it has been necessary to depend upon the history of the attack as submitted for an opinion, some time, it may be, after all the symptoms have passed away.

I am reminded also by the editor, first of a restless disturbed sleep, experienced by some persons, that is really a mild asthma, although not recognised as such by them; secondly, of that curious faucial or laryngeal suffocative spasm, often in gouty people, that awakens the subject of it in the middle of the night in terror lest he should choke.

Mediastinal tumours and aneurysm of the aorta, by leading to paroxysmal dyspnoea of a sort, are sometimes liable to be overlooked in a hasty diagnosis of the more familiar disease. And there are various obstructive maladies in the upper air-passages that may, in like manner, cause difficulty at times. The safeguard against mistake lies in the unfortunate fact that asthma is very common, and therefore in its usual features is very generally known; and in respect of other maladies, even should they be entirely paroxysmal which is uncommon, each one has usually some peculiarity of its own that is sufficient to arrest the attention. Any one of these things may, of course, exist in association with the special nervous proclivities of asthma, and it might then become a matter of the greatest difficulty to distinguish between the morbid occasions of the spasm. Nevertheless, it may be doubted whether in practice this difficulty often arises.

Treatment.—We will first consider the principles and afterwards the details, lest in the multiplicity of the latter, and in the urgency and intractability of the disease, we lose our hold on the principles to which details ought to be subordinate. As I have already said, to me it seems impossible to doubt that asthma is one of those nervous actions of which we see so many examples in our economy, and which have been well called by Dr. Edward Liveing paroxysmal neuroses.

Epilepsy is one of these; some forms of insanity are others; migraine is another; asthma is another, and so on. Now all these more or less obey this law, that the more they come the more they may. Nervous actions, which in their essence and initiation are not abnormal, by excess of energy, or of frequency, or of both, become abnormal; and ultimately a bad habit becomes fixed. Surely, both in epilepsy and asthma there is much of habit in the intractability of the disease; and if control is to be gained over either, it must be by catching it in the earlier days of its appearance, and by arresting it before it becomes confirmed. We think that we can sometimes gain some control over the convulsions of infancy; we can perhaps keep them at bay sometimes, and so stop the child from becoming epileptic. But what case is more hopeless than that of the confirmed epileptic, even though he be persistently stupefied with bromides?

The case of asthma is a parallel one. It has been contended that it is a disease rather of childhood than of adult age; and that to pay
attention to this fact and to the suggestions that flow therefrom, offers the best possible chance of stopping the attack, and of preventing the fixation of the habit and the establishment of chronic asthma. The chronic asthmatic is almost as hard to cope with as the chronic epileptic.

There are two methods of dealing with the asthmatic. On the one hand, we may attempt to make the environment of the patient conform to the conditions required by the individual; or, on the other hand, to harden the individual, to widen his range of accommodation, and so to make him less susceptible. And in the matter of drugs somewhat similar alternatives present themselves; we may either give sedatives to the over-sensitive nerve structures concerned, or give drugs, if such there be, to raise the level of nervous action to that higher platform that shall enable the perceptive centres to take less heed of their unnatural worries.

But the asthmatic paroxysm is so distressing that, almost always, the treatment of it usurps the first place; and too often this urgency of the situation upsets the perspective. If we are called to a patient in the stress of a paroxysm of asthma, clearly, on all accounts, it must be arrested as quickly as may be; there is no time to be very careful and consistent about ways and means. And the quickest way to relieve a paroxysm of asthma is to make the patient inhale some fume or other, as of nitre, nitrite of amyl, or chloroform; or to give him an injection of morphia or a dose of chloral; indeed, as we all know very well, doctors see paroxysms of this kind less often, because various patent powders for creating fumes hold the field so largely that most people do without us, and stick to their patent remedy.

Thus the treatment of asthma too often becomes a repeated sacrifice to the paroxysm; and the patient drags along, thankful for the small mercy of temporary freedom from his troubles, and easy in mind if he can carry in his pocket protection from those that are to come. But this plan of campaign is ultimately a most disastrous one. It unquestionably produces temporary ease; but what happens afterwards is this: the vapour, on reaching the mucous membrane, stupefies or exhausts the nervous centres, and stops the spasm for a time. But at the same time some of these remedies, by stimulating the mucous membrane and provoking the flow of mucus, make the local erythema rather worse than it was before. The more sedative kinds of inhalations do but appease by offering bribes to vicious nervous influences. By and by the nervous centres wake up again to find matters no better, rather the contrary; and then on comes the spasm again, and the whole process is repeated; and, with each repetition of the cycle, the nervous centres, as their nature is, become more exhausted or more irritable, their sleep is shorter, their spasm is more and more quickly repeated, and the poor patient ultimately lands himself, with perhaps some lessening of the severity of each paroxysm, in a more prolonged or persistent stuffiness hardly less distressing to bear: all day long he appeals to his powder, and becomes in fact the slave of an appetite that he has whetted and that he
cannot now control. Thus ends the chronic asthmatic who betakes himself to vapours. But this is not all, for by common consent a repeated application to some of these drugs, whether by making matters worse in the lungs, or by worrying the cardiac ganglia or what not, tends to dilatation of the heart, and is equivalent to a good many nails in the coffin of the asthmatic. Moreover, this dread of the paroxysm itself is carried into the preventive treatment of the disease, and the patient is submitted to what may be called the glass-case treatment; that is to say, the temperament of the patient is ignored, or not considered as of importance, and the disease is supposed to be brought on by chill. If he be wealthy and adventurous, he fights his environment by running away; and thus he may, perhaps, get along pretty well. If the patient be a child, it is probably kept indoors, except in the finest of summer weather; yet, nevertheless, the history too often is that “it has caught another chill,” but no one can say how. At first, perhaps, the child had the whole house to roam about in, but, as the “colds” recur, it is confined to one room with a south aspect; and yet things do not mend. So the doors of the room are carefully screened, the windows perhaps pasted up, and still the success being not all that can be desired, extra clothing may be piled on. And ultimately the doctor finds somewhere hidden under this heap of precautions a pale, moist, flabby, steamy thing, with big eyes, thin cheeks, protruding ribs, and a more or less general bronchitis; a case of “successful” management, because no attack has occurred for some weeks! But is this to be called success? This is to nurse the powers into imbecility; and the inevitable result is, that the first time the patient puts his head outside the door a fresh cold is “caught,” and a fresh term of imprisonment is ordered. I venture to say that if asthma is to be prevented at all, it will never be kept at bay by hothouse treatment such as this. Yet, unfortunately, it is easy to utter destructive criticisms of this sort, but difficult to point to a better way. I think there can be no doubt that the first requirement for the asthmatic is to put him into a climate in which he can be much out in the air. But there is the difficulty: we know so little about climate; and asthma is so individual a disease. No one can foresee in a particular case whether this place or that will be suitable; and, when the issue is doubtful, experiments in moving invalids about are never likely to be made with any great thoroughness. But for most asthmatic persons there is generally for each his own place or places where he is better or well. Thither he should be sent, at any rate, for a time. This place may be at the sea; or it may be inland; sometimes it is a dry place, sometimes a humid; often even it is a large town: “In the great majority of cases an urban air is the air that cures; and of a city air, that seems to be the best which is the most urban—the densest and smokeiest” (Salter); but wherever it be, the patient should be out and about with very little restriction; and an attempt should be made by this means to render the morbid circuit less prone to discharge. Of games and sports, all should be encouraged that
are outdoor and healthful and invigorating. Some further remarks on climate in the treatment of asthma will be found in the first volume of this work (p. 293).

In diet it is necessary to be careful, but not too much so. It is very easy to give a number of restrictions about food; and thus to make matters worse; yet asthma certainly often does seem to start from a meal that has not been digested—one which may have been too large, of an improper character, or taken at some irregular hour: the points to aim at are good, plain, light food in moderate quantity and slowly ingested. The asthmatic, particularly children, are often deprived of potatoes, of starchy puddings, jam and sugar, and goodness knows what else, and on the other hand are put on various meat juices and other good things in the wrong place, so as to remove all rocks of offence from the path of their pneumogastrics. But "if these things be done in the green tree, what shall be done in the dry?" What chance has such a child of reaching old age? Any food that is plain and wholesome and not known to disagree may be allowed. It is a good thing to have the chief meals early in the day, when digestion is vigorous; therefore breakfast and lunch—an early dinner—should be the main meals; anything taken later must be small in quantity and of the most digestible kind. All meals for the asthmatic should be small ones; his stomach should never be distended [vide art. on Dietetics, vol. i. p. 398]. The bowels should be kept carefully regulated and sufficiently open by taking some saline aperient, or other simple laxative. Every effort should be made to keep the patient in as healthy and physically fit a condition as possible. A tepid or cold bath should be taken in the early morning, and the living room well ventilated.

These must be the general principles upon which to deal with the asthmatic; and the more unhesitatingly the younger the patient, and the earlier in the course of the disease that he comes under treatment.

In considering the treatment by drugs, two divisions of the subject naturally suggest themselves; namely, those medicines that are useful in preventing asthma, and those that are so when the actual attack is threatening or in progress. Again, a distinction must be made between the cases which seem to be pure nervous asthma, those which have any degree of persistent bronchial catarrh, and those already mentioned, which come on in later life, and may not irrationally be attributed, on the one hand, to blood conditions that as a group may for convenience be called gouty, and, on the other, to degenerative changes in the tissues.

As a preventive remedy in the pure form of asthma, no drug is in my opinion equal or nearly equal to arsenic. It should be taken for three or four weeks, then omitted, and then resumed after an interval of equal length; and so on for three or four courses: and the drug should from time to time be resorted to in periods when from any cause the nervous centres begin to show signs of lowered tone. I have not made much use of phosphorus, but it has been spoken well of, and it
might also upon occasion be of value; and so likewise with other good nerve tonics, such as bromides or hydrobromic acid.

In cases where a persistent bronchial catarrh is at the bottom of the trouble, there is obviously less to be expected from medicine, and a suitable climate promises best; as a rule such cases do best in dry and bracing air. A friend of mine thus circumstanced found himself almost renewing his youth as he climbed the Malvern Hills. Others again find more relief in such places as Hastings, Ventnor, Bournemouth; some even in Torquay. Good results are claimed in the bronchial cases for the sprays and waters of the sulphurous springs of Mont Dore, of the Pyrenees, of Harrogate, and of the arsenical waters of La Bourboule. The two chief drugs from which much benefit is often derived are strychnine in three to five-drop doses given steadily for some days, and the iodides which often prove of great value. Perhaps the one acts as a stimulant to the respiratory centre, the other as an expectorant. For the asthma that occurs in later life an eliminant treatment is on the whole the best. It is in such cases that blue pill and colocynth in moderate doses once or twice a week are useful, or saline laxatives with careful attention to and restriction of diet. In these cases, again, iodide of potassium, perhaps by a depressing effect upon the arterial pressure, will often help very considerably.

My friend Dr. Kingscote maintains that the asthmatic state is much benefited by brine baths and systematic exercises, such as have been elaborated at Nauheim, for the treatment of certain forms of disease of the heart; one can well understand that means of this kind, by stimulating the circulation and facilitating the flow of blood through the lungs, may prove of much service.

To relieve an attack, or the semi-asthma that forebodes or lingers after an attack, other means must be used. In the threatening of an attack, or in the dyspnoea that lingers when the more acute symptoms have subsided, many drugs have been tried, and at one time or another succeeded. Of these I should put first a combination of iodide of potassium with the ethereal tincture of lobelia; five, ten, or even fifteen grains of the one, and ten or fifteen minims of the other, seem to bring relief when other things may have failed. Some prefer stramonium to the lobelia. The late Dr. Hyde Salter thought very highly of the Datura stramonium and the D. tatula; their best effects are observed when smoked like tobacco; but they may also be given in a pill, extract, or tincture. Sometimes a combination of iodide of potassium and chloral hydrate has been effectual. It is under such conditions as these that the Euphorbia pilulifera and Grindelia robusta are most useful. The former may be given in a decoction, a wineglassful twice a day; or in tincture, ten to thirty minims, twice or three times a day, or as often as may be requisite. The grindelia is in the form of a liquid extract and is given in similar doses to the tincture. This drug is also recommended at the onset of an attack, in half-hourly doses, until relief has been attained. I have known it to produce decided relief.
but I have not, upon the whole, been very successful either with this
drug or with euphobbia. In the thick of an attack the remedies most
in use are inhalations*of various vapours; and of these, perhaps the
commonest, and one of the most harmless, is blotting-paper soaked in
nitrate of potash, which will often relieve and sometimes very con-
spicuously.

There are many other powders made for the production of fumes;
some are stimulating, and seem to act by provoking cough and the free
secretion of mucus; others, and these I believe the less harmful, are of
a sedative nature. Some of them are made into cigarettes for smoking,
and most of them contain stramonium in some form.

Of inhalations available for more strictly medical uses, three may
be mentioned: nitrite of amyl, iodie other spoken well of by Dr.
Thorowgood, and of course chloroform. In severe cases the last named
may be of the greatest possible value, although its effect is apt to be
but transitory, and the attack may resume its severity as the stupor of
the drug wears off. Of all the other drugs that have been recom-
manded for the relief of the paroxysm, morphine probably stands first;
a hypodermic injection of a sixth of a grain will often procure almost
immediate diminution of the violence of the dyspnea, which gradually ends
in complete cessation of the spasm. Pilocarpine is also a valuable drug; a
tenth to a quarter of a grain may be given hypodermically; a free secretion
from the mouth and fauces is the result, and the spasm is thus relieved.
Sometimes the patient is sick, a thing by no means undesirable; for an
emetic is one of the means advocated for arresting an attack, and no
doubt sometimes with marked success. A combination of bromide of
potassium and chloral is also a good sedative to give at the onset of a
paroxysm. Belladonna, hodecanus, and conium, though not of so
much value, are all of use in their way; tobacco is also said, by virtue of
its powerful depressing action, to be a useful palliative drug. I have
heard it said of pilocarpine that the remedy is worse than the disease;
and, considering the distressing nature of the malady, this is a serious
attack upon the benefit derived from it. If this be true as regards
pilocarpine, it must be still more apt for tobacco, which produces a
dreadful malaise, and is a difficult drug to control in those who are
unaccustomed to its use, in whom only it appears to have the effect
wished for.

Of stimulants, too, coffee and alcohol may be mentioned. Strong
coffee is indeed a popular remedy that has often given relief, as also has
citrate of caffeine. As regards alcohol, I have no personal knowledge
of any special virtue, but Hyde Salter says of it, that while in many
cases it does not do much good, in some it has a most powerful effect,
particularly when all other remedies have failed. It should be given
hot and strong. The compressed air treatment of asthma is described
in the first volume of this work (pp. 315, 316), to which the reader is
referred.

Hay fever, or hay asthma, is in the opinion of many a pure form of
asthma, and with this opinion I myself coincide; it is accordingly more or less amenable, as are other forms of asthma, to treatment by drugs of the same character. I refer more particularly to arsenic; and I should maintain this even for nasal cases: it relieves the itching and smarting of the eyes, the aching of the frontal sinuses, the itching of the nasal mucous membrane and of the nose itself, the sneezing, the watery discharge, the obstruction of the nostrils, the dryness and irritation of the lips and throat. But Karl Binz and others have maintained that local remedies, used upon germicide principles, give great relief in many of these cases; and those who have worked in the special department of diseases of the nose and throat declare that, by paying special attention to the morbid erectility of the mucous membrane over the spongy bones of the nose, this disease may be much reduced. Binz advocated the irrigation of the nostrils with a solution of quinine; Sir Andrew Clark suggested some carbolic preparation; and of late many have tried the application of solutions of cocaine, more upon alleviative than upon curative principles, perhaps; unless alleviation be an earnest of cure.

No one can doubt that these various measures are all useful in their proper place, nor can any one doubt that they have their dangers. For instance, I saw but the other day a lady who for the discomfort arising from the frequently recurring turidity of the nostrils, which is characteristic of hay fever, had betaken herself by medical advice or without it to the use of cocaine locally. Accordingly, more or less, both by day and night, she would pack her nostrils with a solution of cocaine, of which one grain at each time was put into each nostril; and thereafter, by means of hawking and spitting, and other contortions of her pharyngeal muscles, the solution was spread all over the affected area, and temporary ease was obtained. At least six grains a day were thus disposed of, and sometimes more. The position to take with regard to local treatment is this, as it seems to me: the local symptoms are not the disease, and therefore, however necessary it may be at times to relieve conditions that cause great distress by means of this kind, they may do harm by inducing other morbid changes in the part, and conditions that were but temporary may thus be rendered permanent. For instance, a paroxysmal sneezing will stop immediately under the influence of some diverting train of thought, just as asthma will stop under any sudden and powerful mental stimulus. All must agree that if there be any actual disease of the mucous membrane, whether due to the existence of polypus, of a deflected septum, or what not, it must be advisable to get the mucous surface into as healthy a state as possible so as to remove one obvious source of possible irritation. But for cases of asthma in which there is no definite nasal worry, the question must at any rate be considered an open one. If the neurotic origin of the disease be accepted, as I think it must, no one can rest content with the treatment of a peripheral symptom. Still, if it can be shown that a large measure of relief is thus obtained, such an experience must of course be utilised upon the principle that half a loaf is better than no bread. The difficulty of
arriving at any souri' conclusion as to the value of such treatment lies in this, that the specialist and the physician see the cases at different times: the one in the first flush of that post-operative quiescence that we all so well recognise as a characteristic of nervous ailments; the other, when that quiescence has passed off, and the old habit has resumed its sway. I have known some patients to be apparently benefited, but others who have not received any adequate reward.

Prognosis.—I hold most strongly that asthma may be treated with a large measure of success if it be taken in hand at the proper time, that is in childhood; and if it be possible to put the patient under suitable conditions,—those conditions being, in brief, such as will allow of the patient being turned into a good healthy animal. It is in childhood, if at any time, that the opportunity offers of educating the patient out of a faulty habit into a better regulated state of his nervous centres. In the case of the adult one cannot be so hopeful. One could not be so hopeful of successfully combating convulsive attacks occurring late in life as in those occurring in infancy; and I fancy that the asthma that begins in adult age is indicative of some deep ingrained nervous fault, which is not readily to be controlled. Moreover, adults fall in less readily with counsels of perfection, such as the radical cure of faulty habits; they are in distress, they insist upon a dose to set them right, and if one man won't give it them, or does not hit upon the right thing, they quickly resort to some one else, who manages things, as they think, better. Still, even the adult asthmatic is sometimes a sensible person, and many agree to desist from inhalations; to take a drug, such as arsenic, patiently; or iodide, when an attack threatens; or such other drug as may seem best suited to the particular case: to act thus is in most cases to procure considerable relief.

With regard, however, to that other group, when spasmodic asthma occurs in middle age, and after; when, as causes or provocatives, certain changes in the tissues and organs, gouty and other, come into prominence; and when age with its paling vigour of function and its conscious or unconscious indiscretions of living and other habits leads to the over-charging of the blood with waste products, and to excess of arterial blood-pressure, and thus to a true spasmodic asthma analogous to the gouty vertigo and gouty convulsion occasionally seen in adult life: then no doubt great relief, and even cure, may result from such drugs as blue pill, iodide of potassium, and other's, given with the purpose of reducing the arterial pressure, or of eliminating waste products. More difficult to speak hopefully of are the cases associated with and perhaps produced by a pre-existing bronchial catarrh. As Hyde Salter remarks, we send such patients to the Riviera to relieve the bronchitis, and the asthma is aggravated; and thereby we see in a measure how essentially independent the two conditions are; and when, after travelling about, they come to the land of promise so far as their asthma is concerned, then perchance the climatic conditions are not suited to the bronchitic affections. * But even in such persons there is no doubt that, by dealing
with the bronchitis and by endeavouring to ameliorate it by means of a suitable climate, the bronchial tubes will become more healthy, and there may yet be scope for carrying out those principles of reinvigoration of the nervous tone upon which I have dwelt; and thus some of the stress of the asthma may be relieved. Nevertheless, when the best has been done, one cannot but regard the disease as serious, and in too many cases baffling; for even in cases where much good seems to have been done, the disease reappears again, perhaps after many years. In looking over notes of a number of cases, it comes out clearly that in several where the disease existed from, say, the age of three to ten years, it reappeared at forty or fifty. I have already alluded to the many points of similarity between asthma and epilepsy; and this is another feature of resemblance. We meet, too, with many people who have lost the tendency, and who are still free; but many of these, although they say they have lost the asthma, are still a little wheezy, and undoubtedly have some slight amount of bronchial catarrh. So that on the whole there is a degree of uncertainty about the fate of those who are asthmatic in early life. As regards the actual duration of life, perhaps all that can be said is that spasmodic asthma is compatible even with a long life. Of those who become asthmatic in later years, excepting the group of cases due to high arterial pressure already mentioned which may be a fairly large one, most are likely to suffer severely; and, their disease is but too likely to become more or less permanent.

JAMES F. GOODHART.

REFERENCES

SYPHILITIC DISEASE OF THE LUNGS

Our knowledge of the anatomical, characters and clinical history of syphilitic disease of the lungs is still very incomplete, notwithstanding that much has been written on the subject. This is due in part to the rarity of the affection, but chiefly to the difficulty until lately experienced in distinguishing between the lesions of syphilis and of tuberculosis.

Up to the date of the discovery of the tubercle bacillus it was very often impossible to determine with certainty during life whether a given case of pulmonary disease were tuberculous or not; and after death appearances which some considered to be distinctive of tubercle were said by others not to possess this significance. Now, however, that we possess a test for tuberculous lesions, it may reasonably be hoped that the whole subject of syphilitic disease of the lungs will be placed upon a secure foundation.

That the disease is of rare occurrence is a fair inference from the fact that the museums of the London hospitals and of the Royal College of Surgeons, all of which I have recently visited, contain only twelve specimens which are believed to illustrate syphilitic lesions of the lungs; and of these, two may be excluded, as either not of that nature, or of a nature so doubtful that in the present state of our knowledge they are inadmissible as evidence. None of these specimens is from a case of congenital syphilis.

Morbid Anatomy.—The following pulmonary lesions have been attributed to syphilis: (a) gumma; (b) white hepatisation (Virchow, Weber), or “epithelioma of the lung” (Lorain, Robin); (c) gray infiltration (Welch, Panceritz); (d) lobular pneumonia or broncho-pneumonia (Forster, Welch); (e) fibroid induration; (f) changes in the lymphatics (Hermann Weber); (g) a destructive disease, the so-called “Syphilitic Phthisis.” It will be convenient to consider separately the lesions of the hereditary and the acquired disease.

Hereditary syphilis.—The pulmonary changes in hereditary syphilis may be either circumscribed or diffuse; to the former the term “gumma” is applied; the latter are classified under the head of “pneumonia.” It is, however, far more common to find the two changes associated than to meet with either separately.

A. Gumma.—As this lesion is of comparatively rare occurrence in congenital syphilis, and when present does not differ either in appearance or in microscopical structure from that found in the acquired disease, a more distinct picture of the morbid anatomy of the two affections will be obtained by describing it under the latter heading.

B. Pneumonia.—Two different lesions are included under this heading—namely, “white pneumonia” and “interstitial pneumonia”; but of these it must again be stated that they occur more often in combination than apart.
(a) *White pneumonia* (Virchow, Weber), *Epitheliomys of the lung* (Lorain, Robin).—This lesion, which in its true form is rare, is found only in the lungs of still-born children, or of such as have survived their birth a very short time. Other unmistakable signs of congenital syphilis are usually present, and in such cases gestation has seldom proceeded to the full term. It is a diffuse change affecting a lobe either as a whole or in part; or one or both lungs may be completely consolidated.

In still-born children the affected part is bloodless and airless; even if force be used, it may be impossible to inflate it; but in infants several days old the lung always contains some air.

The lung is much increased in size, and its surface may be marked by the ribs. It is solid, dry, white, yellowish, or grayish white in colour; but sometimes presents a reddish marbled appearance. The section differs from that of an ordinary pneumonic lung in that the granular appearance characteristic of the latter is absent, the surface being smooth and somewhat shining.

On microscopical examination in true cases the interstitial tissue is not increased. The alveolar walls are thickened, and the small bronchi and the alveoli are filled with masses of cells of which some are round and others have more or less the character of epithelial cells: the cells are for the most part undergoing fatty degeneration and are beginning to break down. The alveoli are markedly enlarged. The colour of the affected area is due partly to the above changes, but in part also to diminished blood-supply the result of pressure upon the capillaries. The lung tissue surrounding the consolidated part may show some degree of emphysema. Ecchymoses may be present in the pleura, pericardium, and thymus gland; but these appearances are probably incidental to the mode of death.

The bronchial glands are as a rule enlarged and on section dense, from a new formation of fibrous tissue enclosing cells arranged in a concentric manner.

White pneumonia is a lesion of purely pathological interest, as, owing to the filling of the alveoli with cells, the subjects of it, if not still-born, are unable to maintain the respiratory function for any length of time, and soon succumb.

(b) *Interstitial pneumonia.*—This is the most common pulmonary manifestation of hereditary syphilis: but it occurs more frequently in association with some of the changes described under "white pneumonia" than as a purely interstitial lesion. In its true form it is distinguished by a small-celled infiltration of the interalveolar connective tissue, the alveolar epithelium remaining unaffected. This change may be present to a very varied extent. In some cases lungs thus affected appear normal to the naked eye, the lesion being only discoverable on microscopic examination.

In well-marked cases the lungs are large and hard and of a pale or dark grayish red tint. The change may be present throughout the organs, or a single lobe or portion of a lobe may be alone affected. To
the naked eye, the lung tissue presents a decidedly coarse appearance. On microscopical examination a marked increase is seen in the inter-
ateolar and interlobular connective tissue, which forms broad meshes in-
cluding small spaces wherein the alveoli are either crowded together or
completely obliterated.

In some cases the interalveolar meshes appear to consist of a dense
capillary network, the vessels being dilated and tortuous. Around the
vessels and bronchi there is a marked increase of the connective tissue,
and the tunica intima of the small arteries is thickened. The alveolar
epithelium may show desquamative changes, and brown and yellow pig-
ment granules may be present.

Interstitial pneumonia is often found in association with congenital
syphilitic lesions in the skin, with interstitial hepatitis, and with changes in
the epiphyses; but it is also found in cases in which gummatas are present
in the lungs, liver and other organs. The change begins during fetal
life, and at birth may have affected the lungs extensively. In such cases
life is of short duration and death occurs from asphyxia, as is shown after
death by the frequent presence of ecyhmoses in the pleura, pericardium
and thymus gland. When the change is less advanced at birth, such
children may die at a later period by a slow process of carbonic acid
poisoning, the first sign of which may be that a child previously fretful
and noisy becomes quiet.

In cases in which the other organs are healthy, or nearly so, life may
be prolonged for months or years; such subjects are, however, specially
liable to acute disease of the respiratory organs, such as pleurisy, acute
bronchitis, and broncho-pneumonia.

From the above description it will be seen that the morbid processes
concerned in the production of the gummatous and diffuse changes found
in the lungs of syphilitic children chiefly affect the connective tissue and
small arteries. They are—(i.) A round-celled infiltration and proliferation
of the interlobular and interalveolar connective tissue, originating in the
acellular tissue around the bronchi, and leading to marked thickening of
the framework of the lung. (ii.) An isolated perivascular cell prolifera-
tion, which begins around the small arteries, and is accompanied by
changes in the tunica intima (Hochsinger). Both the periarteritic and
peribronchial granulations may occur as separate nodules or node-like
foci; or they may be diffused over large portions of the lungs. A well-
marked desquamation of the alveolar and bronchial epithelium is almost
always present, but it is quite a secondary process.

In the account here given of the pulmonary changes found in hereditary
syphilis the descriptions of Heller, Spaundis, and Hochsinger have been
followed, and to these authors I desire to acknowledge my indebtedness.

Association of congenital syphilis and pulmonary tuberculosis.—
Syphilis, by lowering the resisting power of the individual, may dispose
to tuberculosis; and it has recently been shown by Hochsinger that the
virus of syphilis and tuberculosis may be jointly transmitted from
parent to offspring.
This observation is of much importance, and throws a new light upon the nature of the pulmonary lesions found in infants the subjects of congenital syphilis. Hitherto it has often been assumed, on evidence which is now proved to be insufficient, that such lesions are of syphilitic origin; whereas it is clear that they may be due to an associated tuberculous infection.

In three infants suffering from congenital syphilis, and presenting symptoms of infiltration of the lungs, the pulmonary disease was found after death to be due to tuberculosis and not to syphilis. Tubercle bacilli were found in the lungs in all the cases.

The first case was observed in 1891 in a child not quite three weeks old; the second in 1891 in a child twenty-four days old; the third in 1893 in a child eleven weeks old.

Case I.—Anna B., æt. nearly three weeks. The parents had been married nine years. The father acquired syphilis shortly before marriage. The mother died from pulmonary tuberculosis three months after the birth of the child. The first and second children of the marriage were still-born; the third and fourth died during the first week; the fifth and sixth were living, ages four years and two years respectively. The case of the seventh child is here described. From the time of birth she was sickly and suffered from nasal obstruction, snuffles, and dyspnœa. Râles were present in the chest. At the end of the second week a bullous eruption appeared on the nates. The child presented all the ordinary external signs of congenital syphilis, and was shown at the Vienna Dermatological Society as a case of gummatous disease of the viscera.

On examination of the chest there was marked dulness on the left side from the angle of the scapula downwards, with bronchial breathing over the dull area. The respiratory murmur was harsh over both lungs, with rhonchi and coarse râles. The spleen was enormously enlarged, extending as low as the anterior superior spine of the ilium; the liver could be felt four fingers’ breath below the costal margin, it was hard and the edge was rounded. The diagnosis was pulmonary and visceral syphilis. Mercurial treatment was prescribed. The child died on the thirty-first day after birth. On post-mortem examination the internal organs were found extensively infiltrated with tubercle. Both lungs showed tubercles varying in size from a miliary granulation to a walnut. A nodule as large as a hen’s egg occupied the right middle lobe. The left lobe of the liver was almost completely replaced by a caseous nodule; numerous tubercles studded the right lobe. The spleen was enlarged to nearly four times its normal size and contained similar deposits. Tubercles were also present in the kidney, pericardium and peritoneum. The mesenteric and bronchial glands were enormously enlarged, and in many places caseous. Tubercle bacilli were present in all the lesions. None of the lesions in the internal organs was of syphilitic origin.

Case II.—Victoria S., twenty-four days old. The mother had previously brought three children suffering from congenital syphilis to the same clinic. She had previously stated that she had not had syphilis.
Nothing was known of the father, and it is not certain that either parent was tuberculous. The child had snuffles and presented all the characteristic appearances of congenital syphilis. There was a confluent papular syphilitic eruption on the nates and elsewhere. The percussion note over the left lung was dull and the breathing bronchial, with consonating râles. The spleen was slightly, and the liver markedly, enlarged. The temperature was normal. Mercurial treatment was ordered.

The patient was shown at the Vienna Dermatological Society as a case of syphilitic pemphigus and syphilitic pneumonia. The child died on the thirty-eighth day.

On post-mortem examination the left lower lobe was solid from grayish white infiltration. There was acute catarrh of the bronchi of the left upper lobe and throughout the right lung, also of the larynx and trachea. The mediastinal and bronchial glands were enlarged, but not obviously caseous. The liver was large, reddish brown, somewhat indurated and with rounded margin. On microscopical examination of the lungs confluent peribronchial and perivascular tuberculosis was found, with tubercle bacilli. In the liver recent interstitial inflammation was present, with fatty degeneration of the liver cells. No trace of tuberculous lesions were found in any other organ than the lungs.

Case III.—Auguste G., eleven weeks old. The mother, at 28, was suffering from pulmonary tuberculosis. She had had five illegitimate children and denied having had syphilis. Nothing was known of the father. The child presented the characteristic appearances of congenital syphilis, and had snuffles and a syphilitic rash on the buttocks. The rash appeared during the third week. The child had suffered from cough since it was five weeks old. There was doubtful dulness over the right lower lobe with bronchial breathing and abundant moist râles. The liver was very large and hard, with a rounded edge. The spleen extended four fingers' breadth below the costal margin. The temperature was normal. Mercurial inunction was ordered. The child died aged sixteen weeks.

Post-mortem.—The right lower lobe was solid from a homogeneous, grayish white infiltration. Grayish red and yellow tubercles were disseminated throughout the upper lobe. The lower half of the left lower lobe was collapsed. The bronchial glands were enlarged and caseation was beginning. The liver was fatty and slightly granular. In the portal fissure there was a caseous lymphatic gland the size of a hazel-nut. The mesenteric glands were caseous. The spleen contained a large caseous nodule. Microscopic examination showed the characteristic signs of "chronic tuberculous broncho-pneumonia, tuberculosis of the spleen and mesenteric glands, and syphilitic interstitial inflammation of the liver with well-developed inflammation of the vessels."

It is clear from the perusal of these cases that it will be necessary in future, even when the evidence of syphilis in the foetus is undoubted, to examine carefully for tubercle bacilli before a pulmonary lesion is attributed to syphilis.

Acquired syphilis.—To present a trustworthy account of the morbid
anatomy of acquired syphilis of the lungs is a far more difficult task than that just attempted. In considering a matter of such uncertainty I have preferred to rely upon evidence which is at hand, and may be put to the test, rather than upon that to be found in the records of a period when, owing to the absence of any certain test for tuberculosis, the difficulty of distinguishing between the pulmonary lesions of tubercle and syphilis was almost insuperable.

A study of the specimens of pulmonary syphilis contained in the London museums shows that the possibility of the changes being due to tubercle was in nearly all cases carefully considered. These specimens and the records connected with them probably constitute the most trustworthy evidence on which to base a description of the morbid anatomy of the acquired disease, and, as will be seen hereafter, they have been fully utilised.

Pathology and Morbid anatomy.—Bronchial catarrh may occur as a manifestation of the secondary stage of syphilis, and possibly also of the period of incubation (Walshe). The fact that bronchitis, occurring without obvious cause in syphilitic subjects, may be greatly alleviated or cured by the administration of mercury, is strongly in favour of this view. In the late secondary and tertiary stages gummatous infiltration of the submucous tissue of the trachea and bronchi is not infrequent, and may be followed by the formation of fibrous tissue which, subsequently undergoing cicatrisation, produces stenosis, one of the most characteristic syphilitic lesions in the main bronchi.

No definite statement can be made as to the most common period of the occurrence of gumma in the lungs; cases of which the real nature could not be doubted have been recorded as early as two years and as late as twenty years after infection.

The pulmonary lesions of acquired syphilis belong chiefly to the late tertiary stage of that disease.

A. Gumma.—Gummatas may occur either singly or in numbers, and may vary in size from that of a hemp-seed or a hazel-nut to that of a hen's egg, but the latter size is of rare occurrence. A gumma may be found in any part of the lung, but more commonly within its substance than upon the surface; and more often about the root, near the large vessels and bronchi, than elsewhere. The lower lobes are perhaps more often affected than the upper.

A gumma is rarely seen in the very early stage, of which alone the name is in any sense descriptive; but it is said then to present a gelatinous or glutinous appearance (1), thus resembling a similar growth in the liver. At a later stage it is of a gray colour, tinged with various shades of a red, white, or yellow, and presents on section a smooth and semi-transparent appearance. At a still later period a gumma forms a well-defined nodule of a yellowish colour, firm and dry. Inflammatory changes in the surrounding lung may lead to the production of a well-marked fibrous capsule, but this may be absent. The gumma may break down, and, its contents having been discharged, an irregular cavity may result; but this is,
both absolutely and also in comparison with the occurrence of a similar change in caseous tuberculous masses, very rare.

The chief difference between a gumma of the skin, for example, and one of the lung is that whilst the former tends towards necrosis the latter tends to be transformed into a mass of scar tissue, the contraction of which causes puckering of the surrounding lung and overlying pleura. By the deposition of lime salts a gumma may become calcareous.

In histological structure a gumma of the lung does not differ essentially from a similar growth elsewhere. In the early stage it is seen to consist of a granulation tissue composed of small cells about \( \frac{1}{2} \) in. in diameter, arranged concentrically around the sheath of the small vessels, and in some cases around the small bronchi. At a later stage the nodule becomes opaque in the centre, and its cellular structure can no longer be recognised; but fatty and albuminous granules are seen instead in the meshes of a dense fibrous stroma. Finally it becomes converted into a mass of dense cicatricial tissue. A gumma may form a centre from which a small-celled growth may infiltrate the surrounding tissue, spreading chiefly along the bronchioles.

The walls of the neighbouring alveoli are also infiltrated with small cells; and the alveolar spaces contain inflammatory products, due either to epithelial proliferation or to the presence of cells of a character similar to those constituting the nodular masses already described. Giant cells are occasionally present, but are not so characteristic a feature of gumma as of tubercle.

According to Dr. Councilman, the essential process in the production of a gumma in the lung is a pneumonia with fibrinous exudation, accompanied by fibrous change in the alveolar walls, the whole subsequently undergoing caseation. The first step in the process is stated to be a hyaline degeneration of the capillaries of the affected area; this is followed by atrophy of the alveolar walls. The alveoli become distended with large pale epithelial cells and fibrin; the cells also undergo the hyaline degeneration, forming smooth bodies staining with eosin, and varying in size from one-half the diameter of a red blood corpuscle up to that of a large epithelial cell. The capillaries become converted into rigid tubes and their lumen is much narrowed. Similar changes occur in the small veins and arteries. Immediately around the bronchi and arteries there is a formation of connective tissue, and here the alveolar walls show much thickening and contain many small round cells.

The whole of the structures thus altered tend to undergo necrosis, and when that change is complete a caseous-looking mass results.

The following descriptions of specimens in the Museum of Guy's Hospital well illustrate the appearances presented by gummata in the lungs. It will be observed that all the specimens here described were removed from the lungs of adults.

No. 254.—A portion of the upper lobe of a lung showing on the cut surface two masses, one of which was described in the recent state as "consisting of a circumscribed nodule of a firm, yellowish, dry substance,
corresponding in all particulars to that in the liver, (a gumma), except in being somewhat less firm: the other is softening, breaking up, and in process of forming a cavity. Histologically the nodules are seen to consist of fibroid tissue with many areas of caseation and a few giant cells.

From the report of this case by Dr. Wilks the following additional particulars have been derived:—The patient was a sailor, aged 29. No history was obtained; he was moribund from laryngeal obstruction when admitted, and there was profuse expectoration of mucus and blood. There was a scar in the groin, and phimosis from a contracting sore on the penis. The whole mucous membrane of the larynx and trachea was deeply ulcerated, and the walls thickened by an infiltration of fibrous tissue into the submucous structure, producing great induration. The thyroid cartilage was bare at one spot, the lymphatic glands in the neck were enlarged.

The liver contained a dozen hard, round, fibrous tumours—the largest the size of a marble—yellowish white, tough, and of leathery consistence, dry, and emitting no juice on pressure. In two or three the circumference of the tumour consisted of a translucent structure; and this was evidently the more recent formation, the opaque and yellow parts being probably tissue undergoing a degenerative change. At one spot a deep cicatricial appearance was produced by the contraction of a group of these small nodules.

Microscopically the nodules consisted of nucleated fibres and fibrous tissue.

No. 255.—The lower lobe of a left lung from a man, aged 27, who died from erysipelas of the larynx. The specimen shows at its hinder part a large yellowish mass partially separated from the surrounding tissue. Smaller nodules are seen in the adjacent lung. The pleura over the gumma is much thickened. Histologically the nodule consists of fibrous tissue which stains with difficulty. There were many gummata in the liver. With the exception of the above lesions and some bronchitis the lungs were healthy. There was a chancre on the penis and suppurating buboes.

No. 256.—A portion of lung showing scattered through it several small masses of irregular shape, yellowish in colour, and firm on section. These masses are easily separable from the surrounding lung, which is healthy. Histologically the nodules show a central area of caseous material surrounded by a narrow zone of fibrous tissue in which are many small round cells. The liver contained a single gumma, and was in a condition of diffuse syphilitic hepatitis. There were several gummata in the testes. From a man, aged 39, who had suffered from cough and dyspnea for six months. He was admitted for hepatic ascites and slight jaundice.

The following specimen from the same Museum illustrates the appearances presented by a gumma which has undergone fibrous transformation:—

No. 253.—A section of a right lung. From a man, aged 36, admitted for fracture of the cervical spine. At the upper part of the lower lobe is a circumscribed patch of fibroid material with radiating processes extend-
ing into the surrounding pulmonary tissue. The pleura over it is much thickened. The interlobar septum is thickened, and from its upper portion similar fine fibrous strands radiate into the upper lobe. Other portions are very emphysematous (also fibroid and pigmented). No tubercle was found anywhere. There was lardaceous disease of the liver, spleen, and kidneys. Both testes were good specimens of syphilitic orchitis.

The following description of a specimen in the Museum of St. George's Hospital illustrates a combination of the caseous and fibrous stages of a gumma (10):

"Section of a right lung near the root. In the posterior and upper part of the lower lobe, close to the spine, there is an area showing marked fibrosis; situated within it is a caseous mass the size of a marble, somewhat loose. The overlying pleura is adherent and thickened; bands of thick grayish fibrous tissue pass inwards from the pleura, and joining with each other form a meshwork." No tubercle in any organ; surface of the liver scarred from perihepatitis. Large caseous gumma near the portal fissure, with smaller ones in its neighbourhood. Liver cirrhotic and lardaceous. Gummata in both testes. From a male patient who contracted syphilis in 1884, six years previous to his death. In 1886 he suffered from syphilitic disease of the testes and sores on the right elbow. Death was due to uremia.

Lobular or Broncho-pneumonia.—A careful review of the evidence on which it is believed that inflammatory changes of the lobular or broncho-pneumonic type occur as the direct result of syphilis impresses me with the conviction that many of the cases described in the past as presenting such lesions were really cases of tuberculosis.

- In the following case (3), however, such a possibility may be excluded. It will be observed that the pulmonary lesions were secondary to and in continuity with the growth of large gummata in the liver and spleen. The specimen is in St. George's Hospital Museum:

Left lung.—The lower lobe is deeply congested and partially consolidated; the consolidation is in patches as in catarrhal pneumonia. Some of these masses appeared purulent, others fatty or caseous. The size varied from 3 mm. to ½ mm.; each patch or nodule was surrounded by a deeply congested zone. Right lung.—The lower lobe presented changes similar to the above; it was adherent to the diaphragm, through which a large caseous gumma in the liver had extended into the lung. At the upper margin of the caseous mass there was much fibrous induration and exudative consolidation of the pulmonary tissue. For the microscopical changes, which are given in great detail, the reader is referred to the original article. There was a gummatous mass chiefly in the upper part of the right lobe of the liver measuring 5½ in. by 4½ in., and another occupying the upper third of the spleen. That organ was greatly enlarged, weighing 2 lb. 6 oz. Both liver and spleen were firmly adherent to the diaphragm, and the muscular tissue of the latter was in part destroyed by the extension through it of the gumma in the liver. The specimen was
taken from a man, aged 43, who contracted syphilis in 1861, twenty-five years before his death. He had periostitis of the tibia in 1864, left hemiplegia in 1871, and again in 1876.

Fibroid induration.—The following are the more important changes of this nature which have been attributed to syphilis: (a) thickening extending from the hilum around the bronchi and vessels; (b) isolated masses of fibroid tissue in various parts of the lung; (c) diffuse changes occupying the whole or the greater part of one lung.

The marked tendency of gummatous lesions to spread along the vessels and bronchi has already been referred to.

The following case (4) is an example of syphilitic fibrosis illustrating the first variety of this lesion:

Woman aged 50.—Thrombosis of cerebral artery; hemiplegia. Pigmented excavated scars on left leg, due to old syphilitic ulceration. Lungs.—Emphysema. Right lower lobe contained a deep depression and a much-puckered cicatrix due to pigmented fibroid bands running into the lung tissue. No caseous or calcareous nodules. No pleural adhesions. Microscopical examination.—The fibroid tissue is arranged chiefly around the vessels and bronchi with a more or less concentric disposition. The coats of the vessels are much thickened. There is a small-celled growth invading the alveolar walls, which are also much thickened. In places the cells and nuclei are aggregated in heaps.

As an example of fibrosis in the form of scattered areas of induration, the following case may be cited from the same source (4):

Woman aged 25.—Fracture of cervical spine. Pigmented and puckered cicatrix and syphilitic ulcers on left leg. Calcified gumma in the liver. Right lung.—Upper lobe healthy. Middle lobe presented in the centro large irregular patches formed by radiating bands of fibroid tissue; also smaller scattered patches of the same nature: the bands whitish, not pigmented. One patch contains a calcified nodule. No pleural adhesions. Left lung.—Adhesions over lower lobe; and whitish, puckered, depressed fibroid patches with irregular thickening of the pleura. On section extensive fibroid infiltration; bands appear to run into the lungs from the pleura. Some small rounded cascous patches are also present.

The following specimen from the Museum of Guy's Hospital (9) illustrates the appearances met with in "diffuse syphilitic fibrosis of the lungs." The patient was a man, aet. 54, who had suffered from winter cough for some years.

No. 252.—A portion of a right lung in which there is a considerable excess of fibroid material appearing on the cut surface as a delicate network traversing the pulmonary tissue in all directions. The fibroid change is less marked at the apex than at the base, in which latter situation many of the air-vesicles are dilated; over this area the pleura is slightly thickened and is adherent. The dense fibroid tissue that pervades the lung shows, scattered through it, numerous collections of small round cells not undergoing caseation. No giant cells are present. The walls of the small arteries are thickened. One or two small cavities the
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size of peas, with soft caseous contents, were situated near the root of
the right lung, probably softening gummatas; no tubercle bacilli could be
found in them. The condition of the left lung resembled that of the
right. The liver was scarred; the testes were fibroid. Death was due
to bronchitis.

Changes in the bronchial glands and lymphatics of the lung.—In a case
of syphilitic disease of the liver, lungs, dura mater, cranium and sternum,
recorded by Dr. Hermann Weber, the bronchial glands and lymphatics
of the lung presented the following appearances:—The bronchial glands
were much enlarged—some being of the size of a pigeon’s egg, some only
that of a hazel-nut. From the grayish white section of the larger glands,
which were rather soft, a creamy fluid exuded, consisting of fat globules,
granular corpuscles, and an abundance of large cells in a condition of
fatty degeneration. The less enlarged glands were harder, their sections
offered a marbled appearance, large white patches, almost like bacon,
being interspersed with grayish red, very vascular tissue. No juice
exuded spontaneously or could be squeezed from the section. Large
nuclei and nucleated cells were the principal microscopical elements, with
a very small proportion of fibres thickly studded with nuclei. The
lymphatics leading from the lungs to the enlarged glands were dilated
and their ramifications on the surface and throughout the lungs were
distended with creamy fluid.

A similar appearance is described in the case of Drs. Delépine and
Sisley already quoted. “Immediately under the pleura there was
a network composed of ramified tracks. The appearance suggested
lymphatics distended with cells or some fatty products.” The lymphatics of the subserous layer of the pleura were considerably enlarged
over areas corresponding to the yellow patches († of syphilitic broncho-
pneumonia) within the lung.

Dr. Weber rejects the view that the bronchial glands were first
affected by the syphilitic virus, and that the engorgement of the pulmonary
lymphatics resulted from obstruction to the passage of the lymph.

A progressive destructive disease, the so-called “syphilitic phthisis.”

It appears to me that the question of the existence of a syphilitic
lesion of the above form can only be settled by a careful study of cases
which fulfil the following conditions:—

(i.) The cases must be complete; that is, the symptoms observed
during life must be considered in connection with the lesions discovered
on post-mortem examination.

(ii.) The evidence of syphilitic infection must be undoubted.

(iii.) Repeated examinations of the sputum must have been made,
and tubercle bacilli invariably absent; and the absence of tubercle from
the lungs (as the cause of the lesions) must be proved by post-mortem
examination.

(iv.) Syphilitic lesions about the nature of which there can be no
doubt must be found in other organs.

From such evidence alone can we hope to construct the clinical

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history and morbid anatomy of advanced syphilitic disease of the lungs.

The following cases illustrate this variety of the disease:—

Case I.—Charles N., aged 38, bricklayer. In 1892 he suffered from cough, with expectoration and pain on the left side of the chest. In 1893 he had night-sweats and dyspnoea. From January to May 1894 he was an in-patient of the Brompton Hospital under the care of Dr. Mitchell Bruce; the diagnosis then recorded was “Syphilis (?) tracheal stenosis, chronic bronchial catarrh, induration of the left upper lobe and of the left base with pleural adhesions over that area. Cicatrisation of the soft palate and adhesions of the right posterior pillar of the fauces to the back of the pharynx.” There were no bacilli in the sputum. He continued fairly well until October 1894, when he expectorated a large quantity of offensive purulent material for two consecutive days. Cough was very severe at this period. His health subsequently improved, and so remained until 20th February 1895; when in the course of a few days he brought up about a quart of blood-stained sputum. Hæmorrhage then ceased and dyspnoea diminished. On 5th March, cough and dyspnoea increased and he became seriously ill, with constant headache and slight delirium. Edema of the feet subsequently supervened. On 15th March 1895, he was admitted to the Brompton Hospital under the care of Dr. Percy Kidd. On admission he was reported to be fairly well nourished. He stated that he had not lost weight, and, beyond an occasional streak of blood in the sputum, there has been no hæmoptysis. There was marked stridor and severe cough. Right lung resonant everywhere; breath-sounds much exaggerated, expiration prolonged. Loud hoarse inspiratory and expiratory stridor all over the lung; sibilant rhonchi general. Left lung.—Expansion, much diminished; resonance much impaired front and back; breath-sounds weak; expiration prolonged; fine crackling rales over the whole of lung; vocal fremitus and resonance diminished. Expectoration profuse and difficult to expel. No tubercle bacilli found. Temperature, 99° F. It varied between that point and 96° F. during the time the patient was in hospital. The dyspnoea gradually increased, and death occurred on 10th April.

Necropsy.—Sears on tongue, glans penis, and scrotum; and adhesions of skin to left testis. Marked thickening of the right tibia. Larynx normal. Trachea narrowed at the lower end. Recent ulceration from cricoid downwards for two inches; below this, down to point of bifurcation, there was extensive scarring of the cartilaginous portion; and also at its line of junction with the posterior wall. The submucous tissue was extremely thickened. Cartilages bare in several places. The main bronchi were much scarred and showed extreme narrowing. The bronchus to the left upper lobe was impermeable to a probe. Right lung.—Old pleural adhesions over the upper lobe, recent pleurisy with effusion at the base. Emphysema, with reticular fibrosis especially around bronchioles. Deep in the upper lobe at the edge of one of the main bronchi there was a large black fibroid mass, with fibroid radiation into
the surrounding tissue; elsewhere two small, hard, raised masses, one with fibrous strands, running up to it. Base solid from broncho-
pneumonia. No appearance of tubercle. Left lung.—Upper lobe extremely contracted, containing no normal tissue. It consisted of deeply pigmented blackish gray fibrous tissue surrounding the openings of bronchial tubes, and bronchiectasis. At the centre there was a smooth-walled cavity about the size of a small chestnut into which a bronchus opened. No appearance of tubercle. Lower lobe.—Emphysematous, with reticular fibrosis along the margin and at the base. Bronchi dilated, but not to a marked degree. About the centre point of the outer margin there was a small nodule, probably a gumma; white and firm, and surrounded by a pigmented fibrous capsule. The extreme base consisted of indurated fibrous tissue extending from the pleura to a cavity, the size of a marble, into which a small bronchus opened. From this cavity fine fibrous bands radiated in all directions, producing extensive fibrosis of the surrounding lung. Perihepatic and splenic adhesions. Liver scarred and nutmeg. Spleen contained several calcareous masses surrounded by a fibrous capsule. Testes fibrous.

Case II.—R. D., aged 36; coachman. The family history is unimportant. At the age of 18 he had a sore on the penis, for which he was treated for several months with medicine and a lotion. In 1890 he became an out-patient under the care of the writer at the Brompton Hospital; he was suffering from cough and expectoration, which continued. There was an enlarged gland in the inferior triangle of the neck on the right side, dulness at the right apex, with feeble breath-sounds, and bronchial breathing in the right supraspinous fossa. The liver was large, nodular, and very tender. He took iodido of potassium in gradually increasing doses and obtained some relief. He was subsequently an out-patient at the Middlesex Hospital. In March 1893 he caught a severe cold, but remained at work. In following April he noticed oedema of the legs and scrotum. He was admitted into Middlesex Hospital, under Dr. Cayley, on 13th May 1893. He was pale and emaciated, the legs and scrotum were oedematous. He had troublesome cough, accompanied by the expectoration of large quantities of extremely fetid pus. The breath was fetid.

Physical signs.—Expansion deficient on right side. Relative dulness at right apex front and back, breath-sounds feeble over dull area. Absolute dulness from level of fifth interspace in nipple line and in axilla to base; behind from angle of scapula to base. Vocal fremitus and resonance diminished, and breath-sounds scarcely audible over dull area. Left side normal. No displacement of heart. Hepatic region prominent. Liver dulness extended 3 inches below the costal arch in right mammary line and almost to umbilicus in middle line. Liver somewhat soft and elastic. Urine, sp. gr. 1004, neutral, contained albumin and fatty casts. The expectoration consisted of frothy greenish pus, forming thick masses in a watery fluid. It contained no tubercle bacilli.

17th May.—The chest was explored in the axillary and submammary
region. No pus was found. 19th May.—Liver exposed, by incision below costal arch, and a depressed cicatrix seen. The expectoration continued copious, green, and fetid. Absolute dulness appeared over whole of right side up to clavicle, with amphoric breathing and pectoriloquy below clavicle. 15th June.—Offensive pus was evacuated through a canula inserted in third right interspace in mid-axillary line; a portion of the fourth rib resected, lung incised, more pus evacuated, drainage-tube inserted. 19th and 20th June.—Hæmorrhage from wound. 21st June.—Death.

Necropsy. Abstract of notes:—Old syphilitic scar in trachea, six rings above bifurcation, more recent scar at bifurcation, producing stenosis of the main bronchi to the right upper and lower lobes. One bronchial gland enlarged. Pleura over right lower lobe adherent and much thickened. Bronchi much dilated beyond the site of stenosis. At the base of the upper lobe were two large irregular cavities with sinuous outlines communicating with large bronchi, lined by a distinct membrane, and containing sloughy portions of lung tissue. The anterior cavity had been opened by the incision. The section of the lung was smooth and presented a finely speckled yellow appearance. No pus exuded from the yellow spots on pressure. In the anterior part of the lower lobe there was a large irregular cavity, the walls of which showed no sign of any mucous membrane; they were covered with yellowish gray sloughy material. No tubercle present and no caseation. The lung puckered in many places and fibrous almost throughout. Liver enlarged (76 oz.). Large puckered cicatrix on the upper surface of the left lobe and many similar cicatrices elsewhere. A cretaceous and caseous gumma on the posterior aspect of the right lobe. Liver substance fatty and amyloid. Kidneys large, pale, lardaceous, and fatty.

Case III.—T. H., set. 59; painter. Admitted into Middlesex Hospital under Dr. Fowler, 4th February 1893. Father died aged 70; mother aged 75. No history of tuberculosis in family. Accident to left knee set. 19 years, followed by formation of an ulcer. Chancre on penis in 1858 (set. 25), secondary rash and sore throat subsequently. In 1864 ulcers on left leg and twice subsequently. In 1880 ulcer on right leg, near external malleolus. Dry cough since 1887, worse in winter. Since December 1892 severe paroxysmal cough with offensive muco-purulent expectoration. Marked emaciation during this period.

A pale, gray-haired, emaciated man. Breath very fetid. Extensive scars on left leg of old standing, more recent scars on right leg. Scar in right lumbar region where incision was made for "abcess." Right lung.—Hyper-resonant on percussion; breath-sounds at apex bronchial, front and back; crackling rales in supraspinous fossa. Bronchophony and pectoriloquy well marked in same area. Dulness over lower lobe to angle of scapula, breath-sounds bronchial, with coarse crackling rales over same area. Left lung.—Resonance impaired over clavicle and in supraclavicular fossa, elsewhere hyper-resonance. Bronchial breathing over upper lobe, front and back, with crackling rales. Breath-sounds bronchial over upper part of lower lobe, with bubbling and coarse
crackling rales, the latter extending to the base. Urine, sp. gr. 1020; no albumin. Expectoration copious, purulent, and offensive. Frequent examinations made for tubercle bacilli, but none found. No elastic tissue found. Temp. 98°, pulse 84, respirations 44. • 21st February.—Dulness at both apices, and medium crackling rales. Temperature between 99° and 100° F. The respirations between 36 and 48. Severe cough, and the breath and expectoration offensive. Died 23rd February.

Abstract of P.M. notes:—Scar on corona of penis with some induration around. Calvarium thickened, dura mater adherent. Pleural adhesions over both lungs. Right lung.—Emphysema along anterior margin and at base. Apex pigmented and consolidated from pneumonia and edema. In lower part an oval cavity measuring 2½ inches by 2 inches, in communication with main bronchus, and containing greenish yellow, offensive, shaggy material. Below this for 1½ inches the lung gray in colour and almost solid, a few small cavities with curdy contents. No tubercle found. The pleura covering the consolidated area much thickened. Left lung.—Upper lobe pigmented and "nodular." A cavity, from bronchial dilatation, occupies the posterior portion. The lower lobe emphysematous, and contained numerous encapsulated caseous masses about 2 mm. in diameter. Bronchial glands pigmented, but not caseated. No ulceration in air-passages. No gummatas in liver or spleen. Testes scarred and fibrous. Small white fibrous nodule in right kidney.

The following cases are incomplete, and do not attain to the standard of evidence laid down, inasmuch as the patients are believed to be still living:—

Case IV.—Mary G., 23, married. Three children alive, three dead,—one still-born, one died a few hours after birth. Has had four miscarriages. Admitted into the Brompton Hospital, 13th June 1894, under Dr. Fowler. No history of tuberculous disease in the family. Ten years ago had some affection of the liver. Three years ago had an attack of influenza followed by pleurisy (R) and congestion of the lungs. Right pleurisy recurred in August 1893. Has had a slight cough for three years, worse since September 1893. Expectoration has been profuse, and for the last two months fetid and of a bitter taste. In October 1893 it was tinged with blood for three weeks. Dyspnœa worse since September 1893. Catamenia ceased since the birth of the last child on 30th September 1893, at which time she caught a chill. In February 1893 patient noticed a swelling in the left loin, which at first gradually increased in size and subsequently diminished. It is slightly movable and is not tender. It is about equal in size to a small Tangerine orange, is situated rather superficially, and over the erector spinae muscle; whether actually within the muscle cannot be determined. Emaciation, cough, and weakness have been increasing lately, and night-sweats have been continuous.

Physical signs.—Right lung.—Marked flattening of the whole of the right side, particularly in front. Measurement at right nipple level:
right 14½ inches, left 16 inches. Dulness over upper lobe, with distant cavernous breathing and bronchophony front and back. Impaired resonance over upper part of lower lobe posteriorly, where crackling rales are audible; similar rales at the right base where percussion note is dull. Left lung.—Harsh breathing general (? compensatory), no adventitious sounds. Liver much enlarged and nodular on the surface; margin irregular, extends from the fourth space below the umbilicus. Spleen not enlarged. Urine free from albumin. Expectoration profuse and fetid. No tubercle bacilli. From June to September the expectoration was usually fetid. Bacilli repeatedly sought for, but never found. The cavity at the right apex extended. 5th September.—Retraction more marked at right apex. Cavity dry. Numerous crackling rales in axilla, and all over base. General improvement. Liver appears more nodular. October.—Large crackling rales over base and in axilla. Cavity at apex dry. No bacilli to be found. Discharged 13th October. Intra-tracheal injections of menthol appeared at first to have an effect in diminishing and then removing the odour of the expectoration; but subsequently the fætor returned and appeared to be uninfluenced by their continued use. The quantity of expectoration was small during the period over which their administration extended; but it had been steadily diminishing up to the time when this treatment was commenced. The patient considered that she derived benefit from the injections. The nature of the tumour in the back was doubtful, it was believed to be a gumma in the superficial part of the muscle. Inunction of mercurial ointment was made daily into the back from 22nd September onwards.

Case V.—Edward C., set. 47; a waiter. Admitted into St. George's Hospital, 13th April 1894, under Dr. Whipham.¹ His father and mother died of "consumption." Thirty years ago he had a hard chancre. He has had syphilitic psoriasis of the palms. He has not had hæmoptysis, night-sweats, or emaciation. A fortnight before admission he was attacked with severe pain on the right side of the chest and dyspnoea. On admission he was anæmic, and complained of cough and profuse expectoration. The skin was of a brownish tint and presented numerous old rupial scars. Right lung.—Impaired resonance over upper lobe with feeble breathing. Just below the second rib there is a small area of increased dulness and cavernous breathing with whispering pectoriloquy. There are rhonchi all over the right lung and to a smaller extent over the left. The sputum is profuse and mucous-purulent. No tubercle bacilli were found on any occasion; the examinations were made by several observers. 15th April.—Ordered Potassii iodide gr. v., Liq. hydrarg. perchlor. 3j. ter die. 28th April.—Expectoration and cough less. Physical signs at right apex less marked. 2nd May.—Discharged to Convalescent Home.

The following case illustrates the fact, first pointed out by Dr. Pearson Irvine, that stenosis of a main bronchus may give rise to destructive

¹ The writer is indebted to Dr. Whipham for his kind permission to use the notes of this case.
changes in the lung. It will be observed that the case was one in which a recent tuberculosis supervened on old syphilitic disease:—

Case VI.—Margaret S., aged 25. Admitted into the Brompton Hospital, 25th June 1884, under Dr. Reginald Thompson. Family history good. Good health up to two years ago, when after marriage she had "ulcerated legs." No sore throat or skin eruption. Cough, expectoration, pain in left side, dyspncea, night-sweats and emaciation have been present for eight months. On admission the fingers were clubbed; there was a large circular ulcer on the back of the left thigh with some scarring, and coppery staining about the knee and leg on the same side. Cough more or less paroxysmal; expectoration copious, nummular, and purulent. No tubercle bacilli. Right chest 15½ inches, left 16½. Dullness over left lung, absolute at base, where vocal fremitus is absent; elsewhere it is diminished. Bronchial breathing, pectoriloquy, and crepitation over left upper lobe. Breath-sounds absent at base, some rhonchus there. Slight crepitation at right base.

The ulcer on the thigh yielded to antisyphilitic treatment. The physical signs remained much the same, except that the breath-sound at the left apex became cavernous. There was well-marked pyrexia throughout. The expectoration remained copious, at times it averaged a pint in the twenty-four hours. Death occurred on 1st March 1885, and was preceded by anasarca, ascites, and profuse diarrhoea.

Necropsy.—A few small scars in the subglottic portion of the larynx. The lower half of the trachea marked by numerous stellate puckered cicatrices, involving both membranous and cartilaginous portions, but especially the latter. The origin of the left bronchus represented by a small opening just admitting a probe; the surrounding parts of the tracheal wall extremely fibrous and puckered. Slight scarring in the right bronchus about the origin of the upper lobar branch. Left lung excavated from apex to base. Numerous trabeculated cavities in the upper lobe intersected by tough pigmented bands; walls thin and smooth. The cavities larger behind than in front, in the latter region they were more numerous; and the intervening fibroid induration was more pronounced. Some bronchi appeared to expand uninterruptedly into the smaller cavities. Numerous small cavities in the lower lobe situated in indurated fibroid lung. The cavities contained extremely fetid reddish fluid secretion, and in some places some soft putty-like material. No tuberculous nodules in this lung. The contents of the pulmonary cavities, including the liquid and caseous parts, were carefully examined for tubercle bacilli, but none could be found. Right lung crepitant, but studded with large tuberculous groups which were most plentiful in the middle lobe and lower part of the lower lobe. Lardaceous disease of thyroid, mesenteric and mediastinal glands, also of the kidneys, liver and spleen, and mucous membrane throughout the body.

The recent tuberculosis of the right lung was obviously quite unconnected with the disease in the left, which was secondary to the bronchial stenosis.
This case proves very clearly that a progressive destructive disease of the lung may result from syphilitic stenosis of a main bronchus; but it does not prove that this disintegration of the lung is due to the continued action of the specific virus of the disease, as is the case in pulmonary tuberculosis. The fact that lesions similar to those here described may occur when the narrowing of the bronchus is due to pressure from without, as by an aneurysm, shows that the bronchial obstruction is the main factor in their production. Stenosis of the bronchus is followed by retention of secretion in the tubes, and this by bronchiectasis. Decomposition of the retained secretion induces inflammatory changes in the surrounding lung, and finally the part so affected breaks down and cavities are formed.

The cases here described prove that in individuals undoubtedly the subjects of syphilis, widely-spread destructive changes may be found in the lungs; and that such lesions may occur independently of the presence of tubercle. Whether they are such as to entitle the condition to be named "syphilitic phthisis" must be decided by those who continue to use the word "phthisis," a term which many teachers have ceased to employ.

If the name "phthisis" is given to a group of symptoms and morbid changes, it can hardly be denied that a case (see Case I.) which is marked by such symptoms as severe cough, dyspnœa, emaciation, fever, night-sweats, profuse expectoration, and hæmorrhage, and which, on examination after death, is found to present signs of consolidation, fibrosis and excavation of the lungs, belongs to this category. The task before us, however, is to determine the nature of the pathological lesions of pulmonary syphilis and of the symptoms which they produce; whether they are such as to warrant the use of a vague nomenclature which it would be well to discard is a question of little importance. It may be of service, however, to draw attention to the chief points of difference between the pulmonary lesions of tuberculosis and syphilis.

I. Tubercle usually affects the apex of the lung, and subsequently the apex of the lower lobe; and tends to progress along a certain route. The primary lesion of syphilis is often about the root and central part of the lung; the disease follows no definite line of march, and gummata may be found in any position.

II. Both tubercles and gummata may undergo either necrosis and caseation, or fibrous transformation; but with caseous tubercle the tendency towards softening and cavity formation is the rule, whereas a caseous gumma very rarely breaks down.

III. The progressive destruction of the lung by a process of disintegration leading to a gradual increase in the size of a cavity, a change so commonly observed in tuberculous disease, is rarely if ever observed in syphilis, except as a secondary result of stenosis of one of the main bronchi.

IV. In nearly all cases of advanced destruction of the lung, occurring in the subjects of syphilis, stenosis either of the trachea or of one of the
main bronchi is present, whereas this lesion is very rare indeed in tuberculosis.

V. The cavities found in cases of pulmonary syphilis are usually bronchiectatic, but not invariably so; whereas in tuberculosis they are commonly due to progressive destruction of the lung, but may be bronchiectatic.

VI. The tendency to the formation of pulmonary aneurysms, which is so marked a feature in tuberculosis, is rarely observed in pulmonary syphilis.

VII. Pulmonary lesions in tuberculosis are very common, whereas in syphilis they are extremely rare.

The necessity for prolonged specific treatment is certainly more generally appreciated now than formerly; and it is therefore probable that rare as these lesions have been in the past, they will be still rarer in the future. The conditions which favour their development are the neglect of mercurial treatment shortly after infection, and anything which, by lowering the general health, tends to diminish the resisting power of the individual.

When our knowledge of the virus of syphilis is as complete as that we even now possess of the bacillus tuberculosis, it may be possible to state definitely whether the destructive pulmonary lesions found in advanced cases of the acquired disease are directly due to the continued action of a specific micro-organism; at present the problem remains unsolved.

Symptoms.—The only point worthy of mention in respect of syphilitic lesions of the bronchi is that the catarrhal signs which accompany the secondary stage are, as a rule, general in their distribution; whilst in the tertiary stage they are more often localised, owing to the tendency at that period to the formation of gummata in the main bronchi. Should stenosis occur, there may at first be bronchial breathing limited in area, and often most marked about the root of the lung posteriorly. As the lumen of the tube diminishes, the breath-sounds, over the pulmonary area which it supplies, become more and more feeble, and finally disappear when air ceases to pass the obstruction. If bronchiectasis is forming behind the site of stenosis there may be cough with profuse, purulent, and fetid expectoration, accompanied by general signs such as emaciation and moderate pyrexia.

In the cases described in this article it will be observed that cough was, as a rule, the earliest and most prominent symptom. In the early stage it may be due to irritation, the result of laryngeal, tracheal, or bronchial lesions; at a later period it is probably chiefly due to the changes within the lung itself.

Dyspnoea comes next in point of frequency. It varies in severity with the nature of the lesion: slight when this is limited, in cases of extensive fibrosis or stenosis of one of the main bronchi it may be very severe. The dyspnoea tends to become paroxysmal and to assume the characters of bronchial asthma. Haemoptysis has not been of frequent occurrence in
cases observed by myself, but it may occur and may prove fatal. In one case of syphilis of the bronchial glands, profuse and fatal hæmorrhage occurred from softening of the gland and its rupture into a main branch of the pulmonary artery.

Expectoration may be profuse, purulent, and offensive. Fæctor of the expectoration is common in cases of advanced pulmonary syphilis. The sputum will be free from tubercle bacilli.

Pain may be present, but is not a very prominent feature of the disease.

Emaciation is not, as a rule, nearly so extreme as in tuberculosis; but with advanced lesions in the lungs the difference is not so remarkable as to be of any value from a diagnostic point of view.

Night-sweats were present in several of the cases here described.

When extensive lesions are present, pyrexia may be considerable, and of the hectic type commonly observed in tuberculous disease of the lungs; but in the early stages of the disease there may be a complete absence of fever.

The general symptoms, as will be seen on reference to the cases described, do not, in the presence of widely-spread lesions, differ markedly from those of advanced tubercular disease of the lung.

Physical signs and Diagnosis.—The lesions of syphilis are rarely of such a kind as to produce signs by which they can be distinguished from others of an entirely different origin.

Consolidation and excavation will be recognised by their ordinary signs, probably before their syphilitic origin is suspected; and it appears therefore unnecessary to describe them in detail, more particularly as in the cases here recorded the results of the physical examination are given in full.

The features of pulmonary syphilis are certainly not as yet so clear that the disease can be recognised by any positive signs; but by a process of exclusion a diagnosis may generally be made.

The case will probably be regarded at first as one of pulmonary tuberculosis; but repeated examination of the sputum and the failure to discover tubercle bacilli will suggest another origin.

A careful inquiry, previously perhaps omitted, will now be made as to syphilitic infection and as to the occurrence of any secondary or tertiary manifestations of this disease. The absence of such a history in a hospital patient will not exclude syphilis; but it is rare in private practice for a patient to have had syphilis with tertiary symptoms and to be ignorant of the fact.

Evidence of tertiary lesions in the larynx, liver, spleen, or testes is of importance as showing that the viscera are affected.

Careful search should also be made for lesions of the calvarium, of the dura mater, and of the sternum and ribs.

Speaking generally, the diagnosis of pulmonary syphilis from tuberculosis will depend far more upon the examination of the sputum than on the results of physical examination.
SYPHILITIC DISEASE OF THE LUNGS

A careful examination of undoubted specimens of pulmonary syphilis does not bear out the statement that the lesions are generally limited to the middle part of the lung; they are so often found elsewhere that their more frequent occurrence in that part ceases to be a fact of much value in diagnosis. It would be rash indeed to diagnose pulmonary syphilis because of a lesion situated in and apparently limited to the middle of one lung, without having previously demonstrated, by frequent examinations, the absence of tubercle bacilli from the expectoration. Such points, however, are not without importance, as being unusual in a case possibly hitherto regarded as one of "phthisis" or "consumption," they may serve to arrest attention.

Evidence of excavation and the expectoration of a fetid sputum, which does not contain tubercle bacilli, should always suggest the possibility of pulmonary syphilis. When the physical signs indicate stenosis of the trachea, or of one of the main bronchi, and the presence of a growth or an aneurysm can be excluded, it is very probable indeed that syphilis is the main factor in the case.

Those who are content to diagnose "phthisis," and neglect the systematic examination of the sputum, will almost certainly overlook a case of pulmonary syphilis if it should come in their way.

A striking example of this has recently come under my notice. A military officer who had contracted syphilis some years back began to suffer from symptoms of laryngitis; and on examination of the chest well-marked signs of disease were found at the apex of the right lung. The laryngoscopic appearances did not suggest to several competent observers that the lesion was due to syphilis, and the case was regarded as one of "consumption of the throat and lungs." It occurred to a medical man who saw the patient at a later period to examine the sputa for tubercle bacilli, and, as none was found on repeated examination, doubt was cast upon the diagnosis of "phthisis"; mercury and large doses of iodide of potassium were prescribed, and the patient rapidly improved; but the stenosis of the larynx remained.

Prognosis. — Extensive pulmonary lesions, particularly excavation whether of bronchiectatic or disintegrative origin, and fetid expectoration are certainly very grave complications of syphilis. If, moreover, there is evidence also of gummatous hepatitis, albuminuria, and lardaceous disease, recovery is scarcely possible, and life is not likely to be much prolonged.

It is probable, however, that, with our present improved means of diagnosis of tuberculosis of the lungs, syphilitic cases, which formerly would have been considered tuberculous, may be recognised as syphilitic at an earlier stage, and the patients under appropriate treatment may recover. In an undoubted case of pulmonary syphilis, which came under my own care at a late stage of the disease, the affection had been kept in check for many years by repeated visits to Aix-la-Chapelle, and by the active employment of antisyphilitic treatment. In any case seen in an early stage, great improvement, if not complete cure, may reasonably
be expected from the use of similar measures. There are, however, limits to the action even of specific remedies; and it is not to be expected that lesions such as bronchial stenosis and dilatation, extensive fibrosis and excavation, or gummatas in a state of fibrosis will disappear under the administration of mercury or iodide of potassium.

Treatment. — If the disease in the bronchi or lungs is recognised in an early stage, the patient should be advised to undergo a prolonged course of treatment with mercury. Iodide of potassium in gradually increasing doses is generally administered at the same time.

If, however, the disease is advanced, and the patient emaciated, it is better first to try the effect of iodide of potassium alone; giving at the same time cod-liver oil and tonics. To maintain and improve the strength and general nutrition of the patient are matters of as much importance in the treatment of syphilitic as of tuberculous disease of the lungs, and are to be secured by the same means.

The warm sulphur baths of Aix-la-Chapelle, in association with mercurial inunction, enjoy a special reputation in the treatment of syphilis, and are to be recommended to sufferers from pulmonary syphilis who are able to go abroad for treatment.

When tuberculous disease of the lungs occurs in a syphilitic subject, the treatment will be mainly such as is suited to cases of tuberculosis. A mercurial course is rarely admissible, but iodine, in the form of the syrup of the iodide of iron, may be given with advantage.

In cases accompanied by fetid expectoration, creasote vapour baths and intra-tracheal injections of guaiacol should be tried.

Cases of syphilitic disease of the lung accompanied by bronchiectasis have not, in the experience of the writer, been benefited by surgical measures undertaken with a view to drain the cavities.

J. K. Fowler.

REFERENCES


J. K. F.
DISEASES OF THE PLEURA
INTRAPLEURAL TENSION

In health the two layers of the pleura are in close contact, but they are subject to a constant strain, which tends to separate them; this is called the intrapleural tension. It is for all practical purposes equal to the elasticity of the lung, but opposite in direction; and thus the elasticity of the lung is positive and the intrapleural tension negative.

Whether in health there is any force existing between the layers of the pleura—such as that of cohesion, as I suggested some years ago (1), which neutralises the elasticity of the lung when fully expanded—is a matter which is open to question. It is possible, and I think it probable; but the question need not be further considered here.

The forces, of which the intrapleural tension is the resultant, are, first, the rigidity of the chest walls; secondly and chiefly, the elasticity of the lungs; and, thirdly, the movements of respiration.

So far as the condition of the chest walls is concerned, where they are fairly rigid, as in the adult, this factor may practically be disregarded; but not so in infants or little children, in whom the chest walls are soft and yielding; for then, under pathological conditions, part of the force which would otherwise tend to separate the two layers of the pleura is spent in drawing the chest walls in.

The condition of the chest walls and the elasticity of the lungs cannot vary while observations on intrapleural tension are being made; but the third factor, namely, the movements of respiration, is one which is constantly varying, and introducing variations in intrapleural tension which have to be reckoned with. Thus during inspiration the lungs are placed more on the stretch, and consequently the intrapleural tension is greater; during expiration the lungs are less on the stretch, and the intrapleural tension is therefore smaller.

If the air in the tubes were stationary, as it is after death, the pressure in the air-tubes would be that of the atmosphere; but, during respiration, the air, as it passes in and out through the air-tubes, meets with some obstruction, which on inspiration amounts to about half a millimetre of mercury, and on expiration from 2 to 3 millimetres. Thus an oscillation in pressure is produced during the different phases of respiration, which amounts to 2 or 3 millimetres of mercury; that is, 1½ to 2 inches of water. This is called the respiratory oscillation.
If the movements of respiration were left out of account, the intrapleural tension would be equal to that of the atmosphere, minus the elasticity of the lungs; that is to say, it would always give a negative reading on the manometer. It would then be equal to the elasticity of the lungs with the sign changed, that is, $-6$ to $-8$ millimetres of mercury.

During ordinary respiration the intrapleural tension is also negative throughout; for, if it is negative when the air is stationary, it will be more negative still on inspiration, the lung being more on the stretch; and during quiet expiration, even when from the normal elasticity of the lung the $2\frac{1}{2}$ to $3$ millimetres of mercury be deducted which represent the obstruction in the tubes to which the air is subject on expiration, there are still left $4$ to $5$ millimetres of negative pressure.

During violent expiration, of course, the pressure may rise considerably, even to so much as $70$ to $100$ millimetres of mercury (3 to 4 inches); but it must be remembered that under normal conditions this pressure does not fall directly upon the pleura, but is immediately supported by the chest walls. Under pathological conditions, on the other hand, when the two layers of the pleura are not in contact, but are separated by air or by fluid, pressure of any kind will make itself felt directly by the contents.

There are two methods of determining the value of the intrapleural tension. 1. In the one the elasticity of the lung is determined, and the result, with the sign changed, is transferred to the pleura; 2, in the other the intrapleural tension is estimated directly by means of a trocar introduced between the layers of the pleura.

In man both these methods of investigation are available after death, but the latter only during life, and this under pathological conditions.

In either case the reading is made upon a mercury- or water-manometer. Water has been more commonly employed, because the oscillations are larger and are more easily read; but the conversion is easily made from the one to the other: thus 1 inch is equal to 25 millimetres, and 1 millimetre of mercury is approximately equal to half an inch of water or $12\frac{1}{2}$ millimetres of water.

Intrapleural tension is often spoken of as "intrapleural pressure," and thus confusion is introduced both in thought and in expression. This confusion will be avoided if it be remembered that the values stated are not actual pressures but readings on the manometer. For instance, if the pressure in the pleura were equal to that of the atmosphere it might be called 1, but as this would be indicated on the manometer by the position of equilibrium which is marked zero, it is usually spoken of as zero; 1, 2, or 3 inches or millimetres would then represent 1, or 2, or 3 above or below the atmospheric pressure, as the case might be.

The elasticity of the lung was estimated by Donders to be from 6 to 8 millimetres of mercury; this, therefore, with the sign changed, would represent the intrapleural tension.

An important series of observations of a similar kind was made by
Perls (2). After a tube connected with a manometer had been fixed into the trachea, first one pleura and then the other was opened and the pressures registered. The observations were made upon the dead body of a man under a variety of different conditions, and the results are very interesting.

Seeing how closely intrapleural tension is connected with the elasticity of the lung this will be the natural place to consider various pathological conditions under which the normal elasticity of the lung is altered.

When one pleural cavity is laid freely open to the air there will then be atmospheric pressure on both sides of the visceral pleura; the elasticity of the lungs will come into play, and the exposed lung will collapse. But this is not all, for the alterations in pressure do not affect the one lung only; the mediastinum being not a fixed partition, but a movable one, the elasticity of the opposite lung also comes into play; with the result that the mediastinum and the organs therein are drawn over to the sound side. Thus it follows that the opening of one pleura not only satisfies the elasticity of the one lung, but goes a long way to satisfy the elasticity of the other. If, for example, the pressures be reduced to figures, and we assume for the sake of illustration that in a healthy man the total elastic contractility of the two lungs together amounts to 50, the opening of one pleura may satisfy this elasticity to the extent of 40, leaving only 10 for the unsatisfied elasticity of the opposite lung.

Thus, in pneumothorax, which is the corresponding pathological condition, if the lungs are healthy and their elasticity at its maximum, the total respiratory capacity will be suddenly reduced by four-fifths. If, however, the lungs be previously diseased or the pleura adherent, the elasticity of the lungs will be either reduced or prevented from coming into play; and thus the change in respiratory capacity consequent on the pneumothorax will not be so extreme. For these two reasons it is evident why the sudden admission of air to the pleura should produce more severe results in a healthy person than in one whose chest has been previously diseased; and a clinical paradox is explained.

Where the pleura is completely adherent the elasticity of the corresponding lung may be almost abolished; but it is frequently retained, though of course when retained it is unable to come into play. Under these conditions the opposite lung often becomes greatly enlarged. This has often been called "complementary emphysema," but in these cases the elasticity of the enlarged lung is not diminished, as in ordinary emphysema, but actually largely increased, so that the elasticity of that one lung may be almost equal to the combined elasticity of two healthy lungs. Thus it is made evident that this condition is not emphysema, but hypertrophy, as there are also the best of clinical reasons for maintaining. It should therefore be called, not complementary emphysema, but complementary hypertrophy.

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There is good ground for believing that the contractility of the lung is not simply elastic, but is due in some measure to the muscular fibre with which it is so richly provided. If that be so, we may fairly speak of "pulmonary tone" in the same way as we speak of "vascular tone"; and we may expect it to vary not only with local conditions of nutrition in the lung, but also with defects of nutrition which are general.

Thus, in various local affections, of which pneumonia is the most important, Perls found the elasticity of the lung greatly reduced; as well as in general diseases without any local affection of the lung, as for example in typhoid fever, delirium tremens, erysipelas, phosphorus poisoning, and after severe haemorrhage.

If, then, pulmonary tone be not simply elastic in origin, but in part neuro-muscular, the loss of it may be met with under two different clinical conditions: first, as the result of general causes—as an evidence, for instance, of general neuro-muscular failure; and, secondly, as a result of local nutritive-disturbance.

As a neuro-paralytic phenomenon it might be placed in association with the like condition in the abdomen (acute tympanites), which in the same way may be due to general or local causes. For example, just as in pneumonia, acute abdominal tympanites may suddenly manifest itself—a phenomenon of fatal significance; so with typhoid fever, or any other specific fever, a similar condition may appear in the lung which is likewise of fatal import.

The loss of pulmonary tone is indicated during life, just as it is after death, by change in the percussion note; the resonance becoming more tympanitic and of that character which is generally known under the name of "skodaic resonance." Without any local disease of the lung, I have on several occasions seen this acute pulmonary tympanitic set in; whatever the explanation of its occurrence, there is no doubt as to the existence of the condition.

Where there is local disease in the lung, the other parts of the lungs, as is well known, frequently yield a tympanitic percussion note. There are several conditions under which this is met with: the commonest and easiest to explain is that which occurs with pleural effusion, when the lung floating on the fluid yields this skodaic resonance. The conditions and the percussion note are the same as are presented by the lung removed from the body.

With complementary emphysema, where one part of a lung is diseased and the other parts proportionately distended, similar hyper-resonance is obtained. In this case the hyper-resonance is due to the over-distension of some of the air-vesicles.

But besides these there is another condition which requires a different explanation. Nothing is commoner in pneumonia than to find the parts of the lung above or in front of the affected portion yielding a highly tympanitic note; yet the pneumonic portions of the lung are certainly not collapsed or smaller than they should be, nor are they much larger: thus neither of the explanations just given
is applicable; the part of the lung where the hyper-resonance is obtained is not collapsed on one hand, nor over-distended on the other. This condition, it appears to me, can only be explained on the assumption of loss of lung-tone of neuro-paralytic origin and dependent on nutritive disturbance. This view also obtains support from some of Perls' observations, for among his cases are several instances of pneumonia as well as some of embolism and gangrene; and in all of them the elasticity of the lung was very greatly reduced.

It is possible that the elasticity of the lung diminishes after death, but there are no direct observations to prove this. We may assume at any rate that for some hours after death the elasticity of the lungs is not materially affected.

In estimating the elasticity of the lung and the intrapleural tension, the condition of the abdominal muscles and of the diaphragm must not be overlooked. We have to reckon on the one hand, during life, with their respiratory action, and on the other, after death, with rigor mortis; but it is not necessary here to do more than refer to these complicating factors.

**THE PLEURAL CAVITY UNDER PATHOLOGICAL CONDITIONS**

Under pathological conditions the two layers of the pleura may be separated either by air or by fluid, and each of these presents its own peculiarities and difficulties: thus, fluid has weight, but is practically incompressible; air is compressible, but its weight may be disregarded. With fluid, therefore, the height of the column above the point of the trocar will affect the manometer readings, while with air the position of the trocar is immaterial. As in many respects the problem is simpler in the case of air than of fluid, it will be well to begin with pneumothorax.

**Intrapleural tension in pneumothorax.**—Air may gain access into the pleura either from without through the chest walls, as by a wound, or internally from the lung; and in both cases we have to consider, first, the condition in which the air enters more freely on inspiration than it finds issue on expiration, and, secondly, the condition in which there is no abnormal obstruction either on inspiration or on expiration.

A. Where the air finds entrance through the chest walls.

(i.) **By a punctured wound.**—In this case, where the wound is a small one and merely a puncture, though the lung be injured the air does not, as a rule, find access to the pleura, but crosses the pleura and reaches the subcutaneous tissue. The reason of this is very difficult to find, but of the fact there is no doubt; it need not, however, be considered here.

(ii.) **Where the opening is a small one, so that the air finds easier entrance than it finds exit.** This condition will be the same as that in which the air gains access to the pleura through the lung, and will be better considered later.

(iii.) **Where there is a large opening through the chest walls, at least as large as the cross-section of the trachea.** The air then enters and leaves
the pleura without obstruction, that is to say, the pressure on both sides of the visceral pleura is the same—namely, that of the atmosphere—during all phases of respiration. Under these circumstances the elasticity of the lungs comes simply into play, so that the lungs collapse.

It is no doubt true, as Donders said, that in course of time under these circumstances the lungs will become completely collapsed by virtue of their own elasticity; yet we have daily experience that this does not usually occur, and when we consider the matter the reason is clear. It is found in the fact which has already been stated; namely, that the air in the tubes is not subject simply to the atmospheric pressure during the phases of respiration; on inspiration it is under a pressure somewhat less than the atmosphere (by half a millimetre of mercury), and on expiration under a pressure above that of the atmosphere (to the extent of 1 ½ to 2 millimetres of mercury).

During expiration, therefore, the lungs will always be subject to the distending force of 1 ½ to 2 millimetres of mercury. There are no observations to show how far the lung will be expanded under such a pressure, but it cannot well be less than a half, and is probably more; at any rate we have daily demonstration of the fact that the lungs do not collapse completely as the result of opening the side: on the contrary, on opening the side for empyema it is a common experience to find the lungs which have been completely collapsed by the effusion expand again as soon as the pus is evacuated, so as to reach close to the chest walls immediately after the operation. This may at first be the result of the violent respiratory efforts or of the coughing which very frequently follows the operation; but this is not the only explanation, for it occurs when there is no violent expiration or coughing, or persists when they have passed off.

Two cases which I have recently recorded are of interest in this respect, because the lung had been compressed by fluid for a long time—eighteen months and five months respectively, one being a case of serous effusion and the other of pyopneumothorax; in both, immediately after the operation, the lungs were close to the chest walls, and within a week had come into close contact with it everywhere except just round the incision (3).

B. Where the air gains access to the pleura from the lung.

(i.) Theoretically it is possible that the opening through the lung should be large enough for the air to pass freely in and out during inspiration and expiration without obstruction; yet this is a condition which can hardly ever arise, and almost all the cases of pneumothorax therefore come into the second category.

(ii.) That in which the opening through the lung is of such a kind that though the air gains free entrance into the pleura during inspiration it cannot find free issue from it during expiration. The result of this is that during expiration the pressure rises and compresses the lung, which gradually becomes more and more collapsed. Although it is true that the mediastinum may be displaced to the maximum and the lungs
be completely collapsed in cases where there is no expiratory compression, still in the great majority of cases this rise of pressure during expiration plays a very important part in the production of both these phenomena.

The division of pneumothorax into open, closed, and valvular, interesting as it is in some respects, is of no practical importance from the present point of view—that of intrapleural tension; for in a case of recent pneumothorax as soon as the lungs are completely collapsed the hole becomes closed, whether it be permanently sealed or not. During the early stages pneumothorax is always more or less valvular; in other words, the air finds easier access during inspiration than it finds issue during expiration.

The intrapleural pressures during inspiration and expiration require, in the case of pneumothorax, to be considered separately.

1. The inspiratory pressure.—When the lung has ruptured, air finds access to the pleura during inspiration so long as the pressure in the pleura is below that of the pressure in the air-tubes; that is, below the atmospheric pressure:—although this has to be reduced, as already stated, by half a millimetre of mercury, being the value of the obstruction which the air meets with on its way into the lungs. The inspiratory pressure, therefore, can never rise in pneumothorax above that of the atmosphere except under one condition, namely, that in which there has been much dyspnoea; for as then the inspiratory efforts are considerable the air will consequently continue to enter the pleura as long as the pressure at the end of each inspiration is below that of the atmosphere and until it equals that of the atmosphere, after which no more air can enter. It follows, therefore, that if the patient survive and the dyspnoea pass off, the inspiratory pressure might be above that of the atmosphere to the extent of the difference between the pressure on deep inspiration and the pressure on ordinary inspiration. This is not very much, and in all probability the excess of air, which represents the difference of pressure, is rapidly absorbed.

In ordinary simple pneumothorax the inspiratory pressure is therefore not, as a rule, above that of the atmosphere. If it be, some other factor is required to account for it, and this almost without exception proves to be the presence of fluid; we may therefore conclude that whenever the inspiratory pressure is much raised we shall probably find that fluid is present as well as air.

2. The expiratory pressure.—The expiratory pressure in pneumothorax is always positive. It is true that the mediastinum may be displaced to its maximum in a case where the pressure in the pleura is zero. Still the raised expiratory pressure tends to make the displacement extreme or to produce it more rapidly; while, as already stated, it is the expiratory pressure which probably chiefly accounts for the complete collapse of the lung.

3. The respiratory oscillation.—As this is the difference between the pressure on inspiration and the pressure on expiration it will vary according to the amount of dyspnoea or the violence of the respirations at any given time.
It might be thought, considering the violence of respiration in many of these cases, that the respiratory oscillations would always be considerable. As a matter of fact this is not found to be so, and a little consideration will show why this is the case; on the affected side the chest is in a condition of maximum inspiratory expansion and cannot alter from this on expiration; while on the opposite side the lung is prevented from expanding fully by the amount of the reduction of its volume on the displacement of the mediastinum and the organs connected with it; thus its elasticity also is reduced, being, as already stated, partly satisfied. It is evident, therefore, that the total respiratory excursion of the chest will be very considerably diminished and the respiratory oscillation therefore small.

In a recently published paper (4) I have recorded a series of observations upon the pressures in pneumothorax in eleven cases, some of which were tapped several times; so that there are records of twenty different paracenteses.

The inspiratory pressure varied from zero to +9, the several pressures being 0, $\frac{1}{2}$, 1, $1\frac{1}{2}$, 2, 2$\frac{1}{2}$, 4, $4\frac{1}{2}$, 5, 6, 6$\frac{1}{2}$, 6$\frac{3}{4}$, 7, 8$\frac{1}{4}$, 8$\frac{3}{4}$, 9.

In two cases the inspiratory pressure was that of the atmosphere; that is, the reading of the manometer stood at zero. In both of these cases fluid was present as well as air. From this it is evident that as soon as the fluid formed the air must have been absorbed, since the opening into the lung in both cases was closed.

In another case the inspiratory pressure, after having been in the two first paracenteses positive, fell in the last two to zero; and the change in pressure was due to an opening of considerable size having formed into the lung.

In all the other cases the inspiratory pressure was positive, and fluid (sometimes pus, sometimes serum) was present as well as air; thus the statement already made is confirmed, namely, that when the inspiratory pressure is much above that of the atmosphere the conclusion may be drawn that fluid is present as well as air.

It is no matter of wonder that the inspiratory pressure rises when fluid forms; but it is surprising that the pressure is not much higher than we find it. The highest pressure that I observed was nine inches of water, but pressures as high and even higher have been met with in serous effusions. It follows, therefore, that when fluid forms in pneumothorax a large amount of the air present must be absorbed as the fluid forms.

We know, both as the result of experiments on animals and of operations upon man, as well as from observations of pneumothorax in man, that air may be very rapidly absorbed from the pleura.

Even when fluid is present the pressure may not be above that of the atmosphere, as we have already seen; and I think we may possibly even go so far as to say that if the intrapleural pressure remains unusually high in pneumothorax it may be taken as an indication that there is extensive disease both of the lung and the pleura; so that the
absorption of air which would ordinarily occur is prevented from taking place.

The expiratory pressure also varied considerably from zero up to 13 1/2, the actual figures being 0, 0, 1, 1 1/4, 2 1/4, 2 1/2, 4 1/2, 5, 7, 8, 8 1/2, 9, and 13 1/2. The highest expiratory pressures are, as already stated, due to dyspnea; that is, to violent expiratory efforts.

The respiratory oscillations in the same way showed great variations, and fluctuated from zero up to 8. The largest were 8, 6 1/2, 6 1/4, 6, and 4. In all these cases there was dyspnea, and the large respiratory oscillation was the result of the high expiratory pressure.

The lower respiratory oscillations were 0, 1/2, 1, 1 1/2, 1 3/4, and 3 1/4.

Even where the inspiratory and expiratory pressures are high, the respiratory oscillations may be small or absent; thus in one instance where the inspiratory pressure was +9, the expiratory pressure was the same, and the respiratory oscillation therefore 0. Per contra even where the inspiratory pressure is low, the respiratory oscillation may be considerable if there be much dyspnea; for example, in a case in which the inspiratory pressure was 0, the expiratory pressure was +8, and the respiratory oscillation therefore 8.

Where there is no dyspnea the respiratory oscillations are apt to be small, and may be completely absent.

These observations show that, in pneumothorax, whatever general statements may be made, they have to be applied with caution in individual cases, for it is impossible in any given case to forecast what the actual pressures will prove to be; and, finally, that although the results obtained will have to be explained according to the peculiar circumstances of each case, yet if this be done carefully, much information may be obtained concerning the actual condition of the lung and pleura.

Intrapleural tension in serous effusion.—In health the pleural cavity contains no fluid, and we often speak of it as dry; yet this description is somewhat inaccurate, for there is in fact a constant circulation of fluid into the pleura and out of it, the fluid being effused by the blood-vessels and carried away by the lymphatics. The mechanism by which this is performed has been described as "the lymphatic pump." It consists of the lymphatic vessels with their stomata and valves, and is worked by the respiratory movements. The course of the circulation in the lung is from the pleural surface towards the root of the lung, as has been determined by experiment; and there is a similar circulation from the pleural surface through the diaphragm and through the chest walls. It is partly through the action of the lymphatic pump that the negative pressure is maintained in the pleural cavity and the lungs kept fully expanded.

There are two ways, therefore, in which fluid may accumulate in the pleura: either it may be poured out into the pleura in larger quantities than the pump can remove, that is, its amount may be abnormal, or the amount of fluid not being above the normal, the pump may be defective.
In the case of pleural inflammation both these processes come into play; the amount of transudation is considerable, while the stomata and smaller lymphatics are often plugged by deposits of fibrin. Thus in inflammatory cases the fluid may accumulate with very great rapidity and soon reach a large amount.

In the case of dropsy of the pleura consequent, let us suppose, on heart disease, the explanation is probably also in great part mechanical. Exudation under these conditions takes place from the blood-vessels into the lymphatics of the lung, which become water-logged or choked; thus it is unable to carry off the fluid from the pleural cavity, which consequently accumulates in it. With dropsy, however, the accumulation of fluid is much slower and the amount as a rule much less.

When fluid collects in the pleura it falls by its weight to the lowest part; and although the tension in the whole pleural cavity is diminished in proportion to the amount of fluid present, still the effect upon the different parts of the lung is different: thus the lowest parts suffer most and become collapsed, while the upper parts of the lung remain distended; yet the tension in the upper part of the pleural cavity is also lower than it otherwise would be, as is shown by Calvert’s observations. The diminished tone in the lung or tension in the pleura explains the hyper-resonant note which is obtained in those parts of the lungs which are floating upon the fluid.

In determining the intraplestral pressure in cases of fluid effusion something will depend upon the seat of puncture. This Calvert has also demonstrated; for if the mouth of the trocar be 1, 2, or 3 inches respectively below the level of the fluid, there will be the pressure of a column of fluid of this height to allow for. If, for example, the intrapleural tension be equivalent to – 3 inches of water, and the amount of fluid exuded into the pleura be sufficient to reduce this 3 inches negative pressure to 2 inches negative pressure, it follows that if the mouth of the trocar be 2 inches below the level of the fluid, a positive pressure of 2 inches will have to be added to the negative pressure in the rest of the pleura, which will reduce the pressure-reading to zero; or, if the height of the fluid be 3 inches instead of 2 inches, it would convert the pressure at the point of puncture to a positive pressure of 1 inch. It is very difficult to make due allowance for these variable conditions, so that the pressure records in pleural effusions have not anything like the same value as those in pneumothorax.

It might be supposed at first that with large effusions the pressure would be high, with medium-sized effusions moderate, and with small effusions low; but actual observation shows that this is by no means the case, for whatever be the bulk of the effusions the pressures may be high, moderate, zero, or even below zero. Thus, among my own observations, where the effusion was large and a considerable quantity of fluid was drawn off, the pressures were – 1, 2½, 4, 6, 8, 11½, and 18; where the effusion was moderate – 1, 0, 4, 5, 8½; and where it was small, 0, ½, 1½, 3, 5, 14.

The pressures, therefore, vary in a curiously irregular way, and it is
difficult to see what the explanation can be. It is natural to attempt to refer these variations to the different stages of the inflammation. Thus in the early ingравесent stage, when the effusion is rapidly forming, the pressures might be high, and low in the later stages when the fluid is being absorbed. There is some evidence in favour of this view, but the matter is by no means as simple as it would seem.

Respiratory oscillations.—For the reasons given when speaking of pneumothorax the respiratory oscillation with serous effusion is likely to be small; as a matter of fact it is so, and not infrequently it is entirely absent.

Now, as the action of the lymphatic pump depends upon the respiratory movements, and as these are indicated by the respiratory oscillations, it is evident that in these cases the mechanism for the removal of the fluid is at a standstill.

It is interesting to observe in some cases, though the respiratory oscillation is absent when the puncture is first made, that after fluid has been withdrawn the respiratory oscillation begins to return, and at the end of the operation may be fairly considerable. This is important, as it explains what is often observed at the bedside, namely, that the removal of a small quantity of an effusion may lead to the rapid spontaneous disappearance of the rest. What it really means is, that the lymphatic pump has been set to work again.

The intrapleural pressure in serous effusions is the resultant of three forces:—1. The respiratory movements. The effect of these has been already sufficiently considered. 2. The force of inflammatory exudation. We do not know much of the pressures under which the exudation of inflammatory fluid takes place in the pleura; but if we may compare it with the knee-joint, which is more accessible to observation, we may be quite sure that it occurs under very considerable pressure when we remember how tense the synovial sac becomes during the early stages of inflammatory effusion. 3. The action of the lymphatic pump is opposed to the first. We may presume that it is practically equivalent to the elasticity of the lung, and therefore equal to 6 or 8 millimetres of mercury, when the lung is fully distended; but it is a rapidly diminishing force as the lung becomes compressed, the stomata closed, and the lymphatics collapsed; and when the chest is full of fluid it vanishes, for, as the respiratory oscillations show, the lymphatic pump comes to a stop.

In the early stage of acute inflammation we may conclude that the pressure may be very high when the effusion is a large one, or when the effusion, if a small one, is encapsulated, that is, localised and not general.

When the acute stage of the inflammation has passed and exudation ceases, if the fluid begins to be slowly removed the pressure will fall; and it is obvious, since the fluid is ultimately removed completely, and the lungs come out into contact with the chest walls again, that in course of time the pressure will even become negative. I do not see
any way in which this can be brought about except through the intervention of the lymphatic pump.

**Intrapleural tension in empyema.**—This is a much simpler problem than in the case of serous effusions. The pressures here are in accord with what we know of suppuration elsewhere; for the formation of pus goes on under considerable pressure. It is only in the very chronic so-called "cold" abscesses that the tension is low; but even then the pressure is probably above that of the atmosphere.

Thus among my own observations the pressure was considerably raised in all cases, the lowest being +3. The highest was +16, and this was found with a very large effusion; but, as I have said, small effusions may have a very high pressure if they be loculated or encapsulated. An interesting example of this was observed among the cases of serous effusion; for in one in which the pleura had been tapped twice, and the pressure found on each occasion to be not raised, on the third paracentesis the pressure was +3; the effusion, however, was no longer serous, but had become purulent: in other words, the general serous effusion had been followed by a small localised empyema; this was incised and then recovery became complete.

The respiratory oscillation in empyema is always small and frequently entirely absent.

From what has been said it is evident that the problem of intrapleural tension, especially under pathological conditions, is a very complicated and difficult one, and requires much further investigation.

**Samuel West.**

**REFERENCES**


**S. W.**

**Pleurisy**

The name Pleurisy (ὄ πλευρίτις νόσος, morbus lateralis, side-sore of early English) formerly denoted that acute disease which is characterised by fever and severe pain in the side; and the meaning of the word was wholly clinical. After the time of Morgagni, when the influence of morbid anatomy became predominant, the name acquired that anatomical signification which it has since retained; and for the last century or more, pleurisy has been defined to mean inflammation of the pleural membrane.
PLEURISY

I. Etiology.—1. Age and Sex.—Pleurisy occurs at all ages: I have evacuated pleural empyema in infants aged one month, three months, and five months; and I have drawn off three pints of serum from the chest of a woman eighty-seven years old. The annexed table, drawn up from the records of St. Bartholomew's Hospital for ten years (1884-1893), shows the number of patients treated for pleurisy, and in whom pleurisy was the main and foremost disease: it does not include the cases in which pleurisy was secondary to some other disease no less serious. The figures show: that pleurisy is much more frequent in males than in females; that pleurisy with effusion of coagulable lymph or of serum (dilute liquor sanguinis) is most common in patients between twenty and forty years old; and that pleural empyema is most common in patients less than ten years old.

<table>
<thead>
<tr>
<th>Effusion</th>
<th>Males</th>
<th>Females</th>
<th>Deaths</th>
<th>5 yrs. and under</th>
<th>10 y.</th>
<th>15 y.</th>
<th>20 y.</th>
<th>30 y.</th>
<th>40 y.</th>
<th>50 y.</th>
<th>60 y.</th>
<th>60 y. Over 60</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not purulent</td>
<td>465</td>
<td>186</td>
<td>48</td>
<td>25</td>
<td>59</td>
<td>50</td>
<td>54</td>
<td>179</td>
<td>149</td>
<td>85</td>
<td>35</td>
<td>15</td>
<td>651</td>
</tr>
<tr>
<td>Purulent</td>
<td>155</td>
<td>61</td>
<td>48</td>
<td>53</td>
<td>32</td>
<td>15</td>
<td>22</td>
<td>48</td>
<td>17</td>
<td>23</td>
<td>6</td>
<td>0</td>
<td>216</td>
</tr>
<tr>
<td>Totals</td>
<td>620</td>
<td>247</td>
<td>96</td>
<td>78</td>
<td>91</td>
<td>65</td>
<td>70</td>
<td>227</td>
<td>166</td>
<td>108</td>
<td>41</td>
<td>15</td>
<td>867</td>
</tr>
</tbody>
</table>

2. Specific Poisons.—Pleurisy is due to irritation of the pleural membrane by certain morbidic microbes or poisons. It is difficult not to believe that this proposition is universally true; and true, even in the case of pleurisy following upon an injury to the side, or upon exposure of the chest to cold. (i.) A heavy blow upon the chest, not leading to more than bruising of the parts, and not bringing about any solution of continuity, will sometimes be followed by constant pain, and at length by serous effusion into the pleural cavity: in such a case it is reasonable to suppose that the injury affords an opportunity for infection. (ii.) Again, pleurisy, like pneumonia, will sometimes follow so speedily upon great exposure of the whole body, or of the chest in particular, to cold, that it is carrying scepticism to excess to doubt that the exposure has something to do with causing the subsequent disease: in this case, also, the cold may be supposed to bring about such an altered nutrition of the parts as favours invasion by specific microbes.

But in by far the greater number of cases pleurisy is spontaneous, and arises apart from the operation of any obvious antecedent cause. Microbiology has thrown great light upon this spontaneous or idiopathic pleurisy. The microbes which will account for most pleurisies are three,—tubercle bacillus, streptococcus, and pneumococcus.

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*1 The following details concerning bacteriology are taken from an article by Dr. Netter in the 4th vol. of the Traité de médecine, edited by Charcot, Bouchard, and Brousaud.
(i.) Tubercle bacillus.—(a) Even before the discovery of the bacillus of Koch, it was suspected that many cases of pleurisy with serous effusion were due to tuberculosis of the pleura. But now it cannot be doubted that tubercle is the commonest cause of pleurisy with serous effusion, an opinion supported by the following facts:—Many of the patients have inherited a tendency to tuberculous disease. Some of them have suffered from manifest tuberculous disease before the pleurisy began. Many of those who die are found by examination post-mortem to be tuberculous. Many of those who recover from the effusion suffer afterwards from tuberculous disease, and especially from pulmonary consumption. On the other hand, it is admitted that, even in cases which are undoubtedly tuberculous, bacilli are seldom found in the effusion, and cultivation of the fluid gives no result. Inoculation of the pleural serum into the peritoneal cavity of guinea-pigs is more successful; many of the animals are infected thereby. (β) Purulent effusion is less often dependent upon tubercle. Empyema in a tuberculous subject is sometimes due, not to the tubercle, but to streptococci or pneumococci: the distinction depends upon microbiological examination.

(ii.) Pyogenetic streptococcus is the microbe most commonly found in the pleural empyema of the adult. The morbific germ reaches the pleura:—(α) Through the lung, in pneumonia, dilated bronchi, gangrene, pyemic abscess, tubercle, cancer; (β) through mediastinal organs, in pericarditis, disease of oesophagus, abscess spreading from neck or throat; (γ) through walls of chest, in penetrating wounds, abscesses, lymphangitis, disease of breast, and especially cancer; (δ) from caries of vertebrae, which is sometimes quite latent; (ε) through peritoneum, in peritonitis, subphrenic abscess, suppuration of liver or spleen; (ζ) through the blood, in general diseases, scarlet fever, diphtheria, erysipelas. (η) Adjoining local disease sometimes seems to act as a mere irritant of the pleura, and so to render it susceptible to purulent infection by the blood: aneurysm of the aorta is an instance of this kind, the aneurysm itself being possibly quite latent.

(iii.) Pneumococcus is the microbe most commonly found in the empyema of childhood. In most of the cases it cannot be proved that pneumonia preceded or accompanied the empyema, and this is especially true with respect to children. In other words, primary pneumococcos pleurisy is a common disease. When secondary to pneumonia there is usually an interval of some days' duration between the defervescence of the pneumonia and the occurrence of the symptoms and physical signs of pleural effusion; but sometimes there is no interval, the empyema begins before the pneumonia has ended; on the other hand, the interval is sometimes much longer, several weeks or months. The pleuritic effusion which is subsequent to pneumonia is not always purulent, but is sometimes serous: in this serous effusion pneumococci are found. Moreover, serous effusion, which is not secondary to pneumonia, is due in a few cases to pneumococci.

(iv.) There are some other causes of pleurisy, but the specific manner
in which they operate has not been discovered; such are superficial haemorrhagic infarctus of the lung, nephritis, rheumatism, and gonorrhœal rheumatism.

II. Symptoms, that is to say, *signa assidentia*, are the signs which are not pathognomonic or characteristic of the disease. Yet inasmuch as these are the signs which the patient recognises, and which are, therefore, the signs first recognised, they may be aptly discussed in the first place.

1. Onset of the disease.—(i.) Latent.—The occurrence of one or more of these symptoms marks the onset of the disease in most cases. But in other cases the onset is not perceived: the disease at first is latent; and it is most likely to be latent when it is secondary to other serious disease, the symptoms indicative of the onset of pleurisy being masked by pre-existing symptoms dependent upon the primary disease.

(ii.) Manifest.—When the onset of the disease is not latent, the indicative or invasion symptoms either (a) occur suddenly and decisively, clearly marking the time at which the state of health passes into the state of sickness; or (b) they occur gradually, so that it is not easy to say precisely when the disease began. Whether they occur suddenly or gradually, these symptoms, denoting the onset of the disease, are no other than more or fewer of the symptoms which attend the confirmed disease, and which will be described in the next place. The commonest invasion symptoms are fever (with shivering or not), pain in the side, vomiting, cough, quickness and shortness of breathing; in children sometimes convulsions.

2. Fever.—Fever is not a constant symptom; being slight and temporary in pleurisy, with small innocuous exudation; being present in most, and yet not in all cases of larger effusion; being absent sometimes even in empyema.

(i.) In acute pleurisy the temperature seldom rises above 103°. In pleurisy, as compared with pneumonia, the fever is not so high, shivering at the onset is less common, and the duration of the fever is indefinite.

(ii.) In chronic pleurisy no distinction can be drawn between serous and purulent effusion by means of the characters of the fever. (a) What is called serous effusion is not serous in the strict meaning of that word, but is really a dilute lymph or liquor sanguinis¹ not free from leucocytes. In pleurisy with serous effusion the temperature is often (but not always) persistently raised; and the fever not less or less constant than that of a purulent effusion. When fever is present it lasts until the effusion is wholly absorbed; indeed, in cases of febrile serous effusion defervescence is the best evidence that the effusion has been absorbed, for physical examination is often of no avail in determining this point. The type of fever tends to be quotididian remittent. (b) In pleural empyema, when the pus is pent up or

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offensive, the temperature will be raised almost for certain. Evacuation of the pus will be followed by defervescence, temporary or permanent. In fistulous empyema the temperature is usually almost or quite natural; and a rise of temperature means that pus is pent up somewhere. The type of temperature tends to be that of septic fever, namely, quotidian remittent with evening exacerbation. Colliquiative symptoms (heavy sweats, and especially diarrhoea) sometimes attend the fever. Lastly, in some cases of small empyema, even when undrained, the temperature remains normal.

Local temperature.—That the affected side is sometimes hotter than the other was known to ancient Greek physicians, who employed an ingenious means of discovering the fact (see Hippocrates, De Morbis, iii. chapter 16).

3. Pain.—Severe pain in the side was the main and constant sign of the disease called pleurisy in the ancient sense of the word; but the pain of pleurisy, in the modern sense, may be severe, or may be not severe, or there may be no pain at all. The pain is usually felt in the side of the chest; sometimes about the nipple, or above the clavicle, or in the hypochondrium; sometimes about the navel, or even in the iliac fossa and lower belly, on the same side as the disease. The skin over the affected side is often very tender. Marked spinal tenderness, in some part of the vertebral groove in the dorsal region, is common.

The nature of the pain is a matter for debate; probably there are different causes of the pain. Intercostal or diaphragmatic cramp has been suggested as an explanation of the stitch in the side. The pain felt at a distance (namely in the abdomen) is probably conducted along an intercostal nerve.

4. Dyspnœa.—Dyspnœa, manifested by frequent or laboured breathing, is common. Patients kept in bed are apt to become accustomed to the want of breath, and so their dyspnœa may diminish or even disappear; although the quantity of pleural effusion (if present) remain unchanged. Dyspnœa is in some cases greatly due to associated disease, for instance, to chronic pneumonia on the same side as the pleurisy. The dyspnœa of pleurisy without liquid effusion is chiefly shortness of breath; that is to say, inability to breathe freely and deeply because of the pain caused thereby.

5. Cough and Expectoration.—Cough is usually present; but in rare cases there may be no cough, even in pleurisy going on to effusion.

Concerning the sputa.—The terms dry and humoral pleurisy, in the old sense of these words, relate to the absence or presence of expectoration. For sometimes there is no expectoration, and therefore no expulsion. But commonly there is expectoration, although the humours coughed up are not always spat out. (i.) The sputa sometimes consist of mucus nearly pure, judging from their colour and transparency: when the mucus is thin and watery, like gum water, it is called pituitous. (ii.) More often the sputa are mucous-purulent. (iii.) More or less blood in the sputum is not uncommon at the onset of the disease. (iv.) When
an empyema has burst into a bronchus the sputa are almost pure pus. (v.) It is a very uncommon event for a serous effusion to burst through the lung, and so to be expectorated. Yet this seems to have happened in a case narrated by Dr. Vincent Harris in *St. Barthol. Hosp. Reports*, vol. xxiii. p. 34. Less uncommon is the muco-serous (or albuminous) expectoration, which sometimes occurs during or soon after paracentesis thoracis, and which will be described in connection with that operation (p. 376). (vi.) Fœtid expectoration depends upon one of two conditions: either a fœtid empyema has burst through the lung—by far the more common case; or a fœtid empyema, which has certainly not burst into the lung, communicates an offensive smell to the secretions of the air-passages in the neighbourhood, just as abscesses near to the alimentary canal often acquire, for this reason, a disgusting smell.

6. *Vomiting and Diarrhoea.*—Vomiting is common at the onset, especially in children.

Diarrhoea is common in pleurisy with effusion, serous or purulent. Diarrhoea sometimes occurs from the very onset of purulent pleurisy: should vomiting, pain referred to the belly, and tenderness of the belly concur with diarrhoea, peritonitis will be closely simulated at first sight. This diarrhoea tends to be very obstinate, and in many cases cannot be stopped until the empyema is cured. Diarrhoea and marasmus may be the main symptoms of a small empyema. Should the patient die, post-mortem nothing amiss with the intestines will be discovered by the naked eye: it is a septic diarrhoea.

7. *Septic infection of whole body.*—(i.) Symptoms which are called typhoid or putrid, and which are indicative of septic infection of the whole body, are apt to accompany fœtid empyema. The tongue is dry and brown, the secretions become offensive to smell, the eyes are yellowish, the face is dusky, the pulse soft and weak, consciousness blunted, and muscular debility, or prostration, great. (ii.) Like symptoms sometimes occur from the very onset of empyema which is not fœtid. In a state of good health sudden shivering occurs, headache, cough, in some cases much pain in the side; in others no pain at all. The fever is high, the temperature often reaching 104° or more; respiration frequent; sputa not rusty. Consciousness becomes affected; in some cases so much that even as early as on the second day the patient is deeply comatose; but the degree of coma is apt to vary, so that the patient, after deep unconsciousness, may become fairly sensible. More or less delirium occurs in some cases, but in others none at all. Morbilliform motting of the skin (not much like the rash of typhus); temporary redness, swelling, and tenderness of one or several joints; enlargement of the spleen and diarrhoea may occur in some patients. The urine may be albuminous or not. The physical signs of effusion are sometimes late to appear, and are apt to be mistaken for those of pneumonia. The patient will die within ten or twelve days; and whether paracentesis be employed or not seems to make small difference. The pus
has been found to contain pneumococci (21); but pneumococccous pleurisy is seldom attended by these grave signs of universal-poisoning.

8. Latent pleurisy.—Pleurisy is sometimes latent, in the sense that the symptoms of the disease are slight, nay almost absent; and this even in the case of large effusion. But it is only in the neglect of physical examination that pleurisy, unless its extent be very small indeed, can ever be really latent.

III. Signs.—Signs which are pathognomonic, signs by which pleurisy can, with certainty, be distinguished from other diseases, are of two kinds; namely, physical signs and the result of puncture. These signs do more than this; they enable us to distinguish two kinds of pleurisy, which it is important should be distinguished; namely, pleurisy with exudation of coagulable lymph only, and pleurisy with liquid effusion. Moreover, puncture enables us to distinguish the different kinds of liquid effused.

A. Pleurisy with no liquid effusion.—1. This condition often exists unattended by physical signs of disease, or at most attended by signs which are not distinctive; such as some degree of retraction of the chest, some loss of clear tone on percussion, some weakness of breathing sound.

2. The only sign which is quite distinctive is friction sound. But it is very far from being a constant attendant upon pleurisy, even when the effusion is nothing more than coagulable lymph. Indeed it might be said, and probably with truth, that even under these conditions friction sound is more frequently absent than present. Friction sound is to be recognised by its peculiar friction quality, giving the notion either of rubbing to any degree between lightest grazing and harshest scraping, or of creaking like that of leather. Friction is usually a very local sound, heard over a small part of one side only; and that part is mostly where the rib movements are freest, namely, the lower part of the chest, below the nipple or armpit, or about the angle of the shoulder-blade.

B. Pleurisy with liquid effusion.—1. Before the effusion becomes abundant enough to gravitate, a friction sound is sometimes (but seldom) heard. Still more uncommon are signs which attend the onset of pleurisy with effusion in rare cases, and which closely resemble those of bronchitis. The distinction between the two diseases is to be found in the fact that bronchitis very seldom affects one side only, and that pleurisy with effusion very seldom affects both sides. The signs referred to are these:—The affected side moves less freely than the other; the percussion note is raised in pitch and muffled over the greater part or the whole of the side; the sense of resistance to percussion is increased; the breathing sound is weak and attended by widely-spread rale, which is quite indistinguishable from the rale of bronchitis. This rale has been called friction-rale, thereby to indicate the belief that the sound is produced in the pleural sac. But it seems more probable that the rale really is a bronchial and mucous rale
produced in the air-tubes, and that the catarrhal or bronchitic state of
the lung is due to its relaxation or deficient expansion consequent upon
the pleural effusion, small though it be.

2. Much more frequently, however, the earliest signs of pleurisy
with effusion are those which indicate that the effusion is already
abundant enough to have sunk to the lowest place. What constitutes
the lowest place depends upon the attitude assumed by the patient
while effusion is going on. At first, when the quantity is small, the
lung is simply relaxed by virtue of its own elasticity, and swims upon
the effusion; but as the liquid accumulates, it compresses the lung and
renders it more or less empty of air.

(i.) The great sign of liquid effusion is a coextensive dulness to
percussion. This dulness is not wholly due to the effusion, but is
partly dependent upon associated collapse of lung; that is to say, a
layer of liquid an inch or more thick would transmit percussion
resonance of the lung were the lung resonant. Dulness begins at the
lowest part of the chest behind, the note being natural elsewhere.
When the effusion has risen higher than the angle of the scapula, the
lung will have relaxed to such an extent as to give a clear tracheal
note above the nipple of the same side in front—a sign not always
present even in cases watched day by day from the onset. Whether,
by further increase in the quantity of the fluid, the whole back become
dull before the front is so at all, or whether the upper level of the
fluid be comparatively horizontal, depends upon the attitude assumed
by the patient while the effusion is going on. Hence, when the effusion
is small the dulness may be wholly posterior, and sharply defined in
front by the posterior axillary line, the lateral region remaining reso-
nant. On the other hand, the upper limit of a dulness which occupies
the lower rather than the hinder part of the chest often rises higher
in the axillary region than in the back. Even when absolute dulness
is confined to the base, there is usually some impairment of resonance
all over the back on that side. The dulness over the effusion may be
far from absolute. The anterior clear resonance, when present, is
sometimes of cracked-pot quality. The effusion, even when partial,
does not shift its position easily or at all with changes in the position
of the body.

(ii.) In proportion to the amount of effusion the side is enlarged,
diaphragm depressed, and mediastinum displaced. (a) The side, com-
pared with the other, will possess these characters: shape, on horizontal
section, rounder; antero-posterior diameter longer; length from above
downwards diminished; shoulder raised; spine curved towards the
unaffected side. The antero-posterior enlargement becomes very obvious
when the physician stands behind the patient so as to look obliquely
over the shoulders and the front of the chest. Circumferential measure-

1 What the Germans call "tymanitisch." For the exact meaning of the technical
terms used in these pages with reference to percussion and auscultation, I must refer the
reader to my book on those subjects.
ments of the two sides are often made for the sake of comparison, but be it remembered that, by the passage of the elliptical form into the circular, considerable increase in the sectional area of the chest may occur, whilst the length of the periphery remains the same. Moreover, the displacement of the mediastinum thrusts the heart into the unaffected side. Add this consideration, too, that the walls of the healthy side must follow the antero-posterior, projection of the diseased side; and then it will be plain why, as a matter of fact, the perimeter of the affected side often measures very little more, nay, sometimes even less, than that of the side which is not diseased. The cyrtometer, by indicating shape as well as circumference, affords us the true means of recording the amount of unilateral enlargement. (β) Displacement of the mediastinum is indicated by displacement of the heart. Effusion into the right pleura may displace the heart so as to cause its impulse to be felt in the left axillary line, and in any interspace from the second to the sixth. Effusion into the left pleura may displace the heart so as to cause its impulse to be felt anywhere between its natural position and the right nipple line, and in any interspace from the fourth to the seventh, or in the epigastrium. When the impulse is felt to the right of the natural position, it is often some part of the heart, other than the apex, which strikes against the chest; and this part is usually the right conus arteriosus. When the heart is displaced to the right, there is, in most cases, no considerable change in the relative position of base and apex; that is to say, the heart does not swing to the right upon its base as a fixed point. Yet such a change in the attitude of the heart does sometimes occur, and the very ventricular apex may beat in the right nipple line. The displacement of the heart is often more or less than might be expected; for instance, it may remain unmoved by an effusion of not less than a quart of serum into one pleura. Percussion of the sternal region above the heart sometimes affords evidence of displacement of the mediastinum: the upper part of the sternum naturally yields a clear resonance; under the pressure of a copious liquid effusion into either pleura, the mediastinum bulges so much towards the unaffected side as to afford absolute dulness to percussion in the sternal region, and even somewhat beyond it. (γ) Displacement of the diaphragm downwards is determined by ascertaining the position of the liver, spleen, and stomach. When the quantity of fluid in the left pleura is very great, the left half of the diaphragm may possibly be depressed to such a degree that not only can the lower margin of the spleen be felt, but even its upper margin, in fact its whole outline. At the same time, the thrusting of the mediastinum and heart into the right side of the thorax may depress the right wing also of the diaphragm to an almost equal degree; a point ascertained by examination of the liver.

(iii.) Vocal thrill is diminished where dulness to percussion exists, and is wholly abolished in great distension of the side.

(iv.) The respiratory sound is at first weakly vesicular, and sometimes
remains so throughout the disease. But often the breathing soon becomes bronchial, sometimes even before the dulness becomes absolute. With progressive increase of effusion the bronchial breathing tends to become less and less loud, until, at last, it is wholly suppressed. But sometimes, although the quantity of fluid be very great, loud bronchial breathing is heard all over the affected side: the fact being that the loudness depends, not inversely upon the quantity of liquor effused, but directly upon the openness of the air-tubes; for liquid is a good conductor of sound.

(v.) Vocal resonance is weak or bronchial in much the same manner as the breathing sound. When the effusion is partial, with clear resonance in front, the bronchophony is sometimes ægophonic about the angle of the scapula. Ægophony is a sign of little or no value. In the first place, well-marked ægophony is seldom heard; next, it is sometimes heard over simple consolidation of the lung, such as is left by absorption of pleural effusion; and, lastly, ægophony certainly does not always attend thin layers of liquid in the pleural sac.

(vi.) By percussing the chest in front with two coins, and auscultating behind as for the bell sound, a pleural effusion will sometimes be found to transmit a clear metallic sound (penny sound, signe de sou) quite unlike that heard through healthy or solid lung.

(vii.) A small protrusion, in the lateral region, distended during expiration, receding during inspiration, and due to perforation of the pleura and intercostal space, may be met with even in moderate serous effusion.

(viii.) A systolic murmur, having the characters of a pulmonary obstructive murmur, sometimes concurs with pleural effusion; disappearance of the effusion being attended by disappearance of the murmur.

C. Puncture.—Puncture of the chest, by means of a fine tubular needle adapted to a small exhausting syringe, is the most decisive means of determining the presence of pleural effusion. Moreover, puncture ascertains the quality of the effusion. The bare suspicion of a pleural effusion, however small it may seem to be, is a sufficient reason for exploring the chest by puncture, inasmuch as we know that to pierce the lung with a clean, fine needle is harmless.

Puncture is made where the signs of effusion are most marked; due regard being paid to the anatomy of the parts within, so as to beware of wounding the heart, diaphragm, or great vessels. But, if possible, let the puncture be made somewhere between the angle of the scapula and the edge of the pectoralis major, and not much below the nipple level. As matter of fact, the puncture is most usually made somewhere about the angle of the scapula.

Puncture may lead us into error. (i.) In small, old empyemata the enclosing walls are sometimes very thick, and it asks some faith in our power of diagnosis to let us push the needle boldly through them so as to reach the pus. (ii.) Pus is sometimes so thick that it will not pass
through a fine needle: in this case a small quantity (less than a drop perhaps) will probably have entered the needle, and can be blown out and examined; if there be pus, a larger needle must be used next time. (iii.) Pus can sometimes be drawn from a bronchial tube, or from a suppurating cavity within the lung, such as produced by tuberele, destructive pneumonia, or actinomycosis. (iv.) The needle may draw off pus from the pericardium or a subphrenic abscess, after perforating the lung or the diaphragm.

D. Different kinds of liquid effusion.—It is by means of puncture that the kind of effusion is discovered.

1. Serous effusion.—It has been already remarked that what is called a serous effusion consists of diluted liquor sanguinis. The specific gravity is usually from 1018 to 1024; but in proportion as the effusion approximates to the nature of hydrothorax the specific gravity falls, and it may be so low as 1006. Reaction, alkaline. Colour, yellowish from serum-lutein. Proteids present: fibrinogen, serum-globulin, and serum-albumin. A small quantity of sugar is often found. The liquid is seldom or never quite clear and transparent. Opalescence, when slight, is due to a few leucocytes, particles of fibrin, albuminous particles, minute oil globules, cholesterin. When the turbidity is great the effusion is called opaline or chylous, a condition which will be described further on (see p. 357). The fibrin present coagulates soon after the effusion is drawn off. The quantity of fibrin differs much in different cases; it may amount to no more than a few filaments floating in the serum, or it may be so abundant as to coagulate into a firm jelly.

2. Purulent effusion.—Pleural empyema is probably such from the first in most cases. Yet a serous effusion may possibly become purulent, a change which is either spontaneous or the result of operation. Spontaneously the change takes place slowly, a serous effusion becoming gradually purulent in the course of about three weeks. When the change is due to operation upon a serous effusion (that is to say, to infection of the effusion by septic matters), suppuration occurs more quickly, in a few days instead of weeks.

Pus is sometimes remarkably glutinous, so that, as it escapes during paracentesis, it stands in a heap when drawn into an aspiration bottle.

Pus sometimes contains much gas in solution, so that it effervesces in an aspiration bottle. Such pus is not necessarily offensive.

Pus is sometimes very fetid, and the cause is not always the same. (i.) The cause is sometimes obscure, the pus is fetid from the first; I have known such a case to end in recovery after a single paracentesis, without draining. (ii.) The cause is sometimes manifest; the pus becomes offensive through contamination with putrefactive microbes in such manner as the following:—gangrene of lung, perforation of lung, perforation from without (for example, an operation), perforation of diaphragm by hepatic or subphrenic abscess, mere contiguity of an offensive abdominal abscess without actual perforation. Fœtid empyema

1 Serum seu lympha coagulabilis, De Haen, Ratio Med. IV. cap. iii. p. 74.
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is sometimes associated with—(i.) sloughing of pleural false membranes, and even of the pleura itself (an offensive slough lying loose in the empyematosus cavity may be the cause of the fœtus); (ii.) necrosis of one or more ribs.

3. Blood mingled with effusion.—Not hæmorrhage, which signifies extravasation of pure blood into the pleural sac. The effusion, which is bloody, is either serous or purulent, and the proportion of blood differs much in different cases.

The conditions under which an effusion becomes bloody are these:—
(i.) Simple uncomplicated pleurisy, the hæmorrhage being probably due to rupture of embryonic vessels in the false membranes; the hydrothorax of heart disease; the pleurisy of scarlatinal renal dropsy. (ii.) Acute tuberculosi of the pleura. (iii.) Cancer, sarcoma, lymphadenoma of the pleura. (iv.) Hæmorrhagic diathesis. The patient is sometimes markedly anemic from the loss of blood, sallow, cachetic. The prognosis depends upon the cause, the fact of a bloody effusion in itself makes no difference. Cancer of the pleura is by no means the most frequent cause of bloody effusion, and the effusion in cancer is sometimes clear yellow serum.

4. Opaline serous effusion.—The effusion is opaline, milky, in consequence of abundant molecular matter suspended in it; a few leucocytes are often present, and sometimes a few red discs: these latter may be numerous enough to give a reddish colour to the effusion. Specific gravity the same as that of ordinary pleural serum. The conditions of opaline effusion are these:—

(i.) Sometimes the opacity is really chylous; for instance, if the thoracic duct be torn across, so that chyle is diffused into the right pleura: in this case the molecules are all fatty, and rise to the top of the effusion, like cream. Obstruction to the duct may possibly have the same effect.

(ii.) But in most cases there is no reason for suspecting any lesion of the chylous system. The particles are often by no means all fatty, indeed very few may be fatty; they seem to be some ill-known form of proteid. Whence they come is quite uncertain; disintegration of pus globules has been supposed to be the source. Cholesterin crystals may be present, usually very few, but now and then they are very abundant, so that the opacity is chiefly due to them. No deduction can be drawn, as to the nature of the pleurisy, from the fact of the effusion being opaline.

E. Loculated empyema, or pleural abscess; the purulent effusion not occupying the whole of the pleural cavity, and being enclosed by adhesions, the rest of the pleural sac being natural or obliterated by adhesion.

1. The commonest seat of a circumscribed effusion is in the lateral or posterior part of the lower half of the chest on one side. In this case the diagnosis is easy enough by physical examination and puncture.

2. Loculated empyema sometimes lies between the base of the lung
and the diaphragm; mostly on one side only, but occasionally on both sides, without communicating. The diagnosis depends upon the situation of the pain felt, namely, at the attachment of the diaphragm around the lower margin of the thorax, upon immobility of the diaphragm and hypochondrium on the affected side; upon increased resistance of the hypochondrium to pressure; upon the signs of more or less extensive solidification of the base of the lung on the same side, in consequence of collapse of the lung and associated congestion (a small empyema will sometimes cause very extensive collapse, p. 368), indicated by loss of percussion tone (now and then the tone is clear and tubular, the lung being relaxed only), and weakened breath-sounds. The breathing is apt to be painful and difficult. Diaphragmatic empyema is often associated with subphrenic or hepatic abscess, and is often quite latent, found on post-mortem examination only. When a loculated empyema of this kind contains gas, diagnosis is often difficult (see page 370, Subphrenic abscess).

3. Abscess between the lobes of a lung is less common. The pus is very often discharged through the lung and expectorated, as early, it may be, as three or four weeks from the onset of the pleuritic symptoms. As a rule, it is only when the patient has begun to spit pus that the disease can even be suspected; physical signs, if any, are inadequate to the diagnosis.

4. Empyema at the apex only of the pleural cavity is an uncommon event, but one which sometimes occurs. Diagnosis is rendered all the more difficult on account of the reluctance with which we make a puncture in this dangerous region.

F. Pulsating Empyema.—That is to say, empyema which pulsates rhythmically with the heart.

The empyema is commonly very large, occupies and fills the left pleural cavity. (i.) The effusion usually points in one or two places, which alone pulsate. This bulging occurs in the normal heart region or in the lowest interspaces. In rare cases the protrusion has been seen in the loin below the ribs. The bulging is never larger than an orange. (ii.) Less commonly, the effusion nowhere points or bulges through the chest wall. However, in these cases also, the pulsation is usually limited to the normal heart region (to the left of the sternum), or to the lowest three or four intercostal spaces. But sometimes the pulsations are seen and felt over almost the whole of the left side.

Whether the empyema bulge or not, the heart is much displaced to the right. Pericarditis may concur, but usually the heart remains healthy. Auscultation of the pulsating part may detect conducted heart-sounds. Palpation detects no thrill and no expansion like that of an aneurysm.

Paracentesis very much helps the diagnosis. By removing part of the liquid the pulsation ceases; but the heart, being fixed by external pericardial adhesions, does not return to its normal position. The puncture needs not be made at the spot which pulsates.
The effusion is mostly chronic, and the lung wholly collapsed. Pneumothorax often concurs; in this case, pulsation is conveyed by the liquid only. The effusion is purulent in the great majority of cases, but now and then a serous effusion has been known to pulsate.

Very seldom the empyema does not fill the whole pleural cavity, but is loculated and enclosed in adhesions. This kind of pulsating empyema always bulges; it may be to the right of the sternum, but still in close neighbourhood to the heart.

The diagnosis is from intrathoracic aneurysm, and from the very uncommon condition of a pulsating cancerous tumour. Aortic aneurysm and pulsating empyema may coexist.

Pulsating empyema is, in most cases, incurable.

IV. Course and Termination. — 1. Adhesions. — When pleurisy terminates favourably, it is by the formation of more or less extensive adhesions between the opposed pleural surfaces, the pleural cavity being proportionally obliterated. The patient has recovered, and it is assumed, for this reason, that adhesion has occurred. Yet the recovery from pleurisy without effusion, and even from pleurisy with effusion and empyema, is often complete so far as physical signs are concerned; and the most careful examination fails to find contraction of the chest or any other signs of past disease. If adhesion be attended by physical signs, they are those which indicate unilateral contraction of the thorax and imperfect expansion of the lung. The more marked these signs, the more dense and tight may the adhesions be assumed to be.

2. Serous effusion. — (i.) Absorption. Serous effusion tends to be spontaneously absorbed; a large effusion may thus disappear in a week or two.

- The temperature, if it have been raised, usually remains raised until absorption is complete.

- The physical signs which indicate the progress of absorption are these:—The diaphragm and mediastinum go back to their natural position; to follow the retreating heart, liver, and spleen is the best means of marking the process so long as the quantity of effusion remains great. The distension of the affected side becomes less, and accurately to register this fact is an important service rendered by the cyrtometer. When the effusion has so far diminished that the lung again comes into contact with the chest wall, percussion usually enables us to follow the falling level of liquid. And, at the same time, auscultation will sometimes inform us when and where actual contact of the opposed surfaces of the pleura has occurred, friction sound being heard.

The manner in which the effusion is absorbed is not constant, but usually the liquid disappears in something like the following order:—From the vertebral groove near the root of the lung; from the supramammary region; from the rest of the vertebral groove and infrascapular region; from the inframammary region; and, lastly, from the lower lateral region, concerning which it is important to remember that the lowest part of the pleural cavity, in the erect position of the body, is in
the axillary line. Thus, the upper surface of the liquid, when it reaches as high as two inches above the nipple level, is horizontal; when lower than this, the dulness forms irregular parabolic curves, which become smaller and smaller, and last of all disappear in the lowest parts of the thorax. But we must be prepared to meet with exceptions to these rules, and to find the residue of liquid in almost any part of the chest. Moreover, a large pleural effusion is sometimes absorbed, not from above downwards, according to the rule, but equally all over the side at once, friction or pleuritic râle becoming audible all over the side at once.

Disappearance of effused liquid at any spot is sometimes attended, for a day or two, by friction sound, indicative of restored contact between the pleural surfaces, redux friction as it is usually called.

Dulness, practically absolute, and due to unexpanded lung, often remains for a long time after all the effusion has been absorbed. For this reason it is often impossible to say, from physical signs alone, when the effusion has been absorbed. The physician must judge from all the signs and symptoms taken together, and especially from permanent defervescence, if the patient have been febrile. More or less dulness often remains for the rest of life.

The latest physical sign, dependent upon absorption, is retraction of the affected side. Cup-like sinking of the lower part of the sternum occasionally ensues. In some cases these deformities tend to disappear gradually, in others they are permanent.

A systolic murmur, having the characters of a pulmonary obstructive murmur, sometimes concurs with pleural effusion; disappearance of the effusion being attended by disappearance of the murmur. A permanent murmur of the same kind is sometimes heard when one side of the chest is left contracted.

(ii.) Permanence.—If the whole lung be very much reduced in size and quite inexpansible, a serous effusion will probably be permanent and endure to the end of the patient’s life. It is possible that, under these circumstances, the chest walls may contract, and the mediastinum and diaphragm be displaced to such a degree as to allow of absorption of the liquid and obliteration of the pleural cavity; but these events seldom happen in the case of serous effusion. The conditions of lung which lead to its complete inexpansibleity are two: carnification (see p. 368), associated with tight, unyielding thickening of the pulmonary pleura; and contracting cancer, which may reduce the whole lung to a mass not larger than the pancreas.

3. Empyema.—(i.) External rupture.—An empyema, left to itself, will usually perforate the thoracic wall in course of time. The opening mostly occurs in front; a common situation is the fifth interspace in the nipple line. But an empyema may point almost anywhere, from just below the collar-bone to the loin or even the buttock. The first effect of a pointing empyema in some cases is to produce what looks like a mere subcutaneous abscess; in fact an abscess of this kind over
the ribs is often due to the perforation of a pleural empyema, even if there be no signs of pleural effusion.

The course of an empyema (unless it be very small) which has been allowed to discharge spontaneously through the chest wall, and which is left to itself, is very tedious. If the opening close, it takes a long time in doing so, but often it never closes. In either case the patient runs the risk of a ruined state of health, complicated by lardaceous changes in the viscer.a.

(ii.) Rupture through lung.—In this case a small hole, which allows of direct communication between the empyema and a bronchial tube, is made through the lung by ulceration; or else, more seldom, the pus filters through a small portion of lung which is spongy and penetrated by many minute passages.

Empyema, which perforates the lung, is usually loculated, and often so small and deeply seated that it cannot be detected by physical examination. Such loculated empyemata often occur between the lobes of a lung, or between the lung and diaphragm, or in the mediastinum close to the root of the lung.

The expectorated pus is sometimes fœtid, sometimes not. It is sometimes fœtid at first, and afterwards spontaneously ceases to be fœtid. In some cases the opened cavity contains air, in others not. The microbe present is usually pneumococcus.

Recovery often occurs, and in no great space of time, even when the patient is left to the unassisted powers of nature, as is very often the case, it being impossible to open a deeply-seated abscess by simple paracentesis. Death may be very unexpected, the patient being choked by the sudden discharge of a large quantity of pus into the air-passages. Or death may be the termination of a long period of purulent expectoration and gradual exhaustion of the patient’s strength.

(iii.) Rupture into other parts.—Empyema will sometimes perforate the pericardium, and in the case of pneumo-empyema the pericardial sac may contain air as well as pus. The peritoneum may be perforated. The empyema may discharge through the oesophagus. It is probable that the cases narrated by older physicians, cases in which empyema has been accompanied by a discharge of pus from the intestines or with the urine, were really cases of empyema complicated with subphrenic abscess.

(iv.) Incurable empyema.—Empyema is sometimes permanent and incurable because associated with certain local conditions which prevent recovery. The lung may be quite inextensible, either carniﬁed or tightly bound by thickened pleura. Tubercle may have invaded the lung extensively. When empyema follows upon pneumonia the pulmonary inﬂammation sometimes is never resolved, the lung remains hepatised, and if the patient live long enough the hepatisation will tend to pass into cirrhosis. The corresponding branch of the pulmonary artery may be closed by a thrombus. And, lastly, extensive necrosis or erosion of the ribs may ensue, in which case the pus is not necessarily offensive.
Great deformity.—More or less contraction of the affected side is an almost necessary result of a healed empyema which has occupied the whole or the greater part of the pleural cavity. When the lung is totally unexpanded, the contraction will be great, the spine much curved, the mediastinum, heart, other lung, and diaphragm displaced towards the affected side. In course of time, the heart will become dilated, especially the right chambers, and this is one way in which the patient may die at last from the consequences of his empyema, even although it may have closed long ago.

V. Associated diseases.—Pleurisy is often accompanied with other diseases which impede or prevent recovery.

1. The pleura of the other side sometimes becomes inflamed, and the patient suffers from double pleurisy. Recovery, and quick recovery, is not uncommon; and even although the case be one of double empyema, appropriate treatment will usually cure the patient.

2. Collapse of the lung on the other side may occur in an infant and be necessarily fatal.

3. Gray induration (fibrous change, cirrhosis) will sometimes ensue upon collapse of the lung. But collapse may last for many years without being followed by fibrous change, a fact proved by examination post-mortem.

4. Gangrene of a portion of the lung may occur when festid pus penetrates it from an empyema—a serious complication.

5. Pneumothorax is often associated with pleural effusion, and in one of two ways. Either the pneumothorax and effusion occur simultaneously, in consequence of rupture of the lung, in which case the effusion is usually purulent, but may be serous; or the pneumothorax is secondary to the pleural effusion: an empyema has opened up a bronchus by ulceration, or has discharged through the thoracic wall, or, what is more common, a pleural effusion (serous or purulent) has been removed by paracentesis, and air has passed out of the lung into the pleural cavity, not through puncture of the lung, but through rupture of it by atmospheric pressure from within.

6. Tubercle of the lung associated with pleurisy has been already referred to. Also the fact that many cases of pleurisy are due to tubercle of the pleura, the source of infection being, in some cases, the bronchial glands, which lie at the root of the lung covered in places by nothing but pleura. Tuberculous pleurisy is attended by exudation of organisable lymph, or serum, or pus, or by haemorrhagic effusion. But pleural liquid effusion is sometimes concurrent with progressive pulmonary consumption, a complication which cannot be detected by physical examination until the effusion has been absorbed. Examination of the sputa for bacilli affords the only means by which pulmonary disease can be discovered during the presence of pleural effusion. When, as is sometimes the case, the phthisis is on the side opposite to that of the effusion, diagnosis is less difficult. More distant organs sometimes suffer from tubercle during the course of pleurisy, and thus
the patient's death may be hastened; the meninges of the brain are especially apt to be so affected in the young. Lastly, this seems to be a convenient place to say that a considerable proportion of persons who have recovered from pleurisy become tuberculous afterwards, and die within ten or twelve years from pulmonary consumption, or some less common form of tuberculosis.

7. Pericarditis often coexists whether the pleurisy affect the left side or the right. Sometimes, but seldom, perforation of the pericardium has taken place. In any case pericarditis is apt to go on to large effusion of serum or of pus. The pericardial effusion is usually not detected during life, the physical signs of that condition being hidden by those of the pleurisy. This is unfortunate, because the complication is very serious, and the patients generally die. Pneumopericardium as a result of pneumo-empyema has already been mentioned.

8. Peritonitis may concur. It is sometimes acute, purulent, and speedily fatal. Or it is chronic, and in this case is often tuberculous, ascites or universal adhesion being the result; the patient may recover even after his pleural effusion has been complicated by ascites. The certain diagnosis of acute and of chronic peritonitis is often impossible during life. When ascites is present, the legs are sometimes anasarceous; this condition also may end in recovery.

9. Dilatation of the heart sometimes follows pleurisy, especially when both pleuræ are obliterated by old adhesions, and when the lungs are imperfectly expanded. Under these conditions universal dropsy may ensue.

10. Dropsy, that is to say, anasarca and ascites, sometimes occurs even in acute pleurisy with effusion on one side only, there being no evidence of nephritis or of disease of the heart, and the patient recovering completely in about three months. In such cases the dropsy must be due to stagnation of blood in the right side of the heart.

11. Nephritis, indicated by the appearance of blood and tube-casts in the urine, sometimes occurs suddenly in the course of empyema under treatment by drainage. The nephritis will probably last four or six weeks and end in recovery. The cause is probably a morbid poison produced by the empyema. That pleurisy and pleural effusion are frequent complications of chronic nephritis may just be mentioned in this place.

12. Abscess of the brain is a consequence (not very uncommon) of empyema. The abscess is usually single, and occupies either the occipital or temporo-sphenoidal lobe: in a few rare cases many abscesses have been found. The abscess sometimes bursts into the lateral ventricle; and in this way even the subarachnoid space of the spinal cord may become filled with pus. This cerebral abscess is probably metastatic, and due to the transportation of a microbic embolus from the thoracic disease; but any other signs of pyemia are seldom observed either before or after death: why the white matter of the brain alone should be selected for embolism is unknown. The onset of
cerebral abscess is very insidious; for a long time the only symptom is headache of varying severity, sometimes little, sometimes much: so far as any distinctive symptoms go, the disease is latent. Towards the end, a few days or a week before death, much more decisive signs of disease are superadded to the headache; namely, vomiting, optic neuritis, general convulsions, coma. Or, as sometimes happens, the patient dies very unexpectedly, without the occurrence of any grave warning symptoms.

13. Hemiplegia due to softening of the brain is another possible consequence of empyema. No doubt the softening is sometimes caused by embolism of the middle cerebral artery; the embolus being derived from a thrombus which has formed in the heart or pulmonary veins during the stagnation of the circulation which is a necessary result of compression of the lung and displacement of the heart. Sometimes hemiplegia occurs during or soon after paracentesis, a thrombus or a portion thereof being dislodged during the commotion of parts which must follow upon removal of much liquid. In rare cases this hemiplegia is temporary, and the patient recovers in a few hours or days. But the softening of the brain which causes hemiplegia is not always to be explained by embolism; it may be that no arterial lesions of any kind are to be found after death; and a local metastatic encephalitis, not going on to suppuration, seems to afford the most probable explanation (6a). Other symptoms may depend upon the softening, according to its locality; namely, aphasia and associated defects; amaurosis, with a natural condition of the retina. Or the softening may involve both sides of the brain, with the consequences of general paralysis and dementia.

14. Lardaceous disease is a consequence which nowadays is seldom met with. In this case the empyema is usually chronic and fistulous; but even a small empyema which has never been discharged may be attended by this form of degeneration.

15. Clubbing of the finger-ends attracted much attention from the ancient physicians. The symptom may be well marked at the end of a fortnight from the beginning of an empyema. Clubbing will sometimes disappear gradually when empyema has been cured.

Samuel Gee.

Morbld anatomy.—The pleural affection does not necessarily vary with its exciting cause. Pleurisy, whether primary or secondary, may present the same appearances both to the naked eye and to the microscope. As in all serous inflammation, several factors are present: hyperemia, proliferation, and desquamation of the endothelium, proliferation of the sub-endothelial connective tissue cells, exudation of fluid, and escape of leucocytes from the blood-vessels into the cavity, the formation and deposition of lymph on the surface, and finally the organisation of the lymph into fibrous membrane or adhesions. The difference in different cases consists principally in the amount of fluid and in the proportion of fibrin and leucocytes which it contains; but on
the one hand the same exciting cause may produce in one case a "dry" pleurisy, in another a serous, and in a third a purulent effusion; and on the other hand these various forms may pass imperceptibly from one into another. The driest pleurisy is attended with some fluid exudation, and the clearest pleural effusion contains some fibrin and some leucocytes. These are, therefore, but stages in one process.

At the onset of pleurisy the surface of the membrane can just be seen to have lost its polish; and if the inflammation be more advanced, the fingers also can feel a slight roughness. This is due both to endothelial proliferation and to fibrinous deposit. If the disease go no farther, these products may disappear, and the membrane show no sign of the attack. But probably, in all cases which reach beyond the very slightest degree, both the parietal and visceral surfaces become affected, follow the plastic tendency of all serous membranes, and eventually form adhesions with one another. The extent to which this takes place varies from the production of a few fibrous threads to general adhesion of the whole apposed surfaces.

If the inflammation be more intense, there is exudation of fluid containing some fibrinous shreds. It varies from a few drachms up to an amount sufficient to distend the chest and displace the viscera. The amount of fibrin in it also varies greatly; though produced wherever the pleura is inflamed the fluid tends to collect at the lowest part. Occasionally this tendency is counteracted by adhesions, so that a fluid collection is limited to some other part of the chest than the base. Where it lies it takes the place of the lung, which, thus relieved from the suction of the chest wall, collapses beneath the fluid. When the exudation is large enough to exert positive pressure in the thorax, the lung is forcibly compressed also. While the fluid is effused, layers of lymph may at the same time be formed upon the pleura, and the membrane thus formed may so swath the lung that inspiration has not force enough to expand it as the fluid is removed.

An originally serous effusion may become purulent; but the great majority of purulent effusions are probably purulent from the first. The fluid in these cases varies greatly. It is sometimes liquid and laudable with very little fibrin; in others, and especially in chronic cases, the fibrin may form large curdy masses; in others, again, and especially in those of a septic nature, the fluid is much thinner than pus. The purulent or puriform effusion is usually inodorous; but it may become putrid when, by a wound of the chest or through the lung, access has been given to the open air; or when, as occasionally happens, the pleura communicates with an abdominal abscess or with the alimentary tract.

Lastly, the effusion may be bloody or may be almost pure blood.

Pathogeny.—The exciting causes of pleurisy are manifold. Those cases which are secondary to heart disease are, so far as we know, mechanical in origin. Pleurisy occurring in the course of nephritis may be of the same nature (2). But evidence is accumulating that under other conditions pleurisy is directly due to microbes. Purulent effusions were
the first to be studied from this point of view. Ehrlich found micrococi
in three cases of puerperal septicæmia with empyema. Rosenbach, Hoffa,
and Weichselbaum also verified their presence in all the cases of
empyema, eleven in number, which they examined." Kracht confirmed
this in ten cases. Fränkel examined twelve cases; in three cases of
primary pleurisy he found the streptococcus pyogenes; in three the dipl
coccus pneumonise; in four which were of tuberculous origin he discovered
the tubercle bacillus in one alone, the others giving negative results; and
in two cases secondary to other abscesses he again found the strepto
coccus. Meanwhile the influence of tubercle bacilli in the causation of
pleural effusions, whether serous or purulent, on which great stress had
been laid by Landouzy from the clinical point of view, had been studied
by Kelsch and Vaillard, Gombault and Chauffard, and Gilbert and Lion.
They were not very successful. Kelsch and Vaillard, inoculating in
animals, could only reproduce tubercle from two out of four empyemas,
and from one out of ten serous effusions. Gombault and Chauffard
failed nine times and succeeded eight times. Gilbert and Lion failed
altogether in twenty cases. Levy examined fifty-four cases, of which
thirty-seven were serous, seventeen purulent effusions; six were
secondary to typhoid fever, of which three contained staphylococcus
pyogenes, and three were negative; nineteen were secondary to
pneumonia, broncho-pneumonia, or influenza, of which three were nega
tive; fourteen revealed diplococci pneumonia, and two contained the
staphylococcus; in one of these also the diplococcus was found. In
fourteen tuberculous cases Levy failed to find the tubercle bacillus. In
one case secondary to rheumatism, and in seven secondary to heart
disease, nephritis, and cancer, the result was negative; but in one
hemorrhagic effusion secondary to infarct of the lung and in six other
mixed cases the staphylococcus was present. Renvers and Prince
Ludwig Ferdinand confirmed these results. Panseini in fifteen serous
effusions had five negative results, but found tubercle bacilli six times,
diplococcus thrice, and streptococcus or diplococcus once; in eight
empyemas he found tubercle bacilli thrice; and in all but one of the rest
the strepto-, staphylo-, or diplococcus. In one sanguineous effusion
he found the tubercle bacillus. Netter (13) examined 109 cases of
empyema: the diplococcus was present twenty-nine times alone, thrice
with streptococcus; streptococcus was found alone in forty-eight cases,
and staphylococci in two cases. Of fifteen cases of foetid effusion saprop
lytic organisms were found in all, and of twelve tuberculous cases the
tubercle bacillus was present in six. He points out the much greater
benignity of the diplococcus, and explains by this fact the more frequent
recovery of children; for of twenty-eight cases in children the diplococcus
was present alone or with the other two cocci in fifteen—a rate of 53
per cent, which is exactly that of the streptococcus in adults. In
a second paper (14) he stated that he had been able to produce tubercle
by inoculating guinea-pigs with the serous effusion drawn off by a Pravaz
syringe in seven out of twelve cases which could be diagnosed clinic-
ally as tuberculous, and in eight out of twenty cases of "idiopathic" pleurisy. Koplik gives confirmatory evidence of cases in children. Sacaze found tubercle bacilli at the beginning of a serous effusion, but failed to produce it later; which result throws some light on the difficulty always encountered in showing its presence, even when clinical evidence of a tuberculous process is strong. Hanot discovered the bacillus in a hæmorrhagic effusion (7) (21).

Both serous and purulent effusions, when primary, are therefore due to the three micrococci mentioned, and to the tubercle bacillus; and this so frequently, that as observers become more skilled this rule will probably be found universal. More than one of the above authors venture to state that where micrococci are not present the case will almost always prove to be tuberculous. Hæmorrhagic effusions when not due to cancer or to some rarer cause are also probably tuberculous.

The pathology of rheumatic pleurisy is as yet unknown; and the same may be said of the pleurisy which French authors (1) describe as occurring in the secondary stage of syphilis.

W. P. HERRINGHAM.

VI. Diagnosis.—Pleurisy is simulated by certain other diseases in respect either of symptoms or of physical signs.

1. The pain of pleurodynia is, by itself, indistinguishable from that of pleurisy. Diagnosis becomes possible when there are other signs or symptoms of pleurisy; for pleurodynia is mere pain, and pleurisy is sometimes indicated by pain alone.

2. The rale (not friction sound) which in rare cases attends the onset of pleurisy closely resembles the rale of bronchitis (see p. 352). The difference lies mainly in this, that the rale of pleurisy tends to be heard over one side only of the chest, and the rale of bronchitis over both sides. Pleuritic rale is soon superseded by other signs of pleurisy.

3. Acute collapse of extensive portions of lung is a condition which is very apt to occur in young children as a result of obstruction to a bronchial tube. The case of obstruction by an inhaled foreign body need not be considered here, for the whole course and symptoms of this accident are not at all like those of pleurisy with effusion. But bronchial catarrh, and even slight bronchial catarrh, will sometimes cause extensive collapse in a young child, or in a very feeble patient who is not a young child. Bronchitis setting in suddenly, with fever, cough, tightness of the chest, vomiting, and followed in a day or two by the signs of collapse at the lower part of one lung (namely, dulness to percussion and weak breathing), counterfeits pleurisy with effusion very closely. Diagnosis may be impossible at first. Usually the catarrhal infarct soon clears up; if it be deemed necessary a puncture may be made.

4. Chronic collapse of lung and Cirrhosis are two conditions which closely resemble each other in the living subject, and which often cannot be distinguished excepting upon the post-mortem table. Nor is it of any practical importance that they should be distinguished; the
useful term carination (invented by Laennec) may be taken to include them both. The physical signs of carination of the lower lobe of a lung and those of a small pleural effusion are the same, excepting that the chest may be distended on one side, and the heart be displaced away from the disease in some cases of local pleural effusion. But now and then the chest is contracted and the heart not displaced even in a pleural effusion. The symptoms afford no help to diagnosis, and the right understanding of a case may be rendered all the more difficult by the fact that carination is not only a constant result of pleural effusion, but often persists long after the effusion has disappeared (see p. 360). The chief means of distinguishing between the two conditions is puncture. Yet, under these circumstances, puncture sometimes fails to detect pleural effusion, and chiefly for this reason, that carination is often much more extensive than the effusion which causes it. For instance, a small pleural effusion, lying upon the diaphragm or in the posterior mediastinum, will sometimes be attended by collapse of the whole lower lobe of a lung; and this carified lung being the only portion of disease which is in contact with the chest walls, the physical signs will be wholly dependent upon the carination, and if puncture be made it cannot hit the effusion unless the needle go right through the lung. Wherefore it may be impossible to say whether there be an effusion or a mere carination. Sometimes the expectoration of a small empyema occurs so as to clear up our doubts.

5. Tuberculous phthisis of a lower lobe resembles a small pleural effusion in many respects which it seems hardly necessary to enumerate; puncture and microscopic examination of the sputa are the most trustworthy means of distinction. But there is an especial form of pleurisy which, for a time, is indistinguishable from pulmonary tuberculosis. In this case the pleurisy involves the whole of one side, which is retracted, it may be considerably, and moves much less freely than in health. The percussion note is raised in pitch and muffled over the greater part or the whole of the side; the sense of resistance is increased; when the disease affects the left side the superficial area of cardiac dulness is extended. The respiration generally is weak, and attended by friction sound at some part, or by widespread rale indistinguishable from the mucous rale of catarrh or phthisis (see p. 352). At places the breath-sound may be bronchial, in all degrees of intensity, up to perfect cavernous resonance. Add to these signs hectic fever with diarrhea and vomiting, and it is easy to understand why pleurisy of this kind is apt to be mistaken for phthisis more or less advanced. The pleurisy terminates in one of two ways. Either the physical signs of disease gradually disappear, excepting perhaps that a slight unilateral retraction of the chest, or a cup-like depression of the sternum, is left behind, the patient recovering at the same time his former state of health; or signs of a small effusion slowly appear at the base, and, when the chest is punctured, a little pus is withdrawn and the case comes into the category of empyema. Whenever the signs of a case of supposed
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phthisis are in some respects peculiar; whenever they indicate advanced and extensive disease, but limited to one side of the chest; whenever cavernous signs are heard in unusual places;—it is well to weigh the possibility of simple pleurisy, and not to rest confidently in the diagnosis of phthisis until tubercle bacilli have been found in the sputa.

6. Acute pneumonia is seldom mistaken for pleural effusion unless the tubes of the pneumonic lung be so plugged with mucus that conduction of the breath-sounds is obstructed. It much more often happens that a small pleural effusion is mistaken for pneumonia. The physical signs of the two diseases may be the same, and even puncture is not always decisive; should an empyema be confined to the apex of a pleural cavity, so infrequent an occurrence, compared with the frequency of apex pneumonia, will render diagnosis unusually difficult (see p. 358). The symptoms of the two diseases may be the same, especially in the pleurisy which is due to pneumococcus (see p. 352); not seldom in this case the patient dies before certain diagnosis becomes possible: a physician well read in the book of nature knows that he cannot always distinguish between pleurisy and pneumonia.

Chronic pneumonia—that is to say, hepatisation slow to resolve—will resemble in many respects pleural effusion supervening upon pneumonia.

7. Malignant tumour of the lung closely resembles pleural effusion in respect of the physical signs. A tumour does not often cause enlargement of the affected side, or displace any organs, yet now and then a quickly growing tumour will produce these effects. When dulness begins not at the bottom of the chest; when there is a great extent of absolute dulness in front and none behind; when, in the midst of a great extent of dulness, we detect one or more small insulated patches of resonance (perhaps quite clear or even cracked-pot), we may debate the existence of solid tumour. The crucial test is puncture.

A large serous effusion (see p. 362) is sometimes the necessary result of contracting cancer of the lung. The nature of the case may be suspected if cancer can be discovered elsewhere, and especially if large hard glands can be felt above the collar-bone or in the armpit.

8. A large hydatid cyst will yield most of the signs of pleural effusion; namely, unilateral distension of the chest, displacement of the diaphragm and mediastinum, dulness to percussion, and weak or absent breathing sound. An exploratory puncture is the most decisive means of diagnosis; the fluid of hydatid being free from albumin and more watery than that of pleural effusion, to say nothing of the possible discovery of echinococcus hooks. But if the hydatid have suppurred, the nature of the disease is sometimes not suspected until a free opening has been made, such as to permit the escape of hydatid membrane. (For full discussion of Thoracic Hydatid, vide vol. ii. p. 1137.)

9. Actinomycosis of the base of the lung simulates pleurisy with effusion, and is, indeed, sometimes attended therewith. The diagnosis...
cannot be made until the fungus is discovered in the sputum, or until the growth perforates the wall of the chest (vid., vol. ii. p. 81).

10. Subphrenic abscess is much more common on the right side than on the left, for reasons which become clear when the antecedents of the abscess are considered. It is often, if not always, associated with pleurisy on the same side, and usually with empyema, due to perforation of the diaphragm or not. Hence empyema on the right side in a person who has probably suffered from tropical hepatitis, from simple or cancerous ulcer of the stomach, or from other causes of subphrenic abscess, should always lead us to reflect upon the possible coexistence of this disease. The pus of subphrenic abscess and of the empyema is putrid. The abscess, even if there be no empyema, may burst into the lung, and lead to expectoration of most offensive pus. Whether there be an associated thoracic empyema or not makes little difference so far as the physical signs of a subphrenic abscess are concerned; for the empyema is local, enclosed in adhesions, and not nearly filling the pleural cavity. The signs are both abdominal and thoracic, sometimes more the one, sometimes the other. The abdominal signs are: (a) fulness and tightness in the hypochondrium; (b) the liver depressed, sometimes, but by no means always; moreover the liver is sometimes much depressed in uncomplicated thoracic empyema. The thoracic signs are: (a) dulness to percussion and signs of pleural effusion at the base, whether there be a pleural effusion or not; in the latter case the diaphragm is much pushed upwards, and the lung proportionally collapsed; (b) the heart's apex beat is sometimes displaced even in subphrenic abscess without empyema, but more often is not displaced. Puncture, made as for pleural effusion, will probably reveal the presence of pus, but will not tell us whether the pus is above the diaphragm or below it. Uncomplicated subphrenic abscess may be mistaken for simple thoracic empyema, even after the abscess has been emptied of pus by aspiration; the needle having gone right through the diaphragm, which has been pushed much upwards, as high it may be as the third rib. Even resection of a portion of a rib, and exploration of the pus cavity by the finger, do not always enable us to say at first whether we have opened a cavity above or below the diaphragm; or, in the former case, whether the diaphragm be perforated or not.

Subphrenic abscess often contains gas derived from perforation of the alimentary canal or from decomposition. In this case the disease is apt to escape discovery by physical examination, because there is no dulness to percussion. Sometimes the percussion note is clearer than natural; and sometimes the clear note is more extensive also, so that the liver dulness disappears. The resonance may possess amphoric quality. Auscultation usually detects one or more signs of a large cavity containing air; namely, amphoric hum (attending the sounds of breathing, speaking, and of the heart), metallic tinkle, bell sound, and succession splash. If the diaphragm be perforated, the empyema will be a pyopneumothorax.
11. It is sometimes hard to decide whether friction sound heard over the heart region be pleural or pericardial. Pleural friction may be produced by movement of the heart alone; as pericardial friction may be under the influence of breathing movements.

12. Large effusion into the left pleura may cause bulging of the chest in the heart region, such as to raise the question of concurrent pericardial effusion; for the two diseases are often associated (see p. 369). The diagnosis depends mainly upon the result of emptying the left pleura by paracentesis, whereby alone can the signs of pericardial effusion become manifest. When a rib has been resected in the treatment of empyema, the finger passed into the pleural cavity may possibly be able to feel a bulging pericardial sac.

13. When pericardial effusion is attended by extensive collapse of lung, and the chest is punctured with a view to determine the cause of the dulness, the needle may go right through the lung and discharge liquid from the pericardium; and, until examination post-mortem, the physician may rest in the unshaken belief that the liquid came from the pleura.

14. The manner in which pulsating empyema counterfeits aneurysm has been already referred to (p. 358).

15. An abscess in the thoracic walls may be the only evidence of a small empyema (see p. 360) which has penetrated an intercostal space. Even when the abscess has been opened it is not always easy to say whether it communicates with the pleural cavity or not. It is possible that pleurisy may be the cause of abscess in the thoracic walls without actual perforation of the pleura. But more commonly parietal abscess (as distinguished from pointing empyema) is due to such causes as injury, pyæmia, periostitis of a rib, or necrosis of the same; and this "peripleuritis" may perhaps sometimes set up pleurisy. Lastly, in all cases of superficial abscess the question of actinomyosis must be pondered.

VII. Prognosis.—It seems to be unnecessary to reiterate many prognostics, which will be found in their appropriate places in the foregoing and following pages. But one fact of great importance demands special attention, namely, the occurrence of unexpected and speedy death in cases of pleural effusion. The conditions of this unexpected death are not always the same.

(i.) The sudden rupture of an empyema (and it may be quite a small empyema) into the lung is sometimes sufficient to suffocate the patient in a few minutes.

(ii.) Much more often the death occurs apart from any discharge of the effusion. The effusion is usually large, filling up the whole or greater part of the pleural cavity. The effusion is usually serous. Whether it be on the right side or on the left makes no difference. Suddenly, and often after a little exertion, the patient is seized with dyspnoea or faintness, or both. The lipothymial symptoms soon predominate; the skin becomes cold and clammy or sweating, the face and lips assume the wan,
dusky, livid colour of a dying person, the pulse is small and irregular; death ensues within half an hour or an hour. The explanation of the speedy death is mostly found post-mortem in thrombosis of the right side of the heart, consequent upon stagnation of the circulation through it, dependent upon the collapsed state of the lung. This heart thrombus has one of two results: either the thrombus is propagated into the pulmonary artery, and thence into that branch of it which supplies the unaffected lung; or an embolus, derived from the heart thrombus, is driven into the pulmonary artery, or a large branch of it. But thrombosis meet to explain the death is not always found: sometimes a latent pericardial effusion is present; but sometimes nothing sufficient can be found, and in cases of this kind hypothetical explanations have been offered, such as twisting of the large vessels at the root of the heart, bending of the inferior vena cava at an acute angle, compression of one auricle of the heart, degenerative changes in the muscular tissue of the heart.

(iii.) Frothy serous expectoration sometimes suffocates the patient during or soon after paracentesis of the chest (see p. 376); or, in very rare cases, may even supervene upon large effusions apart from paracentesis.

VIII. Treatment.—A. In the treatment of pleurisy with no liquid effusion, the main indication special to the disease is to relieve pain. The most effectual means of doing so are two: subcutaneous injection of morphia at the seat of pain, or the application of a few leeches. In many cases much less decisive means are sufficient: warmth by hot-water fomentations or linseed-meal poultices; a mustard poultice, or a turpentine fomentation.

B. The treatment of pleurisy with effusion relates almost wholly to removal of the effusion.

When the effusion is believed to be recent, not large, and not purulent, it is best to defer operation for a week or two, so as to see whether the liquid can be removed spontaneously without operation. It is probable that absorption may be assisted by sundry means: iodide of potassium in moderate doses should be given; the affected side of the chest should be painted with tincture of iodine two or three times a day; blisters, the size of the palm of the hand, or less according to the size of the patient, may be employed, one blister at a time, and the sore allowed to heal as soon as possible. In the case of children blisters should not be used.

But the question of paracentesis is always foremost in the mind, and may be discussed under four heads: when, where, and how the operation should be performed, and, lastly, certain dangers which sometimes attend the operation. The age of the patient is never taken into consideration. I have treated successfully by paracentesis patients three months old and eighty-seven years old.

I. *When should paracentesis be performed?*—The answer to this question depends upon the quality of the effusion.
1. Pus must be removed as soon as possible. If it be bloody, or if the pleura contain air as well as pus, the same rule holds good. Free evacuation of pus may be expected to bring the patient's temperature down nearly or quite to the normal; if this be not the result, we may assume that there is some retention of pus. Any subsequent rise of temperature, after a fall to the normal, will most likely be due to imperfect drainage. But perfect drainage cannot always be attained, especially when a small quantity of pus is secreted in an inaccessible cavity shut off from the rest; in cases of this kind time usually surmounts the difficulty, the retaining lymph breaking down under persistent drainage.

2. Serum should be removed by paracentesis in all cases which present an effusion so great as to fill the pleura, or which are attended by any distress of breathing, or which show no signs of being absorbed after a week or ten days of the other treatment already described.

II. Where should paracentesis be performed?—1. When the effusion is small the puncture must be made where the effusion is believed to be.

2. When the effusion is great, so that the pleural cavity is full or almost full, the best place for puncture is in the middle line of the axillary region, about the horizontal level of the nipple or a little below it, where the intercostal spaces are wide and the muscular integuments thin. Another part of the chest which is often chosen for puncture is a spot just below the angle of the scapula, but the lung is sometimes adherent to the chest wall here, and will therefore be pierced by paracentesis; in this case pneumothorax is apt to ensue, and, what is a result far worse, but less frequent, sloughing of the perforated lung. Probably no part of the chest can be chosen as being entirely free from the risk that paracentesis may perforate collapsed and adherent lung but the risk is less at the spot first recommended for the place of puncture than at any other situation.

III. How should paracentesis be performed?—1. Serous effusion should be removed by means of a trocar and canula. Whether suction be employed or not is, in most cases, a matter of no great consequence. If suction be not employed, a canula connected with a long india-rubber tube should be used, the free end of the tube being kept under liquid, so that no air can enter the chest. On the whole, suction is to be preferred, for in this way small obstacles due to fragments of lymph floating in the serum can be overcome. It is best to make no more vacuum than is necessary to maintain a gentle flow of liquid. As much liquid is to be drawn off as possible without causing any serious discomfort to the patient. Suction is to be stopped if the flowing fluid become bloody, if the patient feel much pain in his chest, or if he begin to cough much; in which last case there is the risk of serous expectoration (p. 376).

The pain of puncture is diminished if the skin be previously frozen by ice, or by an ether or chloretethyl spray.

It happens sometimes, but not often, that the most powerful suction
can extract no more than a small quantity of the effusion. The usual cause of this difficulty is found in a fragment of lymph which blocks the canula or obstructs its orifice. But sometimes, even when the effusion is free from floating lymph, it is impossible to evacuate the chest. Cases of this latter kind, which are uncommon, are probably to be explained by a lung rendered inexcisable by thickened pleura or by obstructed air-tubes. Nothing more can be done than to draw off as much serum as possible, and to repeat the paracentesis in a day or two.

Very often a single paracentesis cures the patient, the little liquid left being soon absorbed. But sometimes the effusion returns, and the rule of practice is to repeat the operation as often as seems necessary. In rare cases an abundant effusion will continue for an indefinite time, but even then the only treatment is paracentesis repeated as often as necessary. Drainage by a permanent opening is out of the question, and would be certain to convert the serous effusion into empyema, to the great danger of the patient's life.

There is no reason for fear lest paracentesis alone and without drainage should convert serous effusion into pus, provided that all the instruments used be surgically clean.

2. Emphyema is to be treated by incision and drainage.

(i.) When the quantity of pus is not very large it is best to make a permanent opening and drain at once. In some cases thorough and speedy drainage cannot be obtained unless a large opening is made by excising a portion of one of the ribs; and, therefore, to avoid all doubt upon this point it is good practice to resect a rib in all cases.

(ii.) When the empyema fills the pleural cavity it is safer to remove as much of the pus as possible by paracentesis at first, and to make the incision a day or two afterwards. The sudden discharge of a very large quantity of pus from the chest causes a great shock to some patients, and previous paracentesis lessens the shock. Paracentesis, and sometimes even a single paracentesis, can cure empyema. I have known a single paracentesis cure a stinking empyema of considerable size. And I have known paracentesis, which removed more than five pints of pus from the pleura, to be followed within a week or two by effusion of clear serum to the same amount in the same pleura. But cases of this sort are very uncommon, and incision and drainage are the proper treatment of empyema.

In order, then, that the drainage may be thorough it is best to remove a small portion of one of the ribs. Incision is made right down upon the rib, the periosteum is separated all round by an elevator, and the rib is divided in two places by cutting forceps, so that about an inch can be removed. It is not good practice to swill the empyematous cavity out; nothing is gained by removing false membranes in this way; a fetid empyema is soon deodorised by thorough drainage and careful antiseptic dressing, and even if not, washing out does not help. Moreover, injections are dangerous if there be an ulcerous opening through which they can enter the lung; the shock which immediately ensues upon
such an entrance puts life in jeopardy. Even if there be no such ulcer, injections are dangerous. I have known a patient die very suddenly during injection, when but a very small quantity of a weak carbolic acid solution had been injected; no chloroform was given, and nothing could be found post-mortem to explain the death. But sudden syncope coming on in this way is not always fatal. The cause of the syncope is not understood; a case has been recorded in which the right chambers of the heart were found post-mortem to be distended with gas. The syncope is sometimes followed by convulsions and coma; in this case death usually ensues within twenty-four hours. If the temperature rise much above 105° recovery is very unlikely. Yet recovery even after convulsions may occur; temporary palsy of a limb or of one side of the body has been noted upon cessation of the convulsions. In other cases the sudden syncope has been attended by palsy without convulsions, by spastic rigidity of a limb or of the jaw, or by aphasia; these symptoms commonly pass away in an hour or less. Convulsions and paralyses of this kind are probably epileptoid in nature, and quite different from the paralyses which will be spoken of hereafter, and which are due to embolism.

If there be any probability of the coexistence of pulmonary tubercle the line of treatment is not so clear. To release the lung from compression may accelerate the infective and destructive changes going on therein; to say nothing of the debilitating effect of a free purulent discharge, which there is but small chance of stopping. Under these circumstances it is best to resort to paracentesis several times at least, the result being watched before proceeding to drainage.

In dealing with small deeply-seated empyema, such as that which so often leads to fetid expectoration, it is sometimes necessary to remove portions of two or three ribs, so that adhesions can be broken down and the cavity opened by the finger. To cut through the lung in such cases many cost the patient his life.

For a day or two after opening the chest the discharge will probably continue to be abundant. It will then, in most cases, gradually lessen until it ceases altogether in a few weeks, three or more. In proportion as the discharge becomes scantier the drainage-tube must be shortened, so as to allow the sinus to heal from the bottom.

The temperature ought to fall almost or quite to the normal after the pus has been discharged. Should the temperature remain raised, there must be either retention of pus or some other concomitant disease. When, after the fever has ceased, the temperature rises again there is probably retention of pus, and should the temperature not fall again in a few days the sinus should be probed, and a longer piece of tube be inserted if necessary. But the temperature will often rise for a few days without obvious retention of pus, and will fall again without obvious increase in the amount of discharge.

When the sinus shows no tendency to close it is best to wait two or three months before undertaking any further operation. But when
the discharge continues for a longer time (and these remarks apply also to cases of neglected empyema which has been allowed to open spontaneously), and it seems necessary that something else should be done (especially when the discharge remains abundant and the health of the patient suffers), a more extensive operation must be performed. Longer portions (two or three inches) of three, four, or more ribs in the neighbourhood of the sinus must be resected, so as to allow the chest walls to fall in and meet the lung. The cases in which the discharge is not finally arrested by this operation are very few.

IV. Dangers of paracentesis.—1. Serous (or albuminous) expectoration. Paracentesis, by suction, of a pleural effusion is sometimes followed by expectoration of blood-serum. If a patient begin to cough much during the operation it must be stopped at once, and the patient be carefully watched. It is very probable that a small amount of serous expectoration under these circumstances is not uncommon; it is only when the secretion is abundant that the condition is dangerous and apt to end in speedy death. Serous expectoration mostly ensues during or directly after the operation, but sometimes an hour or two will elapse before the secretion becomes dangerously abundant; the latter cases are less serious. When abundant serous expectoration follows rapidly upon paracentesis the patient may die suffocated within half an hour. The stuff expectorated is frothy, viscid, transparent, neutral, or alkaline, yellow or yellowish green, with a specific gravity of about 1020, and rendered almost solid by heat and a drop or two of acetic acid. Chemically the sputum consists of serum-albumin and a little mucin. On standing there falls a scanty deposit of pus and blood corpuscles. Post-mortem the lung is oedematous, and usually fully expanded. Concomitant disease, such as disease of the heart, mediastinal tumour, or hæmoptoic infarcts, favours the occurrence of serous expectoration.

2. Pneumothorax sometimes follows withdrawal of a pleural effusion. The cause is not always the same. (i.) In some cases the lung has been injured by the operation, an accident especially apt to occur when collapsed lung, undiscoverable by physical examination, is adherent to the chest wall, so that the trocar goes through the lung. (ii.) Sometimes the air comes from a spontaneous rupture of the lung; softened tubercle may give way; in empyema there is sometimes a small ulcerous breach in the surface of the lung existing before the operation, or merely collapsed lung may burst in some small spot in the process of expansion during paracentesis by suction. (iii.) The pus of empyema sometimes contains so much gas dissolved in it that in some cases this is a very probable cause of pneumothorax.

3. Hæmorrhage from the pleuritic membranes is sometimes the result of paracentesis. If the blood flow at all freely, the operation must be stopped, and it is seldom that any bad consequence follows. But death has been due to this cause, the pleural cavity being found post-mortem to contain a large quantity of blood.

4. Fatal hæmoptysis has ensued upon evacuation of empyema in
pulmonary phthisis, which has gone on to the formation of cavity containing a small aneurysm.

5. Embolism of distant arteries, by coagula dislodged from the pulmonary veins, may be the result of paracentesis. The most common result is hemiplegia (see p. 364), which is usually incomplete. Embolism of both iliac arteries has been known to occur.

6. Sudden death has followed soon after paracentesis in rare cases, even when the pleura has not been washed out (see p. 376). In one case of this kind the right side of the heart was found to be filled by a clot, formed in all probability during life. In other cases no satisfactory explanation of the death has been forthcoming; all operations involve the possibility of the patient dying suddenly.

The treatment of fetid expectoration from a small deeply-seated empyema, which cannot be laid open by an operation, is the same as that of a similar condition in phthisis.

Subsequent deformity. — Not much can be done to expand the collapsed lung, and to counteract the deformity of the thorax and spine which is apt to follow upon chronic pleurisy. Exercises for the arms should be prescribed, especially such as tend to open the chest in front; for instance, drawing the body up by the arms clinging to a horizontal bar, skipping backwards, the use of a chest-expander behind the back, or of dumb-bells and appropriate drilling.

Samuel Gee.

REFERENCES


S. G.

W. P. H.
PNEUMOTHORAX

Hydropneumothorax; Pyopneumothorax

Definition.—By pneumothorax is meant the presence of air in the pleural sac. Generally speaking, the air or gas is accompanied by serous fluid or pus; hence the synonyms hydro- or pyo-pneumothorax to denote one or other of these composite conditions.

Etiology.—Although causes leading to the production of pneumothorax are fairly numerous, most of them, as detailed by various observers, are of remarkably infrequent occurrence; indeed the disease itself may be said to be rather uncommon.

The oft-quoted statistics of Saussier (12) give the relative frequency of the causes in 131 cases as follows:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phthisis</td>
<td>81</td>
</tr>
<tr>
<td>Empyema</td>
<td>29</td>
</tr>
<tr>
<td>Gangrene of lung</td>
<td>7</td>
</tr>
<tr>
<td>Emphysema of lung</td>
<td>5</td>
</tr>
<tr>
<td>Apoplexy of lung</td>
<td>3</td>
</tr>
<tr>
<td>Fistula between pleura, liver, and intestine</td>
<td>2</td>
</tr>
<tr>
<td>Abscess of lung</td>
<td>1</td>
</tr>
<tr>
<td>Cancer of lung</td>
<td>1</td>
</tr>
<tr>
<td>Hemotherax</td>
<td>1</td>
</tr>
<tr>
<td>Hydatids</td>
<td>1</td>
</tr>
</tbody>
</table>

Even this considerable list is by no means complete, and several additions have to be made—most of them, however, of rare occurrence. For example, pneumothorax may be brought about by external injury—such as a perforating wound of the wall of the chest, by the wounding of the visceral pleura by a fractured rib, or even by heavy blows or falls, apart from wound or fracture. Indeed, cases of pneumothorax occur from time to time of which no cause is discoverable (6).

Internal injury also must be credited with the production of some cases of the disease, as when a bougie, in its passage through a cancerous oesophageal stricture, has perforated the diseased wall of the tube and entered the pleural sac. Ulceration of a bronchial tube, however produced, is another possible cause; perforation of the diaphragm, brought about by suppuration resulting from a perforated gastric ulcer, another; and finally, cases occur in persons, otherwise apparently in robust health, as a result of strain, that is, of strenuous muscular effort with the glottis closed. All these varieties of causes group themselves in two divisions: the one containing those in which the perforation causing pneumothorax results from injury or disease directly affecting the lungs themselves, or the bronchia (and this is the more important); and the other containing those in which the causes of perforation are external to the pleura. An additional variety is attributed by some authors to the gaseous decomposition of liquides, such as pus, pathologically present in the pleura: this, if it ever occur at all, is infinitely rare; there is good reason to doubt whether it does occur.
All observers agree that pulmonary tuberculosis, producing perforation by ulceration of the visceral pleura, is by far the most frequent cause of pneumothorax; and most of them place the proportion of such cases at about 90 per cent of the whole. The relative frequency of the disease in cases of phthisis is variously stated by different authorities as being from 3 to 14 per cent (4). My own experience would suggest the smaller number as being nearer the general average. It was found present twice only in 60 post-mortem examinations made on cases of phthisis at the Middlesex Hospital in the years 1877, 1878, and 1879.

It should be added that men are more apt to be attacked by pneumothorax than women; and it is a disease especially of the earlier periods of adult life. This latter is to be expected from its connection with pulmonary tuberculosis.

Pathology and Morbid anatomy.—The mode in which air gains access to the pleura in such cases as those of external injury, or the bursting of an empyema, are so obvious as to require no explanation. With regard to the tuberculous cases, which, as we have seen, form an enormous majority of the whole, it is in the acute forms that pneumothorax is most apt to occur. Those in which the disease of the lung progresses slowly are comparatively little likely to perforate, owing to the formation of protecting adhesions between the visceral and parietal layers of the pleura. In the former class of cases tuberculous masses become softened, and break down close under the surface of the lung; necrosis of the overlying portion of the pleura takes place; and some effort, or an attack of coughing, is sufficient to determine a rupture, or it may occur without any apparent exciting cause.

In connection with this portion of the subject it is interesting to note how the conservative processes of nature tend to the prevention of pneumothorax. In the more slowly progressive cases of pulmonary tuberculosis perforation of the pleura is anticipated by the formation of inflammatory adhesions—such inflammation being apparently set up by the commencing necrosis of the pleural tissue. Were it not for this, pneumothorax, instead of being a somewhat uncommon event, would be a very frequent if not an invariable incident in the course of caseous tubercle in the lungs.

In a few instances the perforation seems to take place by the extension towards the surface of a cavity itself, or by a sinus proceeding from the cavity. With very rare exceptions the disease is unilateral; and the left pleura is much more frequently the seat of the lesion than the right. Usually there is only one perforation, which may be found at almost any part of the lung. The common site, however, is the lower part of the upper lobe, or the upper part of the lower lobe; and the reason for this is that the higher parts of the lung are usually the seat of pleural adhesions, which, as we have seen, prevent perforation.

The size of the perforation varies much; in great degree according to the length of time the patient survives. It may be large enough to admit the tip of the finger, or so small as to be discerned with difficulty;
indeed, it is often not discovered at all, being overlaid with lymph which has become organised in the repair of the mischief. The opening may be direct or valvular; and these conditions have an important bearing on treatment and prognosis, as well as on the amount of suffering to which the occurrence of the lesion gives rise. When the perforation takes place, the elastic traction of the affected lung is neutralised, and the heart and mediastinum are displaced towards the sound side. If the opening be direct and free, air passes out of the pleural sac as well as into it, and there may be no intrapleural pressure; if, on the other hand, the opening be valvular, air enters the pleura during inspiration, and as the respiratory movement is reversed, the valve closes so that no air can escape: the consequence is that the pleura gradually becomes as full of air as bulging of the chest, shrinking of the lung on the affected side, depression of the diaphragm, and displacement of the mediastinum will permit. (Vide article "Intrapleural Tension," p. 335). The quantity of gas present depends on various circumstances—chiefly on the presence of serum or pus in the pleura, and the condition of the lungs themselves, especially of that which is perforated. Adhesions and consolidation tend to minimise the quantity, while the opposite conditions favour the largest possible accumulation.

The gas itself, as regards its chemical composition, very much resembles expired air; it consists of nitrogen with oxygen and carbonic acid, together with sulphuretted hydrogen in cases where a fetid liquid is also present in the pleural cavity. The proportion of oxygen and carbonic acid may vary from time to time; but this matter, however interesting, is of no practical importance.

When a rupture of the pleura is due to one of the simpler causes—such as injury or the giving way of an emphysematous vesicle, the opening is soon closed, the air becomes absorbed, and the previous state is completely restored. But it is different with the tuberculous perforation: here, owing to the leakage of septic liquid from the pulmonary cavity into the pleura, acute inflammation of the pleural membrane is set up, which may be both intense and widespread. Following this comes more or less rapid effusion, which is most likely to be purulent.

On post-mortem examination the escape of pent-up air, when the cut is made through the chest wall for removal of the sternum and rib cartilages, may bear witness to the intrapleural pressure which sometimes exists. The mediastinum and heart are displaced towards the sound side; and shrinking of the affected lung, much or little according to its condition as regards intrinsic disease or adhesions, will be observed. Where the pneumothorax has lasted for some time the pleural surfaces are covered with quantities of lymph (the result of the pleurisy), which, as before mentioned, may render the discovery of the perforation difficult or even impossible. In ordinary cases the lung may be adherent in part to the chest wall at the apex, and may be the seat of cavities and of nodules of caseous tubercle. The opposite lung may show a similar state; or, if the perforation have occurred early in the history
of the tuberculous condition, it may be perfectly sound. The pleura contains serous fluid or pus, the quality of the liquid as well as its quantity depending to some extent upon the time which has elapsed since the occurrence of the perforation. Exceptions to this rule, however, may be found in cases which have proved rapidly fatal, as there may not have been time for an obvious effusion to take place.

Symptoms.—In the ordinary case the patient, perhaps during a fit of coughing, is attacked by agonising pain in the chest, a feeling as of something having given way, and perhaps of fluid trickling down inside his chest, together with great difficulty of breathing. Any of these symptoms, however, may be wanting; in some cases all of them may be comparatively inconspicuous. Nor will this appear strange when we consider that the accident, as it may be called, of pneumothorax often occurs in patients already acutely ill, with rapidly eacting or softening tubercle, probably confined to bed, and suffering from respiratory discomfort and thoracic pain. Pulse and respiration rate are both increased, the latter more so than the former; the patient is cyanosed, the expression anxious, the alas nasi working, the heart palpitating, the extremities cold, the voice weak, the temperature lowered, and the body bathed in cold sweat—in fact, as regards his general condition the patient is in a state of collapse. Dyspnoea, which is perhaps the most characteristic of the symptoms, is often extreme and distressing, the patient feeling as if he were about to be suffocated. It is most marked when the perforation through the pleura is valvular, because the condition producing dyspnoea is aggravated with every inspiration; and it may readily happen, especially if the function of the opposite lung be impaired by disease, that the case may speedily have a fatal issue. The decubitus of the patient varies a good deal in different cases. There may be orthopnoea, or he may lie half propped up on the back, or on either side. In a case recently observed the position chosen was semi-prone towards the sound side, with the head low.

Physical signs are often more definite than the symptoms. The following points are to be noted:

Inspection.—The shoulder of the affected side is elevated, the intercostal spaces partially or wholly obliterated, the side distended, and the movements of respiration diminished or altogether absent. The respiratory movements of the sound side are correspondingly exaggerated. The heart's maximum impulse may be seen displaced towards the sound side; although, owing to the rapid and disturbed respiratory movements together with the weakness of the heart's action, it may be difficult to make out.

Pulvation.—This means may enable the last-named point to be more distinctly perceived; and by it we can also appreciate the diminution or abolition of respiratory movements: tactile fremitus is also abolished. Displacement downwards of the liver or spleen may be observed according to the side affected; and the displacement may be very considerable in amount if the pleural cavity contain much air or liquid, or both. This change has an important bearing upon treatment, since downward dis-
placement of the diaphragm forms such a large pocket for the accumulation of pus that its amount is very apt to be underestimated; thus steps for its prompt removal may not be taken.

**Percussion.**—The presence of air in the pleura gives rise to a marked change in the percussion resonance; the note is over-resonant, and may generally be described as tympanitic. When the tension of the walls, however, becomes very great, there is a change in the note, so that it is shorter and of higher pitch, and hence of a less tympanitic quality. The "cracked pot" sound might be expected in cases where the perforation between the lung and pleural cavity is open and free; and some writers state that it is present occasionally, although rarely. The characteristic note may not be made out over the whole of the affected side; adhesions fixing a portion of lung to the thoracic wall may prevent it, and this condition is of course most frequently observed at the apex of the lung. Or the presence of an accumulation of liquid—purulent or otherwise—at the base of the pleural cavity will cause a dulness in the percussion note over the area so occupied. In the latter case the dulness and tympanitic resonance may be made to alter their relative positions by changes in the position of the patient's chest.

The normal area of cardiac dulness is abolished in cases of left-sided pneumothorax; and in any case, owing to great displacement of the mediastinum, the tympanitic note often encroaches considerably on the sound side.

Lastly, there is what is known as the bell sound, the "bruit d'airain" of Trousseau, an interesting phenomenon which may be said to belong partly to the domain of percussion and partly to that of auscultation. It is recognised when some part of the side which is distended with air is auscultated, while a coin placed on another part is struck with another coin or some similar hard substance, such as a key. The sound conveyed to the ear of the listener is of a ringing metallic quality often closely resembling the tinkling of a small bell.

**Auscultation.**—When the opening is valvular, and the pleura has become as full of air as possible, no breath-sound may be audible, except perhaps along the spine where the compressed lung lies; but when the opening is patent, breathing of an amphoric quality is well heard, as a rule, both with inspiration and expiration.

It was formerly thought that there must be a passage of air through the perforation in order that breath-sounds may be heard, but this opinion is no longer held. If air enter and leave the lung at all, as it may do in parts where adhesions have prevented complete collapse, breath-sounds of the quality referred to, although distant and feebly conducted to the ear of the observer, may often be heard, even through the pneumothorax.

The amphoric breath-sound, when present, is most likely to be easily detected just over the site of perforation. Voice and cough sounds have a metallic ring in cases where the opening into the pleural cavity is free; and, in connection with the cough especially, the phenomenon known by the name of "metallic tinkling" is often well heard. It is not due, as
was thought by Laennec, to drops of fluid falling in the air-filled cavity; but it may be produced by various adventitious sounds having their origin in the lung.\footnote{In a case seen by me about fifteen years ago in a healthy, athletic young man of some twenty years of age, the air escaped into the pleural cavity with a succession of tinkles or clicks. These were audible in all parts of the large room, and continued until the family medical attendant arrived, probably two hours, so that he also heard them plainly. Before my arrival they had ceased. I suggested that the sounds were due to a rupture of a tiny bubble at each issue of air. Their frequency varied, they came much faster at first and grew rarer. Inspiration, at any rate at first, increased the number and loudness of the tinkles. The rupture was brought about by an attempt to bend the body backwards so as, if possible, to touch the ground with the hands without removing the toes from a line. The patient, whose pneumothorax on our examination was considerable, soon got well and has remained well.—Ed.}

Finally, there is the succussion sound, often associated with the name of Hippocrates, because it was first described by him. To elicit it, the patient, preferably sitting up, is sharply jolted or shaken, while the observer has his ear applied to the chest; or, if not acutely ill, he may be made to shake himself so as to bring out the sound. It is caused by the splashing of the liquid effusion in the cavity containing also air, just as it would be produced in a cask having similar contents; and it is of the metallic ringing quality which characterises all the adventitious sounds of pneumothorax. The patient himself may be conscious of the presence of fluid in his chest, while under examination he may both hear and feel the splashing of the fluid.

**Diagnosis.**—As many of the phenomena accompanying the majority of cases of pneumothorax are of a definite and striking character, the diagnosis, generally speaking, is not a matter of much difficulty. The essential points are: over-resonance; absence or great enfeeblement of breath-sounds (these, if present at all, being of amphoric quality); displacement of the heart, and the bell sound. These are perhaps more than enough for diagnosis; and they are necessarily strengthened if we have a history of sudden attack of pain in the chest with dyspnea. The only class of cases at all likely to give rise to doubt are those in which the pneumothorax is partial, and limited by old adhesions between the pleural layers.

From emphysema, which in some points may seem to resemble pneumothorax, the distinction is easily made by the fact that emphysema is bilateral, and that in it there is no lateral displacement of the heart and no bell sound; also, that the resonance of emphysematous lung is not so tympanitic as is the rule in pneumothorax. It must be admitted, however, that rare instances occur in which the distinction is a fair point for discussion. I can recall two such cases: the diagnosis of emphysema, however, was duly made in both cases.

From a large pulmonary vomica pneumothorax is distinguished by the absence of the bell sound, a duller quality of resonance, even where the conditions of the cavity are most favourable for confusion of diagnosis, and the absence of displacement of the heart; or, at any rate, if the heart be displaced, it is towards the affected side, and is due to contrac-
tion of the lung. The side of chest affected would also be rather retracted than distended. Metallic tinkling and 'amphoric breath-sounds may, of course, both be obtained in cases of cavity; and, indeed, even the succession sound, if the cavity be large and contain a quantity of liquid.

From pyopneumothorax subphrenicus—the name given by Leyden (10) to a condition in which an abscess cavity receiving air through a fistulous perforation from an air-containing viscus (most commonly perforating ulcer of the stomach) is found below the diaphragm, the principal guide to diagnosis is to be found in the history of the case.

Lastly, resonance and breath-sounds somewhat resembling those of pneumothorax are occasionally found at the apex of the lung in cases of pleural effusion; and sometimes over part of a lung consolidated by pneumonia. The site of the physical signs here, and a careful estimation of the condition generally, will probably prevent any mistake in such cases.

A few other rare conditions, such as hernia of a part of the stomach, or colon, through the diaphragm, have simulated pneumothorax: such an accident is usually the result of injury, and it can generally be distinguished without much trouble.

Should there be any difficulty in deciding on the causation of a case of pneumothorax, the withdrawal of a few drops of fluid from the pleural cavity, if such be present, and its examination for tubercle bacilli may be of material help. This was done in a case recently under my own care, and it furnished positive results. Careful attention to the physical signs and symptoms of the case will probably enable the physician to arrive at the correct conclusion as to the nature of the perforation in the lung, which is important from the point of view of treatment.

Prognosis.—The prospects in a case of pneumothorax depend chiefly on its cause. In the simple and traumatic class of cases the opening soon becomes sealed by inflammatory exudation and the air is absorbed. In all other varieties prognosis must be guided practically by the underlying disease. The tuberculous cases, which, as we have seen, form a large majority, end for the most part unfavourably, and that at no distant date. The shock and intensity of the early symptoms may even cut life short in a few hours. At the same time, much depends upon the condition of the opposite lung, as well as upon the presence of adhesions limiting the extent of the pneumothorax in that which has become perforated. Although it may seem paradoxical to say so, patients who, before the occurrence of the pneumothorax, had been in comparatively sound condition are, so far as pneumothorax is concerned, in greater danger than those whose affected lung has been much crippled by disease; and this chiefly due to the fact that in the latter case the system has gradually adapted itself so far to its changed conditions as to tolerate an amount of interference with normal function which would excite much greater disturbance if it fell upon the patient with all its force suddenly. The same thing is seen in cases of ordinary pleuritic effusion. If this occurs very slowly, the physical signs may indicate that one side is practically full of fluid, and no respiratory distress, apart from exertion, may be
complained of; while a second case in which half the quantity of fluid is present may be characterised by great dyspnoea if the accumulation have been rapid.

Both clinical and pathological experience go to show that even in tuberculous cases of pneumothorax rare cures have taken place; but in the great majority the outlook is a very dismal and discouraging one.

Treatment.—In most cases this can only be palliative and symptomatic. So far as drugs are concerned, opiates and stimulants comprise practically all the medicines likely to be useful. Morphine, either by the mouth or subcutaneously, is perhaps the best of the former class; alcohol in some form of the latter, but its effects may be helped by ether and ammonia. The opiate acts beneficially by relieving pain, checking the cough, and diminishing the discomfort of the patient generally, especially that resulting from the dyspnoea; and the stimulants are called for both to counteract the collapse first occurring, and to help the heart to carry on its work in which it is handicapped both by the alteration in its position and the obstruction of the circulation through the compressed lung tissue. Some external applications are useful. Dry cupping may be recommended if the dyspnoea and cyanosis be great; and where pain, resulting from the accompanying pleurisy, is much complained of, the application of two or three leeches and hot fomentations are likely to give relief. Subsequently strapping the side may be thought of.

Sooner or later the question of paracentesis will, in most cases, have to be considered. If there be evidence that the pressure within the thorax is considerable, we have practically no choice; especially if, owing to the valvular character of the perforation, this pressure be increasing. A fine trocar should be used, but no aspiration. The danger, of course, is that the diminution of the intra-thoracic pressure may encourage the reopening of the perforation which may have been closed by lymph, a condition on which our hopes for a cure of the pneumothorax depend; but it is better to run this risk than to allow the patient to die from asphyxia and exhaustion. If the opening should not have closed, the passage of the trocar will at least do no harm, and it will enable the presence or absence of intra-pleural pressure to be demonstrated. After puncture, strapping of the affected side, in order so far to prevent the recurrence of distension, may be employed in some cases with advantage. The only danger which attends puncture is that subcutaneous emphysema, partial or general, may spread from the seat of it; but this rarely happens, and all risk may be practically abolished by keeping up a little pressure on the wound after the puncturing instrument has been withdrawn.

In any case when there is evidence that the pleural cavity is partly occupied with liquid, it is wise to explore from time to time to ascertain the nature of the liquid. If serous, the general condition of the patient will be no worse than if air alone were present; probably indeed better, as the pressure exerted on the lung may tend to check the progress of disease in it, and will promote the effectual sealing up of the perforation. If the liquid be fetid pus, nothing but harm can come from letting it
remain in the pleura, and it ought to be freely evacuated at once. But there is an intermediate class of cases in which the fluid is purulent, not fetid; and it is more difficult to decide what should be done here, and when. In such a case, if the pneumothorax have resulted from the rupture of an empyema into the lung, the chest should be freely opened and drained; and the same would hold good if the empyema had ruptured through the chest wall, the opening which nature makes not being, as a rule, sufficient for free drainage. And even in the case of pyopneumothorax of tuberculous origin, a consideration of general principles dictates the free evacuation of the pus, the case being thus converted into an empyema with some chance of the perforation in the lung being closed, followed by slight re-expansion of lung and obliteration of the pleural cavity. It is true that tuberculous patients in whom this is done rarely recover; this, however, is not because of the removal of the pus, but of the progress of the disease which produced it. On general grounds it is something of an opprobrium to allow a patient to die with a large quantity of pus in his chest.

The diet should be light and nutritious, and the bowels must not be allowed to become constipated. The treatment does not differ otherwise from that of phthisis pure and simple.

The question of prophylaxis is a more difficult one, and has reference, of course, almost solely to tuberculous cases. In them, as has been pointed out by Dr. Henry Thompson (14), there may be a warning of coming danger. His view is that a hint of impending perforation may be found in a persistent and prolonged decubitus on one side, on account of pain and cough when lying on the other side is attempted; and that such a condition suggests the presence of cavities underneath a part of the pleura unprotected by adhesions; for with adhesions there would be no such severe and continuous pain. Under such circumstances strapping of the side is more than ever advisable; medicines should be administered to keep down the cough, which in these cases is apt to be frequent and exhausting, as well as superfluous: this form of cough, says the author, "is imminently dangerous from the strain it puts upon the damaged lungs, and upon their frail investing membranes." Every physician must have seen cases which correspond exactly to his description.

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REFERENCES

PNEUMOTHORAX


D. W. F.

N.B.—Dr. Frederick T. Roberts's article on "Diseases of the Mediastinum and Thymus Gland" has been carried forward to the end of the Diseases of the Chest, in the sixth volume, where fuller justice can be done to the subject.
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